European Journal of Applied Physiology (2019) 119:1959–1970
https://doi.org/10.1007/s00421-019-04184-9

ORIGINAL ARTICLE

# Physiological adaptations to repeated sprint training in hypoxia induced by voluntary hypoventilation at low lung volume

**Xavier Woorons**[1] · **Grégoire P. Millet**[2] · **Patrick Mucci**[1]

Received: 27 March 2019 / Accepted: 27 June 2019 / Published online: 8 July 2019


## Abstract

**Purpose** This study investigated the effects of repeated-sprint (RS) training in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) on physiological adaptations, RS ability (RSA) and anaerobic performance.

**Methods** Over a 3-week period, eighteen well-trained cyclists completed six RS sessions in cycling either with RSH-VHL or with normal conditions (RSN). Before (Pre) and after (Post) the training period, the subjects performed an RSA test ($10 \times 6$-s all-out cycling sprints) during which oxygen uptake $\left(\dot{V}O_2\right)$ and the change in both muscle deoxyhaemoglobin (Δ[HHb]) and total haemoglobin (Δ[THb]) were measured. A 30-s Wingate test was also performed and maximal blood lactate concentration ($[La]_{max}$) was assessed.

**Results** At Post compared to Pre, the mean power output during both the RSA and the Wingate tests was improved in RSH-VHL ($846 \pm 98$ vs $911 \pm 117$ W and $723 \pm 112$ vs $768 \pm 123$ W, $p < 0.05$) but not in RSN ($834 \pm 52$ vs $852 \pm 69$ W, $p = 0.2$; $710 \pm 63$ vs $713 \pm 72$ W, $p = 0.68$). The average $\dot{V}O_2$ recorded during the RSA test was significantly higher in RSH-VHL at Post but did not change in RSN. No change occurred for Δ[THb] whereas Δ[HHb] increased to the same extent in both groups. [$La_{max}$] after the Wingate test was higher in RSH-VHL at Post ($13.9 \pm 2.8$ vs $16.1 \pm 3.2$ mmol $L^{-1}$, $p < 0.01$) and tended to decrease in RSN ($p = 0.1$).

**Conclusions** This study showed that RSH-VHL could bring benefits to both RSA and anaerobic performance through increases in oxygen delivery and glycolytic contribution. On the other hand, no additional effect was observed for the indices of muscle blood volume and $O_2$ extraction.

**Keywords** Hypoventilation · Hypoxia · Hypoxemia · Cycling

## Abbreviations

| | |
|---|---|
| [HHb] | Muscle concentrations of deoxyhaemoglobin |
| [La] | Blood lactate concentration |
| $[La]_{max}$ | Maximal blood lactate concentration |
| $[O_2Hb]$ | Muscle concentrations of oxyhaemoglobin |
| [THb] | Total haemoglobin |
| HR | Heart rate |
| MPO | Mean power output |
| NIRS | Near-infrared spectroscopy |
| $O_2$ | Oxygen |
| PPO | Peak power output |
| Reoxy[HHb] | Amplitude variation between the peak and nadir [HHb] |
| Reoxy[THb] | Amplitude variation between the peak and nadir [THb] |
| RPE | Rating of perceived exertion |
| RSA | Repeated sprint ability |
| RSE | Repeated-sprint exercise |
| RSH | Repeated sprints in hypoxia |
| RSH-VHL | Repeated sprints in hypoxia induced by voluntary hypoventilation at low lung volume |
| RSN | Repeated sprints in normoxia |
| $SpO_2$ | Arterial oxygen saturation |
| VHL | Voluntary hypoventilation at low lung volume |

Communicated by Susan Hopkins.

✉ Xavier Woorons
xavier.woorons@gmail.com

[1] Univ. Lille, Univ. Artois, Univ. Littoral Côte d'Opale, EA 7369 - URePSSS - Unité de Recherche Pluridisciplinaire Sport Santé Société, F-59000, Lille, France

[2] Institute of Sport Sciences (ISSUL), Faculty of Biology and Medicine, Lausanne, Switzerland

| | |
|---|---|
| $\dot{V}O_2$ | Oxygen uptake |
| $\dot{V}O_2$/HR | Oxygen pulse |

## Introduction

The ability to repeatedly perform maximal or near-maximal bouts of exercise with incomplete recoveries is an important physical requirement in a large number of sports. In team and racket sports for instance, this ability, which has been termed repeated-sprint ability (RSA) (Girard et al. 2011), may be useful to maintain the efficiency of the offensive and defensive actions and, therefore, to increase the chances to win the game (Carling 2013). RSA may also be useful in continuous-cyclic sports which require intermittent high-intensity efforts, such as road or track cycling.

Repeated-sprint ability is a multifactorial fitness component involving both the aerobic and anaerobic metabolism as well as neural factors (i.e., generation of motor command in the motor cortex) (Girard et al. 2011). As such, it can be improved using different training methods (Bishop et al. 2011). However, it seems that the best way to increase sprints performance (i.e. better time or power output of the sprints) is to comply with the concept of training specificity, that is by performing repeated-sprint exercise (RSE). Over the past few years, it has been shown that RSE could be even more effective at improving RSA if performed in hypoxic conditions (RSH). The majority of the studies that investigated the effects of RSH have found larger performance gains than with the same training performed in normoxia (Brocherie et al. 2017).

Noteworthy, the findings reported after RSH have been replicated when the hypoxic conditions were created through voluntary hypoventilation at low lung volume (VHL) (Fornasier-Santos et al. 2018; Trincat et al. 2017). A 35% gain in the number of sprints completed until task failure (open-loop test) has been reported in competitive swimmers who performed six sessions of repeated-sprint training with VHL (RSH-VHL) over a two-week period (Trincat et al. 2017). Conversely, swimmers who carried out the same training with normal conditions did not significantly improve their performance. In another open-loop test, RSA was also largely improved in highly-trained rugby players after four weeks of RSH-VHL (64% increase in the number of sprints), whereas no performance gain was recorded in the normoxic group (Fornasier-Santos et al. 2018). These results are in line with those obtained in open-loop tests after RSH in cycling (+38% in the number of sprints until task failure) (Faiss et al. 2013a) and in double-poling (+58% sprints) (Faiss et al. 2015).

One limitation in these RSH-VHL studies is that few physiological measurements were made. Therefore, no solid explanations can be provided for the mechanisms involved in the RSA improvements. While an increase in maximal blood lactate concentration, and probably in the anaerobic glycolysis, has been reported after high-intensity or repeated-sprint training with VHL (Trincat et al. 2017; Woorons et al. 2016), this may not be sufficient to explain the large gains in RSA performance. Anaerobic glycolysis is generally not a major component of the total energy supply during RSE, whereas phosphocreatine and its resynthesis through the aerobic pathway have been shown to play the most important role (Girard et al. 2011). Furthermore, the contribution of the aerobic metabolism increases throughout RSE and becomes predominant in the final repetitions (McGawley and Bishop 2008). So far, however, no beneficial effect for the aerobic metabolism has been reported after training with VHL either at moderate or high intensity (Woorons et al. 2008, 2016).

