



Influence of cerebral and muscle oxygenation on repeated-sprint ability

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**Abstract**

The study examined the influence of cerebral (prefrontal cortex) and muscle (vastus lateralis) oxygenation on the ability to perform repeated, cycling sprints. Thirteen team-sport athletes performed ten, 10-s sprints (with 30 s of rest) under normoxic ($F_{I}O_2$ 0.21) and acute hypoxic ($F_{I}O_2$ 0.13) conditions in a randomised, single-blind fashion and crossover design. Mechanical work was calculated and arterial O_2 saturation (S_pO_2) was estimated via pulse oximetry for every sprint. Cerebral and muscle oxy- (O_2Hb) , deoxy- (HHb) , and total haemoglobin (THb) were monitored continuously by near-infrared spectroscopy. Compared with normoxia, hypoxia induced larger decrements in S_pO_2 and work (11.6% and 7.6%, respectively; $P<0.05$). In the muscle, we observed a fairly constant level of deoxygenation across sprints, with no effect of the condition. In normoxia, regional cerebral oxygenation increased during the first two sprints and slightly fluctuated thereafter. In contrast, this initial cerebral hyper-oxygenation was attenuated in hypoxia. Changes in $[O_2Hb]$ and $[HHb]$ occurred earlier and were larger in hypoxia compared with normoxia ($P<0.05$), while regional blood volume ($\Delta[THb]$) remained unaffected by the condition. Changes in cerebral $[HHb]$ and mechanical work were strongly correlated in normoxia and hypoxia ($R^2=0.81$ and $R^2=0.85$, respectively; $P<0.05$), although the slope of this relationship differed (normoxia: -351.3 ± 183.3 vs. hypoxia: -442.4 ± 227.2 ; $P<0.05$). The results of this NIRS study show that O_2 availability influences oxygenation of the prefrontal cortex during repeated, short sprints. By using a hypoxia paradigm, the study suggests that cerebral oxygenation may impose a limitation to repeated-sprint ability.

Key words: intermittent sprints, brain oxygenation, NIRS, hypoxia, altitude



Introduction

Support for the role of a failure of the central nervous system (CNS) to excite the motor neurons adequately (i.e., central fatigue) in fatigue during tests of repeated-sprint ability (RSA) has been provided by the finding that voluntary activation of skeletal muscles (accessed via the twitch-interpolation technique) is reduced after ten 6-s sprints separated by 30 s of rest (27). This suboptimal muscle activation has also been functionally observed via lowered surface electromyographic (EMG) activity on several occasions during repeated sprints (6, 24). However, what triggers these acute changes in the CNS behaviour remains to be determined.

Central fatigue may be elicited by low brain oxygenation, i.e., by insufficient O₂ delivery and/or low pressure gradient to drive the diffusion of O₂ from the capillaries to the mitochondria. Direct and indirect evidence supports the contention that inadequate cerebral oxygenation depresses cortical neuron excitability, although the mechanisms remain debated (for review see (1, 2, 26). The non-invasive technique of near-infrared spectroscopy (NIRS) offers real-time measurement of oxygenation and haemodynamics in tissues (35), and thus, constitutes a relevant tool to enhance our current knowledge of central (CNS) and peripheral (muscle) determinants of RSA.

Some studies have reported that muscle deoxygenation occurs during cycling and running RSA tests (8, 27). However, exercises of this nature appear to induce a fairly constant level of deoxygenation in prime mover muscles across sprints, and therefore authors have suggested that muscle O₂ uptake is well preserved and is not likely to represent a limiting factor of RSA. Data on cerebral oxygenation changes during RSA tests are currently inexistent. Based on studies conducted during constant workload exercise (3), incremental test to maximal effort (32), and supramaximal exercise (25, 29, 30), the deoxygenation of the cerebral cortex has, in general, been incriminated in the cessation of exercise, or at least the reduction of exercise intensity. This finding, however, is confounded by the availability of O₂ (3, 32). Although an association exists between cerebral oxygenation and performance in varied exercises, no studies have yet determined if a critical level of cerebral deoxygenation impairs RSA.

The aim of this study was therefore to use NIRS to monitor changes in both central and peripheral oxygenation during a RSA test, in order to gain further understanding of the factors associated with fatigue during such task and how best to improve RSA. This study was the first to monitor simultaneous changes in cerebral and muscle oxygenation under both normoxic and hypoxic conditions. The possibility to blind subjects to manipulations of F_IO₂ represents a relevant experimental approach to investigate the effects of tissue oxygenation on RSA. It was hypothesised that cerebral, but not muscle, deoxygenation would be associated with impairments in RSA.

