The Effects of Endurance Training on Muscle Fibre Types and Enzyme Activities

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Abstract/Résumé

Practitioners and scientists have demonstrated great interest in the physiological and biochemical effects of endurance training on the results of the marathon run. It is well documented that athletes with a large proportion of slow twitch and fast twitch aerobic skeletal muscle fibre, high metabolic enzyme activities and concentrations, large mitochondria concentration and, of course, the ability to increase the power output generated for a given rate of oxygen consumption and energy expenditure, are generally highly successful distance runners. Aerobic and endurance training have been shown to bring about significant adaptations to the skeletal muscle and its inclusions as well as to the delivery system. In particular, enzyme activity levels are readily mutable, mitochondrial concentrations increase, and some evidence suggests that the fibre distribution is changed. This article briefly reports on changes in skeletal muscle brought about by endurance training and those changes that appear most effective in yielding success in endurance events.

Praticiens et scientifiques ont manifesté un grand intérêt à l’égard des effets biochimiques et physiologiques de l’entraînement à l’endurance sur les résultats au marathon. D’après de nombreuses études, les athlètes présentant une forte proportion de fibres musculaires lentes et de fibres rapides à caractère aérobie, d’importantes concentrations d’enzymes actives, beaucoup de mitochondries, et une capacité d’accroître la puissance mécanique

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Introduction

Human skeletal muscles generally are mosaics of muscle fibres that play a significant role in determining the contractile and fatigue patterns of the total muscle. Scientists have been well aware, since the work of Ranvier in 1874, that skeletal muscle is composed of different fibre types. It has essentially been accepted that individual motor units (the smallest unit that can be activated in a muscle contraction) are composed of a particular motor nerve and the fibres it innervates. Individual innervated fibres of a motor unit are metabolically homogeneous. However, skeletal muscles generally possess more than one type of motor unit.

Histochemical and histological laboratory techniques have helped to identify not only various fibre types but also substrate concentrations and metabolic enzyme activities. The advent of the skeletal muscle biopsy technique developed by Hultman (1967b) rapidly opened opportunities for research encompassing human tissue taken under nonsurgical conditions and the study of exercise and physical training on skeletal muscle. For an excellent review of skeletal muscle adaptability, see Saltin and Gollnick (1983).

Since the time of the ancient Greeks, the figure of 26 miles and 385 yards (42.195 km) has reserved a unique place in the annals of history. It has evolved from a wartime symbol to one of sport. The marathon is perhaps the most prestigious event in the world of track and field, and the multitude of international marathon runs bring not only instant acclaim but a great deal of profit to the successful competitors and the organizers. As a result, the marathon has been the focus of an overwhelming amount of investigation over the past 20 years. The first major conference dealing with this subject was hosted by The New York Academy of Science in 1977 (Milvy, 1977). Researchers have become fascinated with the athletes who are physiologically equipped to succeed in such a race. This paper examines the topic from the perspectives of fibre types and enzyme activities, and the research completed, for the most part, in the past 30 years.

What Predicts Success in Marathons?

Many factors have been used in the attempt to predict success in numerous athletic events. The marathon is no exception. If a generalization can be permitted here, it could be stated that no single parameter has been shown to predict success in any sporting event, although some have a higher correlation than others. Maximum oxygen uptake, body composition, enzyme activities, and fibre type have all been applied, but with limited success (Costill, 1972, 1974; Saltin et al., 1977). Obviously
the physiology and biochemistry of the elite athlete are dependent upon a complex series of mechanisms, operating within an appropriate time frame, which leads to successful completion of the task. With the marathon in particular, external factors such as temperature, humidity, altitude, and psychological disposition also play a significant role (Costill, 1972; Milvy, 1977).

