Muscle Deoxygenation during Repeated Sprint Running: Effect of Active vs. Passive Recovery

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Key words
- intermittent work
- team-sport performance
- near-infrared spectroscopy
- muscle deoxygenation

Abstract

The purpose of this study was to compare the effect of active (AR) versus passive recovery (PR) on muscle deoxygenation during short repeated maximal running. Ten male team sport athletes (26.9±3.7y) performed 6 repeated maximal 4-s sprints interspersed with 21 s of either AR (2 $m.s^{-1}$) or PR (standing) on a non-motorized treadmill. Mean running speed ($AvSp_{mean}$), percentage speed decrement (Sp%Dec), oxygen uptake ($\dot{V}O_2$), deoxyhemoglobin (HHb) and blood lactate ($[La]_b$) were computed for each recovery condition. Compared to PR, $AvSp_{mean}$ was lower (3.79±0.28 vs. 4.09±0.32 $m.s^{-1}$; $P<0.001$) and Sp%Dec higher (7.2±3.7 vs. 3.2±0.1.3%; $P<0.001$) for AR. Mean $\dot{V}O_2$ (3.64±0.44 vs. 2.91±0.47 L.min^{-1}, $P<0.001$), HHb (94.4±16.8 vs. 83.4±4.8% of HHb during the first sprint, $P=0.02$) and $[La]_b$ (13.5±2.5 vs. 12.7±2.2 $mmol.l^{-1}$, $P=0.03$) were significantly higher during AR compared to PR. In conclusion, during run-based repeated sprinting, AR was associated with reduced repeated sprint ability and higher muscle deoxygenation.

Introduction

Time-motion analysis of team sports such as soccer, rugby, basketball and handball has revealed that successfully decisive moments in a match are often preceded by short high-intensity sprints in the range of 10–30 m or 2–4 s [16, 30, 35]. The ability to repeat these high-intensity short-duration efforts (typically 3–7 times) following short recovery periods is called 'repeated sprint ability' [35]. Recently, repeated sprint ability has been shown to be a strong predictor of match-related physical performance (e.g., total sprint distance or very high-intensity running distance) in top-level professional soccer players [32]. Thus, an understanding of the bioenergetic demands of repeated sprint ability is fundamental to the appropriate design of training programs aimed at improving repeated sprint ability in team sport athletes [10, 19, 32].

Performing active versus passive recovery between high-intensity cycling efforts has been reported to significantly impair performance [14, 15, 36, 37]. In the only two studies yet to examine very short cycle sprints (4 s), Spencer et al. [36, 37] showed that active versus passive recovery was associated with a significantly higher muscle lactate accumulation, a strong trend towards a lower PCr resynthesis, and significantly poorer repeated sprint ability. As experimentally shown by Haseler et al. [23], the finding of a reduced repeated sprint performance with active versus passive recovery is thought to be due to the reduction in oxygen availably that occurs with active recovery, which may limit muscular PCr and ATP resynthesis in the early recovery phase (within 20–30 s) following exercise. Recently, because of the strong relationship between muscle deoxygenation (or reoxygenation) rate and PCr utilization (or recovery) kinetics [22, 26], the noninvasive near-infrared spectroscopy technique (NIRS) has become the preferred method to examine muscle metabolism during exercise [3, 14, 15, 31]. For example, following 15-s high-intensity cycling efforts, active versus passive recovery was shown to lower time-to-exhaustion [14] and decrease power output during successive 30-s Wingate cycling tests [15] while being associated with an increase in muscle deoxygenation [14, 15].

Despite the important contributions that have advanced our understanding of the effects of active versus passive recovery, in all pre-cited studies, participants exercised exclusively on standard [14, 15] or front-access cycle ergometers [36, 37], neither of which replicates specific team

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sport movements. It is well known that exercise mode influences muscle recruitment patterns [7], which in turn affects metabolic cost [27], anaerobic system participation and repeated sprint ability [33]. An assessment of the actual muscle metabolism during repeated sprint running is therefore necessary to fully understand the effects of recovery type under conditions resembling those experienced during team sport activity. It is possible that the greater number of muscle groups and joints engaged during running compared with cycling, due to body-balance and weight-bearing factors [25,27], may elicit a greater usage of muscle substrates and oxygen stores, leading to increased metabolic cost [27] and metabolite accumulations [33]. This, in turn, may be associated with a more pronounced reduction of repeated sprint ability during sprint running [33] (i.e., higher percentage of velocity decrement) than that previously observed during sprint cycling [14,15,36,37]. In contrast, the delta efficiency (i.e., the ratio of change in external mechanical work to energy expenditure) has been shown to be higher in running (42–45%) compared with cycling (25%) [6,7], especially at low running velocities [7]. It is therefore possible that this enhanced muscular efficiency might lower the energetic demands of the recovery run, thus diminishing the detrimental effects of a low-velocity active recovery on repeated sprint running ability.