Materials and methods

Subjects

Thirteen, male soccer and rugby players were recruited from university and local sports clubs (mean \pm SD: 23.6 \pm 3.7 y, 181.5 \pm 5.5 cm, 81.5 \pm 11.3 kg, 13.6 \pm 1.2 % body fat). These subjects were chosen because they were accustomed to high-intensity exercise and familiar with laboratory testing. All subjects were healthy and with no known neurological or cardiovascular diseases. The study was conducted with the ethical approval of the Human Subject Research Committee of the University of Lethbridge. Before the trials, all subjects were informed of the nature of the investigation, after which they gave written informed consent.

Experimental design



Athletes visited the laboratory three times. During the first visit, anthropometric measurements (stature, body mass, and body fat percentage) were recorded, and athletes were re-accustomed with sprint cycling (five, 5-s sprints) until fully confident of producing an all-out effort from a stationary start.

Three days following the familiarisation session, subjects were randomised in a single-blind, cross-over design and asked to perform a RSA test under normoxic ($F_{I}O_2$ 0.21) and acute hypoxic ($F_{I}O_2$ 0.13) conditions. Trials were conducted at the same time of day and were separated by one week.

Exercise testing

All testing was performed on a friction-loaded cycle ergometer (Monark 874E, Stockholm, Sweden) with a braking, resistive force applied on the flywheel set at $0.9 \text{ N}\cdot\text{kg}^{-1}$ of body mass. The instantaneous power output, corrected for flywheel acceleration, was recorded at 50 Hz, and the mechanical work performed (kJ) was calculated by integrating the power curve over the 10 s of the sprint for every sprint. Subjects were instrumented with necessary probes and sensors, and were then asked to close their eyes, eliminate extraneous thoughts, and rest completely in the exercising position on the ergometer to obtain 2 min of baseline measurements. Immediately after the baseline period, subjects were equipped with the breathing apparatus, and a 10-min exposure period to the gas was observed while seating on the ergometer (10 min was enough to reach a steady state in every subject). After a 5-min warm-up at 60–70 watts, subjects rested for another 1 min, and the RSA test (10 x 10-s sprints with 30 s of rest) was initiated. Sprints were initiated with the subject's dominant leg, with the crank arm located 45 deg forward to the vertical axis. Subjects were asked to remain seated during every sprint and during the recovery periods. Following the test, all instrumentation was removed and subjects performed a self-paced cool down under ambient condition.

Humidified, experimental gases (normoxia = compressed air; hypoxia = 13% O_2 , 87% N_2) were administered using a system of plastic tubing and 150-liter Douglas bag reservoir, with the O_2 - N_2 dilution constantly controlled by a PO_2 probe (Hypersphere Pro, AltitudeTech Inc., Kingston, ON, Canada).

Responses to exercise

Arterial oxygen saturation (S_pO_2). S_pO_2 was estimated via pulse oximetry (Nellcor N-200, Nellcor Inc., Hayward, CA) with adhesive optodes placed on the forehead. This technique has been shown to be in good agreement with haemoglobin O_2 saturation based on arterial blood analysis (28), and has been used elsewhere (3, 6, 28). SpO_2 was recorded during baseline, exposure, and immediately after every sprint.

NIRS measurements and analysis. During all tests, subjects were instrumented with two pairs of NIRS probes to monitor absorption of light across cerebral and muscle tissue (Oxymon MKIII, Artinis, The Netherlands). The theory, limitations and reliability of measurement obtained with this device during exercise are detailed elsewhere (32, 35). One NIRS emitter and detector pair was placed over the left prefrontal lobe, between Fp1 and F3 (international EEG 10-20 system), and placement was further adjusted (less than 5 mm) to obtain strong signal strength on every subject. Spacing between optodes was fixed at 5 cm using a black, plastic spacer held in place via double-sided, stick disks and a black, tensioning headband to reduce the intrusion of extraneous light and the loss of transmitted NIR light from the field of investigation. It has

previously been shown that an emitter–detector distance of 5 cm increases spatial resolution, allowing a light penetration depth of ~2.3–2.5 cm (18). It has also been reported that extracranial contribution to the NIRS signal is negligible when the inter-optode distance is > 4.5 cm (31).

