

The cognitive dysregulation of Internet addiction and its neurobiological correlates

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1. ABSTRACT

Individuals with Internet addiction (IA) show loss of control and recurring maladaptive Internet use. This condition has negative consequences and causes significant psychosocial distress. Here, we review neurobiological changes in four key paradigms in cognitive domain in IA including reward processing, impulsivity, cue reactivity, and decision-making. IA is associated with alterations in prefrontal-cingulate region activation during the inhibition of inappropriate responses. Such patterns are also observed in cue-reactivity paradigm tasks, suggesting a relationship with loss of control and deficits in the control of cue-eliciting behavior. Individuals with IA exhibit heightened reward prediction, devalue negative outcomes and have a higher risk-taking propensity under ambiguous

situations. In conclusion, addictive use of the Internet is associated with deficits in cognitive-emotional processing, aberrant sensitivity to rewards and Internet-related cues, poor impulse control, and impaired decision-making. There is a need to examine neural underpinnings of these aberrant behaviors and neurobiological-cognitive perspective in IA.

2. INTRODUCTION

The Internet is now used extensively in our daily lives. But over past decades, there have been rapidly growing concerns that some individuals show a loss of control over Internet use and even suffer from psychological distress and dependence

symptoms, similar to those of substance use disorder(1). However, only Internet Gaming Disorder (IGD) has been seen as synonymous with Internet use disorder (IUD) and Internet addiction (IA) in DSM-5(2). Even in recently published review articles, most examinations of Internet addiction disorder, generally and typically, focus specifically on IGD. Given that IUD can be considered a consequence of the *content* of Internet use, such as online chatting, pornography, and information searching, rather than the medium itself, the Internet activity of individuals making use of addictive phenomena should be of considerable interest(3).

Therefore, we propose the term “Internet addiction” (IA) in this paper, a term that includes Internet gaming addiction and other content having the potential to stimulate addictive Internet usage. Diagnostic criteria for IGD in DSM-5 resemble those of substance dependence, such as the existence of withdrawal symptoms and tolerance, continued use despite negative consequences, and loss of control over the activity. However, neurobiological findings related to IA have only recently begun to be accumulated. Thus, more research is needed regarding the neural correlates of IA through the use of neurocognitive, neurophysiological, and neuroimaging techniques.

Understanding IA as a model of behavioral addiction in neurobiological and cognitive terms is important, because it may reveal how addictive Internet usage can affect brain function in relation to cognitive-emotional processes. To our knowledge, there is no reported comprehensive review of the existing neurobiological studies in IA with regard to the four key paradigms of the cognitive domain in addiction: reward and punishment sensitivity, impulsivity, cue reactivity, and decision-making. In this review, we aim to integrate neurobiological considerations and characteristics of cognitive constructs of IA, based on available data from studies of IA.

3. NEUROBIOLOGICAL STUDIES IN INTERNET ADDICTION: A REVIEW

3.1. Search strategies and criteria

We sought to provide a narrative review on the neurobiological correlates of IA and to offer new insights regarding cognitive-emotional processes in IA. We performed a literature search using Pubmed (US National Library of Medicine) and Google scholar databases using the **Key Words** (“Internet addiction” “problematic Internet use” “excessive Internet use” “Internet gaming disorder” “pathological Internet use” “gaming” “gamer” “Internet addict” in combination with “impulsivity” “inhibition” “cognitive control” “reward/punishment processing” “cue reactivity” “cue-related”

“decision-making”. The final article was published in June 2016. We selected original research papers and review articles.

To be included in this review, the criteria were as follows:

- 1) Studies published from December 2005 to June 2016, peer-reviewed, English as the published language.
- 2) Studies used imaging techniques, including structural and functional magnetic resonance imaging (fMRI).
- 3) Studies used neurophysiological methods, including electroencephalography (EEG) and event-related potentials (ERPs), and neurocognitive tests (such as gambling tasks, color-word Stroop task, Go/NoGo tasks).

We also aimed to focus on the most relevant and recent findings from the perspective of alterations in cognitive-emotional processes in IA on a neurobiological basis.

