

Tommy Fredriksen Skålvik

**Changes in power output, physiological responses and
technique during upper-body repeated sprint exercise**

BEV3901 Master Thesis

Department of Human Movement Science

Norwegian University of Science and Technology (NTNU)

Trondheim, 2013



NTNU – Trondheim
Norwegian University of
Science and Technology

ABSTRACT

Purpose: The purpose of this study was to examine upper-body repeated sprint performance, physiological responses, and the relationships to technique and muscle co-ordination.

Methods: Twelve male elite cross-country skiers (body mass: 75.4 ± 7.1 , VO_2max : 73 ± 4) were tested for repeated sprint ability during 8·8-s maximal poling with 22-s breaks sprint using a modified poling ergometer. Total mean power (i.e. mean power of all sprints added together) and the sprint decrement (i.e. difference in percent between total mean power and best single sprint power times 8) determined performance. Applied forces were measured with a force cell, the movement frequencies and velocity of the movement using high-speed infrared cameras, muscle activation by surface electromyography of biceps and triceps, oxygen consumption was continuously measured breath by breath and blood lactate in the breaks between each sprint. Furthermore, peak power was determined from the first 8-s sprint and VO_2peak during a 3-min all-out ergometer poling test.

Results: The average total mean power was 2246 ± 387 W. The average sprint decrement was 11.7 % after eight sprints. Poling rate did not change, while power per stroke decreased during the eight sprints ($p < 0.05$). Activation of biceps brachii increased- and triceps brachii decreased during the repeated sprint exercise, however these changes did not correlate with the changes in performance. Furthermore there was a change in the timing of the triceps in the last two sprints. The greatest increase in VO_2 occurred between sprint 1 and sprint 2 ($p < 0.05$), after which the increase plateaued at a level of 75% of VO_2peak . VO_2peak during a 3-min all-out poling test correlated with power per stroke in the last three sprints ($r = 0.6 - 0.7$, all $p < 0.05$). Total increase in blood lactate correlated with total mean power ($r = 0.692$, $p < 0.05$) and sprint decrement ($r = 0.604$, $p < 0.05$).

Conclusions: During repeated sprint exercise with 22-s breaks a high decrement in power is apparent after the first sprint. The changes in power are related to reduced power per stroke, without any changes in poling rate over all eight sprints. The change in muscle activation suggests an alteration of technique during the two last sprints in order to withstand fatigue. VO_2 peak seems to be important in the latter stages of RSE. Increase in lactate is a fatiguing factor related to sprint decrement, however a large increase in lactate also seems to cause high total mean power, suggesting that the ability to enable lactic energy sources enhances high power outputs.

Key Words: Repeated sprint ability, upper-body, performance, poling, kinematics, VO_2 , Lactate

INTRODUCTION

In recent years the ability to perform repeated sprint exercise (RSE) has been highlighted as an important factor related to sport performances (1), especially as a critical component of intermittent high intensity sports, such as football, ice hockey and rugby, where the ability to make fast sprints followed by active recovery throughout a game is critical for the game's result (2). Repeated sprint ability (RSA) is described as the ability to produce high sprint speed or power output together with the ability to maintain the speed or power output throughout the entire competition (3). Power output is a product of power per movement cycle and the cycle frequency. In poling exercises, such as cross-country skiing the cycle rate and cycle length increase with increasing intensity, and decrease when athletes are fatigued (4, 5). Sandbakk et al. (6) reported that cycle length throughout the RSE-test was relatively stable, which rely changes in performance on RSE on cycle rate and power per stroke. Together, these findings indicate that power per stroke during RSE is important for performance. However, most of the earlier studies have focused on leg-work, and to the best of our knowledge there is only one study that has examined RSE-kinetics during upper-body work (6).

Earlier studies have shown that RSA is associated with winning games in team sports (7), and the most important factors for RSA are rapid and high energy delivery capacity, the muscle's ability to convert energy to high power outputs, and the ability to resist fatigue over the repeated sprints (2, 8, 9). The main contributors to energy production during RSE in the first sprints are pre-stored phosphocreatine (PCr) followed by Adenosintriphosphate (ATP) from

glycolysis (1-3, 8, 9). Due to limitations in the resynthesis of PCr and depletion of glucose-storage during repeated sprints, the energy sources shift from being mostly anaerobic towards being a mix of anaerobic and aerobic systems. During RSE the aerobic energy sources are important, not only to resynthesize PCr during the recovery process, but also to fuel ATP during contractile activity (3, 10). Recent research shows that the ability to resynthesize PCr correlates with the recovery of repeated sprints and with RSA (11). This may imply that the ability to use anaerobic rather than aerobic energy could determine RSA. However, due to resynthesis of PCr and lack of glycogen in the latter stages of RSE, the aerobic energy could be important during the last sprints.

Several studies have shown correlations between the decrement in RSA (reduced power output), and the decline in intramuscular pH (8, 12). The exact role of acidosis on fatigue during RSE is still not fully understood, because recent reports indicate that lowered pH has little effect on contractile function during exercise (8). The influence of decreased intramuscular pH on fatigue seems to be less than previously thought, but there is still a belief that accumulation of lactate and hydrogen ions (H^+) has an inhibitory effect on performance (12). Hamilton et al. (13) also found that the biggest decrement in mean power throughout a RSE was in explosive team-sport athletes with high post-test lactate values, while endurance runners maintained their sprinting performance better in RSE with lower post-test lactate values. (13). This may indicate that decreased pH values have an effect on performance, but it can also be due to the higher glycolytic rate in team-sport athletes to compensate for lower oxygen uptake compared to the endurance runners (14).

When performing explosive sprint exercises, the ability to fully activate the contracting musculature enhances performance (15). In RSE, the ability to maintain the muscle recruitment and to have rapid firing from pre-motoric cortex is of great importance as a factor to resist fatigue during the latter stages of the exercise (15). During RSE the decline in sprint performance has been reported together with a decrease in EMG-signal, which can be a sign of inhibited neural drive, lower firing frequencies and reduced neuromuscular transmission (sarcolemmal excitability) (16, 17). The mechanism behind the inhibited neural drive is suggested by Racinais et al. (17) to derive from inhibiting muscle afferents to avoid muscle tissue damage because of muscle deoxygenation. Girard et al. (1) suggested that one of the factors contributing to fatigue in RSE may be the impaired cell membrane excitability caused by increased extra-cellular K^+ concentration by reducing the action potential amplitude and slowing of impulse conduction. This reaction in the Na^+/K^+ pump has been observed in intense dynamic contractions. The same study observed decreased M-wave amplitude in a repeated sprint protocol, suggesting that the action potentials' synaptic transmission may be impaired during RSE (1). However, due to limited scientific research on RSE, the physiological demands in RSA still requires further investigation (2).

Although there are several other sports that depend greatly on the upper-body for propulsion, such as kayaking, sledge ice-hockey, wheelchair basket and cross-country skiing (18-21), most scientific research focused on locomotion where mainly the lower extremities are active. The physiological responses during upper-body work seem somewhat different from those obtained during leg exercise (18, 20, 22). The upper-body is characterized by lower muscle mass, which explains the higher accumulation of blood lactate during strenuous exercise

because of the smaller part of active skeletal muscles in the upper-body that can consume lactate during exercise (23). Furthermore maximal oxygen consumption is lower in upper-body exercise, together with slower oxygen uptake kinetics, compared to leg exercise (22, 24). This is likely due to intrinsic factors such as different capillarization in the upper-body which leads to shorter mean transit time and impaired diffusion conditions in the muscle, and a lower proportion of muscle fibre type 1 compared to the lower limbs (22, 25). However, there is to date only one study (6) that has examined upper-body physiology in RSE, and here the only measured physiological responses were heart rate and lactate values. This implies that there is still a lack of understanding regarding upper-body physiology during RSE. Whether there are different factors contributing to performance in upper-body RSE compared to lower- or whole-body RSE requires further investigation.

