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ORIGINAL ARTICLE

The physiological responses to repeated upper-body sprint exercise in highly trained athletes

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Abstract
Purpose To study performance, physiological and biomechanical responses during repeated upper-body sprint exercise.
Methods Twelve male elite cross-country skiers performed eight 8-s maximal poling sprints with a 22-s recovery while sitting on a modified SkiErg poling ergometer. Force, movement velocity, cycle rate, work per cycle, oxygen saturation in working muscles and pulmonary oxygen uptake were measured continuously. A 3-min all-out ergometer poling test determined VO_{2peak}, and 1 repetition maximum (1RM) strength was determined in a movement-specific pull-down.
Results Average sprint power was 281 ± 48 W, with the highest power on the first sprint, a progressive decline in power output over the following four sprints, and a sprint decrement of 11.7 ± 4.1 %. Cycle rate remained unchanged, whereas work per cycle progressively decreased ($P < 0.05$). m. triceps brachii and m. latissimus dorsi were highly desaturated already after the first sprint (all $P < 0.05$), whereas the response was delayed for m. biceps brachii and m. vastus lateralis. Correspondingly, increases in VO_2 mainly occurred over the first two sprints ($P < 0.05$) and plateaued at approximately 75 % of VO_{2peak}. 1RM correlated with power during the first four sprints and with average sprint power ($r = 0.71$–0.80, all $P < 0.05$), whereas VO_{2peak} correlated with power in the last three sprints ($r = 0.60$–0.71, all $P < 0.05$).
Conclusions The main decrement in upper-body sprint performance was evident in the first five sprints, followed by highly desaturated muscles and a plateau in pulmonary oxygen uptake already after the first 2–3 sprints. While high maximal strength seems important for producing power, aerobic capacity correlates with power in the last sprints.

Keywords Cross-country · Skiers · Cycle rate · Maximum strength · Oxygen saturation · Oxygen uptake · Power output

Abbreviations

ATT	Adipose tissue thickness
BB	m. Biceps brachii
CO_2	Carbon dioxide
F	Force
FIS	International Ski Federation
HHb	Deoxyhemoglobin
LAT	m. Latissimus dorsi
NIRS	Near-infrared spectroscopy
O_2	Oxygen uptake
O_2Hb	Oxyhemoglobin

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PCr	Phosphocreatine
RSA	Repeated sprint ability
SD	Standard deviation
SmO_2	Muscle oxygen saturation
TB	m. Triceps brachii
tHb	Total hemoglobin
VL	m. Vastus lateralis
VO_{2max}	Maximal oxygen uptake
VO_{2peak}	Peak oxygen uptake
W	Work
1RM	1 Repetition maximum
Δl	Average displacement

Introduction

Repeated sprint ability/exercise (RSA) is the ability to perform repeated sprints with brief recovery intervals (Girard et al. 2011; Dawson et al. 1993). Consequently, the most important factors for RSA are rapid and high energy delivery, the ability to convert energy into high power outputs, and the ability to resist fatigue over the repeated sprints (Glaister 2005; Spencer et al. 2005; Gaitanos et al. 1993). With recovery periods that are too short to fully resynthesize phosphocreatine (PCr) in repeated sprint exercise, there is a decreasing absolute contribution from PCr to the total ATP production. As a consequence of the additional energy required from glycolysis, repeated sprints may result in high muscle and blood lactate concentrations (Glaister 2008; Spencer et al. 2006a). Additionally, there is an increasing aerobic energy contribution over repeated sprints (Glaister 2005, 2008), with an elevated oxygen uptake due to restoration of homeostasis as explained in detail by others (Bishop et al. 2011; Spencer et al. 2008). Overall, repeated sprint exercise requires contributions from both the aerobic and anaerobic energy systems, shifting from being mostly anaerobic towards a combination of anaerobic and aerobic. However, the physiological responses to repeated sprint exercise are highly dependent upon the test protocol used, such as the number of sprints and the duration of sprint and recovery periods (Balsom et al. 1992), as well as the physiological characteristics of the subjects (Mendez-Villanueva et al. 2008). Additionally, the magnitude of sprint decrement and subsequent physiological responses are likely to be dependent on exercise mode and the amount of active muscle mass. These aspects still require further examination.

Most studies on RSA have so far focused on exercise modes with activity of the lower extremities. However, in various sports the upper extremities are of high importance or even fully responsible for propulsion (Stoggl et al. 2006a; Hawkeswood et al. 2011; Uzun et al. 2012; Tesch 1983). Since the upper-body is characterized by less muscle mass than the lower limbs, physiological responses during upper-body work may be different from those obtained during leg exercise. For example, the peak oxygen uptake is lower in upper-body exercise, together with slower oxygen uptake kinetics, compared to leg exercise (Fukuoka et al. 2002; Calbet et al. 2005). This may be due to intrinsic factors such as lower capillarization in the upper-body which leads to shorter average transit time and impaired diffusion conditions in the muscle (Calbet et al. 2005; Koppo et al. 2002). While several studies have examined determinants of upper-body power (Stoggl et al. 2006b, 2007), only one study has examined upper-body RSA (Sandbakk et al. 2014). In that study high heart rates and blood lactate values were obtained and reduced cycle rates followed the 7 % sprint decrement over eight 7–8 s sprints in ice sledge hockey poling on ice. However, no kinematic or physiological characteristics significantly correlated with RSA or the sprint decrement. These data correspond well with research on team-sport athletes where the legs produce propulsion (Spencer et al. 2006b). However, there is a lack of understanding about performance, physiological and biomechanical responses during upper-body repeated sprint work and research adopting a more laboratory-based approach is required to further explore the physiological mechanisms related to upper-body RSA.

