The metabolic responses of human type I and II muscle fibres during maximal treadmill sprinting

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1. Muscle biopsy samples were obtained from the vastus lateralis of six healthy volunteers before and after 30 s of treadmill sprinting. A portion of each biopsy sample was used for mixed-fibre metabolite analysis. Single fibres were dissected from the remaining portion of each biopsy and were used for ATP, phosphocreatine (PCr) and glycogen determination.

2. Before exercise, PCr and glycogen contents were higher in type II fibres (79·3 ± 2·7 and 472 ± 35 mmol (kg dry matter (DM))$^{-1}$, respectively) compared with type I fibres (71·3 ± 3·0 mmol (kg DM)$^{-1}$, $P < 0·01$ and 375 ± 25 mmol (kg DM)$^{-1}$, $P < 0·001$, respectively).

3. Peak power output was 885 ± 66 W and declined by 65 ± 3 % during exercise. Phosphocreatine and glycogen degradation in type II fibres during exercise (74·3 ± 2·5 and 126·3 ± 15·8 mmol (kg DM)$^{-1}$, respectively) was greater than the corresponding degradation in type I fibres (59·1 ± 2·9 mmol (kg DM)$^{-1}$, $P < 0·001$ and 77·0 ± 14·3 mmol (kg DM)$^{-1}$, $P < 0·01$, respectively). The decline in ATP during exercise was similar when comparing fibre types ($P > 0·05$).

4. Compared with previous studies involving similar durations of maximal cycling exercise, isokinetic knee extension and intermittent isometric contraction, the rates of substrate utilization recorded in type I fibres were extremely high, being close to the rapid rates observed in this fibre type during intense contraction with limb blood flow occluded.

Several studies have investigated the metabolic response of mixed-fibre human skeletal muscle during maximal short-term dynamic exercise (Boobis, Williams & Wooton, 1982, 1983; Jacobs *et al.* 1982; Jacobs, Tesch, Bar-Or, Karlsson & Dotan, 1983; Jones, McCartney, Graham, Spriet, Kowalchuk, Heigenhauser & Sutton, 1985; McCartney, Spriet, Heigenhauser, Kowalchuk, Sutton & Jones, 1986; Cheetham, Boobis, Brooks & Williams, 1986; Nevill, Boobis, Brooks & Williams, 1989; Spriet, Lindinger, McKelvie, Heigenhauser & Jones, 1989). The quadriceps muscle group of man includes muscles composed of at least three distinct types. For example, it has been known for some time that human type II muscle fibres possess higher activities of myosin ATPase, creatine kinase, myokinase, phosphorylase, phosphofructokinase and lactate dehydrogenase than human type I muscle fibres (Essen, Jansson, Henriksson, Taylor & Saltin, 1975; Harris, Essen & Hultman, 1976; Thorstensson, Sjodin, Tesch & Karlsson, 1977), which fits well with the comparatively higher anaerobic ATP turnover rate (Crow & Kushmerick, 1982) and peak power output (Faulkner, Claflin & McCully, 1986) associated with this fibre type. It is perhaps surprising, therefore, that few studies have been undertaken to investigate energy metabolism in different muscle fibre types during maximal short-term sprint exercise in man (Friden, Seger & Ekblom, 1989; Vollestad, Tabata & Medbo, 1992). Those studies that have been reported in the literature have usually relied upon histochemical techniques and have not taken into account the contribution made by phosphocreatine (PCr) to ATP resynthesis. Tesch, Thorsson & Fujitsuka (1989) demonstrated that PCr was markedly reduced in both fibre types during exhaustive short-lasting voluntary isokinetic exercise, with a tendency for utilization to be greater in type II fibres. However, no measure of single fibre glycogen degradation was made in this study. We have recently demonstrated (Greenhaff, Ren, Soderlund & Hultman, 1991; Hultman, Greenhaff, Ren & Soderlund, 1991;

Soderlund, Greenhaff & Hultman, 1992) that during short-lasting intense intermittent (1·6 s stimulation, 1·6 s rest) electrical stimulation in man a rapid rate of glycogenolysis occurs, which is almost totally restricted to type II muscle fibres, in conjunction with a decline in the rate of type II fibre PCr utilization. No corresponding changes were observed in type I fibres dissected from the same muscle biopsy samples.

