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Changes in performance, muscle metabolites, enzymes and fibre types after short sprint training

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Abstract In contrast to endurance training, little research has been carried out to investigate the effects of short (< 10 s) sprint training on performance, muscle metabolism and fibre types. Nine fit male subjects performed a mean of 16 outdoor sprint running training sessions over 6 weeks. Distances sprinted were 30–80 m at 90–100% maximum speed and between 20 and 40 sprints were performed in each session. Endurance (maximal oxygen consumption; $\dot{V}O_{2\,max}$), sprint (10 m and 40 m times), sustained sprint (supramaximal treadmill run) and repeated sprint (6 × 40 m sprints, 24 s recovery between each) performance tests were performed before and after training. Muscle biopsy samples (vastus lateralis) were also taken to examine changes in metabolites, enzyme activities and fibre types. After training, significant improvements were seen in 40 m time ($P < 0.01$), supramaximal treadmill run time ($P < 0.05$), repeated sprint performance ($P < 0.05$) and $\dot{V}O_{2\,max}$ ($P < 0.01$). Resting muscle concentrations of ATP and phosphocreatine did not change. Phosphorylase activity increased ($P < 0.025$), citrate synthase activity decreased ($P < 0.01$), but no significant changes were recorded in myokinase and phosphofructokinase activities. The proportion of type II muscle fibres increased significantly ($P < 0.05$). These results demonstrate that 6 weeks of short sprint training can improve endurance, sprint and repeated sprint ability in fit subjects. Increases in the proportion of type II muscle fibres are also possible with this type of training.

Key words Maximal intensity · Phosphagens · Type I and II muscle fibres · Maximal oxygen consumption · Enzyme activities

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Introduction

Short sprint training is utilised in the physical preparation of athletes for many sports, yet the physiological adaptations to such training, by which performance may be improved, are not well understood. Previous research has concentrated on aerobic training methods; the effects of shorter, high-intensity anaerobic training efforts are much less well known. In particular, the use of brief (< 10 s) sprint efforts as a training stimulus has not been well researched. Only Thorstensson et al. (1975) and, more recently, Esbjornsson et al. (1993), Hellsten-Westing et al. (1993) and Linossier et al. (1993), have exclusively utilised short (10 s or less) maximal sprints as the method of training. Longer-term (15–90 s) high-intensity training efforts have dominated previous anaerobic training studies, either exclusively (Ready et al. 1981; Roberts et al. 1982; Sharp et al. 1986; Jacobs et al. 1987; Bell and Wenger 1988) or in combination with short sprint efforts as part of a mixed training protocol (Houston and Thomson 1977; Nevill et al. 1989). To work with maximal intensity for 15 s or more is extremely demanding and a steep decline in power output will usually be evident over the duration of the sprint. Even in highly motivated subjects it is unlikely that maximum sprint efforts for these duration's could be maintained for many repetitions.

In contrast, brief (< 10 s) sprint efforts separated by relatively short recovery periods provide an exercise challenge where it is more feasible for maximal or near-maximal efforts to be performed on each training repetition (Balsom et al. 1992). By careful manipulation of exercise duration and exercise:recovery ratios, a large (20 or more) number of training repetitions is generally possible in a training session. In this way, short sprint training will provide a regular maximum power stimulus to the exercising muscles as opposed to the speed-endurance stimulus presented when training repetitions are 15 s or longer.

Therefore, the aim of this study was to examine the changes in performance relating to each of the three energy systems that could be achieved by 6 weeks of short (< 10 s) sprint running training. Tests of anaerobic power, anaerobic work capacity, repeat sprint ability and aerobic power, as well as muscle fibre type, enzyme activities and phosphagen storage levels were measured before and after the training period to explore further the nature of the adaptation to short sprint training.

Methods

Subjects

Nine healthy and active, but not well-trained males volunteered as subjects. Their mean (SD) values for age, height and body mass were 22 (2) years, 180.1 (3.7) cm and 77.3 (3.3) kg respectively. Each subject was informed of the purpose of the study prior to giving their written consent to participate in the study. The test procedures were approved by the Human Rights Committee of The University of Western Australia.