Therefore, the purpose of the present study was to examine performance, physiological and biomechanical responses during upper-body repeated sprint exercise, and to investigate the physiological and biomechanical factors that may be responsible for power output and the decrement in power over the repeated sprints.

Methods

Participants

Twelve male elite cross-country skiers [age 25.7 ± 6.2 years, body height 180.4 ± 3.4 cm, body mass 75.4 ± 7.1 kg, maximal oxygen uptake (VO_{2max}) in running 73.0 ± 3.6 ml kg^{-1} min^{-1}, International Ski Federation (FIS) points 76 ± 21 and annual training of 612 ± 37 h $year^{-1}$] volunteered to participate in the study. The experimental procedures employed were approved by the Norwegian Regional Ethics Committee and the protocol and procedures verbally explained to each subject prior to obtaining their written informed consent to participate. All participants had performed upper-body strength and endurance training daily over the last 6 months.

Overall design of the study

After an initial warm-up, the participants performed eight bouts of 8 s all-out sprints during isolated upper-body

Fig. 1 Illustration of the setup used during the experiment

double poling using a modified Concept2 ski ergometer with a resisting backward arm pull and a free forward retrieval movement. A new repetition started every 30 s. This protocol was adopted from a previous study by our group where we tested upper body repeated sprints in ice sledge hockey (Sandbakk et al. 2014) and is also typically used as speed endurance training in cross-country skiers. The athletes were seated in an ice sledge hockey seat, such that they could perform arm movements that resembled synchronous double-poling, without lower body propulsion (Fig. 1). Force, movement velocity, movement frequency and oxygen saturation in the m. biceps brachii (BB), m. triceps brachii (TB), m. latissimus dorsi (LAT) and m. vastus lateralis (VL), as well as pulmonary oxygen uptake were measured continuously. Blood lactate concentration was measured every 30 s directly after each sprint. Additionally, peak oxygen uptake (VO_{2peak}) determined during a 3-min all-out ergometer poling test was performed after 15 min of active recovery following the repeated sprints. On a separate day, a 1 repetition maximum (1RM) strength test was conducted in a movement-specific pull-down exercise and VO_{2max} was measured during treadmill running. All participants provided written information about their date of birth, FIS scores and annual training hours using a questionnaire.

Instruments and materials

Poling was performed using a modified Concept2 SkiErg (Morrisville, Vermont, US). The ergometer was adjusted for seated poling exercise to avoid any contribution of power from the lower extremities. Participants were strapped around the pelvis and thighs in a firmly fixed ice sledge hockey seat. The feet were placed freely in front so that generating any resistance force was not possible. This way, the pelvis region functioned as the final body segment mechanically linked to the surroundings.

Reflective markers were placed at the origin of the pulling ropes, i.e., on the pulley where the ropes leave the Concept2 frame, and in the transition between the handles and the ropes on both left and right side. Movement velocity was determined using the Oqus motion capture cameras and the Qualisys Track Manager software (Qualisys AB, Gothenburg, Sweden). Force was measured by a force cell (N-DTS-FS5, Noraxon USA Inc., Scottsdale, Arizona), which was mounted on the rope system of the Concept2, collected by the same software and, thus, fully synchronized with the motion signals. The force cell was calibrated using weights from 1 to 10 kg. A force offset was measured each trial by leaving the Concept2 unloaded and recording force, which was subtracted from the signal in the data analysis.

Near-infrared spectroscopy (NIRS) was used to continuously measure the changes in muscle oxygenation and saturation. NIRS is based on the different optical properties for light in the near-infrared region that exists for the oxygenated and deoxygenated forms of hemoglobin and myoglobin and is described in detail elsewhere (Beekvelt et al. 2001). Four portable NIRS devices (Portamon, Artinis Medical Systems, Netherlands) generating light at 845 and 761 nm were used on top of the BB, TB, LAT, and VL. All NIRS devices were placed directly on the skin on top of the muscle, fixed with adhesive tape and covered with a bandage. Each of the Portamon systems consisted of three LED transmitters and one receiver with fixed source-detector distances of 30, 35 and 40 mm. In order to limit the amount of data, the source-detector distance of 35 mm was used for further analysis of concentration changes in oxyhemoglobin (O_2Hb), deoxyhemoglobin (HHb) and total hemoglobin (tHb). All three source-detector distances were used for calculation of muscle oxygen saturation (SmO_2). Simultaneous NIRS measurements on all four muscles were done with one operating system. Data were sampled at 10 Hz, displayed real-time and stored on disk for off-line analysis. Adipose tissue thickness (ATT) was measured after the test, on top of each muscle and in between source and detector using a skin fold caliper (Holtain Ltd, Crymmych, UK). ATT was 3.3 ± 0.4 mm for BB, which was lower than for TB (7.1 ± 2.0 mm), LAT (6.8 ± 1.4 mm) and VL (5.5 ± 1.8 mm) (all $P < 0.01$).

