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REVIEW ARTICLE

Neurobiological understanding of gaming disorder: A narrative review

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Abstract

Gaming disorder (GD) is a mental disorder characterized by impaired control over gaming behaviors and the continuation of gaming despite negative consequences, resulting in functional impairments in important areas of life. Based on a growing body of scientific evidence demonstrating its neurobiological similarities to substance use disorders, GD is listed in the latest 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* and the 11th revision of the *International Classification of Diseases* as a behavioral addiction. Therefore, this review aims to understand the neurobiological underpinnings of GD by reviewing current literature on structural and functional changes in the brain. It is suggested that prolonged and excessive gaming may lead to alterations in the structure or function of the brain reward circuit and fronto-striatal circuit, affecting both reward processing and cognitive control. Changes in brain areas involved in executive function have been observed, indicating that GD is associated with reduced response inhibition and impaired decision-making. Furthermore, brain regions associated with craving exhibit heightened activity in response to gaming stimuli. This review highlights the significance of conducting further research to uncover the underlying mechanisms of GD and to develop effective interventions for its prevention and treatment.

Keywords: Internet gaming disorders; Neurobiology; Neuroimaging; Event-related potentials; Electroencephalography

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1. Introduction

Gaming is a popular leisure activity in modern society. Although most individuals engage in gaming in a healthy manner, a subset of individuals exhibit problematic behaviors. Gaming disorder (GD) is defined as a behavioral addiction, with a worldwide prevalence estimated at approximately 3%.[1,2] The *Diagnostic and Statistical Manual of Mental Disorders* 5th edition proposed diagnostic criteria for Internet gaming disorder,[3] and the World Health Organization has formally listed GD in the *International Classification of Diseases* 11th revision (ICD-11).[4] GD has been classified as "disorders due to addictive behaviors" alongside gambling disorder, which is a subcategory of "disorders due to substance use or addictive behaviors in the "mental, behavioral, or neurodevelopmental disorders" category.[4] The changes in the latest versions of the international diagnostic manual and the revisions in the classification

of diseases were guided by epidemiologic, clinical, and neurobiological studies, which reflect growing scientific evidence about the similarities of GD with substance-related and other addictive disorders.[5,6] Initially, GD was considered a subtype of Internet addiction, but a review of the scientific literature on gaming revealed that excessive gaming tends to be distinct and more likely to lead to serious consequences independently.[7-9] Furthermore, since the Internet is merely a medium for accessing potentially problematic activities, GD applies not only to excessive gaming through the Internet but also through non-Internet media.

According to the ICD-11,[4] GD is a pattern of persistent or recurrent gaming behavior ("digital gaming" or "video-gaming") characterized by impaired control over gaming, increasing priority given to gaming over other activities, to the extent that gaming takes precedence over other interests and daily activities, and continuation or escalation of gaming despite the occurrence of negative consequences. Gaming behavior can be either continuous or episodic, and it can result in marked distress or significant impairment in personal, family, educational, occupational, social, or other important areas of functioning. This pattern of behavior has been evident for at least 12 months.

The symptoms of GD, which are similar to those of substance-related and addictive disorders, include tolerance, withdrawal, cravings, repeated unsuccessful attempts to control or discontinue the behavior, and impairment in daily function.[10,11] Individuals with GD may express their tolerance to gaming by increasing gaming hours or feeling a perceived need for upgraded computer equipment and additional software. Withdrawal symptoms can manifest as anger, tension, or depression if access to gaming is denied.[12] Research has demonstrated that individuals with GD experience more intense cravings to play games.[13,14] These suggest the possibility that GD shares similar neurobiological underpinnings with substance use disorders (SUD). However, the neural mechanism underlying GD remains unclear.

