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Biochemical and histochemical adaptation to sprint training in young athletes

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The purpose of the present study was to investigate the effects of 8 months of a specific and controlled sprint training programme on three groups of young athletes (two groups of males and one of females). Biopsies of vastus lateralis were taken before and after the period of training. The type percentage and diameter of the fibres, as well as the glycogen content and the activities of the enzymes of glycogen metabolism (glycogen synthase and glycogen phosphorylase), glycolysis (phosphofructokinase, pyruvate kinase, aldolase and lactate dehydrogenase), oxidative metabolism (succinate dehydrogenase) and creatine kinase and aminotransferases were studied. The results show an increase in the percentage of type I fibres and an increase in the diameter of both fibre types. A significant increase was also observed in glycogen content, and in the activities of glycogen synthase, glycogen phosphorylase, phosphofructokinase, pyruvate kinase, succinate dehydrogenase, aspartate aminotransferase and alanine aminotransferase. We conclude that a long period of sprint training induces a biochemical muscle adaptation to anaerobic exercise. This metabolic adaptation is followed by a morphological adaptation, although this is probably not as specific as the biochemical one.

Key words: fibre type, glycogen, glycolytic enzymes, human sprinters, muscle biopsy.

Muscle adaptation to physical training depends on the mode, intensity and duration of training. It is well documented that endurance training induces biochemical and morphological muscle fibre changes consistent with an increase of energy production via oxidative metabolism (Gollnick *et al.* 1972, 1973, Houston & Thomson 1977, Tesch & Karlsson 1985). On the other hand, fewer studies have been able to find peripheral adaptations to sprint training (Henriksson & Reitmann 1976, Saltin *et al.* 1976, Costill *et al.* 1979, Houston *et al.* 1981, Roberts *et al.* 1982). Possible explanations could be periods of training that were too short, or previous training of the athletes in other disciplines that interfere with the results, or even that slow fibres cannot change their characteristics in response to maximal exercise.

The aim of the present study was to determine if there is any consistent pattern of muscle adaptation after a long period of specific and well-controlled sprint running training (8 months) in a group of teenagers with previous training exclusively devoted to sprint or with no previous specific training.

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MATERIALS AND METHODS

Subjects. Sixteen young athletes, male and female, engaged in a training programme at the Escola Catalana de Velocitat (the athletics school in Barcelona) volunteered to participate in the study after being fully informed of the details, risks and possible hazards associated with the experimental protocol.

Table 1. Training programme for groups A and C

Predominant energetic system	Type of exercise	Training volume*	Training intensity (%)
Anaerobic system (ATP–PC†)			
Power	Multi-hops	3000 hops	98–100
	Multi-throws	1500 throws	98–100
	Speed (30–60 m sprint)	2 km	98–100
	Weight	155 t	98–100
	Muscular power	11,000 repetitions	98–100
Capacity	Speed (60–80 m sprint)	15 km	96–100
Anaerobic system (lactic)			
Power	100–150 m runs	3 km	98–100
Capacity	100–300 m runs	10 km	80–90
		7 km	90–100
	300–500 m runs	15 km	80–90
		10 km	90–100
Aerobic system (oxygen)	Continuous runs, repetition running (progressive), interval training	40 km	180 beats min^{-1}

* Total number of exercises performed in the 8 months.
† Phosphocreatine.

Written consent was obtained from all subjects, parents and instructors. The athletes were divided into three groups. Group A were females. Groups B and C were males. Groups A and B had followed a sprint training programme the previous year, while group C only began the training programme in the current year. The training programme was initiated in October and ended in May.

Age, height, body weight, skinfold and arm perimeter over the midportion of the triceps brachia were recorded for each subject. Approximately 30 mg of muscle samples from the non-dominant vastus lateralis was obtained using the needle biopsy technique (Bergström 1962). The first biopsy from each subject was obtained during the first week of October. The second one was obtained during the first week of June. The athletes did not train for 48 h before the biopsy.

