

The relationship between exercise intensity, cerebral oxygenation and cognitive performance in young adults

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Abstract

Purpose To assess the relationship between exercise intensity, cerebral HbO₂ and cognitive performance (Executive and non-Executive) in young adults.

Methods We measured reaction time (RT) and accuracy, during a computerized Stroop task, in 19 young adults (7 males and 12 females). Their mean \pm SD age, height, body mass and body mass index (BMI) were 24 ± 4 years, 1.67 ± 0.07 m, 72 ± 14 kg and 25 ± 3 kg m⁻², respectively. Each subject performed the Stroop task at rest and during cycling at exercise of low intensity [40 % of peak power output (PPO)], moderate intensity (60 % of PPO) and high intensity (85 % of PPO). Cerebral oxygenation

was monitored during the resting and exercise conditions over the prefrontal cortex (PFC) using near-infrared spectroscopy (NIRS).

Results High-intensity exercise slowed RT in both the Naming ($p = 0.04$) and the Executive condition ($p = 0.04$). The analysis also revealed that high-intensity exercise was associated with a decreased accuracy when compared to low-intensity exercise ($p = 0.021$). Neuroimaging results confirm a decrease of cerebral oxygenation during high-intensity exercise in comparison to low- ($p = 0.004$) and moderate-intensity exercise ($p = 0.003$). Correlations revealed that a lower cerebral HbO₂ in the prefrontal cortex was associated with slower RT in the Executive condition only ($p = 0.04$, $g = -0.72$).

Conclusion Results of the present study suggest that low to moderate exercise intensity does not alter Executive functioning, but that exercise impairs cognitive functions (Executive and non-Executive) when the physical workload becomes heavy. The cerebral HbO₂ correlation suggests that a lower availability of HbO₂ was associated with slower RT in the Executive condition only.

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Keywords Reaction time · Near-infrared spectroscopy · Cerebral HbO₂ · Peak power output · Hyperventilation

Abbreviations

ANOVA	Analysis of variance
BMI	Body mass index
CO ₂	Carbon dioxide
CBF	Cerebral blood flow
CBF	Cerebral blood flow
fNIRS	Functional near-infrared spectroscopy
GLM	General linear model
HbO	Oxy-hemoglobin
HbR	Deoxy-hemoglobin

HbT	Total hemoglobin
HPF	High pass filter
MICE	Moderate-intensity continuous exercise
NIRS	Near-infrared spectroscopy
PaCO ₂	CO ₂ arterial pressure
PFC	Prefrontal cortex
PPO	Peak power output
Ra	Respiratory alkalosis
RA	Respiratory acidosis
RCT	Respiratory compensation threshold
RT	Reaction time
SD	Standard deviation
THT	Transient hypofrontality theory
$\dot{V}O_2$	Oxygen uptake
W	Watts

Introduction

The mechanism by which acute exercise influences cognitive functions has not yet been clearly established (McMorris and Graydon 1997; Brisswalter et al. 2002; McMorris et al. 2003; Pesce et al. 2007a, b). The transient hypofrontality theory (THT) posited to account for this relationship states that the brain, to drive bodily motion, is forced to make profound changes to the way it allocates its metabolic resources (Dietrich 2003). According to this theory, a temporary inhibition in regions such as the prefrontal cortex may emerge. Indeed, research that has combined exercise and cognitive tasks has demonstrated that cognitive tasks that are prefrontal lobe dependent (Executive tasks involving updating, inhibition, switching) are preferentially impaired during endurance exercise when compared with cognitive tasks that are not as prefrontal dependent (non-Executive tasks: simple reaction time tasks) (Dietrich and Sparling 2004). Moreover, a recent study from our group demonstrated that this effect was more pronounced in lower-fit than higher-fit individuals (Labelle et al. 2013).

Studies using in vivo brain imaging techniques have been designed to describe and understand the nature of the relationship between exercise intensity and brain functions. Among the neuroimaging techniques that have been successful at measuring brain activity in the prefrontal cortex during exercise is near-infrared spectroscopy (NIRS). Studies using NIRS have clearly shown the existence of an inverted U-shape relationship between cerebral oxygenation and exercise intensity (Gonzalez-Alonso et al. 2004; Bhambhani et al. 2007; Subudhi et al. 2007). The main physiological trigger of that typical kinetics appears to be the relationship between arterial pressure in CO₂ (PaCO₂) and cerebral hemodynamics. As shown by Bhambhani et al. (2007) in young adults, the decrease in cerebral blood flow and cerebral oxygenation observed at the end of the

