Nucleus Accumbens-Centered Neurotransmitter Imbalances in Instagram Reels Consumption: Linking Reward Dysregulation to Memory and Anxiety Pathology

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Abstract

The proliferation of ultra-short-form video content platforms, particularly Instagram Reels, has introduced novel patterns of digital consumption characterized by rapid content switching and algorithm-driven variable reward schedules. This comprehensive review examines the neurochemical mechanisms underlying compulsive Reels consumption, focusing on nucleus accumbens-mediated reward dysregulation and subsequent disruptions in glutamate-GABA-serotonin homeostasis. We synthesize emerging evidence linking these neurotransmitter alterations to memory recognition impairments and anxiety manifestations through disrupted hippocampal-striatal-prefrontal circuits. The pathophysiology involves excessive dopaminergic signaling triggering compensatory glutamatergic overexcitation and GABAergic inhibitory failure, while serotonergic depletion exacerbates anxiety and cognitive deficits. Pharmacological interventions targeting these systems—including glutamate modulators (memantine, riluzole), GABAergic enhancers (pregabalin, baclofen), selective serotonin reuptake inhibitors, and dopamine stabilizers (N-acetylcysteine)—demonstrate therapeutic potential. Novel strategies involving mGluR5 negative allosteric modulators and GABA-A subunit-selective compounds warrant investigation. This integrative framework addresses a critical gap in understanding digital behavior-induced cognitive-emotional dysfunction and proposes evidence-based therapeutic approaches for this emerging public health concern.

Keywords: Instagram Reels, nucleus accumbens, glutamate, GABA, serotonin, memory recognition, anxiety, behavioral addiction, neurotransmitter dysregulation, digital dopamine.

1. Introduction

The digital media landscape has undergone radical transformation with the emergence of ultra-short-form video platforms, including Instagram Reels, TikTok, and YouTube Shorts **(1, 2).** These platforms deliver algorithmically curated content in 15-90 second segments, creating engagement patterns fundamentally distinct from traditional social media consumption. Recent epidemiological studies indicate that young adults spend an average of 52 minutes daily consuming short-form video content, with approximately 16-20% exhibiting patterns consistent with problematic use (**3, 4).** This behavioral phenomenon raises critical questions regarding neurobiological consequences, particularly concerning memory function and emotional regulation. The neurochemical foundations of behavioral addictions have been extensively characterized in contexts such as gambling disorder, gaming addiction, and compulsive buying **(5, 6, 7).** However, the specific neurotransmitter dynamics associated with ultra-short-form video consumption remain poorly understood. The rapid content switching inherent to Reels viewing—where users swipe to new content every few seconds—creates unique neurobiological challenges distinct from sustained attention paradigms **(8).** Emerging evidence suggests that compulsive Reels consumption induces cascading neurotransmitter alterations centered on nucleus accumbens dysregulation, with downstream effects on glutamatergic, GABAergic, and serotonergic systems **(9, 10).** Memory recognition deficits and anxiety symptoms represent two prominent clinical presentations associated with excessive digital media consumption **(11, 12).** Memory recognition—the capacity to identify previously encountered information—depends critically on hippocampal-prefrontal cortex circuits modulated by dopamine and glutamate signaling **(13, 14).** Anxiety disorders involve complex interactions between serotonergic pathways, GABAergic inhibition, and stress-responsive neurocircuitry **(15, 16).** The convergence of these systems in the nucleus accumbens, a key node in reward and motivation networks, provides a mechanistic framework for understanding how digital behavior patterns influence both cognitive and emotional domains (**17, 18**). This review addresses three primary objectives:

I. To characterize the pathophysiological mechanisms linking compulsive Reels consumption to neurotransmitter dysregulation,

II. To examine relationships between these neurochemical changes and memory recognition impairments with anxiety manifestations

III. To evaluate pharmacological strategies targeting these systems. By integrating findings from behavioural neuroscience, clinical psychiatry, and psychopharmacology, we propose an evidence-based model for understanding and treating digital behaviour-associated cognitive-emotional dysfunction.

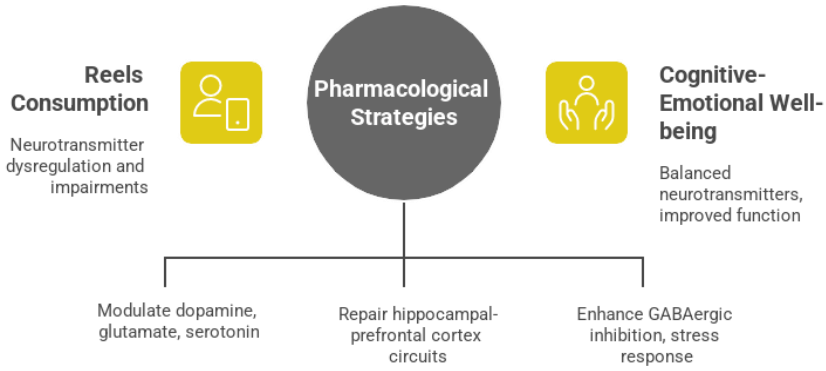


Fig.1. Addressing Cognitive-Emotional Dysfunction from Short-Length Video like Instagram Reels

2. Neurotransmitter Systems and Digital Reward Processing

2.1 Nucleus Accumbens Sensitization and Dopaminergic Dysregulation

The nucleus accumbens (NAc), a central component of the ventral striatum, serves as the primary integration site for reward-related information processing **(19).** Dopaminergic projections from the ventral tegmental area (VTA) to the NAc encode reward prediction errors—the discrepancy between expected and received rewards—forming the neural substrate for reinforcement learning **(20, 21).** Algorithm-driven content delivery on Instagram Reels creates variable ratio reinforcement schedules, where rewards (engaging content) arrive unpredictably, maximizing dopamine release and behavioral persistence **(22, 23).** Neuroimaging studies examining social media use demonstrate NAc activation patterns comparable to those observed in substance use disorders **(24, 25).** Functional magnetic resonance imaging (fMRI) investigations reveal that receiving social media notifications activates the NAc with intensity correlating to behavioral measures of problematic use **(26).** The continuous novelty inherent to Reels viewing maintains elevated dopaminergic tone, as each swipe potentially delivers highly rewarding content **(27).** This sustained activation leads to dopamine receptor desensitization, requiring progressively greater stimulation to achieve equivalent reward responses—a hallmark of addiction-like processes **(28, 29).** The temporal dynamics of Reels consumption further amplify dopaminergic dysregulation. Unlike traditional media consumption with defined start and end points, the infinite scroll design eliminates natural stopping cues, maintaining users in a prolonged state of reward anticipation **(30).** Electrophysiological studies in rodent models demonstrate that unpredictable reward timing produces sustained dopaminergic firing, whereas predictable rewards induce phasic responses that rapidly return to baseline **(31, 32).** The algorithmic unpredictability of engaging Reels content thus creates optimal conditions for dopaminergic pathway sensitization, potentially leading to nucleus accumbens hypersensitivity over time **(33).**

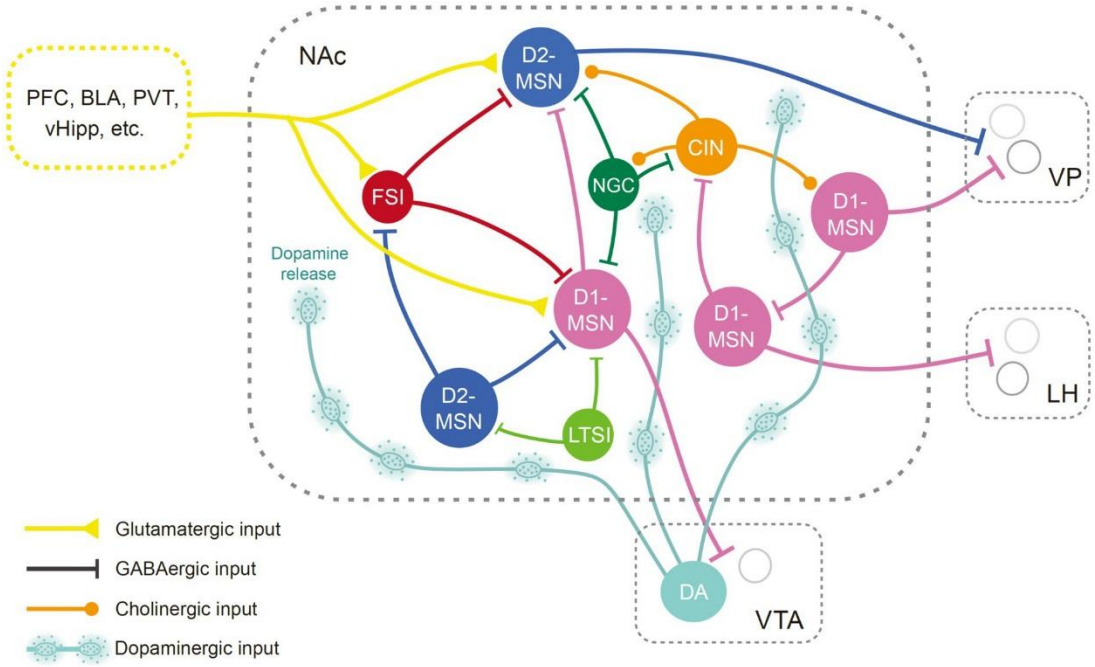


Fig.2. **Anatomical diagram of NAc neuron types**.