The metabolic responses recorded in mixed-fibre skeletal muscle during intense intermittent electrical stimulation (Greenhaff *et al.* 1991; Hultman *et al.* 1991; Soderlund *et al.* 1992) were similar to those recorded during similar durations of maximal short-term dynamic exercise (Jacobs *et al.* 1983; Jones *et al.* 1985; Cheetham *et al.* 1986; McCartney *et al.* 1986). The present experiment was therefore undertaken to quantify PCr and glycogen utilization in type I and II muscle fibres during maximal treadmill sprinting.

METHODS

Subjects

Six healthy volunteers (5 males, 1 female) previously familiarized with treadmill sprinting agreed to take part in the present experiment. Their mean age, height and weight was 26 years (range, 23–29 years), 173·0 cm (range, 158–177 cm) and 71·0 kg (range, 57·8–79·4 kg), respectively. Two of the subjects were involved in a regular training programme and the remaining four were all active. Before commencement of the study, the experimental protocol was explained to all subjects and their written consent was obtained. The study was approved by the Ethical Committee of Loughborough University.

Exercise

All subjects were acquainted with treadmill sprinting on a non-motorized treadmill (Cheetham *et al.* 1986; Nevill *et al.* 1989) during the weeks before the start of the experiment. On the day of the study, all subjects reported to the laboratory after an overnight fast. The study began with two submaximal 30 s bouts of running. This served as both a warm-up and re-familiarization exercise. Five minutes following its completion, a 30 s maximal sprint from a rolling start of 8 km h^{-1} was performed. All subjects were asked to run maximally and received strong verbal encouragement throughout the sprint. The reproducibility of the exercise test used has previously been shown to be satisfactory (Cheetham, Williams & Lakomy, 1985) and the equipment used to perform and monitor sprint exercise has been described by Lakomy (1987). Briefly, treadmill belt velocity was monitored using a DC generator attached to the front rolling drum of the treadmill. The output voltage from the generator was monitored by a microcomputer using an analog-to-digital converter. Restraint force was also monitored by the microcomputer by fixing a non-elastic belt around the waist of each subject and then to a force transducer at the rear of the treadmill. The instantaneous product of the restraint force and the belt velocity was used by the microcomputer to determine the horizontal component of the power generated during exercise. Power was averaged over 1 s time intervals and was displayed on the computer monitor at the end of the sprint. Throughout this manuscript, the maximum 1 s average of the product of treadmill belt speed and restraint force will be referred to as 'peak power output' and the corresponding integral for the whole 30 s of exercise will be referred to as 'mean power output'.

Blood sampling and analysis

Venous blood samples (5 ml) were obtained, from a 21 gauge venous catheter placed in a superficial forearm vein, at rest before exercise, immediately prior to sprint exercise and at 0, 1, 5, 10 and 15 min following the completion of exercise, with subjects resting in a supine position. Upon removal, ~4 ml of blood was mixed with K_3EDTA and the remainder was used for the determination of plasma pH (Corning pH blood/gas 161 meter; Corning, Stone, Staffs, UK). The blood in the K_3EDTA tube was immediately mixed and an aliquot was deproteinized in 2·5 % perchloric acid, centrifuged, frozen and later used for blood lactate determination using the method described by Maughan (1982). A further 2 ml of blood was centrifuged and the plasma was rapidly frozen and stored in liquid nitrogen. The plasma was used for the determination of ammonia concentration (NH_4^+; Sigma Chemical Co., procedure 170-UV) within 3 days of storage.

Muscle sampling and analysis

Needle biopsy samples (Bergstrom, 1962) were obtained from the vastus lateralis muscle of each subject at rest before exercise and immediately after sprinting. All samples were immediately frozen in liquid nitrogen and after removal from the biopsy needle were freeze-dried and stored at −80 °C until analysed at a later date. The time delay between the cessation of sprinting and the freezing of the biopsy sample was ~7 s.

