Repeated Double-Poling Sprint Training in Hypoxia by Competitive Cross-country Skiers

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ABSTRACT

FAISS, R., S. WILLIS, D.-P. BORN, B. SPERLICH, J.-M. VESIN, H.-C. HOLMBERG, and G. P. MILLET. Repeated Double-Poling Sprint Training in Hypoxia by Competitive Cross-country Skiers. *Med. Sci. Sports Exerc.*, Vol. 47, No. 4, pp. 809–817, 2015. **Purpose:** Repeated-sprint training in hypoxia (RSH) was recently shown to improve repeated-sprint ability (RSA) in cycling. This phenomenon is likely to reflect fiber type-dependent, compensatory vasodilation, and therefore, our hypothesis was that RSH is even more beneficial for activities involving upper body muscles, such as double poling during cross-country skiing. **Methods:** In a double-blinded fashion, 17 competitive cross-country skiers performed six sessions of repeated sprints (each consisting of four sets of five 10-s sprints, with 20-s intervals of recovery) either in normoxia (RSN, 300 m; F_iO_2 , 20.9%; $n = 8$) or normobaric hypoxia (RSH, 3000 m; F_iO_2 , 13.8 %; $n = 9$). Before (pre) and after (post) training, performance was evaluated with an RSA test (10-s all-out sprints—20-s recovery, until peak power output declined by 30%) and a simulated team sprint (team sprint, 3×3 -min all-out with 3-min rest) on a double-poling ergometer. *Triceps brachii* oxygenation was measured by near-infrared spectroscopy. **Results:** From pretraining to posttraining, peak power output in the RSA was increased ($P < 0.01$) to the same extent ($29\% \pm 13\%$ vs $26\% \pm 18\%$, nonsignificant) in RSH and in RSN whereas the number of sprints performed was enhanced in RSH (10.9 ± 5.2 vs 17.1 ± 6.8 , $P < 0.01$) but not in RSN (11.6 ± 5.3 vs 11.7 ± 4.3 , nonsignificant). In addition, the amplitude in total hemoglobin variations during sprints throughout RSA rose more in RSH ($P < 0.01$). Similarly, the average power output during all team sprints improved by $11\% \pm 9\%$ in RSH and $15\% \pm 7\%$ in RSN. **Conclusions:** Our findings reveal greater improvement in the performance of repeated double-poling sprints, together with larger variations in the perfusion of upper body muscles in RSH compared with those in RSN. **Key Words:** ALTITUDE TRAINING, REPEATED SPRINTS, CROSS-COUNTRY SKI, PERFORMANCE

For many years now, coaches and athletes have combined the stress of training with hypoxia to elicit even greater physiological adaptations (12,15,27,40,42) initially by combining hypoxia with low-intensity aerobic exercise to enhance oxygen transport and, more recently, with high-intensity interval training or repeated sprints.

However, a recent review concludes that the additional benefits for sea-level performance of intermittent hypoxic training (IHT) compared with those of similar training under normoxic conditions are strikingly small (14). At the same time, three interesting investigations reveal that a novel approach involving repeated-sprint training in hypoxia (RSH)

does provide additional systematic benefits in comparison with the same training under normoxic conditions (15,17,33).

Because RSH is based on repetition of “all-out” efforts of short (≤ 30 s) duration separated by short periods of incomplete recovery, the efficiency of this strategy probably depends on the maximal intensity of the successive bouts of sprinting (separated by very short periods of recovery) to evoke potent adaptations at the molecular level and, possibly, in the delivery of oxygen (O_2) to the mitochondria (15). Indeed, the intensity of hypoxic training *per se* seems to modulate muscle performance at the molecular level with “adaptations that compensate for the reduced availability of oxygen during exercise” (25). Moreover, the limitation of O_2 delivery during exercise triggers compensatory vasodilation, increasing blood flow in an attempt to maintain sufficient delivery of O_2 to muscles to an extent dependent on the intensity of the exercise (10).

Repeated sprints under hypoxic conditions may benefit from such adaptive mechanisms, perhaps stimulating the diffusion of O_2 into working muscles (10,15). We have reported that the repeated-sprint ability (RSA) of trained cyclists improved significantly after as few as eight sessions of RSH (15). In that study, the additional benefit of RSH

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was thought to involve improved O_2 extraction by the fast-twitch fibers (FT) (which are those predominantly recruited during sprints (22)) as a result of the high-intensity repeated sprinting under hypoxic conditions (28). Greater amplitudes of muscle blood perfusion variations suggesting enhanced muscle blood flow supported this hypothesis of greater O_2 use by FT after RSH.

