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THE SPECIFIC NATURE OF TRAINING ON MUSCLE: A REVIEW

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To evaluate the hypothesis that the accumulation of metabolites during training will specifically induce the adaptive synthesis of structural and enzyme proteins, the relationships between the performed exercises and the changes in muscle fibers are discussed. According to the principle of specificity of adaptation to various kinds of muscular activity, various adaptive changes are induced in muscle fiber by different types of exercise. The two main directions of training influences are action on myofibrils, induced by high resistance exercises, and action on mitochondria, induced by endurance exercises. It seems that the effect on myofibrillar hypertrophy depends on the resistance to muscle contraction, and on the total number of contractions performed against high resistance. The effect on mitochondria depends on the total period of persisting high-level oxidation in skeletal muscle, and on the oxidation rate during these periods. Between the two main directions of training effects is the influence of power, sprint, and anaerobic interval training. In these cases, adaptation in the muscle fiber is related, in the first instance, to the sarcoplasmic reticulum and enzymes of anaerobic metabolism. Myofibrillar hypertrophy is pronounced modestly if at all in these cases, as well as in aerobic endurance training. High resistance, power, or sprint exercises do not induce adaptations at the level of mitochondria. Training may induce two different adaptations at the level of myofibrillar adenosine triphosphatase (ATPase): elevation of the enzyme activity, making possible a rapid liberation of energy for contraction (observed in sprint, interval, or strength training), and decreased enzyme activity essential for a more economical utilization of the ATP stores (in endurance training). In muscle fibers of various types the expression of training-induced specific changes depends on involvement of fibers during the performed exercises.

KEYWORDS: exercise training, glycolytic enzymes, mitochondrial enzymes, myofibrillar ATPase, myofibrillar hypertrophy, muscle fibers of various types, sarcoplasmic reticulum, skeletal muscle

The changes induced by exercise training express an adaptation to a condition of increased muscular activity. There is evidence that the development of a continuous stable adaptation is closely related to the genome activation (Meerson, 1975). Accordingly, an adaptive protein synthesis has been assumed as a foundation for fitness improvement (Booth and Thomason, 1991; Hollmann and Hettinger, 1976; Mader, 1988; Poortmans, 1975; Yakovlev, 1976). The training and detraining mirror relationships in growth and decay of cellular capacities (Banister *et al.*, 1992). It has been hypothesized that the accumulation of metabolites during training will specifically induce adaptive synthesis of structural and enzyme proteins related to the most active cellular structures and biochemical reactions, respectively (Viru, 1984). In order to evaluate this hypothesis, the relationship between training and the change in muscle fibers will be discussed. Knowing the precise relationship between the exercise and training effect on tissue is essential for guidance of the training

process, on the one hand, and for operative feedback control on the training procedure, on the other.

The principle of specificity of adaptation to various kinds of muscular activity was first formulated and argued by Yakovlev (1949, 1976, 1977). Later, striking evidence for the specific nature of the training effect was obtained from a great number of studies carried out in several laboratories.

Each exercise determines the degree of activity of various organs, different type of muscles, and motor units. Within each active cell, the main metabolic pathway that permits accomplishment of the necessary functional task also depends on the nature of exercise. The activity of any metabolic control system at various levels as well as the activity of the system directly regulating bodily function is also dependent on the nature of the training exercise. Correspondingly, the organism's adaptation bears the imprint of the type of exercise systematically used in training (Fig. 1).

MYOFIBRILS

Hypertrophy

The most prominent result of training for improved strength is muscular hypertrophy (Hettinger, 1961; Hollmann and Hettinger, 1976). A very pronounced muscle hypertrophy is displayed by athletes exposed to long-term vigorous strength training (Häkkinen *et al.*, 1984; Pipes, 1979; Tesch and Larsson, 1982). The augmented muscle mass is primarily the result of an increased size of the individual fiber (Dons *et al.*, 1978; Häggmark *et al.*, 1978; Häkkinen *et al.*, 1981; MacDougall *et al.*, 1980; Prince *et al.*, 1976; Thorstensson *et al.*, 1976). The latter is based on enlargement of myofibrils themselves (Lüthi *et al.*, 1986; Yakovleva, 1968), and obviously due to the augmentation of myofibrillar protein (Helander, 1961; McDonagh and Davies, 1984; Yakovlev, 1976, 1977). Also an increased number of myofibrils have been found, indicating some hyperplasia (Goldspink, 1964; Yakovleva, 1968). It is assumed that the myogenic response to strength training involves mainly the synthesis of new contractile protein (Goldberg, 1968; Hamosch *et al.*, 1967; Hoppeler, 1986; Yakovlev, 1978).

In the rat a method of strength training, consisting of a repeated series of fast climbing up a slope with an attached load (100 to 400 gm), induced an increased rate of synthesis of actinomyosin protein in fast-twitch glycolytic (FG) and fast-twitch oxidative glycolytic (FOG) fibers (Pehme and Seene, 1988). Thus, a period of strength training resulted in an increase of the muscle cross-sectional area mainly in the fast-twitch fibers of the rat (Yakovlev and Yakovleva, 1971) as well as of the human (Costill *et al.*, 1979a; Dons *et al.*, 1978; Häkkinen *et al.*, 1981; Komi, 1986). Olympic weightlifters may possess fast-twitch fibers that are two times larger in diameter than slow-twitch fibers of the same muscle (Prince *et al.*, 1976; Tesch and Karlsson, 1985). The great growth potential of fast-twitch fibers allows the area of muscle occupied by fast-twitch fibers to increase by 90% with strength training, despite retaining a fiber-type composition within the normal range (Tesch, 1988). However, a moderate enlargement of slow-twitch fibers cannot be excluded from having contributed to this hypertrophy (Komi, 1986; Young *et al.*, 1983).

