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MALLEABILITY OF THE MOTOR SYSTEM: TRAINING FOR MAXIMIZING POWER OUTPUT

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SUMMARY

The training programmes used to improve the strength and/or endurance capacities of athletes are described. Specific stimuli are either high tension in contracting myofibrils or high turnover rates in the oxidative metabolism of the muscle cell. Structural adaptations consist of the synthesis of additional myofibrillar material or increased volume density of both interfibrillar and subsarcolemmal mitochondria. Transformation of fibre types at the level of the molecular structure of myosin seems possible with high-intensity training of long duration. Functional adaptations consist of higher activities of important enzymes in either anaerobic or aerobic energy metabolism and of larger intracellular stores of the respective substrates. All adaptations are highly specific and reversible through detraining or immobilization of the muscle.

Power output is very high in international athletes and their performances are still improving in most sports. Such improvement is possible because of the remarkable malleability of the human motor system in response to highly specific training methods. Skeletal muscle of man can adapt to the needs for either short maximum effort (e.g. weightlifting, sprinting, etc.) or prolonged exercise (e.g. long-distance running, cycling, cross-country skiing, etc.). In many sports, however, performance capacity depends on both strength and endurance of the muscles involved (e.g. in rowing, middle-distance running, swimming and ball games), whereas in others the success of an athlete is mainly a question of optimized neuromuscular control (e.g. in gymnastics and figure skating). At the level of the muscle cell, training-induced adaptations occur in structure as well as in metabolic function and the changes are highly specific to the particular training stimulus. Some of the cellular adaptations are accompanied by adaptive processes in the cardiovascular and respiratory system, since skeletal muscle must be provided with both energy-supplying substrates and oxygen during exercise of more than one or two minutes.

Key words: Strength *vs* endurance, mitochondria, transformation of muscle fibre types.

ADAPTATIONS INDUCED BY STRENGTH TRAINING

Increases in muscle strength can be best obtained following isotonic work against heavy resistances involving six to ten repetitions before the muscle or the muscle group involved is exhausted. Each training session should include two or three of these working series per muscle group and the sessions should be repeated every second day. As soon as the muscle can perform more than ten repetitions, as a result of adaptation, the resistance must be readjusted to the new performance capacity. The training stimulus consists of contracting the muscle fibres at nearly maximal voluntary intensity, thus creating considerable tension in the myosin to actin crossbridges of the myofibrils. Training-induced changes in the ultrastructural composition of muscle can be investigated using the needle biopsy technique, electron microscopy and morphometric analysis (Weibel, 1979). In a recent experiment we found a 15% increase in peak torque of M. quadriceps femoris at $60° s^{-1}$ angular velocity for knee extension in previously untrained adolescents after only 6 weeks of participation in such a training programme. The muscle mass of the thigh (evaluated by morphometric analysis of computed tomography) had increased by 11%. The same percentage increase was calculated for the absolute volume of myofibrils, whereas the volume density of mitochondria decreased significantly (Table 1), confirming the results of a previous study (MacDougall *et al.* 1979). Thus, strength training stimulates the muscle cell to synthesize myofibrillar material and to increase its cross-sectional area (Häggmark, Jansson & Svane, 1978), but does not improve the muscle's oxidative capacity.

The fibre type distribution in M. vastus lateralis is not significantly influenced by a few weeks of strength training. Nevertheless, we found some changes in the myosin light chain pattern of isolated type I fibres, which became more similar to type IIC fibres after 6 weeks of strenuous heavy resistance training (Fig. 1). The IIC fibres are considered to be a transformational type between type I and type IIA fibres, since they

Table 1. *Morphometric analysis of M. vastus lateralis before and after strength training (own unpublished results)*

	Strength training	
	Before	After
M. vastus lateralis		
$Vv_{(myo, f)}$	0·8523 ± 0·0277	0·8644 ± 0·0155
$Vv_{(mt, f)}$	0·0552 ± 0·0095	0·0499 ± 0·0081*
$Vv_{(lip, f)}$	0·0033 ± 0·0019	0·0034 ± 0·0023
$Vv_{(cyt, f)}$	0·0891 ± 0·0241	0·0823 ± 0·0129
% Type I fibres	64 ± 4	66 ± 3

