Power output and muscle metabolism during and following recovery from 10 and 20 s of maximal sprint exercise in humans

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ABSTRACT

On two separate days eight male subjects performed a 10- or 20-s cycle ergometer sprint (randomized order) followed, after 2 min of recovery, by a 30-s sprint. Muscle biopsies were obtained from the vastus lateralis at rest, immediately after the first sprint and after the 2 min of recovery on both occasions. The anaerobic ATP turnover during the initial 10 s of sprint 1 was $129 \pm 12$ mmol kg dry weight$^{-1}$ and decreased to $63 \pm 10$ mmol kg dry weight$^{-1}$ between the 10th and 20th s of sprint 1. This was a result of a 300% decrease in the rate of phosphocreatine breakdown and a 35% decrease in the glycolytic rate. Despite this 51% reduction in anaerobic ATP turnover, the mean power between 10 and 20 s of sprint 1 was reduced by only 28%. During the same period, oxygen uptake increased from $1.30 \pm 0.15$ to $2.40 \pm 0.23$ L min$^{-1}$, which partially compensated for the decreased anaerobic metabolism. Muscle pH decreased from $7.06 \pm 0.02$ at rest to $6.94 \pm 0.02$ after 10 s and $6.82 \pm 0.03$ after 20 s of sprinting (for all changes $P < 0.01$). Muscle pH did not change following a 2-min recovery period after both the 10- and 20-s sprints, but phosphocreatine was resynthesized to $86 \pm 3$ and $76 \pm 3\%$ of the resting value, respectively (n.s. 10- vs. 20-s sprint). Following 2 min of recovery after the 10-s sprint subjects were able to reproduce peak but not mean power. Restoration of both mean and peak power following the 20-s sprint was 88% of sprint 1, and was lower compared with that after the 10-s sprint ($P < 0.01$). Total work during the second 30-s sprint after the 10- and the 20-s sprint was $19.3 \pm 0.6$ and $17.8 \pm 0.5$ kJ, respectively ($P < 0.01$). As oxygen uptake was the same during the 30-s sprints ($2.95 \pm 0.15$ and $3.02 \pm 0.16$ L min$^{-1}$), and [Phosphocreatine] before the sprint was similar, the lower work may be related to a reduced glycolytic ATP regeneration as a result of the higher muscle acidosis.

Keywords aerobic metabolism, anaerobic, fatigue, glycolysis, pH, phosphocreatine, recovery, sprinting.

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It has long been observed that power output during maximal sprint cycling exercise against a fixed load peaks during the first 2–3 s and thereafter declines rapidly at first and becomes slower as the sprint continues (Lakomy 1986, Boobis et al. 1987, Bogdanis et al. 1994, 1995, Nevill et al. 1996). The decline of power output (i.e. fatigue) during such an intense type of exercise has been related to the inability to resynthesize or utilize energy at maximal rates (Hermansen 1981, Hultman et al. 1990). Thus, it is reasonable to assume that ATP resynthesis during a sprint decreases in a similar fashion to power output. The fact that $\approx 44\%$ of the total work done during a maximal 30-s cycling sprint is generated during the initial 10 s (Bogdanis et al. 1996) suggests that there is a high rate of ATP resynthesis during that time and a lower rate afterwards. Previous studies have shown that during a 6-s cycle ergometer sprint there is a high contribution (10–15 mmol kg dry muscle$^{-1}$ s$^{-1}$) by anaerobic sources to ATP resynthesis (Jacobs et al. 1983, Boobis et al. 1987, Gaitanos et al. 1993). However, this rate drops to $\approx 5$ mmol kg dry muscle$^{-1}$ s$^{-1}$ between the 6th and 30th second of the sprint as a result of reduced anaerobic glycolysis (Boobis et al. 1987) and a low

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phosphocreatine level (Gaitanos et al. 1993). Interestingly, power output is not reduced to the same extent. Thus muscles probably utilize aerobic energy sources to compensate for the decreased anaerobic metabolism during the latter part of a single sprint but there is a lack of quantitative data to show this. Studies where the contribution of aerobic metabolism during a single sprint was examined have almost exclusively used a 30-s exercise bout, while the methodologies and calculations used vary and have a relatively large effect on the results. Thus, values ranging between 18 and 46% have been reported as a percentage contribution of aerobic metabolism during a single 30-s sprint (Kavanagh & Jacobs 1988, Serresse et al. 1988, Medbo & Tabata 1989, Granier et al. 1995). If anaerobic energy supply decreases so rapidly as seen in the data of Boobis et al. (1987) then it is logical to assume that the aerobic contribution to energy supply is substantial from the early stages of sprinting. Measurements of oxygen uptake ($\dot{V}O_2$) during a 30-s sprint suggest that the aerobic contribution is increased throughout the sprint with the $\dot{V}O_2$ reaching as high as 85% of the maximal oxygen uptake (Kavanagh & Jacobs 1988). Therefore, in the present study the contribution of aerobic and anaerobic metabolism was examined during the initial and latter stages of a short (20-s) sprint. A 10- and a 20-s sprint was performed on two separate occasions and the anaerobic and aerobic contributions to energy supply (ATP resynthesis) were quantified by measuring changes in muscle metabolites and oxygen uptake ($\dot{V}O_2$) during the sprint. One hypothesis was that within a 20-s sprint, decreases in anaerobic metabolism will be compensated by an increase in aerobic metabolism. It was also hypothesized that the phosphocreatine (PCr) content of the muscle may go down to very low levels in the first 10 s of a maximum sprint. Indeed, in a study by Jones et al. (1985) using only two subjects, the PCr concentration after a 10-s sprint was as low as that measured after a 30-s sprint. However, muscle lactate in their study was almost twice as high after the 30-s sprint compared with after the 10-s sprint. Similar differences in muscle lactate between a 10- and a 30-s sprint have been reported by Jacobs et al. (1983). Thus, if PCr levels are similar at the end of the 10- and the 20-s sprint but muscle lactate and pH are different, the restoration of PCr and power output may be examined following a recovery period, under different levels of muscle acidity and thus, further investigate the aetiology of muscle fatigue during sprinting.