A second NIRS emitter and detector pair was fixed on the distal part of the left vastus lateralis muscle belly (approximately 15 cm above the proximal border of the patella) using a black, plastic spacer with optode distance of 4.5 cm. Skinfold thickness was measured between the emitter and detector using a skinfold calliper (Harpندن Ltd.) to account for the skin and adipose tissue thickness covering the muscle (23). Probes were secured to the skin using double-sided, stick disks and shielded from light using black, elastic bandages.

A modified form of the Beer-Lambert Law was used to calculate micromolar changes in tissue [O₂Hb] and [HHb] across time using received optical densities from two continuous wavelengths of NIR light (763 and 855 nm). An age-dependent differential optical pathlength factor for cerebral cortex (15, 29, 30) and of 4.95 for muscle (16, 32) were used in this study. Changes in total Hb ([THb]) were calculated by the sum of [O₂Hb] and [HHb] and used as an index of change in regional blood volume within the illuminated area (35). When regional blood volume (i.e., Δ [THb]) is constant, [O₂Hb] and [HHb] exist in equilibrium. Thus, decreases in [O₂Hb] and increases in [HHb] reflect relative deoxygenation in the underlying tissue (18, 21, 32).

NIRS data were acquired at 10 Hz and transferred online from the Oxymon MKIII to a PC (Figure 1). Data were averaged over the last 5 s within every sprint to obtain one value per sprint, and normalised to express the magnitude of changes from the baseline period (arbitrarily defined as 0 μ M) (29, 30, 32, 33).

Fig. 1

Statistical analysis

Statistical analyses were performed using Statistica 5.5 for Windows (Statistica, Statsoft Inc., Tulsa, OK). Two-way, repeated measures, analyses of variance (ANOVAs) (sprint x condition) were used to compare the following dependent variables between normoxia and hypoxia across sprints: mechanical work, S_pO₂, Δ [O₂Hb], Δ [HHb], and Δ [THb]. One-way ANOVAs (condition) were used to compare the slope of the relationships between mechanical work and Δ [HHb] and Δ [HHb] between normoxia and hypoxia. Tuckey's HSD *post-hoc* analyses were used to locate differences among pairs of means when ANOVAs revealed a significant *F* ratio for main or interactive effects. Pearson's product–moment coefficients were used to determine relationships. The level of significance was set at $P < 0.05$. Data are reported as mean \pm SD.

Results

Mechanical measurements

The mechanical work values recorded during the RSA tests are displayed in Fig. 2. No effect of the condition was observed for the initial-sprint score (normoxia: 8.6 \pm 1.1 kJ vs. hypoxia: 8.4 \pm 1.1 kJ; $P > 0.05$). When compared with the first sprint of the series, there was a significant decline in work (both conditions compounded) in sprints 2–10 (overall decrement: 31.2%, $P < 0.05$). We also noted a significant main effect of condition on the work performed (normoxia: 67.2 \pm 5.5 vs. hypoxia: 62.1 \pm 5.4 kJ; $P < 0.05$). No significant interaction sprint x condition was observed for this parameter.

Fig. 2



Arterial O₂ saturation

Mean data at rest, exposure, and over the sprints are displayed in Fig. 3. At rest, there was little variation among subjects and conditions (average: $98.6 \pm 0.9\%$) with all participants within the normal range. Compared with resting baseline, S_pO₂ (all conditions compounded) fell significantly in sprints 1–10 (overall decrement: 14.5%, $P < 0.05$). This parameter was profoundly affected by the condition, with lower values recorded in hypoxia than in normoxia (average: 12.1%, $P < 0.05$). Furthermore, the pattern of desaturation across sprints varied between conditions, with significant decrements occurring in sprints 2–10 (overall decrement: 8.3%, $P < 0.05$) in normoxia, and in sprints 1–10 (overall decrement: 20.7%, $P < 0.05$) in hypoxia.

Fig. 3

NIRS measurements

Fig. 4 displays the average concentration changes in cerebral and muscle NIRS signals across sprints during normoxia and hypoxia.

