Effect of training on muscle metabolism during treadmill sprinting

MARY E. NEVILL, LESLIE H. BOOBIS, STEPHEN BROOKS, AND CLYDE WILLIAMS
Department of Physical Education and Sports Science, University of Technology,
Loughborough LE11 3TU, United Kingdom

Nevill, Mary E., Leslie H. Boobis, Stephen Brooks, and Clyde Williams. Effect of training on muscle metabolism during treadmill sprinting. J. Appl. Physiol. 67(6): 2376-2382, 1989.-Sixteen subjects volunteered for the study and were divided into a control (4 males and 4 females) and experimental group (4 males and 4 females, who undertook 8 wk of sprint training). All subjects completed a maximal 30-s sprint on a nonmotorized treadmill and a 2-min run on a motorized treadmill at a speed designed to elicit training). All subjects completed a maximal 30-s sprint on a nonmotorized treadmill and a 2-min run on a motorized treadmill at a speed designed to elicit 110% of maximum oxygen uptake (110% run) before and after the period of training. Muscle biopsies were taken from vastus lateralis at rest and immediately after exercise. The metabolic responses to the 110% run were unchanged over the 8-wk period. However, sprint training resulted in a 12% (P < 0.05) and 6% (NS) improvement in peak and mean power output, respectively, during the 30-s sprint test. This improvement in sprint performance was accompanied by an increase in the postexercise muscle lactate (86.0 ± 26.4 vs. 103.6 ± 24.6 mmol/kg dry wt, P < 0.05) and plasma norepinephrine concentrations (10.4 ± 5.4 vs. 12.1 ± 5.3 nmol/l, P < 0.05) and by a decrease in the postexercise blood pH (7.17 ± 0.11 vs. 7.09 ± 0.11, P < 0.05). There was, however, no change in skeletal muscle buffering capacity as measured by the homogenate technique (67.6 ± 6.5 vs. 71.2 ± 4.5 Slykes, NS).

advesine 5'-triphosphate; buffering; catecholamines; exercise; lactate; fatigue; pH

ALTHOUGH IT is generally accepted that short-term high-intensity training will result in small improvements in the ability to perform sprint exercise, the mechanism of adaptation is little understood. We have recently reported that for recreationally active subjects anaerobic glycolysis is a major contributor to ATP resynthesis during a 30-s treadmill sprint and that the associated decrease in muscle pH may contribute to the development of fatigue during this type of exercise (5, 6). The purpose of the present study was to examine these variables before and after sprint training in an attempt to further understand the mechanisms underlying training-induced improvements in the ability to perform maximal exercise.

METHODS

Sprinting was performed on a nonmotorized treadmill using procedures and equipment previously described (6, 18). A nonmotorized treadmill was interfaced to a microcomputer that allowed the continuous monitoring of the horizontally applied force and treadmill belt velocity throughout the period of the test. The instantaneous product of restraint force and belt velocity was used to determine the horizontal component of power during the test. Results were averaged over 1-s time intervals and displayed by the computer at the conclusion of the sprint. The maximal product of restraint force and belt speed will hereafter be referred to as "peak power output," while the integral of restraint force and belt speed throughout the time period of the test will be referred to as "mean power output." "Fatigue" will refer to the decrease in power output during the test and will be expressed as percent of the peak value.

Subjects. Eight male (M) and eight female (F) recreationally active runners gave their informed consent and volunteered to participate in this study, which was conducted in keeping with the principles embodied in the Declaration of Helsinki for experiments involving human subjects. The subjects were assigned to either an experimental (4 M, 4 F, ht 171.2 ± 7.6 cm, age 30.0 ± 7.6 yr, wt 65.9 ± 10.1 kg) or a control group (4 M, 4 F, ht 171.6 ± 11.6 cm, age 29.7 ± 12.5 yr, wt 66.2 ± 5.3 kg).

Protocol. All subjects were familiarized with treadmill sprinting several days before the experiment. After an overnight fast and standardized warm-up, each subject completed a maximal 30-s sprint on the nonmotorized treadmill. On separate days, a number of other tests were performed. Maximum oxygen uptake (VO2 max) was determined using a standard uphill treadmill running test (26). A 2-min run was also completed on a level treadmill at a speed designed to elicit 110% of uphill VO2 max (110% run). In addition, the subjects completed a track 200-m performance test during which the time taken to run the first 50 m and the time for the total distance were recorded. The posttraining testing commenced 3 days after the last training session. All tests were performed at approximately the same time of day and in the same order before and after training and were completed within 1 wk. The 110% run was performed at the same treadmill speed before and after training.