Training programme. The training programme was anaerobic, and a high percentage of the time was devoted to promoting muscle strength. The programme was divided into three cycles, of which speed and strength were particularly emphasized in the first and the third. Aerobic capacity, power and general strength were developed in the second. Each cycle was followed for 1–2½ months. The training programme finished with a competition over different speed distances (100, 200, 400 and 800 m). Groups A and C trained especially for 100 and 200 m. Group B trained for 400–800 m. The training programme is shown in Tables 1 and 2.

Histochemical and morphometric studies. Two muscle specimens were obtained in each biopsy. One sample was frozen in isopentane precooled to the temperature of liquid nitrogen. Histological sections were stained with haematoxylin–eosin, PAS, oil red O, non-specific esterase and modified Gomori trichrome. They reacted with myofibrillar ATPase after preincubation at pH 4.3, 4.6 and 9.4, and NADH tetrazolium reductase (Dubowitz 1985). Micrographs of the ATPase stains were used to assess the diameter and surface area and type percentage of fibres with a computer-assisted planimeter. At least 150 fibres were evaluated in each case. The hypertrophy concept used is that defined by Dubowitz (Dubowitz 1985).

Biochemical studies. About 20 mg of muscle tissue was directly frozen in liquid nitrogen and stored at −80 °C until analysed.

Glycogen was extracted by alkaline treatment from about 10 mg of human muscle. The hydrolysis and measurement of glucose produced was carried out using the anthrone method (Carroll *et al.* 1956).

For the enzymatic analyses, 15 mg of muscle was homogenized in 30 volumes of ice-cooled extraction medium, using a Polytron (10 s at position 5). The extraction medium contained 50 mM HCl-Tris, 4 mM EDTA, pH 7. The preparation was centrifuged at 100 *g* for 20 min. One hundred microlitres of the supernatant was used to measure succinate dehydrogenase activity. The supernatant was centrifuged

Table 2. Training programme for group B

Predominant energetic system	Type of exercise	Training volume*	Training intensity (%)
Anaerobic system (ATP–PC†)			
Power	Multi-hops	3000 hops	98–100
	Speed (30–60 m sprint)	4.8 km	98–100
	Weight	155 t	98–100
	Muscular power	11,000 repetitions	98–100
Capacity	Speed (60–80 m sprint)	16 km	96–100
Anaerobic system (lactic)			
Capacity	300–500 m runs	10 km	80–90
Aerobic system (oxygen)	Continuous runs, repetition running (progressive), interval training	200 km	180 beats min^{-1}

* Total number of exercises performed in the 8 months.
† Phosphocreatine.

Table 3. Physiological characteristics of the young athletes

	Group A ($n = 5$)		Group B ($n = 3$)	
Sex	Female		Male	
Athletic training	100–200 m		400–800 m	
Previous training	Yes		Yes	
	Before	After	Before	After
Age	16±0.0	17±0.0	17±0.0	18±0.0
Weight (kg)	57±5.1	58.4±4.8	58.7±3.4	60.5±1.5
Height (cm)	168±4.8	169±5.4	173±9.9	173±11
Triceps skinfold (mm)	15.2±2.8	14.5±4	7.2±0.3	6.9±0.3**
Arm perimeter (cm)	24.9±2	25.2±1.9	26.2±1.6	27.0±1.7
	Group C ($n = 8$)		Total ($n = 16$)	
Sex	Male			
Athletic training	100–200 m			
Previous training	No			
	Before	After	Before	After
Age	15.6±0.5	16.6±0.5**	16±0.63	17±0.63**
Weight (kg)	64.5±0.7	66.0±8.5	61.1±7.0	62.6±7.3**
Height (cm)	173±6.2	1.74±6.0	172±7.1	173±7.1
Triceps skinfold (mm)	6.2±0.7	6.3±0.9	9.24±4.5	8.98±4.4
Arm perimeter (cm)	25.0±1.8	25.5±1.3**	25.6±1.8	26.2±1.7**

Values are means±SD. * $P < 0.05$, ** $P < 0.01$.