Accordingly, physical activity involving extensive recruitment of FT may benefit the most from RSH. Of interest in this context is the fact that upper arm muscles contain a high proportion of FT (26). For example, in professional tennis players, two-thirds of all the fibers in the *m. triceps brachii* are FT, whereas the corresponding value for the *m. vastus lateralis* is only one-third (34). Moreover, in elite cross-country (XC) skiers, the proportions of FT were also shown to be higher in the *triceps brachii* than those in the *vastus lateralis* muscles (32).

Consequently, if RSH improves the extraction of O_2 by FT, the benefits seen in trained cyclists should certainly occur in XC skiers as well. During double-poling XC skiing, upper body muscles contribute substantially to power production (24), and even if this sport is thought to be primarily aerobic, recent developments include the introduction of shorter racing formats, such as individual and team sprints. Such events require greater anaerobic capacity and upper body power (35) and, together with the repeated changes in velocity and intermittent sprints that occur during mass start races, underline the decisive role of sprinting ability for the outcome of a competition. Clearly, improving RSA may give XC skiers (including top athletes) a competitive edge.

Nonetheless, the potential associated benefits for the ability to repeat sprints under normoxic conditions after RSH or normoxic training (RSN) have never been compared in a randomized, double-blind, controlled study on XC skiers. Such a comparison revealed that repeated-sprint training under hypoxic conditions can improve subsequent performance. Here, XC ski double poling was selected as a model to elucidate the influence of RSH training on the upper body performance of highly trained athletes in a reproducible and controlled manner. Moreover, de/reoxygenation of the *triceps brachii*, a muscle group containing a high proportion of FT fibers, was measured to evaluate potential fiber type-specific adaptations triggered by RSH.

We hypothesized that repeated-sprint training in hypoxia improves the performance of competitive XC skiers more than the same training under normoxic conditions.

METHODS

Subjects

Seventeen highly trained XC skiers (11 men (29.6 ± 8.8 yr, 1.82 ± 0.08 m) and six women (24.2 ± 5.0 yr, 1.68 ± 0.09 m)) (Table 1) were recruited from Swedish national and regional XC ($n = 15$) and biathlon teams ($n = 2$). Subjects were familiar to the double-poling ergometer and to high-intensity

TABLE 1. Subjects' characteristics before (pre) and after (post) repeated double-poling sprint training in normoxia (RSN) or in hypoxia (RSH).

	RSN ($n = 8$)		RSH ($n = 9$)	
	Pretraining	Posttraining	Pretraining	Posttraining
Body mass (kg)	74.7 ± 7.8	74.7 ± 7.6	72.2 ± 7.0	72.3 ± 7.1
Hematocrit (%)	46.0 ± 3.2	45.7 ± 2.3	46.5 ± 2.0	46.1 ± 1.8
Hemoglobin ($g \cdot L^{-1}$)	154 ± 13	152 ± 15	156 ± 7	152 ± 12
VO_{2peak} ($L \cdot min^{-1}$)	4.2 ± 0.8	4.0 ± 0.8	4.1 ± 0.6	4.2 ± 0.7

exercise bouts from their usual training practice and competitive background. Subjects were all nonsmokers and lowlanders. None of the subjects were acclimatized or recently exposed to altitude, and they were not exposed to an altitude of more than 500 m during the protocol. The responses induced in male and female athletes by RSH might differ. However, a recent study (36) reported that “men and women matched for initial-sprint work experience similar levels of fatigue and systemic, cerebral, and peripheral adjustments” during repeated-sprint exercise under both normoxic and hypoxic conditions. Subjects provided their written informed consent after the state medical ethics committee approved the experiment (regional ethical review board, Umeå, Sweden Agreement 2013-72-31 M) performed according to the Declaration of Helsinki.

Study Design

Experimental protocol consisted in two testing sessions before (pre) and after (post) a specific repeated-sprint training period of 2 wk (three sessions per week). Subjects were assigned into a specific training group, as follows: repeated-sprint training in hypoxia (RSH, $n = 9$; six males and three females) or repeated-sprint training in normoxia (RSN, $n = 8$; five males and three females).