Therefore, the objective of this paper is to understand the neurobiological underpinnings of GD by reviewing current literature on the structural and functional changes of the brain related to key features of addiction. To identify relevant publications, we conducted a search in PubMed for English-language sources using the following keywords and MeSH terms: "gaming disorder," "video game addiction," "Internet addiction disorder," and "neurobiology." Because the study focuses on research involving individuals with problematic Internet gaming or video gaming, this review included studies on GD, limited to papers within the realm of Internet addiction studies where the primary purpose of Internet use has been explicitly identified as gaming. In such cases, the term "GD," instead of the term "Internet addiction," is used to describe research results on Internet addiction. In addition, this review excludes studies focusing on GD with comorbid conditions. Table 1 provides brief explanations for the technical terms related to brain imaging techniques.

2. Changes of brain reward circuit

The primary reward circuit is a dopaminergic pathway that originates from the ventral tegmental area (VTA) in the midbrain and extends to the nucleus accumbens (NA), which is part of the ventral striatum (Figure 1A). Classically, addiction is a disorder of this reward circuit.[15] Communication between the VTA and NA is essential for acute drug reward.[16] Addictive substances, such as alcohol, induce and increase dopamine release in the VTA–NA circuit, resulting in positive reinforcement effects, such as pleasure.[15] Subsequently, repeated and intermittent stimulation of the reward circuit leads to neuroadaptation in the dopamine system, causing changes such as a decrease in the availability of dopamine D2 receptors (D2R) and a reduction in the expression of dopamine transporters. These adaptations are associated with rendering the reward pathway hypersensitive to addictive substances and cues associated with them, thereby manifesting characteristics

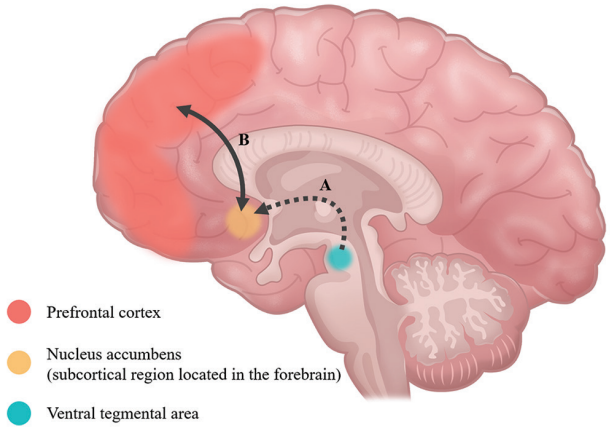


Figure 1. A schematic diagram of the primary reward circuit (A) and fronto-striatal pathway (B). (A) It is well established that primary reward circuitry, such as dopamine outputs from the ventral tegmental area (VTA) to the nucleus accumbens (NA), plays a role in the development of addiction. The addictive properties of drugs and behaviors result from their effects on the dopamine reward pathway, which leads to feelings of reward and reinforcement. The VTA is a group of neurons located close to the midline on the floor of the midbrain. The NA is a region in the basal forebrain rostral to the preoptic area of the hypothalamus. The NA and the olfactory tubercle collectively form the ventral striatum. (B) Fronto-striatal pathways are neural pathways that connect the prefrontal cortex with the striatum and play a crucial role in executive functions and reward processing. This image was created by the author.

Table 1. Brief explanations for the technical terms related to brain imaging techniques

Structural brain imaging	
Voxel-based morphometry (VBM)	Measuring gray matter volume
Diffusion-tensor imaging (DTI)	Observing the anatomical connectivity, primarily by measuring the distribution of nerve fibers in white matter *Fractional anisotropy (FA): An index of integrity and organization of white-matter tracts in DTI, with higher FA suggesting greater structural integrity of white matter
Functional brain imaging	
Functional magnetic resonance imaging (fMRI)	Identifying functionally active regions of the brain involved in various tasks and cognitive processes *Resting-state functional connectivity (rsFC): Functional connectivity between different brain regions during a state of rest in fMRI, reflecting intrinsic functional networks of the brain
Event-related potentials (ERP)	Measuring brain electrical changes recorded through electroencephalography during stimulus presentation to study cognitive process *Late positive potential (LPP): A positive electrical brain response occurring later in time in ERP, often associated with the processing of emotionally significant stimuli *P100: A positive peak in ERP occurring approximately 100 ms after the onset of a visual stimulus, often associated with early visual processing and perceptual mechanisms

of addiction such as tolerance, withdrawal, cravings, and compulsive substance use.[17]

