Neural Network Dynamics and Brain Oscillations Underlying Aberrant Inhibitory Control in Internet Addiction

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***Abstract*— Previous studies have reported a role of alterations in the brain's inhibitory control mechanism in addiction. Mounting evidence from neuroimaging studies indicates that its key components can be evaluated with brain oscillations and connectivity during inhibitory control. In this study, we developed an internet-related stop-signal task with electroencephalography (EEG) signal recorded to investigate inhibitory control. Healthy controls and participants with Internet addiction were recruited to participate in the internet-related stop-signal task with 19-channel EEG signal recording, and the corresponding event-related potentials and spectral perturbations were analyzed. Brain effective connections were also evaluated using direct directed transfer function. The results showed that, relative to the healthy controls, participants with Internet addiction had increased Stop-P3 during inhibitory control, suggesting that they have an altered neural mechanism in impulsive control. Furthermore, participants with Internet addiction showed increased low-frequency synchronization and decreased alpha and beta desynchronization in the middle and right frontal regions compared to healthy controls. Aberrant brain effective connectivity was also observed, with increased occipital-parietal and intra-occipital connections, as well as decreased frontal-paracentral connection in participants with Internet addiction. These results suggest that physiological signals are essential in future implementations of cognitive assessment of Internet addiction to further investigate the underlying mechanisms and effective biomarkers.**

***Index Terms*— Electroencephalography (EEG), internet addiction (IA), brain oscillations, effective connectivity.**

Manuscript received 1 October 2023; revised 5 January 2024; accepted 3 February 2024. Date of publication 9 February 2024; date of current version 28 February 2024. This work was supported in part by the Collaborative Research Project of National Sun Yat-sen University and Kaohsiung Chang Gung Memorial Hospital, Taiwan, under Grant CGMH-NSYSU Joint Research Program-111-06, Grant CORPG8M0261, and Grant CORPG8N0171. *(Corresponding author: Liang-Jen Wang.)*

This work involved human subjects or animals in its research. Approval of all ethical and experimental procedures and protocols was granted by the Institutional Review Board (IRB) at CGMH, Taiwan, under Application No. 202101487A3.

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Digital Object Identifier 10.1109/TNSRE.2024.3363756

I. Introduction

THE development of the Internet and digital information technology has brought new public health issues, such as internet addiction and internet gaming disorder (IGD). Internet addiction (IA), also known as Problematic Internet Use, is defined as a behavioral addiction involving psychological dependence on internet applications, such as online social networking, gambling, gaming, cybersex, and e-shopping [1], [2], [3]. It is characterized by the uncontrolled use of the internet and an excessive craving for online activities [4]. Although there is still considerable controversy surrounding the existence of Internet addiction disorder [5], it is worth noting that only IGD has been included as a proposed disorder within the broader category of Internet addiction in the last version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) in 2013 [6]. Moreover, the prevalence of IGD has been reported to be high, ranging from 0.2% to 57.5%, in general populations [7]. Previous studies have claimed that the risk of internet addiction in children has also become an increasing problem, particularly with the popularity of mobile internet [8]. Furthermore, it has been found that almost one-quarter of early teenagers spend 40 hours online per week [9]. The rapidly increasing incidence makes internet addiction a public health problem [3]. Recent evidence has also shown that internet addiction is associated with various comorbidities, including attention deficit/hyperactivity disorder, depressive disorder, social phobia, hostility, and interpersonal and emotional impairments. Additionally, internet addiction has been linked to dissociative phenomena and suicidal ideation [5].

A. Internet Addiction Disorder and the Inhibitory Mechanism

The relevance of impulsivity and general executive functions is highlighted by empirical findings on addictive behaviors related to IA [10]. IA is claimed to be attributed to deficits

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in inhibitory impulse control and impairment of the frontal executive control network. These deficits can lead to the development of different kinds of behavioral addictions [1], [2], [11]. High impulsivity is a defining feature of IA and is manifested during addiction-related behaviors, such as gambling, gaming, social network use, and binge-watching. Empirical studies have demonstrated deficits in inhibition associated with binge-watching [12], social networks use disorder [10], [13], [14] problematic online pornography use [15], and gaming disorder [16]. These studies have investigated inhibitory control in response to addiction-related cues. Therefore, it is crucial to understand the neurophysiology of the mechanisms underlying this condition in order to establish an effective treatment plan. Theoretical models, such as the Interaction of Person-Affect-Cognition-Execution (I-PACE) model proposed by Brand and colleagues in 2019 [17], have further demonstrated the potential interactions between impulsivity, general executive functions, and specific inhibitory control. According to the I-PACE model, situational factors, such as exposure to addiction-related cues, can interact with individual coping styles (e.g., trait impulsivity) and cognitive biases, leading to affective and cognitive responses, including addiction, craving, and attentional bias [10]. Furthermore, distinct cognitive impairments, such as deficits in decision-making, working memory, attentional control, and motor inhibition, have been demonstrated in individuals with IA [10], [18]. The behaviors of individuals with addictions are also believed to increasingly rely on limbic structures associated with impulsive and reactive neural systems [19], while the involvement of the prefrontal cortex in inhibitory control over urges may decrease during the addiction process [20]. These studies on theoretical perspectives have proposed that the decisions to engage in specific behaviors are influenced by the level of inhibitory control and self-directedness [17], [21], and may be primarily guided by impulsive and reactive responses to triggers. Reductions in inhibitory control and general executive functions can impact various cognitive processes, including attention, information retrieval and integration, planning, monitoring, updating, and strategy evaluation. Moreover, these reductions may exacerbate specific behaviors, such as the excessive use of online applications, and result in a loss of control over behavior and negative consequences in daily life [10].

B. Neuroimaging Evidence for IA and Inhibitory Control

Possible interactions between inhibitory control and executive functions have been relatively understudied in specific IA. Some behavioral and neurophysiological studies have supported the evidence of a deficit in inhibitory control that leads to addictive behavior in IA participants. In functional Magnetic Resonance Imaging (fMRI) studies [22], IA participants have shown aberrant activations and connectivity in the response inhibition network. The IA group exhibited significant hyperactivity during inhibitory control in the left and right superior/medial frontal gyrus, dorsal lateral prefrontal cortex, right anterior cingulate cortex, left inferior parietal lobule, left precentral gyrus, left precuneus, and cuneus [23], [24]. Decreased activation in the left medial temporal gyrus, right supplementary motor area (SMA), and pre-SMA has been observed during inhibitory control and tasks with anxiety interference [25], [26]. Abnormal brain connections have also been found in resting fMRI studies, including decreased effective connections from the parahippocampal gyrus to the prefrontal cortex [27]. However, most of the previous studies have reported results of resting-state brain connectivity instead of functional connectivity during inhibitory control. These studies have suggested that there is abnormal functional connectivity in the cognitive control network, default mode network, and visual attention network in IA participants [3], [4], [28], [29].

Although previous fMRI studies have identified specific brain areas and proposed theoretical models related to addictive processes in IA based on behavioral and neuroimaging studies [17], there remains a significant gap in our understanding of the neural mechanisms underlying brain dynamics and functional connectivity. Since these addictive and inhibitory processes exhibit high temporal dynamics, there is a need for neuroimaging modalities with higher temporal resolution, such as electroencephalography (EEG) and magnetoencephalography (MEG), to explore the underlying brain functional network. However, recent EEG studies focusing on inhibitory control have yielded diverse and inconsistent results [2], [12], [30], [31], [32], [33], with only a few studies investigating brain functional connectivity [34], [35]. Therefore, in future neuroscience research, it is crucial to define and validate reliable neurodynamic features of IA.

In this study, we developed a stop-signal paradigm utilizing Internet-related stimuli for IA participants. EEG signals were recorded using a mobile EEG device to assess brain oscillations and functional connectivity during the experiment. Our aim is to investigate the following hypotheses: 1) the presence of altered brain oscillations and distant functional connectivity in IA participants, and 2) the proposal of neurofeedback indices based on recorded EEG neurophysiological signals for cognitive assessment.

