Anxiety symptoms without depression are associated with cognitive control network (CNN) dysfunction: an fNIRS study

Caihong Yang¹, yan zhang², Pu Wang³, Huifen Wu⁴, Qiang Xiao⁴, Fang Xu⁴, Yueran Bian⁴, Nian Xiang⁴, and Min Qiu⁴

¹Central China Normal University
²Affiliation not available
³The Seventh Affiliated Hospital Sun Yat-sen University
⁴Huazhong University of Science and Technology

July 4, 2023

Abstract

Anxiety is a common psychological disorder associated with affective disorders and associated with other mental disorders, with depression being the most common comorbidity. Few studies have examined the neural mechanisms underlying anxiety after controlling for depressive symptoms. This study aimed to explore whether there are differences in cortical activation in anxiety patients with different severity whose depressive symptoms is normal. In current study, depression levels were normal for 366 subjects—139 healthy subjects, 117 with mild anxiety, and 110 with major anxiety. The Hospital Anxiety and Depression Scale (HADS) and a verbal fluency task (VFT) tested subjects' anxiety and depression and cognitive function, respectively. A 53-channel guided near-infrared spectroscopic imaging technology (fNIRS) detected the concentration of oxyhemoglobin (oxy-Hb). Correlation analysis between anxiety severity and oxy-Hb concentration in the brain cortex was performed, as well as ANOVA analysis of oxy-Hb concentration among the three anxiety severity groups. Results showed that anxiety severity was significantly and negatively correlated with oxy-Hb concentration in the left frontal eye field (lFEF) and in the right dorsolateral prefrontal area (rDLPFC). The oxy-Hb concentration in the lFEF and the rDLPFC were significantly lower in the major anxiety disorder group than that in the control group. This suggests that decreased cortical activity of lFEF and rDLPFC may be neural markers of anxiety symptoms after control depressive symptoms. Anxiety symptoms without depressive symptoms may be result from the dysfunction of cognitive control network (CCN) which includes lFEF and rDLPFC.

Anxiety symptoms without depression are associated with cognitive control network (CNN) dysfunction: an fNIRS study

Caihong Yang¹*, Yan Zhang²*, Pu Wang³, ⁴*, Huifen Wu², Qiang Xiao⁵, Fang Xu⁵, Yueran Bian², Nian Xiang⁵, Min Qiu⁵

¹School of Psychology, Central China Normal University, Wuhan, 430074, China.
²School of Educational Science, Huazhong University of Science and Technology, Luoyu Road No. 1037, Hongshan, Wuhan, P.R. China, 430074.
³Department of Rehabilitation Medicine, The seventh Affiliated Hospital Sun Yat-sen University, Shenzhen, 518107, China.
⁴Department of Rehabilitation Medicine, Tianyang District People’s Hospital, Baise City, Guangxi Province
⁵Hospital of Huazhong University of Science and Technology, Wuhan, China
Anxiety symptoms without depression are associated with cognitive control network (CNN) dysfunction: an fNIRS study

Abstract
Anxiety is a common psychological disorder associated with other mental disorders, with depression being the most common comorbidity. Few studies have examined the neural mechanisms underlying anxiety after controlling for depressive symptoms. This study aimed to explore whether there are differences in cortical activation in anxiety patients with different severity whose depressive symptoms is normal. In current study, depression levels were normal for 366 subjects—139 healthy subjects, 117 with mild anxiety, and 110 with major anxiety. The Hospital Anxiety and Depression Scale (HADS) and a verbal fluency task (VFT) tested subjects' anxiety and depression and cognitive function, respectively. A 53-channel guided near-infrared spectroscopic imaging technology (fNIRS) detected the concentration of oxyhemoglobin (oxy-Hb). Correlation analysis between anxiety severity and oxy-Hb concentration in the brain cortex was performed, as well as ANOVA analysis of oxy-Hb concentration among the three anxiety severity groups. Results showed that anxiety severity was significantly and negatively correlated with oxy-Hb concentration in the left frontal eye field (lFEF) and in the right dorsolateral prefrontal area (rDLPFC). The oxy-Hb concentration in the lFEF and the rDLPFC were significantly lower in the major anxiety disorder group than that in the control group. This suggests that decreased cortical activity of lFEF and rDLPFC may be neural markers of anxiety symptoms after control depressive symptoms. Anxiety symptoms without depressive symptoms may be result from the dysfunction of cognitive control network (CCN) which includes lFEF and rDLPFC.