The effect of training for improved strength differs from that of aerobic endurance training, which does not cause a substantial increase in the cross-sectional area of the muscle fiber (Hoppeler, 1986; Hoppeler *et al.*, 1985; Salmons and Hendriksson, 1981). In

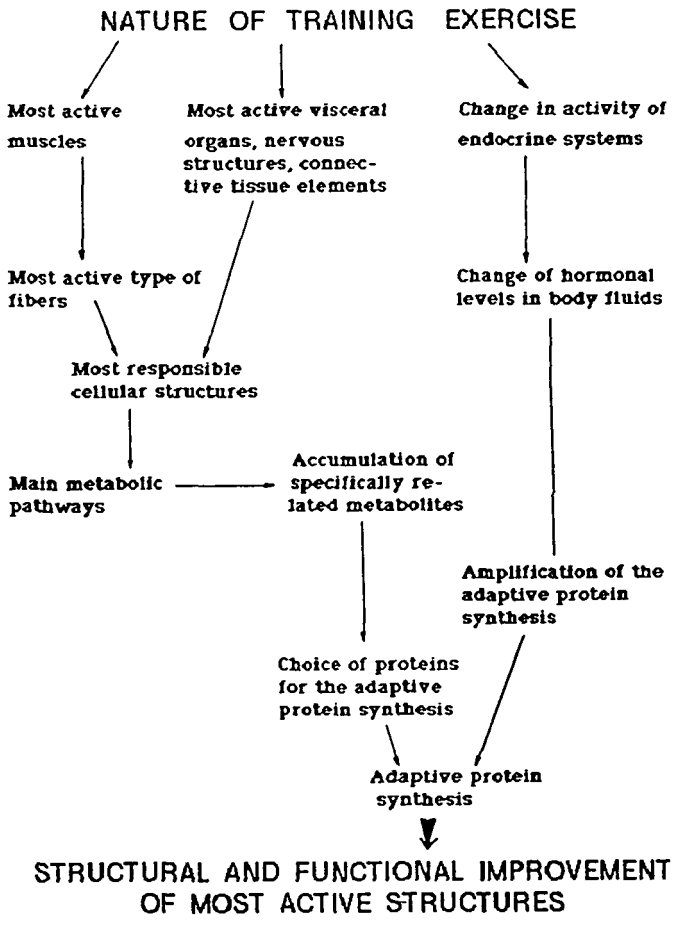

FIGURE 1 Exercise-induced specificity of training effect.

one study even a decrease of the volume fraction of myofibrils was found after endurance training of 2 months' duration (Rösler *et al.*, 1985). However, a selective and moderate enlargement of the slow oxidative fiber (SO), in some cases also of the FOG is possible in endurance training (Bylund *et al.*, 1977; Gollnick *et al.*, 1973; Howald *et al.*, 1985; Sjöström *et al.*, 1982). No increase in the content of myosin or actin has been detected after endurance training (Yakovlev, 1975, 1976).

Endurance training induces an elevated rate of the myosin heavy chain and actin turnover in all fiber types. In sprint-trained rats a more rapid turnover of the myosin heavy chain was found only in FG and FOG fibers (Seene and Alev, 1991).

Sprint training increases the cross-sectional area of both slow-twitch and fast-twitch fibers (Macková *et al.*, 1986). This effect is greatest for the fast-twitch fiber. The end result is that the latter occupies a greater area in the sprint-trained athlete than in the nonathlete (Saltin *et al.*, 1976). The effect of sprint training on fiber size is less pronounced than that of strength training.

The area of slow-twitch fibers constitutes 51 to 76% of muscle in distance runners (percent of slow-twitch fibers, 63 to 74) and in sprinters 15 to 32% (percent of fibers, 21 to 27) with no significant difference in individual fiber area between the two groups of athletes. In middle-distance runners, long and high jumpers, shot-putters, javelin and discus throwers, the corresponding values are somewhere between those in distance runners and sprinters. The area of individual fibers was the largest in shot-putters and discus throwers, particularly in regard to fast-twitch fibers (Costill *et al.*, 1976).

CONTRACTILE MECHANISM

Sarcolemma

No data are available for evaluating the specific training effect on the excitation mechanism. It may only be speculated that there exist individual differences in acetylcholine synthesis and release, cholinesterase activity, and maybe even in ionic gradients. However, the plasma membrane of skeletal muscle has been frequently implicated in the process of fatigue (Tibbits, 1990). It has been suggested that low-intensity fatigue may be the result of an alteration in membrane structure in response to the activation of phospholipases (membrane) or the production of free radicals. The latter can induce lipid peroxidation as well as direct modification of the intracellular transport systems (Tibbits, 1990). A training-induced adaptive response involves lipid peroxidation and alteration in scavenger enzyme activity (Alessio and Goldfarb, 1988). The action on cellular membranes is obviously accentuated by concomitant increase in activity of proteases (Byrd, 1992).

A highly effective ionic pump system is necessary for good performance in athletes. In rats, very long exercise causes decreased activity of sodium-potassium adenosine triphosphatase (Na^+,K^+-ATPase) in association with the accumulation of sodium and water in skeletal muscle and myocardial cells. Endurance training did not alter the activity of myocardial Na^+,K^+-ATPase in the rats but prevented the change induced by the prolonged exercise (Kórge *et al.*, 1974). An 11-week training program decreased the K_m of Na^+-Ca^{2+} exchange in sarcolemmal vesicles of myocardium with no alterations of maximum velocity (Tibbits *et al.*, 1981).

In humans a biopsy study showed that the person who for many years systematically exercised had an increased concentration of Na^+,K^+-pump loci in the vastus lateralis muscle. In comparison with age-matched untrained subjects, the swimming-, running- and strength-trained subjects demonstrated increased concentration of [^{3}H]ouabain binding site of 30, 32, and 40%, respectively (Klitgaard and Clausen, 1989). Intensive cycle-sprint training for 7 weeks also resulted in an increase in the [^{3}H]ouabain binding site in vastus lateralis muscle (McKenna *et al.*, 1991). In rats intensive swimming training for 3 weeks produced a 25 to 64% increase in the concentration of Na^+-K^+ pumps (Kjeldsen *et al.*, 1986). In dogs subjected to a moderate training program, the activity of Na^+,K^+-ATPase in the sarcolemma was found to be 2.6-fold higher than in control dogs (Knochel *et al.*, 1985).

