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Article

Functional near-infrared spectroscopy study of hemodynamic in the prefrontal and motor cortices and its implications for endurance capacity

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Abstract: This study investigates the primary cortical areas that influence endurance capacity in humans by monitoring cerebral hemodynamics in the prefrontal and motor cortices during endurance exercise. Participants engaged in incremental load endurance exercise while equipped with a functional near-infrared spectroscopy to assess cortical activity. Hemodynamic in the prefrontal and motor cortices were continuously monitored, with a particular emphasis on cortical activation at both the onset and termination of exercise, as well as their relationships with exercise duration and other cortical regions. Results showed that both the prefrontal and motor cortices exhibited significant activation during the onset and termination of exercise, with activation intensity and areas increasing in response to elevated exercise loads. Notably, cortical activation in these cortices at the onset of exercise did not show a significant correlation with exercise duration. However, activations in specific aeras of the motor cortex-FC1h, FC2h, C1h, and C2h-at the termination of exercise were significantly correlated with endurance duration and showed extensive interconnections with other areas in both the prefrontal and motor cortices. These results suggest that FC1h, FC2h, C1h, and C2h in the motor cortex might play a crucial role in regulating endurance capacity. Enhancing the functionality of these cortical areas might contribute to further improvements in endurance performance.

Keywords: prefrontal cortex; motor cortex; functional near-infrared spectroscopy; cerebral hemodynamic; endurance capacity

1. Introduction

The central nervous system (CNS) plays a crucial role in sports science, significantly enhancing our understanding of athletic performance [1]. Consequently, exploring the relationship between the CNS and physical performance is essential for improving athletic capabilities. Recent studies indicate that endurance capacity is not solely determined by the musculoskeletal system; rather, it is intricately linked to cortical function [2]. Thus, investigating the role of cortical areas in endurance exercise is vital for optimizing athletic performance.

Among the various cortical regions, the prefrontal cortex and motor cortex, along with the temporal cortex and cerebellum, are recognized as critical in modulating endurance capacity [3–6]. Specifically, the prefrontal and motor cortices have garnered particular attention for their significant contributions to enhancing endurance performance [3]. Research shows that boosting the excitability of these cortical areas can significantly enhance endurance capacity without changing physiological or psychological performance [7]. However, it is important to note that many existing studies on the cortical modulation of endurance capacity have inadequately assessed the activation states of these cortices [8]. This oversight might reduce the effectiveness

of intervention strategies and introduce variability in outcomes. As the cerebral cortex adapts to different exercise demands, it can lead to distinct activation patterns. [9,10,11]. To more effectively optimize endurance capacity, precise assessments of cortical activation should be conducted prior to implementing interventions.Functional near-infrared spectroscopy (fNIRS) has emerged as a powerful tool for examining cortical function, offering high ecological validity and facilitating the exploration of interactions between cortical activity and specific motor behaviors [9,12,13]. Its capability for non-invasive, real-time monitoring of neuronal activity, metabolic processes, and hemodynamic responses within the cortex makes fNIRS particularly suitable for investigating complex, wholebody activities such as cycling and rowing [11,14].

Based on these advantages, this study uses fNIRS to evaluate the activity of the prefrontal and motor cortices during endurance exercise. By employing fNIRS, we aim to analyze hemodynamic responses in relation to the duration of endurance performance, elucidating the impact of both cortices on endurance capacity and identifying the primary cortical areas that influence endurance performance.

2. Methods

2.1. Participants

Thirty healthy adults participated in the endurance exercise tests (mean age: 23.86 ± 3.97 years; height: 170.24 ± 6.83 cm; weight: 63.48 ± 15.47 kg; BMI: 21.72 ± 3.94 kg/m^2). Inclusion criteria for participants included: (1) engaging in physical activity 3 to 5 times per week, with each session lasting a minimum of 60 minutes; (2) no history of cardiovascular or respiratory diseases; (3) no exercise-related injuries or contraindications in the past six months, and (4) no potential risk factors that could compromise exercise safety. Furthermore, participants were required to have head circumferences between 51 and 53 cm, as well as hair characteristics compatible with the application of fNIRS probes on the scalp.

All participants were informed about the benefits and risks associated with taking part in this study and provided signed informed consent. The consent form included information from the Helsinki Declaration, the study's purpose, and detailed protocols. This study received approval from the Ethics Committee of the Ethics Committee of Shanghai University of Sport (number: 102772023RT031).

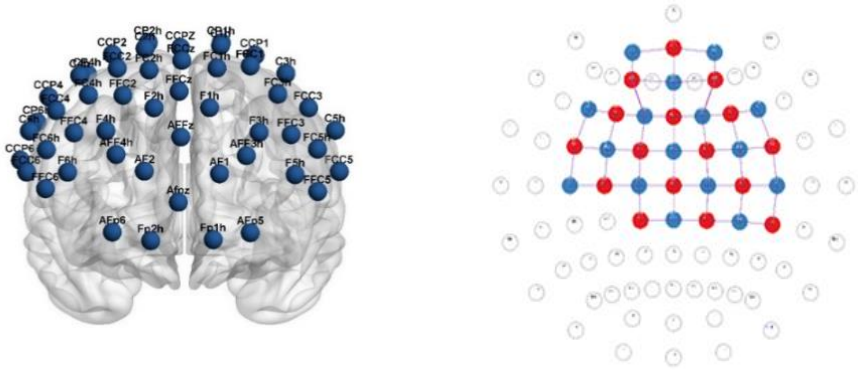
2.2. Experimental design

After acclimatizing to the experimental environment and procedures, participants undertook a graded incremental load exercise test on a stationary cycle ergometer. Throughout the exercise, fNIRS was employed to continuously monitor hemodynamic responses in the bilateral prefrontal and motor cortices. Additionally, heart rate monitors and subjective fatigue scales were utilized to quantify physiological and psychological loads at each stage of exercise. To minimize the impact of fatigue on the results, participants were instructed to avoid high-intensity exercise on the day preceding the test and to ensure a minimum of 7 h of sleep the night prior to testing.