Performance Tests

RSA test. RSA was tested with 10-s all-out double-poling sprints repeated with incomplete recoveries of 20 s until task failure.

Subjects first performed a 4-min 50-s aerobic warm-up at $1 \text{ W} \cdot \text{kg}^{-1}$ followed by an isolated 10-s all-out sprint, 4-min 50-s active recovery at $1 \text{ W} \cdot \text{kg}^{-1}$, and a second isolated 10-s all-out sprint. After 10 min of passive recovery seated (including 5 min with the right arm kept still to calibrate the near-infrared spectroscopy (NIRS) device), the RSA test started with 1 min at $1 \text{ W} \cdot \text{kg}^{-1}$ before the first sprint.

Subjects were given very strong verbal encouragement to perform as many sprints as possible until task failure. Peak power was defined as the highest power recorded in association with a single stroke during all RSA sprints (always the first or second sprint). Task failure was then set at 70% of this peak power. Subjects were given a warning when they did not reach that value the first time and the test was ended the second time. Subjects were not told about the criterion for task failure or any indication on the number of sprints performed. In addition, to avoid any protective pacing

strategy, subjects were requested to reach at least 95% of the best peak power of the two isolated sprints during the first two sprints, which was the case in all subjects. Unfinished sprints (subjects not being able to pull anymore) were not taken into consideration.

Team sprint simulation aerobic test. Exactly 45 min after the end of the RSA test, subjects were asked to warm up again by completing three incremental steps of 4 min at 1, 1.5, and 2 W·kg⁻¹. Then, after an additional 5-min passive rest, subjects were asked to perform a team sprint simulation (TS) consisting of three bouts of 3-min all-out interspersed with 3-min active recovery at 0.5 W·kg⁻¹. Strong verbal encouragement was given throughout the test, and poling frequency was instructed to be as high as possible at all times to elicit maximal all-out performance. Information about elapsed time was only given during the final minute every 15 s to prevent pacing and to further motivate subjects. A peak in oxygen consumption ($\dot{V}O_{2peak}$) was calculated as the highest 30-s average value during the test. Although residual fatigue from the RSA test was probable, TS was performed for all subjects at the same moment of the testing sequence, allowing for a good comparison of subjects' aerobic capacity (average power of all 3-min bouts).

Equipment and Measured Variables

A specially designed double-poling ergometer (SkiErg; Concept2, Morrisville, VT) was used for all training sessions and performance tests. The ergometer is equipped with XC ski handles and straps (Leki, Kirchheim, Germany). Power is produced by pulling cords spinning a wind resistance flywheel. A damper located on the flywheel housing controls the airflow and, thus, the necessary work to accelerate the flywheel during each stroke. The flywheel deceleration rate (called drag factor by the manufacturer) digitally displayed on the ergometer's interface was used to set and reproduce the resistance individually for each test and training session. Drag factor was set at 130% and 110% of body weight in male and female subjects, respectively. These levels, selected on the basis of pilot tests, were designed to allow each athlete to perform 10–15 pre-sprints before task failure. To simulate natural XC skiing, double-poling movement subjects stood with their own XC ski boots in bindings screwed in a wooden plate. The distance between the bindings and the ergometer was freely chosen by the subjects to mimic their habitual position and reproduced for each test and training session. A wide belt around the subjects' hips was additionally attached to the wall behind them with an elastic band to induce some resistance at the hips to better simulate the double-poling sprinting movement (Fig. 1B).

The ergometer displays instantaneous power output and cycle frequency for each stroke that were recorded externally in a spreadsheet (Excel; Microsoft, Redmond, WA) using a Microsoft ActiveX® software component to extract data live. The ergometer's readings of power output during each sprint were displayed purposefully to both the subjects

FIGURE 1—A. Description of one training session. **B.** Ergometer setup for XC ski double poling.

and investigators in an attempt to further motivate optimal performance. Other values such as HR and oxygen saturation were not revealed. $\dot{V}O_{2peak}$ was determined during TS in pretraining and posttraining with an ergospirometry device (AMIS 2001; Innovision A/S, Odense, Denmark) as the highest 30-s average measured.

Muscle oxygenation, blood lactate, blood pH, hematocrit, blood hemoglobin concentration, arterial oxygen saturation (SO_2), HR, and RPE were measured at rest and at the end of each performance test as well as during the first and sixth training sessions (at rest and after the final sprint).