Illustration of different NAc neuron types and their basic connectivity pattern. Neurons in the NAc receive glutamatergic inputs from the cortex, the hippocampus, the amygdala, and thalamic regions, as well as dopaminergic input from the midbrain. The MSNs, the principal neurons in the NAc, are innervated by neighboring GABAergic interneurons and cholinergic neurons. The NAc mainly targets the VP, LH, and VTA regions. BLA, basolateral amygdala; CIN, cholinergic interneuron; D1-MSN, D1 type dopamine receptor-expressing medium spiny neuron; D2-MSN, D2 type dopamine receptor-expressing medium spiny neuron; DA, dopaminergic neurons; FSI, fast-spiking interneurons; LH, lateral hypothalamus; LTSI, low-threshold-spiking interneuron; NGC, neurogliaform cells; NAc, nucleus accumbens; PFC, prefrontal cortex; PVT, paraventricular thalamus; vHipp, ventral hippocampus; VP, ventral pallidum; VTA, ventral tegmental area.

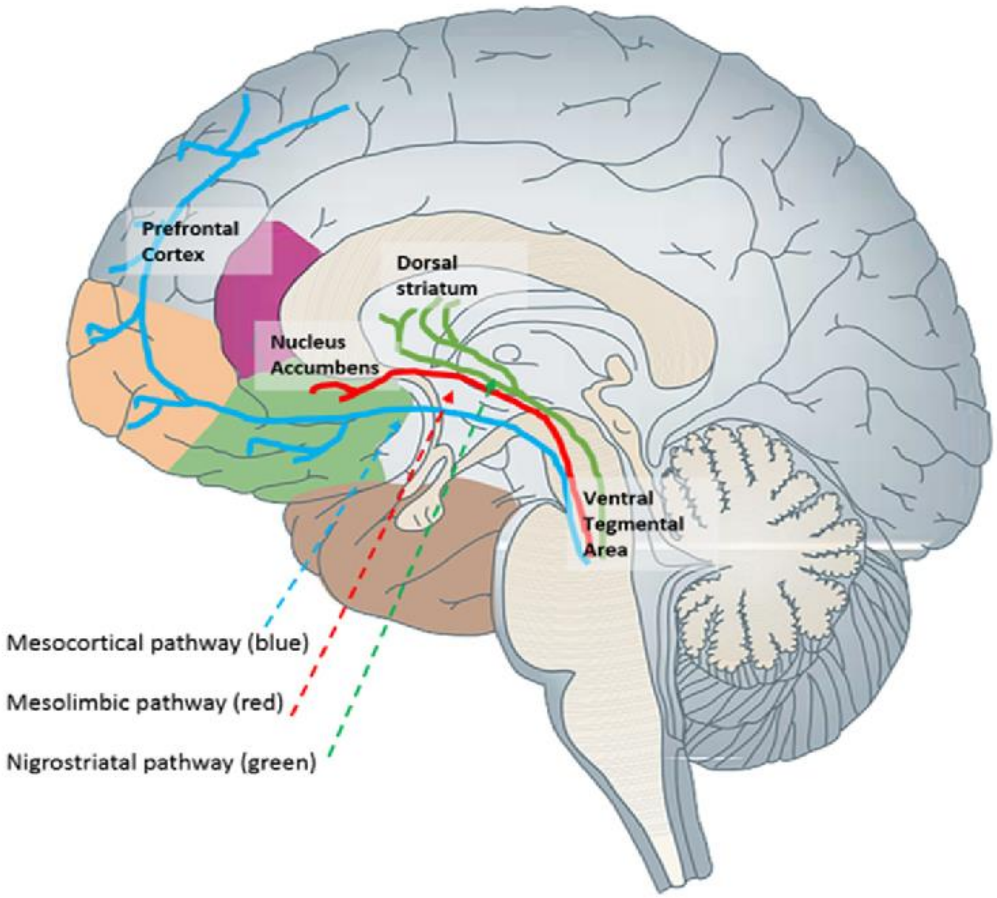


Fig.3. **Main dopaminergic pathways.**
The brain reward system is primarily associated with the Mesolimbic (red) and mesocortical (blue) pathways

2.2 Glutamatergic Overexcitation and Synaptic Plasticity Disruption

Glutamate, the principal excitatory neurotransmitter in the mammalian central nervous system, plays crucial roles in learning, memory consolidation, and reward processing **(34).** The NAc receives dense glutamatergic projections from prefrontal cortex, hippocampus, and amygdala, enabling integration of cognitive, mnemonic, and emotional information **(35, 36).** Dopamine and glutamate interact synergistically in the NAc, where dopamine D1 receptor activation enhances N-methyl-D-aspartate (NMDA) receptor-mediated glutamatergic transmission, facilitating synaptic plasticity underlying reward learning **(37).** Compulsive digital behaviors induce aberrant glutamatergic signaling through multiple mechanisms. Rapid attention switching between Reels activates prefrontal-striatal glutamatergic pathways repeatedly within short timeframes, potentially leading to excitotoxicity when cellular buffering mechanisms become overwhelmed **(38, 39).** Studies of behavioral addictions demonstrate elevated glutamate concentrations in NAc and anterior cingulate cortex, correlating with craving intensity and compulsivity measures **(40, 41).** Chronic glutamatergic hyperactivity triggers homeostatic downregulation of NMDA receptors, compromising long-term potentiation (LTP) mechanisms essential for memory consolidation **(42, 43).** The role of metabotropic glutamate receptor 5 (mGluR5) deserves particular attention in digital behavior contexts. mGluR5 in the NAc modulates both dopamine receptor signaling and synaptic plasticity, with excessive

activation contributing to compulsive reward-seeking behaviors **(44).** Preclinical studies demonstrate that mGluR5 antagonism reduces compulsive behaviors across multiple addiction models, suggesting this receptor as a potential therapeutic target for problematic digital use **(45).** Furthermore, the rapid content transitions characteristic of Reels viewing may induce NMDA receptor desensitization through repeated activation cycles, impairing the stable synaptic changes required for effective memory encoding **(46).**

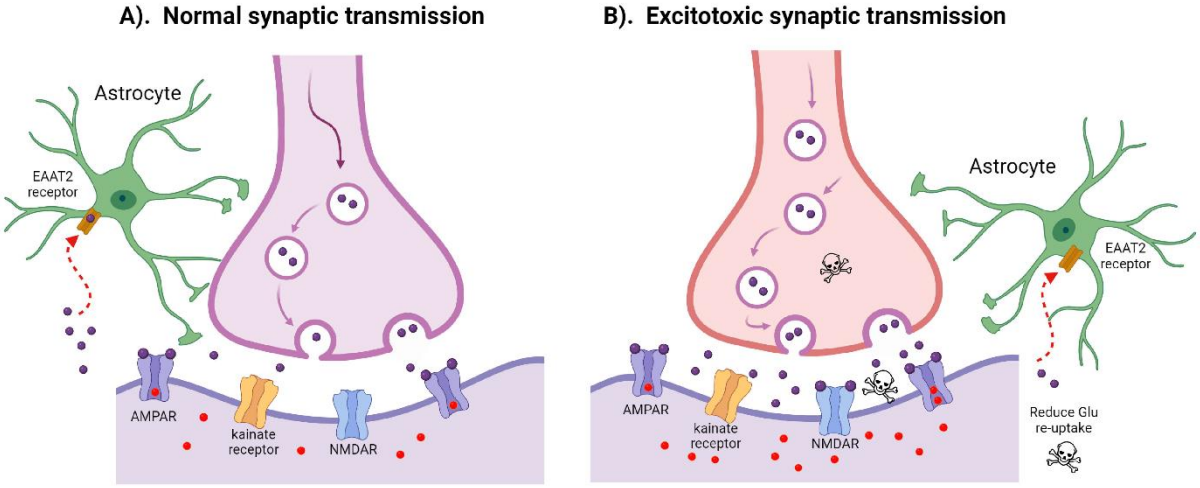


Figure 4. **Physiologic versus excitotoxic synaptic glutamate transmission and re-uptake.**

(**A**) In typical synaptic transmission, physiological levels of glutamate (purple circles) are trafficked within vesicles to the synapse. Synaptic glutamate activates three ionotropic glutamate receptors expressed on the postsynaptic neuron: N-methyl-D-aspartate receptor (NMDAR), α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAR), and kainate receptors. Regulated activation of calcium-permeable vs. calcium-impermeable AMPARs by glutamate facilitates the entry of calcium ions (red dots) into the postsynaptic cell. Balanced re-uptake of glutamate from the synapse is predominantly undertaken by EAAT2 receptors on the astrocytes (red arrow). (**B**) Prominent mechanisms underlying excitotoxic transmission include excessive glutamate release at the synapse, inefficient glutamate re-uptake via astrocytic EAAT2 receptors and increased calcium permeability of AMPARs.