KEYWORDS
Anxiety, depression, fNIRS, cognitive control network

1 | INTRODUCTION
Anxiety is an irrational state of fear of helplessness and insecurity, a state of fear of danger in which the danger is unrealistic. Moderate anxiety can remind people to pay attention to things around them. However, when the fear state becomes irrational and burdens the individual, general anxiety and even panic ensue. Pathological anxiety, also known as anxiety disorder, is a common psychological disorder associated with affective disorders, which is beyond the control of individuals and interfere with their normal functioning (Carey, et al., 2021). Therefore, this study mainly aims to explore whether there are functional abnormalities in the cognitive control network of the brain in anxiety disorders.

The key aspect of cognitive decline in anxiety is the internal sense of loss of control or impaired attention to inhibiting emotional responses to unrealistic danger or unwanted thoughts. This sense of loss of control in anxiety disorders may be due to dysfunction of the top-down control. Some studies have reported that anxiety is associated with decreased neural activity in the frontal cortex (Basten, Stelzel, & Fiebach, 2012; Bishop, Duncan, Brett, & Lawrence, 2004; Bishop, 2009). Bishop found that in threat-related attention tasks, higher trait anxiety is associated with decreased neural activity in the prefrontal (PFC) (Bishop, Duncan, Brett, & Lawrence, 2004). They then allowed participants to perform conflict tasks, and also found that high trait anxiety is associated with decreased neural activity in PFC (Bishop, 2009). This suggests that trait anxiety is associated with broader attention control disorders in the PFC, even without threat-related stimuli. Basten and Comte found that during the working memory task, high trait anxiety levels were related to stronger deactivation of the region related to the brain default pattern network; namely, the rostral-ventral anterior cingulate cortex (ACC) (Basten, Stelzel, & Fiebach, 2012; Comte, et al., 2015). Similarly, Ansari and Derakshan (2011) used ERP research to find that in response-inhibition tasks, high-anxious individuals have a longer latency in response to the inhibited target (Ansari & Derakshan, 2011). Moreover, in a period of time before the inhibited target appears, the ERP activity in the frontal lobe center...
and the central recording site is lower for these individuals than those of the low-anxiety individuals. This shows that anxiety interferes with top-down inhibitory processing.

We still do not fully understand the underlying neural mechanism of anxiety disorder even though there are some studies on social anxiety disorder (SAD) (Holas, Krejtz, Cyprianska, & Nezlek, 2014; Kawashima, et al., 2016; Yokoyama, et al., 2015), trait anxiety (Basten, Stelzel, & Fiebach, 2012; Bishop, 2009), and anxiety accompanied by depression (Delaparte, et al., 2017; Kessler, et al., 2015; Wu, et al., 2022). Whether anxiety causes activation or deactivation of the brain during cognitive processes is still a controversial question (Kawashima, et al., 2016; Yokoyama, et al., 2015). Yokoyama et al., using the fNIRS technique, found that, compared with the healthy control group, the concentration of oxygenated hemoglobin (oxy-Hb) in the ventrolateral prefrontal cortex (VLPFC) of patients with anxiety changed less during a verbal fluency task (VFT) (Yokoyama, et al., 2015). This suggests that the decreased VLPFC activity among the task-related patients may be a neural marker for the SAD patients. However, Kawashima et al. (2016) also used the fNIRS technique to find that the left frontal cortex is overactivated during VFT among anxiety disorders (Kawashima, et al., 2016). The results of these studies are inconsistent regarding changes in neural function in anxiety disorders. Therefore, it is necessary to further explore the changes in its neural function, which will help to further intervene and treat anxiety disorders.