2.3. Hemodynamic monitoring of the prefrontal and motor cortices

A multi-channel continuous-wave functional near-infrared spectroscopy (fNIRS) system (NIRSout, NIRx, USA) was employed to monitor hemodynamic changes in the prefrontal and motor cortices during endurance exercise, following the channel arrangement protocols established by Feng et al. [15] and Liang et al. [16] (**Figure 1**). The hemodynamic metrics analyzed included the relative concentrations of oxyhemoglobin (HbO), deoxyhemoglobin, and total hemoglobin. Previous studies have indicated that HbO is the most sensitive marker of neuronal metabolic activity, regional oxygenation changes, and neural activation [17,18]. Consequently, HbO was designated as the primary outcome variable for assessing cortical activity in this investigation.

Before data collection, the NIRSite 2.0 software (NIRx, USA) was utilized to spatially localize the fNIRS channels. Participants were then instructed to sit quietly with their eyes open for 3 minutes to facilitate relaxation. During the endurance exercise, hemodynamic responses were continuously monitored. The first 20 seconds following the initiation of exercise were analyzed to represent cortical activation at the onset, while the final 20s before exercise cessation reflected cortical activation at termination.



(a) **(b)**

Figure 1. Channel configurations of the functional near-infrared spectroscopy system. **(a)** The anatomical localization of the channel; **(b)** the optodes layout diagram.

Figure 1a indicates the channel anatomical position, **Figure 1b** indicates the channel configuration. In the **Figure 1b** plot, red represents the infrared light source transmitter, and blue represents the infrared light source receiver.

2.4. Endurance exercise protocol

A graded incremental load endurance exercise protocol was utilized to assess maximum endurance capacity [19]. Participants began with a 3-minute warm-up at 0 watts, followed by incremental cycling until they could no longer maintain the prescribed intensity. Participants increased the load by 20 watts per minute for female, 25 watts per minute for male, with a target cycling cadence maintained between 60 and 90 RPM [16]. Exercise was terminated when participants were unable to sustain

the load for more than 5 seconds, reached a heart rate of ≥180 bpm, or reported subjective fatigue scores of ≥17, at which point exercise duration was recorded [16].

2.5. Subjective fatigue and heart rate monitoring

The 20-point Borg Rating of Perceived Exertion (RPE) scale was utilized to assess participants' subjective fatigue at each load [20], Polar heart rate belt (H10, Polar, Finland) were monitored heart rates (HR); both parameters were collected during the last 10 seconds of each load to quantify physiological and psychological loading, as well as to serve as primary outcome measures for exercise cessation.

2.6. Data processing and statistical analysis

2.6.1. Data processing

HbO raw data were processed using Homer2_UI (v2.8, p2.1, MATLAB). Initially, the raw optical intensity data were converted to optical density. Subsequently, motion artifacts were identified, with fluctuations exceeding five times the mean or fifty times the standard deviation within one second classified as motion artifacts. These artifacts were corrected using a 1.5 interquartile range wavelet transform. A bandpass filter (high-pass at 0.01 Hz and low-pass at 0.1 Hz) was then applied to remove baseline drift and physiological noise. Finally, the modified Beer-Lambert law was used to convert optical density into HbO concentration. Following data processing, NIRS_SPM 8.0 (KASIT, Korea) was employed to calculate β.

2.6.2. Statistical analysis

A one-sample t-test was conducted to compare the β values of each channel in the prefrontal and motor cortices against zero, assessing activation levels at both the onset and termination of exercise. Pearson correlation analyses were performed to explore relationships between the β values of activated channels and exercise duration, identifying primary cortical regions influencing maximum endurance capacity within both cortices. Additional Pearson correlations were conducted to examine interconnections among different cortical areas. Paired sample t-tests were employed to analyze the activation intensity of the prefrontal and motor cortices at the onset and termination of exercise, thereby assessing the impact of increased exercise intensity on their activation. For multiple comparisons in the statistical analyses, the False Discovery Rate method was applied to adjust significance levels. All statistical analyses were performed using SPSS 21.0 (IBM, Chicago, IL, United States), with a significance threshold set at $p < 0.05$.

3. Results

3.1. Activation of the prefrontal and motor cortices during exercise

Participants successfully completed the endurance exercise test with an average duration of 799.85 ± 15.67 seconds, and no exercise-induced injuries were reported. Throughout the exercise, HR exhibited a significant increase from baseline (94.20 ± 1.95 bpm) to termination (173.65 ± 2.21 bpm). Similarly, RPE escalated from 7.20 ± 0.29 at the onset to 18.75 ± 0.24 at the conclusion, consistent with the predefined termination criteria.

At the onset of exercise, significant activation was observed in the prefrontal and motor cortices across various channels, including Afpz, AF1, AFF3h, F2h, FFCz, F5h, FC3h, FC1h, FC2h, and CP4h ($p < 0.05$). At the termination of exercise, extensive activation was observed across both cortical aeras ($p < 0.05$) (**Figure 2** and **Table 1**).

Further comparative analysis of activation intensity between the onset and termination phases of exercise revealed significant differences in the activation levels at the channels Fp1h, Fp2h, Afpz, AFp6, AF2, AFF4h, F6h, F4h, FFC4, FFC1, FC1h, FCC1, FC4h, FCCz, C2h, CCPz, FCC4, CCP4, and CCP2 ($p < 0.05$). The activation intensity at the termination of exercise was significantly higher compared to the onset (**Figure 2** and **Table 1**).