Performance tests

In the 10 days before and after the commencement and conclusion of the training period, subjects attended the laboratory or gymnasium on three different occasions (each separated by at least 2 days).

Sprint tests

On one day, after a thorough warm-up, subjects performed two maximal short sprint trials (each separated by at least 4 min) in which 10 m and 40 m were recorded (with the aid of photoelectric cells). A standing start was used, and subjects began to run on a signal from the experimenters. Approximately 10 min after completing the second trial the subjects performed a supramaximal treadmill run to exhaustion (14 km · h^{-1}, 20% gradient), with time recorded on a digital stopwatch. Microsamples (50 μl) of capillary blood were taken from a hyperaemised earlobe (Finalgon ointment) at 3, 6, 9 and 12 min after completing the supramaximal run, to determine peak blood lactate concentration [La_b^-]. These measurements were obtained via an oxidase assay performed on an Analox LM3 multichannel analyser (Analox Instruments, London, UK), which had a coefficient of variation on serial sampling of 1.5%.

Repeated sprint test

On another day, again after a thorough warm-up, the subjects performed a repeated sprint test (RST) which involved 6 × 40 m maximal sprints, departing every 30 s (Fitzsimons et al. 1993). Two sets of timing gates were used, working in opposite directions, so that subjects could start the next 40 m sprint from the end at which they finished the preceding sprint. Therefore, in the approximately 24 s of recovery allowed between each sprint subjects could walk slowly back to the next start point. The time taken to complete each 40 m sprint was recorded (with the aid of photoelectric cells) and 50 μl microsamples of capillary earlobe blood were taken at 3, 6 and 9 min following the last sprint, for determination of peak [La_b^-] (as described previously). Two performance indices were calculated from the RST scores. Overall test performance was scored as the total time taken to complete the six sprints, and a percentage decrement score across the six sprints was also calculated (see Fitzsimons et al. 1993).

Maximal oxygen consumption

On a 3rd day the subjects performed a progressive incremental (speed firstly, then grade) treadmill running test for the determination of maximal oxygen consumption ($\dot{V}O_{2max}$). The test commenced at 12 km · h^{-1} and zero grade; every 2 min the speed was increased by 1 km · h^{-1} until 14 km · h^{-1} was reached, after which the grade was increased by 2% per minute until volitional exhaustion. The duration of this test lasted between 5 and 10 min. Inspired volumes were measured by a Morgan Ventilometer Mark II 225A (P.K. Morgan, UK) and expired fractions of O_2 and CO_2 were continuously sampled from a 4-l mixing chamber and recorded every 30 s by Applied Electrochemistry S-3A O_2 and CD-3A CO_2 analysers, respectively. Gravimetrically determined gases of known concentration were used to calibrate the gas analysers before and after each test. The sum of the two highest consecutive 30 s oxygen consumption ($\dot{V}O_2$) values was taken as the subject's $\dot{V}O_{2max}$, providing that an R value of 1.15 or more and/or the presence of peaking over in the $\dot{V}O_2$ values had been achieved.

In all performance tests the subjects wore running shoes, and the 10 m and 40 m and RST trials were performed indoors in a gymnasium on a wooden floor. As much as possible, depending on subject and venue availability, the order of the tests was randomly assigned both before and after training.

Training program

The 6-week sprint interval training program used for all subjects is presented in Table 1. All distances sprinted (at either 90% or 100% of maximum speed) were between 30 and 80 m, with ≈90% of the training performed over 40–60 m. In the final 3 weeks of training all sprints were over 60 m or less. Progressive overload was applied by increasing the number of sprint repetitions in a session from an initial 22 to a high of 42 in two of the sessions in the 6th week, and by performing more sprint sets at 100% of maximum speed. A gradual reduction in the work:recovery ratio was also applied from an initial 1:6 down to 1:4 on certain sets. The recovery time between sets of sprints was 2–4 min with the longer recovery time allowed between sets of maximum effort. Slow walking and jogging was performed in the recovery periods between sets. When sprint sets at 90% of maximum speed were performed (see Table 1) the experimenter kept time on each sprint as a check on the required intensity. All training sessions were supervised by the same experimenter and were completed in small groups of three or four, except for two occasions when one subject trained without supervision. The training sessions were conducted outdoors on a grassed oval with the subjects wearing running shoes. Microsamples of capillary blood were taken before (post-warm-up), during (2 min after two and four sets), and after (2, 4 and 6 min after six sets) two training sessions (see Table 1; 3 subjects in session 17, 1 subject in session 18) and later analysed for [La_b^-] (as described previously) as an indicator of the training intensity.