II. Materials And Methods

A. Participants

We recruited a total of 53 participants, including 31 healthy control (HC) participants (aged 19.52 $\pm$ 5.01; 24 men) and 22 participants with IA (aged 16.64 $\pm$ 5.88; 18 men). Prior to the experiment, informed written consent was obtained from the legal representatives of all participants, in accordance with the requirements of the human subject research ethics committee/Institutional Review Board (IRB) at CGMH, Taiwan (no. 202101487A3). Participants were assigned to either the HC or IA group based on their scores on the Chen Internet Addiction Scale (CIAS). The CIAS is a 26-item self-reported scale with four-point response options, assessing five dimensions of Internet-related symptoms and problems, including compulsive use, withdrawal, tolerance, and problems in interpersonal relationships and health/time management [36]. The CIAS score ranges from 26 to 104, with higher scores indicating greater severity of Internet addiction. Optimal screening cutoffs of 57/58 and diagnostic cutoffs of 63/64 were applied for adolescents, while optimal screening cutoffs of 63/64 and diagnostic cutoffs of 67/68 were applied for college students [37].

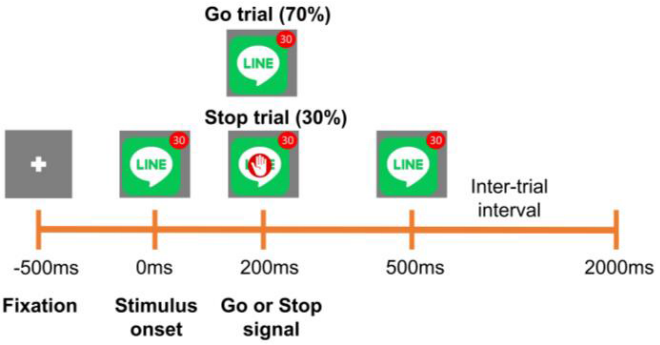

Fig. 1. Stop signal task. Each trial consisted of the presentation of a background picture (neutral or Internet-related image).

Participants' neuropsychiatric comorbidities were determined based on the criteria outlined in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [6] and the International Classification of Diseases (ICD-11; WHO 1994) criteria. Diagnostic scales were used to assess social inference, emotional communication, language and cognitive abilities, and motor coordination skills. Additionally, the participants' full-scale intelligence quotient (FSIQ) was assessed using the Wechsler Intelligence Scale for Children [38] and Adult [39]. The IA and HC groups were matched as closely as possible based on their FSIQ scores.

B. Experimental Design: Stop-Signal Task

The participants were instructed to perform a stop-signal task while their EEG data was recorded. The stop-signal task was a modified version of the experimental task used by Balconi and Finocchiaro [33], [40], [41]. It consisted of four blocks, with each block containing 120 stimuli. Within each block, there were 84 Go trials and 36 Stop trials. The task was conducted in multiple sessions.

During the task, participants viewed randomized background pictures from two different contexts: neutral (N) and Internet (I), each presented for 500 ms. The Internet condition included images related to social platforms and online games. After the presentation of the background picture, the initial background picture returned during inter-trial intervals (ITIs) (see Fig. 1). The duration of ITIs varied randomly between 1300 to 1700 ms, with an average of 1500 ms.

Stop signals were presented in a random order to maintain a consistent percentage of Go (70%) and Stop (30%) trials within each block. The stop signals appeared in the center of the background picture after a delay of 200 ms (see Fig. 1), and remained visible for 300 ms.

Participants were seated on a comfortable chair facing a screen positioned approximately 60 cm away from them. They were instructed to press a button as quickly as possible when they saw the Go stimulus at the center of the screen, and to withhold their response for the Stop stimulus. Additionally, they were instructed to minimize movement and blinking during the task to reduce noise during EEG registration. Each participant completed a total of 480 trials.

C. EEG Recording and Signal Processing

The EEG signals were recorded using the Quick-20 Dry EEG Headset (CGX, Inc., San Diego, CA, USA) from 20 scalp locations. The electrodes were placed according to the international 10-20 system, with reference to the left earlobe (A1). The specific electrode locations were as follows: Fp1, F7, F3, Fz, F4, F8, Fp2, C3, Cz, C4, P7, P3, Pz, P4, P8, T3, T4, O1, and O2. The EEG signals were sampled at a rate of 500 Hz, and the stimulus triggers were synchronized and saved in a single file using the lab streaming layer (LSL) data acquisition framework. The data were saved in extensible data format using LSL LabRecorder software version 1.05. MATLAB R2022a (The MathWorks, Inc., Natick, MA, USA) was used for data analysis.

The recorded EEG data underwent several preprocessing steps. First, a highpass filter with a cutoff frequency of 1 Hz was applied using the EEGLAB toolbox [42], [43]. The Cleanline EEGLAB plug-in was then used to remove line noise from the data. Artifacts in the EEG signals were identified and removed using artifact subspace reconstruction (ASR) with a parameter K set to 20 [44]. The data were then re-referenced to the average of all channels. Signal epochs were extracted from -200 to 1000 ms, and the signal was corrected using the mean value in the pre-stimulus period (-200 to 0 ms). Epochs containing extremely large amplitudes (greater than $\pm 100\ \mu$V or 3 times the standard deviation) were discarded. The adaptive mixture independent component analysis (AMICA) was applied to further remove any remaining unwanted artifacts that could not be identified and removed in previous steps, such as eye blinks, muscle artifacts, and line noise [45].

Finally, the epoched EEG data were subjected to signal averaging and Morlet wavelet transformation to obtain event-related potentials (ERPs) and event-related spectral perturbations (ERSPs). Averaged ERPs and ERSPs were calculated for each EEG sensor relative to the baseline. Synchronized averaging calculations were used to observe the ERPs of each group and condition. Event-related synchronization (ERS) and event-related desynchronization (ERD) were represented by increases and decreases in the power of different frequency bands, respectively, relative to the pre-stimulus baseline. Two-sample t-tests were performed to examine the differences in ERPs and ERSPs between the groups and conditions. Additionally, multivariate analysis of covariance (MANCOVA) was conducted to evaluate the influence of covariates, including comorbidity with Attention-Deficit/Hyperactivity Disorder (ADHD) and age factors.

D. Brain Effective Connectivity

Direct directed transfer function (dDTF) has been used to evaluate the frequency-domain representation of causality by applying the SIFT toolbox in EEGLAB [46]. The grand-mean effective connectivity across independent components (ICs) for the two groups was calculated using the groupSIFT toolbox in EEGLAB. The groupSIFT toolbox was originally developed for the study of chronic tic disorder [47] and has since been applied to several clinical studies [48], [49], [50], [51] and a cognitive study [52]. The technical details of the algorithm can

be found in the appendix, referenced in [47]. To provide a brief explanation of the algorithm, groupSIFT resolves individual subjects' ICs into a common anatomical space by converting each IC's equivalent current dipole coordinates into a 3-D sphere of *dipole density*, which serves as a probabilistic representation of the anatomical localization of the IC. The spheres of dipole densities are segmented into a priori defined anatomical regions of interest (ROIs) using the AAL atlas [53]. The dipole density acts as a weighting factor for the SIFT-derived IC-to-IC connectivity measures, which converts individualized IC-to-IC connectivity matrices into subject-consistent ROI-to-ROI connectivity matrices. Thus, groupSIFT avoids the issue of post-ICA inter-subject inconsistency. The weighted connectivity measure, dDTF in this study, is statistically tested for every time-frequency point and corrected using time-frequency domain cluster-level multiple comparison correction across all surviving graph edges to guarantee omnibus correction across time, frequency, and graph edges. The details steps of groupSIFT are described in the following:

