Training induced physiological and metabolic changes associated with improvements in running performance

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The purpose of the present study was to examine the relationship between improvements in running performance and some of the prominent physiological and metabolic adaptations to endurance exercise training. Twelve male undergraduates agreed to participate in this study (trial group), the aged matched physical education students provided a control group. Running performance, assessed as a five km time trial, improved from 19.69±2.24 to 19.22±2.03 min in the trial group (P < 0.01) after training. Maximal oxygen uptake values increased from 56.0±6.1 to 60.7±5.4 ml.kg⁻¹.min⁻¹; the running speed equivalent to a blood lactate reference concentration of 4 mmol.l⁻¹ (V₄ mM) increased from 3.79±0.77 to 4.04±0.71 m.s⁻¹, and the rate of oxygen consumption at 3.58 m.s⁻¹ (running economy) increased from 43.3±3.2 to 45.0±3.4 ml.kg⁻¹.min⁻¹ (P < 0.01).

The control group did not show any significant changes. The improved five km times in the trial group were significantly correlated (r = −0.71; P < 0.01) with changes in the running economy rather than changes in the VO₂ max (r = −0.07; ns), or V₄ mM (r = −0.13; ns) suggesting the increased rate of oxygen utilization reflected a greater oxidative degradation of metabolic substrates together with a slower rate of lactate production.

Keywords: Endurance exercise, training, five km times, running economy, blood lactate

Introduction

The effective transport to, and uptake of oxygen by, exercising skeletal muscle is crucial to endurance exercise performance. Physical training leads to an increased capacity for the transport of oxygen by the circulatory system¹ and improved performance times in both men and women²,³. Endurance exercise training in humans⁴,⁵ and in animals⁶ leads to modest improvements in maximal oxygen uptake (VO₂ max) but large increases in the capacity to sustain submaximal exercise to exhaustion. These large increases in submaximal endurance capacity is thought to be closely related to the increases in muscle oxidative capacity⁶.

Variations in the activity of oxidative enzymes, rather than small changes the maximal oxygen uptake, have been suggested as being responsible for changes in the running performance of well-trained endurance runners⁷ and may influence substrate utilization during submaximal exercise⁸,⁹. Evidence from animal studies suggests training frequency¹⁰, rather than training intensity¹¹ is responsible for the increase in mitochondrial enzyme concentration and endurance exercise capacity.

Running at faster speeds demands the utilization of a greater proportion of the maximal oxygen uptake (% VO₂ max) and an increased contribution to the energy supply from anaerobic metabolism. The end result is that lactate accumulates in the blood in increasing concentrations. Strong correlations have been reported between the onset of plasma or blood lactate accumulation and distance running performance¹²-¹⁶. Similarly, it is well documented that endurance training results in lowered blood lactate concentrations at the same absolute and relative exercise intensity after training which suggests an increased proportion of the energy supply derived from fat metabolism¹⁷,¹⁸ or greater aerobic catabolism of substrates per se.

Many studies report improvements in the VO₂ max values of their subjects as a result of physical training¹⁹,²⁰. These increases in VO₂ max are generally assumed to be associated with improvements in running performance¹. But how changes in the oxygen cost of submaximal exercise may relate to changes in distance running performance is more difficult to determine²,²¹.

The purpose of the present study was to assess the influence of short term steady-state endurance training on five km performance times in recreationally active men. Some of the more prominent physiological and metabolic characteristics were measured during maximal and submaximal exercise in order to examine the relationship between changes in these functional characteristics and changes in their running performance.

Methods

Twelve male undergraduates, active in recreational sports, agreed to undertake five weeks additional en-
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Endurance training. Measurements of their physiological and metabolic responses to maximal and submaximal exercise were made under controlled laboratory conditions. Their running performance was assessed by their times over a five km distance. This five km time trial consisted of five laps of a measured one km loop on level roads.

The subjects who agreed to undertake the additional endurance training are referred to henceforth as the training group (TG). A control group (CG) of physically active students (n = 7) was reduced to four because of injury and non-compliance with the requirements of this study. The remaining subjects in the control group completed all laboratory and field tests and their results have been included for comparative purposes. All subjects gave informed consent after being fully aquainted with the requirements of this study and the experimental protocol used during laboratory based treadmill exercise.

