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Trait anxiety negatively modulates the coupling of motor event-related desynchronization and event-related synchronization

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Abstract

Background Recent neurophysiological studies showed that patients with psychiatric disorders demonstrated abnormalities in sensorimotor functions in addition to cognitive deficits. These findings intrigued us to investigate whether trait anxiety, a persistent inclination towards being anxious in multiple contexts, would affect motor cortical functions. Event-related desynchronization (ERD) and event-related synchronization (ERS) of α and β oscillations are associated with movement execution and movement termination, respectively. However, no study has comprehensively examined the effects of trait anxiety on motor ERD and ERS. Therefore, this study aimed to determine how trait anxiety influences these motor cortical oscillations.

Methods Twenty subjects (top 10% of the trait anxiety score distribution from 400 college students) with higher trait anxiety (HTA) and 20 subjects (bottom 10% of trait anxiety score distribution from the same sample) with lower trait anxiety (LTA) were recruited to perform a Go-Nogo task during electroencephalographic recordings. ERD and ERS of α and β oscillations to Go responses were compared between these two groups. The associations between ERD and ERS in each group were also examined.

Results Neither ERD nor ERS power changes were significantly different between LTA and HTA groups. Interestingly, a significant correlation between β ERD and α ERS/β ERS was found in the individuals with LTA; however, such functional coupling was not present in the individuals with HTA.

Conclusion Trait anxiety negatively modulates the coupling of motor ERD and ERS.

Keywords Event-related desynchronization (ERD), Event-related synchronization (ERS), Trait anxiety, Alpha oscillations, Beta oscillations

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Introduction

Trait anxiety refers to a persistent tendency to feel anxious in a variety of situations that is separate from state anxiety, which is in response to a temporary situation that causes feelings of nervousness or fear [1, 2]. People who have trait anxiety usually perceive a heightened sense of apprehension even in relatively low-risk circumstances, with the symptoms including fear, avoidance, difficulty concentrating, and muscle tension [3, 4], which subsequently affects their well-being and functioning. A number of studies have dedicated to studying how trait anxiety modulates individuals' cognitive performance, particularly in the executive functioning [1, 5]. For example, previous electrophysiological studies have shown that people with higher trait anxiety (HTA), compared to those with lower trait anxiety (LTA), demonstrated poorer inhibitory functions [6–10]. There were other lines of evidence revealing that HTA was associated with poorer processing efficiency in the tasks requiring working memory [11] or mental flexibility [12, 13]. However, it remains substantially unknown that whether the cortical representations of motor control are modulated by trait anxiety.

Event-related desynchronization (ERD) and event-related synchronization (ERS) are two common neurophysiological indicators of motor control. Pre-movement ERD of α (8–12 Hz) and β (13–30 Hz) oscillations have been suggested as indices of cortical activations during motor activities and are strongly related to the efficiency of motor preparation and motor execution; by contrast, post-movement ERS of α and β oscillations have been more associated with cortical idling or termination of motor movement [14–17]. Up to the date, there has been only one study that comparted the motor ERD/ERS between individuals with LTA and HTA [18]. The authors analyzed event-related spectral power to Go stimuli in the stop-signal task, and found that subjects with HTA showed more α and β ERD (i.e., more negativity of α and β band) as compared to those with LTA [18]. However, it should be noted that the stop-signal task requires subjects' substantial efforts to "cancel" the initiated movements. In this scenario, cortical responses to Go stimuli might not only be operated by motor cortices, but also be governed by other cognition-related regions (e.g., taking strategies to wait for the occurrence of the stop stimulus). Thus, we reasoned that employing a simpler task would allow for a more precise investigation into the effects of trait anxiety on ERD and ERS. In this regard, the first aim of the present study was to compare ERD and ERS of α and β oscillations to Go stimuli in a simple Go-Nogo task between LTA and HTA groups.

