Electroencephalography-detected neurophysiology of internet addiction disorder and internet gaming disorder in adolescents - A review

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ABSTRACT

Introduction: Internet Addiction Disorder (IAD) is an umbrella term for various types of Internet-based behavioural addiction, whereas Internet Gaming Disorder (IGD) addresses a specific type of IAD that is postulated to be due to a lack of control in impulse inhibition. IGD is an area of concern in the Diagnostic and Statistics Manual of Mental Disorders (DSM-5), which can be objectively assessed by dysfunctional behaviour and the increasing time of being online, particularly during the COVID-19 pandemic. Electroencephalography (EEG) identifies amplitude changes in the evoked response potential (ERP) among IGDs, correlated with underlying comorbidities.

Materials and Methods: A scoping review was performed to elaborate on the research regarding resting-state EEG and task-based EEG, particularly for Go/No-go paradigms pertaining to subjects with IAD or specifically IGD. The role of EEG was identified in its diagnostic capability to identify the salient changes that occurred in the response to reward network and the executive control network, using resting-state and task-based EEG. The implication of using EEG in monitoring the therapy for IAD and IGD was also reviewed.

Results: EEG generally revealed reduced beta waves and increased theta waves in addicts. IGD with depression demonstrated increased theta and decreased alpha waves. Whereas increased P300, a late cognitive ERP component, was frequently associated with impaired excessive allocation of attentional resources of the IAD towards addiction-specific cues. IGD had increased whole brain delta waves at baseline, which showed significant reduction post therapy.

Conclusion: EEG can identify distinct neurophysiological changes among Internet Addiction Disorder and Internet Gaming Disorder that are akin to substance abuse disorders.

KEYWORDS:
EEG, ERP amplitude, impulsivity, inhibitory control, P300, resting-state EEG

INTRODUCTION

Internet addiction disorder (IAD) can be defined as a non-chemical, behavioural addiction that involves human-machine interaction.[1] Whereas, electroencephalography (EEG) is a modality that identifies cortical electrical impulses of the brain, hence enabling the detecting of brainwave patterns during rest and thought processing.[2] IAD is comprised of generalised Internet addiction (GIA) and specific Internet addictions (SIA). SIAs are comprised of distinctive online activities such as Internet Gaming Disorder (IGD), social networking sites (SNS) addiction, cyber-pornography addiction, and online shopping addiction.[1] IGD has been proposed as a type of addiction similar to substance use disorders (SUDs).[3] Specifically, IGD can be considered a pathological behavioural addiction, provided that the gaming causes significant impairment or distress in several aspects of a person's life.[3]

The development of tolerance has been implicated in the diagnosis of IGD as it is a progressive and chronic condition. Some of the objective assessment for an individual to be diagnosed with IGD include increased time of being online and having a tendency to download faster software to run their gaming applications.[3] Clearly with the advent of mobile smartphones, problematic smartphone use and internet addiction have become more prevalent among young adults.[4] Moreover, during these challenging times of the novel coronavirus disease 2019 (COVID-19) pandemic and movement restriction orders imposed by governments to enforce social distancing, the urge to be constantly online has become stronger among the adolescents and young adults, as evidenced by a three-fold increase in online mobile gaming and 35% higher usage for multiplayer modes in a survey conducted in India.[5]

Internet Addiction Disorder

The phenomenon of IAD, also known as Problematic Internet Use (PIU), is defined as a behavioural addiction involving a psychological dependence to internet applications and sites, with resultant adverse consequences.[1] IAD is attributed to an impairment of the frontal executive control network (ECN), which leads to a diverse set of behavioural addictions that may comprise of specific applications and activities, such as online pathological gambling, online social networking addiction, cybersex addiction, e-shopping, and online information seeking.[1] The lack of inhibitory impulse control leads to a compulsive behaviour to constantly seek gratifying activities.[2] Thus, understanding the neurophysiology of the mechanisms underpinning this condition can be critical in implementing a proper treatment plan. The theory driven

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model for IAD assumes a similarity with SUDs, whereby the 'positive' reinforcement factor is the cravings for rewarding stimuli (impaired response to reward in the limbic system); and the 'negative' factor is the impulsiveness or lack of inhibitory control that leads to withdrawal and tolerance (lack of impulse control in the ECN).[2,3]

Internet Gaming Disorder

Playing videogames online provides a conducive platform for gaining mastery of skills needed to win the games, escaping from the realities of life, and achieving socializing needs while providing anonymity, all of which are vastly appealing to adolescents and young adults.[4] Nevertheless, the downside of this is the development of dependence and tolerance, particularly among adolescents and young adults. The neurobiology of IGD is deemed analogous to the pathways involved in SUDs.[3] Neuroimaging studies and epidemiological studies point to a dual processing model of digital technology addictions characterized by an imbalance between the reflective ECN, which is 'hijacked' by the over-reactive cortico-limbic reward system.[3,6-9] Moreover, by using cue-reactivity paradigms, e.g., presenting game-related stimuli, activations of the ventral striatum, amygdala and hippocampus were noted in IGD cases compared to controls.[3]

Neurophysiological evaluation using electroencephalography

There is growing recognition that the EEG activity, which is recorded under either the resting-state eyes-closed (EC) or eyes-open (EO) condition or simultaneously during the presentation of a task-based stimulus, acts as a brain-activity correlate of behaviour and cognition.[7] The high temporal resolution, mobility, non-invasive scanning, and relatively low cost of EEG make it a valuable tool to study addictive behaviours compared to neuroimaging techniques.[2] Current scalp EEG systems are typically 64-channel electrodes placed in several predetermined positions on the scalp of the subjects (Figure 1a). The interpretation of EEG waves is categorized into frequency bands that denote different degrees of brain activity. Commonly discernible frequency bands include the delta wave, δ wave (0.3–3.5 Hz), theta wave, θ-wave (4–7.5 Hz), alpha wave, α-wave (8–13 Hz), beta wave, β-wave (14–30 Hz) and gamma wave, γ-wave (>30 Hz).[2] Beta band activity are high-frequency waves that are related to exerting inhibitory control, whereby the activation of attention will cause an increase in β-wave amplitude in the frontal area and a decrease in the β-wave amplitude in the posterior regions.[2] The α-wave amplitude increases with an increase in cognition, memory and attention, and is reduced when an inhibitory control is exerted by the ECN.[2] Contrarily, an increase in θ-wave amplitude indicates cognitive dysfunction.[2]