Training. The experimental and control groups were recreationally active (running ~20 miles/wk) before the study, and both groups maintained this prior activity during the study. In addition, the experimental group trained in the laboratory three to four times per week. The sessions were comprised of two 30-s maximal sprints 10 min apart (completed twice per week), six to ten 6-s sprints 54 s apart (completed once per week), and two to
five 2-min runs at 110% of $\dot{V}O_2_{max}$ 5 min apart (completed once per week).

**Blood sampling and treatment.** For both the 30-s sprint and the 110% run, venous blood samples were taken from an antecubital vein at rest and at 3 min after the run for the determination of blood pH and plasma catecholamines while capillary blood samples were taken after the warm-up and at 1 and 5 min after both tests for blood lactate and blood glucose determinations. Venous blood samples were placed in tubes containing lithium-heparin and blood pH was determined immediately (Radiometer PHM73 pH/blood gas monitor). The remaining blood (8–10 ml) was centrifuged, and the plasma was treated with 200 µl of a mixture of 100 mmol/l ethylene glycol-bis(β-aminoethyl ether)-N,N,N',N'-tetraacetic acid and 100 mmol/l glutathione. The treated plasma was frozen at −20°C and analyzed at a later date for epinephrine and norepinephrine using high-performance liquid chromatography with electrochemical detection (14). Capillary samples were deproteinized in 2.5% perchloric acid, stored at −20°C, and subsequently analyzed for blood lactate and blood glucose concentrations using the method described by Maughan (20).

**Muscle sampling and analysis.** All of the experimental group (n = 8) and six of the controls agreed to have muscle biopsies taken. However, full muscle metabolite data are presented for only six of the six experimental group because of the inadequate sample size obtained from the two remaining subjects. Needle-biopsy samples were taken under local anesthesia (1% plain lidocaine) from the vastus lateralis at rest and immediately after the 30-s sprint and the 110% run. The time taken from cessation of the sprint for the subjects to stop and for the biopsy sample to be taken averaged 8.7 ± 1.4 s. The samples were instantly immersed in liquid nitrogen, removed from the needle, and split into two pieces by a strong metal rod. One piece was stored in liquid nitrogen for subsequent determination of muscle pH and buffering capacity, and the other portion was freeze-dried for muscle metabolite analysis. The freeze-dried sample was dissected free of connective tissue and blood and powdered, and the fat was removed by ether extraction. An acid extract of the muscle was obtained (16), and the neutralized extract was assayed enzymatically for creatine, phosphocreatine (PCr), ATP, ADP, AMP, glucose 1-phosphate (G1P), glucose 6-phosphate (G6P), fructose 6-phosphate (F6P), fructose 1,6-diphosphate (FBP), triose phosphates (1P), pyruvate, and lactate by fluorometric analyses (19). Glycogen was determined both on the neutralized extract and on the muscle pellet left after the extraction procedure, giving an acid-soluble and acid-insoluble fraction. Muscle metabolite concentrations are expressed with respect to dry weight.

Samples for the muscle pH and buffering capacity determinations were weighed while frozen and homogenized in a mixture of 145 mmol/l KCl, 10 mmol/l NaCl, and 5 mmol/l iodoacetic acid, using a dilution ratio of 25 µl of homogenizing solution per milligram wet weight muscle. Muscle pH was determined as previously described (10, 21). Muscle buffering capacity was determined by adjusting a 100-µl volume of the homogenate to a common starting pH of 7.2 and titrating the sample to 6.2 and back to 7.2 with 0.01 M HCl and 0.01 M NaOH. Using animal muscle, we had found that although the pH of muscle at different dilutions (10–50 µl) remains constant, buffering capacity increased with increasing dilution (70 ± 3, 78 ± 3, 88 ± 3, and 101 ± 12 Slykes at dilutions of 10, 15, 25, and 50 µl/mg, respectively, unpublished observations). Thus care must be taken when comparing buffer values between studies using different dilution ratios.

**Statistical analyses.** Two and three way analyses of variance were used to evaluate the training response. Probabilities are shown in the text and on tables only where there was a significant (P < 0.05) group-by-training or a group-by-exercise-by-training interaction. A Pearson product moment correlation was used to examine the relationship between variables. Means are presented with standard deviations.