Sarcoplasmic Reticulum

The protein content of sarcoplasmic reticulum (SR) increased in rat muscle as a result of swimming training when the exercise intensity was gradually elevated by an increasing work rate, but not when the exercise intensity remained modest, even if its duration was

gradually prolonged (Yakovlev, 1978). In this latter study the capacity of the SR to absorb Ca^{2+} remained unchanged when calculated per milligram of protein. If calculated per unit of muscle weight, the capacity increased after training at an elevated intensity of swimming. In another study calcium transport capacity was found to have decreased as a result of an analogous training program (Sembrovich *et al.*, 1978). During endurance training Ca^{2+} uptake by SR decreased in FG fibers, but did not change in FOG or SO fibers (Kim *et al.*, 1981).

A comparison of the effect of various training regimens in rats indicates that there is an increased rate of Ca^{2+} accumulation by the SR (calculated per 1 mg protein) in the SO soleus muscle after training by continuous aerobic running, sprint training, interval training, and strength training in fast climbing (Fig. 2). In the white portion of quadriceps muscle (FG) a significantly increased rate of Ca^{2+} accumulation was obtained after sprint, interval, or strength training, but not after continuous running. In both muscles the highest change was caused by sprint and strength training. Training in continuous swimming decreased the Ca^{2+} accumulation rate in both muscles (Viru, 1992). Consequently, the training effect on the SR depends on the exercise intensity used. As might be expected, adaptation to the systematic use of highly intensive exercise stimulated an improvement in the function of the SR. The lack of effect on SR in endurance training seems to be related to the fact that the increased rate of Ca^{2+} accumulation is easily overcome by continuous heavy exercise when an elevated Ca^{2+} remains in muscle hours after exercise (Byrd, 1992).

Actomyosin Adenosine Triphosphatase Activity

In rats the activity of muscle actomyosin ATPase is increased (Wilkerson and Evonuk, 1971) or remains unchanged (Bagby *et al.*, 1972) after endurance training. Strenuous running training (continuous running with periodic sprints) decreased the enzyme activity by 20% in FOG fibers and increases it approximately to the same extent in the muscle containing predominantly SO fibers. No change has been found in FG fibers (Baldwin *et al.*, 1975). When training exercise consists in repeated jumps, the myofibrillar ATPase activity has been shown to increase in rats (Yakovlev and Yakovleva, 1971). In humans, training with intensive exercise has increased the activity of myofibrillar ATPase (Thorstenson *et al.*, 1975).

Myofibrillar Ca^{2+}-ATPase activity is increased in the soleus muscle after sprint, interval, or strength training. In the white quadriceps the same changes have also been found after continuous running in addition to the effect obtained after sprint, interval, and strength training (Fig. 2). The greatest increase has been caused by strength training in glycolytic muscle. The effect of repeated short-term intensive running (sprint and interval training) was more pronounced than after continuous running. Continuous swimming caused a decrease in enzyme activity (Viru, 1992).

In humans the effect of strength training on the activity of myofibrillar ATPase is variable: showing both an increase (Costill *et al.*, 1979a) and a decrease (Tesch *et al.*, 1987). Tesch *et al* (1990) also showed that neither concentric nor combined eccentric and concentric resistance training caused any change in Mg^{2+}-ATPase activity.

Thus, training may induce two different adaptations at the level of myofibrillar ATPase. One of them consists in an elevation of the enzyme activity, making possible a rapid liberation of energy for muscular contraction at the level of a high-power output. This adaptation seems to be common for sprint and interval training as well as for strength

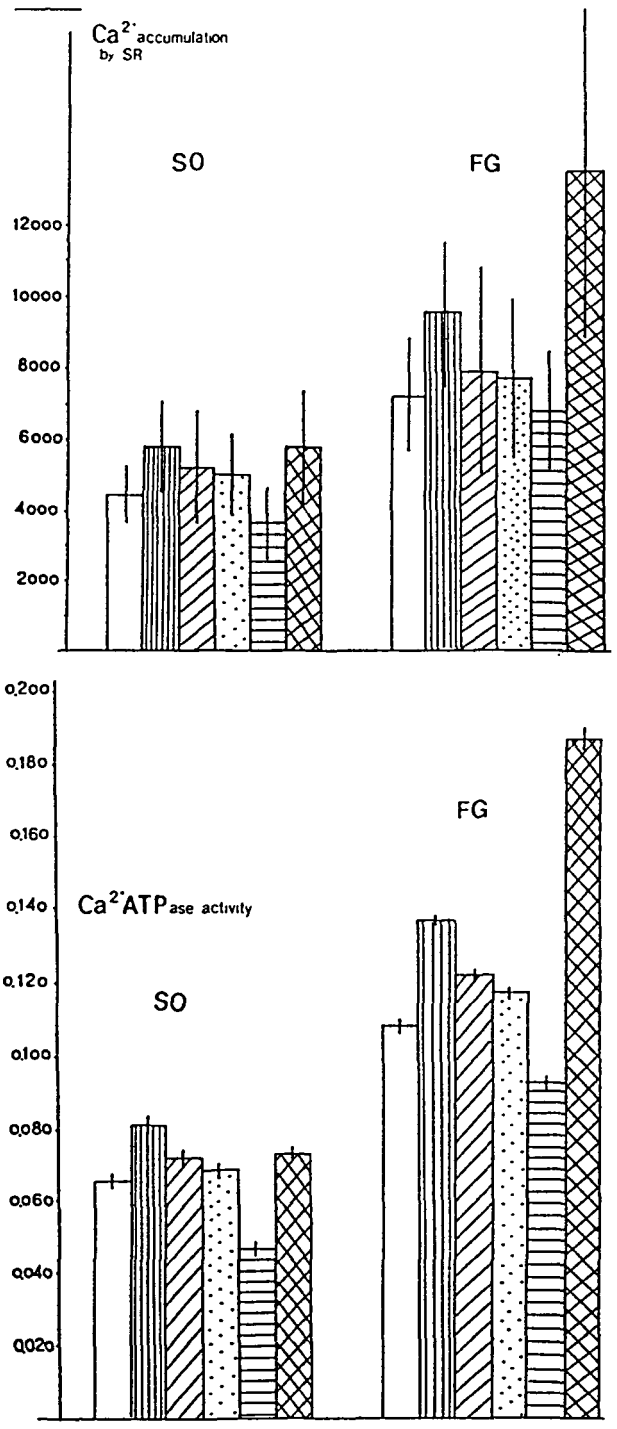

FIGURE 2 Effect of training by sprint (▥), anaerobic interval (▨), aerobic continuous running (▤) or swimming (▤), or fast climbing (▩) on Ca^{2+} accumulation by SR (top) and myofibrillar Ca^{2+} ATPase activity (bottom) in rats. Ca^{2+} accumulation was evaluated by cpm and Ca^{2+} ATPase activity by micromoles of released phosphate per milligram of protein in 1 minute. (Mean ± SEM.) □ sedentary control.