Table 4. Glycogen and enzyme activities in skeletal muscle of the athletes

	Group A ($n = 5$)		Group B ($n = 3$)		Group C ($n = 8$)		Total ($n = 16$)	
	Before	After	Before	After	Before	After	Before	After
Glycogen	1.5±0.2	3.0±1.1**	1.2±0.5	1.9±0.9**	1.7±0.3	3.4±1.4**	1.5±0.5	3.0±1.2**
Glycogen synthase	1.4±0.4	2.1±0.9**	3.4±0.9	6.0±2.9**	1.5±0.6	6.0±2.4**	1.7±1.2	5.1±3.1**
Glycogen phosphorylase	27.4±9.9	67.6±16.2**	41.7±11.8	103.6±40.2**	26.4±7.9	85.4±42.7**	29.1±11.5	92.7±39**
Phosphofructokinase	11.9±4.9	21.8±8.6*	21.9±3.8	29.3±11.4*	20.8±12.3	29.0±14.4*	19.9±11.5	28.4±15.6*
Aldolase	16.8±4.5	18.0±7.4	24.0±0.0	20.0±5.7	22.5±12.3	18.7±5.4	17.9±7.5	19.7±5.4
Pyruvate kinase	45.8±8.0	92.7±8.4**	41.7±13.1	95.2±20.6**	52.1±18.8	102.4±26.4**	47.8±11.9	100.9±26.1**
Lactate dehydrogenase	198.0±54.1	264.2±111.1	330.6±113.7	283.4±68.1	332.7±111.3	333±109.8	297.3±115.7	318.5±140.4
Succinate dehydrogenase	0.16±0.05	0.17±0.05	0.21±0.06	0.30±0.1*	0.17±0.1	0.27±0.13*	0.11±0.04	0.17±0.08*
Creatine kinase	4000±832	5150±1272	4596±1527	3800±1533	4931±873	3539±760	4209±1065	4136±1391
Aspartate aminotransferase	84.2±21.0	125.5±22.7**	115.0±30.5	131.6±13.2**	97±21.4	128.9±29.7**	91.8±23.9	134.9±40.8**
Alanine aminotransferase	7.1±2.4	14.3±2.8**	12.8±2.8	16.7±3.3**	10.1±2.9	14.0±5.0**	100±3.6	15.6±5.9**

The results are expressed in units g^{-1} of tissue except for glycogen, which is expressed in mg glucose 100 mg^{-1} tissue. Value are means±SD. * $P < 0.05$, ** $P < 0.01$.

Table 5. Percentage of isozymes of lactate dehydrogenase

	Group A ($n = 5$)		Group B ($n = 3$)		Group C ($n = 8$)	
	Before	After	Before	After	Before	After
LDH-1	7.2±4.6	1.7±2.3	3.3±1.5	2.7±4.6	2.3±2.6	1.5*±4.2
LDH-2	13.0±4.7	4.0*±4.9	9.3±2.5	12.7±11.0	6.3±3.1	5.4±7.7
LDH-3	19.6±3.9	26.2±10.9	14.0±1.0	26.0±2.0	12.9±5.3	16.5±7.7
LDH-4	12.6±2.6	23.2*±3.3	14.3±3.5	18.7±6.7	17.6±5.9	15.4±3.8
LDH-5	47.6±13.3	44.7±10.6	59.0±3.0	40.0±8.9	61.0±16.0	61.2±13.2
H-subunit	29.9±9.7	23.6±6.5	20.9±2.7	29.8±10.3	17.8±8.5	17.6±9.8
M-subunit	70.1±9.7	76.3±6.5	79.1±2.7	70.1±10.3	82.2±8.5	82.3±9.8

Values are means±SD. * $P < 0.05$, ** $P < 0.01$.

again at 6800 *g* for 20 min. The new supernatant was used to measure the other enzymatic activities.