Each freeze-dried muscle sample was divided into two portions. One was dissected free from blood and connective tissue, powdered and used for the determination of mixed-fibre muscle ATP, PCr, glucose, glycogen, glucose 1-phosphate (G1P), glucose 6-phosphate (G6P), fructose 6-phosphate (F6P), glycerol 3-phosphate (Gly3P) and lactate content according to the methods of Harris, Hultman & Nordesjo (1974). Fragments of single muscle fibres (about 60–100 from each biopsy) were dissected free from the remaining portion of muscle with the aid of low power microscopy. The ends of the fragments were cut off and stained for myofibrillar ATPase, enabling type I and II fibres to be identified (Brooke & Kaiser, 1970; Essen *et al.* 1975). The remainder of each fragment was then weighed on a quartz fibre fishpole balance (Lowry & Passonneau, 1972), which had been previously calibrated spectrophotometrically by determining the weight of *p*-nitrophenol crystals that had been weighed on the balance. After fibre characterization, eight to fifteen fibres of each type were pooled to form two 20–25 μg pools. Each pool of fibres was then digested by adding 20 μl KOH (1 M) and glycogen was extracted by vigorously mixing and warming the samples at 50 °C for 15 min. The extract was then neutralized by adding HCl (0·25 M) and was assayed for glucosyl units using a fluorimetric modification of the method of Harris *et al.* (1974). A minimum of ten fibres of each type were used for the luminometric determination of ATP and PCr in individual fibre fragments (i.e. non-pooled) according to the method of Wibom, Soderlund, Lundin & Hultman (1991).

Statistical analysis

A comparison of pre- and post-exercise blood and muscle metabolite levels was achieved using Student's paired *t* test. Values in the text, tables and figures represent means ± S.E.M.

RESULTS

Power output

Performance variables are shown in Table 1. Power output is shown in absolute terms (W) and relative to body weight (W kg^{-1}). On all occasions peak power output was achieved within the first 3 s of exercise and declined thereafter. The mean power output throughout the 30 s of exercise was equal to 552 ± 40 W ($7{\cdot}7 \pm 0{\cdot}4$ W kg^{-1}). The percentage fatigue during exercise was calculated as:

$$\frac{\text{Peak power output} - \text{end power output}}{\text{Peak power output}} \times 100,$$

and averaged $65{\cdot}0 \pm 3{\cdot}4$ %.

Blood pH and metabolites

Sprinting resulted in a marked decline in plasma pH and increases in blood lactate and plasma ammonia. The greatest decline in plasma pH was recorded immediately after exercise ($7{\cdot}01 \pm 0{\cdot}03$) and values had still not returned to 'normal' following 15 min of recovery. Peak blood lactate ($17{\cdot}08 \pm 0{\cdot}76$ mmol l^{-1}) and plasma ammonia (232 ± 14 μmol l^{-1}) concentrations were recorded following 5 and 10 min of recovery, respectively. Levels of both metabolites were still markedly elevated at the end of the recovery period (Table 2).

Mixed-fibre muscle metabolites

Exercise resulted in a 3·3, 60·1 and 82·0 mmol (kg dry matter (DM))$^{-1}$ decline in muscle ATP, PCr and glycogen contents, respectively (Table 3). Muscle free glucose content increased by 3·4 mmol (kg DM)$^{-1}$ and the 24·0 mmol (kg DM)$^{-1}$ rise in G6P accounted for 76 % of the observed increase in hexose monophosphates. Muscle Gly3P increased by 6·2 mmol (kg DM)$^{-1}$ and muscle lactate by 86·6 mmol (kg DM)$^{-1}$. The total number of glucosyl units produced as a result of glycogen degradation was 82·0 mmol (kg DM)$^{-1}$, of which 34·6 mmol (kg DM)$^{-1}$ was in the form of phosphorylated glycolytic intermediates and at least 43·3 mmol (kg DM)$^{-1}$ was in the form of lactate (Table 3). Thus, even assuming that all of the lactate produced during exercise was retained in muscle, very little of the degraded glycogen was used for oxidative ATP resynthesis.