Pulmonary gas exchange was continuously measured during the test by indirect calorimetry using a Metamax 3 portable analyser (Cortex Biophysik GmbH, Leipzig, Germany) in breath-by-breath mode. The instruments were calibrated against ambient air and a commercial gas of known concentrations of O_2 (16.00 %) and CO_2 (4.00 %) before the start of each test day. The concentration of O_2 and CO_2 of room air was read and the flow transducer was calibrated using a 3 l high-precision calibration syringe (Calibration syringe D, Sensor Medics, Yorba Linda, CA) before testing

a new subject. The flow transducer was exchanged and recalibrated before each test. During testing, the data were transmitted telemetrically and stored on a lab computer after each test. The instruments were carried in a vest which was placed around the athletes upper body during testing. Heart rate was continuously measured using a heart rate sensor (Polar T34, Polar Electro OY, Kempele, Finland). Heart rate and VO_2 were collected simultaneously by the Metamax system (and thereby synchronized by the internal software). The start of each sprint was marked manually on the Metamax system, with the event being simultaneously identified in the motion capture data. Blood lactate concentration was measured using a Biosen C-Line Sport lactate measurement system (EKF Industrial Electronics, Magdeburg, Germany) collecting 20 μL blood samples from the fingertip.

1RM in a seated pull-down was performed on a cable apparatus (Technogymcorp, New Jersey, US) with a custom made handlebar attached to the grip as standardized previously (Losnegard et al. 2011)

Prior to testing, body mass was measured using a digital body composition scanner (Tanita BC-545, Tanita Corp Inc., Tokyo, Japan) and height was measured using a stadiometer (Harpenden Portable Stadiometer, Holtain Limited, Crymych, Dyfed, UK).

Test protocols and measurements

After a 10-min low-intensity warm-up while running on a treadmill, the participants were familiarized with the poling ergometer, and performed 20 min of movement-specific warm-up at a low to moderate intensity. The RSA test included eight 8 s maximal sprints with 22 s recovery between each sprint. During the RSA test the participants were instructed to perform maximally for all sprints. The participants had a passive recovery, making it possible to gather blood samples during each break. The poling exercise was done in a synchronous manner, imitating the double poling technique. Thus the average displacement (Δl) of left and right handle marker relative to the origin marker was used for further calculations. Sample rate was set at 100 Hz for the motion capture system and at 1,500 Hz for the force (F) measurements. All data were up-sampled to 1,500 Hz before calculating instantaneous work as $W = F \times \Delta l$. The power output was calculated as the time differential of work. The cycle rate was the reciprocal of cycle time, and work per cycle was calculated as the cumulative sum of W over a movement cycle. The power outputs for each of the eight sprints were calculated as average power over the whole sprint, and the average of all sprints determined repeated sprint performance. The sprint power decrement was calculated by using the method from Spencer et al. (2006b). Here, the difference between the highest 8 s power and the average sprint power divided by the highest 8 s power, and presented as a percentage.

Muscle oxygen saturation, heart rate and breath-by-breath VO_2 measurements (resampled at 1 Hz) were sampled continuously during the whole test protocol. In order to capture the dynamic response of VO_2 and heart rate, an eighth order elliptic bandpass filter with 0.03 and 0.037 Hz cut-off frequencies (i.e., around 1/30 = 0.033 Hz) was used on these traces.

Using NIRS, the light of the specific wavelengths is mainly absorbed by hemoglobin and myoglobin, though due to their identical spectral characteristics, it is not possible to distinguish between the two. Changes in absorption at the discrete wavelengths were converted into concentration changes of O_2Hb and HH using the modified Lambert–Beer law (Livera et al. 1991), and incorporating a differential path-length factor of 4.0 to correct for scattering of photons within the tissue (Ferrari et al. 1992; Duncan et al. 1996). tHb was derived from the sum of O_2Hb and HHb, where changes in tHb reflect changes in blood volume. SmO_2 was derived by means of spatially resolved spectroscopy based on the photon diffusion theory (Patterson et al. 1989) and using three source–detector distances. The movement artefacts that were present in the NIRS signals due to the contraction–relaxation pattern during poling exercise were smoothed with a Butterworth filter (50 Hz, eighth order, zero-lag) prior to calculation of mean values and group responses. Baseline values for O_2Hb, HHb, tHb and SmO_2 were calculated over the 30-s period prior to the start of the repeated sprint protocol. End-exercise values for each of the eight sprints were calculated as the mean over the last 2 s of each sprint, relative to baseline values. Group responses for O_2Hb, HHb, tHb and SmO_2 were calculated as the mean ± standard deviation (SD) over all individual responses after filtering of the data.

To determine VO_{2peak}, a 3-min all-out poling ergometer test was performed. The participants were told to perform the test at an even maximal pace to exhaustion. The average power output determined performance and the average of the three highest consecutive 10-s values of VO_2 determined VO_{2peak}. A similar test has previously been shown to be valid for measuring VO_{2peak} in cross-country sit skiers (Forbes et al. 2010). Blood lactate was measured 1 and 3 min after the 3-min all-out test, of which the highest value was used for further analyses. All participants had regularly performed this type of test and could therefore use previous experience to find their optimal pacing strategy.

After a 15-min warm-up at 60 % of maximal heart rate, VO_{2max} was measured while running on a motorized treadmill according to standardized procedures for testing cross-country skiers in Norway (Ingjer 1991). The test lasted for 5–6 min and was performed at a constant inclination of 10.5 % with individual starting speeds and

an increase of 1 km h^{-1} every minute. The maximal level of effort was considered to be attained when a plateau in VO_2 was achieved, despite increasing intensity, a respiratory exchange ratio >1.10 and a peak blood lactate concentration >8 m mol l^{-1}. VO_2 and ventilation were monitored continuously and the averages of three consecutive 10-s intervals with the highest values were used to determine maximal values.