1) **AMICA for extracting ICs from subjects** A total of 737 ICs were preselected as brain sources (HC: 14.16 ± 1.61 ICs; IA: 14.90 ± 1.48 ICs). The grand-mean effective connectivity across ICs for the two groups was calculated.
2) **Calculation of effective connectivity** Time-frequency decomposed dDTF was calculated across ICs with a sliding window length of 0.5 s, sliding window stop size of 0.025 s, 30 frequency bins ranging from 2 to 50 Hz, and a Hamming window.
3) **Validation of models** The grand-average optimum model order was 13.35 ± 2.78 and 12.15 ± 3.25 for HC and IA, respectively, indicating delayed effective connectivity up to approximately 25 ms.
4) **Conversion to group anatomical ROIs** The estimated equivalent dipole locations of the corresponding ICs were convolved with a 3-D Gaussian kernel with a full width at half maximum (FWHM) of 20 mm to obtain a probabilistic *dipole density*. The dipole density within the brain space was segmented into anatomical regions defined by the modified AAL [53]. The difference from the original AAL is that subcortical regions are re-organized into two larger subgroups, namely 'upper basal' and 'lower basal'. The purpose of this modification is to avoid showing specific names of the subcortical anatomical structures that is unlikely to be sources of scalp-measurable EEG signals. The cause of this problem is that IC dipoles are often (in our unpublished observations, about 25% of the 'Brain' ICs classified by ICLabel) localized at subcortical regions. One possible explanation for this issue is that the EEG sources identified by ICA may distribute to significant area of the cortex, which introduces depth bias in fitting a single dipole to their scalp projections. The modified AAL consists of 76 X 76 anatomical regions. The full list of the regions is found in the groupSIFT repository (https://github.com/sccn/groupSIFT).
5) **Statistical Analysis** Overlapped graph nodes calculated from the total dipole density between the two groups were further compared. The frequency range of interest selected for analysis was 1-50 Hz. The time-frequency representations of the data were tested against the baseline period and then across conditions. The mass univariate analysis using t-test was used. Multiple comparison correction was made using weak family-wise error rate (wFWER) control [54]. To obtain threshold values that are effective for omnibus corrections, surrogate distributions were built from data across all of the time, frequency, and connectivity edges. Thus, the results are protected from inflation of the Type I error arising from the repeated application of t-test across all time, frequency, and connectivity edges. Statistical significance was set at $p < 0.05$ with false discovery rate adjustment.

TABLE I
CHARACTERISTICS OF THE PARTICIPANTS WITH INTERNET ADDICTION (N=22) AND THE COMPARISON GROUP (N=31)

Variables	Internet addiction (n=22)	Comparison group (n=31)	χ or t	p-value
Sex, n (%)			0.151 [a]	0.697
Males	18 (81.8)	24 (77.4)		
Females	4 (18.2)	7 (22.6)		
ADHD, n (%)			8.727 [a]	0.003
With	11 (50)	4 (12.9)		
Without	11 (50)	27 (87.1)		
Age, years	16.6 ± 5.9	19.5 ± 5.0	1.918	0.061
FSIQ	106.6 ± 12.8	105.8 ± 13.1	0.199	0.843
CIAS	70.9 ± 9.6	51.1 ± 7.8	8.299	<0.001
SPAI	67.4 ± 12.5	54.6 ± 11.6	3.845	<0.001

Notes: Data are expressed as mean ± SD or n (%); [a]Pearson Chi-square; ADHD, Attention-Deficit / Hyperactivity Disorder; CIAS, Chen Internet Addiction Scale, FSIQ, Full-Scale Intelligence Quotient; SPAI, The Smartphone Addiction Inventory

III. RESULTS

A. Behavioral Results

The clinical characteristics of the participants are presented in Table I. The two groups were matched in terms of FSIQ, with no significant difference between them. The total accuracy rates for HC and IA participants were 94% ± 5% and 92% ± 7%, respectively, and there was a significant difference between the two groups ($p = 0.043$). The task accuracy for IA participants in the Stop trials was 91% ± 9%, slightly lower than that of HC participants (95% ± 5%), but the difference was not significant ($p = 0.122$). Similarly, the task accuracy for IA participants in the Go trials was 91% ± 9%, also lower than that of HC participants (94% ± 8%), but with no significance ($p = .215$). The mean reaction time for IA participants in the Go and Stop trials was 1.15 ± 0.06 s and 0.95 ± 0.04 s, respectively, slightly slower than that of HC participants (1.15 ± 0.07 s and 0.93 ± 0.19 s), but with no significance ($p = .963$ and .519).

B. ERP Results

The ERPs in the two conditions of the stop signal task were compared between IA and HC participants (Fig. 2). The total

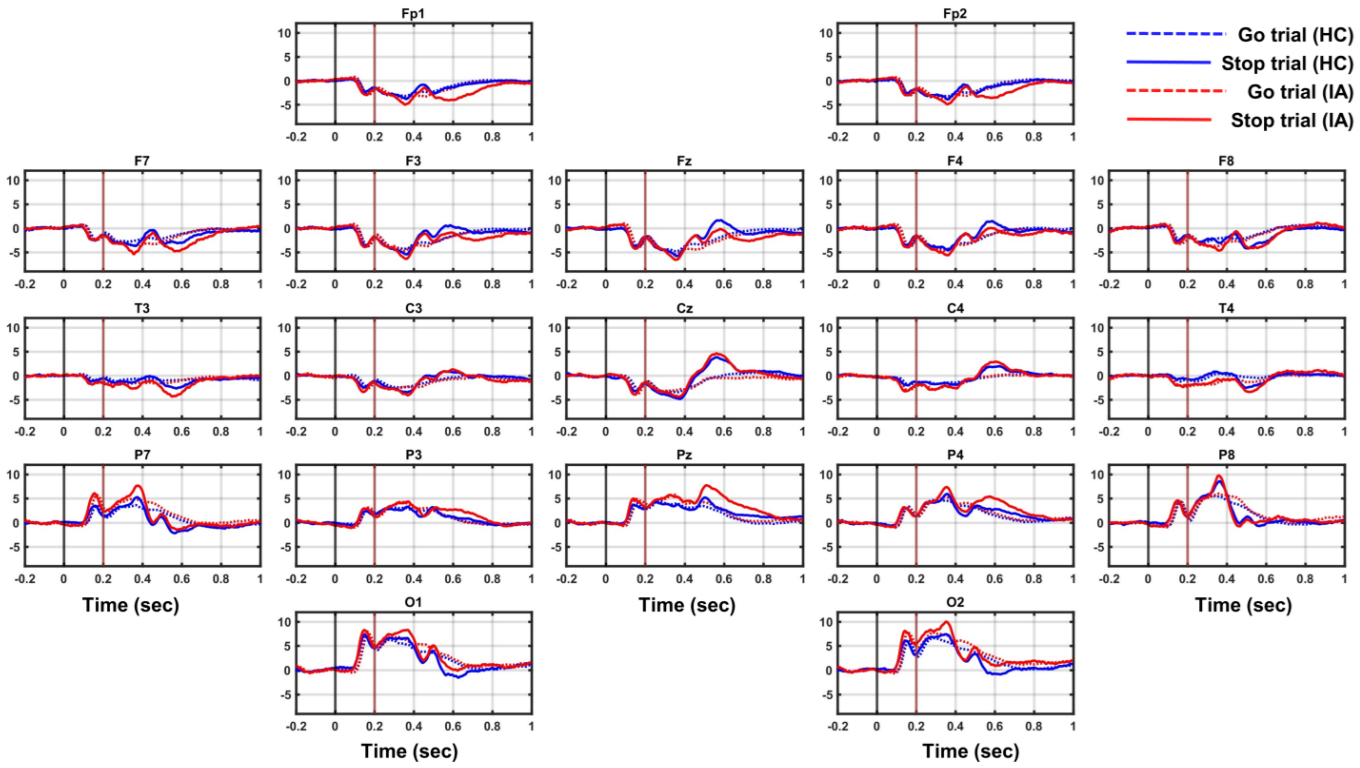

Fig. 2. The 19-channel ERPs of the Go (dotted lines) and Stop (solid lines) trials were compared between the HC (blue lines) and IA (red lines). No significant difference was observed between the two groups in the Go trials.

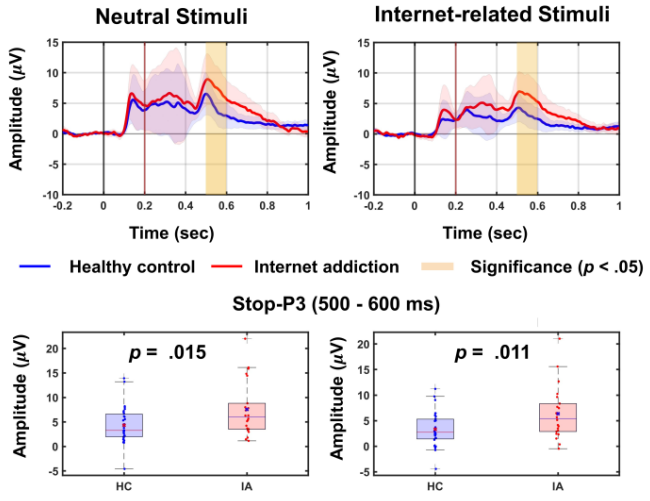

Fig. 3. The ERPs of the Stop trials during the two conditions (Neutral: left column; Internet: right column) of inhibitory control at Pz between 500 and 600 ms (Stop-P3).

