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ABSTRACT

Purpose: This study investigates the effects of normobaric hypoxia on repeated sprint exercise (RSE) with different balance between oxidative (phosphocreatine and oxidative pathway) and glycolytic contributions. Therefore, performance and psycho-physiological responses were compared during RSE to exhaustion with the same exercise-to-rest ratio (1:2) but different sprint durations (5, 10 or 20s) either in normoxic (RSN) or hypoxic (RSH; FiO_2 = 0.13) conditions. **Methods:** On separate visits, 10 active participants completed in random order three cycling RSN (5:10; 10:20 and 20:40) and three similar RSH sessions to exhaustion. *Vastus lateralis* muscle oxygenation was recorded by near infrared spectroscopy. Blood lactate concentration, limb and breathing discomfort, and ratings of perceived exertion (RPE) were measured. **Results:** Total sprint number was smaller in hypoxia than in normoxia for 5:10 (20.8±8.6 vs 14.7±3.4; p=0.014) and 10:20 (13.7±6.3 vs 8.8±2.5; p=0.018) but not 20:40 (5.6±1.9 vs 5.6±2.5). The fatigue index was larger in hypoxia only for 5:10 (-43.5%, p<0.001). Irrespective of condition, blood lactate concentration increased with the sprint duration with higher values for 20:40 than 5:10 (13.1±2.7 vs 11.5±2.2 $mmol.l^{-1}$; p=0.027). Limb and breathing discomfort and RPE did not differ in all RSE. Muscle oxygenation was mainly impacted by sprint duration (i.e., main effect of sprint duration on [HHb] min, [tHb] max, Δ[HHb] and Δ[tHb]) but not by hypoxia. The normoxia-to-hypoxia percentage decrease for total sprint number for 5:10 was correlated with the highest power output over 5s (R^2=0.55; p=0.013) and 10s (R^2=0.53; p=0.016). **Conclusions:** Hypoxia impairs repeated sprint ability when the oxidative but not the glycolytic contribution is substantial. The oxidative-glycolytic balance, influenced partly by sprint duration, is key during repeated sprint in hypoxia. **Key Words:** HYPOXIC TRAINING, REPEATED SPRINT ABILITY, INTERMITTENT SPORTS, NEAR INFRARED SPECTROSCOPY, FIBER TYPOLOGY

INTRODUCTION

Repeated sprint ability, which involves 'all-out' efforts interspersed with incomplete recovery periods, is a key performance determinant of intermittent activities (i.e., team-, racket-, or combat- sports). Repeated sprint exercise (RSE) performed in hypoxia (RSH) induces greater performance improvement than in normoxia (1). Improved fatigue resistance following RSH could be explained by hypoxia-induced upregulation of several regulatory mechanisms: i) the modifications at cellular level following the decrease in oxygen (O_2) availability and the increased expression of hypoxia induced factors and target genes (i.e., phenotypic changes in favor of fast-twitch fibers and increase in myoglobin content) (2, 3); ii) the hypoxia-induced compensatory nitric-oxide related vasodilation (4, 5); iii) the behavioral change of fast twitch (FT) fibers which benefit more of increased blood perfusion than slow twitch (ST) fibers (6). Muscle de-/reoxygenation changes have been previously use to non-invasively estimate adaptations (i.e., increased blood volume, estimated from total hemoglobin, during RSH (3)) triggered by RSE in normoxia and hypoxia (7–10). Total hemoglobin also depends on oxygen fractional consumption and on the combined influence of sympatholysis and nitric oxide bioavailability, that impact both muscle perfusion and oxygenation levels.

Both sprint duration and exercise-to-rest ratio likely influence repeated sprint ability, notably in hypoxia (1), so that performance improvement post-RSH may in turn depend on the oxidative-glycolytic balance (11, 12). Indeed, the normoxia-to-hypoxia decrease in mean power output during RSE is influenced by the exercise-to-rest ratio (13). When three sets of 6 x 15-s sprints (exercise-to-rest ratios of 1:3, 1:2, and 1:1 for the first, second, and third set, respectively; with 3 min of recovery between each set) were performed separately in both hypoxia (simulated

altitude of ~2100 m) and normoxia, hypoxia-to-normoxia decrements in mean power output occurred for 1:2 (-5.1%) and 1:1 (-7.4%), but not 1:3 ratios (13). This suggests that with a smaller exercise-to-rest ratio the additional impact of hypoxia on RSE is reduced. Although there are several RSH studies that focused on different exercise-to-rest ratios (13–16), the comparison of different sprint durations, which largely impact the oxidative glycolytic balance, has been scarcely investigated. While longer sprint duration (>15 s) mainly relies on the glycolytic pathway, shorter sprints have a larger dependence on the oxidative system (phosphocreatine (PCr) breakdown and resynthesis) and oxygen reserve (17, 18). During a 3-s sprint, 55% of the energy supply comes from PCr, while 32% is derived from anaerobic glycolysis (19). These contributions are 26% and 47% for a 12-s sprint and 17% and 45% for a 30-s effort, respectively (19). During RSE with short sprints interspersed with incomplete recovery, the oxidative system and oxygen reserve are mobilized to resynthesize phosphocreatine (20). Oxygen bound to myoglobin plays a decisive role in muscle metabolism during sprints relying mainly on the oxidative pathway (up to 10 s duration) (17). Overall, there is a wide range of high-intensity intermittent exercises (21, 22) with short (< 30 s) work durations and various exercise-to-rest ratios that may rely differently on the oxidative-glycolytic balance. In this context, it would be impossible to compare RSE with different sprint durations and exercise-to-rest ratios since each one influence performance.

