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Modulating inhibitory control in test-anxious individuals via tDCS: An ERP study

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ABSTRACT

Test anxiety is a common emotional problem that often negatively affects academic performance. To examine the potential of transcranial direct current stimulation (tDCS) in modulating neural inhibitory control efficiency in individuals with high test anxiety, this study investigated the intervention effects and underlying neural mechanisms. A total of 42 participants with high or low levels of test anxiety were recruited. Each participant received both active (anodal) and sham tDCS targeting the left dorsolateral prefrontal cortex (DLPFC), while event-related potentials (ERPs) were recorded during a Flanker task that indexes inhibitory control by requiring participants to respond to a central target while ignoring distracting flankers. Results showed that, compared to sham stimulation, active tDCS significantly reduced the amplitude of the P3 component—a late positive potential associated with attentional allocation—in the high test anxiety group, but had no significant effect in the low test anxiety group. These findings suggest that tDCS modulates neural inhibitory control in individuals with high test anxiety by activating the left DLPFC. This study provides electrophysiological evidence for non-invasive neuromodulation as a potential intervention strategy and identifying a promising neural target.

1. Introduction

Test anxiety refers to the physiological and behavioral responses individuals exhibit in evaluative situations, often accompanied by concerns about potential negative outcomes or failure (Zeidner, 1998). It is a situation-specific form of anxiety, in which individuals with high test anxiety tend to perceive evaluative contexts (such as examinations) as threatening, leading to a sustained state of heightened anxiety (Spielberger and Vagg, 1995). Globally, an estimated 15%–40% of students experience varying degrees of test anxiety (Thomas et al., 2018). In China, the prevalence of high test anxiety among university students has increased by 13.2 percentage points over the past 15 years (Huang and Zhou, 2019). Test anxiety has been shown to significantly impair cognitive control, particularly inhibitory control, which manifests as difficulties in suppressing distracting stimuli and irrelevant information (Coy et al., 2011; Sarason, 1987; Song et al., 2021; Hu et al., 2021). Such deficits in inhibition make it challenging for test-anxious individuals to focus on task-relevant content during learning and examinations, potentially leading to decreased working memory efficiency and emotional regulation difficulties, ultimately undermining academic performance (Moran, 2016). Recent theoretical developments have shifted the understanding of test anxiety from early attention bias theories to more integrative models that emphasize deficits in inhibitory control as a core mechanism (Eysenck et al., 2007; Wei et al., 2022), proposing that impaired inhibitory function in individuals with high test anxiety may underlie their broader cognitive difficulties.

Inhibitory control, a core component of executive function, is primarily responsible for suppressing irrelevant information to ensure the efficient execution of goal-directed behavior (Diamond, 2013). Its neural basis involves a distributed brain network, with the dorsolateral prefrontal cortex (DLPFC) playing a central role (Miller and Cohen, 2001). The prefrontal cortex can be conceptualized as a system of "cognitive bandwidth," wherein the capacity to allocate executive control resources is inherently limited. When individuals face multiple tasks or emotionally demanding situations, cognitive resources must be distributed among competing demands. If the demands exceed the available bandwidth, cognitive performance deteriorates (Miller and Buschman, 2015). Recent evidence suggests that test anxiety modulates neural oscillatory activity during working memory encoding, particularly reducing frontal theta power under moderate cognitive load, likely

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due to impaired top-down attentional control (Wei et al., 2025). Compared to individuals with low anxiety, those with high anxiety often exhibit a distinctive pattern of neural compensation during emotion regulation. Specifically, they rely more heavily on the DLPFC to perform functions that are typically mediated by the lateral frontal pole (Bramson et al., 2023). These functions involve the higher-order control of emotional responses, particularly the suppression of negative emotional information. This reallocation of neural resources depletes the limited cognitive bandwidth of the DLPFC, potentially reducing the efficiency of cognitive control. In the context of test anxiety, the emotionally charged nature of exams demands increased DLPFC engagement for emotional regulation. This, in turn, consumes resources that would otherwise support essential cognitive functions such as comprehension, distraction suppression, and strategy execution. Their cognitive bandwidth in the DLPFC is strained by the simultaneous demands of emotional regulation and cognitive control, resulting in impaired inhibitory function, misallocation of attentional resources, and ultimately, diminished test performance.

Although traditional interventions for test anxiety—particularly cognitive-behavioral therapy (Reiss et al., 2017; Ugwuanyi et al., 2020), as well as mindfulness training (Priebe and Kurtz-Costes, 2022), expressive writing (Relojo-Howell and Stoyanova, 2019), and working memory training (Minihan et al., 2021)—have shown some effectiveness in alleviating symptoms, these approaches primarily focus on psychological strategies and do not directly target the underlying neural mechanisms in test-anxious individuals. In light of this, transcranial direct current stimulation (tDCS) has emerged in recent years as a promising non-invasive neuromodulation technique that allows for targeted regulation of cortical excitability. Specifically, anodal stimulation increases cortical excitability, whereas cathodal stimulation reduces it (Zhou et al., 2023). In anxiety-related conditions, an imbalance in activity between the left and right DLPFC has been observed, characterized by hypoactivity in the left hemisphere and hyperactivity in the right (Grimm et al., 2008; Nitschke and Heller, 2005). This interhemispheric imbalance is thought to contribute to the processing of negative emotions and the generation of anxiety (Sagliano et al., 2016). Previous research has shown that anodal tDCS targeting the left DLPFC can significantly enhance inhibitory control (Dubreuil-Vall et al., 2019). Moreover, tDCS has been widely applied in the treatment of various anxiety-related disorders, including generalized anxiety disorder (Lin et al., 2019), social anxiety disorder (Heeren et al., 2017), and even math anxiety (Sarkar et al., 2014). These findings suggest that tDCS, by directly modulating left DLPFC function, may enhance processing efficiency and expand cognitive bandwidth, thereby improving inhibitory control. However, the specific effects of tDCS on neural inhibitory control in individuals with high test anxiety, along with its underlying electrophysiological mechanisms, remain largely unexplored.