Changes in the reward circuit accompanied by positive reinforcement were also observed in individuals with GD. Koepp *et al.*[18] conducted studies comparing dopamine release in the striatum during gaming and non-gaming periods in healthy adult males without GD. The results showed increased dopamine release in the striatum during gaming compared to non-gaming periods, with higher levels of gaming associated with greater dopamine release, particularly in the ventral striatum. Regarding D2R availability, studies on men with GD found decreased D2R availability in the striatum compared to individuals without GD. Furthermore, the D2R availability negatively correlated with the severity of addiction assessed by the Young's Internet Addiction Test (YIAT).[19,20]

Structural brain imaging studies have shown differences in NA volume between individuals with GD and controls. A study using voxel-based morphometry (VBM) demonstrated a decrease in the volume of the NA in subjects with GD compared to recreational game users (RGU) and its association with lifetime gaming usage.[21] In contrast, in a study by Yuan *et al.*,[22] increased gray matter (GM) volume of the NA was observed in subjects with GD compared to healthy controls, and the NA volume was positively correlated with YIAT scores.

A study using diffusion-tensor imaging (DTI), which may characterize microstructural white-matter organization, showed that online game playing is associated with increased fractional anisotropy (FA), regarded as an index of coherence of white-matter fiber tracts, in the reward circuity and sensory and motor control systems relative to RGU. These findings suggest that individuals with GD may have higher reward sensitivity and improved coordination between sensory and motor processing.[23] In contrast, other studies on GD have used DTI-based structural connectivity and resting-state functional connectivity (rsFC), which have shown a decrease rsFC and structural connectivity of VTA–NA in GD when compared to healthy controls.[24,25] Furthermore, rsFC of these regions was significantly negatively associated with the craving rating for Internet gaming.[24] These findings suggest a decrease in sensitivity to reward, making the individuals prone to seeking more intense rewards in GD.

3. Frontostriatal circuit dysfunction

Changes in the dopamine system also trigger neuroplasticity in the frontostriatal circuits (Figure 1B). These circuits can be divided into two pathways in addiction: the ventral valuation pathway, which is responsible for reward processing, and the dorsal control pathway, which is involved in cognitive inhibitory control.[26] As addiction progresses, the locus of control within the striatum shifts from the ventral to the dorsal region, and the influence of the prefrontal cortex (PFC) weakens.[27]

Previous studies have shown evidence of dysfunction in fronto-striatal circuits in GD, including structural and functional impairments. In a longitudinal study[28] that evaluated structural brain imaging and functional connectivity in individuals with GD, follow-up assessments were conducted after an average of 21.2 months. The results did not show any difference in changes in GM volume of both the dorsal and ventral striatum. However, subjects with GD exhibited a decrease in functional connectivity between the left dorsal putamen and the left medial PFC (mPFC), as well as an increase in functional connectivity

between the right dorsal putamen and the right middle occipital gyrus (MOG) over time, when compared to healthy controls. The mPFC is a region associated with self-regulation and is responsible for maintaining goal-directed behaviors over a long period of time for a larger reward. The MOG is a region responsible for visuospatial processing and is associated with the sensorimotor network. These findings suggest that GD is associated with a weakening of the PFC, whereas the sensorimotor circuit, which is connected to habit formation, strengthens.

In addition, previous cross-sectional studies have also reported differences in the rsFC of fronto-striatal circuits between individuals with GD and controls. Yuan *et al.*[22] demonstrated abnormal rsFC within the ventral and dorsal striatum networks. Specifically, they found a decrease in the strength of rsFC between the dorsolateral PFC (DLPFC) and caudate, as well as between the orbitofrontal cortex (OFC) and NA. Jin *et al.*[29] observed more extensive abnormal prefrontal-striatal circuits in individuals with GD, including the anterior cingulate cortex (ACC)–striatal, OFC–striatal, and DLPFC–striatal circuits. Specifically, the study found negative correlations between the rsFC of the right ACC–left putamen, left OFC–left caudate, and left DLPFC–left pallidum and YIAT scores in the GD group. A previous review on addiction proposed that unbalanced prefrontal-striatal circuits may reflect observable behaviors that characterize the addiction, such as impaired self-control and compulsive drug consumption.[30]