Muscle biopsy sampling

Prior to warming up for the RST, both before and after the 6 weeks of training, muscle tissue samples (≈50 mg wet mass) were obtained

Table 1 The sprint training program, showing the number of repetitions and distances covered for each session. Five subjects completed 16 sessions while the other 4 subjects each completed 14, 15, 17 and 18 sessions, respectively. Each subject completed at least three sessions of 40–42 repetitions. Where a set of intervals are *underlined* those efforts were run at maximum speed. (*W*:*R*) work:recovery ratio)

Week	Session							% Maximum effort	W:R	Number of reps.
1	1	6 × 80	6 × 60	6 × 40	4 × 40			90	1:6	22
	2	6 × 80	6 × 60	6 × 40	4 × 40			90	1:6	22
	3	6 × 80	6 × 60	6 × 40	6 × 40			90	1:6	24
2	4	6 × 80	6 × 60	6 × 40	8 × 30			90	1:5	26
	5	6 × 80	6 × 60	6 × 40	8 × 30			90	1:5–6	26
	6	4 × 80	6 × 50	8 × 40	6 × 40	6 × 30		90/100	1:5–6	30
3	7	4 × 80	6 × 50	8 × 40	6 × 40	6 × 30		90/100	1:6	30
	8	8 × 30	6 × 50	8 × 30	6 × 40	6 × 30		90/100	1:5–6	34
	9	8 × 30	6 × 50	8 × 30	6 × 40	6 × 30		90/100	1:5–6	34
4	10	6 × 60	8 × 50	6 × 40	8 × 50	6 × 60		90/100	1:5	34
	11	6 × 60	8 × 50	6 × 40	8 × 50	6 × 60		90/100	1:6	34
	12[a]	6 × 60	8 × 50	6 × 40	6 × 40			90/100	1:5	24
5	13	8 × 50	8 × 40	8 × 40	8 × 40	8 × 50		90/100	1:5–6	40
	14	8 × 50	8 × 40	8 × 30	8 × 40	8 × 50		90/100	1:5–6	40
	15	8 × 50	8 × 40	8 × 30	8 × 40	8 × 50		90/100	1:4–6	40
6	16	8 × 50	8 × 40	8 × 30	6 × 50	6 × 40	6 × 30	90/100	1:4–6	42
	17	8 × 30	8 × 40	8 × 50	6 × 50	6 × 40	6 × 30	90/100	1:4–6	42
	18	8 × 30	8 × 40	6 × 50	6 × 50	6 × 40	6 × 30	90/100	1:4–6	40

[a] Very windy day–session reduced

from the vastus lateralis muscle. The percutaneous needle biopsy technique (Bergstrom 1962), with suction applied manually, was used to obtain the samples after first incising the skin and fascia under local anaesthesia (1% xylocaine). Two samples, with the needle angled differently on each occasion, were taken from each subject from a single incision. One sample was immediately immersed in liquid N_2 and later stored at −80°C before freeze-drying and enzymatic analysis of ATP and phosphocreatine (PCr) according to the method of Harris et al. (1974). The activity of phosphorylase (PHOS), phosphofructokinase (PFK) and citrate synthase (CS) was determined using the methods employed by Green et al. (1984). Myokinase (MK) activity was determined using the procedures of Costill et al. (1979). Muscle homogenate protein concentration was determined by the method of Lowry et al. (1951). The other muscle sample was prepared for histochemical analysis by first being placed on a piece of dental wax before being mounted onto cork and frozen in isopentane cooled in liquid N_2. These samples were also stored at −80°C until all post-training samples were taken, so that pre- and post-training samples for each subject could be stained as a batch to avoid possible errors due to differential pH effects during staining. Subsequently, 12-μm-thick sections were cut on a cryostat, then mounted onto clean slides before being stained for myofibrillar ATPase activity to identify type I and II fibres using a modification of the method of Dubowitz and Brooke (1973). A progressive count of type I and type II fibres was made using a Kontron Minimop (Carl Zeiss, Munich, Germany) computerised image analysis system. Subtypes (i.e. a or b) of the type II fibres could not be clearly identified using this system and therefore are not reported. The same experimenter counted all fibres for all subjects by tracing around the perimeter of each fibre on a projected image of the slide. The Minimop system then calculated both the number of fibres and fibre area. The software then converted the measured area into a circle, from which the diameter was recorded. A total of 150–280 (mean = 191) fibres was counted in determining the fibre types for each subject.