MATERIALS AND METHODS

Subjects

Eight male university students volunteered to participate in this study. Their age, height and body mass (mean ± SD) were 26 ± 3 years, 179 ± 6 cm and 77 ± 5 kg, respectively. All subjects were recreational athletes.

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. A medical history questionnaire was also completed in the presence of the experimenter, and subjects with medical problems were excluded. The protocol was approved by the Ethical Committee of Loughborough University.

Equipment

A modified friction-loaded cycle ergometer (Monark, model 864), 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 was passed around the subject’s waist to restrict exercise to the leg muscles during the cycle ergometer sprints. The two side straps of the belt were fixed to a metal rail, bolted on the floor behind the bicycle frame.

Preliminary tests

During a preliminary visit, the maximum oxygen uptake ($\dot{V}O_{2\text{max}}$) of each subject was determined using a continuous incremental test on the Monark cycle ergometer. The subjects performed a 2-min warm-up at 60 W (60 revs min$^{-1}$) and then work rate was increased by 30–60 W every 3 min until exhaustion ($\approx$11–14 min).Expired air was collected during the last 60 s of each stage using the Douglas bag technique. Heart rate was monitored throughout the test using short range telemetry (Sports Tester, Polar Electro Fitness, PE3000). Samples of expired air were analysed for fractions of $O_2$ and $CO_2$ using a paramagnetic oxygen analyser (Servomex-Sybron/Taylor, model 570 A) and a carbon dioxide analyser (Lira infra-red Gas analyser, model...
and the volume of expired air was measured by a Harvard dry gas meter. All gas volumes were corrected to STPD.

Main tests

The subjects were familiarized with sprint cycling by completing three separate sprint practice sessions. The two randomly assigned main tests were performed at the same time of the day, one week apart. 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. A standardized warm-up consisting of 4-min pedalling at 60 W followed by 2 × 30 s at 80 and 100 W, preceded each test. Five minutes after the completion of the warm-up, subjects performed two maximal cycle ergometer sprints, separated by 2 min of passive recovery on the cycle ergometer seat. On one occasion the duration of the first sprint was 10 s (10–30 condition) and on the other 20 s (20–30 condition). The second sprint lasted 30 s on both occasions (Fig. 1). The resistive load was 75 g kg\(^{-1}\) body mass (average load: 5.8 ± 0.1 kg) and each sprint started from a rolling start of ≈70 revs min\(^{-1}\). Strong verbal encouragement was given during each sprint.

The following performance parameters were obtained for each sprint: peak power output (PPO) and peak pedal speed, mean power output between 0 and 10 s (MPO\(_{10}\) ), between 10 and 20 s (MPO\(_{10-20}\) ) and between 0 and 30 s for sprint 2 only (MPO\(_{30}\) ). Mean pedal speed between 0 and 10 s and between 10 and 20 s of sprint 1 was also recorded. The fatigue index (FI) for power output was calculated as the percentage decline from peak to end power output for each sprint.

Expired air samples were collected in Douglas bags during each sprint. The test/re-test reliability for \(\dot{V}_O_2\) measurements during sprinting was determined in separate experiments. For a 30-s sprint, test 1 vs. test 2 \(\dot{V}_O_2\) values were: 2.53 ± 0.28 vs. 2.59 ± 0.29 L min\(^{-1}\) (n.s., \(n = 19\)), and the correlation coefficient (\(r\)) was 0.94 (standard error of estimate: 0.12 L min\(^{-1}\)). For a 10-s sprint, test 1 vs. test 2 \(\dot{V}_O_2\) values were: 1.64 ± 0.20 vs. 1.69 ± 0.19 L min\(^{-1}\) (n.s., \(n = 7\)), and the correlation coefficient (\(r\)) was 0.96 (standard error of estimate: 0.15 L min\(^{-1}\)).

Muscle samples

On arrival at the laboratory, subjects rested on a couch for 30 min, while small incisions were made through the skin and fascia over the vastus lateralis muscle of both legs, under local anaesthesia (1% plain lidocaine). The resting biopsy was then taken. Only one resting biopsy was obtained during one of the two main trials (balanced randomization). Further biopsies were taken immediately after the first sprint and 11.0 ± 2.1 s before the second sprint on both occasions, while the subject was sitting on the cycle ergometer (Fig. 1). The time delay between the end of sprint 1 and the time when the muscle sample was frozen in liquid nitrogen was 6.3 ± 0.5 s. The biopsy leg and testing order were randomized in a balanced design so that biopsies were taken from the same leg in each condition. Muscle samples were kept in liquid nitrogen until they were freeze dried (within 24 h of sampling).