$Vv_{(myo, f)}$, volume density of myofibrils.
$Vv_{(mt, f)}$, volume density of total mitochondria.
$Vv_{(lip, f)}$, volume density of intracellular lipid.
$Vv_{(cyt, f)}$, volume density of residual sarcoplasmic components.
Values are means and standard deviations.
* Significant, $2P < 0·05$, paired *t*-test.

contain both slow and fast myosin (Billeter *et al.* 1980; Billeter, Heizmann, Howald & Jenny, 1981).

On the functional side, the cellular stores of energy-rich phosphates and glycogen are larger after strength training (MacDougall, Ward, Sale & Sutton, 1977), and the activities of the enzymes needed for splitting of these substrates are higher than in untrained muscle (Costill *et al.* 1979).

Strength training does not improve the functional capacity of either the cardiovascular or the respiratory system.

ADAPTATIONS INDUCED BY ENDURANCE TRAINING

A training programme designed to improve endurance capacity should clearly define intensity, duration and frequency of exercise. The most effective intensity seems to be in the range of power outputs eliciting 70–80 % of an individual's maximum oxygen uptake capacity ($\dot{V}_{O_2}$max) or 80–90 % of the maximal heart rate. Duration varies from 15 min per day in beginners to several hours in top athletes. Frequency should reach at least three times per week, best results being obtained with one or more exercise sessions every day. The training stimulus consists of accelerating the oxidative energy turnover of the muscle cell to a maximum without unduly stressing the anaerobic processes. A critical threshold is reached at the onset of blood lactate accumulation, which then starts respiratory compensation of exercise-induced metabolic acidosis.

The most important adaptation of the muscle cell to endurance training is a marked increase in the volume density of both interfibrillar and subsarcolemmal mitochondria (Hoppeler *et al.* 1973; Howald, 1976). The ultrastructural changes occurring in the muscle cells after 6 weeks of high-intensity bicycle ergometer exercise are listed in Table 2. The slightly higher mitochondrial volume density in untrained M. vastus lateralis of our strength training group (Table 1) compared to the endurance training group is most probably due to the lower percentage of mitochondria-rich type I muscle fibres in the latter. The mitochondrial volume in the M. vastus lateralis of top class cyclists was found to be around 12 % of the cell volume. This very high value is

Table 2. *Morphometric analysis of M. vastus lateralis before and after strength training (Hoppeler* et al. *1984)*

	Endurance training	
	Before	After
M. vastus lateralis		
$Vv_{(myo, f)}$	0·8279 ± 0·0236	0·7755 ± 0·0343*
$Vv_{(mt, f)}$	0·0474 ± 0·0030	0·0655 ± 0·0055*
$Vv_{(lip, f)}$	0·0068 ± 0·0017	0·0099 ± 0·0018
$Vv_{(cyt, f)}$	0·1057 ± 0·0374	0·1473 ± 0·0286
% Type I fibres	52 ± 4	56 ± 5

Symbols are explained in Table 1.
* Significant, $2P < 0·05$, paired t-test.