training if we do not consider the negative result of the study mentioned before. The second adaptation taking place is that to continuous exercise of moderate intensity. A decreased myofibrillar ATPase may be considered essential for more economical utilization of ATP stores.

Characteristics of Contraction

Strength training induces a change in the force-velocity relationship of muscle (Häkkinen, 1989). After heavy resistance training, an increase in the maximal voluntary force was most pronounced at a slow velocity of contraction (Caiozzo *et al.*, 1981; Coyle *et al.*, 1981; Häkkinen and Komi, 1985a). After explosive strength (power) exercise, the improvement was greater in the high-velocity portion of the curve (Coyle *et al.*, 1981; Häkkinen and Komi, 1985b).

The time to production of a 30, 60, or 90% force level was also found to be shorter for wrestlers and bodybuilders than for power lifters (MacDougall *et al.*, 1977). These differences were explained from the various training practices followed by each group. The training of a bodybuilder and especially that of a wrestler involves submaximal lifting at a higher speed, whereas training of the power lifter involves high resistance, slow contraction velocity exercise.

A program of sprint training shortened the time to peak tension of the rat soleus muscle but did not alter the contractile properties of the fast-twitch rectus femoris muscle (Staude *et al.*, 1973). Treadmill endurance training resulted in a 14% decrease in the time to peak tension of the rat soleus muscle (Fitts and Holloszy, 1977) and increased the ability to maintain tetanic tension during a series of fatiguing contractions (Barnard *et al.*, 1970; Fitts and Holloszy, 1977).

MITOCHONDRIA

Typical of endurance training is the augmented number and volume of mitochondria observed (Bylund *et al.*, 1977; Gollnick and King, 1969; Hoppeler *et al.*, 1985; Howald, 1976; Krause *et al.*, 1969; Yakovlev *et al.*, 1972) and the increased activity of oxidative enzymes (Holloszy, 1967, 1973; Holloszy and Booth, 1976; Holloszy *et al.*, 1970; Kraus *et al.*, 1969; Palladin, 1935; Saltin and Gollnick, 1983; Yakovlev, 1949, 1975, 1976, 1977). A preferential proliferation of subsarcolemmal mitochondria in comparison with interfibrillar mitochondria has also been found (Hoppeler *et al.*, 1973, 1985; Müller, 1976; Rösler *et al.*, 1985).

Increased activity of the enzymes of β-oxidation (Baldwin *et al.*, 1972; Costill *et al.*, 1979b; Holloszy, 1973; Holloszy and Coyle, 1984; Hoppeler *et al.*, 1973; Jansson and Kaijser, 1977; Kraus *et al.*, 1969) as well as a general enhancement of oxidative potential of muscle fibers (Fitts *et al.*, 1975; Gollnick and Saltin, 1982; Gollnick *et al.*, 1985; Saltin and Gollnick, 1983) makes possible an elevated use of lipid during prolonged exercise, even despite a high level of muscle lactate (Green *et al.*, 1979; Holloszy, 1973, Yakovlev, 1977).

When endurance exercise is sufficiently intense, the increase in mitochondrial enzymes occurs somewhat in parallel in all fiber types in the muscle (Baldwin *et al.*, 1972; Terjung, 1976). Subsequently, glycolytic fibers become "more oxidative" (Holloszy and Coyle, 1984). This fact is related to the peculiarity in motor unit recruitment during prolonged exercise. Endurance exercise at approximately 60% maximal oxygen consumption rate

($\dot{V}O_{2max}$) is initially performed through involvement of the activity of slow-twitch motor units. As the exercise continues, there is a progressive recruitment of fast-twitch motor units. If exercise is carried out until exhaustion, all the motor units in a muscle may be utilized (Gollnick *et al.*, 1973b, 1974). However, if the training exercise is moderate in intensity and duration, a difference may be found in the enzymatic profile between various types of muscle fiber, such as the β-hydroxybutyrate dehydrogenase activity increased 2.6-fold in SO and sixfold in FOG fibers, whereas no change could be detected in FG fibers (Winder *et al.*, 1974). An endurance training effect on carnitine palmityltransferase activity has been found in slow-twitch but not in fast-twitch fibers of the gastrocnemius muscle (Tikkanen *et al.*, 1989).

Training for improved strength usually does not cause a significant change in mitochondria number (Costill *et al.*, 1979a; Howald, 1982; Hoppeler, 1986; MacDougall *et al.*, 1977, 1979). Even a significant reduction has been found in mitochondrial volume density following strength training (MacDougall *et al.*, 1977, 1979, 1980). The mitochondrial density is reduced also in the muscles of bodybuilders and power lifters (MacDougall *et al.*, 1982). In some reports an increased activity of enzymes catalyzing oxidative processes has been reported (Komi *et al.*, 1978). However, these findings are not consistent (Häkkinen *et al.*, 1981; Houston *et al.*, 1983; Tesch *et al.*, 1990). Decreased activity of citrate synthetase has been found in vastus lateralis muscle after 6 months of strength training (Tesch *et al.*, 1987).