One possible explanation for the RSA improvement after RSH-VHL is that this approach would induce similar physiological adaptations as after RSH. In this latter method, a significant increase in muscle blood perfusion has been found after only a few weeks of training (Faiss et al. 2013a, b, 2015). This adaptation would take place mainly in the fast-twitch fibres (Faiss et al. 2013b) and would be the consequence of an enhanced nitric oxide-mediated vasodilation under the effect of hypoxic exposure (Casey et al. 2011). Such phenomenon is particularly interesting for both phosphocreatine resynthesis (Haseler et al. 1999) and the removal of waste metabolites (i.e., inorganic phosphate, hydrogen ions) and, consequently, for the maintenance in repeated-sprint performance. Even though an improved muscle perfusion may not occur to the same extent after RSH-VHL as after RSH, due to the lower hypoxic dose (Woorons 2014), an improved tissue $O_2$ utilization is still possible.

The main goal of the present study was, therefore, to investigate the effects of three weeks of cycling RSH-VHL on oxygen uptake and the indices of muscle oxygenation during a repeated sprint exercise. The second objective was to determine the effects of the same intervention on anaerobic performance using for the first time the Wingate test. We first hypothesized that RSH-VHL could bring an additional beneficial effect for RSA performance through an improvement in $O_2$ utilization (i.e., higher muscle blood volume and/or oxygen extraction). We also assumed that RSH-VHL would improve anaerobic performance to a greater extent than the same training performed in normal conditions.

## Methods

### Subjects

Eighteen male cyclists, competing at regional or national level were recruited to participate in this study. Their physical characteristics (mean ± SD) were age 34.6 ± 11 years, height 177.7 ± 6 cm and body mass 73.2 ± 9 kg. At the time of the experiment the subjects were already well-trained with a training volume of four to five sessions per week on average for a total duration of about 10–12 h. All of the participants were non-smokers, lowlanders and not acclimatized or exposed to altitude (above 500 m) during or over the few months preceding the experiment. Furthermore, none of them had used hypoventilation training in the previous few months of the study. All the subjects were informed about the nature, conditions, and risks of the experiment and gave their written informed consent. The study was approved by the French Ethics Committee for Research in Sports Science and complied with the Declaration of Helsinki (2008).

### Study design

The experimental protocol consisted of performing six fully-supervised training sessions of repeated sprints in cycling over a three-week period. Before and after the training period, an RSA test was performed, followed 48 h later by a Wingate test. All training and testing sessions were carried out at sea level, in an air-conditioned gym which temperature was maintained at around 20 °C. The subjects were matched into pairs for performance level and then randomly assigned to a group that performed the repeated-sprint training either with normal breathing conditions (RSN, $n = 9$) or in hypoxia induced by VHL (RSH-VHL, $n = 9$). Before the start of the experiment, subjects participated in one or two sessions to familiarize with the testing procedures and with the VHL technique which has been used and well described in several previous studies (Woorons et al. 2017).

### Testing sessions

Repeated-sprint ability was tested with a 10 × 6-s all-out cycling sprints (maximal pedalling) with a departure every 30 s (passive rest). The RSA test was undertaken on a magnetically and air-braked ergometer (WattBike Pro, WattBike, UK) which was mechanically calibrated using the principles dynamic calibration rig (Australian Institute of Sport, Belconnen, ACT, Australia). This cycle ergometer has been found to be an accurate and reliable tool for training and performance assessments (Wainwright et al. 2017). Both groups performed the test with normal breathing. Before starting the trial, the subjects completed a standardized warm-up (10 min at low to moderate intensity followed by one single sprint at maximal intensity). The RSA test began after a 5-min period of rest. At the first sprint of the test, subjects were required to achieve at least 95% of the mean power output (MPO) reached in the single sprint, as recommended by Girard et al. (2011). If they failed to do so, they had to restart the set again after a 5-min period of rest. Forty-eight hours after the RSA test, the subjects performed a standard 30-s Wingate test on the same cycle ergometer. This test was preceded by a 10-min warm-up conducted at progressive intensity. For both the RSA and the Wingate test, participants used their own cycling shoes and the Wattbike was set to replicate the dimensions of each participant's own bicycle as closely as possible. This cycle ergometer provides two types of resistance: an air and a magnetic resistance. The load was selected using only the air resistance which ranges from 1 to 10 and provides powers up to 1500 W depending on the pedal rate (WattBike Pro, WattBike, UK). For each subject, the resistance was set relative to his body weight and the maximal power reached over a single 6-s sprint during the familiarization sessions. All participants were strongly encouraged during the tests to maintain their maximum pedal rate. Within the 24 h preceding the tests, they were instructed to avoid high-intensity training and to refrain from consuming caffeine and alcohol. Each single individual subject performed the same test at the same time and day before and after the training period to limit the physiological variations.

### Training sessions

Over a 3-week training period, the subjects had to complete six specific repeated-sprint sessions (two sessions per week separated by 48–72 h) on the same cycle ergometer and with the same resistance as for the testing sessions (i.e., Wattbike Pro). Each repeated-sprint session was preceded by a 10-min warm-up performed at low to moderate intensity. The training sessions consisted of performing 6-s all-out sprints with a start given every 30 s. At the first two sessions, all subjects had to complete two sets of 6 to 8 × 6-s sprints. The number of repetitions was then progressively increased over the course of the training period (two more repetitions per week on average) to reach 3 × 8 sprints at the last session. Each set was separated by three minutes of active recovery (i.e., cycling at low intensity). The RSN group performed the repeated-sprint training with normal breathing while the RSH-VHL group completed the repetitions with VHL (except the recovery between sets which was performed with normal breathing). In this training modality, the subjects were told to start each repetition by doing a normal exhalation and then to hold their breath until the end of the 6-s sprint. A verbal countdown was given in the last 5 s before

the start of each sprint. After each 6-s sprint, the subjects observed a passive rest of 24 s, remaining seated on the cycle ergometer without pedalling.

## Measurements

### RSA test

#### Performance

Peak power output (PPO) and MPO of each 6-s sprint of the RSA test were measured and analysed with the Wattbike Expert software (Wattbike Ltd., Nottingham, UK). The fatigue index was also assessed by calculating the percentage decrement score as follows:

$$(100 \times (\text{total sprint MPO of the set} / \text{ideal sprint MPO of the set})) - 100$$

where total sprint MPO = sum of sprint MPO from all sprints of the set.

ideal sprint MPO = number of sprints (i.e., 10) × highest sprint MPO of the set.

This formula has been found to be the most valid and reliable method for quantifying fatigue in tests of multiple-sprint performance (Glaister et al. 2008).

#### Gas exchange

Gas exchange was continuously recorded during the entire RSA test through a breath-by-breath portable system (K4b$^2$, Cosmed, Rome, Italy). The standardized calibration procedures were performed before the beginning of exercise as recommended by the manufacturer. They included air calibration, turbine calibration with a standard 3000-mL syringe, gas calibration with a certified commercial gas preparation (oxygen 16%, carbon dioxide 5%) and delay calibration to ensure accurate readings during the testing and to check the alignment between the gas flow and gas concentrations. The breath-by-breath measurements included tidal volume, breathing frequency, expired ventilation, oxygen uptake ($\dot{V}O_2$) and carbon dioxide production. Data were analysed over the 6-s sprints as well as during the first 15 s of the recovery periods, in which the main effects were detected.

#### Heart rate

Heart rate (HR) was continuously measured using the K4b$^2$ device (Cosmed, Rome, Italy) presented above, associated with a Polar T34 strap (Polar Electro Inc, Lake Success, NY, USA) which was placed on the chest of the participants. Data were averaged over 6 s, which corresponded to the duration of each sprint. For each sprint, the highest mean values of HR were assessed using a rolling approach since the peak values are often recorded in the few seconds following the sprints. We then analyzed the mean HR values over the entire RSA test and we calculated the oxygen pulse ($\dot{V}O_2$/HR) to obtain a valid index of stroke volume (Whipp et al. 1996).

