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The anxiety-specific hippocampus–prefrontal cortex pathways links to procrastination through self-control

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Abstract

Procrastination, which is defined as delaying an intended course of action despite negative outcomes, is demonstrated to have a deal with negative emotion including trait anxiety. Although highly anxious individuals showed impoverished control ability, no studies have indicated the role of self-control in the relationship between trait anxiety and procrastination, and its neural correlates. To this end, we used the sliding window method to calculate the temporal deviation of dynamic functional connectivity (FC) in 312 healthy participants who underwent the resting-state functional magnetic resonance imaging (fMRI) scanning. In line with our hypothesis, higher trait anxiety is linked to more procrastination via poorer self-control. Besides, the dynamic FC analyses showed that trait anxiety was positively correlated with dynamic FC variability in hippocampus–prefrontal cortex (HPC–PFC) pathways, including left rostral hippocampus–left superior frontal gyrus (left rHPC–left SFG), and left rHPC–right middle frontal gyrus (left rHPC–-MFG). Furthermore, the structural equation modeling (SEM) uncovered a mediated role of self-control in the association between the anxiety-specific brain connectivity and procrastination. These findings suggest that the HPC–PFC pathways may reflect impoverished regulatory ability over the negative thoughts for anxious individuals, and thereby incurs more procrastination, which enhances our understanding of how trait anxiety links to procrastination.

KEYWORDS
dynamic functional connectivity, procrastination, self-control, structural equation modeling, trait anxiety

1 | INTRODUCTION

Procrastination is referred to as "to voluntarily delay an intended course of action despite expecting to be worse off for the delay" (Steel, 2007). Approximately 20% of adults procrastinate problematically, suggesting its high prevalence among the public (Harriott & Ferrari, 1996). In the long run, it exerts an adverse influence on aspects of life, including poorer academic performance (Kim & Seo, 2015), less career/financial success (Mehrabian, 2000), and even increased health problems (e.g., sleep; Przepiórka, Błachnio, & Siu, 2019). Given the detrimental impact, researchers seeking to probe the nature of procrastination propose that procrastination is a quintessential form of self-regulatory failure, resulting from the primacy of repairing short-term mood that derived from task aversiveness (e.g., anxiety) over pursuing the goal-directed behavior (Sirois & Pychyl, 2013). Trait anxiety, which is often used as an analog for the

presence of an anxiety disorder (Sylvester et al., 2012), is defined as predisposition to keep intense anxious experience despite no stressors (Spielberger & Gorsuch, 1983). Prior studies uncover a positive association between trait anxiety and procrastination (Constantin, English, & Mazmanian, 2018; Eerde, 2003). Moreover, people with higher anxiety levels are proposed to show the weakened control ability, one that may regulate the resources occupied in pursuing goal-directed behaviors (Eysenck, Derakshan, Santos, & Calvo, 2007). However, less is known about whether control ability plays a role in the association between trait anxiety and procrastination, and its underlying neural basis.

More specifically, anxious individuals are characterized by capturing attention towards potential signals of danger (Bishop, 2007). As attentional control theory suggests, anxiety impedes the processing efficiency of goal-directed system, biasing the preference for stimulus-driven processing (Eysenck et al., 2007). For example, trait anxiety is associated with impoverished deficiencies in the executive control network that incorporates inhibition in paradigms such as attention network test (ANT; Pacheco-Unguetti, Acosta, Callejas, & Lupiáñez, 2010) and Go-No-Go task (Edwards, Edwards, & Lyvers, 2017), suggesting greater difficulties in high-trait-anxiety participants in controlling interference efficiently. Importantly, following temporal decision model, to procrastinate or not relies on weighing between task aversiveness and incentive outcomes (S. Zhang, Liu, & Feng, 2019), which may be modulated by self-control, a capacity to allocate resources for supporting the pursuit of long-term goals (Baumeister, Vohs, & Tice, 2007). In line with this connotation, procrastination is the consequence of failure of top-down control over task aversiveness (Sirois & Pychyl, 2013; Steel, 2007). However, options with greater delayed rewards will be preferred when the prefrontal self-control is exerted (Ballard et al., 2017; Figner et al., 2010). These findings indicate procrastination occurs when self-control fails to override the task aversiveness, or cannot help people direct attention to incentive outcomes a task will yield. Collectively, we assume that higher trait anxiety may weaken individuals' control ability, which thereby engenders more unnecessarily delay.