The purpose of the present study was to examine changes in power output, physiological responses, and technique during upper-body RSE. Furthermore, power output was correlated with the physiological and technical responses. In addition it would be interesting to examine whether upper-body physiology differed from whole-body physiology in repeated sprint. It was hypothesized that the sprint decrement would be associated with decreased muscle activation, increased blood lactate and a decrease in power per stroke, rather than change in poling rate. Furthermore, it was hypothesized that VO_{2max} in running or VO_{2peak} in poling would not correlate with performance.

METHODS

Subjects

12 Norwegian male cross-country skiers competing at national level volunteered to participate in the study. Their demographic, anthropometric, and performance characteristics (in accordance with the FIS system) are documented in Table 1. The experimental procedures employed were pre-approved by the Norwegian Regional Ethics Committee and the protocol and procedures explained verbally to each subject prior to obtaining his written informed consent prior to participate. In order to participate, the subjects had to perform upper-body strength training twice a week over the last 3 months and include upper-body training in their daily endurance training.

Table 1: Anthropometric and physiological characteristics and levels of performance for the twelve male cross-country skiers.

Parameter	Mean \pm SD
Age (years)	25.7 \pm 6.2
Body height (cm)	180.4 \pm 3.4
Body mass (kg)	75.4 \pm 7.1
VO ₂ peak poling (ml/kg·min ⁻¹)	47.9 \pm 8.3
VO ₂ max running (ml/kg·min ⁻¹)	73.0 \pm 3.6
Peak BLa poling (mmol·L ⁻¹)	14.6 \pm 3.4
Peak heart rate poling (bpm)	176 \pm 9
FIS-points	76 \pm 21
Training (h·year ⁻¹)	612 \pm 37

Overall Design of the Study

After an initial warm-up, 8·8-s maximal sprints with 22-s recovery in a modified double-poling ergometer with locked legs were performed. The applied forces were measured with a force cell together with the movement frequencies and velocity of the arm movement using the Qualisys Oqus system. Muscle activation patterns were measured by surface electromyography of the biceps and triceps muscles, oxygen consumption was continuously measured breath by breath and blood lactate in the breaks between sprints each 30 seconds. Furthermore, peak power was determined from the first 8-s sprint and peak oxygen consumption was measured during a 3-min all-out ergometer poling test.

Instruments and materials

By using a Concept2 SkiErg (Concept2 Inc., Morrisville, Vermont, US) a poling system was built consisting of an ice sledge hockey seat mounted to a platform (Figure 1). The seat elevated the athlete's sitting position so that the athletes could imitate a similar poling angle as used in poling when skiing. The ice sledge hockey seat was used to make it possible to lock the athlete's legs in order to ensure that only the upper-body could actively contribute to force production during the test protocol. The ergometer was equipped with a force cell on the rope pulling-system.



Figure 1: 1A: Picture of the modified poling ergometer with the ice sledge-hockey seat mounted to a platform. The red rings are marking the positions of the reflective markers used with the Qualisys Oqus camera-system. 1B: Picture of the force cell mounted inside the ergometer. 1C: Picture of an athlete using the ergometer.

Gas exchange was continuously measured during the test by indirect calorimetry using the 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 O₂ (16.00%) and CO₂ (4.00%) before the start of each test day. The concentration of O₂ and CO₂ of room air was read and the flow transducer was calibrated using a 3-L high-precision calibration syringe (Calibration syringe D, SensorMedics, Yorba Linda, CA) before testing a new subject. The flow transducer was switched and recalibrated before each test. During the testing the data were transmitted telemetrically and stored on a lab computer after each test. The instruments were carried in a vest mounted to the athletes' upper-body during the tests. Heart rate was continuously measured using a heart rate sensor (Polar T34, Polar Electro OY, Kempele, Finland) synchronized with the VO₂ measurement system. Blood lactate concentration was measured using Biosen C-Line Sport lactate measurement system (EKF Industrial Electronics, Magdeburg, Germany) collecting 20 µL blood in each sample from the fingertip.

Kinetics was measured with a Noraxon force cell (Noraxon USA Inc., Scottsdale, Arizona) mounted inside the concept2 SkiErg poling ergometer. The displacement and velocity of the movement was captured by the Qualisys Oqus camera system (Qualisys AB, Gothenburg, Sweden). Reflective markers were attached on both poling ropes, on the top where the ropes leave the body of the ergometer (top markers), and markers on the right and left rope handles (handle markers) (Figure 1A). Displacement was calculated as the change in distance between handle markers and top markers. Velocity was determined using a 5 point differentiating filter. The average of right and left displacement was used for further analysis. All data was analysed in a self-written Matlab-script. Muscle activity was recorded using surface

electromyography (EMG) (Noraxon USA Inc., Scottsdale, Arizona) synchronized with the Qualisys infrared camera-system. EMG-electrodes were attached to the right hand biceps brachii (long head) and triceps brachii (short head) according to the Seniam guidelines (26). The electrodes were pre-gelled disposable self-adhesive AG/AGCL snap electrodes for surface-EMG (Noraxon USA Inc., Scottsdale, Arizona).

Prior to testing, body weight was measured using a digital body composition scanner (Tanita BC-545, Tanita Corp Inc., Tokyo, Japan) and height was measured using a stadiometer (Harpden Portable Stadiometer, Holtain Limited, Crymych, Dyfed, UK). The subjects provided written information about their birthdate, FIS Scores and training hours per year using a questionnaire.

Test protocols and measurements

After a 10 minute low intensity warm-up while running on a treadmill, the subjects were familiarized with the poling ergometer for five minutes before 20-min of movement specific warm-up on a low to moderate intensity. The RSA test included 8·8 seconds maximal sprints with 22 seconds recovery between each sprint. During the RSA test the subjects were told to perform maximally at all sprints. The subjects had a passive recovery, making it possible to gather blood samples during each break. The muscle activity (EMG), force and recording of movements were synchronous sampled during the repeated sprint test. The infrared camera-system's sample rate was 100Hz, while force and EMG was measured at a sample rate of 1500Hz. EMG was rectified and averaged according to the root-mean-square method (RMS). The mean was calculated using a 30-ms moving average time window. Movement velocity was calculated using a differentiating filter on displacement. The force cell measured all force

produced in the ergometer, which was used in combination with time and velocity data to calculate power kinetics during RSE. For every poling cycle, force per stroke was measured from the start of the pulling phase until the end of the pulling phase, which was calculated into power in watts by multiplying the force per stroke and the velocity of this movement. During the sprints, forces were continuously measured, and based on each cycle (pull phase and retrieval phase) mean force was calculated in each sprint. Combining force with movement velocity the mean power per sprint was calculated as the product of force and movement velocity. Mean power was calculated for each of the 8 sprints, and the sum of all sprints determined the total mean power. Ideal power was defined as the highest mean power times 8 repetitions. Repeated sprint performance was defined as total mean power during the 8 sprints and the power decrement from the ideal sprint power to the total power actually produced. Sprint decrement was calculated by using the method from Spencer et al. (27). The percent sprint decrement was calculated as the total mean power divided by ideal power and multiplied by 100. Power per stroke was defined as the mean power in the pull-phase during each sprint. Kinematics was sampled during the entire RSE-test, and was determined by establishing the change from positive displacement (pull) and negative displacement (retrieval). Poling excursion is the total displacement from the start to the end of the pull, and poling rate is the inverse of time ($1/\text{time}$) between start of each pull to the start of the next pull.

Heart rate and VO_2 measurements were sampled continuously during the whole test protocol. In order to capture the response on 30-s interval sprint repetitions of VO_2 and heart rate an elliptic (bandpass) filter was used, set at $1/30$ seconds = 0.033Hz (range: $0.9 - 1.1 \cdot 0.033\text{Hz}$).

The amplitude of this 0.033Hz band was used as a measure of change in heart rate and VO_2 as a dynamic response on RSE load.