SKELETAL MUSCLE FIBRE TYPES AND ENDURANCE RUNNING

Research with skeletal muscle fibre types has evolved from the early red and white muscle classification (Abernathy et al., 1990; Saltin and Gollnick, 1983) to identify a broad spectrum of fibres differentiated by contractile properties, enzyme activities, myosin content, and other morphological, biochemical, and physiological differences (Booth and Baldwin, 1996; Connett and Sahlin, 1996; Howald et al., 1985; Saltin and Gollnick, 1983). Classification schemes based on two fibre types—Type I (slow twitch) and Type II (fast twitch)—soon expanded to Type I, Type IIa, and Type IIb, and more recently added Type IIx (Booth and Baldwin, 1996). Probably more fibre types have yet to be identified.

From the outset it became obvious that athletes with a high preponderance of slow twitch fibres or a mixture of Type I and Type IIa would have more success in endurance events (Ahlborg and Brohult, 1967; Benzi et al., 1975; Billeter et al., 1976; Holloszy, 1973; Holloszy et al., 1977; Horowicz et al., 1994). When compared to IIb fibres, these aerobic fibres display greater utilization of aerobic metabolic pathways, extensive capillarization, greater mitochondria concentration, and different substrate utilization. A high percentage of slow twitch fibres improves endurance performance ability by significantly increasing the power output generated for a given rate of oxygen consumption and energy expenditure (Holloszy,

Table 1 Effects of Endurance Training on Selected Skeletal Muscle Fibre Characteristics

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Training effect</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I fibre area</td>
<td>↑</td>
<td>Costill (1972); Gollnick et al. (1973); Hakkinen &amp; Keskinen (1989); Patton et al. (1990); Saltin et al. (1976)</td>
</tr>
<tr>
<td></td>
<td>→</td>
<td>Thayer et al. (1993)</td>
</tr>
<tr>
<td></td>
<td>↓</td>
<td>Jansson &amp; Kajser (1977)</td>
</tr>
<tr>
<td>Type II fibre area</td>
<td>↓</td>
<td>Howald et al. (1985)</td>
</tr>
<tr>
<td>Fiber conversion</td>
<td>↑</td>
<td>Jansson &amp; Kajser (1977)</td>
</tr>
<tr>
<td>Mitochondria number and size</td>
<td>↑</td>
<td>Holloszy (1967); Saltin &amp; Gollnick (1983)</td>
</tr>
<tr>
<td>Capillary density</td>
<td>↑</td>
<td>Hermansen &amp; Wachtlova (1971); Saltin &amp; Gollnick (1983)</td>
</tr>
</tbody>
</table>
1973; Horowitz et al., 1994; Patton et al., 1990). Significant positive correlations have also been found between the percentage of slow twitch fibres and the absolute and relative lactate threshold (Ivy et al., 1980; MacDougall, 1977; Sjodin, 1976).

Most of the literature further suggests there is a large interindividual variation in muscle fibre composition, but the variation is far less in different muscles in the same person (Gollnick et al., 1973; Saltin and Gollnick, 1983; Thayer et al., 1993). As well, fibre percentages differ between men and women, with men having larger fibres and a greater distribution of Type IIb in most locomotory muscles (Boros-Hatfaludy et al., 1986; Costill et al., 1979; Fournier et al., 1982; Gollnick et al., 1973; Saltin and Gollnick, 1983). It has recently been noted that the ratio of Type I to Type II is greater near the central portion of the muscle (Booth and Baldwin, 1996; Thayer et al., 1993). Most elite distance runners have a range of 56–100% (Saltin and Gollnick, 1983; Saltin et al., 1977) Type I and Type IIa fibres in the locomotion muscles of the legs. This very large range further suggests that factors other than fibre type are important manifestations of endurance training.

