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Pushing to the limits: The dynamics of cognitive control during exhausting exercise

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ABSTRACT

This study aimed at investigating concurrent changes in cognitive control and cerebral oxygenation (Cox) during steady intense exercise to volitional exhaustion. Fifteen participants were monitored using prefrontal near-infrared spectroscopy and electromyography of the thumb muscles during the completion of an Eriksen flanker task completed either at rest (control condition) or while cycling at a strenuous intensity until exhaustion (exercise condition). Two time windows were matched between the conditions to distinguish a potential exercise-induced evolutive cognitive effect: an initial period and a terminal period. In the initial period, Cox remained unaltered and, contrary to theoretical predictions, exercise did not induce any deficit in selective response inhibition. Rather, the drop-off of the delta curve as reaction time lengthened suggested enhanced efficiency of cognitive processes in the first part of the exercise bout. Shortly before exhaustion, Cox values were severely reduced – though not characteristic of a hypofrontality state – while no sign of deficit in selective response inhibition was observed. Despite this, individual's susceptibility to making fast impulsive errors increased and less efficient online correction of incorrect activation was observed near exhaustion. A negative correlation between Cox values and error rate was observed and is discussed in terms of cerebral resources redistribution.

1. Introduction

It is now well-accepted that physical exercise has a positive effect on basic cognitive functions (Tomporowski, 2003; Lambourne and Tomporowski, 2010), however its impact on higher cognitive processes (*e.g.* selective inhibition) is less clear. According to the different meta-analyzes on exercise and cognition, it seems that exercise would lead to a rather small positive effect on cognitive functioning due to large variations of the reported results (Chang et al., 2012; McMorris et al., 2011). It has been proposed that exercise intensity would be the most important factor to explain this variability. Specifically, an inverted-*U* function has been suggested in such a form that exercise above a certain intensity is no longer beneficial to cognitive functioning (McMorris and Hale, 2012). In other words, while moderate exercise is associated with a positive effect, intense exercise (*i.e.* above the second ventilatory threshold, VT2) is associated to a null or negative effect on cognitive functioning. This view has been supported by the main theoretical models. According to the arousal-cognitive performance (Yerkes and Dodson, 1908), the catecholamine (Cooper, 1973; McMorris et al., 2008) and the reticular-activating hypofrontality (Dietrich, 2003, 2009; Dietrich and Audiffren, 2011) theories, intense exercise, by inducing high levels of arousal, increasing neural noise, or down-regulating prefrontal cortex activity, respectively, is predicted to impede higher cognitive processes.

Evidence of the detrimental effect of intense exercise mostly come from studies using incremental protocols in which the effects of exercise, probed at the end of the exercise, are confounded with the effect of exhaustion (*e.g.* Ando et al., 2005; Chmura and Nazar, 2010; McMorris et al., 2009). The present study aimed to dissociate the effect of the intensity from the state of exhaustion using a steady state intense exercise performed until exhaustion. Exhaustion is a psychophysiological state concluding a fatigue development process. It is a predictable consequence to any strenuous exercise. The state of exhaustion can also be considered as the time spent on the task. In a concomitant realization of a choice reaction time task and a fatiguing submaximal contraction,

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Lorist et al. (2002) showed that the more the time-on-tasks elapsed, the more error rate and force variability of hand muscles increased, suggesting an increasing detrimental effect of the dual-task on the dual-performance.

The detrimental effect of exercise on cognition occurring near exhaustion can be explained as neural competition for the limited cerebral resources between different centers of the brain (Dietrich, 2003, 2009; Dietrich and Audiffren, 2011). Specifically, the hypofrontality theory predicts that the prefrontal cortex (PFC), in response to the development of muscular fatigue, would be down-regulated to favor the allocation of resources to motor areas, which would in turn result in weaker cognitive functioning. The resource redistribution hypothesis has been supported by animal studies assessing cerebral activity during exercise. Tracing regional cerebral blood flow and local cerebral glucose utilization as indexes of cerebral activity showed a selective cortical and subcortical recruitment of brain areas during exercise (Delp et al., 2001; Gross et al., 1980; Holschneider et al., 2003; Vissing et al., 1996). Specifically, while motor and sensory cortices, basal ganglia, cerebellum, midbrain and brainstem nuclei were consistently activated at high intensities, the frontal cortex rather showed signs of deactivation. Positron emission tomography studies have also provided a similar pattern of findings in human (Tashiro et al., 2001, Kemppainen et al., 2005). If such neural balance may rationalize down-regulated cognitive performances during exercise (Dietrich and Audiffren, 2011), previous result remain based on an instantaneous description of the neural pattern during exercise, which does not inform about changes related to the proximity of exhaustion.

Near-infrared spectroscopy (NIRS) is a continuous tissue-monitoring technique which is able of tracking cerebral oxygenation (Cox) during exercise due to its relative robustness during movement (see Perrey, 2008). The NIRS method has been validated and correlates highly with both electro-encephalographic and functional magnetic resonance imaging responses (Timinkul et al., 2010; Toronov et al., 2001). NIRS studies during exercise have shown a very dynamic Cox pattern. A meta-analysis by Rooks et al. (2010) proposes an intensity based account with the second ventilatory threshold as the critical reversing point in the inverted-*U* relation between exercise intensity and PFC oxygenation determined through oxyhemoglobin concentration [HbO_2]. Nevertheless, this pattern is specific to untrained participants, as trained participants do not show any [HbO_2] decline at high intensities. In addition, most of the studies reviewed employed incremental protocols. In studies that push participants until exhaustion, results suggest that [HbO_2] reduction may be related to the individual time course to exhaustion. Timinkul et al. (2008) reported an individual timing of Cox desaturation, occurring before VT2 for some participants and after for others. During steady intense exhausting exercise, Shibuya et al. (2004a,b) reported Cox patterns identical to those observed in incremental exercise (e.g., Bhambhani et al., 2007; Rupp and Perrey, 2008). However, the paucity of the exercise-NIRS studies on intense as well as prolonged exercise to exhaustion in humans cannot ensure the validity of a fatigue-based PFC deactivation hypothesis.

The second aim of the present study was to investigate concurrent changes in cognitive performance and Cox in PFC while performing strenuous exercise until volitional exhaustion. More specifically, the protocol was designed to determine whether cognitive performance and Cox follow a similar dynamic. In an initial period of intense exercise, it was anticipated that cognitive performance would be facilitated and Cox elevated compared to the same initial period at rest. Then, in a critical period occurring just before exhaustion, we expected a decrease in cognitive performance and a drop of PFC [HbO_2] in comparison to the same period at rest. Cognitive performances were assessed using a modified version of the Eriksen flanker task (Eriksen and Eriksen, 1974), consisting in overcoming the irrelevant dimension of the stimulus to give the correct response, to probe the efficiency of selective response inhibition. The flanker task has been largely used to investigate the effects of exercise on cognitive control (Davranche et al., 2009a,b; McMorris et al., 2009; Pontifex and Hillman, 2007). Although mean RT and average error rate do provide valuable information relative to cognitive processes, more-detailed data analyzes uncover modulations that the sole consideration of central tendency indices cannot reveal. Indeed, combined to RT distribution analyzes, conflict tasks have proved to be powerful for assessing the processes implemented during decision-making tasks while exercising (Davranche and McMorris, 2009; Davranche et al., 2009a,b; Joyce et al., 2014).