Analytical methods

The freeze dried samples were dissected free of connective tissue and blood and homogenized. Muscle powder was then divided in two parts. One part of the powder was extracted with 0.5 M HClO\(_4\) and the extract was neutralized with 2.1 M KHCO\(_3\) (Harris et al. 1974). Phosphocreatine (PCr), creatine (Cr), ATP, free glucose, glucose 1-phosphate (G1P), glucose 6-phosphate (G6P), fructose 6-phosphate (F6P), pyruvate (Pyr), and lactate (Lac) were assayed enzymatically by fluorometric analysis (Lowry & Passoneau 1972). Glycogen was determined both in the neutralized extract (acid-soluble glycogen fraction), and in the muscle pellet left after the extraction procedure by prior HCl hydrolysis (acid-insoluble glycogen fraction). Muscle metabolite contents (except lactate and glucose) were adjusted to the individual mean total creatine content (PCr + Cr, range of corrections 0–11%). As the sum PCr + Cr does 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 as mmol kg dry muscle\(^{-1}\).
Muscle pH was determined in the second part of the freeze dried muscle powder after homogenization at 4 °C with a solution containing 145 mmol L$^{-1}$ KCl, 10 mmol L$^{-1}$ NaCl and 5 mmol L$^{-1}$ iodoacetic acid (Sahlin et al. 1976). The dilution ratio used was 100 μL of homogenate solution per milligram dry muscle. 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 anaerobic ATP turnover was calculated from the values of ATP, PCr, lactate (Lac) and pyruvate (Pyr) before and immediately after each sprint, using the formula:

\[
\text{ATP turnover} = 2(-\Delta \text{ATP}) - \Delta \text{PCr} + 1.5\Delta \text{Lac} + 1.5\Delta \text{Pyr}
\]

Two active phosphates are cleaved per ATP utilized; 1.5 mmol ATP is produced for every mmol Lac and Pyr. The mean anaerobic ATP turnover rate (mmol kg dry muscle$^{-1}$ s$^{-1}$) was obtained by dividing the anaerobic ATP turnover by the duration of the sprint (e.g. 10 or 20 s).

Glycogenolytic and glycolytic rates during each sprint (mmol glucosyl units kg dry muscle$^{-1}$ s$^{-1}$) were calculated from accumulation of glycolytic metabolites:

\[
\text{glycogenolysis} = (\Delta \text{G1P} + \Delta \text{G6P} + \Delta \text{F6P})
\]
\[
0.5(\Delta \text{Lac} + \Delta \text{Pyr})
\]

\[
\text{glycolysis} = 0.5(\Delta \text{Lac} + \Delta \text{Pyr})
\]

As the exercise was dynamic and blood flow to the quadriceps muscle was not restricted by any mechanical means (e.g. tourniquet), some lactate diffused into the circulation during the sprints. However, because of the short duration of the exercise bouts, the underestimation in the above calculations is thought to be minimal.

Changes in the concentration of inorganic phosphate (ΔPi) in the muscle after the sprints and during recovery, were calculated from changes in ATP, PCr and hexose monophosphates (G1P, G6P, F6P). ΔPi is expressed in mmol kg dry muscle$^{-1}$.

\[
\Delta \text{Pi} = 2(-\Delta \text{ATP}) - \Delta \text{PCr} - (\Delta \text{G1P} + \Delta \text{G6P} + \Delta \text{F6P})
\]

The concentration of Pi in the di-protonated form ($\text{H}_2\text{PO}_4^-$) was calculated from muscle pH and the pK of Pi (6.8), using the Henderson–Hasselbalch equation.

The contribution of aerobic metabolism to energy supply during each sprint was estimated from the $\dot{V}_\text{O}_2$ measurements and the following assumptions:

(1) Working muscle mass was calculated as 20% of body mass (Jones & Pearson 1969, Wootton, S.A., unpublished thesis).

(2) The conversion of wet to dry muscle weight was done assuming that muscle water content at rest was 77% of the total weight.

(3) The P/O$_2$ ratio was assumed to be 6.5 (6.5 mmol ATP mmol O$_2$)$^{-1}$ (Medbo & Tabata 1989).

(4) The ATP resynthesized from oxygen stored in the working muscle’s myoglobin and local capillary blood (1.5–2 mmol O$_2$ kg dry muscle$^{-1}$) was assumed to be 10 mmol ATP kg dry muscle$^{-1}$ (Harris et al. 1975). This contribution was assumed to take place during the first 10 s of the sprint (Medbo & Tabata 1989)

(5) The $\dot{V}_\text{O}_2$ before each sprint was subtracted from the $\dot{V}_\text{O}_2$ measured during the sprint, to give the net increase in oxygen uptake. The mean $\dot{V}_\text{O}_2$ value before sprint 1 was 0.55 ± 0.05 while the mean $\dot{V}_\text{O}_2$ before sprint 2 was 0.57 ± 0.05 when sprint 2 was 10 s and 0.66 ± 0.06 when sprint 2 was 20 s. These values were not significantly different and were measured in a separate experiment (n = 8).

Although estimation of aerobic energy supply from pulmonary (whole body) $\dot{V}_\text{O}_2$ may not be as accurate as local (working leg) $\dot{V}_\text{O}_2$ determinations, recent studies have shown that changes in whole body $\dot{V}_\text{O}_2$ reflect closely those occurring within the exercising legs both during submaximal and maximal cycling exercise (Knight et al. 1992, Poole et al. 1992).