To precisely evaluate the effects of tDCS on neural inhibitory control in individuals with high test anxiety, event-related potentials (ERPs) serve as an ideal neurophysiological tool for capturing task-related cognitive processes. Among ERP components, the N2 and P3 elicited by the Flanker task (i.e., a classic paradigm used to assess inhibitory control by requiring participants to respond to a central target while ignoring distracting flankers) are widely regarded as reliable neural markers of inhibitory control (Groom and Cragg, 2015). The N2 is a negative wave occurring approximately 200–350 ms post-stimulus, primarily generated in the anterior cingulate cortex and the DLPFC, reflecting conflict monitoring and the initiation of inhibitory processes (Siemann et al., 2016). The P3, a positive wave peaking around 300–600 ms post-stimulus, is associated with stimulus evaluation, attentional resource allocation, and the execution of inhibition (Xie et al., 2017). Prior studies using Stroop or AX-CPT task have reported increased or sustained P3 amplitudes in test-anxious individuals (Wang and Zhou, 2025; Zhang et al., 2019). However, P3 alterations in test anxiety may be task-dependent, and findings from flanker tasks appear less consistent. For example, using a flanker paradigm, Wei et al. (2022) did not observe reliable group differences in P3 amplitude between high and low test-anxiety individuals. The present study examined whether anodal tDCS over the left DLPFC modulates neural indices of inhibitory control in individuals with different levels of test anxiety. We hypothesized that, compared to sham stimulation, active tDCS would modulate ERP components associated with inhibitory control (specifically N2 and P3) during the flanker task, with effects expected to be more evident in individuals with high test anxiety.

2. Methods

2.1. Participants

This study employed a 2 (Group: High Test Anxiety vs. Low Test Anxiety) × 2 (Stimulation Type: Active vs. Sham) × 2 (Time: Pre-test vs. Post-test) mixed factorial design. Group was treated as a between-subjects factor, while stimulation type and time were treated as within-subjects factors. To estimate the required sample size for detecting a three-way interaction effect, a power analysis was conducted using G*Power 3.1 ($\alpha = 0.05$, $\beta = 0.05$; ANOVA: repeated measures, within-between interaction). Given the lack of direct prior studies on this specific population, we followed Cohen's (1988) guidelines, a medium effect size of $f = 0.25$ was assumed, yielding a required total sample size of 36 participants. The protocol of the present study was not preregistered.

Before the experiment, participants completed the following self-report measures: the Beck Depression Inventory–Second Edition (BDI-II; Beck et al., 1996), the Trait subscale of the State-Trait Anxiety Inventory (STAI-T; Spielberger et al., 1983), and the Test Anxiety Scale (TAS; Newman, 1996). In accordance with Newman's (1996) criteria, participants scoring above 20 on the TAS were assigned to the High Test Anxiety (HTA) group, and those scoring below 12 were assigned to the Low Test Anxiety (LTA) group. A total of 152 students volunteered for the study. After screening, 42 participants were included in the final sample (HTA: $n = 20$; LTA: $n = 22$). The remaining 110 participants were excluded based on the following criteria: (a) TAS scores between 12 and 20; (b) history of neurological disorders or brain injury; (c) uncorrected visual impairment or left-handedness. All participants provided written informed consent before the experiment. The study protocol was approved by the Ethics Committee of the Department of Psychology, Nanjing University.

Preliminary analyses confirmed that the two groups did not significantly differ in age, gender, depression (BDI-II), or trait anxiety (STAI-T). However, a significant group difference was found in test anxiety levels (TAS), as presented in Table 1.

2.2. Experimental procedure and task

Each participant visited the laboratory twice, with a minimum interval of 72 h between sessions. In each session, participants received

Table 1
Demographic characteristics and baseline questionnaire scores of the two groups.