Muscle analysis. In normoxia, regional muscle oxygenation decreased rapidly during the first sprint ($\downarrow \Delta[\text{O}_2\text{Hb}]$, $\uparrow \Delta[\text{HHb}]$), and this was accompanied by a reduction in regional blood volume ($\downarrow \Delta[\text{THb}]$). From sprints 2–10, $\Delta[\text{HHb}]$ remained unchanged, yet $\Delta[\text{O}_2\text{Hb}]$ and $\Delta[\text{THb}]$ rose significantly, indicating a reduction in the rate of muscle deoxygenation in subsequent sprints. In hypoxia, regional muscle oxygenation followed a very similar pattern as seen in normoxia (interaction sprint x condition: $P > 0.05$). The magnitude of muscle deoxygenation ($\downarrow \Delta[\text{O}_2\text{Hb}]$, $\uparrow \Delta[\text{HHb}]$) was the same in both conditions (main effect of condition: $P > 0.05$).

Cerebral analysis. In normoxia, regional cerebral oxygenation increased rapidly in sprints 1–2 ($\uparrow \Delta[\text{O}_2\text{Hb}]$, $\leftrightarrow \Delta[\text{HHb}]$, and $\uparrow \Delta[\text{THb}]$; Table 1), indicating cerebral vasodilation during initial sprints. Thereafter, NIRS signals fluctuated without showing clear evidence of deoxygenation between sprints 3 and 10 (from \leftrightarrow to $\uparrow \Delta[\text{O}_2\text{Hb}]$ and $\uparrow \Delta[\text{HHb}]$), while regional blood volume displayed a slight, although not significant, increase from sprint 1 to 10 ($P = 0.21$). In contrast, regional cerebral oxygenation was markedly attenuated during sprint 1 in the hypoxic trial ($\leftrightarrow \Delta[\text{O}_2\text{Hb}]$, $\uparrow \Delta[\text{HHb}]$, and $\uparrow \Delta[\text{THb}]$), and decreased progressively across subsequent sprints ($\leftrightarrow \Delta[\text{O}_2\text{Hb}]$, $\uparrow \Delta[\text{HHb}]$), whereas regional blood volume did not show a significant alteration ($P = 0.21$). Changes in $\Delta[\text{O}_2\text{Hb}]$ and $\Delta[\text{HHb}]$ were larger (main effect of condition) in hypoxia compared with normoxia throughout the RSA test (average: 118.5% and 45.1%, respectively; $P < 0.05$; Table 1). Between normoxia and hypoxia, there was no change in $\Delta[\text{THb}]$ across the sprints (main effect of condition: $P = 0.58$).

Fig. 4 and Table 1

Discussion

The major finding in this study was that acute hypoxia induced by a blinded change in F₁O₂ profoundly affected the pattern of prefrontal cortex oxygenation during a RSA test. Results demonstrated for the first time that repeated, short sprints interspersed with incomplete recovery intervals elicited an earlier and larger degree of cerebral deoxygenation in hypoxia compared with normoxia. These findings suggest that changes in cerebral, unlike muscle, oxygenations appear to limit RSA in team-sport athletes under hypoxic conditions, since hypoxia-induced performance decrements were not accompanied by an aggravated muscle deoxygenation.



Technical considerations

We acknowledge that prefrontal cortex oxygenation is a regional measurement that may not be reflective of global cerebral oxygenation. However, the deoxygenation that occurs during whole-body, strenuous exercise is not confined to the prefrontal cortex but seems widespread to motor areas as well (33). Also, while this area of the brain is not directly involved in the neural control of movement as motor areas do, the deoxygenation of the prefrontal cortex has been purported to contribute to fatigue on different occasions (3, 29, 30, 32). Finally, since the differential pathlength factors were only estimated, it must be reminded that absolute NIRS measurements and actual tissue oxygenation values remain unknown.

As far as the muscle tissue is concerned, no post-exercise, vascular occlusion was performed in the current study because Chance et al. (11) reported little additional deoxygenation (< 2%) during supra-systolic cuff occlusion of the vastus lateralis muscle immediately following strenuous exercise. We also chose not to perform arterial occlusion to stay consistent with previous studies that have investigated muscle oxygenation trends during RSA tests (8, 27). Nevertheless, if a leg cuff ischemia technique had allowed obtaining a low-oxygenation reference point it would have been similar in both conditions, and thus, would not have altered our conclusions.