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 a Tukey’s post-hoc test. Relationships between variables were examined by calculating the product moment correlation coefficient (r). Results are presented as mean ± standard error (SE).

RESULTS

Power output

The power output profiles during sprint 1 and sprint 2 in the two experimental conditions are shown in Fig. 2. The duration of sprint 1 was 10 s in one condition and 20 s in the other. Consequently, power output and pedal speed were similar during the initial 10 s of sprint 1 in both experimental conditions. For example, PPO and mean power output during the first 10 s of sprint 1 (MPO$_{10}$) were 1262 ± 46 and 886 ± 26 W in the 10–30 condition and 1271 ± 55 and 893 ± 29 W in the 20–30 condition (NS). The mean power between 10
and 20 s (MPO_{10-20}) in the 20–30 condition was 643 ± 19 W, which was ≈28% lower than the power generated during the first 10 s of the sprint. As expected from the longer duration of the 20-s sprint, the FI (% decline from peak to end power output) was higher compared with the 10 s sprint (56 ± 1 vs. 41 ± 2%; \( P < 0.01 \)).

Following the 2-min recovery after the 10-s sprint the subjects were able to reproduce the PPO achieved during sprint 1 (Fig. 3). However, mean power output during the first 10 s of the second sprint (MPO_{10}) did not recover fully (95 ± 1% of sprint 1; Fig. 3).

Restoration of PPO and MPO_{10} following the 20-s sprint was incomplete (89 ± 3 and 88 ± 1% of sprint

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**Figure 2**  Power output profiles (mean ± SE, \( n = 8 \)) for Sprint 1 (●) and Sprint 2 (◎), during the 10–30 (left) and the 20–30 condition (right).

**Figure 3**  Peak Power Output (PPO) and mean power output during the first 10 s (MPO_{10}) for each sprint during the two experimental conditions. (■), when sprint 1 was 10 s; (□), when sprint 1 was 20 s; * \( P < 0.01 \) from Sprint 1; † \( P < 0.01 \) from corresponding Sprint 2 in the other condition.

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Table 1 Muscle metabolites (mmol kg dry muscle⁻¹) in vastus lateralis at rest, immediately after the 10- and 20-s sprints, and following 2 min of recovery after the 10- (Rec 10 s), and the 20-s sprints (Rec 20 s)

<table>
<thead>
<tr>
<th>Muscle metabolites</th>
<th>Rest</th>
<th>10-s sprint</th>
<th>20-s sprint</th>
<th>Rec 10 s</th>
<th>Rec 20 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP</td>
<td>25.6 ± 0.7</td>
<td>20.2 ± 1.3</td>
<td>19.8 ± 1.4</td>
<td>21.8 ± 1.2*</td>
<td>19.8 ± 1.3*</td>
</tr>
<tr>
<td>PCr</td>
<td>80.7 ± 3.2</td>
<td>36.1 ± 3.0*</td>
<td>21.4 ± 2.2*</td>
<td></td>
<td>69.5 ± 3.3*</td>
</tr>
<tr>
<td>Creatine</td>
<td>37.0 ± 3.5</td>
<td>81.8 ± 5.7</td>
<td>96.4 ± 5.3</td>
<td>48.2 ± 4.3</td>
<td>56.4 ± 3.9</td>
</tr>
<tr>
<td>Total Cr</td>
<td>117.7 ± 5.0</td>
<td>117.9 ± 3.7</td>
<td>117.8 ± 4.3</td>
<td>117.7 ± 3.9</td>
<td>117.8 ± 4.0</td>
</tr>
<tr>
<td>ΔPi (dry)</td>
<td>–</td>
<td>38.8 ± 5.3</td>
<td>43.6 ± 6.1</td>
<td>84.8 ± 2.9</td>
<td>119.5 ± 4.5*</td>
</tr>
<tr>
<td>ΔH₂PO₄⁻</td>
<td>–</td>
<td>16.0 ± 2.9</td>
<td>21.4 ± 3.7</td>
<td>4.3 ± 1.5</td>
<td>5.8 ± 2.8</td>
</tr>
<tr>
<td>Glycogen</td>
<td>403.8 ± 20.1</td>
<td>357.4 ± 18.6</td>
<td>329.7 ± 21.4f</td>
<td>364.1 ± 25.0f*</td>
<td>328.4 ± 24.5f,g,d</td>
</tr>
<tr>
<td>Glucose</td>
<td>1.6 ± 0.1</td>
<td>3.0 ± 0.3</td>
<td>3.6 ± 0.3</td>
<td>4.0 ± 0.3</td>
<td>6.5 ± 0.5f,g,d</td>
</tr>
<tr>
<td>G1P</td>
<td>0.13 ± 0.02</td>
<td>0.85 ± 0.14*</td>
<td>1.09 ± 0.15*</td>
<td>0.35 ± 0.12c</td>
<td>0.30 ± 0.14e</td>
</tr>
<tr>
<td>G6P</td>
<td>1.2 ± 0.1</td>
<td>16.8 ± 1.8</td>
<td>22.5 ± 1.5b</td>
<td>9.6 ± 1.0h,b,c</td>
<td>16.7 ± 1.0e,b,c</td>
</tr>
<tr>
<td>F6P</td>
<td>0.18 ± 0.03</td>
<td>3.6 ± 0.4</td>
<td>5.5 ± 0.4b</td>
<td>2.0 ± 3.4e,c</td>
<td>3.5 ± 0.2e,c</td>
</tr>
<tr>
<td>HMP</td>
<td>1.5 ± 0.1</td>
<td>21.2 ± 2.3e</td>
<td>29.0 ± 1.7ab</td>
<td>11.9 ± 1.3h,e</td>
<td>20.5 ± 1.1e,b,c</td>
</tr>
<tr>
<td>Pyruvate</td>
<td>0.4 ± 0.1</td>
<td>2.8 ± 0.4</td>
<td>3.8 ± 0.3f</td>
<td>1.0 ± 0.2h,c</td>
<td>1.4 ± 0.3h,b,c</td>
</tr>
<tr>
<td>Lactate</td>
<td>4.5 ± 0.4</td>
<td>51.0 ± 4.6*</td>
<td>81.7 ± 4.7ab</td>
<td>38.2 ± 2.8e</td>
<td>66.2 ± 4.8f,g,d</td>
</tr>
</tbody>
</table>