To date, neural bases underpinning the processes linking trait anxiety to procrastination are as yet unclear. Specifically, anxiety is characterized by the dysfunction of some brain regions, especially the cortico-limbic systems including prefrontal cortex (PFC) and hippocampus (HPC) in rodents and humans (Chavanne & Robinson, 2021; Daviu, Bruchas, Moghaddam, Sandi, & Beyeler, 2019; Mobbs & Kim, 2015). In particular, the direct monosynaptic projection from HPC, especially the ventral part, to PFC is crucial for the expression of the anxiety-like behavior. For instance, oscillatory synchrony (theta-frequency, 4–12 Hz) in the HPC–PFC network emerges when the rodents are exposed to anxious environments (e.g., elevated plus maze; Adhikari, Topiwala, & Gordon, 2010). Inhibition of the direct HPC input to PFC decreases anxiety (Padilla-Coreano et al., 2016), highlighting the importance of HPC-to-PFC circuit for anxiety. Generally speaking, HPC is preferentially recruited in contextual association, and episodic prospection or memory (Godsil, Kiss, Spedding, & Jay, 2013; Maguire & Hassabis, 2011; Martin, Schacter, Corballis, & Addis, 2011), while PFC, especially its dorsal and anterior parts, supports top-down control for the focal planning (Gläscher et al., 2012; Momennejad & Haynes, 2012). Thus, the HPC–PFC circuit is identified as attending to not only episodic memory (Chao, de Souza Silva, Yang, & Huston, 2020), but also working memory that is a core element of executive function (Bilek et al., 2013). The immediate route between these regions allows HPC to generate the spatiotemporal construction of event, and deliver information to PFC where serves as an executor to exert regulation (Chao et al., 2020). Moreover, resting-state functional magnetic resonance imaging (fMRI) studies from humans find that trait anxiety is negatively associated with the static functional connectivity (FC) between default mode network (i.e., middle temporal gyrus that embedded HPC) and frontoparietal cortex (Modi, Kumar, Kumar, & Khushu, 2015; R. Zhang, Chen, Liu, & Feng, 2019). Recently, researches uncover that the spontaneous brain activation during the resting-state changes over time rather than being stationary, and dynamic FC is thus a good metric to quantify the variability in strength or spatial dynamic organization of brain connectivity pattern (Hutchison et al., 2013), which is sensitive to distinguish patients with anxiety disorders (e.g., GAD and PTSD) and health controls precisely (Jin et al., 2017; Yao et al., 2017). The dynamic measures of FC enable the researchers to understand the transient variation of neural correlates for trait anxiety. However, no studies have investigated the neurocognitive basis underlying how trait anxiety links to procrastination from the dynamic view thus far.

In line with the findings mentioned above, emerging researches focusing on the dynamic nature of spontaneous brain activation reveal that patients with anxiety disorders, especially generalized anxiety disorder (GAD), exhibit the increased dynamic amplitude of low-frequency fluctuation (ALFF) and increased dynamic functional connectivity density in brain regions implicated in the prefrontal lobes (i.e., dorsomedial PFC) and HPC (Y. Chen et al., 2020; Cui et al., 2020). Moreover, greater variability of the dynamic FC between these regions, namely parahippocampal gyrus–superior frontal gyrus, is associated with higher scores of posttraumatic stress disorder (PTSD) across all traumatized subjects (H. J. Chen et al., 2021). Importantly, individuals with highly trait anxiety, who are susceptible to developing the anxiety disorders, are lack of the engagement of prefrontal control, and incapable of inhibiting the distractor efficiently even in the absence of threats (Bishop, 2009), which indicates impoverished control among anxious individuals. These findings suggest that individuals with higher trait anxiety may be accompanied by amplified dynamic brain communication of the cortico-limbic circuit, which reflects a tendency for preoccupation of negative future thoughts to distract attention from goals. Hence, we propose the HPC–PFC pathway that underlies trait anxiety may impair individuals' control ability, and thereby causes more procrastination.

The current study consequently investigated the neurocognitive bases accounting for how trait anxiety is linked to procrastination. We conducted a mediation analysis to explore the role of self-control in anxiety–procrastination linkage. To further depict the neural pattern underlying trait anxiety, a sliding window method was applied to calculate the seed-to-voxel dynamic FC analysis. Specifically, based on

the previous findings (Spampinato, Wood, De, & Grafman, 2009; Zhang, Chen, et al., 2019), the subregions of HPC were defined as seed regions for testing whether trait anxiety was accompanied by dysfunction of hippocampal connectivity with other brain regions (e.g., PFC). Finally, we performed the structural equation modeling (SEM) analyses to testify whether anxiety-specific brain pattern impacted procrastination through self-control.

2 | METHODS

2.1 | Subjects

Three hundred and fifty-nine healthy participants (males, $n = 99$; age $= 20.03 \pm 1.89$), who were the college students as well as the native Chinese speakers from the Southwest University, Chongqing, China, were recruited via online advertisements. Among them, 340 subjects underwent the resting-state functional magnetic resonance imaging (fMRI) scanning. Participants were excluded for excessive head motion ($n = 27$; >2.5 mm in translation and >2.5° in rotation), or functional images with lower than 50% of total temporal points ($n = 1$), yielding a final sample of $n = 312$ subjects (males, $n = 83$; age $= 20.06 \pm 1.68$).

All participants were free of neurological impairment and psychiatric disability. The human procedures were approved by the Institutional Review Board of Southwest University. All subjects provided the written informed consent before the experiments, and were paid after the experiment.

2.2 | Psychological assessment

2.2.1 | Trait anxiety

The subscale of Spielberger State–Trait Anxiety Inventory (STAI) was applied to measure trait anxiety levels (Spielberger & Gorsuch, 1983). This scale includes 20 items, such as "I get in a state of tension or turmoil as I think over my recent concerns and interests". Participants rated on a 4-point Likert scale ranging from 1 (almost never) to 4 (almost always). Higher total scores indicated higher levels of trait anxiety. The scale showed a good internal consistency (Cronbach's $\alpha = .884$).

2.2.2 | Procrastination

The study administered the Pure Procrastination Scale (PPS) to assess procrastination (Svartdal & Steel, 2017). The scale consists of 12 items, such as "I always say, I will do it tomorrow". Subjects responded to the items following a 5-point Likert rating (1, strongly disagree; 5, strongly agree). The total scores on all items were calculated, with higher scores indicating higher tendency of procrastination. This scale showed adequate reliability (Cronbach's $\alpha = .847$).