More importantly, it is worth noting that most anxiety disorders are associated with other mental disorders, with depression being the most common comorbidity. According to the World Health Organization’s World Mental Health Survey, the comorbidity rate of anxiety and depression was 45.7% (Kessler, et al., 2015). Therefore, in most existing studies, anxiety and depression are often studied together, and anxiety disorders are often regarded as accompanying symptoms of depression (Delaparte, et al., 2017; Kessler, et al., 2015; Wu, et al., 2022). However, functional impairment is more severe in patients with combined anxiety and depressive disorders (Crane, et al., 2016), and their brain functions may be different from those of patients who suffer from anxiety alone. Few studies have focused on the symptoms of anxiety after controlling for depression to study the underlying neural mechanisms of its pathogenesis. Therefore, in this study, we control for depressive symptoms and the concerned about whether the severity of anxiety symptoms affects brain cortical activation.

Multi-channel fNIRS is an optical neuroimaging technology that has been applied to the research of several neuropsychiatric disorders (Rahman, Siddik, Ghosh, Khanam, & Ahmad, 2020), such as anxiety disorders (Ishikawa, et al., 2014). The fNIRS technology uses a NIR light source and detector placed on the scalp to measure the change in reflected NIR light intensity, which is mainly due to the difference in the absorbance of oxyhemoglobin (oxy-Hb) and deoxyhemoglobin (deoxy-Hb) in the subcortical blood vessels. Therefore, fNIRS can quantify concentration changes in oxy-Hb and deoxy-Hb. Previous studies have shown that the cerebral cortex hemoglobin signal recorded by fNIRS is similar to that recorded by functional MRI, and this hemodynamic signal is considered a representation of brain activity (Duan, Zhang, & Zhu, 2012; Pinti, et al., 2018; Sato, et al., 2013). The prefrontal cortex is closely associated with the primary formation of fear memories, thus offering new possibilities for studying the neurobiology of the underlying abnormal fear in anxiety disorders (Dejean, et al., 2015). Therefore, in this study, the fNIRS system channel covers the frontotemporal region.

Impaired verbal fluency is common in patients with mental disorders; therefore, the verbal fluency task (VFT) is usually used to diagnose them (Raucher-Chéné, Achim, Kaladjian, & Besche-Richard, 2017; Yeung & Lin, 2021). The VFT is a commonly used neuropsychological tool that can measure executive function (Whiteside, et al., 2016) and is often used to assess cognitive changes and impairment (McDonnell, et al., 2020). Neuroimaging studies have found that the VFT depends on frontotemporal lobe function, especially on the dominant side (Henry & Crawford, 2004). Therefore, in this study, we investigated whether changes in frontal activation during the VFT correlate with anxiety disorder severity. To exclude the effect of depression, we selected only individuals with different levels of anxiety whose depression was at normal levels. We allowed the participants to perform the VFT while recording the hemodynamic response of the fNIRS system.

Based on the evidence of an association between anxiety disorders and frontal dysfunction found in previous
studies (Basten, Stelzel, & Fiebach, 2011; Basten, Stelzel, & Fiebach, 2012; Bishop, Duncan, Brett, & Lawrence, 2004; Bishop, 2009; Conte, et al., 2015), we predicted that generalized anxiety severity during the VFT would be associated with the degree of frontal function activation, as well as significantly lower frontal function activity in patients with severe anxiety disorders.

2 | MATERIALS AND METHODS

2.1 | Participants

Participants were recruited from the psychiatry department of the hospital of the Huazhong University of Science and Technology in Wuhan, China from September 2020 to May 2023. All subjects were assessed with the Mini International Neuropsychiatric Interview (MINI-Chinese version) and the Hospital Anxiety and Depression Scale (HADS).

According to the Hospital Anxiety and Depression Scale (HADS), there were 366 subjects whose depression levels were normal (HAD, D [≥] 7). The 139 healthy subjects (HAD, A [≥] 7) comprised 75 males and 64 females (mean age ± SD: 20.34 ± 2.55). The 117 mild anxiety subjects (HAD, 0 [≥] A < 11) comprised 57 males and 60 females (mean age ± SD: 21.59 ± 2.25). The 110 major anxiety subjects (HAD, A [≥] 11) comprised 55 males and 55 females (mean age ± SD: 21.02 ± 2.17).

