

Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man

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1. The recovery of power output and muscle metabolites was examined following maximal sprint cycling exercise. Fourteen male subjects performed two 30 s cycle ergometer sprints separated by 1.5, 3 and 6 min of recovery, on three separate occasions. On a fourth occasion eight of the subjects performed only one 30 s sprint and muscle biopsies were obtained during recovery.
2. At the end of the 30 s sprint phosphocreatine (PCr) and ATP contents were 19.7 ± 1.2 and $70.5 \pm 6.5\%$ of the resting values (rest), respectively, while muscle lactate was 119.0 ± 4.6 mmol (kg dry wt)⁻¹ and muscle pH was 6.72 ± 0.06 . During recovery, PCr increased rapidly to $65.0 \pm 2.8\%$ of rest after 1.5 min, but reached only $85.5 \pm 3.5\%$ of rest after 6 min of recovery. At the same time ATP and muscle pH remained low (19.5 ± 0.9 mmol (kg dry wt)⁻¹ and 6.79 ± 0.02 , respectively). Modelling of the individual PCr resynthesis using a power function curve gave an average half-time for PCr resynthesis of 56.6 ± 7.3 s.
3. Recovery of peak power output (PPO), peak pedal speed (maxSp) and mean power during the initial 6 s (MPO₆) of sprint 2 did not reach the control values after 6 min of rest, and occurred in parallel with the resynthesis of PCr, despite the low muscle pH. High correlations ($r = 0.71$ – 0.86 ; $P < 0.05$) were found between the percentage resynthesis of PCr and the percentage restoration of PPO, maxSp and MPO₆ after 1.5 and 3 min of recovery. No relationship was observed between muscle pH recovery and power output restoration during sprint 2 ($P > 0.05$).
4. These data suggest that PCr resynthesis after 30 s of maximal sprint exercise is slower than previously observed after dynamic exercise of longer duration, and PCr resynthesis is important for the recovery of power during repeated bouts of sprint exercise.

The decline in force during maximal, short-term contractions has been associated with several metabolic changes in the exercising muscle such as a decrease in muscle phosphocreatine (Sjoholm, Sahlin, Edstrom & Hultman, 1983; Katz, Sahlin & Henriksson, 1986), a corresponding increase in inorganic phosphate (P_i) and its diprotonated form, H₂PO₄⁻ (Baker, Carson, Green, Miller & Weiner, 1992) and a marked fall in muscle pH (Cady, Elshove, Jones & Moll, 1989). Although the accumulation of H⁺, P_i and H₂PO₄⁻ in the muscle cell may directly impair the activation of the contractile mechanism (Hermansen, 1981), other experiments have suggested that the decline in force may be related to the inability to regenerate ATP at the required rates (Sahlin & Ren, 1989; Soderlund, Greenhaff & Hultman, 1992).

Maximum rates of ATP regeneration are required during sprint exercise. The average rate of ATP regeneration from anaerobic sources during a 6 s sprint on a cycle ergometer is as high as 14.9 mmol (kg dry muscle)⁻¹ s⁻¹ (Gaitanos, Williams, Boobis & Brooks, 1993), and a mean value for a 30 s sprint is ~ 7.5 mmol (kg dry muscle)⁻¹ s⁻¹ (Bogdanis, Nevill, Lakomy & Boobis, 1994). These high anaerobic ATP regeneration rates result in a 60–80% fall in phosphocreatine (PCr), a $\sim 30\%$ fall in ATP and a severalfold increase of glycolytic intermediates and lactate, as glycolysis supplies 65–70% of the anaerobic energy during a 30 s sprint (Cheetham, Boobis, Brooks & Williams, 1986; McCartney, Spriet, Heigenhauser, Kowalchuk, Sutton & Jones, 1986; Nevill, Boobis, Brooks & Williams, 1989).

Since PCr can regenerate ATP at very high rates, and its concentration in the muscle is limited, fatigue during short-term high intensity exercise may be related to PCr availability. However, the simultaneous drop in PCr and pH in this type of exercise, and the fact that PCr and H⁺ are linked via the creatine kinase reaction (Sahlin, Harris & Hultman, 1975), make it difficult to separate their individual contributions to fatigue.

A very useful model for assessing the importance of the different muscle metabolite changes during high intensity exercise is the study of the relationship between force or power output and metabolic state of the muscle during recovery from fatigue. During the initial recovery period the relationship between PCr and [H⁺] disappears, allowing the study of their separate effects on power generation. A study by Harris, Edwards, Hultman, Nordesjo, Nylinde & Sahlin (1976) has shown that PCr resynthesis following exhaustive cycling (~9 min) or isometric exercise is a rapid process, with a half-time of ~22 s. On the other hand, several studies using similar exercise modes have found that the half-time for muscle lactate disappearance or pH restoration is in the region of 3–9.5 min (Sahlin *et al.* 1975; Sahlin, Harris, Nylinde & Hultman, 1976). Interestingly, the pattern of peak force restoration after fatiguing isometric exercise is rapid and resembles that of PCr resynthesis (Sahlin & Ren, 1989; Baker *et al.* 1992). However, only a limited number of studies have followed power output recovery, which reflects variations in both force and velocity of contraction in the fatigued condition. The exercise intensities used in order to induce fatigue in these studies examining power output were either submaximal or ~120% maximum O₂ uptake ($\dot{V}_{O_{2,max}}$), and muscle metabolites and muscle pH were not measured (Sargeant & Dolan, 1987; Hitchcock, 1989).

The purpose of the present study was to follow PCr resynthesis, and the recovery of muscle pH, lactate and other muscle metabolites after maximal sprint exercise lasting 30 s. The relationship between PCr resynthesis, muscle acidosis and muscle function during the recovery period was also examined by parallel power output measurements, which may provide more information about muscle function than isometric force restoration alone.

METHODS

Subjects

Fourteen male university students volunteered to participate in this study. Their mean (\pm s.d.) age, height and body mass were 23 ± 2 years, 178 ± 7 cm and 75.3 ± 8 kg, respectively. All subjects were involved in regular training (athletics or games players; 5–6 times per week). Eight of the subjects participated in the second part of the study, which involved muscle biopsies. Subjects were informed in writing about the purpose of the study, any known risks, and the right to

terminate participation at will. Each expressed understanding by signing a statement of informed consent. The protocol was approved by the Ethical Committee of Loughborough University of Technology.

Equipment

A modified friction-loaded cycle ergometer (Monark, model 864, Varberg, Sweden), interfaced with a microcomputer, was used to attain high frequency logging of the flywheel angular velocity. The instantaneous power generated during the sprints was corrected for the changes in kinetic energy of the flywheel (Lakomy, 1986), and results were averaged over 1 s intervals. By taking into account the work done in accelerating the flywheel during the initial seconds of the sprint, peak power was always reached before peak speed. A restraining harness, passed around the subject's waist, was used during the cycle ergometer sprints in order to limit the exercise to the lower limbs. The two side straps of the belt were fixed to a metal rail, bolted on the floor behind the bicycle frame.

Experimental procedures and protocol

Prior to any experimental testing each subject completed at least two practice sessions. Subjects were requested to repeat their pre-recorded normal diet and refrain from any form of intense physical exercise for 48 h prior to each test. Each subject performed all tests at the same time of day, which was at least 4 h after any meal. The study consisted of two parts: performance of repeated 30 s sprints and the muscle biopsy condition.

Repeated sprints performance. All subjects ($n = 14$) were required to perform two 30 s maximal cycle ergometer sprints against a resistance of $75 \text{ g (kg body mass)}^{-1}$ (average resistive load: $5.6 \pm 0.2 \text{ kg}$) from a rolling start of approximately seventy pedal revolutions per minute (r.p.m.), on three occasions. On each occasion the two sprints were separated by either 1.5, 3 or 6 min of passive recovery on the bicycle seat (Fig. 1). Experimental tests were carried out in a random order, 1 week apart. A standardized warm up consisting of 4 min pedalling at 60 W followed by $2 \times 30 \text{ s}$ at 80 and 100 W separated by 30 s of rest, preceded each test. Strong verbal encouragement was given during each sprint. Peak power output (PPO; attained 2 s into the sprint), pedal speed at which peak power was attained (SpPPO), maximum pedal speed (maxSp), mean power output for the first 6 s and the whole sprint (MPO₆ and MPO₃₀), and the percentage decline from peak to end power output (fatigue index: $FI = \{[(PPO - \text{end power output})/PPO] \times 100\}$) were calculated for each 30 s sprint.