**RESULTS**

**Performance changes.** As a result of sprint training, peak power output during the 30-s sprint was increased by 12% [experimental group (Ex) 606 ± 36 vs. 681 ± 134 W, control group (Con), 576 ± 112 vs. 565 ± 93, P < 0.05], and mean power output was increased by 6% (Ex 378 ± 76 vs. 399 ± 72 W, Con 367 ± 60 vs. 383 ± 61; NS). However, end power output was similar before and after training, leading to a change in the power profile as shown in Fig. 1. Thus the decline in power output tended to be greater after training (Ex 49.7 ± 6.3 vs. 60.5 ± 6.7%, Con 50.3 ± 19.7 vs. 48.6 ± 12.6; NS). The experimental group also improved their track 50- and 200-m times by 0.17 s (Ex 7.72 ± 0.91 vs. 7.55 ± 0.77 s, Con 7.46 ± 0.38 vs. 7.67 ± 0.46; P < 0.05) and 1.46 s (Ex 31.11 ± 4.88 vs. 29.65 ± 4.26 s, Con 29.34 ± 1.65 vs. 29.37 ± 1.86; P < 0.01), respectively.

**Muscle metabolites.** The resting and postexercise muscle metabolite concentrations for the experimental and control groups are shown in Tables 1 and 2. Total creatine was constant at −114 mmol/kg dry wt before and after exercise and before and after training. Both the 30-s sprint and the 110% run resulted in decreases in PCr.
bic glycolysis was increased by 20% (Ex 4.23 t 1.35 vs. 4.08 t 1.12; P < 0.05). Before training, a significant change in muscle metabolites as previously described (17, 21).

Values are means ± SD from 7 experimental subjects. * P < 0.05, significant difference between experimental and control groups in response to exercise after training (group X exercise interaction).

Muscle and blood pH. Muscle pH at rest and after the 30-s treadmill sprint and a 2-min run at 110% of Vo_2max pre- and posttraining

<table>
<thead>
<tr>
<th>n</th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-s Sprint</td>
<td>6</td>
<td>7.0 ± 0.02</td>
</tr>
<tr>
<td>Control</td>
<td>6</td>
<td>7.0 ± 0.05</td>
</tr>
<tr>
<td>110% Run</td>
<td>6</td>
<td>7.0 ± 0.04</td>
</tr>
<tr>
<td>Control</td>
<td>6</td>
<td>7.0 ± 0.03</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. * P < 0.05, significant difference between experimental and control groups in response to exercise after training (group X exercise X training interaction).

and peak power output in relation to body weight and the resynthesis of ATP from anaerobic sources (r = –0.70, P < 0.01 and r = 0.65, P < 0.05, respectively; n = 13).

Muscle and blood pH. Muscle pH at rest and after exercise for the 30-s sprint and the 110% run is shown in Table 3. For both the experimental and control group the decrease in muscle pH during the 30-s sprint (Ex 0.24 ± 0.05, Con 0.20 ± 0.07) was approximately twice that for the 110% run (Ex 0.13 ± 0.06, Con 0.10 ± 0.06), and no additional changes in muscle pH could be detected as a result of training. However, there was a further decrease in blood pH after the 30-s sprint after training (Table 4). Before training, those individuals with the largest changes in blood pH also had the highest peak power output in relation to body weight during the 30-s sprint (r = 0.70, n = 13, P < 0.01).

Muscle buffering capacity. Buffering capacity, as deter-
minded by the homogenate technique, was unchanged as a result of training (Ex 67.6 ± 6.5 vs. 71.2 ± 4.5 Slykes, n = 8, Con 63.8 ± 4.0 vs. 64.9 ± 4.5; n = 6). However, when buffering capacity was calculated from the changes in pH and lactate during the 30-s sprint, a higher value was obtained than that determined by the homogenate technique, and there was a tendency for the value to be increased after training in the experimental group [Ex 87.9 ± 24.7 vs. 126.7 ± 38.9 Slykes (n = 4), Con 138.8 ± 68.1 vs. 113.3 ± 18.8 (n = 6); NS]. Before training, those individuals with the highest buffering capacity, as determined by the homogenate technique, also had the fastest 200-m times, the highest peak power output, and the largest increases in muscle lactate during the treadmill sprint (r = -0.57, P < 0.05; r = 0.69, P < 0.01; and r = 0.67, P < 0.01, respectively; n = 13).