2.2 | Measures

In the area of anxiety, there are several outcome tools to measure the overall level of anxiety, such as the Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown, & Steer, 1988), the Hamilton Rating Scale for Anxiety (HAMA) (Hamilton, 1959), the State-Trait Anxiety Inventory (STAI-S trait and STAI-Trait) (Spielberger, 1970) and Hospital Anxiety and Depression Scale (HADS) (Snaith, 1992). This study used the HADS to test depression and anxiety. HADS is a scale designed as a short self-reported measure depression and anxiety. It performs well in assessing the symptoms of anxiety disorder and depression in physical, psychiatric and the general population (Bjelland, Dahl, Haug, & Neckelmann, 2002; White, Leach, Sims, Atkinson, & Cottrell, 1999). The reliability and validity of the scale were verified in different languages (including Chinese) (Herrero, et al., 2003; Whelan-Goodinson, Ponsford, & Schonberger, 2009). HADS is a 14 item self-reported scale including 7 items measure depression and 7 items measure anxiety (Snaith, 2003). Each item is scored on a four-point scale, ranging from 0 to 3. 3 indicates a high frequency of symptoms. The total score of each subscale ranges from 0 to 21 points: normal (0 to 7 points), suspicious (8 to 10 points) and diagnostic (11 to 21 points).

2.3 | Activation task

We selected VFT task to distinguish the cognitive executive function of anxious depression and non-anxious depression. VFT requires subjects to perform word formation task according to the given Chinese characters when they hear specific Chinese characters such as “上,” “时,” “说,” and “家”). This block contains a 30 second pre task rest, a 60 second VFT task, and a 60 second post task rest (Figure 1).

FIGURE 1 The verbal fluency task protocol.

2.4 | fNIRS measurement
A 53-channel fNIRS device (BS-7000, Wuhan Znion Technology Co., Ltd., China) was used to measure the three types of relative concentration of oxy-Hb, deoxy-Hb, and total-Hb by near-infrared light of a specific wavelength. The instrument has 16 pairs of emission and detector probes (Fig. 2). The wavelengths of light emitted are 760 nm and 850 nm, and the frequency is 100 Hz. The distance between each emitter and detector is 2.9–3.1 cm. The area between the emitter and detector probes consists of a channel. Each probe was positioned on the scalp of the forehead. The optode arrangement is shown in Figure 2 and is based on the 10/20 System of Electrode Placement method, which is also commonly used in EEGs (Okamoto, et al., 2004).

2.5 | Regions of interest (ROIs)

ROIs were set at the following regions: Left Broca’s area (detected by the 2nd, 3rd, 5th, 7th, 8th and 13th channels), Right Broca’s area (detected by the 44th, 49th, 50th, 46th, 51st and 53rd channels), Left Dorsolateral prefrontal cortex (detected by the 6th, 11th, 14th, 17th, 18th and 20th channels), Right Dorsolateral prefrontal cortex (detected by the 31st, 32nd, 34th, 39th, 42nd and 45th channels), Left Frontal eye fields (detected by the 12nd and 24th channels), Right Frontal eye fields (detected by the 26th and 38th channels), Left Frontopolar area (detected by the 9th, 15th, 16th, 19th, 21st, 22nd, 23rd, 27th and 28th channels), Right Frontopolar area (detected by the 30th, 33th, 35th, 36th, 37th, 41st, 43rd and 48th channels), Left Motor and Supplementary Motor Cortex (detected by the 1st, 4th and 10th channels) and Right Motor and Supplementary Motor Cortex (detected by the 40th, 47th and 52nd channels) (Figure 2).

FIGURE 2 Locations of the 53-channels for the functional near-infrared spectroscopy (fNIRS). Estimated cortical areas corresponding to each channel using the virtual registration method in (A) the right parietal and temporal areas, (B) the frontal area, and (C) the left parietal and the temporal areas.