On a substantial amount of trials, although the correct response was given, a subthreshold electromyographic (EMG) activity in the muscles involved in the incorrect response could be observed. Such subthreshold EMG activities, named "partial errors", reflect incorrect action impulses that were successfully corrected in order to prevent a response error (Hasbroucq et al., 1999). To evaluate the efficiency of the cognitive control during exercise, electromyographic (EMG) activity of response effector muscles were monitored to estimate the number of partial EMG errors. In order to measure Cox, NIRS recording was centered on the right inferior frontal cortex (rIFC) as this brain area is a main region involved in the brain network supporting the inhibition function (Aron et al., 2004, 2014), and has been described as the most responsive region while performing the Eriksen flanker task (Hazeltine et al., 2000). Additionally, the distribution-analytical technique and the delta plot analysis (Ridderinkhof, 2002; Ridderinkhof et al., 2004) were used to assess the efficiency of cognitive control and the propensity to make fast impulsive reactions through the analyzes of the percentage of correct responses (CAF) and the magnitude of the interference effect (delta curve) as a function of the latency of the response (van den Wildenberg et al., 2010). If exhausting exercise impairs the efficiency of cognitive control, the drop-off of the delta curve should be less pronounced in the terminal period than in the initial period. If the propensity to commit impulsive errors increases before exhaustion, more errors are expected for fast RT trials on distributional analyzes of response errors.

2. Method

2.1. Participants

Fifteen volunteers took part in this experiment. They were mostly classified as untrained following the $\dot{V}O_2$ max criteria of de Pauw et al. (2013) and had basic cycling experience (< 1 h a week). Informed written consent was obtained according to the declaration of Helsinki. Participants' anthropometrical and

Table 1
Anthropometrical and physiological characteristics of participants.

Variables	Mean ± SD		
	All	Women	Men
Sample size	15	5	10
Age [years]	22.1 ± 0.6	23.2 ± 1.2	21.5 ± 0.6
Height [cm]	175.9 ± 2.6	166.3 ± 1.8	180.7 ± 2.6
Body mass [kg]	66.5 ± 3.1	52.9 ± 2.1	73.3 ± 2.3
$\dot{V}O_2$ max [ml kg^{-1} min^{-1}]	44.5 ± 1.9	45.2 ± 2.9	44.1 ± 2.6
Maximal HR [bpm]	178 ± 2.6	180 ± 1.9	177 ± 3.0
MAP [W]	261.3 ± 14.2	204.3 ± 10.3	290.2 ± 12.2

Results are presented as the mean group ± SD.
Notes. SD = standard deviation; MAP = maximal aerobic power; $\dot{V}O_2$max = maximal oxygen consumption; HR = heart rate.

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physiological characteristics are presented in Table 1.

2.2. Apparatus and display

All sessions were performed on a cycle ergometer (Brain-bike NeuroActive, Motion Fitness, 1775 Winnetka Circle, Rolling Meadows, IL 60008) equipped with a handlebar and soft padding supports to comfortably support forearms. Two thumb response keys were fixed on the top of the right and left handle grips. A screen mounted on the ergometer at head height faced participants at a mean distance of 70 cm.

2.3. Cognitive task

The cognitive task consisted of a modified version of the Eriksen flanker task (Eriksen and Eriksen, 1974). Participants were required to complete 20 blocks of 40 trials during the control session and as many blocks as possible until exhaustion in the exercise session. There were two types of trials in each block: congruent trials (CO, 50%, all arrows uni-directional) and incongruent trials (IN, 50%, center arrow contra-directional). Each trial began with the presentation of a cross at the center as a fixation point. After 800 ms, the stimulus was presented and participants had to respond according to the direction pointed by the central arrow. The delivery of the response turned off the stimulus. When participants failed to respond within 2000 ms, the stimulus was terminated and the next trial began. The inter-stimulus interval was 800 ms. In this modified version, each group of arrows could randomly be displayed either at the top or at the bottom of the screen (Fig. 1). This modification ensured a higher processing of the flankers as participants could not anticipate the location of the central arrow.

2.4. Electromyographic measurement

Surface EMG activity of the *flexor pollicis brevis* (FPB) of each thumb and of the *vastus lateralis* (VL) of the right thigh were recorded using bipolar Ag/AgCl electrodes (diameter: 10 mm, inter-electrode distance (FPB, VL): 12 mm, 20 mm). The common reference electrode was situated on the head of the second metacarpal of the right hand. Electrodes locations were determined as proposed by Hasbroucq et al. (1999) and were marked in order to ensure congruent positioning in both sessions. Low inter-electrode impedance ($<3\ k\Omega$) was obtained via skin preparation. EMG signals were amplified (gain x1000), filtered (pass-band 10–500 Hz) and recorded at 2000 Hz (MP100 Biopac® Systems Inc., Holliston, MA, USA). During both sessions, the experimenter continuously monitored the signal of the thumbs and asked the subjects to relax their muscles if the signal became noisy through increased preactivation.

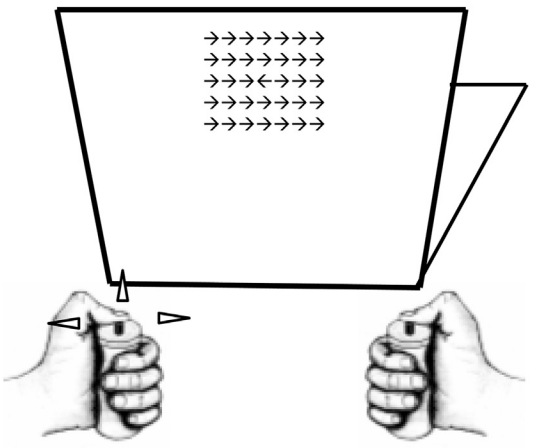

Fig. 1. Schematic drawing of an incongruent trial displayed at the top of the screen. In this example, participants had to respond by pressing the left response key according to the direction indicated by the central arrow.

2.5. Cerebral oxygenation measurement

Cox was monitored using a Near-infrared Spectroscopy (NIRS) system (Artinis Medical, PortaMon, The Netherlands). The NIRS evaluates the amount of infrared light that effectively traverses an investigated tissue from an emitter to a receptor. The received signal allows the description of a quantitative change from baseline in chromophore concentration. The PortaMon continuously emits two wavelengths of IR light, 773 and 853 nm, which are situated within the absorption spectrum of hemoglobin (Hb) and Myoglobin (Mb). Using two wavelengths allows for determination of the changes in [HbO_2] and deoxyhemoglobin ([HHb]) concentration. The inter-optode distance (IOD) was fixed through the apparatus at 30, 35 and 40 mm. Positioning of the NIRS probe was considered paramount to the study design (Strangman et al., 2003; Boas et al. 2001; Mansouri et al. 2010) and great care was taken that the probe was tangential to the curvature of the cranium and that no contamination through ambient light was present. Fixation was obtained through a custom-designed multi-density foam receptacle which was secured using a system of straps to prevent movement during exercise. Location of the rIFC was determined using AF8 references from the electro-encephalic 10–20 international system and measured in duplicate. The probe position was then extensively marked on the skin using a surgical marker and was also documented photographically for later reference to ensure congruent placement in the following session. Data was acquired at 10 Hz. Concentrations were calculated using the standard form of the modified Beer–Lambert Law (Beer, 1851; Delpy et al. 1988). For this calculation, the wavelength-specific extinction coefficients were extracted from Cope (1991) and the equally specific differential pathlength factors (DPF) were adopted from Duncan et al. (1995). HbO_2 concentration from the deepest source (IOD_{40}, ca. 2 cm) has been previously reported to be the most sensitive indicator of regional cerebral oxygenation changes and of neural activity (Hoshi, Kobayashi and Tamura, 2001) and was therefore selected as the primary outcome measure. Post-acquisition, the data was normalized to the 2-min rest period and the control data was truncated to retain the same duration as cognitive task time completion in the exercise condition. Data was subsequently reduced to 20 datapoints using a spline interpolation.