Variable	HTA Group ($n = 20$)	LTA Group ($n = 22$)	Statistical Results
Age (years)	20.15 ± 2.23	21.14 ± 1.58	$t = -1.67$, $p = 0.104$
Gender (Female/Male)	8/12	12/10	$\chi 2 = 0.89$, $p = 0.346$
Depression (BDI-II)	5.00 ± 1.12	5.09 ± 1.02	$t = -0.28$, $p = 0.785$
Trait Anxiety (STAI-T)	47.45 ± 7.07	46.36 ± 5.44	$t = 0.56$, $p = 0.578$
Test Anxiety (TAS)	27.40 ± 4.76	6.00 ± 3.24	$t = 16.87$, $p < 0.001$

Note: Values are presented as M ± SD.

either active or sham tDCS. The order of stimulation (active vs. sham) was counterbalanced across participants to control for potential confounding effects due to task familiarity. Before and after each tDCS intervention, participants completed a Flanker task, during which electroencephalographic (EEG) data were recorded. The full experimental procedure is illustrated in Fig. 1. In the Flanker task, each trial consisted of an array of five white arrows presented on a black background. The central arrow served as the target, while the flanking arrows acted as distractors. Two conditions were included: Congruent condition (50%): all arrows pointed in the same direction; Incongruent condition (50%): the target arrow pointed in the opposite direction to the flanking arrows. Participants were instructed to respond as quickly and accurately as possible to the direction of the target arrow. They were instructed to press the "F" key with the left hand if the target pointed left, and the "K" key with the right hand if it pointed right. Each stimulus was presented for 300 ms, with an interstimulus interval of 2000 ms, and the trials were presented in a randomized order (Wei et al., 2022). Participants completed the task individually in a quiet room, seated comfortably approximately 70 cm from a 21-inch monitor. The task included 20 practice trials followed by 200 formal trials.

2.3. tDCS parameters

High-definition tDCS was administered using a device developed by Soterix Medical. The electrode configuration and current intensity settings are illustrated in Fig. 2. A total of five high-definition electrodes were used to deliver stimulation over the left DLPFC—one central electrode and four peripheral return electrodes forming a closed-loop circuit. Following the international 10–20 EEG system, the central electrode was positioned at site F3, while the peripheral electrodes were placed at F1, FC3, F5, and AF3. During stimulation, electrode impedance was kept at or below 5 kΩ. In the active stimulation condition, the central electrode delivered a current of 1.52 mA, and each of the four surrounding electrodes delivered −0.38 mA. Stimulation lasted for 25 min, including a 30-s fade-in and 30-s fade-out period. In the sham condition, only the initial 30-s fade-in and final 30-s fade-out were applied, with no current delivered during the rest of the session. This procedure mimicked the physical sensations of active stimulation while avoiding neuromodulatory effects. The order of active and sham stimulation was counterbalanced across participants. Participant feedback collected after the sessions indicated no significant difference in perceived sensations between active and sham conditions, suggesting that blinding was successful.

2.4. EEG data acquisition and analysis

EEG data were recorded using a Grael amplifier with the Curry 8 system (32-channel configuration). The EEG reference was placed at the midpoint between FPz and Fz. The sampling rate was set to 1024 Hz, and electrode impedance was maintained below 10 kΩ throughout the experiment. EEG preprocessing was performed using EEGLAB 13_0_0b. The raw EEG data were band-pass filtered with a high-pass filter at 0.1 Hz and a low-pass filter at 30 Hz. Data were re-referenced to the average of M1 and M2 electrodes. Continuous EEG signals were segmented into epochs from −200 ms to 1000 ms relative to stimulus onset, with baseline correction applied using the −200 to 0 ms window. Ocular artifacts (e.g., blinks and saccades) were corrected using Independent Component Analysis (ICA) based on the Infomax algorithm. Artifactual components were identified and removed manually via visual inspection of their scalp topographies and time courses. Following ICA, trials with voltage fluctuations exceeding ±150 μV were rejected. No participants were excluded from the final analysis due to excessive artifacts. Based on the procedure used by Dubreuil-Vall et al. (2019), the present study focused on incongruent trials only. Specifically, the N2 component was analyzed in the 240–310 ms window at the Fz electrode, and the P3 component was analyzed in the 390–490 ms window at the Pz electrode.

2.5. Statistical analysis

Statistical analyses were conducted using SPSS Statistics 26.0 (IBM, Somers, USA). Descriptive statistics are reported as mean ± standard deviation (SD). The significance level was set at 0.05, and partial eta squared (η_p^2) was used to report effect sizes for ANOVA. A three-way mixed-design ANOVA was performed on the following dependent variables under the incongruent condition: accuracy, reaction time (RT), mean N2 amplitude, and mean P3 amplitude. The between-subjects factor was Group (High vs. Low Test Anxiety), and the within-subjects factors were Stimulation Type (Active vs. Sham) and Time (Pre-test vs. Post-test). To interpret the significant three-way interaction, we conducted comprehensive simple effects analyses. We examined pairwise comparisons for all factors (Group, Stimulation, and Time) at each level of the interacting variables.