The investigation on ERD or ERS alone might not thoroughly reflect the functional integrity of motor cortex. Take amyotrophic lateral sclerosis (ALS) as an example, a previous study has found that post-movement β ERS was reduced while pre-movement α ERD was relatively preserved in patients with ALS as compared to healthy controls [19]. Another study, however, revealed a reverse pattern where patients with ALS exhibited a reduced α ERD but not β ERS as compared to healthy controls [20]. These findings intrigued us to propose a research question that whether the coupling between ERD and ERS might be another potential indicator to reflect the integrity of motor cortical function. Although the physiological meaning of the relationship between ERD and ERS is complex and remains elusive, we proposed that in the motor domain, ERD and ERS must function properly in order for a movement to be accurate. At the beginning of the movements, a more magnitude of ERD would result in more readiness of motor programming. A higher level of ERS, presenting several hundreds of milliseconds following ERD, would be considered to more efficiently terminate the movements. The second aim of the present study was to examine whether a more magnitude of ERD would be associated with a more ERS in individuals with LTA and HTA when they performed simple movements (i.e., respond to Go stimuli in a simple Go-Nogo task).

The hypotheses of the research aims were in 2 folds. For the first aim, we predicted that there would be significant differences in motor ERD and/or ERS of α and β oscillations in response to Go stimuli between the LTA and HTA individuals (*Hypothesis 1*). A previous study found that, as compared to healthy controls, patients with obsessive–compulsive disorder (OCD) showed significantly attenuated β ERS during self-paced movements of the right thumb [21]. However, another study revealed that subjects with HTA exhibited more α and β ERD compared to those with LTA [18]. Due to the mixed results in the literature for the effects of anxiety on the motor ERD and ERS, we did not set a directional hypothesis in the present study. For the second aim, we predicted that there would be a significant correlation between ERD and ERS in the subjects with LTA, but not in those with HTA (*Hypothesis 2*). This reasoning was based on the inference from our previous study showing that in the healthy controls, the somatosensory gating ratios (i.e., an indicator of automatic inhibitory function in the primary somatosensory cortex [SI]) were significantly associated with peak frequency values of gamma oscillations (i.e., a kind of inhibition-related indicators) in the SI; however, such a relationship disappeared in the patients with panic disorder [22]. Although no study, to the best of our knowledge, directly tested the association between ERD and ERS in the motor cortex, we borrowed the interesting findings from our previous study to postulate this hypothesis.

Methods

Subjects

This study was the secondary analysis of our previous research project, which aimed to investigate the effects of trait anxiety on inhibitory control and error monitoring [8, 23]. In brief, a total of 400 subjects completed the survey of State-Trait Anxiety Inventory (STAI) [24]. Based on the score distribution of STAI–trait (STAI-T), we invited 20 subjects with HTA from the top 10% and 20 subjects with LTA from the bottom 10% to undergo event-related potential (ERP) recordings. They were self-reportedly right-handed and free of substance use and any history of neurological/psychiatric disorders.

Experimental procedures

The participants performed Go-Nogo and stop-signal tasks with a counterbalanced sequence. The detailed experimental procedures of stop-signal task were described previously [23]. In the Go-Nogo task, the subjects were told to respond to Go trials (a white "O", 270 trials, $p = 75\%$) with their right index finger and to withhold responses to Nogo trials (a white "X", 90 trials, $p = 25\%$).

Each stimulus was presented for 250 ms, and the inter-trial interval was jittered between 1000 and 2000 ms to avoid anticipation effects. Both speed and accuracy were emphasized in the instructions. A short practice was arranged for the participants to familiarize with the procedure prior to the formal experiment.

Electrophysiological recordings and data analysis

Electrophysiological signals were recorded using a 34-channel elastic cap (EasyCap, GmbH, Herrsching, Germany) following the international 10–20 system. Eye blinks and movements were recorded using electrooculograms (EOGs). Two channels were designated for EOG recordings, and two channels served as reference electrodes (i.e., A1 and A2). Thus, electrophysiological responses were recorded and analyzed from a total of 30 active channels (Fp1, Fp2, F7, F8, F3, F4, Fz, FT7, FT8, FC3, FC4, FCz, C3, C4, Cz, T7, T8, CP3, CP4, CPz, TP7, TP8, P7, P8, P3, P4, Pz, O1, O2, Oz). The acquisition of data was performed by a 40-channel QuickAmp amplifier and BrainVision Recording software (Brain Products GmbH, Gilching, Germany). The online digitization rate was 1000 Hz and the impedance was kept below 5 kΩ. The recordings were conducted in the afternoon for all the subjects.