A comprehensive study performed by Dundley and coworkers (1982) on rats used the concentration of cytochrome c as an index of mitochondrial adaptation. In rats running at speeds of 10 or 20 $m \cdot min^{-1}$ (final duration of exercises from 30 to 90 minutes) no change in cytochrome c concentration of FG fibers was observed. When the running velocity was 30, 40, 50, or 60 $m \cdot min^{-1}$, the training effect appeared despite the decrease of final exercise duration to 5 to 27 minutes (Fig. 3). In FOG fibers an increased cytochrome c concentration was found at all exercise intensities. The training effect increased with running velocity up to 30 $m \cdot min^{-1}$ and then leveled off at a steady-state level. In SO fibers the training effect was negligible when the running velocity was 10 $m \cdot min^{-1}$. The highest effect was at a running velocity of 30 or 40 $m \cdot min^{-1}$. Likely, the variable effect found in different types of fiber was related to the extent of motor unit recruitment, depending on exercise intensity. An important point for rats is that at a running velocity of 30 $m \cdot min^{-1}$ rats use 83% of their $\dot{V}O_{2max}$ at this intensity (Shephard and Gollnick, 1976). This percent value seems to be close to their anaerobic threshold. If this is the case, then from the anaerobic threshold onward:

1. Training becomes effective in increasing oxidation potential in FG fibers
2. The training effect levels off in regard to FOG fibers
3. A further increase in exercise intensity reduced the training effect in SO fibers

The abbreviated running time in rats when exercise intensity was inordinately high (100 $m \cdot min^{-1}$) yielded a minimal or no mitochondrial change (Hickson *et al.*, 1975; Saubert *et al.*, 1973). Accordingly, in man sprint exercise does not improve the oxidation capacity of the muscle (Fournier *et al.*, 1982; Macková *et al.*, 1986; Sharp *et al.*, 1986). However, some studies indicate that a modest adaptation of mitochondrial enzyme is not excluded following sprint training (Jacobs *et al.*, 1987; Saltin *et al.*, 1976; Thorstensson *et al.*, 1975). The possibility of a positive effect of sprint training seems to be related to the intermittent character of sprint exercises when a short period of activity with great power output is

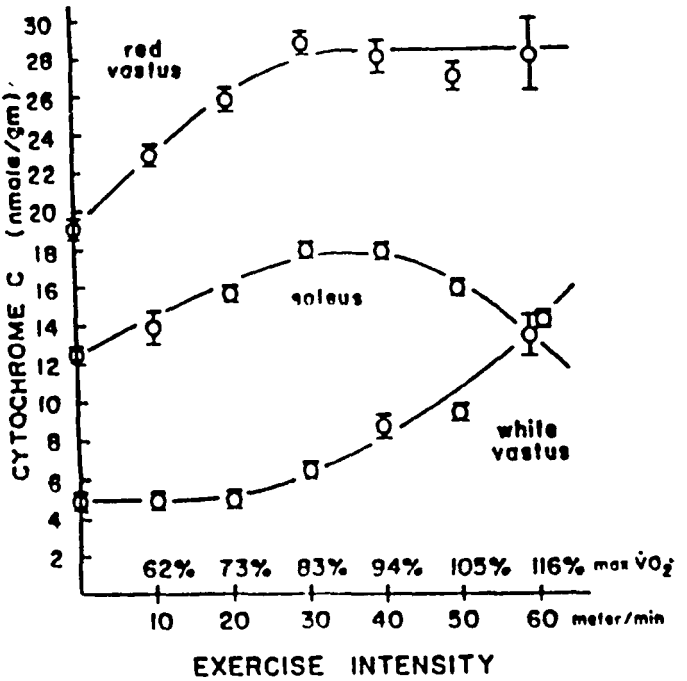

FIGURE 3 Effect of training on cytochrome c concentration in various muscles showing its dependence on training intensity, indicated on the abscissa by percent $\dot{V}O_{2max}$ or running velocity $m \cdot min^{-1}$. Reprinted with permission from Dudley *et al.*, 1982.)

followed by a more prolonged recovery period for restoration of the muscle concentration of high-energy phosphate furnished from oxidation energy.

In this regard it is understandable that anaerobic interval training increases the activity of the enzymes catalyzing both anaerobic glycogenolysis and oxidation processes (Pfister *et al.*, 1981). A greater mitochondrial effect has been found in a low-power compared with a high-power interval training program (Hickson *et al.*, 1975).

ENZYMES OF ANAEROBIC PATHWAYS OF ADENOSINE TRIPHOSPHATE RESYNTHESIS

Creatine Kinase and Myokinase

Animals adapted to rapid dashes possess a high percent of fast-twitch fibers and a very high activity of muscle creatine kinase, myokinase, and glycolytic enzymes in FG fibers (Hochachka and Somero, 1984). It might be expected that sprint training also increases the activity of these enzymes. However, the results of corresponding studies vary. In rats sprint training induced an increased activity of creatine kinase in the soleus muscle but not in the rectus femoris muscle (Staude *et al.*, 1973). Thus, the enzyme activity was stimulated only in SO fibers, which possess a lower activity of the enzyme than other fiber types. However, in another study no changes were found in SO, FOG, and FG fibers as a result of a sprint training (Gillespie *et al.*, 1982). No change was found either as a result of daily prolonged

exercise of moderate intensity (Gillespie *et al.*, 1982; Oscai *et al.*, 1971). Both creatine kinase and myokinase activity remained constant after daily exercise of high intensity (Bagby *et al.*, 1972).

In humans sprint training did not change the activity of muscle creatine kinase activity (Jacobs *et al.*, 1987). When fast maximal contractions were repeated five to eight times with brief rest intervals, creatine kinase and myokinase activity in muscle increased (Thorstensson *et al.*, 1975). Another program of strength training did not cause such a change, however (Thorstensson *et al.*, 1976). In SO fibers endurance training increased the creatine kinase MB isozyme content of muscle (Apple and Tesch, 1989; Jansson and Sylvén, 1985).