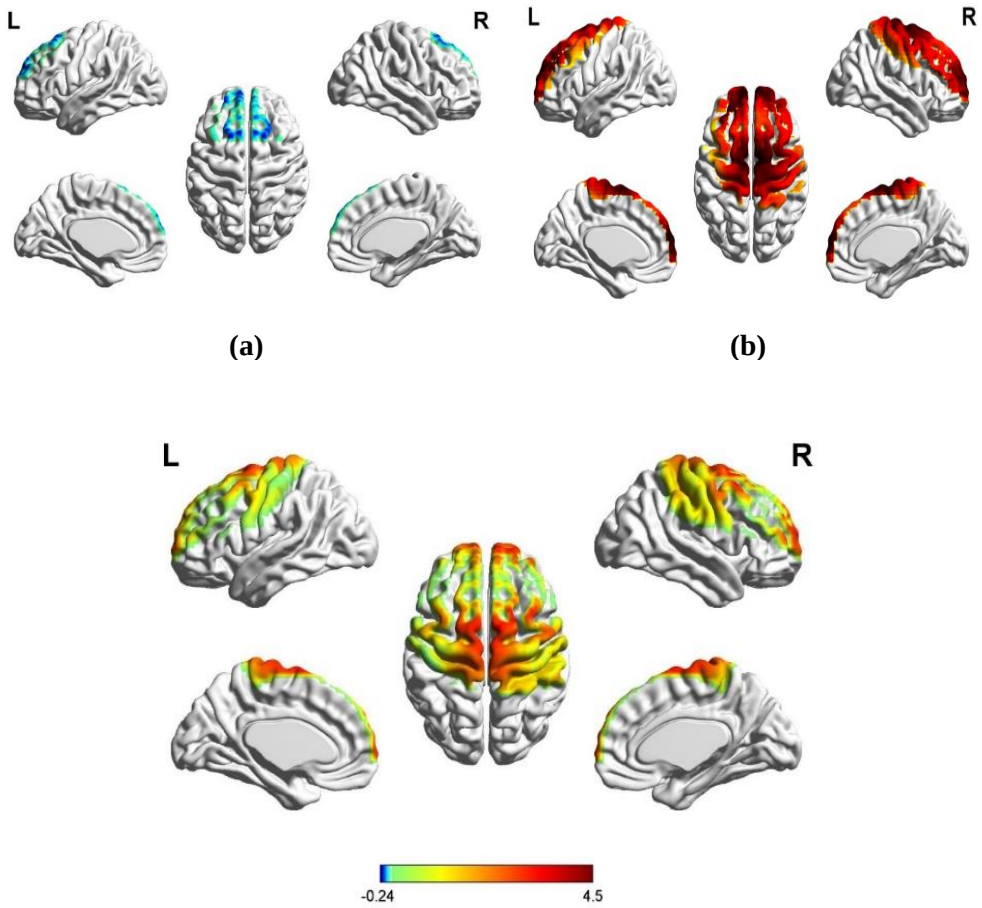


Figure 2. Cortical activations of the prefrontal and motor cortices during endurance exercise. **(a)** The activation in the prefrontal and motor cortices observed at the onset of exercise; **(b)** the activation in the prefrontal and motor cortices observed at the termination of exercise; **(c)** the comparison of cortical activation in the prefrontal and motor cortices between the onset and termination of exercise.

L represents the left hemisphere, and R represents the right hemisphere. The colors in panels **Figure 2a**,**b** depict the activation areas of the prefrontal cortex and motor cortex at the onset and the termination of exercise. The colors in panel **Figure 2c** illustrate the differences in activation of the prefrontal cortex and motor cortex

between the onset and termination of exercise, with a gradient from cool to warm colors indicating increasing activation intensity.

Table 1. Cortical activations of the prefrontal and motor cortices during endurance exercise.

Onset			**Termination**						**Onset vs termination**		
Channel	***t***	***p***	**Channel**	***t***	***p***	**Channel**	***t***	***p***	**Channel**	***t***	***p***
Afpz	−2.14	0.042	Fp1h	−6.25	<0.001	FC3h	−3.87	0.002	Fp1h	3.09	0.020
AF1	−2.32	0.028	Fp2h	−4.33	<0.001	FC1h	−5.42	<0.001	Fp2h	2.71	0.034
AFF3h	−2.65	0.014	AFpz	−7.22	<0.001	FCC1	−3.87	0.002	AFpz	3.28	0.020
F2h	−2.40	0.024	AFp6	−5.70	<0.001	FFC2	−2.94	0.010	AFp6	3.07	0.020
FFCz	−2.87	0.008	AF2	−5.05	<0.001	FC2h	−6.53	<0.001	AF2	3.33	0.020
F5h	−2.14	0.042	AFF4h	−5.00	<0.001	FC4h	−5.19	<0.001	AFF4h	2.93	0.024
FC3h	−2.49	0.019	AFp5	−3.45	0.003	FCC2	−6.27	<0.001	F6h	3.07	0.020
FC1h	−2.49	0.019	AF1	−2.76	0.014	FCCz	−7.35	<0.001	F4h	2.73	0.033
FC2h	−2.47	0.020	AFF3h	−2.27	0.042	C2h	−6.65	<0.001	FFC4	3.34	0.020
CP4h	−2.26	0.033	F6h	−5.03	<0.001	C1h	−3.97	0.002	FFC1	3.02	0.022
			F4h	−4.95	<0.001	CCPZ	−5.31	0.001	FC1h	3.11	0.020
			FFC4	−4.86	<0.001	FCC4	−5.09	<0.001	FCC1	3.09	0.020
			AFFz	−3.84	0.002	C6h	−3.28	0.004	FC4h	4.14	<0.001
			F2h	−3.26	0.004	CCP4	−3.86	0.002	FCCz	3.53	0.020
			F1h	−3.96	0.002	CCP1	−4.46	<0.001	C2h	3.72	0.020
			FFCz	−4.22	<0.001	CP1h	−3.32	0.004	CCPZ	2.86	0.026
			F3h	−6.78	<0.001	CCP2	−4.96	<0.001	FCC4	3.04	0.020
			F5h	−2.27	0.032	CP2h	−4.34	<0.001	CCP4	3.15	0.020
			FCC1	−3.75	0.002	CP4h	−4.12	<0.001	CCP2	2.55	0.046

3.2. Correlation between activation of the prefrontal and motor cortices with exercise duration and other brain regions

During the onset of exercise, no significant correlation was found between the activation of Afpz, AF1, AFF3h, F2h, FFCz, F5h, FC3h, FC1h, FC2h, and CP4h and exercise duration ($p > 0.05$). During the termination of exercise, a significant correlation was observed between the activation of FC1h, FC2h, C2h, and C1h and exercise duration ($p < 0.05$) (**Figure 3** and **Table 2**).