exercise intensity spectrum is concomitant with the respiratory compensation threshold and its resulting hypocapnia. This phenomenon has been reported in a localized fashion over the frontal cerebral cortex, and this has been put forward as an explanation for the specific impairments observed in Executive function tasks reported in some studies. Several reports using functional NIRS (fNIRS) have described the relationship between exercise intensity, cerebral oxygenation and Executive performance through the prefrontal-dependent Stroop task (Lucas et al. 2010; Yanagisawa et al. 2010; Ando et al. 2011). Whatever the exercise intensity [40–80 % peak $\dot{V}O_2$ (Ando et al. 2011), 50 % peak power output (Yanagisawa et al. 2010) and 30–70 % heart rate range (Lucas et al. 2010)], there was consensus across these studies that an increase in cerebral oxygenation resulted in improved Executive performance. These studies support the first portion of the inverted U-shape relationship between exercise intensity and cerebral oxygenation, and demonstrate that low to moderate exercise intensity can improve Executive functions through a modification of cerebral hemodynamics. However, they did not measure the effect of higher exercise intensities, particularly beyond the respiratory compensation threshold. Clarifying the effect of exercise intensity (particularly high intensity) on cerebral oxygenation and its relationship to Executive performance might help further understand the basic mechanisms of the effect of aerobic exercise on cognition.

The aim of the present study was to address this gap in the literature and investigate the relationship between Executive performance and cerebral oxygenation during exercise intensities that cover a wide intensity spectrum (i.e., before and after the respiratory compensation threshold). We hypothesized that: (1) Executive performance would be impaired during high-intensity exercise when compared with low or moderate exercise intensity and that (2) this alteration of Executive performance would be associated with significant changes in prefrontal cerebral oxygenation.

Methods

Participants

In this study, 19 young adults (7 males and 12 females) gave their written informed consent to participate in the study. Their mean \pm SD age, height, body mass, body mass index (BMI), peak power output and $\dot{V}O_{2peak}$ were, 24 ± 4 years, 1.67 ± 0.07 m, 72 ± 14 kg, 25 ± 3 kg m⁻², 200 ± 64 W and 36 ± 10 ml min⁻¹ kg⁻¹, respectively. All participants were healthy, had normal-to-corrected vision and were right handed. None of the participants had a history of neurological or psychiatric disorder, color blindness, a surgery with general anesthesia in the past 6 months, involuntary

tremors, epilepsy or drug/alcohol problems. The protocol was reviewed and approved by the Institutional Research Ethics Board in Health Sciences of the Montreal Geriatrics Institute and was conducted in accordance with recognized ethical standards and national/international laws.

Experimental design

The experiment involved two sessions. Following a thorough briefing and medical screening, participants completed a maximal continuous graded exercise test. In the second session, they completed a constant intensity exercise test during which they performed a cognitive test. Both sessions were separated by a minimum 24 h and took place in a laboratory room kept at constant temperature (22 °C) and humidity (45 %). Participants were asked to refrain from strenuous exercise the day before the test and to arrive fully hydrated to the laboratory and at least 3 h after their last meal. No attempt was made to control the content of this meal. All exercises were performed on an electro-mechanically braked bicycle ergometer (Lode B.V., Medical Technology, Goningen, Netherlands). Saddle height was individually adjusted so that there was a slight bend in the knee joint, even when the pedals were at their furthest distance.

Exercise testing

Maximal continuous graded exercise test

The initial workload was set at 1 W/kg body mass, for example 75 W for an individual with a body mass of 75 kg. The workload was increased by 15 W every minute until voluntary exhaustion. Strong verbal encouragement was given throughout the test. The power of the last completed stage was considered as the peak power output (PPO, measured in W). Oxygen uptake ($\dot{V}O_2$, in ml min⁻¹ kg⁻¹) was determined continuously on a 30-s basis using an automated cardiopulmonary exercise system (Moxus, AEI Technologies, Naperville, IL, USA). Gas analyzers were calibrated before each test using a gas mixture of known concentrations (15 % O₂ and 5 % CO₂). The turbine was calibrated before each test using a 3-l syringe at several flow rates. The highest over a 30-s period during the test was considered as the peak oxygen uptake ($\dot{V}O_{2\text{peak}}$, in ml min⁻¹ kg⁻¹).