Muscle oxygenation was measured using an NIRS technique as described elsewhere (6). The NIRS device (Portamon;

Artinis, Zetten, The Netherlands) was used to measure changes in muscle oxygenation by placing a triple optode sensor on the muscle belly of the right arms *m. triceps brachii* with an interoptode spacing of 40 mm. The probe was protected with a thin transparent plastic sheet and then attached to the skin with a double-sided tape and firmly fastened with an opaque cotton elastic band wrapped around the subjects' arms. The position of the probe was marked during pretraining with a permanent pen for accurate repositioning. Differential pathlength factor was set to 3.79 and 4.67 in males and females, respectively (13). All signals were recorded with a sampling frequency of 20 Hz. They were downsampled at 10 Hz using MATLAB (MATLAB Software, Natick, MA) routine resample. Then, a 10th-order low-pass zero-phase Butterworth filter (cutoff frequency, 0.1 Hz) was applied to the resampled signals to remove possible artifacts and smooth the movement-induced perturbations. Automatic detection of the start and end times of the successive sprints was obtained by estimating the filtered deoxyhemoglobin signal upper and lower envelopes using local minima and maxima in a sliding window of 400 samples length (4 s). The starting and end times were obtained as the times of contact between the envelopes and the signal. This allowed determination of maximum and minimum for each signal during the successive sprint and recovery phases.

Concentrations for deoxyhemoglobin ([HHb]) and total hemoglobin/myoglobin ([tHb]) were recorded. Because [HHb] values were proposed to be less sensitive to blood flow variations than oxyhemoglobin (O₂Hb) (11) and changes in O₂Hb signals might be confounded by rapid blood volume changes during sprints (7), only [HHb] and [tHb] were analyzed for relevant interpretations. Differences between maximum and minimum concentrations were defined as the amplitude of the variation for each sprint ($\Delta[tHb]$ and $\Delta[HHb]$), and $\Delta[tHb]$ was used as an index of blood perfusion (15). Thus, for example, at the beginning of each sprint, a maximum in [tHb] is observed (i.e., end of each recovery period) and [tHb] decreases to reach a minimum value at the end of each sprint. The concentration recorded during a 3-min seated rest with no arm movement was set as 100% for standardization (Fig. 3). So, amplitudes for each sprint (i.e., $\Delta[tHb]$ and $\Delta[HHb]$) and the average value for all sprints throughout the RSA test (i.e., $\Delta[tHb]_{av}$ and $\Delta[HHb]_{av}$) were calculated.

Surface EMG signals were recorded from the *triceps brachii*, *latissimus dorsi*, *rectus abdominis*, and *soleus* muscles from one side of the body (randomized, right, or left) using surface electrodes (Ambu A/S, Ballerup, Denmark), TeleMyo 2400 T G2™ data logger (Noraxon, Cologne, Germany), and MyoResearch XP Master Edition® software version 1.08.27 (Noraxon, Scottsdale, AZ). Surface electrodes (Ambu® Blue Sensor N; Ambu A/S, Ballerup, Denmark) were positioned with 20 mm of interelectrode distance on the prepared skin (impedance, <5 k Ω) after muscle belly identification by palpation of the contracted muscle and marked for proper repositioning after testing. Athletes performed a series

of previously practiced 5-s maximal isometric voluntary contractions (MVC) to normalize the data to each individual. Before calculation of EMG variables, all raw signals were processed first to determine the MVC using the IIR filter with a band pass 20- to 400-Hz Butterworth approximation then treated with EMG reduction in the *latissimus dorsi* channel, followed by RMS smoothing with a window of 100 ms and amplitude normalization to the peak value with a window of 250 ms. Calculations were performed to assess the mean amplitude expressed in percentage of MVC during both performance tests. Analyses of the RSA included calculation of the average of all cycles performed during each 10-s sprint period. Thirty-second averages were used for the analysis of the TS data.

Blood lactate concentration was measured using a Biosen lactate measurement device (C-Line Sport; EKF Industrial Electronics, Magdeburg, Germany) from 20- μ L fingertip capillary blood. Single values for blood pH, hematocrit, hemoglobin concentration, and SO₂ were assessed by collecting 20- μ L earlobe capillary blood analyzed immediately in a blood analyser (ABL800 CO-OX Flex; Radiometer, Copenhagen, Denmark). HR was recorded at 1 Hz by telemetry (RS800; Polar Oy, Kempele, Finland). RPE was assessed for the legs, arms, and breathing using a 6–20 Borg scale (4).

