Received: 24 October 2020 | Revised: 4 May 2021 | Accepted: 8 May 2021

DOI: 10.1111/jsr.13398

Journal of Sleep Research

RESEARCH ARTICLE

Altered brain activity related to inhibitory processing in youth with insomnia

Jiefan Ling[1] | Xuanyi Lin[2] | Xiao Li[1] | Ngan Yin Chan[3] | Jihui Zhang[4] | Yun Kwok Wing[3] | Xiaoqing Hu[2,5] | Shirley Xin Li[1,5]

[1]Sleep Research Clinic and Laboratory, Department of Psychology, The University of Hong Kong, Hong Kong, Hong Kong

[2]The Social and Cognitive Neuroscience Lab, Department of Psychology, The University of Hong Kong, Hong Kong, Hong Kong

[3]Department of Psychiatry, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, Hong Kong

[4]Guangdong Mental Health Center, Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, Guangzhou, China

[5]The State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong, Hong Kong, Hong Kong

Correspondence
Shirley Xin Li and Xiaoqing Hu, Department of Psychology, The University of Hong Kong, Pokfulam Road, Pokfulam, Hong Kong, Hong Kong.
Email: shirley.li@hku.hk; xiaoqinghu@hku.hk

Funding information
University Research Committee, The University of Hong Kong, Grant/Award Number: 27613017; General Research Fund, Grant/Award Number: 17601318; National Natural Science Foundation of China, Grant/Award Number: 31922089

Summary

Insomnia has been shown to negatively affect one's cognitive functioning. While there has been some evidence suggesting sleep disruption in relation to impaired inhibitory control, a major component of executive function, little is known about the underlying neural processing in insomnia. The current study aimed to examine the differences in the behavioral responses and electroencephalography (EEG) correlates of inhibitory control between youths with insomnia and healthy sleepers. Twenty-eight participants with insomnia disorder and 31 healthy sleeper controls aged between 15 and 25 completed the study. Electroencephalographic activity was recorded during the Cued Go/NoGo (CGNG) task, a task assessing inhibitory control. Although insomnia group exhibited comparable behavioral performance to the healthy sleeper group, they showed impaired attention preparation, as displayed by a smaller contingent negative variation (CNV) component (F = 4.10, p = 0.048) after cue onset; and demonstrated impaired inhibitory control, as evidenced by smaller N2 and theta power on 200–350 ms (MANCOVA multivariate Group effect, F = 5.85, p < 0.001). The results suggested that youths with insomnia demonstrated altered brain activity during inhibitory control, despite their comparable behavioral performance. Given that impaired inhibitory control is often implicated in psychopathology, future studies with a longitudinal design are needed to further explore the long-term impacts and trajectory of altered inhibitory control in youths with insomnia.

KEYWORDS
adolescents, electroencephalography, inhibitory control, insomnia, sleep, youth

1 | INTRODUCTION

Insomnia, presented as problems with initiating or maintaining sleep, or early morning awakening, affects about 33.7% to up to 69% of the youth population, depending on the defining criteria and age of the study samples (Blank et al., 2015; Chung, Kan, & Yeung, 2014; Sing & Wong, 2011). Insomnia in youth entails an increased risk of developing a wide range of negative outcomes, including interpersonal problems, poor perceived physical health, somatic health problems, as well as depressive and anxiety symptoms (Blank et al., 2015). Meanwhile, youth is a developmental period that is especially vulnerable to impulsivity and poor impulse control due to the immature development of the forebrain circuits during adolescence (Geier & Luna, 2009). In particular, insomnia in youth has been linked to the behavioral problems associated with impaired inhibitory control, such as substance abuse, self-harm and suicidal risk (Hysing, Sivertsen, Stormark, & O'Connor, 2015).

Jiefan Ling and Xuanyi Lin contributed equally to this work.

Inhibitory control is a key feature of executive functions that helps individuals to resist impulses, habitual responses, and ultimately impulsivity (Diamond, 2012). Previous studies that assessed the performance on inhibition task in individuals with insomnia were mostly conducted using adult samples and have yielded mixed findings, with some reporting a significant behavioral impairment (e.g., Zhao et al., 2018), while others showing no difference (e.g., Muscarella, Mairesse, Hughes, & Neu, 2019). On the other hand, functional neuroimaging studies found that prefrontal cortex, one of the key brain regions involved in inhibitory control (Swick, Ashley, & Turken, 2011), showed reduced activities during the wake time positron emission tomography scan (Nofzinger et al., 2004) and lower activation during category and letter fluency task (Altena et al., 2008) in patients with insomnia as compared with healthy sleepers.

Recent research has turned to examine the EEG correlates of inhibitory control to better understand its neurocognitive basis. Event-related potentials (ERPs) studies have identified the N2 component and the P3 component during the performance on the inhibition task, such as Go/NoGo task, to reflect the neuronal processes in relation to inhibitory control (Bokura, Yamaguchi, & Kobayashi, 2001). One study found that although adults with insomnia disorder (N = 18, aged 20–53, Mean = 32) showed comparable performance as the healthy sleepers (N = 15, aged 21–53, Mean = 32) on the AX-continuous performance task assessing cognitive control, they showed altered neural activities of inhibitory functioning as indicated by reduced amplitudes of the P3 component and the contingent negative variation (CNV) component (Muscarella et al., 2019). Another study that assessed ERP during an auditory stop-signal task found that adults with insomnia disorder (N = 12, aged 18–65, Mean = 49) showed slower reaction time and reduced P3 amplitude as compared with healthy sleepers (N = 13, aged 18–65, Mean = 41) (Zhao et al., 2018). Collectively, these studies have provided preliminary evidence showing electrophysiological correlates of inhibitory deficits in insomnia. However, both studies were constrained by relatively small sample sizes and a wide age range of the study participants.

Meanwhile, some other studies have examined event-related dynamics of inhibitory control based on the time-frequency domain using event-related spectral perturbation. Theta (e.g. 4–7 Hz) oscillations have been increasingly recognized as important correlates of cognitive flexibility, including goal updating (Cooper, Wong, McKewen, Michie, & Karayanidis, 2017) and selective attention (Phillips, Vinck, Everling, & Womelsdorf, 2014). A previous study showed that adults with primary insomnia, as compared with their aged-matched controls, had decreased theta band power in the prefrontal region during a wake period 2-min resting EEG (Wolyńczyk-Gmaj & Szelenberger, 2011). Another study found that, as compared to the healthy sleepers (N = 10, Mean age = 18.8), poor sleepers (defined by self-reported sleep complaints and a score of five or above on the Pittsburgh Sleep Quality Index; N = 10, Mean age = 18.8) showed lower theta suppression during the rest condition with eyes closed, as compared to the sensory attentive condition, which indicated a down-regulation of attention in the poor sleeper group (Buckelew, DeGood, Roberts, Butkovic, & MacKewn, 2009). Researchers have also found increased theta power in multiple interference situations including during NoGo of Go/NoGo task (Nigbur et al., 2011). Although previous findings suggested a link between sleep disruption and altered theta oscillations, there has been no study conducted to examine the event-related theta dynamics during inhibition tasks in individuals with insomnia.

Given that insomnia is often linked to the behavioral problems with inhibitory control in youths, there is a need to further delineate the underlying electrophysiological mechanisms. The current study aimed to examine the EEG correlates during a Cued Go/NoGo task assessing inhibitory control in youths with insomnia. We hypothesized that as compared with the healthy sleepers, youths with insomnia would show impaired inhibitory processing on both behavioral as well as electrophysiological levels, such as along time domain (e.g. reduced ERP components: cue related CNV, and target related N2 and P3) and time-frequency domain (e.g. reduced event-related theta band power) EEG correlates of inhibitory control.

2 | METHODS

2.1 | Participants

Two groups of participants were separately recruited via mass emails, online advertising, posters and flyers in the local community (Figure S1.). Chinese youths aged between 15 and 25 with insomnia disorder and their age- and gender-matched healthy sleeper controls were recruited into the present study. The age range of 15–25 was selected to cover a wider developmental range, derived from the definition of youth by World Health Organization (World Health Organization, 1986). For the recruitment of insomnia cases, interested participants responded to an advertisement that invited individuals who had insomnia complaints (i.e. difficulty in initiating sleep, difficulty maintaining sleep, or early morning awakening) to sign up and join this study. For the recruitment of healthy sleeper group, interested participants responded to another advertisement that aimed to recruit healthy sleepers without any mental health problem. All the interested participants were asked to register for the study online, followed by completing a clinical interview for those potential eligible participants to ascertain their eligibility. For the insomnia group, the inclusion criteria included: (1) having a score on Insomnia Severity Index (ISI) ≥ 9, the suggested cut-off score for clinical insomnia symptoms in adolescents (Chung, Kan, & Yeung, 2011), and (2) meeting the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) diagnostic criteria of insomnia disorder (American Psychiatric Association, 2013). For the healthy sleeper group, the inclusion criteria included having no sleep or neuropsychiatric disorder as confirmed by the screening interview. Exclusion criteria for both groups included: (1) night-shift workers, (2) a current diagnosis of any neuropsychiatric or sleep disorder (other than insomnia for the insomnia group) as confirmed by the clinical interview, (3) current use of medications or having any prominent medical condition affecting sleep or cognition, (4) impaired vision and hearing deficit.