Constant intensity exercise test

Participants had to perform a Stroop task under four conditions: at rest (no exercise) and during a constant intensity exercise at low (40 % of PPO), moderate (60 % of PPO) and high intensities (85 % of PPO). The cognitive task was

performed using a standard laptop that was adjusted for each participant and located in front of him or her. The rest condition was performed first, after which intensity levels were counterbalanced across participants according to a 3 × 3 repeated measures Latin square design, as presented elsewhere (Labelle et al. 2013). Each exercise sequence had to be maintained for 9 min and was separated from the other by a 6-min recovery period during which participants remained seated on the bicycle ergometer without pedaling. The first 2 min of each sequence was devoted to reaching a $\dot{V}O_2$ steady state and then the computerized modified Stroop task was performed until the end of each exercise sequence.

Cognitive test

The computerized Stroop test was based on the Modified Stroop task used by Bohnen et al. (1992) and included two experimental conditions: Naming (non-Executive) and Executive. Each run of the task involved two blocks of each condition composed of 15 trials in each block, resulting in 30 trials per experimental condition. Experimental blocks were interspersed with a 1-min condition named Rest. During this condition, participants were asked to keep pedaling at the required intensity, but were not presented with any trials. All experimental trials began with a fixation cross for 500 ms, and all visual stimuli appeared in the center of the computer screen for 2000 ms duration. In the Naming block, participants were presented with 15 trials involving a visual stimuli (XXX) colored in green or in blue. Each color was linked to a button located on the handles of the ergocycle (i.e., left button = green response; right button = blue response). In each block, participants had to press the button that corresponded to the color of the ink of the stimulus being presented. In the Executive block, there were two trial types in the block: inhibition and switch trials. In the inhibition trials, the participant was presented the word BLUE in green ink (or GREEN in blue ink) and they had to restrain from reading the word and respond to the ink color. In the switch trials, there was a box surrounding the word presented (i.e., BLUE in green ink), and when this box appeared around the word the participants were instructed to change their response set and now read the word, ignore the color of the ink, and respond to the word they read (i.e., BLUE). In the Executive block, 75 % of the trials were inhibition trials and the remaining 25 % of trials were switch trials. The switch trials appeared randomly throughout the block.

Prior to the experimental blocks, to get familiarized with the apparatus and experimental procedure, the participants completed three practice blocks of 20 trials in the Naming condition, while seated at the ergocycle (without pedaling). Throughout practice, visual feedback was provided

after each trial, with the words “correct answer” or “incorrect answer” appearing in the center of the screen. After the three practice conditions were completed, participants began the four experimental blocks, which involved cycling while performing the task. The conditions were always presented in the same order (ABAB design): Rest–Naming–Rest–Executive–Rest–Naming–Rest–Executive. Baseline NIRS data were acquired for 1 min prior to each of the exercise segments. Reaction times (in milliseconds) and accuracy values were recorded for all conditions.

Measurement of cerebral oxy- and deoxy-hemoglobin

Changes in the concentration of oxy-hemoglobin (HbO₂) and deoxy-hemoglobin (HbR) were measured by a multichannel, continuous-wave spectrometer (CW6, TechEn Inc., Milford, MA, USA), which emits an 830-nm wavelength more sensitive to HbO₂ and a 690-nm wavelength more sensitive to HbR. Two bands of NIRS sources and detectors were positioned on the subject’s head, one on each frontal lobe. Each band consisted of three rows of four optodes each, with one central row of four sources, and one inferior/ventral row and one superior/dorsal row of four detectors. The 16 detectors were placed optimally 2.8 cm away from the nearest sources. The two bands were placed symmetrically over the lateral prefrontal cortex, and the most anterior and most ventral source–detector pair of each band was placed on Fp1/Fp2 using the 10/20 system. The NIRS transmitters were tightly secured with a tensor bandage wrapped around the forehead to limit movement during exercise and to ensure that there was no infiltration of background light (Fig. 1).

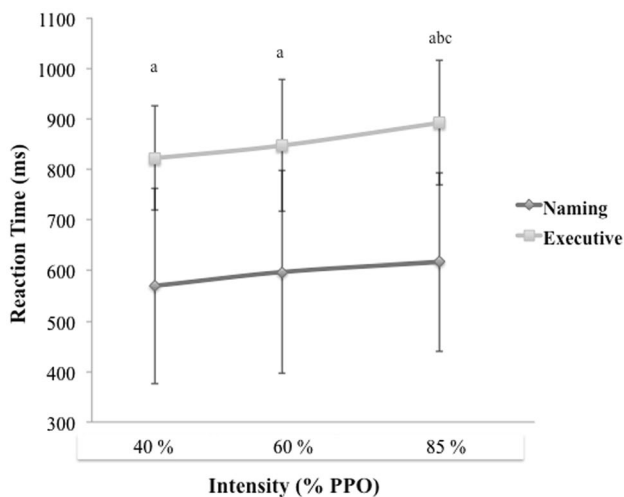


Fig. 1 Mean reaction times as a function of exercise intensity in the Naming and Executive condition. PPO peak power output. Compared with the Naming condition: ^a $p < 0.01$, Compared to 40 % PPO: ^b $p < 0.05$. Compared to 60 % PPO: ^c $p < 0.05$