The mode of endurance training used in this study was outdoor running which was best described as steady state or aerobic exercise. The intensity and frequency of training during the five week period were freely chosen. The subjects' aim in training was to increase the duration of continuous running. They were encouraged to train a minimum of three times per week and to record their training in a daily diary.

Maximal oxygen consumption was determined directly using a motor-driven treadmill (Quinton, 24–72) using a modification of the procedure described by Taylor and co-workers. The speed of the treadmill was adjusted on the basis of familiarization runs and stayed constant throughout the test. The elevation of the treadmill increased 2.5 per cent every three minutes from an initial inclination of 3.5 per cent. Expired air samples were taken during the third minute of each exercise period until the subject indicated he could only maintain the exercise intensity for a further one minute, when a final one minute sample of expired air was taken.

Determinations of oxygen consumption (\(\dot{VO}_2\)) and carbon dioxide production (\(\dot{VCO}_2\)) were made using procedures previously described by Williams and Nute. A 16 minute run on a level treadmill was performed by each subject at a subsequent occasion to determine the oxygen cost of submaximal running. This test was continuous and a sample of the subject's expired air was taken during minute three to four for analysis of \(\dot{VO}_2\) and \(\dot{VCO}_2\). The subjects' heart rates were continuously measured on an oscilloscope (Cambridge Instruments Ltd.) during each laboratory test using suitably placed chest electrodes.

From the relationship between running velocity and oxygen consumption individual treadmill running speeds representing 60, 70, 80 and 90 per cent of the subject's \(\dot{VO}_2\) max were calculated. The subjects returned to the laboratory after a rest day and overnight fast to run at these speeds during a continuous 16 minute test. A sample of the subject's expired air was collected during the fourth minute of each exercise period together with duplicate samples (25 s) of arterialized capillary blood taken from the finger. These samples were deproteinized and later assayed for the determination of blood lactic acid. The values for blood lactic acid presented in this paper are delta values, i.e. exercise minus resting values.

The oxygen cost of running at a fixed or reference speed has been used in many previous studies, either to further characterize the subjects or investigate its validity as a physiological determinant of running performance. In the present study the oxygen cost of running at 3.58 m.s\(^{-1}\) was compared before and after training using calculated values from a regression equation relating oxygen consumption and running velocity for each subject. This velocity was chosen because it represented a mid-point in the running speeds for the subjects in this study and because the same velocity had been used in a previous study to compare running economy in male and female recreational runners.

Statistical analysis was performed on the Minitab software package (Minitab Inc. 1985). Post-training comparisons were made using a Student's t-test for correlated data and comparison between training and control groups was made using a two sample test. In the results section the measurements or values attained by the training group (TG) are presented first, followed by the corresponding values for the control group (CG).

Results

There were no significant pre-training differences between the TG and CG during maximal (Table 1) or submaximal exercise (Table 2). Running performance during the five km time trial was also similar, 19.69±2.24 vs 20.13±0.68 min, TG and CG respectively (ns). Before the onset of training the estimated rate of oxygen consumption and estimated per cent \(\dot{VO}_2\) max utilized during the five km run were very similar in both groups (Table 2).

After five weeks endurance training the TG improved their five km times by an average 28.2 s (P < 0.01), from 19.69±2.24 to 19.22±2.03 min. The last three one km laps were significantly faster after, compared with before, training. The control group also ran slightly faster, 20.13±0.68 vs 20.06±1.14 min (4.2 s; ns). Analysis of the training diaries submitted by members of the training group (n = 8) showed the average weekly training distance had increased from 31.3±7.0 to 40.8±9.5 km during weeks one and five respectively. This increase just failed to attain statistical significance because of the large standard deviation.

Although \(\dot{VO}_2\) max in the TG increased from 56.0±6.1 to 60.7±5.4 ml.kg\(^{-1}\).min\(^{-1}\) (P < 0.01), there were no significant changes in either the maximum heart rate (197±10 vs 194±6 b.min\(^{-1}\)) or the maximum ventilatory rate (123.2±12.8 vs 128.0±14.31.min\(^{-1}\)) after training. The CG showed slight, non-significant increases in maximal oxygen uptake (three per cent) and maximum rate of ventilation (two per cent). There was no significant change in the estimated % \(\dot{VO}_2\) max utilized during the five km run by either the TG or the CG (Table 2).