To determine the peak oxygen uptake ($\text{VO}_{2\text{peak}}$), a 3-min all-out poling ergometer test was performed. The subjects were told to perform the test at an even maximal pace to exhaustion. All subjects had performed this test regularly as a part of their monthly testing and could therefore use previous experience to find their optimal pacing strategy. A similar test has previously been shown to be valid for measuring $\text{VO}_{2\text{peak}}$ in cross-country sit skiers (28). The athletes' $\text{VO}_{2\text{max}}$ in running was tested in our laboratory prior to the study. After a 15-minute warm up at 60% of HR_{max} , the athletes' $\text{VO}_{2\text{max}}$ in running was tested in our laboratory prior to the study when running on a motorized treadmill, according to a traditional method of monitoring cross-country skiers in Norway (29, 30). The test duration was 5-6 minutes, performed at a constant inclination of 10.5% with individual starting speeds and a stepwise increase by 1 km/h every minute. These tests were considered to represent maximal effort if the following criteria were met: 1) a RER value above 1.10, and 2) BLa exceeding 8 $\text{mmol}\cdot\text{L}^{-1}$. VO_2 was measured continuously and the average of the highest minute defined as $\text{VO}_{2\text{peak}}$ (poling) and $\text{VO}_{2\text{max}}$ (running). The highest heart rate values attained during the tests were defined as peak and maximal heart rate, and the blood lactate concentration was measured 1 min after the tests were finished.

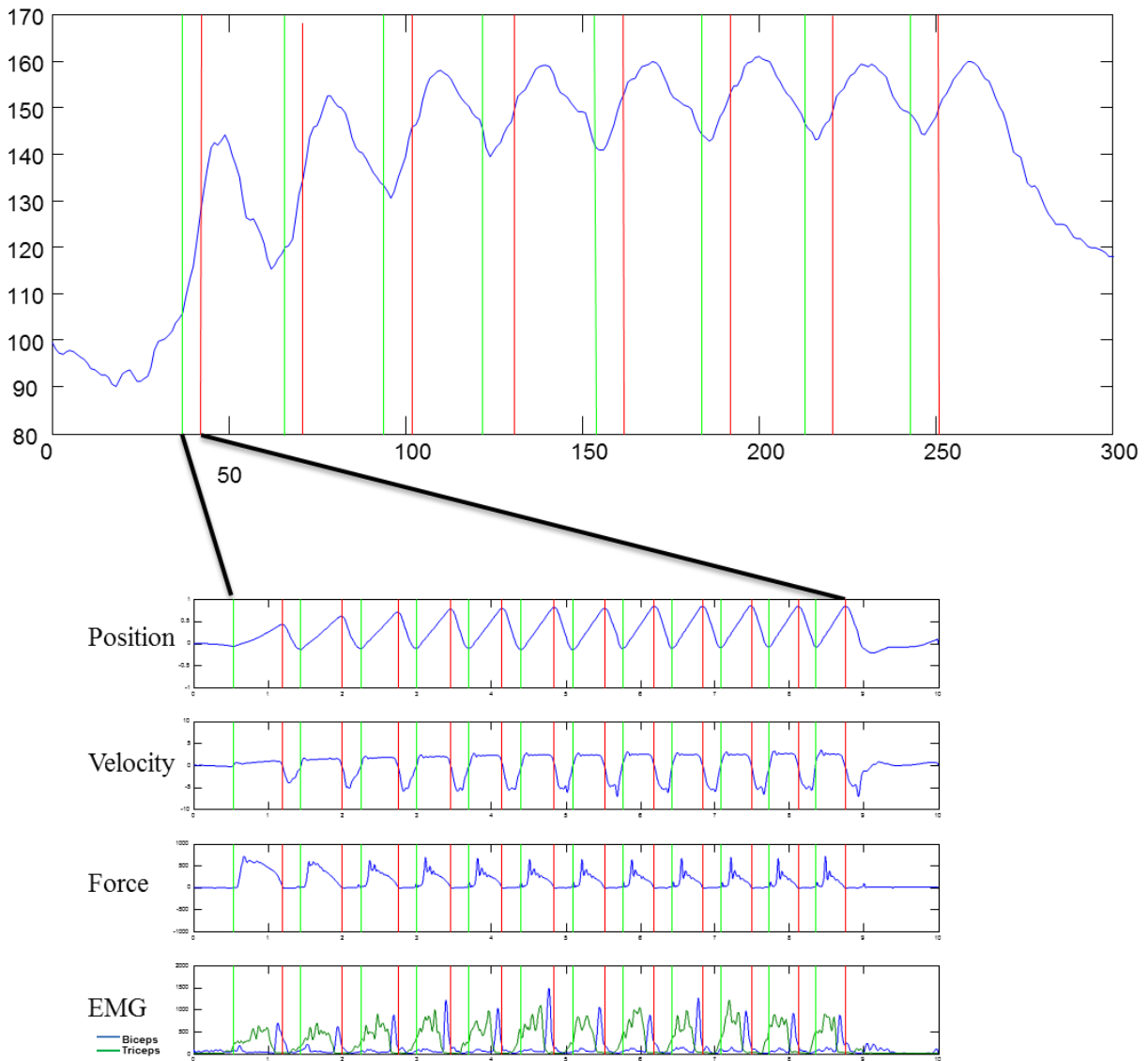


Figure 2: Heart rate kinetics in a representative subject with movement kinetics in the upper picture and EMG-signal from the first sprint in the picture below. Vertical green lines indicate start and vertical red lines indicate stop at each sprint in the upper picture. In the picture below, vertical green and red lines indicate start and stop at each pull.

Statistical analyses

All data were checked for normality and presented as mean and standard deviation (SD). Statistical significance was set at $p < 0.05$. All statistical tests were processed using SPSS 20.0 Software for Windows (SPSS Inc., Chicago, IL). A one-way repeated measures ANOVA was used to look for changes in sprint performance across all sprints. This included identifying changes in kinetics, EMG-signals, VO_2 and HR. Pearson's product-moment correlation coefficient test was used to measure correlations between performance variables and physiological variables.

RESULTS

Repeated sprint performance

The mean power output for each 8-s sprint is shown in Figure 3. The mean power was highest in the first sprint, after which there was a significant decline in power output from sprint one to six ($p < 0.05$), before a slight (non-significant) increase in power occurred in the last two sprints. Total change in sprint power gave a significant negative mean slope of 5.22 W per sprint from sprint 1 to 8 ($p < 0.05$). The total decrease from first to last sprint in mean power output was 12% ($p < 0.05$). A 9.9 ($p < 0.01$) decrease occurred from first to the fourth sprint. After that changes were not significant.

Table 2: Total mean power and sprint decrement during an 8·8-s upper-body repeated sprint exercise in 12 elite cross-country skiers.

Parameter	Power
Peak Mean Power	319 ± 62 W
Ideal Mean Power	2555 ± 493 W
Total Mean Power	2246 ± 387 W
Sprint Decrement (%)	11.7 ± 4.3 %