In light of our limited understanding of performance during team sport-specific repeated sprint running, we sought to investigate muscle deoxygenation during a repeated sprint ability test using either active or passive recovery to understand possible differences in repeated sprint performance between both conditions. In contrast to previous cycling studies, we hypothesized that repeated sprint running would be associated with a progressive muscle deoxygenation, but due to the high delta efficiency of running at low intensity (i.e., during recovery), only minor differences in muscle oxygenation and performance between active and passive recovery conditions would be apparent.

Methods

Subjects

Ten male subjects (26.9±3.7 y, 81.5±8.9 kg, 180.9±4.9 cm) volunteered to participate in this study. All were involved (5±3.2 $h \cdot wk^{-1}$ in various intermittent sporting activities (soccer, handball or basketball) and had no history or clinical signs of cardiovascular or pulmonary diseases. Participants were not currently taking prescribed medications and presented with normal levels of blood pressure and electrocardiographic patterns. The study conformed to the recommendations of the Declaration of Helsinki and participants gave voluntary written consent to participate in the experiment, which was approved by the local human research ethics committee.

Experimental overview

On three distinct occasions (separated at least by 48 h) participants performed a graded aerobic test and, in a randomized order, two sets of 6 repeated maximal 4-s sprints interspersed with 21 s of either active (AR, running at 2 $m.s^{-1}$ [38]) or passive recovery (PR, standing) on a non-motorized treadmill (Force model, Woodway, Waukesha, WI, USA). Prior to the study, all subjects were familiarized with sprinting on the treadmill. For all tests, ventilation parameters (i.e., oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory exchange ratio (RER), respiratory rate (RR), minute ventilation ($\dot{V}_E$,)) and heart rate (HR) were recorded. Deoxyhemoglobin and total hemoglobin of the vastus lateralis were additionally measured via near-infrared spectroscopy (NIRS) during the two repeated sprint ability tests. Participants indicated their rating of perceived exertion (RPE, 6–20 Borg's scale) immediately at the end of each exercise.

Maximal graded exercise test

The maximal graded exercise test was performed on a motorized treadmill (Trackmaster TM500E, JAS Manufacturing; Carrollton, Texas, USA). After a 2-min warm-up at 6 $km.h^{-1}$, the test began at an initial speed of 10 $km.h^{-1}$ and speed was increased by 0.5 $km.h^{-1}$ every minute until fatigue [28]. The test was terminated when the subject was unable to maintain the required running speed.

Repeated sprint ability tests

Within 5 min of completing a 6-min running exercise at 60% of $\dot{V}O_2$max on the non-motorized treadmill (sampling frequency of 200 Hz, s), subjects performed 2–3 maximal 3-s warm-up sprints, followed 2 min after by the repeated sprint ability test. Subjects performed 6 repetitions of maximal 4-s sprints on the treadmill every 25 s, so that recovery lasted 21 s. During the first 18 s of recovery between sprints, subjects performed either active running recovery (2 $m.s^{-1}$ [38], ~45% of v $\dot{V}O_2$max, see below) or a passive standing recovery. Three seconds prior to the commencement of each sprint, subjects were asked to assume the ready position and await the start signal. During the active recovery, visual (i.e., actual treadmill speed displayed on a screen in front of the subject) and audio (i.e., time countdown) feedback was given to the subjects so that they maintained a running speed of 2 $m.s^{-1}$. Subjects were instructed to complete all sprints as fast as possible, and strong verbal encouragement was provided to each subject during all sprints. This test was adapted from a previous running test (i.e., 6 × 25 m) [38], which has been shown to be reliable when performed in the field (CV = 0.7%, 95% CI [0.5–1.2] for total sprint time [38]) or on a non-motorized treadmill (i.e, CV = 2.8%, 95% CI [1.9–3.9] for maximal speed [24]). Maximal speed (MxSp; $m.s^{-1}$), average speed (AvSp; $m.s^{-1}$), acceleration (Acc; $m.s^{-2}$), distance covered (Dist; m) and stride frequency (StFq; Hz) were calculated for each sprint via the software provided by the Woodway manufacturer. Over the 6 repetitions, the fastest and mean AvSp were noted as $AvSp_{best}$ and $AvSp_{mean}$, respectively. To assess fatigue during the repeated sprint ability protocol, the percentage of sprint speed decrement (Sp%Dec) was calculated as follows: 100 – ([$AvSp_{mean}$/$AvSp_{best}$] × 100). This method has been shown to provide the most reliable method (typical error = 4.6%, 95% CI [3.7–6.2]) of assessing fatigue during repeated sprint ability tests [18].