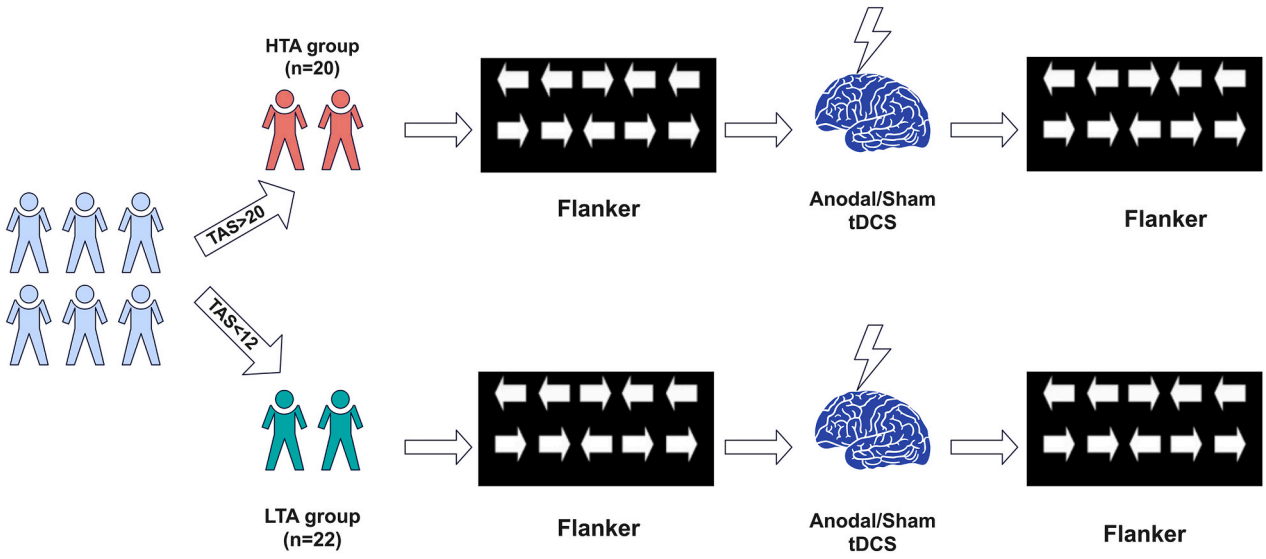

Fig. 1. Schematic of the experimental task procedure. HTA = High Test Anxiety; LTA = Low Test Anxiety; TAS = Test Anxiety Scale.

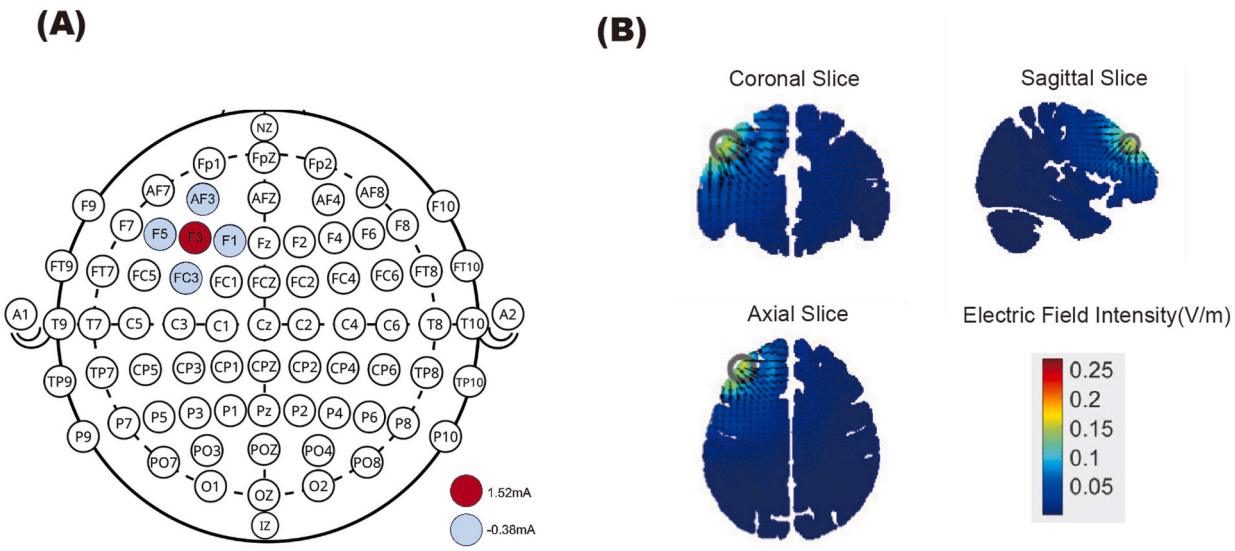

Fig. 2. (A) Configuration of tDCS electrode placement and current settings. (B) Electric field simulation of tDCS using the TesView software (Ma et al., 2024).

3. Results

3.1. Behavioral results

Descriptive statistics for accuracy and reaction time are presented in Table 2.

For accuracy, there was no significant main effect of Group, $F(1, 40) = 1.65$, $p = 0.206$; no significant main effect of Stimulation Type, $F(1, 40) = 2.43$, $p = 0.127$; and no significant main effect of Time, $F(1, 40) = 0.90$, $p = 0.349$. None of the interaction effects were significant: Group × Stimulation Type, $F(1, 40) = 0.64$, $p = 0.428$; Group × Time, $F(1, 40) = 3.41$, $p = 0.072$; Stimulation Type × Time, $F(1, 40) = 1.76$, $p = 0.192$; and the three-way interaction, $F(1, 40) = 0.17$, $p = 0.687$.