Finally, 15 studies used fMRI, one used structural MRI, 11 used neurocognitive tests, and seven were electrophysiological studies. This review provides neurobiological findings with regard to neurocognition, neurophysiology, and neuroimaging in the domains of reward processing, impulsivity, cue reactivity, and decision-making.

3.2. Reward processing

Many researchers have suggested alterations in the functioning of the normal reward-related neurocircuitry, so-called reward-based learning processes, in substance addiction(4) (5). Under the past experience of reward and reinforcement, the mesocortico-limbic reward system is responsible for subsequent reactions to external stimuli(6). Development of addictive behavior, such as pathological gambling, has been suggested to be linked to behavioral conditioning due to a variable, intermittent pattern of reinforcement(7), and reduced activation of the mesolimbic dopaminergic reward system in response to monetary reward(8, 9). Indeed, there have been studies searching for differences in the function of reward- and punishment-related systems with a view to assessing neurobiological characteristics in behavioral addiction.

3.2.1. Neurocognition

Little is known about reward processing in IA through neurocognitive measures. More research is needed to explore this issue.

Table 1. Overview, characteristics and main results of neurobiological studies in Reward processing

Study	Subject	Subtype of online behavior	Task & method	Results
Neurophysiology				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results (IAD>HC)</i>
(10)	N=14 IGD ¹ N=13 casual gamers	Not specified	Reward-seeking computer game(token search)	Delayed and Enhanced N100, Reduced P200 and P300
Neuroimaging				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results(IAD>HC)</i>
(11)	N=14 IAD ² N=13 HC ³	Not specified	Monetary card guessing task (fMRI) ⁴	B: no differences Hyperactivation in gain trial; OFC ⁵ Hypoactivation in lose trial; ACC ⁶
(12)	N=16 IAD N=15 HC	Not specified	Monetary card guessing task (fMRI)	Hyperactivation after continuous win; SFG ⁷ Hypoactivation after continuous loss; PCC ⁸

Abbreviations: Internet gaming disorder,¹ Internet addiction disorder,² Healthy control,³ Functional Magnetic resonance imaging,⁴ Orbitofrontal cortex,⁵ Anterior cingulate cortex,⁶ Superior frontal gyrus,⁷ Posterior cingulate cortex⁸

3.2.2. Neurophysiology

In an electrophysiological study, Duven *et al.*(10) explored changes in reward processing in 14 pathological computer gamers and 13 casual online gamers. They used a computer game task where participants had to find tokens in a virtual situation on ERP, being time-locked to the moment when participants found a token. The results showed that pathological gamers presented delayed and enhanced N100, and reduced P200 and P300 at the moment following token discovery. Thus, the authors concluded that pathological gamers had to consume more neural resources for the initial orienting towards the reward, as evidenced by the changes in N100. Additionally, tolerance effects were observed, as shown by the reduction in the later P300 component amplitude, suggesting that pathological gamers may invest less attention in evaluating the reward.

3.2.3. Neuroimaging

Dong *et al.* (11) investigated reward and punishment processing in IA versus normal controls using functional MRI during the performance of a monetary card-guessing task designed to stimulate subjectively experienced reality-simulated loss and gain(11). The study found that Internet addicts showed hyperactivation of the orbitofrontal cortex, which is important for value-guided behavior, in gain trials, and deactivation of the anterior cingulate cortex (ACC), which is known to be a component of a circuit mediating emotional responses to pain, in loss trials, versus normal controls. They concluded that Internet addicts had different sensitivity in both win and loss situations, and had difficulty in worrying about the negative consequences of their behaviors.

To investigate reward and punishment sensitivities after repeated wins and losses, another

study by the same research team(12) designed a gambling task paradigm to simulate extreme win and loss situations in Internet addicts while participants underwent fMRI. The results showed that IA subjects had higher superior frontal gyrus activation after continuous wins compared with controls, and were not disturbed by their continued losses. Moreover, Internet addicts showed decreased posterior cingulate activation after continuous losses. Consistent with previous findings(11), these results suggested that Internet addict subjects had a preference for winning situations, while neglecting their losses, and needed less executive effort to control their negative emotions, even after continued losses.