Cardiorespiratory measures

During all exercise tests, respiratory gas exchanges were measured breath-by-breath using an automated metabolic system (Medgraphics CPX Gas Analysis System; St. Paul, MN). Prior to each test, the O_2 and CO_2 analyzers were calibrated using gases of a known concentration and the flow meter was adjusted using a 3 L syringe (Hans Rudolph, Inc., Kansas, MO). This particular metabolic system was verified to an accuracy of less than 3% across all possible flow rates by the Australian Laboratory Standards Assistance Scheme [20]. During the graded maximal aerobic test, maximal oxygen uptake was defined as the highest $\dot{V}O_2$

attained in one 30 s period [29]. Confirmation of $\dot{V}O_2$ max was established when HR peak was within 10 beats.min^{-1} of the age-predicted maximum (i.e., 220- age), when respiratory exchange ratio (RER) was greater than 1.1 and when post exercise blood lactate concentration was higher than 8 mmol.L^{-1} [39]. The velocity associated with O_2max (v O_2max) was determined as the lowest running speed which elicited a O_2 value equal to O_2max (within 3 ml.kg^{-1}.min^{-1}) [8]. A Polar S810 HR monitor (Polar Electro, Kempele, Finland) was used to continuously record beat-to-beat HR during each exercise session. Maximal HR (HR_{max}) was defined as the average of the 5 highest 1-s averaged consecutive values.

Cardiorespiratory analysis during repeated sprint ability tests

During the repeated sprint ability tests, all cardiorespiratory measures were automatically filtered for aberrant data points, interpolated to 1-s intervals and time-synchronized. Maximal (O_2peak) and minimal (O_2 min) values for O_2 uptake were computed after each sprint (i.e., during the successive 21-s recovery periods) as the average of the 5 highest or 5 lowest 1-s consecutive values. Changes in O_2 during recovery ($O_2\Delta$) were calculated for each subject as their mean delta values (O_2peak – O_2 min) observed during each of the first 5 recovery periods (recovery after the sixth sprint was not taken into account). Due to low variations in HR during the recovery periods (HR inertia), recovery index for HR was not calculated.

Near-infrared spectroscopy measurements

The NIRS apparatus (Niromonitor NIRO-200, Hamamatsu Photonics, Japan) used in this study was a 3-wavelength continuous wave system, which simultaneously uses the modified Beer–Lambert and spatially resolved spectroscopy methods. Changes in tissue deoxyhemoglobin (HHb) and total hemoglobin (tHb) were measured using the differences in absorption characteristics of light at 775, 810, and 850 nm. Because of uncertainty in differential path length factors for the quadriceps muscle, these were not used in the present study. The two photodiodes mounted 40 mm apart detect changes in the transmission of radiation as a function of time, distance and wavelength. During all tests, the NIRS system was connected to a personal computer for data acquisition (6 Hz), analog-to-digital conversion, and subsequent analysis. To allow comparisons with previous cycling studies [14, 15, 31], NIRS probes were positioned on the left vastus lateralis muscle, approximately 12 cm from the knee joint along the vertical axis of the thigh. A surgical marker was used to mark the probe placement for accurate repositioning. The probe and the skin were covered with black tape to prevent contamination from ambient light. Skinfold thickness at the site of application of the NIRS probe was determined before the testing sessions using Harpenden skinfold calipers (British Indicators Ltd, UK). The calculated value of skin and subcutaneous tissue thickness was less than half the distance between the source and the detector. Although the analysis of other NIRS-derived measures could have been considered (i.e., oxyhemoglobin (O_2Hb)), we paid specific attention to HHb (and tHb) to remain consistent with other repeated high-intensity exercise studies [14, 15, 31], and because HHb has been shown to be essentially unaffected by changes in blood volume [12, 21]. Moreover, it is possible that changes in O_2Hb signals might be confounded by perfusion variations and abrupt blood volume changes during sprints due to fast muscle contractions and the 'muscle pump' phenomena [12]. NIRS data were time-synchronized with cardiorespiratory data. Resting HHb and tHb values before the repeated sprint ability test (120 s averaging) were standardized as 0 %, and the maximum values reached during the first sprint were standardized as 100 %. Changes in HHb during each recovery period (i.e., muscle Δdeoxygenation, HHb_Δ) was calculated for each subject as HHb_{max}–HHb_{min} values (HHb_{max} and HHb_{min} were calculated as an average of the 5 highest or 5 lowest 1-s consecutive values) observed during each of the 5 first recovery periods (recovery after the sixth sprint was not taken into account).

Blood lactate measurement

Three and five minutes after the end of each exercise set, a fingertip blood sample (5 µL) was collected and blood lactate concentration ($[La]_b$) was determined (Lactate Pro, Arkray Inc, Japan). The accuracy of the analyzer was checked before each test using standards.

Statistical analyses

Statistical analyses were carried out using Minitab 14.1. Software (Minitab Inc., Paris, France) and data are presented as means and standard deviations (SD). As data were normally distributed, paired *T*-tests were used to compare mean values for all cardiorespiratory, HHb and repeated sprint ability data between the two repeated sprint ability trials. A two-way repeated measures ANOVA with Tukey's post-hoc tests were used to determine the effect of 'recovery mode' and 'sprint repetition' on cardiorespiratory, HHb and repeated sprint ability parameters throughout the two 6-sprint trials with either active or passive recovery. Significance was set at $P<0.05$.

Results

Graded maximal aerobic test

Mean values for $\dot{V}O_2$max, RER, v $\dot{V}O_2$max, HRmax, $[La]_b$ 3 min after exercise and RPE were 55.1 ± 7.7 ml.min^{-1}.kg^{-1}, 1.3 ± 0.1, 15.4 ± 2.1 km.h^{-1}, 191 ± 8 bpm, 12.2 ± 1.2 mmol.L^{-1} and 18.1 ± 1.1 a.u.