Training Protocol. Training consisted of six sessions of repeated double-poling sprint over 2 wk on an XC ski ergometer including each time four sets of five sprints of 10 s interspersed with 20 s of recovery (Fig. 1A). Warm-up and recovery periods between sets were performed with an individualized resistance of 1 W·kg⁻¹.

Between the first and last two sets, recovery was allowed for 4 min 50 s, whereas to promote the highest possible peak power in the final two sets, 9 min 50 s of recovery was allowed between sets 2 and 3.

For all training sessions, subjects wore a comfortable oronasal mask allowing normal breathing. Subjects breathed either room air (for RSN) or hypoxic air (for RSH) provided via the mask through a hose (suspended to the ceiling by a supportive elastic band) connected with a three-way valve to an altitude simulation device (Altitrainer; SMTech, Nyon, Switzerland). For RSH, ambient air was mixed with nitrogen (from pressurized tanks) and inspired oxygen fraction was reduced to 13.8% $\pm$ 0.1% to simulate an altitude of 3000 m. Two independent researchers assigned subjects randomly to RSN or RSH, with groups matched upon subjects' peak oxygen uptake measured during the TS aerobic test in pretraining. Helpers then handled the altitude simulation device to provide either normal air or hypoxic air during training by turning the three-way valve so that two subjects training simultaneously inspired either air from the altitude simulator or from the room in the well-ventilated laboratory at a constant temperature of approximately 24°C. This way, the altitude simulator (with the nitrogen outlet sounding) was running during all training sessions. Subjects and investigators were subsequently unable to know whether training was performed in hypoxia or normoxia, and the

TABLE 2. Parameters measured during supervised double-poling training in the RSN and RSH training groups.

	RSN	RSH
Total work of all training sessions (kJ)	1290 ± 197	1335 ± 137
Mean peak power output (W)	482 ± 149	481 ± 131
Mean HR (bpm)	149 ± 11	144 ± 13
Mean SO ₂ after last sprint of each session (%)	94.3 ± 1.3	86.0 ± 4.2*
Mean RPE (arms)	17.6 ± 0.7	16.9 ± 1.1
Mean RPE (legs)	16.7 ± 1.4	17.0 ± 1.8
Mean RPE (breathing)	18.3 ± 0.4	18.0 ± 1.0

RPE was measured using Borg 6–20 scale.

* $P < 0.01$ for difference with RSN.

protocol was run in a double-blind condition. Subjects were told that all training sessions were to be performed at a given altitude without being told exactly which altitude. Subjects were asked to avoid any strenuous training outside the protocol but to keep their usual aerobic training sessions that were reported in a training diary.

Subjects filled a questionnaire reporting their physical activity and food and drink intake during the 48 h before each testing session. During these 48 h, subjects were asked to refrain from any training or exhaustive activity. All testing sessions were carried out at the same time of the day, starting at 8:00 a.m., and subjects were requested to sleep at least 8 h the night before each test. Because of the extreme intensity of the tests, subjects were asked not to report to the laboratory on an empty stomach. A standard breakfast (bread with jam and water) was therefore advised. Subjects were asked to replicate the last meals and drinks, avoiding alcohol and caffeine intake during the 24 h before each test. A preliminary visit allowed subjects to familiarize with the ergometer by completing a 15-min trial including 2×3 repeated sprints of 10 s.

Statistical Analyses

Data are presented as mean (SD). Performance and blood perfusion changes during RSA test were first evaluated with a two-way (training group–sprint number) general linear model repeated-measures ANOVA with all pairwise multiple comparison procedures (Holm–Sidak method). Performance improvement, muscle oxygenation during RSA, blood

lactate, and other single-variable tests were then evaluated with two-way (training group–time (pre- vs posttraining)) general linear model repeated-measures ANOVA with all pairwise multiple comparison procedures (Holm–Sidak method). All analyses were made using Sigmaplot 11.0 software (Systat Software, San Jose CA). Null hypothesis was rejected at $P < 0.05$.