2.6. Experimental procedure

Three sessions (one training session and two experimental sessions) were conducted at standard (± 1 h) morning hours (from 8 to 12 pm) within close intervals (4 ± 3 days). Participants were instructed to abstain from any vigorous exercise for 24 h pre-session, to sleep at least seven hours in the night before each session and to avoid caffeinated drinks in the morning.

During the training session, subjects performed four blocks of 80 trials of the cognitive task to prevent a potential learning effect. Additional blocks were completed if the participant did not fulfill the following learning criteria: (a) RT intra-block variability below 5%, (b) RT variability with the previous block below 5%, (c) mean RT inferior to 600 ms, and (d) response accuracy superior to 85%. Participants then performed a maximal aerobic power (MAP) test on the cycle ergometer. The resistance was automatically regulated to ensure constant power output independent of pedal frequency. Power output was increased by 10 W every 30 s after a 4 min

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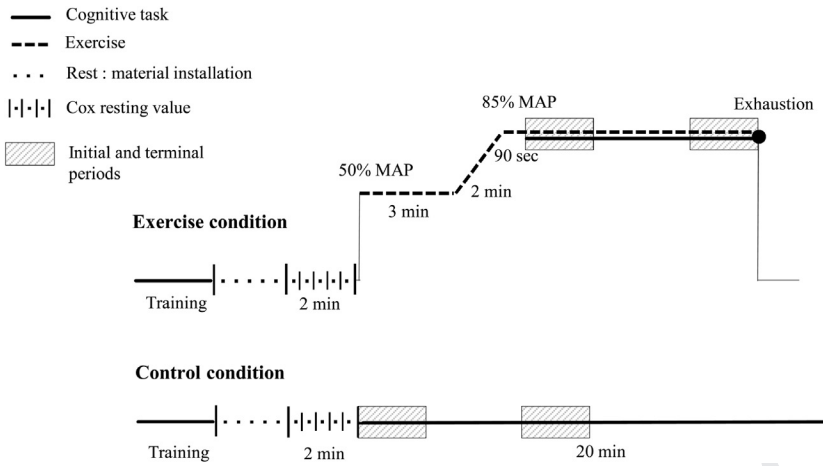

Fig. 2. Schematic representation of the experimental sessions which consisted of performing an Eriksen flanker task for 20 min without exercising (control condition) or while cycling at 85% of maximal aerobic power (MAP) (exercise condition). The initial period of exercise corresponds to the first hundred trials and the terminal period to the last hundred trials in the exercise condition. These two time windows were matched with the control condition. Cox resting values were measured prior to any cognitive and/or physical solicitation.

warm-up at light intensity (women: 70 W; men: 80 W). Oxygen consumption (ml kg^{-1} min^{-1}) and ventilatory output (L min^{-1}) were recorded using the Fitmate Pro gas analyzer (COSMED, Miami, USA) validated by Nieman et al. (2007). Cardiac frequency was recorded by a Polar system (Polar RS800CX, Polar Electror Oy, Kempele, Finland). Voluntary exhaustion was defined as the point where participants voluntarily stopped or when they could no longer maintain a pedaling frequency above 50 rotations per minute (RPM) more than 10 s despite strong verbal encouragement.

The experimental sessions consisted of a control condition and an exercise condition (Fig. 2). The order of the sessions was counterbalanced across participants. Each session began with two training blocks of 80 trials of the cognitive task (about 3 min). Following the installation of the NIRS and EMG sensors (about 30 min), the measurement of Cox resting values was conducted. The recording of baseline values only began if Cox level was visually stable during the last 2 min. Participants then performed the cognitive task for twenty minutes[1] without pedaling (control condition) or while pedaling at 85% of MAP until exhaustion (exercise condition). The intensity (regulated independently from pedal frequency) was chosen to be intense enough to exceed the second ventilatory threshold (VT2) while allowing an exercise duration long enough to obtain sufficient data to define two time periods: an initial period and a terminal period. In each condition, the initial period corresponded to the first hundred trials of the cognitive task performed by the participant. In the exercise condition, the terminal period corresponded to the last hundred trials performed just before exhaustion and was determined according to individual time to exhaustion. To obtain a matching terminal period in the control condition, the time frame corresponding to the cessation of pedaling during the exercise condition was extracted and the period calculated from there. The exercise condition began after a 3-min warm-up at 50% of MAP. The intensity was then increased every 20 s for 2 min until reaching 85% of MAP. To ensure that a steady-state of oxygen consumption was attained, a 90 s delay was imposed between the attainment of target intensity and the beginning of the cognitive task. Cox, heart rate (HR), pedaling frequency and electromyographic activity of the agonist muscles involved in the task were continuously monitored from rest to exhaustion.

2.7. Psychological measurement

Participants were asked to provide a verbal rating of perceived exertion (RPE) between each block of the cognitive task, using a visual Borg (6–20) scale (Borg, 1998). RPE was defined as the "perceived difficulty to exert at the same time the physical plus the cognitive tasks". The next block followed immediately after participants' RPE responses.

2.8. Data analysis

The EMG signals of the *flexor pollicis brevis* recorded during each trial were aligned to the onset of the imperative stimulus and the onsets of the changes in activity were visually determined. EMG signals with a background activity superior to 10% of the burst peak were rejected (Rejection rate: 20%). RT for pure correct trials–trials with no sign of EMG activation associated with the incorrect activation-was measured for each condition from onset of the stimulus to the onset of the EMG involved in the response. RTs of less than 100 ms and RTs higher than 1500 ms (3% of the total number of trials) were considered anticipated responses and omissions, and were excluded from further analysis. The incorrect activation trials were differentiated into two categories of trials: errors and partial EMG errors. Partial EMG errors were incorrect action impulses, mostly undetected consciously, that were successfully corrected (Hasbroucq et al., 1999; Rochet et al., 2014). The force exerted by the non-required effector was not sufficient to elicit an error and was followed by a correct activation which reached the response threshold. Trials containing a partial error are of particular interest, since they indicate that although an error was about to be made, the nervous system was able to overcome and provide the correct action. Error rate and partial EMG error rate were calculated according to the number of trials after artifact rejection. Additionally, the correction rate which represents the efficiency of the nervous system to overcome and provide the correct action after incorrect activations was calculated. It corresponds to the number of partial EMG errors divided by the total number of incorrect activations (partial EMG errors and errors).

For the exercise condition, the root mean square (RMS) from EMG signal of the VL was calculated using a custom signal detection routine in Matlab between the onset and the end of each burst recorded from the beginning of the cognitive task and until

[1] This corresponds to 1.5 times the best time-to-exhaustion performed during pre-tests and thus is ensured to completely cover the cognitive task duration during the exercise condition for all participants.

exhaustion. Results of each subject were reduced to 20 measurement points using a spline interpolation function in Matlab (R2012b, the MathWorks, Inc., Natick, MA) in order to depict the mean evolution of VL muscular activation during the task.