Concerning task-based EEG, several evoked response potentials (ERPs) are implicated in the neurophysiology of addiction. The ERPs are defined by the amplitude, latency, polarity, and scalp distribution of the electrical wave recordings. A fronto-central negative wave that peaks at 200 to 350 ms after the onset of the stimulus is termed the N2 or N200 wave.[6] The N200 amplitude has a negative deflection from the baseline EEG recordings and is larger during negative feedback and perceived adverse outcomes in neuropsychological tasks.[6] Figure 1b shows an example of the N200 wave. In Go/No-go tasks, the N200 is observed predominantly in the fronto-central regions and is caused by infrequent/ 'target' stimuli in the No-go task. The N200 amplitude is noted to increase as the frequency of the eliciting stimulus decreases.[6] This is inferred to be caused by the mental need to control incorrect responses and involved in the automatic novelty sensing process.[6] Accentuated novelty sensing process has been implicated in IAD due to increased impulsiveness.[22] Whereas, the feedback related negativity (FRN) wave reflects the monitoring process associated with feedback inputs and is often elicited by negative stimuli. This negative potential of FRN is predominantly distributed over the fronto-central scalp area and occurs at a latency of 200–300 ms after the feedback (Figure 1c).

FRN is defined as the negative deflection of the EEG recording produced by the subtraction of the waveform for the positive feedback from that for the negative feedback, or purely from the negative waveform obtained from the negative feedback.[2] Studies have revealed reduced amplitude and prolonged latency of the FRN in pathological conditions related to impaired attentional states.[2] Then, there is the P300 or P3 wave, which is an ERP that corresponds to activations of the anterior cingulate cortex (ACC) is postulated to reflect premotor decisional processes, including updating of memory, providing cognitive closure, and activation of inhibitory processes over widespread cortical areas.[2] The P300 ERP is a large positive amplitude waveform, having a maximum amplitude over the parietal area with a peak latency of about 300 - 350 ms (auditory stimulus) and 350 - 450 ms (visual stimulus) (Figure 1c). The P300 is involved in the activation of cortical inhibitory processes. Its amplitude is affected by the level of difficulty of the task, motivation, significance of the stimulus, and vigilance.

The hypotheses of IAD and IGD are that they are similar to SUDs, i.e., both are influenced by (i) impaired inhibitory control exerted by the ECN/ increased impulsivity and (ii) overactive response to addiction-specific cues that are perceived as rewards.[2,3] However, little is known regarding the underpinnings of the neurophysiological changes that are involved in these disorders. EEG can provide an excellent temporal resolution in detecting abnormal brain wave patterns. Thus, this scoping review aims to identify relevant articles that have evaluated the role of resting-state and task-based EEG in determining the neurophysiological changes that occur in IAD and IGD. Subsequently, this review aims to summarize recommendations to improve this technique and to propose a conceptual framework regarding the role of EEG in evaluating the neurophysiology of IAD and IGD.

MATERIALS AND METHODS

This review adopted the framework of scoping reviews proposed by Arksey and O'Malley's, 2005.[26] The initial research questions were identified, relevant studies were sourced, studies that fulfilled the inclusion criteria were selected, the data was plotted in tables, and the report and finally a summary of the salient findings and recommendations were presented in the discussion section.

Identifying the initial research questions
This review aimed to identify critical neurophysiological changes that occur in IAD and IGD subjects using resting-state EEG and task-based EEG, respectively. We also aimed to determine trait features of IAD and IGD, while identifying potential trait features of comorbid conditions such as Attention Deficit Hyperactivity Disorder (ADHD), which is also an impulse control disorder and depressive symptoms that can co-exist with IAD or IGD.

Identifying relevant studies
A wide range of keywords and their combination was used to source the published literature in Scopus and PubMed databases. Advanced search tools and the use of Boolean operators were employed to broaden the scope to cover the specific objectives of this review. The search terms included a combination of the keywords 'Internet Addiction Disorder', 'IAD', 'Internet Gaming Disorder', 'IGD', 'electroencephalography', 'EEG', 'resting-state EEG', 'evoked response potential', and 'inhibitory control'.

The inclusion criteria were all peer-reviewed, original research articles, written in the English language, evaluating the role of EEG in determining the neurophysiological changes that occur in IAD or IGD, compared with healthy controls (HC) or any other suitable, comparable group. The exclusion criteria were all articles that were not original research articles, e.g., review papers, proceedings, technical reports, and case series. Furthermore, the exclusion criteria involved full texts that were not written in the English language, articles that did not evaluate IAD or IGD, and studies that did not utilise EEG as the primary investigation to evaluate IAD or IGD. We did not apply any time constraint for our search strategy. Review of the literature was completed over four months, ending in January 2021.

Articles selection
The initial search of the databases identified 114 articles by applying the inclusion criteria. Deduplication was done and 59 articles were removed. Fifty-five articles were evaluated based on the title and abstract, and subsequently 14 articles were removed. A review of the abstracts revealed large numbers of articles that were not original articles (n=10). The full texts of a further 41 articles were screened using the exclusion criteria. The Preferred Reporting of Items for Systematic Reviews and Meta-Analyses (PRISMA) method was utilised for the whole process of evaluating the articles. Finally, 35 studies were identified as being relevant to the research topic, as shown in the PRISMA flowchart (Figure 2). We did not evaluate the potential bias of the eligible articles as this process is not considered essential for scoping reviews.

RESULTS

Overall, our search of the databases identified 12 eligible resting-state EEG articles and 23 eligible task-based EEG articles that evaluated IAD or IGD. Of the former 12 articles, five evaluated IAD[7-8,10-12] and seven evaluated IGD.[13-19] Of the latter 23 articles that utilised task-based EEG, 16 evaluated IAD[6,20-33] and seven evaluated IGD,[9,34-39] respectively.

