Title: **Performance and metabolism in repeated sprint exercise: Effect of recovery intensity**

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Running Head: Recovery intensity and repeated-sprint bouts

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Abstract

This study investigated the effects of a moderate (MI) and a low intensity (LI) active recovery (both compared to a passive recovery) on repeated-sprint performance and muscle metabolism. Nine, male, subjects performed three repeated-sprint cycle tests (6 x 4-s sprints, every 25 s) in a semi-randomized, counter-balanced order. Recovery after each sprint for the MI and LI trials respectively was 60 W ($\sim 35\% \dot{V}O_{2\max}$) and 20 W ($\sim 20\% \dot{V}O_{2\max}$). Biopsies were taken from the vastus lateralis pre- and immediately post-test during the MI and LI trials to determine adenosine triphosphate (ATP), phosphocreatine (PCr) and lactate (MLa⁻) content. Compared to passive, significant reductions in peak power of 3.4 to 6.0% were recorded in the MI trial (4 of 6 sprints; $P < 0.05$) and reductions of 3.5 to 3.7% in the LI trial (2 of 6 sprints; $P < 0.05$), with no differences between the two active trials. No significant differences were evident in ATP, PCr and MLa⁻ between the two active recovery trials. In summary, peak power indices during the repeated-sprint test were inferior in the MI and LI active recovery trials, compared to passive. The minimal differences in performance and muscle metabolites between the MI and LI trials suggest that any low-to-moderate level of muscle activation will attenuate the resynthesis of PCr and the recovery of power output during repeated short-sprint exercise.

Keywords: Peak power output, intermittent exercise, phosphocreatine, muscle lactate

Introduction

Studies that have documented the sprint activity of field-based team sports (via time-motion analysis), have typically reported only mean data (i.e., on average a 2- to 3-s sprint is performed every 1 to 3 min during a game) (Docherty et al. 1988; Lothian and Farrally 1994; Mohr et al. 2003). However, while mean data is useful, it does not provide sufficient information regarding intense periods of play that may be critical to the outcome of a game. Through a detailed analysis of international field-hockey competition, we have recently reported that while players perform a sprint on average every 2 min, they may perform 3 to 7 sprints with little recovery, within a repeated-sprint bout (defined as a minimum of three sprints, with a mean recovery duration between sprints of less than 21 s) (Spencer et al. 2004). Furthermore, we reported that most of the recovery within the repeated-sprint bouts was of an active nature (i.e., jogging, walking and striding) (Spencer et al. 2004), with the remaining time consisting of passive recovery.

To date, very few studies have compared the effects of active versus passive recovery on repeated-sprint ability. Studies that have compared a passive versus an active recovery (i.e., 30 to 40% of $\dot{V}O_{2max}$) on subsequent exercise performance have generally reported an improved performance or power output recovery for the active condition (Bogdanis et al. 1996; Connolly et al. 2003). However, these aforementioned studies have not used sprint and recovery durations specific to the team-sport situation. One study that did use a protocol more specific to team-sport performance (i.e., 8 x 6-s cycling sprints with 30 s recovery between sprints) reported an increase in both peak power output and total work when an active (cycling at 60 W; ~30 to 40% $\dot{V}O_{2max}$), rather than passive recovery was completed (Signorile et al. 1993). In contrast, we have recently reported that active (approx 32% $\dot{V}$

$\dot{V}O_{2\max}$), compared to passive recovery, resulted in a lower peak power for the final sprint and a greater power decrement during a repeated-sprint test consisting of 6 x 4-s cycle sprints departing every 25 s (Spencer et al. 2006a). The improved repeated-sprint performance during the passive, compared to the active, recovery trial was associated with a strong trend for lower phosphocreatine (PCr) and a significantly higher muscle lactate (MLa⁻) (Spencer et al. 2006a).

