Acute exercise increases oxygenated and deoxygenated hemoglobin in the prefrontal cortex

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Both acute and chronic exercise is consistently associated with a number of benefits to physical and mental health, including cardiovascular function, body weight, mood, and cognition. Near-infrared spectroscopy is an ideal method to measure changes in oxygenated and deoxygenated hemoglobin $(O_2Hb$ and dHb) levels in the prefrontal cortex (PFC) during exercise, to better understand the locus of such changes in affective and cognitive processes. The present study tracked time-dependent changes in O_2 Hb and dHb levels in the PFC as a function of parametrically manipulated target exercise intensity. Near-infrared spectroscopy was conducted as regular exercisers completed a 30-min bout of exercise with one of three target intensities: 52% (low condition), 68% (moderate condition), or 84% (high condition) of age-adjusted maximum heart rate. Heart rate data confirmed that the participants reached their goal intensities immediately, after 10 min, or after 20 min, respectively. Data showed that O_2Hb and dHb levels in the PFC increased as a function of both

Introduction

Aerobic exercise has consistently been shown to improve a number of physical and psychological health factors. Physically, regular exercise improves cardiovascular function, reduces the risk-factors for and complications of type 2 diabetes, and protects against obesity [\[1](#page-5-0)]. Psychologically, single bouts of exercise enhance positive affect [\[2\]](#page-5-0) and cognitive performance [\[3](#page-5-0)].

Despite the wealth of knowledge on the beneficial effects of exercise to physical and mental health, the neural substrates underlying such effects remain to be discovered. Changes in oxygenated and deoxygenated hemoglobin levels $(O₂Hb$ and dHb, respectively) in the prefrontal cortex (PFC), which accompany exercise, may be one such neural substrate. The PFC is associated with cognitive domains including those for emotional processing, memory, and performance monitoring [\[4](#page-5-0)]. Research has used functional near-infrared spectroscopy (fNIRS), a method that measures the changes in $O₂Hb$ and dHb levels in the superficial layers of the cortex, to investigate real-time changes in cortical oxygenation during exercise. Compared with other neuroimaging techniques, fNIRS is noninvasive and less sensitive to movement, making it a suitable candidate for measuring differences in regional cortical activity during exercise [\[5\]](#page-5-0).

exercise load and duration. An 84% > 68% > 52% difference was evident after 18 min of cycling for O_2 Hb and after 23 min of cycling for dHb. The present results add to the growing body of literature showing that at submaximal levels, increasing exercise intensities reliably promote prefrontal cerebral oxygenation. NeuroReport 25:1320–1325 © 2014 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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The direction and magnitude of changes in cerebral oxygenation in the frontal cortex as a function of exercise partially depend on the duration and intensity of exercise [\[6\]](#page-5-0). At relatively low to moderate intensities (up to ∼ 60% VO_{2max}), exercise increases the O₂Hb level in the PFC [\[7](#page-5-0)–[9\]](#page-5-0). At high intensities, when exercise reaches or exceeds VO_{2max} , the O₂Hb level often declines, especially as the duration or the number of repetitions at such intensities progresses [\[10](#page-5-0)–[12](#page-5-0)]. The effects of exercise on dHb levels are less consistent, as exercise between 30 and 60% VO_{2max} may increase [\[7\]](#page-5-0) or have little influence on dHb levels [\[8](#page-5-0)]. Cerebral oxygenation, the ratio of O_2Hb to total Hb (tHb = O_2Hb + dHb), provides a measure of relative changes in $O₂Hb$ and dHb levels. Cerebral oxygenation during exercise follows a pattern similar to that of O_2Hb , declining at high intensities such as 80% VO_{2max} [\[10](#page-5-0),[13\]](#page-5-0).