After training, the TG and CG ran at the same absolute treadmill speeds, equivalent to 60, 70, 80 and 90 per cent of their pre-training \(\dot{VO}_2\) max values. The TG showed a trend, during this submaximal exercise test, to a decrease in the heart rate at each running speed, which was significant at the third speed. Their rate of
Table 1. Physiological characteristics of the TG (n = 12) and CG (n = 7) before the onset of training (mean±S.D. and range)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Ht. (cm.)</th>
<th>Wt. (kg.)</th>
<th>VO2max (ml.kg⁻¹.min⁻¹)</th>
<th>VEmax (l.min⁻¹)</th>
<th>HRmax (b.min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Training Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>177.8</td>
<td>71.0</td>
<td>4.0</td>
<td>56.8</td>
<td>123.2</td>
</tr>
<tr>
<td>±SD</td>
<td>4.5</td>
<td>8.0</td>
<td>0.5</td>
<td>6.1</td>
<td>12.8</td>
</tr>
<tr>
<td>Range</td>
<td>172.9–185.8</td>
<td>63.3–87.5</td>
<td>2.8–4.9</td>
<td>47.9–69.4</td>
<td>101.3–150.0</td>
</tr>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>176.9</td>
<td>70.3</td>
<td>4.0</td>
<td>56.8</td>
<td>115.3</td>
</tr>
<tr>
<td>±SD</td>
<td>5.7</td>
<td>4.2</td>
<td>0.3</td>
<td>3.0</td>
<td>13.4</td>
</tr>
<tr>
<td>Range</td>
<td>164.3–180.5</td>
<td>65.8–77.1</td>
<td>3.5–4.4</td>
<td>51.8–60.3</td>
<td>95.2–136.7</td>
</tr>
</tbody>
</table>

Physiological characteristics of those members of the control group who participated in both pre- and post-training tests (n = 4) (mean±S.D. and range)

<table>
<thead>
<tr>
<th>Subjects</th>
<th></th>
<th></th>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>174.8</td>
<td>70.1</td>
<td>4.0</td>
<td>57.4</td>
<td>120.8</td>
</tr>
<tr>
<td>±SD</td>
<td>7.1</td>
<td>3.6</td>
<td>0.4</td>
<td>3.8</td>
<td>13.2</td>
</tr>
<tr>
<td>Range</td>
<td>164.3–180.1</td>
<td>65.8–73.5</td>
<td>3.5–4.4</td>
<td>51.8–60.3</td>
<td>106.9–136.7</td>
</tr>
</tbody>
</table>

Table 2. The estimated rate of oxygen consumption and %VO₂ max at 5 km pace, (V-5 km (m.s⁻¹)), V-2mM (m.s⁻¹), V-4mM (m.s⁻¹) and at 3.58 m.s⁻¹ for the training group and control group pre- and post-training (mean±S.D.)

<table>
<thead>
<tr>
<th>Subjects</th>
<th>V-5 km (ml.kg⁻¹.min⁻¹)</th>
<th>%VO₂ max (%)</th>
<th>V-2 mM (ml.kg⁻¹.min⁻¹)</th>
<th>%VO₂ max (%)</th>
<th>V-4 mM (ml.kg⁻¹.min⁻¹)</th>
<th>%VO₂ max (%)</th>
<th>3.58 m.s⁻¹ (ml.kg⁻¹.min⁻¹)</th>
<th>%VO₂ max (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Training Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-training</td>
<td>51.4±5.6</td>
<td>91.8±4.9</td>
<td>39.5±8.3</td>
<td>72.1±1.8</td>
<td>46.0±7.4</td>
<td>81.5±4.2</td>
<td>43.3±3.2</td>
<td>78.2±9.9</td>
</tr>
<tr>
<td>Post-training</td>
<td>54.6±4.3**</td>
<td>90.2±4.1</td>
<td>45.2±6.5**</td>
<td>74.3±5.7</td>
<td>49.9±6.0**</td>
<td>82.7±3.6</td>
<td>45.0±3.4**</td>
<td>74.8±9.9**</td>
</tr>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-training</td>
<td>49.9±0.9</td>
<td>87.3±5.4</td>
<td>38.7±1.5</td>
<td>67.6±2.3</td>
<td>45.0±2.0</td>
<td>78.5±2.6</td>
<td>43.7±1.4</td>
<td>76.5±7.6</td>
</tr>
<tr>
<td>Post-training</td>
<td>52.0±2.8</td>
<td>88.0±2.7</td>
<td>39.6±3.4</td>
<td>67.1±6.7</td>
<td>46.3±3.2</td>
<td>78.4±4.6</td>
<td>44.5±1.2</td>
<td>75.5±5.6</td>
</tr>
</tbody>
</table>

