Biological Psychology 186 (2024) 108742

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

Subsyndromal depression leads to early under-activation and late over-activation during inhibitory control: an ERP study

Weiyi Zhou [a], Fangfang Long [a,d], Fang Wang [a], Renlai Zhou [a,b,c,*,1]

[a] Department of Psychology, Nanjing University, Nanjing 210023, China
[b] Department of Radiology, Nanjing Drum Tower Hospital, the Affiliated Hospital of Nanjing University Medical School, Nanjing 210008, China
[c] State Key Laboratory of Media Convergence Production Technology and Systems, Beijing, China
[d] School of Psychology, Guizhou Normal University, Guiyang 550025, China

ARTICLE INFO

Keywords:
Depressive tendency
Inhibitory control
Response inhibition
Interference control
ERP

ABSTRACT

Individuals with depressive disorders have deficits in inhibitory control and exhibit symptoms of impaired cognitive and emotional functioning. Individuals with subsyndromal depression are intermediate between the healthy group and clinically diagnosed patients with depressive disorders, and studying the characteristics of their inhibitory control functioning can help to investigate the mechanisms underlying the development of depressive disorders. Using two classical paradigms of inhibitory control, Flanker and Go/NoGo, the present study explored the differences in inhibitory control between individuals with subsyndromal depression and healthy individuals from the perspectives of both response inhibition and interference control. Behavioral results showed that both groups did not differ in response time and accuracy; in terms of event-related potentials, individuals with subsyndromal depression presented smaller N2 amplitudes as well as larger P3 amplitudes in the NoGo condition of the Go/NoGo paradigm; and smaller N2 amplitudes in the incongruent condition of the Flanker paradigm. Moreover, the depression-prone group showed lower theta power compared to the healthy group in the NoGo condition of the NoGo paradigm and the incongruent condition of the Flanker paradigm. The present study reveals that the depression-prone group may have a compensatory mechanism in the response inhibition, which is mainly manifested as early under-activation as well as late over-activation.

1. Introduction

Inhibitory control, an important component of the central executive system, refers to the cognitive process of continuously recognizing and evaluating stimuli in the environment, selecting task-relevant stimuli, and ignoring irrelevant stimuli during information processing (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Wiecki & Frank, 2013). As an important cognitive ability, there are large individual differences in inhibitory control; individuals with weaker inhibitory control have difficulty inhibiting distracting irrelevant stimuli and flexibly controlling their attention, which leads to altered behavioral patterns (Miyake & Friedman, 2012). It has been suggested that an individual's ability to inhibit inappropriate or irrelevant thoughts and behaviors can not only be used to predict task performance but also largely determine the impact of a negative event on their mental health (Gotlib & Joormann, 2010).

Depressive disorder is a common mental disorder, affecting 4–7% of the global population (Liu et al., 2020). The core symptoms of depressive disorders include long-lasting negative moods, lack of pleasure, and significant impairment of cognitive and social functioning (Cambridge et al., 2018), and at least 30% of people with depressive disorders relapse after being cured (Richards and Borglin, 2011). Because of the serious burden depressive disorders place on individuals, families, and even society, vulnerability factors for depression have been a focus of research. Cognitive models of depression and negative affect point to deficits in inhibitory control functions as a central component in the pathogenesis of depressive disorders (MacQueen et al., 2000) and subsyndromal depression (Joormann & Gotlib, 2010).

Inhibitory control can be categorized into two parts: response inhibition and interference control (Friedman & Miyake, 2004; Diamond, 2013). Response inhibition refers to the inhibition of automatized or dominant responses that are not relevant to the current task, whereas

* Correspondence to: Department of Psychology, Nanjing University, Room 418, Heren Hall, 163 Xianlin Avenue, Nanjing 210023, China.
E-mail address: rlzhou@nju.edu.cn (R. Zhou).
[1] ORCID ID: 0000-0001-7035-7097

https://doi.org/10.1016/j.biopsycho.2024.108742
Received 6 November 2023; Received in revised form 2 January 2024; Accepted 3 January 2024
Available online 6 January 2024

interference control refers to the inhibition of informational distractions that are not relevant to the current task and maintain the current task goal. Previous research has examined the association between inhibitory control and depression through a response inhibition or interference control paradigm, in depressive disorder or subsyndromal depression groups versus healthy group populations. For interference control, Holmes and Pizzagalli's (2008) study found that people with depressive disorder responded slower than the healthy group in the Flanker task, but there was no difference in accuracy; Dillon et al.'s (2015) study found that people with depressive disorders responded slower to incongruent trials in the Flanker paradigm and had a higher accuracy. For response inhibition, a study by Shimony et al. (2021) found that in a non-clinically depressed population, diminished Go/Nogo behavioral results were significantly associated with increased depressive disorder symptoms and excessive interference with negative affect, and a study by Yitzhak et al. (2023) found that in a less symptomatic depressed population, deficits in inhibitory control were associated with difficulties in the regulation of negative affect. However, fewer studies have compared both response inhibition and interference control in depressed populations with normal populations; Li et al. (2021) conducted a behavioral experiment of both response inhibition and interference control and noted that patients with depressive disorders had significant deficits in both dimensions, as evidenced by slower response time in the stop-signal task and lower accuracy in incongruent conditions of the Flanker task. In summary, behavioral results suggest that both inhibitory control functions are deficient in patients with depressive disorders and that worsening inhibitory control deficits may be accompanied by more depressive symptoms.

The observed discrepancies in the behavioral research results may stem from differences in the selected study participants and employed research paradigms. For instance, studies conducted by Holmes and Pizzagalli (2008) and Dillon et al. (2015) measured interference control using the Stroop and Flanker paradigms, respectively. On the other hand, Shimony et al. (2021) and Yitzhak et al. (2023) measured response inhibition with the Go/NoGo paradigm, but their participants were non-clinical. Li et al. (2021) simultaneously investigated interference control and response inhibition, but their chosen paradigm for response inhibition differed from the aforementioned studies. Neurophysiological methods somewhat compensate for the limitations of experimental design. N2 is an ERP component that is closely associated with inhibitory control (Larson et al., 2014; Hsieh et al., 2022; Pires et al., 2014). For example, the N2 component is a symbol in the classic Go/NoGo or Flanker paradigm (Cheng et al., 2019; Erb & Cavanagh. 2019; Overbye et al., 2021), where the appearance of a NoGo stimulus or a Flanker incongruent stimulus is characterized by a significant increase in the amplitude of N2 so that the N2 amplitude increase reflects an elevated functional demand of inhibition. Monitoring the emergence of a conflicting stimulus and creating a need for inhibition, the brain will be activated, and the P3 component that appears after the N2 demonstrates the brain's processing of the target stimulus (Erb & Cavanagh. 2019; López Zunini et al., 2016; Pires et al., 2014). The amplitude of the P3 component showed a significantly greater increase in the cognitive conflict condition (Overbye et al., 2021; Rueda et al., 2004). The neural significance of the P3 lies in what extent top-down control is involved in the conflict resolution process (Erb & Cavanagh, 2019; Groom & Cragg, 2015). A study by Alderman et al. (2015) found that patients with depressive disorders had significantly lower N2 amplitudes during the Flanker task and that only the healthy group could show a significant Flanker effect. With a larger sample of depressed patients, a research group found that the P3 amplitude during the Flanker task was significantly smaller in patients with depressive disorders (Klawohn et al., 2020). The study by Santopetro et al. (2021) used the P3 of the Flanker paradigm to track the progression of patients with depressive disorders and found that patients with smaller P3 amplitudes showed more severe depressive disorder symptoms in the follow-up. As for response inhibition, Kaiser et al. (2003) found that patients with depressive disorders presented smaller NoGo-N2 amplitudes during the Go/NoGo paradigm. A study by Ruchsow et al. (2008) found that patients with depressive disorders would exhibit smaller NoGo-N2 and NoGo-P3.

Neural oscillations in the theta frequency band (4–7 Hz) provide neural communication between the functional brain networks involved in inhibitory control. Inhibitory control involves the coordinated activity of multiple brain regions, regardless of the type of subfunction (Cavanagh & Frank, 2014). As a large-amplitude and low-frequency temporal scheme, the theta band is suitable for organizing activities across large spatial distances (Buzsáki & Draguhn, 2004). The significance of the theta band lies in its integration of sensory and response-related information during inhibitory control (Hoffmann & Beste, 2015; Chmielewski & Beste, 2017). Although there is a lack of studies comparing the theta oscillations in the inhibitory control task directly between the depressive disordered/depressively inclined group and the healthy group, an interventional study by Schoenberg and Speckens (2014) found that meditation therapy ameliorated symptoms and rumination in patients with depressive disorders while enhancing theta oscillations during an emotional Go/NoGo task, suggesting the possibility of theta oscillatory attenuation in patients with depressive disorders. Although evidence on neural oscillations is scarce, previous studies have more consistently demonstrated a deficient activation in inhibitory control in patients with depressive disorders (Kato et al., 2022; Vahid et al., 2018).