Muscle biopsy condition. After completion of the first part of the study, eight of the subjects agreed to have muscle biopsies taken. Subjects reported to the laboratory 4 h after a light meal and rested on a couch for 30 min. A cannula was placed in an antecubital vein, and small incisions through the skin and fascia over the vastus lateralis muscle of both legs were made under local anaesthesia (1% lidocaine). Then subjects performed the standardized warm up, and one maximal 30 s cycle ergometer sprint. Needle biopsy samples were obtained from the vastus lateralis muscle before the 30 s sprint, immediately after, and again after 1.5, 3 and 6 min, while the subject was lying on a couch. In half of the subjects the resting muscle biopsy was taken from the left leg, and subsequent biopsies were taken from alternate legs. All biopsies were obtained through different incisions in the skin. The time delay

between cessation of the sprint and freezing of the sample in liquid nitrogen was 7.5 ± 1.6 s. Muscle samples were then removed from the needle under liquid nitrogen, and stored in plastic screw-top tubes in liquid nitrogen, until freeze dried (within 24 h).

Analytical methods

The freeze-dried samples were dissected free of connective tissue and blood, and homogenized. The muscle powder was extracted with 0.5 M HClO₄ and the extract was neutralized with 2.1 M KHCO₃ (Harris, Hultman & Nordesjo, 1974). Phosphocreatine (PCr), creatine (Cr), ATP, ADP, free glucose, glucose-1-phosphate (G-1-P), glucose-6-phosphate (G-6-P), fructose-6-phosphate (F-6-P), pyruvate (Pyr), and lactate (Lac) were assayed enzymatically by fluorometric analysis (Lowry & Passoneau, 1972). Glycogen was determined both on the neutralized extract (acid-soluble glycogen fraction), and on the muscle pellet left after the extraction procedure by prior HCl hydrolysis (acid-insoluble glycogen fraction). Muscle metabolite contents were corrected to the individual mean total creatine content (PCr + Cr; range of correction 0–11%). Since the sum PCr + Cr should not change during exercise, this acts as an internal reference in order to account for errors in muscle metabolite concentrations arising from the variable inclusion in the muscle samples of any remaining connective tissue, fat or blood (Harris *et al.* 1974). All muscle metabolite concentrations are expressed per kilogram of dry muscle.

Muscle pH was determined after homogenization of the freeze-dried muscle powder at 4 °C with a solution containing (in mmol l⁻¹): 145 KCl, 10 NaCl and 5 iodoacetic acid (Sahlin *et al.* 1976). The dilution ratio used was 100 μl of homogenizing

solution per milligram of dry muscle, which is the maximum dilution for reliable pH determinations (Marlin & Harris, 1991). Homogenates were equilibrated to 37 °C for 5 min and the pH was measured using a MI-410 microelectrode (Microelectrode, Inc.) connected to a Radiometer acid–base analyser (Radiometer PHM73).

Calculations

The time course of PCr resynthesis during the recovery time (*t*), PCr(*t*), was modelled for each subject separately, as described in the Appendix. Briefly, an exponential model with a power function exponent in time was fitted to the data of each subject. The general form of the model is:

$$PCr(t) = R - R \exp(-at^b), \tag{1}$$

where *R* denotes PCr at rest, and the parameters *a* and *b* (location and shape parameters) characterize each subject's PCr resynthesis curve. For the present data a common group resynthesis model was shown to be inappropriate.

The anaerobic ATP utilization was calculated from the values of ATP, ADP, PCr, lactate and pyruvate before and immediately after the sprint, using the formula (Katz *et al.* 1986):

$$ATP \text{ utilization} = 2(-\Delta ATP) - \Delta ADP - \Delta PCr + 1.5 \Delta Lac + 1.5 \Delta Pyr.$$

Two active phosphates are cleaved per ATP utilized; 1.5 mmol ATP is produced for every millimole of lactate and pyruvate.

The mean anaerobic ATP utilization rate (mmol (kg dry muscle)⁻¹ s⁻¹) was obtained by dividing the anaerobic ATP utilization by the duration of the sprint (30 s).

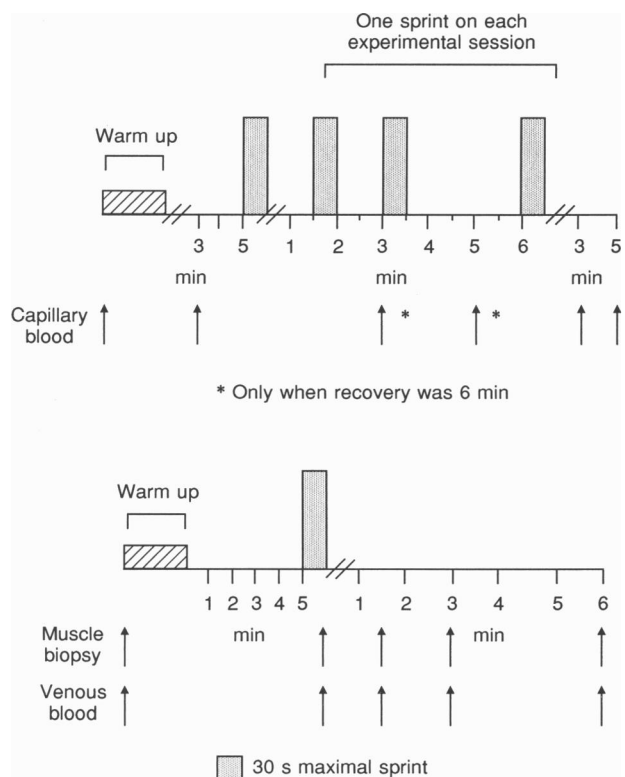


Figure 1. Experimental design of the study

Part 1 (top), repeated sprints performance (*n* = 14) and part 2 (bottom), biopsy occasion (*n* = 8).

Glycogenolytic and glycolytic rates during the 30 s sprint (mmol glucosyl units (kg dry muscle)⁻¹ s⁻¹) were calculated from accumulation of glycolytic metabolites as previously reported (Spriet, Soderlund, Bergstrom & Hultman, 1987):

$$\text{Glycogenolysis} = (\Delta\text{G-1-P} + \Delta\text{G-6-P} + \Delta\text{F-6-P}) \\ + 0.5 (\Delta\text{Lac} + \Delta\text{Pyr}),$$

$$\text{Glycolysis} = 0.5 (\Delta\text{Lac} + \Delta\text{Pyr}).$$

Since the exercise was dynamic and the circulation was not restricted by any mechanical means (e.g. tourniquet), some lactate diffused into the circulation during the 30 s of the sprint. However, due to the short duration of the exercise bout the underestimation in the above calculations is thought to be minimal.

The concentration of inorganic phosphate ([P_i]) in the muscle after the sprint and during recovery, was calculated from changes in ATP, ADP, PCr and hexose monophosphates (G-1-P, G-6-P, F-6-P), as in Bergstrom & Hultman (1988):

$$P_i = 2.9 + \{2(-\Delta\text{ATP}) - \Delta\text{ADP} - \Delta\text{PCr} \\ - (\Delta\text{G-1-P} + \Delta\text{G-6-P} + \Delta\text{F-6-P})\}/3,$$

where the resting value from Chasiotis (1983) was 2.9; 3 l of intracellular water per kilogram of dry muscle. P_i concentration is expressed in mmol (l muscle water)⁻¹.