19-channel ERPs of the Go and Stop trials (solid lines) were compared between the HC and IA groups. No significant difference was observed between the two groups in the Go trials. A significant difference was observed at Pz in the ERPs of the Stop trials during the two conditions of inhibitory control, as shown in Fig. 3. The ERPs of the Stop trials in both the Neutral and Internet-related stimuli during inhibitory control showed a difference in the Stop-P3 component in the parietal regions between 300 and 400 ms after the onset of Stop stimuli at 200 ms. The grand averages of the Stop-P3 waveforms at Pz and their corresponding mean amplitudes are shown in the lower portion of Fig. 3. Participants with IA exhibited increased Stop-P3 amplitudes (Neutral: 7.56 ± 5.62 μV; Internet-related: 6.37 ± 5.14 μV) compared to HC (Neutral: 4.34 ± 3.68 μV, $p = 0.015$; Internet-related: 3.33 ± 3.29 μV, $p = 0.011$), suggesting that IA participants have altered attention processing mechanisms. No significant difference was observed between the two conditions. When combining the Internet-related and neutral conditions, increased Stop-P3 amplitudes were observed in IA (6.85 ± 5.25 μV) than HC (3.76 ± 3.40 μV, $p = 0.012$). However, the significance between the two groups diminished after considering age and comorbid factors ($p = .170$), suggesting that age is also an important covariate influencing the amplitude of Stop-P3.

C. Time-Frequency Analysis

The increases in the power of different frequency bands relative to the pre-stimulus baseline, known as ERS, as well as the decreases in the power or ERD, are compared between the two groups in Fig. 4. Significant differences in ERS and ERD between the two groups were observed specifically in the middle and right frontal regions. The average ERSPs in the frontal regions of HC and IA are shown in Fig. 4A and 4B. Theta and alpha synchronization often occurred in the first 200-ms window after the onset of Go stimuli. Another ERS was observed around 400-600 ms, corresponding to the onset of Stop stimuli. Alpha and beta desynchronization were

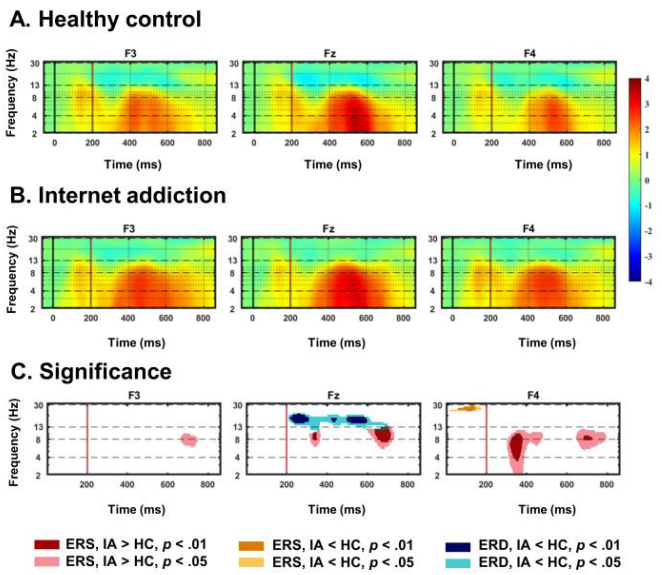

Fig. 4. The ERSPs of the Stop trials during inhibitory control are depicted in three channels of the middle frontal regions for (A) healthy control and (B) participants with internet addiction, with (C) the significance levels indicated.

observed in the time interval after 200 ms, indicating motor activity preparation and intention. The time intervals with significance and the corresponding frequency bands are displayed in Fig. 4C during inhibitory control. There was a pronounced difference in ERS and ERD between the two groups in brain oscillations of the delta, theta, alpha, and beta frequency bands. IA showed consistent increases in theta (4-8 Hz) and alpha (8-13 Hz) synchronization in the middle and right frontal regions after the onset of Stop stimuli (200 ms), as indicated by the red color in Fig. 4C. Sustained ERS was observed in the IA group during the late period of inhibitory control (after 600 ms). Additionally, a notable decrease in lower beta (13-21 Hz) desynchronization was observed in IA during motor reaction between 200 and 600 ms, represented by the blue color in Fig. 4C. The results of MANCOVA indicated that after considering the influence of covariates including comorbid ADHD and age factors, significant differences in ERSPs were observed in specific brain regions and frequency bands. These differences included post-stimulus theta synchronization at F4 between 300 and 400 ms (IA: 1.56 ± 0.91 dB, HC: 0.77 ± 1.02 dB; $p = 0.027$), alpha synchronization at Fz between 600 and 700 ms (IA: 0.97 ± 1.18 dB, HC: 0.01 ± 1.19 dB; $p = 0.008$), and beta desynchronization at Fz between 200 and 600 ms (IA: -0.13 ± 0.65 dB, HC: -0.74 ± 0.81 dB; $p = 0.044$). These results suggest that ERSPs are more reliable than ERPs in discriminating between IA and HC when considering the influence of comorbid and age factors.

D. Effective Connectivity

Fig. 5 displays the neural networks showing increased or decreased effective connectivity in participants with IA compared to healthy controls. In the group comparisons, a total of 28 out of 76 graph nodes exhibited overlap between the two groups, accounting for 66.6% of the dipole density

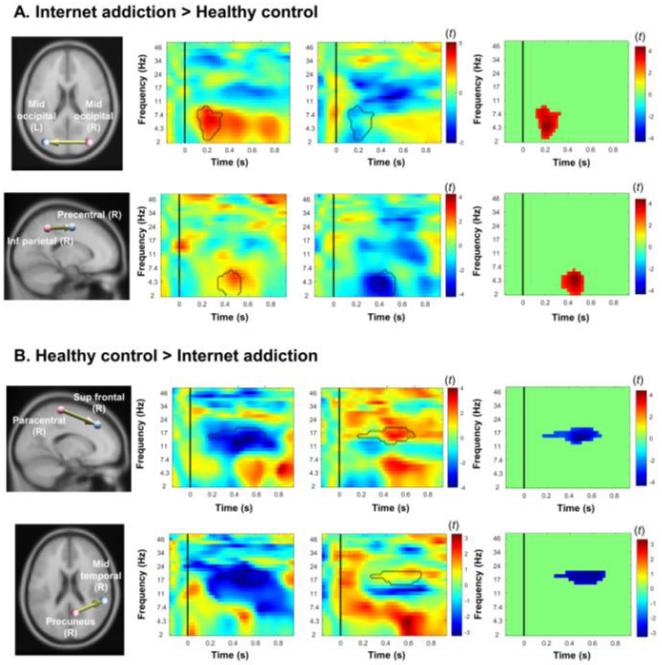

Fig. 5. The brain effective connectivity during inhibitory control. (A) The interhemispheric connection in the occipital region and connectivity from the parietal to precentral regions were observed to increase in the IA group. (B) Decreased connectivity from the paracentral to superior frontal regions, as well as from the precuneus to middle temporal regions, was found in IA.

from ICs. Aberrant connectivity patterns were observed in IA participants during impulsive control. As depicted in Fig. 5, IA participants exhibited increased connectivity from the right middle occipital to the left middle occipital regions in the delta and theta frequency bands immediately after the onset of Stop signals (with a centroid frequency of approximately 5.72 Hz and a centroid time of around 225 ms). Furthermore, there was increased information flow from the right inferior parietal to the right precentral regions, predominantly characterized by a centroid frequency of approximately 3.66 Hz and a centroid time of around 450 ms (Fig. 5A). Conversely, decreased connectivity was observed from the right paracentral lobule to the right superior frontal regions (with a centroid frequency of approximately 18.44 Hz and a centroid time of around 525 ms), as well as from the right precuneus regions to the middle temporal regions in IA (with a centroid frequency of approximately 15.33 Hz and a centroid time of around 450 ms), as illustrated in Fig. 5B. The most significant difference between HC and IA occurred in the beta frequency band between 0 and 500 ms after the onset of stop stimuli. As depicted in Fig. 6, boxplots illustrate the mean values of significant clusters in brain effective connectivity (Fig. 5, right column) during inhibitory control, indicating increased information flow in one group compared to the other.