RESULTS

Total work and training intensity during supervised training were similar during RSN and RSH (Table 2). In addition to specific training in the laboratory, subjects completed a similar amount of aerobic training between pretraining and post-training of 16.6 ± 6.6 and 16.3 ± 6.5 h in RSH and RSN, respectively. Total hypoxic exposure was 4.8 ± 0.2 h in RSH.

RSA performance. Performance results at the pretraining and posttraining are summarized in Table 3. No significant differences between groups were observed in any variable in pretraining. During the RSA test, peak power output [$29\% \pm 13\%$ vs $26\% \pm 18\%$, nonsignificant (NS)] and likewise the average power of all sprints ($18\% \pm 16\%$ vs $22\% \pm 14\%$, NS) increased ($P < 0.01$) to the same extent from pretraining to posttraining in RSH and in RSN. The number of sprints before exhaustion was increased in RSH (from 10.9 ± 5.2 to 17.1 ± 6.8 , $P < 0.01$) but not in RSN (11.6 ± 5.3 and 11.7 ± 4.3 , NS) (Fig. 2). In posttraining, compared with that in pretraining, 10-s peak power in the successive sprints was significantly improved until the 15th sprint in RSH and until the 11th in RSN. Significant group (RSH vs RSN)–time (pretraining vs posttraining) interactions were found in the number of sprints ($F = 11.22$, $P = 0.004$) and total work ($F = 8.11$, $P = 0.01$) performed during the RSA test.

Isolated 10-s sprint peak power output and TS performance increased ($P < 0.01$) similarly in RSH and RSN (Table 3). For instance, the average power output during all TS improved similarly by $11\% \pm 9\%$ and $15\% \pm 7\%$, respectively.

Muscle oxygenation. Successive values in $\Delta[\text{tHb}]$ during the RSA test are displayed in Figure 3. After training,

TABLE 3. Performance results before (pre) and after (post) repeated double-poling sprint training in RSN or in RSH.

	RSN		RSH	
	Pretraining	Posttraining	Pretraining	Posttraining
Single 10-s sprint average power (W)	441 ± 153	539 ± 158*	421 ± 122	534 ± 123*
RSA peak power (W)	437 ± 156	532 ± 158*	415 ± 117	528 ± 121*
RSA best average power (W)	365 ± 124	440 ± 128*	350 ± 91	437 ± 98*
RSA mean power of all sprints (W)	315 ± 107	376 ± 111*	308 ± 94	352 ± 78*
RSA total work of all sprints (kJ)	33.1 ± 11.9	42.0 ± 16.6	30.6 ± 12.8	58.8 ± 24.4***
RSA [La] (mmol·L ⁻¹)	11.0 ± 2.5	12.6 ± 2.1	11.1 ± 2.0	12.3 ± 2.9
RSA SO ₂ (%)	93.2 ± 0.8	93.6 ± 1.4	94.2 ± 1.2	93.5 ± 2.2
RSA blood pH	7.23 ± 0.04	7.22 ± 0.06	7.21 ± 0.05	7.22 ± 0.04
3 × 3 average power (W)	218 ± 62	239 ± 64*	219 ± 49	240 ± 46*
3 × 3 average power during first minute (W)	239 ± 64	264 ± 69*	243 ± 49	272 ± 50*
3 × 3 stroke rate (per minute)	60 ± 4	57 ± 4	64 ± 8	62 ± 9
3 × 3 [La] (mmol·L ⁻¹)	11.7 ± 3.0	11.9 ± 2.8	13.1 ± 2.2	14.1 ± 2.9

RSA parameters were measured immediately after repeated-sprint test.

3 × 3, three times 3-min all-out TS; [La], blood lactate concentration.

* $P < 0.01$ for difference with pretraining.** $P < 0.05$ for difference with RSN.

FIGURE 2—Peak power output (W) in successive sprints during the repeated double-poling sprint test before (pre) and after (post) specific repeated double-poling sprint training in hypoxia (RSH) (A) or in normoxia (RSN) (B).

$\Delta[\text{tHb}]_{\text{av}}$ increased to a greater extent ($F = 35.9$, $P < 0.001$) in RSH (from $212\% \pm 12\%$ to $713\% \pm 21\%$, pretraining to posttraining) than in RSN (from $186\% \pm 19\%$ to $361\% \pm 30\%$, pretraining to posttraining) (Fig. 3B). From pretraining to posttraining, $\Delta[\text{HHb}]_{\text{av}}$ increased significantly in RSH ($+225\%$, $P < 0.01$) but decreased in RSN (-27% , $P < 0.01$). There was a significant training group–time interaction for $\Delta[\text{HHb}]_{\text{av}}$ ($F = 551.8$, $P < 0.001$).