Glycolytic enzymes

In exercise lasting more than 30 seconds and less than 5 minutes anaerobic glycogenolysis becomes critical for ATP resynthesis. In the practice of sports training the main tool for improvement of anaerobic working capacity is interval training. In a number of biopsy studies adaptation of glycolytic enzymes due to interval training has been found (Green *et al.*, 1979; Komi *et al.*, 1977; Pfister *et al.*, 1981; Roberts *et al.*, 1982). An increase has been detected in the activity of phosphorylase, phosphofructokinase (PFK), glycerolaldehyde phosphate dehydrogenase, lactate dehydrogenase (LDH), and malate dehydrogenase (Roberts *et al.*, 1982).

Interval training methods used on rats indicate that in the case of a more moderate program the effect on phosphorylase, PFK and pyruvate kinase (PK) is shown only in SO fibers (Baldwin *et al.*, 1977; Staude *et al.*, 1973). In more strenuous forms of interval training phosphorylase, PFK, and PK activity increased in FG and FOG but not in SO fibers (Gillespie *et al.*, 1982).

Sprint training differs from interval training by being an exercise less able to be carried on for a prolonged period (10 to 20 second dashes), carried out at almost the highest possible intensity of exercise, and necessarily followed by a prolonged rest interval between repetitions. In humans an increased PFK activity (Fournier *et al.*, 1982; Jacobs *et al.*, 1987; Sharp *et al.*, 1986), but constant phosphorylase activity (Sharp *et al.*, 1986) were found following sprint training. In rats sprint training increased the PFK activity in FOG and FG but not in SO fibers; the activity of LDH did not alter (Gillespie *et al.*, 1982).

Endurance training with continuous exercises does not change PFK activity in SO fibers, but decreases the activity in FOG and FG fibers (Tikkanen and Härkönen, 1989). In another similar study PFK activity did not change and LDH activity decreased in all types of muscle fibers (Gillespie *et al.*, 1982). A small but significant reduction in LDH activity of SO and FG fibers has been reported after both sprint and endurance training (Hickson *et al.*, 1975). The muscle of a sprinter and jumper contains a relatively high percent of LDH_{4-5}, whereas muscle of an endurance athlete is high in LDH_{1-2} (Apple and Rogers, 1986; Sjödin *et al.*, 1976).

Strength training usually does not produce an alteration in muscle activity of phosphorylase, PFK, and LDH (Costill *et al.*, 1979a). Shot-putters, weightlifters, and discus throwers have a muscle phosphorylase, PFK, and LDH activity well within the range of sedentary subjects, whereas sprinters, jumpers, and runners of middle distances usually have an elevated muscle concentration of these enzymes (Costill *et al.*, 1976).

The turnover rate of the muscle glycolytic enzymes lies between one-half hour and a few days (Illg and Pette, 1979). Therefore any observation about the effect of training on

glycolytic enzymes is influenced by the time at which the tissue sample was obtained after the final training session. A principal question arises as well: Does the adaptation of a muscle glycolytic enzyme signify an increased number of enzyme molecules or an enhanced sensitivity of a rapidly renewing enzyme under the regulatory influence of exercise? The latter possibility is underlined by the following results.

In rat muscle PFK activity decreased as a result of a 10-week period of interval training or continuous aerobic training both in SO and FG fibers. Sprint training also caused this change in SO but not in FG fibers. Since 48 hours passed after the final training session before a muscle sample was obtained, the time elapsed was enough to suggest that a rapid enzyme turnover eliminated the increased enzyme activity. However, an important result of this study also was the fact that 4 minutes of intensive running (at 60 $m \cdot min^{-1}$) changed the muscle PFK activity differently, depending on the training regimen used. In untrained control rats the test exercise induced a decrease of the muscle enzyme activity in oxidative muscle. In glycolytic muscle the activity did not change. Instead a two- to threefold increase was found in muscle of rats trained both by interval or continuous running (Fig. 4). The effect of sprint training was different: The effect of a test exercise was to decrease muscle enzyme activity in both types of fibers, but in SO fibers the change was greater than in the control group (Viru, 1993).

There are a great number of factors controlling muscle PFK activity. The enzyme concentration itself may also modulate the activity: A cell with a high enzyme concentration exhibits a higher activity than could be expected from the simple linear relationship between enzyme concentration and activity (Bosca *et al.*, 1985). It may be speculated that a training-induced increase in the enzyme concentration sensitizes the enzyme activity to stimulatory factors. This explanation implies that the enzyme sensitivity to inhibitory factors is also increased. Thus, despite the elevated number of enzyme molecules, the activity was reduced in the resting condition. The end conclusion of the obtained results points to an enhanced effectiveness of the regulation of PFK activity in the organism, trained by interval or continuous exercise. Newsholme (1986) suggests that one reason for the remarkable performance of elite sportsmen is the fact that their metabolic control mechanisms are so well developed that they provide maximum sensitivity when required in the control of the energy-producing pathways in muscle.

It is a rather complicated task to test the hypothesis on the training-induced changes in regulation of PFK activity, since to determine increased molecular activity the enzyme has to be purified and thus removed from its normal operating milieu with cofactors.

The specific adaptation to anaerobic exercise may concern the formation of isozymes less sensitive to a lowered pH value. However, this question has only been investigated in regard to hexokinase and only in one study (Goldberg, 1985). The increase of total hexokinase activity has been established in rats after various kinds of training in all fiber types (Baldwin *et al.*, 1973; Barnard and Peter, 1969; Sauber *et al.*, 1973, Yakovlev, 1975, 1976, 1977). An interval training program (training sessions caused an increase of lactate concentration in blood up to 18 to 22 $mM \cdot 1^{-1}$ and decrease of blood pH to 6.89 to 6.90) resulted in an increase of hexokinase activity in the range of medium pH from 8.0 to 6.5 This change was found in both SO and FG fibers as well as in brain tissue. In FG fibers the enzyme activity increased most of all at pH 6.5. After training with continuous exercise, the increase in the enzyme activity was also found at various medium pH, but at pH 6.5 a predominating increase was not observed. In association with these events, an augmentation of muscle type hexokinase II was detected in muscle and brain tissue (Goldberg, 1985).