#### Arterial oxygen saturation

Arterial oxygen saturation ($SpO_2$) was estimated throughout the RSA test with the pulse oximeter Nellcor N-595 (Pleasanton, CA, USA) and with the adhesive forehead sensor Max-Fast (Nellcor, Pleasanton) which was applied above the right orbital area. This sensor has already been used during intense cycling exercise in hypoxia (Amann et al. 2007) and has been shown to provide accurate and reliable estimations of arterial oxygen saturation (Fernandez et al. 2007). An adjustable headband was placed over the forehead sensor to ensure gentle, consistent pressure on the sensor device. $SpO_2$ was recorded in real time at a frequency of 0.5 Hz and collected using a data acquisition system (Score Analysis Software, Nellcor, Pleasanton). Data were then averaged and analyzed over the 6 s in which the minimum values of $SpO_2$ were reached.

#### Near-infrared spectroscopy

During the RSA test, muscle oxygenation was assessed using a near-infrared spectroscopy (NIRS) technique which was well described elsewhere (Boushel and Piantadosi 2000). The NIRS device (Portamon Artinis, Zetten, The Netherlands) was used to estimate changes in muscle oxygenation by placing a triple optode sensor at the lower third of the right-leg vastus lateralis muscle, parallel to the long axis of the muscle and with an interoptode spacing of 40 mm. The probe was attached to the skin with double-sided tape and firmly fastened with an opaque cotton elastic band wrapped around subjects' thigh. Position of the probe was marked at the first testing session with a permanent pen. Subjects were asked to maintain this mark during the whole training period for accurate repositioning at the second testing session. A standard differential path length factor of 4.0 was used in lack of any clear standard value for human quadriceps muscle during cycling sprints (Racinais et al. 2007). All signals were recorded with a sampling frequency of 10 Hz. We then applied a 10th-order low-pass zero-phase Butterworth filter (cut-off frequency 0.1 Hz) (Faiss et al. 2013a) to reduce artefacts and smooth the pedalling-induced perturbations. Concentrations of oxyhaemoglobin ([$O_2$Hb]) and deoxyhaemoglobin ([HHb]) were recorded. Total haemoglobin ([THb]) was calculated by summing [$O_2$Hb] and [HHb] and used as an

index of the regional blood volume. The change (Δ) in [$O_2$Hb], [HHb] and [THb] were assessed from the resting values recorded over the 2 min preceding the start of the test. The measurements were, therefore, normalized from these recordings (arbitrarily defined as 0 µM). Since [$O_2$Hb] might be confounded by perfusion variations and abrupt blood volume changes during sprints due to rapid and forceful muscle contractions (De Blasi et al. 1993), we chose to restrict our analysis to [HHb] and [THb]. In particular, [HHb] has been shown to be independent of [THb] and is closely associated with changes in venous $O_2$ content (De Blasi et al. 1993; Grassi et al. 2003). For both variables, data were averaged over 6 s. During repeated sprint exercise, muscle [HHb] generally reaches its maximum value at the end of the sprints whereas, in the same time, the minimum values of muscle [THb] are recorded (Faiss et al. 2013a). Conversely, nadir and peak values of [HHb] and [THb], respectively, are reached at the end of the recovery period. For each sprint repetition, peak [HHb] (reflecting maximum $O_2$ extraction) and [THb] (reflecting maximum blood volume) were analysed. Furthermore, to determine the muscle reoxygenation capacity during the recovery periods, which is an essential component of RSE, we also calculated and analysed for each sprint the amplitude variations between the peak and nadir [HHb] (Reoxy[HHb], µM) and [THb] (Reoxy[THb], µM) as previously done (Faiss et al. 2013a; 2015; Billaut and Buchheit 2013). To ensure that the peak and nadir values were actually recorded, we adopted a rolling approach, as shown previously (Faiss et al. 2013a). A sliding window averaging data over a 6-s period was thus used for all NIRS variables. It has recently been demonstrated that the use of a digital filter to smooth NIRS data combined with a rolling approach was the best method to determine peaks and nadirs for accurate interpretation of muscle oxygenation trends during repeated sprint exercise (Rodriguez et al. 2018).

#### Rate of perceived exertion and blood lactate concentration

Immediately after the completion of the last sprint, the subjects were asked to evaluate the rating of perceived exertion (RPE) using the Borg scale (0–10). At the third and fourth minute after the end of the test, a blood sample was collected at the earlobe (5 µL) to measure blood lactate concentration ([La]) (Lactate Pro, Akray, Japan). The highest values of the two samples were recorded as the maximal blood lactate concentration ($[La]_{max}$).

### Wingate test

PPO, MPO and the total work (kJ) reached over the 30-s Wingate test were analysed. RPE was assessed just after the end of the test and a blood sample was collected at the 3rd, 4th and 5th minute following its cessation to assess $[La]_{max}$.

### Training data

To evaluate the overall training stimulus, all participants were asked to report both the duration and RPE of each of their training sessions (and competitions if any) over the 3-week training period. Total training stimulus was calculated using the method developed by Foster et al. (2001) which consists of multiplying the RPE of the global session by its duration. Furthermore, PPO and MPO of each sprint of the whole repeated sprint sessions were measured (Wattbike Expert software, Wattbike Ltd., Nottingham, UK). In each subject, $SpO_2$ was continuously measured during two training sessions with the pulse oximeter Nellcor N-595 (Pleasanton, CA, USA) associated to the adhesive forehead sensor Max-Fast (Nellcor, Pleasanton) presented above. The average $SpO_2$ over an entire set as well as the mean $SpO_2$ at the end of the sprints were analysed.

### Statistics

All the results are expressed as mean ± SD. Data were first tested for distribution normality and variance homogeneity. To determine whether there was an effect of training intervention (RSH-VHL vs RSN) and time (Pre vs Post) on all the means of the variables measured during the testing sessions, a two-way repeated-measures ANOVA was used. We also used a two-way ANOVA for repeated-measures (time × sprint number) to analyse within each group the change in MPO, $\dot{V}O_2$ and NIRS data throughout the RSA test. When a significant interaction effect was found, the Bonferroni post hoc procedure was performed to localize the difference. The comparisons between groups of the variables measured during training were made with Student's *T* tests. All analyses were performed with Sigmastat 4.0 software (Systat Software, CA, USA). Null hypothesis was rejected at $p < 0.05$.

## Results

### Training data

All subjects of both groups completed the six specific training sessions within the 3-week period. The total training load of the entire training period was not different between groups (RSH-VHL 3432 ± 465 vs RSN 3513 ± 412, $p = 0.45$). There

was also no difference between RSH-VHL and RSN in the mean number of 6-s sprints completed per subject (107 ± 6 vs 108 ± 8 repetitions, $p = 0.82$) and in the mean power per repetition (873 ± 93 vs 872 ± 71 W, $p = 0.99$). The average $SpO_2$ over an entire set (including the recovery periods) (94.1 ± 2.4 vs. 96.5 ± 0.8%, $p < 0.01$) and the mean $SpO_2$ at the end of the sprints (87.7 ± 3.5 vs 95.6 ± 1.2%, $p < 0.01$) were significantly lower in RSH-VHL than in RSN.

## RSA test

### Performance

The results are presented in Table 1 and Fig. 1.