IV. Discussions

A. Increased Stop-P3 Suggests Altered Processing of the Stop Signal in Internet Addiction

In the present study, we incorporated behavioral and EEG measures to characterize inhibitory control in Internet

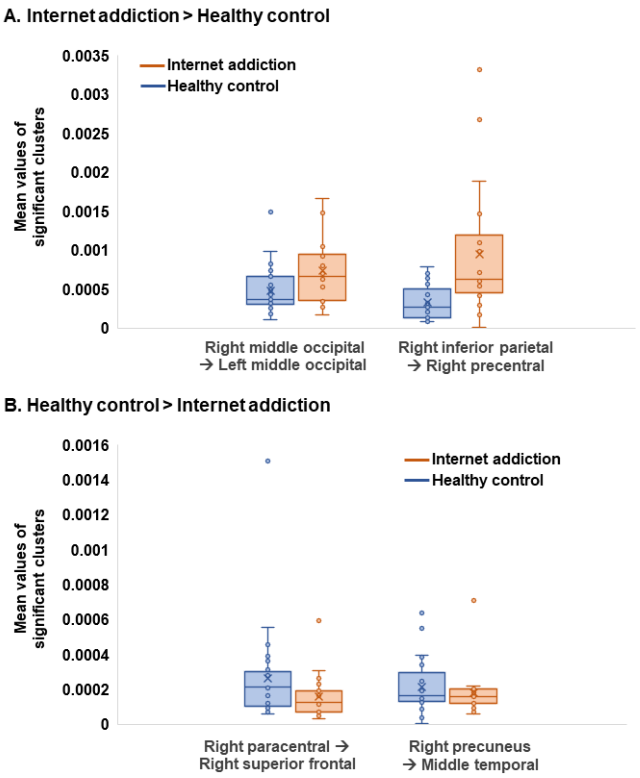

Fig. 6. The mean values of significant clusters in brain effective connectivity during inhibitory control are presented for the (A) IA group and (B) HC group.

addiction. IA participants exhibited lower accuracy in the stop-signal paradigm compared with HC, implying that they were not able to efficiently receive and utilize the information provided during inhibitory control. Underlying the lower task accuracy, our ERP analysis revealed that the stop signals elicited an increased Stop-P3 component in IA compared with HC participants. Since Stop-P3 is associated with implementing inhibition or stopping [55], the increase in Stop-P3 suggests altered neural activities of impulsive control in IA. As illustrated in Table II, previous EEG studies investigating inhibitory control effects on addiction-related cues demonstrated inhibition deficits to be associated with different kinds of IA. IA participants show atypical ERP in N170, N200, P300, and feedback-related negativity (FRN) components [2]. Echoing these studies, we found that Stop-P3 was attenuated in participants with Internet addiction. These findings provide evidence to support the theory that the required cognitive resources are aberrant in IA to successfully suppress response impulses or the stopping processes [55], [56]. However, it is worth mentioning that the significance of Stop-P3 between the two groups diminished after taking age factor as a covariate in the present study. Although many studies claim the possibility of using P300 as a neurophysiological biomarker for IA [57], our results suggest that the age factor may be another parameter that affects the amplitude of P300 and should be controlled with caution.

B. Sustained Frontal ERS in Internet Addiction

The investigation of EEG brain oscillations in Internet addiction during inhibitory processes has received limited attention. Previous studies have primarily focused on analyzing the power spectral analysis of EEG signals during resting states [31]. These studies have reported higher gamma activity in internet addicts, which is associated with the integration of perceptual and conceptual information and dysfunction in the dopaminergic system [31], [61]. Additionally, decreased beta and delta activity during resting states have been observed in Internet addiction compared to healthy controls, which has been linked to inattention and impulsivity [31], [61], [62]. However, it is important to note that the patterns of brain oscillations may be altered during different cognitive processes.

In the present study, we specifically focused on brain oscillations during inhibitory control using a stop-signal task. Additionally, there was a decrease in beta activity during inhibitory control in IA. Specifically, we found robust sustained theta and alpha ERS in the right and midline frontal regions in IA, independent of comorbid and age factors. Sustained frontal theta power has been associated with the initial phase of learning and subsequent decline [63]. Previous studies on ADHD have also reported sustained theta and alpha ERS activity during post-stimulus periods, indicating incremental retention for task requirements in memory and stop-signal tasks [64], [65].

Consistent with prior research, we observed a prolonged increase in alpha ERS at later post-stimulus time points, suggesting altered mechanisms in the active suppression of

TABLE II
PREVIOUS COGNITIVE STUDIES OF TASK-RELATED DEMONSTRATED EEG FINDINGS IN INTERNET ADDICTION

Study	Tasks	Participants	Findings
Dong (2010) [58]	Go/No-go task	IA (n=12) HC (n=12)	Increased P300 and longer P300 peak latency
Zhou (2010) [59]	Visual Go/No-go task	IA (n=26) HC (n=26)	Decreased N200
Balconi (2017) [41]	Attentional inhibitory task (Go/No-go task)	High IAT (n=12) Low IAT (n=13)	Decreased N170 and FRN, increased P300
Gao (2019) [32]	Go/No-go task	Excessive social network users (n=23)	Increased N100 and N200, decreased P300
Killian (2020) [12]	Go/No-go task	High binge watching (n=35) No binge watching (n=33)	Increased P300
Gao (2020) [60]	Go/No-go task	Problematic mobile phone use (n=15); HC (n=15)	Decreased P300
Moretta (2021) [14]	Emotional Go/No-go task	Problematic social network sites use (n=22); HC (n=23)	Decreased P300
Dieterich (2021) [55]	Go/ No-go task Stop signal task	High binge watching (n=32) No binge watching (n=31)	Increased P300 (stopping trials)
Chen (2022) [56]	Go/ No-go task	Gaming disorder (n=25) Control group (n=24)	Decreased nogo-N2 and increased nogo-P3

cognitive operations [64]. Recent studies have suggested that mid-frontal theta power is modulated by switch costs and is related to reaction time, with enhanced theta power during task preparation [66]. The sustained frontal theta and alpha activations observed in IA during post-stimulus periods may be attributed to the longer time required for successfully performing cognitive processes with additional neural resources. These activations could be associated with increased attention processes and compensatory mechanisms for insufficient vigilance during the early stages of perception [65].

Moreover, increased theta oscillations have been reported in patients with obsessive–compulsive disorder during a flanker task [67], while decreased theta oscillations were observed in patients with schizophrenia during a stop-signal task [68]. Although recent studies have reported different patterns of theta oscillations in IA during a Stroop task [69], further investigation is needed to validate the patterns of brain oscillations across different task contexts.

To the best of our knowledge, this is the first study to investigate brain oscillations in IA during impulsive control using a stop-signal task. These findings, which remained robust after controlling for comorbid ADHD and age factors, may serve as more reliable biomarkers of Internet addiction compared to the previously discussed Stop-P3 signals in the existing literature.

C. Hypoconnectivity of Frontal-Parietal Network in Internet Addiction

Aberrant connectivity patterns have been found in this study during impulsive control in IA. IA participants have demonstrated increased connectivity in the posterior brain regions, whereas decreased connectivity has been found in the frontal-parietal and central executive networks during inhibitory control. However, most previous connectivity studies of IA have focused only on resting-state analysis. Studies on structural connectivity using diffusion tensor imaging have shown a consistent increase in the thalamus, anterior thalamic radiation, corticospinal tract, and inferior longitudinal fasciculus in individuals with IGD, along with decreased nodal and global efficiencies [70]. In the past decade, more studies have investigated brain networks in IA and IGD using resting fMRI, revealing impaired connections in cortico-subcortical circuits, including the prefrontal and parietal cortices [71], interhemispheric functional connectivity in the prefrontal lobe [72], the reward system and executive control network [73], the frontal-parietal network and cingulo-opercular network [74], and the executive-cerebellar networks [75]. Conversely, increased occipital-putamen connectivity has been reported in IA [75]. Consistent findings of aberrant connectivity between the prefrontal cortex and other brain regions have been reported in these resting-state fMRI studies. Furthermore, very few fMRI studies have focused on task-based connectivity in IA during impulsive control, but the results also suggest the involvement of the prefrontal cortex in modulating impulsivity [76]. In the present study, advanced source reconstruction and connectivity methods were incorporated for EEG data. Taking advantage of better temporal resolution, task-based EEG signals were analyzed, and brain effective connectivity was evaluated during inhibitory control. The findings align with previous studies, showing decreased connectivity in the frontal-parietal and central executive networks, particularly from the right paracentral lobule to the right superior frontal regions, as well as from the right precuneus regions to middle temporal regions in the IA group during impulsive control. The precise time interval of the decrement was also indicated in the present study, suggesting that aberrant information flow occurs within the first 500 ms of inhibitory control after the onset of stop stimuli.