2.6 | Statistical analysis

In the fNIRS data preprocessing section. The fNIRS data were analyzed using Homer2 package based on Matlab (M., et al., 2016). Firstly, the raw light intensity file has been converted to homer2 file format (.nirs). Then, the raw NIRS data were first converted to optical density (function: hmrIntensity2OD), and using the manufacturer’s recommendations, channels with a variation coefficient greater than 7.5% are considered bad channels and deleted from the analysis. A wavelet transform was used to correct for motion artifact (function: hmrMotionCorrectWavelet) using the default interquartile range (0.1), as this is optimal for motion correction. Any remaining motion artifact was then removed through the motion artifact detection tool (function: hmrMotionArtifact, tMotion = 0.5, tMask = 1.0, STDEVthresh = 15, AMPthresh = 3.0). The signal was then bandpass filtered (function: hmrBandpassFilt, hpf = 0.00, lpf = 0.10) to remove baseline drift and physiological noise. Finally, the concentration changes of oxy-Hb and the deoxy-Hb were then computed according to the Modified Beer-Lambert Law. The oxy-Hb and the deoxy-Hb values were then saved as text files for each subject. Finally, the oxy-Hb and the deoxy-Hb time series for each subject was z-scored by channel.

3 | RESULTS
3.1 | Correlation between anxiety severity and cortical activity

According to previous studies, we predicted that the severity of anxiety was negatively correlated with the activation of the prefrontal cortex during the VFT. Pearson correlation results showed that there was a negative trend between anxiety severity and the mean oxy-Hb concentration during VFT in the right dorsolateral prefrontal area (rDLPFC) \( r = -0.226, p = 0.066 \) (Figure 3AB) and in the left FEF during VFT \( r = -0.258, p = 0.035 \) (Figure 3 CD). This shows that the anxiety may result from the decrease of cortical activity in the prefrontal lobe (including right DLPFC and left FEF).

3.2 | Comparison of cortical activity of oxy-Hb among study groups

According to previous studies and the Pearson correlation results in this study, we predicted that the activation in the right DLPFC and the left FEF during the VFT in major anxiety disorder is decreased compared with healthy individuals. Therefore, first, we took the mean oxy-Hb concentration in the right DLPFC during VFT as the dependent variable, did ANOVA analysis between the three anxiety severity groups, and found that the main effect of groups was also significant, \( R^2 < 0.001, F_{(df=2)} = 3.587, p = 0.033 \). The post analysis results showed that the mean oxy-Hb concentration in the right DLPFC during VFT in the major anxiety group \( m = 0.047, SE = 0.606 \) was significantly lower than that in the control group \( m = 0.896, SE = 0.942, p = 0.009 \) and was lower than that in the mild anxiety group \( m = 0.730, SE = 1.019 \), and the statistical results reached the marginal significant level \( p = 0.059 \) (Figure 4A). This suggests that the decreased cortical activity in right DLPFC in patients with major anxiety may be a neural marker of their anxiety symptoms.

Second, we took the mean oxy-Hb concentration in the left FEF during VFT as the dependent variable and...
did ANOVA analysis between the three anxiety severity groups. The results show that the main effect of groups is significant, $R^2 = 0.000$, $F_{(df=2)} = 4.935$, $p = 0.010$. Post analysis showed that the mean oxy-Hb concentration in the left FEF of the major anxiety group ($m = -1.255$, $SE = 2.215$) was significantly lower than that in the control group ($m = 0.601$, $SE = 1.646$) ($p = 0.003$) and was lower than that in the mild anxiety group ($m = 0.050$, $SE = 1.608$), and the statistical results reached the marginal significant level $p = 0.057$ (Figure 4B). This suggests that the decreased cortical activity in left FEF in patients with major anxiety may be a neural marker of their anxiety symptoms.

FIGURE 4 (A) The oxy-Hb concentration in the rDLPFC during VFT between three anxiety severity groups; (B) The oxy-Hb concentration in the lFEF during VFT between three anxiety severity groups.