Hypoxia and Repeated Sprint Ability

The ability to generate maximal power during isolated, all-out exercise is preserved in acute hypoxia (9) owing to an enhancement of the anaerobic energy supply. The current results are in good agreement with this finding since the initial-sprint performance was not affected by hypoxia. In the hypoxic trial, every subject was able to reproduce the initial mechanical score achieved during the trial performed in normoxia although S_pO_2 was significantly lowered. In contrast, and in accord with previous research on RSA (4), the ability to reproduce total mechanical work in subsequent sprints was impaired in the current hypoxic conditions (Fig. 2) after we induced large changes in S_pO_2 by the experimental treatment (Fig. 3). Therefore, our results confirm that a reduction in O_2 availability impairs RSA (4), and further strengthen the recent finding that arterial O_2 desaturation may be considered as a limiting factor of RSA in team-sport athletes (6). This is also in keeping with the hypothesis that the subsequent, potential mismatch between O_2 delivery and requirement explains part of the progressive reduction in the absolute contribution from aerobic sources and mechanical output observed during such exercise (4, 19).

In this perspective, it is worth mentioning that a correlation between the initial mechanical score and performance decrement over subsequent sprints has consistently been reported and demonstrated as a confounding factor when investigating the mechanisms underlying RSA impairment (7, 24). Since the initial-sprint performance was similar in normoxia and hypoxia in the current study, it is likely that the lower RSA observed in hypoxia has been elicited by the decrease in S_pO_2 , i.e., by O_2 -dependent mechanisms of fatigue.

Muscle oxygenation and Repeated Sprint Ability

Muscle oxygenation patterns have been described during cycle- and run-based RSA tests (8, 27). In agreement with these reports, our data showed that under normoxic conditions muscle oxygenation decreased rapidly at the beginning of the exercise and plateaued across sprints. Since NIRS measurements have been correlated with changes in intracellular O_2 tension (34) and



venous O₂ saturation (17), a plateau in oxygenation may be interpreted as evidence of maximal O₂ extraction (17, 34). A unique feature of the current study was to use a hypoxia paradigm to exacerbate the reduction in O₂ availability (as shown in Fig. 3), and thereby test directly the influence of muscle oxygenation on RSA. Our results showed that patterns of change and the magnitude of tissue deoxygenation were nearly identical in normoxia and hypoxia (Fig. 4). This indicates no additional deoxygenation in the quadriceps muscle during the hypoxic trial compared with the normoxic trial, which demonstrates that muscle O₂ uptake was maintained in acute hypoxia despite a reduced O₂ availability. Interestingly, by comparing whole-body (cycling) with small-muscle (one-leg, knee extension) exercises, Calbet and colleagues (10) have demonstrated that the main mechanism limiting performance in hypoxia appears to be the systemic delivery of O₂, whereas muscle O₂ diffusing capacity (and therefore muscle O₂ uptake) may only have a secondary role. The current findings suggest that the level of muscle oxygenation is not likely to limit RSA, as previously hypothesised (8, 27), and highlight that the hypoxia-induced RSA decrement in the current hypoxic conditions was caused by factors other than purely the muscle aerobiosis.

Our assessment is primarily based upon muscle Δ [HHb] because it is shown to be essentially independent of changes in blood volume during exercise (14, 21). Δ [HHb] is therefore a reliable estimator of changes in intramuscular oxygenation and O₂ extraction in the area investigated (13). Our results showed, however, that regional blood volume (given by Δ [THb]) fluctuated across sprints, as previously reported (27), which may raise a concern about the current physiological conclusions. That being said, because subjects exhibited very similar patterns and magnitudes of change for every NIRS parameter under normoxic and hypoxic conditions, our conclusion would be unaffected: these observed levels of tissue deoxygenation would not be directly responsible for RSA impairments. Although an association exists between O₂ availability and RSA (6), other avenues for potential physiological mechanisms must be considered, which include O₂-dependent CNS fatigue (1, 2, 26, 32).

Cerebral oxygenation and Repeated Sprint Ability

We observed that regional cerebral oxygenation and blood volume were adequately maintained during ten, 10-s sprints performed in normoxia and interspersed with 30 s of rest (Fig. 4 and Table 1). In contrast, Shibuya and colleagues (30) reported a progressive cerebral deoxygenation during intermittent exercises. Specifically, these authors observed a reduction in Δ [O₂Hb] and Δ [THb], while Δ [HHb] increased, over the course of seven, 30-s cycling exercises performed at an intensity corresponding to 150%VO₂max and interspersed with 15-s of rest. It was thus concluded that fatigue resulting from such intermittent, supramaximal efforts was related to a decrease in the cerebral oxygenation level (30). At first glance, the results of the current study and that of Shibuya et al. (30) may appear contradictory, but could be explained by major differences in protocols such as duration and intensity.