Compared to fMRI, there have been only a few EEG-based investigations with divergent results reported in the brain connectivity of IA and IGD. Some studies that have investigated brain connectivity using resting-state EEG have reported enhanced intra-hemisphere connectivity within the fronto-temporal area in IA/IGD participants, specifically in the delta, beta, and gamma frequency bands [34], [35], [77], as well as higher alpha coherence in the right hemisphere [31], [78]. Consistent with previous findings from resting fMRI studies, a recent study using resting EEG also demonstrated the important role of frontal and parietal areas in the neuropathological mechanism of IA, showing a decreased shortest path length of brain connectivity patterns [79]. In line with the aforementioned results of the present study, we suggest that the impairment of connectivity found in the frontal-parietal and central executive networks not only exists in the resting state but can also be observed during cognitive processes involved in impulsive control. Further investigation of task-based EEG connectivity is required to achieve a convergent result since only a few task-based EEG studies have mentioned brain networks in IA. In 2020, Wang et al. reported an altered brain network during working memory processes in IA, with the aberrant connection mainly found in the frontal and limbic lobes and in the alpha frequency band [80]. To the best of our knowledge, this is the first EEG study conducting a stop-signal investigation to evaluate the brain's effective connection in Internet addiction. In summary, our results suggest that increased occipital-parietal and intra-occipital connections in the delta and theta frequency bands are found in IA right after the onset of stop signals. In contrast, decreased frontal-paracentral and parietal-temporal connections in participants with IA have been reported after the first 500 ms of stop signals in the beta frequency band. Echoing previous studies, our findings demonstrate the possibility of using EEG signals as neurophysiological markers for Internet addiction [57].

V. Conclusion

An Internet-related stop-signal game has been developed to characterize the altered ERPs, ERSPs, and brain effective connectivity in participants with Internet addiction. Consistent with previous studies, altered Stop-P3 ERP components have been reported in this study. However, our results have also indicated that age should be considered as a covariate to reduce the sensitivity of using Stop-P3 as a neurophysiological marker for evaluating IA. Furthermore, our findings suggest that the sustained frontal ERS is associated with aberrant impulsive control in IA and may be a more reliable factor, unaffected by

age. Additionally, our results demonstrate increased connectivity in the posterior brain regions and decreased connectivity in the frontal-parietal and central executive networks during inhibitory control in IA. Further investigation is needed in this area to establish the underlying brain functional network during impulsive control in Internet addiction.

REFERENCES

[1] N. S. Nasser et al., "Cue-reactivity among young adults with problematic Instagram use in response to instagram-themed risky behavior cues: A pilot fMRI study," *Frontiers Psychol.*, vol. 11, p. 2386, Nov. 2020.

[2] H. Sharifat and S. Suppiah, "Electroencephalography-detected neurophysiology of internet addiction disorder and internet gaming disorder in adolescents—A review," *Med. J. Malaysia*, vol. 76, no. 3, pp. 401–413, May 2021.

[3] Y. Wang et al., "Identifying internet addiction and evaluating the efficacy of treatment based on functional connectivity density: A machine learning study," *Frontiers Neurosci.*, vol. 15, Jun. 2021, Art. no. 665578, doi: 10.3389/fnins.2021.665578.

[4] Y. Wang et al., "Abnormal functional connectivity in cognitive control network, default mode network, and visual attention network in internet addiction: A resting-state fMRI study," *Frontiers Neurol.*, vol. 10, p. 1006, Sep. 2019.

[5] C. Lai et al., "Neural correlate of internet use in patients undergoing psychological treatment for internet addiction," *J. Mental Health*, vol. 26, no. 3, pp. 276–282, May 2017, doi: 10.1080/09638237.2017.1294745.

[6] *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*, American Psychiatric Association, Richmond, VA, USA, 2013.

[7] N. Darvesh et al., "Exploring the prevalence of gaming disorder and internet gaming disorder: A rapid scoping review," *Systematic Rev.*, vol. 9, no. 1, pp. 1–10, Dec. 2020.

[8] M. Mihajlov and L. Vejmelka, "Internet addiction: A review of the first twenty years," *Psychiatria Danubina*, vol. 29, no. 3, pp. 260–272, Sep. 2017.

[9] D. Ayar et al., "The effect of adolescents' internet addiction on smartphone addiction," *J. Addictions Nursing*, vol. 28, no. 4, pp. 210–214, 2017.

[10] E. Wegmann, S. M. Müller, O. Turel, and M. Brand, "Interactions of impulsivity, general executive functions, and specific inhibitory control explain symptoms of social-networks-use disorder: An experimental study," *Sci. Rep.*, vol. 10, no. 1, p. 3866, Mar. 2020, doi: 10.1038/s41598-020-60819-4.

[11] N. S. Nasser, B. Ibrahim, H. Sharifat, A. A. Rashid, and S. Suppiah, "Incremental benefits of EEG informed fMRI in the study of disorders related to meso-corticolimbic dopamine pathway dysfunction: A systematic review of recent literature," *J. Clin. Neurosci.*, vol. 65, pp. 87–99, Jul. 2019, doi: 10.1016/j.jocn.2019.03.054.

[12] C. Kilian, K. L. Bröckel, R. Overmeyer, R. Dieterich, and T. Endrass, "Neural correlates of response inhibition and performance monitoring in binge watching," *Int. J. Psychophysiol.*, vol. 158, pp. 1–8, Dec. 2020.

[13] S. M. Müller, E. Wegmann, M. G. Arias, E. B. Brotóns, C. M. Giráldez, and M. Brand, "Deficits in executive functions but not in decision making under risk in individuals with problematic social-network use," *Comprehensive Psychiatry*, vol. 106, Apr. 2021, Art. no. 152228, doi: 10.1016/j.comppsych.2021.152228.

[14] T. Moretta and G. Buodo, "Response inhibition in problematic social network sites use: An ERP study," *Cognit., Affect., Behav. Neurosci.*, vol. 21, no. 4, pp. 868–880, Aug. 2021, doi: 10.3758/s13415-021-00879-9.

[15] S. Antons and M. Brand, "Trait and state impulsivity in males with tendency towards internet-pornography-use disorder," *Addictive Behaviors*, vol. 79, pp. 171–177, Apr. 2018, doi: 10.1016/j.addbeh.2017.12.029.

[16] Z. Zhou, G. Yuan, and J. Yao, "Cognitive biases toward internet game-related pictures and executive deficits in individuals with an internet game addiction," *PLoS ONE*, vol. 7, no. 11, Nov. 2012, Art. no. e48961, doi: 10.1371/journal.pone.0048961.

[17] M. Brand et al., "The interaction of Person-affect-cognition-execution (I-PACE) model for addictive behaviors: Update, generalization to addictive behaviors beyond internet-use disorders, and specification of the process character of addictive behaviors," *Neurosci. Biobehavioral Rev.*, vol. 104, pp. 1–10, Sep. 2019, doi: 10.1016/j.neubiorev.2019.06.032.

[18] K. Ioannidis et al., "Cognitive deficits in problematic internet use: Meta-analysis of 40 studies," *Brit. J. Psychiatry*, vol. 215, no. 5, pp. 639–646, Nov. 2019, doi: 10.1192/bjp.2019.3.

[19] X. Noël, M. Van Der Linden, and A. Bechara, "The neurocognitive mechanisms of decision-making, impulse control, and loss of willpower to resist drugs," *Psychiatry*, vol. 3, no. 5, p. 30, 2006.

[20] N. D. Volkow and M. Morales, "The brain on drugs: From reward to addiction," *Cell*, vol. 162, no. 4, pp. 712–725, Aug. 2015.