Blood sampling and analysis

During the repeated sprints performance (part 1 of study), duplicate samples of arterialized capillary blood (20 μl each) were taken from a pre-warmed thumb for lactate determination at rest, after the standardized warm up and at the third and fifth minute after the second sprint in each condition (see Fig. 1). Capillary samples were also taken at the third and fifth minute after the first sprint when the recovery between sprints was 6 min. Venous blood samples were obtained during the biopsy condition (part 2 of the study), at rest, and at the same time as the muscle biopsies (i.e. immediately after, 1.5, 3 and 6 min after the 30 s sprint; Fig. 1). Venous samples were placed in tubes containing lithium-heparin, and blood pH was measured immediately (Radiometer PHM73 pH/blood gas monitor, Copenhagen, Denmark). Changes in plasma volume were estimated from pre- and post-sprint haematocrit and haemoglobin values (Dill & Costill, 1974). Duplicate (20 μl) samples from the venous blood were also taken for lactate determination. These samples and the capillary samples collected during part 1 of the study were immediately deproteinized in 2.5% perchloric acid, stored at -20 °C and analysed at a later date using a fluorometric method described by Maughan (1982). The remaining blood (~1.5 ml) was placed in a Ca²⁺ heparinized tube, centrifuged at 12 000 r.p.m. for 3 min and then stored at -70 °C. Plasma ammonia was determined enzymatically within 24 h of sampling (Boehringer Mannheim GmbH enzymatic UV method).

Statistical analysis

One-way or two-way analyses of variance (ANOVA) for repeated measures on both factors were used where appropriate for statistical analysis. Where significant *F* ratios were found (*P* < 0.05), the means were compared using Tukey's post-hoc test. Relationships between variables were

examined by calculating the product moment correlation coefficient (*r*). Results are presented as means ± s.e.m.

RESULTS

Power output during the first sprint

As this study had two parts, results will be reported for *n* = 14 (repeated sprints performance) or *n* = 8 (biopsy condition).

There were no significant differences in any of the power output indices of the first sprints between the three experimental conditions (1.5, 3 and 6 min recovery intervals, *n* = 14). Consequently, the mean values of the three first sprints will be presented. Values for subjects who were subsequently biopsied (*n* = 8) are in parentheses.

The peak power output during the first sprint was 1264 ± 55 W (1310 ± 65 W), and was attained at a pedal speed of 148 ± 3 r.p.m. (151 ± 4 r.p.m.). The highest pedal speed (maxSp) was attained 4 s after the start of the sprint, and averaged 160 ± 4 r.p.m. (165 ± 5 r.p.m.), while the mean power output over the first 6 and 30 s of the sprint was 931 ± 33 W (954 ± 39 W) and 647 ± 18 W (660 ± 24 W), respectively. The fatigue index (FI) was 65 ± 2% (66 ± 2%).

High correlations were found in all three conditions between PPO and SpPPO during the first sprint (*r* = 0.84–0.90, *P* < 0.01, *n* = 14), indicating that the more powerful subjects generated their PPO at higher pedal speeds. The subjects with the higher PPO during the first sprint also had the higher fatigue index in all conditions (*r* = 0.84–0.88, *P* < 0.01, *n* = 14).

The power output indices of the 30 s sprint performed during the biopsy condition (*n* = 8) were not significantly different from the corresponding values during the first sprints of the repeated sprints sessions.

Peak power output was 1360 ± 58 W and was generated at 152 ± 3 r.p.m. The mean power outputs for 6 and 30 s were 988 ± 37 and 664 ± 24 W, respectively, and the FI was 68 ± 1%. A maximum pedal speed of 170 ± 3 r.p.m. was again attained on the fourth second of the sprint. Since the power output indices during sprint 1 were the same during all four occasions, and there was no order effect (ANOVA), the muscle metabolites measured during the biopsy condition were considered to be representative of all occasions.

Power output recovery

Figure 2 shows the PPO, maxSp, MPO₆ and MPO₃₀ attained during the second sprint, after 1.5, 3 and 6 min of recovery. Values are expressed as a percentage of the values attained during the corresponding sprint 1 (*n* = 14). For maxSp and PPO, the zero time point represents the

corresponding value at the last second of sprint 1. There was no difference between the recovery curves for $n = 8$ and $n = 14$ (n.s.).

During the first 3 min of recovery, PPO and maxSp showed a rapid rate of restoration reaching 88.7 and 93.5% of sprint 1 values. However, no further increase in PPO and maxSp was observed during the remaining 3 min of recovery (Fig. 2). The recovery pattern for MPO_6 was similar to that of PPO and maxSp, but with a significant 3% increase between 3 and 6 min ($P < 0.01$). In contrast, MPO_{30} restoration followed a more linear pattern lacking the initial fast recovery. None of the sprint performance variables reached the control (sprint 1) values by the sixth minute of recovery.

Strong negative correlations ($r = -0.74$ to -0.93 ; $P < 0.01$, $n = 14$) were found between PPO generated during the first sprint and %PPO, %SpPPO, %maxSp, % MPO_6 and % MPO_{30} attained at the second sprint after 1.5 and 3 min of recovery. These correlations were lower ($r = -0.61$ to -0.77 ; $P < 0.05$ to $P < 0.01$, $n = 14$) during the 6 min recovery occasion. Similar relationships were

obtained when the FI of the first sprint was correlated with all the above recovery variables, implying that the more powerful subjects have the lower rate of power output recovery.

Muscle metabolites

The muscle metabolite concentrations at rest, immediately after the 30 s sprint, and 1.5, 3 and 6 min into recovery are shown in Table 1.

Muscle glycogen decreased by 35% (~ 110 mmol glucosyl units (kg dry muscle) $^{-1}$) and remained at that level during the rest of the recovery time. Interestingly, 85% of the glycogen utilized during the 30 s sprint could be accounted for by the accumulation of the measured glycolytic intermediates, pyruvate and lactate. The PCr content of the muscle ~ 7.5 s after the sprint was $19.7 \pm 1.0\%$ of the resting value, but PCr was rapidly resynthesized with a mean half-time of 56.6 ± 7 s (Fig. 3). However, resynthesis of PCr was not complete after 6 min of recovery ($85.5 \pm 3.5\%$ of the resting value). Predictions from the model gave an average time of 13.6 min (range 3.9–25 min)