Resting-state Electroencephalography in IAD and IGD
The frequency bands detected from the EEG recordings can be evaluated for their power spectral density (PSD) or absolute power, representing the power distribution of the EEG series in the frequency domain. Additionally, the scalp recording sites can also be statistically correlated using coherence analysis to estimate the functional connectivity between areas of the brain cortices in the temporal domain of IGD subjects.[16] The data can be recorded using either the eyes closed (EC), or eyes open (EO) condition, whereby the EC condition permits the evaluation of a functional baseline because there are no external tasks demands that can distract the subjects' attention. Alternatively, the EO condition allows the subjects to engage passively with an external visual input without performing a specific task. Fast Fourier Transformation (FFT) is used to convert the continuous EEG data recordings into the frequency domain, and subsequently, the data from each scalp electrode will be computed for the respective frequency bands.[16]

Studies that have evaluated the resting-state EEG coherence among IAD and IGD have identified similarities with SUD regarding the regional brain connectivity abnormalities. It is postulated that the altered γ phasic synchrony is explained by the abnormal excitatory system and hyper-aroused sensory system in addicts.[10]

Decreased absolute β power is a consistent finding among subjects with IAD and IGD and is significantly correlated with the severity of the addiction and impulsivity.[13] It is also postulated that the resting-state EEG findings of IAD patients with comorbid depression are altered because of the effect of depression, whereby an increased relative θ-wave power was found in these cases.[8]

A summary of studies that evaluated resting-state EEG in IAD and IGD can be found in Table I.

Electroencephalography and event related potentials
EEG responses that are time-locked to more complex processing of a given stimulus is defined as the event-related potential (ERP).[2] As mentioned earlier, the commonly identified ERPs, namely the N200, P300 and FRN, occur after a stimulus is presented, and can provide detailed measures of the processes that occur between the stimulus input and response output.

The N100 and P200 peaks are auditory cortical responses that reflect bottom-up information such as stimulus features.[9] Frequently, the P300 wave is elicited by two types of ERP tasks, namely the oddball task and the Go/No-go task. The subject's response is typically recorded by eliciting the button-pressing response. Furthermore, the P300 is sensitive to the occurrence probability of a stimulus as well as task complexity.[9] The P300 amplitude is increased when a more salient and relevant event is observed that produces an automatic attentional response.[9] Hence, an increased P300 amplitude is considered as a biomarker of inhibitory deficit in IAD.[9] This is frequently observed in the Go/No-go task, whereby the No-go stimulus is considered as the rare one, in which the subjects are required to avoid it. Thus, the presence of an increased P300 amplitude during the No-go task is a marker for response inhibition.

Table I: Studies that assessed Internet Addiction Disorder / Internet Gaming disorder using resting-state EEG

(A) Internet Addiction Disorder

Author (Year)	Region	Type of tests to diagnose IAD/ IGD and comorbids	Participants, Sample size	Findings
1. Choi JS et al. (2013)[10]	South Korea	- IAT - BIS-11 test	• IAD (n=21) • HC (n=20)	-↓ absolute power on the β band, ↑ absolute power on the γ band among IAD - increased impulsivity and impaired inhibitory control among the IAD
2. Lee J et al. (2014)[8]	South Korea	- IAT - SCID - BDI - BAI	• IAD (N=35; IAD with MDD: n=18, IAD without MDD: n=17) • HC (N=34)	- IAD without MDD had ↓ absolute power of δ and β waves - IAD with MDD had ↑ θ but ↓ α waves - This indicates that changes in the δ and β waves can act as a neurobiological marker of IAD. Whereas ↑ absolute power of θ waves may represent trait markers of IAD with comorbid MDD because θ waves are predominantly associated with emotional memory retrieval and meditative states.
3. Kim JW et al. (2017)[11]	South Korea	- DISC-IV - CDI - K-scale	• Pure ADHD (n=22) • ADHD with depression (n=11) • ADHD with PIU (n=19)	- ADHD with PIU showed ↓ absolute θ power at the central and posterior zones compared to pure ADHD - ADHD with depressive symptoms did not show any significant changes. - This indicated that ↓ absolute θ power at fronto-parietal regions may represent trait markers for PIU.
4. Wang GY et al. (2018)[7]	New Zealand	-IAT	• Healthy subjects using the Internet for recreational purposes	- IAT score was positively correlated with α power obtained during eyes closed resting-state EEG recordings - There was a positive correlation between BDI score with α asymmetry at mid-frontal regions
5. Kamaruddin N et al. (2021)[12]	Malaysia / Indonesia	- validated questionnaire for cyber-pornography addiction - EEG pattern derived classification of learning disorders	• Not having porn addiction (n=7) • Not addicted to porn (n=7)	- ADHD was identified as those with high θ/β ratio in frontal regions - ASD was identified as those with high α/β ratio in frontal regions - Dyslexia was identified by ↑ power of θ on the left hemisphere of the brain including the frontal regions - ASD was significantly correlated with risk for cyber-pornography.

(B) Internet Gaming Disorder

Author (Year)	Region	Type of tests to diagnose IAD/IGD and comorbids	Participants, Sample size	Findings
1. Son KL et al. (2015)[13]	South Korea	-IAT	• IGD (n=34) • AUD (n=17) • HC (n=25)	- ↓ absolute β power among IGD - ↑ absolute power on the γ band in AUD - No significant correlation between severity of IGD with QEEG
2. Kim YJ et al. (2017)[14]	South Korea	- IAT at baseline and and post SSRT therapy	• IGD (n=20) • HC (n=20)	- there was ↑ absolute power of θ at central zone and whole brain δ bands at baseline, which showed significant ↓ at 6 months post SSRT - the extent of θ band reduction correlated with the change in IAT scores
3. Park JH et al. (2017)[15]	South Korea	- YIAS - K-ARS	• ADHD only (n=15) • ADHD with IGD (n=15) • HC (n=15)	- ADHD only group showed ↑ power of θ at the frontal regions - ADHD and IGD showed ↑ power of β at the temporal regions - interhemispheric coherence was ↑ in ADHD with IGD (likely due to repetitive activation of the brain reward and working memory systems during continuous gaming can result in an ↑ in neuronal connectivity within the temporal and parieto-occipital regions