4 | DISCUSSION

Anxiety is an irrational state of fear of helplessness and insecurity, or a state of fear of unrealistic danger that deeply impacts our psychological functioning (Nechita, Nechita, & Motorga, 2018), leading to problems such as the decreasing ability to read text (Calvo & Carreiras, 1993), feeling a lack of success or failure (Yajima & Arai, 1996), making decisions with low risk but low return (Raghunathan & Pham, 1999), and having lower levels of learning aptitudes (Cassady, 2004). The global incidence rate of anxiety disorder within one year is as high as 13% (Steel, et al., 2014), and the lifetime prevalence has reached 5.7% (Kessler, et al., 2005). Although anxiety disorders deeply impact psychological functioning and their prevalence is high, the underlying neural mechanism is still largely unclear. Although previous studies have examined the neural mechanisms of social anxiety, trait anxiety, and anxious depression, few have examined the neural mechanisms of anxiety after controlling for depressive symptoms. Therefore, this study aimed to explore whether there are differences in brain cortical activity based on severities of anxiety disorders among patients with normal depressive symptoms.

We selected people aged 18–24 years as the subjects because, among people of all ages, the proportion of anxiety disorders among this age group is the highest (Alonso, Angermeyer, Bernert, Bruffaerts, & Vollebergh, 2004). The Hospital Anxiety and Depression Scale (HADS) was used to test anxiety and depression. fNIRS was used to detect oxy-Hb concentration during the VFT. Anxiety severity was significantly and negatively correlated with oxy-Hb concentration in the IFEF. The oxy-Hb concentration in the IFEF of patients with major anxiety disorder was significantly lower than that of healthy controls. In addition, there was a negative correlation between anxiety severity and oxy-Hb concentrations in the rDLPFC. The oxy-Hb concentration in the rDLPFC among patients with major anxiety disorders was significantly lower than that in the control group. These results indicate that decreased cortical activity in the rDLPFC and IFEF in major anxiety disorder patients may be a neural marker of anxiety symptoms.

4.1 | Anxiety is closely related to dysfunctional cortical activity in the cognitive control network
Previous studies have found that the severity of GAD is related to the uncontrollability of unrealistic worries (Hallion & Ruscio, 2013; Hallion, Tolin, Assaf, Goethe, & Diefenbach, 2017), and cognitive control can inhibit such unrealistic worries. Therefore, cognitive control plays an important role in anxiety disorders. The core network of cognitive control is the CCN, comprising the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), and posterior parietal cortex (Niendam, et al., 2012). Previous fNIRS-related studies of individuals with high social anxiety found that in state rumination, the activation of the brain region of the CCN was reduced, which resulted in reduced cognitive and attention control in individuals with higher social anxiety (Laicher, et al., 2022). Our results showed a negative correlation between anxiety severity and cortical activation of the rDLPFC, a part of the CCN. Our results also found that anxiety severity was significantly and negatively correlated with cortical activation of the IEF, a part of the frontoparietal cortex. The frontoparietal region is the brain region predominantly involved in cognitive control (Niendam, et al., 2012). These results indicate that anxiety is closely related to cognitive dysfunction during the VFT.

4.2 Decreased cortical activity of the CCN in patients with major anxiety disorder may be a neural marker for anxiety symptoms

The oxy-Hb concentrations in both the IEF and rDLPFC in the major anxiety disorder group were significantly lower than that in the control group. Both the rDLPFC and IEF are part of the brain region predominantly involved in cognitive control. Consistent with our results, some fNIRS studies found that cognitive control is impaired in anxiety-related disorders. For example, Ohta et al. used fNIRS to find that in the non-emotional VFT experiment, panic disorder showed hypofrontality activation (Ohta, et al., 2008), indicating that cognitive control impairment does not seem to be related to emotional processes. Nishimura et al., using fNIRS, found that the cortical activity in the left inferior frontal cortex in patients with panic disorder during the VFT was significantly less than that among the healthy control group (Nishimura, et al., 2007); thus, they believed that the left frontal lobe, which is critical for active attention control, may be damaged in patients with panic disorder. Bishop also found that trait anxiety is related to impaired prefrontal control of attention (Bishop, 2009). In addition, some ERP-related studies have reported that cognitive control is impaired (Righi et al., 2009; Cavanagh et al., 2017. Righi et al. (2009), using ERP, found that in a continuous attention task, N2 amplitude increased among participants with high trait and state anxiety, and so they believed that the attention function of anxiety disorder is impaired (Righi, Mecacci, & Viggiano, 2009). Cavanagh et al. (2017), using ERP, found that in the executive control task, ERR, or error related to $\theta$ power, and the correlation between reaction time and $\theta$ power, could predict GAD status (Cavanagh, Meyer, & Hajcak, 2017). Further, they believed that the executive control function is impaired with GAD. Thus, decreased cortical activity of the CCN in major anxiety disorders may be a neural marker for anxiety symptoms.