The enzymatic activities assayed were glycogen synthase (GS; Thomas *et al.* 1968), glycogen phosphorylase (GPh; Gilboe *et al.* 1972), phosphofructokinase (PFK; Beutler 1975a), aldolase (Ald; Beutler 1975b), pyruvate kinase (PK; Beutler 1975c), lactate dehydrogenase (LDH; Bass *et al.* 1969), succinate dehydrogenase (SDH; Gella *et al.* 1981), aspartate aminotransferase (ASAT; Bergmeyer *et al.* 1976), alanine aminotransferase (ALAT; Wrobleski & Ladue 1956) and creatine kinase (CK; Oliver 1955). Isozymes of LDH (Market & Moller 1959) and CK (Gerhart & Waldenstrom 1979) were analysed by electrophoretic methods.

Statistics. Data were analysed by means of a repeated measures MANOVA including two factors: group (between-subject factor) and training or time (within-subject factor). When significant 'group training' interaction was present, comparisons between groups were analysed using a one-way ANOVA test with pre and post measurements carried out separately, and the training effect was evaluated by means of paired *t*-tests. In this case, we are aware that because of the small number of subjects in groups A and B the statistical tests have a very low power and should be considered only indicative.

Computation was carried out with the SPSS-X package on an IBM-3083/XE computer. Results are expressed as means±standard deviation (SD).

RESULTS

The physical characteristics of the subjects, as well as the biochemical and morphometric findings are presented in Tables 3, 4 and 5, and Figs. 1 and 2. Except for the defining characteristics of each group and for the tricipital skinfold, the groups did not differ significantly for any of the anthropometric, enzymatic or histochemical parameters under consideration before or after training. No interactions between factors (group training) were found either, except for the isozymes of lactate dehydrogenase.

The athletes gained weight ($P < 0.005$) and their arm perimeter increased ($P < 0.001$). They grew in height, though the increase was not statistically significant ($P = 0.08$). Taking into account the previous considerations about the statistical value of intergroup comparisons, the tricipital skinfold was the only parameter which differentiated the groups before and also after the training period, being higher in females than in males ($P < 0.001$ in both cases). In the whole group, the skinfold decreased after the training, although the difference was not statistically significant ($P = 0.39$).

Biochemical findings

The athletes significantly increased the muscle glycogen content and the activities of GS, GPh, PFK, PK, SDH and transaminases (Table 4). On the other hand, the activities of Ald, LDH and CK did not change.

When considering the isozymes of LDH, some interactions between groups were found (while in some groups the activity increased, in others it decreased). In this case, the MANOVA test could not be applied and the effect of training in the whole group is not computable. The univariate statistical results have to be considered only indicative and can be found in Table 5.

Histological findings

After training there was a slight increase in the number of split fibres in the tissue sections. One necrotic fibre per specimen in the second biopsy