For RT, there was no significant main effect of Group, $F(1, 40) = 0.42$, $p = 0.519$; and no significant main effect of Stimulation Type, $F(1, 40) = 0.09$, $p = 0.772$. However, the main effect of Time was significant, $F(1, 40) = 7.31$, $p = 0.010$, $\eta_p^2 = 0.154$, indicating that RTs were shorter at post-test compared to pre-test. The interaction effects were not significant: Group × Stimulation Type, $F(1, 40) = 3.16$, $p = 0.083$; Group × Time, $F(1, 40) = 1.32$, $p = 0.258$; Stimulation Type × Time, $F(1, 40) = 2.51$, $p = 0.121$; and the three-way interaction, $F(1, 40) = 0.57$, $p = 0.456$.

3.2. EEG results

The topographical map and ERP waveform at the Fz electrode for the N2 component are presented in Fig. 3. Descriptive statistics for N2 amplitudes are provided in Table 2. There was no significant main effect of Group, $F(1, 40) = 0.12$, $p = 0.728$; no significant main effect of Stimulation Type, $F(1, 40) = 0.83$, $p = 0.369$; and no significant main effect of Time, $F(1, 40) = 3.55$, $p = 0.067$. None of the interaction effects reached significance: Group × Stimulation Type, $F(1, 40) = 0.26$, $p = 0.612$; Group × Time, $F(1, 40) = 0.10$, $p = 0.750$; Stimulation Type × Time, $F(1, 40) = 0.56$, $p = 0.460$; and the three-way interaction, $F(1, 40) = 0.05$, $p = 0.882$.

The topographical map and ERP waveform at the Pz electrode for the P3 component are presented in Fig. 4, and the P3 amplitude results are shown in Fig. 5. There was no significant main effect of Group, $F(1, 40) = 1.32$, $p = 0.258$; no significant main effect of Stimulation Type, $F(1, 40) = 0.08$, $p = 0.778$; and no significant main effect of Time, $F(1, 40) = 3.59$, $p = 0.065$. Additionally, none of the two-way interactions were significant: Group × Stimulation Type, $F(1, 40) = 0.01$, $p = 0.918$; Group × Time, $F(1, 40) = 1.19$, $p = 0.281$; Stimulation Type × Time, $F(1, 40) = 2.59$, $p = 0.116$. Importantly, the three-way interaction among Group, Stimulation Type, and Time was significant, $F(1, 40) = 5.81$, $p = 0.021$, $\eta_p^2 = 0.127$. Follow-up simple effects analysis revealed that only in the HTA group, P3 amplitude significantly decreased from pre-test (5.60 ± 4.47) to post-test (4.20 ± 4.83) under the active condition, $F(1, 40) = 6.16$, $p = 0.017$, $\eta_p^2 = 0.133$. No other condition showed significant differences in P3 amplitude ($ps > 0.05$).

Table 2
Descriptive statistics (Mean ± SD) for behavioral performance and N2 amplitudes across groups and conditions.

Measure	Group	Condition	Pre-test	Post-test
Accuracy	HTA (n = 20)	Active	0.97 ± 0.04	0.98 ± 0.02
		Sham	0.97 ± 0.03	0.97 ± 0.05
	LTA (n = 22)	Active	0.99 ± 0.01	0.98 ± 0.03
		Sham	0.98 ± 0.02	0.97 ± 0.05
Reaction Time (ms)	HTA (n = 20)	Active	452.52 ± 74.71	470.37 ± 77.30
		Sham	471.84 ± 72.55	470.17 ± 59.35
	LTA (n = 22)	Active	449.03 ± 80.15	472.56 ± 65.02
		Sham	439.19 ± 49.64	455.77 ± 54.15
N2 Amplitude (μV) *(Electrode Fz)*	HTA (n = 20)	Active	−3.70 ± 6.57	−4.15 ± 7.77
		Sham	−3.07 ± 6.77	−4.34 ± 8.67
	LTA (n = 22)	Active	−3.37 ± 4.38	−3.76 ± 5.19
		Sham	−2.37 ± 5.44	−3.20 ± 6.07

Note. HTA = High Test Anxiety; LTA = Low Test Anxiety.

3.3. Control analyses

To rule out potential confounding variables, we conducted preliminary analyses on baseline affect and stimulation order effects. Detailed statistical results are provided in the **Supplementary Materials**. Results confirmed equivalent baseline affect and the absence of stimulation order effects, ensuring that the findings were not confounded by emotional fluctuations or habituation.

4. Discussion

The present study revealed a significant Group × Stimulation × Time interaction on the P3 component during the Flanker task, such that only individuals with high test anxiety showed a significant reduction in P3 amplitude following active (anodal) tDCS over the left DLPFC compared to sham stimulation. In contrast, no significant effects were observed in the low test anxiety group or in N2 amplitude.