Treadmill data during repeated run-based sprinting

Fig. 1 shows that $AvSp_{mean}$ was lower ($P<0.001$) and Sp %Dec was significantly higher ($P<0.001$) for the 6 sprints during AR compared with PR conditions. All other kinematic parameters were significantly higher ($P<0.001$) for PR compared to AR (**Table 1**). When considering the overall repeated sprint performance, there was a significant (all $P<0.001$) 'recovery mode' effect for all parameters (e.g., MxSp and StFq are presented in **Fig. 2**), whereas a 'sprint repetition' effect was only significant for StFq ($P<0.001$) and MxSp ($P<0.01$). No 'recovery mode' × 'sprint repetition' interactions were identified for any parameter (all $P>0.125$).

Cardiorespiratory data during repeated run-based sprinting

When considering the entire repeated sprint session, mean HR, $\dot{V}O_2$, $\dot{V}CO_2$, RER, RR and V_E values were all significantly higher for AR compared to PR (**Table 2**, $P<0.001$). There was no significant ($P=0.09$) difference in the HR_{max} reached during the trials between AR (178 ± 10 beats.min^{-1}) and PR (174 ± 10 beats.min^{-1}). Mean values of HR, $\dot{V}O_2$ (expressed as percentage of the HR_{max} and $\dot{V}O_2$max), $[La]_b$ (3 min after exercise) and RPE were significantly (all $P<0.05$) higher for AR compared to PR (**Fig. 3**).

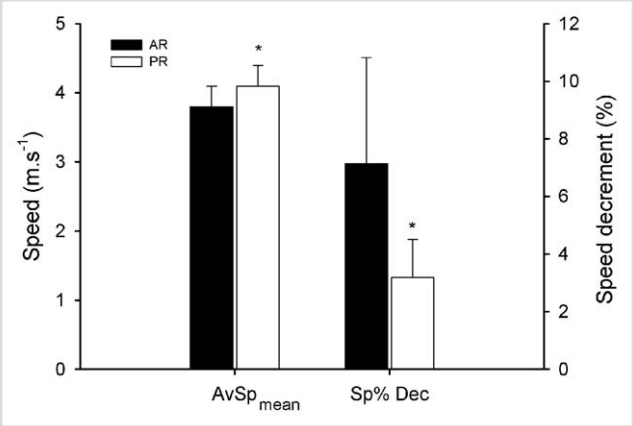

Fig. 1 Mean average speed ($AvSp_{mean}$, $m.s^{-1}$) and percentage of speed decrement (Sp%Dec, %) during the six 4-s all-out sprints with 21 s of active (AR) or passive recovery (PR) between sprints. Values are mean ± SD (n = 10). *Significant difference vs. AR ($P<0.05$).

Table 1 Kinematic data during repeated sprint running with active or passive recovery periods. Comparison between active recovery (AR) and passive recovery (PR) for maximal speed (MxSp; $m.s^{-1}$), acceleration (Acc; $m.s^{-2}$), distance covered (Dist; m) and stride frequency (StFq; Hz).

	AR	PR
MxSp ($m.s^{-1}$)	4.59 ± 0.47	4.85 ± 0.32**
Acc ($m.s^{-2}$)	3.39 ± 0.61	3.73 ± 0.61**
Dist (m)	15.24 ± 1.54	16.48 ± 1.32**
StFq (Hz)	1.84 ± 0.22	1.95 ± 0.19**

** Significant difference vs. AR ($P<0.001$)

Table 2 Cardiorespiratory values during repeated sprint running with active or passive recovery periods. Comparison between active recovery (AR) and passive recovery (PR) for mean (± SD) HR, oxygen uptake ($\dot{V}O_2$), carbon dioxide uptake ($\dot{V}CO_2$), respiratory exchange ratio (RER), respiratory rate (RR) and minute ventilation (V_E) and during repeated sprint running.

	AR	PR
HR (bpm)	160 ± 8	155 ± 8**
$\dot{V}O_2$ ($L.min^{-1}$)	3.64 ± 0.44	2.91 ± 0.47**
$\dot{V}CO_2$ ($L.min^{-1}$)	3.89 ± 0.60	2.96 ± 0.60**
RER	1.1 ± 0.1	1.0 ± 0.1*
RR ($breaths.min^{-1}$)	52.8 ± 6.3	44.6 ± 6.6**
V_E ($L.min^{-1}$)	118.7 ± 11.6	92.2 ± 18.9**

*Significant difference vs. AR ($P<0.05$); ** Significant difference vs. AR ($P<0.001$)