Because continuous-wave technology does not measure optical path lengths (Ekkekakis 2009), only changes in concentration of HbR, HbO₂ and HbT relative to baseline could be inferred assuming both a path length factor and partial volume. Total hemoglobin (HbT), the sum of HbR and HbO₂, was used as a measure of cerebral blood volume (CBV) assuming constant hematocrit (Ekkekakis 2009).

Statistical analysis

Behavioral data were analyzed using SPSS v. 16.0 for Mac. NIRS data were analyzed using custom-built Matlab code. A 5 % confidence level for statistical significance was targeted after corrections for multiple comparisons. Two-way 3×2 repeated measures ANOVAs were performed on each of HbR, HbO₂ and HbT responses, intensity (40, 60, 85 %) and task (Naming and Executive) as within-subject factors. Two corrections for multiple comparisons were applied. (1) The Euler characteristic method (Ye et al. 2009) was applied to account for the spatial extent of NIRS topographic maps. (2) Furthermore, the degrees of freedom of the F tests were corrected using the method of Greenhouse–Geisser when the assumption of sphericity was violated [except when this violation was mild ($\epsilon > 0.75$)], then the method of Huynh–Feldt was used. The Maxwell Delaney guidelines for analyzing two-way ANOVAs were followed. (1) First, the interaction was investigated. Since it was significant after Greenhouse–Geisser correction (2) we then performed Bonferroni-corrected F tests of the effect of either factor on each level of the other factor (Huynh–Feldt corrected). Behavioral data, RT and accuracy data were analyzed using a 2 (Stroop condition) \times 3 (exercise intensity) repeated measure ANOVA. Follow-up analyses were conducted with pairwise comparisons using a Bonferroni correction (Ye et al. 2009).

Concentration changes were calculated using the modified Beer–Lambert law and a path-length factor of 5.9, from measured optical intensities, which were normalized by the time-wise median value of each NIRS channel calculated separately for each intensity segment (Strangman et al. 2003).

The data of each individual participant were analyzed using a general linear model (GLM) approach (Ye et al. 2009), using an in-house Matlab code based on SPM and NIRS_SPM frameworks. The design matrix was built from a convolution of the protocol with the standard SPM8 hemodynamic response function. To this design matrix, regressors representing a linear trend and a constant were added for the high pass filter (HPF). A Gaussian low pass filter of 1.5-s width was applied to the NIRS data. Individual responses for each intensity level and task were obtained, from the GLM inversion.

To assess the direct relationship between cerebral HbO₂ and cognitive function, we performed a median split of our sample. We divided our population sample based on cerebral HbO₂ [individuals with low cerebral HbO₂ ($n = 9$) and individuals with high cerebral HbO₂ ($n = 10$)]. We then attested the relationship between the level of cerebral HbO₂ and the reaction time in both cognitive conditions using the magnitude of difference and a paired t test between both conditions.

The magnitude of the difference was assessed by the Hedges g (g), as presented elsewhere (Dupuy et al. 2014). The scale proposed by Cohen (1988) was used for interpretation. The magnitude of the difference was considered either small ($0.2 < ES \leq 0.5$), moderate ($0.5 < ES \leq 0.8$), or large ($ES > 0.8$)

Results

Maximal continuous graded exercise test

Peak power output and $\dot{V}O_{2\text{peak}}$ reached by the participants during the test were 200 ± 64 W and 36 ± 10 ml min⁻¹ kg⁻¹, respectively.

Cognitive test

Reaction time

Results for mean reaction time as a function of exercise intensity in the Naming and Executive conditions are presented in Fig. 1. The analysis revealed a main effect of Stroop condition [$F(2, 17) = 6.25$, $p < 0.05$], with longer reaction times (RT) in the Executive compared to the Naming condition. Analysis of reaction times also revealed a main effect of exercise intensity [$F(1, 18) = 139.27$, $p < 0.001$], where high-intensity exercise induced an increase in RT when compared with all intensity conditions. There was no interaction between the Stroop condition and exercise intensity [$F(2, 17) = 1.09$, $p = 0.357$]. In fact, high-intensity exercise significantly increased RT in both the Naming ($p = 0.04$) and the Executive condition ($p = 0.04$).