The aforementioned studies use exercise tasks in which participants complete short bouts of exercise (30–90 s) [\[9,14](#page-5-0),[15\]](#page-5-0) or incrementally increase exercise intensity for longer durations (10–90 min) [\[7,10](#page-5-0)–[12](#page-5-0)]. In both designs, changes are typically compared within the same exercise bout rather than across sessions, making it difficult to disentangle intensity effects from any cumulative effect of exercise duration. To date, only one study

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has investigated the influence of parametrically varying exercise intensities (across at least three levels) on PFC oxygenation in a within-participant design involving extended exercise durations [\[13\]](#page-5-0). In that study, the authors found evidence for reduced PFC oxygenation when the participants exercised at 80% VO_{2max} relative to 40% or 60% $\rm VO_{2max}$; however, they only measured right hemisphere oxygenation and had a restricted sample size. Thus, although evidence is accumulating on the influence of exercise on PFC oxygenation, it is both limited in scope and often equivocal. Investigating changes that occur over time as a function of the goal intensity of the exercise bout would contribute to the literature in at least two ways. First, a carefully controlled experiment with a more substantial sample size may reveal patterns of cerebral oxygenation in the PFC that have yet to be elucidated. Second, such a study would be characteristic of typical exercise plans, in that exercise regimes often comprise workouts that vary in intensity, and both highintensity interval training [\[16\]](#page-5-0) and moderate sustained exercise show benefits to overall health [\[17](#page-5-0)].

Hence, the purpose of the present study was to examine how graded-intensity exercise with target loads of low, moderate, and high work rates influences PFC oxygenation. fNIRS was performed over the PFC regions as regular exercisers completed three 30-min bouts of exercise with a goal of reaching 52% (low condition), 68% (moderate condition), or 84% (high condition) ageadjusted maximum heart rates (HR_{max}) . On the basis of recent meta-analytical results suggesting a quadratic relationship between exercise intensity and cerebral oxygen levels [\[6\]](#page-5-0), we predicted a dose-dependent increase in $O₂Hb$ and dHb levels in the PFC between low-intensity, moderate-intensity, and high-intensity exercise.

Methods

Twenty-four students (14 male, 10 female, mean \pm SD age 20.21 ± 2.38 years, mean \pm SD BMI 22.51 \pm 2.72) participated for monetary compensation. All participants were right-handed and in good health. All participants exercised regularly, defined by the American College of Sports Medicine recommendation of at least 30 min of moderate-intensity cardiorespiratory exercise up to 5 days/week, or at least 20 min of vigorous-intensity cardiorespiratory exercise at least 3 days/week [\[24\]](#page-5-0). Written informed consent was obtained, and all procedures were approved by the Tufts University Institutional Review Board and the Army Human Research Protections Office.

Functional near-infrared spectroscopy

fNIRS was performed using the fNIR Imager 1100 (fNIR Devices LLC, Potomac, Maryland, USA). This continuous-wave fNIRS system consists of four light sources and 10 detectors, with a 2.5 source–detector separation, comprising 16 channels across the dorsal and anterior PFC [\[18\]](#page-5-0). Each LED light source emitted light at two wavelengths (730 and 850 nm). Data were recorded at a temporal resolution of 2 Hz. The sensor pad was positioned on the forehead by centering the bottom of the probe at the Fpz, according to the international 10–20 system [\[19](#page-5-0)], such that the probe was aligned with F7, F_{P1} , F_{P2} , and F8. The sensor pad was secured at the back of the head and covered with opaque self-adhering tape to block ambient light. Fluorescent overhead lights were turned off during data acquisition to reduce potential interference. Cognitive Optical Brain Imaging Studio was used to adjust the LED current and gains to ensure that raw signal levels were greater than 700 mV and less than 1400 mV. Manual markers were used to denote the start and end of the 30-min cycling task.