While other studies have also reported the effects of recovery mode (active vs passive) on repeated-sprint performance, no study has investigated if there is an intensity effect of active recovery (i.e., a jogging versus walking intensity) on metabolism and subsequent performance. The higher oxygen cost of a more intense active recovery (Yoshida et al. 1996) may result in a decreased availability of mitochondrial ATP for PCr resynthesis (Bessman and Carpenter 1985) and subsequently greater impairments in sprint performance (Bogdanis et al. 1995). Furthermore, while both the accumulation of metabolites and muscle activation are required to stimulate anaerobic glycolysis (Crowther et al. 2002), it is not known if different intensities of active recovery will affect MLa⁻ accumulation. Therefore, the purpose of this study was to investigate the effect of a low intensity (LI) versus a moderate intensity (MI) active recovery (with passive recovery used as the control condition) on sprint performance and muscle metabolism, during a repeated-sprint test that mimics the sprint and recovery durations commonly seen in team sports.

Methods

Subjects

Nine moderately-trained, male, team-sport athletes (age, 19 ± 2 y; body mass, 70.7 ± 9.2 kg; $\dot{V}O_{2\max}$, 3.75 ± 0.50 L·min⁻¹, mean $\pm$ SD) volunteered for this investigation, which was

approved by the institutional Human Research Ethics Committee. All subjects participated in intermittent sports and trained regularly (6 ± 2 sessions per week). After being fully informed of the risks associated with participation, each subject gave his written informed consent.

Experimental Overview

The subjects were required to undertake five testing sessions over a four-week period. The initial testing session familiarized the subjects with the procedures of the repeated-sprint test.

On a separate day, a graded exercise test was conducted to determine $\dot{V}O_{2\max}$. Within four to seven days of the familiarisation session, subjects undertook the first of three semi-randomised, counter-balanced experimental trials on a sprint-cycle ergometer. These trials were conducted seven days apart, over a three week-period. Trial PASSIVE consisted of 6 x 4-s maximal sprints separated by 21 s of passive recovery. Trial MI consisted of the same repeated-sprint test, however, a MI active recovery (cycling at 60 W, approximately 35% of $\dot{V}O_{2\max}$) was undertaken between the sprints. Trial LI also involved active recovery between the sprints, with a LI recovery (cycling at 20 W, approximately 20% of $\dot{V}O_{2\max}$) being undertaken between the sprints. A total of four muscle biopsies were taken from each subject; pre- and immediately post-test for both the LI and MI recovery trials. No biopsies were taken in the passive recovery trial. This was done in order to minimise the invasive demands on the subjects and also due to the fact that we have previously investigated the muscle metabolite differences between a MI active recovery trial and a passive recovery trial (Spencer et al. 2006a). All experimental trials were conducted at the same time of day (± 2 h) for each subject, to minimise the effect of diurnal variation. Subjects were asked to maintain their normal diet throughout the study and to abstain from training on the day prior to each

test. They were also instructed to be adequately hydrated and not to eat for three hours prior to each test. Diaries were administered to record food and fluid consumption during the two days prior to each test and all subjects were instructed to replicate this for each subsequent testing session.

Cycle Ergometers

Air-braked cycle ergometers were used to conduct all sprint tests, interfaced with a computer system to allow for the collection of data for the calculation of work and power generated during each flywheel revolution (Cedaa, Western Australian Institute of Sport, Perth, Australia). These ergometers require subjects to pedal against air resistance caused by rectangular vanes attached perpendicular to the axis of rotation of the flywheel. The power output of the air-braked, cycle ergometer is proportional to the cube of the flywheel velocity. A magnetic sensor monitored the velocity of the flywheel at a sampling rate of one pulse per flywheel revolution. Before testing, each ergometer was dynamically calibrated on a mechanical rig (Bio-Med Electronic Services & South Australian Sports Institute, Adelaide, Australia) across a range of power outputs (100-2000 W) and a range of flywheel velocities (approximately 4 to 25 Hz) for cadences of 50 to 120 rpm (with increments of 10 rpm).