[21] E. Hahn, M. Reuter, F. M. Spinath, and C. Montag, "Internet addiction and its facets: The role of genetics and the relation to self-directedness," *Addictive Behaviors*, vol. 65, pp. 137–146, Feb. 2017.

[22] H. Sharifat, A. A. Rashid, and S. Suppiah, "Systematic review of the utility of functional MRI to investigate internet addiction disorder: Recent updates on resting state and task-based fMRI," *Malaysian J. Med. Health Sci.*, vol. 14, no. 1, pp. 21–33, 2018.

[23] C.-H. Ko et al., "Altered brain activation during response inhibition and error processing in subjects with internet gaming disorder: A functional magnetic imaging study," *Eur. Arch. Psychiatry Clin. Neurosci.*, vol. 264, no. 8, pp. 661–672, Dec. 2014.

[24] W.-N. Ding et al., "Trait impulsivity and impaired prefrontal impulse inhibition function in adolescents with internet gaming addiction revealed by a Go/No-go fMRI study," *Behav. Brain Functions*, vol. 10, no. 1, pp. 1–9, Dec. 2014.

[25] C. Chen et al., "Brain correlates of response inhibition in internet gaming disorder," *Psychiatry Clin. Neurosci.*, vol. 69, no. 4, pp. 201–209, Apr. 2015.

[26] J. Dieter et al., "The role of emotional inhibitory control in specific internet addiction—An fMRI study," *Behavioural Brain Res.*, vol. 324, pp. 1–14, May 2017.

[27] Z. Wang, H. Dong, X. Du, J.-T. Zhang, and G.-H. Dong, "Decreased effective connection from the parahippocampal gyrus to the prefrontal cortex in internet gaming disorder: A MVPA and spDCM study," *J. Behav. Addictions*, vol. 9, no. 1, pp. 105–115, Apr. 2020, doi: 10.1556/2006.2020.00012.

[28] J.-W. Chun et al., "Altered core networks of brain connectivity and personality traits in internet gaming disorder," *J. Behav. Addictions*, vol. 9, no. 2, pp. 298–311, Jun. 2020, doi: 10.1556/2006.2020.00014.

[29] B. Li et al., "Impaired frontal-basal ganglia connectivity in adolescents with internet addiction," *Sci. Rep.*, vol. 4, no. 1, pp. 1–8, May 2014.

[30] J. Ha, W. Park, S. I. Park, C.-H. Im, and L. Kim, "EEG response to game-craving according to personal preference for games," *Social Cognit. Affect. Neurosci.*, vol. 16, no. 9, pp. 995–1005, Oct. 2020, doi: 10.1093/scan/nsaa131.

[31] T. L. Burleigh, M. D. Griffiths, A. Sumich, G. Y. Wang, and D. J. Kuss, "Gaming disorder and internet addiction: A systematic review of resting-state EEG studies," *Addictive Behav.*, vol. 107, Aug. 2020, Art. no. 106429, doi: 10.1016/j.addbeh.2020.106429.

[32] Q. Gao, G. Jia, J. Zhao, and D. Zhang, "Inhibitory control in excessive social networking users: Evidence from an event-related potential-based Go-Nogo task," *Frontiers Psychol.*, vol. 10, p. 1810, Aug. 2019.

[33] G. Petit, C. Kornreich, X. Noël, P. Verbanck, and S. Campanella, "Alcohol-related context modulates performance of social drinkers in a visual Go/No-go task: A preliminary assessment of event-related potentials," *PLoS ONE*, vol. 7, no. 5, May 2012, Art. no. e37466.

[34] S. Park et al., "Longitudinal changes in neural connectivity in patients with internet gaming disorder: A resting-state EEG coherence study," *Frontiers Psychiatry*, vol. 9, p. 252, Jun. 2018, doi: 10.3389/fpsyt.2018.00252.

[35] S. M. Park et al., "Neural connectivity in internet gaming disorder and alcohol use disorder: A resting-state EEG coherence study," *Sci. Rep.*, vol. 7, no. 1, p. 1333, May 2017, doi: 10.1038/s41598-017-01419-7.

[36] S.-H. Chen, L.-J. Weng, Y.-J. Su, H.-M. Wu, and P.-F. Yang, "Development of a Chinese internet addiction scale and its psychometric study," *Chin. J. Psychol.*, vol. 45, no. 3, pp. 279–294, Sep. 2003.

[37] C. Ko, C. Yen, C. Yen, J. Yen, C. Chen, and S. Chen, "Screening for internet addiction: An empirical study on cut-off points for the Chen internet addiction scale," *Kaohsiung J. Med. Sci.*, vol. 21, no. 12, pp. 545–551, Dec. 2005.

[38] D. Wechsler, *Wechsler Intelligence Scale for Children*, 5th ed. Bloomington, MN, USA: Pearson, 2014.

[39] D. Wechsler, "The measurement and appraisal of adult intelligence," Tech. Rep., 1958.

[40] M. Balconi and R. Finocchiaro, "Deficit in rewarding mechanisms and prefrontal left/right cortical effect in vulnerability for internet addiction," *Acta Neuropsychiatrica*, vol. 28, no. 5, pp. 272–285, Oct. 2016, doi: 10.1017/neu.2016.9.

[41] M. Balconi, S. Campanella, and R. Finocchiaro, "Web addiction in the brain: Cortical oscillations, autonomic activity, and behavioral measures," *J. Behav. Addictions*, vol. 6, no. 3, pp. 334–344, Jul. 2017.

[42] S. Makeig, "Auditory event-related dynamics of the EEG spectrum and effects of exposure to tones," *Electroencephalogr. Clin. Neurophysiol.*, vol. 86, no. 4, pp. 283–293, Apr. 1993.

[43] A. Delorme and S. Makeig, "EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis," *J. Neurosci. Methods*, vol. 134, no. 1, pp. 9–21, Mar. 2004.

[44] T. R. Mullen et al., "Real-time neuroimaging and cognitive monitoring using wearable dry EEG," *IEEE Trans. Biomed. Eng.*, vol. 62, no. 11, pp. 2553–2567, Nov. 2015, doi: 10.1109/TBME.2015.2481482.

[45] J. A. Palmer, K. Kreutz-Delgado, and S. Makeig, "AMICA: An adaptive mixture of independent component analyzers with shared components," Swartz Center Computatonal Neursosci., Univ. California, San Diego, CA, USA, Tech. Rep., 2012.

[46] A. Delorme et al., "EEGLAB, SIFT, NFT, BCILAB, and ERICA: New tools for advanced EEG processing," *Comput. Intell. Neurosci.*, vol. 2011, pp. 1–12, Jan. 2011, doi: 10.1155/2011/130714.

[47] S. K. Loo et al., "Neural activation and connectivity during cued eye blinks in chronic tic disorders," *NeuroImage, Clin.*, vol. 24, Jan. 2019, Art. no. 101956, doi: 10.1016/j.nicl.2019.101956.

[48] J. Jurgiel, M. Miyakoshi, A. Dillon, J. Piacentini, S. Makeig, and S. K. Loo, "Inhibitory control in children with tic disorder: Aberrant fronto-parietal network activity and connectivity," *Brain Commun.*, vol. 3, no. 2, Apr. 2021, Art. no. fcab067, doi: 10.1093/braincomms/fcab067.

[49] D. Koshiyama et al., "Abnormal effective connectivity underlying auditory mismatch negativity impairments in schizophrenia," *Biol. Psychiatry: Cognit. Neurosci. Neuroimaging*, vol. 5, no. 11, pp. 1028–1039, Nov. 2020, doi: 10.1016/j.bpsc.2020.05.011.

[50] D. Koshiyama et al., "Neurophysiologic characterization of resting state connectivity abnormalities in schizophrenia patients," *Frontiers Psychiatry*, vol. 11, Nov. 2020, Art. no. 608154, doi: 10.3389/fpsyt.2020.608154.

[51] D. Koshiyama et al., "Auditory-based cognitive training drives short- and long-term plasticity in cortical networks in schizophrenia," *Schizophrenia Bull. Open*, vol. 1, no. 1, Jan. 2020, Art. no. sgaa065, doi: 10.1093/schizbullopen/sgaa065.

[52] M. Miyakoshi, L. Gehrke, K. Gramann, S. Makeig, and J. Iversen, "The AudioMaze: An EEG and motion capture study of human spatial navigation in sparse augmented reality," *Eur. J. Neurosci.*, vol. 54, no. 12, pp. 8283–8307, Dec. 2021, doi: 10.1111/ejn.15131.