Statistical analyses

All data were checked for normality and presented as mean ± SD. Statistical significance was set at an alpha level of <0.05. A one-way repeated measures ANOVA was used to look for changes in sprint performance, physiological and biomechanical responses across sprints. Paired samples *t* tests with Bonferroni-correction for multiple comparisons were used to analyse pairwise differences. Pearson's product-moment correlation coefficient test was used to measure correlations between performance, physiological and biomechanical variables. In addition to the traditional statistics, magnitude-based differences were analysed for the main sprint performance data (i.e., power output, cycle rate and work per cycle). The percentage likelihood of difference between individual sprints was calculated and considered almost certainly not (<0.5 %), very unlikely (<0.5 %), unlikely (<25 %), possibly (25–75 %), likely (>75 %), very likely (>99 %), or most likely (>99.5 %). Threshold chances of 5 % for substantial magnitudes were used, meaning likelihood with >5 % in both positive and negative manner was considered an unclear difference. Pilot tests with repeated measurements of the performance and physiological variables used in this study demonstrated intraclass correlation coefficients >0.90 and CVs <5 %. All traditional statistical tests were processed using SPSS 20.0 Software for Windows (SPSS Inc., Chicago, IL).

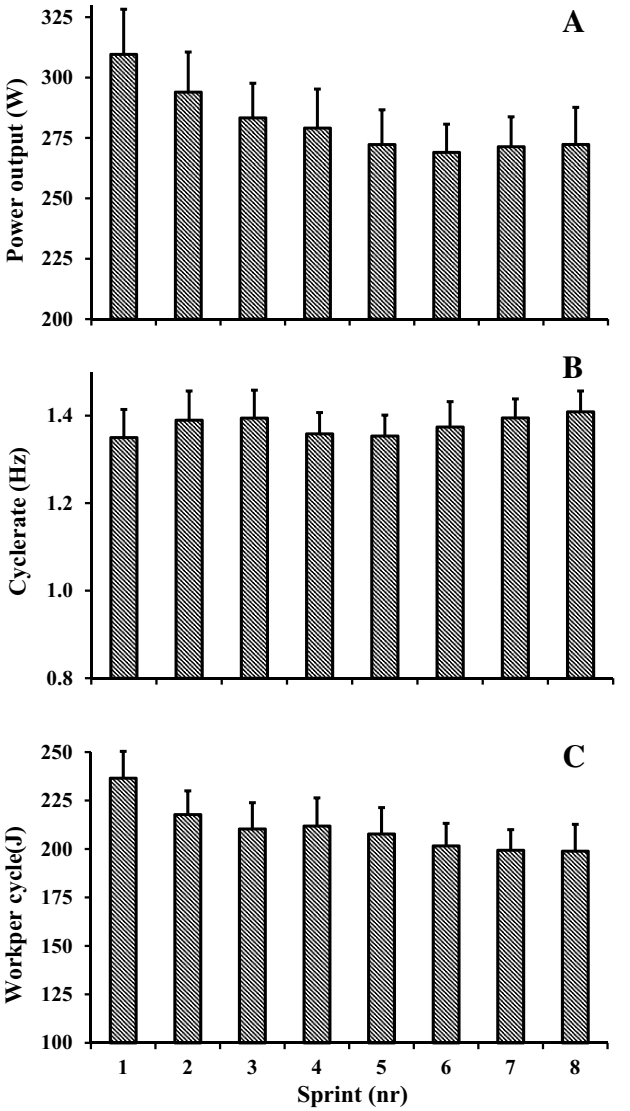

Fig. 2 The development of power output (**a**), cycle rate (**b**) and work per cycle (**c**) during the eight repeated 8-s upper body sprints

Results

Repeated sprint performance

Power output was highest in the first sprint for all athletes, after which there was a very likely to most likely decline in power output over the following four sprints (Fig. 2a; $P < 0.05$). There were no significant changes in cycle rate during the sprint protocol (Fig. 2b), with all individual sprint comparisons being rated at most likely trivial. In contrast, there was a most likely decrease in work per cycle over the first five sprints (Fig. 2c; $P < 0.05$). The average power output over all sprints was 281 ± 48 W and the sprint decrement 11.7 ± 4.1 % ($P < 0.05$).

There was a correlation between power output in the first sprint and both average power output ($r = 0.70$, $P < 0.05$) and the sprint decrement ($r = 0.58$, $P < 0.05$), but no significant correlation was found between sprint decrement and average power output. Average power correlated with average work per cycle for all sprints ($r = 0.65$–0.86, all $P < 0.05$), but not with cycle rate. There was no significant correlation between the sprint power decrement and any of the cycle characteristics.

1RM in pull-down (39 ± 4 kg) correlated with power during the first four sprints and with average sprint power ($r = 0.71$–0.80, all $P < 0.05$).