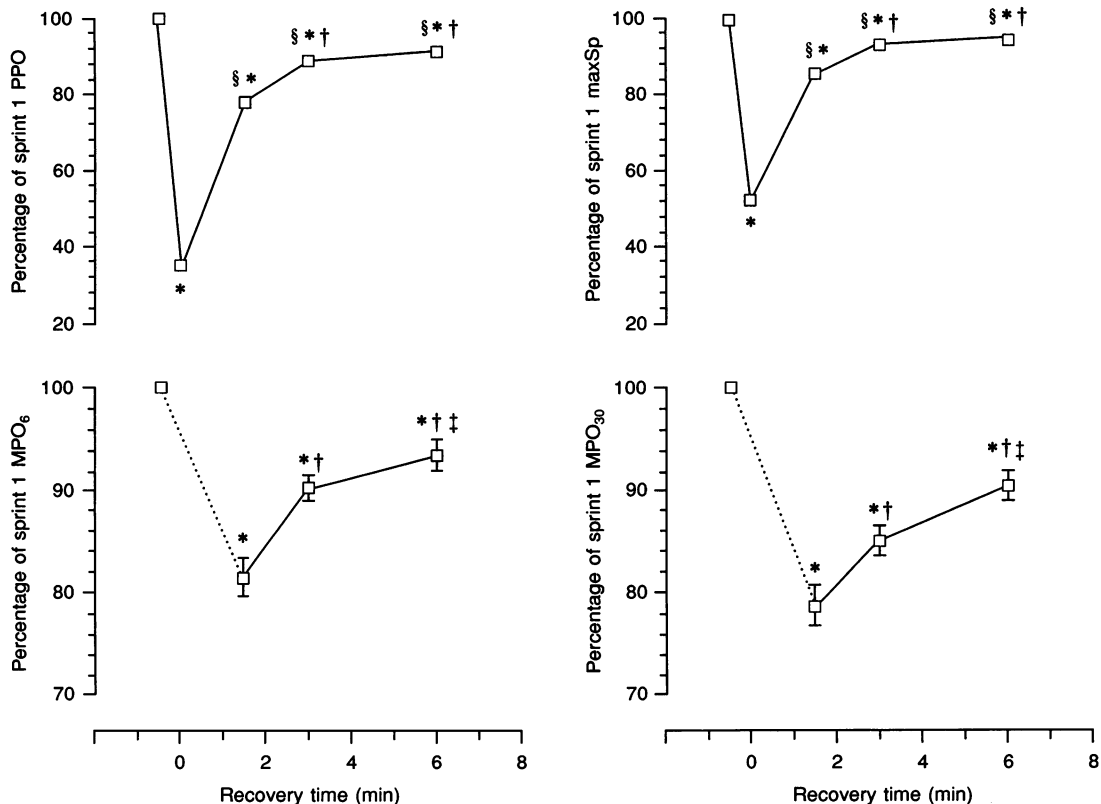


Figure 2. Restoration of power output and peak pedal speed (maxSp) during recovery from maximal 30 s cycle ergometer sprint

PPO, peak power output; MPO_6 and MPO_{30} , mean power output during the first 6 and 30 s of the sprint. For PPO and maxSp the zero time point represents the corresponding value at the last second of sprint 1 (End S1). Values means \pm s.e.m., $n = 14$. * $P < 0.01$ from sprint 1; § $P < 0.01$ from End S1. † $P < 0.01$ from 1.5 min. ‡ $P < 0.01$ from 3 min.

Table 1. Muscle metabolites in vastus lateralis at rest, immediately after (Post) and 1.5, 3 and 6 min after a 30 s cycle ergometer sprint

| | Rest | Post | 1.5 min | 3 min | 6 min |
|------------------|--------------|---------------------------|---------------------------|---------------------------|----------------------------|
| Glycogen (total) | 321.5 ± 18.2 | 211.6 ± 18.5 ^a | 223.2 ± 19.5 ^a | 217.2 ± 21 ^a | 221.0 ± 18.3 ^a |
| Acid insoluble | 258.7 ± 12.8 | 176.5 ± 12.2 ^a | 183.2 ± 13.8 ^a | 184.4 ± 15.2 ^a | 187.5 ± 12.9 ^a |
| Acid soluble | 62.8 ± 8.4 | 35.1 ± 8.3 ^a | 40.0 ± 7.3 ^e | 32.8 ± 6.8 ^a | 33.5 ± 6.6 ^a |
| PCr | 77.1 ± 2.4 | 15.1 ± 1.0 ^a | 49.7 ± 1.1 ^{ab} | 57.2 ± 2.0 ^{abc} | 65.5 ± 2.2 ^{abcd} |
| Cr | 30.7 ± 1.8 | 91.2 ± 2.4 ^a | 56.5 ± 2.7 ^{ab} | 48.6 ± 2.9 ^{abc} | 41.8 ± 2.8 ^{abcd} |
| Total creatine | 107.8 ± 3.2 | 106.3 ± 2.5 | 106.2 ± 2.9 | 105.8 ± 2.1 | 107.3 ± 2.7 |
| P _i | 2.9 | 18.5 ± 1.4 ^a | 7.7 ± 1.1 ^{ab} | 7.4 ± 1.3 ^{ab} | 6.4 ± 0.7 ^{ab} |
| ATP | 25.6 ± 0.4 | 18.1 ± 1.7 ^a | 19.1 ± 0.9 ^a | 18.8 ± 1.1 ^a | 19.5 ± 0.9 ^a |
| ADP | 2.2 ± 0.2 | 2.6 ± 0.4 | 2.2 ± 0.3 | 2.1 ± 0.3 | 2.3 ± 0.3 |
| Glucose | 1.7 ± 0.2 | 5.3 ± 0.4 ^a | 6.6 ± 0.4 ^a | 7.4 ± 0.5 ^{ab} | 7.8 ± 0.6 ^{ab} |
| G-1-P | 0.12 ± 0.01 | 2.12 ± 0.3 ^a | 1.35 ± 0.3 ^{af} | 1.07 ± 0.2 ^{ab} | 0.84 ± 0.2 ^{ab} |
| G-6-P | 1.21 ± 0.2 | 22.8 ± 1.2 ^a | 20.9 ± 0.6 ^a | 16.6 ± 0.8 ^{abc} | 11.0 ± 1.2 ^{abcd} |
| F-6-P | 0.13 ± 0.02 | 6.4 ± 0.3 ^a | 5.2 ± 0.2 ^{ab} | 4.0 ± 0.3 ^{abc} | 2.6 ± 0.3 ^{abcd} |
| Pyruvate | 0.95 ± 0.1 | 4.6 ± 0.4 ^a | 1.9 ± 0.2 ^{eb} | 1.6 ± 0.1 ^b | 1.5 ± 0.1 ^b |
| Lactate | 3.8 ± 0.3 | 119.0 ± 4.6 ^a | 107.3 ± 3.8 ^a | 95.4 ± 5.6 ^{ab} | 81.9 ± 6.0 ^{abcg} |

Values are means ± s.e.m. for 8 subjects, expressed in mmol (kg dry muscle)⁻¹. Muscle glycogen expressed in mmol glucosyl units (kg dry muscle)⁻¹; acid insoluble and acid soluble, muscle glycogen fractions; PCr, phosphocreatine; P_i, calculated inorganic phosphate (mmol (l muscle water)⁻¹); G-1-P, glucose-1-phosphate; G-6-P, glucose-6-phosphate; F-6-P, fructose-6-phosphate. Significant differences: a, *P* < 0.01 from Rest; b, *P* < 0.01 from Post; c, *P* < 0.01 from 1.5 min; d, *P* < 0.01 from 3 min; e, *P* < 0.05 from Rest; f, *P* < 0.05 from Post; g, *P* < 0.05 from 3 min.

for PCr resynthesis to 95% of the resting value. The large range of recovery times stresses the importance of considering the differences between individual PCr resynthesis curves (Fig. 4).

PPO, maxSp and MPO₆ restoration occurred in parallel with the resynthesis of PCr. In addition, there were high correlations between the individual percentage PCr resynthesis (relative to the resting value) and the percentage PPO, maxSp and MPO₆ attained during the second sprint, both after 1.5 and 3 min of recovery (*r* = 0.71–0.86; *P* < 0.05). An example from the correlations found is shown in Fig. 5. These correlations became lower and not significant for the 6 min recovery interval (percentage PCr – PPO; *r* = 0.66, n.s.).

Muscle lactate increased to 119.0 ± 4.6 mmol (kg dry muscle)⁻¹ immediately after the sprint, and subsequently decreased to ~90, 80 and 70% of the peak value after 1.5, 3 and 6 min of recovery (Fig. 3). The concomitant muscle acidosis, as quantified by muscle pH, is shown in Fig. 3. Muscle pH remained at the immediate post-sprint levels for the first 3 min of recovery and then increased slightly to 6.79 ± 0.02 after 6 min. A high correlation was found between the muscle pH and Lac + Pyr contents after the

sprint (*r* = -0.94; *P* < 0.01). Since muscle pH remained depressed and [Lac] was high during recovery, they were unrelated with the percentage sprint performance restoration (*P* > 0.05)

Muscle ATP was decreased by ~30% after the sprint, and was not restored during recovery (Table 1). Muscle glucose was elevated at the end of the sprint and continued to increase until the end of the observation period. The concentrations of G-1-P, G-6-P and F-6-P increased 18-, 19- and 49-fold, respectively, and were still elevated after 6 min of recovery. The subjects with the higher MPO (kg body mass)⁻¹ had the highest post-sprint [G-6-P] (*r* = 0.84; *P* < 0.01).

Calculated P_i concentration (Table 1) increased considerably after the sprint, and was still twice the theoretical resting value of 2.9 mmol (l muscle water)⁻¹ at the end of the recovery period.