Graded Exercise Test

The graded exercise test for $\dot{V}O_{2\max}$ determination was performed on an air-braked cycle ergometer (Evolution, Melbourne, Australia) and consisted of graded exercise steps, using a continuous protocol. The test commenced at 25 W and thereafter the intensity increased by 50 W every 2 min until volitional exhaustion. The criteria for achieving $\dot{V}O_{2\max}$ was a plateau in $\dot{V}O_2$ despite an increase in exercise intensity, with a respiratory exchange ratio

above 1.1. During the graded exercise test, expired air was continuously monitored for the analysis of O₂ and CO₂ concentrations (Ametek gas analysers SOV S-3A and COV CD3A respectively, Pittsburgh, Pa., USA) and data was reported as an average of 30-s intervals. Ventilation was recorded every 30 s using a turbine ventilometer (Morgan, Model 096, Kent, England). The gas analysers were calibrated immediately before and verified after each test using three certified gravimetric alpha-grade gas mixtures (BOC Gases, Chatswood, Australia); the ventilometer was calibrated pre-exercise and verified post-exercise using a mechanical syringe pump of one to four litres.

Repeated-sprint cycle tests

A standardized warm-up was performed prior to all repeated-sprint cycle tests. This consisted of 5 min of cycling at 60 W followed by 1 min of passive rest. Two, 2-s maximal sprints were then performed followed by 2 min of slow cycling (approximately 10 to 20 W). Finally, one 4-s maximal sprint was performed followed by 2 min of slow cycling (approximately 10 to 20 W) and 4 min of passive rest. The repeated-sprint test comprised 6 x 4-s maximal sprints departing every 25 s (i.e., 21 s of recovery was completed between each sprint). The recovery during the passive trial required the subjects to decelerate to a stop as soon as possible (approximately 1 to 2 s) after the preceding sprint and to remain stationary. The recovery during the active trials required the subjects to decelerate (approximately 1 to 2 s) on immediate completion of the sprint and then cycle at either 60 W (MI trial) or 20 W (LI trial) for 15 s. The remaining 6 s of recovery, in all trials, required the subjects to assume the start position and await the countdown to the next sprint. The starting position of each sprint was controlled by standardising the pedal and crank angle, with the crank angle being approximately 45 degrees to the vertical. The feet were secured on the pedals via toe clips and heel straps. The active deceleration after each sprint was included in an attempt to mimic

the extra energy expenditure that would most likely occur during repeated-sprint bouts in team-sport performance. All repeated-sprint cycle tests were performed in the standing position, on an air-braked, front-access cycle ergometer (Model Ex-10, Repco, Victoria, Australia), which is more specific to the muscle actions of running than a conventional cycle ergometer (i.e., subjects are seated). A repeated-sprint cycle test, similar to the present study, has been reported to be valid for assessing over-ground running repeated-sprint ability (Bishop et al. 2001).

Muscle biopsies

The skin and fascia overlaying the vastus lateralis was anaesthetised (5 mL, 1% Xylocaine) prior to the incision being made. One incision was used for both the pre-test and the post-test biopsies, with the needle inserted at different angles. The pre-test samples were taken during supine rest, approximately 5 min prior to the warm-up. The post-test samples were taken on immediate completion of the repeated-sprint tests, for both the MI and LI active recovery trials. The post-test biopsies were taken while the subject was supported on the cycle ergometer. The percutaneous needle biopsy technique (Bergstrom 1962), with suction applied manually, was used to obtain the samples. Each sample (50 to 80 mg wet muscle per sample) was immersed in liquid nitrogen within 10 s of being taken, then removed from the needle, and stored at -80°C until freeze dried for analysis. An extract of the muscle was obtained after the blood, fat and connective tissue were removed from the sample.

Muscle analysis

Freeze-dried rest and post-exercise muscle samples (3 – 3.8 mg) were enzymatically assayed for [ATP], [PCr] and [MLa⁻] after being extracted by the addition of 6% perchloric acid, before being centrifuged (10,000 g x 10 min). The supernatant was removed and neutralized

by the addition of 2.4 mol/L potassium hydroxide and 3 mol/L potassium chloride. Samples were centrifuged again and the supernatant was stored at -80°C. The metabolites, [ATP], [PCr] and [MLa⁻] were measured using a method described previously (Arthur and Hochachka 1995).

For the analysis of muscle pH, freeze-dried muscle samples (2.5 to 3.2 mg) were homogenized on ice for 2 min in a solution containing sodium fluoride (10 mM) at a dilution of 30 mg dry muscle·mL⁻¹ of homogenizing solution (Mannion et al. 1993). The muscle homogenate was then placed in a circulation water bath at 37°C for 5 min prior to and during the measurement of pH. The pH measurements were made with a microelectrode (MI-415, Microelectrodes Inc, Bedford, NH, USA) connected to a pH meter (SA 520, Orion Research Inc, Cambridge, MA, USA).