Time–frequency analyses of the Go trials were performed to obtained ERD and ERS. Raw data were re-referenced to the average of mastoid electrodes with a high-pass filter of 0.1 Hz. Although all the subjects were right-handed, the data from both C3 and C4 electrodes were analyzed and then compared between groups. Response-locked epochs from −1000 ms prior to the movement (i.e., button press to Go trials) to 800 ms after the movement were analyzed using BrainVision Analyzer2 (Brain Products GmbH, Gilching, Germany). Each raw epoch was computed by Morlet wavelet-based transformation from 1–50 Hz, using 40 logarithmical steps with 3 cycles. The output values were computed as spectral power (uV^2). Subsequently, the percentage change (%) method, which was first baseline-corrected and then rescaled relative to the mean norm within a reference interval [−1000 to 0 ms]), was applied.

ERD and ERS typically last for 200 to 300 ms or even longer after movement onset. To better represent their power strength, we identified the most reactive 100 ms time window for each subject. Given the known variability in the precise onset and duration of ERD and ERS across individuals, we adjusted the placement of these time windows accordingly for each subject. More specifically, α ERD and ERS were derived from the 8–12 Hz band, while β ERD and ERS were calculated from the most reactive 8 Hz segment within the 13–30 Hz β range. For each component, the power was averaged over a 100 ms window centered around the peak latency of the respective frequency band response [25–28]. Additionally, the mean power of α and β ERD was estimated within the −200 to 200 ms time window, and α and β ERS within the 300 to 800 ms time window.

In addition to the conventional analysis method that extracted the maximal power changes in ERD and ERS, we also determined the temporal dynamics of ERD and ERS on every 100-ms time window.

Statistical analysis

The numerical data were presented as mean ± standard of the error mean (SEM). The differences in demographic data were compared between LTA and HTA groups using the independent t tests (e.g., age) or chi-square tests (e.g., sex) when appropriate. Since the data of ERD and ERS were normally distributed (all p values >0.05) as evaluated by one-sample Kolmogorov–Smirnov test, the differences in ERD and ERS were compared using independent t tests between LTA and HTA groups. P values of less than 0.05 were considered statistically significant. In addition, Cohen's d effect sizes were reported.

For the correlational analyses, we firstly determined whether α ERD was associated with α ERS and β ERS in the LTA and HTA groups using Pearson correlation coefficients. We then determined whether β ERD was associated with α ERS and β ERS in each group using the same method. Here, we applied Benjamini and Hochberg

method [29] to adjust p values due to multiple comparisons, and the false discovery rate (FDR) was set at 0.05.

Results

Table 1 lists the demographic information and behavioral results of the Go-Nogo task. Age and sex distribution were similar between these two groups. As expected, individuals with HTA had higher scores on the STAI-T than those with LTA ($p < 0.001$). The behavioral responses to Go trials were also similar between LTA and HTA groups (Go accuracy, $p = 0.337$; Go RT, $p = 0.254$), suggesting that trait anxiety did not significantly affect behavioral performance in a simple motor task.

Table 1 Demographic information and behavioral data of the Go-Nogo task (mean ± SEM) in subjects with lower trait anxiety (LTA) and higher trait anxiety (HTA)

	LTA ($n = 20$)	HTA ($n = 20$)	*P* value
Age (years)	21.95 ± 0.72	21.2 ± 0.32	0.347
Gender (male/female)	8/12	4/16	0.176
STAI-T	26.1 ± 0.68	62.45 ± 1.05	< 0.001
STAI-S	28.00 ± 1.51	44.25 ± 1.89	< 0.001
Go RT (ms)	377.99 ± 13.13	360.76 ± 6.98	0.254
Failed Nogo RT (ms)	328.83 ± 8.67	330.79 ± 7.91	0.868
Go miss rate (%)	1.68 ± 0.40	2.38 ± 0.57	0.326
Nogo accuracy (%)	83.83 ± 3.80	78.48 ± 4.00	0.338

SEM Standard error of the mean, *STAI-T* State-Trait Anxiety Inventory-Trait, *STAI-S* State-Trait Anxiety Inventory-State, *RT* Reaction time

Since all the subjects were right-handed, we demonstrated the grand-averaged time–frequency responses to Go trials from C3 electrode in both LTA and HTA groups (Fig. 1). An obvious decrease in spectral power of α and β oscillations (i.e., α ERD and β ERD) was observed between 150 ms before and 200 ms after the button-press in both groups. An obvious increase in spectral power of α and β oscillations (i.e., α ERS and β ERS) was found between 400 and 700 ms after the button-press in both groups. The scalp topographies representing the peaks of α ERD, β ERD, α ERS, and β ERS in the LTA and HTA groups are also illustrated. The patterns of topographic maps are similar between these two groups. Supplementary Fig. 1 further displays the time–frequency maps for Go trials from C3 and C4 electrodes in each participant.