Accuracy

Results for mean accuracy as a function of exercise intensity in the Naming and Executive conditions are presented in Fig. 2. Analysis revealed a main effect of Stroop condition [$F(1, 17) = 40.27$, $p < 0.001$], with a reduced accuracy in the Executive condition when compared with the Naming condition. Results also show a main effect of exercise intensity [$F(2, 16) = 4.94$, $p < 0.021$], where high-intensity

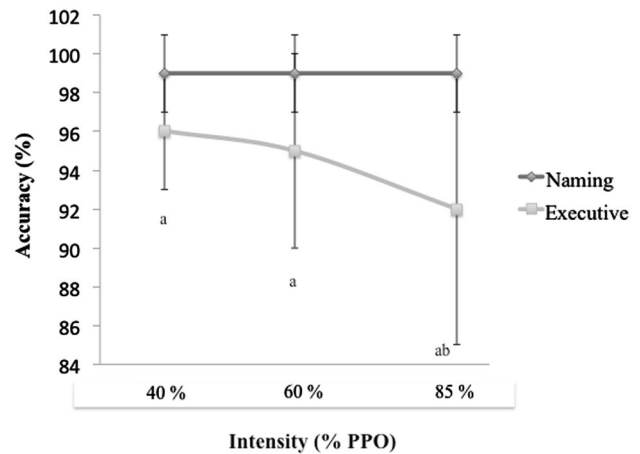


Fig. 2 Mean accuracy as a function of exercise intensity in the Naming and Executive condition. PPO peak power output. Compared with Naming condition: ^a $p < 0.01$, Compared to 40 % PPO: ^b $p < 0.05$

exercise was associated with a decreased accuracy when compared with low-intensity exercise. The interaction between exercise intensity and task was not significant [$F(2, 16) = 2.70$, $p = 0.098$].

Cerebral responses

Due to technical problems (poor signal in the prefrontal cortex), the data from one male and three females were excluded from the NIRS analysis. Illustration of NIRS results for the left side of the brain at the pixel where the main effect of intensity on HbO₂ was highest are presented in Table 1.

HbO₂ response

In HbO₂ values, there was a main effect of intensity [$F(2, 30) = 8.45$], $p < 0.001$]. Cerebral oxygenation was significantly lower during high-intensity exercise (85 % PPO) when compared with low-intensity exercise (40 % PPO) ($p = 0.004$) and moderate-intensity exercise (60 % PPO) ($p = 0.003$). There was also a significant interaction between Stroop task and exercise intensity [$F(2, 30) = 4.82$, $p = 0.015$]. Pairwise comparisons analysis revealed that both Naming and Executive conditions demonstrated lower cerebral oxygenation during high-intensity exercise.

HbR response

The HbR analysis revealed a significant interaction between exercise intensity and Stroop condition [$F(2, 30) = 4.958$, $p = 0.014$]. Pairwise comparisons revealed that at moderate intensity (60 % PPO), the change in HbR

