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Repeated sprint training in hypoxia induced by voluntary hypoventilation improves running repeated sprint ability in rugby players

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The authors declare that they have no conflict of interest.

The authors declare that this research has been carried out in accordance with "The Code of Ethics of the World Medical Association" for experiments involving humans.

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(verso) C. Fornasier-Santos et al.

(recto) Effect of repeated sprint training with hypoventilation

ABSTRACT

PURPOSE: The goal of this study was to determine the effects of repeated sprint training in hypoxia induced by voluntary hypoventilation at low lung volume (VHL) on running repeated sprint ability (RSA) in team-sport players.

METHODS: Twenty-one highly trained rugby players performed, over a 4-week period, **7 sessions** of repeated 40-m sprints either with VHL (RSH-VHL, n = 11) or with normal breathing (RSN, n = 10). Before (Pre-) and after training (Post-), performance was assessed with a RSA test (40-m all-out sprints with a departure every 30 s) until task failure (85% of the peak velocity of an isolated sprint).

RESULTS: The number of sprints completed during the RSA test was significantly increased after the training period in RSH-VHL (9.1 ± 2.8 **vs.** 14.9 ± 5.3; + 64%; $p < 0.01$) but not in RSN (9.8 ± 2.8 **vs.** 10.4 ± 4.7; + 6 %; $p = 0.74$). Maximal velocity was not different between Pre- and Post- in both groups whereas the mean velocity decreased in RSN and remained unchanged in RSH-VHL. The mean SpO_2 recorded over an entire training session was lower in RSH-VHL than in RSN (90.1 ± 1.4 vs. 95.5 ± 0.5 %, $p<0.01$).

CONCLUSION: RSH-VHL appears to be an effective strategy to produce a hypoxic stress and to improve running RSA in team sport players.

Key Words: *Hypoventilation, hypoxia, repeated sprints, training, team-sports, rugby union*

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Introduction

Repeated sprint ability (RSA), which represents the ability to reproduce performance during maximal or near maximal efforts interspersed with brief recovery intervals, is considered a key factor in team sports (Bishop, Girard, & Mendez-Villanueva, 2011; Girard, Mendez-Villanueva, & Bishop, 2011)**.** In sport disciplines such as rugby or soccer, the ability to recover and to repeat sprints is an important fitness requirement. It may for instance influence the final outcome of a game by giving the possibility to win possession of the ball or by preventing the opponents from scoring.

Over the recent years, it has been shown that RSA could be significantly improved when using a new approach of hypoxic training, namely the repeated sprint training in hypoxia (RSH). As compared with the same training performed in normoxic conditions (RSN), sea-level repeated-sprint performance has been found to be more largely increased after 3-4 weeks of RSH in cycling (Faiss, Léger et al., 2013) and in double poling cross-country skiing (Faiss et al., 2015). The improvement in RSA was also greater after RSH in soccer and rugby players (exercise mode = running) (Gatterer et al., 2014; Hamlin, Olsen, Marshall, Lizamore, & Elliot, 2017) as well as in female cyclists (Kasai et al., 2015). A greater increase in the distance covered during an intermittent test has also been reported in rugby players (RSH: +33% vs. RSN: +14%) (Galvin, Cooke, Sumners, Mileva, & Bowtell, 2013). Even though some studies have not reported any additional effect of RSH over RSN for RSA (Brocherie, Girard, Faiss, & Millet, 2015; Goods, Dawson, Landers, Gore, & Peeling, 2015; Montero & Lundby, 2017), a recent meta-analysis has shown that RSH is more efficient than RSN to significantly improve mean repeated-sprint performance (Brocherie, Girard, Faiss, & Millet, 2017).

RSH is based on the repetition of short ‘all-out’ exercise bouts (generally <10 s) interspersed with incomplete recoveries under hypoxic conditions. Its efficiency would rely on the fact that during sprints in hypoxia, the intensity-dependent compensatory vasodilation (Casey & Joyner,

2012) is likely to be maximum (Faiss, Girard & Millet, 2013). These conditions thus favour fast twitch fibres that have a better oxygen extraction capacity than their slow counterparts (McDonough, Behnke, Padilla, Musch, & Poole, 2005). Furthermore, an upregulation of the genes involved in pH control (i.e. monocarboxylate transporter-4 and carbonic anhydrase) has been reported after RSH (Faiss, Léger et al., 2013). This may also participate in the performance improvement induced by this approach.