2.3 GABAergic Inhibitory Failure and Executive Dysfunction

Gamma-aminobutyric acid (GABA), the principal inhibitory neurotransmitter, provides essential counterbalance to glutamatergic excitation and maintains neural network stability (47). GABAergic interneurons in the prefrontal cortex regulate executive functions including impulse control, decision-making, and attentional allocation—processes frequently compromised in behavioral addictions **(48, 49).** The NAc contains distinct populations of GABAergic medium spiny neurons that gate reward-related behaviors, with D1 receptor-expressing neurons promoting approach behaviors and D2 receptor-expressing neurons mediating avoidance responses **(50).** Chronic reward system activation disrupts GABAergic function through compensatory mechanisms. As glutamatergic excitation increases with repeated Reels exposure, GABAergic systems attempt to restore homeostasis by

downregulating GABA-A receptor expression or altering receptor subunit composition **(51).** This adaptation, while initially protective, ultimately reduces inhibitory capacity, manifesting as impaired impulse control and difficulty disengaging from rewarding stimuli **(52).** Magnetic resonance spectroscopy studies in individuals with internet gaming disorder reveal reduced GABA concentrations in prefrontal regions, correlating with symptom severity and executive dysfunction **(53, 54).** Prefrontal GABAergic interneurons, particularly parvalbumin-positive fast-spiking cells, regulate working memory and attentional control through gamma oscillation generation **(55).** Disruption of these interneurons impairs the ability to maintain goal-relevant information while filtering distractions—precisely the cognitive profile observed in excessive digital media users **(56).** The rapid context switching required for Reels consumption may overwhelm prefrontal inhibitory circuits, leading to lasting alterations in GABAergic tone that compromise executive control even during offline activities **(57).** This GABAergic dysfunction contributes to the loss of inhibitory control over intrusive thoughts and anxiety-related rumination commonly reported in problematic users **(58).**

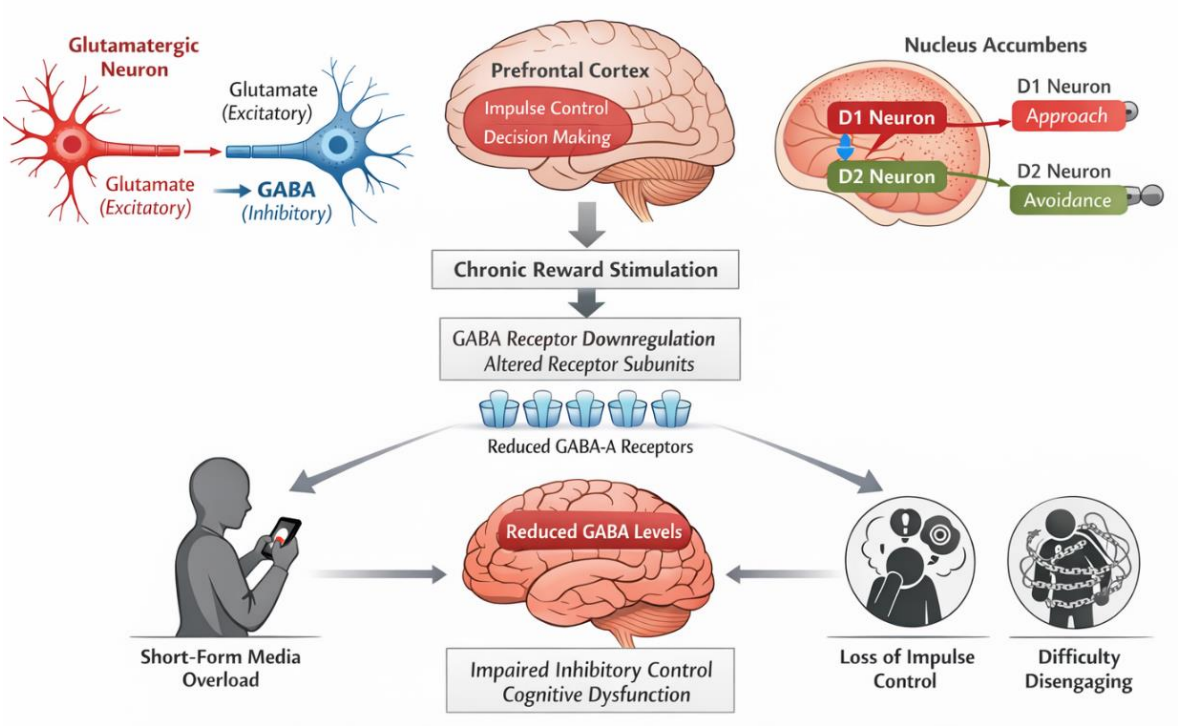


Figure 5. **GABAergic Dysfunction Cycle in Addiction**

2.4 Serotonergic Dysregulation and Mood-Cognition Interactions

The serotonergic system, originating primarily from dorsal and median raphe nuclei, extensively modulates mood, anxiety, impulsivity, and cognitive flexibility **(59).** Serotonin (5-HT) interacts with dopaminergic reward circuits, with 5-HT2C receptors on VTA dopamine neurons providing inhibitory control over dopamine release in the NAc **(60).** Dysregulation of this serotonergic brake mechanism contributes to compulsive reward-seeking behaviors observed in various addiction contexts **(61, 62).** Multiple lines of evidence implicate serotonergic dysfunction in problematic digital media use. Individuals with internet addiction show reduced serotonin transporter availability in multiple brain regions, similar to patterns observed in major depressive disorder **(63).** Polymorphisms in the serotonin transporter gene (5-HTTLPR) associate with vulnerability to behavioral addictions, suggesting genetic predisposition mediated through serotonergic pathways **(64).** Furthermore, chronic stress from social comparison and fear of missing out (FOMO) associated with social media use may deplete serotonergic resources, creating vulnerability to both mood and cognitive symptoms **(65).** Serotonin's role in anxiety and memory provides additional mechanistic links to clinical presentations. 5-HT projections to the hippocampus modulate memory consolidation, with both excessive and insufficient serotonergic activity impairing optimal memory formation (**66**). The 5-HT1A receptor, highly expressed in hippocampus, regulates anxiety-like behaviors and contextual memory processing, with dysregulation potentially contributing to the anxiety-memory comorbidity frequently observed in problematic digital users **(67).** The interplay between 5-HT2A receptors and glutamate signaling in cortical regions further influences cognitive function, with altered receptor expression patterns reported in behavioral addiction populations **(68).**