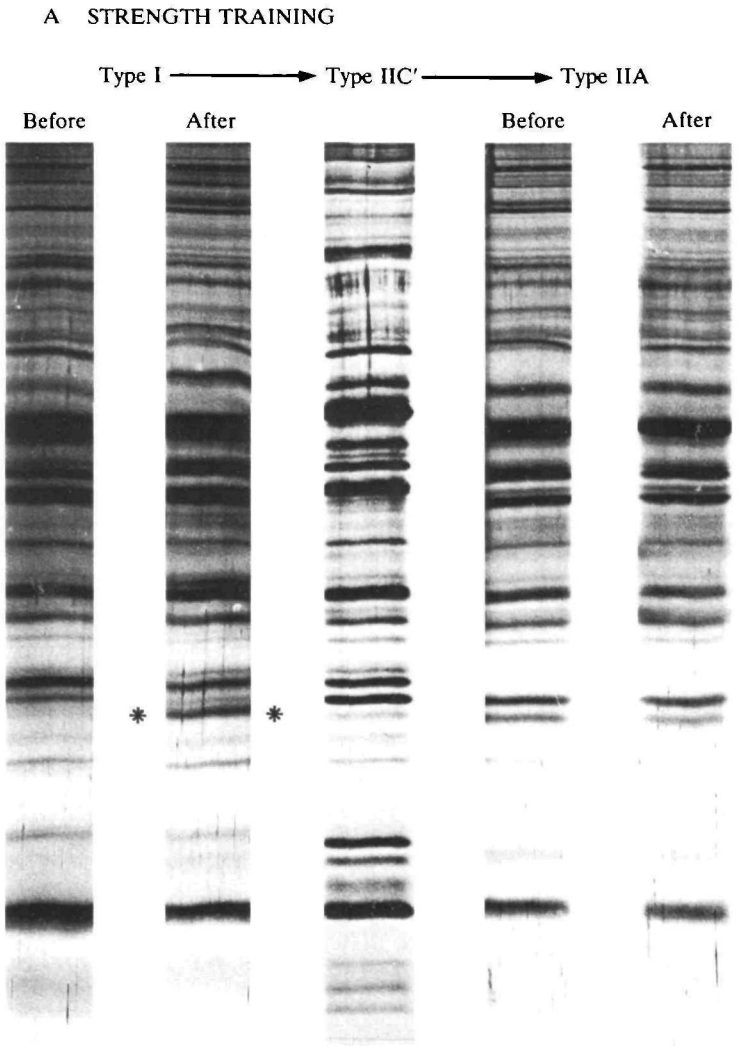

Fig. 1. One-dimensional SDS polyacrylamide gel electrophoresis (Baumann, Cao & Howald, 1984) of type I and type IIA muscle fibres before and after (A) strength or (B) endurance training. Asterisks indicate training-induced changes in the myosin light chain pattern of the different fibre types. Arrows indicate the direction of possible fibre type transformation.

mainly a result of a striking predominance of type I fibres in the muscles of these athletes who have been training for many years. Their muscles contain as much as 90–95 % type I fibres, but the largest mitochondrial volume density of isolated type I fibres does not exceed 14–15 % of the cell volume.

After endurance training, the volume density of mitochondria is increased not only in the slow-oxidative type I muscle fibres, but also in the fast-oxidative-glycolytic type