Blood lactate and blood glucose responses. Although the increase in blood lactate concentration was slightly greater after the 30-s sprint (5 min post-Ex 13.0 ± 2.9 vs. 13.7 ± 3.3 mmol/l, Con 12.8 ± 1.3 vs. 12.6 ± 1.3; NS) and slightly less after the 110% run (Ex 8.1 ± 3.2 vs. 6.8 ± 3.2 mmol/l, Con 9.7 ± 2.0 vs. 9.1 ± 2.6; NS) for the experimental group after training, there was no statistically significant difference at 1, 3, or 5 min postexercise after both tests. Similarly, there was no further increase in blood glucose concentration after the 30-s sprint as a result of training at 1, 3, or 5 min postexercise (5 min post-Ex 1.6 ± 0.7 vs. 1.8 ± 0.7 mmol/l, Con 1.3 ± 0.3 vs. 1.2 ± 0.2; NS). However, the increase in blood glucose concentration after the 110% run was lessened as a result of training at 1 and 3, but not at 5 min postexercise (1 min post-Ex 1.3 ± 0.4 vs. 1.0 ± 0.5 mmol/l, Con 1.0 ± 0.4 vs. 1.4 ± 0.4; P < 0.05; 3 min post-Ex 1.4 ± 0.6 vs. 1.0 ± 0.5 mmol/l, Con 1.5 ± 0.4 vs. 1.6 ± 0.5; P < 0.05). Before training, those individuals with the largest increases in blood lactate concentration as a result of the 30 s sprint also had the largest decreases in ATP and increases in muscle lactate (r = 0.69, P < 0.01 and r = 0.73, P < 0.01, respectively; n = 13).

Plasma catecholamines. Plasma catecholamine concentrations before and after the 30-s sprint and 110% run before and after training are shown in Table 5. Norepinephrine was increased by a further 20% after the 30-s sprint after training (P < 0.05). Before training, those individuals with the greatest changes in norepinephrine during the 30-s sprint also had the highest peak power output (r = 0.67, P < 0.01; n = 13), whereas those with the highest plasma epinephrine concentrations after the 30-s sprint also had the highest buffering capacity (r = 0.83, P < 0.01; n = 13), fastest 50-m speed (r = -0.75, P < 0.01), and highest rates of ATP resynthesis from anaerobic sources (r = 0.75, P < 0.01; n = 13).

\[ \text{VO}_2\text{max} \]

\[ \text{VO}_2\text{max} \] was slightly increased by sprint training (Ex 52.6 ± 10.9 vs. 53.5 ± 10.1 ml·kg\(^{-1}\)·min\(^{-1}\), Con 56.7 ± 6.2 vs. 55.4 ± 7.9; P < 0.05). Run time during the uphill running test to determine \( \text{VO}_2\text{max} \) was increased by ~1 min, i.e., by 8.6% when expressed as percent of the pretraining run time, but this increase did not reach statistical significance (Ex 8.9 ± 1.2 vs. 9.7 ± 1.8 min, Con 8.9 ± 1.2 vs. 8.7 ± 0.9; NS).

Oxygen uptake during treadmill sprinting and recovery. There was no change in oxygen uptake during the 30-s sprint (Ex 15.5 ± 3.3 vs. 15.7 ± 3.9 ml/kg, Con 15.4 ± 2.8 vs. 14.9 ± 2.2; NS) or 110% run (Ex 69.2 ± 18.7 vs. 68.0 ± 17.8 ml/kg, Con 72.3 ± 12.4 vs. 72.1 ± 9.9; NS) as a result of sprint training. For the experimental group the oxygen debt (defined for the purposes of this study as the elevation in oxygen consumption above post-warm-up values after exercise) was increased by 18.4% after the 30-s sprint after training (Ex 55.8 ± 16.4 vs. 66.5 ± 21.3 ml/kg, Con 49.9 ± 12.8 vs. 55.4 ± 17.8; NS), whereas the oxygen debt after the 110% run was unchanged (Ex 49.5 ± 15.6 vs. 45.4 ± 17.8 ml/kg, Con 52.6 ± 14.2 vs. 53.8 ± 17.3; NS). For the experimental group the actual oxygen uptake achieved during the last 30 s of the 110% run was 91.9 ± 5.3 and 88.3 ± 4.4% of \( \text{VO}_2\text{max} \) before and after training, respectively. For the control group the respective values were 85.4 ± 5.1 and 89.1 ± 3.1% of \( \text{VO}_2\text{max} \) on the two testing occasions.