ADAPTATION OF FIBRE TYPES TO ENDURANCE TRAINING (Table 1)

Type I skeletal muscle fibres have been found to hypertrophy in postpubertal subjects with endurance training (Howald et al., 1985; Saltin et al., 1977), but to remain constant in distribution and size in prepubertal children (Fournier et al., 1982). No significant longitudinal studies (+10 yrs) have been carried out from puberty to early adulthood in order to observe the effects of training on fibre distribution, although work by Jansson and Kaijser (1977, 1987) with professional orienteers, and by Howald et al. (1985) with sedentary subjects, did demonstrate that fibre distribution changes moderately with endurance training.

Some recent findings reviewed by Booth and Baldwin (1996) show that fibres in other mammals may be undergoing inclusive changes that are not identified by simple histochemical techniques but are noted via myosin identification and other more sophisticated techniques such as protein translation, cell signaling, direct plasmid injection, and homologous recombination, which will someday be carried out in the human model. Interindividual variation in fibre distribution appears to be genetically determined (Hamel et al., 1986). Whether endurance training can bring about substantial changes in fibre composition remains to be determined.

METABOLIC ENZYMES AND ENDURANCE TRAINING

Regardless of changes in muscle fibre types, endurance training results in many metabolic adaptations in the muscles of elite athletes. These adaptations include: improved ability to produce ATP via oxidative phosphorylation, increased size and number of mitochondria, less lactic acid produced per given amount of exercise, increased myoglobin content, increased intramuscular triglyceride content, increased lipoprotein lipase activity, increased proportion of energy derived from fat and less from carbohydrate, lower rate of glycogen depletion during exercise, and improved efficiency in extracting oxygen from the blood.

Many of these adaptations are regulated by or are at least affected by changes in specific enzyme activities. For example, endurance training produces a sequence of biochemical adaptations in skeletal muscle which maximizes its capacity specifically for oxidative metabolism, including increased mitochondrial size and
### Table 2: Summary of Effects of Endurance Training on Selected Energy Pathway Enzyme Activities

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Site</th>
<th>Training effect</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myosin ATPase</td>
<td>Types I and II fibers</td>
<td>→</td>
<td>Taylor et al. (1974a)</td>
</tr>
<tr>
<td>Hexokinase</td>
<td>Muscle</td>
<td>↑</td>
<td>Holloszy (1975)</td>
</tr>
<tr>
<td>PFK (phosphofructokinase)</td>
<td>Muscle</td>
<td>↑</td>
<td>Gollnick et al. (1973); Hamel et al. (1986); Taylor et al. (1978)；Costill et al. (1976); Fournier et al. (1982)</td>
</tr>
<tr>
<td>LDH&lt;sub&gt;1&lt;/sub&gt; (lactate dehydrogenase, heart type)</td>
<td>Type I</td>
<td>↑</td>
<td>Sjodin (1976)</td>
</tr>
<tr>
<td></td>
<td>Type II</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>LDH&lt;sub&gt;Ttot&lt;/sub&gt; (total lactate dehydrogenase)</td>
<td>Muscle</td>
<td>↓</td>
<td>Sjodin (1976)</td>
</tr>
<tr>
<td>BCAA family of enzymes</td>
<td>Serum</td>
<td>↑→↓</td>
<td>Eastmann &amp; Lehmann (1998)</td>
</tr>
</tbody>
</table>

number (Billeter et al., 1976; Essén et al., 1975; Holloszy, 1967; Howald et al., 1985; Saltin and Gollnick, 1983; Saltin et al., 1976). But moreover, the changes enable the athletes to be more effective competitors. One cellular adaptation to endurance training is the synthesis of more enzymes and the subsequent appearance of higher enzyme activities. These adaptations appear to be a function of the daily workload and the total training time (Conley and Krahenbuhl, 1980; Foster et al., 1977; Patton et al., 1990; Siegel et al., 1980).

In elite endurance athletes, especially those who use the Fartlek system of training or those who enjoy the sprint home after a long race, several enzymes play a key role including ATPase, glycogen cycle enzymes, glycolytic enzymes utilized primarily under aerobic conditions, Kreb's cycle enzymes, those involved in fat oxidation, and to a lesser extent, amino acid transferases, synthetases, dehydrogenases, and decarboxylases, in particular those affecting branched-chain amino acids (BCAA).