Data analysis was carried out on changes in $O₂Hb$ and dHb levels, which were calculated with Cognitive Optical Brain Imaging Studio using the modified Beer–Lambert law. All channels were visually inspected, and faulty channels were removed from subsequent analyses. To remove motion artifact, any value greater than or less than 2 SDs from the mean of 30 s surrounding that value was excluded from subsequent analyses (comprising 7.6% of all data). Total blood flow (tHb) was calculated as the summed absolute values of $O₂Hb$ and dHb.

Cycling task

Participants visited the laboratory for three sessions, one for each load condition. During each session they cycled for 30 min on a Life Fitness recumbent bicycle ergometer (Rosemont, Ilinois, USA) at either a low, a moderate, or a high load, followed by a 5-min cool down. The exertion loads were delineated using target HR, on the basis of age-adjusted HR_{max} , calculated as $HR_{\text{max}} = 207 - 0.7 \times \text{age}$ [\[20](#page-5-0)]. Under the low exertion condition, the participants pedaled for 30 min with minimal resistance and maintained a target HR of 52% HR_{max} . Under the moderate-exertion and high-exertion conditions, the resistance was increased until the participants reached ∼ 68% (moderate load) and 84% (high load) HR_{max} , respectively. Once the target HR was achieved, participants rode at a steady state until a total of 30 min had elapsed; ergometer resistance was manually adjusted throughout the session to maintain target HR ranges $(\pm 7.5\%)$. HR data were collected by Polar telemetry. Exercise load order was counterbalanced $(3! = 6$ orders) across participants to circumvent order effects (see [Fig. 1](#page-2-0) for a schematic representation of the study schedule).

To reduce diurnal variation in cognitive and physical performances, the test sessions were scheduled at the approximate same time of day within participants $(\pm 1 h)$. To reduce the influence of hydration status on physical performance, we asked the participants to consume half a liter of water the night before a test session, and half a

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Schematic representation of the study schedule. During the study sessions, participants first donned the heart rate monitor and fNIRS sensor. They began the cycling task, at one of three exercise loads (low, moderate, or high). During the 30-min cycling task, participants completed the Borg Scale of Perceived Exertion (RPE) every 5 min. Finally, participants completed a 5-min cool down. fNIRS, functional near-infrared spectroscopy. RPE, rated perceived exertion.

liter of water on the morning of the test session. Participants were also required to consume at least one meal before a morning test session (i.e. breakfast), and at least two meals before an afternoon test session (i.e. breakfast and lunch).

Rated perceived exertion

The Borg Scale of Perceived Exertion [rated perceived exertion (RPE)] [\[21](#page-5-0)] is a commonly used self-report measure of perceived physical exertion. The scale ranges from 6 (none) to 20 (very very hard) in RPE. Participants reported RPE every 5 min during the cycling task to verify perceived differences between the three exercise loads.

Statistical methods

O2Hb, dHb, tHb, and HR were averaged into 30 1-min time intervals and were then analyzed using analyses of variance (ANOVA) with exercise load 3 (52%, 68%, 84% HR_{max}) and time 30 (minutes 1, 2,..., 30) as withinparticipant factors. RPE was analyzed using an ANOVA with exercise load 3 (52%, 68%, 84% HR_{max}) and time 7 (minute 0, 5, 10, 15, 20, 25, 30) as withinparticipant factors. An effect was deemed statistically significant if the likelihood of its occurrence by chance was P less than 0.05. When sphericity was violated, Greenhouse–Geisser-corrected P-values were used. When an ANOVA yielded a significant main effect, posthoc tests using the Bonferroni correction were performed. All statistical analyses were carried out using SPSS 12.0 (SPSS Inc., Chicago, Illinois, USA).

Results

Functional near-infrared spectroscopy Oxygenated hemoglobin

Analysis showed that $O₂Hb$ increased as a function of exercise load $[F(2,46) = 13.493, P < 0.001; \eta^2 = 0.037]$, where O_2Hb was higher with a high rather than a moderate target HR_{max} ($P < 0.05$) and a moderate rather than a low target HR_{max} ($P < 0.01$). Likewise, O₂Hb increased as a function of time across all loads $[F(29,667) = 101.632]$, $P < 0.001$; $\eta^2 = 0.175$. Here, O₂Hb did not change within

the first minute of pedaling $(P> 0.41)$, but increased each minute thereafter (all $P's < 0.001$).