Single muscle fibre metabolites

The pre- and post-exercise single fibre metabolite measures are shown in Table 4.

Prior to exercise, no difference was observed when comparing the ATP content of each fibre type ($P > 0{\cdot}05$). However, type II fibre PCr ($P < 0{\cdot}01$) and glycogen contents ($P < 0{\cdot}001$) were higher than the corresponding levels in type I fibres.

The decline in ATP during exercise was similar when comparing fibre types ($P > 0{\cdot}05$), amounting to $3{\cdot}5 \pm 0{\cdot}5$ and $5{\cdot}0 \pm 1{\cdot}1$ mmol (kg DM)$^{-1}$ in type I and II fibres,

Table 1. Performance variables obtained during 30 s maximal sprint exercise in humans ($n = 6$)

	Mean	S.E.M.	Range
Peak power (W)	885	66	620–1061
Peak power (W kg^{-1})	12·4	0·7	10·6–15·2
Mean power (W)	552	40	383–650
Mean power (W kg^{-1})	7·7	0·4	6·6–9·3
Fatigue index (%)	65·0	3·4	55·8–75·9

Table 2. Plasma pH, blood lactate and plasma ammonia concentrations at rest, following warm-up exercise and at 0, 1, 5, 10 and 15 min after 30 s of maximal sprint exercise

	pH	Lactate (mmol l^{-1})	Ammonia (μmol l^{-1})
Rest	$7{\cdot}39 \pm 0{\cdot}01$	$0{\cdot}84 \pm 0{\cdot}04$	20 ± 7
Warm-up	$7{\cdot}38 \pm 0{\cdot}01$	$1{\cdot}52 \pm 0{\cdot}10$	19 ± 5
0 min	$7{\cdot}01 \pm 0{\cdot}03$	$12{\cdot}86 \pm 1{\cdot}10$	207 ± 19
1 min	$7{\cdot}09 \pm 0{\cdot}04$	$12{\cdot}26 \pm 1{\cdot}22$	171 ± 20
5 min	$7{\cdot}06 \pm 0{\cdot}02$	$16{\cdot}58 \pm 0{\cdot}50$	232 ± 14
10 min	$7{\cdot}10 \pm 0{\cdot}02$	$17{\cdot}08 \pm 0{\cdot}76$	225 ± 16
15 min	$7{\cdot}15 \pm 0{\cdot}02$	$16{\cdot}12 \pm 0{\cdot}95$	206 ± 20

Values represent means ± S.E.M.

Table 3. Selected metabolite contents measured in mixed-fibre muscle at rest immediately prior to and after 30 s maximal sprint exercise in humans

	Pre-exercise content (mmol (kg DM)$^{-1}$)	Post-exercise content (mmol (kg DM)$^{-1}$)
ATP	$25{\cdot}7 \pm 0{\cdot}5$	$22{\cdot}4 \pm 1{\cdot}2$
PCr	$81{\cdot}2 \pm 4{\cdot}2$	$21{\cdot}1 \pm 2{\cdot}4$
Glycogen	417 ± 33	335 ± 27
Glucose	$1{\cdot}97 \pm 0{\cdot}34$	$5{\cdot}34 \pm 0{\cdot}47$
G1P	$0{\cdot}22 \pm 0{\cdot}09$	$1{\cdot}60 \pm 0{\cdot}07$
G6P	$2{\cdot}58 \pm 1{\cdot}37$	$26{\cdot}60 \pm 1{\cdot}30$
F6P	$0{\cdot}67 \pm 0{\cdot}41$	$6{\cdot}82 \pm 0{\cdot}34$
Gly3P	$0{\cdot}86 \pm 0{\cdot}61$	$7{\cdot}02 \pm 0{\cdot}32$
Lactate	$3{\cdot}1 \pm 1{\cdot}1$	$89{\cdot}7 \pm 3{\cdot}7$
Total glucosyl units degraded from glycogen		82·0
Total phosphorylated glucosyl units		34·6
Total glucosyl units in form of lactate		43·3
Total glucosyl units in muscle		77·9

Values represent means ± S.E.M. $n = 6$.