Calculation of test scores

An absolute (total work) and relative (percent decrement over the repeated efforts) repeated-sprint ability score was calculated for the 6 x 4-s cycle test. The calculated total work was the sum of all six sprint bouts (e.g., 270 J·kg⁻¹). The percent sprint decrement was calculated as [100 – (total work / ideal work x 100)]. The ideal work was the highest individual work bout, usually the first sprint bout, multiplied by six (e.g., 49 x 6 = 294 J·kg⁻¹). The ideal work was calculated separately for each subject, for each experimental trial. An example of the calculated percent sprint decrement is presented below;

$$\begin{aligned}\text{Sprint decrement (\%)} &= 100 - (\text{total work} / \text{ideal work} \times 100) \\ &= 100 - (270 / 294 \times 100) \\ &= 100 - 91.83 \\ &= 8.17 \%\end{aligned}$$

We have previously reported the reliability of the 6 x 4-s cycle test using the same procedures and cycle ergometer as the present study, with the typical error for total work and work decrement being 4.5 and 17.2%, respectively (Spencer et al. 2006a). The formula used for calculating the work decrement was the same for calculating the power decrement.

Statistical Analysis

Simple group statistics were presented as means $\pm$ between-subject standard deviations. First, the traditional approach of determining statistical significance, via the *P*-value, was conducted. Analysis of the performance and physiological data from the recovery conditions were assessed using a two-factor (recovery and time) analysis of variance (ANOVA) with repeated measures. A significant interaction was then analyzed by Newman-Keuls post-hoc tests to determine at which time points an effect was observed between trials. Differences in total work, work decrement and power decrement were compared using paired-sample t-tests. Significance was accepted at $P < 0.05$. Statistica software for Windows (version 6.0, StatSoft, Inc., Tulsa, OK) was used for the analyses.

Precision of estimation and magnitude-based inferences were also conducted. Confidence limits for the true mean values of effects were estimated with a spreadsheet (Hopkins 2003) via the unequal-variances t statistic computed for the change scores between the three trials (MI, LI and passive recovery). Each subject's change score, for the three comparisons, was presented as a percentage via analysis of log-transformed values. The spreadsheets provide a precision of the estimate as 90% confidence limits. The magnitude of differences between recovery trials were expressed as standardized mean differences (Cohen effect sizes). The criteria to interpret the magnitude of the effect sizes were; < 0.2 trivial, 0.2–0.6 small, 0.6–1.2

moderate, 1.2-2.0 large and > 2.0 very large (Hopkins 2000). In test measures related to team sports, where there is no direct relationship between fitness-test performance and team performance, it is suggested that an appropriate default for the smallest worthwhile change in test performance is one-fifth (Cohen effect size = 0.20) of the between-subject standard deviation (Hopkins 2004). The smallest worthwhile change for the main performance variables in the present study (i.e., total work and peak power for each of the six sprint bouts) was approximately the same as the typical error of measurement of this exact laboratory based, repeated-sprint test (Spencer et al. 2006a). For the test variables of percent work decrement and percent peak power decrement, where we have reported the typical error of measurement to be greater than the previously mentioned variables (Spencer et al. 2006a) substantial changes in test performance were only interpreted when the change was greater than the typical error of measurement. To estimate the chances that effects were substantial for the physiological variables (i.e., muscle metabolites, muscle pH), the smallest worthwhile changes were also calculated as one-fifth of the between-subject standard deviation (Hopkins 2004).

Results

Performance data – peak power & total work

Compared to the passive trial, the MI trial resulted in significantly lower peak power outputs for sprints 2, 3, 4 and 6 ($P = 0.03$, 0.02 , 0.004 and 0.046 , respectively) and the LI trial resulted in significantly lower peak power outputs for sprints 4 and 6 ($P = 0.045$ and 0.01 , respectively) (Figure 1). There were no significant differences in peak power output between the MI and LI trials. However, there was a strong trend for a lower peak power in the MI trial, compared to LI, in sprint 3 ($P = 0.051$). There were no significant differences for total work between the three conditions of passive, MI and LI recovery (255.6 ± 41.5 , 245.6 ± 30.5

and $245.2 \pm 29.5 \text{ J}\cdot\text{kg}^{-1}$, respectively). The magnitude of differences, via effect sizes, between trials for the performance data are presented in Table 1.