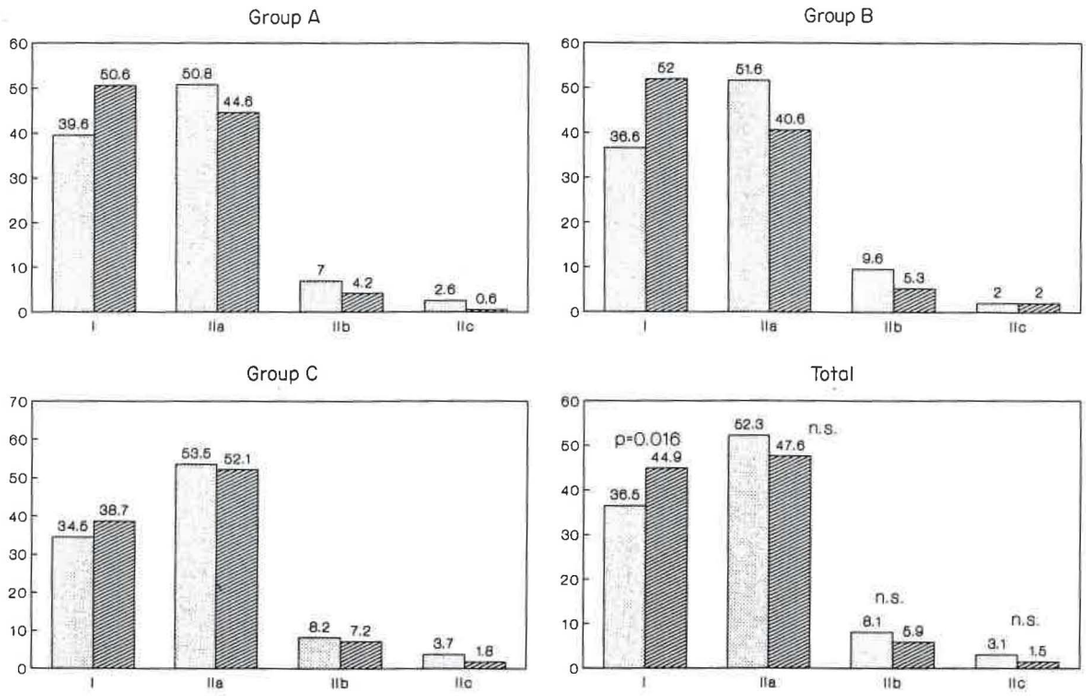

Fig. 1. Fibre-type distribution. Results are expressed as mean percentage of fibres. Statistics are only indicated in the TOTAL group. ▨, before training; ▩, after training.

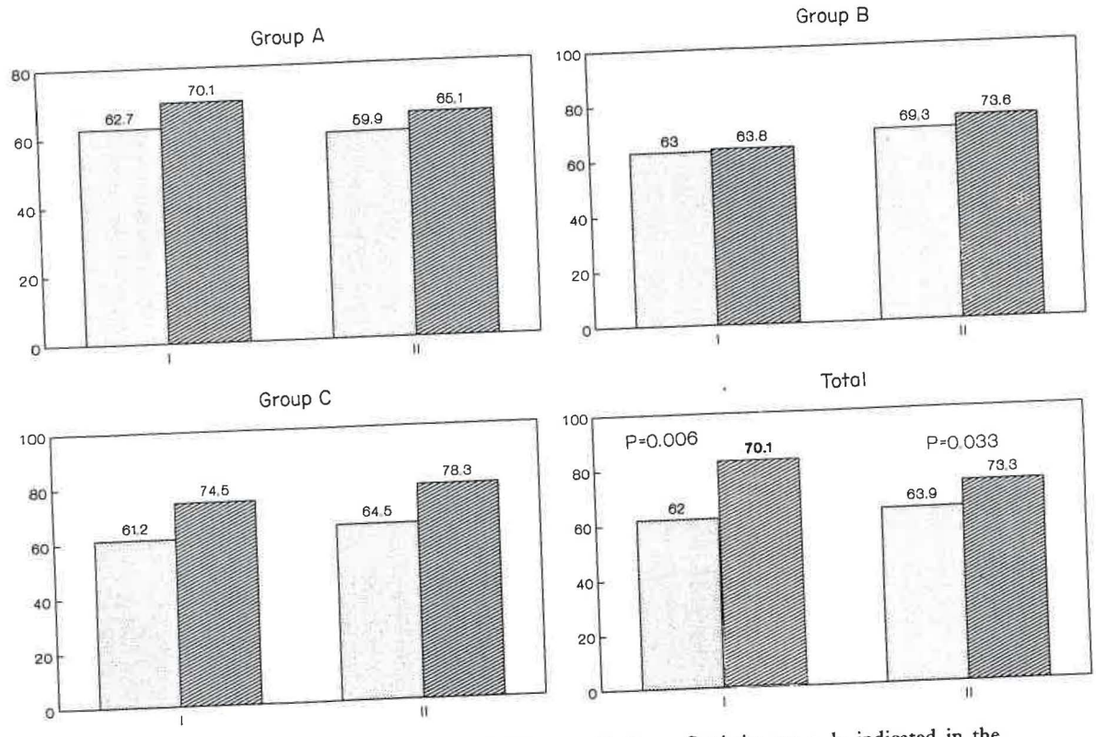

Fig. 2. Diameter in microns of type I and type II fibres. Statistics are only indicated in the TOTAL group. ▨, before training; ▨, after training.