Significant difference between pre- and post-training means; **P < 0.01

Table 3. Treadmill running speed, heart rate, oxygen consumption, %VO₂ max and respiratory exchange ratio (R value) equivalent to 60, 70, 80 and 90 per cent of the pre-training VO₂ max values for the TG pre- and post-training. (mean±S.D.)

| Running velocity | VE (l.min⁻¹) | VO₂ (ml.kg⁻¹.min⁻¹) | %VO₂ | R value | VE (l.min⁻¹) | VO₂ (ml.kg⁻¹.min⁻¹) | %VO₂ | R value | VE (l.min⁻¹) | VO₂ (ml.kg⁻¹.min⁻¹) | %VO₂ | R value | VE (l.min⁻¹) | VO₂ (ml.kg⁻¹.min⁻¹) | %VO₂ | R value |
|------------------|-------------|---------------------|------|---------|-------------|---------------------|------|---------|-------------|---------------------|------|---------|-------------|---------------------|------|---------|-------------|---------------------|------|---------|
| Pre-training | 146±11 | 33.1±3.8 | 59.1±4.6 | 0.90±0.06 | 146±11 | 33.1±3.8 | 59.1±4.6 | 0.90±0.06 | 146±11 | 33.1±3.8 | 59.1±4.6 | 0.90±0.06 | 146±11 | 33.1±3.8 | 59.1±4.6 | 0.90±0.06 |
| Post-training | 143±10 | 33.8±3.9 | 55.8±4.1* | 0.86±0.04* | 143±10 | 33.8±3.9 | 55.8±4.1* | 0.86±0.04* | 143±10 | 33.8±3.9 | 55.8±4.1* | 0.86±0.04* | 143±10 | 33.8±3.9 | 55.8±4.1* | 0.86±0.04* |

Significant difference between pre- and post-training means *P < 0.05; **P < 0.01.

oxygen consumption was slightly higher, significantly so, during the last three running speeds after training. The respiratory exchange ratio (R value) was significantly lower at each running velocity (Table 3), together with significantly lowered blood lactic acid concentrations (Figure 1). The oxygen cost of running at 3.58 m.s⁻¹ increased (P < 0.01) although the %VO₂ max utilized at this reference speed decreased (P < 0.01) after training (Table 2).

In the CG the rate of oxygen consumption increased slightly (P < 0.05) during submaximal exercise at speeds equivalent to 80 per cent and 90 per cent of their pre-training VO₂ max values. However, there was no significant change in their respiratory exchange ratios (R values), heart rates or the blood lactate concentrations during this submaximal exercise test. The CG showed no significant change in the oxygen cost of running at 3.58 m.s⁻¹, nor in the %VO₂ max utilized at this speed (Table 2).

There was no significant change in the pulmonary ventilation (VE) utilized by the TG during this test, although the trend was for lower values after training. However, the ventilatory equivalent for oxygen (VE.VO₂⁻¹) decreased significantly at running speeds...
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Figure 1. The delta blood lactic acid concentration and the \%VO2 max utilized at four running speeds before and after training for the TG. (●): pre-training; (○): post-training. *P < 0.05; **P < 0.01 (Mean±SEM; n = 12)

equivalent to 70, 80 and 90 per cent of their pre-training VO2 max values. The average decreases were, 0.93±0.30 (P < 0.05), 1.4±0.95 (P < 0.05) and 1.84±0.60 l.min⁻¹ (P < 0.01) respectively. These changes were not observed in the CG. There was no significant change in the ventilatory equivalent for carbon dioxide (VE/VO2) for either the TG or CG after the training period.