While most of the RSH studies used simulated altitude (i.e. normobaric hypoxia), two recent repeated-sprint studies induced arterial desaturation through voluntary hypoventilation at low lung volume (VHL) (Trincat, Woorons, & Millet, 2017; Woorons, Mucci, Aucouturier, Anthierens, & Millet, 2017). It has been shown that this breathing modality could lead to both a significant arterial and muscle deoxygenation during exercise (Woorons et al., 2010; 2017), leading to a hypoxic state similar to what is obtained at altitudes above 2000 m (Woorons et al., 2011). Although the hypoxic dose (i.e. scale and time spent at low arterial oxygen saturation) is low with VHL (Woorons, 2014), this kind of approach was effective for improving RSA after RSH induced by VHL (RSH-VHL) in competitive swimmers (Trincat et al., 2017). This improvement was significantly greater than in the group who performed the same repeated sprint training in normoxia (RSN). Of interest is that the magnitude of the RSA enhancement after RSH-VHL in swimming (+ 35%) was in line with what has been previously reported after RSH in cycling (+38%; Faiss, Léger et al., 2013) and in double poling cross-country skiing (+58%; Faiss et al., 2015).

Whether or not an improved RSA could be obtained with RSH-VHL in land based activities is a matter of potential interest. In team sports, the vast majority of the disciplines involve running as main exercise mode (e.g. soccer, rugby, basketball …). The aim of the present study was therefore to determine the effects of four weeks of RSH-VHL on running RSA in highly trained rugby union players. We tested the hypothesis that under these training conditions, the RSA improvement would be greater than with RSN.

Methods

Subjects

Thirty-five highly trained male rugby union players, competing at national level, were selected to participate in this study. Their characteristics were (mean ±SD) age 18.3 ± 1.3 years, height 182 ± 7 cm and body mass 94.9 ± 15.1 kg. At the time of the experiment, which was conducted during the competitive season, the weekly training volume of the participants was ~14 $h.wk^{-1}$ (i.e. three sessions of strength training, five sessions of specific rugby training, one session of physical conditioning and one rugby match). The subjects were all non-smokers, sea-level residents and not acclimatized or recently exposed to altitude (>500 m). None of them had ever used VHL training before the study. All participants had a full medical examination two months before the beginning of the study which did not reveal any respiratory, pulmonary or cardiovascular diseases**.** They presented no sign of respiratory, pulmonary or cardiovascular disease. All subjects (or their parents for the minors) gave their written informed consent after being fully informed about the nature, the conditions and the risks of the study which was approved by the Ethical Commission for Human Research (CER-VD 138/15) and conducted in accordance with the Declaration of Helsinki (2008).

Study design

The experimental protocol consisted of one RSA testing session performed before and after four weeks of repeated sprint training in running (two sessions per week). To include the training sessions with repeated sprints to the training programme of the rugby players without increasing the risk of injuries or overtraining, two usual weekly sessions at high exercise intensity were suppressed. One of these sessions involved lactic anaerobic exercises in cycling and the other running exercises at maximal aerobic velocity.

The subjects were matched into pairs for performance level and then randomly assigned to a group that performed the repeated sprint training either in normoxia (RSN, n=17) or in hypoxia induced

by VHL (RSH-VHL, n=18). Before the start of the experiment, two sessions were organized in order to familiarize the participants with the VHL technique. This breathing technique has recently been used in a cycling repeated sprint study (Woorons et al., 2017). Briefly, it consists of repeating (while exercising) short bouts of breath holding after exhaling down to around the functional residual capacity. Each breath holding is followed by a second exhalation down to residual volume in order to evacuate the carbon dioxide accumulated in the lungs. The VHL technique has recently been included in the updated nomenclature of altitude training methods (Girard, Brocherie, & Millet, 2017).