In some cases insufficient tissue was obtained from the biopsy sample to permit all assays to be done, therefore $n = <9$ for certain results.

Statistics

Paired *t*-tests were used to assess the significance of the differences between pre- and post-training mean values. Pearson product moment correlation coefficients were also calculated between certain measured variables. Statistical significance was accepted at the $P < 0.05$ level.

Results

Performance

Body mass was not significantly changed after the training period [mean (SE); 76.9 (1.6) kg pre-training to 77.6 (1.8) kg post-training]. Table 2 presents the pre- and post-training results for the 10 m and 40 m sprints, supramaximal treadmill run, 6 × 40 m RST and $\dot{V}O_{2max}$. Significant improvements were noted in the scores for all tests post-training, except for the 10 m time ($P = 0.12$).

Table 2 Performance test scores [mean (SE)] measured before and after training ($n = 9$ except for repeated sprint test where $n = 6$). (*RST* repeated sprint test, $\dot{V}O_{2\,max}$ maximal oxygen consumption)

	Pre-training	Post-training
10 m Time (s)	1.87 (0.02)	1.81 (0.03)
40 m Time (s)	5.50 (0.05)	5.37 (0.08)***
Supramaximal run (s)	49.9 (3.5)	55.5 (4.0)*
RST total time (s)	35.66 (0.65)	34.88 (0.49)*
RST % decrement	7.1 (2.6)	5.9 (1.2)
$\dot{V}O_{2\,max}$ ($l \cdot min^{-1}$)	4.40 (0.18)	4.67 (0.16)***
$\dot{V}O_{2\,max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	57.0 (2.4)	60.5 (1.9)***

*$P < 0.05$
***$P < 0.01$, significantly different from pre-training scores

Muscle metabolites and enzyme activities

The resting levels of ATP and PCr were unchanged after training (see Table 3). Enzyme activities were decreased (CS; 36%, $P < 0.01$), increased (PHOS: 40%, $P < 0.025$; MK: 19%, but not significant, $P = 0.28$), and unchanged (PFK; $P = 0.81$) by the end of the 6-week training period and did not correlate with any performance test variables either pre- or post-training.

Fibre types

The training induced a significant increase (≈10%, $P < 0.05$) in the percentage of type II fibres (% type II fibres) and the percentage area occupied by them (see Table 4). The % type II fibres was correlated with 40 m time ($n = 6$: $r = -0.82$; $P < 0.05$) and $\dot{V}O_{2max}$ ($n = 6$, $1 \cdot min^{-1}$: $r = -0.75$; $P < 0.05$) before training; after training stronger correlations were recorded (40 m; $r = -0.97$; $P < 0.01$; $\dot{V}O_{2max}$: $r = -0.83$; $P < 0.05$) and the 10 m time was also correlated with % type II fibres ($n = 6$: $r = -0.93$; $P < 0.01$). No association was found between % type II fibres and supramaximal treadmill run time before ($r = 0.20$) or after ($r = 0.22$) training. Correlations between performance on the RST and % type II fibres were not calculated due to incomplete results ($n = 4$; only for subjects having both pre- and post-training measures). Enzyme activities were not correlated with % type II fibres either before or after the training period.