APASE (Table 2)

In 1967 Bárány observed that the intrinsic speed of muscle contraction, with and without load, was (and is) a characteristic property of the ATPase activity of myosin in skeletal muscle of several mammalian species. ATP hydrolysis has a higher maximum velocity in fast twitch fibres and significantly different biochemical properties from slow twitch fibres. This discovery was significant in that contractile properties were directly related to enzyme activity, but perhaps more important,
Table 3  Summary of Effects of Endurance Training on Glycogen Cycle Enzyme Activities

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Muscle</th>
<th>Training effect</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphorylase (total)</td>
<td>Gastrocnemius</td>
<td>↑</td>
<td>Costill et al. (1979)</td>
</tr>
<tr>
<td>Phosphorylase a (active form)</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Taylor (1975); Taylor et al. (1972)</td>
</tr>
<tr>
<td>Debranching</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Taylor et al. (1972); Taylor (1974c)</td>
</tr>
<tr>
<td>Synthetase (active form)</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Taylor et al. (1972); (1971b)</td>
</tr>
<tr>
<td>Branching</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Taylor et al. (1972); (1974b)</td>
</tr>
</tbody>
</table>

that differences were noted between Type I and Type II muscle. Saltin’s group (Essén et al., 1975; Taylor et al., 1974a) noted that these differences extended to human fibres. In a classic paper (Essén et al., 1975), the first to deal with single human fibres, Saltin’s group further demonstrated that the relationships held with single Type I and Type II fibres. The importance of this finding to endurance running is not obvious, since only minimal changes in ATPase activity have been reported with endurance training (Saltin et al., 1977).

GLYCOGEN CYCLE ENZYMES (TABLE 3)

Little work has been carried out with human tissue and glycogen cycle enzymes. The first series of reports on the relationship to training came from our laboratories (Taylor, 1975; Taylor et al., 1971; 1972; 1974b; 1974c). We found that with endurance training, all measured enzymes in the glycogen cycle increased in activity at least twofold. The importance of this finding is reflected by the need for skeletal muscle to rapidly utilize glycogen with exercise, and the immediate need to resynthesize glycogen after the cessation of exercise. It was apparent that regular exercise, of necessity, contributed to increased activity levels of glycogen anabolic and catabolic enzymes. It is well known that endurance training and diet can increase muscle glycogen stores (Essén et al., 1975; Hultman, 1967a, 1967b; Taylor et al., 1971a), and that carbohydrate is used even during endurance exercise (Roberts et al., 1985; 1988a). Therefore it is important for elite endurance athletes that the glycogen cycle enzymes be utilized to maximum to guarantee an adequate supply of muscle glycogen, and for the subsequent rapid resynthesis of these carbohydrate stores.

GLYCOLYTIC ENZYMES

Although marathon running is primarily an endurance event, one would suspect that for the start and perhaps for a relatively long sprint at the end of the race, increased glycolytic enzyme activity levels would be advantageous. There are conflicting results as to the adaptation of glycolytic marker enzymes (Saltin and
### Table 4: Summary of Effects of Endurance Training on Oxidative Enzyme Activities

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Muscle</th>
<th>Training effect</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>SDH (succinic dehydrogenase)</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Fink et al. (1977); Fournier et al. (1982); Gollnick et al. (1973); Holloszy (1967, 1973, 1975); Hoppler et al. (1985); Saltin et al. (1976); Taylor et al. (1978)</td>
</tr>
<tr>
<td>CS (citrate synthase)</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Anderson &amp; Henriksson (1977); Boros-Hatfaludy et al. (1986); Henriksson &amp; Reitman (1977); Holloszy et al. (1977); Swedenhag et al. (1983)</td>
</tr>
<tr>
<td>CYTOX (cytochrome oxidase)</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Henriksson &amp; Reitman (1977)</td>
</tr>
<tr>
<td>βHAD (β-hydroxyacyl CoA dehydrogenase)</td>
<td>Vastus lateralis</td>
<td>↑</td>
<td>Saltin &amp; Gollnick (1983); Jansson &amp; Kaijser (1977)</td>
</tr>
</tbody>
</table>