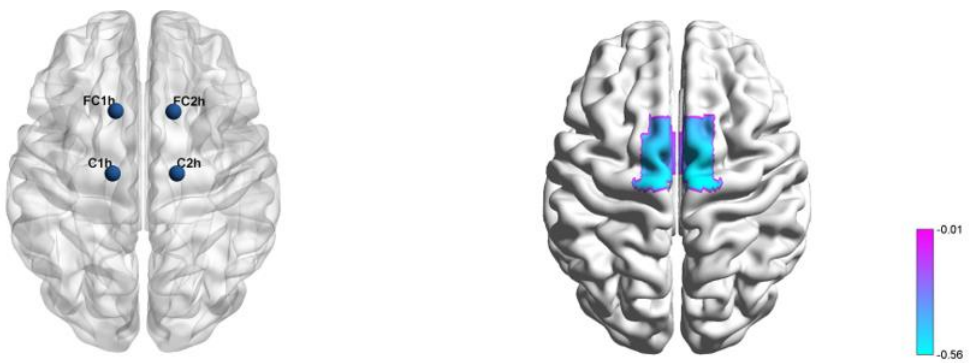


Figure 3. Localization of primary cortical areas in the prefrontal and motor cortices.

Table 2. Correlation between activation of the prefrontal and motor cortices at different stages and exercise duration.

Onset			**Termination**					
Channel	***R***	***p***	**Channel**	***R***	***p***	**Channel**	***R***	***p***
Afpz	0.04	0.847	Fp1h	−0.21	0.287	FC3h	−0.37	0.057
AF1	−0.03	0.894	Fp2h	−0.10	0.614	FC1h	−0.39	0.042
AFF3h	−0.05	0.819	AFpz	−0.31	0.119	FCC1	−0.35	0.070
F2h	0.16	0.440	AFp6	−0.11	0.590	FFC2	−0.19	0.337
FFCz	0.21	0.282	AF2	−0.35	0.073	FC2h	−0.40	0.040
F5h	0.06	0.783	AFF4h	−0.23	0.244	FC4h	−0.23	0.251
FC3h	−0.12	0.558	AFp5	−0.33	0.090	FCC2	−0.22	0.267
FC1h	−0.15	0.456	AF1	−0.35	0.077	FCCz	−0.32	0.105
FC2h	0.07	0.719	AFF3h	−0.28	0.156	C2h	−0.56	0.002
CP4h	0.22	0.269	F6h	−0.25	0.205	C1h	−0.50	0.008
			F4h	−0.33	0.096	CCPZ	−0.26	0.184
			FFC4	−0.15	0.445	FCC4	−0.03	0.874
			AFFz	−0.35	0.074	C6h	−0.07	0.747
			F2h	−0.25	0.204	CCP4	−0.38	0.051
			F1h	−0.32	0.099	CCP1	−0.38	0.051
			FFCz	−0.27	0.172	CP1h	−0.20	0.326
			F3h	−0.13	0.529	CCP2	−0.21	0.294
			F5h	−0.14	0.480	CP2h	−0.22	0.272
			FCC1	−0.01	0.985	CP4h	−0.20	0.328

The correlation between primary cortical areas and other regions within the prefrontal and motor cortices at the termination of exercise was showed in **Figure 4** and **Table 3**. Significant correlations were found as follows: FC1h showed significant correlations with Fp1h, F1h, FC5h, FFC1, FC3h, FCC1, FC2h, C1h, Fp2h, AFp6, F2h, FC4h, FCC2, C2h, CCP2, AFPZ, AFFz, FFCz, and CCPZ ($p < 0.05$). FC2h showed significant correlations with F1h, FC3h, FC1h, FCC1, C1h, F4h, FFC4, F2h, FC4h, FCC2, C2h, CCP4, CCP2, and FFCZ ($p < 0.05$). C1h was significantly correlated with AFp5, AF1, AFF3h, F1h, FFC1, FC3h, FC1h, FCC1, C1h, CP1h, CP1-C1-CCP1h, AF2, AFF4h, F4h, F2h, FC2h, FC4h, FCC2, C4h, CCP4, CCP2, CP2h, CP4h, AFPZ, AFFz, FFCz, and CCPZ ($p < 0.05$). C2h was significantly correlated with AFF3h, FFC1, FC1h, FCC1, CP1-C1-CCP1h, AF2, FC2h, FC4h, FFC6, C2h, CP2h, and CP4h ($p < 0.05$).