Table 6. Athletic performances

	Group A ($n = 5$)		Group B ($n = 3$)		Group C ($n = 8$)	
	Before	After	Before	After	Before	After
60 m	8.34±0.33	7.84±0.30	7.50±0.28	7.25±0.37	7.34±0.28	7.30±0.31
300 m	46.06±1.89	43.80±1.73	38.59±1.97	37.00±2.01	39.72±1.65	38.42±1.32

Values are means±SD. Time is expressed in seconds.

of three athletes was found. Marked subsarcolemmal deposits were observed with the NADH in the second biopsy of 11 subjects compared with three in the first one. With the same oxidative reaction, fibre type differentiation in eight athletes after the period of training was difficult because the fibres seemed to acquire an almost uniform increase in coloration, while on ATPase staining reaction fibre type differentiation was, as usual, easily accomplished. No other diffuse or focal abnormality was noted.

Figure 1 shows the fibre type distribution in the three groups. The percentage of type I fibres increased significantly ($P < 0.05$) in the whole group. Consequently, the percentage of type II fibres decreased significantly after the period of training. When considering type II fibre subtypes, IIA decreased in groups A and B, and IIB and IIC decrased in the three groups. None of these changes were, nonetheless, significant.

The diameter of both fibre types increased significantly after the period of training (type I, $P < 0.01$; type II, $P < 0.05$) (Fig. 2).

DISCUSSION

In addition to phosphagen splitting, anaerobic glycolysis seems to be the most important source of energy in athletic events requiring maximal exercise and lasting for up to a few minutes, such as sprint track and field competition (Agnevic *et al.* 1967, Hermansen & Medbö, 1984). Unfortunately, adaptive muscle response to sprint remains controversial in view of the results previously reported (Saltin *et al.* 1976, Costill *et al.* 1979, Houston *et al.* 1981, Roberts *et al.* 1982). The reasons could be periods of training which are too short, previous training of the athletes in other disciplines that interfere with the results, changes so subtle that they are difficult to extract from the short series usually reported, or even that the muscle cannot adapt to sprint under physiological conditions. In this sense, in experimental situations in which motor units from a laboratory animal are electrically stimulated, muscle fast fibres may convert to slow fibres by means of a superimposed chronic nerve stimulation; on the other hand, slow fibres do not change their characteristics in response to high-frequency stimulation unless there is an exclusion of the original slow-firing innervation (Edström & Grimby 1986). The present investigation was undertaken to document and eventually quantify the adaptive response to sprint, trying to avoid, at least in part, the above-mentioned problems.

Our athletes gained weight and increased their arm perimeter. Although they also increased in height, this was not statistically significant, and these changes are at least partly attributable to an increase in muscle mass. The athletes also improved their dynamic performance, as shown in Table 6.

Both the increase in muscle mass and the enhancement in athletic performances correlated with the changes in intramuscular metabolites. We found a statistically significant increase in the muscle glycogen content. This finding is consistent with a protective effect against depletion of carbohydrate stores during sprint exercise (glycogen-sparing effect; James & Kraegen 1984). It is interesting to note that the increase was especially evident in groups A and C, both trained for the shortest distance sprint.

The increase in glycogen stores is related to an increase in GS and GPh. This is consistent with an improved capacity of muscle for rapid glycogen mobilization and repletion (Hultman 1967, Urbano-Marquez *et al.* 1987). In the female group the GS increase was threefold lower than in males, which may explain the slower rate of recuperation of females after intense exercise (Holloszy 1984). The glycolytic profile is different between males and females, as previously described (Komi & Karlsson 1978). The key enzymes of glycolysis, PFK and PK, increased significantly after the period of training. Both are regulatory enzymes, and the increase in their concentration permits an increase in glycolytic flux. This could explain why both enzymes increased significantly while other non-limiting enzymes of glycolysis such as ALD and LDH did not change.