Deficits in cognitive and emotional functioning in patients with depressive disorders stem from deficits in inhibitory control to some extent. Previous studies have largely clarified the abnormalities of inhibitory control in patients with depressive disorders utilizing behavioral or neurological experiments. However, there are some problems unsolved as follows: (1) studies tend to measure inhibitory control in a single paradigm; (2) previous studies have focused more on inhibitory control deficits in clinically depressive populations and may overlook the changes in inhibitory control during the development of depression. In this study, a depression-prone population is selected to explore the characteristics of inhibitory control from two perspectives, response inhibition and interference control. In this study, we will test the response inhibition and interference control ability of the depression-prone group through two paradigms of Flanker and Go/Nogo respectively, and combined with the EEG test, we will observe the characteristics of neural activity of inhibitory control of the group by three neural indicators of N2, P3 and theta in the middle frontal area. N2 and theta oscillations in the middle frontal area characterize the conflict monitoring in the early stage of the inhibitory control, and P3 characterizes the conflict resolution. The expected results of this study are as follows: (1) the depression-prone group does not show significant differences compared with the healthy group in behavioral indices; (2) the NoGo-N2 and N2 in Flanker incongruent trials of the depression-prone group are smaller than that of the healthy group, and the P3 is smaller as well; and (3) the mid-frontal theta power during cognitive conflict is lower in the depression-prone group.

2. Methods

2.1. Participants

Recruitment for university students was conducted through the Internet and posters. Volunteers who willingly participated in the experiment were screened using the Center for Epidemiologic Studies Depression Scale (CES-D) and the Beck Depression Inventory—II—Chinese (BDI-II-C). Inclusion criteria for volunteers in this study were as follows: (1) For the depression-prone group: BDI-II-C scores greater than or equal to 15 and CES-D scores greater than or equal to 20, and not meeting the diagnostic criteria for depression according to the Chinese Classification and Diagnostic Criteria of Mental Disorders, Third Edition (CCMD-3); (2) For the healthy control group: BDI-II-C less than 15 and CES-D scores less than 20. All volunteers also needed to meet the

following criteria: right-handedness, normal or corrected-to-normal vision, no history of neurological or psychiatric disorders, no prior use of psychiatric medication, and no prior experience with psychological therapy. Before the formal initiation of the experiment, a total of 232 university students were screened, and 65 individuals initially met the criteria for the depression-prone group. Among them, 43 individuals ultimately passed the screening and participated in the experiment. Additionally, 45 healthy participants were selected from the initial pool of 232 university students to serve as controls. During the course of the experiment, 2 participants from the healthy group and 1 participant from the depression-prone group withdrew from the study. Furthermore, the data from 2 participants in the healthy group and 1 participant in the depression-prone group were deemed ineligible for further analysis due to substantial artifacts in the EEG data. Therefore, the final analysis included data from 41 participants with depression tendencies and 41 healthy participants, with ages ranging from 18 to 27 years. All volunteers signed informed consent forms before the start of the experiment and received compensation upon completion. This study was approved by the Ethical Evaluation of Research Projects of the Department of Psychology in the School for Social and Behavioral Sciences at Nanjing University. All participants provided informed written consent and received a cash payment at the end of the experiment.

2.2. Flanker task

This study used the classic arrow Flanker paradigm to measure interference control (as shown in Fig. 1). During the task, participants sat in front of a 21-inch display screen at a distance of approximately 70 centimeters. The formal experiment consisted of 4 blocks, with each phase containing 80 trials. In each trial, a row of 5 arrows was presented simultaneously for a duration of 200 ms. Half of the arrows were congruent, with no visual conflict, while the other half were incongruent, involving visual conflicts. The inter-trial interval fluctuated between 2000 and 2400 ms, and the presentation order was random. Volunteers were required to quickly determine the direction of the middle arrow, pressing the "F" key on the keyboard if the middle arrow pointed to the left, and the "K" key if it pointed to the right. To ensure that volunteers understood the task rules, they needed to complete 16 practice trials before the formal experiment, achieving an accuracy of 75%.

窗体顶端.

2.3. Go/NoGo task

This study employed a classic letter Go/NoGo paradigm to measure response inhibition (as illustrated in Fig. 1). During the task, participants sat in front of a 21-inch display screen at a distance of approximately 70 centimeters. The formal experimental procedure consisted of 2 blocks, with each block containing 240 trials. Within each block, there were 200 trials in the Go condition and 40 trials in the NoGo condition: in the Go condition, participants were required to respond as quickly as possible by pressing the spacebar when the letter "X" appeared in the center of the screen; in the NoGo condition, the letter "Y" appeared in the center of the screen, and participants were instructed to simply gaze at the letter without making a response. The letter stimuli were presented for 200 ms, and the inter-trial interval varied between 2500 ms and 3000 ms. To ensure that volunteers understood the task rules, they underwent 24 practice trials before the formal experiment, and they were required to achieve an accuracy of 75%.

2.4. EEG data collection and analysis

During the Flanker and Go/NoGo paradigms, EEG data were collected using Curry 8–40 scalp electrodes placed according to the International 10–20 system. Scalp EEG was recorded using Ag/AgCl electrodes, with bilateral mastoids as the reference and AFz as ground lead. The bandpass filter settings were 0.01–100 Hz, with a sampling rate of 1000 Hz, and scalp impedance was kept below 10 kΩ.

EEG data was processed using the EEGLAB 14.1.1b toolbox on the Matlab 2018a platform (Delorme & Makeig, 2004). The offline analysis involved using bilateral mastoids as reference, applying a 30 Hz low-pass filter and a 0.1 Hz high-pass filter, and segmenting the data from 1000 ms before stimulus onset to 1500 ms after stimulus onset. The data of bad electrode sites was replaced with the arithmetic mean of adjacent electrode sites using the linear interpolation method, and epochs with large drift at any electrode were manually removed. Eye movements, head movements, cardiac artifacts, and other components in the EEG data were manually identified and removed using the Independent Component Analysis (ICA) algorithm. Finally, any trials with amplitude values exceeding ± 75μV on any electrode were excluded.

For event-related potentials (ERP) data analysis, baseline correction was applied using the − 200 ms-0 ms time window before stimulus onset. Based on previous literature (Folstein & Van Petten, 2008; Wei et al., 2022) and visual inspection, the analysis electrodes for the N2

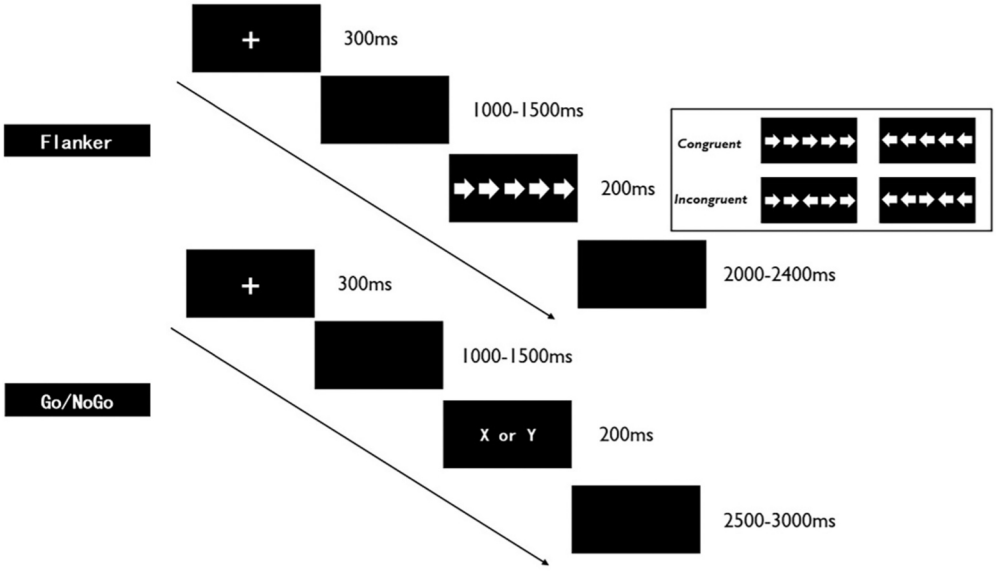

Fig. 1. Schematic representation of Flanker and Go/NoGo task.

component in both Go/NoGo and Flanker paradigms were selected as Fz, and the time window was set from 250 ms to 350 ms after stimulus onset. The analysis electrodes for the P3 component were CPz, and the time window was set from 300 ms to 600 ms after stimulus onset.