Moreover, RSE using a fixed number of repetitions (i.e., close-loop design) are very popular. One potential drawback of this approach, however, is that pacing may occur due to prior knowledge of the expected number of sprints (23). A growing number of RSE to exhaustion

protocols (i.e., open-loop design) are now implemented, for instance, to assess the effects of systemic hypoxia (7).

The purpose of this study was therefore to investigate the acute responses to RSE until exhaustion with different sprint durations (5, 10 and 20 s) but same exercise-to-rest ratio, performed in normoxia and in hypoxia. By manipulating sprint duration but keeping a constant exercise-to-rest ratio during RSE, we intended to alter the oxidative-glycolytic balance in order to assess to which extent it modifies RSH responses. We hypothesized that the higher the oxidative component of RSE (i.e., the shorter the sprint duration and consequently the recovery duration), the larger the hypoxia-induced decrement in RSE performance to exhaustion.

METHODS

Participants

Twelve healthy males regularly engaged in physical activity (weekly training volume: 4-7 h; weekly number of sessions: 2-4) volunteered to take part in the study. None of them was of elite level. Among them, 10 completed the protocol (age: 29.2 ± 6.0 y; height: 1.84 ± 0.04 m; body mass: 76.5 ± 3.0 kg; body fat 11.0 ± 1.8 % (Tanita Balance MC-980MA, Tokyo, Japan)), one was excluded because of frequent vasovagal episodes and another because abnormal high heart rate peaks in hypoxia. Participants gave their written consent, and the study was approved by the Ethical Commission for Human Research (CER-VD 308/13) in compliance with the Declaration of Helsinki.

Study design

A randomized, single-blind, repeated-measures design was used. Participants first completed a preliminary visit where they performed a 10-min warm-up at 1.5 $W.kg^{-1}$ followed by one 5-s, one 10-s and one 20-s maximal sprints (rest = 3 min between sprints) on the same ergometer and in the same environment (i.e., in the chamber in normoxia). Ergometer dimensions were recorded for standardization during subsequent sessions. On separate visits (3–7 days apart, same time of day ± 1 h), participants completed six trials to assess RSE to exhaustion. Testing visits were performed with three different sprint durations (5 s, 10 s, and 20 s) and two FiO_2 (20.9% and 13.6%, corresponding about to ~3600 m simulated altitude) in a normobaric hypoxic chamber (SL-400, ATS, Sydney, Australia). The hypoxic chamber is a well-ventilated 30 m^3 room (5 m x 2.5 m x 2.4 m) with transparent glass panels. The system consists of compressor storing air in pressurized tanks with serial connection to air filters allowing oxygen reduction (altitude simulation) in the air input flow to the chamber. Air input flow (up to 1000 l/min) was sufficient for safe, comfortable and stable training conditions. The same exercise-to-rest ratio (1:2) was used for all RSE and all sessions (in hypoxia and in normoxia) were performed in the chamber. All cycling bouts were conducted on a cycle ergometer (Lode Excalibur Sport Ergometer, Lode B.V., The Netherlands). Room temperature and relative humidity were 19.6 ± 1.2°C and 33.2 ± 4.1%, respectively.

Experimental trials

Upon arrival, participants completed a 12-min warm-up (6 min at 50 W followed by 6 min at 100 W; pedaling frequency 85 rpm) in the same environmental condition (i.e., hypoxia or normoxia) than the subsequent trial. Subsequently, two maximal warm-up sprints of 5 s, 10 s or

one of 20 s (rest = 3 min) were performed depending on the RSE session; this was followed by 5 min of passive seated rest. All sprints were then performed in "Wingate mode" with a fixed resistance (torque factor of 0.8 $Nm.kg^{-1}$). The first two sprints were checked to be at least > 95% of the best warm-up sprints. Exercise bouts were initiated from a rolling start, with participants seated on the bike and targeting a pedaling frequency of 85 rpm with 15 W resistance, which was also automatically adjusted during each recovery period to 15 W. Strong verbal encouragement was provided, and there was no indication of the number of sprints performed. The test was terminated when peak power output dropped below 70% of the maximal peak power output or if a pedaling cadence of at least 70 rpm could not be achieved in the first half of the sprint.

Responses to exercise

Total number of sprints, peak and mean power output (average of all sprints) were recorded. Fatigue index (FI) was determined by the following equation (24):

$$FI = \frac{100 \times \ (sum\ of\ total\ power\ output\ from\ all\ sprints)}{(number\ of\ sprints\ x\ the\ most\ powerful\ sprint)} - 100$$

Participants indicated their ratings of perceived exertion (RPE) using the 6–20 Borg scale (25). Participants' perceived leg discomfort ('*How uncomfortable do your legs feel?*') and difficulty breathing ('*How uncomfortable does it feel to breathe?*') were also immediately recorded at exhaustion using 0–100 mm visual analogic scales.

Blood lactate concentration was assessed from a hyperemic fingertip (Lactate Scout, SensLab, GmbH, Leipzig, Germany), exactly 3 min after protocol termination.

Vastus lateralis muscle oxygenation was recorded by a NIRS system (Oxymon, Artinis Medical Systems BV, Elst, The Netherlands). This NIRS technique has been extensively described and validated in the literature (26). The NIRS light-emitting diodes (emission wavelengths: 760 and 850 nm) enable the measurement of micromolar changes in oxygenated hemoglobin and deoxygenated hemoglobin ([O_2Hb] and [HHb], respectively). The sum of the changes at 760 and 850 nm gives the micromolar change in total hemoglobin ([tHb]), which in turn reflects the variation in regional blood volume. A differential path length factor of 6.5 was assumed for all tests (27) and muscle oxygenation was measured at 50 Hz. Optodes were placed on the right belly of the *vastus lateralis*, midway between the greater trochanter and the lateral epicondyle of the femur. The *vastus lateralis* muscle was chosen to assess exercise-induced changes in [HHb] concentrations since this muscle is highly active during cycling (28). After the first placement, pen marks were made on the skin at the margins of the probe to verify that it did not move during the trial and to enable the exact same placement in the next test session. The probe was then firmly fixed to the thigh with tape, a bandage, and the aid of the subject's shorts, reducing the influence of ambient light on the measurements.