Training protocol

Over a four-week training period, the rugby players had to complete eight specific repeated sprint training sessions in running (two sessions per week separated by at least 48 h). All sessions were conducted outdoors, on a rugby field, except one which took place in indoor conditions (concrete floor) in order to take both physiological and performance measurements. Each repeated sprint session was preceded by a 10-min standardized warm-up including active mobilisation, dynamic stretching, running drills and three progressive accelerations. During the training sessions, the participants had to perform 40-m all-out sprints with a start given every 30 s. At the first two sessions, subjects performed two sets of 8 x 40 m. 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 x 8 sprints at the last session. Each set was separated by three minutes of semi-active recovery (i.e. walking). The RSN group performed the repeated sprint training with normal breathing while 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 do a normal exhalation just before the start of each sprint, then to hold their breath until the end of the 40-m sprint and finally to perform the second exhalation to empty the remaining air from the lungs. A verbal countdown was given in the last 5 seconds before the start of the

sprints. After each 40-m sprint, the subjects observed a semi-active recovery by walking slowly near the finish line and then started again from this spot.

Testing protocol

One week before (Pre-) and one week after (Post-) the training period, a running RSA test until exhaustion was implemented at sea level and in indoor conditions, on a concrete floor**.** Before starting the test, the subjects completed the same warm-up as during the training sessions. Then they performed two single all-out 40-m sprints to obtain the reference velocity which was calculated from the best time of the two sprints. The RSA test consisted of the repetition of 40-m all-out running sprints with normal breathing, with a start every 30 s. Task failure was declared when peak velocity of the subjects dropped to 85% of the reference velocity or below for the second time (i.e. after a first verbal warning). To avoid any pacing strategies, the subjects were asked to reach at least 95% of the reference velocity in the first sprint. If they did not, they had to start the test again after a resting period of five minutes. To complete as many sprints as possible, the subjects were given very strong verbal encouragements during the entire test. Within the 24 h preceding the RSA test, all participants were instructed to avoid high-intensity training and to refrain from consuming caffeine and alcohol.

Measurements

Testing data. Time of the single 40-m sprint as well as the time of each sprint completed in the RSA test were measured with photocells (Brower timing systems, Draper, Utah 84020 USA). The total number of sprints completed during the RSA test until task failure was evaluated as well as maximal velocity (V_{max}, calculated from the fastest time of the 40-m sprints) and mean velocity (V_{mean}, calculated from all the 40-m sprints). Heart rate (HR) was continuously measured during the RSA test (Polar Oy, Kempele, Finland) while immediately after the completion of the last sprint, the subjects were asked to evaluate the rating of perceived exertion (RPE) using a Borg scale (0-10). At the 3^{rd} and 4^{th} minute after the end of the test, a blood sample was collected at the

finger (5 µL) to measure blood lactate concentration ([La]) (Lactate Pro, Akray, Japan). The highest values of the two samples was recorded as the maximal lactate concentration ($[La]_{max}$).

Training data. To evaluate the overall training stimulus, all participants were asked to report both the duration and RPE of each session (including the rugby matches) over the 4-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. During the repeated sprint training sessions, time of each repetition was measured by the same photocells as in testing sessions and recorded. To assess and compare the acute effects of RSH-VHL and RSN, physiological and performance measurements were made during one repeated sprint session including two sets of 8x40 m. Arterial oxygen saturation (Nonin WristOx2, Minnesota, USA) and HR were continuously measured to obtain the minimum and maximum values respectively. Data were recorded every second and averaged over 6 s for the analyses. A blood sample was taken two minutes after each set for blood lactate concentration. Peak velocity (GPS Unit S4, Catapult, Victoria, Australia) and mean velocity of each set were calculated while RPE was evaluated just after the completion of the second set.

Statistics

Data analysis included only the subjects who completed at least 6 sessions of repeated sprints over the 4-week training period. All data recorded during the testing session in Pre- and Post- were analysed by two-way ANOVA for repeated measures (time x condition). When a significant effect was found, the Bonferroni post-hoc procedure was performed to localize the difference. Training data were also analysed by two-way repeated measures ANOVA (same post-hoc) and completed with student t-tests when necessary. All analyses were made with Sigmastat 3.5 software (Systat Software, CA, USA). Data are presented as mean ± SD. Null hypothesis was rejected at $P<0.05$.