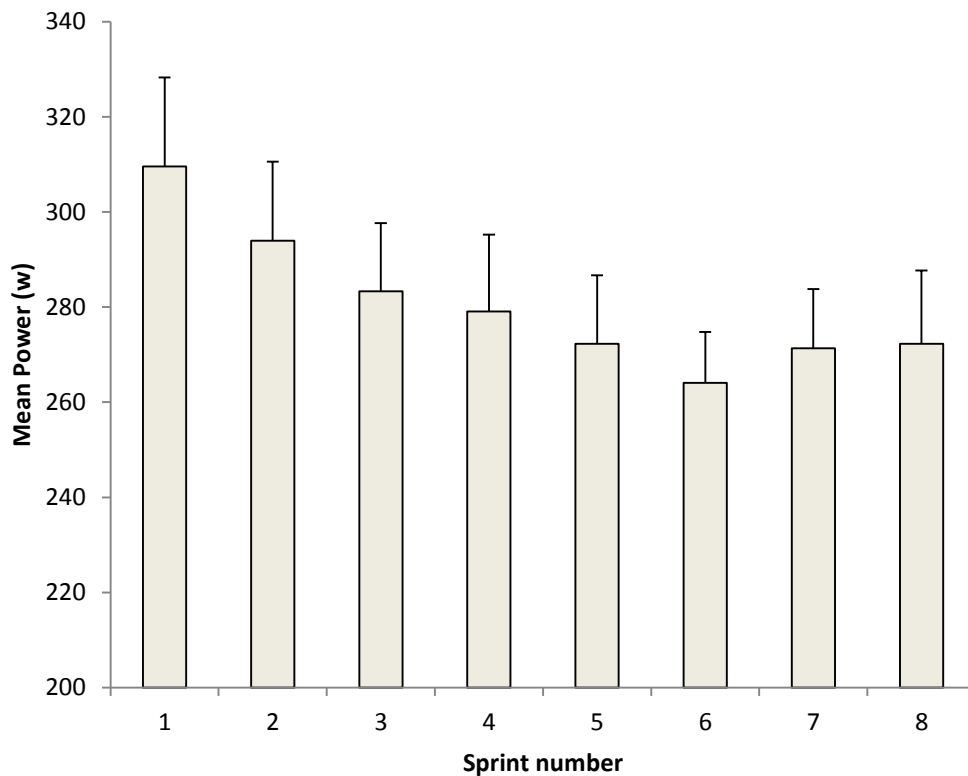


Figure 3: Overall development of mean power (W) during eight repeated sprints.

There were no significant changes in poling rate and poling excursion during the sprint protocol, however power per stroke changed from sprint 1 to 8, with a mean slope of -11.7 W per sprint ($p < 0.05$). Total mean power correlated with power per stroke whereas total mean power did not correlate with poling rate and poling excursion (Table 3). There was a significant correlation between mean power in sprint 1 and the sprint decrement ($r = 0.577$ $p < 0.05$).

Table 3: Overview of correlation between performance variables in upper-body repeated sprints and movement kinetics. All variables are the mean for all 8 sprints.

Correlations	Total Mean Power	Sprint Decrement
Power per stroke	R = 0.945, p = 0.0001*	R = 0.160, p = 0.619
Poling rate	R = 0.266, p = 0.404	R = 0.458, p = 0.134
Poling excursion	R = 0.330, p = 0.295	R = -0.153, p = 0.636

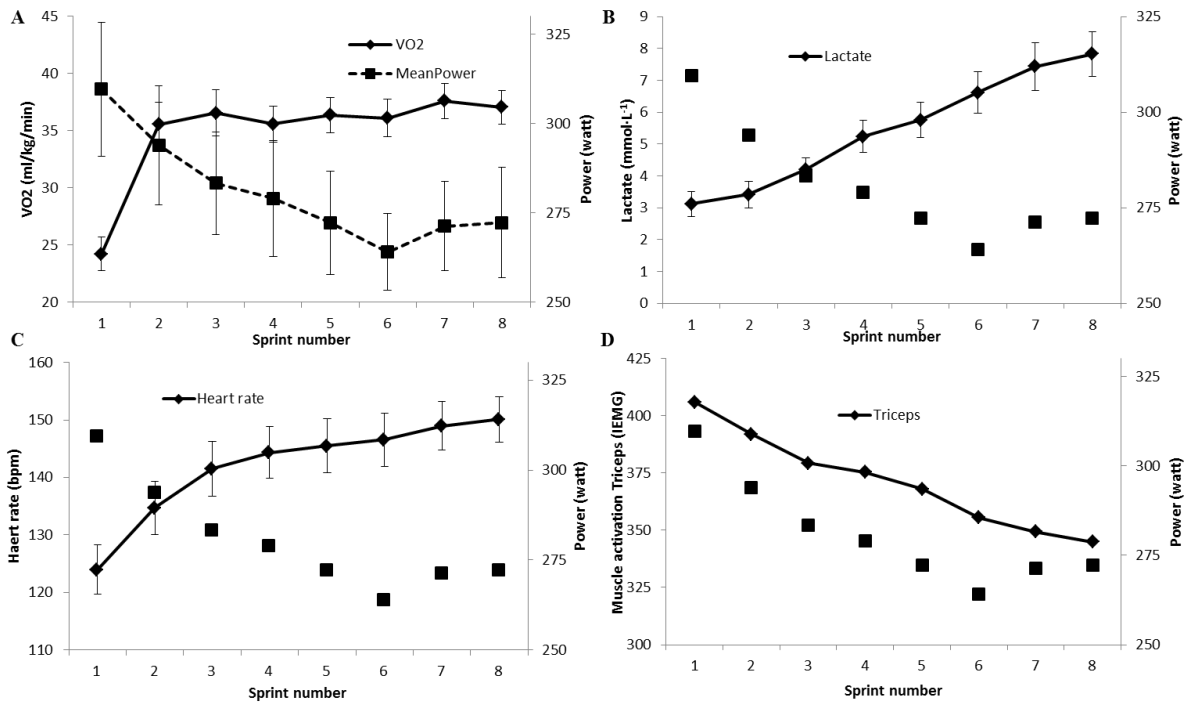
*Significant at a level of $p < 0.01$

Physiological responses to repeated sprints

The average VO_{2peak} in poling, as obtained in the all-out test, was 47.9 ± 8.3 ml/kg/min. There was no significant relationship between VO_{2peak} poling and total mean power or between VO_{2peak} poling and sprint decrement ($r = 0.408$ and 0.199 , $P > 0.05$). There was a significant correlation between VO_{2peak} poling and power per stroke during the last three sprints (sprint six: $r = 0.621$, sprint seven: $r = 0.595$, sprint eight: $r = 0.682$, all $p < 0.05$). There was a significant increase in VO_2 in the RSE-test from the first to the second sprint ($p < 0.01$). However there was no significant increase after sprint 2 (Figure 4). During the repeated sprint test, the mean use of VO_2 was at a level of 75 ± 7.6 % of the VO_{2peak} in poling. The athletes with the highest use of their VO_{2peak} in poling had the lowest power per stroke during the three last sprints (sprint six: $r = -0.592$, sprint seven: $r = -0.649$, sprint eight: $r = -0.718$, $p < 0.05$). In addition, the individual percentage used of VO_{2peak} showed a negative significant correlation with VO_{2peak} ($r = -0.647$, $p < 0.05$). In other words, the lower the VO_{2peak} , the higher was the used percentage. There were no significant correlations between VO_{2max} in running and performance variables in repeated sprint. VO_{2max} in running did not

correlate with VO_{2peak} in poling ($r = -0.152$, $p = 0.637$). The athletes' average VO_{2peak} in poling was at a level of $65 \pm 12\%$ of their VO_{2max} in running.

Changes in average VO_2 (over the entire 30-s) from sprint to sprint were only significantly different from sprint 1 to the rest of the sprints, while there were significant changes in HR between every sprint (Figure 4). There is a relationship between fluctuations in VO_2 and HR with starts and stops in the repeated sprint test (Figure 5 and 6).



*All ranges in SEM.

Figure 4: A-D: Development of physiological variables compared to mean power output during 8 repeated sprints. Mean power pattern are kept in figure 4A-D for comparison reasons. A: Development of VO_2 . B: Development of blood lactate. C: Development of heart rate. D: Development of muscle activation in triceps.