Through the analyzes of the percentage of correct responses (conditional accuracy functions, CAF) and the magnitude of the interference effect (delta curve) as a function of RT, the activation-suppression model provides a powerful framework to assess conflict resolution (for details see, van den Wildenberg et al., 2010). This model specifically allows for the assessment of both the initial phase linked to an individual's susceptibility to making fast impulsive errors (early automatic response activation) and, the later phase associated with the efficiency of the cognitive control (build-up of a top-down response suppression mechanism). Reaction time distribution was obtained using individual RTs "vincentized" into four equal-size speed bins (quartiles) for CO and IN trials separately. The lack of data for three participants did not permit a relevant vincentization, consequently they have not been taken into account in this analysis. Delta plots were constructed by calculating interference effect as a function of the response speed (average of difference between RT in IN and RT in CO trials for each quartile). Curve accuracy functions (CAF) were constructed by plotting accuracy as a function of the response speed. The data presented are the mean values of each set averaged across participants.

Separated ANOVAs were performed on each dependent variable (i.e., mean RT, partial EMG errors, errors and correction rates). The analyzes involved conditions (control vs. exercise) congruency (CO vs. IN) and periods (initial vs. terminal) as within-subject factors. In order to control for the effect of the order of the experimental session, this was initially included as a between-subject factor along with all its interaction terms with the other predictors in the analyzes. However, given that none of these variables reached significance ($ps > .10$), the order and its interaction terms were removed from the analyzes to optimize the parsimony of the models. An ANOVA including condition (control vs. exercise) and period (initial vs. terminal) as within-subjects factors was performed on hemoglobin concentration measures to determine whether cerebral oxygenation diverged. The analyzes conducted on the RMS of VL activation and RPE data recorded during the exercise session only included period (initial vs. terminal) as a within-subject factor. Exploratory Pearson correlations were performed between [HbO_2] concentration, cognitive performances (mean RT, error rate, partial error rate, correction rate) and root mean square (RMS) from EMG signal of the VL. Data presented on these measures correspond to mean values averaged across participants. The SPSS software (IBM® SPSS® Statistics 20) was employed for all analyzes. Planned comparisons were used in the GLM as post-hoc analyzes when significant p-values ($p < .05$) were found. Values are expressed as mean ± standard deviation (SD).

3. Results

3.1. Time to task failure, heart rate and rating of perceived exertion

In the exercise condition, the strenuous exercise until exhaustion lasted 360 ± 43 s, which enabled participants to complete 5.5 ± 0.4 blocks of the cognitive task (*i.e.*, average of 220 trials, ranged from 200 to 320 trials). Participants always stopped cycling at the end of a block of the cognitive task, meaning they always fully completed all the blocks they have started.

HR showed a main effect of condition ($F(1,14) = 1507.95$, $p < .001$, $\eta_p^2 = .99$), a main effect of period ($F(1,14) = 43.83$, $p < .001$, $\eta_p^2 = .76$) and an interaction between these two factors ($F(1,14) = 33.27$, $p < .001$, $\eta_p^2 = .70$). In the control condition, HR remained stable from the initial (67 ± 4 beat per minute, bpm) to the terminal period (66 ± 3 bpm, $F(1,14) < 1$, $p = .73$), but increased from 162 ± 2 bpm to 176 ± 2 bpm in the exercise condition ($F(1,14) = 152.12$, $p < .001$, $\eta_p^2 = .92$).

RPE results showed a main effect of period ($F(1,14) = 387.18$, $p < .001$, $\eta_p^2 = .96$) and condition ($F(1,14) = 450.92$, $p < .001$, $\eta_p^2 = .97$). An interaction between these two factors was observed ($F(1,14) = 6.91$, $p = .02$, $\eta_p^2 = .33$). The increase in RPE was greater in the exercise condition (from 14.2 ± 0.6 to 19.4 ± 0.3) compared to the control condition (6.5 ± 0.2 to 8.9 ± 0.7).

3.2. Reaction time

Results showed a main effect of congruency ($F(1,14) = 29.72$, $p < .001$, $\eta_p^2 = .68$) with longer RT for IN trials (454 ± 13 ms) than for CO trials (410 ± 8 ms). No other main effect or interaction was significant.

3.3. Error rate

Results showed a main effect of congruency ($F(1,14) = 60.18$, $p < .001$, $\eta_p^2 = .81$), a main effect of condition ($F(1,14) = 9.54$, $p < .01$, $\eta_p^2 = .40$) as well as an interaction between these two factors ($F(1,14) = 11.74$, $p < .01$, $\eta_p^2 = .45$, Fig. 3). The frequency of errors was lower during rest than when exercising for IN trials (Ctrl: 9.93 ± 1.14%; Exer: 18.15 ± 3.67%; $p < .01$) but not for CO trials (Ctrl: 0.86 ± 0.53%; Exer: 1.21 ± 0.52%; $p = .58$) (Fig. 3). Interestingly, the interaction between condition and period showed a trend ($F(1,14) = 3.37$, $p = .08$, $\eta_p^2 = .19$) and suggests that the evolution of accuracy differs between control and exercise conditions. In the terminal period, participants committed more errors during exercise (10.73 ± 3.84%) than at rest (5.32 ± 1.59%, $p < .01$, $\eta_p^2 = .40$), whereas they had an equivalent error rate in the initial period (Exer: 8.31 ± 2.54%; Ctrl: 5.88 ± 1.41%; $p > .05$).

3.4. Partial EMG error

Results showed a main effect of congruency ($F(1,14) = 143.76$, $p < .001$, $\eta_p^2 = .91$), condition ($F(1,14) = 11.32$, $p < .01$, $\eta_p^2 = .45$) and period ($F(1,14) = 7.21$, $p = .01$, $\eta_p^2 = .34$). The number of partial EMG errors was increased for IN trials (36.92 ± 4.13%) compared to CO trials (10.65 ± 2.81%), exercise (28.09 ± 5.58%) compared to rest (19.49 ± 4.76%) and greater in the terminal period (25.83 ± 5.90%) than in the initial period (21.74 ± 4.26%). No interaction reached

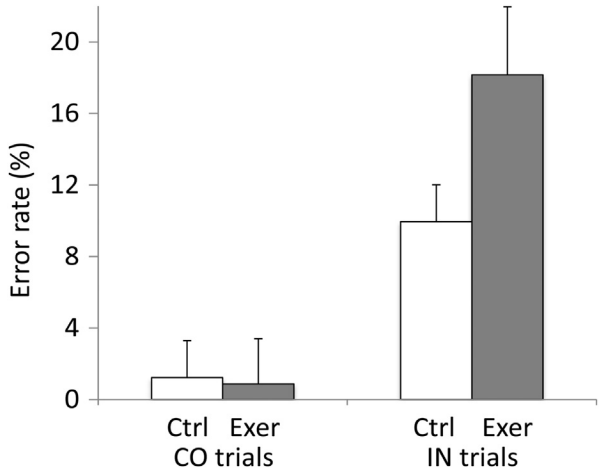

Fig. 3. Error rate (in percentage) during control (Ctrl, white bars) and exercise (Exer, grey bars) conditions for congruent (CO) and incongruent (IN) trials. Error bars represent standard deviation.