Surface EMG. From pretraining to posttraining, the mean amplitude of the activity of all muscles taken together (expressed as the sum of the percentage of MVC of all muscles) did not change in RSA test neither in RSH ($119.1\% \pm 22.2\%$ vs $121.6\% \pm 17.7\%$, NS) nor in RSN ($118.2\% \pm 27.7\%$ vs $121.3\% \pm 76.2\%$, NS). Similarly, the mean amplitude (all 3-min bouts taken together) did not change during TS neither in RSH ($205.8\% \pm 76.0\%$ vs

$216.9\% \pm 69.6\%$, NS) nor in RSN ($204.0\% \pm 49.8\%$ vs $176.0\% \pm 34.0\%$, NS).

DISCUSSION

The main finding of the present investigation was that with competitive XC skiers, repeated double-poling sprint training in hypoxia (involving large upper body muscle groups) resulted in greater improvement in repeated-sprint performance than analogous normoxic training (RSN). Secondly, the amplitude of variations in blood perfusion during sprints increased to a greater extent in RSH than in RSN. Lastly, these differences were specific to repeated sprinting, i.e., performance in connection with an aerobic simulated team sprint was enhanced to a similar degree by RSH and RSN.

FIGURE 3— $\Delta[tHb]$, successive values in total hemoglobin concentrations' amplitude during sprints (expressed in percent compared with the resting baseline set to 100%) measured by NIRS (A), and $\Delta[tHb]_{av}$, average of all amplitude values during the repeated double-poling sprint test to exhaustion (B) before (pre) and after (post) the specific repeated double-poling sprint training in hypoxia (RSH) or in normoxia (RSN).

The present study confirms the potential of RSH as an innovative hypoxic training method in line with similar benefits and underpinning mechanisms recently reported in cyclists (15). Interestingly, RSH in XC skiers seemed to delay fatigue during an RSA test, with 58% more sprints performed after training. This compares well with the 38% increase in sprint repetition reported in cycling in a strictly similar RSA test (10-s sprints, 20-s recovery period) (15). Whereas the cyclists in the latter performed sprints to exhaustion, XC skiers in this study were stopped during RSA when maximal power output during sprinting did not reach 70% of the best sprint anymore. In fact, this threshold was similar in the cycling study, so it can be argued that RSH in XC skiers was more efficient than that in cyclists. This was expected because we hypothesized that the maximal solicitation of FT during sprints in RSH would challenge adaptive mechanisms at the muscular level for sufficient O_2 extraction/delivery. With a higher proportion of FT in upper arm muscles (32,34) contributing greatly to power production during double poling (24), the mechanism of compensatory vasodilation particularly important in FT when O_2 tension falls (10) is enhanced after RSH. This may thus explain why fatigue was more delayed during RSA in XC skiers than in cyclists.

However, the NIRS measurement methods allowed to record local variations in total hemoglobin/myoglobin content ($\Delta[tHb]$) in the muscle, thus reflecting blood perfusion and possibly blood flow variations (20,39). So, this method provided only an indirect estimation of muscle perfusion variations (and thus blood flow), and our interpretations should be considered with care. Nevertheless, the gains in the amplitude of the variations after RSH may be more important when measured here in *m. triceps brachii* compared with *m. vastus lateralis* we reported recently (15). Eventually, the latter is in accordance with our hypothesis that

performing RSH in activities soliciting more FT fibers (e.g., cross-country double poling with the upper body comprising more FT (22)) would potentiate larger benefits in RSA performance gains. Research on isolated animal muscles containing almost exclusively FT might help elucidate the adaptive mechanisms involved in greater detail.

We hypothesized that XC double poling would substantially benefit from fiber-type selective mechanisms, allowing better O_2 extraction by FT after RSH because upper body muscles are largely contributing to power production (24).