INSERT TABLE 1 AND FIGURE 1 ABOUT HERE

Performance data – work & power decrement

There were no significant differences in work decrement between the three recovery trials; passive vs MI ($P = 0.28$; ES = 0.58, small), passive vs LI ($P = 0.30$; ES = 0.58, small) and MI vs LI ($P = 0.83$; ES = 0.10, trivial) (Figure 2a). Similarly, there were no significant differences in power decrement between the three recovery trials; passive vs MI ($P = 0.07$; ES = 1.03, moderate), passive vs LI ($P = 0.12$; ES = 0.57, small) and MI vs LI ($P = 0.17$; ES = 0.46, small) (Figure 2b).

INSERT FIGURE 2 ABOUT HERE

Muscle data

The pre- and post-test muscle metabolite concentrations, as well as the delta change (i.e., differences between pre- and post-test values), for the LI and MI recovery trials are presented in Table 2. There were no significant differences between the LI and the MI trials, for resting [ATP] (2.3%; $P = 0.37$; ES = 0.18, trivial), [PCr] (1.4%; $P = 0.71$; ES = 0.13, trivial) or [MLa⁻] (7.2%; $P = 0.85$; ES = 0.06, trivial). Similarly, there were no significant differences between the two recovery trials for the delta change for [ATP] (11.3%; $P = 0.86$; ES = 0.08, trivial), [PCr] (2.1%; $P = 0.79$; ES = 0.12, trivial) and [MLa⁻] (19.4%; $P = 0.59$; ES = 0.17, trivial). There were also no significant differences between the LI and MI recovery trials for resting muscle pH (7.02 ± 0.08 and 7.02 ± 0.09 , respectively; 1.1%; $P = 0.59$; ES = 0.08,

trivial) or for the delta change of muscle pH (0.15 ± 0.14 and 0.21 ± 0.11 , respectively; 34.5%; $P = 0.26$; ES = 0.53, small).

INSERT TABLE 2 ABOUT HERE

Discussion

The major finding of this study was that there were significant and substantial reductions in the individual peak power outputs within the repeated-sprint test, during both the MI and LI active recovery trials, when compared to the passive recovery trial. However, there was no difference in performance between the MI and LI active recovery trials. Furthermore, there were no significant or substantial differences in muscle metabolite content (i.e., ATP, PCr and MLa^-) between the MI and LI recovery trials.

Effects of MI and LI active recovery on repeated-sprint performance, compared to passive recovery

To date, only two studies have investigated the effects of active versus passive recovery on repeated-sprint performance, specific to the sprint and recovery durations associated with field-based team sports (i.e., sprint and recovery durations of 4-6 s and 20-30 s, respectively) (Signorile et al. 1993; Spencer et al. 2006a). Although the exercise protocols in the two aforementioned studies were quite similar, contrasting results were reported. The study of Signorile et al. (1993) reported that both peak power output and total work were significantly greater in the active recovery trial. In contrast, our previous study, revealed both a significantly lower peak power output for the last sprint and a greater power decrement in the active, compared to passive recovery trial, with no differences between trials for total work (Spencer et al. 2006a).

The data from the present study support our previous findings, with both studies reporting reductions in peak power indices during the active recovery trials. For example, individual peak power outputs were lower in the MI trial, compared to the passive trial (i.e., 4 of the 6 sprints were significantly lower with small to moderate effect sizes). In addition, two of the individual peak power outputs were significantly lower in the LI trial, compared to the passive trial. When the two active recovery trials were compared, there were no significant differences in the individual peak power outputs. These peak power data suggest that active recovery of low- to moderate-intensity is detrimental to repeated-sprint performance, when compared to a passive recovery.