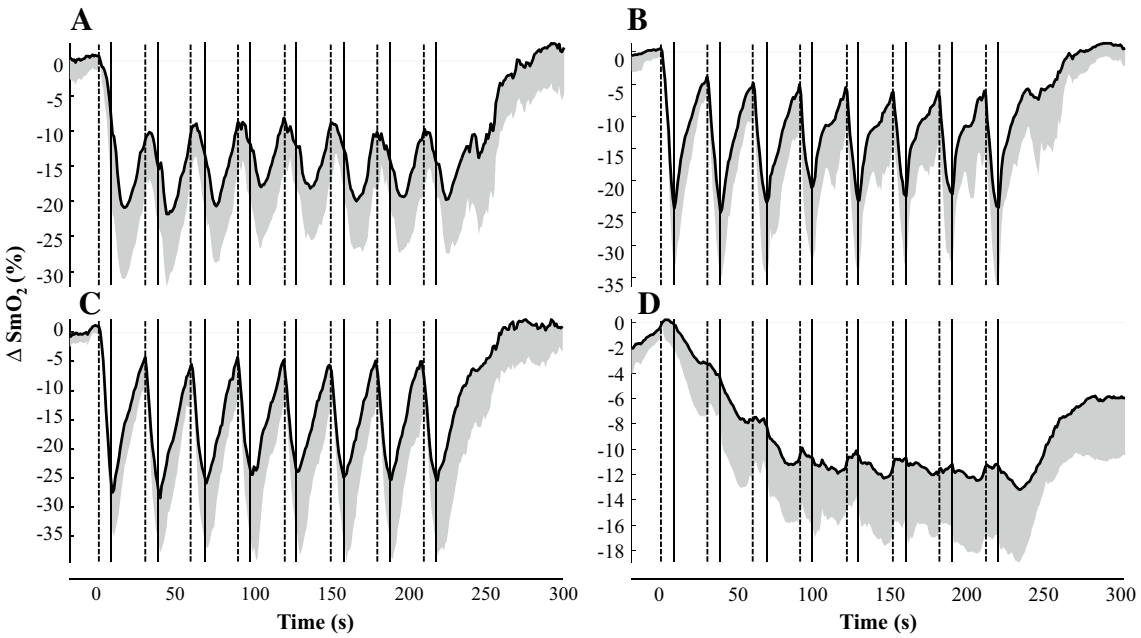

Fig. 3 Mean (SD) group response for muscle O_2 saturation (SmO_2) in **a** m. biceps brachii, **b** m. triceps brachii, **c** m. latissimus dorsi, and **d** m. vastus lateralis during the eight repeated 8 s upper body sprints. *Broken and continuous vertical lines* indicate start and stop of each sprint

Physiological responses

The mean group response for SmO_2 during the eight 8-s sprints in the four muscles is shown in Fig. 3. In TB and LAT, a rapid desaturation occurred during each sprint followed by rapid resaturation during the recovery phase. A similar pattern was seen in BB, though less pronounced and desaturation was delayed in this muscle. In the VL desaturation was less pronounced and resaturation during the recovery phase was not apparent. End-exercise values for O_2Hb, tHb and SmO_2 are shown in Fig. 4. The amount of deoxygenation at the end of the sprints increased from the first to the second sprint ($P = 0.01$) and remained constant throughout the remaining sprints ($P > 0.05$). Moreover, NIRS responses over the eight sprint periods were different between muscles (Fig. 4a, c; $P < 0.001$), apart from the response in tHb (Fig. 4b; $P = 0.30$). Pairwise comparisons showed that deoxygenation at the end of each of the eight sprints was larger in the three upper-body muscles compared to the VL (all $P \leq 0.01$). In addition, maximum deoxygenation was reached within the first sprint period for the LAT and TB, while this occurred later in the BB (second sprint) and the VL (third sprint) (Fig. 4a). Similar results of the pairwise comparisons were found for SmO_2, with the largest desaturation in the three upper-body muscles compared to the VL (Fig. 4c; $P < 0.01$), and an increase in desaturation from the first to the second sprint ($P < 0.01$), with no further change throughout the remaining sprints ($P > 0.05$) apart from a small drop in desaturation during the last two sprints ($P > 0.05$). Desaturation was larger in TB and LAT compared to BB (both $P = 0.01$). The overall decrease in end-exercise SmO_2 was largest in TB (-23.9 ± 3.4 %) and LAT (-24.1 ± 2.3 %), and smaller in both BB (-13.5 ± 2.4 %) and VL (-6.1 ± 1.1 %). Similar to the changes in oxygenation, TB and LAT directly reached maximum desaturation during the first sprint, while this occurred later for BB and VL (Fig. 4c). No NIRS variables correlated with the performance variables in repeated sprint.

The $\dot{V}O_{2peak}$ in poling was 65 ± 12 % of the $\dot{V}O_{2max}$ in running. The average utilization of pulmonary $\dot{V}O_2$ during the repeated sprints was 75 ± 7 % of the $\dot{V}O_{2peak}$ in poling (47.9 ± 8.3 ml kg^{-1} min^{-1}). On average, $\dot{V}O_2$ for each 30-s period (i.e., 8 s sprint + 22 s recovery) increased from the first to the second and third sprints (both $P < 0.01$) before a plateau was reached (Figs. 5, 6a). The peak and average heart rate over each 30-s period increased continuously from sprint to sprint ($P < 0.05$; Figs. 5, 6b). The average $\dot{V}O_2$ and heart rate fluctuation amplitudes were 9.3 ± 5.5 ml kg^{-1} min^{-1} and 13.3 ± 4.6 bpm for the 30-s periods including an 8-s sprint and a 22-s recovery period. Heart rate and $\dot{V}O_2$ showed similar patterns, but $\dot{V}O_2$ tended to respond faster and typically showed the highest value directly after each sprint was finished, whereas the highest values for heart rate were achieved approximately mid-way in the recovery periods.