Not supporting *Hypothesis 1*, the statistical results (Table 2) revealed that subjects with LTA and HTA did not significantly differ in peak powers of α ERD (C3: $p = 0.994$, Cohen's d = 0.002; C4: $p = 0.513$, Cohen's d = 0.208), β ERD (C3: $p = 0.665$, Cohen's d = 0.143; C4: $p = 0.713$, Cohen's d = 0.116), α ERS (C3: $p = 0.974$, Cohen's d = 0.010; C4: $p = 0.918$, Cohen's d = 0.033), and β ERS (C3: $p = 0.120$, Cohen's d = 0.502; C4: $p = 0.437$, Cohen's d = 0.248). Additional analyses were performed on every 100-ms time window in terms of ERD and ERS. The

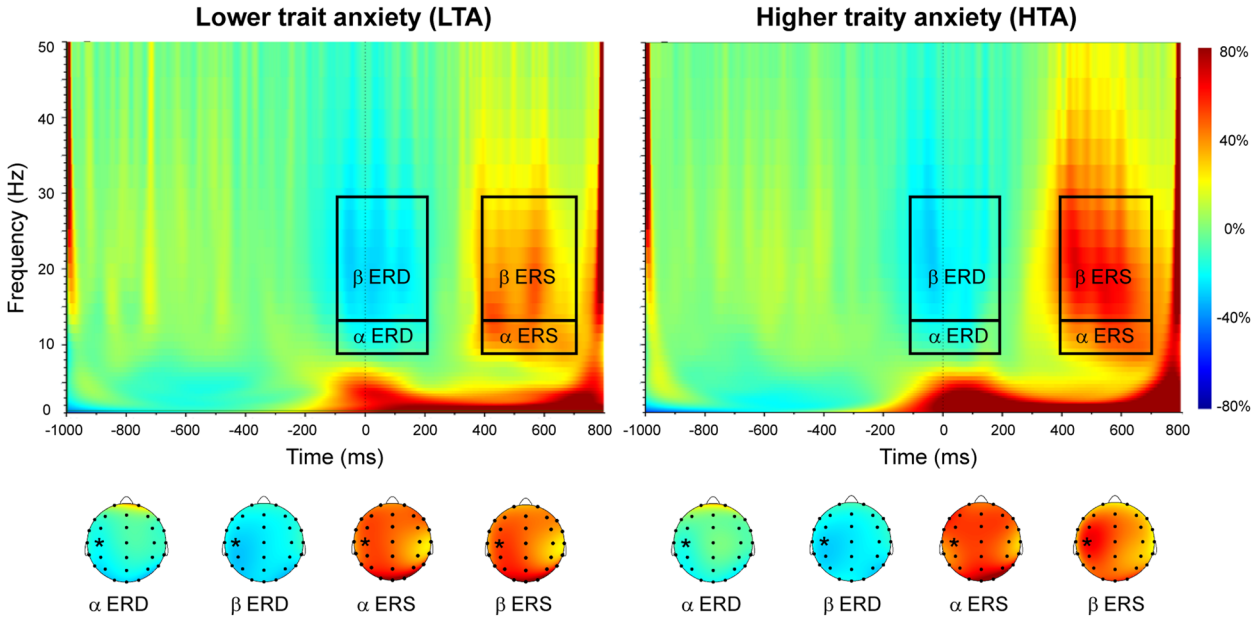

Fig. 1 Upper panel: Grand-averaged time–frequency maps over C3 electrodes for the subjects with lower trait anxiety (LTA, $n = 20$) and higher trait anxiety (HTA, $n = 20$) during the time window from −1000 to 800 ms relative to button-press to Go trials. The diagrams were converted into percentage changes of event-related desynchronization (ERD) and event-related synchronization (ERS) relative to baseline. Lower panel: The scalp topographies of α ERD, β ERD, α ERS, and β ERS in each group. The asterisks represent the C3 electrodes

Table 2 Motor cortical oscillations (mean ± SEM) during Go trials in subjects with lower trait anxiety (LTA) and higher trait anxiety (HTA)