Time–frequency distributions of EEG trials were obtained using a windowed Fourier transform (WFT) with a fixed 200 ms Hanning window. This was done using the Letswave7 toolbox (Mouraux & Iannetti, 2008), in the frequency range of 1–30 Hz, with a 1 Hz step, and data from − 1000 ms to 1500 ms were analyzed with a 1 ms step. To obtain average time-frequency representation, single-trial time-frequency data were averaged. To analyze the changes in energy of post-stimulus brain activity in the time-frequency representation, Event-Related Spectral Perturbation (ERSP) was calculated for each time-frequency point. The resulting spectrogram, $P(t, f) = ||F(t, f)||^2$, which represents the signal power as a joint function of time and frequency at each time–frequency point, contained brain responses both phase-locked (ERP) and non-phase-locked (event-related synchronization and desynchronization, ERS and ERD, respectively) to laser stimulation. Based on previous literature (Cavanagh & Frank, 2014; Nigbur et al., 2011; Wei et al., 2022) and visual inspection, analysis of theta power in the Flanker and Go/NoGo tasks was conducted using EEG data from the Fz electrode, in the frequency range of 4–7 Hz. The time window was set from 200 ms to 400 ms after stimulus onset.

2.5. Statistical analysis

The statistical analysis was performed using SPSS 26.0 software. Independent samples t-tests were conducted for the scores on the Center for Epidemiologic Studies Depression Scale (CES-D) and the Beck Depression Inventory-II-C (BDI-II-C).

For the Flanker paradigm, repeated measures analysis of variance (ANOVA) was used to analyze reaction times, accuracy, N2 amplitude, P3 amplitude, and theta power, with a 2 (Group: depression-prone group, healthy group) * 2 (Task condition: congruent stimulus, incongruent stimulus) design. If the assumption of sphericity was violated in the repeated measures ANOVA, the Greenhouse-Geisser correction was applied. The significance level was set at $p < 0.05$, and effect sizes were reported using ηp^2.

For the Go/NoGo paradigm, repeated measures ANOVA was also used to analyze reaction times, accuracy, N2 component amplitude, P3 component amplitude, and theta band energy with a 2 (Group: depression-prone group, healthy group) * 2 (Task condition: Go condition, NoGo condition) design. Similarly, if the sphericity assumption was violated, the Greenhouse-Geisser correction was applied. The significance level was set at $p < 0.05$, and effect sizes were reported using ηp^2. Post hoc multiple comparisons were conducted using the Bonferroni test.

3. Results

3.1. CES-D and BDI-II-C

The results of the demographic variables and scores on the Center for Epidemiologic Studies Depression Scale (CES-D) and the Beck Depression Inventory-II (BDI-II) are presented in Table 1. The results indicate that there were no significant differences in age between the two groups *($t = -1.62$, $p = .277$)*, and there were no differences in gender distribution *($X^2 = 2.242$, $p = .134$)*.

However, there were significant differences in the depression questionnaire scores between the two groups. The depression-prone group had significantly higher CES-D scores compared to the healthy group *($t = -16.47$, $p < .001$)*, and the depression-prone group had significantly higher BDI-II scores compared to the healthy group *($t = -13.25$, $p < .001$)*.

3.2. Flanker

3.2.1. Behavioral results

The behavioral results of the Flanker task for the depression-prone group and the healthy group are presented in Table 2. A repeated measures analysis of variance (ANOVA) was conducted for reaction time and accuracy of the Flanker task with a 2 (Group: depression-prone, healthy) * 2 (Task condition: congruent stimulus, incongruent stimulus) design.

The results showed that the main effect of group was not significant, $F(1, 80) = 1.148$, $p = .288$, $\eta_p^2 = .027$, indicating that there were no significant differences in reaction time between both groups. However, the main effect of task condition was significant, $F(1, 80) = 18.266$, $p < .000$, $\eta_p^2 = .291$, indicating that reaction time was significantly slower in the incongruent condition compared to the congruent condition. The interaction effect of Group * Task condition was not significant, $F(1, 80) = 1.664$, $p = .2$, $\eta_p^2 = .019$.

A repeated measures ANOVA was also conducted for accuracy in the Flanker task with the same 2 (Group: depression-prone, healthy) * 2 (Task condition: congruent stimulus, incongruent stimulus) design. The results showed that the main effect of Group was not significant, $F(1, 80) = .438$, $p = .51$, $\eta_p^2 = .005$, indicating that there were no significant differences in accuracy between both groups. However, the main effect of Task condition was significant, $F(1, 80) = 5.691$, $p = .037$, $\eta_p^2 = .112$, indicating that accuracy was significantly lower in the incongruent condition compared to the congruent condition. The interaction effect of Group * Task condition was not significant, $F(1, 80) = 1.576$, $p = .213$, $\eta_p^2 = .031$.

In addition, we explored the potential influence of gender and did not find the interaction effect of gender, group and task conditions, (Fs < 1.700, ps >.200).

3.2.2. ERP

The ERP results are shown in Fig. 2. A repeated measures analysis of variance (ANOVA) was conducted for the N2 component amplitude with a 2 (Group: depression-prone, healthy) * 2 (Task condition: congruent stimulus, incongruent stimulus).

The results revealed a significant main effect of Group, *$F(1, 80) = 9.817$, $p = .002$, $\eta_p^2 = .111$*, indicating that the N2 component amplitude was greater in the healthy group compared to the depression-prone group. There was also a significant main effect of Task condition, *$F(1, 80) = 7.541$, $p = .007$, $\eta_p^2 = .087$*, showing that the N2 component amplitude was greater in the incongruent condition compared to the congruent condition. Moreover, there was a significant interaction effect of Group * Task condition, *$F(1, 80) = 4.370$, $p = .040$, $\eta_p^2 = .052$*. Further simple effect analysis revealed that in the incongruent

Table 1
Demographic variables, CES-D and BDI-II scores of both groups (M ± SD).

	Depression-prone N = 41	Healthy N = 41	*p*
Age	22.19 ± 3.68	22.45 ± 4.14	.277
Gender (Female%)	60%	52.5%	.134
CES-D scores	29.47 ± 4.98	16.89 ± 3.90	< .001
BDI-IIscores	22.21 ± 5.58	4.66 ± 4.39	< .001

Table 2
Behavioral results of Flanker task (M±SD).

	Depression-prone N = 41	Healthy N = 41
Congruent RT (ms)	420.98 ± 58.17	411.18 ± 51.00
Imcongruent RT (ms)	471.70 ± 70.00	460.97 ± 44.39
Congruent ACC (%)	.96 ± .060	.98 ± .039
Imcongruent ACC (%)	.90 ± .088	.92 ± .075