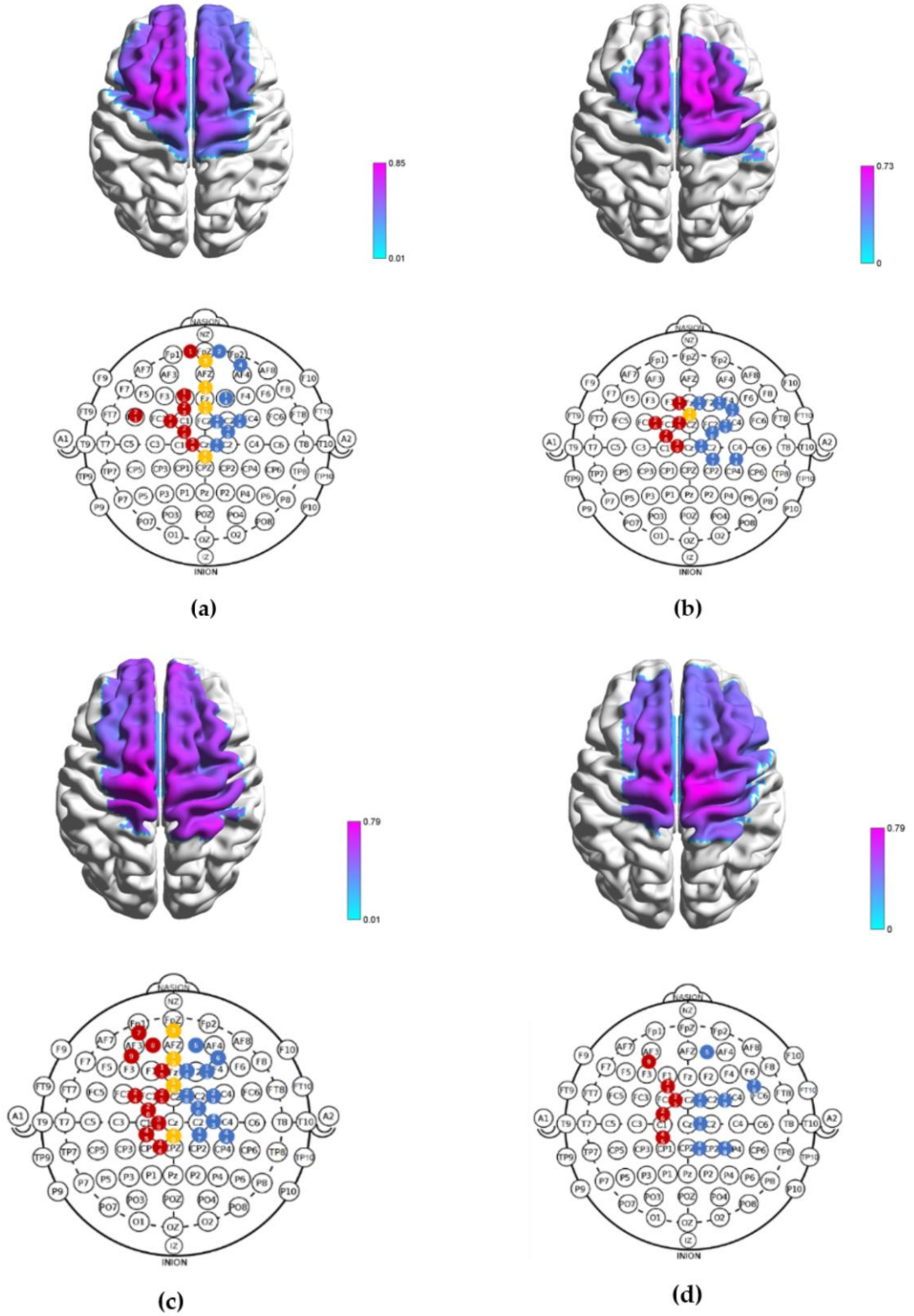


Figure 4. Correlation between the primary cortical areas and the rest of cortical areas. **(a)** The correlation between FC1h and the rest of the prefrontal and motor cortices; **(b)** the correlation between FC2h and the rest of the prefrontal and motor cortices; **(c)** the correlation between C1h and the rest of the prefrontal and motor cortices; **(d)** the correlation between C2h and the rest of the prefrontal and motor cortices.

Red indicates the distribution of correlated channels in the left hemisphere of the brain, blue indicates the distribution of correlated channels in the right hemisphere of the brain, and yellow indicates the distribution of correlated channels in the cerebral longitudinal fissure.

Table 3. Correlation between primary cortexand other regions in the prefrontal and motor cortices

Channel	**Left hemisphere**			**Right hemisphere**			**Cerebral longitudinal fissure**		
	Channel	**R**	***p***	**Channel**	**R**	***p***	**Channel**	**R**	***p***
FC1h	Fp1h	0.49	0.009	Fp2h	0.50	0.008	AFPZ	0.41	0.036
	F1h	0.60	0.001	AFp6	0.39	0.045	AFFz	0.49	0.010
	FC5h	0.42	0.028	F2h	0.51	0.007	FFCz	0.67	0.001
	FFC1	0.82	<0.001	FC4h	0.50	0.008	CCPZ	0.40	0.039
	FC3h	0.65	<0.001	FCC2	0.46	0.017			
	FCC1	0.85	<0.001	C2h	0.61	0.001			
	FC2h	0.60	0.001	CCP2	0.38	0.048			
	C1h	0.49	0.009						
FC2h	F1h	0.52	0.006	F4h	0.53	0.005	FFCZ	0.601	0.001
	FC3h	0.39	0.044	FFC4	0.46	0.016			
	FC1h	0.60	0.001	F2h	0.41	0.032			
	FCC1	0.59	0.001	FC4h	0.58	0.002			
	C1h	0.38	0.049	FCC2	0.73	0.001			
				C2h	0.56	0.002			
				CCP4	0.43	0.027			
				CCP2	0.433	0.024			
C1h	AFp5	0.52	0.005	AF2	0.67	0.001	AFpZ	0.49	0.010
	AF1	0.52	0.006	AFF4h	0.51	0.007	AFFz	0.59	0.001
	AFF3h	0.49	0.009	F4h	0.54	0.004	FFCz	0.51	0.007
	F1h	0.55	0.003	F2h	0.51	0.007	CCPZ	0.56	0.002
	FFC1	0.50	0.009	FC2h	0.56	0.002			
	FC3h	0.42	0.031	FC4h	0.58	0.001			
	FC1h	0.61	0.001	FCC2	0.54	0.003			
	FCC1	0.62	0.001	C4h	0.42	0.031			
	C1h	0.79	0.001	CCP4	0.56	0.002			
	CP1h	0.48	0.011	CCP2	0.65	<0.001			
	CP1-C1-CCP1h	0.64	0.001	CP2h	0.61	0.001			
				CP4h	0.67	0.001			
C2h	AFF3h	0.45	0.018	AF2	0.42	0.030			
	FFC1	0.46	0.015	FC2h	0.38	0.049			
	FC1h	0.49	0.009	FC4h	0.52	0.006			
	FCC1	0.63	0.001	FFC6	0.43	0.027			
	CP1-C1-CCP1h	0.46	0.017	C2h	0.79	0.001			
				CP2h	0.41	0.033			
				CP4h	0.43	0.027			