[53] N. Tzourio-Mazoyer et al., "Automated anatomical labeling of activations in SPM using a macroscopic anatomical parcellation of the MNI MRI single-subject brain," *NeuroImage*, vol. 15, no. 1, pp. 273–289, Jan. 2002, doi: 10.1006/nimg.2001.0978.

[54] D. M. Groppe, T. P. Urbach, and M. Kutas, "Mass univariate analysis of event-related brain potentials/fields I: A critical tutorial review," *Psychophysiology*, vol. 48, no. 12, pp. 1711–1725, Dec. 2011.

[55] R. Dieterich, V. Wüllhorst, J. Berghäuser, R. Overmeyer, and T. Endrass, "Electrocortical correlates of impaired motor inhibition and outcome processing are related in high binge-watching," *Psychophysiology*, vol. 58, no. 6, Jun. 2021, Art. no. e13814, doi: 10.1111/psyp.13814.

[56] Y. Chen, H. Yu, and X. Gao, "Influences of emotional information on response inhibition in gaming disorder: Behavioral and ERP evidence from Go/Nogo task," *Int. J. Environ. Res. Public Health*, vol. 19, no. 23, p. 16264, Dec. 2022, doi: 10.3390/ijerph192316264.

[57] S. Kashif, S. Pandey, and Z. I. Warriach, "Neurophysiological markers of internet gaming disorder: A literature review of electroencephalography studies," *Cureus*, vol. 13, no. 9, Sep. 2021, Art. no. e17866, doi: 10.7759/cureus.17866.

[58] G. Dong, Q. Lu, H. Zhou, and X. Zhao, "Impulse inhibition in people with internet addiction disorder: Electrophysiological evidence from a Go/NoGo study," *Neurosci. Lett.*, vol. 485, no. 2, pp. 138–142, Nov. 2010, doi: 10.1016/j.neulet.2010.09.002.

[59] Z.-H. Zhou, G.-Z. Yuan, J.-J. Yao, C. Li, and Z.-H. Cheng, "An event-related potential investigation of deficient inhibitory control in individuals with pathological internet use," *Acta Neuropsychiatrica*, vol. 22, no. 5, pp. 228–236, Oct. 2010.

[60] L. Gao, J. Zhang, H. Xie, Y. Nie, Q. Zhao, and Z. Zhou, "Effect of the mobile phone-related background on inhibitory control of problematic mobile phone use: An event-related potentials study," *Addictive Behav.*, vol. 108, Sep. 2020, Art. no. 106363, doi: 10.1016/j.addbeh.2020.106363.

[61] J.-S. Choi et al., "Resting-state beta and gamma activity in internet addiction," *Int. J. Psychophysiol.*, vol. 89, no. 3, pp. 328–333, Sep. 2013.

[62] J. Lee et al., "Differential resting-state EEG patterns associated with comorbid depression in internet addiction," *Prog. Neuro-Psychopharmacol. Biol. Psychiatry*, vol. 50, pp. 21–26, Apr. 2014.

[63] A. Clarke, B. M. Roberts, and C. Ranganath, "Neural oscillations during conditional associative learning," *NeuroImage*, vol. 174, pp. 485–493, Jul. 2018, doi: 10.1016/j.neuroimage.2018.03.053.

[64] P. Missonnier et al., "EEG anomalies in adult ADHD subjects performing a working memory task," *Neuroscience*, vol. 241, pp. 135–146, Jun. 2013, doi: 10.1016/j.neuroscience.2013.03.011.

[65] A. V. Bocharov et al., "Associations of hyperactivity and inattention scores with theta and beta oscillatory dynamics of EEG in stop-signal task in healthy children 7–10 years old," *Biology*, vol. 10, no. 10, p. 946, Sep. 2021, doi: 10.3390/biology10100946.

[66] P. S. Cooper et al., "Frontal theta predicts specific cognitive control-induced behavioural changes beyond general reaction time slowing," *NeuroImage*, vol. 189, pp. 130–140, Apr. 2019, doi: 10.1016/j.neuroimage.2019.01.022.

[67] A. Riesel, N. Kathmann, and T. Endrass, "Overactive performance monitoring in obsessive–compulsive disorder is independent of symptom expression," *Eur. Arch. Psychiatry Clin. Neurosci.*, vol. 264, no. 8, pp. 707–717, Dec. 2014, doi: 10.1007/s00406-014-0499-3.

[68] P. S. Cooper and M. E. Hughes, "Impaired theta and alpha oscillations underlying stopsignal response inhibition deficits in schizophrenia," *Schizophrenia Res.*, vol. 193, pp. 474–476, Mar. 2018, doi: 10.1016/j.schres.2017.08.002.

[69] F. Rostami, A. Esteki, and A. Sarabi-Jamab, "Investigating the interference control in internet addiction disorder evidence from brain oscillatory activity associated with stroop task," Tech. Rep., 2023.

[70] S. Mohammadi, A. Jahanshahi, M. A. Salehi, R. Darvishi, H. Seyedmirzaei, and L. P. Luna, "White matter microstructural changes in internet addiction disorder: A systematic review of diffusion tensor imaging studies," *Addictive Behav.*, vol. 143, Aug. 2023, Art. no. 107690, doi: 10.1016/j.addbeh.2023.107690.

[71] S.-B. Hong et al., "Decreased functional brain connectivity in adolescents with internet addiction," *PLoS ONE*, vol. 8, no. 2, Feb. 2013, Art. no. e57831, doi: 10.1371/journal.pone.0057831.

[72] Y. Wang et al., "Decreased prefrontal lobe interhemispheric functional connectivity in adolescents with internet gaming disorder: A primary study using resting-state fMRI," *PLoS ONE*, vol. 10, no. 3, Mar. 2015, Art. no. e0118733, doi: 10.1371/journal.pone.0118733.

[73] S. Kuriki et al., "Neurobiological influence of comorbid conditions in young patients diagnosed with gaming disorder: A whole-brain functional connectivity study based on a data driven method," *PLoS ONE*, vol. 15, no. 5, May 2020, Art. no. e0233780, doi: 10.1371/journal.pone.0233780.

[74] N. Zeng et al., "Gender-related differences in frontal-parietal modular segregation and altered effective connectivity in internet gaming disorder," *J. Behav. Addictions*, vol. 10, no. 1, pp. 123–134, Apr. 2021, doi: 10.1556/2006.2021.00015.

[75] A. U. Patil, D. Madathil, and C. Huang, "Age-related and individual variations in altered prefrontal and cerebellar connectivity associated with the tendency of developing internet addiction," *Hum. Brain Mapping*, vol. 42, no. 14, pp. 4525–4537, Oct. 2021, doi: 10.1002/hbm.25562.

[76] W.-N. Ding et al., "Trait impulsivity and impaired prefrontal impulse inhibition function in adolescents with internet gaming addiction revealed by a Go/No-go fMRI study," *Behav. Brain Functions*, vol. 10, no. 1, p. 20, Dec. 2014, doi: 10.1186/1744-9081-10-20.

[77] J. Youh et al., "Comparison of electroencephalography (EEG) coherence between major depressive disorder (MDD) without comorbidity and MDD comorbid with internet gaming disorder," *J. Korean Med. Sci.*, vol. 32, no. 7, p. 1160, Jul. 2017, doi: 10.3346/jkms.2017.32.7.1160.

[78] J.-Y. Lee, J.-S. Choi, and J. Kwon, "Neurophysiological mechanisms of resilience as a protective factor in patients with internet gaming disorder: A resting-state EEG coherence study," *J. Clin. Med.*, vol. 8, no. 1, p. 49, Jan. 2019, doi: 10.3390/jcm8010049.

[79] Y. Sun, H. Wang, and S. Bo, "Altered topological connectivity of internet addiction in resting-state EEG through network analysis," *Addictive Behav.*, vol. 95, pp. 49–57, Aug. 2019, doi: 10.1016/j.addbeh.2019.02.015.

[80] H. Wang, Y. Sun, F. Lan, and Y. Liu, "Altered brain network topology related to working memory in internet addiction," *J. Behav. Addictions*, vol. 9, no. 2, pp. 325–338, Jun. 2020, doi: 10.1556/2006.2020.00020.