However, inconsistent with our results, some studies have reported anxiety is associated with increased neural activity (Basten, Stelzel, & Fiebach, 2011; Basten, Stelzel, & Fiebach, 2012; Comte, et al., 2015) and decreased functional connectivity (Basten, Stelzel, & Fiebach, 2011; Comte, et al., 2015). Anxiety tendency is closely related to changes in emotional function, showing that, in selective attention, anxious individuals are highly sensitive to threat-related and vague emotional stimuli. Some researchers have found that, in affective-related tasks, the DLPFC (Basten, Stelzel, & Fiebach, 2011) and ACC (Comte, et al., 2015) of individuals with high trait anxiety showed stronger task-related activation; however, the connectivity between the PFC and ACC was reduced (Basten, Stelzel, & Fiebach, 2011; Comte, et al., 2015). This shows that, when faced with incompatible emotional information, the neural processing efficiency of attention control in trait anxiety disorders is inefficient. The overactivated DLPFC attempts to compensate for inefficient connectivity. Basten et al. (2012) found that in working memory tasks, trait anxiety is related to stronger functional coupling between the right DLPFC and ventrolateral prefrontal cortex, and higher levels of trait anxiety are related to stronger activation of the rDLPFC and left inferior frontal sulcus (Basten, Stelzel, & Fiebach, 2012). This shows that anxiety can affect cognitive processing even in the absence of threat-related stimuli. The overactivation of the rDLPFC and left inferior frontal sulcus is a compensatory effect. These findings, which
are inconsistent with our results, may reflect the different task types used. In conclusion, patients with anxiety disorders showed low efficiency in terms of cortical functional activation and cognitive processing efficiency.

4.3 | Limitations

However, this study also has some limitations. First, Hospital Anxiety and Depression Scale (HADS) was used to evaluate the symptoms of anxiety and depression among all groups. However, the mainstream psychiatric diagnostic scale Hamilton Depression Scale (HAM) was not used (Ho, et al., 2020). Different measurements may lead to different research results. Therefore, HAM, Montgomery Asperger Depression Scale or Beck Depression Scale should be used for further verification. Second, in this study, the 53 channels of fNIRS only covered the frontal and temporal cortex, but not the posterior and deeper brain regions, such as amygdala.

5 | CONCLUSION

The anxiety is closely related to the dysfunction of cortical activity of the cognitive control network (CCN). The decreased cortical activity of CCN in major anxiety disorders may be a neural marker of their anxiety symptoms.

AUTHOR CONTRIBUTIONS

Yan Zhang: Supervision; conceptualization; review and editing.
Caihong Yang: Conceptualization; data collection; data analysis; methodology; visualization; writing.
Pu Wang: supervision; conceptualization; review and editing.
Huifen Wu: Methodology; review.
Yueran Bian: Data collection.
Fang Xu: Data collection.
Qiang Xiao: Data collection.
Nian Xiang: Data collection.
Min Qiu: Data collection.

ACKNOWLEDGMENTS

This research was supported by Philosophy and Social Science Research in the Universities of Hubei Province. Project number: 21Q247. We gratefully acknowledge the staff of the Department of Psychiatry, Hospital of Huazhong University of Science and Technology in China. We would like to appreciate all the participants in this study.

CONFLICT OF INTEREST STATEMENT

We have no known conflict of interest to disclose.

DATA AVAILABILITY STATEMENT

Raw data within this article will be made available upon request. If you would like to access the raw data and analysis, please email Caihong Yang at the following e-mail address: ychpsychology@163.com.

REFERENCES