Results

Training data

Fourteen out of the 35 rugby players could not complete at least 6 training sessions because of injuries (mostly occurring during the matches; n=8), illness (n=4) or missed training sessions (n=2). They were therefore excluded of the data analysis. The number of training sessions performed by the remaining players was not different between the RSH-VHL (n=11) and the RSN group (n=10) (6.9 ±0.9 vs. 6.6 ±0.8 sessions; p=0.27). The total training load of the 4-week training period was not different between groups (RSH-VHL: 7784± 1460 u.a; RSN: 7306± 1712 u.a; p=0.34). Over the entire training period, there was no difference between RSH-VHL and RSN in the number of 40-m repetitions completed (132± 23 vs. 127± 8 repetitions, p=0.61) and the mean velocity per repetition (6.1± 0.4 vs. 6.1± 0.4 $km.h^{-1}$, p=0.99).

During the 2 sets of 8 x 40 m all-out sprints, we did not find any difference between groups in V_{mean}, V_{peak} and HR (Figure 2(a), 2(b) and 2(d)). On the other hand, SpO_2 was lower in RSH-VHL than in RSN during most of the repeated sprint exercise (Figure 2(c)). The mean SpO_2 recorded over the two sets was lower in RSH-VHL than in RSN (90.1 ±1.4 vs. 95.5 ±0.5%, p<0.01). The time spent at different levels of SpO_2 is presented in Table II. Blood lactate concentration was not different between RSH-VHL and RSN both at set 1 (9.2 ±3.0 vs. 10.2 ±3.3 $mmol.L^{-1}$, p=0.31) and set 2 (9.8 ± 2.8 vs. 11.0 ± 3.7 $mmol.L^{-1}$, p=0.53). Finally, RPE at the end of the repeated sprint exercise was not different between RSH-VHL and RSN (8.7 ±0.7 vs. 8.3 ±0.5; p=0.23).

Testing data

The results are presented in Table I and Figure 1. No significant differences between groups or between Pre- and Post- within each group were found in the reference velocity that was recorded during the single sprint that preceded the RSA test. The number of sprints completed during the RSA test was greater in Post- than in Pre- in RSH-VHL (+64%; p<0.01) and not different in RSN (+6%; p=0.74). Furthermore, while the number of sprints was not different between groups in Pre-

(p=0.76), it was higher in RSH-VHL in Post- (p=0.02). The percentage of the reference velocity at which task failure occurred was not different between RSH-VHL and RSN both at Pre- (83.1 ±1.3 vs 82.7 ±1.6%) and Post- (83.0 ±1.6 vs 82.9 ±1.2%) (p=0.70) and not different between Pre- and Post- within each group (p= 0.73). The relative velocity (expressed as % of the reference velocity) was not different in Post- compared to Pre- for all sprints of the RSA test in RSN (p=0.27) whereas it was higher in RSH-VHL at sprints 5, 8 and 9 (p=0.04; Figure 1). Moreover, while the relative velocity was not different between groups in the first nine sprints in Pre- (p=0.86), it was higher in RSH-VHL than in RSN during most of the first ten sprints in Post- (p<0.01). We did not find any difference in V_{max} in Post- compared with Pre- in each group and between each group both in Pre- and Post-. On the other hand, V_{mean} decreased in Post- compared to Pre- in RSN (p=0.02) while it remained unchanged in RSH-VHL (p=0.23). The mean velocity was also higher in RSH-VHL than in RSN in Post- (p=0.03) and not different between the two groups in Pre- (p=0.23). Maximal heart rate was higher in RSN than in RSH-VHL in Pre- (p=0.02) and not different between groups in Post- (p=0.45). There was no difference in HR_{max} between Pre- and Post- in both groups. Maximal blood lactate concentration was lower in Post- compared to Pre- in RSN (p=0.01) whereas it remained unchanged in RSH-VHL (p=0.23). Furthermore, $[La]_{max}$ was not significantly different between groups both in Pre- and Post-. The rate of perceived exertion was not different between RSH-VHL and RSN either in Pre- or Post- and not different between Pre- and Post- within each group.