A load \times time interaction $[F(58, 1334) = 26.673, P < 0.001;$ η^2 = 0.022; [Fig. 2\]](#page-3-0) showed that O₂Hb was similar between exercise loads until minute 15. At minute 15, $O₂Hb$ increased with a high and moderate target HR_{max} compared with a low target HR_{max} . At minute 18, O₂HB also increased with a high target HR_{max} compared with a moderate target HR_{max}.

Deoxygenated hemoglobin

Analysis of dHb showed that dHb increased as a function of exercise load $[F(2,46) = 8.245, P < 0.01; \eta^2 = 0.091]$, in that dHb was higher for high rather than moderate HR_{max} $(P<0.05)$ and marginally higher for moderate rather than low target HR_{max} ($P = 0.060$). Likewise, dHb increased as a function of time across all loads $[F(29,667) = 12.940,$ $P < 0.01$; $\eta^2 = 0.048$]. Here, dHb did not change within the first 12 min of pedaling $(P > 0.10)$, but increased each minute thereafter (all $P's < 0.01$).

A load \times time interaction $[F(58, 1334) = 10.443, P < 0.001;$ η^2 = 0.039; [Fig. 3\]](#page-3-0) showed that dHb was similar between exercise loads until minute 23, when dHb became greater with a high > moderate > low HR_{max} (all $P's < 0.001$).

Total hemoglobin

Analysis of tHb showed that tHb increased as a function of exercise load $[F(2,46) = 19.543, P < 0.001; \eta^2 = 0.054]$, in that tHb was higher for high rather than moderate HR_{max} ($P < 0.001$), as well as when the target HR_{max} was moderate rather than low $(P<0.01)$. Likewise, tHb increased as a function of time across all loads $[F(29,667) = 108.01, P < 0.001; \eta^2 = 0.172]$. Here, tHb increased each minute upon commencing cycling (all $P's < 0.01$).

A load \times time interaction [$F(58, 1334) = 26.153$, $P < 0.001$; η^2 = 0.027] showed that tHb was similar between exercise loads until minute 16, when dHb became greater with a high > moderate > low HR_{max} (all $P's < 0.001$).

 O_2 Hb: mean (SE). The graph represents the change in O_2 Hb signals from the 30-s baseline intervals at each minute upon commencing exercise across the low, moderate, and high exercise loads $(n=24)$. Topographic plots indicate the effect of exercise load on the change in O₂Hb levels (centered on moderate load) for each channel across the 25–30-min interval, with darker shaded areas indicating higher parameter estimates (i.e. steeper slopes). O_2Hb , oxygenated hemoglobin.

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Table 1 Heart rate means (SE)

	Minutes 1-5	Minutes 6-10	Minutes 11-15	Minutes 16-20	Minutes 21-25	Minutes 25-30
Low	96.62 (1.70)	100.19 (1.73)	101.86 (1.88)	102.31 (1.59)	103.17 (1.65)	103.97 (1.56)
Moderate	98.28 (1.85)	106.41 (1.78)	112.35 (1.78)	119.86 (1.84)	122.71 (1.74)	127.62 (1.74)
High	104.47 (2.32)	113.37 (2.25)	120.29 (2.40)	127.64 (2.12)	133.35 (2.38)	139.39 (2.61)

The graph represents heart rate (in beats/min) at each 5-min interval upon commencing exercise across the low, moderate, and high exercise loads (n=24).