Table 1 Cerebral hemodynamics during cognitive tasks at various workloads

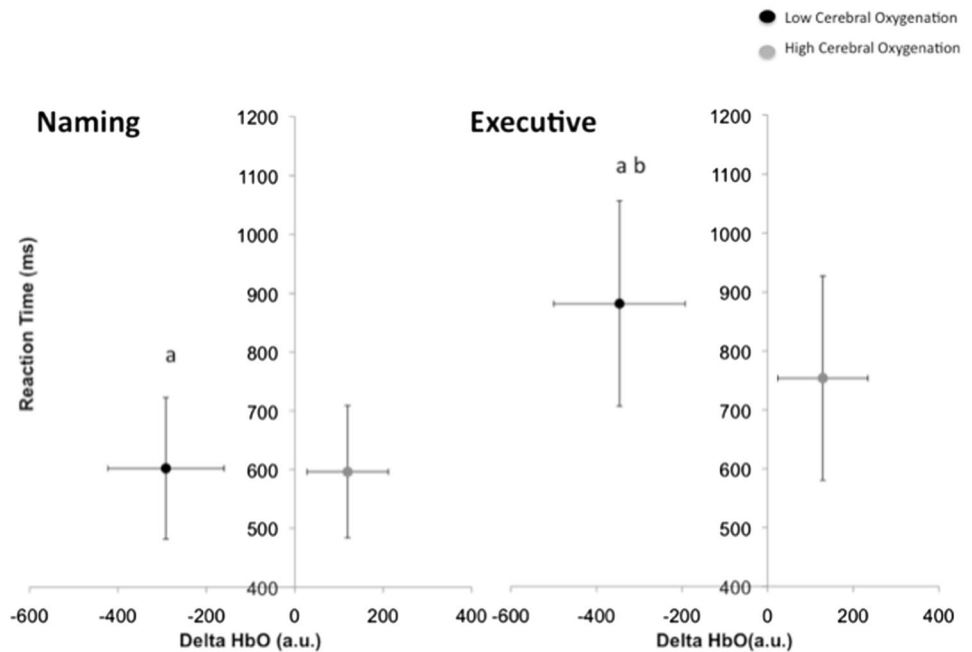
	HbO		HbR		HbT	
	Naming	Executive	Naming	Executive	Naming	Executive
40 % PPO	-32 ± 142	-31 ± 182	30 ± 73	22 ± 82	-2 ± 127	-9 ± 158
60 % PPO	2 ± 176	30 ± 187	13 ± 77	-17 ± 69	16 ± 170	13 ± 170
85 % PPO	-195 ± 208 ^a	-287 ± 326 ^a	49 ± 129	90 ± 195	-146 ± 158 ^a	-197 ± 209 ^a

Values are expressed as the mean (SD) difference from baseline

PPO peak power output

Compared to 40 % PPO and 60 % PPO : ^a $p < 0.05$

Fig. 3 The overall relationship between cerebral oxygenation and cognitive tasks. Values are expressed as mean (SD). Compared to low cerebral oxygenation: ^a $p < 0.05$. Compared to low oxygenation reaction time during the Executive task: ^b $p < 0.05$



was significantly different ($p = 0.024$) between the Executive condition ($M = -17.46$; $SE = 17.34$) and the Naming condition ($M = 13.22$, $SE = 19.40$).

HbT response

Cerebral HbT analysis disclosed a main effect of intensity [$F(2, 105) = 21.4$, $p < 0.05$]. HbT was significantly lower during high-intensity exercise when compared with low-intensity exercise ($p = 0.003$) and moderate-intensity exercise ($p = 0.009$). There was no significant difference when low and moderate intensity were compared ($p = 0.72$).

Relationship between Stroop conditions and cerebral HbO₂

Results examining the relationship between the cognitive task and cerebral HbO₂ are illustrated in Fig. 3. The analysis revealed that a higher cerebral HbO₂ in the pre-frontal cortex was selectively associated with faster RT in

the Executive condition only ($p = 0.04$, $g = -0.72$). When comparing the levels of cerebral HbO₂ and RT in the Naming condition, we found no significant relationship between the variables ($p = 0.89$, $g = -0.04$).

Discussion

The aim of this study was to investigate the relationship between cognitive function and cerebral HbO₂ over a wide range of exercise intensities in healthy young individuals. We hypothesized that (1) Executive function would be impaired during an exercise of vigorous intensity when compared with low or moderate exercise intensity and that (2) the relationship between Executive performance and exercise intensity would be associated with significant changes in cerebral HbO₂. In accordance with these hypotheses, we found a significant decrease in cerebral HbO₂ during exercise at vigorous intensity, as well as a

significant decrease in Executive and non-Executive performance. The results of our correlations indicate that there is a significant relationship between the increase of reaction time in the Executive condition and decrease in cerebral oxygenation observed.

The behavioral results of our study are in accordance with existing hypotheses and theories (i.e., THT) which state that a greater impairment in cognitive functions, which are heavily supported by prefrontal regions, will be observed during high-intensity exercise (Brisswalter et al. 2002; Dietrich and Audiffren 2011; McMorris et al. 2009a, b). In our study, however, we found that both Executive and non-Executive tasks were affected at high intensities and that this effect was not specific to Executive tasks. The non-Executive finding is similar to simple reaction time research by Brisswalter et al. (2002). Brisswalter and colleagues examined how simple reaction time performance was influenced by pedaling at different rates at an identical power output on a cycle ergometer. The results of this study demonstrated that a higher pedaling rate (80 rev min⁻¹) corresponded to a greater simple reaction time (Brisswalter et al. 2002). Chmura et al. (1994) determined that participants' reaction times were related to exercise intensity. Reaction times increased dramatically when the resistive load was increased to 300 W. Similar to our findings, this study demonstrates that even performances on simple reaction time tasks (non-Executive tasks) can be altered by high-intensity exercise.