After evaluating the rsFC during a stimulus-response association learning task in GD, Kim and Kang[31] found that, for monetary reward, the rsFC of the ventromedial PFC (vmPFC) with the left caudate and various prefrontal regions, such as the ventrolateral PFC and dorsal ACC with the ventral striatum, was weaker in the GD group relative to controls. In addition, the vmPFC rsFC with the right NA was elevated. Dong *et al.*[32] investigated the directional functional connectivity between the ACC and the lentiform nucleus (putamen and globus pallidus as part of the basal ganglia) during a cue-craving task in individuals with GD. The study found enhanced brain responses in the lentiform nucleus, which may suggest reward sensitivity or cravings in response to specific cues. In addition, the lentiform nucleus typically exhibits greater activity in GD subjects relative to non-GD subjects when exposed to gaming-related cues. The results demonstrated that individuals with GD showed reduced ACC-to-lentiform and lentiform-to-ACC connectivity relative to RGU. Furthermore, when presented with gaming cues, GD subjects showed a trend toward decreased left-hemispheric modulatory effects in ACC-to-lentiform connectivity compared to RGU. The study also found that self-reported cue-related cravings before scanning showed a negative correlation with GD severity measured by DSM-5 scores and the left-hemispheric modulatory effect from the ACC to the lentiform.

A study was conducted to examine glucose metabolism and metabolic connectivity using positron emission tomography (PET) in individuals with GD.[33] The results showed hypometabolism in the ACC in the GD group compared to both healthy controls and those with alcohol use disorder (AUD). In addition, a negative correlation was found between glucose metabolism in the ACC and the duration of gaming use in the GD group. Significant reductions in metabolic connectivity were observed between prefrontal and limbic regions, including the striatum, in both the GD and AUD groups compared to the healthy controls. Another PET study[20] was conducted to assess D2R availability of the striatum and glucose metabolism in individuals with GD under the game task state. The study showed that the GD group had reduced D2R availability in the striatum. In addition, the level of D2R in the striatum was significantly associated with decreased glucose metabolism in the OFC, indicating dysregulation of the prefrontal-striatal circuit.

4. Impairments in executive control

Impairments in executive function, such as reduced response inhibition, increased impulsivity, and impaired decision-making, are closely associated with the pursuit of compulsive addictive behaviors. These impairments play a crucial role in the mechanism of addiction, contributing to its maintenance and relapse.[15]

Several studies have reported structural and functional changes in brain areas, including the DLPFC, OFC, ACC, and insula, suggesting a decline in executive function in the context of GD. Individuals with GD exhibited a significant decrease in GM volume or cortical thickness in the DLPFC,[21,29] ventrolateral PFC,[34] OFC,[29,35,36] ACC,[29,34] insula,[35] and the supplementary motor area[29,34,35] compared to healthy controls or RGU. The left DLPFC's GM density and the ventrolateral PFC's GM volume were associated with lifetime usage of Internet gaming,[21,34] whereas the GM volume in the OFC and insula was negatively correlated with the YIAT scores in the GD group.[35] Another study, which compared GM density among three groups – non-gamer, RGU, and GD group – indicated a marked decrease in the DLPFC in the GD group compared to the other two groups, and this density was associated with lifetime usage of Internet gaming.[21]

Regarding white matter integrity, reduced FA has been identified in the bilateral frontal lobe, corpus callosum, and external capsule in individuals with GD compared

to controls.[35] These findings suggest that Internet game overuse may damage the microstructure of both GM and white matter fibers connecting executive control.

In studies using fMRI, individuals with GD exhibited less activation in the DLPFC and ACC.[37] In addition, they showed decreased brain activation in the ACC when viewing game-related pictures during a forced break[38] compared to non-GD subjects.