Together, these findings provide important insights into aberrant processes in reward prediction and outcome evaluation, as well as the devaluation of loss situation, in IA. Collectively, these observations at the neurophysiological and neuroimaging levels suggest an initially heightened expectation of rewards and goal-directed behavior in IA. Previous findings of correlations between diminished neural activity of the cingulate cortices and reduced dopamine transmission in the striatal area in substance use disorder (SUD) may represent in motivational disturbances(13) Therefore, it is plausible that an imbalance in the reward system may set the stage for the development and maintenance of IA.

3.3. Impulsivity

Impulsivity, often equated with disinhibition, is recognized as a failure of top-down control mechanisms, which would normally be expected to suppress automatic responses. Inhibitory control, the ability to successfully suppress thoughts, behaviors, and inadequate stimuli, consists of several components, including impulsive choice and action(14). The potential impairment of response

Table 2. Overview, characteristics and main results of neurobiological studies in Impulsivity

Study	Subject	Subtype of online behavior	Task & method	Results
Neurocognition				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results (IAD vs. HC)</i>
(18)	N=21 IAD N=20 HC	Not specified	Stop-signal task	More direction error on go trial; lower proportion successful stop on stop trial
(20)	N=23 IA ¹ N=24 HC	Not specified	Stop-signal task	More direction error on go trial; Lower proportion successful stop on stop trial
(19)	N=50 IAD N=50 HC	Not specified	GoStop Impulsivity paradigm	Higher failure to inhibit responses
(25)	N=14 IAD N=14 HC	Not specified	Delay discounting task	Faster discounting delayed reward
Neuroimaging				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results(IAD>HC)</i>
(36)	N=12 IAD N=12 HC	Online gaming	Color-word stroop task (fMRI)	Hyperactivation; ACC, PCC During incongruent trial
(34)	N=18 IAD N=18 HC	Not specified	Color-word stroop task(SMRI) ¹	Negative correlation between cortical thickness and response errors; OFC
(37)	N=18 IAD N=18 HC	Not specified	Color-word stroop task(resting-state fMRI)	Increased ALFF ² values; mOFC ³ ; correlated with response errors
(35)	N=17 IGD N=17 HC	Online gaming	Color-word stroop task(resting-state fMRI)	Abnormal FA ⁴ (White matter fiber connecting ACC ⁵ -Rt insula); Negative correlation with response errors
(38)	N=17 IGD N=17 HC	Online gaming	Go/Nogo task (fMRI)	Hyperactivation in nogo trials; SFC ⁶ , ACC ⁵ , PCG ⁷ , Precuneus Hypoactivation in nogo trials; SPC ⁸ , MFG/IFG ⁹
(39)	N=11 IGD N=11 HC	Online gaming	Go/Nogo task (fMRI)	Hypoactivation in nogo trials; DLPFC ¹⁰ , SPC ⁸
Neurophysiology				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results(IAD>HC)</i>
(32)	N=17 PIU ¹¹ N=17 HC	Not specified	Color-word stroop task (ERP) ¹²	B: longer RT ¹³ more response errors in incongruent conditions EEG; Reduced MFN deflection in incongruent conditions
(29)	N=12 PIU N=12 HC	Not specified	GO/Nogo with letters(ERP)	B: no differences EEG; Lower Nogo N2 amplitude, Higher Nogo P3 amplitude, Longer Nogo P3 latency
(31)	N=26 PIU N=26 HC	Not specified	GO/Nogo with 8 two-digit numbers(ERP)	B: lower Nogo accuracy EEG; Reduced Nogo N2 amplitude

Abbreviations: Structural MRI¹, Amplitude of low frequency fluctuation², medial orbitofrontal cortex, ³Fractional anisotropy⁴, Anterior cingulate cortex,⁵ Superior frontal cortex,⁶ Precentral gyrus,⁷ Superior parietal cortex,⁸ Medial/Inferior frontal gyrus,⁹ Dorsolateral prefrontal cortex,¹⁰ Pathological Internet use,¹¹Event-related potential,¹² Reaction time,¹³

inhibition related to impulsivity and the concepts of self-regulation and goal-directed behavior are of major interest in IA, because this has been considered as an endophenotype in SUD and gambling disorder(15).

3.3.1. Neurocognition

At the behavioral level, individual differences in impulsivity may potentially interact with general task measures, errors, and reaction times in inhibitory performances (16).