Though the LDH content was not significantly modified, it is interesting to note the isozyme pattern in the different groups (Table 5). Before training, we found that the percentages of LDH-1, LDH-2 and LDH-3 in the three groups were lower than those previously described (Apple & Rogers 1986). This could be because our athletes were adolescents (Erikson & Saltin 1974, Bouchard *et al.* 1986). On the other hand it seems that LDH-1, LDH-2 and LDH-3 were higher in females than in males. Differences in the LHD isozymes pattern depending on the sex have also been described (Astrand 1952, Apple & Rogers 1986). After the period of training the modifications were in general not significant, but there was a tendency towards a decrease in the isozymes 1 and 2, which favour the oxidation of lactate to pyruvate during aerobic metabolism (group A showed a decrease in LDH-2 and group C showed a decrease in LDH-1), simultaneously with an increase in isozymes 4 and 5, which reduce pyruvate under anaerobic conditions (group A showed an increase in the content of LDH-4) (Sjodin *et al.* 1976, Apple & Rogers 1986). According to the modifications of LDH isozymes, it seems to be an adaptation to anaerobic metabolism.

Modifications in the activity of CK have been described occasionally with training (Thorstensson *et al.* 1975), but usually this activity is not modified (Jacobs *et al.* 1987), as occurred in our athletes. As CK is present in large quantities in muscle, it may be that in sedentary situations there is a 'relative excess' of CK, the stress induced by exercise being insufficient to stimulate its increase. Nonetheless, the phosphagens required for immediate energy supply were possibly increased as a result of the augmented muscle mass.

The significant increase in SDH and amino acid transferases suggests an enhancement of oxidative metabolism. It is well known that during maximal exercise of short duration both aerobic and anaerobic processes play an important role in energy supply (Hermansen & Medbö 1984), and therefore the enhancement of both energy pathways is not surprising. It has to be taken into account, also, that our athletes did not follow an exclusively anaerobic training. The increase in amino acid transferase activities results in an increased removal of pyruvate by conversion to alanine and in an increase of intermediates of the Krebs cycle (Holloszy & Booth 1976). Besides facilitating the oxidative metabolism, the removal of pyruvate prevents an excess of lactate from accumulating, and this could account for a certain delay in the appearance of fatigue.

The increase in fibre splitting and the enhancement of oxidative reactions in all fibre subtypes have already been reported (Urbano-Marquez *et al.* 1987) and are probably non-specific findings. There was a significant increase in the percentage of type I fibres after training accompanied by a commensurate decrease in type II fibres. All type II fibre subtypes decreased in the three groups except in group C, in which type IIA fibres also increased, although these changes were not statistically significant. Such results were not expected in groups of sprint-trained athletes. The training may not have been sufficient to modify the natural tendency to increase the aerobic capacity with training from childhood to adulthood (Fournier *et al.* 1982). It could also be that the oxidative metabolism enhancement that the athletes underwent may have prevented a pure anaerobic histochemical adaptation. Whatever the case, the finding implies that the increase observed in anaerobic enzyme activity does not correspond to an increase in type II fibre percentage.

Both type I and II fibres underwent hypertrophy. The size of individual muscle fibres increases with heavy resistance training and correlates with strength (Costill *et al.* 1979). Type II fibre hypertrophy is in accordance with an increase in muscle strength and therefore with acceleration capability, which is fundamental to excellence in sprint.

In summary, this study demonstrates biochemical and also morphological muscle adaptation to exercise. It appears that sprint training induces sprint-specific anaerobic biochemical modifications, but also less specific aerobic enhancement. Histological modifications, although clear-cut, seem to be more unspecific. It is possible that sprint-specific biochemical changes affect both fibre types.

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