2.2 | Study procedures

To ascertain one's eligibility for this study, potential participants underwent a screening session using two validated semi-structured clinical interviews in Chinese version: Diagnostic Interview for Sleep Patterns and Disorder (DISP) to screen for major sleep disorders such as narcolepsy, restless leg syndrome, obstructive sleep apnea, and parasomnias (Yu, Zhang, & Wing, 2015), and Mini-International Neuropsychiatric Interview (MINI)/Mini International Neuropsychiatric Interview for Children and Adolescents (MINI-KID) for those aged below 18 (Si et al., 2009) to screen for psychiatric disorders, such as major depression, anxiety disorders, and psychotic disorders (Amorim, 2000). Eligible participants completed a battery of self-report questionnaires, and were instructed to complete a daily sleep diary while wearing a wrist actigraphy (Actiwatch Spectrum PRO, Philips—Respironics) to measure their sleep at home for seven consecutive days before the EEG experiment. To eliminate time-of-the-day effect, all the EEG experiments were scheduled from around 10:30 a.m. to 12:30 p.m. Four participants were rescheduled from 2:30 p.m. or 3:30 p.m. due to either the equipment or the participant being unavailable in the morning (n = 3 in the insomnia group) and they were excluded from the EEG analyses. Written informed consent forms were signed by all the participants, while parent/guardian consent forms were additionally collected from those aged below 18. The study protocol was approved by the Institutional Ethics Committee (Ref. EA1804003). Participants who completed the study were given HKD$400 cash as remuneration. Data collection was conducted during Dec 2018 to May 2019.

2.3 | Measures

2.3.1 | Subjective sleep and mood measures

Insomnia Severity Index (ISI) (Morin, Belleville, Bélanger, & Ivers, 2011) is a 7-item scale to measure different symptom dimensions of insomnia (e.g. difficulty with sleep onset, sleep maintenance, and early morning awakening). The Chinese version of ISI has been shown to be a reliable and valid instrument for measuring insomnia in Chinese adolescents (Chung et al., 2011). An ISI score ≥ 9 was found to be a cut-off score suggestive of the presence of clinical insomnia in adolescents (Chung et al., 2011). Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) is a 19-item scale to assess self-reported sleep quality, which has been validated in the Chinese population (Shochat et al., 2014). Chronotype preference was measured by the Chinese version of the Reduced Morningness/Eveningness Questionnaire (rMEQ; Carciofo, Du, Song, Qi, & Zhang, 2012). The Chinese version of the Hospital Anxiety and Depression Scale (HADS) (Leung, Wing, Kwong, Lo, & Shum, 1999) consists of a 7-item anxiety subscale (HADS-A) and a 7-item depression subscale (HADS-D). HADS has been validated for assessing anxiety and depression in the general population, showing good internal consistency and external validity (e.g. Cronbach's alpha for HADS-A = 0.83, ranging from 0.68 to 0.93; Cronbach's alpha for HADS-D = 0.82, ranging from 0.67 to 0.90; sensitivity and specificity of both subscales were in the range of 0.70–0.90; Bjelland, Dahl, Haug, & Neckelmann, 2002).

2.3.2 | Objective sleep measures

Actigraphy (Philips Respironics) was used to objectively measure sleep. Actigraph was configured to record in 1-min epochs. The analysis in Actiware software was based on sleep diary and/or event markers. Sleep parameters generated for the analysis in this study included total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), and sleep efficiency (SE).

2.3.3 | Measure of impulsivity

The Chinese version of Barratt Impulsiveness Scale (BIS-11), which has been validated in the Chinese adolescent sample (Shuqiao et al., 2007), is a 30-item scale designed to assess trait impulsivity. It consists of three factors related to impulsivity: attention (inattention and cognitive instability), motor (impulsive behaviors and inconsistent lifestyle), and non-planning (self-control and cognitive complexity).

2.4 | Experimental paradigm: Cued Go/NoGo task

During the experiment, participants were comfortably seated in a sound attenuated room, while wearing the 64-channel waveguard original cap (ANT Neuro) for electroencephalography (EEG) recording. The Cued Go/NoGo (Hong, Wang, Sun, Li, & Tong, 2017) experimental paradigm (Figure 1) is a spatial-cueing Go/NoGo task designed to probe top-down selective attention and response inhibition. The task was implemented in E-Prime (Version: 2.0). Participants were asked to keep a central fixation throughout the task. Each trial began with a 200-ms cue, i.e., a left or right pointing arrow instructing the participants to attend to the cued location and ignore the uncued location. To avoid habitual expectation that may potentially influence target processing, following a cue-target interval (CTI, between cue offset to target onset) randomly selected as 1000/1100/1200 ms, a target (either the plus sign '+' as Go or the letter 'x' as NoGo), was presented for 200 ms at either the cued or uncued location. Participants were required to respond to the Go target at the cued location (Attend-Go) as quickly and accurately as possible by pressing the spacebar with the right index finger, and to inhibit from responding to the NoGo target at the attended location (Attend-NoGo). Participants were also instructed to refrain from responding to both the Go target (Ignore-Go) and the NoGo target (Ignore-NoGo) at the ignored location. The type of the cues, and the location and type of the targets were all varied randomly with 50% probability, which has been suggested to eliminate bias towards Go stimuli and minimize the confounding

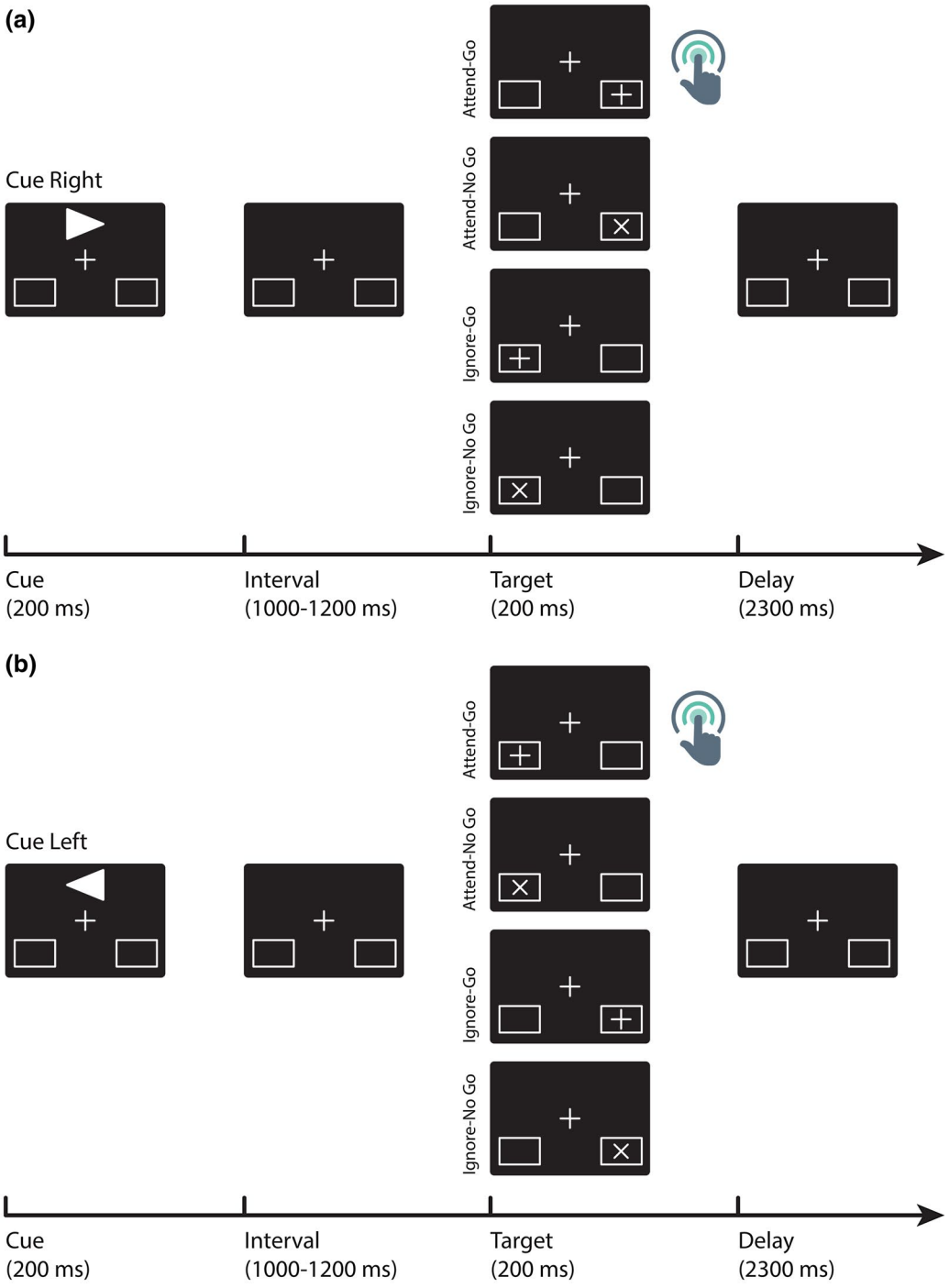


FIGURE 1 Experimental paradigm for the Cued Go/No-go task. The Cued Go/NoGo task is a spatial-cueing task with equal probability of Go and NoGo stimuli. In the beginning of each trial, a left or right pointing arrow is presented for 200 ms as a cue, instructing the participants to attend to the cued location and ignore the other. Following interval varying from 1000 to 1200 ms from cue offset, a target is presented for 200 ms. Participants should respond to the plus sign '+' (Go target) at the cued location (Attend-Go), whilst inhibit from responding to the letter 'x' (NoGo target) at the cued location (Attend-NoGo). For any target appearing at the ignored location, participants should not respond to either the Go target (Ignore-Go) or the NoGo target (Ignore-NoGo). At the end of each trial, there is a fixed delay of 2300 ms