Choi *et al.* used a stop signal task, a measurement of the ability to inhibit a prepotent response (17), considering impulsivity to be key to the neuropsychological profile required to investigate inhibitory control in IA(18). They found that IA subjects made significantly more direction errors and failures at a successful stop, and reported higher scores on the Barratt impulsiveness scale-11 (BIS-11). Similarly, a study using a GoStop impulsivity paradigm showed that IA subjects had a higher failure in inhibiting responses than did matched controls. The degree of

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failure to inhibit responses was positively correlated with BIS-11 scores(19). In another study using a neuropsychological test to explore impulsivity in IA, the results also showed significantly poorer task performance during a stop signal task, reflecting poor inhibitory control in IA (20).

Also via a delay discounting task to examine a subject's devaluation of a reward, which was expected to be delayed in time(21), the derived constant that indicates the rate at which a reward loses subjective value as a function of delay has been used as a behavioral measure of impulsivity in substance-dependent populations, including opioid, alcohol, and nicotine(22) (23, 24). Researchers found that IA subjects discounted delayed rewards at a faster rate than controls, suggesting an impulsive tendency in IA(25).

3.3.2. Neurophysiology

It has been suggested that impulsive behavior reflects a deficit in the ability to inhibit prepotent responses, as reflected in ERP(26). Previous studies have demonstrated that response inhibition is associated with distributed network activation, involving the prefrontal area and the anterior cingulate gyrus, which are involved in impulse control(27, 28). A study examined response inhibition in IA on ERPs via EEG with a Go/NoGo paradigm in which participants had to respond to Go stimuli and inhibit responses to No-Go stimuli(29). The IA subjects showed lower NoGo N2 amplitudes than controls. The NoGo N2 component is relevant to inhibitory control, by way of triggering preparation for an incorrect response that must then be withheld, and overcoming a strong habitual response(30). Also, the IA subjects had delayed NoGo P3 amplitudes, reflecting dysfunction in conflict detection in the later stage during inhibitory tasks(29). The same group also investigated deficient inhibitory control using a visual Go/NoGo task with eight two-digit numbers and compared the inhibition-related ERP components of IA subjects and controls(31). The IA subjects showed reduced N2 amplitude during the NoGo condition versus the controls.

Another study(32) focused on medial frontal negative-polarity ERP (MFN, occurring 400-500 ms after stimulus), probably generated in a medial-frontal region near the ACC, which is thought to be increased by stimuli that elicit the potential needed to adjust response control in conflicting situations(33). They found that the IA reduced the MFN amplitude, delayed the reaction, and caused more errors in incongruent conditions during a color-word Stroop task(32).

3.3.3. Neuroimaging

In a structural neuroimaging study, cortical thickness of the orbitofrontal cortex was correlated

with impaired task performance during the color-word Stroop task(34).

In another study that measured alterations in structural connections of white-matter tracts connecting the ACC-bilateral insula, the so-called salience network, which regulates dynamic communication among brain neurocognitive networks to modulate cognitive control, the researchers focused on the relationship between connections within the salience network and cognitive control deficits using the color-word Stroop task(35). They found that the IA group showed decreased fractional anisotropy (FA) values, suggesting reduced white matter integrity in the right salience network versus controls, which correlated negatively with response errors in the color-word Stroop task(35).

A task-related fMRI study using the color-word Stroop task to measure inhibition of an automatic response showed that the IA group had hyperactivation in the anterior and posterior cingulate cortices versus healthy controls following incongruent stimuli(36). Additionally, there have been studies investigating the relationship between resting-state abnormalities using fMRI and impaired cognitive control ability at the behavioral level using the color-word Stroop task. An fMRI study used the amplitude of low frequency fluctuation (ALFF) method, which reflects neuronal brain activity(37). The results demonstrated that abnormal ALFF values in the left medial orbitofrontal cortex correlated with response errors during incongruent color-word Stroop trials in the IA group(37).