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4. Park SM et al. (2017)[16]	South Korea	- DSM-5 - IAT - AUDIT	• IGD (n=30) • AUD (n=30) • HC (n=32)	- AUD showed ↑ θ band - IGD showed ↑ interhemispheric γ coherence compared to AUD and HC regardless of psychological features evaluated such as depression, anxiety, and impulsivity - Right fronto-central coherence predicted the IAT scores
5. Youh J et al. (2017)[17]	South Korea	- DSM-5 - IAT	• MDD (n=15) • MDD + IGD (n=14)	- MDD+IGD had ↓ α band coherence between the interhemispheric frontal regions - MDD+IGD had ↑ intra-hemispheric coherence for α band between parietal and occipital regions - MDD+IGD had ↑ intra-hemispheric β band coherence between fronto-temporal regions
6. Park S et al. (2018)[18]	South Korea	- DSM-5 - YIAS	• IGD (n=30 out of which 18 completed treatment) • HC (n=12)	- At baseline recording, IGD showed ↑ β and γ inter-hemispheric coherence, and ↑ δ intra-hemispheric coherence of the right hemisphere - At 6 months post SSRI interventional therapy, IGD did not demonstrate any significant EEG changes compared to baseline but continued to show ↑ β and γ inter-hemispheric coherence despite improvements in IGD symptoms (likely to indicate that ↑ β and γ inter-hemispheric coherence are neuro-physiological trait markers of IGD.
7. Hong JS et al. (2020)[19]	South Korea	- DSM-5 - Beck Depression Inventory - Beck Anxiety Inventory - K-ARS	• IGD with no comorbids, treated with CBT and PE (n=25) • IGD with no comorbids, treated with CBT only (n=25)	-↑ FAA, addiction score, and ↓ depression scores that were more marked in the CBT and PE group, which indicates greater left PFC activation during PE alleviates the mood of IGD subjects

Footnote: ↓: reduced, ↑: increased, ADHD: attention deficit hyperactivity disorder, ASD: autism spectrum disorder, AUD: alcohol use dependence, AUDIT: Alcohol Use Disorders Identification Test, BAI: Beck's Anxiety Inventory, BDI: Beck's Depression Inventory, BIS-11: Barratt's impulsiveness scale, DSM-5: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, FAA: frontal alpha asymmetry, CBT: cognitive behaviour therapy, PE: physical exercise, PFC: prefrontal cortex, K-ARS: Korean version of ADHD Rating Scale, YIAS: Young's Internet Addiction Scale, IAT: Internet Addiction Test, MDD: major depressive disorder, DISC-IV: ADHD Diagnostic Interview Schedule for Children Version IV, CDI: Children's Depression Inventory, K-scale: Korean Internet Addiction Self-scale, SSRT: selective serotonin inhibitor, SCID: Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders-IV, CPz: central-parietal zone. QEEG: quantitative EEG.

Table II: Studies that elicited evoked response potential in Internet Addiction Disorder /Internet Gaming Disorder during task-based EEG

(A) Internet Addiction Disorder

Author (Year)	Region	Type of stimulus	Participants, Sample size	Findings
1. Yu H et al. (2009)[20]	China	Auditory oddball task with simultaneous 19-channel scalp electrodes EEG monitoring	• young adults with IAD (n=10) • HC (n=10)	- PIU showed ↓ P300 amplitude and ↑ P300 latency in all electrodes. - γ oscillation occurred at 300 ms after stimuli presentation at 40–50 Hz on the CPz electrode. - PIU affects information coding and integration in the brain.
2. Dong G et al. (2010)[21]	China	Go/No-go task	• IAD (n=12) • HC (n=12)	- IAD had ↑ P300 amplitude, which indicated that more cognitive endeavours were required to complete the inhibitory task because of impaired inhibitory control - longer P300 peak latency noted in IAD, indicated less efficient information processing due to impaired inhibitory control towards No-go tasks - N200 amplitude was ↓ in IAD, likely due to poor attentional resources
3. Zhou ZH et al. (2010)[22]	China	Visual Go/No-go task	• IAD (n=26) • HC (n=26)	- BIS-11 total scores, attentional key, and motor key scores in IAD group were higher than that of the HC group. - N200 amplitude was significantly ↓ in the frontal regions during the No-go task among the IAD and correlated with impulsivity scores. - This indicates that IAD shares neuropsychological and ERPs characteristics of compulsive-impulsive spectrum disorder.
4. Ge L et al. (2011)[23]	China	Auditory oddball task	Middle-aged adults: • IAD (n=38) • HC (n=48)	- ↓P300 amplitude and longer P300 latency in IAD, was postulated to be due to attentional resource allocation of cognitive processing being of greater importance in the development of IAD in older people compared with younger people. - shorter/ reduced latency of P300 after CBT may be indicative of quicker response and improved attentional resource allocation.
5. Zhou Z et al. (2013)	China	Erikson flanker task	• IAD (n=23) • HC (n=23)	- IAD made ↑ total error rates than HC - IAD had ↓ Reactive times for total error responses - IGD had ↓ mean ERN amplitudes of total error response conditions at frontal and central electrode sites (likely to indicate deficient response monitoring function characteristics, i.e. compulsive-impulsive spectrum disorder characteristic.
6. Ling Z et al. (2015)[25]	China	Visual oddball task	• IAD (n=10) • HC (n=10)	- IAD showed ↓ P300 wave amplitude and longer latency period when tested for addiction-related stimuli (likely due to impaired memory abilities of the IAD)
7. Yau YHC et al. (2015)[26]	USA	BART task	• PIU (n=39) • HC (n=27)	- PIU showed ↓ FRN and P300 amplitudes to both negative and positive feedback (implying that impaired feedback processing can be a neural correlate of PIU).
8. Zhang F et al. (2016)[27]	China	Face vs. non-face object	• IAD (n=20) • HC (n=20)	- The N110 and the P200 ERP amplitude in response to faces were ↑ in the IAD group than HC group. - The N170 to faces ↓ in the IAD group than in the HC group.
9. Balconi M et al. (2017)[28]	Italy	Attentional inhibitory task (Go/No-go task)	- High IAT scores (n=12) -Low IAT scores (n=13)	- High-IAT young participants had specific responses to IAD-related cues (videos representing online gambling and videogames) in terms of cognitive performance (↓ Response Times, and Error Rates) with ↓ FRN and increased P300.
10. Jiao C et al. (2017)[29]	China	Visual stimuli (person's hands/ forearms/feet in painful or non-painful situations)	• IAD (n=16) • HC (n=16)	- Painful pictures elicited ↑ N200 and P300 amplitudes than the non-painful pictures only in the HC group but not in the IAD group. - Both early automatic and of the later cognitive processes of pain empathy may be impaired in IADs. - Psychophysical evidence of empathy deficits in association with IAD.