There were no significant or substantial differences in total work between the three trials, which support the results of our previous study (Spencer et al. 2006a). There was also no significant difference in the peak power decrement between the MI trial and the passive trial ($P = 0.07$), which contrasts with the findings of our previous study (Spencer et al. 2006a). However, there was a moderate effect size in the peak power decrement ($ES = 1.03$) with the MI trial being greater than the passive trial. There was a small effect size of a greater peak power decrement for the LI compared to the passive trial. In addition, there was a small effect size suggesting a greater decrement in the MI compared to the LI recovery trial. The peak power decrements of all three recovery trials in the present study (i.e., range of 6 to 9%), are within the range of other repeated-sprint studies using similar protocols conducted on a sprint cycle ergometer (Fitzsimons et al. 1993; Spencer et al. 2006a) and also in over-ground running (Dawson et al. 1993; Fitzsimons et al. 1993; Spencer et al. 2006b).

Effects of MI versus LI active recovery on muscle metabolism

There is evidence reporting an effect of recovery type (active or passive) on muscle metabolite changes during repeated bouts of high-intensity exercise (McAinch et al. 2004; Yoshida et al. 1996). We have previously reported greater changes in muscle metabolites (i.e., lower PCr and higher MLa^-) when a MI active, compared to a passive recovery, was performed during a test of repeated-sprint ability (Spencer et al. 2006a). The present study, to the authors' knowledge, is the first to investigate changes in muscle metabolites when two different intensities of active recovery are performed during a test of repeated short-sprint ability. There were no significant or substantial differences between the MI and LI active recovery trials for the changes in [ATP], [PCr] and [MLa^-]. These data may partly explain the minimal differences in performance measures between the two active recovery trials.

In our previous study, we suggested that the lower PCr content, in the active, compared to passive recovery, may have contributed to the reduced performance in peak power output indices (Spencer et al. 2006a). A strong correlation between percent of resting PCr content and sprint performance has previously been reported (Bogdanis et al. 1995). Other studies have also reported lower PCr content following exercise bouts with active, compared to passive recovery (McAinch et al. 2004; Yoshida et al. 1996), even though the exercise was not of maximal intensity. However, in the present study, there was no significant or substantial difference in the delta change (i.e., difference between rest and post-test value) for PCr content between the MI and LI active recovery trials. Thus, while changes in PCr content are less when repeated sprints are interspersed with passive recovery, as compared to active recovery (Spencer et al. 2006a), the intensity of active recovery (low vs moderate) does not affect changes in PCr content. This is an interesting finding and suggests that the amount of mitochondrial ATP available for PCr resynthesis was not affected by the active recovery intensities used in this study.

In the present study, there was no significant or substantial difference in the delta change for $[MLa^-]$ between the MI and LI active recovery trials. Our previous study, using the same repeated-sprint protocol, reported a significantly higher $[MLa^-]$ post-test in the active, compared to the passive recovery trial (Spencer et al. 2006a). We speculated that the higher $[MLa^-]$ may have been associated with the lower $[PCr]$ in the active recovery trial, as accumulation of metabolites in the process of ATP-Cr splitting (i.e., P_i , ADP and AMP) are suggested to be the stimuli for anaerobic glycolysis (Chasiotis et al. 1982; Crowther et al. 2002). The similar changes in $[MLa^-]$ between MI and LI active recovery trials are therefore probably associated with the similar changes in ATP and PCr during trials with low- to moderate-intensity active recovery.

Conclusion

In summary, significant and substantial reductions in peak power indices were evident in both the MI and LI active recovery trials, compared to the passive recovery trial, during exercise that mimics the sprint and recovery durations of an isolated bout of repeated-sprint activity. However, no significant or substantial differences were evident in total work between the three recovery trials. The minimal differences in performance and muscle metabolite measures between the MI and LI trials suggest that any low-to-moderate level of muscle activation will attenuate the resynthesis of PCr and the recovery of power output during repeated short-sprint exercise.

Although performance is potentially hindered when low-to-moderate level active recovery is conducted before and during repeated-sprint efforts, the team-sport athlete is likely to undertake the most appropriate action to ensure optimal positioning (e.g., marking an

opposition player) regardless of the energy cost of this action. Thus, these findings are unlikely to influence the choice of recovery mode during match play. However, these results have potentially important implications regarding the specificity of training to improve repeated-sprint performance. Furthermore, it is obvious that more research is required to understand the factors that control PCr resynthesis and the recovery of team sport specific, repeated-sprint performance.