In a study using fMRI and a Go/NoGo paradigm to investigate deficits in response inhibition and differential brain activation patterns in individuals with IA(38), the IA group showed increased signals in the superior/middle frontal lobule, ACC, precentral gyrus, and precuneus during NoGo trials. It was argued that hyperactivation in the superior/middle frontal gyrus, typically deactivated during active task performance, suggested reduced response-inhibition efficacy in attempts to inhibit inadequate responses. Regarding findings that the IA showed decreased signal in the superior parietal lobule and medial/inferior frontal gyrus during NoGo trials, it was suggested that altered visual and auditory functions occurred under chronic exposure to online game stimuli.

In contrast, another study using the same paradigm, while exposing participants to a gaming cue picture found that the IA group showed lower activation in the dorsolateral prefrontal cortex (DLPFC) and superior parietal cortex (SPC) in NoGo trials(39). Moreover, brain activation in these two areas was negatively associated with response inhibition performance. Given these findings, it was suggested that failure to activate

Table 3. Overview, characteristics and main results of neurobiological studies in cue reactivity

Study	Subject	Subtype of online behavior	Task & method	Results
Neurocognition				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results(IAD>HC)</i>
(48)	N=49 EIG ¹ N=19 HC	Online gaming	Modified stroop task	B: longer RT, game-related word compared to neutral word
(47)	N=12 EIG N=30 HC	Online gaming	GO/Nogo task	Higher D ² and C ³ toward game-related words
(49)	N=64 IAD N=71 HC	Online gaming	Implicit association task	B: Faster reaction; congruent condition(positive motivational implicit response)
Neurophysiology				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results(IAD>HC)</i>
(50)	N=15 EIG N=15 causal gamer	Online gaming	Cue reactivity task (ERP)	Increased LPC ⁴ at parietal for game-related pictures
Neuroimaging				
<i>Study</i>	<i>Subject</i>	<i>Subtype of online behavior</i>	<i>Task & method</i>	<i>Results(IAD>HC)</i>
(60)	N=39 IGD N=23 control	Online gaming	Cue reactivity task (FMRI)	Hyperactivation: Ventral striatum, Dorsal striatum; Positive correlation between DS ⁴ activation and IGD duration
(56)	N=10 IGD N=10 control	Online gaming	Cue reactivity task (FMRI)	Hyperactivation: OFC, DLPFC, ACC, MFC ⁵ , Caudate, NAc ⁶
(58)	N=15 IGD N=15 IGD (remitted)	Online gaming	Cue reactivity task (FMRI)	Hyperactivation: DLPFC, parahippocampus, (IGD>remitted)
(57)	N=10 IGD N=10 HC	Online gaming	Cue reactivity task (FMRI)	Hyperactivation: DLPFC, ACC, IPG, ITG ⁷ , Insular, angular gyrus, cerebellum

Abbreviations: Excessive internet gamer,¹ Target Discrimination², Response disinhibition,³ medial prefrontal cortex,⁴ Dorsal striatum, ⁴ medial prefrontal cortex,⁵ Nucleus accumbens,⁶ Inferior temporal gyrus⁷

these two regions, known to contribute to both cognitive control and attention distribution, over a gaming cue, showed distraction in the IA group(39).

In summary, when measuring response inhibition with behavioral tasks, some differences have been found between IA and control subjects in behavioral-control-related actions in the studies reviewed. The other dimension of impulsivity has been assessed by delay discounting tasks and the results showed a tendency to choose an immediate smaller reward rather than a delayed larger reward in IA subjects.

Neurophysiological studies have also suggested impairment in adjusting response control under conflicting situations in IA subjects during inhibitory tasks. In neuroimaging studies, these preliminary findings suggest dysfunction in the prefrontal cortex and cingulum.

Neuroimaging studies combining cognitive tasks on impulsivity also revealed structural and functional dysfunction in subregions of the prefrontal region (dorsolateral, orbitofrontal cortex (OFC), anterior cingulate) during inhibition of an automatic response. These findings may indicate inefficient operation of the prefrontal-cingulate network involved

in impulse control through performance monitoring and filtering inappropriate behavior in IA.

3.4. Cue reactivity

Cue reactivity, which refers to abnormal neural responses to the salience of addiction-related stimuli that often result in strong urges and craving, has been studied extensively in SUD; such studies have involved alcohol, nicotine, and cocaine(40-43) and gambling disorders(44, 45).