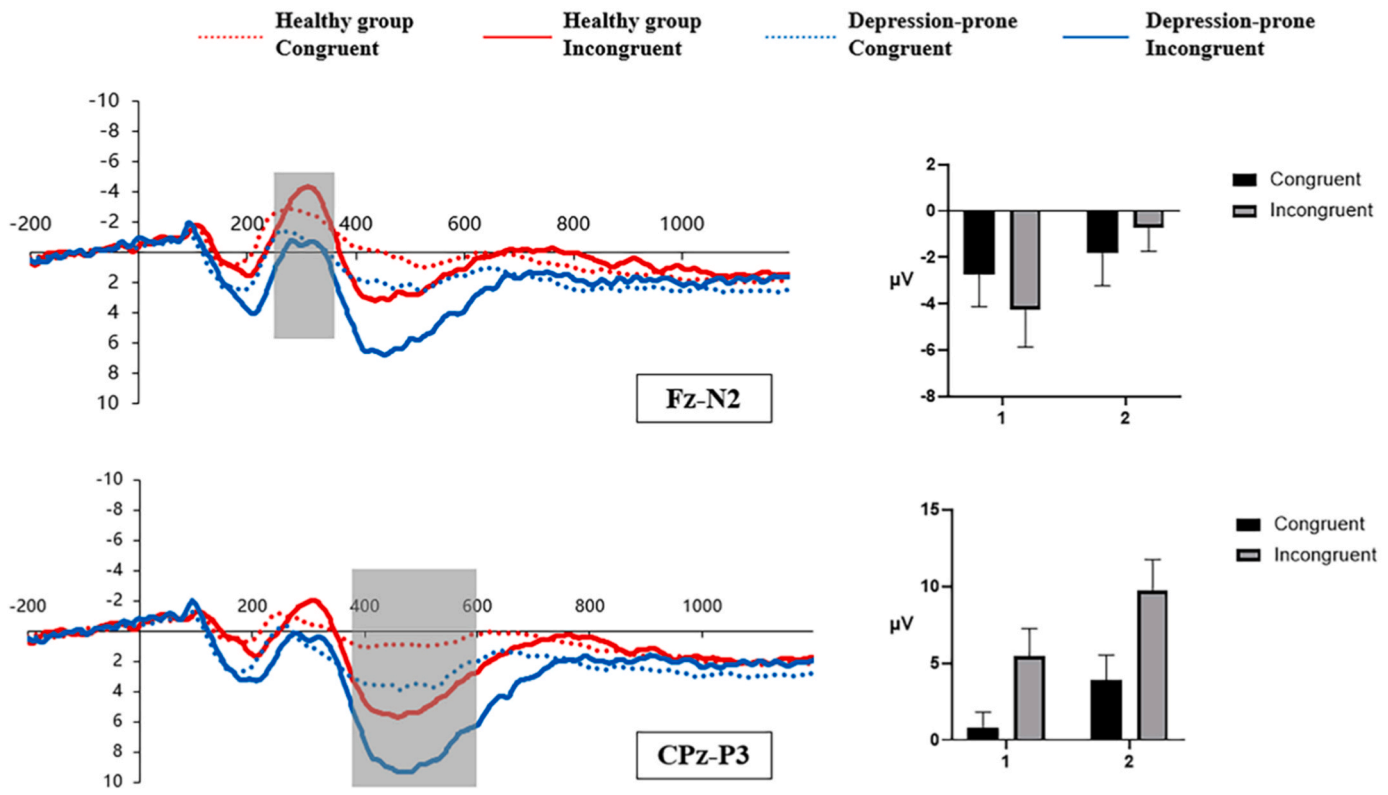

Fig. 2. Flanker related ERP waveforms at electrode site FZ and CPz.

condition, the N2 component amplitude was significantly greater in the healthy group compared to the depression-prone group, $F(1, 80) = 14.70$, $p < .001$, $\eta_p^2 = .157$.

Similar analyses were conducted for the P3 component amplitude, and the results indicated a significant main effect of Group, $F(1, 80) = 12.872$, $p < .001$, $\eta_p^2 = .140$, with the depression-prone group showing greater P3 component amplitude compared to the healthy group. There was also a significant main effect of Task condition, $F(1, 80) = 5.898$, $p = .024$, $\eta_p^2 = .080$, with the P3 component amplitude being greater in the incongruent condition compared to the congruent condition. However, there was no significant interaction effect between Group and Task condition for the P3 component amplitude.

For the peak latency, we conducted statistical analyses consistent with those for the N2 and P3 amplitude, and found no differences in the peak latency. Hereby, we also explored the potential influence of gender factors and did not find the interaction effect of gender, group and task conditions (Fs < 1, ps >.320).

3.2.3. *Theta Power*

The time-frequency results are depicted in Fig. 3. A repeated

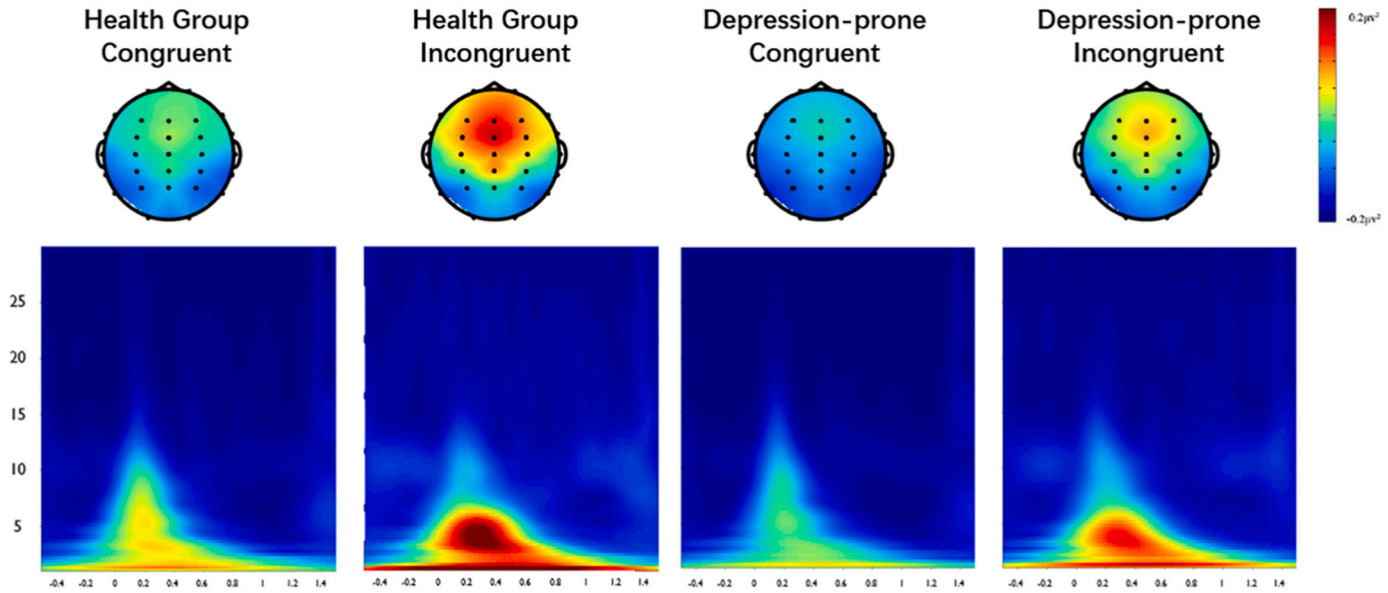

Fig. 3. The spectrograms of the mean power at electrode Fz and the topographies of theta power between 200 ms and 400 ms in the Flanker paradigm.

measures analysis of variance (ANOVA) was conducted for theta power with a 2 (Group: depression-prone, healthy) * 2 (Task condition: congruent stimulus, incongruent stimulus).

The results showed a significant main effect of Group, $F(1, 80) = 8.400$, $p = .012$, $\eta_p^2 = .095$, indicating that theta power was greater in the healthy group compared to the depression-prone group. There was also a significant main effect of Task condition, $F(1, 80) = 7.065$, $p = .018$, $\eta_p^2 = .084$, showing that theta power was greater in the incongruent condition compared to the congruent condition. Furthermore, there was a significant interaction effect of Group * Task condition, $F(1, 80) = 4.837$, $p = .033$, $\eta_p^2 = .061$. Further simple effect analysis revealed that in the incongruent condition, theta power was significantly stronger in the healthy group compared to the depression-prone group, $F(1, 80) = 6.70$, $p = .022$, $\eta_p^2 = .077$. Similarly, we did not find an effect of gender here, $F(1, 160) = .440$, $p = .511$, $\eta_p^2 = .020$.

3.3. Go/NoGo

3.3.1. Behavioral results

The results indicate that there were no significant statistical differences in Go accuracy, NoGo accuracy, and Go reaction time between the depression tendency group and the healthy group. These results are presented in Table 3.

In addition, we explored the potential influence of gender and did not find the interaction effect of gender, group and task conditions (Fs < 0.8, ps >.400).

3.3.2. ERP

The ERP results are displayed in Fig. 4. A repeated measures analysis of variance (ANOVA) was performed for the N2 component amplitude with a 2 (Group: depression-prone, healthy) * 2 (Task condition: Go condition, NoGo condition) design.

The results revealed a significant main effect of Group, $F(1, 80) = 5.426$, $p = .023$, $\eta_p^2 = .078$, indicating that the N2 component amplitude was greater in the healthy group compared to the depression-prone group. There was also a significant main effect of Task condition, $F(1, 80) = 10.175$, $p < .001$, $\eta_p^2 = .122$, showing that N2 component amplitude was greater in the NoGo condition compared to the Go condition.

Furthermore, there was a significant interaction effect of Group * Task condition, $F(1, 80) = 5.285$, $p = .029$, $\eta_p^2 = .072$. Further simple effect analysis revealed that in the NoGo condition, the N2 component amplitude was significantly greater in the healthy group compared to the depression-prone group, $F(1, 80) = 6.872$, $p = .011$, $\eta_p^2 = .083$.

Similar analyses were conducted for the P3 component amplitude, and the results indicated a significant main effect of Group, $F(1, 80) = 10.786$, $p < .001$, $\eta_p^2 = .125$, with the healthy group showing smaller P3 component amplitude compared to the depression-prone group. There was also a significant main effect of Task condition, $F(1, 80) = 4.547$, $p = .037$, $\eta_p^2 = .060$, with the NoGo condition having greater P3 component amplitude compared to the Go condition.