Discussion

This study is the first to investigate the effects of repeated sprint training in hypoxia induced by VHL on running repeated sprint ability in team-sport players. The main finding is that RSA performance was largely improved in highly trained rugby players after four weeks of RSH-VHL whereas it did not change in the RSN group. Another interesting outcome of the present study is the fact that VHL is able to produce a hypoxic stress in a simple fashion applicable in team sports.

The improvement in such RSA test (i.e. "open-loop" carried out until a predefined exhaustion criteria) is similar with those reported in competitive cross-country skiers (Faiss et al., 2015) and in trained cyclists (Faiss, Léger et al., 2013) after RSH in normobaric hypoxia or in swimmers after RSH-VHL (Trincat et al., 2017). In a "close-loop" test (i.e. carried out with a fixed number of repetitions), RSA was also improved compared with RSN in well-trained rugby players (8 x 20 m in running; Hamlin et al, 2017) and in female cyclists (10 x 7 s in cycling; Kasai et al., 2015) after respectively three (6 sessions) and four weeks (8 sessions) of RSH. Furthermore, Brocherie et al. (2015) reported that the ability to repeat maximal sprints including direction changes was enhanced to a greater extent after 5 weeks of RSH than after the same training in normoxia. Finally, Gatterer et al. (2014) found a reduced fatigue during a RSA test after a 5-week RSH compared with RSN in elite youth soccer players.

Conversely, few studies did not report any additional benefit of RSH over RSN (Brocherie et al., 2015; Goods et al., 2015; Montero & Lundby, 2017). This may be potentially explained by some methodological limitations such as the lack of protective pacing measures or an inappropriately large number of performance tests before and after intervention (Girard et al., 2017; Millet, Brocherie, Faiss, & Girard, 2017; Montero & Lundby, 2017). More particularly, it seems that the key factor for obtaining an improvement in RSA performance is the specificity of training relatively to the test implemented in pre- and post-intervention. It is remarkable that in most of the studies in which RSA was not greater after RSH than after RSN, the procedures of the RSA tests

were different from those implemented during the training sessions (Brocherie et al., 2015; Galvin et al., 2013; Goods et al., 2015). For instance, differences in the exercise mode (e.g. cycling vs. running; Goods et al., 2015), in motion direction (e.g. straight-line vs. direction changes; Brocherie et al., 2015), in sprint duration (or distance) (Galvin et al., 2013) or in work-to-rest ratio (Goods et al., 2015) are likely to be responsible for the absence of increased RSA performance after RSH. Contrastingly, to the best of our knowledge, almost all of the studies that reported an improved RSA used the same procedures during training and testing sessions (Brocherie et al., 2015; Faiss, Léger et al., 2013; Faiss et al., 2015; Gatterer et al., 2014; Kasai et al., 2015). Besides, the present study reinforces this assertion. These considerations have important practical implications for the implementation of RSH or RSH-VHL in team sports. Indeed, in these sports, the repeated sprint patterns (i.e. duration, starting velocity, changes in direction, number of sprints, work-to-rest ratio etc…) are rarely predictable. Therefore, it may be recommended to vary the pattern of the repeated sprint sessions for improving the effectiveness of this kind of training in team sports players.

The efficiency of adding a hypoxic stimulus when repeatedly sprinting would rely on an improved vascular conductance and blood perfusion through an enhanced nitric oxide-mediated vasodilation (Casey & Joyner, 2012), which would be both intensity and fibre type dependent (Faiss, Léger et al., 2013; Faiss et al., 2015). In the present study, according to the SpO_2 data collected during training, it is remarkable that the hypoxic stimulus was rather low when performing RSH-VHL. Over the entire session, arterial O_2 desaturation was severe (i.e. SpO_2 <88%) for only about one third of the time and the mean SpO_2 was slightly above 90%. However, interestingly, the hypoxic stimulus was not that low when compared with what happens during RSH performed under ambient hypoxic conditions. Some studies have reported mean SpO_2 values ranging from 91-93% during RSH sessions performed at a simulated altitude of 3000 m (inspired oxygen fraction [FiO_2]=14.5%) (Brocherie et al., 2015; Kasai et al., 2015). Furthermore, Goods et al. (2014) have shown a more potent stress at 3000 m rather than 2000 m with end-exercise values of arterial

oxygen saturation (i.e. 83.6% on average over 3 sets) quite similar to the lowest mean SpO_2 values recorded in the present study (i.e. 84.1%).