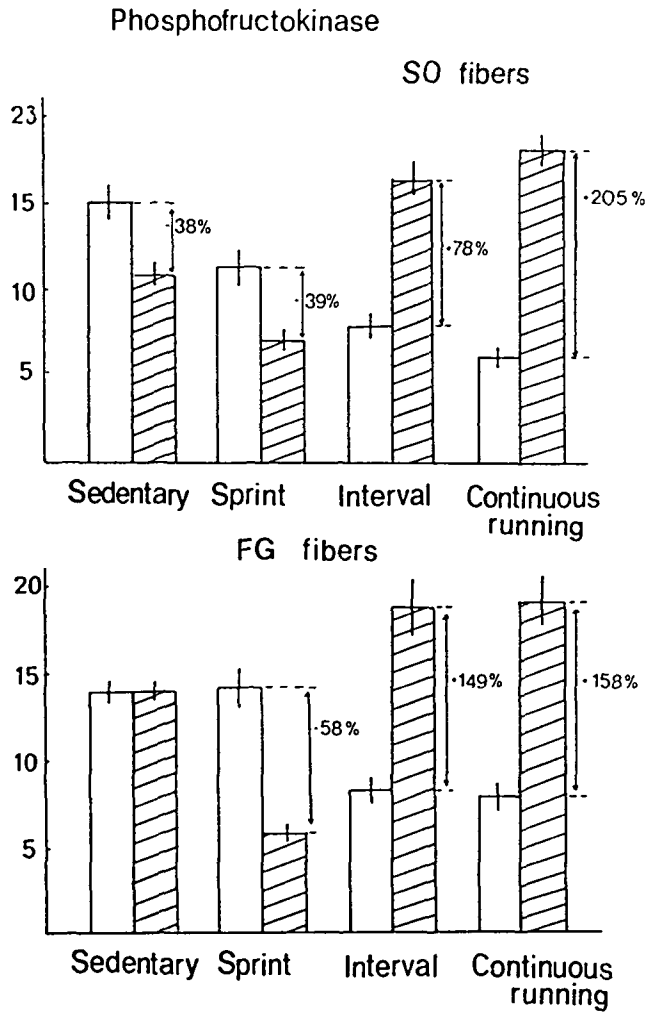

FIGURE 4 Effects of sprint, interval, or continuous running on phosphofructokinase activity in the resting state (□) and after a 4-minute test running at 60 $m \cdot min^{-1}$ (□) in rats (Viru, 1992). Phosphofructokinase activity is evaluated by the change in optical density during 30 seconds per 1 g protein. (Mean ± SEM.)

Buffer Capacity

A significant result of anaerobic interval training as well as of sprint training is an increased buffer capacity of skeletal muscle (Parkhouse and McKenzie, 1984; Sharp *et al.*, 1986). Accordingly, a runner for 800 m has a significantly higher muscle buffer capacity than the untrained person or marathon runner (McKenzie *et al.*, 1983). However, in regard to this question, more information is necessary.

ENERGY STORES AND MYOGLOBIN

Adenosine Triphosphate

There is no convincing evidence that training increases the ATP store in muscle. No changes have been detected in rat muscle with either endurance or strength training (Yakovlev, 1976, 1977). A mild increase in ATP concentration has been reported in the limb muscle of the adolescent (Eriksson *et al.*, 1973) and adult (Karlsson *et al.*, 1972) male after a program of endurance training. However, these reports seem to describe the initial effect of training.

Phosphocreatine

Rats trained by repeated short-term intensive exercise increased the phosphocreatine content in skeletal muscles, but the effect of continuous exercise was only modest (Yakovlev, 1976, 1977). Some of the human biopsy studies confirmed these changes (Eriksson *et al.*, 1973, Karlsson *et al.*, 1972). However, the increased phosphocreatine store is not considered as a common result of training (Saltin and Gollnick, 1983).

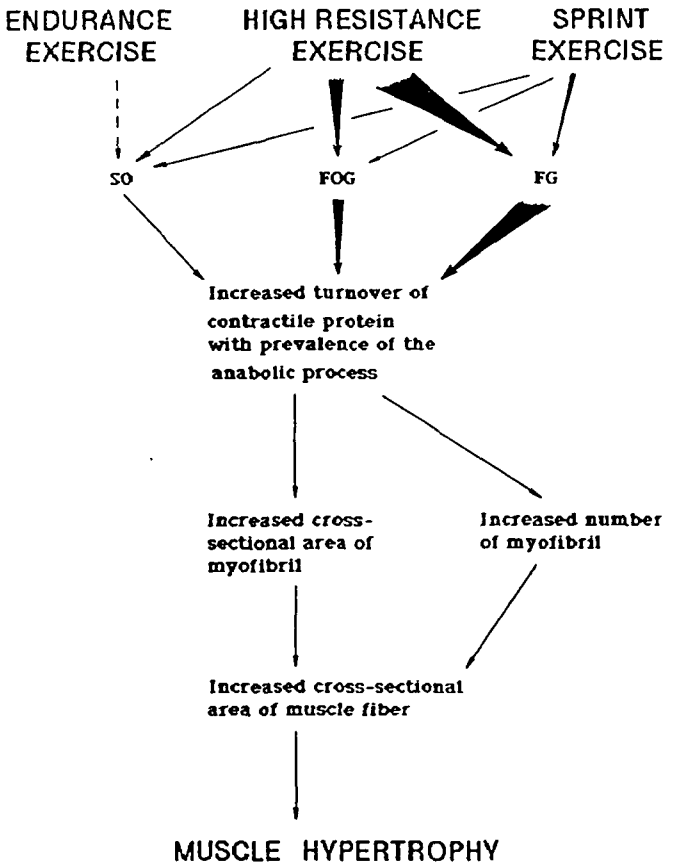

FIGURE 5 Exercise-dependent specificity of effect on muscle hypertrophy and hyperplasia.