A previous study investigated the differences in electroencephalography (EEG) activity between the GD group and healthy controls. The study revealed that subjects with GD exhibited lower activity in the left frontal theta, alpha, and beta bands compared to controls while gaming. Furthermore, a negative correlation was found between left frontal theta power and YIAT scores.[39] Another study compared the resting-state quantitative EEG, which reflects fundamental brain function during rest, among patients with GD or AUD and healthy controls.[40] The results showed that individuals with GD had lower absolute beta power compared to patients with AUD and normal controls.

These findings indicate that individuals with GD may have weaker executive control compared to those without GD.

4.1. Reduced efficiency in response inhibition

Response inhibition is the ability to inhibit inappropriate responses[41] and is evaluated using tasks such as the Go/No-go task, stop-signal task, and color-word Stroop test. Impulsivity is a crucial factor in response inhibition, referring to a tendency to react hastily without considering negative consequences. It is closely related to the loss of control, which is a central symptom of GD.[42]

In individuals with GD, characteristic features include a decline in response inhibition and high impulsivity. They exhibited more errors in the No-go condition of the Go/No-go task and the incongruent condition of the Stroop test when compared to the control group.[22,43-46] Furthermore, they demonstrated higher impulsivity scores, as measured by the Barratt Impulsiveness Scale-11 (BIS-11) or Eysenck Impulsivity Questionnaire.[35,46,47]

Numerous studies have reported structural and functional changes in brain regions that suggest a decline in response inhibition in individuals with GD. The main regions affected are the DLPFC, OFC, and ACC. A previous study using VBM found a negative correlation between GM volume in the ACC and impulsivity, as measured by the BIS-11.[34] Yuan *et al.*[22] found that reduced rsFC between the DLPFC and caudate was associated with more incongruent errors in the Stroop task in individuals with GD. Similarly, Ko *et al.*[48] found that individuals with GD exhibited reduced rsFC between frontal regions, including the DLPFC and OFC, and the amygdala, compared to the control group, and the disrupted rsFC was negatively correlated with impulsivity.

The OFC has been linked to impulse control and is activated during response inhibition.[49] A longitudinal study was conducted to investigate the impact of Internet gaming on brain structure. The study recruited not only excessive Internet gamers but also gaming-naïve subjects. The gaming-naïve subjects were randomly divided into two groups. One group was assigned to 6 weeks of daily gaming, whereas the other group was assigned to a non-gaming condition. Initially, the GM volume in the OFC was lower in excessive gamers than in the gaming-naïve group, and the volume further decreased during the 6-week follow-up interval. Furthermore, the longitudinal analysis revealed that the control group's mean GM volume of the OFC did not significantly change over time, whereas the GM volume in this region reduced in the 6-week daily gaming group.[50] In accordance with the longitudinal study, a cross-sectional study found that individuals with GD exhibited reduced cortical thickness in brain regions such as the OFC, insula, and lingual gyrus. The cortical thickness of the OFC was found to have an inverse correlation with the number of errors made during the incongruent condition of the color-word Stroop task in the GD group.[44] In an event-related fMRI study, the group with GD exhibited higher brain activation when processing the Go/No-go task over the left OFC and bilateral caudate compared to the control group.[47] Although the GM volume and cortical thickness decreased in the OFC of individuals with GD, the activity in the OFC and caudate increased during the Go/No-go task, indicating the need for greater cognitive effort due to reduced efficiency of cognitive control.

The decrease in cognitive control efficiency was also observed in the ACC, a brain region linked to conflict monitoring and error detection in response inhibition. Although individuals with GD demonstrated lower GM density[51] and volume,[28,29,34] as well as lower white matter density[51] compared to the control group, increased activity was observed in the ACC while performing the Stroop test in the incongruent condition[52] or No-go trials,[53] indicating a need for greater cognitive effort in response inhibition.