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Table 1. Mean changes in performance between the passive, MI (moderate intensity) and LI (low intensity) recovery trials and the magnitude of differences between trials.

	Change in performance (%)	Magnitude of differences	
	Effect; $\pm$ 90% CL	ES	Rating
MI - Passive			
Total Work	-3.1; $\pm$ 4.6	0.19	Trivial
Peak Power 1	-1.0; $\pm$ 2.5	0.09	Trivial
Peak Power 2	-3.4; $\pm$ 2.3	0.26	Small
Peak Power 3	-6.0; $\pm$ 3.8	0.55	Small
Peak Power 4	-5.8; $\pm$ 2.6	0.54	Small
Peak Power 5	-3.4; $\pm$ 3.5	0.35	Small
Peak Power 6	-5.7; $\pm$ 4.5	0.63	Moderate
LI - Passive			
Total Work	-1.4; $\pm$ 6.0	0.09	Trivial
Peak Power 1	-0.3; $\pm$ 3.4	0.03	Trivial
Peak Power 2	-2.0; $\pm$ 3.4	0.15	Trivial
Peak Power 3	-1.7; $\pm$ 3.3	0.15	Trivial
Peak Power 4	-3.5; $\pm$ 2.9	0.32	Small
Peak Power 5	-2.8; $\pm$ 3.4	0.28	Small
Peak Power 6	-3.7; $\pm$ 2.3	0.41	Small
LI - MI			
Total Work	-1.7; $\pm$ 5.5	0.10	Trivial
Peak Power 1	0.7; $\pm$ 1.5	0.06	Trivial
Peak Power 2	1.5; $\pm$ 2.3	0.11	Trivial
Peak Power 3	4.5; $\pm$ 3.5	0.39	Small
Peak Power 4	2.4; $\pm$ 2.4	0.19	Trivial
Peak Power 5	0.7; $\pm$ 3.1	0.07	Trivial
Peak Power 6	2.1; $\pm$ 4.1	0.23	Small

CL = confidence limits; ES = effect size: <0.2, trivial; 0.2-0.6, small; 0.6-1.2, moderate; 1.2-2.0, large; >2.0, very large.

Table 2. Muscle metabolites in the vastus lateralis at rest, post-test and delta change (Δ) during the low-intensity and moderate-intensity recovery trials.

	Low-Intensity Recovery			Moderate-Intensity Recovery		
	Rest	Post-test	Δ	Rest	Post-test	Δ
[ATP]	21.3 $\pm$ 2.4	18.0 $\pm$ 2.7	3.4 $\pm$ 2.3	21.8 $\pm$ 2.4	17.6 $\pm$ 4.0	3.9 $\pm$ 3.5
[PCr]	75.8 $\pm$ 9.2	38.0 $\pm$ 12.3	37.8 $\pm$ 15.6	76.7 $\pm$ 7.1	36.8 $\pm$ 17.7	39.9 $\pm$ 17.6
[Lactate]	4.7 $\pm$ 3.1	50.9 $\pm$ 20.1	46.2 $\pm$ 19.8	4.9 $\pm$ 3.3	46.8 $\pm$ 26.1	41.9 $\pm$ 24.5

Values are means $\pm$ SD, expressed in mmol $\cdot$ kg dm⁻¹ (n = 9). ATP, adenosine triphosphate; PCr, phosphocreatine.

Figure Legends

Figure 1. Relative peak power output ($\text{W} \cdot \text{kg}^{-1}$) during the 6 x 4-s repeated sprint test with either low-intensity (LI), moderate-intensity (MI) or passive recovery. Values are means $\pm$ SD ($n = 9$). Significantly different ($P < 0.05$): * Passive vs MI; [†] Passive vs LI.

Figure 2. Work decrement (A) and power decrement (B) during the 6 x 4-s repeated sprint test, with moderate-intensity (MI), low-intensity (LI) or passive recovery. Values are mean $\pm$ SD ($n = 9$).

Figure 1.

Figure 2.