Additionally, there was a significant interaction effect of Group * Task condition, $F(1, 80) = 5.645$, $p = .027$, $\eta_p^2 = .074$. Further simple effect analysis revealed that in the NoGo condition, the healthy group had significantly smaller P3 component amplitude compared to the depression-prone group, $F(1, 80) = 7.386$, $p = .009$, $\eta_p^2 = .085$.

For the peak latency, we conducted statistical analyses consistent with the N2 and P3 amplitude, and no differences were found in the peak latency. Moreover, We did not find the interaction effect of gender, group and task conditions (Fs < 1.860, ps >.180).

Table 3
Behavioral results of Go/NoGo task (M±SD).

	Depression-prone N = 41	Healthy N = 41	*p*
Go ACC (%)	.99 ± .02	.99 ± .01	.683
NoGo ACC (%)	.82 ± .11	.87 ± .10	.244
Go RT (ms)	360.29 ± 48.17	387.47 ± 89.36	.198

3.3.3. Theta Power

The Go/NoGo time-frequency results are presented in Fig. 5. A repeated measures analysis of variance (ANOVA) was conducted for theta power with a 2 (Group: depression-prone, healthy) * 2 (Task condition: Go condition, NoGo condition) design.

The results showed a significant main effect of Group, $F(1, 80) = 9.308$, $p = .004$, $\eta_p^2 = .102$, indicating that theta power was greater in the healthy group compared to the depression-prone group. There was also a significant main effect of Task condition, $F(1, 80) = 7.583$, $p = .007$, $\eta_p^2 = .086$, showing that theta power was greater in the NoGo condition compared to the Go condition.

Furthermore, there was a significant interaction effect of Group * Task condition, $F(1, 80) = 5.340$, $p = .028$, $\eta_p^2 = .072$. Further simple effect analysis revealed that in the incongruent condition, the healthy group had significantly stronger theta power compared to the depression-prone group, $F(1, 80) = 6.320$, $p = .020$, $\eta_p^2 = .075$. There is no effect of gender on theta oscillations, $F(1, 160) = .527$, $p = .472$, $\eta_p^2 = .009$.

4. Discussion

This study used the classic paradigms of Flanker and Go/NoGo, which measure inhibitory control functions, in conjunction with the electroencephalographic event-related potential (ERP) technique to explore inhibitory control differences between subsyndromal individuals with depressive tendency and a healthy group. It was found that, in terms of behavioral indices, individuals with depressive tendency did not show significant differences in response time and accuracy compared to the healthy group in both paradigms. However, in terms of ERP components, individuals with depressive tendency exhibited smaller N2 amplitudes and larger P3 amplitudes in the Go/NoGo paradigm's NoGo condition compared to the healthy group. They also showed smaller N2 amplitudes in the Flanker paradigm's incongruent condition. In terms of neural oscillations, individuals with depressive tendency showed lower theta power compared to the healthy group in both the Go/NoGo paradigm's NoGo condition and the Flanker paradigm's incongruent condition.

This study did not find significant differences in response times and accuracy between individuals with depressive tendency and the healthy group, and there were no similar experimental results available for comparison in previous research. In studies on depressive disorders, Alderman et al. (2015) did not find behavioral differences between individuals with depressive disorders and the healthy group. On the other hand, Dillon et al. (2015), Holmes and Pizzagalli (2008), and Li et al. (2021) found more pronounced differences, but their conclusions were not entirely consistent. Dillon et al.'s study (2015), which used the drift-diffusion model, found it challenging to directly establish the presence of a clear trade-off relationship in the current results. Therefore, it can be observed that, whether it is response inhibition or interference control, the current research faces difficulties in determining the extent of behavioral differences between individuals with depressive disorders and the healthy group. The observed discrepancies in the behavioral research results may stem from differences in the selected study participants and employed research paradigms. The characteristics of volunteers recruited in previous studies on depressive disorders, such as age, gender, and illness duration, varied to some extent, and inhibitory control abilities also undergo significant changes with age

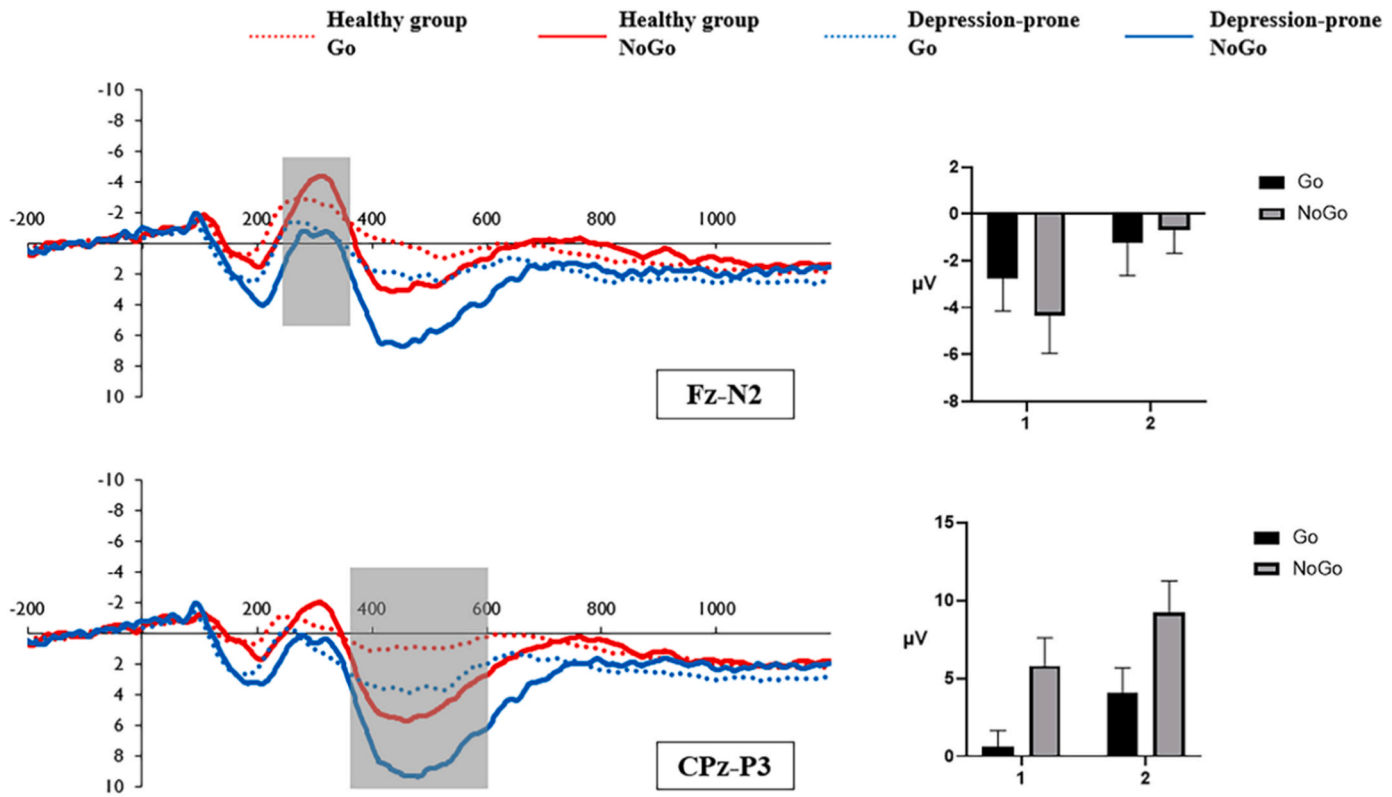

Fig. 4. Go/NoGo related ERP waveforms at electrode site FZ and CPz.