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11. Lai C et al. (2017)[30]	Italy	Visual task of viewing pictures with Internet themes and emotional images having neural, positive, and negative themes as conditions	• IAD (n=16) • HC (n=14)	- there was no significant difference between IAD and HC when presented with Internet themed cues (likely due to heterogeneous group of IAD, who may have different motivations for their Internet addiction) - IAD subjects had ↑ activations detected using the somato-sensorial cortex on LoRETA software (likely due to dissociative symptoms in pathological Internet use, i.e., disruption of the normal psychological functioning caused by the IAD.
12. Gao et al. (2019)[31]	China	Go/No-go task	• excessive SNS users (n=23) • HC (n=20)	- N100 amplitude was ↑ following SNS images than control images in excessive SNS users. - Excessive users showed ↑ N200 amplitude and ↓ No-go P300 amplitude than non-excessive users irrespective of stimuli. - Excessive SNS users are inefficient in allocating monitoring resources in the No-go task (reflected by enhance N200) and showed difficulty in late inhibitory control procedure (reflected by ↓ No-go P300) compared to non-excessive users. - Excessive SNS users pay more attention to SNS-related images compared to non-SNS-related images (reflected by the N100).
13. Karapetsas AV et al. (2020)[32]	Greece	IAD related visual stimuli	• IAD (n=14) • HC (n=14)	- longer latency of P300 that normalised in post rehabilitation EEG recordings in the IAD - Improved IAT score was observed post rehabilitation programme and correlated with shorter latency period of P300.
14. Killian C et al. (2020)[33]	Germany	Go/No-go task and response to reward assessment Flanker task	• HBW (n=35) • NBW (n=33)	- Internet entertainment programmes streaming binge watching subjects who were HBW showed ↑ P300a and P300b during response inhibition and ↑ MMN/ERN for Flanker task errors.
15. Wang J et al. (2020)[6]	China	Two-choice Oddball task (neutral stimuli and deviant pornographic images)	• TCA (n=36) • HC (n=36)	- TCA showed ↓ N200 and P300 waves amplitude for deviant compared to neutral stimuli. - TCAs were more impulsive/ showed lack of inhibitory control and shared similar neurophysiological ERP changes with substance use disorder and other behavioural addictions.

(B) Internet Gaming Disorder

Author (Year)	Region	Type of stimulus	Participants, Sample size	Findings
1. Duven ECP et al. (2015)[34]	Germany	Cue induced reactivity/ Monetary reward computer game task	• IGD (n=14) • HC (n=13)	- IGD showed ↑ N100 amplitude, ↓ P300 amplitude, likely caused by an initial ↑ attention status that required more cognitive capacity to process the gaming reward, but later evaluating that the reward required less attention (this is contradictory to other findings but is explained by the maintenance phase of addiction, whereby habitual processing of the addictive stimuli might lead to less attentional processing of the addiction-specific cues).
2. Kim SN et al. (2018)[35]	South Korea	Gaming related cues	• IGD: 20 • OCD: 20 • HC: 23	- IGD group showed ↑ amplitude of P300 at the CPz in game-related cues (indicating ↑ arousal/attentional resources to gaming-related cues).
3. Kim J et al. (2019)[36]	South Korea	Comparison of band power during playing online games	• IGD (n=24) • HC (n=35)	- IGD showed ↓ left frontal θ, α, and β band activities - left frontal θ power negatively correlated with IGD severity. - Proposed that left frontal θ power could be used as a neurophysiological biomarker for the detection of diminished cognitive control in IGD.
4. Park M et al. (2017a)[9]	South Korea	Auditory oddball task	• IGD (n=26) • AUD (n=22) • HC (n=29)	- IGD and AUD groups had ↓ P300 amplitudes at the midline central and parietal area compared with the HCs. - IGD had ↓ N100 amplitudes at the midline frontal area compared with the HCs. - ↓ P300 was correlated with a higher spatial span error rate in the IGD.

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5. Park M et al. (2017b) [37]	South Korea	Auditory oddball task at baseline and 6 months post SSRI interventional therapy	• IGD (n=18) • HC (n=29)	- At baseline recording, IGD had ↓ P300 amplitude and longer latency at the CPz - At post treatment recording, although there were no significant changes in EEG waves, there was significant reduction in the symptoms and improvements in the addiction scores - This indicates that ↓ P300 amplitude and longer latency at the CPz are likely to be candidate endophenotypes in the pathophysiology of IGD.
6. Ha J et al. (2020)[38]	South Korea	Cue induced reactivity towards watching preferred gaming video vs. control	• IGD (n=20) • HC (n=20)	- When watching the preferred type of gaming video (addiction-specific stimulus), the IGD showed ↑ absolute θ power and ↓ absolute β power at the parieto-occipital region that correspond to cravings related features.
7. Raiha S et al. (2020)[39]	South Korea	Response to reward cue reactivity task	• IGD (n=35) • HC (n= 39)	- IGD showed blunted FRN for losses vs gains - when presented with high-risk choices, there was increased FRN amplitude in HC, but not in IGD (likely to represent impaired risk avoidance tendency/ inability of the ECN to exert an inhibitory control).

Footnote: ↓: reduced, ↑: increased, AUD: alcohol use disorder, BART: Balloon Analogue Risk Task, CBT: cognitive behaviour therapy, CPz: central-parietal zone, DISC-IV: ADHD Diagnostic Interview Schedule for Children Version IV, CDI: Children's Depression Inventory,DSM-5: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, ECN: executive control network, ERN: error related negativity, FRN: feedback related negativity, MMN: mismatch negativity, HBW: high binge watching, NBW: no binge watching, PE: physical exercise, PFC: prefrontal cortex, K-ARS: Korean version of ADHD Rating Scale, IAT: Internet Addiction Test, MDD: major depressive disorder, K-scale: Korean Internet Addiction Self-scale, SSRT: selective serotonin inhibitor, SCID: Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders-IV, BDI: Beck's Depression Inventory, BAI: Beck's Anxiety Inventory, ADHD: attention deficit hyperactivity disorder, ASD: autism spectrum disorder, SSRI: selective serotonin receptor inhibitor, TCA: tendencies towards cybersex addiction.