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Table 2
Mean reaction times, accuracies and partial errors per condition and period in the Eriksen flanker task.

Variables		Control Initial	Control Terminal	Exercise Initial	Exercise Terminal
CO	RT (ms)	411 ± 34	414 ± 43	406 ± 40	410 ± 49
	Acc (%)	1.7 ± 2.3	0.7 ± 1.7	1 ± 1.9	0.7 ± 2.1
	PE (%)	7 ± 6.8	8.2 ± 6.5	12.8 ± 9.1	14.7 ± 10.3
IN	RT (ms)	454 ± 50	461 ± 46	447 ± 63	456 ± 76
	Acc (%)	10 ± 3.7	9.9 ± 4.9	15.7 ± 8.2	20.6 ± 14.2
	PE (%)	27.6 ± 14.8	35.2 ± 14.5	39.6 ± 14	45.3 ± 17.3

Results are presented as the mean group ± SD.
Notes. SD=standard deviation; CO=congruent trials; IN=incongruent trials; RT=reaction time; Acc=accuracy; PE=partial error.

significance. The interaction between condition and period did not reached significance ($F(1;14)=.04$; $p=.84$; $\eta_p^2=.003$). All the results for RT, error rate (accuracy) and partial EMG error are presented in Table 2.

3.5. Correction rate

Correction rate trended towards significant interaction between condition and period ($F(1,14)=4.12$, $p=.06$, $\eta_p^2=.23$, Fig. 4). In the initial period, the correction rate was equivalent at rest and during exercise ($F(1,14)=0.82$, $p=.37$, $\eta_p^2=.05$). However, in the terminal period, participants were less capable to correct incorrect action impulses during exercise (68.92 ± 3.42%) than in the control condition (78.05 ± 3.56%) ($F(1,14)=7.03$, $p=.01$, $\eta_p^2=.33$). This finding suggests a deficit in cognitive control just before exhaustion, incorrect action impulses were not corrected effectively and more errors were committed.

3.6. Distributional analysis

Reaction time distributions were submitted to an ANOVA involving condition (control vs. exercise), congruency (CO vs. IN), period (initial vs. terminal) and quartile (Q1, Q2, Q3, Q4) as within-subjects factors. Results confirmed the main effect of congruency previously observed on mean RT ($F(1, 11)=73.07$, $p<.001$, $\eta_p^2=.87$). More interestingly, the analysis showed an interaction between condition and quartile ($F(3,33)=5.16$, $p<.01$, $\eta_p^2=.32$) which revealed that exercise differently affects RT performance as a function of the response speed (Fig. 5A). A beneficial effect of exercise was actually observed for the first quartile (Q1: −27 ms, $p<.01$), and the second quartile (Q2: −21 ms, $p<.05$) and disappeared for the last two quartiles (Q3: −11 ms, $p=.19$; Q4, +13 ms, $p=.10$).

According to van den Wildenberg et al. (2010), the magnitude of interference as RT lengthened is associated with the efficiency of the cognitive control (build-up of a top-down response suppression mechanism). Then, a second analysis focused on delta plot slopes was conducted to examine whether exercise altered the magnitude of the interference. The analysis involved condition (control vs. exercise), period (initial vs. terminal) and quartile (Q1, Q2, Q3, Q4) as within-subject factors. Results showed that the magnitude of interference did not fluctuate as RT lengthened, except during the initial period of exercise (Fig. 5B). In this period, the interference decreased from 64 ms (± 9 ms) in the first quartile to 25 ms (± 12 ms) in the last quartile ($F(1,11)=14.19$, $p<.01$) suggesting that non-exhausting intense exercise enhances cognitive control. It is noteworthy that no sign of any deficit in cognitive control was observed in the terminal period of exercise when exhaustion was about to occur ($F(1,11)=0.07$, $p=.79$).

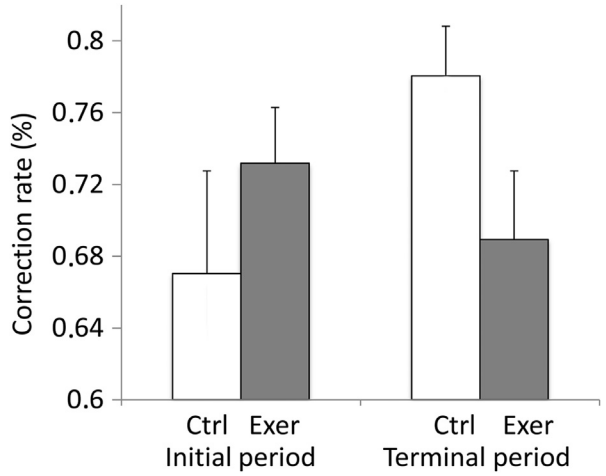

Fig. 4. Correction rate (in percentage) during rest (Ctrl, white bars) and exercise (Exer, grey bars) conditions for the initial and terminal periods. Error bars represent standard deviation.

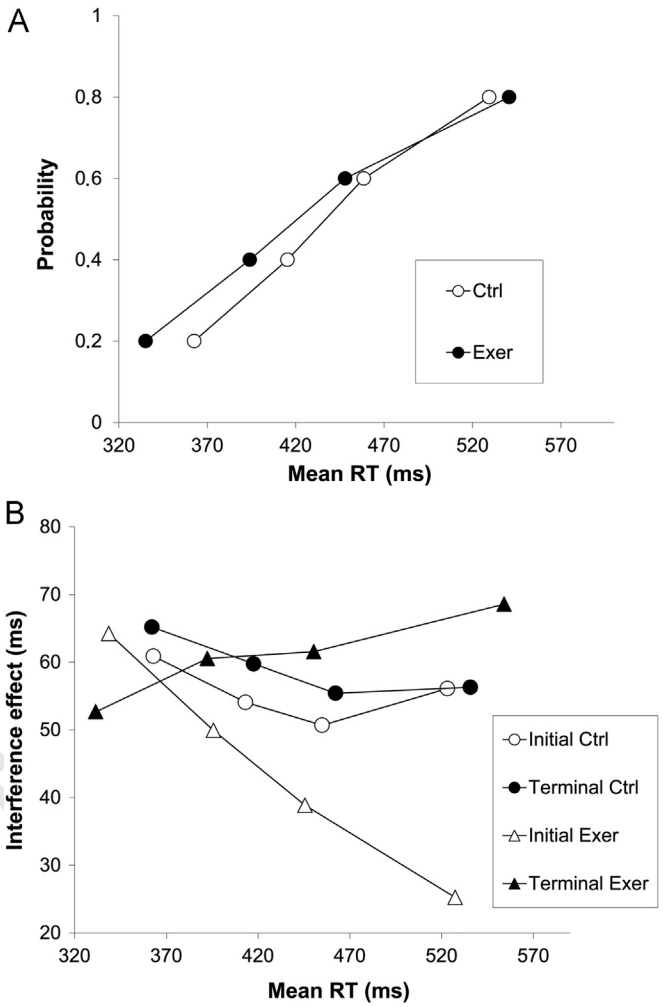

Fig. 5. (A) Cumulative density functions as a function of reaction time (RT) during rest (Ctrl, empty symbols) and exercise (Exer, full symbols) conditions. (B) Delta plots of RT illustrating the magnitude of the interference (in ms) as a function of RT during rest (Ctrl, circle) and exercise (Exer, triangle) conditions for the initial (empty symbols) and terminal (full symbols) periods.