	LTA ($n=20$)	HTA ($n=20$)	*P* value	Cohen's d
C3 α ERD	−18.78 ± 4.32	−18.74 ± 3.40	0.994	0.002
β ERD	−31.39 ± 4.01	−29.30 ± 2.28	0.665	0.143
α ERS	50.85 ± 13.08	50.34 ± 8.21	0.974	0.010
β ERS	56.09 ± 8.30	81.90 ± 13.92	0.120	0.502
C4 α ERD	−15.21 ± 3.28	−12.46 ± 2.56	0.513	0.208
β ERD	−24.63 ± 2.94	−23.23 ± 2.37	0.713	0.116
α ERS	43.93 ± 11.23	45.30 ± 6.95	0.918	0.033
β ERS	40.76 ± 5.87	46.86 ± 5.07	0.437	0.248

SEM Standard error of the mean

results showed that neither ERD nor ERS was significantly different between LTA and HTA groups.

At the C3 electrode, α ERD showed no significant association with either α ERS or β ERS in both the LTA and HTA groups. Interestingly, in the LTA group, β ERD was significantly correlated with β ERS (r = −0.599, $p = 0.005$, FDR = 0.020); however, this association was absent in the HTA group (Fig. 2A). A similar pattern was observed at the C4 electrode. In the LTA group, β ERD was significantly correlated with both α ERS (r = −0.584, $p = 0.007$, FDR = 0.022) and β ERS (r = −0.554, $p = 0.011$, FDR = 0.022), but these associations were not present in the HTA group (Fig. 2B). These findings support *Hypothesis 2*.

Discussion

This study aimed to determine the effects of trait anxiety on motor ERD and ERS. The results showed that there were no significant differences in the spectral power of α ERD, β ERD, α ERS, and β ERS between LTA and HTA groups. Interestingly, we found a significant association between β ERD and α ERS/β ERS in the LTA group, and such functional coupling was absent in the HTA group.

Although motor signs were not a major clinical manifestation in psychiatry disorders, several empirical studies showed that these patients still demonstrated abnormalities in motor cortical functions [30]. For example, patients with schizophrenia exhibited reduced post-movement β ERS as compared to healthy controls [31, 32]. Furthermore, another study revealed that among the individuals with schizotypal personality, which refers to a set of traits quantitatively within normal range but qualitatively similar to schizophrenia, lower β ERS was significantly associated with higher scores of Schizotypal Personality Questionnaire [33]. In the anxiety disorder, one study recruiting a small sample of patients with OCD found that they demonstrated decreased β ERS as compared to healthy controls [21]. Based on this result, we further examined the effects of trait anxiety on motor ERD and ERS. Our data showed that motor ERD and ERS did not significantly differ between LTA and HTA groups,