Yet, Dietrich and Sparling (2004) showed that compared to a resting condition, younger adults performed poorer at an Executive task requiring switching abilities (i.e., Wisconsin Cards Sorting Test) than at an intelligence assessment when exercising at 60 % of their HR_{max}. In both cases, tasks requiring the involvement of the prefrontal lobes (to different degrees) were disrupted by exercise. Compared to a resting condition, other studies have reported a decrease in inhibition abilities while exercising at 60 % (Pontifex and Hillman 2007) and 70–85 % (Dietrich and Sparling 2004) of their HR_{max}. Some authors also reported a change to an easier strategy in the inhibition component of a random generation task (RNG) at an average of 46 % of PPO compared to rest (Audiffren et al. 2009). One must remember that intensity levels and the index of exercise intensity compared in those studies (i.e., rest vs. exercise; HR_{max} and PPO) were different from the ones used in our study (i.e., different intensities during exercise; PPO). Our cognitive results demonstrate that both the Executive and Naming conditions were impaired during high-intensity exercise. This non-significant difference between Naming and Executive RT at high intensities could possibly be the consequence of our lower-fit sample size ($\dot{V}O_{2\max}$ of 36 ± 10). A recent study from our group has demonstrated that when compared with their lower-fit

counterparts ($\dot{V}O_{2\max}$ of 38.33 ± 5.16), higher-fit individuals ($\dot{V}O_{2\max}$ of 50.62 ± 7.86) showed an increase in cognitive performance during high-intensity exercise. Results from this study also indicate that both lower- and high-fit young adults exhibit a difficulty to switch attention between two sets of rules while performing a high-intensity exercise (Labelle et al. 2013). In addition, we found a significant main effect of exercise intensity on the accuracy, as indicated by a decrease in accuracy at 85 % PPO. Although it was not significant, we observed a trend where participants seemed to have poorer accuracy in the Executive function during higher workloads. Nevertheless, there was no difference between conditions at lower workloads (40 % PPO and 60 % PPO). Taken together, these findings suggest that at high exercise intensities, it may be the case that lower-fit individuals will be unable to maintain behavioral performances even for the simple non-Executive tasks.

Studies provide a viable explanation for the negative cognitive performances observed during this protocol designed to test the inverted-U hypothesis. McMorris et al. (2009a, b) have found that neuroendocrinological changes could be potential underpinnings of the effect of acute cardiovascular exercise on cognition via the influence of catecholamines and cortisol. During high-intensity exercise, an increase in cortisol level can lead to an important increase in arousal, which might then lead to a decrease in cognitive performances. The catecholamine increase can lead to an activation of the limbic system at the expense of the prefrontal lobes and therefore cause a decrease in performance in Executive control tasks (McMorris et al. 2009a, b). In the current experiment, we counterbalanced our different exercise intensities (40, 60, 85 %) with a Latin square design, to control for arousal effects across the different intensity levels. since we compared across different intensities. This design has been used by previous studies to control for arousal affects across exercise intensity (Labelle et al., 2013).

Compared with the other neuroimaging methods, NIRS has both some important strengths and some notable limitations. On the one hand, NIRS is a noninvasive and relatively low-cost optical technique that has become a widely used instrument for measuring changes in HbO and, indirectly, brain blood flow. On the other hand, NIRS does not have a good spatial resolution and the depth coverage is not good, so most NIRS investigations are limited by skull thickness, scalp flow or adipose tissue thickness (Ekkekakis 2009).

While caution should be taken when interpreting cerebral HbO₂ during exercise, previous studies have shown that fNIRS signal can be used as a reliable index of cerebral response during maximal and submaximal exercise (Bhambhani et al. 2007; Ando et al. 2011). Results from a recent literature review regrouping the last decade of NIRS

studies have indicated an inverted U-shape relationship between cerebral blood oxygenation and exercise intensity during an incremental test (Rooks et al. 2010). The mechanism that subtends this relationship relies on a number of contributors, among which the regulatory effect of PaCO₂ on CBF appears to be the most important (Querido and Sheel 2007). The kinetics of PaCO₂ during an exercise of increasing intensity is biphasic. Until the attainment of the ventilatory compensation threshold, the failure to eliminate extra CO₂ because of a drop in alveolar ventilation induces an increase in PaCO₂, which in turn increases cerebral blood flow (Querido and Sheel 2007) and cerebral blood oxygenation (Rooks et al. 2010). Once exercise intensity exceeds the ventilatory compensation threshold, and the large increase in alveolar ventilation that takes place induces a decrease in PaCO₂ that results in a vasoconstriction of cerebral arteries (Bhambhani et al. 2007) and a decrease of cerebral blood oxygenation (Ogoh et al. 2010; Rooks et al. 2010). The decrease of HbT observed during high-intensity exercise represents very well the decrease of blood flow caused by vasoconstriction of the cerebral arteries.