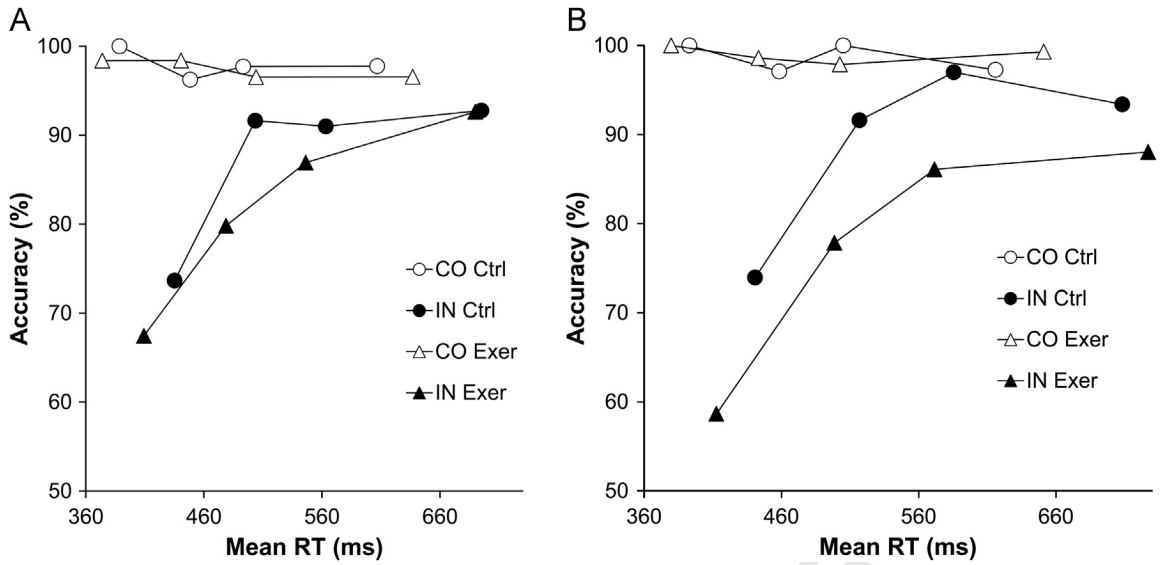

Fig. 6. Conditional accuracy function (CAF) representing the percentage of accuracy for congruent (CO, empty symbols) and incongruent (IN, full symbols) trials as a function of reaction time (RT) during control (Ctrl, circle) and exercise (Exer, triangle) conditions for the initial (A) and terminal (B) periods.

3.7. Conditional accuracy functions (CAF)

An ANOVA involving condition (control vs. exercise), congruency (CO vs. IN), period (initial vs. terminal) and quartile (Q1, Q2, Q3, Q4) as within-subject factors was conducted on error rates (Fig. 6). Results confirmed the main effects of condition ($F(3,14)=9.78$, $p<.01$, $\eta_p^2=.41$), congruency ($F(3,14)=40.35$, $p<.001$, $\eta_p^2=.74$) and the interaction between the two ($F(3,14)=11.14$, $p<.01$, $\eta_p^2=.44$) previously observed on mean error rate. In line with the activation–suppression model (Ridderinkhof, 2002), the interaction between congruency and quartile, which illustrated the strength of the automatic response triggered by the flankers, was significant ($F(3, 42)=25.02$, $p<.001$, $\eta_p^2=.64$). The interference was more pronounced for the first quartile than for the second quartile (Q1: 31 ms *vs.* Q2: 12 ms, $p<.001$) and for the second quartile compared to the third quartile (Q2: 12 ms vs. Q3: 8 ms, $p<.01$), whereas the interference was equivalent for the last two quartiles (Q3: 8 ms vs. Q4: 6 ms, $p=.23$). The interaction between condition, congruency and period ($F(1,14)=3.89$, $p=.07$, $\eta_p^2=.22$) and the interaction between condition, congruency and quartile ($F(3,14)=2.71$, $p=.07$, $\eta_p^2=.16$) tended to be significant.

A second series of analyzes, focusing on the first quartile of the CAF, was conducted to examine whether exercise alters the rapid response impulse (van den Wildenberg et al., 2010). The analysis carried out on the initial period of exercise did not reveal an interaction between condition and congruency ($F(1,14)=1.05$, $p=.32$, Fig. 6A). However, in the terminal period of exercise, there was a significant interaction ($F(1,14)=12.68$, $p<.01$, $\eta_p^2=.49$). Just before exhaustion, participants committed about 15% more errors than at rest in IN trials (Fig. 6B) while the accuracy rate in CO trials remained unchanged.

3.8. Cerebral oxygenation

An interaction between condition and period was observed on Cox fluctuations ($F(1,14)=5.98$, $p<.001$, $\eta_p^2=.30$) (Fig. 7). During the control condition, the time spent on the task did not impact Cox ($F(1,14)=0.71$, $p=.98$, $\eta_p^2=.04$). During exercise, $\Delta[HbO_2]$ linearly (linearity, $p<.05$) decreased from 2.71 ± 0.05 μmol cm in the initial period to 0.21 ± 0.06 μmol.cm in the terminal period

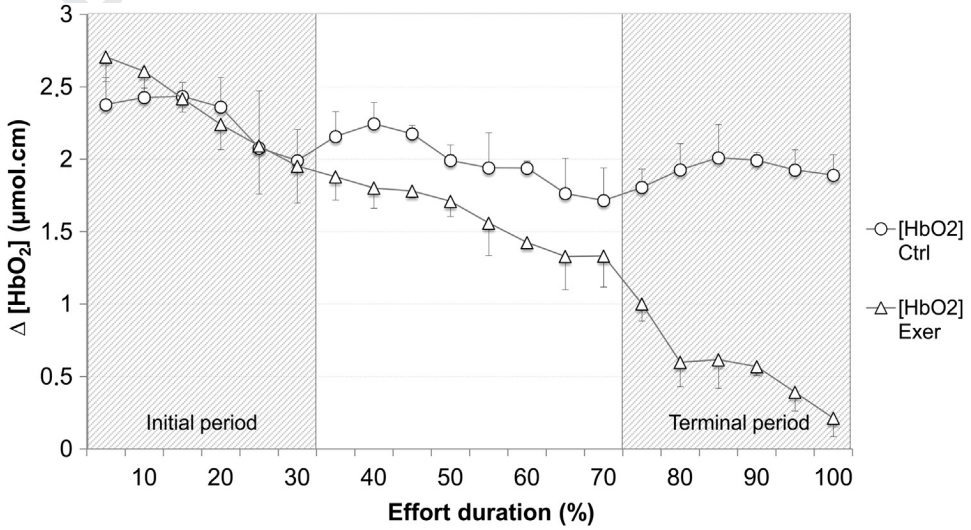

Fig. 7. Changes from resting values (baseline) in cerebral oxyhemoglobin [HbO_2] during control (Ctrl, circles) and exercise (Exer, triangles) conditions. Error bars represent standard deviation.