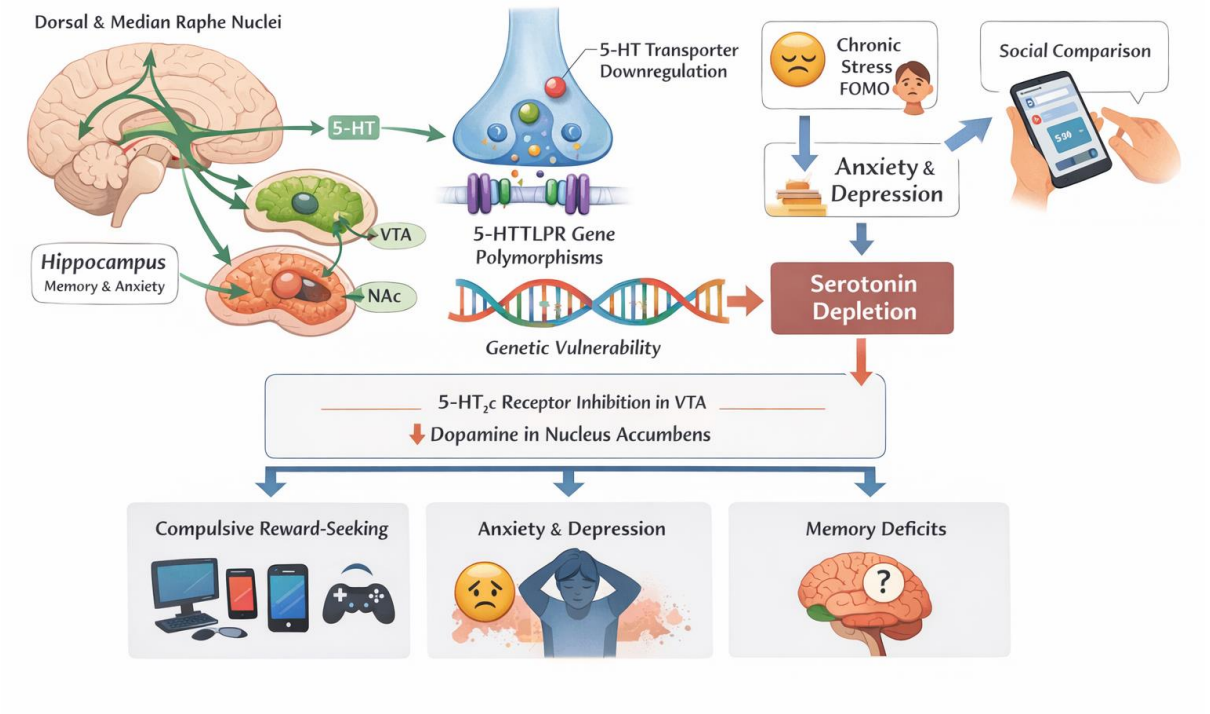
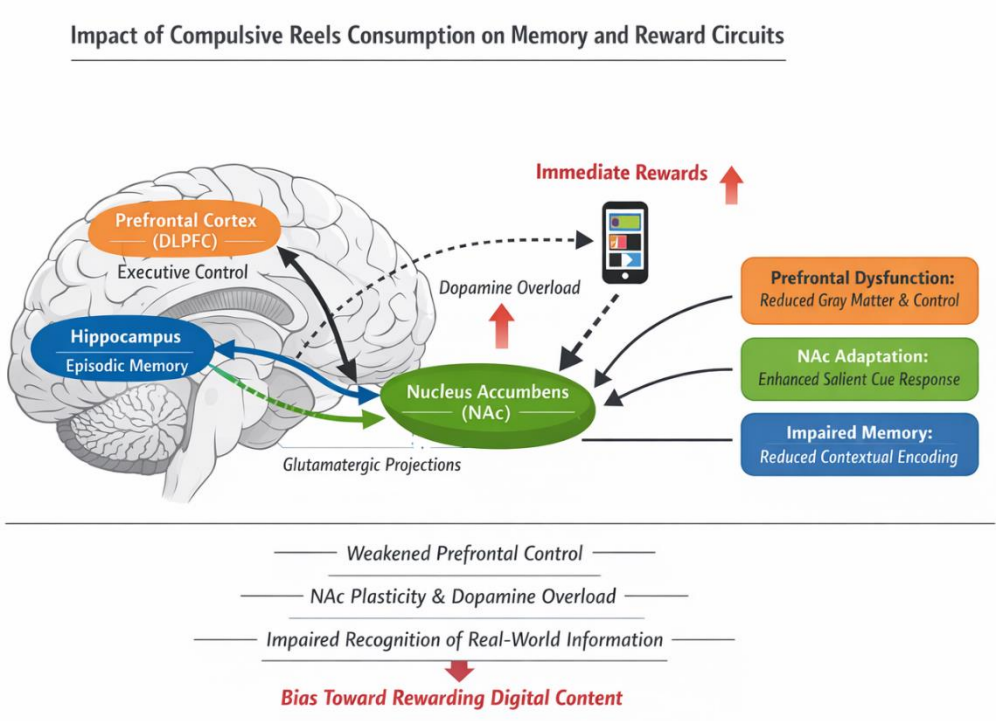


Figure 6. **Serotonergic Activity Spectrum from Inhibition to Excitation**

3. Pathophysiological Mechanisms of Memory Recognition Impairment

3.1 Hippocampal-Nucleus Accumbens-Prefrontal Cortex Circuit Disruption

Memory recognition depends on coordinated activity across distributed brain networks, with the hippocampus encoding episodic details, the prefrontal cortex maintaining contextual



frameworks, and the striatum modulating motivational salience of information **(69, 70).** The hippocampus-NAc pathway represents a critical interface where mnemonic and reward information converge, enabling organisms to remember which behaviors led to positive outcomes **(71**). Excessive dopaminergic signaling in the NAc, as induced by compulsive Reels consumption, disrupts this circuit by biasing processing toward immediate reward-related stimuli at the expense of neutral information consolidation **(72).** Ventral hippocampal glutamatergic projections to the NAc undergo experience-dependent plasticity that normally supports adaptive reward learning **(73**). However, chronic overstimulation of this pathway—through repeated engagement with highly salient digital content—may induce maladaptive plasticity characterized by strengthened responses to reward-predictive cues and weakened encoding of contextually rich but less immediately rewarding information **(74).** This altered circuit function manifests behaviorally as enhanced memory for social media content but impaired recognition of non-digital information, a phenomenon increasingly reported in clinical settings **(75).** The prefrontal cortex, particularly the dorsolateral prefrontal cortex (DLPFC), provides top-down control over both reward and memory processes **(76).** Excessive digital media use associates with reduced gray matter volume and functional connectivity in prefrontal regions, potentially reflecting glutamate-induced excitotoxicity or metabolic stress **(77, 78).** Weakened prefrontal control allows subcortical reward circuits to dominate behavior, creating difficulty disengaging from digital content even when such use conflicts with other goals **(79).** This executive dysfunction extends to memory processes, as prefrontal regions normally support strategic encoding and retrieval that enhance recognition performance **(80).**

3.2 Context-Dependent Memory Impairment and Attention Residue

The cognitive phenomenon of "attention residue"—where attention remains partially allocated to a previous task even after switching to a new one—provides a mechanism linking rapid Reels switching to memory encoding failure **(81).** Each Reel potentially captures attention through emotional salience, humor, or novelty, leaving residual activation that interferes with subsequent information processing **(82).** When users rapidly switch between Reels, attention becomes fragmented across multiple competing representations, preventing the sustained neural activity required for effective memory consolidation **(83).** Context-dependent memory, which relies on hippocampal binding of item information with contextual details, proves particularly vulnerable to the attention-switching demands of Reels consumption **(84).** The brief duration of each Reel (15-90 seconds) may be insufficient for complete contextual encoding, resulting in isolated memory traces lacking the rich associative networks that support robust recognition **(85).** Furthermore, the algorithm-driven content delivery disrupts natural semantic clustering that typically facilitates memory organization, as successive Reels often share no thematic relationship **(86).** Recognition memory performance depends critically on

Figure 7. **Hippocampal-Nucleus Accumbens-Prefrontal Cortex Circuit Disruption**

similar content—as occurs during extended Reels sessions—may overwhelm pattern separation capacity, leading to interference between memory traces and impaired discrimination between previously encountered and novel information **(88).** This mechanism may underlie subjective reports of "digital blur," where users struggle to remember specific content despite hours of consumption **(89).**