Gollnick, 1983). However, high intensity endurance training has been shown to produce increased PFK (Gollnick et al., 1973) and hexokinase (a nontraditional glycolytic enzyme) activity (Holloszy, 1975), and endurance runners have high LDH$_h$ values (Saltin and Gollnick, 1983). It has yet to be determined whether the differences are caused by the different activity levels found in different fibre types, but positive correlative data are available for total LDH (Sjödin, 1976). Clearly, further investigation is required to study the effects of endurance training on specific glycolytic enzymes.

**MITOCHONDRIAL ENZYMES (TABLE 4)**

The marathon is an aerobic endurance event. Thus it is not surprising that mitochondrial enzyme activity levels increase rather dramatically with endurance training. The capacity of skeletal muscle to conserve glycogen is directly related to the level of activity of the Krebs cycle and electron-transport system (Saltin and Gollnick, 1983). Rate limiting and/or regulatory enzymes including cytochrome oxidase (CYTOX), succinic dehydrogenase (SDH), and citrate synthase (CS) have been shown to increase as much as four- to sixfold under endurance training conditions (Saltin and Gollnick, 1983). In fact, oxidative capacity increases in both fibre types with endurance training (Green et al., 1983; Saltin et al., 1977). It is interesting that sprint training also causes a definite elevation of the mitochondrial enzymes (Saltin et al., 1976), and demonstrates that muscle contractility, a mechanical factor, may be the stimulus for increased enzyme production for all pathways.
Table 5  Selected Adaptations of Skeletal Muscle to Endurance Training

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Measurement site</th>
<th>Training effect</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycogen content</td>
<td>Muscle</td>
<td>↑</td>
<td>Gollnick et al. (1973); Taylor (1975); Taylor et al. (1971a, 1971b, 1972, 1974a)</td>
</tr>
<tr>
<td>FFA use (non-esterified fatty acids)</td>
<td>Serum-muscle</td>
<td>↑→</td>
<td>Saltin &amp; Gollnick (1983)</td>
</tr>
<tr>
<td>Triglyceride content</td>
<td>Muscle</td>
<td>↑</td>
<td>Hoppler et al. (1985); Howald et al. (1985)</td>
</tr>
<tr>
<td>BCAA use</td>
<td>Serum-muscle</td>
<td>↓</td>
<td>Eastmann &amp; Lehmann (1988)</td>
</tr>
<tr>
<td>Amino acid use</td>
<td>Serum-muscle</td>
<td>↑</td>
<td>Eastmann &amp; Lehmann (1988)</td>
</tr>
</tbody>
</table>

There is a close relationship for the time course between changes in oxidative enzyme activities and the concomitant change in total body oxygen uptake (VO₂ max) over 3 to 4 weeks of endurance training. As well, the relationship is maintained with detraining (Henriksson and Reitman, 1977). The adaptive response of the enzyme activity level is a local phenomenon and appears regardless of age or sex.

OTHER ENZYMES RELATED TO ENERGY PRODUCTION (TABLES 2 AND 5)

The respiratory capacity of the muscle is of primary importance in determining the work rate at which lactate accumulation begins (Ivy et al., 1980). The slow twitch fibre has been shown to have a high mitochondrial density and mitochondrial enzyme activity (Howald, 1985), which favors oxidative energy production. Regular enforced rigorous exercise has been found to result in enhanced liver and muscle glycogen stores and increased plasma free fatty acids (Bergstrom, 1962; Bergstrom et al., 1967; Hultman, 1967a). The use of free fatty acids as a fuel, by slow twitch fibres, is vital in sparing the glycogen stores in the fibres and hence prolonging the work time at submaximal levels, while decreased hexokinase activity spares glucose.