Error processing is a crucial aspect of response inhibition, and the ACC and insula are the primary areas involved in this process.[54] Error processing refers to the ability to detect errors and evaluate performance,[55] and impairment of this function has been previously reported in patients with SUD.[56] Similarly, an event-related potential (ERP) study found that individuals with GD exhibited significantly diminished error-related

negativity (ERN) following errors. The GD group showed lower ERN amplitudes observed in response to incorrect trials compared to correct trials during a Go/No-go task.[46] The ERN is a negative peak that occurs after incorrect behavioral responses, reflecting rapid and automatic initial error detection. It is assumed to originate in the ACC.[46] In another ERP study during a Go/No-go task, the NoGo-N2 latency at the central electrode site was delayed in the GD group compared to the healthy control. It was also positively correlated with impulsivity and YIAT scores.[57] The N2 ERP component in a Go/No-go task reflects an early stage of response inhibition and conflict monitoring. The latency of the N2 reflects the effort required to inhibit a response.[57] This suggests a reduced ability to handle performance errors in individuals with GD, similar to what is observed in patients with SUD.

4.2. Impairment of decision-making ability

Decision-making is the cognitive process of selecting the best choice among several options, taking into consideration goals, certainty, and risk. The process of decision-making involves both risk evaluation and outcome processing. Cognitive tests, such as the Iowa Gambling Task, the Cups Task, and the Probability Discounting Task, are used to evaluate the decision-making ability of individuals by assessing their choices between high-gain but risky options and low-gain but safe options.[58]

Individuals with SUD have been found to make more disadvantageous choices under both risk and ambiguity[59,60] and to exhibit greater sensitivity to reward.[61] Previous behavioral studies have shown that individuals with GD tend to select risky options more frequently and make decisions more quickly than healthy participants or RGU.[62,63] In addition, GD subjects tend to make more disadvantageous risky choices in the loss domain compared to healthy controls, but not in the gain domain, due to insensitivity to losses and levels of uncertainty.[64]

Several brain regions are associated with decision-making processes. The vmPFC, OFC, and ventral striatum are brain regions engaged in reward anticipation and outcome processing during decision-making.[65,66] The DLPFC is associated with self-control during risky decision-making,[67] and the inferior parietal lobule plays a role in decision-making under uncertainty.[68]

Functional changes were also observed in those brain regions during the Cups Task in subjects with GD.[67] The Cups Task evaluates risk decision-making in both gain and loss domains through elements of uncertainty and reward/punishment outcomes associated with different choices. In comparison to the healthy controls, the GD group exhibited less activation in the DLPFC (weaker modulation) and inferior parietal lobule (insensitivity regarding uncertainty) during risk perception for potential losses. This reduced activation was correlated with the severity of GD, as indicated by higher YIAT scores. During the process of outcomes, the GD group showed enhanced responses to experienced reward in the ventral striatum, OFC, and vmPFC for potential gains, indicating hypersensitivity to reward outcomes. In the GD group, there was a positive association between the severity of GD and increased reward-related activity in the OFC.[65] These findings support a previous study that showed individuals with GD exhibit increased activation in the OFC during gain trials and decreased activation in the ACC during loss trials in a reality-simulated guessing task compared to normal controls.[69] These results suggest decision-making deficits in individuals with GD and indicate an imbalance between the bottom-up system associated with hypersensitivity to reward and the top-down system associated with self-regulation.

5. Increased incentive salience and cravings from gaming stimuli

Cravings in patients with SUD can intensify upon exposure to stimuli associated with the substance, even without its administration.[70] This increased sensitivity to conditioned stimuli associated with substance use is linked to the expectation of obtaining the substance, memories of its positive reinforcing effects from the past, and anticipation of its rewarding effect.[70] Cravings involve not only the fronto-striatal pathway but also the hippocampus and amygdala, which are associated with memory and learning, as well as the OFC and insula, which are associated with incentive salience. Incentive salience refers to the motivational and desirable aspects of a stimulus, such as a drug or addictive behavior, that make it attractive and desirable to an individual. It enhances the perceived value or "wanting" of the addictive stimulus, contributing to the triggering of intense cravings. The DLPFC is also involved in anticipating the reward response.[71] This attractiveness is initially rooted in the "liking" condition. As addiction progresses, the transition from "liking" to "wanting" occurs, accompanied by obsessive-compulsive aspects regarding the addictive substance or behavior. This transition is characterized by cravings that dominate thoughts and behaviors, manifesting as intrusive, persistent thoughts that compel individuals to seek out the addictive substance or engage in the addictive behavior.[72,73]