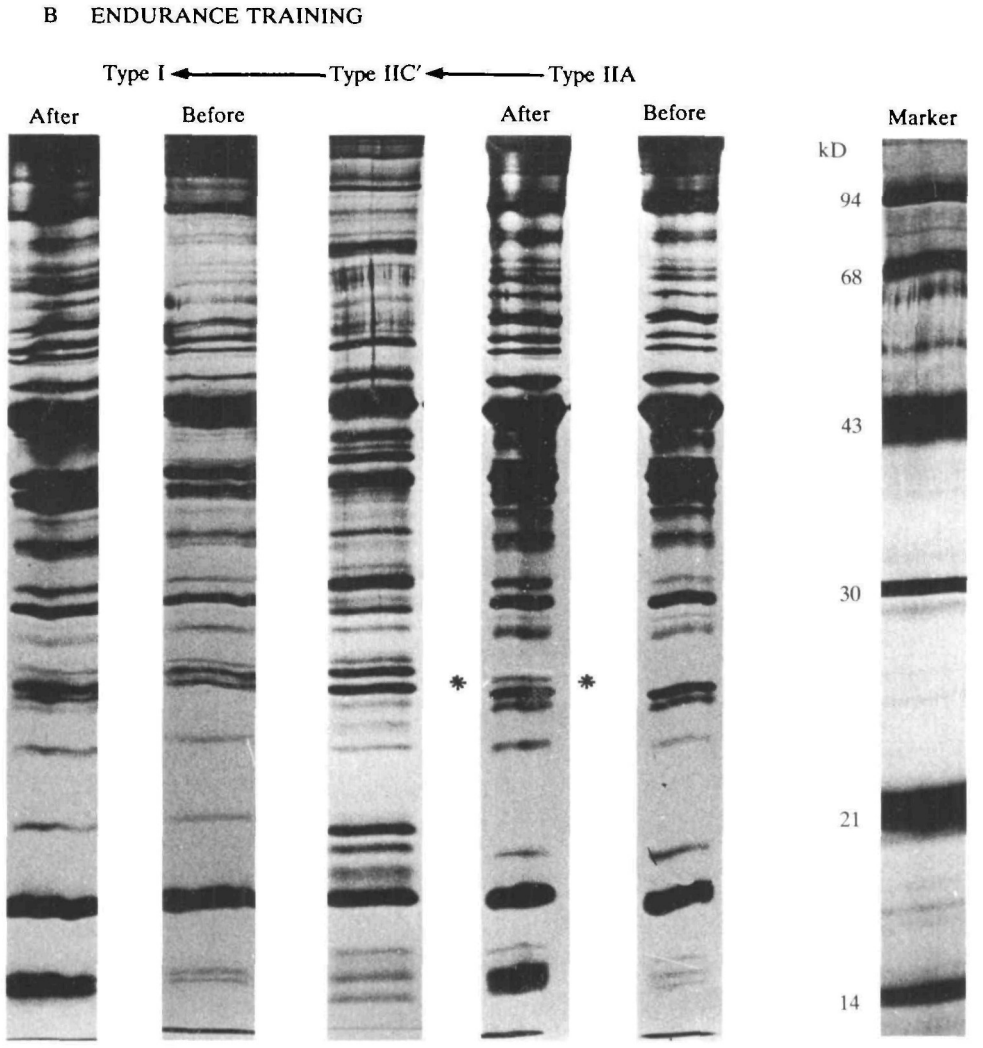

Fig. 1B

IIA and in the fast-glycolytic type IIB fibres (Table 3). The oxidative capacity of all three major fibre types is, therefore, improved and more power output can be generated without lactate accumulation since a smaller proportion of pyruvate must be converted to lactate (Hurley *et al.* 1984).

The activities of intramitochondrial enzymes involved in both citric acid cycle and respiratory chain are higher in endurance trained than in sedentary muscles (Holloszy, 1967). Intracellular lipid stores (Hoppeler *et al.* 1973; Oberholzer, Claassen, Moesch & Howald, 1976) and the activity of enzymes responsible for the

beta-oxidation of free fatty acids (Moesch & Howald, 1975; Green *et al.* 1979) are also increased as a result of endurance training, thus enabling the trained muscle cell to supply energy by using more fat and less glycogen as a substrate. Again, this phenomenon can be observed in the type IIA and type IIB fibres as well as in the type I fibres (Table 3). As a consequence, the activities of extramitochondrial enzymes involved in the anaerobic glycolysis are lower in the muscles of trained than untrained subjects (Moesch & Howald, 1975; Green *et al.* 1979). It is clear that these mechanisms also contribute to a lower lactate accumulation during exercise.

Endurance training does not influence the cross-sectional area of the muscle fibre nor its content of myofibrils. Transformation of type IIB into type IIA and of type IIA into type I fibres as a result of long-term, high-intensity training has been postulated in analogy to experiments with chronic low-frequency nerve stimulation (Salmons & Henriksson, 1981; Howald, 1982), but such a training-induced change of the molecular structure of myosin has only very recently been demonstrated in skeletal muscles of the rat (Green *et al.* 1984). Although the percentage of type I fibres did not significantly increase in our 6-week training studies, there were changes in the myosin light chain pattern of isolated type IIA fibres, which became more similar to the transitional type IIC fibre as a result of the training (Fig. 1). Whether a complete transformation of type IIA into type I fibres in human muscle can occur with training needs to be further investigated with exercise programmes lasting several months or even years.