The neurobiology of this observation correlates with the hypothesis that the ECN fails to exert an inhibitory effect in addicts, often causing decreased P300 amplitude.[2] Nevertheless, the P300 amplitude increases with the rarity of the stimulus, is sensitive to changes in the working memory, and is affected by the preferential allocation of attentional resources.[9] In SUD, particularly alcoholics, P300 ERP is observed to have a reduced amplitude and a delayed latency, particularly in the parietal region.[9] In fact, a reduced P300 has been observed in both Go and No-go tasks in alcoholics, indicating a dysfunction of the response to reward among addicts.[2] Additional data is available pertaining to the P300 amplitude in SUD, whereby a larger P300 amplitude was provoked by alcohol-related pictures in alcohol dependent patients but not in controls.[9] Conversely, reduced P300 amplitudes and delayed P300 latency periods have been noted in the centro-parietal zone (CPz) among IGD subjects compared with healthy controls.[9]

The feedback related negativity (FRN) is an ERP, which can elucidate the neurocognitive correlates of decisional behaviour in addiction. FRN effects are characteristically negative amplitude deflections that occur at the mediofrontal regions and peak at approximately 200–350 ms after the onset of the feedback stimulus, which codes for reward predicting error.[9] FRN acts to monitor performances and is generated at the medial prefrontal cortex (mPFC) and ACC.[2] Varying results have been observed pertaining to the significance of the FRN, however there is consensus that this ERP is an adaptive mechanism that analyses the outcome expectancies and provides a feedback control mechanisms that alters the rewarding power of responses.[9] In combination, the P300 and FRN act as biomarkers of the increased inability to adopt an adequate cognitive strategy in response to a decisional context. This occurs in the presence of some rewarding bias, which occurs concomitantly with an anomalous automatic attentional response.[9]

A summary of studies that evaluated task-based stimuli with simultaneous EEG in IAD and IGD can be found in Table II.

Electroencephalography during stimulus presentation

The main components of working memory are visuo-spatial scratchpad and attention allocation. Many neuropsychological paradigms assess for working memory, the cues are often visual cues and preferably are addiction-specific cues.[2] Stroop Colour and Word Test (SCWT), also known as the Stroop Test for short, is an example of a neuropsychological test, which is extensively used to assess the ability to inhibit cognitive interference. A Stroop task is administered with alternating 'congruent' and 'incongruent' stimuli, whereby a congruent stimulus occurs when what the words say, compared to what the displayed colours of words portray are matched (congruent), hence the frequency of the congruent and incongruent stimuli can be designed to form a Go/No-go task. Conversely, an incongruent stimulus is when the words and displayed colours are mismatched/contradictory. Notably, a previous Stroop task study has detected that children had longer reaction time (slower to respond) and more inferior results accuracy (made more mistakes) compared to adults when they had mixed tasks, i.e., neutral, congruent, and incongruent trials all mixed in every block of the paradigm.[17]

The oddball task, which is used to detect short-term memory retrieval, is performed by requiring the subject to respond mentally or physically to an infrequent target presented amidst frequently occurring standard stimuli and infrequently occurring distracters.[6] He et al., 2018 also utilised an oddball paradigm in an ERP experiment that induced mismatch negativity (MMN).[40] MMN was significantly induced in the subjects with IAD as evidenced by more significant negative deflection of the waveform occurring at the timing of the amplitude at the time of display of the Internet-related pictures.[40]

In a study by Park et al. 2017a, IGD exhibited reduced N100 amplitudes at the midline frontal area compared with the controls.[9] Among the IGD, the reduced P300 was associated with a higher spatial span error rate. However, the reduced P300 and N100 amplitudes were not correlated with the Internet addiction severity scores in the IGD. These results indicate that IGD is associated with abnormalities in the P300 comparable to those with alcohol use disorder.[9]

Zhou et al., 2010 evaluated Problematic Internet Use (PIU), which is like IAD, and its association with impulsivity by performing a Go/No-go task along with simultaneous EEG.[22] It was identified that the N200 amplitude was significantly decreased in the frontal regions during the No-go task among the PIU and correlated with impulsivity scores. This indicates that PIU shares similar neuropsychological characteristics of compulsive-impulsive spectrum disorder.[28] Zhang et al. 2016 conducted an ERP experiment to evaluate dysfunctional face processing in IAD patients and identified that the underlying mechanism of processing faces could be different in IAD compared to healthy individuals.[27]

Role of electroencephalography in monitoring treatment response

A study by Lai et al., 2017 recorded EEG data while presenting images related to the internet and visual emotional cues among IAD patients undergoing psychological treatment.[30] The study utilised a software, i.e., the GeoSource (version 2.0; EGI, Eugene, OR) that is based on the standardized Low Resolution Electromagnetic Tomography (LoRETA). LoRETA is a software that enables the identification of the neural sources of the measured scalp potentials.[2,30] Probabilistic maps are generated and averaged in the Talairach space using an anatomical atlas, then the mean intensity of the Brodmann Areas are extracted based on the identified ERP components. Prior to therapy, IAD patients have a higher primary somatosensorial cortex and lower paralimbic, temporal, and orbito-frontal activation in response to both internet and emotional images as compared to the healthy population.[30]

DISCUSSION

Internet Addiction Disorder comprises of a broad range of addictive online stimuli that induce cravings and reveal a lack of inhibition among both genders of addicted adolescents and young adults. Conversely, Internet Gaming Disorder, which is a specific type of internet addiction, particularly afflicts young males. Teenagers are in the most vulnerable age group, as they develop more severe complications compared to other age groups when addicted to the internet. The deterioration of the inhibitory control