Here, we questioned whether a reduction in cerebral oxygenation influences RSA, which is a major fitness requirement for team-sport athletes (7, 24). To test this hypothesis, we compared the patterns of change in cerebral oxygenation obtained during normoxia and hypoxia. The deoxygenation of the prefrontal cortex occurred earlier and to a larger extent in hypoxia than in normoxia. All subjects exhibited a fall in cerebral oxygenation when performing the hypoxic trial. In addition, the relationship of Δ [HHb] to mechanical work exhibited a strong coefficient of determination in both conditions (Fig. 5A), which suggests a link between the oxygenation of the brain and the ability to perform work at supramaximal intensity (25, 29) and repeatedly (30). The



slope change of this relationship in normoxia vs. hypoxia further suggests that the larger RSA decrement observed in the hypoxic trial was caused, in part, by the larger metabolic disturbances of the cerebral function recorded in this trial. In addition to a lower O_2 availability in hypoxia, hypoxia-induced hyperventilation and subsequent reductions in arterial CO_2 tension may have resulted in cerebral vasoconstriction and diminished cerebral blood flow (25). In addition, the increase in cerebral O_2 uptake coupled with a small decrease in cerebral blood flow has been incriminated in the decrease in cerebral oxygenation near exhaustion during constant work-rate and incremental tests (20, 32). The current results support a similar conclusion for repeated sprints, since the rate of change in regional cerebral blood volume was similar between conditions, whereas the rate of O_2 uptake/extraction was accelerated in hypoxia ($\downarrow \Delta[O_2Hb]$ and $\uparrow \Delta[HHb]$). Overall, these data add to the current knowledge that, during repeated sprints in normoxia, the changes in cerebral oxygenation *per se* may not reach a level low enough to cause the reduction in work. However, larger changes seen in acute hypoxia ($F_I O_2$ 0.13) may become critical to RSA. This subsequently raises the question: how can the lower oxygenation of the brain explain part of the greater RSA decrement in hypoxia?

Insufficient oxygenation of the cerebral cortex has been shown to affect neurotransmitter turnover and depress neuronal electrical activity of the brain (12), which has been incriminated in the occurrence of central fatigue in challenging levels of arterial hypoxaemia (i.e., $S_p O_2$ levels $< 75\%$) (2, 3). However, since the entire RSA test was performed with reduced $F_I O_2$, this likely also accelerated the development of peripheral, limb fatigue (4, 19). Consequently, there is also the possibility that the greater RSA impairment in hypoxia is explained by sensory afferent feedback (groups III/IV muscle afferents), originating in the fatiguing locomotor muscles, to the CNS, which have a powerful input to the regulation of the CNS response in moderate hypoxia (1, 2, 5). Therefore, it is impossible to determine whether the RSA impairment in hypoxia was attributable to a directly-mediated decrease in CNS motor output (i.e., due to reduced cerebral oxygenation) or to inhibitory, peripheral feedback to the CNS from increased disturbances in the fatiguing muscles. Quantifying the relative contributions of these central fatigue-causing mechanisms will advance the current body of knowledge in the area of RSA.

Conclusion

The results of the current study have shown that it is unlikely that changes in cerebral oxygenation limit RSA in normoxia, yet such changes may contribute to performance decrement during repeated-sprint efforts in hypoxia. The consequences of reduced oxygenation of the brain are not only reflected in a debilitated exercise capacity, but also in compromised fine, psychomotor skills and cognitive abilities (2, 22). The implications that these findings have for team-sport performance warrant further investigation, specifically in altitude training and tissue deoxygenation during games at altitude.



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Table 1. NIRS cerebral concentration changes during the RSA test in normoxia and hypoxia.