2.2.3 | Self-control

The levels of self-control were measured by the widely adopted Self-control Scale (Tangney, Baumeister, & Boone, 2004). The scale includes 36 items, such as "I am able to resist the temptation well". The participants responded to the items based on a 5-point Likert rating (1, utterly uncharacteristic; 5, utterly characteristic). Higher total scores indicated higher levels of self-control. The scale showed adequate reliability (Cronbach's $\alpha = .710$).

2.3 | fMRI data acquisition

All subjects underwent the resting-state scanning on a TRIO 3.0 T scanner (Siemens Medical, Erlangen, Germany). Before scanning, participants were instructed to keep their eyes closed, stay relaxed, and remain physically still, but not to fall asleep. The whole scanning lasted 12 min, resulting in 360 volumes for each participant. The scanning parameters for functional images were as followed: TR = 2,000 ms; TE = 30 ms; flip angle = 90°; resolution matrix = 64×64; FOV = 200×200; slices = 33; voxel size = $3.1 \times 3.1 \times 3.6$ mm^3.

2.4 | Data preprocessing

The functional images were preprocessed in Data Processing Assistant for resting-state fMRI protocol (DPARSF, http://rfmri.org/DPARSF; C. Yan & Zang, 2010). The initial 10 volumes were removed for ensuring steady-state magnetization. The remaining frames underwent slice timing correction for temporal shifts, and realignment for head motion. Subsequently, high-resolution T1-weighted images were coregistered to the mean functional image, after which the transformed images were segmented into gray matter (GM), white matter (WM), and cerebrospinal fluid (CSF; Ashburner & Friston, 2005). Next, the functional images were normalized to Montreal Neurologic Institute (MNI) space via the DARTEL tool (Ashburner, 2007), resliced at a resolution of $3 \times 3 \times 3$ mm^3 voxel size, as well as spatially smoothed with a Gaussian Kernel of 4 mm full-width at half-maximum (FWHM). Besides, multiple linear regression analyses were conducted to exclude the nuisance signals including Friston 24-parameter motion (i.e., 6 head motion parameters, 6 head motion parameters one-time point before, and the 12 corresponding squared items), and averaged signals from WM, CSF, and global signal (Friston, Williams, Howard, Frackowiak, & Turner, 1996). Moreover, we performed temporal filtering (0.01–0.08 Hz) and liner detrending (Fox, Zhang, Snyder, & Raichle, 2009). Finally, given that motion was unable to be fully controlled by regression analysis (Power, Barnes, Snyder, Schlaggar, & Petersen, 2012), the voxel-specific head motion at the individual level was conducted by data scrubbing to remove the time points that were affected by excessive motion (Satterthwaite et al., 2013), where bad time points in the frame-wise displacement (FD) censoring was flagged following the criteria of any volume with FD > 0.2 mm, as well as volumes 2 forward and 1 back from these volumes (Power et al., 2012; C.-G. Yan, Wang, Zuo, & Zang, 2016).

2.5 | Dynamic functional connectivity variability analysis

Prior studies have found that trait anxiety is related to the structural abnormalities in HPC (Spampinato et al., 2009; Zhang, Liu, & Feng, 2019). We therefore defined the subregions of the rostral and caudal HPC bilaterally obtained from the brain atlas (http://atlas.brainnetome.org/bnatlas) as four seed regions including the left/right rostral hippocampus (rHPC), left/right caudal hippocampus (cHPC; ROI, see Figure 1; Fan et al., 2016).

To explore the seed-to-voxel analysis, a sliding window approach was utilized to construct dynamic FC using the DynamicBC toolkit (Liao et al., 2014). Previous studies have suggested that window size length of about 30–60 s provides a much more robust observation for dynamic FC (Hutchison et al., 2013; Preti, Bolton, & Van De Ville, 2017). Hence, we selected a sliding window length of 30 TR (60 s), and shifted with a step size of 1 TR. To test the reliability of our findings, a window size of 50 TR (step size = 1 TR) was applied as well to avoid the spurious fluctuations derived from short time segments. The averaged BOLD time series in each ROI was calculated to compute the seed-to-voxel dynamic FC in each sliding window, which generated a set of sliding window correlation maps for each participant (Tomasi & Volkow, 2010). All maps applied the Fisher's *r*-to-*z* transformation for acquiring distribution normality. The dynamic FC variability was obtained by computing the standard deviation (*SD*) of *z* values at each voxel, and thereby the spatial map of SD for each participant would be generated.

Moreover, to specify the brain regions that connected with the seed ROI, we conducted the second-level analyses in SPM 12 software (https://www.fil.ion.ucl.ac.uk/spm/software/spm12/). Specifically, scores of trait anxiety were entered as a covariate of interest in the multiple linear regression model, while gender, and age were included as covariates of no interest. The statistical maps underwent multiple comparisons correction by Gaussian random filed theory correction (GRF; voxel significance: $p < .005$; cluster significance: $p < .05$).