Mean post-exercise $[La]_b$ values were also significantly higher for AR compared to PR conditions at 5 min (13.5 ± 2.5 vs. 12.7 ± 2.2 $mmol.l^{-1}$, $P=0.03$). However, there were no differences between the 3 and 5 min post exercise $[La]_b$ values for either condition (all $P>0.72$). Mean $\dot{V}O_2\Delta$ was significantly lower for AR (13.1 ± 0.5 $ml.min^{-1}.kg^{-1}$) compared to PR (19.7 ± 4.6 $ml.min^{-1}.kg^{-1}$). ▶ **Table 3** shows the mean HR, as well as the maximal and minimal $\dot{V}O_2$ values recorded immediately after each sprint (i.e. during the successive 21 s recovery periods). Except for RR, there were significant 'recovery mode' and 'sprint repetition' effects for all parameters (e.g. $P<0.001$ for $\dot{V}O_2$ relative to body weight), but a 'sprint repetition' × 'recovery mode' effect was not found (e.g., $P=0.79$ for $\dot{V}O_2$) (▶ **Table 3**). For both trials, compared with after sprint 1, $\dot{V}O_2$ was higher after sprint 2, sprint 3, sprint 4, sprint 5 and sprint 6 (▶ **Fig. 4**).

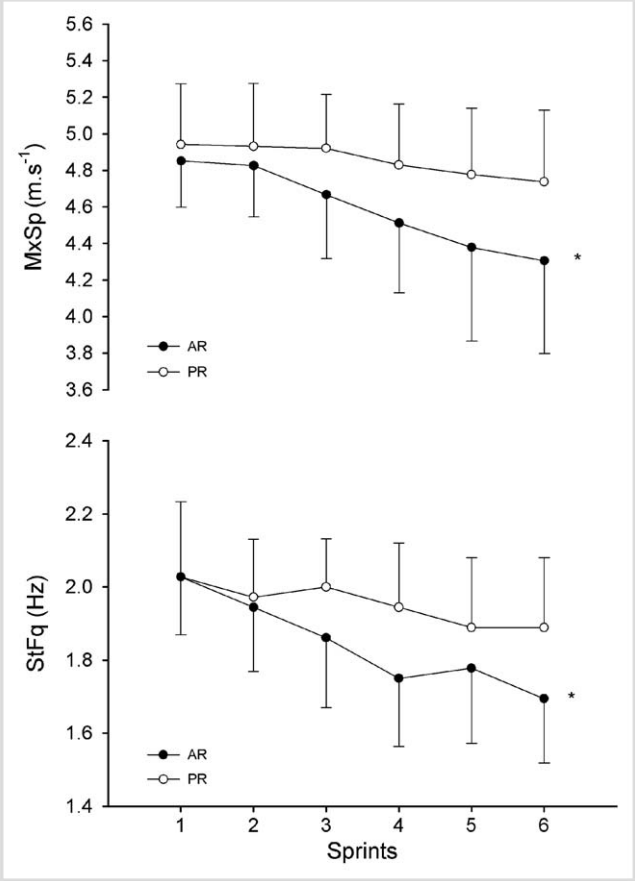

Fig. 2 Maximal speed (MxSp, $m.s^{-1}$) and stride frequency (StFq, Hz) during the six 4-s all-out sprints with 21 s of active (AR) or passive recovery (PR) between sprints. Values are expressed as mean ± SD (n = 10). * Significant difference vs. AR ($P<0.05$).

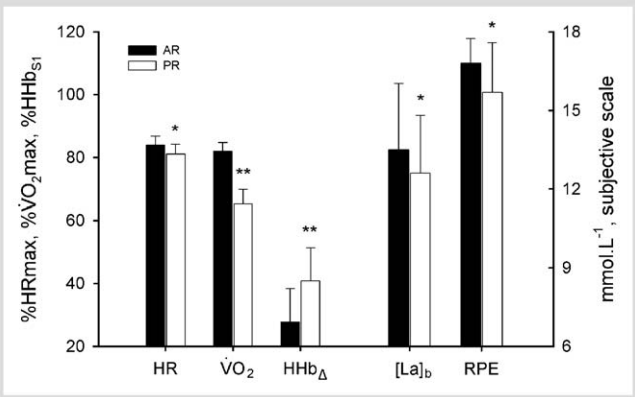

Fig. 3 Percentage of maximal heart rate (%HRmax), percentage of maximal oxygen uptake (% $\dot{V}O_2$ max), mean recovery of HHb during the 21 s recovery periods (expressed as a percentage of maximal HHb during the first sprint (HHb_{rec})), blood lactate ($[La]_b$) and rating of perceived exertion (RPE) measured for the six 4-s all-out sprints with 21 s of active (AR) or passive recovery (PR) between sprints. Values are mean ± SD ($n=10$). * Significant difference vs. AR ($P<0.05$). **Significant difference vs. AR ($P<0.001$).

Deoxyhemoglobin during repeated sprints

There was neither a 'sprint repetition' ($P=0.32$). nor 'recovery mode' ($P=0.81$) effect on tHb, which remained stable during the 6 repetitions for both conditions (100 ± 0, 99.9 ± 8.2, 99.8 ± 7.5, 100.1 ± 10.1, 101.2 ± 11.7, 103.1 ± 12.9% and 100 ± 0, 99.8 ± 9.2,

Table 3 Cardiorespiratory and deoxyhemoglobin values during the 21 s recovery periods after each 4-s sprint. Mean (±SD) minimal and maximal cardiorespiratory and NIRS values during the 21 s active and passive recovery periods following each 4-s sprint. Heart rate (HR), oxygen uptake ($\dot{V}O_2$) and deoxyhemoglobin, (HHb, expressed as a percentage of HHb during the first sprint).