Values are mean ± SE for eight subjects. ΔPi, calculated changes in inorganic phosphate; ΔH₂PO₄⁻, changes in the di-protonated form of inorganic phosphate; HMP, hexose monophosphates (G1P + G6P + F6P); Significant differences: *P < 0.01 and P < 0.05 from rest; b,f P < 0.01 and P < 0.05 from 10-s sprint; c,g P < 0.01 and P < 0.05 from 20-s sprint; d P < 0.01 between Rec 10 s and Rec 20 s.

1, respectively; Fig. 3), and values were significantly lower compared with those after the 10-s sprint (P < 0.01). The mean power during the second 30-s sprint after the 10- and the 20-s sprint was 644 ± 19 W and 592 ± 17 W, respectively (P < 0.01). However, the FI during the 30-s sprint was the same in the two conditions (63 ± 1%).

Peak pedal speed was reached 3.5 ± 0.2 s into the sprint in all sprints and was 166 ± 5 revs min⁻¹ during sprint 1. Peak pedal speed followed the same pattern of recovery as peak power output. The mean pedal speed during the first 10 s of sprint 1 was 149 ± 4 revs min⁻¹ and it dropped to 120 ± 4 revs min⁻¹ during the subsequent 10 s.

Subjects who had the higher FI during sprint 1, recovered less during sprint 2 (r = −0.93, P < 0.01, between FI and %PPO recovery in both conditions).

Muscle metabolites

Table 1 shows muscle metabolite concentrations at rest, after sprint 1 and before sprint 2 in the two experimental conditions. During the first 10 s of sprint 1, muscle glycogen decreased by 46 ± 6 mmol glucosyl units kg dry muscle⁻¹. There was a very good agreement between the calculated rate of glycogenolysis (from changes in La, Pyr and hexose monophosphates) and the measured rate of glycogen degradation (Table 2). During the second 10-s period of sprint 1, the rate of muscle glycogen degradation was ≈40% slower than that during the initial 10 s, and calculated glycogenolysis dropped from 4.4 ± 0.4 to 2.4 ± 4 mmol glucosyl units kg dry muscle⁻¹ s⁻¹ (P < 0.01). No resynthesis of muscle glycogen was observed during the 2-min recovery period following the 10- or the 20-s sprint.

A rapid rate of decrease of PCr (4.5 ± 0.5 mmol kg dry muscle⁻¹ s⁻¹) was observed during the initial 10 s of sprint 1, resulting in a 55 ± 2% fall in PCr concentration. However, only 14.7 ± 2.6 mmol PCr kg dry muscle⁻¹ were utilized between the 10th and 20th second of the first sprint, and PCr concentration at the end of 20 s was 27 ± 3% of the resting value. Following 1 min 49 s of recovery after the 10- and the 20-s sprint, PCr was resynthesized to 86 ± 3% and 76 ± 3% of the resting value, respectively (Table 1). These two values were not significantly different, but they were both significantly lower than the resting value (P < 0.01).

Changes in calculated Pi generally followed those of PCr. However, a large part of Pi was ‘trapped’ in the hexose monophosphates (HMP: G1P + G6P + F6P)

Table 2 Calculated anaerobic glycogenolytic and glycolytic rates, and measured rate of glycogen degradation (mmol glucosyl units kg dry muscle⁻¹ s⁻¹) during the first 10 s (0–10 s), the second 10 s (10–20 s) and the whole 20 s of sprint 1

<table>
<thead>
<tr>
<th>Rates</th>
<th>0–10 s</th>
<th>10–20 s</th>
<th>0–20 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycogenesis</td>
<td>4.4 ± 0.4</td>
<td>2.4 ± 0.4*</td>
<td>3.4 ± 0.2</td>
</tr>
<tr>
<td>Glycolysis</td>
<td>2.4 ± 0.2</td>
<td>1.6 ± 0.2**</td>
<td>2.0 ± 0.1</td>
</tr>
<tr>
<td>Ratio</td>
<td>1.8 ± 0.1</td>
<td>1.5 ± 0.1**</td>
<td>1.7 ± 0.1</td>
</tr>
<tr>
<td>Rate of ↓ of glycogen</td>
<td>4.6 ± 0.6</td>
<td>2.8 ± 0.5*</td>
<td>3.7 ± 0.4</td>
</tr>
</tbody>
</table>