ATP utilization rate and glycogenolytic and glycolytic rates

The mean ATP utilization rate was 8.5 ± 0.9 mmol (kg dry muscle)⁻¹ s⁻¹ (total ATP utilization was 255 ± 10 mmol (kg dry muscle)⁻¹), of which 69.9 ± 1% was supplied from

Figure 3. Time course of phosphocreatine (PCr) resynthesis and muscle lactate disappearance (top) and muscle pH changes (bottom) during recovery from a 30 s maximal cycle ergometer sprint

Values for PCr are expressed as a percentage of the resting concentration and for lactate as a percentage of the peak content (mean \pm s.e.m., $n = 8$). The curve fitted on the PCr data represents the mean of the curves fitted on the individual data for each subject.
 * $P < 0.01$ from Rest. † $P < 0.01$ from 1.5 min. Due to lack of biopsy material, pH was determined for $n = 6$ for the Post and 6 min sampling points, and $n = 7$ for the 1.5 min point. The resting and 3 min pH determinations were for $n = 8$.

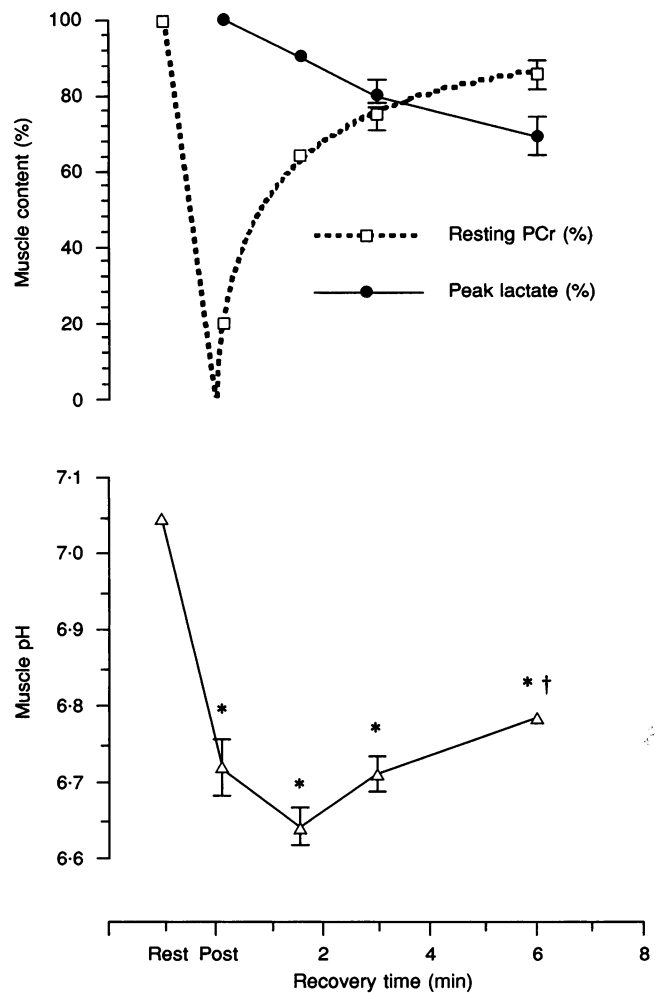
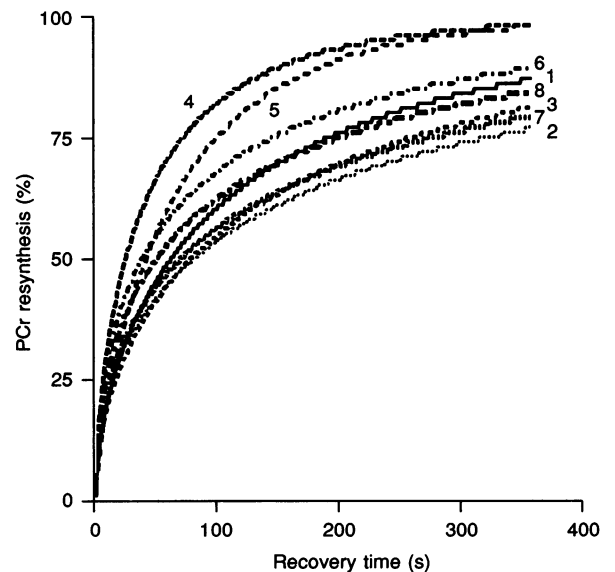


Figure 4. Individual PCr resynthesis curves fitted using the model described in Methods

Values for PCr are expressed as a percentage of the resting concentration. Numbers 1–8 represent individual subjects.



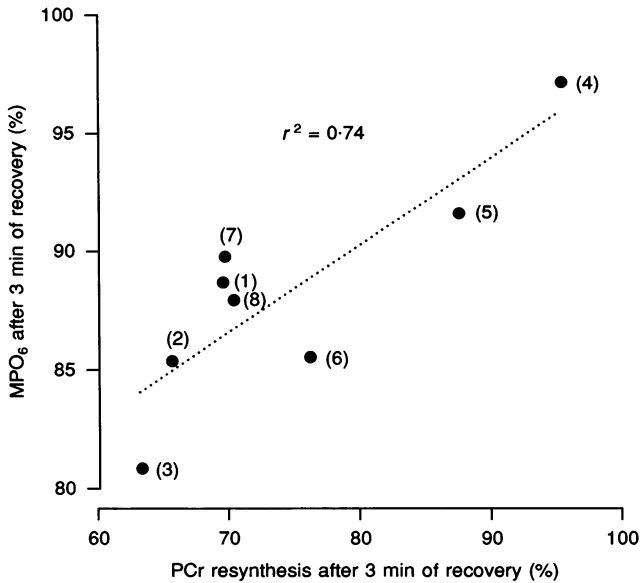


Figure 5. Relationship between percentage PCr resynthesis after 3 min of recovery and the corresponding percentage restoration of MPO₆

Numbers 1–8 represent individual subjects. The coefficient of determination (r^2) for the correlation is also shown.

glycolysis, and $24.4 \pm 1\%$ from PCr. The ATP utilization rate was closely related to the glycogenolytic rate ($r = 0.87$; $P < 0.01$). The glycogenolytic and glycolytic rates during the 30 s sprint were 3.0 ± 0.1 and 2.0 ± 0.1 mmol glucosyl units (kg dry muscle)⁻¹ s⁻¹, resulting in a glycogenolysis to glycolysis ratio of 1.5 ± 0.02 . This was a consequence of a large build-up of hexose monophosphates (G-1-P, G-6-P, F-6-P).

Blood lactate and blood pH

The standardized warm-up did not result in a significant increase of blood lactate concentration ([BL]; rest = 0.7 ± 0.1 vs. post-warm up = 1.1 ± 0.1 mmol l⁻¹; n.s.). The peak lactate values, observed 5 min after the second 30 s sprint (repeated sprints performance), were similar in the 1.5 and 3 min recovery conditions (16.9 ± 0.5 and 16.6 ± 0.4 mmol l⁻¹), but a higher [BL] was seen in the 6 min recovery condition (17.4 ± 0.4 mmol l⁻¹; $P < 0.01$).

The blood lactate responses (venous blood) to one 30 s cycle ergometer sprint were examined during the biopsy condition. The highest [BL] was observed 6 min after completion of the sprint, and was 13.6 ± 0.9 mmol l⁻¹. The blood pH was also measured on this occasion (Fig. 6). Blood pH dropped from a resting value of 7.38 ± 0.01 to 7.08 ± 0.03 , and remained depressed until the end of the observation period. The subjects with the higher PPO and MPO₃₀ also had the higher [BL] on the sixth minute of recovery ($r = 0.93$; $P < 0.01$). Blood lactate and pH were inversely correlated throughout recovery ($r = -0.95$ to -0.96 ; $P < 0.01$). There was no relationship between blood and muscle lactate or blood and muscle pH for corresponding time samples throughout recovery ($r < 0.35$; n.s.).