Since [HHb] values are less sensitive than [O_2Hb] to blood flow variations (29) and changes in O_2Hb signals might be confounded by rapid blood volume changes during sprints (9), only [HHb] and [tHb] were considered for subsequent analysis. A 4^{th}-order low-pass zero-phase Butterworth filter (cutoff frequency 0.2Hz) was applied to the resampled signals in order to remove possible artifacts and smooth the pedaling-induced perturbations. Maximum and minimum [HHb] and [tHb] values were identified during each sprint. Typically, maximum [tHb] ([tHb] max) and minimum [HHb] ([HHb] min) occurred at the beginning of each sprint, while

minimum [tHb] ([tHb] min) and maximum [HHb] ([HHb] max) were located at the end of each sprint. Differences between beginning and end sprint concentrations of [tHb] and [HHb] were defined as the amplitude of the variation for each sprint (Δ[tHb] and Δ[HHb]). The values of Δ[HHb] and Δ[tHb] were expressed as a change from baseline in micromoles (μM).

Statistical analysis

Values are expressed as means ± standard deviations (SD). The normality and equality of variances of the data were verified using the Shapiro–Wilk test. Data were compared with two-way repeated-measures ANOVA (sprint duration [5:10, 10:20 and 20:40] × condition [FiO_2 = 20.9% and 13.6%]), followed by *Bonferroni*-adjusted post hoc comparisons. The relationships between the best mean power output over 5, 10 or 20 s and the normoxia-to-hypoxia decrease in number of sprints were investigated using *Pearson* coefficient after normality check. Statistical significance was set at $p < 0.05$. Effect sizes were calculated using partial eta squared (η_p^2). All statistical analyses were performed using SPSS software (IBM statistic V27, Chicago, Il, USA).

RESULTS

There was a significant sprint duration x condition interaction for the total number of sprints and fatigue index ($p = 0.015$, $\eta_p^2 = 0.375$ and $p < 0.001$, $\eta_p^2 = 0.653$; respectively) (Figure 1 and Table 1). As expected, the number of sprints to exhaustion decreased with increasing RSE sprint durations, but hypoxia led to a significant decrement only for 5:10 ($p = 0.016$) and 10:20 ($p = 0.018$; Figure 1). Fatigue index was more pronounced in hypoxia than in normoxia only for 5:10 (Table 1). Post-exercise blood lactate concentration only showed a main effect of sprint duration ($p = 0.029$, $\eta_p^2 = 0.325$). Compared to 5:10 (11.4 ± 2.2 $mmol.l^{-1}$), blood

lactate accumulation was significantly higher for 20:40 (13.1 ± 2.7 mmol.l^{-1}; p = 0.027), while statistical significance was not reached for 10:20 (12.5 ± 2.3 mmol.l^{-1}; p = 0.056).

Muscle oxygenation values are reported in Table 2 and Figure 2. There was a main effect of sprint duration for [HHb] min (p = 0.003, $\eta_p^2 = 0.696$), [tHb] max (p = 0.001, $\eta_p^2 = 0.623$), Δ[HHb] (p = 0.003, $\eta_p^2 = 0.813$) and Δ[tHb] (p = 0.036, $\eta_p^2 = 0.341$). During recovery, [HHb] min decreased while [tHb] max increased from 5:10 to 20:40 (-61% and +27%, respectively) and from 10:20 to 20:40 (-58% and +73%, respectively). During exercise, Δ[HHb] increased from 5:10 to 20:40 (+210%), 10:20 to 20:40 (+92%), and 5:10 to 10:20 (+61%). There was also a Δ[tHb] decrease from 5:10 to 20:40 (-25%). However, no significant interaction effect was observed for any index derived from [HHb], [tHb] min and for Δ[tHb]. Conversely, there was a significant interaction effect for [tHb] max (p < 0.001, $\eta_p^2 = 0.632$).

The highest mean power output over 5 s and 10 s was positively correlated with the normoxia-to-hypoxia percent decrease in total number of sprints to exhaustion for 5:10 (R^2 = 0.55, p = 0.013 and R^2 = 0.53, p = 0.016; respectively) (Figure 3). These relationships remained significant even with the removal of the extremely high value in power output (rightmost black circle; Figure 3A and 3B) from the analysis (R^2 = 0.45, p = 0.048). Contrastingly, there was no significant relationship between the power output over 20 s and the normoxia-to-hypoxia percent decrease in number of sprints to exhaustion during all RSE sessions.

DISCUSSION

This study investigated the acute responses to RSE until exhaustion with different oxidative-glycolytic balances, using different sprint durations (5, 10 and 20 s) but the same exercise-to-rest ratio, performed in normoxia and in hypoxia. The main finding is that hypoxia reduced performance for 5:10 and 10:20, but not 20:40 RSE. Blood lactate concentration and muscle de-/reoxygenation were impacted by sprint duration but not by hypoxia. Moreover, there was a positive relationship between the maximum power over 5 s or 10 s (but not 20 s) and the normoxia-to-hypoxia percent decrease in number of sprints during 5:10 RSE (but not 10:20 and 20:40). Overall, our results highlight the importance of the oxidative-glycolytic balance in acute physiological responses to RSH indicating that hypoxia impacts to a larger extent RSH with a predominant oxidative component.