4. Discussion

This study employed fNIRS to monitor the activation of the prefrontal cortex (PFC) and motor cortex (MC) during incremental load endurance exercises, focusing on the relationship between cortical activation and endurance capacity. The results reveal that the activation area and intensity of PFC and MC increase with the exercise load, the cortex consisting of FC1h, FC2h, C1h, and C2h within the MC emerged as primary aeras influencing endurance capacity. Therefore, a deeper understanding of the intricate relationships between these cortical areas and endurance performance may provide valuable insights into the central mechanisms that enhance endurance capacity.

4.1. Cortical activation during endurance exercise

While investigating cortical activation in the PFC and MC during incremental load endurance exercise is not novel, it remains an important area of inquiry. Prior research has explored cortical activation from various perspectives, including exercise intensity and regional specificity. For instance, Seidel et al. [21] analyzed motor cortex activation during incremental load endurance exercises at varying intensities (20%, 40%, and 60% of peak power), identifying significant activation in the supplementary motor area (SMA), premotor cortex (PMC), and primary motor cortex (M1). Similarly, Jung et al. [22] utilized fNIRS to monitor activation in the PFC, MC, and occipital cortex during incremental load exercises, observing activation in the PFC and MC, but not in the occipital cortex. However, a notable limitation in prior research is the predominant focus on isolated cortical regions, neglecting the monitoring of cortical function throughout the entirety of endurance exercise.

In this study, we monitored the activation of the PFC and MC throughout the entire duration of endurance exercise. This approach corroborates previous findings [21,22] while revealing that both the area and intensity of cortical activation expand with increasing exercise load. Previous research has indicated that cortical activation during physical activity reflects its role in motor control [23]. The coactivation of the PFC and MC during endurance exercise underscores their essential roles in regulating performance, marking them as critical areas influencing endurance capacity.

Moreover, the observed increase in cortical activation intensity with exercise load emphasizes the significant impact of exercise intensity on cortical function. Consistent with our findings, studies by Suzuki et al. [24] and Tempest et al. [25] further confirmed the relationship between cortical activation intensity and exercise intensity during incremental load endurance activities such as running and cycling. These findings highlight a positive correlation between cortical activation and exercise intensity [26]. Generally, during low-intensity exercise, cortical activation primarily facilitates basic neural control and execution, as peripheral sensory input often lacks the intensity to elicit substantial cortical responses. This results in relatively smaller activation areas and lower intensity. As exercise intensity escalates, greater demands for coordination, control, and attentional focus necessitate increased cortical involvement. Consequently, the CNS expands cortical activation and enhances intensity to improve response speed, movement coordination, and attentional focus [25].

4.2. The key role of the motor cortex in endurance performance

While both the PFC and MC were activated during exercise, only the channels FC1h, FC2h, C1h, and C2h at exercise termination demonstrated a significant correlation with exercise duration, highlighting them as the primary cortical areas influencing maximum endurance capacity. These channels are predominantly situated in the MC; specifically, FC1h and FC2h encompass the PMC, SMA, and frontal eye fields, while C1h and C2h also cover the PMC, SMA, and M1. These findings also support the conclusions drawn by Jung et al. [22] that improvements in endurance performance rely on the MC.

Prior studies have emphasized the roles of both the PFC and MC in movement selection, preparation, planning, and execution [21]. The PMC, SMA, and M1 function as advanced motor centers responsible for complex motor planning, preparation, and execution [27,28]. Enhancing the functions of these regions could yield significant benefits for endurance exercise. Furthermore, sustaining long-term endurance exercise may involve mechanisms that increase cortical excitability and enhance intracortical facilitation systems [29]. The balance between excitatory and inhibitory systems within the cortex decides neural output, thereby affecting cortical excitability [30]. Therefore, increasing the excitability of the PMC, SMA, and M1 theoretically enhances CNS output to peripheral systems, which can mitigate corticospinal tract fatigue and ultimately improve endurance capacity.

Further analysis of the interconnections among primary cortical areas within the PFC and MC revealed extensive connectivity networks. This complexity may be attributed to the brain's multiscale organization and functional characteristics. The CNS operates as a network of interconnected brain regions, where nodes are both anatomically and functionally linked, allowing them to compensate for one another. Consequently, interventions targeting one node can influence adjacent or even distant nodes [31]. Typically, limb movement results from these cortical interactions, involving not only the MC but also higher-order brain networks [32]. Thus, we propose that the extensive connections between primary cortical areas and other regions of the PFC and MC may facilitate the integration of diverse neural resources and functions, playing a pivotal role in the control of endurance exercise.