Variable		Exposure		Sprints									
		1	2	3	4	5	6	7	8	9	10		
Δ [THb]	Normoxia	1.9±4.3	11.4±0.6	9.6±9.0	9.1±8.4	8.0±10.1	8.6±9.7	9.7±9.2	10.8±8.9	12.5±8.6	14.3±7.8	14.6±7.6	
	Hypoxia	2.0±4.2	9.2±5.2	5.8±4.0	4.9±3.6	5.7±4.5	6.6±5.2	9.3±7.2	9.8±7.7	11.8±8.5	12.4±8.2	13.1±10.0	
Δ [O ₂ Hb]	Normoxia	1.6±3.4	9.39±7.7*	6.4±6.5*	4.4±4.8	3.3±6.9	3.5±6.9	3.4±6.1	3.7±5.8	4.6±6.0	6.2±5.1*	5.6±4.5*	
	Hypoxia	-0.9±2.2	2.8±4.8	-1.7±2.3	-3.2±2.8	-3.2±4.4	-2.7±4.9	-1.2±6.0	-1.1±5.8	0.2±6.8	0.2±6.8	1.1±7.9	
Δ [HHb]	Normoxia	0.8±1.2	2.2±4.7	3.3±4.1	4.2±4.1	4.7±4.5*	5.1±4.3*	5.6±3.8*	6.3±3.5*	7.0±3.8*	7.9±2.6*	8.4±3.1*	
	Hypoxia	2.9±2.5*	6.6±1.9*	7.7±3.4*	8.3±3.8*	9.0±3.8*	9.3±4.3*	10.5±4.9*	10.9±5.7*	11.5±5.4*	12.1±5.8*	12.0±6.1*	

Values are micromolar changes from resting baseline ($n = 13$). O₂Hb: oxy-haemoglobin; HHb: deoxy-haemoglobin; THb: total haemoglobin. Brackets indicate concentration. * Different from resting baseline value (interaction sprint x condition: $P < 0.05$).

**Figure captions**

Fig. 1 Representative concentration changes in cerebral oxy-haemoglobin ($[O_2Hb]$), deoxy-haemoglobin ($[HHb]$), and total haemoglobin ($[THb]$) from a single subject during the sprints in normoxia.

Fig. 2 Mechanical work performed during the sprints in normoxia (●) and hypoxia (Δ). There was a decrease in work during the sprints (main effect of sprint: $P < 0.05$); however, decrements were larger in hypoxia than in normoxia (main effect of condition: $P < 0.05$).

Fig. 3 Mean arterial O_2 saturation at baseline (BL), exposure (EXP), and throughout the sprints in normoxia (●) and hypoxia (Δ). There was a decrease in arterial saturation during the sprints (main effect of sprint: $P < 0.05$); however, changes were larger (main effect of condition: $P < 0.05$) and occurred earlier (interaction sprint x condition: $P < 0.05$) in hypoxia than in normoxia.

Fig. 4 Near-infrared spectroscopy concentration changes from resting baseline (BL) during exposure (EXP) and throughout the sprints in normoxia (●) and hypoxia (Δ). O_2Hb : oxy-haemoglobin; HHb : deoxy-haemoglobin; THb : total haemoglobin. Brackets indicate concentration.