Psycho-physiological differences between normoxia and hypoxia

Hypoxia reduced the total number of sprints completed during 5:10 (-29.3%) and 10:20 (-35.7%), while it had no impact when sprint duration was longer (20:40). Only one hypoxic condition (FiO_2 = 0.136) was compared to normoxia, while the severity of hypoxia is also paramount. Indeed, severe hypoxia (FiO_2 < 14%) induced more pronounced alterations than either normoxia or low-to-moderate hypoxia (FiO_2 > 16%) (30). Using a comparable RSE (10-s cycling sprints interspersed by 20-s rest) test to exhaustion, Willis et al. (2017) showed that the total number of sprints in moderate (FiO_2 = 16.5%) and severe hypoxia (FiO_2 = 13.6%) was only two-thirds and half of the number of sprints performed in normoxic conditions, respectively (7). In line with this result, fatigue index was significantly larger only for 5:10, when compared to normoxia. This may be due to the higher oxidative component of open-ended RSE with short

sprints durations and a ratio ≥ 1:2, leading therefore to short recovery periods. RSE with both very short work and recovery intervals was shown to stimulate the oxygen consumption to a very high level; i.e., maximal oxygen consumption was maintained during 14 min for 15:15 (ratio 1:1) RSE sessions (21, 31). During short sprints, the energy supply comes mainly from phosphocreatine breakdown (32, 33). Hence, PCr resynthesis during recovery depends on the O_2 availability, leading to an altered performance in hypoxia in RSE with a high oxidative component (i.e., 5:10 and 10:20 in our study) (17, 32). Moreover, a pioneering study highlighted the importance of the O_2 availability from myoglobin for muscle metabolism, which is directly impacted by sprint durations (17). Regarding long sprint durations (>10 s), the relative proportion of energy generated from glycolysis increased compared to oxidative phosphorylation, even if the intensity was identical (17).

In the present study, higher blood lactate concentrations were measured for 20:40 than 5:10; suggesting an increased reliance on the glycolytic pathway with longer RSE. This confirms previous observations of increased glycolytic supply with longer sprint durations during all out exercise > 15 s (17, 18). Since hypoxia impacts phosphagen and oxidative pathway, glycolysis is stimulated to a higher degree in this condition involving greater lactate production in hypoxia than in normoxia (34, 35). The lack of either a condition effect or an interaction could be attributed to the open loop design of our study. Indeed, regardless of the condition, participants were instructed to continue exercise until they met exhaustion criteria, which may have blunted any difference in glycolytic contribution between RSE performed in normoxia and in hypoxia. Exhaustion was reached since all perceptual responses at exercise cessation were rated ‘high’ (Table 1). Of importance, all the subjective perceptual responses (i.e., RPE, limb discomfort,

difficulty breathing) were close to maximal values, and with no meaningful differences between trials, despite a large inter-individual variability. These results confirm that all RSE were performed at (or near) perceptual maximum effort, and are in line with previous reports of comparable exercise-related sensations imposed on both locomotor and respiratory systems during repeating cycling sprints until exhaustion (7). Perceived effort and exercise-related discomfort are not increased by hypoxia or sprint duration since no effect on RPE, limb discomfort, and difficulty breathing were found. Overall, our results suggest that hypoxia impacts RSE mainly when the primary sources of energy supply come from phosphagen and oxidative pathways.

Regardless of sprint duration or condition (normoxia or hypoxia), participants were able to maintain their peak power output (Table 1). This is in agreement with previous studies suggesting that peak power output is generated during the first 5–10 s during all out efforts (36, 37). Hence, it seems that participants had sufficient time to reach their maximal power output even for RSE with the shorter sprint duration. This is one of the paramount requirements to generate anaerobic system adaptations, such as enhanced glycolytic activity, upregulation of anaerobic metabolism, increased buffering capacity, modified FT fibers behavior, and increased end-product metabolite removal (1, 3, 12, 33, 35). Our results show that peak power output did not differ between RSE with distinct sprint durations and hence no pacing occurred during testing.

Muscle oxygenation and blood volume variation

In the present study, [HHb] min, [tHb] max, Δ[HHb] and Δ[tHb] showed a main effect of sprint duration. This highlights that the amplitude of muscle deoxygenation/perfusion directly depends on sprint duration, regardless of the condition, as previously reported (7). Muscle [tHb] at the optode location dropped during sprinting phases to reach a minimum at the end of each sprint. These fluctuations were reported by others (3, 8) and may be attributed to the decreased muscle blood flow during maximal cycling exercise due to intra-muscle vascular occlusion. The intramuscular pressure on *vastus lateralis* blood vessels during maximal contractions (all out cycling sprints) seems to restrain blood perfusion due to vessel squeezing, resulting in a reduction of blood volume during the exercise phase (38, 39), hence the main effect of duration on [HHb] and [tHb] was expected. Moreover, 20:40 induced a more complete reoxygenation during recovery phase (i.e., lower [HHb] min and higher [tHb] max) compared to 5:10 and 10:20. This would suggest a greater inhibitory effect of the nitric oxide on vascular vasoconstriction, inducing a larger hyperemia (40). The higher metabolite (K^+, lactate) accumulation during 20:40 may contribute to both vasorelaxation and hyperemia (41).