The most widely used method for assessing sensitivity to gaming cues in IA is to measure attentional bias towards salient versus neutral cues. Additionally, attentional bias, preferential neural processing in attentional distribution in the presence of addiction-related stimuli, and neutral stimuli have been proposed to contribute to aspects of cue reactivity (46).

3.4.1. Neurocognition

A study investigating attentional bias in IA through a word-matching Go/NoGo paradigm showed that excessive gamers had significantly faster reactions under conditions with correctly matched game-related words relative to matched neutral words(47).

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The results also showed higher target discrimination towards game-related words than common English words, reflecting cognitive bias towards game-related words in these excessive gamers(47).

Another study used a modified Stroop task, where participants were asked to name the color of presented words that were game-related, negatively balanced, and neutral words(48). Similarly, response rates to game-related words were faster than those to neutral words, demonstrating an attentional bias towards gaming cues in addicted gamers, but not in controls(48).

Likewise, Yen *et al.*(49) explored the potential effect of gaming-cue reactivity on uncontrollable Internet use by examining the positive motivational implicit response(49). During matching affectively positive or negative words under a gaming-related picture, the result showed that IA subjects reacted faster under conditions that paired positive words and gaming cues. This suggests higher positive motivational implicit responses towards online gaming cues in IA versus control subjects. Moreover, it was suggested that the lack of awareness of an automatic positive implicit process may affect addictive behavior, such as logging on to the Internet without thinking and beginning an online game(49).

3.4.2. Neurophysiology

Only one reported study has investigated ERP correlates of attentional attribution toward salient cues in excessive online gamers versus casual player controls using a cue-reactivity paradigm(50). The IA group showed an increased late positive component at a parietal site for game-related cues, indicating hyperarousal of emotional processing towards the cues. The functional significance of a late positive complex (LPC) has been suggested in previous studies to represent increased late positive potential amplitudes, reflecting a temporary increase in attention, that serves to facilitate the processing of emotionally irrelevant cues(51-53).

Some researchers have argued that conditioned incentive salience can lead to Pavlovian conditioned cues to wanted stimuli(54). From this point of view, it has been suggested that increased LPC of game-related cues in pathological gamers may reflect a motivational component or reward in IA, so-called “wanting” components, as differentiated from casual players, who may deal with gaming cues as “liking” components(50).

3.4.3. Neuroimaging

The utility of fMRI cue reactivity has been established, given previous findings on cue-induced

activation in several brain regions, including the ventral and dorsal striatum, the ACC and the prefrontal area in SUD(55). Several studies have demonstrated abnormal responses to gaming-related cues in IA compared with normal controls using fMRI.

Ko and colleagues (2009) studied the neural correlates of cue-induced gaming urges through exposing participants to gaming pictures in fMRI scans. The IA subjects had higher brain activation in the right OFC, right DLPFC, right caudate nucleus, and bilateral ACC compared to controls(56).

Similarly, another study made comparisons of gaming urge induced by gaming cue pictures in IA male adolescents(57). The results showed hyperactivation in the DLPFC, ACC, inferior frontal cortex, insula, angular gyrus, and cerebellum in IA versus controls(57). In a study to compare craving induced by gaming-related cues in IA, remitted IA subjects and normal controls, the IA group showed higher activity in the bilateral DLPFC and parahippocampal area than the remitted IA group(58).

These results are similar to the brain regions of interest that have been demonstrated to generate the craving for salient cues in SUD and pathological gambling(40, 5, 45). A recent study investigated a transition in processing of cue reactivity from the ventral to the dorsal component of the striatum using a cue-related task with fMRI(60). Higher cue-induced activations within both the ventral striatum (VS) and dorsal striatum (DS) were observed in IA. Furthermore, a positive correlation was observed between DS activation (right putamen, pallidum, and left caudate) and duration of illness within IA. Consistent with previous findings in SUD(61), it was suggested that a transition occurred from ventral to dorsal striatal processing in IA(60).

In summary, brain activations in motivational and emotional processing under craving states were similar between IA and other addictive disorders, such as gambling disorders and SUD. Together, given these findings, it seems plausible that IA subjects have a tendency to reallocate attentional resources towards motivationally salient stimulus. These findings lead to the notion that IA is characterized and maintained through a strong emotional-motivational state induced by incentive salience and lack of awareness of an automatic process when processing specific addiction-related cues.