In the single sprint preceding the RSA test, there was no difference in PPO between RSH-VHL and RSN or between Pre and Post within each group. On the other hand, while MPO was not different between groups at Pre ($p = 0.91$) and Post ($p = 0.75$), it was higher at Post than at Pre in RSH-VHL ($p < 0.01$) but remained unchanged in RSN ($p = 0.66$).

In the RSA test, at either Pre or Post, the percentage decrement score ($p = 0.77$ and 0.26) and the average MPO ($p = 0.86$ and 0.4) were not different between groups. Conversely, both MPO and the percentage decrement score were higher at Post than at Pre in RSH-VHL ($p < 0.01$), whereas they remained unchanged in RSN ($p = 0.2$ and 0.54). For the mean PPO, the ANOVA revealed only a time effect. The analysis of each of the ten 6-s sprints throughout the RSA test showed that MPO was greater at Post than at Pre from the 2nd sprint in RSH-VHL, the magnitude of the difference being more important in the second part of the test (Fig. 1). On the other hand, the ANOVA showed no effect of training intervention for the RSN group.

### Gas exchange

The results are presented in Table 2 and Fig. 2.

During the 6-s sprints, the ANOVA showed no significant effect for the average tidal volume and breathing frequency. On the other hand, there was a time effect for both the mean

**Table 1** Performance results before (Pre) and after (Post) the training period

| | RSH-VHL | | RSN | | ANOVA $p$ value | | |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | *T* | *C* | *T*×*C* |
| Single 6-s PPO (W) | 1120 ± 230 | 1149 ± 249 | 1136 ± 213 | 1146 ± 225 | 0.3 | 0.96 | 0.59 |
| Single 6-s MPO (W) | 995 ± 175 | **1042 ± 186*** | 1006 ± 154 | 1011 ± 134 | **< 0.01** | 0.92 | **0.03** |
| RSA mean PPO (W) | 940 ± 117 | 995 ± 111 | 970 ± 65 | 996 ± 60 | **< 0.01** | 0.88 | 0.29 |
| RSA mean MPO (W) | 846 ± 98 | **911** ± 117* | 834 ± 52 | 852 ± 69 | **< 0.01** | 0.6 | **0.03** |
| RSA $S_{dec}$ (%) | − 12.3 ± 9 | **− 8.2** ± 7* | − 13.4 ± 9 | − 12.0 ± 9 | **0.01** | 0.47 | **0.04** |
| 30-s Wingate PPO (W) | 1136 ± 234 | 1150 ± 258 | 1141 ± 192 | 1142 ± 197 | 0.43 | 0.98 | 0.46 |
| 30-s Wingate total work (kJ) | 92.8 ± 12 | 97.3 ± 14 | 91.3 ± 8 | 91.8 ± 8 | **0.04** | 0.61 | 0.09 |

Significant statistical difference between variables are indicated in bold

Values are mean ± SD

*RSH-VHL* repeated sprints in hypoxia induced by voluntary hypoventilation at low lung volume, *RSN* repeated sprints with normal breathing, *T* time effect, *C* condition effect, *T*×*C*, interaction effect (time × condition), *PPO* peak power output, *MPO* mean power output, *RSA* repeated sprint ability, $S_{dec}$ percentage decrement score

*Significantly different from Pre within group; $p < 0.05$

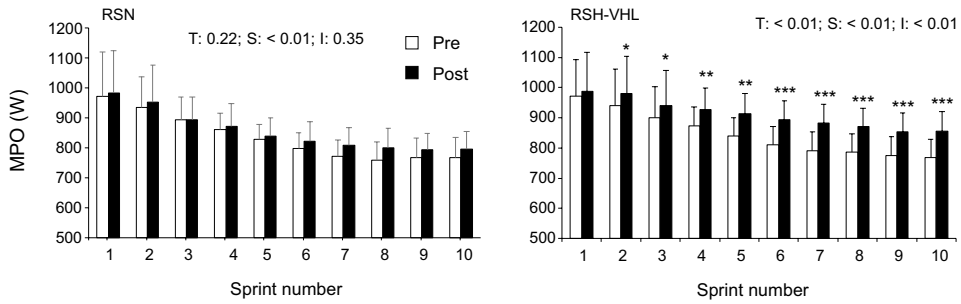



**Fig. 1** Mean power output (MPO) expressed in watts (W) in successive sprints during the repeated-sprint ability test before (Pre) and after (Post) repeated-sprint training with normal breathing (RSN) and in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL). *T* ANOVA time (Pre vs Post) effect, *S* ANOVA sprint number effect, *I* ANOVA interaction effect. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ for significant difference with the same sprint at Pre

**Table 2** Gas exchange before (Pre) and after (Post) the training period

| | RSH-VHL | | RSN | | ANOVA *p* value | | |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | *T* | *C* | *T*×*C* |
| 6-s Spirnts | | | | | | | |
| Vt (L) | 2.67 ± 0.4 | 2.87 ± 0.6 | 2.28 ± 0.4 | 2.52 ± 0.3 | 0.07 | 0.09 | 0.93 |
| Bf (breaths $min^{-1}$) | 42.4 ± 6 | 41.3 ± 5 | 43.6 ± 8 | 41.1 ± 9 | 0.52 | 0.85 | 0.81 |
| $\dot{V}E$ (L $min^{-1}$) | 113 ± 22 | 118 ± 23 | 100 ± 11 | 104 ± 18 | **0.02** | 0.11 | 0.38 |
| $\dot{V}O_2$ (L $min^{-1}$) | 2.37 ± 0.5 | **3.03 ± 0.6***† | 2.39 ± 0.2 | 2.47 ± 0.2 | **< 0.01** | 0.13 | **0.01** |
| $\dot{V}O_2$ (mL $min^{-1}$ $kg^{-1}$) | 33.4 ± 4 | **42.2 ± 6***† | 32.6 ± 6 | 33.5 ± 4 | **< 0.01** | **0.04** | **< 0.01** |
| $\dot{V}CO_2$ (L $min^{-1}$) | 2.75 ± 0.5 | 2.96 ± 0.4 | 2.72 ± 0.2 | 2.84 ± 0.3 | **< 0.01** | 0.90 | 0.79 |
| Recovery periods | | | | | | | |
| Vt (L) | 2.47 ± 0.3 | 2.56 ± 0.4 | 2.40 ± 0.2 | 2.56 ± 0.2 | 0.12 | 0.81 | 0.63 |
| Bf (breaths $min^{-1}$) | 57.3 ± 12 | 61.3 ± 13 | 53.7 ± 10 | 50.8 ± 9 | 0.91 | 0.23 | 0.09 |
| $\dot{V}E$ (L $min^{-1}$) | 141 ± 22 | **157 ± 24*** | 129 ± 15 | 130 ± 20 | **< 0.01** | 0.20 | **0.03** |
| $\dot{V}O_2$ (L $min^{-1}$) | 2.70 ± 0.5 | **3.30 ± 0.7***† | 2.79 ± 0.3 | 2.78 ± 0.2 | **0.03** | 0.32 | **0.03** |
| $\dot{V}O_2$ (mL $min^{-1}$ $kg^{-1}$) | 38.2 ± 8 | **46.2 ± 7***† | 38.1 ± 8 | 37.6 ± 5 | **0.03** | 0.2 | **0.02** |
| $\dot{V}CO_2$ (L $min^{-1}$) | 3.03 ± 0.5 | 3.17 ± 0.3 | 3.13 ± 0.2 | 3.22 ± 0.3 | 0.22 | 0.61 | 0.74 |