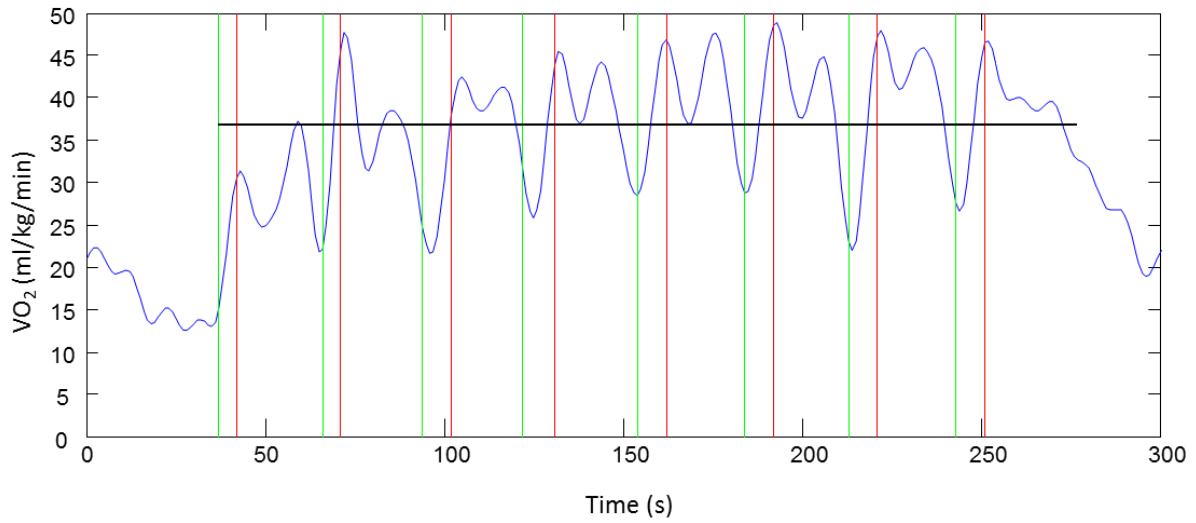


Figure 5: VO₂ kinetics during RSE in a representative subject. Blue line indicates VO₂ (ml·kg⁻¹·min⁻¹). Green vertical lines indicate start of each sprint, red vertical lines indicate stop of each sprint. The black line indicates mean VO₂ during the whole sprint.

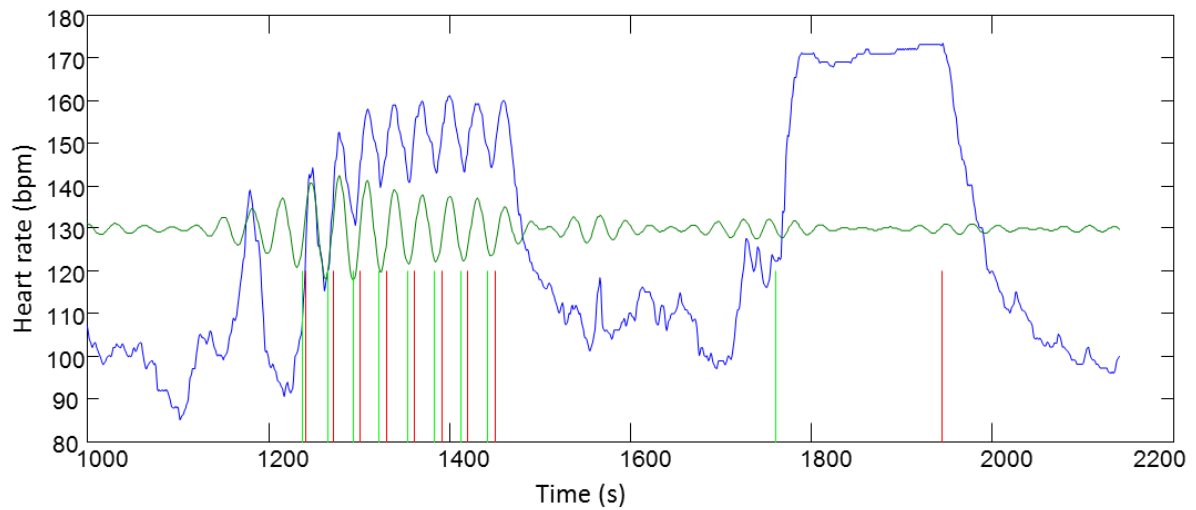


Figure 6: Heart rate kinetics during RSE and all-out test in a representative subject. The blue line indicates the heart rate, while the green horizontal line indicates fluctuations in heart rate signal every thirtieth second. Green vertical lines indicate start of each exercise, red vertical lines indicate stop of each exercise.

HR and VO₂ showed a clear fluctuation in the RSE at 0.033 Hz, which was lacking in the all-out exercise. In the RSE-test the HR-amplitude was 13.3 ± 4.6 bpm, while VO₂-amplitude was 9.3 ± 5.5 ml/kg/min. This indicates that both VO₂ and heart rate responded dynamically to the repeated load. However, the amplitude of the fluctuations did not correlate with any of the performance variables (all $p > 0.05$).

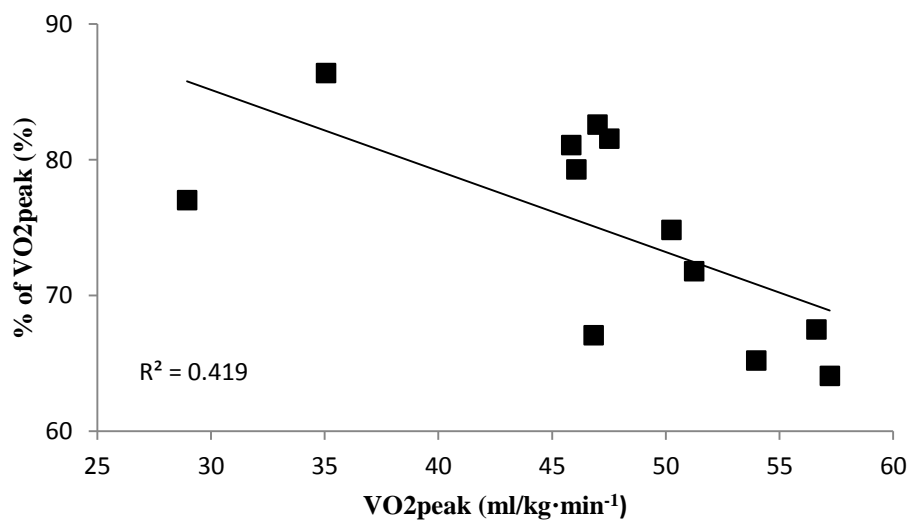


Figure 7: The mean use of VO₂ in RSE presented as percent of VO₂peak in poling, plotted against VO₂peak in poling.

During the RSE-test the blood lactate values increased from sprint 1 to 3 min post sprint 8, with a mean increase of 5.2 ± 2.8 mmol·L⁻¹. Lactate tests showed a significant relationship between increase in lactate, from the first sprint to three minutes post the last sprint, with both sprint performance variables, (total mean power: $r = 0.692$, sprint decrement: $r = 0.603$, both $p < 0.05$). In addition there was a significant correlation between peak mean power and absolute increase in lactate ($r = 0.790$, $P < 0.01$). Average peak lactate value in the repeated

sprint test was $8.5 \pm 3 \text{ mmol}\cdot\text{L}^{-1}$, while the average peak lactate value in the all-out test was $14.6 \pm 3.4 \text{ mmol}\cdot\text{L}^{-1}$.

The EMG-signal from both biceps and triceps show a significant change from sprint to sprint, with a mean negative slope of 6.8 IEMG per sprint ($P < 0.05$) in triceps and a mean positive slope of 5.6 IEMG per sprint ($P < 0.05$) in biceps. However, there was no correlation between performance variables and individual changes in EMG-signal ($P > 0.05$). In addition, the ratio of integrated EMG during pull only and entire cycle, a measure for the functional timing of triceps activity, decreased in the two last repetitions ($1.31 \pm 0.03 \text{ IEMG}$) from the first six ($1.37 \pm 0.04 \text{ IEMG}$). This decrease indicates that triceps brachii activation is relatively less active in the pull phase of the movement and more active in the retrieval phase. Such a clear pattern was not found for biceps.