While $[La]_{max}$ surprisingly decreased after RSN, it was maintained after RSH-VHL. The lower $[La]_{max}$ measured in RSN at the end of the RSA test at Post-, associated with a lower V_{mean}, may be due to a detraining of the anaerobic glycolytic system. Indeed, the rugby players were highly trained at the commencement of the experiment, in particular regarding the anaerobic metabolism. Yet training sessions at high intensity had to be suppressed to include the repeated sprint sessions whereas the work-to-rest ratio used in the present protocol (i.e. 1:4) may have mainly involved the ATP-Pcr rather than the anaerobic glycolytic system. Another possible explanation is that the repeated sprint training led to a greater lactate clearance. If it was so, this could also explain the maintenance of $[La]_{max}$ in RSH-VHL which would therefore be the result of both a greater lactate tolerance (as a consequence of the glycolytic feature of VHL exercise [Woorons et al., 2010; Woorons, Mucci, Richalet, & Pichon, 2016]) and, possibly, greater lactate clearance.

In addition to the hypoxic effect, an elevated carbon dioxide partial pressure (i.e. hypercapnic effect) has been shown to systematically occur during exercise with VHL (Woorons et al., 2011; Woorons, Gamelin, Lamberto, Pichon, & Richalet, 2014). The main consequence of this phenomenon is to induce a blood, and probably muscle acidosis (Woorons et al., 2010). When this kind of exercise is regularly repeated, it may be possible that physiological adaptations, such as improved buffer capacity, occur within blood or muscle tissue, therefore leading to better pH regulation (Woorons et al., 2008). This would reinforce the potent effects of RSH over both an improvement in buffer capacity and an upregulation of genes involved in pH control (Faiss, Léger et al., 2013). Since the accumulation of hydrogen ions within muscle has been argued to be an important factor leading to fatigue during repeated sprint exercise (Bishop et al., 2011; Girard et al., 2011), one could assume that an improved pH regulation may have played a role in the large performance increase in the RSH-VHL group.

The main limitation of the present field study was that few physiological parameters were measured. The factors involved in the increased RSA performance cannot therefore be clearly identified. We recently reported that RSH-VHL induces a significant muscle deoxygenation (Woorons et al., 2017). Further investigation would thus be useful to determine whether, as reported after RSH (Faiss, Léger et al., 2013; Faiss et al., 2015), RSH-VHL could lead to an improved muscle blood perfusion, which would be favourable to both the speed of phosphocreatine resynthesis (Faiss, Girard & Millet, 2013) and to the removal of waste metabolite (e.g. inorganic phosphate and hydrogen ion). Another limitation of this study was that, like in all VHL studies, it could not be conducted single or double blind. Psychological factors may therefore have influenced the outcomes in the RSH-VHL group. However, this assumption may be rejected in most of the subjects on the basis of the results of RPE or HR_{mean} which were unchanged in Post- compared to Pre- in the RSH-VHL group and which were not different from the RSN group. One may also postulate that a nocebo effect might have occurred in the RSN group, although the subjects were not given any information regarding the method they were tested and that the investigators kept a neutral attitude towards the subjects.

In conclusion, this study demonstrated that RSH-VHL could represent an effective "on-field outdoor" hypoxic training strategy for improving running RSA in team sport players. From a practical point of view, this RSA enhancement can be obtained in specific conditions (running, on grass if needed and without the use of any hypoxic device) and can therefore be easily integrated in a conditioning program. Further investigation is required to assess the underlying physiological mechanisms responsible for the large increase in RSA performance and to determine the optimal parameters (work-to-rest ratio, sprint duration…) of RSH-VHL across the different team sports.

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Figure 1: Mean relative velocity (expressed as % of the reference velocity) in successive sprints during the repeated-sprint ability test before (Pre-) and after (Post-) repeated-sprint training in hypoxia induced by voluntary hypoventilation at low lung volume (RSH-VHL) or with normal breathing (RSN). * $p < 0.05$ for difference with sprint of same number in Pre- within the RSH-VHL group; † $p < 0.05$ for difference with same sprint in the RSN group in Post-.