Heart rate

HR also increased as a function of load across all time points $[F(2,46) = 68.02, P < 0.001; \eta^2 = 0.364]$, in that HR was higher when the target HR_{max} was moderate rather than low, as well as when it was high rather than moderate – that is, high > moderate > low (all $P's < 0.001$). HR also increased with time across exercise loads $[F(29,667) = 125.795, P < 0.001; \eta^2 = 0.310]$. These main effects were qualified by an interaction between load and time $[F(58, 1334) = 29.524, P < 0.001; \eta^2 = 0.081],$ in which HR was higher with high > moderate > low HR_{max} at all time points after 3 min of cycling (all $P's < 0.01$; Table 1).

Rated perceived exertion

RPE increased as a function of load across all time points $[F(2,46) = 52.782, P < 0.001; \eta^2 = 0.286]$, in that RPE was higher when the target HR_{max} was high rather than moderate $(P<0.001)$, and moderate rather than low $(P<0.001)$. Likewise, RPE increased with time across all exercise loads $[F(6,138) = 91.618, P < 0.001; \eta^2 = 0.351]$, in that RPE was significantly greater at each subsequent 5-min period compared with the previous one (all P 's < 0.01), except between minutes 25 and 30, at which RPE remained constant $(P> 0.47)$. These main effects were qualified by an interaction between load and time $[F(12,276) = 15.569, P < 0.001; \eta^2 = 0.073]$. At 5 min of cycling, RPE was higher when the target HR_{max} was moderate and high, rather than low. At 20 min of cycling, RPE also increased when the target HR_{max} was high rather than moderate (all $P's < 0.01$).

Discussion

In the present study, participants cycled for 30 min to attain a target HR_{max} of 52, 68, or 84%, which they typically reached immediately, after 10 min, or after 20 min, respectively. This dose–response pattern of HR was mirrored in ratings of perceived exertion. $O₂Hb$ and dHb levels, and hence tHb level, in the PFC increased as a function of exercise load and duration (Figs 2 and 3). $O₂Hb$ levels increased throughout the exercise bout. This increase was magnified when the target HR_{max} was moderate and high, rather than low, at 15 min, as well as when the target HR_{max} was high rather than moderate – that is, high $>$ moderate $>$ low at 18 min. dHb showed the same general pattern, although the levels did not increase from rest until 12 min of cycling had elapsed. Further, dHb levels were higher when target HR_{max} was high rather than moderate, as well as when target HR_{max} was

moderate rather than low – that is high > moderate > low – at 23 min into the cycling task.

Enhanced total frontal cortical blood flow during lowintensity to moderate-intensity aerobic exercise is consistent with previous findings [\[7,11](#page-5-0),[12\]](#page-5-0). However, the present results somewhat diverge from prior results, in that $O₂Hb$ and dHb levels continued to increase when participants exercised at 84% HR_{max}, whereas previous reports have shown reductions in $O₂Hb$ levels in tandem with increases in dHb levels at such high intensities. For instance, incremental cycling exercise at 80% VO_{2max} reduced cerebral oxygenation over the right frontal cortex (reduced $O₂Hb$ and dHb levels relative to the resting state) [\[13](#page-5-0)]. Intermittent (30 s) bouts of exercise at 150% VO_{2max} resulted in increased $O₂Hb$ levels during the first three of seven sets, and decreased $O₂Hb$ levels compared with baseline during the final three sets, suggesting that increases in prefrontal blood flow may be attenuated over repeated bouts [\[14](#page-5-0)].

Similarly, exercise reduced cerebral oxygenation after participants reached the respiratory compensation threshold (RCT) or ventilatory threshold (VT), until they reached voluntary fatigue or indicators of VO_{2max} [\[10](#page-5-0)–[12](#page-5-0)]. The threshold at which exercise changes from 'moderate' to 'heavy' is referred to as the first VT (VT_1), and the threshold at which it changes from 'heavy' to 'unsustainable' is the second VT (VT₂), also called the RCT. Exercise between ~85% and 91% HR_{max} has been found to be sufficient to meet or exceed the RCT [\[22,23](#page-5-0)]. Given that our target $%$ HR_{max} is below this range, and that HR_{max} and the degree to which it is associated with other parameters of exercise intensity vary with fitness level and body composition, among other factors [\[24](#page-5-0)], our 84% HR_{max} condition may not have been sufficiently strenuous to induce a reduction in prefrontal oxygenation, as found in previous studies [\[10](#page-5-0)–[12](#page-5-0)].