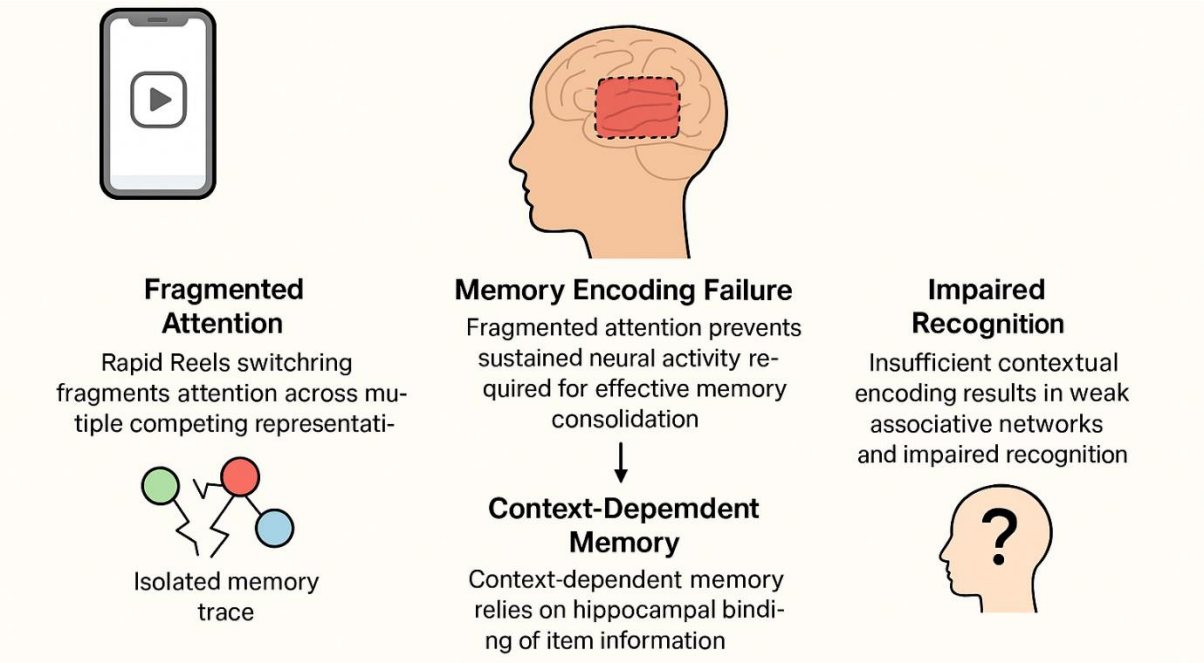


Figure 8. **Context-Dependent Memory Impairment and Attention Residue**

3.3 Working Memory Capacity Reduction

Working memory, the cognitive system responsible for temporarily maintaining and manipulating information, shows particular sensitivity to digital media multitasking **(90).** The rapid switching between Reels requires continuous updating of working memory contents, potentially exceeding capacity limits and inducing cognitive overload **(91).** Neuroimaging studies demonstrate that media multitasking associates with reduced DLPFC activation during working memory tasks, suggesting impaired recruitment of neural resources necessary for optimal performance (**92**). The relationship between working memory and long-term memory consolidation provides an additional pathway through which Reels consumption may impair recognition memory. Working memory serves as a gateway to long-term storage, with information requiring sufficient working memory processing for successful encoding **(93).** When working memory resources are depleted by continuous Reels switching, incoming information receives inadequate processing depth, resulting in weak memory traces vulnerable to rapid forgetting **(94).** This mechanism may explain why students often report difficulty remembering academic material after social media study breaks, despite subjective feelings of relaxation **(95).**

4. Clinical Phenomenology: Anxiety Manifestations

4.1 State Versus Trait Anxiety in Heavy Reels Users

Anxiety manifestations associated with compulsive Reels consumption encompass both state anxiety (temporary anxiety responses to specific situations) and trait anxiety (stable tendency to experience anxiety across situations) **(96).** State anxiety frequently emerges during Reels use itself, driven by social comparison with idealized content, exposure to anxiety-provoking news or trends, and platform-induced time pressure to keep up with rapidly changing content **(97).** Neurobiologically, this state anxiety reflects acute serotonergic and noradrenergic activation in amygdala and bed nucleus of the stria terminalis, regions mediating threat detection and sustained anxiety states **(98).** Trait anxiety develops through chronic alterations in anxiety-regulating neurocircuitry. Prolonged Reels consumption may sensitize amygdala-prefrontal circuits, lowering thresholds for anxiety responses even in offline contexts **(99).** The fear of missing out (FOMO) phenomenon represents a specific anxiety manifestation particularly relevant to social media use, characterized by persistent concern that others are having rewarding experiences from which one is absent **(100).** FOMO associates with increased dopaminergic activity in reward anticipation circuits combined with heightened amygdala reactivity to social cues, creating a state of hypervigilance for social information (**101).** Comparison with generalized anxiety disorder (GAD) pathophysiology reveals similarities in neurotransmitter dysfunction. Both conditions involve reduced GABAergic

inhibition in prefrontal cortex, impaired serotonergic regulation of anxiety circuits, and heightened glutamatergic excitation in amygdala **(102).** However, Reels-associated anxiety may show greater specificity to social and digital contexts, with anxiety symptoms intensifying during periods of forced abstinence from the platform—a pattern resembling withdrawal states in substance use disorders **(103).**

4.2 Neurobiology of Fear of Missing Out (FOMO)

FOMO represents a psychosocial phenomenon with distinct neurobiological underpinnings increasingly recognized in digital media research **(104).** The algorithmic presentation of curated highlights from others' lives activates social comparison processes mediated by dorsomedial prefrontal cortex and posterior superior temporal sulcus **(105).** Simultaneously, awareness that content is constantly being generated and potentially missed triggers anticipatory anxiety through amygdala activation and hypothalamic-pituitary-adrenal (HPA) axis engagement **(106).** Dopaminergic mechanisms in FOMO involve tonic versus phasic signaling patterns. Tonic dopamine provides a baseline motivational state, while phasic dopamine signals specific reward events (**107)**. Chronic Reels use may reduce tonic dopamine while maintaining or enhancing phasic responses, creating a state of low baseline motivation punctuated by intense reactions to salient social content (**108).** This neurochemical profile generates persistent restlessness and checking behaviors aimed at capturing the next dopamine surge from engaging content (**109).** Serotonergic dysfunction exacerbates FOMO through impaired emotional regulation and increased sensitivity to social rejection cues. 5-HT1A receptor downregulation in limbic regions reduces capacity to modulate anxiety responses, while altered 5-HT2A receptor function affects perceptual processing of social information **(110).** The combination of dopaminergic reward anticipation and serotonergic anxiety dysregulation creates a powerful motivational state driving compulsive platform checking despite negative emotional consequences **(111).**

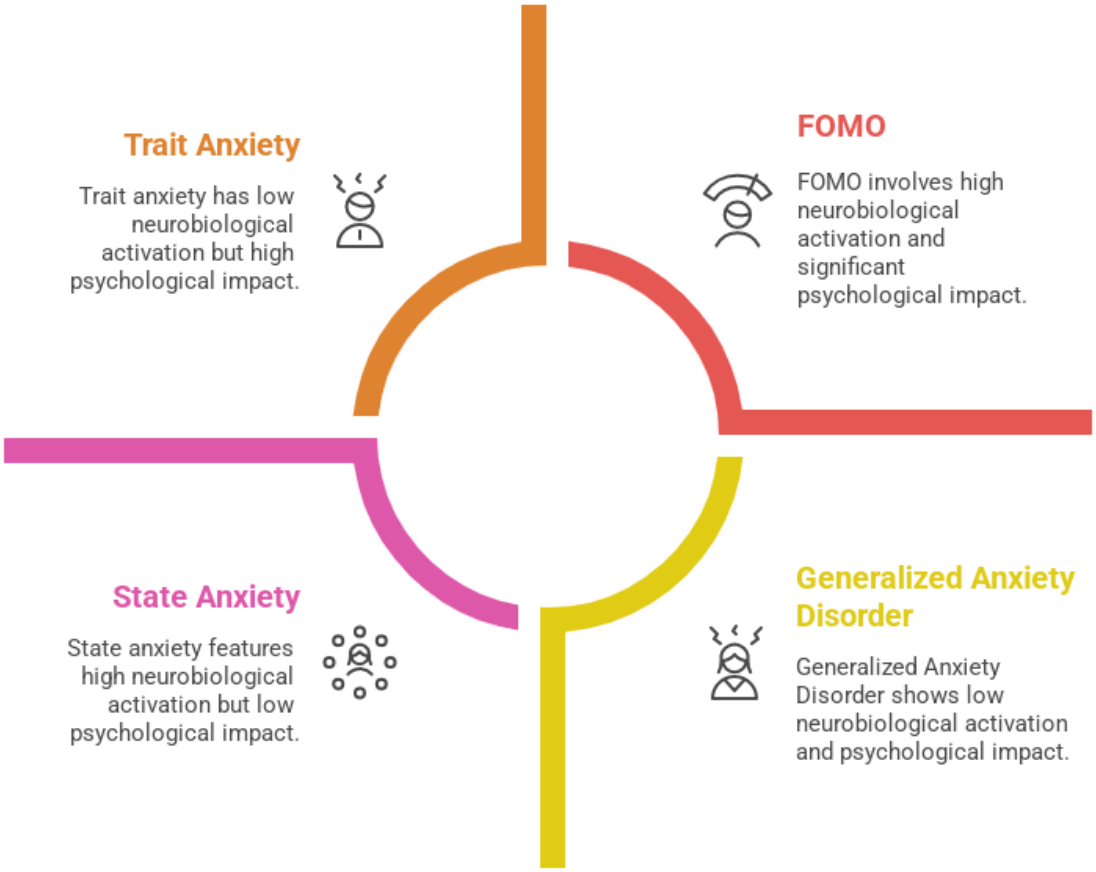


Figure 9. **Neurological and psychological aspects of Reels associated anxiety**

5. Integrated Neurotransmitter Model

Table 1 presents an integrated summary of neurotransmitter alterations associated with compulsive Instagram Reels consumption, their neuroanatomical substrates, and resulting behavioral manifestations.