The ratio of glycogenolysis to glycolysis is also presented for each time interval. Values are means ± SE (n = 8). • P < 0.01 and ** P < 0.05 from 0–10 s.
and therefore became unavailable for PCr resynthesis. Inorganic phosphate (in both the mono- and di-protonated forms) increased similarly after both the 10- and 20-s sprints, and was well above the resting value before sprint 2. There was no difference in Pi (and H₂PO₄⁻) concentration before sprint 2 between the two conditions (Table 1). It is noteworthy that ≈21 mmol kg dry muscle⁻¹ of inorganic phosphate appeared as HMP at the end of recovery following the 20-s sprint (Table 1).

All the decrease in ATP during sprint 1 (21 ± 2%) occurred during the first 10 s. No further changes in ATP were seen either after 10 more seconds of sprinting or after 2 min of recovery (Table 1).

The high glycolytic rate during the first 10 s of sprint 1 (Table 2) resulted in a marked increase in muscle lactate (to 51.0 ± 4.6 mmol kg dry muscle⁻¹). A significant reduction of the glycolytic rate during the second 10 s of sprint 1 (by ≈35%) was reflected as a decreased rate of muscle lactate accumulation (Table 1). Recovery of muscle lactate was slow, with a small drop of ≈13–16 mmol lactate kg dry muscle⁻¹ at the end of the recovery period after both sprints. However, this decrease was only statistically significant in the 20–30 condition. As can be seen in Table 1, subjects started the second sprint with high muscle lactate in both conditions, but muscle lactate was almost two times higher when the 20-s sprint preceded the 30-s sprint (Table 1). No correlations were found between muscle lactate and the recovery of PCr.

**ATP turnover**

The anaerobic ATP turnover during the initial 10 s of sprint 1 was 129 ± 12 mmol kg dry weight⁻¹ but was decreased to 63 ± 10 mmol kg dry weight⁻¹ between the 10th and 20th second of sprint 1. This was a result of a 300% decrease in the rate of PCr breakdown and an ≈35% decrease in the glycolytic rate. Despite this 51% reduction in anaerobic ATP turnover, the mean power output between 10 and 20 s of sprint 1 was reduced by only 28% (Fig. 4). During the same period, oxygen uptake increased from 1.30 ± 0.15 L min⁻¹ during the first 10 s to 2.40 ± 0.23 L min⁻¹, thereby increasing the contribution of aerobic metabolism to energy supply (Table 3). A further increase in VO₂ was observed during sprint 2 which was similar in both conditions (2.95 ± 0.15 and 3.02 ± 0.16 L min⁻¹, NS). The calculated ATP resynthesis from aerobic metabolism during sprint 1 is shown in Table 3. Aerobic metabolism contributed 13 ± 2% of the total energy during the first 10 s, and its contribution was doubled during the next 10-s period (27 ± 5%). The subjects with the higher VO₂ max had the higher percentage aerobic contribution to both sprint 1 (20 s; r = 0.79, P < 0.05) and sprint 2 (r = 0.83, P < 0.05). Glycolytic ATP represented 50–55% of the total energy during both 10-s periods, and the contribution of PCr was only 17% between the 10th and 20th second of the sprint.

When the contribution of aerobic metabolism was taken into account, the decrease in the rate of ATP turnover from the first 10-s to the second 10-s period of sprint 1 was ≈40% as opposed to 51% calculated only from anaerobic sources. This value is closer to the ≈28% decrease in power output between the same time intervals.
Muscle pH after 10 and 20 s of sprinting and at the end of the 2-min recovery period are shown in Fig. 5. Muscle pH decreased significantly from 7.06 ± 0.02 at rest to 6.94 ± 0.02 after 10 s of sprinting (P < 0.01). A further drop of muscle pH to 6.82 ± 0.03 (P < 0.01 from 10 s) was observed at the end of the 20 s sprint. No recovery of muscle pH was seen following a 2-min recovery period after both the 10- and the 20-s sprints (Fig. 5).

**DISCUSSION**

This study examined two separate aspects of muscle metabolism and power output during sprinting: development of fatigue during the first sprint, and recovery under different metabolic environments (higher vs. lower muscle acidity). One main finding was that the calculated ATP turnover rate from anaerobic sources decreased by 51% during the second half (10–20 s) of the first 20-s sprint. This was mainly due to the 3-fold decrease of PCr breakdown during the second 10 s compared with the initial 10 s of the sprint (15 vs. 45 mmol kg dry muscle\(^{-1}\)), and a 35% decrease in glycolysis (Fig. 4). This large decrease in the calculated anaerobic energy supply after only 10 s of sprinting, was associated with a less severe (28%) decrease in mean power output during the same time period. Similar findings have been reported by Gaitanos et al. (1993) during 10 6-s cycle ergometer sprints separated by 30 s of recovery. They found a 27% decline in mean power output from sprint 1 to sprint 10, which was accompanied by a 65% decrease in the calculated anaerobic ATP production.

Two possible explanations may be put forward for this mismatch between the decrease in power output and the calculated energy supply from anaerobic sources. Firstly, the contribution of aerobic energy metabolism may constitute a significant part of energy supply even during an all-out sprint of short duration. Secondly, an increase in efficiency expressed as less energy (ATP turnover) per unit power may have occurred during the second 10 s of the sprint.