Neither the changes in average $\dot{V}O_2$ over each 30 s period nor the dynamic responses in $\dot{V}O_2$ correlated with

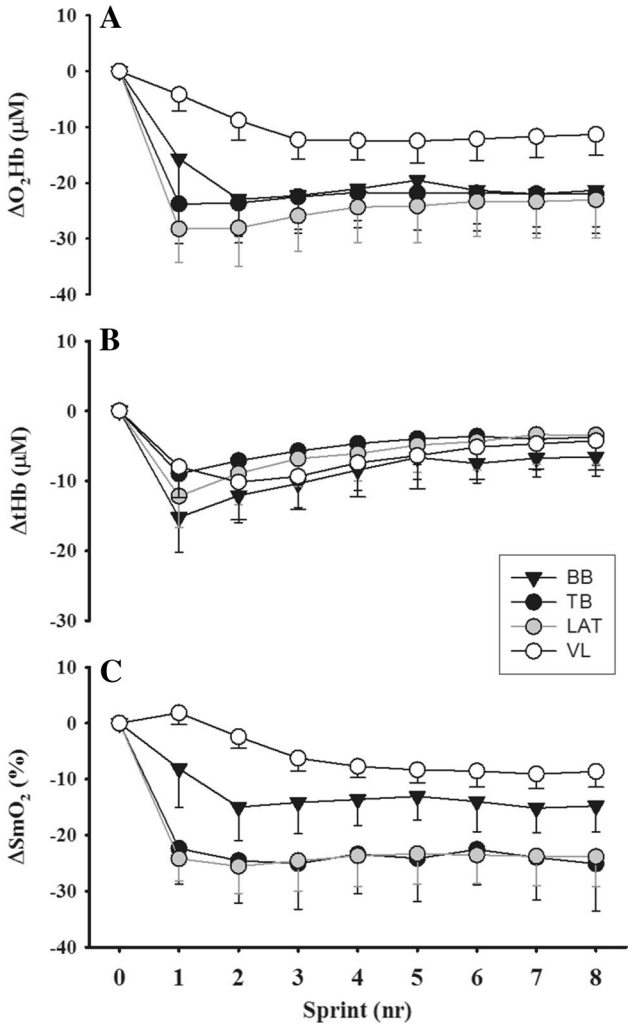

Fig. 4 Group mean (SD) changes for **a** oxyhemoglobin (O_2Hb), **b** deoxyhemoglobin (HHb), **c** total hemoglobin (tHb), and **d** muscle O_2 saturation (SmO_2) in the m. biceps brachii (BB), m. triceps brachii (TB), m. latissimus dorsi (LAT), and m. vastus lateralis (VL) at the end of each of the eight repeated 8-s upper body sprints

any of the performance variables. There was no significant relationship between VO_{2peak} in poling and average power or the sprint decrement. However, there was a correlation of VO_{2peak} with power output and work per cycle in the last three sprints ($r = 0.60$ and 0.71, respectively, both $P < 0.05$). There was no significant correlation between VO_{2max} in running and performance variables in repeated sprint or with VO_{2peak} in poling.

During the repeated sprint test, the blood lactate concentration progressively increased every 30 s ($P < 0.05$), reaching a peak value of 8.5 ± 3.0 m mol l^{-1} after the last sprint (Fig. 6c), which is lower than the 14.6 ± 3.4 m mol l^{-1} that was reached after the VO_{2peak} test ($P < 0.05$). There were positive correlations between the absolute increase in blood lactate concentration versus average power output and the sprint decrement ($r = 0.79$ and 0.61, both $P < 0.01$).

Discussion

While most previous studies on repeated sprint ability have focused on leg exercises, this was the first laboratory-based study that investigated repeated sprints during isolated upper-body work. Here, the majority of the 12 % power decrement in upper-body sprint performance was evident in the first five sprints, followed by reductions in work per cycle at stable cycle rates. The subsequent responses of deoxygenation/desaturation and increased pulmonary oxygen uptake were remarkably quick, reaching plateaus already after the first and second sprint, respectively. Maximal strength was correlated with power output in the first four sprints, whereas maintenance of power over the last three sprints correlated to aerobic capacity.

Repeated sprint performance

Although the upper extremities are of high importance or even fully responsible for propulsion in many sports (Stoggl et al. 2006a; Hawkeswood et al. 2011; Uzun et al. 2012; Tesch 1983), only one previous study has focused on repeated sprint ability during upper-body exercise (Sandbakk et al. 2014). Since the previous study on upper-body RSA was performed with limited physiological measurements, the current research adopted a laboratory-based approach to explore the physiological mechanisms in more detail. In this case, elite cross-country skiers performed eight bouts of 8 s isolated upper-body poling with an average power of approximately 280 W during a single sprint. The sprint decrement was on average 12 %, with most of the loss in power occurring during the first five sprints. This decrement is slightly greater than the decrements of 5–10 % found in previous research on repeated sprint exercise in running and cycling using relatively similar protocols (Spencer et al. 2005; Perrey et al. 2010; Dupont et al. 2005). However, even small differences in test protocols (i.e., number of sprints as well as sprint and recovery times) and physiological profiles of the subjects may influence the outcome. Still, the current decrement is higher than the 7 % sprint decrement found in upper-body trained ice sledge hockey players during maximal double poling on ice using an almost identical protocol (Sandbakk et al. 2014). Even though the majority of the decrements during such protocols have been explained by incomplete resynthesis of PCr between the sprints (Mendez-Villanueva et al. 2012; Bishop et al. 2003), the larger sprint decrement found here compared to previous studies requires further explanation. First, this study examined power output decrement and not