2.6 | Structural equation modeling analysis

The current study sought to testify whether the dynamic FC pattern of trait anxiety could impact procrastination through self-control. To this end, SEM analyses were conducted in AMOS 23.0, with the tested variables listed below: X = anxiety-specific brain pattern, M = self-control, Y = procrastination. Specifically, following attention control theory, anxiety was characterized by the bottom-up neural pattern, resulting in deficient resources allocated in the goal-directed behaviors (Eysenck et al., 2007). We thus hypothesized that the neural pathways underlying trait anxiety might impede people's control ability over negative stimuli, causing failure of self-regulation. Hence, a latent variable named "anxiety-specific brain pattern" was constructed to represent the HPC-MFG and HPC-SFG dynamic FC for testing its role in the hypothesized mediation model. The variability values of HPC–PFC pathways including the HPC-MFG and HPC-SFG that were derived from window size of 30 TR (50 TR) were extracted based on the "ROI signal extractor" function in DPARSF toolkit. Moreover, two types of fit indices were applied for evaluating the goodness of model fit. On the one hand, the absolute fit indices, which determined how well a priori model fitted the sample data (McDonald & Ho, 2002), were assessed: (a) The normed chi-square value (χ^2/df) was a measure for evaluating overall model fit, whose values not exceeding three indicated good model fitness (Chin & Todd, 1995); (b) The root mean square error of approximation (RMSEA) illustrated how well the model would fit the populations covariance matrix, with values not exceeding 0.06 representing good model fitness (Hu & Bentler, 1999); (c) The Goodness-of-Fit statistic (GFI) was created to calculate the proportion of variance that was explained by the estimated population covariance, whose cut-off values more than 0.95 was appropriate (Tabachnick & Fidell, 2007); (d) The adjusted goodness-of-fit (AGFI) statistic was improved by adjusting the GFI based on degrees of freedom, values of which were more than 0.90 or greater suggesting well model fitness (Hu & Bentler, 1999; Tabachnick & Fidell, 2007). Besides, the incremental fit indices were adopted for comparing the χ^2 value to a baseline model (McDonald & Ho, 2002): (a) The normed-fit index (NFI) evaluated the

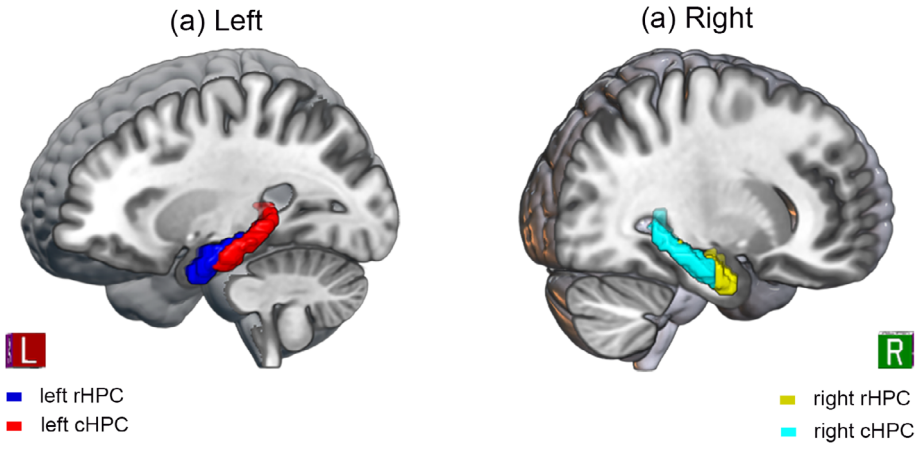

FIGURE 1 The seed regions for dynamic FC analyses. The four subregions of HPC were defined as regions of interest to calculate the seed-to-voxel dynamic FC analyses. HPC from bilateral hemispheres were displayed. R, right; L, left

model by comparing χ^2 of this model to χ^2 of the null model, and its values that were greater than 0.95 indicated a good fit (Hu & Bentler, 1999); (b) The comparative fit index (CFI) was a revised version of NFI considering the effect of sample size, and a value of CFI > 0.95 was known as well fitness (Byrne, 1998; Hu & Bentler, 1999). Notably, we tested the direct and indirect effects of the model by bootstrap estimation procedure (biased-corrected, with 95% confidence intervals; number of bootstrap sample = 1,000; S. Kim, Sturman, & Kim, 2015; Preacher & Hayes, 2004, 2008).

3 | RESULTS

3.1 | The behavioral results

To validate the trait anxiety–procrastination linkage, a Pearson correlation analysis was conducted, and results showed that trait anxiety was positively correlated with procrastination [$r = .480$, $p < .001$; 95% confidence interval (CI): 0.393–0.566]. Besides, to explore whether age and gender affected the variables, we performed another Pearson correlation analysis and the independent sample *t*-test. The results showed that age was not statistically related to either trait anxiety [$r = .032$, $p = .573$; 95% CI: −0.075–0.137], or procrastination [$r = .003$, $p = .954$; 95% CI: −0.111–0.110]. Moreover, there were no gender differences in trait anxiety [$t_{(310)} = -.297$, $p = .766$], and procrastination [$t_{(310)} = 1.568$, $p = .118$]. Moreover, controlling for the covariates of no interest like age, and gender, our results still found a robust positive correlation between such relationships [$r = .376$, $p < .001$; 95% CI: 0.267–0.470].

Collectively, these findings suggest that people with higher trait anxiety tend to procrastinate more.