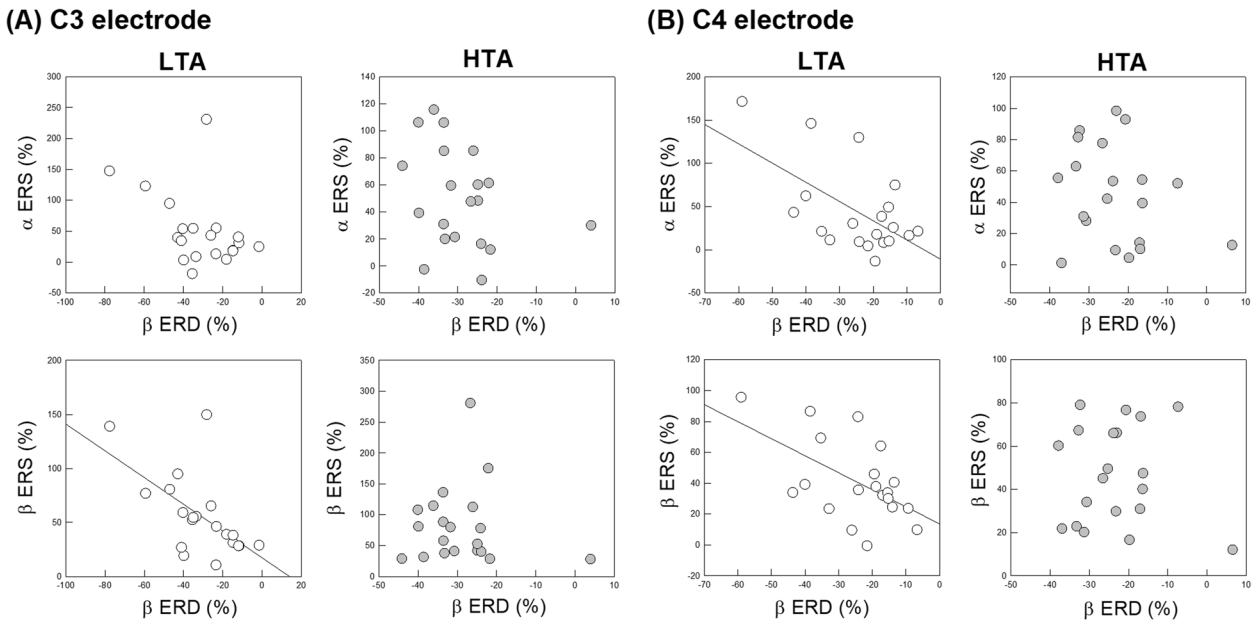

Fig. 2 **A** Scatter plots showing the relationship between β ERD and α ERS, as well as β ERS, in the lower trait anxiety (LTA) and higher trait anxiety (HTA) groups at the C3 electrode. **B** Scatter plots showing the relationship between β ERD and α ERS, as well as β ERS, in the LTA and HTA groups at the C4 electrode. ERD, event-related desynchronization; ERS, event-related synchronization

suggesting trait anxiety did not directly and simply affect the movement-related ERD and post-movement ERS. We reasoned that the lack of significant differences in spectral power between the LTA and HTA groups may be due to the task used in this study not sufficiently challenging the participants'central nervous system. For instance, research has shown that alterations in beta and gamma oscillations in patients with multiple sclerosis are evident during cognitively demanding tasks but not during the resting state. [34].

Despite no significant between-group differences in the ERD and ERS, we found that trait anxiety modulated motor cortical activities through the form of ERD–ERS coupling. More specifically, a more magnitude of β ERD (i.e., more negativity of β oscillations) was concomitant with a more magnitude of α ERS or β ERS in the individuals with LTA, whereas such a link was broken in those with HTA. The observed ERD–ERS coupling in the LTA group represents a novel finding. To date, no study has directly examined the relationship between ERD and ERS. However, existing evidence suggests that ERD and ERS often operate as a sequential neural process, with desynchronization during motor activation typically followed by synchronization during relaxation or inhibition. This dynamic balance between cortical activation (ERD) and inhibition (ERS) is crucial for optimal motor performance. Pfurtscheller and Lopes da Silva provided foundational insights into this process, demonstrating how ERD occurs during motor imagery and execution, while ERS emerges during rest phases [17]. This temporal sequence reflects a functional coupling within the motor cortex that supports efficient motor control. Further reinforcing this concept, recent studies in patients with neurological and psychiatric disorders have also highlighted the importance of ERD–ERS dynamics in maintaining motor and cognitive function [30, 35].

The absence of ERD–ERS coupling observed in the HTA group is also a novel finding while the underlying mechanism is under investigated. It has been hypothesized that α oscillations are generated through the thalamo-cortical loops, while the β oscillations appear only in the cortex [16, 36]. Previous magnetoencephalographic (MEG) studies employing beamforming methods identified that the neural sources of β ERD in the postcentral gyrus [27, 37] or sensorimotor cortex [38, 39]. In contrast, β ERS has been attributed to neural activity originating from the precentral gyrus, and/or supplementary motor area (SMA) [27, 37]. Furthermore, we reasoned that the lack of ERD–ERS association was not the primary source of the differences in trait anxiety. Instead, it was more possible that the brain regions associated with trait anxiety (e.g., anterior cingulate cortex [ACC], prefrontal cortex [PFC], etc.) were altered in subjects with HTA, which further led to the disruption of motor cortical oscillations.

The ACC and PFC play pivotal roles in emotional regulation and motivational processes. Neuroanatomical studies have mapped pathways through which reward and outcome information from the ACC, along with action-related information from the posterior cingulate cortex, are relayed to the midcingulate motor area. This area subsequently projects to premotor regions, including the premotor cortex and SMA [40]. Functional magnetic resonance imaging (fMRI) studies further support these connections, demonstrating that individuals with HTA exhibit reduced functional connectivity between the dorsolateral PFC and ACC [41, 42], as well as between the precuneus and PFC [43]. Notably, these regions have also been shown to connect with the precentral gyrus, premotor cortex, and SMA [44]. Although these studies do not directly investigate the ACC-PFC-motor cortex connectivity in individuals with trait anxiety or anxiety disorders, they underscore the complex interconnectedness of these regions in both emotional and motor functions. To deepen our understanding of how these networks are altered in anxiety, further research focusing on the ACC-PFC-motor cortex pathways in anxiety populations is warranted.