Glycogen

The training effect on muscle glycogen store has been known since 1927 (Embden and Habs, 1927; Saltin and Gollnick, 1983; Yakovleva, 1977). In rats no difference was found between training with continuous exercise and interval training, but the effect of high-power exercise was less pronounced (Yakovlev, 1975, 1976, 1977). In one study, no effect of sprint training on the glycogen content of any of the fiber types was found (Saubert *et al.*, 1973). A comparison of various kinds of training effects confirmed that sprint training does not alter the glycogen store of either SO and FG fibers. Aerobic training with continuous running or swimming was highly effective in this process. In SO fibers these effects were more pronounced than the response to interval or strength training. In FG fibers all the training variants produced approximately the same change. In this study, glycogen compartmentalized in the SR was detected, too. It increased as a result of all the training variants used, including the sprint training that was otherwise ineffective in regard to inducing the total glycogen increase. The largest increase in glycogen compartmentalized to the SR resulted from interval training both in SO and FG fibers (Viru, 1993).

In humans a higher value of muscle glycogen storage in trained than sedentary individuals was demonstrated repeatedly from the time of the first biopsy studies (Hermansen *et al.*, 1967; Hultman, 1967): subjects undergoing strength, sprint, or endurance training possess a larger store of muscle glycogen (Gollnick *et al.*, 1973a; Karlsson *et al.*, 1972; MacDougall *et al.*, 1977).

Triglycerides

In humans an increase in the triglyceride content of the quadriceps muscle was detected after endurance training (Morgan *et al.*, 1969). This change was not confirmed in a one-leg training experiment (Henriksson, 1977).

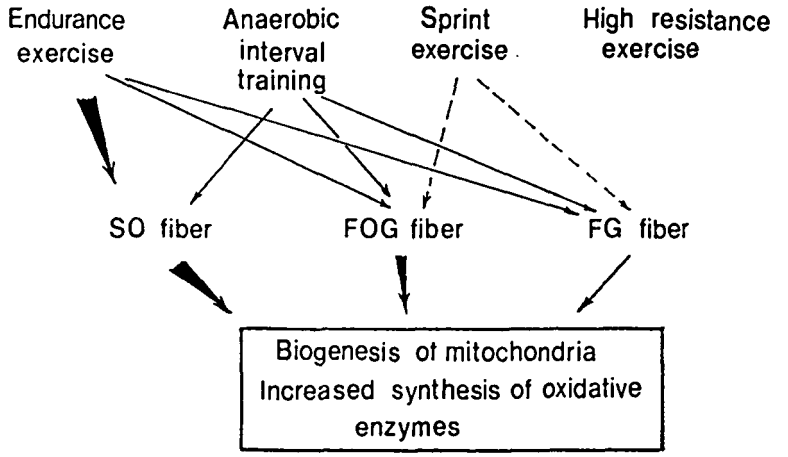

FIGURE 6 Exercise-dependent specificity of effect on mitochondrial proliferation.

Myoglobin

The endurance training increased the concentration of myoglobin in skeletal muscles of rats (Hickson, 1981; Lawrie, 1983; Pattengale and Holloszy, 1967; Yakovlev, 1975, 1976, 1977). In contrast, biopsy studies have not demonstrated an increased myoglobin content in endurance trained humans (Jansson *et al.*, 1982, Svedenhag *et al.*, 1983).

CONCLUSIONS

Muscle fibers are capable of the synthesis of various proteins. Realization of this possibility depends on the inductive stimulus. It seems that various adaptive changes are induced in muscle fiber by different types of exercise. The inductors generated by endurance exercise do not influence the genes responsible for an increase in the amount of myofibrillar protein (Fig. 5), whereas inductors generated by high-resistance exercise do not stimulate the genes related to an increase in the synthesis of mitochondrial protein (Fig. 6). Between these two main directions of training effect is the influence of power, sprint, and anaerobic interval training (Fig. 7). In these cases, adaptation in the muscle fiber is related, in the first

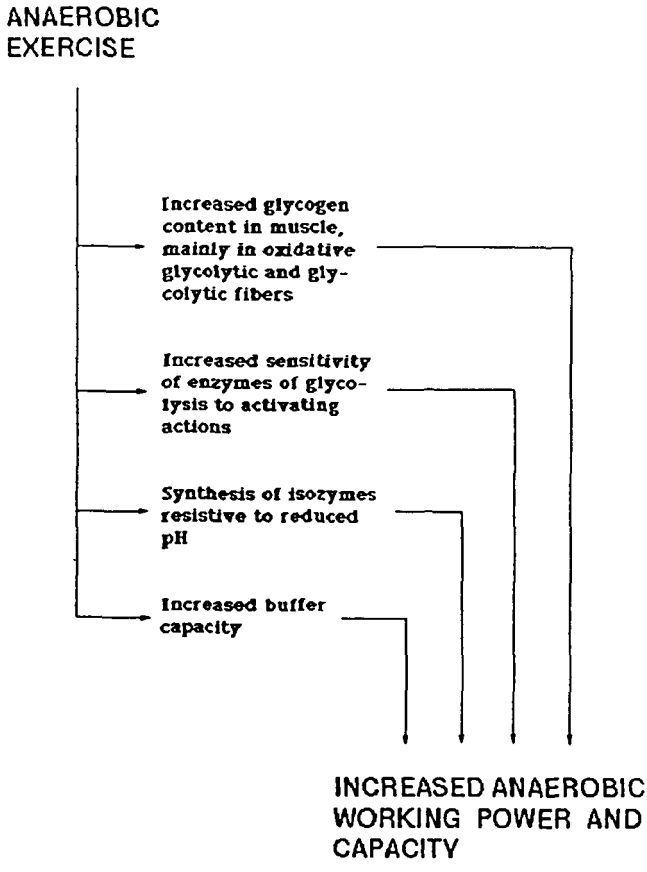

FIGURE 7 Adaptation to anaerobic exercise.

instance, to the SR and enzymes of anaerobic metabolism. Myofibrillar hypertrophy is pronounced only modestly if at all in these cases.

The effect of training on muscle fibers of various types depends on their involvement during training.

The stimulus for muscular hypertrophy depends on the resistance to muscle contraction as well as the total number of contractions performed against high resistance. The stimulus for the mitochondrial adjustment depends: (1) on the total period of persisting high level oxidation in skeletal muscle, including the time for contractile activity, and the time for restitution based on a high oxidation rate; and (2) on the oxidation rate during these periods (the closer to maximum of the oxidation rate, the more effective is the training).

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