No condition or interaction effect was reported for any [HHb] signal. This suggests that hypoxia did not exacerbate muscle deoxygenation during RSE to exhaustion. Although surprising, these results are in line with previous studies which reported no difference in [HHb] during repeated sprint at 3800 m vs 400 m (7). A recent study reported no change in muscle oxygenation during RSH compared to normoxia despite a drop in systemic saturation (10). It was hypothesized that the fitness level of participants and the associated physiological mechanisms (muscle recruitment and O_2 availability) trigger similar magnitudes of fatigue and muscle

oxygenation despite hypoxemia (42). An increased [tHb] in RSH was also expected, when compared to normoxia, as reported previously by our research group for 10:20 RSE (3, 43). Indeed, hypoxia is known to induce a compensatory nitric oxide-related vasodilation (4, 5). Our results (i.e., significant interaction effect on [tHb] max and [tHb] max higher in hypoxia only during 10:20 RSE) partly confirm the compensatory vasodilation during RSH compared to normoxia, at least with appropriate sprint and recovery durations (3, 12). However, the lack of difference during 5:10 and 20:40 should reflect an inappropriate combination of sprint duration and ratio to induce optimal hypoxia-related response. Hence, further study should confirm if the optimal sprint duration with a 1:2 ratio should be comprised between 6 and 10 s. The increased [tHb] during sprint and recovery in hypoxia during 10:20 suggests that blood volume in the muscle was higher (44). This could act as a defensive mechanism to blunt hypoxia effect by improving muscle waste metabolite removal and O_2 delivery. However, these mechanisms are not sufficient during sprints with a high oxidative component (i.e., 5:10 and 10:20) since number of sprints to exhaustion decreased in hypoxia, highlighting the additional stress induced by hypoxia.

Potential influence of muscle fiber typology.

In fact, our findings showed that there were significant relationships between best mean power output over 5 s or 10 s and the normoxia-to-hypoxia percent decrease in sprint number for 5:10 (R^2 = 0.55, p = 0.013 and R^2 = 0.53, p = 0.016; respectively). Fast twitch fibers are preferentially recruited during maximal intensity short sprints (45) and consequently in such exercise, individuals generating higher power output typically possess a greater proportion of FT fibers (46, 47). One can make the assumption that participants with the highest best mean power

over 5 or 10 s are also the ones with the greater proportion of FT fibers (46, 48, 49). Hence, the present relationships highlighted that individuals with predominantly FT fibers (and consequently less ST fibers) were less impacted by the decreased O_2 availability than their counterparts (with more ST fibers), but only during the 5:10 RSE; i.e., relying more on the oxidative pathway. This confirms the hypothesis of pioneering investigations (3, 12, 43), which suggested that FT fibers are more likely to benefit from the hypoxia-induced compensatory vasodilation and the associated higher perfusion (6). Therefore, FT fibers may benefit to a larger extent of the hypoxic stimulus and this was the core mechanistic difference between RSH and interval hypoxic training (12). FT fibers are known to have a greater fractional O_2 extraction (compared to ST fibers) if highly perfused (6). Overall, it can be argued that RSH would require a maximal recruitment of FT fibers and second a severe hypoxic stimulus ($FiO_2 \leq 0.13$) for a large desaturation/deoxygenation (50). Although the relationships were significant, there are several other factors, not measured in the present protocol, that can affect the normoxia-to-hypoxia percent decrease in sprint number (e.g., maximal oxygen consumption, amplitude of the hypoxia-induced VO_2 decrease, ventilatory response to hypoxia, chemosensitivity).

Limitations

This study is not without limitations. First, it did not include the quantification of skin and adipose tissue thickness at the NIRS measurement location. The manner in which these measures relate to the distance between optodes on the NIRS monitor is known to affect NIRS signal depth penetration (51). However, body fat (11.0 ± 1.8 %) was low, suggesting a reduced inter-subject variability in tissue thickness and consequently a small effect on the NIRS assessments. Moreover, the exercise-to-rest ratio was constant and set at 1:2 in the present study.

Further study should compare the acute performance and psycho-physiological responses of RSE to exhaustion with varying sprint durations, smaller exercise-to-rest ratios, and inspired oxygen fractions. For ethical reason, fiber typology was estimated from the highest power output developed over 5 s (48, 49). Future muscle biopsy studies are needed to confirm the present relationship between fiber typology and hypoxia-related RSH impairment. There is a well-known inter-subject and inter-trial variability in metabolic responses, particularly on the kinetics of the respective glycolytic vs oxidative contribution during exercise. While it was beyond the scope of this study, one cannot rule out the influence of fast or slow VO_2 kinetics or lactate production that may vary between subjects and consequently influence the global responses to RSE with different sprint durations. Finally, a common indirect measurement of anaerobic/aerobic energy contribution, such as accumulated oxygen deficit or gross efficiency method, was not included. In the absence of gas exchange measurements, blood lactate concentrations were used to estimate the anaerobic contribution.

CONCLUSIONS

This study investigates the effects of normobaric hypoxia on repeated sprint exercise to exhaustion with different oxidative-glycolytic balance. The main finding is that hypoxia deteriorated performance for 5:10 and 10:20, but not 20:40. Blood lactate increased with sprint duration, confirming that glycolytic component increases with sprint duration. Muscle oxygenation was not impacted by RSH but increased sprint duration induced higher muscle deoxygenation. Moreover, there was a relationship only between the maximal power output over 5 or 10 s and the normoxia-to-hypoxia percent decrease in number of sprints during 5:10 RSE. Finally, this study – even indirectly – confirms previous assumptions of the importance of the FT

fibers recruitment during RSH to induce chronic muscle adaptations. With a 1:2 exercise-to-rest ratio, sprint duration up to 10 s should be used to induce optimal response from hypoxia. The present study highlights the importance of the oxidative-glycolytic balance on the acute physiological responses to RSH.

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FIGURE LEGENDS

Figure 1 - Maximal number of sprints to exhaustion realized during repeated sprint exercise (RSE) performed in normoxia (white bar) or in hypoxia (black bar).

D: main sprint duration effect; C: main condition effect; D × C: sprint duration × condition interaction effect. * $p < 0.05$ for difference with normoxia; # $p < 0.05$ for difference with 5:10 RSE; † $p < 0.05$ for difference with 10:20 RSE.