'oddball effect' associated with a low frequency of the NoGo stimuli (Masharipov, Kireev, Korotkov, & Medvedev, 2019). The inter-trial interval was set to be 2300 ms. For Attend-Go targets, participants were instructed to respond as soon as possible, and only responses made within 1000 ms after the target offset were included as valid trials. Each testing block contained 72 trials (2 cue types × 3 CTIs × 2 target locations × 2 targets × 3 repeats) and lasted for about 5 min, with self-determined breaks between blocks. Each participant went through a practice block of 16 trials before completing 7 formal blocks. Behavioral outcomes of the

task included: the percentage of errors in all trials (total error %), the percentage of errors in Attend-NoGo trials (commission errors, indicating inhibition error %), and the reaction time in Attend-Go trials with accurate responses (reaction time).

2.5 | Electrophysiological (EEG) data processing

2.5.1 | EEG recording

Continuous EEG signals were recorded using the eego mylab system™ (ANT Neuro) and a 64-channel waveguard original cap™ (ANT Neuro). The signals were amplified and digitized at 500 Hz sampling rate. Impedance of each electrode was maintained below 10 kΩ during the recording.

2.5.2 | Preprocessing

EEG preprocessing was performed in Matlab-based (Version: R2016b) EEGlab (Version: 14.1.1b) and ERPlab (Version: 7.0) toolboxes. Raw EEG data were first filtered using a band-pass filter between 0.1 and 40 Hz and a notch filter at 50 Hz, and were re-referenced to common average. Ocular artifacts were corrected by independent component analysis based on the Infomax (runica) algorithm. Continuous EEG data were then segmented into −200 to 800 ms epochs relative to target onset for N2 and P3 analyses, and into −200 to 1200 ms epochs relative to cue onset for contingent negative variation (CNV) analysis. Longer epochs (−1000–2000 ms) relative to target onset were used for time-frequency analyses. Artifact detection using ERPlab were performed for all the EEG epochs to examine (1) maximally allowed amplitude difference (threshold: 150 μV) for all channels within a moving window (width: 200 ms; step: 50 ms) using the peak-to-peak function; (2) maximally allowed absolute amplitude (threshold: ±100 μV) for all the channels throughout the whole epoch. All the EEG epochs were then visually inspected to ensure the quality before subsequent analysis. Across participants, the averaged acceptance rate of EEG epochs among all trials was 83.33% (SD = 14.85%) for cre-related ERP, 85.35% (SD = 12.13%) for target-related ERP, and 73.89% (SD = 20.14%) for target-related time-frequency analysis.

2.5.3 | Time domain analysis

To analyze the contingent negative variation (CNV), cue-related epochs were grouped and averaged with the 200 ms pre-cue interval as baseline. We focused cue-related CNV at the FPz electrode, given the topographical distributions and CNV's typical frontal distributions (Figure 2) Amplitudes of CNV were then identified as the mean within 800–1200 ms following cue onset, consistent with a previous study using the same experimental paradigm (Hong et al., 2017). The averaged cue-related ERP waveforms from other midline frontal-central electrodes (Fz, Cz, FCz) were presented in the supplemental figure (Figure S2). To analyze the N2 and P3 components, target-related epochs were grouped and averaged according to the type of targets of interest (Attend-NoGo and Attend-Go) with the 200 ms pre-target interval as baseline, yielding the target-related ERP for each electrode and participant. Given N2 and P3's different topographical distribution, target-related ERPs were averaged across frontal-central electrodes (Fz, Cz, FCz) for N2 and central-parietal electrodes (CP1, CP2, Pz, P1, P2) for P3, respectively. The individual peak latencies of the N2/P3 were identified as the time points of the most negative/positive voltages during pre-defined time windows, namely 200–350 ms for the N2 and 300–600 ms for the P3. We identified the grand averaged ERP peak latencies as follows: 288 ms for N2-Attend-Go; 278 ms for N2-Attend-NoGo; and 422 ms for P3-Attend-Go and 312 ms for P3-Attend-NoGo. Amplitudes of the N2 and P3 were then calculated as the mean amplitude of the 50 ms time window, centering around the peak latency for each type of targets, respectively.

2.5.4 | Time-frequency domain analysis

To analyze the event-related theta band activities, the epochs from −1000 ms to 2000 ms were first transformed using the Morlet wavelet-based analysis with 3 cycles per wavelet generating 80 log-spaced frequencies from 4 Hz to 40 Hz with a scaling factor of 0.5 (using minimal cycle of 3 at frequency 4, and maximal cycle of 10 at max frequency 40). Baseline normalization using decibel transform was performed using a baseline period from −500 to −200 ms relative to target onset. Target-related 4–7 Hz theta band power (hereafter Theta) were extracted from the N2 time window (200–350 ms) over the same frontal-central electrodes (Fz, FCz, Cz).

2.6 | Statistical analysis

A priori power analysis ('pwr' package in R, Champely et al., 2018, R Core Team, 2014) suggested that to detect an estimated effect size of 0.75 (Muscarella et al., 2019), 28 participants per group would be required to achieve a power of 0.80 while controlling type I error as 0.05 in the two-group comparison. A one-way ANCOVA was conducted for CNV, with group (Insomnia vs. Healthy Sleeper) as dependent variable, controlling for age and mood (HADS-A and HADS-D). N2, P3 and Theta were submitted to one MANCOVA with group (Insomnia vs. Healthy Sleeper) and condition (Attend-NoGo vs. Attend-Go, within-subject) as independent factors, with age and mood (HADS-A and HADS-D) as covariates. Post-hoc analyses following significant multivariate and univariate effects were performed in *t*-tests (independent samples *t*-tests for Healthy Sleeper vs. Insomnia group effects and paired samples *t*-tests for Go vs. NoGo condition effects). Pearson correlation analyses were used

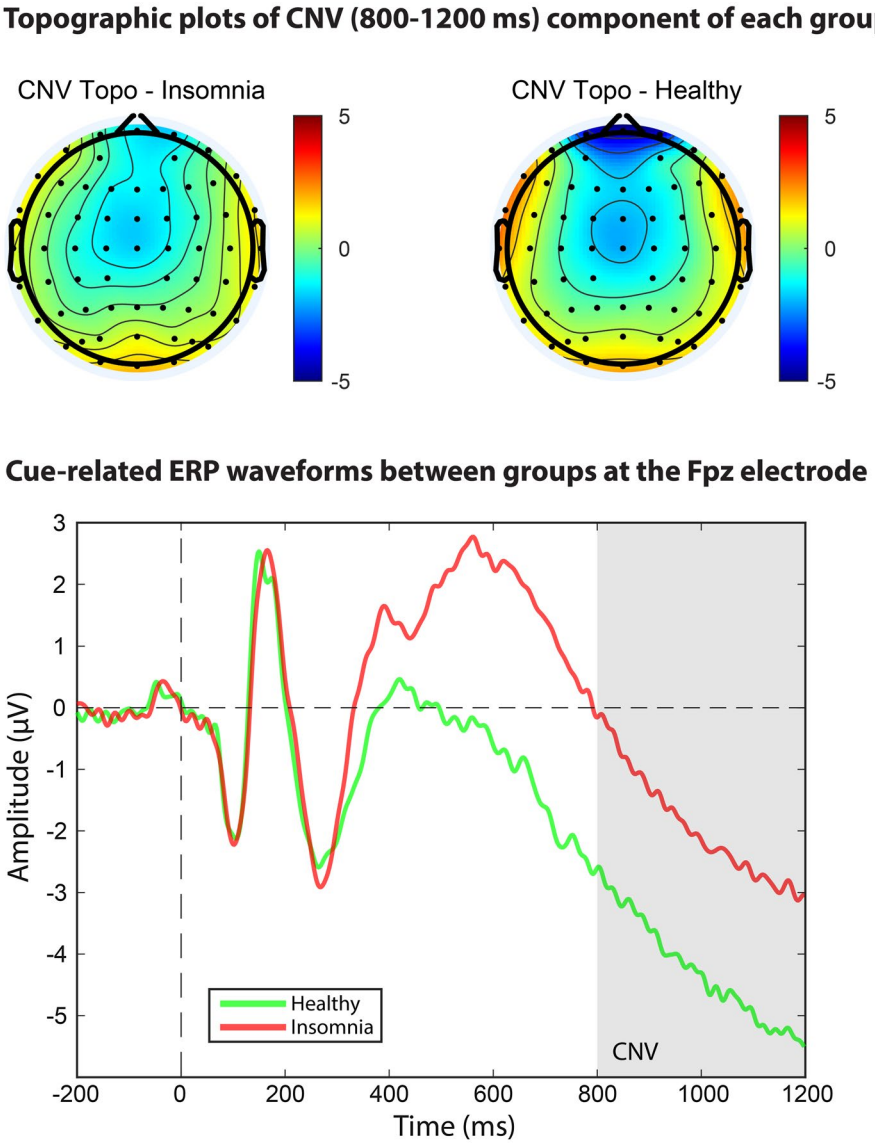


FIGURE 2 Cue-related ERPs. (a) Topographic plots of CNV (800–1200 ms) component. (b) Cue-related ERP waveforms between groups at the Fpz electrode

to examine the relationship between ERP components (CNV, NoGo N2, and NoGo P3) and the measures of sleep, mood, and impulsivity among all the participants. Statistical significance was set at $p < .05$ and all the statistical analyses were conducted using jamovi (The jamovi project, 2021).