Significant statistical difference between variables are indicated in bold

Values are mean ± SD

*RSH-VHL* repeated sprints in hypoxia induced by voluntary hypoventilation at low lung volume, *RSN* repeated sprints with normal breathing, *T* time effect, *C* condition effect, *T*×*C*, interaction effect (time × condition), *Vt* tidal volume, *Bf* breathing frequency, $\dot{V}E$ expired ventilation, $\dot{V}O_2$, oxygen uptake, $\dot{V}CO_2$ carbon dioxide production

*Significantly different from Pre within group

†Significantly different from RSN; $p < 0.05$

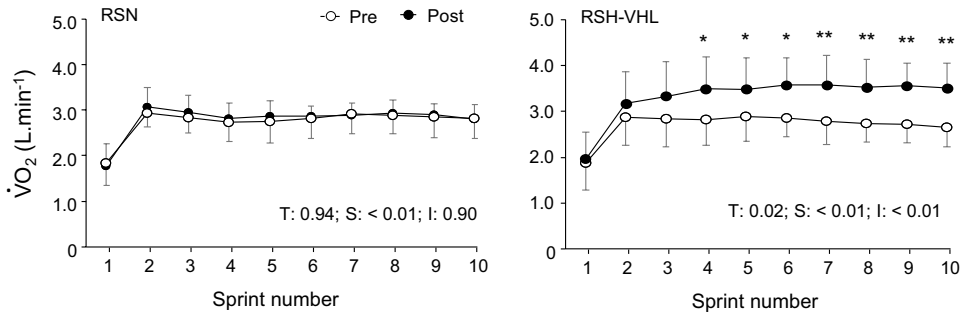
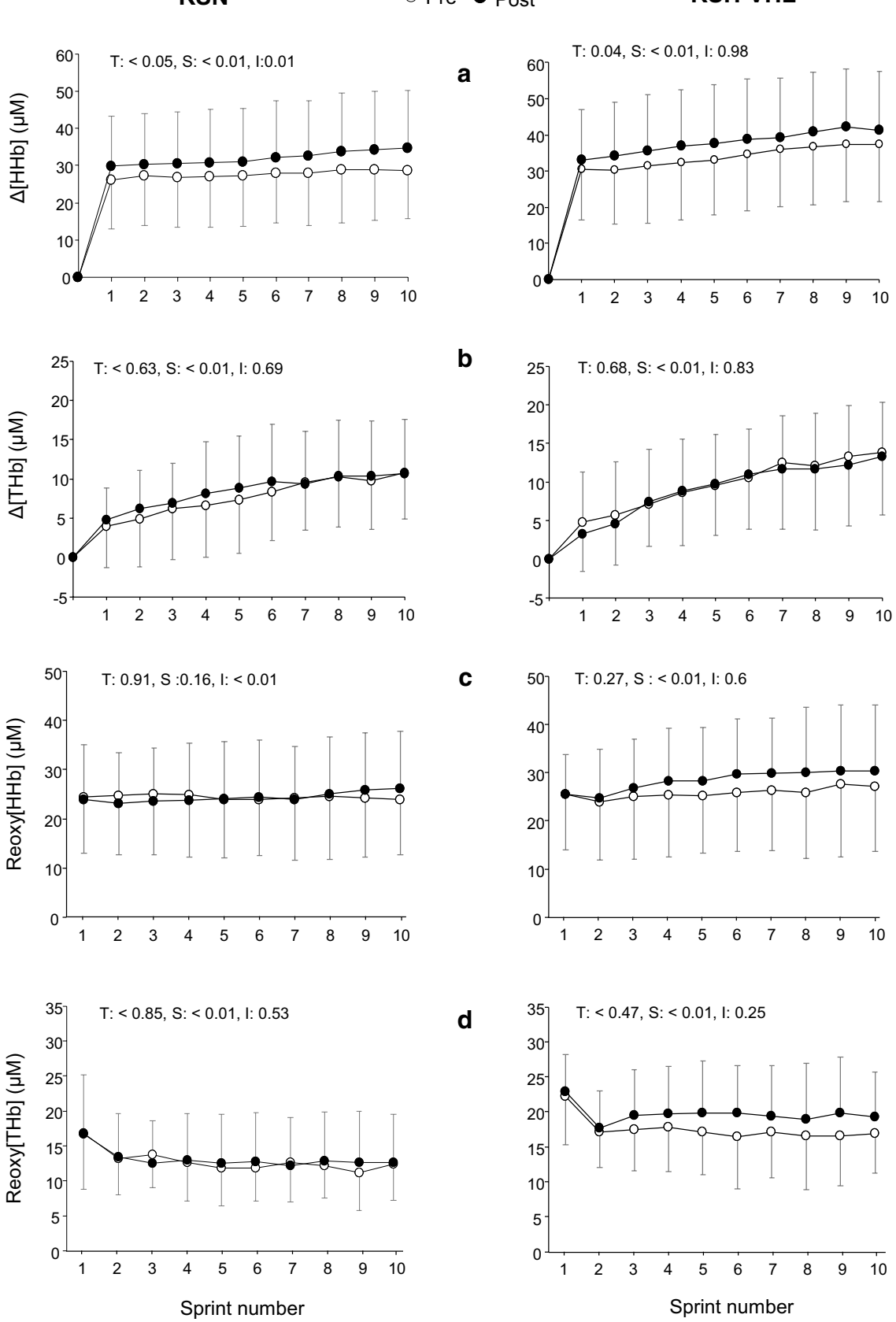



**Fig. 2** Oxygen uptake ($\dot{V}O_2$) measured over the first 15 s of the recovery periods of each sprint during the repeated-sprint ability test before (Pre) and after (Post) repeated-sprint training with normal breathing (RSN) and in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL). *T* ANOVA time (Pre vs Post) effect, *S* ANOVA sprint number effect, *I* ANOVA interaction effect. *$p < 0.05$, **$p < 0.01$ for significant difference with the same sprint at Pre

ventilation and carbon dioxide production and an interaction effect for the average $\dot{V}O_2$. The post-hoc test revealed that while $\dot{V}O_2$ was not different between conditions at Pre ($p = 0.94$), it was higher in RSH-VHL at Post ($p < 0.01$). Furthermore, $\dot{V}O_2$ was also higher at Post than at Pre in RSH-VHL ($p < 0.01$) and remained unchanged in RSN ($p = 0.54$).