The enzymes of beta oxidation increase with training, and as fatty oxidation increases, the ratio of acetyl CoA increases and inhibits pyruvate oxidation. Thus there is an increased rate of fat oxidation which is shown to slow the rate of glycolysis. In addition to a decreased rate of lactate accumulation, the marathoner also develops an increased tolerance for peak exercise lactate levels, a greater work capacity, a prolonged time to fatigue, and a shortened recovery time (Costill, 1972; Ivy et al., 1980). Endurance training is associated with a preferential use of triglycerides for slow twitch fibres, and endogenous triglycerides may account for over 50% of the total lipid oxidized during exercise (Abernethy et al., 1990; Essén, 1977).
Highly trained endurance athletes have been shown to possess higher lipoprotein lipase activity (Roberts et al., 1988b), increased uptake by muscle of branched-chain amino acids (Eastmann and Lehmann, 1998), and an increased rate of oxidation of BCAA during exercise (Wagennakers et al., 1989). Thus all energy producing systems appear to be ameliorated by endurance training. In the trained state, the increased number and volume of mitochondria offer an increased surface area for the exchange of metabolites, cofactors, and end products. However, all these adaptations fail to explain the large increase in the oxidative capacity of skeletal muscle (Saltin and Gollnick, 1983).

ADAPTATION OF ENZYMES TO ENDURANCE TRAINING

Some of these adaptations can be explained by the increased number and size of mitochondria, and the increased muscle enzyme synthesis and turnover (Holloszy et al., 1977). Endurance training results in a rise in the activities of the enzymes included in the citric acid cycle, terminal oxidation and the beta oxidation of free fatty acids, contributing to oxidative energy production in skeletal muscle.

A variation in the activity level causes the oxidative potential of muscle to vary (Saltin et al., 1976). This finding complements the fibre cross-sectional data and demonstrates the capacity of skeletal muscle for adaptation (Table 5). Few enzymes of the metabolic pathways do not increase in concentration and/or activity with endurance training (Fowler et al., 1968; Hearn and Wainio, 1956, 1957; Saltin and Gollnick, 1983).

Summary

1967 was a very important year for scientists studying the effects of endurance exercise on skeletal muscle. The development of the biopsy needle, the relationship between contractile properties and ATPase activity, and early studies on metabolic enzyme concentrations and activity levels were presented. Although it has been some 30 years since these findings were published, the era of research related to fibre typing, histochemistry, and enzymology is far from finished. For example:

- System analysis has yet to brave the frontier of assumed equilibrium with exercise—a kinetic model is required (Connett and Sahlin, 1996).
- Control mechanisms are required for many metabolic pathways, but in particular the glycolytic pathway.
- Kinetic models do not yet adequately explain the quantitative effects of Pi and pH in vivo and the relationship to ATP hydrolysis.
- The role of super activity levels of metabolic pathway enzymes is yet to be defined.
- The true interchange between substrates under different exercise conditions is not clear, i.e., when and why a muscle changes substrate and the mechanisms causing this phenomenon.
- Transport of reducing equivalents across the mitochondrial membrane via the shuttle systems remains a mystery (Connett and Sahlin, 1996).
- The role of BCAA with exercise has only recently been investigated.
- Mechanisms causing signaling pathways must be identified.
• Cause and effect relationships of signaling pathways must be established.
• Mechanisms modulating gene expression in exercising muscle is a burgeoning field.
• And as Booth and Baldwin (1996, p. 1111) have stated, perhaps one of the most difficult tasks of "delineating signalling pathways from the exercise signal to the changes in gene expression" may be the major area of research for the future.

References


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