3.5. Decision-making

Dysfunctional decision-making processing has been suggested to be a key feature of SUD and gambling disorders, characterized by a tendency to choose an immediate reward despite the expectation

of severe negative consequences(62-64). This concept includes multi-dimensional perspectives, involving, primarily, risk taking, impulsivity, cognitive inflexibility, and difficulty evaluating in the face of an immediate reward and harmful consequences(65, 66).

3.5.1. Neurocognition

Behavioral studies have been conducted with gambling tasks adapted to explore adaptive decision-making processes in IA, such as the IOWA gambling task (IGT)(67), the balloon analog task (BART)(68), and game of dice tasks(69). These tasks were created to mimic real-life decision-making under ambiguous situations between safe and risky options.

Most findings have provided evidence of impaired decision-making in IA in a range of gambling tasks. For example, Pawlikowski and Brand (2011) reported that individuals with IA made more risky choices on the game of dice task, which may have resulted from a failure to use feedback regarding the negative consequences(70). These findings are consistent with previous studies of opiate dependence and pathological gambling(71, 72).

In the IGT, participants are asked to choose four virtual decks of cards on a computer screen, which lead to losses in the long run, and would also lead to gains. The goal of the game was to win as much money as possible(73). Following this approach in a study, IA subjects selected significantly less net decks and changed selection strategy more slowly, suggesting an inability to learn from task contingencies versus the normal controls(74). Conversely, a study using BART and IGT demonstrated that there was no behavioral difference between IA and control subjects on BART, and better performance of IA subjects on IGT(75). Also, using a delay discounting task to examine the extent of the subjects' devaluation of a reward, which is expected to be delayed in time, it was found that IA subjects discounted delayed rewards faster than controls(25).

In a study adopting the cups task, IA subjects made more disadvantageous choices in the loss domain and showed more favor for the expected value of a risky option than that of the safe option(76). Results indicated greater risk-taking tendencies and insensitivity to losses in the face of harmful decisions relative to controls(76).

3.5.2. Neurophysiology

There have been few studies investigating neurophysiological correlates in decision-making processes in IA. Enlarged error-related negativity (ERN), a negative deflection generated in the ACC that peaks ~50 ms after an unintended response, has been

suggested to be an index of defensive reactivity after mistakes and, thus, to reflect decision conflict(77, 78). In an ERP study, decision-making processing in IA was studied during a Go/NoGo task with letters(79). It was found that the IA subjects demonstrated decreased ERN in NoGo trials compared with the controls. Additionally, in behavioral results, the IA subjects made more errors and showed delayed reaction time in NoGo trials.

Another ERP study used a modified Erikson flanker task and also revealed decreased ERN in IA compared with controls under incongruent conditions (80). In total, these findings demonstrate learning inability *in situations* of an impaired defensive reaction after errors in individuals with IA.

3.5.3. Neuroimaging

Imaging studies have been used in attempts to assess impaired decision-making in IA through combined neuropsychological and physiological assessments. Using probability discounting tasks while undergoing fMRI, the results revealed hypoactivation of the inferior frontal gyrus while choosing the risky option in IA. The IA group also showed faster reaction times and a preference for the fixed option(81). These findings suggest abnormal processing of risk evaluation in IA subjects. In another study using a monetary card guessing task to simulate continuous and extreme win and loss situations, IA subjects demonstrated hyperactivation in the inferior frontal cortex, insular, and anterior cingulate in continuous win trials and hypoactivation in continuous loss trials in the posterior cingulate cortex(82).

In a recent study using BART with fMRI to evaluate the modulation of the risk level – that is, the probability of balloon explosion – the results showed decreased modulation by risk level in the right DLPFC activation during the active decision-making process in IA(83). It was concluded that IA subjects had less sensitivity in the right DLPFC, a key region for decision-making, leading them to encounter more adverse situations relative to healthy controls.