	Sprint #	HR (%HRmax)	$\dot{V}O_2$ (ml.min^{-1}.kg^{-1})		HHb (%)	
			Max	Min	Max	Min
passive	1	63.8±4.7	28.0±9.8	10.8±4.2	100±00	61.4±12.0
	2	79.2±3.2	48.0±12.9	24.8±7.2	101.1±11.7	67.9±7.6
	3	84.7±3.5	52.9±9.8	30.8±7.1	101.9±11.7	60.6±9.5
	4	87.8±2.8	50.9±7.9	32.9±7.1	100.5±14.5	57.5±12.6
	5	88.9±3.2	50.9±9.2	33.5±5.9	100.6±17.1	57.1±12.3
	6	89.1±3.5	50.7±8.2	–	109.1±14.9	–
active	1	66.9±9.2	37.7±11.1	19.4±3.3	100±00	78.1±8.5
	2	83.8±4.4	53.3±9.2	37.6±6.3	109.4±14.2	89.2±13.3
	3	88.4±3.2	54.3±7.6	42.7±6.4	111.3±16.4	87.2±16.4
	4	90.8±3.5	54.9±7.9	44.4±4.9	111.1±18.0	86.6±14.2
	5	92.1±3.2	54.6±7.6	45.3±5.4	113.7±17.4	88.2±17.7
	6	92.5±3.8	54.5±6.6	–	128.8±22.5	–

Data are obtained by averaging the five highest (or lowest) consecutive 1 s values during the considered period. No sprint by recovery mode effect. Since post-exercise recovery was not controlled for, data for the last recovery period (after sprint 6) was not computed

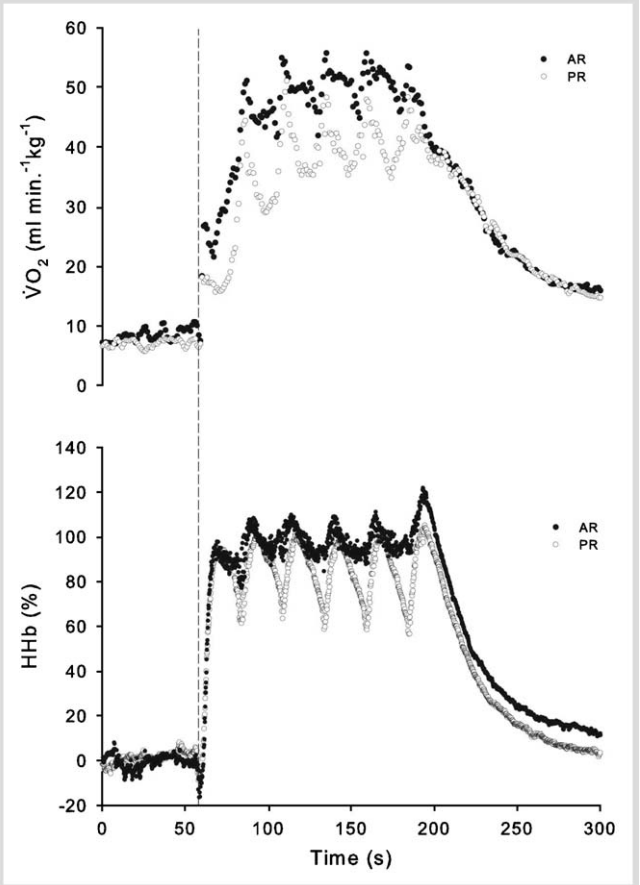

Fig. 4 Mean oxygen uptake ($\dot{V}O_2$) and deoxyhemoglobin (HHb, expressed as a percentage of HHb level at the end of the first sprint) after each of the 6 sprints interspersed with active (AR) or passive recovery (PR) for the ten subjects. Dashed line represents the start of the repeated sprint ability tests. For the sake of clarity, error bars have been omitted.

99.9±9.8, 101.2±9.1, 100.3±11.6, 102.6±13.3% for AR and PR, respectively). Mean HHb level was significantly higher for AR compared with PR (94.4±16.7 vs. 83.4±4.7%, for AR vs. PR, respectively, $P=0.02$), whereas mean HHb_{Δ} was significantly lower for AR versus PR ($P<0.001$, **Fig. 3**). There was no 'sprint repetition' effect for HHb_{Δ} ($P=0.47$), whereas the 'recovery mode' effect was significant ($P<0.01$). There was also no interaction between both factors for HHb_{Δ} ($P=0.62$). The time-course of HHb measured throughout the 6 sprints is presented in **Fig. 4** for both recovery conditions, with values detailed in **Table 3**. There was a significant 'recovery mode' ($P<0.001$) and 'sprint repetition' ($P<0.01$) effect for HHb_{max} as well as a 'recovery mode' effect for HHb_{min} ($P<0.001$). However, there was no 'sprint repetition' x 'recovery mode' effect ($P=0.45$ and $P=0.72$ for HHb_{max} and HHb_{min}, respectively).