Plasma ammonia

Resting plasma ammonia concentration was 29.1 ± 4.7 μ mol l⁻¹ and was elevated to 94.0 ± 17.6 μ mol l⁻¹ after

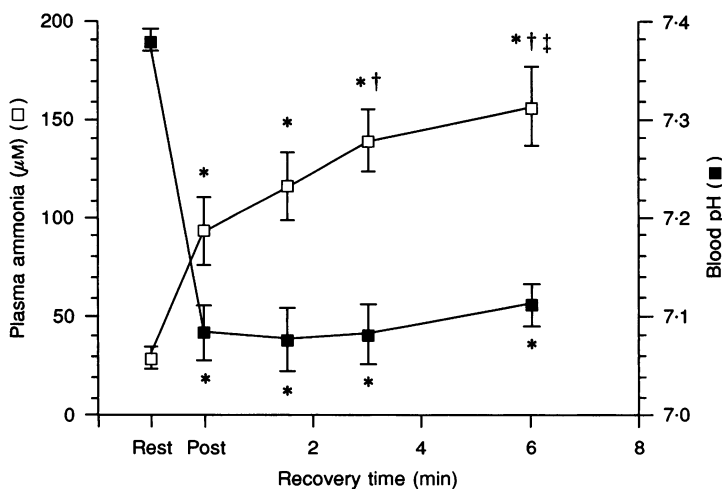


Figure 6. Plasma ammonia and blood pH at rest and during recovery from a 30 s maximal cycle ergometer sprint

Values are means \pm s.e.m., $n = 8$. * $P < 0.01$ from rest. † $P < 0.01$ from Post; ‡ $P < 0.01$ from 1.5 min.

the 30 s sprint. The plasma ammonia concentration increased throughout recovery, reaching $156.9 \pm 20.0 \mu\text{mol l}^{-1}$ after 6 min (Fig. 6). The individuals with the higher PPO and MPO_{30} also had the higher peak plasma ammonia concentration ($r = 0.85$ and 0.86 ; $P < 0.01$). Strong correlations were found between blood lactate and pH, and ammonia concentration throughout recovery ($r = 0.84$ to 0.90 ; $P < 0.01$, ammonia and lactate; and $r = -0.80$ to -0.86 ; $P < 0.05$ to 0.01 , ammonia and pH).

DISCUSSION

This study examined muscle metabolism and performance restoration after a maximal 30 s cycle ergometer sprint. The main findings were that the resynthesis of PCr and the restoration of peak performance (PPO, maxSp, MPO_6) proceeded in parallel, even though muscle pH remained low (~ 6.7) throughout the recovery. In addition, PCr resynthesis was slower than that previously observed after dynamic exercise of longer duration (Harris *et al.* 1976). These observations allow some comments to be made on the relative importance of energy supply and the direct effect of H^+ on the contractile mechanism in the aetiology of fatigue during sprinting.

During the first few seconds of a maximal sprint, energy is made available through the rapid degradation of PCr and anaerobic glycolysis. For example, during a 6 s sprint on a cycle ergometer the mean rate of PCr degradation averaged $7.3 \text{ mmol (kg dry muscle)}^{-1} \text{ s}^{-1}$ and PCr contributed approximately 50% to the total anaerobic ATP production (Gaitanos *et al.* 1993). However, if exercise is continued for 30 s, as in the present study, PCr will be so low after 10 s (Jones *et al.* 1985; Bogdanis *et al.* 1994) that ATP production during the remaining 20 s will be dependent predominantly on the glycolytic rate and aerobic metabolism. Thus if energy supply is more critical to power generation than the direct effect of H^+ on the contractile mechanism, then performance during the initial seconds of the second sprint will reflect PCr resynthesis and any reduction in the glycolytic rate, rather than the recovery in muscle pH. Furthermore, peak and mean (MPO_{30}) power output restoration may be differently affected as a result of the varying contributions of PCr and glycolysis to energy supply in the first few seconds and over the entire 30 s of the sprint.

In the present study the similar patterns of, and the statistically significant correlations between, PCr and peak performance restoration support the idea that PCr availability is critical for power generation during the initial seconds of the sprint. A possibility exists that PCr resynthesis reflects the removal of inorganic phosphate and its acidic fraction (H_2PO_4^-), which may be related to power recovery, rather than PCr availability *per se*. However, the calculated P_i and its acidic fraction H_2PO_4^- (from the

Henderson–Hasselbalch equation, pH and a $\text{p}K$ for P_i of 6.83) did not change between 1.5 and 6 min after the sprint, while power and PCr recovered significantly.

Furthermore, it is possible that changes in PCr may account for changes in power output in early recovery as a result of similar inhibition of glycolysis at 1.5 and 3 min, due to the lack of change in muscle pH. While the interaction of the variables controlling the rate of glycolysis are not fully understood, it has been reported that the glycolytic rate is reduced by 40–60% when muscle pH is decreased to ~ 6.7 (Spriet *et al.* 1987; Spriet, Lindinger, McKelvie, Heigenhauser & Jones, 1989).

Thus the initial slower restoration of mean power output (MPO_{30}) in comparison with peak power output may be explained by the continued inhibition of glycolysis, while PCr is rapidly resynthesized. At this point it must be noted that a significant part of MPO_{30} recovery can be attributed to MPO_6 recovery (and therefore PCr resynthesis), since 30% of the total work done during the 30 s sprint is generated during the initial 6 s. After 6 min of recovery muscle pH increased, and the inhibitory effect on the glycogen phosphorylase and phosphofructokinase activity (Chasiotis, 1983; Spriet *et al.* 1987) was possibly less, allowing more ATP to be regenerated through glycolysis and further recovery of MPO_{30} .

The suggestions that energy supply and particularly PCr availability are critical for short-term power production are supported by recent studies which have shown that oral creatine supplementation increased muscle PCr content (Harris, Soderlund & Hultman, 1992) and enhanced performance during repeated bouts of high-intensity exercise (Greenhaff, Casey, Short, Harris, Soderlund & Hultman, 1993; Balsom, Ekblom, Soderlund, Sjodin & Hultman, 1993).

Although PCr and power output recovered at an initial rapid rate in the present study, 6 min of rest following a maximal 30 s sprint was inadequate for muscle metabolism and power output to recover fully. The incomplete recovery of all power output indices may be related to the low pH which impairs glycolysis, and also to the PCr which after 6 min was only $\sim 85\%$ of the resting value. Support for the notion that the effect of $[\text{H}^+]$ on the ATP-generating process (i.e. glycolysis) is more important than an effect on the contractile mechanism itself is given by a study which followed the recovery of isometric force after a fatiguing static contraction (Sahlin & Ren, 1989). Maximum voluntary force (MVC) was back to the pre-fatigue value 2 min after isometric knee extension to exhaustion (~ 52 s duration) at 66% MVC, despite high muscle lactate. Furthermore, in the present study some subjects exhibited almost full recovery (96–98%) of PPO, maxSp and MPO_6 values after 3 min of rest, which was matched by a high PCr resynthesis, despite a pH of ~ 6.79 .

A possible explanation for the apparent plateau in PPO and maxSp recovery may be given by selective fatigue of fast twitch fibres. During all-out sprint cycling exercise, fast twitch motor units are important for high power output generation (McCartney, Heigenhauser & Jones, 1983). However, due to the higher glycogenolytic and PCr degradation rates in the fast twitch (FT) compared with the slow twitch (ST) fibres (Greenhaff, Casey, Short, Harris, Soderlund & Hultman, 1994), the FT fibres may accumulate more H^+ and be subject to an 'energy crisis' which will result in lower force and therefore power generation (Soderlund *et al.* 1992). Moreover, it has been reported that PCr resynthesis is slower in the FT fibres (Tesch, Thorsson & Fujitsuka, 1989; Soderlund & Hultman, 1991) possibly due to higher $[H^+]$ and a poorer capillary network supplying the FT fibres (Tesch & Wright, 1983). Therefore an impaired ability of FT fibres to regenerate ATP at high rates, due to slower PCr resynthesis in these fibres, may explain the levelling off of PPO and maxSp recovery observed in the present study after the third minute of rest. The finding that the subjects with the higher PPO during the first sprint had the lower power recovery may be related to the above hypotheses, since subjects with high PPO usually have a high percentage of FT fibres (McCartney *et al.* 1983).