Before training a large oxygen debt after the 30-s sprint was associated with high muscle (r = 0.71, P < 0.01; n = 13) and blood lactate concentrations (r = 0.72, P < 0.01; n = 13) and with high peak (r = 0.60, P < 0.05; n = 13) and mean (r = 0.76, P < 0.01; n = 13) power

### Table 4. Blood pH before and after a 30-s treadmill sprint and a 2 min run at 110% of \( \text{VO}_2\text{max} \) pre- and posttraining

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Pretraining 30-s Sprint</td>
<td>Experimental 7.38±0.02</td>
<td>7.17±0.11</td>
</tr>
<tr>
<td></td>
<td>Control 7.38±0.02</td>
<td>7.15±0.07</td>
</tr>
<tr>
<td>110% Run</td>
<td>Experimental 7.39±0.02</td>
<td>7.25±0.06</td>
</tr>
<tr>
<td></td>
<td>Control 7.37±0.02</td>
<td>7.18±0.09</td>
</tr>
</tbody>
</table>

### Table 5. Plasma epinephrine and norepinephrine concentrations before and after a 30-s treadmill sprint and a 2 min run at 110% of \( \text{VO}_2\text{max} \) pre- and posttraining

<table>
<thead>
<tr>
<th></th>
<th>Pretraining</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Pretraining 30-s Sprint</td>
<td>Epinephrine 0.4±0.2</td>
<td>1.5±0.6</td>
</tr>
<tr>
<td></td>
<td>Control 0.4±0.2</td>
<td>1.4±0.4</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Experimental 2.4±1.0</td>
<td>10.4±6.4</td>
</tr>
<tr>
<td></td>
<td>Control 2.0±0.8</td>
<td>11.8±7.8</td>
</tr>
<tr>
<td>110% Run</td>
<td>Epinephrine 0.5±0.2</td>
<td>1.4±0.6</td>
</tr>
<tr>
<td></td>
<td>Control 0.8±0.2</td>
<td>1.5±0.4</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Experimental 1.9±0.5</td>
<td>6.4±2.2</td>
</tr>
<tr>
<td></td>
<td>Control 1.7±1.1</td>
<td>3.5±3.5</td>
</tr>
</tbody>
</table>

Values are means ± SD from 8 experimental and 8 control subjects. *P < 0.05, significant difference between experimental and control groups in response to exercise after training (group × exercise × training interaction).
output in relation to body weight during the treadmill sprint. The total oxygen cost (oxygen uptake during the sprint plus the oxygen debt) of the 30-s sprint was most highly correlated with mean power output in relation to body weight \((r = 0.82, P < 0.01; n = 13)\). Oxygen debt after the 110% run was not associated with the increase in muscle lactate \((r = -0.24; NS)\) but was related to the changes in blood pH \((r = 0.68, P < 0.01; n = 13)\) and blood lactate concentration \((r = 0.69, P < 0.01; n = 13)\) and to the treadmill speed \((r = 0.71, P < 0.01; n = 13)\).

**DISCUSSION**

The purpose of this study was to identify the metabolic changes that accompany training-induced improvements in sprint running performance. In the light of these observations, we have attempted to describe the mechanism of the adaptation to training, and in doing so, to understand more clearly the metabolic events that underlie performance during high-intensity exercise.

Short-term sprint or interval training has previously been shown to result in improvements in performance during sprint tests of between 5 and 28% \((15, 23)\). The 6–12% improvement in power output during treadmill sprinting in the present study falls within this range. Because a significant relationship was found between ATP resynthesis from anaerobic sources and performance during treadmill sprinting in the present study, it would be reasonable to expect the improvement in sprint running performance to be accompanied by an increased contribution from anaerobic metabolism to ATP resynthesis. One possible mechanism by which an increase in ATP resynthesis from anaerobic metabolism could be achieved is by an increase in the resting muscle content of PCr and ATP, as has been found previously after general conditioning \((13)\). However, in the present study, both the resting concentration and the degradation of PCr and ATP were unchanged by training. Furthermore, total creatine was unchanged both before and after exercise and before and after training, which indicates that a common reference base was achieved for comparison of the pre- and postexercise and pre- and postraining samples. However, these findings do not rule out the possibility that the rate of PCr degradation was greater in the first few seconds of the sprint test after training, which would contribute both to the higher peak power output and to the tendency for a greater decrease in power output found during the 30-s test after training (Fig. 1).