Table 3 Resting muscle metabolite concentrations [mean (SE): $mmol \cdot kg^{-1}$ D^M and enzyme activities ($\bar{x} \pm SE$: $umol \cdot g^{-1} \cdot min^{-1}$ WW) recorded before and after training. (PCr phosphocreatine, MK myokinase, PHOS phosphorylase, PFK phosphofructokinase, CS citrate synthase)

	Pre-training	Post-training
[ATP] ($n = 7$)	26.1 (1.3)	25.7 (0.9)
[PCr] ($n = 9$)	80.5 (4.5)	81.2 (3.0)
MK ($n = 7$)	145.4 (9.7)	172.4 (16.2)
PHOS ($n = 9$)	15.1 (0.8)	21.3 (1.9)**
PFK ($n = 9$)	37.3 (3.1)	38.0 (2.1)
CS ($n = 7$)	10.0 (1.4)	6.8 (0.9)***

**$P < 0.025$, significantly different from pre-training values
***$P < 0.01$, significantly different from pre-training values

Table 4 Changes ($\bar{x} \pm$ SE) in percentage and area of type I and type II muscle fibres before and after training ($n = 7$)

	Pre-training	Post-training
% Type I	45.8 (6.9)	36.2 (4.3)*
% Type II	54.2 (6.9)	63.8 (4.3)*
$\bar{x}$ Diameter type I (μm)	50.7 (0.8)	52.7 (1.0)
$\bar{x}$ Diameter type II (μm)	57.9 (2.0)	57.8 (3.8)
% Area type I	43.0 (6.6)	34.4 (4.0)*
% Area type II	57.0 (6.6)	65.6 (4.0)*

*$P < 0.05$, significantly different from pre-training values

Peak blood lactate

No significant differences were recorded between the peak [La_b^-] measured before and after training following both the RST [$n = 8$: 16.4 (1.3) $mmol \cdot l^{-1}$ vs 14.0 (1.3) $mmol \cdot l^{-1}$; $P = 0.14$] and supramaximal treadmill run [$n = 7$: 15.9 (1.1) $mmol \cdot l^{-1}$ vs 16.0 (0.9) $mmol \cdot l^{-1}$; $P = 0.99$]. Peak [La_b^-] was also not correlated before or after training with either the total time on the RST ($n = 6$: $r = 0.44$ and $r = 0.74$) or the supramaximal treadmill run time ($n = 9$: $r = 0.64$ and $r = 0.01$).

The [La_b^-] recorded before, during and after training session 17 or 18 for four subjects demonstrated that the short sprint training resulted in a peak [La_b^-] of 10 $mmol \cdot l^{-1}$ (range: 10.2–13.9 $mmol \cdot l^{-1}$) or more in these sessions.

Discussion

The energetics of repeated short sprints requires some degree of ATP resynthesis from each of the energy systems (Balsom et al. 1992; Dawson et al. 1993; Bogdanis et al. 1996). Therefore, a wide range of performance and metabolic adaptations can be expected to result from such training, especially in subjects who are not well-trained either aerobically or anaerobically. Such was the case in the present study, where single (40 m) and repeated (6 × 40 m) short sprint performance, as well as supramaximal treadmill run time and $\dot{V}O_{2max}$ were improved after training, representing positive adaptations in all three of the energy systems. Underlying these improvements were, most importantly, an increase in % type II muscle fibres and some alterations in muscle enzyme activities.

The short sprint training used in this study was designed to provide a repeated near-maximal and/or maximal power stimulus to the active muscles, rather than a less intense speed-endurance stimulus (i.e. 15- to 90 s efforts) which has typified most of the previous studies on high-intensity training. Improvements in power after training were reflected in the improved 40 m and RST total times. Short sprint time and/or power improvements have also been reported to occur following training involving repeated 5 s sprints over 7–8 weeks (Thorstensson et al. 1975; Linossier et al. 1993).