Table 1. Neurotransmitter Dysregulation in Compulsive Reels Consumption

Neurotransmitter System	Primary Brain Regions	Pathophysiological Changes	Behavioral Manifestations	Key Receptor Subtypes
Dopamine	Nucleus accumbens, VTA, prefrontal cortex	Receptor desensitization, sensitized phasic responses, altered prediction errors	Compulsive checking, reduced baseline motivation, craving	D1, D2
Glutamate	NAc, hippocampus, prefrontal cortex, amygdala	Chronic overexcitation, NMDA receptor downregulation, impaired LTP	Memory encoding deficits, attention fragmentation, excitotoxicity	NMDA, mGluR5
GABA	Prefrontal cortex, NAc, amygdala	Reduced inhibitory tone, GABA-A downregulation, interneuron dysfunction	Impaired impulse control, executive dysfunction, intrusive thoughts	GABA-A (α2, α3 subunits)
Serotonin	Raphe nuclei, hippocampus, amygdala, prefrontal cortex	Transporter downregulation, receptor alterations, depletion	Anxiety, mood dysregulation, cognitive inflexibility, FOMO	5-HT1A, 5-HT2A, 5-HT2C

VTA = ventral tegmental area; **NAc** = nucleus accumbens; **LTP** = long-term potentiation; **NMDA** = N-methyl-D-aspartate; **mGluR5** = metabotropic glutamate receptor 5; **GABA =** gamma-aminobutyric acid; FOMO = fear of missing out.

Table 2 delineates the specific memory system dysfunctions resulting from neurotransmitter imbalances in compulsive Reels users.

Table 2. Memory System Dysfunction in Compulsive Reels Consumption

Memory Type	Neural Substrate	Neurotransmitter Mechanisms	Observed Impairments	Contributing Factors
Recognition Memory	Hippocampus, perirhinal cortex	Impaired glutamatergic LTP, disrupted dopamine modulation	Difficulty identifying previously encountered information	Attention residue, pattern separation failure
Working Memory	DLPFC, parietal cortex	Reduced GABAergic inhibition, compromised dopamine signaling	Decreased capacity, impaired maintenance	Cognitive overload, rapid switching
Contextual Memory	Hippocampus, prefrontal cortex	Altered glutamate-GABA balance, serotonergic dysfunction	Weak item-context binding, decontextualized memories	Insufficient encoding time, semantic disruption

Memory Type	Neural Substrate	Neurotransmitter Mechanisms	Observed Impairments	Contributing Factors
Episodic Memory	Medial temporal lobe, prefrontal cortex	Disrupted consolidation through NAc interference	Fragmented autobiographical memory, temporal confusion	Motivational bias, consolidation interference

DLPFC = dorsolateral prefrontal cortex; **LTP =** long-term potentiation; **NAc** = nucleus accumbens.

6. Pharmacological Implications and Therapeutic Strategies

6.1 Glutamate Modulators for Excitotoxicity Prevention

Glutamate modulators represent a promising therapeutic approach for addressing the excitotoxicity and synaptic dysfunction associated with compulsive digital behaviors. Memantine, an uncompetitive NMDA receptor antagonist approved for Alzheimer's disease, shows potential in reducing compulsive behaviors by normalizing glutamatergic transmission **(112).** Case reports and small trials suggest memantine may reduce internet addiction severity and associated cognitive symptoms **(113).** The drug's mechanism—preferentially blocking excessive NMDA receptor activation while preserving physiological signaling—makes it theoretically suited to counteracting the glutamatergic overexcitation induced by rapid Reels switching **(114).** Riluzole, a glutamate release inhibitor with multiple mechanisms including mGluR activation and sodium channel blockade, has demonstrated efficacy in various compulsive disorders **(115).** Preclinical studies show riluzole reduces cue-induced reinstatement of reward-seeking behaviors and normalizes prefrontal-striatal glutamate levels **(116).** Its neuroprotective properties may additionally address the excitotoxic damage potentially resulting from chronic digital overstimulation **(117).** Clinical trials investigating riluzole for behavioral addictions report improvements in compulsivity and craving, suggesting applicability to problematic social media use (**118).**

mGluR5 negative allosteric modulators represent a novel therapeutic strategy specifically targeting compulsivity mechanisms. Compounds such as mavoglurant and basimglurant modulate glutamatergic transmission without complete receptor blockade, potentially offering improved tolerability compared to direct antagonists **(119**). Animal studies demonstrate that mGluR5 negative allosteric modulators reduce compulsive reward-seeking and facilitate extinction of maladaptive behaviors **(120).** Phase II clinical trials for substance use disorders show promising efficacy and safety profiles, warranting investigation in digital behavior contexts **(121).**

6.2 GABAergic Enhancement for Anxiety and Compulsivity

GABAergic pharmacotherapy addresses both the anxiety manifestations and impulse control deficits associated with compulsive Reels consumption. Pregabalin, an α2δ ligand that enhances GABAergic transmission, demonstrates efficacy in generalized anxiety disorder and shows promise in addiction contexts **(122)**. Its anxiolytic effects combined with potential anti-craving properties make it relevant for treating the anxiety-compulsivity comorbidity common in problematic users **(123).** Clinical observations suggest pregabalin reduces both anxiety symptoms and compulsive checking behaviors in individuals with internet addiction (124).

Baclofen, a GABA-B receptor agonist, modulates dopamine release in the NAc and reduces craving across multiple addiction models **(125**). Its ability to normalize reward circuit function while providing anxiolytic effects positions it as a candidate treatment for Reels-associated pathology **(126).** Small clinical trials demonstrate baclofen efficacy in reducing alcohol and stimulant use, with mechanisms likely applicable to behavioral addictions **(127)**. However, careful monitoring is required due to potential for dependence and withdrawal with long-term use (**128**). GABA-A receptor subunit-selective compounds represent a sophisticated approach targeting specific GABAergic circuits. α2 and α3 subunit-preferring compounds provide anxiolysis without the sedation, dependence, and cognitive impairment associated with non-selective benzodiazepines (**129**). These compounds enhance GABAergic inhibition in prefrontal cortex and limbic regions while sparing α1 subunit-mediated sedative effects (**130**). Preclinical studies show α2/α3-selective compounds improve impulse control and reduce anxiety-like behaviors without tolerance development **(131)**. Clinical development of these agents may provide safer long-term options for managing digital behavior-associated anxiety and compulsivity **(132).**

6.3 Serotonergic Agents for Mood and Impulse Regulation

Selective serotonin reuptake inhibitors (SSRIs) represent first-line pharmacotherapy for both anxiety disorders and compulsive behaviors, making them relevant to problematic Reels consumption **(133).** Clinical trials demonstrate SSRI efficacy in reducing internet addiction severity, with effects mediated through improved impulse control and reduced anxiety **(134).** Escitalopram and sertraline show particular promise, with studies reporting decreased online time and improved quality of life measures (**135**). The therapeutic mechanism likely involves enhanced serotonergic inhibition of dopaminergic reward circuitry combined with normalization of prefrontal executive function **(136**). 5-HT2C receptor agonists offer targeted modulation of dopamine-serotonin interactions. Lorcaserin, though withdrawn from market for other indications, demonstrated efficacy in reducing impulsive behaviors through 5-HT2C-mediated inhibition of VTA dopamine neurons **(137).** Next-generation 5-HT2C agonists with improved selectivity profiles are under development for obesity and addiction, potentially applicable to compulsive digital behaviors (**138)**. These agents normalize reward processing without the broad serotonergic effects that may limit SSRI tolerability (**139).**

Combination approaches targeting multiple serotonergic mechanisms may provide superior outcomes. Augmentation of SSRIs with buspirone, a 5-HT1A partial agonist, enhances anxiolytic effects while potentially improving cognitive function **(140).** This combination addresses both the anxiety and memory impairment dimensions of Reels-associated pathology **(141).** Preliminary evidence suggests serotonergic augmentation strategies improve treatment response rates in behavioral addictions resistant to monotherapy **(142).**

6.4 Dopamine Stabilization and Reward Circuit Normalization

N-acetylcysteine (NAC), a glutathione precursor with multiple neurochemical actions, has emerged as a promising treatment for behavioral addictions **(143).** NAC normalizes glutamate homeostasis through cystine-glutamate exchange and modulates dopamine release in the NAc **(144).** Clinical trials demonstrate NAC efficacy in reducing compulsive behaviors across gambling disorder, trichotillomania, and internet addiction **(145).** Typical dosing ranges from 1200-2400 mg daily, with good tolerability and minimal adverse effects **(146).** The dual mechanism addressing both glutamatergic excitotoxicity and dopaminergic dysregulation makes NAC particularly suited to the pathophysiology of compulsive Reels consumption

(147). Bupropion, a norepinephrine-dopamine reuptake inhibitor, offers antidepressant effects while modulating reward circuitry function **(148).** Its dopaminergic activity may help normalize the low tonic dopamine state hypothesized in behavioral addictions, reducing anhedonia and compulsive reward-seeking **(149).** Case reports suggest bupropion may reduce internet addiction severity, though controlled trials are needed **(150).** The drug's cognitive-enhancing properties additionally address the attention and memory deficits associated with excessive digital use **(151).**

Table 3 summarizes current and emerging pharmacological strategies for treating compulsive Reels consumption and associated cognitive-emotional symptoms.