In individuals with GD, exposure to game-related stimuli resulted in greater cravings compared to healthy controls or even the RGU.[32,74,75] Furthermore, individuals with GD exhibited increased activity in several brain

regions, including the striatum (including NA),[8,65] superior/medial frontal cortex,[8] OFC,[8] ACC,[8,76] DLPFC,[8,76] parahippocampal gyrus,[76] precuneus,[76] and posterior cingulate cortex,[76] when exposed to game stimuli. These activations were positively correlated with subjective gaming urges.[76] These brain regions are similar to those associated with craving in SUD.

In a study comparing cue-reactivity among individuals with GD, subjects with remitted GD for longer than 6 months, and normal controls,[76] the GD group scored higher on craving than the remission group. Furthermore, the GD group exhibited greater activation in the right DLPFC, left parahippocampus, and left middle temporal gyrus when exposed to gaming cues, in contrast to the remission group. This study suggests that these activated areas could serve as potential markers for gaming addiction. A longitudinal study was conducted on RGU who subsequently developed GD.[77] The study showed that the GD group exhibited relatively increased bilateral lentiform nucleus activation following a forced break from gaming compared with the remaining RGU group. Significant correlations were observed between the activation of the right lentiform nucleus and self-reported craving for gaming cues during periods of deprivation in the GD group. The evidence indicates that activation of the lentiform nucleus in response to cues related to deprivation may be a predictor of the transition from RGU to GD.

Incentive salience amplifies the significance of substance-related stimuli compared to other stimuli, leading to an attentional bias toward these cues. In a study comparing cue-related attentional bias between GD, obsessive-compulsive disorder (OCD), and controls,[78] individuals with GD exhibited increased late positive potential (LPP) amplitude in response to game-related pictures, indicating the enhanced salience of game-related cues. The GD group did not exhibit LPP changes in response to OCD-related cues. The LPP is a component of the ERP that arises following specific stimuli and represents motivated attention to salient stimuli. Another study[79] used ERP to observe changes in the brain during face recognition in a group of subjects with GD. Participants were presented with images of both human faces and cartoon faces resembling characters from a game. The study found that individuals with GD showed a significant increase in P100 peak amplitude when presented with cartoon faces compared to the control group. In addition, within the GD group, cartoon faces elicited a larger P100 peak amplitude than human faces. The P100 component is associated with the early automatic perception of stimuli. These results suggest that individuals with GD have an attentional bias toward stimuli related to gaming.

6. Conclusion

GD is characterized by persistent gaming behavior that leads to impairment in various life domains. Neurobiological investigations have revealed alterations in the brain reward circuit, fronto-striatal circuit, and executive control among individuals with GD. These changes parallel those observed in SUD and are associated with heightened sensitivity to gaming-related cues, increased cravings, and impaired decision-making abilities. These neurobiological findings provide insights into the treatment of GD. Pharmacological interventions that modulate the dopaminergic system, such as naltrexone or bupropion, may reduce cravings by targeting neurobiological pathways such as the reward circuit. Approaches to treatment for diminished response inhibition and impaired decision-making may include cognitive-behavioral therapy (CBT). CBT can help individuals recognize maladaptive cognitions and manage cravings. Moreover, CBT can facilitate a transition from short-term decision-making to longer-term goals or more adaptive decision-making. Research on GD is still in its early stages, and further in-depth studies across various fields are required in the future. In particular, longitudinal neurobiological studies are important, as well as studies to understand the interactions with comorbid conditions. The results of these studies will provide the fundamental knowledge needed to enhance understanding of GD and to develop effective prevention and treatment strategies.

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Conflict of interest

The author declares that she has no competing interests.

Author contributions

This is a single-authored article.

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