DISCUSSION

The purpose of the present study was to examine changes in power output, physiological responses and changes in technique during upper-body RSE. While most previous studies on RSE have focused on leg exercises, this study is amongst the first to investigate repeated sprints based solely on isolated upper body work. The biggest decrement in performance took place after the first sprint, and 9.9 of total 12 % decrease in power occurred after sprint 4, indicating a rapid exercise-induced fatigue in upper-body RSE. Our hypothesis that power per stroke rather than movement frequency would predict sprint performance was confirmed by the present findings. Poling rate and poling excursion did not change over the sprints. There was a significant change in triceps activity during the last two sprints in the retrieval phase of the movement, suggesting an alteration of muscle co-ordination during the latter sprints. VO_2 increased rapidly from sprint 1 to sprint 2, however after this increase the VO_2 plateaued at a level of 75% of athletes' average VO_{2peak} poling. In contrast to the increased pattern of VO_2 , the heart rate increased at a steady rate during the RSE-test, reaching its highest level after sprint 8. VO_{2peak} correlated positively with sprint performance during the last three sprints, and lactate increase correlated positively with total mean power and sprint decrement.

Performance

In this study both total mean power and sprint decrement was used as performance variables during RSE. Sprint decrement and total mean power did not relate to each other, which indicate that these variables describe different aspects of RSE performance. The results show an 11.7 % sprint decrement in power from the ideal sprint power to the actual sprint power, indicating that fatigue is occurring. The decrement in power is consistent with previous

research on repeated sprints (2, 16, 31). Most of the loss in power came after the first sprints, which could be due to incomplete resynthesis of PCr between the sprints, and as previous studies reported (11, 32); the ability to resynthesize PCr correlates with RSE performance. Further, Bishop et al. (32) showed that a 24-s recovery period may not be sufficient for complete resynthesis of PCr, which explains the sprint decrement already in early phases of RSE. This is in contrast with findings reporting that 30-s recovery is sufficient to restore power output for up to four consecutive sprints (9). However, these contrasts may be linked to different types of ergometers, training status and initial power output (33, 34). Differences in resynthesis of PCr may be even greater due to contrasting types of movement. This could be evident in our research in upper-body work, and could be likely due to the anatomy of the upper-body, consisting of much smaller muscles and therefore has a smaller oxidative capacity than leg-muscles meaning that the oxygen demanded in resynthesis of PCr could be inadequate or at least more sensitive to fatigue in strenuous exercise (22).

Technique/Kinematics

It was hypothesized that force per stroke rather than poling rate and poling excursion would determine upper-body RSE performance. This is confirmed by our findings, showing that power per stroke did have a strong correlation with total mean power. There was no significant change of poling rate or poling excursion from sprint to sprint, and no significant correlation between poling excursion and poling frequencies with total mean power or sprint decrement. This is in contrast with earlier studies in ice sledge-hockey poling exercise (6), where poling rate was reduced during the sprints. Zory et al. (35) reported from sprint in cross-country skiing that both cycle rate and cycle length were reduced when fatigued. However, these contradictory findings may be due to the Concept 2 mechanical properties

where a flywheel adjusts the air-resistance when higher power is produced and therefore holding the mechanic resistance almost constant, which influence the athletes' ability to increase their poling rate at high intensities. Poling excursion is also influenced by the ergometer's predetermined work path, which is only changeable by the athletes' ability to alter their upper-body position by stretching out more in the retrieval phase or extend the pull behind the trunk. By taking these opportunities in mind, the athletes' options to alter their technique during RSE in the ergometer used in this study are limited.

By measuring EMG-activity in Biceps brachii and Triceps brachii during the repeated sprint test, it was possible to analyse the change in net motor neuron activity (11), which could represent alteration of technique together with fatigue. The results indicate a change in the triceps brachii activation during the pull- and retrieval phase in the last two sprints, where the EMG-signal of the biceps and triceps co-ordination was changed, suggesting a change in triceps timing of activation during the pull and retrieval phase in these sprints. The triceps is seem to act like the agonist in the pull phase and the biceps like the antagonist; however the function of these seems to be modified during the two last sprints. This could be a sign of fatigue or alteration of technique due to fatigue, however this is unclear and should be investigated in future studies. It should be noticed that mean power during the last two sprints increased compared to sprint six, which indicates that the change of co-ordination beneficially affects the performance.

Several studies have concluded that peripheral fatigue like impaired intramuscular conditions (i.e. PCr depletion and decreased pH) is the main cause for sprint decrement (2, 3, 11). However, the change in EMG-signal from sprint to sprint in triceps (and biceps) shows that

alteration of activation is present. The reduction of EMG-signal could be a sign of central fatigue (i.e. reduced muscle activation) which is shown to inhibit repeated sprint performance (1). The reduced muscle activation as a factor of fatigue is not a universal finding (16), however, it is supported by other studies showing that muscle activity decreases during RSE (17, 33). This may be due to reduced motor unit activity and firing rates. However, the exact mechanism behind the decreased EMG-signal cannot be ascertained from surface EMG (33). The reduction of muscle activation did not correlate to sprint decrement in the present study, indicating that there are other limiting factors that are more important to RSE-performance. This is supported by several studies (11, 16, 17), who concluded that fatigue in RSE was most likely present due to failure of excitation contraction coupling and impairment of sarcolemmal excitability (peripheral factors). Mendez-Villanueva et al. (11) suggested that decreased EMG-activity could be the consequence rather than cause of the reduced power output, due to other fatiguing factors, which could also be the case in this study. This could be explained by the properties of muscle fiber type IIX (fast-twitch), which is the main contributor during high-intensity exercise and has the highest twitch tensions, larger action potentials and faster conduction velocities compared to slow-twitch fibers (33). The fast-twitch fibers are highly exposed to fatigue and could thereby result in decreased EMG amplitude in the latter stages of RSE, when slower fibers compensate for the fatigued fast-twitch fibers (33). In contrast to the decrease in triceps-activity during the repeated sprint test, the biceps-activity is significantly increasing during the test. As the biceps are mostly active as an antagonistic stabilizing muscle during the poling phase it could indicate more inhibition from the antagonist during fatiguing processes, or the biceps are more activated during the retrieval-phase in order to compensate for the loss of triceps-activation in the pulling phase. However,

our data are not suitable in order to conclude in which part the increasing activity of the biceps brachii affects performance.

The blood lactate levels increased at a steady rate during the RSE-test, which could be explained by the short breaks between the sprints, leading to incomplete resynthesis of PCr, which decrease the contribution from PCr to ATP-production (36). Therefore a greater contribution from anaerobic lactic sources is demanded (3). The results of this study show a significant correlation between sprint decrement and an absolute increase in lactate which is in compliance with the study by Mendez-Villanueva et al. (33) 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. This leads to the theory that most of the decrement in RSE derive from metabolic disturbances such as decreased pH-values due to high muscle lactate concentration (9). Interestingly, the present study also shows that the athletes with the highest peak power had the greatest increase in absolute lactate, suggesting that a high peak power derives from the contribution of anaerobic lactic sources to ATP. However, results from the 3-min all-out test show higher peak values of lactate than the repeated sprint test, indicating that anaerobic glycogenolysis is not fully activated during RSE, and that the latter stages of RSE is mostly fuelled by ATP from PCr degradation and oxidative metabolism (9). Furthermore, it has been suggested that athletes' with the lowest rate of anaerobic power have the lowest sprint decrement and the lowest peak power (33), which both derive from athletes' ability to fuel energy supply with aerobic power and thereby are less exposed to metabolic disturbances during strenuous exercise. In this study no such relationship was found between mean VO_2 during repeated sprints, sprint decrement and peak power. In line with the findings by Bishop and Spencer (34) our results imply that the athletes with the highest sprint

decrement also had the highest peak power in the first sprint, which Bishop and Spencer concluded was due to high utilization of pre-stored energy, which leads to higher demand of PCr resynthesis. Due to the even shorter breaks in this study compared to the test in the study by Bishop and Spencer (22-s versus 24-s); this is also likely to be evident here.