Figure 2 – Deoxyhemoglobin [HHb] amplitude (A) and total hemoglobin [tHb] amplitude (B) during repeated sprint exercise (RSE) performed in normoxia (white circle) or in hypoxia (black circle).

D: main sprint duration effect; C: main condition effect ; D × C: sprint duration × condition interaction effect.

Figure 3 – A/ Relationship between the best mean power output over 5 s (circle), 10 s (square) and 20 s (triangle) and the percent normoxia-to-hypoxia decrease in sprint number to exhaustion during repeated sprint exercise (RSE) with corresponding sprint duration; i.e., 5:10 RSE (black symbols), 10:20 RSE (grey symbols), and 20:40 RSE (white symbols).

B / Relationship between the best mean power output over 5 s (circle), 10 s (square) and 20 s (triangle); and the percent decrease in sprint number to exhaustion performed in 5:10 RSE.

Figure 1

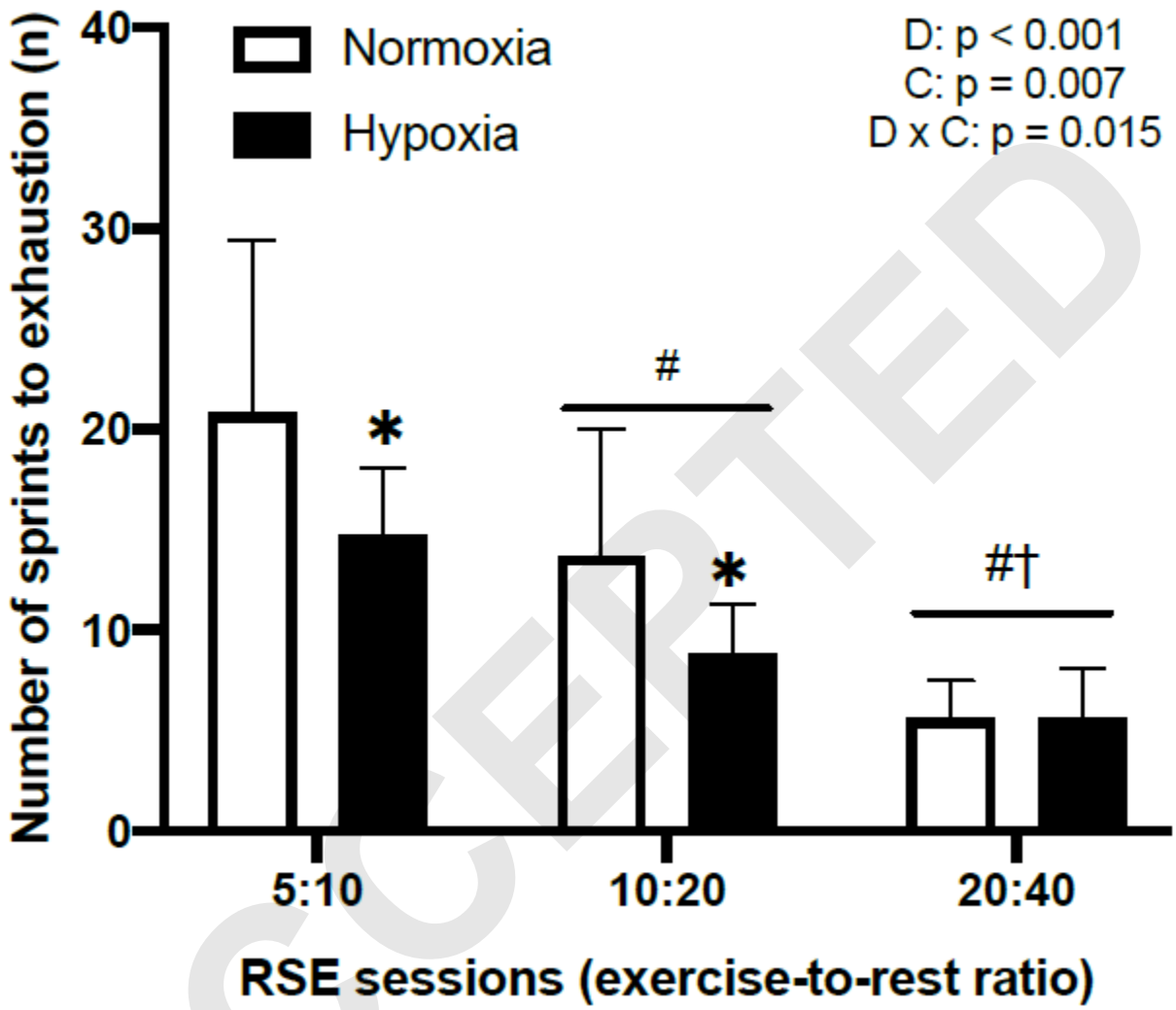

Figure 2

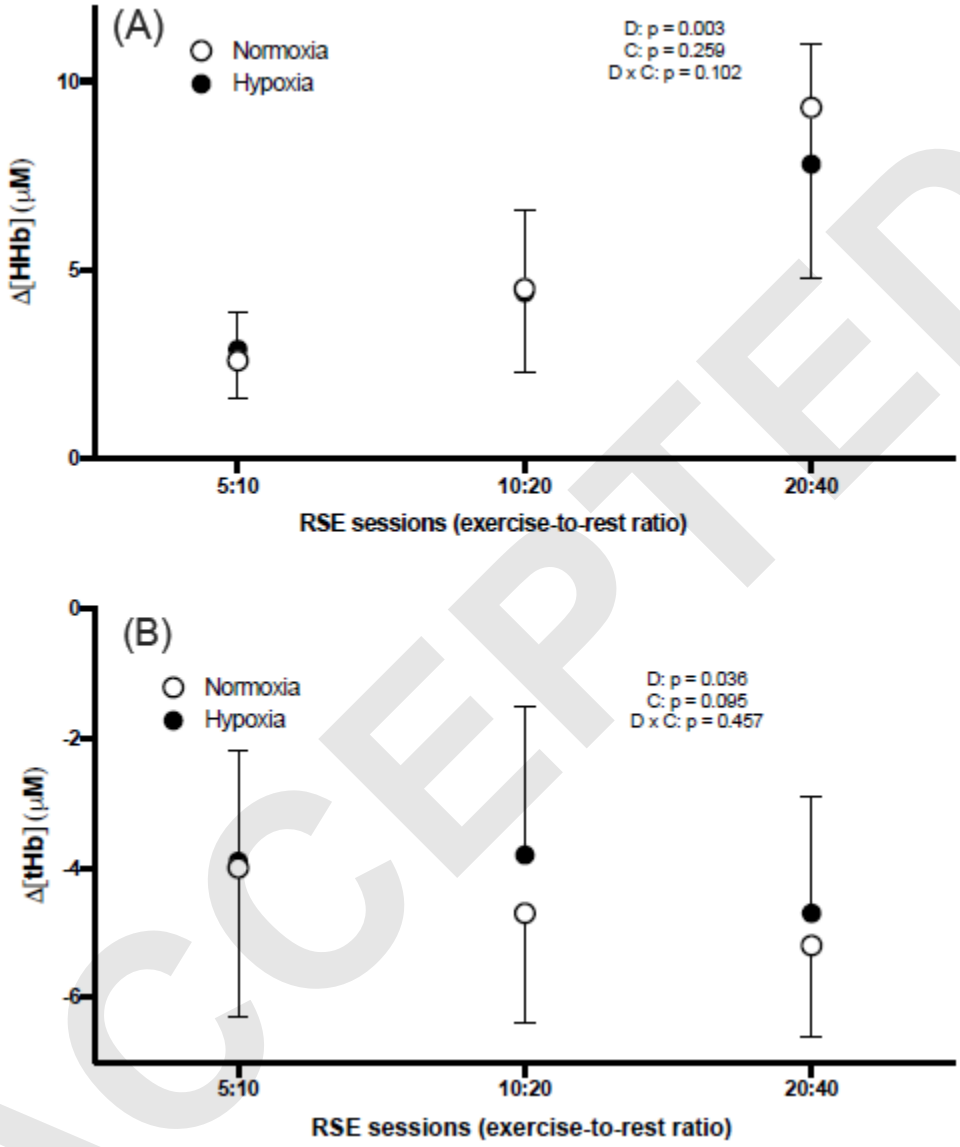

Figure 3

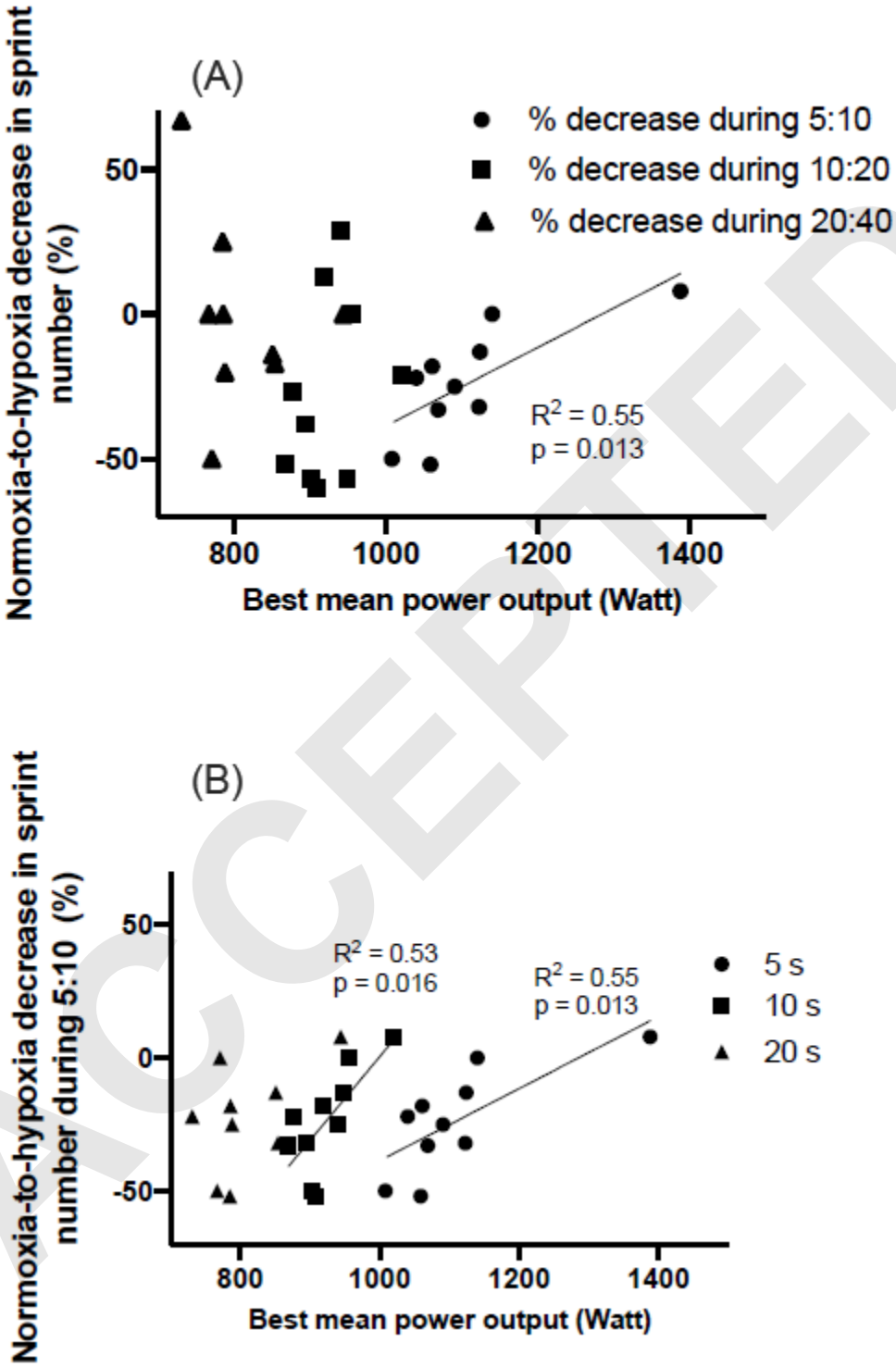

Table 1 - Comparison of psycho-physiological responses during repeated sprints exercise (RSE) with several sprint durations performed in normoxia or in hypoxia.

				Anova p-values		
			Mean ± SD	Sprint duration effect	Condition effect	Sprint duration × condition interaction