However, the major metabolic change accompanying the training-induced improvement in sprint running performance was a 20% increase in the ATP resynthesized from anaerobic glycolysis during the 30-s sprint. It has previously been shown that sprint-trained athletes tend to have higher muscle and blood lactate concentrations after high-intensity exercise than endurance-trained athletes \((7, 23)\), which has been attributed, in part, to the higher proportion of type II fibers in the sprint-trained athletes \((9)\). However, in this study sprint training resulted in an increase in muscle lactate content during sprinting, when it is unlikely that any changes in fiber type had occurred. In earlier sprint-training studies a 7% (NS) increase in muscle lactate content was found during a 30-s cycle ergometer sprint test, and a marked increase in the blood lactate concentration was found after a 45-s isokinetic bike test after training \((4, 23)\). The results of these studies may suggest that the supply of energy from anaerobic glycolysis was limiting to performance before training. If this were the case a possible mechanism of adaptation would be an increase in phosphofructokinase (PFK) activity, because the activity of this enzyme is known to be high in sprint-trained compared with endurance-trained or untrained individuals \((9)\) and has been shown to increase with strength and sprint training \((8, 23)\). However, if the activity of PFK had been rate-limiting before training, a reduced accumulation of the glycolytic intermediates would be expected after training, but the ratio of G6P to lactate remained constant before and after training. One could claim that the activity of phosphorylase had increased in parallel with the activity of PFK, but with such a large accumulation of G6P both before and after training, it seems unlikely that the activity of phosphorylase could have been limiting. In addition, the activity of phosphorylase has been shown to be unaltered by sprint training in humans \((23)\). Thus it is possible, but by no means certain, that a greater contribution from anaerobic glycolysis to energy supply during the sprint after training was the result of an increase in PFK activity.

A further and favored hypothesis of the mechanism of the increased ATP resynthesis rate observed in this study is that the buffering capacity of skeletal muscle is improved, allowing anaerobic glycolysis to make a greater contribution to energy supply for the same change in muscle pH. In the present study muscle lactate was increased, but no changes could be detected in muscle pH, resulting in a tendency \((n = 4\) only, NS) for an improvement in buffering capacity as determined by calculation, as has been previously reported after sprint-cycling training in humans \((23)\). However, buffering capacity as determined by the homogenate technique was unchanged by training. A possible explanation for these findings lies in the fact that buffering capacity as determined by the homogenate technique takes no account of transmembrane fluxes of ions. It is possible that the production of more H⁺, per se, may cause a change in membrane characteristics, such as a decrease in membrane potential, that allows more H⁺ to leave the cell \((21)\), while the efflux of lactate may occur at a slower rate \((3)\). Such a decrease in membrane potential has been recorded during high-intensity cycling exercise \((24)\). The greater decrease in blood pH found after the 30-s sprint after training would suggest that H⁺ efflux may have been enhanced by sprint training. The generally higher buffer capacity for all subjects, when determined by calculation, may additionally reflect contamination of the postexercise biopsy samples with blood, even though every effort was made to select a relatively blood-free portion of the sample. However, these individuals with a high buffering capacity, as measured by the homogenate technique, also had the highest power output and the largest increases in muscle lactate during the 30-s sprint, suggesting that either buffering capacity may be im-
proved by longer-term training or that individuals possessing a high buffering capacity and the capacity to generate lactate during sprinting have a third variable in common, such as a high proportion of type II fibers. Finally it should be noted that the coefficient of variation for the determination of buffering capacity using the homogenate technique was 7%, which may mean that small but significant improvements in buffering capacity were obscured.