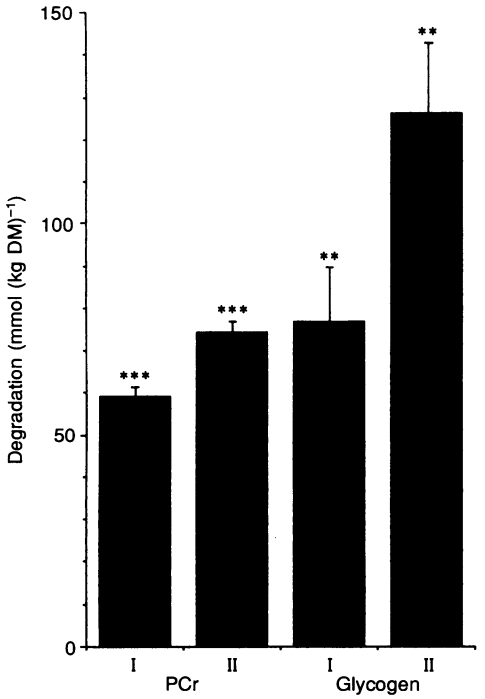

Figure 1. Phosphocreatine (PCr) and glycogen degradation in type I and type II muscle fibres during 30 s maximal treadmill sprinting in humans
Values represent means ± S.E.M. Significant differences between fibre types are indicated as follows: $^{**}P < 0.01$; $^{***}P < 0.001$. $n = 6$.

respectively. The decline in PCr during exercise was greater in type II fibres (74·3 ± 2·5 mmol (kg DM)$^{-1}$ than in type I fibres (59·1 ± 2·9 mmol (kg DM)$^{-1}$, $P < 0.001$; Fig. 1). This resulted in a 25 % higher rate of PCr degradation in type II fibres (2·47 ± 0·08 mmol (kg DM)$^{-1}$ s^{-1}) compared with type I fibres (1·97 ± 0·10 mmol (kg DM)$^{-1}$ s^{-1}, $P < 0.001$). The decline in type II muscle fibre glycogen content during exercise was also greater in type II fibres (126 ± 16 mmol (kg DM)$^{-1}$) when compared with type I fibres (77 ± 14 mmol (kg DM)$^{-1}$, $P < 0.01$; Fig. 1). As a consequence, the glycogenolytic rate in type II fibres (4·21 ± 0·53 mmol (kg DM)$^{-1}$ s^{-1}) was 64 % higher than in type I fibres (2·57 ± 0·48 mmol (kg DM)$^{-1}$ s^{-1}, $P < 0.01$).

DISCUSSION

Several studies have demonstrated very high rates of muscle PCr and glycogen utilization in mixed-fibre skeletal muscle during maximal short-term dynamic exercise (Boobis *et al.* 1982, 1983; Jacobs *et al.* 1982, 1983; Jones *et al.* 1985; Cheetham *et al.* 1986; McCartney *et al.* 1986; Nevill *et al.* 1989; Spriet *et al.* 1989), which are not maintained when repeated bouts of exercise are undertaken (McCartney *et al.* 1986; Spriet *et al.* 1989). It has been previously suggested from the analysis of mixed-fibre muscle biopsy samples that fatigue during short-term electrically evoked isometric contraction (Hultman & Spriet, 1986), voluntary isometric contraction (Katz, Sahlin & Henriksson, 1986) and maximal voluntary dynamic exercise (Jones *et al.* 1985) may be due to a depletion of muscle PCr stores and a decline in the rate of anaerobic muscle glycogen utilization during contraction. The main aim of the present experiment was to quantify the contribution made by PCr and glycogen to ATP resynthesis during short-term maximal exercise in different muscle fibre types. Exercise resulted in a substantial decline in PCr and glycogen in type I fibres (59·1 ± 2·9 and 77 ± 14 mmol (kg DM)$^{-1}$, respectively), but the extent of