In the present study, we found an increase in HbO and a decrease in HbT in the prefrontal cortex during exercises of low and moderate intensity, and an opposite pattern during vigorous intensity exercise suggesting a decrease in cerebral HbO₂ and cerebral blood flow. We know that increases in neuronal activity elicit an augmentation in blood flow that is not matched by an equal increase in oxygen consumption (Ekkekakis 2009). This is in accordance with a model that predicts a higher oxygen extraction fraction for reduced CBF, and the overall findings of this study are consistent with previous reports that have assessed the cognitive response to different intensity exercise while measuring cerebral response (Lucas et al. 2010; Ando et al. 2011).

These results confirm the importance of cerebral oxygenation availability in the prefrontal cortex during cognitive tasks.

Despite no differences in cerebral oxygenation at higher intensities between both cognitive tasks, our correlations suggest that higher cerebral HbO₂ in the prefrontal cortex was selectively associated with faster overall RT in the Executive condition only. Our findings are in line with Dupuy et al. (2015), who found that higher-fit individuals who demonstrate better cardiorespiratory functions show faster reaction times and greater cerebral oxygenation. Similarly, our results align with Prakash et al. (2011), who reported a greater blood oxygen level-dependent (BOLD) signal increases in the prefrontal cortex during the Executive Stroop conditions for individuals who demonstrated a higher cardiorespiratory function. Although the purpose of this study was not to observe the effects of fitness on cognition and cerebral oxygenation, our results are in accordance

with these findings since we found, regardless of exercise intensity, a higher cerebral oxygenation during the Executive condition of the Stroop task. However, within the current study, we do not have all the measures necessary to examine the role of physical fitness on cerebral oxygenation and future research is needed to clarify this result.

A few limitations in this study warrant discussion. We measured cerebral HbO₂ in the prefrontal cortex during exercise. Although the frontal cortex is activated during a Stroop task, activity in the anterior cingulate cortex also plays a key role in the inhibition response (Colcombe et al. 2004). Therefore, our interpretation of the cognitive data is limited to our choice of brain area (PFC) and observing the contributions of additional areas involved in inhibition might extend the current findings. Although we tried controlling for arousal by using a Latin square design, a more appropriate control would have been to measure skin conductance for fluctuations in arousal during exercise and cognitive task performance.

Oxygen uptake, RPE, HR and ventilatory parameters were not measured during submaximal intensity exercises. This choice was justified by the fact that many studies have already described the response of these variables during this kind of exercise. We therefore decided to focus on cerebral HbO₂ and Executive performance.

Practical implications

The results of this study opened doors to understanding the influence of cardiovascular exercise on cognition and will help optimize training in younger and older adults hoping to attenuate the drop of physical and cognitive functions associated with an aging population. A well-based training program may delay the the drop in cerebral oxygenation and permit individuals to have better cognitive functions in their daily activities. While the current study focused on the relationship between exercise and cognition in a healthy population, these findings may also be beneficial in symptomatic population research. It is important to consider the role of exercise and cognition in a rehabilitation protocol in a population with a cardiovascular burden.

Conclusion

Although the relationship between cerebral response and cognition requires further investigation, the current study took a step in assessing the relationship between cerebral HbO₂ and cognitive functions. The results of this study opened doors to a better understanding of the influence of cerebral HbO₂ on cognitive workload and will help understand the mechanisms by which cognitive functions are

altered during exercise. The methodological framework that was established by this study can be adapted for studies having objectives, but in different populations (patients with Alzheimer disease, coronary heart disease, etc.). More research is needed, particularly in samples that are low fit or that may have Executive function decline, to assess how exercise intensity, cerebral oxygenation and cognitive performance relate. While the current study focused on the relationship of cerebral HbO₂ and cognition in a healthy population, these findings may also be beneficial in other symptomatic population research.

Conflict of interest The authors of this paper have no conflicts of interest.

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