Perhaps the most likely explanation of the increased contribution from anaerobic glycolysis to energy supply during sprinting after training is that the improvement in performance itself demanded an increased rate of ATP resynthesis. This line of argument demands that the rate of ATP resynthesis from anaerobic glycolysis can be dictated by contractions per se. Traditionally, the rate of energy supply in the cell has been considered to be determined by the energy charge or phosphate potential of that cell (1). However, in this study the relationship between ATP, ADP, and AMP was the same before and after training, while the rate of ATP resynthesis was higher, although it is recognized that changes may have occurred in ADP and AMP that were too small to detect by chemical methods. Evidence that contractions per se may determine the glycolytic rate has been presented by Dawson (12), who reported that, in human muscle during ischemic conditions and during contractions, there was a similar change in ATP, ADP, and AMP but that the contractions produced a several hundredfold greater increase in the glycolytic rate. However, if the argument is accepted that the improved performance demanded an increased rate of ATP resynthesis, which was provided by an increase in the glycolytic rate, then questions concerning the cause of the improvement in performance must be addressed.

An attractive suggestion for the mechanism of the improved performance is an increased recruitment of motor units after training. Alternatively, there may have been hypertrophy or a change in the metabolic characteristics of existing fibers, both of which could result in greater tension development. The marked increase in the blood lactate and norepinephrine concentration and the greater decrease in blood pH after training could equally reflect any one of the above-mentioned adaptations. However, if increased recruitment of motor units was the explanation of the improved performance, as well as the increased muscle lactate concentration found in this study, one would expect to see a further decrease in the post sprint muscle PCr and ATP concentrations, because more of the fibers within the biopsy sample would have been active after training. Because the postexercise PCr and ATP were unchanged after training, these findings do not support the possibility that an increased recruitment of more fibers from a given area contributes to the training-induced improvements in sprint performance. Increased recruitment from an increased muscle mass after training is a possibility, but anthropometric measurements made in a previous study in humans (28) have shown unchanged lean leg volumes and cross-sectional areas after sprint training. Also it has been reported that the muscle mass of rats is unchanged by sprint training (11). It is also possible that the improved performance was contributed to by a more active involvement of other primary and synergistic muscle groups not examined in this study. However, such suggestions do not begin to explain the finding of increased muscle lactate in the vastus lateralis observed in this study. Therefore the results of this study would suggest that the increased glycolytic rate after training must be attributed to a change in the metabolic characteristics of individual muscle fibers recruited.

Such metabolic changes within a muscle fiber may obviously relate to the provision of energy during maximal exercise, but equally important, they may reflect changes in the contractile properties of muscle. One explanation for the improvement in power output could be that more contractions occurred during the 30-s time period of the test after training, which would be facilitated by a decrease in the time to peak tension of fibers after training. A 14% decrease in the time to peak tension of rat soleus muscle has been found after sprint training (25). A prime determinant of this contractile characteristic of muscle is the myofibrillar adenosinetriphosphatase activity. The activity of myofibrillar ATPase has been shown to increase 30-34% in response to sprint-cycling and sprint-running training in humans (2, 27). Furthermore the increase in adenylate kinase identified after strength training in humans (8) may also facilitate an enhanced activity of myofibrillar ATPase by hastening the removal of ADP from the contraction site, which would otherwise inhibit ATP utilization by product inhibition. The mechanism by which contractions during training are translated into expression of the gene for the appropriate protein synthesis is at present unknown.

In summary it is suggested that sprint training resulted in a change in some aspect of the contractile characteristics of the involved muscle groups which improved sprint-running performance. Thus a higher rate of ATP resynthesis was demanded during the sprint. Because the rate of ATP resynthesis from PCr is limited by its concentration in muscle, which was unaltered by training, and oxygen uptake during the sprint was unchanged by training, the only available means by which ATP resynthesis could be marked increased was by an enhanced flux through anaerobic glycolysis. This increase in ATP resynthesis from anaerobic glycolysis may have been facilitated by an increase in the activity of PFK and by an increased efflux of H+ from the muscle cell after training.

The authors thank Dr. A. M. Nevill for statistical advice, Dr. H. K. A. Lakomy for instrumentation of the nonmotorized treadmill, the Sports Science Research Group at Loughborough University for their assistance on experimental days, and the subjects for their tremendous commitment to this demanding study.

L. H. Boobis is a Porritt Fellow of the Royal College of Surgeons of England, which provided financial support for this study.

Address for reprint requests: M. E. Nevill, Dept. P.E. and Sports Science, University of Technology, Ashby Road, Loughborough, Leicestershire LE11 3TU, UK.

Received 30 March 1988; accepted in final form 1 August 1989.
REFERENCES