Table 4. Adenosine triphosphate (ATP), phosphocreatine (PCr) and glycogen contents measured in type I and II muscle fibres obtained immediately before and after 30 s maximal sprint exercise

	ATP content (mmol (kg DM)$^{-1}$)	PCr content (mmol (kg DM)$^{-1}$)	Glycogen content (mmol (kg DM)$^{-1}$)
Pre-exercise			
Type I	24·0 ± 0·8	71·3 ± 3·0	375 ± 25
Type II	24·0 ± 0·4	79·3 ± 2·7**	472 ± 35***
Post-exercise			
Type I	20·6 ± 0·5	12·2 ± 2·1	298 ± 30
Type II	19·0 ± 1·1	5·0 ± 1·3**	346 ± 27*

Values represent means ± S.E.M. Significant difference between fibre types: $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$; $n = 6$.

breakdown was a further 25 and 64 % greater in type II fibres. Tesch *et al.* (1989) demonstrated that substantial PCr hydrolysis occurs in both fibre types during maximal isokinetic knee extension, with a trend for depletion to be greater in type II fibres ($P > 0{\cdot}05$). The results of the present experiment are partly in agreement with these findings, but show clearly that PCr utilization was significantly greater in type II fibres during maximal sprint exercise ($P < 0{\cdot}001$). At the end of exercise, PCr was almost totally depleted in this fibre type, despite the pre-exercise concentration in this fibre type being 10 % higher than the corresponding value in type I fibres ($P < 0{\cdot}01$). This 25 % greater utilization of PCr by type II fibres during exercise fits well with the 26 % higher creatine kinase activity observed in human fibres of this type (Thorstensson *et al.* 1977). The greater extent of PCr degradation observed in type I and II fibres during the present study when compared with the study of Tesch *et al.* (1989) (43 and 57 mmol (kg DM)$^{-1}$, respectively) probably reflects the highly anaerobic nature of treadmill sprinting.

The similarity in the rates of single fibre glycogen degradation during sprinting and those observed during electrical stimulation with circulation occluded (Greenhaff, Soderlund, Ren & Hultman, 1993) suggests that glycogenolysis is maximally activated in both fibre types during sprinting. This is supported by the finding that the rates of glycogen utilization in type I and II fibres during sprinting were 55 and 5 % greater, respectively, than the highest *in vitro* phosphorylase activities measured in type I (1·66 mmol (kg DM)$^{-1}$ s^{-1}) and type II (4·02 mmol (kg DM)$^{-1}$ s^{-1}) fibres by Harris *et al.* (1976). Any suggestion that type I fibres were not maximally recruited during exercise in the present study is not supported by the marked changes observed in the ATP, PCr and glycogen stores of this fibre type. The high blood lactate concentration recorded immediately following exercise in the present experiment (Table 2) and the close similarity between the number of glucosyl units derived from glycogen degradation and the number present in muscle in the form of glycolytic intermediates or lactate (Table 3), clearly indicate that oxidative phosphorylation contributed very little to type I fibre ATP resynthesis during the 30 s of maximal sprinting.

Single muscle fibre glycogen utilization during maximal sprint exercise has been investigated recently using histochemical techniques (Friden *et al.* 1989; Vollestad *et al.* 1992). In the latter study, the authors calculated the rates of single fibre glycogen utilization during 30 s of cycling exercise from changes in optical density of stained sections. The pattern of single fibre glycogen utilization in this particular study was similar to that seen during the present experiment. However, the rates of glycogenolysis in type I (1·51 mmol (kg DM)$^{-1}$ s^{-1}) and type II (2·2 mmol (kg DM)$^{-1}$ s^{-1}) fibres were ~40 and 45 % lower, respectively, than the corresponding rates in the present study. The reasons for this disparity are unclear but may be related to the differences in the analytical techniques employed, i.e. histochemical as opposed to biochemical, or to differences in the exercise modes, i.e. bicycle exercise as opposed to sprint running. It is worth noting that the rate of glycogen degradation in type II fibres in the study of Vollestad *et al.* (1992) fell at the lower end of the range of *in vitro* phosphorylase activities (2·12–4·02 mmol (kg DM)$^{-1}$ s^{-1}) measured in this fibre type by Harris *et al.* (1976), suggesting that glycogenolysis may not be maximally activated in this fibre type during bicycle exercise at 90 r.p.m.