Type II muscle fibres are more metabolically suited to sprint efforts by virtue of their having higher myosin ATPase activity than type I fibres (Essen et al. 1975). Their importance to short sprint performance is demonstrated by the higher correlations found between % type II fibres and 10 m and 40 m time after training (than before), when sprint times were faster. Increased enzyme activities can enhance the rate of ATP resynthesis (in particular MK and creatine phosphokinase [CPK]) and hydrolysis (Mg^{2+}-stimulated ATPase), and therefore sustain greater power. Only Thorstensson et al. (1975) have previously measured alactic enzyme activities before and after short sprint training. They found

increases in Mg^{2+}-stimulated ATPase, MK and CPK of 30%, 20% and 36%, respectively, after training. The 19% increase in MK activity found after training in the present study, although non-significant, is of the same relative magnitude as that recorded by Thorstensson et al. (1975). It is also possible that CPK activity, which was not measured here, may have increased after the training. Muscle fibre types that were identified by staining for myosin ATPase activity showed a 10% increase in type II fibres after training, which may indirectly reflect an increase in the levels of this enzyme.

In addition to these physiological changes, it is also possible that neural adaptations, which were not measured here, may have some contribution to the observed improvements in power (MacDougall et al. 1991). It is clear though that the power changes were not due to any increase in [ATP] and [PCr], as these were unchanged after training. This result confirms the findings of other sprint training studies (Thorstensson et al. 1975; Belcastro et al. 1981; Sharp et al. 1986; Linossier et al. 1993) which have also failed to show any increase in phosphagen storage after training. However, a faster rate of PCr resynthesis after training appeals as a more functionally significant adaptation than any absolute increase in PCr storage. In the RST used here the rate of PCr resynthesis in the ≈24 s between sprints would be an important factor in determining overall performance. Bogdanis et al. (1996) have recently shown that the rate of PCr resynthesis following a 30-s sprint is related to endurance fitness, and McCully et al. (1991) have demonstrated that this factor can be improved by endurance training. The effects of short sprint training on the rate of PCr repletion are unknown, but the endurance fitness ($\dot{V}O_{2max}$) of the subjects here was significantly increased after training, and Yoshida and Watari (1993) have shown PCr resynthesis to be faster in endurance-trained athletes. Therefore, the improved performance on the RST after training may have been at least partly assisted by the increases measured in aerobic fitness. Changes in performance on a RST after a program of short sprint training have not previously been reported and these results would have most relevance to multiple sprint team sports, which require repeated short maximal sprints over the course of a match.

The longer supramaximal treadmill run time recorded after training demonstrates that short sprint training can also improve anaerobic work capacity, which Linossier et al. (1993) have also reported (30 s Wingate test). The greater PHOS activity measured after training may have increased the ATP supply via glycolysis and hence produced a longer run time before exhaustion. Other possible reasons for the improved anaerobic work capacity found here after training may be a greater muscle buffering capacity, which has been demonstrated in repeated 20- to 30 s sprint training studies (Sharp et al. 1986; Bell and Wenger 1988) and the increased $\dot{V}O_{2max}$ recorded after training. While muscle buffering capacity was not measured here, increases in aerobic power may have led to a greater aerobic energy contribution to the supramaximal run. If this was the case, then a lesser reliance on glycolytic energy supply would have occurred, which may explain the lack of change in $[La_b^-]$ after the supramaximal run post-training, despite a greater time to exhaustion. The much weaker association between $[La_b^-]$ and supramaximal run time after training ($r = 0.01$) than before ($r = 0.64$) may also point to a greater aerobic energy contribution in the run post-training. Denis et al. (1992) have reported that the ability to sustain supramaximal work lasting 30–45 s was related more to muscle oxidative capacity than to glycolytic capacity and may explain the lack of association found in the present study before and after training between supramaximal run time and % type II fibres. Previous studies employing a training stimulus of exclusively <10 s sprints have not measured $[La_b^-]$ responses to a sustained sprint test after training. Typically, $[La_b^-]$ is increased post-exercise in training studies where longer duration sprints have been performed (Houston and Thompson 1977; Sharp et al. 1986; Jacobs et al. 1987). Often PFK activity is also increased by this training (Sharp et al. 1986; Jacobs et al. 1987); the lack of change in PFK activity recorded in the study reported here may also be a factor underlying the unchanged $[La_b^-]$ found after the supramaximal run.