3 | RESULTS

3.1 | Sample characteristics

A hundred and nine participants registered for this study (Figure S1). Sixty-seven potentially eligible participants aged 15–25 were invited to attend a screening session; five were excluded following the screening session ($N = 1$ not meeting insomnia diagnosis, $N = 1$ with delayed sleep phase, $N = 2$ with major depression, $N = 1$ with obsessive compulsory disorder). Three participants dropped out before the completion of the study. All the participants were students from local secondary schools and universities. Finally 28 participants (15 females, mean age 20.5 years) in the insomnia group and 31 participants (18 females, mean age 21.2 years) in the healthy sleeper group were recruited and completed the Go/NoGo task. In the final analysis, we further excluded the four participants who started late in the afternoon (3 in the insomnia group and 1 in healthy sleeper group) to control for time-of-day effects on EEG activities (Aeschbach et al., 1999). Note that all statistical results patterns and conclusions remained the same when using late-start as a covariate. There were no significant differences in age, gender, and chronotype preference between the two groups (insomnia group $N = 25$ vs. healthy sleeper group $N = 30$; See Table 1). As compared with healthy sleepers, participants in the insomnia group had significantly more sleep disturbances (as assessed by PSQI, $p < 0.001$), more severe insomnia symptoms (as assessed by ISI, $p < 0.001$), lower sleep efficiency (as assessed

TABLE 1 Between-group comparisons on demographic characteristics, sleep, mood disturbances, and impulsivity measures

	Insomnia group	Healthy sleeper group	*t*	
	N = 25	*N* = 30	(χ2 for gender)	*p*
Demographics				
Female, *N* (%)	14 (56.00)	17 (56.67)	0.002	0.960
Age, M (SD)	20.88 (2.09)	21.17 (2.44)	−0.46	0.645
Self-report sleep measures, M (SD)				
PSQI	8.04 (2.37)	3.67 (1.24)	8.77	<0.001***
ISI	14.08 (3.51)	3.70 (2.40)	12.98	<0.001***
rMEQ	12.90 (3.86)	13.37 (3.44)	−0.47	0.638
Actigraphy sleep measures, M (SD), min				
TIB	490.40 (47.98)	472.90 (44.79)	1.40	0.168
TST	470.79 (43.77)	459.40 (46.14)	0.93	0.355
SOL	12.62 (16.24)	7.16 (5.30)	1.74	0.088^
SE	77.44 (10.72)	82.58 (7.31)	−2.11	0.040*
WASO	92.89 (49.08)	70.85 (38.45)	1.87	0.067^
Mood disturbances, M (SD)				
HADS-depression	7.04 (6.99)	2.67 (2.47)	3.20	0.002**
HADS-anxiety	8.52 (3.96)	4.83 (3.02)	3.92	<0.001***
Impulsivity measure, M (SD)				
BIS	65.28 (6.74)	60.60 (8.35)	2.26	0.028*
BIS-attention	18.76 (3.32)	15.53 (2.83)	3.89	<0.001***
BIS-MOTOR	21.08 (3.93)	20.83 (3.33)	0.25	0.802
BIS-non-planning	25.44 (2.80)	24.23 (4.99)	1.08	0.287

Abbreviations: BIS: Barratt Impulsiveness Scale; HADS: Hospital Anxiety and Depression Scale; ISI: Insomnia Severity Index; PSQI: Pittsburgh Sleep Quality Index; rMEQ: Reduced Morningness -Eveningness Questionnaire; SE: Sleep efficiency; SOL: Sleep onset latency; TIB: Time in bed; TST: Total sleep time; WASO: Wake after sleep onset.

^$p < 0.1$.

*$p < 0.05$.

**$p < 0.01$.

***$p < 0.001$.

TABLE 2 Between-group comparisons on the behavioral outcomes in the Cued Go/No-go task

	Insomnia group	Healthy sleeper group		
	Mean (SD)	Mean (SD)	*t*	*p*
Inhibition error %	1.87 (2.71)	2.57 (4.49)	−0.68	0.500
Total error %	9.43 (20.06)	4.93 (6.10)	1.15	0.255
Reaction time (ms)	483.32 (110.37)	447.15 (84.55)	1.36	0.179

by actigraphy, $p < 0.05$), and marginally longer wake after sleep onset (as assessed by actigraphy, $p = 0.067$). Insomnia group also scored higher on the measures of mood disturbances (as assessed by HADS-D, $p < 0.01$; and HADS-A, $p < 0.001$) than healthy sleeper group. Participants with insomnia disorder also had higher trait impulsivity (as assessed by BIS $p < 0.05$), especially on the attention subscale ($p < 0.001$).

3.2 | Behavioral performance

There were no significant differences between insomnia group and healthy sleeper group in inhibition error percentage, total error percentage, and reaction time in the Cued Go/NoGo task (See Table 2).

3.3 | Time domain results

Grand averaged cue related ERPs are shown in Figure 2. One-way ANCOVA showed a significant main effect of group on CNV, $F = 4.099$, $p = 0.048$, $\eta^2 = 0.07$. As shown in Figure 2., healthy sleeper group had significantly greater CNV amplitude (Mean = −4.35, SD = 4.02) than the insomnia group (Mean = −1.52, SD = 2.53).

Grand averaged target related ERPs for Attend-Go and Attend-NoGo conditions are plotted in Figure 3. We conducted one MANCOVA on N2, P3 and Theta, with Condition (Go vs. NoGo,