In the TG the treadmill running speeds equivalent to blood lactate reference concentrations of 2 mmol.l⁻¹ (V-2 mM) and 4 mmol.l⁻¹ (V-4 mM) increased from 3.25±0.87 to 3.57±0.74 m.s⁻¹ and from 3.79±0.77 to 4.04±0.71 m.s⁻¹ respectively after training (P < 0.01). The rate of oxygen consumption at these blood lactate reference concentrations also increased significantly although the %VO2 max utilized showed no significant change. The control group did not demonstrate such changes (Table 2).

Maximal oxygen uptake showed strong correlations with running performance, expressed as five km times, for the TG and CG both pre- and post-training. Although VO2 max is widely accepted as an indicator of performance capacity, strong correlations with five km times, both pre- and post-training, were also found between V-2 mM, V-4 mM, the rate of oxygen consumption at these two blood lactate reference concentrations, the %VO2 max at 3.58 m.s⁻¹, and the estimated rate of oxygen consumption at five km pace (Table 4).

The changes in five km times were correlated with the changes in the physiological and metabolic responses measured during laboratory based exercise. This analysis revealed that the improved five km times in the training group were significantly correlated (r = -0.71; P < 0.01) with changes in the oxygen cost of running at 3.58 m.s⁻¹, rather than changes in the VO2 max (r = -0.07; ns), V-2 mM (r = -0.08; ns), V-4 mM (r = -0.13; ns) or changes in the rate of oxygen consumption at V-2 mM (r = -0.28; ns) and V-4 mM (r = -0.47; ns).

Discussion

The TG demonstrated slightly faster five km times (2.5 per cent) and higher VO2 max values (eight per cent) after the training period. The greatest improvements in VO2 max were shown by those subjects who were the least well trained at the start of the study, which is consistent with the results of other studies. Improvements in running performance may have matched the improvements in VO2 max achieved by the subjects in the present study if high intensity interval training had complemented the increased training volume.

The TG ran the last three laps of the five lap five km time trial significantly faster after, compared with before, training. The ability to maintain a faster average running velocity after training meant that if the TG ‘post-training’ could have raced the TG ‘pre-training’ run 123.5 m in front of the latter. However, the TG were unable to utilize a significantly greater proportion of their post-training VO2 max values during the five km race nor during treadmill running at reference blood lactate concentrations of 2 mmol.l⁻¹ or 4 mmol.l⁻¹. These results suggest that this ability may be a long term adaptation rather than a short term response of physical training.

During submaximal exercise of increasing intensity the TG demonstrated a decrease in the ventilatory equivalent for oxygen (VE/VO2). This decrease represents a ‘more efficient utilization of oxygen’ and, for the subjects in the present study, may reflect an increased mechanical efficiency of the running action.

Table 4. Pearson product moment correlations between physiological and metabolic characteristics and 5 km times (min) for the TG (n = 12) and CG (n = 4) both pre- and post-training

<table>
<thead>
<tr>
<th>Physiological and metabolic characteristics</th>
<th>Training Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pre-tr.</td>
<td>post-tr.</td>
</tr>
<tr>
<td>VO2 max (ml.kg⁻¹.min⁻¹)</td>
<td>r = -0.82**</td>
<td>r = -0.86**</td>
</tr>
<tr>
<td>V2 mM (m.s⁻¹)</td>
<td>r = -0.71*</td>
<td>r = -0.77**</td>
</tr>
<tr>
<td>V4 mM (m.s⁻¹)</td>
<td>r = -0.81**</td>
<td>r = -0.91**</td>
</tr>
<tr>
<td>VO2 at V-2 mM (ml.kg⁻¹.min⁻¹)</td>
<td>r = -0.68*</td>
<td>r = -0.69</td>
</tr>
<tr>
<td>VO2 at V-4 mM (ml.kg⁻¹.min⁻¹)</td>
<td>r = -0.76**</td>
<td>r = -0.85**</td>
</tr>
<tr>
<td>VO2 at 3.58 m.s⁻¹ (ml.kg⁻¹.min⁻¹)</td>
<td>r = 0.49</td>
<td>r = 0.72**</td>
</tr>
<tr>
<td>%VO2 max at 3.58 m.s⁻¹</td>
<td>r = 0.87**</td>
<td>r = 0.90**</td>
</tr>
<tr>
<td>Est. VO2 at V-5 km (ml.kg⁻¹.min⁻¹)</td>
<td>r = -0.83**</td>
<td>r = 0.87**</td>
</tr>
<tr>
<td>Est. %VO2 max at V-5 km</td>
<td>r = -0.09</td>
<td>r = 0.21</td>
</tr>
</tbody>
</table>