Energy Consumption

The VO_2 in the RSE-test increased significantly from sprint 1 to sprint 2, after which the VO_2 was stable at around 75 % of VO_{2peak} after each sprint. This shows that the VO_2 in high intensity poling increases rapidly and indicates that the oxygen dependent processes are influential at an early stage of RSE. Furthermore, the 75 % level of VO_{2peak} is in compliance with the findings by Balsom et al. (10) in cycling, further confirming that oxidative metabolism contributes to ATP resynthesis during exercise and to fuel recovery processes during breaks. Despite the increase in VO_2 there is a significant decrease in power output during the test. This supports our earlier remarks that decrement in power derives from a decrease in anaerobic energy sources, mainly PCr, partly anaerobic glycogenolysis which is supported by Racinais et al. (17). Like several previous studies on RSE (6, 32) this study found no significant correlation between VO_{2peak} poling or VO_{2max} running and RSE performance; however the results of this study showed that VO_{2peak} did correlate significantly with power per stroke in the last three sprints in our test. This indicates that oxygen capacity could be an important factor during the latter stages of RSE, influencing in both the recovery phase between sprints and energy formation during sprints. It is evident that PCr is the most important energy source, making the oxidative processes in the resynthesis of PCr an important part of the recovery-phase in RSE (32). It should be noticed that oxygen-dependent processes in the muscles (peripheral), like resynthesis of PCr and removal of accumulated intracellular phosphate (6), could be the most crucial parts of RSE recovery (8).

This suggests that the recovery is both depending on oxygen delivery from central systems and peripheral processes in the muscles.

In contrast to the findings showing a correlation between VO_2peak and performance during the latter stages of RSE, our results show a negative correlation between the ability to use a high percentage of VO_2peak during RSE and power per stroke in the last three sprints. This suggests that athletes with the lowest use of their oxygen capacity during RSE perform better during the latter stages of a RSE. This may be due to their better anaerobic capacities such as higher rate of energy from phosphocreatine and enhancement of glycolytic energy sources. However, it may suggest that high VO_2peak in poling could be beneficial for athletes in RSE, since having a high VO_2peak seems to make the athletes use a lower percentage of their individual VO_2peak in average use of VO_2 during the RSE test (Figure 7). Thereby the athletes are not exposed to a lack of oxygen, which may cause the lowered power per stroke during the last three sprints. The physiological mechanism behind this could be a more efficient use of oxygen-dependent processes like resynthesis of PCr together with better removal of accumulated intracellular inorganic phosphate (6), in athletes' with a high VO_2peak . Sandbakk et al. (6) reported that aerobic fitness did not correlate with performance in their upper-body exercise test, and suggests that this response derives from the relatively low muscle mass that is active in the upper-body during exercise, making more peripheral factors responsible for recovery of the upper limbs.

The kinetics of VO_2 between sprints did not correlate to sprint performance. This indicates that the ability to have a fast response in the cardiorespiratory system in response to changing

intensities do not affect upper-body RSA. However, this is in contrast to what was earlier suggested in a study on leg exercise by Dupont et al. (31).

The current study provides some different data on the importance of oxidative metabolism in repeated sprint performance. Bishop et al. (32), reported that differences in oxidative capacity would likely be more influential on performance in RSE on untrained to moderately trained individuals. However, in elite athletes like those used in this study, the oxidative capacity has less influence on RSA.

There is a clear response in the heart rate signal in every sprint with the heart rate increasing at a steady rate from sprint 1-8. However, it seems that the heart rate recovers to near the same baseline-level in each break, indicating that the cardiorespiratory system adapts fast to the intensity level. The fast recovery of HR is normal in well-trained athletes, as reported by Lee (37), and indicates that upper-body work has the same cardiorespiratory response as leg-work, which is confirmed by the findings of Sandbakk et al. (6).

Upper-body repeated sprints

In comparison with whole-body RSE this study shows that upper-body RSE has the same level of sprint decrement during the test protocol (2). This implies that the same level of fatigue is occurring during RSE in upper-body and whole-body exercise. However, there are some different findings on what is causing the fatigue and how recovery of fatigue works. It is evident from the results of this study that upper-body VO_{2peak} accounts for approximately 65% of the athletes VO_{2max} in running, suggesting that upper-body activity is not enabling all the potential of the cardiorespiratory system. There are no other studies comparing VO_{2-}

capacity in upper-body poling exercise with VO_2 -capacity in running. Interestingly, individual capacity in upper-body and whole-body VO_2 represented by $\text{VO}_{2\text{peak}}$ in poling and $\text{VO}_{2\text{max}}$ in running did not correlate. This shows that cardiorespiratory capacity in whole-body exercise does not necessarily determine upper-body aerobic capacity. This was expected taking results of other research on upper-body in mind (18). This indicates that there are differences between whole-body and upper-body cardiorespiratory capacity. In addition, differences between upper-body and whole-body physiological response could be more evident because of the upper-body's inability to consume the lactate in the same rate as the legs. Sandbakk et al. (6) referred to the upper-body as the main producer of lactate, while the legs are the main consumers of lactate. In our repeated sprint test the athletes' legs were inactive and could therefore not contribute much in lactate consumption, making the athletes more sensitive to decreased pH because of accumulated lactate. Differences between in upper-body versus whole-body physiological responses (i.e. VO_2 and lactate) may be influenced by the athletes' ability to withstand high lactate levels when performing work where both the upper-body and the legs are active. However, when they are not able to actively use their lower limbs their capacity to remove lactate may be reduced. This may have influenced the relationship between lactate increase and sprint decrement.

Limitations

In this study, the sprint decrement method could be flawed by the fact that an increase in power was found in the last two sprints compared with the sprint before. This could be due to "pacing", which means that the athletes allocates an even distribution of effort during the sprints in order to assure that they can perform well during the last sprints as well as the first. This will affect the sprint decrement score by its artificial low power output during the middle

phase of the repeated sprints, and further affects the total power from which the decrement score is calculated. However, in well-trained cross-country skiers, the ability to resist fatigue is high, and compared to their normal form of competition, they are used to high power outputs in the latter stages.

Conclusions

The current study provides new insight on repeated sprint performance and physiology during isolated upper-body work. The sprint decrement was significant already after the first sprint, and 10 % of the total 12 % decrease appeared after sprint 4 in the current protocol with 8-s work periods and 22-s breaks between sprints. VO_2peak correlated to power per stroke in the latter stages of RSE-test indicating that oxidative processes are influential during the latter stages of RSE. The increase in blood lactate correlated with total mean power, sprint decrement and peak power, implying that athletes with the highest increase in lactate have the ability to produce the highest power. These results indicate that high maximal output is beneficial in order to perform well in upper-body RSE, even if the decrement is high. Furthermore it seems like a change in the activation of triceps during the retrieval phase is indicating an alteration of technique in order to withstand fatigue. However, this is not confirmable by the measurements of this study, and should be investigated in more detail in future studies.

Acknowledgements

This study was supported financially by the Mid-Norway Department of the Norwegian Olympic Committee. I would like to thank my supervisors Øyvind Sandbakk and Gertjan Ettema for their help and guidance. I would also like to thank Xiangchun Tan, Knut Skovereng, Matthew Spencer, Boye Welde, Terje Gjøvåg, Mads Hansen and Ann Magdalen Hegge for help in the laboratory. Finally, I would like to thank the subjects for their valuable cooperation and participation in this study.