Fig. 1

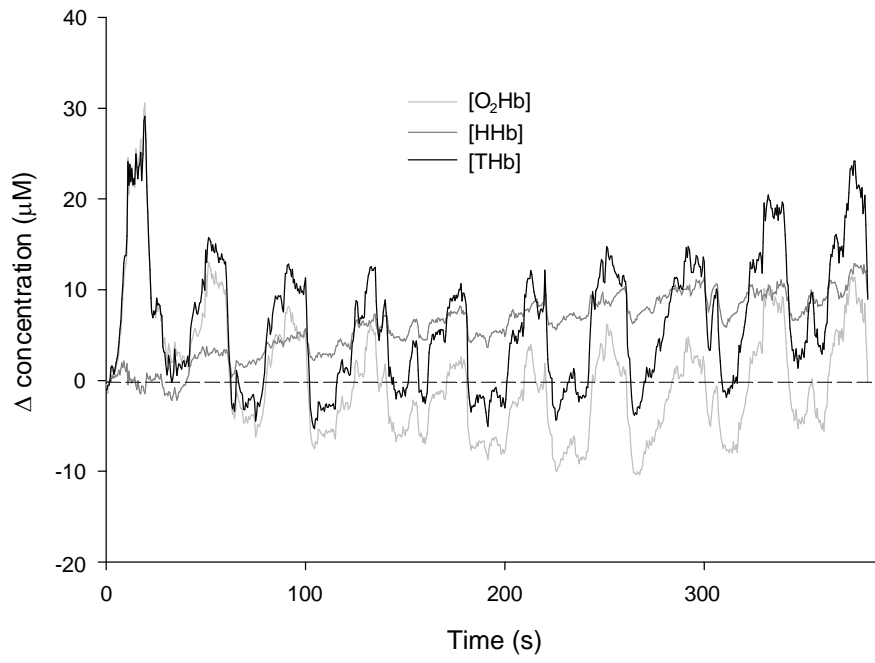




Fig. 2

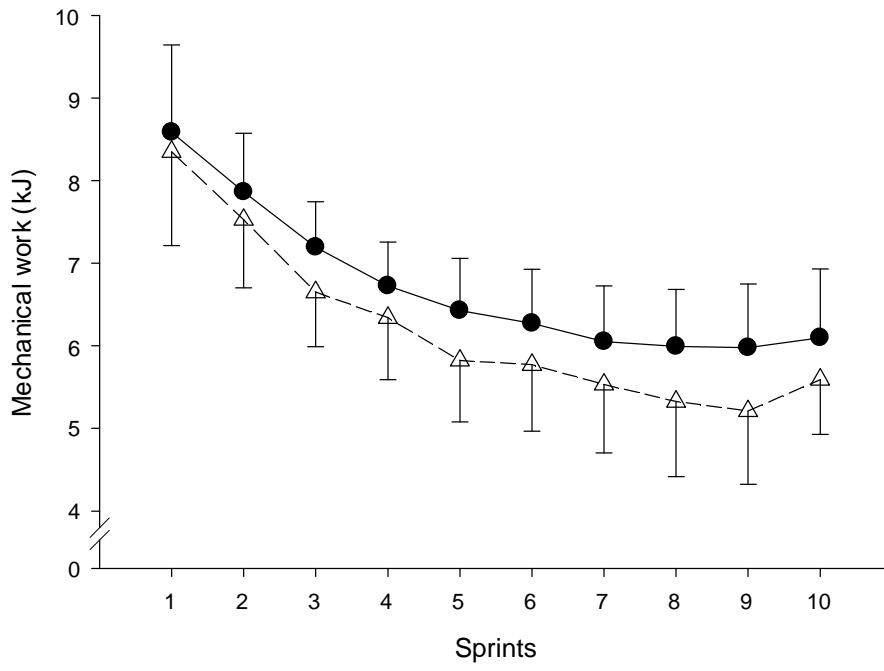




Fig. 3

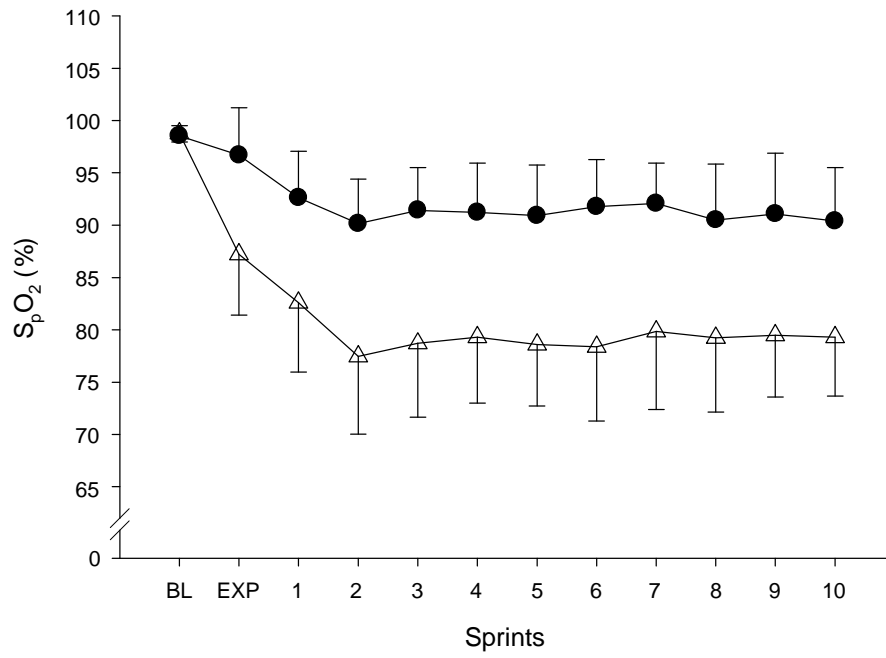




Fig. 4