In the first 15 s of the recovery periods, the average tidal volume, breathing frequency and carbon dioxide production were not different between RSH-VHL and RSN both at Pre and Post and not different between Pre and Post within each group. The mean ventilation was also not different between the two training interventions at Pre ($p = 0.37$) and Post ($p = 0.11$) but was higher at Post than at Pre in RSH-VHL only ($p < 0.01$). Likewise, compared to Pre, the average $\dot{V}O_2$ was greater at Post in RSH-VHL ($p < 0.01$) but not in RSN ($p = 0.95$). Furthermore, while the mean $\dot{V}O_2$ was not different between groups at Pre ($p = 0.75$), it was significantly higher at Post in RSH-VHL ($p = 0.04$). When analysing each sprint repetition, it appears that $\dot{V}O_2$ was significantly higher at Post than at Pre in RSH-VHL from the 4th sprint, with a stronger statistical difference from the 7th sprint (Fig. 2). On the


RSN
Pre
Post
RSH-VHL
a
T: < 0.05, S: < 0.01, I:0.01
T: 0.04, S: < 0.01, I: 0.98
Δ[HHb] (μM)
b
T: < 0.63, S: < 0.01, I: 0.69
T: 0.68, S: < 0.01, I: 0.83
Δ[THb] (μM)
c
T: 0.91, S :0.16, I: < 0.01
T: 0.27, S : < 0.01, I: 0.6
Reoxy[HHb] (μM)
d
T: < 0.85, S: < 0.01, I: 0.53
T: < 0.47, S: < 0.01, I: 0.25
Reoxy[THb] (μM)
Sprint number
Sprint number

◂**Fig. 3** Changes in the concentrations of deoxyhaemoglobin (Δ[HHb]) (**a**) and total haemoglobin (Δ[THb]) (**b**) from resting baseline and amplitude variations between the peak and nadir [HHb] (Reoxy[HHb]) (**c**) and [THb] (Reoxy[THb]) (**d**) measured by near infrared-spectroscopy at each sprint of the repeated-sprint ability test before (Pre) and after (Post) repeated-sprint training with normal breathing (RSN) and in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL). *T* ANOVA time (Pre vs Post) effect, *S* ANOVA sprint number effect, *I* ANOVA interaction effect

other hand, the ANOVA showed no training effect for $\dot{V}O_2$ in RSN.

### $SpO_2$ and HR

The mean $SpO_2$ ($p=0.86$) and HR ($p=0.33$) recorded at the end of the sprints were not different between Pre and Post both in RSN ($SpO_2$ 96.7 ± 0.5 vs 96.4 ± 0.8%; HR 164 ± 12 vs 162 ± 11 bpm) and RSH-VHL (96.6 ± 1.1 vs 96.9 ± 0.9%; 162 ± 9 vs 162 ± 8 bpm). The average $SpO_2$ ($p=0.45$) and HR ($p=0.77$) were also not different between groups both at Pre and Post. On the other hand, while $\dot{V}O_2$/HR was not different between RSH-VHL and RSN at Pre (16.7 ± 2.8 vs 17.1 ± 2.5 mL $beat^{-1}$; $p=0.81$) it was higher in the former condition at Post (20.4 ± 4.5 vs 17.2 ± 1.8 mL $beat^{-1}$; $p=0.04$). $\dot{V}O_2$/HR was also higher at Post than at Pre in RSH-VHL ($p<0.01$), whereas it remained unchanged in RSN ($p=0.92$).

### NIRS data

Over the ten 6-s sprints, the mean Δ[HHb] was higher at Post than at Pre both in RSH-VHL (33.9 ± 16 vs 37.9 ± 16 µM; $p=0.04$) and in RSN (27.7 ± 13 vs 32.1 ± 15 µM; $p=0.03$). On the other hand, no change was observed in both groups for the average values of Δ[THb] (RSH-VHL 9.8 ± 7 vs 9.4 ± 7 µM; RSN 7.8 ± 6 vs 8.5 ± 6 µM; $p=0.86$), Reoxy[HHb] (RSH-VHL 25.8 ± 13 vs 28.5 ± 11 µM; RSN 24.5 ± 12 vs 24.2 ± 11 µM; $p=0.43$) and Reoxy[THb] (RSH-VHL 17.5 ± 9 vs 19.6 ± 8 µM; RSN 12.9 ± 5 vs 13.1 ± 7 µM; $p=0.45$). There was no difference between groups both at Pre and Post in the mean Δ[HHb] ($p=0.43$), Δ[THb] ($p=0.65$), Reoxy[HHb] ($p=0.63$) and Reoxy[THb] ($p=0.13$). The analysis of the 6-s sprints throughout exercise showed a condition effect for Δ[HHb] both in RSN and RSH-VHL (Fig. 3). Conversely, the ANOVA revealed no effect of the training period for Δ[THb], Reoxy[HHb] and Reoxy[THb] in both groups.

### RPE and maximal blood lactate concentration

At Post compared to Pre, there was no change in both RPE ($p=0.1$) and $[La]_{max}$ ($p=0.74$) in RSH-VHL (RPE 18.2 ± 0.5 vs 18.3 ± 0.9; $[La]_{max}$ 14.0 ± 2.4 vs 14.9 ± 3.7 mmoL $L^{-1}$) and RSN (RPE 17.5 ± 1.1 vs 18.5 ± 0.7; $[La]_{max}$ 14.2 ± 3.0 vs 13.7 ± 4.1 mmoL $L^{-1}$). RPE ($p=0.27$) and $[La]_{max}$ ($p=0.76$) were also not different between groups both at Pre and Post.

### Wingate test

PPO (Table 1) and RPE (RSH-VHL 18.2 ± 0.7 vs 18.6 ± 0.6; RSN 18.4 ± 0.9 vs 18.5 ± 0.7) were not different between Pre and Post within each group ($p=0.43$ and 0.11) and not different between RSH-VHL and RSN both at Pre and Post ($p=0.98$ and 0.79). On the other hand, while MPO was not different between groups both at Pre and Post ($p=0.81$ and 0.35), it was higher at Post than at Pre in RSH-VHL ($p<0.01$) (Fig. 4) and remained unchanged in RSN ($p=0.68$). As for the total work, the ANOVA showed only a time effect. Finally, compared to Pre, $[La]_{max}$ was significantly increased at Post in RSH-VHL ($p<0.01$), whereas it tended to be lower in RSN ($p=0.10$) (Fig. 4). Furthermore, while $[La]_{max}$ was not different between groups at Pre (0.84), it tended to be higher in RSH-VHL at Post (0.08).

## Discussion

This study is the first to investigate the physiological adaptations induced by repeated-sprint training with voluntary hypoventilation. The main finding is that six sessions of cycling RSH-VHL led to significant improvement in RSA which was accompanied by a large increase in $\dot{V}O_2$ without any change in muscle tissue oxygenation. Conversely, the same training carried out with normal conditions did not alter any of these factors. A second important result is that RSH-VHL, unlike RSN, significantly improved the anaerobic performance over a Wingate test.

The improvement in cycling RSA after RSH-VHL confirms the outcomes recently reported after using this approach. In an open-loop test, competitive swimmers increased the number of sprints until task failure by 35% after only a two-week period including six sessions of repeated sprints with VHL (Trincat et al. 2017). In the same kind of test, highly-trained rugby players also significantly improved running RSA with 64% more sprints after four weeks (seven sessions) of RSH-VHL (Fornasier-Santos et al. 2018). In the present study, we used for the first time a close-loop test which allowed the comparison between sprints throughout the entire RSA test. While the results show an average performance gain of 7.7% for MPO after RSH-VHL, it is interesting to note that the improvement occurred as early as the second sprint and was larger with the increasing number of sprints. On the other hand, it is noticeable that in this study, as well as in the previous ones, RSA performance remained unchanged in the group who performed

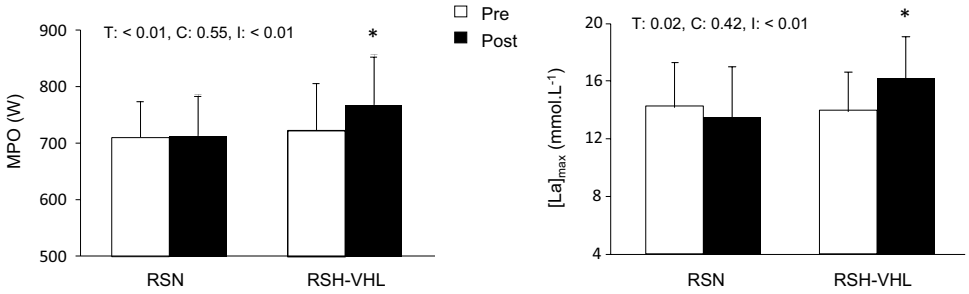



**Fig. 4** Mean power output (MPO) expressed in watts (W) and maximal blood lactate concentration ($[La]_{max}$) reached over the 30-s Wingate test before (Pre) and after (Post) repeated-sprint training with normal breathing (RSN) and in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL). *T* ANOVA time (Pre vs Post) effect, *C* ANOVA condition (RSN vs RSH-VHL) effect, *I* ANOVA interaction effect. $*p < 0.05$ for significant difference with Pre

the training with normal breathing conditions. This is probably due to the fact that the subjects who participated in all these studies were well or highly trained before starting the experiments. Thus, based on the present outcomes and on what has been reported before, it is possible to conclude that RSH-VHL can bring an additional beneficial effect for RSA performance in already well-trained athletes after only a few weeks of training.