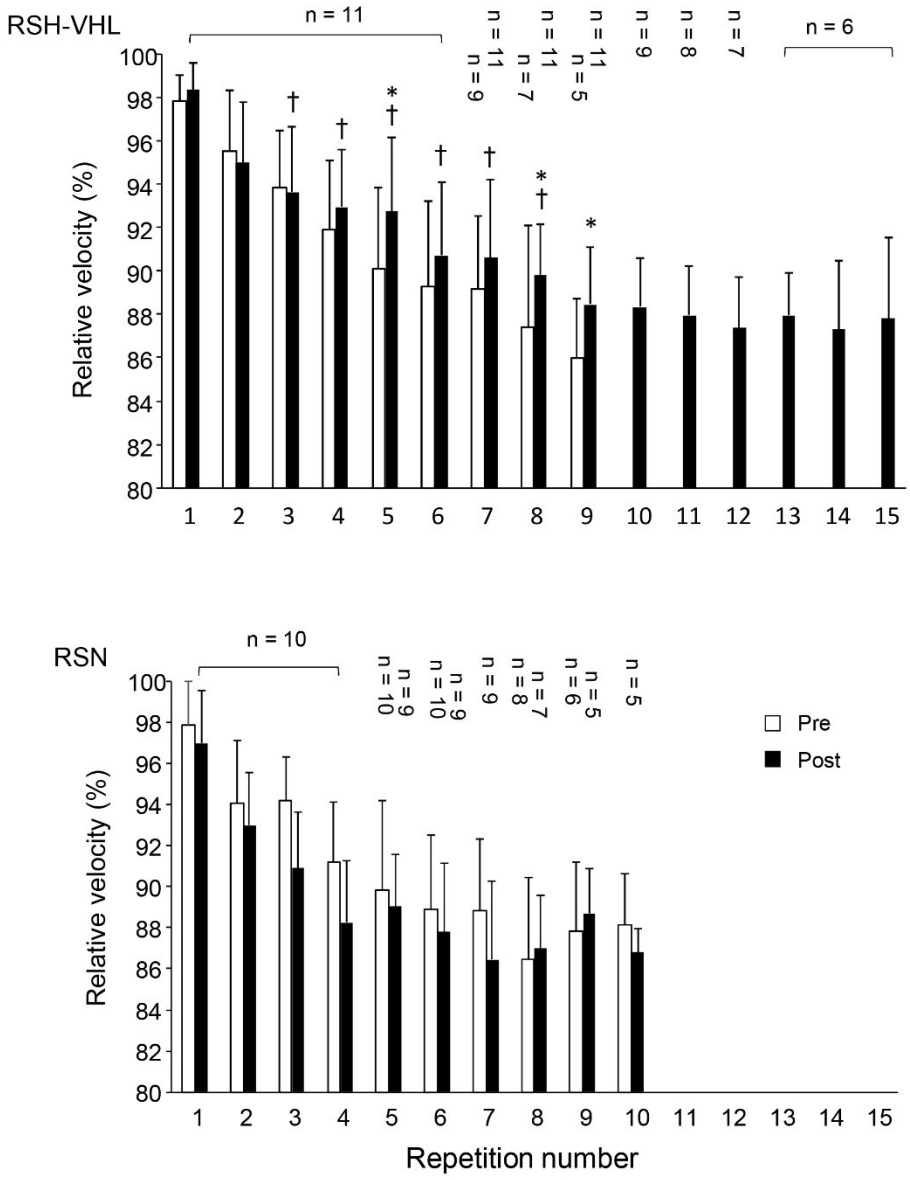

Figure 2: Mean velocity (V_{mean}) (a), peak velocity (V_{peak}) (b), arterial oxygen saturation (SpO_2) (c) and heart rate (HR) (d) during or just after each repetition of repeated sprint training session consisting of performing 2 sets of 8 x 40m all-out sprints with normal breathing (RSN) or in hypoxia induced by voluntary hypoventilation at low lung volume (RSH