The DLPFC plays a critical role in both higher-order cognitive control

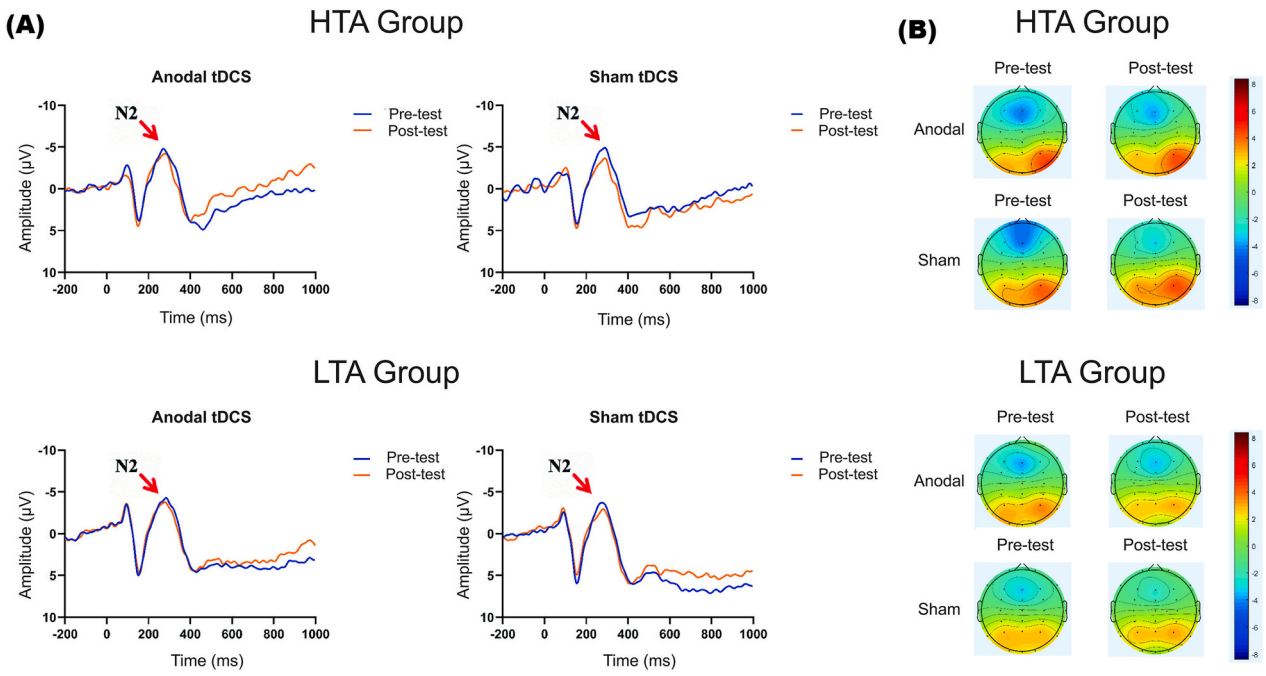

Fig. 3. (A) ERP waveforms at the Fz electrode before and after active/sham stimulation in high and low test anxiety groups. (B) Topographical maps of the N2 time window (240–310 ms) before and after stimulation. HTA = High Test Anxiety; LTA = Low Test Anxiety.

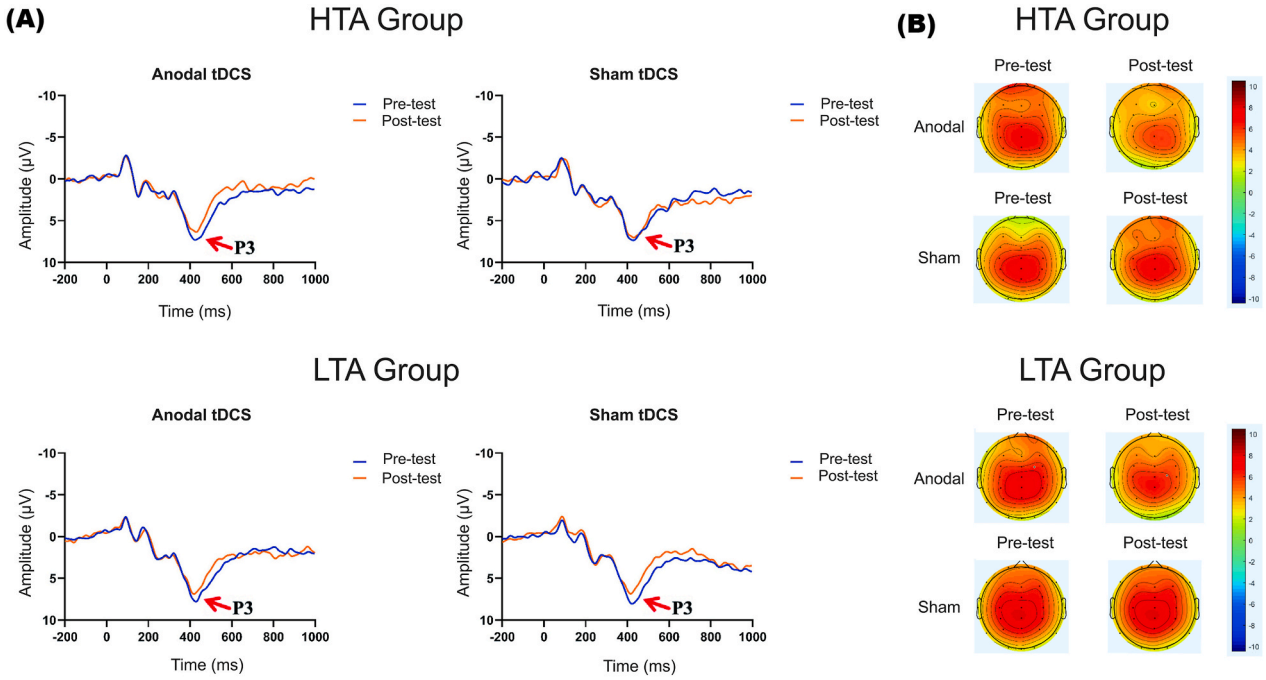

Fig. 4. (A) ERP waveforms at the Pz electrode before and after active/sham stimulation in high and low test anxiety groups. (B) Topographical maps of the P3 time window (390–490 ms) before and after stimulation. HTA = High Test Anxiety; LTA = Low Test Anxiety.