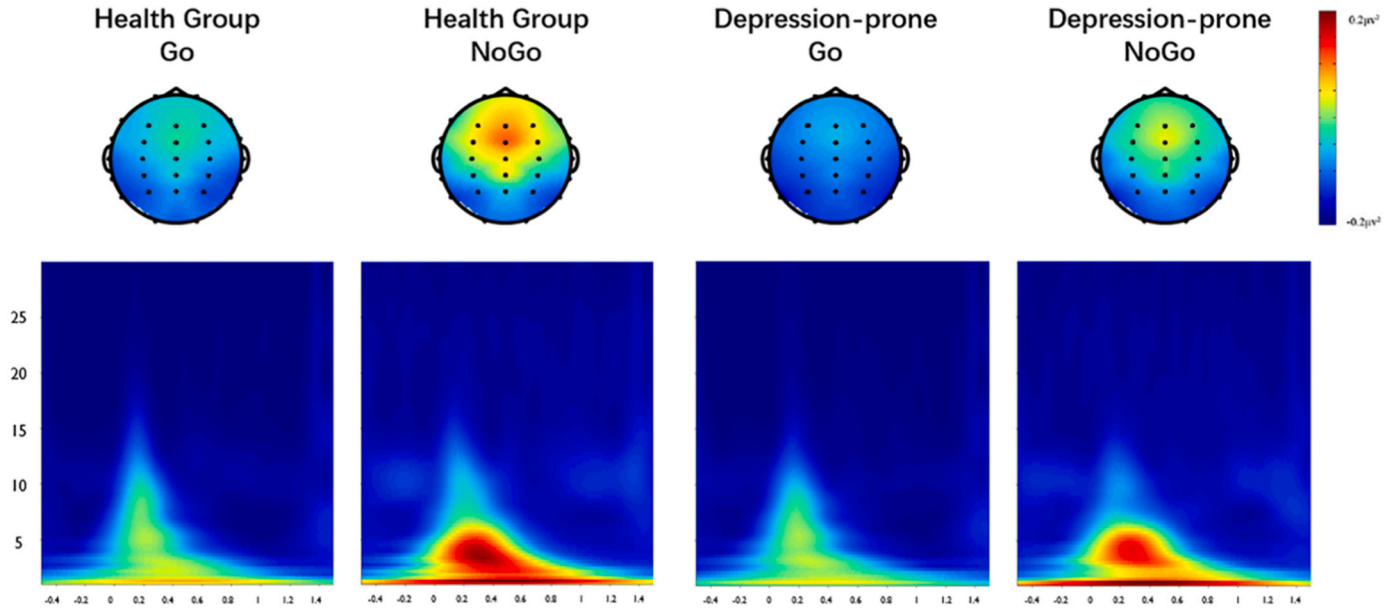

Fig. 5. The spectrograms of the mean power at electrode Fz and the topographies of theta power between 200 ms and 400 ms in the Go/NoGo paradigm.

(Hillman et al., 2006), further affecting the consistency of different research results.

Regarding EEG indices, the depression-prone group exhibited a different neurophysiological pattern during the completion of inhibitory control tasks compared to the healthy group. Previous studies that focus on depression and EEG recording often used a single inhibitory control paradigm (measuring only response inhibition or interference control) to investigate clinically diagnosed individuals with depressive disorders. The results from these studies typically showed that individuals with depressive disorders had smaller N2 and P3 amplitudes than healthy individuals under conditions of cognitive conflict (Klawohn et al., 2020; Santopetro et al., 2021; Ruchsow et al., 2008). The results of this study align with the expected findings, as individuals with depressive tendency exhibited smaller N2 amplitudes in the NoGo condition of the Go/NoGo paradigm and the incongruent condition of the Flanker paradigm. Previous research has already established that, in both response inhibition and interference control processes, an increase in N2 amplitude under conditions of cognitive conflict signifies an individual's

awareness of conflicting stimuli (Jonkman et al., 2004; Smith et al., 2010) and an early inhibitory process against inappropriate behavioral attempts before responding (Falkenstein et al., 1999). The neural source of the N2 component is the anterior cingulate cortex (Nieuwenhuis et al., 2003), which monitors the appearance of conflicting stimuli and generates stronger mid-frontal theta-band oscillations. Enhanced theta oscillations indicate an increased demand for top-down control, with theta oscillations being transmitted from the anterior cingulate cortex to the lateral frontal cortex. Subsequently, the prefrontal regions exert control and resolve conflicting stimuli (Eschmann et al., 2018; Helfrich & Knight, 2016). The inability of depression-prone individuals to exhibit the corresponding enhancement process based on task demands suggests a decrease in network connectivity between the anterior cingulate cortex, prefrontal cortex, and parietal cortex. Individuals with depressive disorders also exhibit reduced theta activity during the maintenance phase of working memory, which reflects the loss of their top-down inhibitory function (Murphy et al., 2019). Working memory and inhibitory control both fall under the category of executive functions (Diamond, 2013), and their relationship is one of mutual support (Diamond, 2013). Therefore, the results of this section also provide further evidence of the impairment caused by depression on executive functions.

Previous research in the field of depression has focused on the changes in theta activity in the anterior cingulate cortex following brain stimulation, with individuals benefiting from such interventions often exhibiting enhanced theta activity in this region (Bailey et al., 2018; Widge et al., 2019). Based on the conclusions drawn from these studies, both theta oscillations and N2 amplitudes during inhibitory control processes are neural indicators of early conflict monitoring and processing. The results of this study indicate that individuals with depressive tendency exhibit deficits in monitoring and processing conflict stimuli at the neural level, regardless of whether it's in the context of response inhibition or interference control. These findings are consistent with the previous research conducted by Alderman and colleagues in individuals diagnosed with depressive disorders in 2015. The blunting of N2 responses reflects impaired early conflict processing abilities, suggesting that deficits in early conflict monitoring and processing occur before the development of depressive disorders. Clawson et al., (2013) proposed that individuals who exhibit some symptoms of depressive disorders but do not meet the diagnostic criteria for depression also display cognitive impairments, including deficits in conflict monitoring and adaptation. The results of this section of the study corroborate the findings of that research, providing further evidence that even in individuals with depressive tendency who do not meet the diagnostic criteria for depression, there are still observable deficits in cognitive functions related to conflict processing and adaptation.

We hypothesized that the P3 amplitude in the NoGo and Flanker incongruent conditions would be smaller in the depression-prone group compared to the healthy group. However, the research results indicate that the depression-prone group exhibited larger P3 amplitudes in the NoGo condition of the Go/NoGo task compared to the healthy group, while there were no significant P3 amplitude differences in the incongruent condition of the Flanker paradigm. Larger-sample experiments involving individuals with depressive disorders have shown that P3 amplitudes in the Flanker task are significantly smaller in depressive patients (Klawohn et al., 2020). Previous studies using the Oddball paradigm (for measuring response inhibition) have found associations between increased depressive symptoms and reduced P3 amplitudes (Nan et al., 2018; Zhou et al., 2019). These previous studies were conducted on clinical depression populations, whereas the depression-prone group recruited for this study did not meet the diagnostic criteria for depressive disorders. Therefore, we speculate that a compensatory mechanism may exist as depressive symptoms gradually worsen.

The P3 amplitude represents the awareness of conflict stimuli and the subsequent control processes over the motor system after the need for inhibition arises (Kok et al., 2004). The amplitude reflects the degree of top-down engagement (Erb & Cavanagh, 2019; Groom & Cragg, 2015). Results from this study, including the N2 and theta power findings, suggest that individuals with a tendency toward depression exhibit deficits in monitoring and early processing of conflict stimuli during inhibitory control, with insufficient early attentional resources. Since the depressive symptom is not yet severe, despite their deficits in early control abilities, individuals with depressive tendency may compensate by allocating more cognitive resources during subsequent inhibitory control processes, which could explain why their behavioral responses do not appear weaker than those of the healthy group.

In a study conducted with bipolar disorder individuals (Morsel et al., 2017), it was found that they exhibited larger P3 amplitudes in the NoGo condition compared to the healthy group, while their N2 amplitudes during NoGo were significantly smaller than those of the healthy group, which is consistent with the results of our study. Previous research has shown that compared to bipolar disorder, both depression and bipolar disorder are more likely to produce cognitive deficits due to excessive negative emotional perception (Martínez-Arán et al., 2011; Morsel et al., 2018). Negative emotional perception makes individuals with depression excessively concerned about their behavioral performance and prone to avoiding errors (Langenecker et al., 2007). Some researchers (Morsel et al., 2014) have found that depression increases the amplitude of error-related negativity, which reflects an individual's perception of errors and the monitoring process of conflicts. The similarities in inhibitory control patterns between individuals with depressive tendency and those with bipolar disorder may suggest that the group selected through depression scales may not necessarily develop into depressive disorders, and differences in early diagnosis and intervention strategies should be considered among different disorders.

At present, there is a lack of dynamic longitudinal data in research, which limits researchers' ability to dynamically observe inhibitory control functions in populations at risk for mental disorders and to distinguish between different mental disorders. Cognitive function impairment has a significant impact on the emotional and social functioning of individuals with depressive disorders; therefore, the N2 and P3 components in EEG may serve as a biomarker for depressive disorders. These two indicators may be used to predict the development of depressive disorders and recovery after intervention. This study found that the inhibitory control function of individuals with depressive tendency is negatively affected earlier than their interference control function, and individuals with depressive tendency have already shown some compensatory phenomena during inhibitory control.