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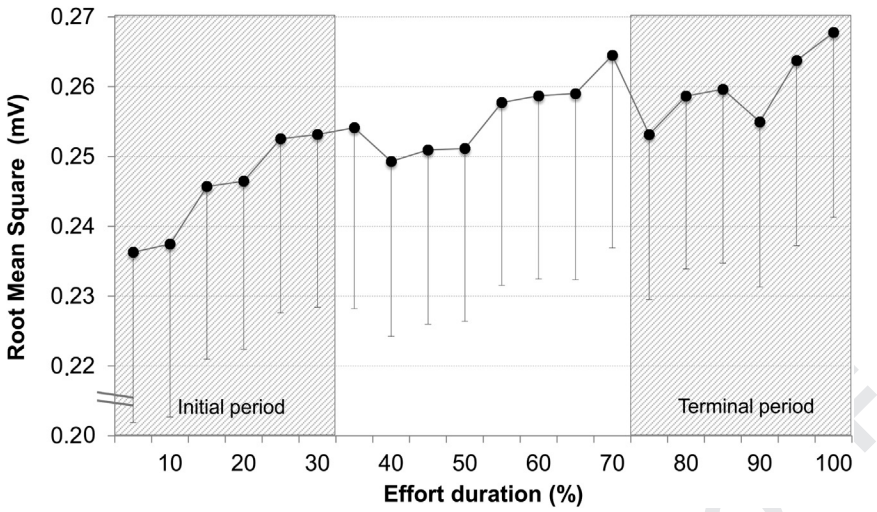

Fig. 8. Electromyographic root mean square (in mV) activity of the *vastus lateralis* muscle during the time-to-exhaustion cycling test. Error bars represent standard errors.

($F(1,14)=9.16$, $p<.001$, $\eta_p^2=.40$)[2]. In the terminal period, Cox was lower while exercising than in the control condition (1.88 ± 0.1 μmol cm, $F(1,14)=5.64$, $p=.03$, $\eta_p^2=.30$).

[HbO_2] levels recorded per condition and period negatively correlated with error rate ($r=-.40$; $p=.001$) and, more specifically, with error rate on IN trials ($r=-.39$; $p<.01$). [HbO_2] levels also negatively correlated with partial errors both on CO ($r=-.38$; $p<.01$) and IN trials ($r=-.26$; $p<.05$). Mean RT ($r=.04$; $p=.79$) and the correction rate ($r=.17$; $p=.19$) were not associated to changes in [HbO_2] levels.

3.9. *Vastus lateralis* activation

A significant main effect of time was observed on the VL activity ($F(1,14)=3.92$, $p<.001$, $\eta_p^2=.23$), which illustrates an increased activation of the muscle throughout exercise duration (Fig. 8). Furthermore, it is interesting to note that the RMS values are significantly and negatively correlated with exercise [HbO_2] ($r=-.23$; $p<.05$).

4. Discussion

This study aimed at investigating concomitant changes in cognitive control and Cox in the prefrontal area during strenuous exercise performed until exhaustion. In an initial period, Cox recorded from the rIFC remained unchanged by intense exercise. Also, the more pronounced drop-off of the delta curve suggested that the cognitive processes, underpinning selective response inhibition, are fully efficient in the first part of the exercise bout. Throughout intense exercise and until exhaustion Cox linearly decreased but without falling below baseline values. In the terminal period, no sign of deficit in selective response inhibition was observed. However, individual's susceptibility to making fast impulsive errors increased and less efficient online correction of incorrect activation was observed just before exhaustion. The main findings of this study are that (i) cognitive functioning evolves during exercise, (ii) intense exercise does not systematically impair cognitive performances, (iii) selective response inhibition efficiency and PFC Cox do not follow a similar dynamic, (iv) the propensity to commit impulsive errors increases and online correction of incorrect activation are disrupted near exhaustion, and (v) Cox patterns suggest a decline in hyperfrontality instead of a hypofrontality.

4.1. *Intense exercise and cognitive performance*

The experimental as well as theoretical literature on the cognitive effect of intense exercise converges towards an impairment of cognitive performances (Ando et al., 2005; Chmura et al., 1994; Chmura and Nazar, 2010; Cooper, 1973; Dietrich and Audiffren, 2011; McMorris et al., 2008; Yerkes and Dodson, 1908). By investigating changes in cognitive functioning through distributional analyzes, the present study highlights a facilitating effect of intense exercise on cognitive control. This effect was localized in the initial part of the exercise bout. Both RT performances and selective response inhibition were indicative of this improvement. More precisely, the exercise-related speeding effect focused on the first two quartiles of the RT distribution, *i.e.* the fastest RT. This does not appear surprising since faster RT have been consistently reported from several meta-analyzes and integrative reviews (Brisswalter et al., 2002; McMorris and Hale, 2012, Tomporowski, 2003). In contrast, the benefit of intense exercise on selective response inhibition constitutes an innovative result. Concretely, the steep negative slope of the delta plots indicates an exercise-related lower interference effect compared to rest. The activation-suppression model of Ridderinkhof (2002) proposes that such pronounced leveling-off in the delta curve is indicative of a greater ability to suppress the automatic response generated by task-irrelevant aspect of the stimulus. The fact that the correction rate remained constant during the initial part of the exercise suggests that online control mechanisms, involved in the correction of incorrect activation, are fully efficient. Together, these findings reveal that the facilitating effect usually reported during moderate exercise can also occur in the first moments of intense exercise.

A cognitive facilitation during intense exercise is not in discrepancy with the main theories on exercise and cognition. Specifically, the catecholamine theory predicts cognitive functioning impairment above the "catecholamine threshold" *i.e.* a pivotal point into the exercise-induced increase in adrenaline, noradrenaline and dopamine (Cooper, 1973; McMorris et al., 2008). During steady exercise, since the level of monoamines increases over time regardless the intensity (Chmura et al., 1997), a certain amount of time is necessary before overreaching this threshold. Accordingly, the first stages of our exercise bout may have spared

[2] During exercise, the same pattern was observed in total hemoglobin ([HbO_2]+[HHb]) which is considered another index of neural activity (e.g., Perrey, 2008). Total hemoglobin decreased from 5.49 ± 0.15 μmol cm in the initial period to 0.69 ± 0.36 μmol cm in the terminal period ($F(1,14)=28.35$, $p<.001$, $\eta_p^2=.67$).

central processes from neural noise, while simultaneously arousing central nervous system and benefiting RT. The transient hypofrontality theory (Dietrich, 2003; Dietrich and Audiffren, 2011) purports another perspective, in which exercise leads to rearrangement of neural resources within the brain. Given the limited cerebral resources, exercise would lead to their redistribution from the brain regions that are not directly involved in the management of exercise such as the PFC to motor areas. Our fatiguing exercise required an increasing magnitude of motor cortex output to increase the firing rate of motor neurons in order to compensate muscle fiber fatigue, as supported by the recorded increase in RMS value of the VL muscle. Instead of exercise intensity *per se*, maintaining the steady power output over time may thus have progressively acted in favor of a PFC down-regulation.

4.2. Exhausting exercise and cognitive performance

Based on the distribution-analytical technique and the delta plot analysis, the present results show that, when exhaustion was about to occur, selective inhibition processes remained unaltered and were thus not responsible for inferior behavioral performances. The delta curve indeed highlights that the selective inhibition is as efficient as in the control condition. Nevertheless, the number of incorrect response activations – including both overt and partials EMG errors – increased. The analyzes of the percentage of correct responses (CAF) showed that, when exhaustion was about to occur, the individual's susceptibility to produce incorrect responses increases. In other words, exhaustion state increases the strength of the automatic response capture activated by irrelevant information and more overt errors are committed.