Evidence that aerobic metabolism may contribute significantly to energy supply during high intensity exercise of short duration has previously been reported mostly for 30-s bouts (Kavanagh & Jacobs 1988, Serresse et al. 1988, Medbo & Tabata 1989, 1993, Withers et al. 1991, Granier et al. 1995, Bogdanis et al. 1996). Measurements of the oxygen consumption in the present study indicated that aerobic energy sources became more important as sprinting was continued because \(\dot{V}_O_2\) was almost doubled from the first to the second 10 s of sprint 1, increasing the percentage contribution of aerobic metabolism from 13% to 27%.

This finding highlights the importance of aerobic metabolism even during sprints of short duration, where it partially compensates for the decreased PCr levels and the impaired anaerobic glycolysis.
Although the inclusion of the aerobic contribution estimates to the total ATP turnover reduced the discrepancy between the decrease in ATP turnover and the decrease in power output from the first 10-s to the second 10-s period of sprint 1, it was not adequate to totally account for it (Fig. 4). The remaining 12% difference, after the inclusion of aerobic metabolism, may be explained by a change in efficiency as the subjects cycled slower during the later stages of sprint 1. The issue of efficiency during sprint cycling is complex, especially when sprinting on a friction loaded cycle ergometer. Experiments using isokinetic cycle ergometers, where the pedal speed is constant throughout the sprint, have suggested that there is a parabolic relationship between power and pedal speed (Sargeant et al. 1981, McCartney et al. 1983, 1985). This implies two things: (1) there is an ‘optimum’ pedal speed at which the highest power is generated and (2) cycling faster or slower than that speed would result in a reduced power output purely as a result of the shape of the power velocity curve. Furthermore, it has been suggested that the percentage contribution of slow twitch fibres to power generation will be high when a muscle of ‘mixed’ composition is contracting at slow speeds (Faulkner et al. 1986). The mean pedal speed during the first 10 s of sprint 1 in the present study was 149 ± 4 revs min⁻¹ and it dropped to 120 ± 4 revs min⁻¹, during the second 10 s. This latter value is closer to an optimum value of 110 revs min⁻¹ suggested by Sargeant et al. (1981) and therefore it can be argued that the remaining 12% discrepancy between the decline in total (aerobic + anaerobic) ATP turnover and power output can be explained by that improvement in ‘efficiency’.

An interesting finding of this study was that all the decrease in muscle ATP content occurred during the first 10 s of sprint 1. Loss of ATP during intense muscle contractions occurs when the rate of ADP rephosphorylation fails to keep in pace with the rate of ATP turnover, and serves to prevent large increases in free ADP and AMP and maintain a high ATP/ADP ratio which is important for cell energetics. The activity of the enzyme AMP deaminase, which catalyses AMP conversion to IMP and ammonia, is increased by free ADP and H⁺, but availability of substrate (AMP) is the primary stimulus for deamination (Tullson & Terjung 1991). In the present study, the drop in muscle pH during the first 10 s of sprint 1 was only 0.12 pH units which corresponds to 28 nmol H⁺ L⁻¹. If muscle pH was an important factor for AMP deamination, it would be expected that the major losses in ATP would be seen later in the sprint when pH drops even further (pH: 6.82 or 152 nmol H⁺ L⁻¹ at the end of the 20-s sprint). Therefore, the loss of ATP at the beginning as opposed to later in the sprint was probably related to the extremely high ATP turnover rates observed (≈13 mmol ATP kg dry muscle⁻¹ s⁻¹) during the first 10 s of the sprint. Comparison with muscle metabolite data from similar cycle ergometer sprints of longer (30 s) duration reveals that only a small decrease in ATP occurs between the 10th and 30th second of a sprint, or even when a 30-s sprint is repeated after a 4-min recovery interval despite the decreases of muscle pH to 6.6–6.7 (McCartney et al. 1986, Boobis et al. 1987, Spriet et al. 1989, Bogdanis et al. 1995, 1996). The common observation in all those studies is that the rate of ATP turnover drops dramatically after the initial seconds of sprinting and also when a sprint is repeated. This finding points to the important role of the ATP turnover rate in AMP deamination and ATP loss (Katz et al. 1986).

The second aspect examined by the present study was the recovery of muscle metabolism and power output following a short rest (2 min) period after the 10- and the 20-s sprint. It was hypothesized that PCR would drop to similar levels after 10 and 20 s of sprinting, and that muscle lactate would be higher and muscle pH lower after the 20-s sprint. Although a large decrease in PCR was observed after both sprints, PCR was ≈15 mmol kg dry muscle⁻¹ higher after the 10 s compared with after the 20 s sprint. Interestingly, PCR was resynthesized to the same level in both conditions following the 2 min of rest. Comparison of the number of millimoles of PCR resynthesized after the 10- and 20-s sprint (33 ± 4 vs. 40 ± 3 mmol kg dry muscle⁻¹, respectively) also showed no significant difference. Considering that muscle lactate and [H⁺] were higher after the 20-s sprint, the similar PCR resynthesis would imply that during this short recovery interval PCR resynthesis was not affected by the extent of muscle acidity observed in this study. This is in agreement with the suggestion that the initial PCR resynthesis is mainly limited by O₂ availability, and not pH (Sahlin et al. 1979).