Discussion

The present study is the first to compare the effect of active versus passive recovery on repeated sprint running ability using simultaneous recordings of cardiorespiratory and muscle oxygenation parameters. Because of the specificity of exercise mode on muscle recruitment and repeated performance, we expected a progressively greater muscular deoxygenation across repetitions for both active and passive recovery, and that there would only be minor differences in muscle deoxygenation and performance between recovery conditions. In contrast to our hypothesis, results showed that passive recovery was likely to be associated with a constant level of muscle deoxygenation and only a mild impairment of repeated sprint ability. Moreover, repeated sprint ability was significantly reduced when active recovery was performed, as mean running speed was lower and percent speed decrement was higher. This decrease in repeated sprint ability under the active recovery condition was concomitant with an increased oxygen uptake, a greater blood lactate accumulation and a higher level of muscle deoxygenation.

Run-based repeated sprint ability

Due to the higher anaerobic system participation [33] and greater number of muscle groups and joints engaged during running compared with cycling exercise [25], we expected repeated sprint running to be associated with more of a decline in repeated sprint performance than that previously reported for repeated cycling sprints of similar work/rest durations [36]. In contrast, our results showed that the percentage of sprint decrement for active and passive recovery tended to be similar to that of Spencer et al. [36] (7.1±1.1 and 3.2±2.4% vs. 7.4±2.2 and

5.6 ± 1.8 % in the present study versus Spencer's study, respectively). Nevertheless, only repeated comparisons using a unique population can draw accurate and definitive conclusions, so our suggestions should be considered carefully. Comparing recovery conditions during our two distinct run-based repeated sprint ability tests, we found that passive recovery was associated with a low Sp%Dec (3.2 ± 2.4 %). Conversely, active recovery was associated with a more pronounced decrease in repeated sprint ability, as inferred by the significantly lower $AvSp_{mean}$ and significantly higher Sp%Dec (**Fig. 1**). Although active recovery has previously been shown to improve subsequent 6- [34] or 30-s [9] cycle-sprint performance, our findings corroborate on several observations made during intermittent cycling efforts lasting 4 [36,37] and 15 s [14,15], which highlight the detrimental effect of active recovery on repeated high-intensity exercise performance. Inconsistencies amongst previous investigations might be related to recovery durations (4 min in the study by Bogdanis et al. [9]): long periods of active recovery are likely to enhance sprint performance, presumably by enhancing lactate metabolism [1] while allowing sufficient time for oxygen and phosphocreatine to recover. Differences could also be related to inactive deceleration procedures used after the sprint in some studies [34]. Given the positive relationship between $\dot{V}O_2$max and repeated sprint ability [17,35], it is possible that active recovery would have had a lower impact on the findings had our subjects been fitter. Nevertheless, our athletes had similar $\dot{V}O_2$max values to those participants in previous repeated sprint ability studies (i.e., 55.1 vs. 54.2 [9], 53.4 [37] and 54.6 [36] $ml.min^{-1}.kg^{-1}$). Finally, contrary to our hypothesis, we can advance that participation of the stretch-shortening cycle inherent to running [2] is not likely to minimize the detrimental effect that low-velocity active recovery has on performance during the repeated sprint ability test. Cardiorespiratory and NIRS measurements presented below support this suggestion.

Cardiorespiratory parameters during a run-based repeated sprint ability test

Results from the present study show that cardiorespiratory stress (i.e., mean HR, $\dot{V}O_2$, $\dot{V}CO_2$, V_E, RER and RR) was significantly higher during active compared with passive recovery conditions (**Table 1**, **Fig. 3**). Mean rating of perceived exertion was also significantly higher. For the active recovery condition, the increase in $\dot{V}O_2$ required to run at 2 $m.s^{-1}$ resulted in an elevated $\dot{V}O_2$ during the recovery periods (even immediately after the first sprint, **Fig. 4**), as inferred by the significantly lower $\dot{V}O_2\Delta$. This higher O_2 cost of exercise, together with an inadequate O_2 delivery, was likely responsible for the increased muscle deoxygenation observed during active recovery (see below). Spencer et al. [36] suggest that active recovery effectively results in a competition between the O_2 required for both PCr resynthesis and lactate oxidation, as well as that required for the additional recovery work rate. The rationale used by practitioners to support the use of active recovery to improve repeated sprint ability was in part motivated by the observation of a higher rate of $[La]_b$ clearance with active recovery [1], which was thought to be due to an increased oxidation of lactate. Nevertheless, the present run-based study, as well as similar cycling-based studies [36,37], showed to be significantly higher for active compared with passive recovery. Since the measurement of $[La]_b$ reflects the balance between lactate production and removal, it is difficult to determine whether the higher values measured in the present study were attributable to the higher production of lactate (i.e., due to the additional energetic needs), or instead due to a lower capacity for oxidation (i.e., due to O_2 competition). From a metabolic viewpoint, we can speculate that the lower PCr concentration likely to have occurred with active recovery would have created for elevated levels of metabolites (e.g. Pi, adenosine diphosphate (ADP) and adenosine monophosphate (AMP) due to ATP–PCr splitting) that are known to stimulate anaerobic glycolysis. Additionally, the increased levels of muscle deoxygenation (as detailed below) during active recovery might also have triggered further anaerobic participation [11].