and emotion regulation. Enhancing activity in this region has been shown to improve cognitive control abilities and facilitate emotional regulation (Chen et al., 2023). In the present study, active tDCS may have increased the excitability of neuronal membranes in the left DLPFC, thereby improving functional connectivity between the prefrontal and parietal cortices, which are often disrupted by anxiety (Modi et al., 2015). This modulation was reflected in the reduced P3 amplitude, a component generated through coordinated activity within the prefrontal–parietal network (Brydges et al., 2020). The P3 component is widely regarded as an electrophysiological index of attentional resource allocation, with its amplitude closely linked to the amount of cognitive resources recruited for processing task-relevant stimuli (Li et al., 2015). A larger P3 amplitude generally reflects greater resource mobilization (Ramchurn et al., 2014). However, from the perspective of processing efficiency, a stronger neural response does not necessarily indicate more effective cognitive functioning.

Importantly, the reduction in P3 amplitude occurred in the absence of significant changes in behavioral accuracy or reaction time,

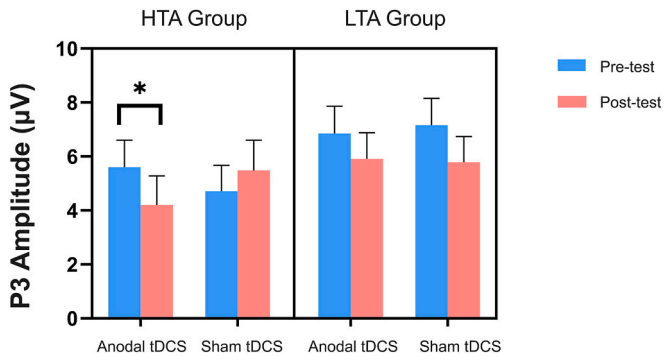

Fig. 5. P3 amplitudes at the Pz electrode in the incongruent condition before and after active/sham stimulation in high and low test anxiety groups. Error bars indicate standard errors. HTA = High Test Anxiety; LTA = Low Test Anxiety.

suggesting a dissociation between neural indices and overt performance. According to theories of attentional control, anxiety predominantly impairs processing efficiency rather than observable performance. Individuals with high test anxiety often engage additional cognitive resources to compensate for impaired control in order to maintain task performance levels comparable to those with low test anxiety (Zhang et al., 2019). From this perspective, the observed P3 reduction may reflect altered late-stage resource allocation or reduced neural recruitment demands during conflict resolution; however, given the absence of behavioral improvement, this pattern should be interpreted as modulation rather than evidence for enhanced processing efficiency.

Notably, the reduction in P3 amplitude observed in the high test anxiety group differs from findings reported in many prior tDCS studies conducted in healthy samples. For instance, recent studies have documented enhanced P300 amplitudes alongside improved cognitive performance in healthy adults during flanker (Chen et al., 2023) and oddball tasks (Liu et al., 2022). Similar patterns of increased neural recruitment have been observed in clinical or sub-clinical populations, such as individuals with substance use disorders (Chen et al., 2023) and chronically stressed adults (Liu et al., 2023). In these contexts, the increased P3 is generally interpreted as an enhancement of cognitive capacity or a greater mobilization of attentional resources to overcome deficits. This discrepancy might be interpreted within a task- and context-dependent framework, whereby the direction and magnitude of tDCS effects may vary with task demands (e.g., difficulty and control requirements) and/or the motivational salience of performance (e.g., evaluative pressure and the perceived importance of performing well) (Hsu et al., 2016; Yee et al., 2022). In healthy participants, anodal tDCS has been associated with larger P3 amplitudes accompanied by faster responses (Dubreuil-Vall et al., 2019), suggesting increased recruitment of attentional resources with behavioral gains. In contrast, under elevated test-related anxiety, the same stimulation may alter late-stage control implementation, yielding a smaller P3 without concurrent behavioral improvement. Future studies that manipulate task difficulty or performance incentives could help test these possibilities.