Furthermore, to testify the role of self-control in the relationship between trait anxiety and procrastination, we performed a mediation analysis using the PROCESS toolkit implemented in SPSS 23 (1,000

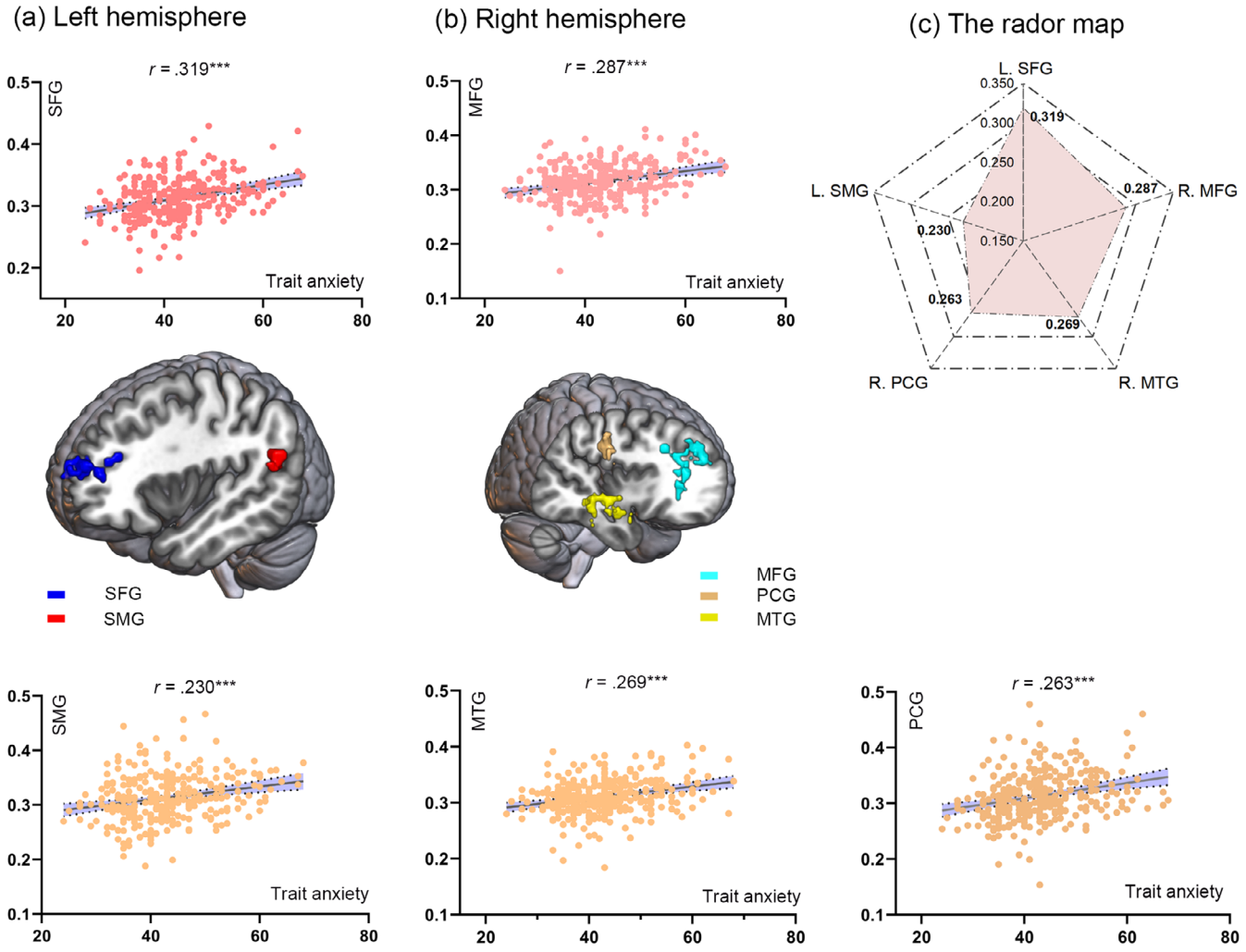

FIGURE 2 The neural connectivity pattern underlying trait anxiety. Higher trait anxiety was related to greater variability of FC between left rHPC and brain regions including left SFG, left SMG, as well as right MFG, right MTG, right PCG, which was illustrated in *panel A* and *panel B*, respectively. Moreover, the radar map in *panel C* further showed the correlation coefficients for displaying the relationship between trait anxiety and temporal variability in the identified regions. Notably, the scatter plots were presented for visual inspection, but not for statistical inference. ***$p < .001$

bootstrap samples; Hayes, 2009; Preacher & Hayes, 2008). The results showed that self-control mediated the relationship between trait anxiety and procrastination (indirect effect = 0.277; biased-corrected CI: 0.209–0.357). When self-control was included in the model, there was also a significant direct effect of trait anxiety on procrastination (direct effect = 0.204; biased-corrected CI: 0.094–0.252, $p \leq .001$), suggesting a partially mediating role of self-control. Taken together, higher trait anxiety is linked to more procrastination through impoverished self-control ability.