Interestingly, the recording of the EMG of the FPB muscle involved in the cognitive task allowed us to identify partial error trials *i.e.* incorrect action impulses that were detected and successfully corrected. Accordingly, partial errors provide a direct measure of the effectiveness of the control mechanism involved in the suppression of the activation of an incorrect response. The present results showed that, in the terminal period, participants were less capable to correct incorrect action impulses during exercise than in the control condition and more overt errors were committed. This finding suggests that, near exhaustion, the online correction mechanism is disrupted and the nervous system seems not able to overcome the incorrect activation and provide the correct action. Since the participants aimed at pursuing exercise as long as they could, it is possible that, near exhaustion, motor task-related regulations (cardio-respiratory, velocity, coordination, power output) became such imperative that they were managed in priority at the expense of cognitive task-related regulations (see Section 4.4).

By dissociating our exhausting exercise into analysis periods, we also investigate a new perspective of exercise-cognition studies. This perspective, actually based on a fatigue-induced reorganization, may help to clarify current inconsistencies in the literature. Concretely, it rationalizes why a sustained cognitive solicitation during last stages of a 65-min exercise bout may reveal diminished performance (Dietrich and Sparling, 2004) while 40 min of an intermittent assessment does not (Lambourne et al., 2010), in spite of similar moderate intensities and cognitive tasks. According to this proposal, the impaired cognitive performance associated with intense exercise do not appear illogical. Indeed, fatigue development is obviously quicker at such higher intensities. Considering this accumulation of fatigue, other moderators should also be taken into account. Fitness level, for example, may explain the better (Chang et al., 2012) and steadier (Labelle et al., 2012) cognitive performance of trained participants. Beyond this, we would like to encourage the general idea of temporal differentiation within data sets to better understand integrated fatigue development.

4.3. Cerebral oxygenation and cognitive performance

In the present study, Cox recorded during exercise from the rIFC was at first at a similarly elevated level as in the control condition before linearly declining until exhaustion. This type of $[HbO_2]$ decrease is common during exhausting exercise and in accordance with previous reports from PFC NIRS-monitoring studies (for details see Ekkekakis, 2009).

More particularly, we found that the Cox level was reduced during the terminal period of exercise compared to the control condition where the cognitive task was conducted at rest. In spite of this decline, we observed that the implementation of selective response inhibition remained fully efficient (comparable to the score of the control condition). This is intriguing since rIFC activity is an important component in inhibition processes (Aron et al., 2004) and a debilitative cognitive effect might thus be expected from its down-regulation. This report is not isolated though. A recent study observed a similar discrepancy: cognitive performance improved at moderate intensity (60% $\dot{V}O_2$) in the absence of any changes in Cox values (Ando et al., 2011). This might lead to the suggestion that an uncoupling of Cox level in PFC areas and corresponding cognitive processes may be happening. This type of uncoupling would not ineluctably hamper, but could maintain PFC functionality. As an explanation, the PFC may preserve its metabolic activity by increasing oxygen extraction from arterial vessels to compensate for reduced perfusion (Nybo and Secher, 2004). It is also possible that the Eriksen flanker task was not demanding enough to elicit observable behavioral effects from reduced Cox level.

Exercise-induced hyperventilation is considered to be the main mechanism for the lowering of cerebral blood flow and, in turn, Cox level (Ogoh and Ainslie, 2009). In our study, ventilatory muscle fatigue may have led to this progressive drift into ventilation and hypocapnia. In spite of this process, the $[HbO_2]$ concentration never reached values lower than baseline (*i.e.* a state that could be characterized as hypofrontal). Since $[HbO_2]$ level consistently remained positive, our Cox pattern rather supports the decline of a hyperfrontality state. This contrasts with the reticular-activating hypofrontality theory (Dietrich and Audiffren, 2011) but not with some of its principles. Specifically, when viewed in light of the redistribution of cerebral resources, some findings may be considered as a support to the theory. Indeed, one possible explanation is that PFC was progressively inhibited as a side-effect of fatigue development to favor activity in motor areas, as supported by the Cox-RMS correlation. In this case, both the correlations between $[HbO_2]$ and error rates, between $[HbO_2]$ and partial error rates, and CAF results near exhaustion support the hypothesis of a reallocation, since impulsive errors relate to activity of the pre-supplementary motor area (Forstmann et al., 2008).

4.4. Rationalize the relation between exercise and cognitive performance

Our results reinforce the idea of an interaction between exercise and cognition for the complete duration of an exercise bout. This interference has previously been proposed using strength (Lorist et al., 2002; Schmidt et al., 2009) and aerobic exercises (Marcora et al., 2009; McCarron et al., 2013). Accordingly, we assume that behavioral performance relative to cognitive tasks is punctual and systemic and depends on the constraints supported by the subject at a given time. This idea of a dynamical cognitive control is supported from several perspectives.

Marcora (2008, 2009) proposes a psychobiological model of Q7

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exercise, within which the anterior cingulate cortex (ACC) appears as the keystone of both exercise and cognitive parameters. ACC is known to be involved in cognitive functioning (Carter et al., 1998), the pain matrix (Peyron et al., 2000), perceived effort (Williamson et al., 2006) and effort-related decision-making. Regarding our protocol and its demands, a hyper-solicitation of the ACC over time may compromise its efficacy to deal with interfering stimuli.

The insular cortex and hypothalamus are other brain areas that are increasingly activated with fatigue development (Meyniel et al., 2013). In response to exercise duration and increasing body afferences, it is possible that these regions act to reduce basal ganglia activation. Such inhibition would prevent the subject from experiencing untolerable perceived effort or any excessive homeostasis disruption, but would be enforced at the expense of the overall performance. Indeed, basal ganglia (specifically the ventral striatum) activation determines both cognitive and motor efforts (Schmidt et al., 2012). Near exhaustion the subject may thus, voluntarily or not, opt for a facilitating strategy leading him to progressively act on the basis of impulsive activations rather than on the basis of high-order processes.

The neuro-hormonale rationale of the "catecholamines hypothesis" may also determine the way participants respond to a cognitive task during exercise (McMorris et al., 2009). Due to the role of monoamines in glycolysis, lipolysis and cardio-respiratory regulation (Borer, 2003), sustained exercise induces increases in adrenaline, noradrenaline and dopamine irrespective of its intensity (Chmura et al., 1997). Such accumulation may progressively lead to overreach the "catecholamine threshold" that would induce neural noise and contributes to the cessation of exercise-induced cognitive facilitation.

5. Conclusion

In conclusion, this study is innovative in that changes in cognitive performances during a steady exercise were characterized. The benefit of intense exercise on selective response inhibition constitutes an original result. Moreover, the use of the distribution-analytical technique highlighted that, when exhaustion was about to occur, selective inhibition processes remained unaltered. Despite this, individual's susceptibility to making fast impulsive errors increased and less efficient online correction of incorrect activation was observed, suggesting that the online correction mechanism is disrupted. Interestingly, the dynamical pattern of selective response inhibition efficiency did not follow the same pattern as [HbO_2], letting Cox-related explanations of cognitive functioning during exercise uncertain. These results reinforce the idea of a complex interaction between exercise and cognition and include fatigue stressors as a determinant component into cognitive performances.

Declaration of interest

The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.

Uncited references

Q8

Chmura et al. (1998), Davranche and Pichon (2005), Meeusen and De Meirleir (1995), Subudhi et al. (2007), Yamashita et al. (2001)

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