Table 3. Pharmacological Interventions for Compulsive Instagram Reels Consumption

Drug Class	Specific Agents	Primary Mechanisms	Target Symptoms	Evidence Level	Typical Dosing
Glutamate Modulators	Memantine, Riluzole	NMDA antagonism, glutamate release inhibition	Compulsivity, cognitive deficits, excitotoxicity	Case reports, small trials	Memantine: 10-20 mg/day; Riluzole: 50-100 mg/day
mGluR5 Modulators	Mavoglurant, Basimglurant	Negative allosteric modulation	Compulsive behaviors, craving	Preclinical, early clinical	Investigational dosing
GABAergic Agents	Pregabalin, Baclofen	α2δ modulation, GABA-B agonism	Anxiety, compulsivity, craving	Clinical trials, case series	Pregabalin: 150-600 mg/day; Baclofen: 30-80 mg/day
Subunit-Selective GABA	α2/α3-selective compounds	Selective GABA-A enhancement	Anxiety, impulse control deficits	Preclinical	Under development
SSRIs	Escitalopram, Sertraline	Serotonin reuptake inhibition	Anxiety, compulsivity, mood symptoms	Randomized trials	Escitalopram: 10-20 mg/day; Sertraline: 50-200 mg/day
5-HT Agonists	Buspirone, 5-HT2C agonists	5-HT1A partial agonism, 5-HT2C agonism	Anxiety, impulsivity	Clinical trials, preclinical	Buspirone: 15-60 mg/day
Dopamine Modulators	N-acetylcysteine, Bupropion	Glutamate homeostasis, dopamine-norepinephrine reuptake inhibition	Compulsivity, anhedonia, cognitive deficits	Clinical trials, case reports	NAC: 1200-2400 mg/day; Bupropion: 150-300 mg/day

NMDA = N-methyl-D-aspartate; **mGluR5** = metabotropic glutamate receptor 5; **GABA** = gamma-aminobutyric acid; **SSRI** = selective serotonin reuptake inhibitor; **5-HT** = serotonin; **NAC** = N-acetylcysteine.

6.5 Non-Pharmacological Interventions

Cognitive behavioral therapy (CBT) adapted for internet addiction addresses maladaptive cognitions and behaviors maintaining compulsive use **(152).** CBT interventions demonstrate efficacy in reducing social media use time and associated distress **(153).** Neurobiological studies suggest CBT modifies prefrontal cortex activation patterns and normalizes striatal dopamine responses to digital cues **(154).** The combination of CBT with pharmacotherapy may provide synergistic benefits through complementary neuroplasticity mechanisms **(155).** Digital detox interventions—periods of voluntary abstinence from digital platforms—show promise in

restoring neurotransmitter balance and cognitive function **(156).** Even brief detox periods (72 hours to one week) associate with improvements in working memory, attention, and anxiety symptoms **(157).** Neuroimaging studies reveal that digital detox normalizes prefrontal-striatal connectivity and reduces NAc hyperreactivity to social cues **(158).** However, relapse rates remain high without ongoing behavioral support or pharmacological maintenance **(159).** Mindfulness-based interventions demonstrate effects on GABAergic tone and anxiety regulation relevant to digital behavior pathology **(160).** Regular mindfulness practice increases prefrontal cortex GABA concentrations and enhances GABAergic inhibitory control over limbic reactivity **(161).** Mindfulness training improves attentional control and reduces compulsive behaviors through strengthened prefrontal-striatal connectivity **(162).** Eight-week mindfulness programs show comparable efficacy to pharmacotherapy for anxiety reduction, with neuroplasticity benefits persisting months after intervention completion **(163).**

7. Future Research Directions

7.1 Neuroimaging Biomarkers and Diagnostic Criteria

Development of objective neuroimaging biomarkers represents a critical priority for advancing the field. Positron emission tomography (PET) studies measuring dopamine receptor availability and serotonin transporter density could identify individuals at high risk for problematic Reels consumption **(164).** Functional MRI paradigms assessing NAc reactivity to digital cues versus natural rewards may provide diagnostic specificity **(165).** Resting-state functional connectivity analyses can characterize circuit-level disruptions in hippocampal-striatal-prefrontal networks **(166).** Magnetic resonance spectroscopy quantifying glutamate, GABA, and other metabolites offers non-invasive assessment of neurotransmitter alterations **(167).**

Standardized diagnostic criteria for problematic short-form video consumption remain absent from current nosology. Proposed criteria should incorporate:

- Time spent exceeding intended limits.
- Unsuccessful attempts to reduce use.
- Preoccupation with content between sessions.
- Use to escape negative mood states.
- Tolerance requiring increasing consumption for satisfaction.
- Withdrawal symptoms during abstinence.
- Continued use despite awareness of negative consequences and
- Functional impairment in academic, occupational, or social domains **(168).** Neurobiological validators including neurotransmitter markers would strengthen diagnostic validity and enable precision medicine approaches **(169).**

7.2 Individual Vulnerability Factors and Genetic Polymorphisms

Genetic variation contributes substantially to behavioral addiction vulnerability. Polymorphisms in dopamine-related genes—including DRD2 (dopamine D2 receptor), DAT1 (dopamine transporter), and COMT (catechol-O-methyltransferase)—associate with reward sensitivity and addiction risk **(170).** The DRD2 Taq1A polymorphism, linked to reduced

striatal D2 receptor density, predicts internet addiction severity **(171).** COMT Val158Met polymorphism affects prefrontal dopamine availability, influencing executive control capacity and vulnerability to compulsive behaviors (**172)**. Serotonergic gene variants show similar associations. The 5-HTTLPR short allele, associated with reduced serotonin transporter expression, confers increased anxiety sensitivity and behavioral addiction risk **(173).** Variations in HTR2A (5-HT2A receptor) and TPH2 (tryptophan hydroxylase 2) genes affect serotonergic function and relate to impulsivity and mood regulation **(174).** Gene-environment interactions likely determine actual phenotypic expression, with genetic vulnerabilities manifesting primarily under conditions of high digital exposure **(175)**. Understanding genetic risk profiles enables targeted prevention and personalized treatment. Individuals with high genetic loading for addiction might benefit from earlier intervention, more intensive monitoring, or specific pharmacological approaches matched to their neurochemical profile **(176).** Polygenic risk scores integrating multiple genetic variants could identify at-risk populations for preventive interventions before problematic patterns develop **(177).**

7.3 Age-Dependent Susceptibility Windows

Adolescence represents a critical developmental period for both brain maturation and digital behavior establishment (**178)**. The adolescent brain undergoes extensive remodeling of dopaminergic, glutamatergic, and GABAergic systems, with prefrontal cortex development continuing into the mid-twenties **(179**). This neurobiological immaturity creates heightened vulnerability to reward-related learning and reduced capacity for impulse control **(180).** Adolescent exposure to compulsive digital behaviors may induce lasting circuit alterations with enduring consequences for cognitive and emotional function (**181).**

Animal models demonstrate that adolescent exposure to reward-related stimuli produces more persistent neuroplastic changes than equivalent adult exposure **(182**). Adolescent rats exposed to variable reward schedules show enhanced NAc dendritic spine density and altered glutamate receptor expression persisting into adulthood **(183).** These structural changes associate with increased compulsivity and impaired behavioral flexibility in later life (**184**). Translating these findings to human development suggests that adolescent onset of problematic Reels consumption may predict particularly severe and treatment-resistant presentations (**185)**. Age-specific interventions should account for developmental stage. Adolescent-focused treatments might emphasize family involvement, school-based components, and psychoeducation about neurodevelopmental vulnerability **(186**). Pharmacological approaches require special consideration in adolescent populations, with careful risk-benefit assessment given ongoing brain maturation **(187**). Early intervention during adolescence may prevent transition to severe adult pathology through disruption of reinforcement learning during critical periods **(188).**