Nevertheless, the results are generally in line with previous reports of increased total blood flow in the PFC during exercise, suggesting increased metabolic resource availability to sustain PFC-dependent cognitive task performance during exercise. Indeed the dosedependent and time-dependent increases in prefrontal O2Hb and dHb levels in the present report suggest that neural activity in this region is progressively enhanced during exercise, and such changes are likely specific to the PFC rather than reflective of total blood flow, which may become decoupled from regional blood flow during exercise [25]. The majority of research, including the present study, assessing exercise-dependent changes in PFC oxygenation focuses on young adults performing incremental cycling tasks, although exercise has been shown to enhance PFC oxygenation and associated executive function at other ages, as well as during different exercise tasks. For instance, older adults show augmented PFC oxygenation and associated cognitive performance during exercise [26,27]. During a treadmill exercise task, $O₂Hb$ levels in the PFC increased in a dose-dependent manner as participants accelerated from rest to either 3, 5 (both walking), or 9 km/h (running), suggesting a role for the PFC in adapting to the cognitive demand of the exercise task [15]. Age may moderate the extent to which the PFC is able to adapt to the cognitive demand of the task, as older adults may not recruit the PFC during exercise in tasks requiring executive function, such as walking while talking, to the same extent as younger adults [9]. Given the present findings that blood flow in the PFC incrementally increases with escalating exercise load and duration, future research should use similar designs to determine whether bouts of exercise with varying intensities influence PFC-associated affective and cognitive processing during and after exercise.

Concluding remarks

The present findings suggest that in habitual exercisers, cerebral blood flow in the PFC increases with higher exercise intensity and duration, as evidenced by both $O₂Hb$ and dHb measures during 30-min bouts of exercise with low, moderate, and high-intensity target loads. The results add to the growing literature supporting enhanced prefrontal blood flow during exercise and provide a novel design characteristic of typical exercise plans that could further be utilized to examine cognitive changes associated with such cortical changes.

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Conflicts of interest

There are no conflicts of interest.