3.2 | Dynamic FC variability results

To depict the neural patterns of trait anxiety from a dynamic FC perspective, a sliding window method (window length = 30 TR; see Figure 2, Table 1) was adopted to calculate the seed-to-voxel connectivity. We found that trait anxiety was positively correlated with temporal variability of dynamic FC between the seed region—left rHPC—and left superior frontal gyrus or rostral prefrontal cortex (SFG or rPFC; $r = .319$, $p < .001$), right middle frontal gyrus or dorsolateral prefrontal cortex (MFG or dlPFC; $r = .287$, $p < .001$), right middle temporal gyrus (MTG; $r = .269$, $p < .001$), right precentral gyrus (PCG; $r = .263$, $p < .001$), and left supramarginal gyrus (SMG; $r = .230$, $p < .001$). Notably, the other three seed regions (i.e., left cHPC, right rHPC, and right cHPC) showed no significant clusters (GRF; voxel significance: $p = .005$; cluster significance: $p = .05$; see Supporting Information for more information).

Furthermore, to validate our findings, the seed-to-voxel analyses were conducted with a window size of 50 TR. In the verification analyses, the findings showed a similar connectivity pattern. More specifically, trait anxiety was positively associated with the dynamic FC between the left rHPC and left SFG, right MFG, right MTG, right PCG, and left posterior cingulate cortex (see Supporting Information for more information), which replicated the results mentioned above (window size = 30 TR). Together, these findings show a specific role of left rHPC connectivity for trait anxiety (see Supporting Information for more information).

3.3 | The structural equation modeling results

The SEM analyses were conducted to verify whether the anxiety-specific brain connectivity pattern affected procrastination through self-control. Above all, the results (30 TR) suggested a well fitness of the hypothesized model ($\chi^2 = 3.536$; $p = .171$; CMIN/DF = 1.768, RMSEA = 0.05, GFI = 0.994, AGFI = 0.972, NFI = 0.988, CFI = 0.995). More specifically, the bootstrap analyses showed a significant mediating effect of self-control in linking HPC–PFC dynamic FC to procrastination (indirect effect = 0.128; biased-corrected CI: 0.045–0.224, $p = .004$). Besides, the direct effect of dynamic FC on procrastination was no longer significant when mediator was included in the model (direct effect = 0.044; biased-corrected CI: −0.057 to 0.145, $p = .368$; Figure 3, and Table 2). To test the robustness of our findings, we conducted another SEM analysis to test whether the dynamic FC pattern derived from 50 TR could relate to procrastination through self-control, and the finding replicated what we reported before (see Supporting Information for more information). Collectively, the results indicate that the anxiety-specific brain pattern, namely the combined dynamic FC of left rHPC–left SFG, left rHPC–right MFG, may impact procrastination through self-control.

4 | DISCUSSION

In the current study, we found that self-control mediated the relationship between trait anxiety and procrastination. Besides, the dynamic FC analyses revealed higher trait anxiety was specifically related to greater variability of dynamic FC between left rHPC and other regions, especially the HPC–PFC circuits including left rHPC–left SFG, and left rHPC–right MFG. More importantly, the SEM results demonstrated that HPC–PFC pathways that underlay the neural correlates of trait anxiety impacted procrastination through self-control. Taken together, these findings shed lights on how trait anxiety links to procrastination from a novel perspective, and provide initial evidence for further interventions.

TABLE 1 The dynamic FC between left rHPC and identified regions underlay the neural substrates of trait anxiety (GFR correction; voxel $p = .005$, cluster $p = .05$)

		MNI				
Identified regions	**BA**	***x***	***y***	***z***	**Voxels**	***t***
L. SFG/rPFC	10	−18	60	18	62	4.544
R. MFG/dlPFC	46	27	54	21	83	3.962
R. MTG	22	63	−12	−15	58	3.949
R. PCG	6	54	−6	21	48	3.954
L. SMG	40	−60	−51	24	34	4.013

Abbreviations: BA, Brodmann area; L. SFG/rPFC, left superior frontal gyrus or rostral prefrontal cortex; L. SMG, left supramarginal gyrus; R. MFG/dlPFC, right middle frontal gyrus or dorsolateral prefrontal cortex; R. MTG, right middle temporal gyrus; R. PCG, right precentral gyrus.

4.1 | Self-control mediated the association between trait anxiety and procrastination

Higher trait anxiety was found to show more procrastination through poorer self-control. Growing evidence demonstrated the crucial role of self-control in procrastination (Przepiórka et al., 2019; Zhao et al., 2019). For example, executive functions (e.g., inhibition) that facilitated self-control were significant predictors of procrastinators (Rabin, Fogel, & Nutter-Upham, 2011). Compared to non-procrastinators, procrastinators showed a reduced ability to override the immediate rewards that disrupted the goal pursuits (Chu & Choi, 2005; Wolters, 2003), which suggested a lack of self-control ability among those who procrastinated more (Steel, 2007; Wijaya & Tori, 2018). People, who were trained for strengthening self-control, benefited from sustaining the pursuit of long-term goals with a reduced intention-action gap (Grunschel, Patrzek, Klingsieck, & Fries, 2018; Job, Friese, & Bernecker, 2015). More broadly, self-control was proposed to engage in the value-based decision-making to enact a more valued option via weighing between gains (e.g., money) and costs (e.g., effort; Berkman, Hutcherson, Livingston, Kahn, & Inzlicht, 2017), which modulated the intro-processes underlying procrastination. Specifically, the motivation to act hinged on weighing between engagement utility (i.e., task aversiveness) and outcome utility (i.e., task outcome; S. Zhang, Liu, & Feng, 2019). For highly anxious individuals, they were susceptible to negative repetitive thoughts (i.e., rumination; Miloyan, Bulley, & Suddendorf, 2016; Miloyan, Pachana, & Suddendorf, 2014), and such preoccupation accounted for anxiety–procrastination linkage (Constantin et al., 2018). The results suggested that negative mental simulation, irrespective of directing toward the negative task engagement or task outcome, would not promote a timely action. Thus unsurprisingly, individuals suffering from higher trait anxiety, who were lack sufficient self-control (Basten, Stelzel, & Fiebach, 2011; Edwards et al., 2017; Eysenck et al., 2007), might be incapable of inhibiting the negative future-oriented thoughts, thereby showing heightened proneness to procrastinate.