4.3. Role of the prefrontal cortex in endurance performance

Monitoring cortical activity during movement tasks has been considered as a pivot way to understand the links between central and peripheral systems, crucial for enhancing athletic capacity [33]. Consistent with Billaut et al. [34], this study also did not observe significant PFC activation directly linked to exercise duration. This suggests that while the PFC may have an association with endurance capacity at the termination phase, it may not be the primary cortical area responsible for regulating endurance capacity [22]. This could be attributed to the unique functional mechanisms and oxygenation patterns of the PFC. PFC, as a crucial component of the motor control system, integrates cognitive processes with peripheral info to plan and organize movements, enabling motor actions or inhibiting control to reduce discomfort [35,36]. Particularly, when MC function declines, the PFC compensates by integrating peripheral info into motor commands, maintaining exercise. In addition, the nonlinear

relationship between the PFC's oxygenation and exercise intensity have impact on performance. Baed on previous study, PFC oxygenation is able to boost endurance capacity at low to moderate intensity but drops past the ventilatory threshold, causing dysfunction and shifting resources to motor regions. [25]. In this study, the exercise intensity surpassed the ventilatory compensation point, resulting in insufficient PFC oxygenation to support endurance capacity [37].

Recent studies have highlighted the promise of lower-limb surface electromyography and the theory of physiologic complexity in exploring the relationship between the PFC and endurance athletic capacity. Caliandro et al. [33] reported in their study that the PFC activity is mainly conditioned by the muscle activation pattern of the lower limb. Hong et al. [38] identified PFC complexity as a key parameter for evaluating adaptive responses to physical demands during endurance activities. Thus, monitoring the lower-limb surface electromyography and calculating PFC complexity during endurance exercise would increase the understanding of the role and interplay of PFC activation and endurance capacity.

Although the findings in this study diverge from Hong et al. [38], who found that PFC activation can enhance endurance capacity, this discrepancy offers a valuable perspective for further investigation into the PFC's role in endurance. Hong et al. [38] found that 80% peak power output (PPO) intensity positively influenced endurance capacity through PFC activation, whereas 100% PPO at present study did not observe such an effect. This variance in exercise intensity suggests that the PFC impact on endurance capacity may vary with activation levels at 80%–100% PPO. Therefore, elucidating the activation of the PFC at the 80%–100% PPO and its correlation with endurance capacity may aid in further enhancing endurance athletic performance.

5. Conclusion

This study employed fNIRS to investigate the activation patterns of the PFC and MC during maximal endurance exercise, aiming to identify the primary cortical areas influencing endurance capacity and their relationship with performance. The findings highlight that both the PFC and MC are crucial for regulating endurance exercise, with endurance capacity being predominantly influenced by specific regions within the MC, namely FC1h, FC2h, C1h, and C2h. These areas appear to impact on endurance performance through the integration of resources across various cortical regions. Therefore, targeted interventions aimed at enhancing the functionality of FC1h, FC2h, C1h, and C2h through neuromodulation techniques may significantly improve endurance capacity.

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References

1. Hou L, Song Z, Yu Y, et al. Influence of exercise-induced fatigue on brain activity during voluntary movement. J Beijing Sport Univ. 2012; 35(3): 71-9. doi: 10.19582/j.cnki.11-3785/g8.2012.03.015
2. Gandevia, SC. Spinal and supraspinal factors in human muscle fatigue. Physiol Rev. 2001; 81(4): 1725–89. Doi: 10.1152/physrev.2001.81.4.1725
3. Angius L, Pascual-Leone A, Santarnecchi E. Brain stimulation and physical performance. Prog Brain Res. 2018; 240: 317–39. doi: 10.1016/bs.pbr.2018.07.010
4. Angius L, Pageaux B, Hopker J, et al. Transcranial direct current stimulation improves isometric time to exhaustion of the knee extensors. Neuroscience. 2016; 339: 363–375. doi: 10.1016/j.neuroscience.2016.10.028
5. Liang Z, Zhou J, Jiao F, et al. Effect of Transcranial direct current stimulation on endurance performance in elite female rowers: a pilot, single-blinded study. Brain Sci. 2022; 12(5): 541. doi: 10.3390/brainsci12050541
6. Okano AH, Fontes EB, Montenegro RA, et al. Brain stimulation modulates the autonomic nervous system, rating of perceived exertion and performance during maximal exercise. Br J Sports Med. 2015; 49(18):1213-8. doi: 10.1136/bjsports-2012-091658.
7. Machado DGDS, Unal G, Andrade SM, et al. Effect of transcranial direct current stimulation on exercise performance: A systematic review and meta-analysis. Brain Stimul. 2019;12(3):593-605. doi: 10.1016/j.brs.2018.12.227
8. Machado S, Jansen P, Almeida V, et al. Is tDCS an adjunct ergogenic resource for improving muscular strength and endurance performance? A systematic review. Front Psychol. 2019; 10:1127. doi: 10.3389/fpsyg.2019.01127
9. Seidel-Marzi O, Ragert P. Neurodiagnostics in sports: investigating the athlete's brain to augment performance and sport-specific skills. Front Hum Neurosci. 2020; 14:133. doi: 10.3389/fnhum.2020.00133
10. Perrey S, Besson P. Studying brain activity in sports performance: Contributions and issues. Prog Brain Res. 2018; 240:247-67. doi: 10.1016/bs.pbr.2018.07.004
11. Tempest GD, Reiss AL. The utility of functional near-infrared spectroscopy for measuring cortical activity during cycling exercise. Med Sci Sports Exerc. 2019; 51(5):979-87. doi: 10.1249/MSS.0000000000001875
12. Arenth PM, Ricker JH, Schultheis MT. Applications of functional near-infrared spectroscopy (fNIRS) to neurorehabilitation of cognitive disabilities. Clin Neuropsychol. 2007; 21(1):38-57. doi: 10.1080/13854040600878785
13. Balardin JB, Zimeo Morais GA, Furucho RA, et al. Imaging brain function with functional near-infrared spectroscopy in unconstrained environments. Front Hum Neurosci. 2017;11: 258. doi: 10.3389/fnhum.2017.00258
14. Possamai LT, Borszcz FK, de Aguiar RA, et al. Comparison of NIRS exercise intensity thresholds with maximal lactate steady state, critical power and rowing performance. Biol Sport. 2024; 41(2):123-130. doi: 10.5114/biolsport.2024.129486
15. Li F, Bi J, Liang Z, et al. Functional near-infrared spectroscopy-based evidence of the cerebral oxygenation and network characteristics of upper limb fatigue. Bioengineering. 2023; 10(10):1112. doi: 10.3390/bioengineering10101112
16. Liang Z, Wang Y, Liu W, et al. Effects of multifocal transcranial direct current stimulation based on motor cortex activation on cycling endurance performance. China Sport Sci 2024; 3(44):41-50. doi: 10.16469/j.css.202403005
17. Obrig H, Villringer A. Beyond the visible--imaging the human brain with light. J Cerebr Blood F Met. 2003; 23(1): 1–18. doi: 10.1097/01.WCB.0000043472.45775.29
18. Hoshi Y, Kobayashi N, Tamura M. Interpretation of near-infrared spectroscopy signals: a study with a newly developed perfused rat brain model. J Appl Physiol. 2001; 90(5): 1657–62. doi: 10.1152/jappl.2001.90.5.1657
19. Bentley DJ; Newell J, Bishop D. Incremental exercise test design and analysis: implications for performance diagnostics in endurance athletes. Sports Medicine. 2007; 37(7): 575–86. doi: 10.2165/00007256-200737070-00002
20. Borg GA. Psychophysical bases of perceived exertion. Med Sci Sport and Exer. 1982; 14(5): 377–381.
21. Seidel O, Carius D, Roediger J, et al. Changes in neurovascular coupling during cycling exercise measured by multi-distance fNIRS: a comparison between endurance athletes and physically active controls. Exp Brain Res. 2019; 237(11):2957-72. doi: 10.1007/s00221-019-05646-4
22. Jung R, Moser M, Baucsek S, et al. Activation patterns of different brain areas during incremental exercise measured by near-infrared spectroscopy. Exp Brain Res. 2015; 233(4):1175-80. doi: 10.1007/s00221-015-4201-4
23. Hou L, Zhou L, Song Z, et al. Whole-brain functional magnetic resonance imaging analysis of motor control in human brain. China Sport Sci. 2010; 30(8): 62–68. doi: 10.16469/j.css.2010.08.003