In summary, most behavioral decision-making task studies have demonstrated that IA is associated with a higher risk-taking propensity under ambiguous situations compared with controls. Neuroimaging findings have provided evidence of abnormal functioning in the prefrontal cortex associated with the planning and execution of subsequent motor responses during decision-making. Moreover, as to the guessing task, which involves an emotionally guided context, the ACC and the insular seem to be engaged in decision-making in IA while modulating the activities of neural processes with the processing of emotional stimuli. These findings are consistent

with the neurophysiological findings discussed above of decreased ERN in IA, which is generated in the medial-frontal region or the nearby ACC, reflecting decision conflict while learning from previous errors. However, inconsistencies in the available findings may reflect differences between behavioral tasks, because it remains uncertain whether they all indicate the same cognitive step.

4. PERSPECTIVE

Despite recently accumulated evidence helping us to understand IA on a neurobiological basis, there remain substantial gaps in the relationship(s) between altered brain function and behavior. The findings reviewed here suggest that individuals with IA can be differentiated from normal controls or casual users in terms of dysfunction in cognitive-emotional processes, which is supported by evidence at the neurocognitive, neurophysiological, and neuroimaging levels. With regard to findings on reward sensitivity, this may be important in the formation of erroneous perceptions of reward and risk probability in IA subjects, which may explain their sustained online gaming behavior, resulting from reduction in perceived excitability towards rewards. However, as few studies have investigated directly any role for dopamine in IA, more research is needed to determine whether an altered reward processing mechanism, such as dopaminergic dysfunction, is a consequence or a vulnerability in IA.

Enhanced reactivity to Internet game-related cues in IA may also contribute to abnormal expectations of immediate rewards and subsequent impulsive reactions. Moreover, from findings using implicit cognitive tasks, even if they perceive given stimuli around them, individuals with IA may be unaware of the implicit activation of cognitive processes. Such alterations in the way of responding to appetizing cues in a given situation in IA may resemble those in SUD, because implicit cognition is supposed to be a reliable factor in SUD(84).

Regarding impulsivity, based on animal studies that explored the predictability of impulsive choices and actions towards vulnerability to addictive behavior, behavioral findings that measured response-inhibition ability as well as preferences for immediate rewards can be interpretable as impulsive behavioral aspects of IA. At the neurophysiological level, studies suggest that IA subjects have abnormal executive control-related neural reactivity, based on tasks triggering incompatible motor activation during response selection under conflict.

Neuroimaging studies combining cognitive tasks on impulsivity also showed structural and functional dysfunction in subregions of the prefrontal

region, including the DLPFC, OFC, and ACC during inhibition of an automatic response. These findings may suggest inefficient operation of the prefrontal-cingulate network involved in impulse control, and in performance monitoring and filtering inappropriate behavior in IA subjects. Additional studies are needed with varied impulsivity measurements and dimensions to further clarify the theoretical and clinical implication of these findings.

Poor decision-making serves as a core feature of certain mental health problems, including drug addiction and gambling disorders. However, the data are very limited regarding the neural substrates of poor decision-making in IA. Given that decision-making is a complex multifaceted construct, investigating how certain aspects relate to the underlying pathophysiology of IA should attract considerable attention. For example, the overevaluation of outcomes towards gaming-related stimuli can be attributed to motivational drives related to immediate reward-seeking behavior, such as stress reduction from gaming, or reducing their craving. The inability to learn from mistakes and to make rational judgments may affect the way of evaluating the pros and cons of subsequent motivated behaviors.

Moreover, conceptualizing IA in the cognitive-emotional concepts may support the establishment of individualized cognitive-behavioral therapeutic interventions, beyond the current pharmacological approaches focusing on comorbid psychiatric disturbances, such as depression and anxiety. For example, targeted interventions can focus on helping to inhibit maladaptive behaviors in those with impaired inhibition abilities and impulsivity, recognizing their attentional biases toward Internet cues and the underlying implicit cognitive processes, and understanding their irrational judgments when exposed to conflicting situations.

It remains unclear whether maladaptive Internet use is more related to the vulnerability of individuals or the negative consequences of prolonged Internet use. Further research focusing on similarities in alterations in cognitive processes in IA and SUD may point to common underlying pathological pathways and vulnerabilities to addictive behaviors. It is hoped that understanding brain-behavior relationships from this cognitive-emotional perspective may enhance our understanding of the neural underpinnings of IA.

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