Peak Power output (W)	5:10	Normoxia	1370 ± 106	P = 0.251	P = 0.217	P = 0.558
		Hypoxia	1333 ± 108			
	10:20	Normoxia	1343 ± 103			
		Hypoxia	1315 ± 144			
	20:40	Normoxia	1380 ± 171			
		Hypoxia	1385 ± 145			
RPE (6-20)	5:10	Normoxia	18.0 ± 1.1	P = 0.124	P = 0.371	P = 0.135
		Hypoxia	17.1 ± 1.3			
	10:20	Normoxia	17.5 ± 1.2			
		Hypoxia	16.9 ± 1.4			
	20:40	Normoxia	17.8 ± 1.1			
		Hypoxia	17.9 ± 1.7			
Limb discomfort (1-100)	5:10	Normoxia	76.4 ± 16.3	P = 0.539	P = 0.980	P = 0.726
		Hypoxia	78.8 ± 9.3			
	10:20	Normoxia	75.7 ± 13.1			
		Hypoxia	73.0 ± 12.7			
	20:40	Normoxia	78.1 ± 14.7			
		Hypoxia	78.0 ± 14.7			
Difficulty breathing (1-100)	5:10	Normoxia	72.2 ± 23.9	P = 0.313	P = 0.976	P = 0.337
		Hypoxia	82.0 ± 16.7			
	10:20	Normoxia	75.6 ± 15.6			
		Hypoxia	65.3 ± 20.8			
	20:40	Normoxia	70.0 ± 22.5			
		Hypoxia	71.0 ± 22.9			
Blood lactate concentration ($mmol.l^{-1}$)	5:10	Normoxia	11.8 ± 2.1	**P = 0.029**	P = 0.954	P = 0.234
		Hypoxia	11.1 ± 2.28			
	10:20	Normoxia	11.8 ± 1.8			
		Hypoxia	13.2 ± 2.9			
	20:40	Normoxia	13.4 ± 1.8			
		Hypoxia	12.8 ± 3.6			
Fatigue index (%)	5:10	Normoxia	-27.6 ± 4.8	**P < 0.001**	**P = 0.003**	**P < 0.001**
		Hypoxia	-39.6 ± 7.6*			
	10:20	Normoxia	-27.6 ± 7.5#			
		Hypoxia	-25.5 ± 5.7#			
	20:40	Normoxia	-20.5 ± 5.0			
		Hypoxia	-21.3 ± 6.2#			