Mechanistically, the increase in cardiac output ($\dot{Q}$) tightly matches the O_2 delivery by elevating blood flow, and interestingly, the rise in $\dot{Q}$ is mainly regulated by peripheral vasodilation changes (with less importance of HR increase) (2). Then, high-intensity intermittent training (e.g., knee extension) was reported to increase the capacity for maximal exercise vasodilation by 20%–30% (9). The latter may in turn partly explain the twofold higher amplitude of blood perfusion variations observed after RSN (Fig. 3B). Whereas vascular conductance may not be a limitation to O_2 transport to the thigh muscle (1), arm muscles may incur a blood flow limitation when other muscles are involved during the exercise (41) such as during intense double-poling exercise known to greatly recruit not only upper body muscles but also trunk and leg muscles (24). This hypothesis is in accordance with the vasoconstrictor signals opposing the vasodilatory metabolites observed during upright whole body exercise (8).

Furthermore, hypoxia *per se* was shown to increase blood flow and vascular conductance during exercise with a regulation by the carotid chemoreceptors (37). Consequently, the threefold increase in arm muscle (*triceps brachii*) perfusion variations during repeated sprinting observed after RSH (Fig. 3B) indicates the cumulative role of hypoxia, possibly challenging to a further extent the adaptations

observed in RSN. Accordingly, a recent study reported a 16% increase in arm blood flow and 6% in arm O₂ extraction after prolonged low-intensity (60% of HR_{max}) XC ski training (5). Because FT have been shown to optimally increase their fraction O₂ extraction during intensive exercise (28), RSH may thus recruit FT maximally and maximize vascular conductance and O₂ extraction to delay fatigue during subsequent RSA in normoxia.

In addition, we stated that the exercise/rest ratio plays an important role in the putative benefits after RSH (29) because it modifies the energetic contribution of glycolysis and the FT recruitment/activation during high-intensity exercise (3,38). Then the rate of postsprint phosphocreatine (PCr) resynthesis is paramount for maintaining power production during sustained RSA (18). If RSH allows increasing muscle blood perfusion and vascular conductance as mentioned previously, it may in turn also elevate locally microvascular PO₂ that is known to reduce PCr breakdown and speed up PCr resynthesis (21). Because hypoxia *per se* was shown to modulate PCr recovery (23), RSH may accordingly optimally delay fatigue during RSA if an optimal exercise/rest ratio is found. In addition, the lack of differences in both groups from pretraining to posttraining in the surface EMG measurements supports the hypothesis of improved metabolic glycolytic activity rather than higher power outputs due to greater recruitment of motor units. Besides, the lack of difference in the aerobic team sprint test between RSH and RSN illustrated that a prolonged intense effort (that is more aerobic by definition) does not benefit to the same extent from RSH with the lesser activation of FT. However, we point out that both isolated sprint and team sprint were improved similarly after RSH and RSN and that there is no downregulation in maximal anaerobic alactic or aerobic exercises after RSH.

Interestingly, a recent update of the current panorama of the hypoxic training methods now distinguishes RSH from traditional IHT methods, predominantly on the basis of other expected adaptive mechanisms (e.g., improvement of the muscle oxidative or buffering capacities) (30). Indeed, a current review questioned the efficiency of IHT and highlighted

that of 20 studies involving IHT, only four bring additional benefits in performance-related variables compared with similar training in normoxia (14).

Finally, a recent study investigated different severity of the hypoxic stress (e.g., at simulated altitude of 2000, 3000, and 4000 m), highlighting that “the higher may not be the better” in the training quality for RSH to improve repeated-sprint performance (19). Further studies could consequently focus on the exercise/rest ratio to optimize RSH benefits and find the optimal balance between maximal exercise intensity and the added hypoxic stress. So far, we can only observe that the chosen simulated altitude (e.g., normobaric hypoxia) of 3000 m seems to be providing a stimulus large enough for inducing additional benefits in RSH. One cannot rule out that in hypobaric hypoxia, the adaptations would be even higher (31) because we reported different responses in nitrosative and oxidative stress between normobaric and hypobaric hypoxia at 3000 m (16).

In conclusion, our present findings demonstrate for the first time that greater improvement in the performance of repeated double-poling sprints can be attained with RSH compared with RSN, with larger variations in the perfusion of active muscles in the former. This adds to the accumulating evidence that RSH represents a promising and potent training strategy promoting repeated-sprint performance enhancement by adding hypoxic stress to repeated-sprint training. We propose that by including RSH sessions in their training regimen, XC skiers can improve their work capacity during sessions of repeated sprints. However, further examination is required to confirm that the underlying mechanisms are fiber specific and to establish definitively the potential for such training to improve performance of team sports.

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