Improvements in the oxidative capacity of the muscle cells are very closely matched by increases in capillary density (Zumstein, Mathieu, Howald & Hoppeler, 1983; Hoppeler *et al.* 1984) and in cardiac output (Ekblom & Hermansen, 1968). Thus, the capacity of the oxygen transport system is adjusted to the increased rate of oxygen utilization in the working muscle cell.

COMBINATION OF STRENGTH AND ENDURANCE TRAINING

In many sports the athlete needs both optimal muscle strength and very high oxidative capacity to produce a maximum power output for a certain time. The training

Table 3. *Morphometric analysis of the different fibres histochemically typed for ATPase before and after endurance training (Howald* et al. *1984)*

	$Vv_{(mt, f)}$		$Vv_{(lip, f)}$	
	Before	After	Before	After
M. vastus lateralis				
Type I fibres	0·0647 ± 0·0063	0·0844 ± 0·0095*	0·0065 ± 0·0038	0·0069 ± 0·0032
Type IIA fibres	0·0492 ± 0·0112	0·0708 ± 0·0169*	0·0027 ± 0·0015	0·0045 ± 0·0040
Type IIB fibres	0·0210 ± 0·0036	0·0338 ± 0·0094*	0·0010 ± 0·0011	0·0029 ± 0·0020*

Symbols are explained in Table 1.
* Significant, $2P < 0·05$, paired t-test.

programme must, therefore, stimulate the synthesis of both myofibrils and mitochondria in the muscle fibres involved. There is no biological reason to believe that the muscle cell is not able to do this. A larger volume of contractile elements can then be provided with sufficient energy from aerobic sources resulting in an optimum power output. Usually by empirism, the athletes and their coaches have developed many kinds of combined strength and endurance training programmes for their specific sport. The scientist can only try to explain what happens in the athlete's body and suggest modifications in case he feels that basic biological principles have not been observed. Best results are obtained when muscles are loaded as precisely as possible and this is why a cyclist's performance capacity cannot be improved by a runner's training programme and *vice versa*, although the same muscle groups seem to be working in both of these activities.

DETRAINING AND IMPAIRMENT OF OXYGEN SUPPLY

All the adaptational processes described in this article are induced by increased physical activity. Inactivity or immobilization of a muscle leads to a rapid loss of power output which is due to detraining effects at both the structural and the functional level. Fibre atrophy (Häggmark, Jansson & Eriksson, 1981) is a consequence of a decrease in myofibrillar volume content. The markedly increased mitochondrial volume density found after a few weeks of vigorous endurance training is reduced to nearly normal values within a short time after cessation of the training stimulus (Table 4). Mitochondrial volume density was also found to be in the normal range of M. gastrocnemius of patients who had to undergo leg amputation due to severe arterial occlusion. Thus, it seems clear that the trigger mechanism for increasing the volume density of mitochondria in muscle is not hypoxia but the exercise-related increase in oxidative metabolism of the cell.

The activities of mitochondrial enzymes involved in both carbohydrate and fat oxidation are decreased as a result of inactivity (Booth, Seider & Hugman, 1980) and the muscle's content of energy-rich phosphates and glycogen is also reduced

Table 4. *Morphometric analysis of M. vastus lateralis or M. gastrocnemius after detraining or arterial occlusion (unpublished data)*

	$Vv_{(mt,\ f)}$
M. vastus lateralis	
Before endurance training	0·0500 ± 0·0039
After endurance training	0·0693 ± 0·0086*
After detraining	0·0502 ± 0·0113*
M. gastrocnemius	
Patients with arterial occlusion	0·0564 ± 0·0074

Symbols are explained in Table 1.
* Significant, $2P < 0·05$, paired *t*-test.

(MacDougall *et al.* 1977). Type I muscle fibres are more severely affected by immobilization and there is a possibility that this fibre type is transformed into type IIC and IIA fibres as a result of complete inactivity (Häggmark *et al.* 1981).

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