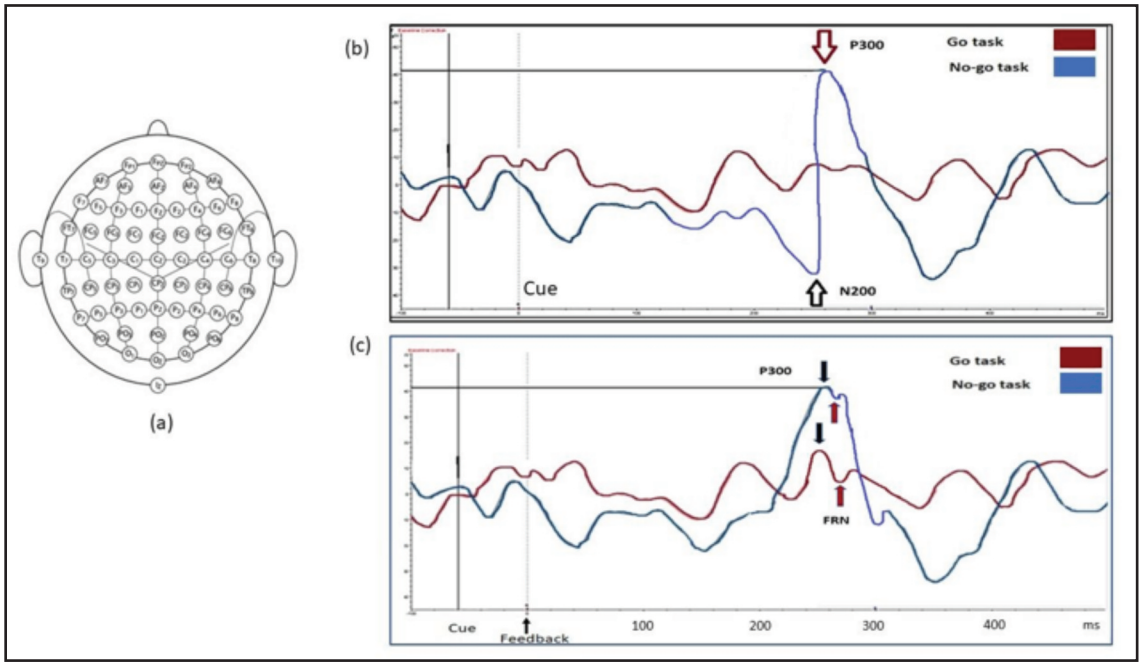

Fig. 1: (a) EEG electrodes designated positions for 64-channels setup. (b) Task-based EEG recording at the central electrodes demonstrated the negative deflection of N200 wave occurring at approximately 220-260 ms from the time of cue presentation, with significantly larger amplitude when performing the No-go task (addiction-specific task) compared to the Go task. The P300 positive deflected wave, occurring at approximately 280-320 ms, also showed a larger amplitude when performing the No-go task compared to the Go task. (c) Task-based EEG recording at the frontal electrodes, demonstrated the P300 positive deflected wave, occurring at approximately 280-290 ms from the time of feedback presentation, which showed a larger amplitude when performing the No-go task (addiction-specific task) compared to the Go task. The feedback related negativity (FRN) evoked response potential demonstrated the negative deflection at approximately 2280-300 ms from the time of feedback presentation, with significantly larger amplitude when performing the No-go task compared to the Go task.

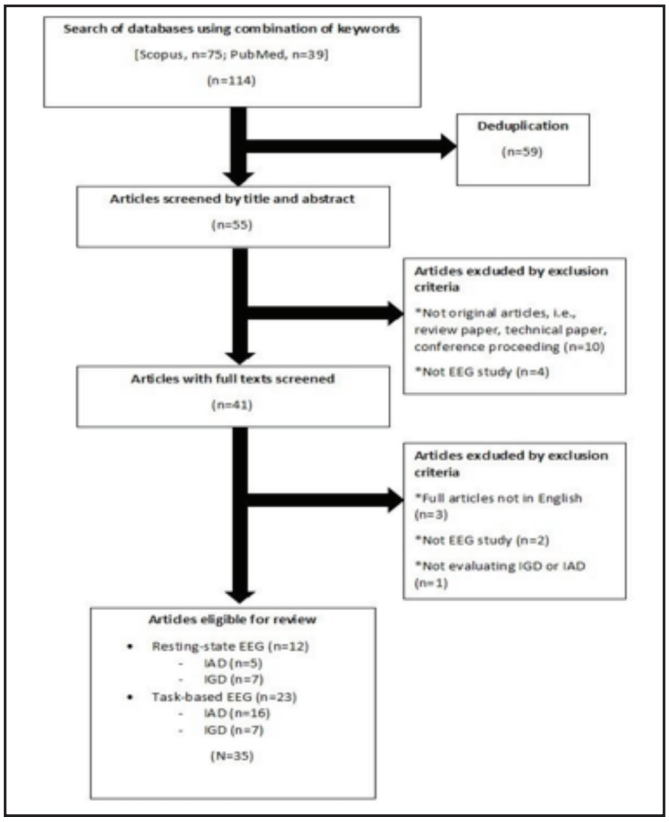

Fig. 2: PRISMA flowchart for selection of eligible articles in this scoping review.

exerted by the ECN, coupled with an inherent risk approach caused by impulsivity, make the IGD subjects susceptible to seeking rewarding stimuli persistently.

Resting-state EEG commonly demonstrates increased θ band activity in the frontal brain regions of the addicts. Hence, a decreased absolute power of the θ band in the frontal brain region may be a trait marker of IAD and IGD, which can act as a biomarker for detecting diminished cognitive control.[11,36] Alternatively, increased absolute power of the θ band has been hypothesized to represent trait markers of IAD with comorbid MDD, because θ waves are predominantly associated with emotional memory retrieval and meditative states.[8]

Furthermore, abnormal ERPs, i.e., the N200, P300 and FRN, were observed in both the IAD and IGD subjects. Nevertheless, there is conflicting evidence from various task-based EEG studies regarding the influence of IAD and IGD on the P300 and FRN amplitude waveforms. Essentially, ERPs on cue reactivity, such as the P300 and FRN, can be utilised as biomarkers for IGD. Deficits in behavioural feedback are believed to be related to rewarding bias, whereby the modulations of FRN and P300 are postulated to be affected by salience detection and motivational states of the subject.[534,39] The subjects' anticipation and prediction of the rewarding stimuli is believed to exert a significant FRN amplitude reduction.[39]