It was interesting to note that the association between ERD and ERS occurred in the β but not in the α oscillations. A growing body of research indicates a measurable relationship between local gamma-aminobutyric acid (GABA) concentrations and beta amplitude in the motor cortex [45–47]. Using magnetic resonance spectroscopy, a previous study revealed that higher GABA concentration in the primary motor cortex was associated with higher power of β ERS [48]. More recently, a study using MEG examined the effects of GABAergic modulation on sensorimotor beta oscillations during a finger abduction task. Participants were administered gaboxadol and zolpidem, two GABA-A positive allosteric modulators. The findings revealed that gaboxadol produced stronger modulation of beta dynamics than zolpidem, leading to deeper beta desynchronization during movement and a more pronounced post-movement beta rebound [49]. Additionally, elevated GABA levels have been reported in individuals with trait anxiety [50–52], though these increases were primarily observed in the prefrontal cortex. Based on these findings, we propose that the functional relationship between ERD and ERS in beta oscillations is governed by the fine-tuned balance of excitation and inhibition modulated by GABA. However, this interpretation remains speculative. Future research should directly manipulate GABA levels and assess their

effects on the ERD–ERS relationship to more conclusively test this hypothesis.

Several limitations should be considered in the present study. Firstly, the sample size was small. However, our study design has dealt with this limitation where the subjects with HTA ($n = 20$) and LTA ($n = 20$) were invited from the top 10% and the last 10% of 400 volunteers who completed the STAI-T. In contrast to the previous studies that separated the subjects into LTA and HTA groups using the median split method [18, 53, 54], the current samples of LTA and HTA extracted using our sampling method were much more likely to represent the characteristics in the whole population [55, 56]. Secondly, voluntary movement can be categorized into self-paced and cue-guided movement. The cue-guided movement indicates that the movement is made in response to external stimuli (e.g., responses to Go in the present study). Although recruiting similar brain substrates as compared to self-paced movement, cue-guided movement is considered to involve more processes including motor (e.g., motor preparation and execution) and cognitive processes (e.g., stimulus processing). Thus, the relationship between ERD and ERS of β oscillations need further replication using self-paced paradigms. Thirdly, the lack of electromyographic (EMG) measures in the present study is a potential methodological limitation. EMGs can precisely detect the timing of muscle contraction and termination, which is very important to study the peak latencies of ERD and ERS. However, we only compared the maximal powers of ERD and ERS within the time window of interest between LTA and HTA groups in the present study. There have been several studies examining the motor-related oscillations with the design of button press. For example, An and colleagues found that the children with autism spectrum disorder (ASD) showed lower evoked gamma oscillations and more β ERD in the ipsilateral primary motor cortex as compared to typically-developing children [57, 58]. Despite these insights, the absence of EMG measures remains a potential source of bias in our findings. Finally, one might argue that motor ERD and/or ERS would be affected by different movement variables, such as speed, duration, or strength. Although the muscle activities of the index fingers were not recorded in the present study, previous literature has shown that strength load or movement speed did not modulate power changes of ERD and ERS [59–61].

In conclusion, trait anxiety did not significantly affect power changes of motor ERD or ERS. However, trait anxiety negatively modulates the coupling of motor ERD and ERS.

Supplementary Information

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Supplementary Material 1.

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Declaration of generative AI and AI-assisted technologies in the writing process
During the preparation of this work, the authors used ChatGPT in order to improve language and readability. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Authors' contributions
Conceived and designed the work: CHC. Acquired the data: CHC, HL. Analyzed the data: PYC, YHC, SYC, HL. Participated in the discussion and provided the comments: PYC, YHC, SYC, HL, CYL. Wrote the paper: CHC. All of the authors have read and approved the manuscript.

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Data availability
The original contributions presented in the study are included in the article. Further inquiries can be directed to the corresponding author.

Declarations

Ethics approval and consent to participate
The study was conducted in accordance with the Declaration of Helsinki. The study was approved by the Institutional Review Board of Chang Gung Memorial Hospital (IRB No.: 201701600 A3), Taiwan. Written informed consent was obtained from all participants.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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