5. Conclusion

The study found that individuals with a tendency towards depression exhibited smaller N2 amplitudes and larger P3 amplitudes in the NoGo condition of the Go/NoGo paradigm compared to the healthy group. They also showed smaller N2 amplitudes in the incongruent condition of the Flanker paradigm. Regarding neural oscillations, the depression-prone group displayed lower theta power in the NoGo condition of the Go/NoGo paradigm and the incongruent condition of the Flanker paradigm. This study suggests that individuals with a tendency towards depression have a compensatory mechanism in their response inhibition function. This compensation is characterized by a deficit in the early processing of conflict awareness, but they compensate by overactivation during the control execution phase to achieve task control.

CRediT authorship contribution statement

Wang fang: Writing – original draft, Validation, Formal analysis, Data curation, Conceptualization. **Zhou Renlai:** Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization. **Zhou Weiyi:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Formal analysis, Data curation, Conceptualization. **Long**

Fangfang: Writing – original draft, Visualization, Validation, Methodology, Formal analysis.

Declaration of Generative AI and AI-assisted technologies in the writing process

The author(s) did not use generative AI technologies for preparation of this work.

Declaration of Competing Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Data Availability

Data will be made available on request.

Acknowledgements

This research was supported by Space Medical Experiment Project of China Manned Space Program (HYZHXM03008) and Brain Health Program of Jiangsu Boerya Science and Technology (2024200013).

References

Alderman, B. L., Olson, R. L., Bates, M. E., Selby, E. A., Buckman, J. F., Brush, C. J., Panza, E. A., Kranzler, A., Eddie, D., & Shors, T. J. (2015). Rumination in major depressive disorder is associated with impaired neural activation during conflict monitoring. *Frontiers in Human Neuroscience, 9*, 269. https://doi.org/10.3389/fnhum.2015.00269

Bailey, A. P., Hetrick, S. E., Rosenbaum, S., Purcell, R., & Parker, A. G. (2018). Treating depression with physical activity in adolescents and young adults: a systematic review and meta-analysis of randomised controlled trials. *Psychological Medicine, 48*(7), 1068–1083. https://doi.org/10.1017/S0033291717002653

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological review, 108*(3), 624–652. https://doi.org/10.1037/0033-295x.108.3.624.

Buzsáki, G., & Draguhn, A. (2004). Neuronal oscillations in cortical networks. *Science (New York, N York), 304*(5679), 1926–1929. https://doi.org/10.1126/science.1099745

Cambridge, O. R., Knight, M. J., Mills, N., & Baune, B. T. (2018). The clinical relationship between cognitive impairment and psychosocial functioning in major depressive disorder: A systematic review. *Psychiatry Research, 269*, 157–171. https://doi.org/10.1016/j.psychres.2018.08.033

Cavanagh, J. F., & Frank, M. J. (2014). Frontal theta as a mechanism for cognitive control. *Trends in Cognitive Sciences, 18*(8), 414–421. https://doi.org/10.1016/j.tics.2014.04.012

Cheng, C. H., Tsai, H. Y., & Cheng, H. N. (2019). The effect of age on N2 and P3 components: A meta-analysis of Go/Nogo tasks. *Brain and cognition, 135*, Article 103574. https://doi.org/10.1016/j.bandc.2019.05.012

Chmielewski, W. X., & Beste, C. (2017). Testing interactive effects of automatic and conflict control processes during response inhibition - A system neurophysiological study. *NeuroImage, 146*, 1149–1156. https://doi.org/10.1016/j.neuroimage.2016.10.015

Clawson, A., Clayson, P. E., & Larson, M. J. (2013). Cognitive control adjustments and conflict adaptation in major depressive disorder. *Psychophysiology, 50*(8), 711–721. https://doi.org/10.1111/psyp.12066

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods, 134*(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Diamond, A. (2013). Executive functions. *Annual Review of Psychology, 64*, 135–168. https://doi.org/10.1146/annurev-psych-113011-143750

Dillon, D. G., Wiecki, T., Pechtel, P., Webb, C., Goer, F., Murray, L., Trivedi, M., Fava, M., McGrath, P. J., Weissman, M., Parsey, R., Kurian, B., Adams, P., Carmody, T., Weyandt, S., Shores-Wilson, K., Toups, M., McInnis, M., Oquendo, M. A., Cusin, C., … Pizzagalli, D. A. (2015). A computational analysis of flanker interference in depression. *Psychological Medicine, 45*(11), 2333–2344. https://doi.org/10.1017/S0033291715000276

Erb, C. D., & Cavanagh, J. F. (2019). Layers of latent effects in cognitive control: An EEG investigation. *Acta psychologica, 195*, 1–11. https://doi.org/10.1016/j.actpsy.2019.02.004

Eschmann, K. C. J., Bader, R., & Mecklinger, A. (2018). Topographical differences of frontal-midline theta activity reflect functional differences in cognitive control abilities. *Brain and cognition, 123*, 57–64. https://doi.org/10.1016/j.bandc.2018.02.002

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

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: a review. *Psychophysiology, 45*(1), 152–170. https://doi.org/10.1111/j.1469-8986.2007.00602.x

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: a latent-variable analysis. *Journal of Experimental Psychology General, 133*(1), 101–135. https://doi.org/10.1037/0096-3445.133.1.101

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: current status and future directions. *Annual Review of Clinical Psychology, 6*, 285–312. https://doi.org/10.1146/annurev.clinpsy.121208.131305

Groom, M. J., & Cragg, L. (2015). Differential modulation of the N2 and P3 event-related potentials by response conflict and inhibition. *Brain and cognition, 97*, 1–9. https://doi.org/10.1016/j.bandc.2015.04.004

Helfrich, R. F., & Knight, R. T. (2016). Oscillatory Dynamics of Prefrontal Cognitive Control. *Trends in Cognitive Sciences, 20*(12), 916–930. https://doi.org/10.1016/j.tics.2016.09.007

Hillman, C. H., Kramer, A. F., Belopolsky, A. V., & Smith, D. P. (2006). A cross-sectional examination of age and physical activity on performance and event-related brain potentials in a task switching paradigm. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology, 59*(1), 30–39. https://doi.org/10.1016/j.ijpsycho.2005.04.009

Holmes, A. J., & Pizzagalli, D. A. (2008). Response conflict and frontocingulate dysfunction in unmedicated participants with major depression. *Neuropsychologia, 46*(12), 2904–2913. https://doi.org/10.1016/j.neuropsychologia.2008.05.028

Hoffmann, S., & Beste, C. (2015). A perspective on neural and cognitive mechanisms of error commission. *Frontiers in Behavioral Neuroscience, 9*, 50. https://doi.org/10.3389/fnbeh.2015.00050

Hsieh, M. T., Lu, H., Chen, L. F., Liu, C. Y., Hsu, S. C., & Cheng, C. H. (2022). Cancellation but not restraint ability is modulated by trait anxiety: An event-related potential and oscillation study using Go-Nogo and stop-signal tasks. *Journal of Affective Disorders, 299*, 188–195. https://doi.org/10.1016/j.jad.2021.11.066

Jonkman, L. M., Kenemans, J. L., Kemner, C., Verbaten, M. N., & van Engeland, H. (2004). Dipole source localization of event-related brain activity indicative of an early visual selective attention deficit in ADHD children. *Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology, 115*(7), 1537–1549. https://doi.org/10.1016/j.clinph.2004.01.022.

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: relation to cognitive inhibition. *Cognition & Emotion, 24*(2), 281–298. https://doi.org/10.1080/02699930903407948

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, 122*(3), 169–184. https://doi.org/10.1016/s0925-4927(03)00004-0

Kato, R., Balasubramani, P. P., Ramanathan, D., & Mishra, J. (2022). Utility of Cognitive Neural Features for Predicting Mental Health Behaviors. *Sensors (Basel, Switzerland), 22*(9), 3116. https://doi.org/10.3390/s22093116

Klawohn, J., Santopetro, N. J., Meyer, A., & Hajcak, G. (2020). Reduced P300 in depression: Evidence from a flanker task and impact on ERN, CRN, and Pe. *Psychophysiology, 57*(4), Article e13520. https://doi.org/10.1111/psyp.13520

Kok, A., Ramautar, J. R., De Ruiter, M. B., Band, G. P., & Ridderinkhof, K. R. (2004). ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology, 41*(1), 9–20. https://doi.org/10.1046/j.1469-8986.2003.00127.x

Langenecker, S. A., Kennedy, S. E., Guidotti, L. M., Briceno, E. M., Own, L. S., Hooven, T., Young, E. A., Akil, H., Noll, D. C., & Zubieta, J. K. (2007). Frontal and limbic activation during inhibitory control predicts treatment response in major depressive disorder. *Biological Psychiatry, 62*(11), 1272–1280. https://doi.org/10.1016/j.biopsych.2007.02.019