An increase in $\dot{V}O_{2max}$ has been reported to occur after sprint training involving efforts of 15 s or more in duration (Ready et al. 1981; Sharp et al. 1986; Simoneau et al. 1987; Bell and Wenger 1988), but not after exclusively short sprint training (Thorstensson et al. 1975; Linossier et al. 1993). All subjects in the present study showed an increase in $\dot{V}O_{2max}$ after training, and their improvement in this variable may reflect their training status pre-training in that they were fit, but not well-trained aerobically. The large number of sprints performed during training would have indirectly placed a significant strain on the aerobic energy system, as oxidative metabolism is important in the recovery between sprints (Bogdanis et al. 1995, 1996).

The significant improvements in aerobic power recorded in the present study after training occurred, unexpectedly, in conjunction with a significant decrease in both CS activity and % type I fibres. With respect to CS activity, Linossier et al. (1993) reported no change in this enzyme after their repeated maximal 5 s sprint training regime, but their recorded activities pre- and post-training were no different to the values recorded after training here. It is possible that CS activity was not a limiting factor to $\dot{V}O_{2max}$ before training and that the decreases noted after training were not of sufficient magnitude to interfere with the maximal production of ATP via oxidative metabolism. The CS activities did not correlate with $\dot{V}O_{2max}$ either before or after training. An increase in % type II muscle fibres (with a corresponding decrease in % type I fibres) after 6 weeks of sprint training has also been reported by Jansson et al. (1990) and Esbjornsson et al. (1993). Their training involved repeated 10 s and 15- to 30 s sprints, respectively, and the subjects were similar to those used in the present

study (i.e. fit, but not well-trained). Andersen et al. (1994) reported similar findings for sprint athletes after a 12-week period of strength and interval running training. All of these studies found the training-induced increase to be in type IIa rather than type IIb fibres and, while fibre type sub-groups were not identified here, the same pattern of change is likely to have occurred in the present study. This may partly explain the improvements in anaerobic power and work capacity seen after training, which occurred in conjunction with an increase in $\dot{V}O_{2max}$. An increased frequency of stimulation of the muscle fibres (as provided by the repetitive nature of multiple sprints) may lead to a greater synthesis of fast myosin within the previously type I fibres. However, increased plasma catecholamine levels, which are known to result after repeated short sprints (Gaitanos et al. 1993), may also provide a stimulus for fibre type transformation (Jansson et al. 1990).

In contrast to these results, Linossier et al. (1993), who used repeated 5 s sprints in training, found, paradoxically, that there was an increase in % type I fibres after training, with a corresponding decrease in % type IIb fibres. They speculated that this response may reflect the role of the type I fibres in the recovery periods between the short sprints, where aerobic metabolism is important in the recovery of PCr stores and the removal of La^- from the muscle cells. In a more recent study, Linossier et al. (1997) confirmed a decrease in % type IIb fibres after 9 weeks of short sprint training, but found evidence of a corresponding increase in either type IIa or type I fibres within their subject group. Cadefau et al. (1990) and Simoneau et al. (1985) have also reported increased % type I fibres after sustained sprint training, with Simoneau et al. (1985) finding that there was a decrease in % type IIb fibres. The results are difficult to reconcile with those recorded in the present study and by Jansson et al. (1990) and Esbjornsson et al. (1993). While no control group was used here, sampling of muscle was performed pre- and post-training by the same person using identical procedures and, more importantly, all samples were stained as a batch to avoid any errors due to differential pH affects during staining. Six of the seven subjects who had fibre typing performed increased their % type II proportion after training; the remaining subject had an unchanged result. Therefore, we are confident that the changes in fibre type proportion observed here are due to the short sprint training performed and are not the result of variability within the procedures. Fibre type transformation resulting from various types of sprint training remains a fertile topic for further research.

In conclusion, the short sprint running training performed in this study with fit, but not well-trained, subjects over a 6-week period, produced improvements in single and repeated short sprint performance, prolonged time to exhaustion in a sustained sprint, and also increased endurance fitness. These performance changes occurred in association with an increase of approximately 10% in type II fibre proportion.

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