Comparison of the results of the present study with the few studies examining power recovery, reveals that the recovery of power output may be related to the duration

and intensity of prior exercise: shorter duration and lower intensity of previous exercise is associated with a faster recovery of muscle function. In the study of Sargeant & Dolan (1987), maximal peak power reached the control values after only 1 min of recovery. The previous exercise was cycling for 6 min at an intensity equivalent to 87% $V_{O_{2,max}}$. Similarly, Hitchcock (1989) reported full recovery of power output 1 min after a 2 min cycling bout at 60 and 80% $V_{O_{2,max}}$. However, power remained at ~87% of the initial value for 4 min after cycling at 120% $V_{O_{2,max}}$ for 1.5–2 min (Hitchcock, 1989). In the present study, the average intensity is about 2.5 times greater than that which would elicit the subjects' $V_{O_{2,max}}$. Furthermore, the high energy demand during sprint exercise results in a mismatch between energy production and utilization, and the magnitude of the metabolic stress is reflected by low muscle pH and a 30% decrease in ATP, which is accompanied by an increase in AMP deamination and a corresponding increase of plasma ammonia concentration (Sahlin & Katz, 1988), as observed in the present study. Therefore the magnitude of the metabolic disturbances may explain the slower power recovery in the present study.

In addition to the slower power recovery, PCr resynthesis was also slower than that previously observed after cycling exercise at 60 r.p.m. to exhaustion lasting ~8.7 min (Harris *et al.* 1976). From the model of PCr resynthesis, it can be calculated that PCr was restored at an initial average rate of 2–4 mmol (kg dry muscle) $^{-1}$ s $^{-1}$. This initial

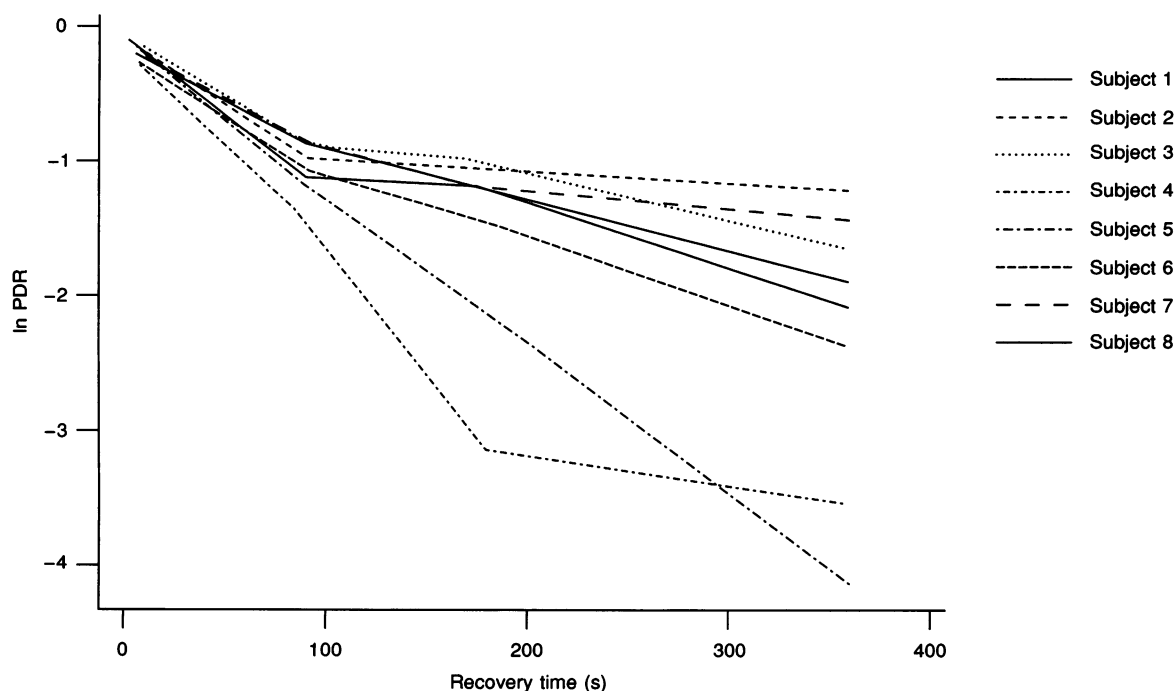


Figure 7

Logarithmic-transformed phosphocreatine depletion ratio ln PDR, by recovery time (s), for all eight subjects

resynthesis rate value was similar to that observed by Harris *et al.* (1976), but the half-time of PCr resynthesis was much longer in the present study (22 vs. 56.6 ± 7.3 s). In earlier studies it has been postulated that the initial fast phase of PCr resynthesis is an oxygen-dependent process, linked with oxidative phosphorylation and the mitochondrial creatine kinase activity, while the subsequent slow phase is limited by the recovery of muscle pH because of the effect of H^+ on the creatine kinase equilibrium (Sahlin *et al.* 1975; Sahlin, Harris & Hultman, 1979). The common regulator for oxygen availability and clearance of H^+ from the muscle is blood flow. The slow resynthesis of PCr and pH recovery in the present study may have been due to the passive recovery between sprints, resulting in pooling of blood in the legs, thereby reducing blood flow, H^+ clearance and O_2 supply (Spriet *et al.* 1989). The importance of blood flow in the recovery of muscle metabolites has been previously demonstrated by occluding the circulation immediately after exercise, which completely prevented PCr resynthesis and pH restoration (Harris *et al.* 1976; Sahlin *et al.* 1979).

The large variation in PCr resynthesis between subjects in the present study (Fig. 4) stresses the importance of following the individualized modelling approach described here, as opposed to a common curve fit for all the subjects. The variability among subjects may be related to differences in muscle fibre composition (Tesch *et al.* 1989; Soderlund & Hultman, 1991) and training status, since an

increased capillary density, which accompanies endurance training, is related to a faster recovery of isokinetic knee extension torque following fatiguing contractions (Tesch & Wright, 1983).

In summary, this study demonstrates that the restoration of peak power output parameters (PPO, maxSp, MPO₆) during recovery from a 30 s bout of maximal sprint exercise occurs in parallel with PCr resynthesis, in spite of the low muscle pH. However, neither PCr nor sprint performance recovery were complete after 6 min of rest. The slow PCr resynthesis after this type of exercise may be related to the high exercise intensity and to a reduced blood flow in the legs during recovery. From the results of this study, it seems that the inability to regenerate ATP at high rates from PCr and glycolysis is related to the decreased power output during recovery from maximal sprint exercise.