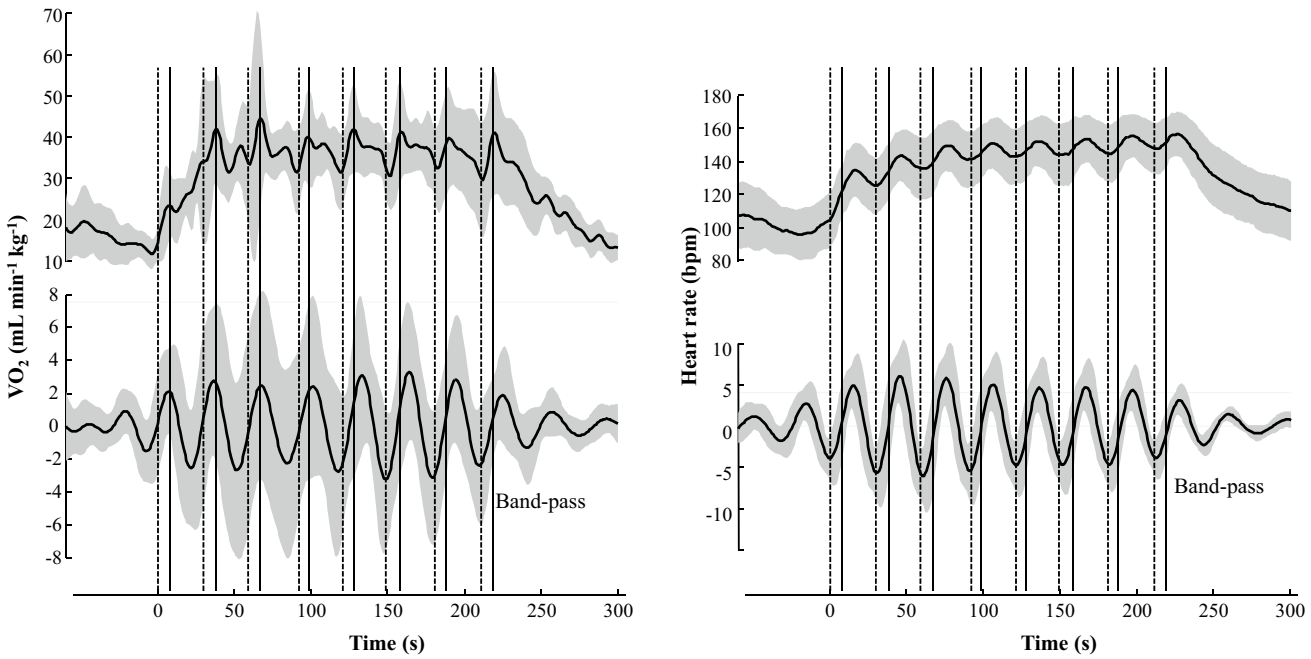

Fig. 5 Mean (SD) group response for oxygen uptake and heart rate kinetics during the eight repeated 8-s upper body sprints with 22-s recovery. The *upper graphs* indicate the absolute values in VO_2 and heart rate, whereas the *lower graphs* illustrate the normalized fluctuations in these variables (as described in detail in the methods). *Broken and continuous vertical lines* indicate start and stop of each sprint

time decrement. A given reduction in power does not necessarily lead to a similar increase in time in sports where athletes work against various environmental resistances. Another potential factor may be the low muscle mass that produces these high workloads. This differs from running and cycling where larger muscle groups produce the workloads and from the previous upper-body study in ice sledge hockey where shorter poling times and more technically demanding execution limited the work rate production (Sandbakk et al. 2014). Additionally, the previous study in ice sledge hockey was performed on disabled athletes, who may respond differently than the cross-country skiers examined here.

In correspondence with the power decrement examined here, the work per cycle decreased whereas the cycle rate was kept at a constant level. This is fundamentally different from the previous study on RSA in ice sledge hockey where cycle rate decreased with increasing sprint time (Sandbakk et al. 2014). The greater importance of work per cycle in the current study may be explained by the constraints of the locomotion. In the Concept2 SkiErg a flywheel adjusts the air resistance when power is produced and therefore the movement speed is almost constant across workloads. Thus, the propulsive phase is longer than during double poling on ice or snow outdoors when the poling time is reduced with increasing speed. These ergometer constraints might also be one of the reasons why the magnitude of work per cycle showed strong correlations with power output in this study. Thus, mainly work per cycle determines average power and sprint decrements during repeated sprint exercise in ergometer poling.

Power output in the first sprint correlated significantly both with the sprint decrement and with the average sprint power. This indicates that a high power capacity is of importance for the average power in repeated sprints, but also that the most explosive athletes, who are able to produce high initial powers, consecutively have a greater decrement over the sprints. This has previously been demonstrated during leg exercise (Mendez-Villanueva et al. 2008), and is confirmed for upper-body work in this study.

Physiological responses

Although NIRS has recently been used to study repetitive work during running (Buchheit et al. 2010; Ihsan et al. 2013; Jones et al. 2013; Ufland et al. 2013) and cycling (Billaut and Buchheit 2013; Racinais et al. 2007; Smith and Billaut 2010, 2012), this is the first study employing NIRS to determine physiological responses in the working muscles during upper-body repeated sprints. Moreover, it is the first study where the responses to repeated sprints are simultaneously measured in multiple muscles varying from highly active to supposedly inactive contributors. The NIRS measurements demonstrated rapid desaturation/resaturation patterns within the main working muscles, LAT and TB, during the sprints. This indicates