Larson, M. J., Clayson, P. E., & Clawson, A. (2014). Making sense of all the conflict: a theoretical review and critique of conflict-related ERPs. *International Journal of Psychophysiology, 93*(3), 283–297. https://doi.org/10.1016/j.ijpsycho.2014.06.007

Li, F. F., Chen, X. L., Zhang, Y. T., Li, R. T., & Li, X. (2021). The role of prepotent response inhibition and interference control in depression. *Cognitive Neuropsychiatry, 26*(6), 441–454. https://doi.org/10.1080/13546805.2021.1987872

Liu, Q., He, H., Yang, J., Feng, X., Zhao, F., & Lyu, J. (2020). Changes in the global burden of depression from 1990 to 2017: Findings from the Global Burden of Disease study. *Journal of Psychiatric Research, 126*, 134–140. https://doi.org/10.1016/j.jpsychires.2019.08.002

López Zunini, R. A., Knoefel, F., Lord, C., Dzuali, F., Breau, M., Sweet, L., Goubran, R., & Taler, V. (2016). Event-related potentials elicited during working memory are altered in mild cognitive impairment. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology, 109*, 1–8. https://doi.org/10.1016/j.ijpsycho.2016.09.012

MacQueen, G. M., Tipper, S. P., Young, L. T., Joffe, R. T., & Levitt, A. J. (2000). Impaired distractor inhibition on a selective attention task in unmedicated, depressed subjects. *Psychological Medicine, 30*(3), 557–564. https://doi.org/10.1017/s0033291799001981

Martínez-Arán, A., Torrent, C., Solé, B., Bonnín, C. M., Rosa, A. R., Sánchez-Moreno, J., & Vieta, E. (2011). Functional remediation for bipolar disorder. *Clinical Practice and Epidemiology in Mental health: CP & EMH, 7*, 112–116. https://doi.org/10.2174/1745017901107010112

Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions: Four general conclusions. *Current Directions in psychological Science, 21*(1), 8–14. https://doi.org/10.1177/0963721411429458

Morsel, A. M., Dhar, M., Hulstijn, W., Temmerman, A., Morrens, M., & Sabbe, B. (2017). Inhibitory control in euthymic bipolar disorder: Event related potentials during a Go/NoGo task. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology, 128*(4), 520–528. https://doi.org/10.1016/j.clinph.2016.12.006

Morsel, A. M., Morrens, M., Dhar, M., & Sabbe, B. (2018). Systematic review of cognitive event related potentials in euthymic bipolar disorder. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology, 129*(9), 1854–1865. https://doi.org/10.1016/j.clinph.2018.05.025

Morsel, A. M., Morrens, M., Temmerman, A., Sabbe, B., & de Bruijn, E. R. (2014). Electrophysiological (EEG) evidence for reduced performance monitoring in euthymic bipolar disorder. *Bipolar disorders, 16*(8), 820–829. https://doi.org/10.1111/bdi.12256.

Mouraux, A., & Iannetti, G. D. (2008). Across-trial averaging of event-related EEG responses and beyond. *Magnetic Resonance Imaging, 26*(7), 1041–1054. https://doi.org/10.1016/j.mri.2008.01.011

Murphy, O. W., Hoy, K. E., Wong, D., Bailey, N. W., Fitzgerald, P. B., & Segrave, R. A. (2019). Individuals with depression display abnormal modulation of neural oscillatory activity during working memory encoding and maintenance. *Biological Psychology, 148*, Article 107766. https://doi.org/10.1016/j.biopsycho.2019.107766

Nan, C., Wang, G., Wang, H., Wang, X., Liu, Z., Xiao, L., Bai, H., & Wu, S. (2018). The P300 component decreases in a bimodal oddball task in individuals with depression: An event-related potentials study. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology, 129*(12), 2525–2533. https://doi.org/10.1016/j.clinph.2018.09.012

Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: effects of response conflict and trial type frequency. *Cognitive, Affective & Behavioral Neuroscience, 3*(1), 17–26. https://doi.org/10.3758/cabn.3.1.17

Nigbur, R., Ivanova, G., & Stürmer, B. (2011). Theta power as a marker for cognitive interference. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology, 122*(11), 2185–2194. https://doi.org/10.1016/j.clinph.2011.03.030

Overbye, K., Walhovd, K. B., Fjell, A. M., Tamnes, C. K., & Huster, R. J. (2021). Electrophysiological and behavioral indices of cognitive conflict processing across adolescence. *Developmental Cognitive Neuroscience, 48*, Article 100929. https://doi.org/10.1016/j.dcn.2021.100929

Pires, L., Leitão, J., Guerrini, C., & Simões, M. R. (2014). Event-related brain potentials in the study of inhibition: cognitive control, source localization and age-related modulations. *Neuropsychology Review, 24*(4), 461–490. https://doi.org/10.1007/s11065-014-9275-4

Richards, D. A., & Borglin, G. (2011). Implementation of psychological therapies for anxiety and depression in routine practice: two year prospective cohort study. *Journal of Affective Disorders, 133*(1-2), 51–60. https://doi.org/10.1016/j.jad.2011.03.024

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: Official Journal of the International Organization of Psychophysiology, 68*(3), 209–218. https://doi.org/10.1016/j.ijpsycho.2008.01.010

Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., & Posner, M. I. (2004). Development of attentional networks in childhood. *Neuropsychologia, 42*(8), 1029–1040. https://doi.org/10.1016/j.neuropsychologia.2003.12.012

Santopetro, N. J., Brush, C. J., Burani, K., Bruchnak, A., & Hajcak, G. (2021). Doors P300 moderates the relationship between reward positivity and current depression status in adults. *Journal of Affective Disorders, 294*, 776–785. https://doi.org/10.1016/j.jad.2021.07.091

Schoenberg, P. L., & Speckens, A. E. (2014). Modulation of induced frontocentral theta (Fm-θ) event-related (de-)synchronisation dynamics following mindfulness-based cognitive therapy in Major Depressive Disorder. *Cognitive Neurodynamics, 8*(5), 373–388. https://doi.org/10.1007/s11571-014-9294-0

Shimony, O., Einav, N., Bonne, O., Jordan, J. T., Van Vleet, T. M., & Nahum, M. (2021). The association between implicit and explicit affective inhibitory control, rumination and depressive symptoms. *Scientific Reports, 11*(1), Article 11490. https://doi.org/10.1038/s41598-021-90875-3

Smith, J. L., Smith, E. A., Provost, A. L., & Heathcote, A. (2010). Sequence effects support the conflict theory of N2 and P3 in the Go/NoGo task. *International journal of psychophysiology : official journal of the International Organization of Psychophysiology, 75*(3), 217–226. https://doi.org/10.1016/j.ijpsycho.2009.11.002.

Vahid, A., Mückschel, M., Neuhaus, A., Stock, A. K., & Beste, C. (2018). Machine learning provides novel neurophysiological features that predict performance to inhibit automated responses. *Scientific Reports, 8*(1), Article 16235. https://doi.org/10.1038/s41598-018-34727-7

Wei, H., Beuckelaer, A., & Zhou, R. (2022). EEG correlates of neutral working memory training induce attentional control improvements in test anxiety. *Biological Psychology, 174*, Article 108407. https://doi.org/10.1016/j.biopsycho.2022.108407

Widge, A. S., Zorowitz, S., Basu, I., Paulk, A. C., Cash, S. S., Eskandar, E. N., Deckersbach, T., Miller, E. K., & Dougherty, D. D. (2019). Deep brain stimulation of the internal capsule enhances human cognitive control and prefrontal cortex function. *Nature Communications, 10*(1), Article 1536. https://doi.org/10.1038/s41467-019-09557-4

Wiecki, T. V., & Frank, M. J. (2013). A computational model of inhibitory control in frontal cortex and basal ganglia. *Psychological Review, 120*(2), 329–355. https://doi.org/10.1037/a0031542

Yitzhak, N., Shimony, O., Oved, N., Bonne, O., & Nahum, M. (2023). Less inhibited and more depressed? The puzzling association between mood, inhibitory control and depressive symptoms. *Comprehensive Psychiatry, 124*, Article 152386. https://doi.org/10.1016/j.comppsych.2023.152386

Zhou, L., Wang, G., Nan, C., Wang, H., Liu, Z., & Bai, H. (2019). Abnormalities in P300 components in depression: an ERP-sLORETA study. *Nordic Journal of Psychiatry, 73*(1), 1–8. https://doi.org/10.1080/08039488.2018.1478991