References

- Penedo FJ, Dahn JR. Exercise and well-being: a review of mental and physical health benefits associated with physical activity. Curr Opin Psychiatry 2005; 18:189–193.
- 2 Reed J, Ones DS. The effect of acute aerobic exercise on positive activated affect: a meta-analysis. Psychol Sport Exerc 2006; 7:477–514.
- 3 Tomporowski PD. Effects of acute bouts of exercise on cognition. Acta Psychol (Amst) 2003; 112:297–324.
- Wood JN, Grafman J. Human prefrontal cortex: processing and representational perspectives. Nat Rev Neurosci 2003; 4:139–147.
- 5 Ekkekakis P. Illuminating the black box: investigating prefrontal cortical hemodynamics during exercise with near-infrared spectroscopy. J Sport Exerc Psychol 2009; 31:505–553.
- 6 Rooks CR, Thom NJ, McCully KK, Dishman RK. Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy: a systematic review. Prog Neurobiol 2010; 92:134–150.
- 7 Ide K, Horn A, Secher NH. Cerebral metabolic response to submaximal exercise. J Appl Physiol (1985) 1999; 87:1604–1608.
- 8 Kounalakis SN, Geladas ND. Cardiovascular drift and cerebral and muscle tissue oxygenation during prolonged cycling at different pedalling cadences. Appl Physiol Nutr Metab 2012; 37:407–417.
- 9 Holtzer R, Mahoney JR, Izzetoglu M, Izzetoglu K, Onaral B, Verghese J. fNIRS study of walking and walking while talking in young and old individuals. J Gerontol A Biol Sci Med Sci 2011; 66:879–887.
- 10 Bhambhani Y, Malik R, Mookerjee S. Cerebral oxygenation declines at exercise intensities above the respiratory compensation threshold. Respir Physiol Neurobiol 2007; 156:196–202.
- 11 Rupp T, Perrey S. Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. Eur J Appl Physiol 2008; 102:153–163.
- Timinkul A, Kato M, Omori T, Deocaris CC, Ito A, Kizuka T, et al. Enhancing effect of cerebral blood volume by mild exercise in healthy young men: a near-infrared spectroscopy study. Neurosci Res 2008; 61:242–248.
- 13 Ando S, Kokubu M, Yamada Y, Kimura M. Does cerebral oxygenation affect cognitive function during exercise? Eur J Appl Physiol 2011; 111:1973–1982.
- 14 Shibuya K, Tanaka J, Kuboyama N, Ogaki T. Cerebral oxygenation during intermittent supramaximal exercise. Respir Physiol Neurobiol 2004; 140:165–172.
- 15 Suzuki M, Miyai I, Ono T, Oda I, Konishi I, Kochiyama T, Kubota K. Prefrontal and premotor cortices are involved in adapting walking and running speed on the treadmill: an optical imaging study. Neuroimage 2004; 23:1020–1026.
- 16 Gillen JB, Gibala MJ. Is high-intensity interval training a time-efficient exercise strategy to improve health and fitness? Appl Physiol Nutr Metab 2014; 39:409–412.
- 17 Armstrong N, Barker AR. Endurance training and elite young athletes. Med Sport Sci 2011; 56:59–83.
- 18 Ayaz H, Onaral B, Izzetoglu K, Shewokis PA, McKendrick R, Parasuraman R. Continuous monitoring of brain dynamics with functional near infrared spectroscopy as a tool for neuroergonomic research: empirical examples and a technological development. Front Hum Neurosci 2013; 7:871.
- 19 Jasper HH. The ten-twenty electrode system of the International Federation. Electroencephalogr Clin Neurophysiol 1958; 10:370–375.
- 20 Gellish RL, Goslin BR, Olson RE, McDonald A, Russi GD, Moudgil VK. Longitudinal modeling of the relationship between age and maximal heart rate. Med Sci Sports Exerc 2007; 39:822–829.
- 21 Borg GA. Psychophysical bases of perceived exertion. Med Sci Sports Exerc 1982; 14:377–381.
- 22 Neder JA, Stein R. A simplified strategy for the estimation of the exercise ventilatory thresholds. Med Sci Sports Exerc 2006; 38:1007-1013.
- 23 Coplan NL, Gleim GW, Nicholas JA. Using exercise respiratory measurements to compare methods of exercise prescription. Am J Cardiol 1986; 58:832–836.
- 24 Garber CE, Blissmer B, Deschenes MR, Franklin BA, Lamonte MJ, Lee IM, et al. American College of Sports Medicine. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc 2011; 43:1334–1359.
- 25 Lucas SJ, Ainslie PN, Murrell CJ, Thomas KN, Franz EA, Cotter JD. Effect of age on exercise-induced alterations in cognitive executive function: relationship to cerebral perfusion. Exp Gerontol 2012; 47:541-551.
- 26 Hyodo K, Dan I, Suwabe K, Kyutoku Y, Yamada Y, Akahori M, et al. Acute moderate exercise enhances compensatory brain activation in older adults. Neurobiol Aging 2012; 33:2621–2632.
- 27 Tsujii T, Komatsu K, Sakatani K. Acute effects of physical exercise on prefrontal cortex activity in older adults: a functional near-infrared spectroscopy study. Adv Exp Med Biol 2013; 765:293–298.