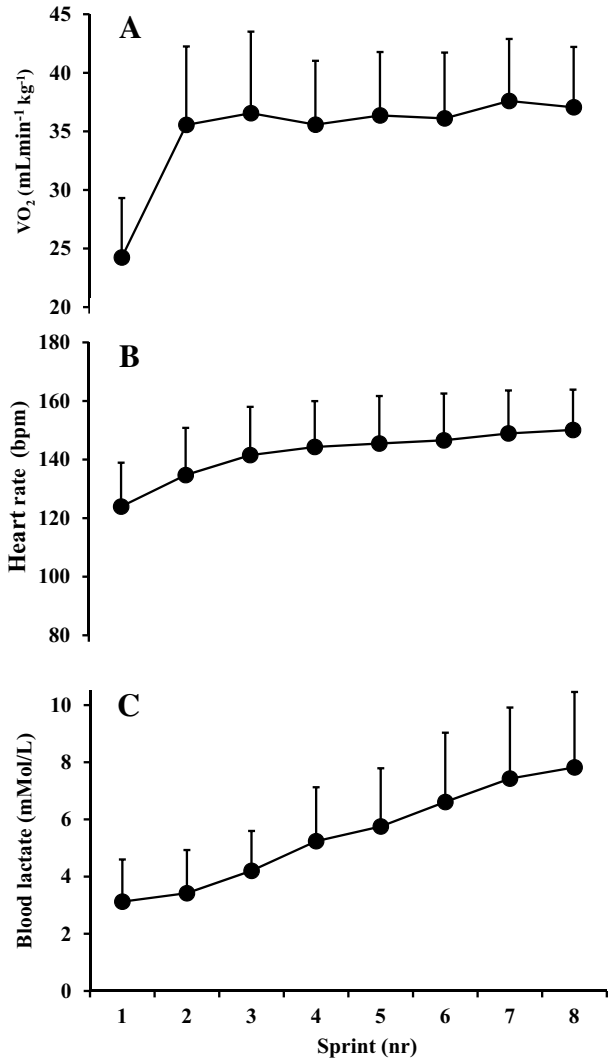

Fig. 6 Development of VO_2 (**a**), heart rate (**b**) and blood lactate concentration (**c**) throughout the eight repeated 8-s upper body sprints

an optimal use of the available oxygen during sprints and a fast restoration of depleted oxygen during recovery in the main muscles involved in the poling phase of the movement for these highly upper-body trained participants. Already during the first sprint, LAT and TB were highly desaturated suggesting large oxygen extraction within the first of these short exhaustive sprints. The maximum desaturation in the BB was less and delayed compared to LAT and TB, indicating that BB is less involved in the power production in this locomotion. Although the leg muscles were not actively involved in the poling exercise, a gradual mild desaturation occurred in the VL, while resaturation during the 22-s recovery periods was minimal. This indicates that, even though the legs were situated below the heart, most of the blood circulation during the recovery phases was directed to the upper-body muscles, thereby preventing resaturation of VL throughout the test.

Pulmonary VO_2 increased significantly following the two first sprints, after which the VO_2 fluctuated around 75 % of VO_{2peak} while the blood lactate concentration increased with a steady rate. The fluctuation of VO_2 around 75 % of VO_{2peak} during repeated sprint exercise found here confirms the findings by Balsom et al. (1999) in cycling. In the current study, we added fluctuation amplitudes of VO_2 and heart rate to indicate the dynamic physiological responses. However, possible differences in VO_2 fluctuations across exercise modes needs to examined in future studies. Although many factors influence the concentration of lactate in the blood, the steady increase in blood lactate levels found here may indicate an incomplete resynthesis of PCr and thereby increasing requirements from lactic anaerobic energy together with the aerobic energy sources utilized (Bogdanis et al. 1995). Several studies have shown correlations between the decrement in RSA and the decline in intramuscular pH (Glaister 2005; Edge et al. 2006). However, the exact role of acidosis on fatigue during repeated sprints is still not fully understood and recent reports indicate that lowered pH has little effect on contractile function during exercise (Glaister 2005). Taken together, the current study demonstrates a remarkably fast response in muscle oxygen desaturation during maximal upper-body work, a slightly delayed but well-timed pulmonary response and further illustrates the gradual shift in energy system contributions during repeated sprints.

Maximal upper-body strength showed large correlations with power output during the first four sprints, as well as with the average power output and the sprint decrement. This indicates that maximal strength is a determining factor for RSA, but also that the most explosive athletes have problems to maintain power over repeated sprints. This is supported by, e.g., Mendez-Villanueva et al. (2008) who reported that athletes with the highest initial power output had the highest decrement due to their high level of non-oxidative pathways of ATP resynthesis. Correspondingly, the athletes with the highest maximal strength also had the greatest increases in absolute lactate concentration as reflected in a significant correlation between sprint decrement and the increase in lactate. The high importance of anaerobic energy pathways in the initial phase of the repeated sprints may explain why VO_2 kinetics did not correlate with any performance parameters which is in line with Buchheit (2012) in running. However, the correlation between VO_{2peak} and power in the last three sprints indicates that aerobic capacity is still a complementary factor for RSA and potentially linked to its role for recovery between sprints. In this context upper-body VO_{2peak} accounted for only 65 % of the athletes VO_{2max} in running and VO_{2max} did not correlate significantly with any

performance parameters. Thus, peripheral rather than central cardiovascular factors may be mainly responsible for recovery of the upper limbs during such exercise modes (Sandbakk et al. 2014).

Limitations of the study

While this study is the first to examine upper body RSA in detail, future studies are recommended to investigate the differences in physiological responses between upper and lower body exercise modes directly using comparable test protocols. Another limitation of the present study is the relatively small sample size and, therefore, the correlation values should be interpreted with caution.

Conclusions

The current study provides new insights into repeated sprint performance and physiological responses during isolated upper-body work. The sprint decrement was already significant after the first sprint, with the majority of the 12 % sprint decrement occurring over the first five sprints in the current protocol of 8 s work periods and 22 s breaks between sprints. The subsequent physiological responses were remarkably quick, with the main working muscles being highly desaturated already after the first sprint, followed by pulmonary oxygen uptake reaching a plateau after two sprints and gradual increases in blood lactate concentration towards the end. Together, this illustrates the gradual shift in energy system contributions during repeated sprints. With regard to performance determining factors, high maximal strength seems important for producing power in the first sprints, whereas aerobic capacity is important for producing high power in the last sprints.

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