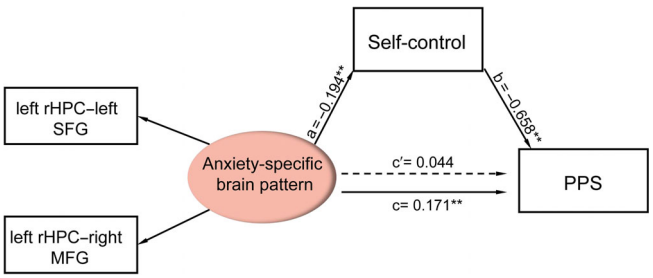

FIGURE 3 The structural model with standardized coefficients. The SEM analyses showed that the anxious brain connectivity pattern, which was implicated in the left rHPC–left SFG and left rHPC–right MFG, impacted procrastination through self-control. $^{**}p < .01$; $^{*}p < .05$; PPS, procrastination

4.2 | The anxiety-specific brain pattern—HPC–PFC pathways—impacted procrastination through self-control

The dynamic FC results showed that individuals high in trait anxiety were accompanied by greater variability in HPC–PFC pathways, namely, left rHPC–left SFG and left rHPC–right MFG. The rostral portion of the HPC (also known as the anterior, head, ventral regions) was distinguished from the caudal segment (also known as the posterior, tail, dorsal regions) by sharp changes in the functional characteristics, the pattern of which was termed as "long-axis specialization" (Poppenk, Evensmoen, Moscovitch, & Nadel, 2013; Zeidman & Maguire, 2016). Relative to the dorsal/posterior HPC, the CA1 cells in the ventral/anterior zone were shown to drive the avoidance behaviors in anxiogenic environments (Jimenez et al., 2018). The genes expressed in the same region were specifically associated with the brain regions supporting emotion and stress like the amygdala (Fanselow & Dong, 2010). Moreover, direct reciprocal connections between anterior HPC and the amygdala, insula, as well as medial PFC conspired to offer it a privileged interface with affect, especially anxiety and stress (Zeidman & Maguire, 2016). For example, human studies showed activation in the anterior HPC occurred in the paradigms like contextual fear conditioning (i.e., a spatial location associated with aversive stimulus; Pohlack, Nees, Ruttorf, Schad, & Flor, 2012), and

TABLE 2 The relationship between HPC–PFC dynamic FC and procrastination was completely mediated by self-control

Model pathways	Estimated effects	Biased-corrected CI		*p*
		Lower	Upper	
Total effect				
X → Y	0.171	0.039	0.295	.01
Direct effect				
X → M	−0.194	−0.333	−0.068	.004
M → Y	−0.658	−0.724	−0.591	.002
X → Y	0.044	−0.057	0.145	.368
Indirect effect				
X → M → Y	0.128	0.045	0.224	.004

Abbreviations: M, self-control; X, anxiety-specific brain pattern; Y, procrastination.

approach-avoidance conflict anxiety task which was central to the genesis of anxiety (Abivardi, Khemka, & Bach, 2020; Loh et al., 2017). Taken together, these findings indicated why it was the left rostral hippocamps, rather than its caudal zone, that anatomically functioned with the prefrontal regions to anxiety-processing. More specifically, anxiety was characterized by the structural and functional dysfunctions in a well-defined set of brain circuits that were implicated in the corticolimbic system, especially HPC and PFC (Daviu et al., 2019; Mobbs & Kim, 2015; Yamasue et al., 2008; R. Zhang, Chen, et al., 2019). For instance, neuroimaging studies linked trait anxiety, or even pathological anxiety in populations, such as adolescents and adults, to regional gray matter volumes abnormalities (Mueller et al., 2013; Spampinato et al., 2009; R. Zhang, Chen, et al., 2019), as well as to the dysfunctional intrinsic brain activity, such as ReHo, in these brain structures (Hahn, Dresler, Pyka, Notebaert, & Fallgatter, 2013; Tian et al., 2016). Moreover, individuals with higher trait-anxiety-predisposition showed hyper-activation in the prefrontal cortex, especially its lateral and ventromedial parts, and HPC during fear extinction (Belleau, Pedersen, Miskovich, Helmstetter, & Larson, 2018), processing threats (Fung, Qi, Hassabis, Daw, & Mobbs, 2019), or even anticipation of uncertain threats (Bijsterbosch, Smith, & Bishop, 2015; Geng et al., 2018). The extent of trait anxiety was associated with strength of functional coupling between the core components of corticolimbic system, including the ventral HPC–medial PFC and medial PFC–amygdala circuits, when participants were asked to process the threats (Fung et al., 2019), and anticipate the threats (i.e., shock; Bijsterbosch et al., 2015), suggesting the insufficient regulation over the subcortical regions. Further, from the dynamic perspective, greater variability FC between default mode network and ventral attention network, the frontoparietal network predicted higher levels of negative affect (Li et al., 2020). Hence, these findings suggested that highly variable HPC–PFC FC in anxious individuals might signify an increased tendency to simulate future in a negative manner with less stable regulatory resources exerted, which could impact the way of pursuing long-term goals.