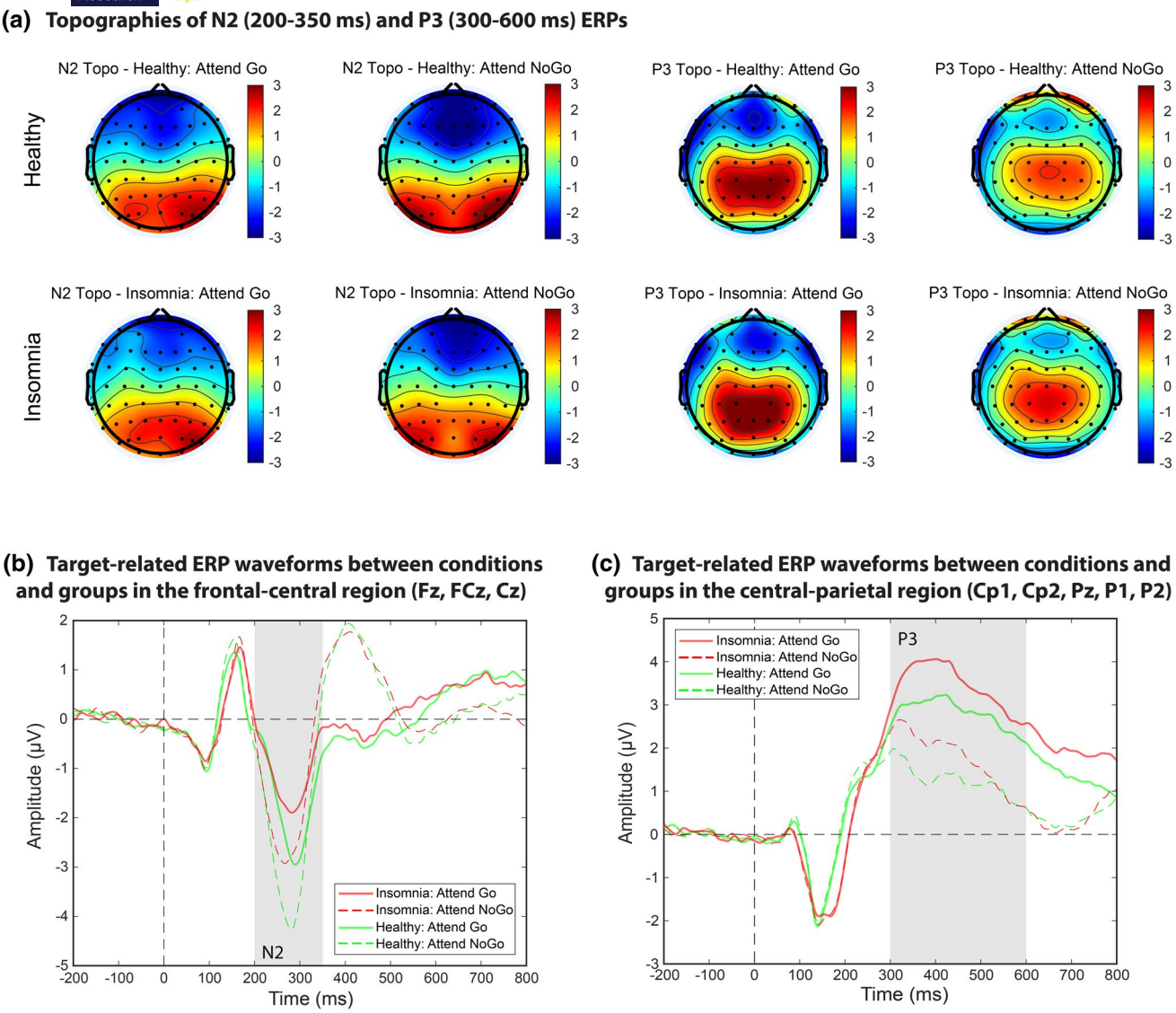


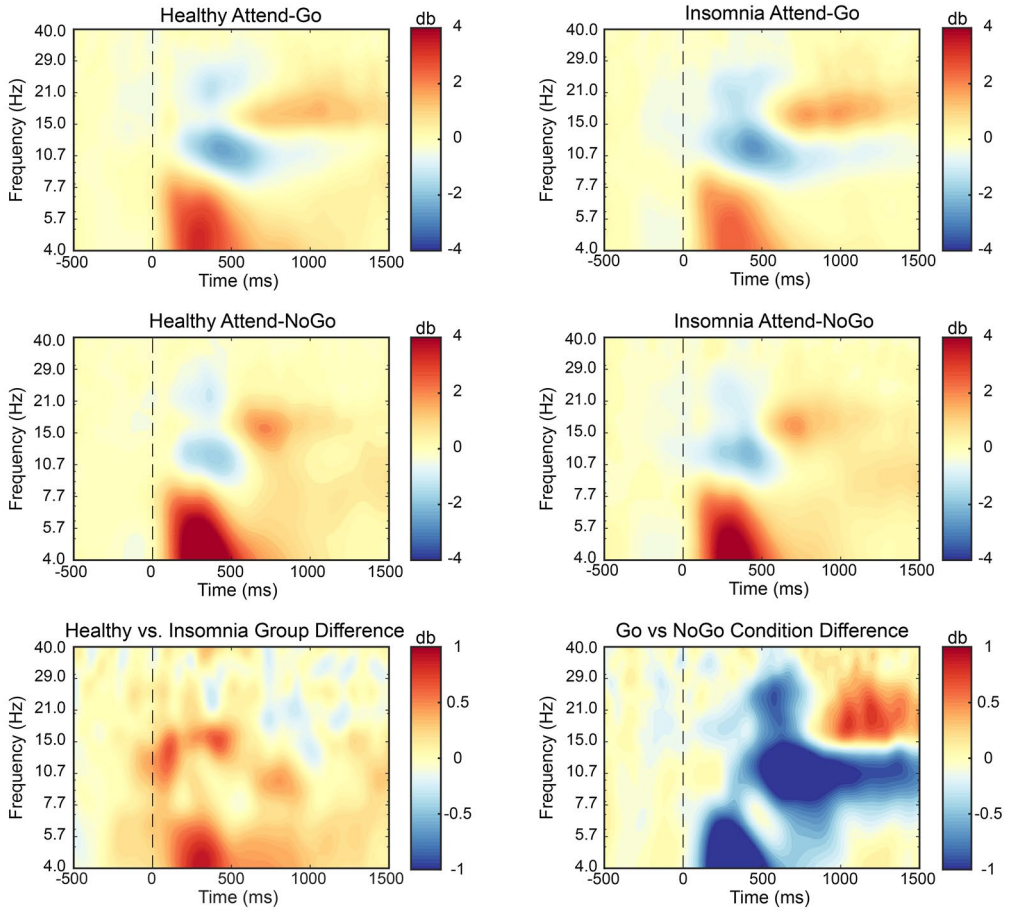
FIGURE 3 Target-related ERPs between conditions and groups (a) Topographies of N2 (200–350 ms) and P3 (300–600 ms) ERPs. (b) Target-related ERP waveforms between conditions and groups in the frontal-central region. (c) Target-related ERP waveforms between conditions and groups in the central-parietal region

within-subject variable) and Group (Insomnia vs. Healthy Sleeper, between-subject variable) as independent variables, controlling for age and mood disturbances as assessed by HADS-Depression and HADS-Anxiety scores. The multivariate tests revealed significant effects of Group (F = 5.854, p < 0.001) and Condition (F = 22.075, p < 0.001), but not their interaction (F = 0.038, p = 0.990). The univariate tests found that, Condition effect was significant for N2 (F = 7.681, p = 0.007) and marginally significant for P3 (F = 3.403, p = 0.068), while Group effect was only significant for N2 (F = 6.753, p = 0.011) but not P3 (F = 1.763, p = 0.187). Post hoc analyses of Group effect using independent samples t-test showed that Healthy Sleepers have a larger N2 compared to Insomnia Group (t = 2.47, p = 0.015, d = 0.472). Post hoc analyses of Condition effect using paired samples t-test found larger N2 (t = 6.55, p < 0.001, d = 0.883) and smaller P3 (t = 4.44, p < 0.001, d = 0.599) in NoGo compared to Go condition.

3.4 | Time-frequency domain results

Increased midfrontal theta power has been found to be related to the recruitment of inhibitory control (Nigbur et al., 2011). Here we examined theta power on the same time window as N2 over the same frontal-central electrodes (Fz, FCz, Cz). The time-frequency data and event-related theta power are shown in Figure 4. As shown in Figure 4, higher theta band (4–7 Hz) power was observed in the NoGo condition (healthy sleeper group, Mean = 5.48, SD = 1.89; insomnia group, Mean = 4.56, SD = 2.05) than the Go condition (healthy sleeper group, Mean = 3.27, SD = 1.29; insomnia group, Mean = 2.44, SD = 1.44). The univariate tests of the same abovementioned MANCOVA revealed a significant Condition effect (F = 49.574, p < 0.001) and a Group effect (F = 8.006, p = 0.006) for theta. Post hoc t-tests showed that insomnia group had a significantly decreased theta power

(a) Time-frequency plots of Attend Go/No-Go in each group, group difference, and condition difference at frontal-central area (Fz, FCz, Cz)



(b) Event-related theta band (4 - 7 Hz) activities across the N2 time window (250 - 400 ms) at frontal-central area (Fz, FCz, Cz)

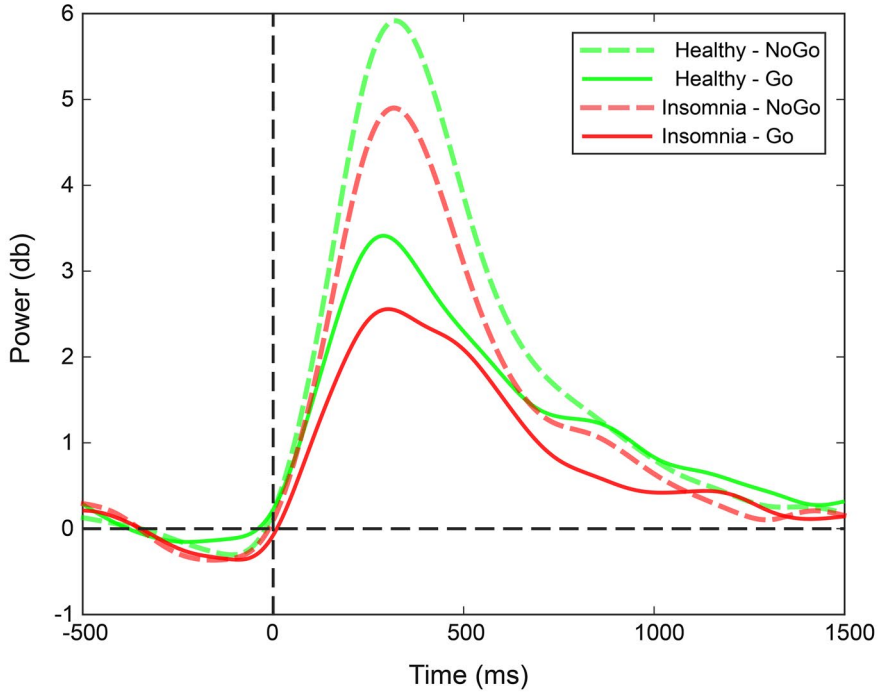


FIGURE 4 Time-frequency domain results. (a) Time-frequency plots and *p*-values with False Detection Rate (FDR) correction. (b) Event-related theta band (4–7 Hz) activities across the N2 time window (250–400 ms)

TABLE 3 Correlation coefficients between ERP components and sleep, mood and impulsivity measures