During the present experiment, a similar decline was observed in the ATP content of both fibre types during exercise (Table 4). When comparing individuals, no relationship was observed between the loss of single fibre ATP stores and the relative decline in power output during exercise. This is in agreement with the suggestion that the loss of muscle ATP stores *per se* in mixed-fibre tissue, or a particular muscle fibre type, is not directly related to the development of fatigue (Jansson, Dudley, Norman & Tesch, 1987), and supports findings indicating that the degradation of muscle ATP to IMP (inosine monophosphate) and ammonia serves primarily as a mechanism of maintaining muscle AMP and ADP levels at a minimum (Sahlin & Broberg, 1990). Unfortunately, single fibre contents of AMP, IMP and ammonia were not determined during the present experiment. However, a marked increase in plasma ammonia concentration was observed following exercise, which, in this situation, was probably mainly attributable to the deamination of AMP to IMP (Table 2).

A further interesting finding of the present experiment is that the metabolic response of type I skeletal muscle fibres during 30 s of maximal sprinting differs markedly from that observed during 64 s of intense intermittent electrical stimulation, involving 1·6 s of stimulation followed by 1·6 s of rest (Greenhaff *et al.* 1991; Soderlund *et al.* 1992). The rate of glycogenolysis observed in type I fibres during the present experiment was ~14 times greater than the negligible rate observed during electrical stimulation. The corresponding rate in type II fibres was only slightly increased, being ~1·2 times greater. The mechanisms responsible for the marked acceleration of type I fibre glycogenolysis during sprinting are not immediately clear. We would like to suggest that a limitation in muscle oxygen delivery during maximal dynamic exercise may be responsible. A reduction in oxygen availability would increase the anaerobic ATP turnover rate and availability of free AMP in type I fibres and thereby increase the activity of glycogen phosphorylase (Ren & Hultman, 1990; Sahlin, Gorski & Edstrom, 1990). This suggestion is supported by the similarity observed when comparing the rate of type I fibre glycogenolysis in the present experiment (2·57 ± 0·08 mmol (kg DM)$^{-1}$ s^{-1}) with the corresponding rate recorded after 30 s of intense electrical stimulation with the circulation occluded (2·05 ± 0·70 mmol (kg DM)$^{-1}$ s^{-1}; Greenhaff *et al.* 1993). It is

probable that the low rate of type I fibre glycogen degradation previously observed during intermittent electrical stimulation with the circulation intact (Greenhaff *et al.* 1991; Soderlund *et al.* 1992) can be attributed to oxygen delivery being sufficient during the 1·6 s of rest between each train of stimulation to accomplish ADP and creatine rephosphorylation in type I fibres. This suggestion can be evaluated by undertaking future electrical stimulation studies with a reduced recovery period between each bout of stimulation.

In conclusion, the results of the present experiment indicate that during 30 s of maximal treadmill sprinting in man the rates of PCr and glycogen degradation are markedly greater in type II muscle fibres compared with type I fibres, while the decline in ATP is similar between fibre types. However, compared with previous studies involving similar durations of near-maximal electrically evoked isometric contraction (Greenhaff *et al.* 1991; Hultman *et al.* 1991; Soderlund *et al.* 1992), isokinetic knee extension (Tesch *et al.* 1989) and cycling exercise (Vollestad *et al.* 1992), the rates of substrate utilization recorded in type I fibres are still extremely high, being close to the very rapid rates observed in this fibre type during intense contraction with limb blood flow occluded (Greenhaff *et al.* 1993).

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Received 11 June 1993; accepted 18 November 1993.