RPE: rating of perceived exertion. * for difference with normoxia; # for difference with 5:10 RSE

Table 2 - Comparison of muscle oxygenation responses during repeated sprints exercise (RSE) with several sprint durations performed in normoxia or in hypoxia.

				Anova p-values		
			Mean ± SD	Sprint duration effect	Condition effect	Sprint duration × condition interaction
[HHb] min (μM)	5:10	Normoxia	9.7 ± 5.4	**P = 0.002**	P = 0.124	P = 0.159
		Hypoxia	9.1 ± 4.3			
	10:20	Normoxia	6.3 ± 3.5			
		Hypoxia	11.17 ± 9.9			
	20:40	Normoxia	2.9 ± 1.5			
		Hypoxia	4.3 ± 3.8			
[HHb] max (μM)	5:10	Normoxia	11.9 ± 6.1	P = 0.676	P = 0.323	P = 0.210
		Hypoxia	11.5 ± 5.1			
	10:20	Normoxia	10.4 ± 5.2			
		Hypoxia	14.7 ± 11.0			
	20:40	Normoxia	11.9 ± 5.6			
		Hypoxia	11.6 ± 5.0			
[tHb] min (μM)	5:10	Normoxia	4.8 ± 3.8	P = 0.372	P = 0.144	P = 0.179
		Hypoxia	4.1 ± 4.5			
	10:20	Normoxia	2.4 ± 7.2			
		Hypoxia	12.6 ± 18.2			
	20:40	Normoxia	3.8 ± 3.4			
		Hypoxia	4.9 ± 4.0			
[tHb] max (μM)	5:10	Normoxia	8.4 ± 5.1	**P = 0.001**	**P = 0.016**	**P < 0.001**
		Hypoxia	7.8 ± 4.9			
	10:20	Normoxia	1.7 ± 3.9#			
		Hypoxia	10.2 ± 5.1*			
	20:40	Normoxia	10.0 ± 4.1†			
		Hypoxia	10.7 ± 3.8			

HHb: deoxyhemoglobin, tHb: total hemoglobin. * $p < 0.05$ for difference with normoxia; # $p < 0.05$ for difference with 5:10 RSE; † $p < 0.05$ for difference with 10:20 RSE.