A major finding of the present study was that following the 2-min recovery after the 10-s sprint the subjects were able to reproduce the PPO achieved during sprint 1, despite the elevated muscle lactate, Pi, H₂PO₄⁻ and [H⁺]. This shows that increased acidity at this level (muscle lactate 30 mmol kg dry muscle⁻¹ and muscle [H⁺] 120 nmol L⁻¹) and inorganic phosphate do not significantly affect the ability of the muscle to generate maximum power over very short intervals of time. A similar conclusion was reached by Sahlin & Ren (1989) for the recovery of force. They observed full restoration of the maximum isometric voluntary contraction 2 min after fatigue, although exercise in spite of a high muscle lactate content. The complete recovery of peak power in the present study is an important finding, because it shows that both components of power...
output, force and velocity of contraction, had recovered.

In contrast, the restoration of mean power output (MPO10) following the 2 min of recovery after the 10-s sprint was not complete (95 ± 1% of sprint 1). Restoration of MPO10 would require similar high rates of ATP regeneration as those seen during the first 10 s of sprint 1. The incomplete resynthesis of PCr before sprint 2 and a possible reduction of glycolysis because of elevated H+ are expected to reduce the anaerobic ATP regeneration during the first 10 s of sprint 2, while the increase in aerobic metabolism is probably not large enough to compensate (Bogdanis et al. 1996). Thus, it seems that following recovery from the 10-s sprint, muscle could reproduce its performance only during the initial 1–4 s of the sprint when PCr and glycolysis were possibly contributing energy at higher rates than during the next few seconds. As can be seen in Fig. 2, the power output profile during sprint 2 was getting further apart from that of sprint 1 as the sprint progressed.

In the 20–30 condition, recovery of all power indices was incomplete and also lower than that after the 10-s sprint. Figure 2 shows the power output profiles for sprint 1 and sprint 2 in this condition. It is interesting that PCr and calculated inorganic phosphate and H2PO4− were similar before sprint 2 in the two conditions (following the 10- and 20-s sprints). However, muscle lactate was 38 and 66 mmol kg dry muscle−1, respectively, and muscle [H+] was 120 and 156 nmol L−1. Although no muscle biopsies were taken after sprint 2 to allow a direct assessment of the effects on muscle metabolism, the similar PCr before and the same oxygen consumption during sprint 2 in the two conditions point to a greater reduction in glycolytic ATP regeneration as a result of a higher muscle acidity after the 20-s sprint.

An alternative, very attractive approach that may contribute to the interpretation of the observed changes in metabolism and the decrease in power output during the first and the second sprints, is to view the contracting muscle as a population of fibres a part of which is fast contracting/fast fatiguing and another part consisting of slow contracting/slow fatiguing fibres. Soderlund et al. (1992) have shown a rapid decline in PCr degradation rates in fast twitch fibres in the second half of 19.2 s of intense electrical stimulation of human quadriceps muscle and attributed fatigue to the rapid loss of PCr in this fibre population. Similarly, Greenhaff et al. (1993) found that glycoenerolysis is twice as high in fast twitch than in slow twitch fibres during intense electrical stimulation lasting 32 s. Consequently, the disturbance in metabolism may be greater in fast twitch fibres and the whole muscle performance may be impaired as result of ‘selective fatigue’ of this fibre type. This may, of course, have an impact on subsequent bouts of exercise as PCr resynthesis is slower in fast twitch fibres (Casey et al. 1996) and glycolysis in fast twitch fibres may not be as high in a second compared to the first bout of exercise. A decreased contribution from fast twitch fibres may also explain the lack of further decrease in ATP observed between 10 and 20 s of sprinting, as it has been shown that fast twitch fibres possess a higher capacity for AMP deamination than the slow twitch fibres (Meyer & Terjung 1979). These notions may provide further explanation of the results of the present study and give support to a mainly metabolic basis of fatigue during this type of exercise. However, impairment of excitation–contraction coupling, especially in the fast twitch/highly glycolytic fibres, should not be disregarded (Westerblad et al. 1991) but the results of the present study do not allow any speculations regarding this theory of muscle fatigue.

In summary, this study has shown that the rate of ATP turnover from anaerobic sources was reduced by approximately 51% during the second half of a 20-s sprint, as a result of low PCr and a 35% decrease in glycolysis. However, mean power output decreased by only 28% because of a 2-fold increase aerobic energy contribution (from 13 to 27% of total ATP turnover) which partially compensated for the decreased anaerobic metabolism. This finding demonstrates the importance of aerobic metabolism even during the early stages of a single bout of sprint exercise. At the end of the recovery period following the 10-s sprint, peak power could be generated despite the increased muscle lactate, [H+] and inorganic phosphate, indicating that both the force and velocity components of muscle function were unaffected. However, mean power output during the first 10 s of the second sprint could not be reproduced as the total rate of ATP resynthesis was probably reduced at this period. The resynthesis of PCr following 10 or 20 s of sprint exercise was similar and was not affected by the different levels of muscle acidosis. However, performance during the second 30-s sprint was possibly affected by muscle acidosis, since power output was lower following recovery from the 20-s sprint. Since PCr before, and $V_{\text{O}_2}$ during the 30-s sprints were similar, the lower power output may be related to a reduced glycolytic ATP regeneration because of the higher muscle acidosis.

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