We assumed that a greater improvement in RSA after RSH-VHL, if any, may have the same physiological origins as after RSH. Faiss et al. (2015) reported dramatic increases in the amplitude of Δ[THb] during successive sprints after only two weeks (six sessions) of RSH. These larger amplitudes in Δ[THb], that occurred to a much lower extent in the RSN group, reflected an improved blood perfusion and were supposed to be the main cause of the performance gain. Acute exposures to hypoxia are known to provoke compensatory vasodilation to the vascular beds of the skeletal muscle (Casey and Joyner 2012). It has been hypothesized that the repetitions of 'all-out' repeated sprints under hypoxic conditions may induce improvements of $O_2$ utilization, mainly in the fast-twitch fibres (Faiss et al. 2013b), which may consequently induce gains in repeated sprint performance. In the present study, however, none of these physiological adaptations occurred. The change in Δ[THb] from the resting period as well as the reoxygenation levels (i.e., amplitude variations in [THb] and [HHb]) did not vary over the 10 sprints of the RSA test after RSH-VHL and were not different between conditions. Furthermore, while Δ[HHb] was greater after RSH-VHL, the same phenomenon also occurred after RSN. Therefore, this adaptation does not constitute an additional effect of the VHL intervention and probably just reflects a better $O_2$ extraction by the fast-twitch fibres.

The fact that RSH-VHL did not lead to higher muscle blood volume or greater muscle $O_2$ extraction than the RSN group was not expected and contrary to our hypothesis. Unlike an exposure to a hypoxic environment, VHL does not provide a continuous drop in $SpO_2$ since the periods with breath holding must be interspersed with periods with normal breathing (Woorons 2014). In particular, during the periods of recovery following sprints with VHL, subjects have to breathe normally and are, therefore, in normoxic conditions, unlike in RSH. This makes $SpO_2$ return to normal levels or close. Thus, the hypoxic dose is low when exercising with RSH-VHL, as previously quantified in a cycling study (Woorons et al. 2017), and it might be too low to induce specific physiological adaptations at the muscle level, at least for better $O_2$ utilization. The higher RSA performance reported in the present study should, therefore, have other origins than adaptations at the muscle tissue level.

The significant increase in $\dot{V}O_2$ that occurred during the RSA test after RSH-VHL is probably the most interesting outcome of this study. This increase was recorded both during the 6-s sprints and in the recovery periods while, in the same time, $\dot{V}O_2$ remained unchanged in RSN. Noteworthy, when analysing each of the recovery periods following sprints in RSH-VHL, it appears that $\dot{V}O_2$ was higher at Post compared to Pre from the 4th sprint, and that the difference was larger in the second part of the test. This result is consistent with the kinetics of the change in MPO throughout the test. While a larger $\dot{V}O_2$ during sprints probably contributed to the improvement in RSA performance in RSH-VHL, its role may be minor with regards to the high levels of power output that were developed. On the other hand, it has been shown that a greater $\dot{V}O_2$ and, therefore, higher $O_2$ availability are paramount during the recovery periods of RSE (Girard et al. 2011). Indeed, they improve phosphocreatine resynthesis, which is the main energy component in repeated sprints, as well as the removal of waste metabolites (i.e., inorganic phosphate, hydrogen ions). This phenomenon should have, therefore, played a major role in the increase in performance after RSH-VHL.

Considering that RSH-VHL did not increase the index of muscle blood volume and did not improve the index of muscle $O_2$ extraction to a greater extent than in RSN, it is likely

that the higher $\dot{V}O_2$ was due to an increased cardiac output, and subsequently a higher muscle blood flow. It is important to note that previous studies dealing with the acute effects of VHL exercise have reported a higher $\dot{V}O_2$ during the periods of recovery (Woorons et al. 2011, 2017, 2019). This phenomenon would be mainly due to an augmented stroke volume as a consequence of the large and brief inspirations that follow the cessation of the breath holding and that create a "pump effect" (Woorons et al. 2011). Thus, when regularly performing repeated sprints with VHL, one might expect that physiological adaptations occur at the heart level, leading to a greater stroke volume. We hypothesize that this may have happened in this study. Heart rate remained unchanged in RSH-VHL whereas $\dot{V}O_2$/HR was largely augmented. The mechanisms by which RSH-VHL may increase stroke volume are unclear, but one may assume that the "pump effect" could have positive effects on the preload and may, therefore, improve the ventricular diastolic filling.

The 6% gain in anaerobic performance reached in the Wingate test is another interesting result of this study. While it has been shown that VHL training seems to be advantageous for improving the anaerobic glycolysis (Trincat et al. 2017; Woorons et al. 2008, 2010, 2016), no study had ever assessed performance on a specific anaerobic test. Previously, increases of 4% in swimming performance over trials mainly involving the anaerobic glycolysis (100- and 200-m front crawl) have been reported after VHL training at supramaximal intensity (Woorons et al. 2016). These improvements were accompanied by greater lactate concentration and higher rate of lactate accumulation. The scale in the performance gain as well as the significant increase in $[La]_{max}$ recorded in the Wingate test are in accordance with the previous outcomes. Based on all VHL studies carried out so far, one can assert that this approach, although inducing a low hypoxic dose, is beneficial for improving anaerobic performance. This improvement could be due to higher lactate tolerance and possibly to better enzyme activities such as phosphofructokinase or lactate dehydrogenase. Noteworthy, it is likely that the greater effectiveness of the glycolytic system after RSH-VHL was responsible for the improvement in MPO in the first sprints of the RSA test as well as in the single 6-s sprint that preceded it. Anaerobic glycolysis has been shown to contribute to about 40% of the total energy during a single 6-s sprint, its role then declining as sprints are repeated (Girard et al. 2011).

Like in all VHL studies, the main limitation of the present one is that subjects could not be blinded from the RSH-VHL intervention. Thus, the performance gains might be attributable, at least in part, to a placebo effect. If it was so, one could argue that the increase in $\dot{V}O_2$ was the consequence, rather than the cause, of the larger power output after the training period with RSH-VHL. Another limitation is the fact that muscle blood volume and $O_2$ extraction were indirect and local indices derived from NIRS measurements on a single site of a single muscle. It is, therefore, possible that microvascular changes occurred but were not detected. Finally, stroke volume was also indirectly estimated through the calculation of the oxygen pulse and one must remain cautious in the interpretation of the data relative to this variable.