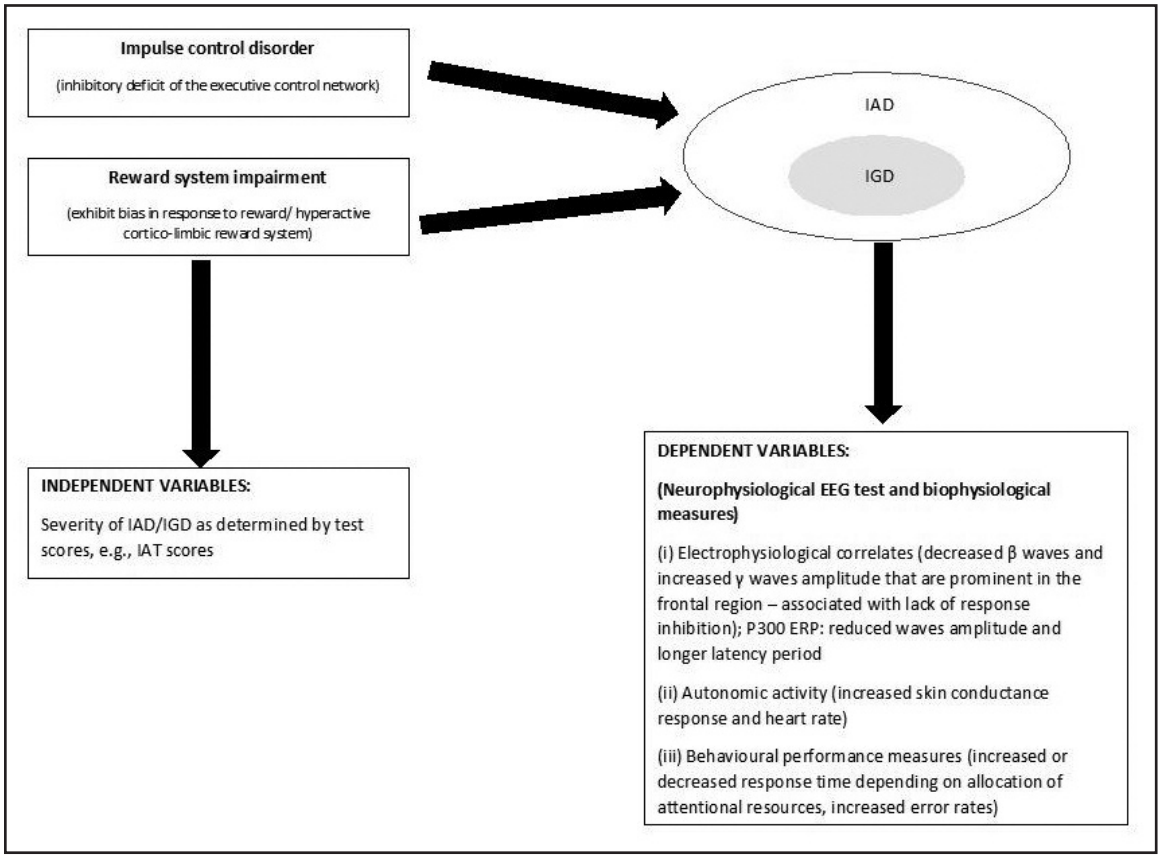

Fig. 3: Conceptual framework of the role of EEG in identifying the neurophysiological underpinning of IAD and IGD.

Implicitly, altered P300 modulations, specifically reduced amplitude, can act as a biomarker of IGD that identifies impaired attentional states when presented with perceived rewarding stimuli in comparison with the non-rewarding stimuli for these subjects.[9,34,37] On the contrary, larger P300 amplitudes have been observed in both IGD and IAD subjects after the presentation of the perceived rewarding cues, which is hypothesized to be caused by an impaired salience assignment and increased arousal or attentional resources to addiction-related cues.[21,33,35]

It is also important to note that comorbid conditions such as ADHD may affect the EEG waveforms, e.g. IGD with concomitant major depressive disorder (MDD) had reduced α band coherence between the interhemispheric frontal regions.[17] As for ADHD only subjects, there is increased power of θ at the frontal regions, however for the ADHD with IGD there is increased power of β at the temporal regions, likely caused by the repetitive activation of the brain reward and working memory systems during continuous gaming.[15]

The conceptual framework regarding the role of EEG in evaluating the neurophysiological changes that occur in IAD and IGD is elucidated by this scoping review (Figure 3). It is hypothesized that the underlying neural mechanism of the overactive cortico-limbic reward system and the deficient inhibitory control exhibited by the ECN collectively give rise to IAD and IGD. Both the conditions share similar neural underpinnings of impulsivity but differ in the type of rewarding stimuli that the addicts respond to.

The limitations include the fact that most studies were performed in either South Korea (mainly for IGD) or China (mainly for IAD). Hence, the recognition of IGD as a true pathological condition is still controversial. More multinational studies are needed to evaluate this disorder to evaluate the consistency of the findings. Another limitation includes the lack of standardisation of the diagnostic criteria and cut-off values to identify the subjects at risk of IAD and IGD. There is also heterogeneity in the observed P300 wave deflections, whereby some studies observed reduced wave amplitude and others observed increased wave amplitude of the P300.[6] The former observation is believed to be caused by reduced attentional resources, whereby habitual processing of the addictive stimuli might lead to less attentional processing of the addiction-specific cues, particularly in the maintenance phase of the addiction. The latter observation can be frequently seen in addicts because of the increased arousal and recruitment of attentional resources towards addiction-specific cues. The interpretation of these findings can be harmonised if the selection of the test subjects is made with care, having considered the phase of addiction that they are currently in.

Moreover, many previous EEG studies pertaining to IAD and IGD have focused on assessing male participants. Hence,

more effort needs to be made to evaluate females, despite the challenges that are associated with performing scalp EEG testing in this group. Additionally, although EEG provides excellent temporal resolution and can provide a gross localization of the neural source using LoRETA, the incorporation of simultaneous EEG-correlated functional magnetic resonance spectroscopy (EEG-fMRI) may pave the way for the improved understanding of the neurophysiology and neuropathology of this condition.[2]

CONCLUSION

EEG can identify distinct neurophysiological changes among Internet Addiction Disorder and Internet Gaming Disorder in adolescents and young adults that are akin to substance abuse disorders.

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CONFLICT OF INTEREST

The authors confirm that there is no conflict of interest to declare.

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