		NoGo N2		NoGo P3		NoGo Theta		Go Theta		CNV	
	Measures	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Sleep	PSQI	0.18	0.194	0.09	0.539	−0.08	0.543	−0.21	0.119	**0.35****	**0.009**
	ISI	0.26$^{\wedge}$	0.061	0.09	0.536	−0.19	0.167	**−0.31***	0.023	**0.39****	**0.003**
	rMEQ	0.13	0.331	−0.24$^{\wedge}$	0.073	−0.08	0.575	−0.06	0.675	−0.15	0.270
Actigraphy	TIB	0.12	0.367	−0.07	0.619	−0.17	0.226	−0.09	0.531	0.09	0.494
	TST	0.18	0.197	−0.07	0.634	−0.17	0.218	−0.07	0.632	0.01	0.919
	SOL	−0.11	0.408	0	0.991	−0.02	0.879	−0.09	0.508	0.24$^{\wedge}$	0.077
	SE	−0.04	0.802	0.12	0.391	0.24$^{\wedge}$	0.072	0.15	0.291	**−0.33***	**0.015**
	WASO	0.08	0.565	−0.15	0.286	**−0.28***	0.042	−0.14	0.317	**0.28***	**0.037**
Mood	HADS-D	0.14	0.328	0.01	0.940	**−0.32***	0.018	**−0.31***	0.024	0.17	0.213
	HADS-A	0.21	0.127	−0.05	0.723	−0.23$^{\wedge}$	0.086	**−0.32***	0.018	**0.35***	**0.009**
Impulsivity	BIS	0.11	0.445	0.02	0.873	−0.02	0.859	−0.08	0.549	0.01	0.951
	Attention	0.26$^{\wedge}$	0.059	0.16	0.254	−0.08	0.564	−0.04	0.749	0.07	0.631
	Motor	0.01	0.951	−0.01	0.932	0.09	0.530	0.01	0.973	−0.02	0.873
	Non-Planning	−0.02	0.894	−0.08	0.575	−0.06	0.687	−0.13	0.360	−0.02	0.888

Abbreviations: BIS: Barratt Impulsiveness Scale; HADS: Hospital Anxiety and Depression Scale, HADS-D: Depression subscale, HADS-A: Anxiety subscale; ISI: Insomnia Severity Index; PSQI: Pittsburgh Sleep Quality Index; rMEQ: Reduced Morningness -Eveningness Questionnaire; SE: Sleep efficiency; SOL: Sleep onset latency; TIB: Time in bed; TST: Total sleep time; WASO: Wake after sleep onset.

Bold values denote statistical significance at the $p<0.05$ level.

^ $p < 0.1$.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

compared to healthy sleeper group ($t = 2.29$, $p = 0.024$, $d = 0.438$), and NoGo target elicited greater theta power than Go target ($t = 13.37$, $p < 0.001$, $d = 1.803$).

3.5 | Correlations between EEG measures and measures of sleep, mood, and impulsivity

Table 3 shows the correlations between the ERP measures (amplitudes of NoGo-N2, NoGo-P3, NoGo Theta, Go Theta and CNV) and the measures on sleep, mood, and impulsivity (BIS). Reduced amplitude of NoGo-N2 was associated with greater insomnia severity (ISI: $r = 0.26$, $p = 0.061$), and higher attention impulsivity (BIS-attention, $r = .26$, $p = 0.059$) at a trend significance level, but not motor or non-planning subscales. NoGo condition theta band activities were associated with wake after sleep onset ($r = -0.28$, $p < 0.5$) and depressive mood (HADS-D: $r = -0.32$, $p < 0.05$), and showed a trend of correlation with anxiety symptoms (HADS-A: $r = -0.23$, $p = 0.086$). Go condition theta band activities were associated with insomnia severity (ISI: $r = -0.31$, $p < 0.05$), depressive mood (HADS-D: $r = -0.31$, $p < 0.05$) and anxiety symptoms (HADS-A: $r = -0.32$, $p < 0.05$). Reduced amplitude of CNV was correlated with a higher level of subjective sleep disturbance (PSQI: $r = 0.35$, $p < 0.01$; ISI: $r = 0.39$, $p < 0.01$), poorer objectively measured sleep based on actigraphy (sleep efficiency: $r = -0.33$, $p < 0.05$; wake after sleep onset: $r = 0.28$, $p < 0.05$; sleep onset latency: $r = 0.24$, $p = 0.077$), and greater levels of anxiety (HADS-A: $r = 0.35$, $p < 0.01$).

4 | DISCUSSIONS

The current study examined the behavioral performance and EEG activities related to response inhibition in both time and time-frequency domains in youths with insomnia disorder as compared to healthy sleepers. We did not observe any between-group behavioral difference in the CGNG task. Compared to healthy sleeper group, however, insomnia group showed reduced cue-related CNV, indicating impaired attention allocation during the preparation phase. The insomnia group also showed blunted N2 amplitudes and attenuated theta activities to Go/NoGo signals, potentially suggesting disrupted inhibitory control.

In this study, we found reduced CNV and N2 amplitudes with medium effect size in the insomnia group as compared to the healthy sleeper group in a task assessing inhibitory control. Our time-domain finding was generally in line with those of previous ERP studies conducted in the adults with insomnia. It was found that adult insomnia patients had reduced CNV, N2, and P3 components during the AX-continuous performance task (Muscarella et al., 2019) and reduced P3 during the Stop Signal Task (Zhao et al., 2018) than healthy sleepers (median to large effect size, $\eta^2 = 0.13–0.21$). These ERPs findings collectively suggested

insomnia is associated with altered neural processing related to inhibitory control. However, we only observed the group differences in the CNV and N2 (median effect size), but not the P3 component. The lack of P3 differences might be due to the different tasks being used and the varying levels of cognitive demands. The smaller effect size in our data might be related to the relatively shorter duration of insomnia in our sample, which consisted of younger participants with insomnia, as compared with the middle-aged sample in the earlier studies (Muscarella et al., 2019; Zhao et al., 2018). Our pattern of results was similar to that of a previous study based on a non-clinical sample, which reported that self-identified poor sleepers (as defined by a sleep disturbance index) had reduced N2 but not P3 in a Go/NoGo task (Breimhorst, Falkenstein, Marks, & Griefahn, 2008). Based on previous research on NoGo N2 (Falkenstein, Hoormann, & Hohnsbein, 1999; Pornpattananangkul, Hu, & Nusslock, 2015), our data suggested that insomnia was associated with altered top-down inhibition mechanisms prior to motor response.

We also found that insomnia group showed reduced event-related theta activity than healthy sleeper group. To our knowledge, this was the first study supporting the hypothesis that insomnia was associated with inhibition deficits, based on EEG time-frequency responses. A recent study has suggested that the increase in frontal midline theta band oscillations during the Cued Go/NoGo task might be reflecting not only response inhibition but also top-down attention processes such as conflict monitoring (Hong, Sun, Wang, Li, & Tong, 2020). Our finding extended the previous observation that insomnia patients had decreased theta band power during awake resting EEG assessment (Wolyńczyk-Gmaj & Szelenberger, 2011), and showed that insomnia patients also had lower theta power during the performance on a Go/NoGo task involving inhibitory control. However, it is worth noting that despite the lower theta activity in the insomnia group, there was a lack of behavioral abnormalities on task performance. Research has shown that attenuated theta power may reflect higher hyperarousal (Wolyńczyk-Gmaj & Szelenberger, 2011), supporting the notion that insomnia was associated with hyperactive physiological, neurocognitive, and emotional systems around the clock (Kay & Buysse, 2017). In other words, attenuated theta band activity in the insomnia group might reflect compensatory mechanisms related with hyperarousal in insomnia, and thus might explain the maintained level of behavioral performance.

In this study, insomnia group was found to show elevated levels of depressive and anxiety symptoms compared with the healthy sleeper group, which were in line with previous reports showing an association between insomnia and the risk of psychopathology (Blake, Trinder, & Allen, 2018; Soldatos, 1994). We also found that lower theta activity in both Go and NoGo conditions, which indicated a deficit in response activation and inhibition, were associated with higher levels of depression and anxiety in our samples. Previous research has shown altered N2-P3 activities and inhibitory deficits in individuals with depression (Kaiser et al., 2003; Ruchsow, Groen, Kiefer, Beschoner, et al., 2008) and anxiety (Righi, Mecacci, & Viggiano, 2009; Xia, Mo, Wang, Zhang, & Zhang, 2020). The findings collectively suggested a link between psychopathology and altered neural processing underlying inhibitory control. In a previous study that measured ERP during an Emotional Go/NoGo Task, it was found that increased rumination, which is a robust cognitive risk factor for depression, was associated with specific alterations in both N2 and P3 amplitudes in response to non-emotional faces in girls of mothers with histories of depression (Connell, Danzo, Magee, & Dawson, 2020). It is possible that inhibitory control deficits might play a role in the cognitive processes linking to the risk of depression and anxiety in individuals with insomnia. Due to the cross-sectional design of the present study and the multidimensional relationship between sleep and psychopathology, future longitudinal studies would be needed to further investigate the neurocognitive processes underlying the link between insomnia and psychopathology.