In conclusion, this study demonstrated that RSH-VHL could bring a beneficial effect for both the anaerobic and repeated-sprint performance in cycling. In particular, the RSA gain was likely the consequence of the significant increases in $\dot{V}O_2$ recorded after the training period, possibly due to a greater stroke volume. On the other hand, RSH-VHL did not improve the index of blood perfusion nor led to a greater index of $O_2$ extraction than in the RSN group. In that sense, the physiological adaptations induced by this method distinguish from those of the RSH approach. The exact mechanisms by which RSH-VHL may lead to a higher $\dot{V}O_2$ are yet to be investigated.

**Acknowledgements** We sincerely thank all the subjects who participated in this study for their efforts, time and dedication. We are also grateful to Adrien Combes for his assistance.

**Author contributions** XW and PM conceived and designed research and conducted the experiments. XW, GM and PM analysed data and interpreted the results of the experiments. XW wrote the manuscript. All authors edited, revised and approved the final version of the manuscript.

**Funding** The authors declare that they received no funding for this work.

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

## References

Amann M, Romer LM, Subudhi AW, Pegelow DF, Dempsey JA (2007) Severity of arterial hypoxaemia affects the relative contributions of peripheral muscle fatigue to exercise performance in healthy humans. J Physiol 581:389–403

Billaut F, Buchheit M (2013) Repeated-sprint performance and vastus lateralis oxygenation: effect of limited $O_2$ availability. Scand J Med Sci Sports 23:e185–193

Bishop D, Girard O, Mendez-Villanueva A (2011) Repeated-sprint ability—part II: recommendations for training. Sports Med 41:741–756 **(Review)**.

Boushel R, Piantadosi CA (2000) Near-infrared spectroscopy for monitoring muscle oxygenation. Acta Physiol Scand 168:615–622

Brocherie F, Girard O, Faiss R, Millet GP (2017) Effects of repeated-sprint training in hypoxia on sea-level performance: a meta-analysis. Sports Med 47:1651–1660 **(Review)**.

Carling C (2013) Interpreting physical performance in professional soccer match-play: should we be more pragmatic in our approach? Sports Med 43:655–663

Casey DP, Joyner MJ (2012) Compensatory vasodilatation during hypoxic exercise: mechanisms responsible for matching oxygen supply to demand. J Physiol 590(Pt 24):6321–6326

Casey DP, Curry TB, Wilkins BW, Joyner MJ (2011) Nitric oxide-mediated vasodilation becomes independent of beta-adrenergic receptor activation with increased intensity of hypoxic exercise. J Appl Physiol 110:687–694

De Blasi RA, Cope M, Elwell C, Safoue F, Ferrari M (1993) Noninvasive measurement of human forearm oxygen consumption by near infrared spectroscopy. Eur J Appl Physiol Occup Physiol 67:20–25

Faiss R, Léger B, Vesin JM, Fournier PE, Eggel Y, Dériaz O, Millet GP (2013a) Significant molecular and systemic adaptations after repeated sprint training in hypoxia. PLoS 8:e56522

Faiss R, Girard O, Millet GP (2013b) Advancing hypoxic training in team sports: from intermittent hypoxic training to repeated sprint training in hypoxia. Br J Sports Med 47(Suppl 1):i45–i50

Faiss R, Willis S, Born DP, Sperlich B, Vesin JM, Holmberg HC, Millet GP (2015) Repeated double-poling sprint training in hypoxia by competitive cross-country skiers. Med Sci Sports Exerc 47:809–817

Fernandez M, Burns K, Calhoun B, George S, Martin B, Weaver C (2007) Evaluation of a new pulse oximeter sensor. Am J Crit Care 16:146–152

Fornasier-Santos C, Millet GP, Woorons X (2018) Repeated-sprint training in hypoxia induced by voluntary hypoventilation improves running repeated-sprint ability in rugby players. Eur J Sport Sci 18:504–512

Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA, Parker S, Doleshal P, Dodge C (2001) A new approach to monitoring exercise training. J Strength Cond Res 15:109–115

Girard O, Mendez-Villanueva A, Bishop D (2011) Repeated-sprint ability—part I: factors contributing to fatigue. Sports Med 41:673–694 **(Review)**.

Glaister M, Howatson G, Pattison JR, McInnes G (2008) The reliability and validity of fatigue measures during multiple-sprint work: an issue revisited. J Strength Cond Res 22:1597–1601

Grassi B, Pogliaghi S, Rampichini S, Quaresima V, Ferrari M, Marconi C, Cerretelli P (2003) Muscle oxygenation and pulmonary gas exchange kinetics during cycling exercise on transitions in humans. J Appl Physiol 95:149–158

Haseler LJ, Hogan MC, Richardson RS (1999) Skeletal muscle phosphocreatine recovery in exercise-trained humans is dependent on $O_2$ availability. J Appl Physiol 86:2013–2018

McGawley K, Bishop D (2008) Anaerobic and aerobic contribution to two, 5 × 6-s repeated-sprint bouts. Coach Sport Sci J 3:52

Racinais S, Bishop D, Denis R, Lattier G, Mendez-Villaneuva A, Perrey S (2007) Muscle deoxygenation and neural drive to the muscle during repeated sprint cycling. Med Sci Sports Exerc 39:268–274

Rodriguez RF, Townsend NE, Aughey RJ, Billaut F (2018) Influence of averaging method on muscle deoxygenation interpretation during repeated-sprint exercise. Scand J Med Sci Sports 28:2263–2271

Trincat L, Woorons X, Millet GP (2017) Repeated sprint training in hypoxia induced by voluntary hypoventilation in swimming. Int J Sports Physiol Perform 2:329–335

Wainwright B, Cooke CB, O'Hara JP (2017) The validity and reliability of a sample of 10 Wattbike cycle ergometers. J Sports Sci 35:1451–1458

Whipp BJ, Higgenbotham MB, Cobb FC (1996) Estimating exercise stroke volume from asymptotic oxygen pulse in humans. J Appl Physiol 81:2674–2679

Woorons X (2014) Hypoventilation training, push your limits!. Arpeh, Lille

Woorons X, Mollard P, Pichon A, Duvallet A, Richalet J-P, Lamberto C (2008) Effects of a 4-week training with voluntary hypoventilation carried out at low pulmonary volumes. Respir Physiol Neurobiol 160:123–130

Woorons X, Bourdillon N, Vandewalle H, Lamberto C, Mollard P, Richalet JP, Pichon A (2010) Exercise with hypoventilation induces lower muscle oxygenation and higher blood lactate concentration: role of hypoxia and hypercapnia. Eur J Appl Physiol 110:367–377

Woorons X, Bourdillon N, Lamberto C, Vandewalle H, Richalet JP, Mollard P, Pichon A (2011) Cardiovascular responses during hypoventilation at exercise. Int J Sports Med 32:438–445

Woorons X, Mucci P, Richalet JP, Pichon A (2016) Hypoventilation training at supramaximal intensity improves swimming performance. Med Sci Sports Exerc 48:119–128

Woorons X, Mucci P, Aucouturier J, Anthierens A, Millet GP (2017) Acute effects of repeated cycling sprints in hypoxia induced by voluntary hypoventilation. Eur J Appl Physiol 117:2433–2443

Woorons X, Dupuy O, Mucci P, Millet GP, Pichon A (2019) Cerebral and muscle oxygenation during repeated shuttle run sprints with hypoventilation. Int J Sports Med 40:376–384. https://doi.org/10.1055/a-0836-901110.1055/a-0836-9011 **[Epub ahead of print]**