24. Suzuki M, Miyai I, Ono T, Oda I, et al. Prefrontal and premotor cortices are involved in adapting walking and running speed on the treadmill: an optical imaging study. Neuroimage. 2004; 23(3):1020-6. doi: 10.1016/j.neuroimage.2004.07.002
25. Tempest GD, Eston RG, Parfitt G. Prefrontal cortex haemodynamics and affective responses during exercise: a multi-channel near infrared spectroscopy study. PloS One. 2014; 9(5): e95924. doi: 10.1371/journal.pone.0095924
26. Rooks CR, Thom NJ, McCully KK, et al. Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy: a systematic review. Prog Neurobiol. 2010; 92(2):134-50. doi: 10.1016/j.pneurobio.2010.06.002
27. Christensen LO, Johannsen P, Sinkjaer T, et al. Cerebral activation during bicycle movements in man. Exp Brain Res. 2000; 135(1):66-72. doi: 10.1007/s002210000493
28. Orgogozo JM, Larsen B. Activation of the supplementary motor area during voluntary movement in man suggests it works as a supramotor area. Science. 1979; 206(4420): 847–50. doi: 10.1126/science.493986
29. Reidler JS, Zaghi S, Fregni F. Neurophysiological effects of transcranial direct current stimulation. San Diego: Academic Press. 2011: 319–49. doi:10.1016/B978-0-12-382235-2.00012-3
30. Tanaka M, Watanabe Y. Supraspinal regulation of physical fatigue. Neurosci Biobehav R. 2012; 36(1): 727–34. doi: 10.1016/j.neubiorev.2011.10.004
31. Bradley C, Nydam AS, Dux PE, et al. State-dependent effects of neural stimulation on brain function and cognition. Nat Rev Neurosci. 2022; 23(8):459-75. doi: 10.1038/s41583-022-00598-1
32. Thiebaut de Schotten M, Forkel SJ. The emergent properties of the connected brain. Science. 2022; 378(6619):505-10. doi: 10.1126/science.abq2591
33. Caliandro P, Molteni F, Simbolotti C, et al. Exoskeleton-assisted gait in chronic stroke: An EMG and functional near-infrared spectroscopy study of muscle activation patterns and prefrontal cortex activity. Clin Neurophysiol. 2020 ;131(8):1775-81. doi: 10.1016/j.clinph.2020.04.158
34. Billaut F, Davis JM, Smith KJ, et al. Cerebral oxygenation decreases but does not impair performance during self-paced, strenuous exercise. Acta Physiol. 2010; 198(4):477-86. doi: 10.1111/j.1748-1716.2009.02058.x
35. Robertson CV, Marino FE. A role for the prefrontal cortex in exercise tolerance and termination. J Appl Physiol. 2016; 120(4):464-6. doi: 10.1152/japplphysiol.00363.2015
36. Menotti F, Berchicci M, Di Russo F, et al. The role of the prefrontal cortex in the development of muscle fatigue in Charcot-Marie-Tooth 1A patients. Neuromuscul Disord. 2014; 24(6):516-23. doi: 10.1016/j.nmd.2014.03.010
37. De Wachter J, Proost M, Habay J, et al. Prefrontal cortex oxygenation during endurance performance: A systematic review of functional near-infrared spectroscopy studies. Front Physiol. 2021; 12:761232. doi: 10.3389/fphys.2021.761232
38. Hong Y, Bao D, Manor B, et al. Effects of endurance exercise on physiologic complexity of the hemodynamics in prefrontal cortex. Neurophotonics. 2024; 11(1):015009. doi: 10.1117/1.NPh.11.1.015009