Muscle deoxygenation during a run-based repeated sprint ability test

Although performing arterial occlusion during recovery periods has allowed others to confidently estimate muscle oxidative metabolism during exercise [3], this was not feasible in the present experiment because of the running nature of sprint exercises (i.e., time to decelerate, standing position during recovery, etc). Taken as it is (i.e., with no blood flow interruption), NIRS enables an evaluation of the balance between O_2 delivery and $\dot{V}O_2$ in the area of investigation [12]. We chose to focus on HHb as opposed to other NIRS-derived measures (e.g. HbO_2) due to the fact that it can be regarded as being essentially independent of changes in blood volume during exercise [12,21]. Our results showed however that blood volume (inferred from tHb values) was likely stable during the 6 repetitions for both trials, so that interpretation of muscle oxygenation from HHb changes would have been consistent. This contrasts with the changes in tHb reported by Racinais et al. [31] (tHb results shown in a correspondence letter [13]) during repeated cycling sprints, where tHb was shown to vary throughout repetitions. Exercise mode, as well as sprint (i.e., 4 vs. 6 s) or recovery (i.e., 21 vs. 30 s) durations could however account for these differences. It is worth noting that because we did not use a differential pathlength factor, as required for quantitative measurements, we instead normalized our data ('physiological calibration' set based on HHb delta on the first sprint) in order to present accurate and qualitative information suitable for comparison of all subjects across both trials. Thus, HHb (%) was used conceptually to compare muscle deoxygenation changes during the two repeated sprint ability tests [14,15,31].

Although muscle oxygenation has been observed during 6 [31], 15 [14] or 15 to 30 s [15] of repeated sprint-cycling exercises, the present study is the first to show the muscle oxygenation levels during repeated maximal running sprints of 4-s duration under active and passive recovery conditions. Because run-based repeated sprints are associated with high levels of blood lactate accumulation [33] and are responsible for a large recruitment of muscle mass [25], we hypothesized that muscle deoxygenation would increase across sprint repetitions, even for passive recovery. Since we did not find a 'sprint' x 'recovery' interaction, it was not possible to analyze changes between recovery conditions throughout the 6 sprints. Thus, our hypothesis could not be fully examined. Nevertheless, visual examination of HHb time course (**Fig. 4**), as well as the low Sp%Dec under the passive recovery condition suggests that, for very short run-based repeated sprints, the level of muscle oxygenation reached might not be as important to preserve repeated sprint ability.

In contrast to the passive recovery findings, when active recovery was performed, as is done in many sporting activities [16,30,35], muscle deoxygenation was significantly higher

(▶ **Figs. 3, 4**). The impairment of repeated sprint ability with active compared with passive recovery was associated with a significantly higher mean muscle deoxygenation that occurred throughout the 6 sprints, combined with the lower muscle 'reoxygenation' during each of the recovery periods (inferred by the lower HHb_{Δ}). The active running velocity of 2 $m.s^{-1}$, chosen in accordance with a previous investigation into repeated sprint running [38], represented ~45% of our subjects' v $\dot{V}O_2max$; an intensity shown to limit muscle reoxygenation during cycling [15]. Because of the good delta efficiency of running [6,7], especially at low running velocities [7], we hypothesized our low-intensity active recovery to have only a minor effect on muscle deoxygenation and repeated sprint performance. In contrast, present findings suggest that running exercise is not likely to minimize the detrimental effect of active recovery on muscle oxygenation. Recently, Spencer et al. [37] have shown that a low recovery intensity performed on an ergocycle (20% $\dot{V}O_2max$) had comparable effects on muscle metabolism and repeated performance than a moderate one (35% of $\dot{V}O_2max$), suggesting that "any low-to-moderate level of muscle activation will attenuate the resynthesis of PCr and the recovery of power output during repeated short-sprint exercise". Nevertheless, given the complex delta efficiency of running [6,7], it is uncertain as to whether similar results would be observed in the field. Research exploring various recovery intensities or modalities is still needed.

In conclusion, the present study has shown that active compared with passive recovery conditions were associated with a higher oxygen uptake, blood lactate accumulation, and muscle deoxygenation, as well as a reduced repeated sprint running ability. Although the precise influence of system stress metabolite accumulation and muscle deoxygenation cannot be differentiated in the present study, our results confirm the negative impact of active recovery on muscle (re)oxygenation when very short team-sport specific running efforts are repeated. Our findings imply that 'lowering' recovery intensity (i.e., walking or standing, if possible, rather than jogging) during team sport events might be an effective strategy for improving repeated sprint running performance.

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