In this study, we found higher impulsivity score in the insomnia group as compared to the healthy sleeper group, which was in line with the observed association between poor sleep and higher impulsivity in the previous research (Bauducco, Salihovic, & Boersma, 2019). Meanwhile, previous research, as well as the present study, found an association between higher impulsivity and EEG correlates of inhibitory control (Ruchsow, Groen, Kiefer, Hermle, et al., 2008; Zhao et al., 2018). It has been argued that reduced inhibition and increased impulsivity might serve as part of the mechanisms underlying the link between insomnia and suicidal risk (McCall & Black, 2013). In a previous study that measured ERP during the Go/NoGo task, it was found that individuals with a history of suicide attempts also showed lower NoGo N2 amplitude, but no difference in P3 component and behavioral outcomes, as compared with the controls without a history of suicide attempts (Albanese et al., 2019). Taken together, the findings from the current study and previous research suggested a unique possibility that inhibitory control and impulsivity might play a role in contributing to the elevated risk of suicide in the individuals with insomnia (Liu et al., 2019). While it is plausible that the neurocognitive mechanism underlying the link between suicidal risk and insomnia might be partly explained by inhibition deficits in individuals with insomnia, the present study was limited by a lack of measure of suicidality, which precluded us from directly testing this hypothesis. Future study with a longitudinal design would be needed to further investigate whether certain neural pathways and brain activity alterations in insomnia might have the prognostic implications in predicting suicidal risk, and if so, whether these altered brain responses could be utilized as a potential marker to screen for individuals with insomnia at high risk of suicide.

Our findings contributed to the current literature by adding novel information on response inhibition in the context of insomnia using time-frequency domain analysis of the theta band activities. To address the problem of limited statistical power in the previous studies owing to the small sample size, we conducted *a priori* power analysis to ensure adequate sample size. Also, the present study included a sample of medication-free young people. Nonetheless, there were some limitations to consider. One might argue that the timing of the experiment, which was fixed to start at 10:30 a.m. across almost all the participants, failed to take into consideration the individual differences in

chronotype preferences. However, this study did not include the participants with extreme chronotype preference (e.g., advanced or delayed sleep phase syndrome) based on prescreening. In addition, this study only used clinical interview to rule out comorbid sleep disorders. Future research could use polysomnographic (PSG) sleep assessment to confirm the comorbidity of sleep problems and to provide objective sleep parameters for analysis. Finally, there were some other potential confounders that were not assessed and controlled in the analyses, including the handedness of the participants, fatigue symptom severity, consumption of activating substances (e.g., caffeine).

In conclusion, the present study found that insomnia was associated with impaired attention preparation as indicated by attenuated CNVs, and inhibition deficits as indicated by N2 amplitude and theta power, despite of comparable behavioral performance from the Cued Go/NoGo task. The temporal dynamics of inhibition alterations in insomnia might be associated with inadequate top-down cognitive control processes. Future studies with longitudinal designs are needed to further explore the long-term impacts of altered inhibition processing in relation to the development of comorbid psychopathology, especially the risk of suicidality, in individuals with insomnia.

ACKNOWLEDGEMENTS

This work was supported by the Seed Fund for Basic Research (to SX Li), University Research Committee, The University of Hong Kong, Early Career Scheme (Ref. 27613017, to SX Li) and General Research Fund (Ref. 17601318, to X Hu) of Hong Kong Research Grants Council, and National Natural Science Foundation of China (No. 31922089, to X Hu).

CONFLICT OF INTEREST

The authors do not have any conflicts of interest to disclose.

AUTHOR CONTRIBUTION

All the authors made substantial contributions to the work leading to this article. Dr. Ling conceptualized the study, conducted data collection, managed the data and conducted the statistical analyses, drafted the initial manuscript, and made critical revisions to the manuscript. Ms. Lin conceptualized the study, managed the data and conducted the statistical analyses and contributed to writing and critical revisions of the manuscript. Dr. Hu, and Dr. S Li conceptualized the study, provided the supervision of the implementation of the study, drafted the initial manuscript, and made critical revisions to the manuscript. Dr. X Li, and Dr. Chan helped with study design, participated in the data collection and management. Dr. Zhang, and Dr. Wing conceptualized the study, and critically reviewed the manuscript.

DATA AVAILABILITY STATEMENT

Author elects to not share data.

ORCID

Ngan Yin Chan https://orcid.org/0000-0002-3009-3565
Jihui Zhang https://orcid.org/0000-0002-4976-3039
Yun Kwok Wing https://orcid.org/0000-0002-5745-5474
Xiaoqing Hu https://orcid.org/0000-0001-8112-9700
Shirley Xin Li https://orcid.org/0000-0002-7085-1850

REFERENCES

Aeschbach, D., Matthews, J.R., Postolache, T.T., Jackson, M.A., Giesen, H.A., & Wehr, T.A. (1999). Two circadian rhythms in the human electroencephalogram during wakefulness. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *277*(6), R1771–R1779.

Albanese, B.J., Macatee, R.J., Gallyer, A.J., Stanley, I.H., Joiner, T.E., & Schmidt, N.B. (2019). Impaired conflict detection differentiates suicide attempters from ideating non-attempters: Evidence from event-related potentials. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *4*(10), 902–912. https://doi.org/10.1016/j.bpsc.2019.05.015

Altena, E., Van Der Werf, Y.D., Sanz-Arigita, E.J., Voorn, T.A., Rombouts, S.A.R.B., Kuijer, J.P.A., & Van Someren, E.J.W. (2008). Prefrontal hypoactivation and recovery in insomnia. *Sleep*, *31*(9), 1271–1276. https://doi.org/10.5665/sleep/31.9.1271

(2013). American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders. In *Diagnostic and Statistical Manual of Mental Disorders* (5th edn). American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890425596.893619

Amorim, P. (2000). Mini International Neuropsychiatric Interview (MINI): Validation of a short structured diagnostic psychiatric interview. *Mini International Neuropsychiatric Interview (MINI): Validacao De Entrevista Breve Para Diagnostico De Transtornos Mentais.*, *22*(3), 106–115. https://doi.org/10.1590/S1516-44462000000300003

Bauducco, S.V., Salihovic, S., & Boersma, K. (2019). Bidirectional associations between adolescents' sleep problems and impulsive behavior over time. *Sleep Medicine: X*, *1*, 100009. https://doi.org/10.1016/j.sleepx.2019.100009

Bjelland, I., Dahl, A.A., Haug, T.T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale: An updated literature review. *Journal of Psychosomatic Research*, *52*(2), 69–77. https://doi.org/10.1016/S0022-3999(01)00296-3

Blake, M.J., Trinder, J.A., & Allen, N.B. (2018). Mechanisms underlying the association between insomnia, anxiety, and depression in adolescence: Implications for behavioral sleep interventions. *Clinical Psychology Review*, *63*, 25–40. https://doi.org/10.1016/J.CPR.2018.05.006

Blank, M., Zhang, J., Lamers, F., Taylor, A.D., Hickie, I.B., & Merikangas, K.R. (2015). Health correlates of insomnia symptoms and comorbid mental disorders in a nationally representative sample of US adolescents. *Sleep*, *38*(2), 197–204. https://doi.org/10.5665/sleep.4396

Bokura, H., Yamaguchi, S., & Kobayashi, S. (2001). Electrophysiological correlates for response inhibition in a Go/NoGo task. *Clinical Neurophysiology*, *112*(12), 2224–2232. https://doi.org/10.1016/S1388-2457(01)00691-5

Breimhorst, M., Falkenstein, M., Marks, A., & Griefahn, B. (2008). The relationship between poor sleep and inhibitory functions indicated by event-related potentials. *Experimental Brain Research*, *187*(4), 657. https://doi.org/10.1007/s00221-008-1354-4

Buckelew, S.P., DeGood, D.E., Roberts, K.D., Butkovic, J.D., & MacKewn, A.S. (2009). Awake EEG disregulation in good compared to poor sleepers. *Applied Psychophysiology Biofeedback*, *34*(2), 99–103. https://doi.org/10.1007/s10484-009-9080-7

Buysse, D.J., Reynolds, C.F., Monk, T.H., Berman, S.R., & Kupfer, D.J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4

Carciofo, R., Du, F., Song, N., Qi, Y., & Zhang, K. (2012). Age-related chronotype differences in Chinese, and reliability assessment of a reduced version of the Chinese Morningness-Eveningness Questionnaire. *Sleep and Biological Rhythms*, *10*(4), 310–318. https://doi.org/10.1111/j.1479-8425.2012.00577.x

Champely, S., Ekstrom, C., Dalgaard, P., Gill, J., Weibelzahl, S., Anandkumar, A., … De Rosario, H. (2018). *Package "pwr": Basic functions for power analysis*. CRAN Repository.

Chung, K.F., Kan, K.K.K., & Yeung, W.F. (2011). Assessing insomnia in adolescents: Comparison of Insomnia Severity Index, Athens Insomnia Scale and Sleep Quality Index. *Sleep Medicine*, *12*(5), 463–470. https://doi.org/10.1016/j.sleep.2010.09.019

Chung, K.F., Kan, K.K.K., & Yeung, W.F. (2014). Insomnia in adolescents: Prevalence, help-seeking behaviors, and types of interventions. *Child and Adolescent Mental Health*, *19*(1), 57–63. https://doi.org/10.1111/camh.12009

Connell, A., Danzo, S., Magee, K., & Dawson, G. (2020). Rumination in early adolescent girls: An EEG study of cognitive control and emotional responding in an emotional Go/NoGo task. *Cognitive, Affective and Behavioral Neuroscience*, *20*(1), 181–194. https://doi.org/10.3758/s13415-019-00761-9.