APPENDIX

Modelling phosphocreatine resynthesis

The simple exponential model to describe a subject's phosphocreatine PCr(*t*) recovery at time *t* is given by:

$$PCr(t) = R - R \exp(a - bt), \quad (A1)$$

where *R* is the subject's phosphocreatine value at rest, the parameter *a* indicates the subject's proportion of depleted

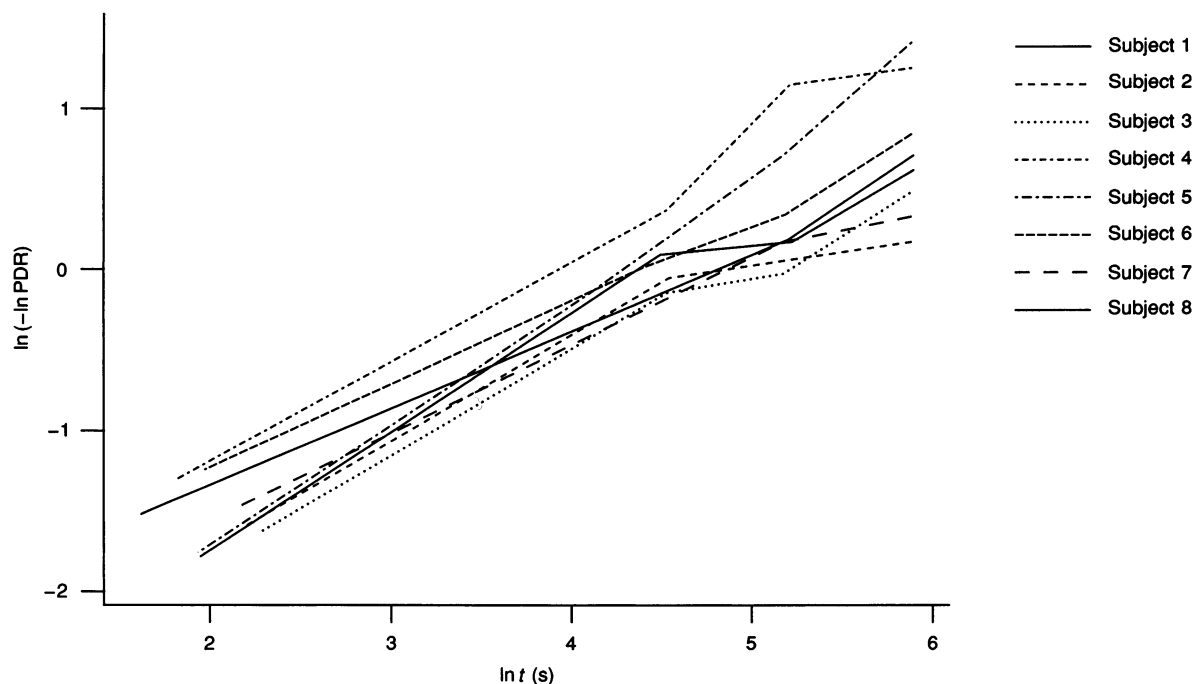


Figure 8

Double logarithmic-transformed phosphocreatine depletion ratio, $\ln(-\ln PDR)$, by logarithmic-transformed recovery time, $\ln t$, for all eight subjects

Table 2. Each subject's estimated parameters, r^2 and half-recovery times, for the phosphocreatine resynthesis ratio model (A6)

| Subject number | a | b | r^2 (%) | Half-recovery time (s) |
|----------------|-------|------|-----------|------------------------|
| 1 | 0.054 | 0.62 | 98 | 62.6 |
| 2 | 0.076 | 0.50 | 94 | 82.0 |
| 3 | 0.055 | 0.58 | 99 | 80.0 |
| 4 | 0.083 | 0.66 | 98 | 25.3 |
| 5 | 0.036 | 0.80 | 100 | 42.0 |
| 6 | 0.102 | 0.52 | 100 | 38.9 |
| 7 | 0.080 | 0.51 | 99 | 71.2 |
| 8 | 0.095 | 0.51 | 99 | 51.0 |

phosphocreatine at time $t = 0$ (proportional to their resting value) and the parameter b describes the subject's 'rate of resynthesis'. By rearranging eqn (A1), a phosphocreatine depletion ratio (PDR) can be defined as:

$$\text{PDR} = (R - \text{PCr}(t))/R = \exp(a - bt). \quad (\text{A2})$$

Assuming that the exponential model (A1) is appropriate to describe phosphocreatine resynthesis, when the natural logarithm of the ratio, $\ln \text{PDR}$, is plotted against time t , the result should be approximately linear. As such, parameters a and b can be estimated using simple linear least-squares regression.

However, supporting the findings of Harris *et al.* (1976), when the log-transformed phosphocreatine depletion ratio, $\ln \text{PDR}$, was plotted against time t for the results reported here, the simple exponential model was found to be unsatisfactory for all but one of the subjects (see Fig. 7).

Rather than indicating a possible linear model, the subjects' plots would appear to better describe a power function curve, $\ln \text{PDR} = -at^b$, where the parameter b would need to be less than unity to accommodate the concave nature of the curves.

The problem with incorporating this power function into either eqns (A1) or (A2), is that the resulting model automatically assumes the subject's phosphocreatine store is entirely depleted at time $t = 0$, i.e. the subject's phosphocreatine depletion ratio is unity. Although this

assumption would appear to be reasonable (Sahlin *et al.* 1979), an alternative model should allow for an additional parameter, a_0 , that can be fitted and subsequently tested for its contribution (and significance) to the prediction of the ratio PDR. Hence, the following model was proposed to represent the phosphocreatine depletion ratio PDR:

$$\text{PDR} = (R - \text{PCr}(t))/R = \exp(a_0 - at^b). \quad (\text{A3})$$

Unfortunately, after taking natural logarithms, the parameters in eqn (A3) can no longer be fitted using the usual linear least-squares regression. However, non-linear least-squares routines are now readily available to fit the parameters in such models. When the model (A3) was fitted separately to all eight subjects using non-linear least-squares, not surprisingly the quality of fit as measured by the coefficient of determination r^2 and the standard error, improved substantially in all but one of the subjects' models. Six of the subjects' coefficients of determination r^2 were greater than 99% leaving the remaining two subjects' r^2 at 98 and 96%. Of equal importance to the quality of fit, none of the subjects' fitted a_0 parameters deviated significantly from zero. This finding supports the assumption that phosphocreatine is entirely depleted during maximum exercise of this type.

Hence the model for the phosphocreatine depletion ratio (A3) was refitted excluding the parameter a_0 , i.e.:

$$\text{PDR} = (R - \text{PCr}(t))/R = \exp(-at^b). \quad (\text{A4})$$

Table 3. ANOVA to compare the subjects' phosphocreatine resynthesis ratio model (A6) parameters

| Source of variation | Sum of squares | Degrees of freedom | Mean square | F | P |
|--------------------------|----------------|--------------------|-------------|-------|--------|
| Overall regression | 23.59 | 1 | 23.59 | 982.9 | < 0.01 |
| Difference in intercepts | 2.09 | 7 | 0.299 | 12.46 | < 0.01 |
| Difference in slopes | 0.64 | 7 | 0.091 | 3.79 | < 0.05 |
| Residual error | 0.39 | 16 | 0.024 | | |
| Total | 26.71 | 31 | | | |

As before, by taking natural logarithms of the ratio PDR, we obtain the following power function relationship between \ln PDR and t :

$$\ln \text{PDR} = -at^b. \quad (\text{A5})$$

Acknowledging that the ratio PDR will always be less than unity and, as such, the parameter a will always be negative, by taking natural logarithms of the positive component of eqn (A5), i.e.:

$$\ln(-\ln \text{PDR}) = \ln a + b \ln t, \quad (\text{A6})$$

the parameters a and b can be fitted separately for each subject using linear least-squares regression. As can be seen in Fig. 8, the relationship between $\ln(-\ln \text{PDR})$ and $\ln t$ is acceptably linear. Each subject's estimated parameters, r^2 and half-recovery times are given in Table 2.

Compared with model (A3), the quality of fit remained almost unchanged with five of the subjects' coefficients of determination (r^2) greater than or equal to 99%, and the remaining three subjects' r^2 98, 98 and 94%.

Using the methods of Nevill, Cooke, Holder, Ramsbottom & Williams (1992), analysis of variance (ANOVA) can be used to test for the homogeneity of subjects' regression line parameters. A significant difference was found between the subjects' slope (shape) and intercept (location) parameters a and b of model (A6) (see Table 3). This finding suggests that each subject's phosphocreatine resynthesis model is sufficiently different to preclude a common group resynthesis model. By fitting model (A6) separately to each subject, the explained variance in the transformed phosphocreatine resynthesis ratio can be obtained (see Table 3) as $R^2 = (26.71 - 0.39)/26.71 = 98.5\%$.

By rearranging eqn (A4), the model for phosphocreatine resynthesis becomes:

$$\text{PCr}(t) = R - R \exp(-at^b), \quad (\text{A7})$$

as described in eqn (1) in the main text.

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