* Significant at *P < 0.05, **P < 0.01.

The decrease in blood lactate concentrations and respiratory exchange ratios (R values) at the same absolute and relative exercise intensity after training have been well documented. These adaptive responses to endurance exercise training suggest an increased aerobic capacity of human skeletal muscle. However, many studies which have used cycle ergometry usually report either unaltered or slightly lower VO2 during submaximal exercise after training, rather than any increase in VO2.

Therefore, the increase in the oxygen cost of submaximal exercise (VO2 submax) after training was an unexpected result in this group of relatively untrained subjects. Previous studies using highly trained athletes have either shown no significant change in VO2 submax with further training, or a slight decrease. Evidence from animal studies has suggested that lactate accumulation occurs in fully aerobic working muscle. Therefore, the concentration of lactic acid alone is not necessarily a signal of hypoxia per se. It may, however, reflect regulatory subcellular adaptation to endurance training, which in turn is manifest in the training status of the individual. Wasserman and co-workers provide evidence to support the notional and close relationship between oxygen supply and consumption by muscle. Similarly Katz and Sahlin suggest that lactate production during exercise depends mainly on the availability of oxygen in the active tissue.

The increased rate of oxygen consumption during submaximal exercise after training, in this study, suggests that more of the energy needs of the muscle cell are met by aerobic metabolism. Further support for this suggestion is provided by the decreased R values and lower lactate concentrations at the same running speeds after, compared with before, training. The increased rate of oxygen consumption after training may suggest an increased utilization of free fatty acids, or intramuscular triglycerides to support energy production, and of the more efficient oxidation of blood borne and muscle carbohydrates.

The respiratory exchange ratio at 60 per cent VO2 max has been used as an indicator of relative substrate utilization. In the present study the R values were 0.90±0.06 and 0.86±0.04 pre- and post-training respectively. At 60 per cent VO2 max pre-training, 34.0 per cent of the energy expenditure was derived from the catabolism of fat. This compares with 47.6 per cent of the energy expenditure derived from fat catabolism after training. These values are similar to those reported for untrained and trained men respectively exercising at 65 per cent VO2 max. If one litre of oxygen were to completely oxidize one gramme of fat and one gramme of carbohydrate respectively, the energy difference would be 8.1 per cent. The difference in oxygen consumption at 60 per cent VO2 max, after compared with before training, in the present study was 2.1 per cent. Therefore, an increased submaximal oxygen consumption per se does not explain totally the difference in respiratory exchange ratios pre- and post-training.

The lack of a significant correlation between the change in VO2 and the change in R values during submaximal exercise in the present study may be because the change in VO2 does not mirror the time course of change in the R values. Changes in the respiratory exchange ratio associated with training maybe more strongly related to changes in muscle oxidative capacity.

The well documented adaptation within endurance trained muscle with respect to its microstructure and associated microvasculature suggest greater perfusion and an increased transit time for blood through muscle after training. Such adaptations would facilitate an improved availability of oxygen and substrates to the active muscle cell while promoting the rapid removal of metabolic waste products. Such adaptations within human skeletal muscle may be especially important during marathon and ultra running events.

In summary, previous studies have shown good correlations between the oxygen cost of submaximal exercise and endurance performance. The present study extends these observations in that a similar correlation was obtained between the increase in aerobic metabolism during submaximal exercise and the improvement in running performance.

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