7.4 Longitudinal Studies and Recovery Trajectories

Limited longitudinal data constrains understanding of natural history and treatment outcomes. Prospective cohort studies following individuals from initial Reels engagement through potential problematic use and recovery would illuminate risk factors, progression patterns, and resilience mechanisms **(189).** Such studies should incorporate repeated neurotransmitter assessments, cognitive testing, and neuroimaging to characterize neurobiological changes over time **(190).** Identification of trajectory subgroups—rapid versus gradual onset, stable versus fluctuating severity, spontaneous recovery versus chronic course—would inform prognosis and treatment selection **(191).** Recovery trajectory research should examine both pharmacological and behavioral interventions. Time courses for neurotransmitter normalization following

treatment initiation remain unknown. Do dopamine receptor levels recover weeks or months after reducing Reels consumption? How quickly do glutamate-GABA balances restore? Does serotonergic function normalize with abstinence alone, or is pharmacological intervention necessary? (**192)**. Answers to these questions will guide treatment duration recommendations and relapse prevention strategies **(193).** Neuroplasticity mechanisms underlying recovery warrant investigation. Adult neurogenesis in hippocampal dentate gyrus may contribute to memory function restoration **(194).** Synaptic reorganization in prefrontal-striatal circuits could support improved executive control **(195).** Understanding which interventions—pharmacological, behavioral, or combined—optimally promote beneficial neuroplasticity will enhance treatment efficacy **(196).** Long-term follow-up assessing sustained recovery versus relapse patterns will inform maintenance treatment needs **(197).**

8. Clinical Implications and Recommendations

Table 4 presents a clinical decision algorithm for assessment and treatment of individuals presenting with concerns related to compulsive Instagram Reels consumption.

Table 4. Clinical Assessment and Treatment Algorithm

Assessment Domain	Evaluation Tools	Threshold for Intervention	First-Line Treatment	Second-Line Options
Usage Patterns	Time tracking apps, self-report diaries	>3 hours daily OR inability to reduce despite attempts	Motivational enhancement, psychoeducation	Digital detox protocols
Memory Impairment	Cognitive screening (MoCA, memory subtests)	Subjective complaints with objective deficits	CBT for compensatory strategies, possible NAC	Memantine for severe cases
Anxiety Symptoms	GAD-7, FOMO scales	GAD-7 ≥10 OR significant functional impairment	SSRI (escitalopram 10-20 mg) + CBT	Pregabalin augmentation, mindfulness
Compulsivity	Y-BOCS adapted for internet use	Moderate to severe compulsive symptoms	SSRI + NAC (1200-2400 mg)	Riluzole or mGluR5 modulators
Executive Dysfunction	Wisconsin Card Sort, Stroop test	Performance <1.5 SD below norm	Cognitive remediation, bupropion	Methylphenidate (if ADHD comorbidity)
Comorbid Conditions	Structured clinical interview	Any DSM-5 diagnosis	Treat primary disorder, address Reels use secondarily	Integrated treatment approach

MoCA = Montreal Cognitive Assessment**; GAD-7** = Generalized Anxiety Disorder 7-item scale; **FOMO** = fear of missing out; **CBT** = cognitive behavioral therapy; **NAC** = N-acetylcysteine; **SSRI** = selective serotonin reuptake inhibitor; **Y-BOCS** = Yale-Brown Obsessive Compulsive Scale; **ADHD** = attention-deficit/hyperactivity disorder; **DSM-5** = Diagnostic and Statistical Manual of Mental Disorders, 5th edition**; SD** = standard deviation.

Table 5 summarizes the integrated neurotransmitter model linking pathophysiology to clinical presentation and therapeutic targets.

Table 5. Integrated Pathophysiology-to-Treatment Model

Pathophysiological Mechanism	Neurotransmitter Systems	Clinical Manifestations	Pharmacological Targets	Non-Pharmacological Approaches	Expected Outcome Timeline
NAc dopamine dysregulation	Dopamine ↑ (phasic), ↓ (tonic)	Compulsive checking, craving, anhedonia	NAC, bupropion	CBT, contingency management	4-8 weeks
Glutamatergic excitotoxicity	Glutamate ↑, NMDA desensitization	Memory encoding failure, attention deficits	Memantine, riluzole, mGluR5 modulators	Cognitive training, reduced multitasking	6-12 weeks
GABAergic inhibitory failure	GABA ↓ in PFC	Impulse control deficits, executive dysfunction, intrusive thoughts	Pregabalin, α2/α3-selective compounds	Mindfulness, inhibitory control training	4-10 weeks
Serotonergic depletion	5-HT ↓, receptor alterations	Anxiety, mood dysregulation, FOMO	SSRIs, 5-HT2C agonists, buspirone	Exposure therapy, acceptance-based interventions	6-12 weeks
Hippocampal-PFC disconnection	Dopamine, glutamate, GABA imbalance	Recognition memory impairment, context-dependent deficits	Combined treatments targeting multiple systems	Memory strategy training, environmental modifications	8-16 weeks

NAc = nucleus accumbens; **PFC** = prefrontal cortex; **NAC** = N-acetylcysteine; **NMDA** = N-methyl-D-aspartate; **mGluR5** = metabotropic glutamate receptor 5; **GABA** = gamma-aminobutyric acid; **SSRI** = selective serotonin reuptake inhibitor; 5-HT = serotonin; FOMO = fear of missing out; **CBT** = cognitive behavioral therapy.

9. Conclusion

This comprehensive review synthesizes emerging evidence linking compulsive Instagram Reels consumption to neurotransmitter dysregulation across dopaminergic, glutamatergic, GABAergic, and serotonergic systems. The nucleus accumbens emerges as a critical integration site where algorithm-driven variable reward schedules induce dopamine receptor sensitization and altered reward processing. These dopaminergic alterations trigger cascading effects on glutamatergic excitation and GABAergic inhibition, compromising the synaptic plasticity mechanisms essential for memory consolidation. Simultaneously, serotonergic dysfunction contributes to anxiety manifestations and cognitive flexibility deficits, creating the clinical phenomenology of memory recognition impairment with anxiety comorbidity. The pathophysiological model presented here—centered on hippocampal-striatal-prefrontal circuit disruption mediated by neurotransmitter imbalances—provides mechanistic insight into an emerging public health concern. Rapid content switching inherent to Reels viewing creates unique neurobiological challenges through attention fragmentation, excitotoxicity risk, and disrupted pattern separation processes. The infinite scroll design maintains users in sustained reward anticipation states, preventing natural satiation and promoting compulsive engagement despite negative consequences.

Pharmacological interventions targeting these systems show promise across multiple domains. Glutamate modulators address excitotoxicity and compulsivity, GABAergic enhancers improve anxiety and impulse control, serotonergic agents normalize mood and reduce FOMO-

related distress, and dopamine stabilizers restore reward circuit function. Novel therapeutic strategies including mGluR5 negative allosteric modulators and GABA-A subunit-selective compounds warrant clinical investigation. Non-pharmacological approaches—particularly cognitive behavioral therapy, digital detox protocols, and mindfulness training—offer complementary neuroplasticity-promoting benefits.

Critical knowledge gaps remain, particularly regarding:

- Neuroimaging biomarkers for diagnosis and treatment monitoring.
- Genetic vulnerabilities and gene-environment interactions.
- Age-dependent susceptibilities and optimal intervention timing.
- Longitudinal recovery trajectories and relapse predictors and
- Comparative efficacy of different treatment approaches. Addressing these gaps requires coordinated research efforts integrating clinical, neurobiological, and epidemiological methodologies.

The clinical implications are substantial. Psychiatrists, neurologists, and primary care providers increasingly encounter patients presenting with cognitive-emotional complaints related to digital media overconsumption. The framework presented here enables evidence-based assessment and treatment planning targeting underlying neurotransmitter pathophysiology. Prevention strategies informed by neurobiological mechanisms—including public health campaigns, platform design modifications, and school-based psychoeducation—may reduce population-level burden.

As ultra-short-form video platforms continue proliferating globally, understanding their neurobiological consequences becomes increasingly urgent. This review establishes a foundation for mechanistic research, clinical innovation, and policy development addressing digital dopamine dysregulation and its cognitive-emotional sequelae. Future investigations should prioritize translational approaches connecting basic neuroscience discoveries to improved patient outcomes, while maintaining attention to ethical considerations surrounding digital technology, mental health, and human flourishing.

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