The present study did not find evidence that active tDCS over the left DLPFC modulated the N2 component, which may be attributed to the fact that N2 and P3 reflect different stages of neural inhibitory control. Specifically, N2 is associated with early conflict monitoring, whereas P3 is linked to later-stage processes such as conflict resolution and resource allocation (Siemann et al., 2016; Xie et al., 2017). This functional dissociation suggests that tDCS may selectively influence the execution stage of neural inhibitory control rather than the monitoring stage. This result aligns with findings from Verveer et al. (2020), who also reported that tDCS targeting the DLPFC significantly modulated later cognitive control markers (i.e., P3 amplitude) but had limited effects on earlier components like N2. Moreover, prior research has shown that the N2 component is sensitive to the proportion of congruent trials. When congruent trials are more frequent, the presence of incongruent distractors elicits stronger response conflict and thus greater N2 amplitudes due to premature response preparation for expected targets (Jost et al., 2022). In the current study, the congruent trial proportion was set at 50%. By contrast, Dubreuil-Vall et al. (2019) used a higher congruent-to-incongruent ratio (2:1) to build a tendency toward congruent responses and increase the difficulty of conflict detection on incongruent trials. This difference in trial structure may have reduced expectancy effects in our task and thus diminished the sensitivity of N2 to tDCS modulation. Future studies could manipulate the proportion of congruent trials to create a higher-conflict expectancy context, potentially enhancing N2 amplitude and allowing for a more comprehensive assessment of how tDCS influences different stages of neural inhibitory control processing.

The absence of significant P3 modulation in the LTA group contrasts with findings from Dubreuil-Vall et al. (2019), who reported increased P3 amplitudes in healthy participants following anodal tDCS. This discrepancy may suggest a state-dependent effect potentially influenced by baseline anxiety levels. It is important to note that our LTA group was selected using a rigorous criterion (TAS <12), representing individuals with comparatively low levels of test anxiety. It is possible that such individuals possess a baseline state characterized by high neural efficiency, which could result in a ceiling effect that limits the modulatory impact of tDCS. For average healthy individuals (e.g., Dubreuil-Vall et al., 2019), tDCS might still be able to enhance capacity (increasing P3). However, for our low-anxiety group, the neural system may have been operating at near-optimal efficiency, potentially leaving limited room for further tDCS-induced modulation. Supporting this, resting-state fMRI studies have shown that individuals with low test anxiety exhibit higher local gyrification indices in cortical regions associated with executive control, including the right lateral occipital gyrus (Hou et al., 2022), as well as stronger functional connectivity between the superior frontal gyrus and anterior cingulate cortex (Huang et al., 2022). Such findings suggest that homeostatic brain mechanisms may protect low-anxiety individuals from unnecessary neural modulation. When brain activity is already operating within an optimal range, additional stimulation is likely to be counteracted in order to maintain internal equilibrium, resulting in a saturated or diminished response to external input.

The present findings contribute to a more nuanced understanding of inhibitory control in test anxiety by highlighting the role of neural efficiency and compensatory processing demands rather than overt behavioral deficits. The findings suggest that the level of test anxiety not only shapes attentional resource allocation, but also influences individual responsiveness to neuromodulatory interventions, supporting the notion that emotional states moderate the efficacy of tDCS (Gibson et al., 2021). Importantly, this study highlights the potential of high-definition tDCS as a form of neuro-targeted precision intervention. Unlike pharmacological treatments, non-invasive brain stimulation techniques such as tDCS offer a safe and immediate means of regulating neural activity (Brunoni et al., 2012).

This study has several limitations that warrant consideration. First, the experiment did not incorporate a realistic testing context or stress-inducing paradigm. Future research could benefit from introducing simulated exam environments or evaluative tasks to examine the effects of tDCS under conditions more closely aligned with actual high-stakes testing situations. Second, this study did not assess state anxiety or stress levels immediately before and after tDCS, preventing a direct evaluation of stimulation-induced changes in affect. Although we confirmed that baseline emotional states were equivalent prior to the active and sham sessions (see **Supplementary Materials**), the lack of post-stimulation affective measures remains a limitation. Future research should incorporate real-time anxiety assessments to explicitly examine the interaction between neuromodulation and acute stress responses. Third, this study employed a single stimulation protocol in

terms of current intensity and electrode configuration, without exploring the dose–response relationship. Prior research has suggested that the effects of tDCS may exhibit nonlinear characteristics, with some studies reporting paradoxical neural outcomes at higher intensities—for example, 2 mA stimulation may induce effects opposite to those expected (Batsikadze et al., 2013). Future studies should systematically investigate varying stimulation intensities, polarities, and dosing parameters to optimize tDCS protocols for improving neural inhibitory control in test-anxious individuals. Fourth, regarding the sample size estimation, we assumed a medium effect size ($f = 0.25$) due to the lack of direct prior data on tDCS effects in this specific population. This assumption may have resulted in insufficient statistical power to detect smaller effect sizes, which could explain the absence of significant findings regarding the N2 component or subtle modulation effects within the LTA group. Lastly, the study primarily relied on ERP measures to infer underlying neural mechanisms, which limits insight into whole-brain network dynamics. Integrating functional neuroimaging techniques such as fMRI in future work may help validate and extend our understanding of tDCS-induced changes in brain connectivity at the network level.

5. Conclusions

This study investigated the effects of tDCS on inhibitory control and its underlying neural mechanisms in individuals with high test anxiety. The results demonstrated that active tDCS over the left DLPFC significantly reduced P3 amplitudes during the Flanker task in the high-anxiety group.

CRediT authorship contribution statement

Peibing Liu: Writing – review & editing, Writing – original draft, Validation, Methodology, Formal analysis, Conceptualization. **Zhaonian Hu:** Writing – original draft, Formal analysis, Data curation. **Renlai Zhou:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Funding acquisition, Conceptualization.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2026.01.055.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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