Cooper, P.S., Wong, A.S.W., McKewen, M., Michie, P.T., & Karayanidis, F. (2017). Frontoparietal theta oscillations during proactive control are associated with goal-updating and reduced behavioral variability. *Biological Psychology*, *129*, 253–264. https://doi.org/10.1016/j.biopsycho.2017.09.008

Diamond, A. (2012). Executive functions. *Annual Review of Psychology*, *64*(1), 135–168. https://doi.org/10.1146/annurev-psych-113011-143750

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychologica*, *101*(2–3), 267–291. https://doi.org/10.1016/s0001-6918(99)00008-6

Geier, C., & Luna, B. (2009). The maturation of incentive processing and cognitive control. *Pharmacology Biochemistry and Behavior*, *93*(3), 212–221. https://doi.org/10.1016/j.pbb.2009.01.021

Hong, X., Sun, J., Wang, J., Li, C., & Tong, S. (2020). Attention-related modulation of frontal midline theta oscillations in cingulate cortex during a spatial cueing Go/NoGo task. *International Journal of Psychophysiology*, *148*, 1–12. https://doi.org/10.1016/j.ijpsycho.2019.11.011

Hong, X., Wang, Y., Sun, J., Li, C., & Tong, S. (2017). Segregating top-down selective attention from response inhibition in a spatial cueing Go/NoGo task: An ERP and source localization study. *Scientific Reports*, *7*(1), 9662. https://doi.org/10.1038/s41598-017-08807-z

Hysing, M., Sivertsen, B., Stormark, K.M., & O'Connor, R.C. (2015). Sleep problems and self-harm in adolescence. *British Journal of Psychiatry*, *207*(4), 306–312. https://doi.org/10.1192/bjp.bp.114.146514

Kaiser, S., Unger, J., Kiefer, M., Markela, J., Mundt, C., & Weisbrod, M. (2003). Executive control deficit in depression: Event-related potentials in a Go/Nogo task. *Psychiatry Research - Neuroimaging*, *122*(3), 169–184. https://doi.org/10.1016/S0925-4927(03)00004-0

Kay, D.B., & Buysse, D.J. (2017). Hyperarousal and beyond: New insights to the pathophysiology of insomnia disorder through functional neuroimaging studies. *Brain Sciences*, *7*(12), 23. https://doi.org/10.3390/brainsci7030023.

Leung, C.M., Wing, Y.K., Kwong, P.K., Lo, A., & Shum, K. (1999). Validation of the Chinese-Cantonese version of the Hospital Anxiety and Depression Scale and comparison with the Hamilton Rating Scale of Depression. *Acta Psychiatrica Scandinavica*, *100*(6), 456–461. https://doi.org/10.1111/j.1600-0447.1999.tb10897.x

Liu, J.W., Tu, Y.K., Lai, Y.F., Lee, H.C., Tsai, P.S., Chen, T.J., … Chiu, H.Y. (2019). Associations between sleep disturbances and suicidal ideation, plans, and attempts in adolescents: A systematic review and meta-analysis. *Sleep*, *42*(6), zsz054. https://doi.org/10.1093/sleep/zsz054

Masharipov, R., Kireev, M., Korotkov, A., & Medvedev, S. (2019). Non-selective response inhibition during an equal probability Go/NoGo task: Bayesian analysis of fMRI data. *BioRxiv*, https://doi.org/10.1101/823625

McCall, W.V., & Black, C.G. (2013). The link between suicide and insomnia: theoretical mechanisms. *Current Psychiatry Reports*, *15*(9), 389. https://doi.org/10.1007/s11920-013-0389-9

Morin, C.M., Belleville, G., Bélanger, L., & Ivers, H. (2011). The insomnia severity index: Psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, *34*(5), 601–608. https://doi.org/10.1093/sleep/34.5.601

Muscarella, C., Mairesse, O., Hughes, G., & Neu, D., & Van den Bussche, E. (2019). Recruitment dynamics of cognitive control in insomnia. *Sleep*, *42*(5), zsz039. https://doi.org/10.1093/sleep/zsz039

Nigbur, R., Ivanova, G., & Stürmer, B. (2011). Theta power as a marker for cognitive interference. *Clinical Neurophysiology*, *122*(11), 2185–2194.

Nofzinger, E.A., Boysse, D.J., Germain, A., Price, J.C., Miewald, J.M., & Kopfer, D.J. (2004). Functional neuroimaging evidence for hyperarousal in insomnia. *American Journal of Psychiatry*, *161*(11), 2126–2129. https://doi.org/10.1176/appi.ajp.161.11.2126

Phillips, J.M., Vinck, M., Everling, S., & Womelsdorf, T. (2014). A long-range fronto-parietal 5- to 10-Hz network predicts "top-down" controlled guidance in a task-switch paradigm. *Cerebral Cortex*, *24*(8), 1996–2008. https://doi.org/10.1093/cercor/bht050

Pornpattananangkul, N., Hu, X., & Nusslock, R. (2015). Threat/reward-sensitivity and hypomanic-personality modulate cognitive-control and attentional neural processes to emotional stimuli. *Social Cognitive and Affective Neuroscience*, *10*(11), 1525–1536.

R Core Team (2014). R: A language and environment for statistical computing, Vienna, Austria: R Foundation for Statistical Computing.

Righi, S., Mecacci, L., & Viggiano, M.P. (2009). Anxiety, cognitive self-evaluation and performance: ERP correlates. *Journal of Anxiety Disorders*, *23*(8), 1132–1138. https://doi.org/10.1016/j.janxdis.2009.07.018

Ruchsow, M., Groen, G., Kiefer, M., Beschoner, P., Hermle, L., Ebert, D., & Falkenstein, M. (2008). Electrophysiological evidence for reduced inhibitory control in depressed patients in partial remission: A Go/Nogo study. *International Journal of Psychophysiology*, *68*(3), 209–218. https://doi.org/10.1016/j.ijpsycho.2008.01.010

Ruchsow, M., Groen, G., Kiefer, M., Hermle, L., Spitzer, M., & Falkenstein, M. (2008). Impulsiveness and ERP components in a Go/Nogo task. *Journal of Neural Transmission*, *115*(6), 909–915. https://doi.org/10.1007/s00702-008-0042-7

Shochat, T., Cohen-Zion, M., Tzischinsky, O., Dahl, R.E., Lewin, D.S., Olds, T., … Sigfúsdóttir, I.D. (2014). Functional consequences of inadequate sleep in adolescents: a systematic review. *Sleep Medicine Reviews*, *18*(1), 75–87. https://doi.org/10.1016/j.smrv.2013.03.005

Shuqiao, Y., Huiqin, Y., Xiongzhao, Z., Auerbach, R.P., Abela, J.R.Z., Pulleyblank, R.W., & Xi, T. (2007). An examination of the psychometric properties of the Chinese version of the barratt impulsiveness scale, 11th version in a sample of Chinese adolescents. *Perceptual and Motor Skills*, *104*(3 II), 1169–1182. https://doi.org/10.2466/PMS.104.4.1169-1182

Si, T.M., Shu, L., Dang, W.M., Se, Y.A., Chen, J.X., Dong, W.T., … Zhang, W.H. (2009). Evaluation of the reliability and validity of Chinese version of the Mini International Neuropsychiatric Interview in patients with mental d isorders. *Chinese Mental Health Journal*, *23*(7), 493–503.

Sing, C.Y., & Wong, W.S. (2011). Prevalence of insomnia and its psychosocial correlates among college students in Hong Kong.

Journal of American College Health, *59*(3), 174–182. https://doi.org/10.1080/07448481.2010.497829

Soldatos, C.R. (1994). Insomnia in relation to depression and anxiety: Epidemiologic considerations. *Journal of Psychosomatic Research*, *999*(94), 3–8. https://doi.org/10.1016/0022-3999(94)90130-9

Swick, D., Ashley, V., & Turken, U. (2011). Are the neural correlates of stopping and not going identical? Quantitative meta-analysis of two response inhibition tasks. *NeuroImage*, *56*(3), 1655–1665. https://doi.org/10.1016/j.neuroimage.2011.02.070

The jamovi project. (2021). jamovi (Version 1.6) [Computer Software]. Retrieved from https://www.jamovi.org

Wolyńczyk-Gmaj, D., & Szelenberger, W. (2011). Waking EEG in primary insomnia. *Acta Neurobiologiae Experimentalis*, *71*(3), 387–392.

World Health Organization. (1986). *Young people's health - a challenge for society*.

Xia, L., Mo, L., Wang, J., Zhang, W., & Zhang, D. (2020). Trait Anxiety Attenuates Response Inhibition: Evidence From an ERP Study Using the Go/NoGo Task. *Frontiers in Behavioral Neuroscience*, *14*, http://dx.doi.org/10.3389/fnbeh.2020.00028.

Yu, W., Zhang, J., & Wing, Y. (2015). Validation of Chinese version of structured diagnostic interview for insomnia disorder: A preliminary report. *Sleep Medicine*, *16*, S248–S249. https://doi.org/10.1016/j.sleep.2015.02.1536

Zhao, W., Gao, D., Yue, F., Wang, Y., Mao, D., Chen, X., & Lei, X. (2018). Response inhibition deficits in insomnia disorder: An event-related potential study with the stop-signal task. *Frontiers in Neurology*, *9*(AUG), 610. https://doi.org/10.3389/fneur.2018.00610

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Ling J, Lin X, Li X, et al. Altered brain activity related to inhibitory processing in youth with insomnia. *J Sleep Res*. 2021;00:e13398. https://doi.org/10.1111/jsr.13398