In support of this hypothesis, the SEM results showed that the anxiety-specific brain pattern impacted procrastination through self-control. Following the attentional control theory, anxiety exerted adverse impacts on the top-down system, thereby impeding the efficiency of executive functioning (i.e., inhibition; Eysenck et al., 2007). Importantly, self-control, the capacity to resist undesired responses and support the pursuit of long-term goals (Baumeister et al., 2007), underlay a core process of goal-directed behaviors (i.e., decision making; Peters & Büchel, 2011; S. Zhang, Liu, & Feng, 2019). Such faculty relied on the prefrontal regions identified in current study, including anterior and dorsolateral parts (Gläscher et al., 2012; Knoch & Fehr, 2007). Utilizing the low frequency repetitive transcranial magnetic stimulation (rTMS), individuals with disruption of the function in left lateral PFC attenuated their self-control ability, preferring the short-sighted choices rather than the delayed benefits (Figner et al., 2010). Moreover, studies showed that recalling the past experiences and imaging the scenes activated the anterior HPC, which was the part of a well-established "core network" of brain regions (Zeidman & Maguire, 2016). The increased HPC–PFC coupling helped people make choices with a long-term pay-off (Benoit, Gilbert, & Burgess, 2011). Coupled with this evidence, current findings indicated that invariable HPC–PFC FC for anxious individuals reflected impoverished regulatory ability over the negative mental simulation, thereby weakening self-control for pursuing goals. Besides, in terms of procrastination, the triple networks underlying procrastination held that the prefrontal regions, especially lateral PFC, engaged in the top-down regulation, while HPC was a hub for supporting episodic prospection (Z. Chen, Liu, Zhang, & Feng, 2019). For instance, procrastination was closely associated with the spontaneous activity (i.e., ALFF) in parahippocampus, and anterior PFC (W. Zhang, Wang, & Feng, 2016). The enhanced functional connectivity within the frontal regions (i.e., vmPFC-SFG and vmPFC-dlPFC) linked to less procrastination (Wu, Li, Yuan, & Tian, 2016; W. Zhang et al., 2016), which indicated an efficient top-down control would attenuate unnecessary delay. Taken together, the findings collectively suggested the anxiety-specific brain pattern, which was implicated in the HPC–PFC circuits, could damage self-control ability, consequently resulting in the failure of acting in time.

4.3 | Future directions

The current study has some limitations. First, the selection of window length has been in debate. Too short window lengths can cause the spurious fluctuations of functional connectivity, while too long lengths are likely to weaken the precision of detecting the temporal variations in seconds (Preti et al., 2017). Thus, the selection of window length may to some extent determine the accuracy of depicting dynamic functional connectivity. Researchers recently have proposed an alternative method to acquire the node-wise temporal variability via calculating an averaged-value map across different window lengths for each participant (Yang et al., 2021). Such method can be adopted in our future work to explore the neural correlates of cognition from the dynamic perspectives. Second, as temporal decision model of procrastination proposes, procrastination often occurs when task utility (i.e. task aversiveness) outperforms outcome utility (i.e. task values; S. Zhang, Chen, et al., 2019), which depends on the dynamic trade-off between different task properties in real life. Procrastination, which is measured by the widely adopted questionnaire in our work, can only reflect chronic tendency of delaying. Further investigations should be performed in the frame of this model, for verifying the role of trait anxiety and self-control in individuals' decisions to delay. Finally, present findings reveal the importance of self-control in accounting for the relationship between anxiety-specific brain pattern (i.e., HPC–PFC pathway) and procrastination. Importantly, brain stimulation on the lateral PFC has been demonstrated to enhance cognitive performance for people with high trait anxiety (Ironside et al., 2019). Hence, it is worthwhile to testify whether procrastination can be significantly decreased through stimulating the dlPFC via high precision transcranial direct current stimulation (tDCS) for anxious individuals.

5 | CONCLUSIONS

As a whole, our work still provides evidence that self-control played a mediated role in the relationship between trait anxiety and procrastination. Furthermore, the anxiety-specific brain connectivity pattern, namely the left rHPC–left SFG and left rHPC–right MFG, could impact procrastination via self-control. Collectively, our findings extend our understanding of how individual differences in trait anxiety and its spontaneous brain activation affected procrastination, and offer new evidence for the interventions for mitigating procrastination among anxious individuals.

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CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

AUTHOR CONTRIBUTIONS

Rong Zhang: Design, data collection, data analyses, writing, and revised the manuscript; **Zhiyi Chen:** Data collection, data analyses, revised the manuscript; **Bowen Hu:** Data analyses, revised the manuscript; **Feng Zhou:** Revised the manuscript; **Tingyong Feng:** Design, data collection, revised the manuscript.

ETHICS STATEMENT

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

CONSENT TO PARTICIPATION

All the participants have provided the written informed consents prior to the experiment.

DATA AVAILABILITY STATEMENT

To date, the data is still not accessed online because the project still lasts.

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SUPPORTING INFORMATION

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