REFERENCES

1. Girard O, Mendez-Villanueva A, Bishop D. Repeated-sprint ability - part I: factors contributing to fatigue. *Sports Med.* 2011;41(8):673-94. Epub 2011/07/26.
2. Spencer M, Bishop D, Dawson B, Goodman C. Physiological and metabolic responses of repeated-sprint activities: specific to field-based team sports. *Sports Med.* 2005;35(12):1025-44. Epub 2005/12/13.
3. Glaister M. Multiple-Sprint Work: Methodological, Physiological, and Experimental Issues. *International journal of sports physiology and performance.* 2008;3:107-12.
4. Lindinger SJ, Holmberg HC, Muller E, Rapp W. Changes in upper body muscle activity with increasing double poling velocities in elite cross-country skiing. *European journal of applied physiology.* 2009;106(3):353-63. Epub 2009/03/13.
5. Stoggl T, Muller E, Ainegren M, Holmberg HC. General strength and kinetics: fundamental to sprinting faster in cross country skiing? *Scandinavian journal of medicine & science in sports.* 2011;21(6):791-803. Epub 2010/05/25.
6. Sandbakk O, Spencer M, Ettema G, Sandbakk SB, Skovereng K, Welde B. The Physiology and Biomechanics of Upper-Body Repeated Sprints in Ice Sledge Hockey. *International journal of sports physiology and performance.* 2013. Epub 2013/05/01.
7. Gabbett TJ. The development of a test of repeated-sprint ability for elite women's soccer players. *Journal of strength and conditioning research / National Strength & Conditioning Association.* 2010;24(5):1191-4. Epub 2010/04/14.
8. Glaister M. Multiple sprint work : physiological responses, mechanisms of fatigue and the influence of aerobic fitness. *Sports Med.* 2005;35(9):757-77. Epub 2005/09/06.
9. Gaitanos GC, Williams C, Boobis LH, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol.* 1993;75(2):712-9. Epub 1993/08/01.
10. Balsom PD, Gaitanos GC, Soderlund K, Ekblom B. High-intensity exercise and muscle glycogen availability in humans. *Acta physiologica Scandinavica.* 1999;165(4):337-45. Epub 1999/06/01.
11. Mendez-Villanueva A, Edge J, Suriano R, Hamer P, Bishop D. The recovery of repeated-sprint exercise is associated with PCr resynthesis, while muscle pH and EMG amplitude remain depressed. *PloS one.* 2012;7(12):e51977. Epub 2013/01/04.

12. Edge J, Hill-Haas S, Goodman C, Bishop D. Effects of resistance training on H⁺ regulation, buffer capacity, and repeated sprints. *Medicine and science in sports and exercise*. 2006;38(11):2004-11. Epub 2006/11/11.
13. Hamilton AL, Nevill ME, Brooks S, Williams C. Physiological responses to maximal intermittent exercise: differences between endurance-trained runners and games players. *Journal of sports sciences*. 1991;9(4):371-82. Epub 1991/01/01.
14. Góralczyk R, Mikolajec K, Poprzecki S, Zajac A, Szyngiera W, Waskiewicz Z. Kinematic analysis of intermittent sprints of elite soccer players. *Journal of Human Kinetics*. 2003;10:107-20.
15. Bishop D, Girard O, Mendez-Villanueva A. Repeated-sprint ability - part II: recommendations for training. *Sports Med*. 2011;41(9):741-56. Epub 2011/08/19.
16. Perrey S, Racinais S, Saimouaa K, Girard O. Neural and muscular adjustments following repeated running sprints. *European journal of applied physiology*. 2010;109(6):1027-36. Epub 2010/04/02.
17. Racinais S, Bishop D, Denis R, Lattier G, Mendez-Villaneuva A, Perrey S. Muscle deoxygenation and neural drive to the muscle during repeated sprint cycling. *Medicine and science in sports and exercise*. 2007;39(2):268-74. Epub 2007/02/06.
18. Stoggl T, Lindinger S, Muller E. Biomechanical validation of a specific upper body training and testing drill in cross-country skiing. *Sports biomechanics / International Society of Biomechanics in Sports*. 2006;5(1):23-46. Epub 2006/03/09.
19. Hawkeswood J, Finlayson H, O'Connor R, Anton H. A pilot survey on injury and safety concerns in international sledge hockey. *International journal of sports physical therapy*. 2011;6(3):173-85. Epub 2011/09/10.
20. Uzun S, Pourmoghaddam A, Hieronymus M, Thrasher TA. Evaluation of muscle fatigue of wheelchair basketball players with spinal cord injury using recurrence quantification analysis of surface EMG. *European journal of applied physiology*. 2012;112(11):3847-57. Epub 2012/03/08.
21. Tesch PA. Physiological characteristics of elite kayak paddlers. *Canadian journal of applied sport sciences Journal canadien des sciences appliquees au sport*. 1983;8(2):87-91. Epub 1983/06/01.
22. Calbet JA, Holmberg HC, Rosdahl H, van Hall G, Jensen-Urstad M, Saltin B. Why do arms extract less oxygen than legs during exercise? *American journal of physiology*

Regulatory, integrative and comparative physiology. 2005;289(5):R1448-58. Epub 2005/05/28.

23. Van Hall G, Jensen-Urstad M, Rosdahl H, Holmberg HC, Saltin B, Calbet JA. Leg and arm lactate and substrate kinetics during exercise. *American journal of physiology Endocrinology and metabolism*. 2003;284(1):E193-205. Epub 2002/10/22.

24. Fukuoka Y, Endo M, Kagawa H, Itoh M, Nakanishi R. Kinetics and steady-state of VO₂ responses to arm exercise in trained spinal cord injury humans. *Spinal cord*. 2002;40(12):631-8. Epub 2002/12/17.

25. Koppo K, Bouckaert J, Jones AM. Oxygen uptake kinetics during high-intensity arm and leg exercise. *Respiratory physiology & neurobiology*. 2002;133(3):241-50. Epub 2002/11/12.

26. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 2000;10(5):361-74. Epub 2000/10/06.

27. Spencer M, Fitzsimons M, Dawson B, Bishop D, Goodman C. Reliability of a repeated-sprint test for field-hockey. *Journal of science and medicine in sport / Sports Medicine Australia*. 2006;9(1-2):181-4. Epub 2006/04/04.

28. Forbes SC, Chilibeck PD, Craven B, Bhambhani Y. Comparison of a double poling ergometer and field test for elite cross country sit skiers. *North American journal of sports physical therapy : NAJSPT*. 2010;5(2):40-6. Epub 2011/05/19.

29. Sandbakk O, Welde B, Holmberg HC. Endurance training and sprint performance in elite junior cross-country skiers. *Journal of strength and conditioning research / National Strength & Conditioning Association*. 2011;25(5):1299-305. Epub 2010/11/18.

30. Ingjer F, Myhre K. Physiological effects of altitude training on elite male cross-country skiers. *Journal of sports sciences*. 1992;10(1):37-47. Epub 1992/02/01.

31. Dupont G, Millet GP, Guinhouya C, Berthoin S. Relationship between oxygen uptake kinetics and performance in repeated running sprints. *European journal of applied physiology*. 2005;95(1):27-34. Epub 2005/06/25.

32. Bishop D, Lawrence S, Spencer M. Predictors of repeated-sprint ability in elite female hockey players. *Journal of science and medicine in sport / Sports Medicine Australia*. 2003;6(2):199-209. Epub 2003/08/30.

33. Mendez-Villanueva A, Hamer P, Bishop D. Fatigue in repeated-sprint exercise is related to muscle power factors and reduced neuromuscular activity. *European journal of applied physiology*. 2008;103(4):411-9. Epub 2008/03/28.
34. Bishop D, Spencer M. Determinants of repeated-sprint ability in well-trained team-sport athletes and endurance-trained athletes. *The Journal of sports medicine and physical fitness*. 2004;44(1):1-7. Epub 2004/06/08.
35. Zory R, Vuillerme N, Pellegrini B, Schena F, Rouard A. Effect of fatigue on double pole kinematics in sprint cross-country skiing. *Human movement science*. 2009;28(1):85-98. Epub 2008/10/07.
36. Bogdanis GC, Nevill ME, Boobis LH, Lakomy HK, Nevill AM. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *The Journal of physiology*. 1995;482 (Pt 2):467-80. Epub 1995/01/15.
37. Lee CM, Mendoza A. Dissociation of heart rate variability and heart rate recovery in well-trained athletes. *European journal of applied physiology*. 2012;112(7):2757-66. Epub 2011/11/30.