

PHYSIOLOGICAL ROLE OF MYOSIN LIGHT AND HEAVY CHAIN ISOFORMS IN FAST- AND SLOW-TWITCH MUSCLES: EFFECT OF EXERCISE

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Abstract: The aims of the present study were to show the distribution of individual myosin light chain (MyLC) isoforms in fast-twitch (FT) and slow-twitch (ST) muscles and between FT muscles in order to find differences between MyLC isoforms in these muscles, to identify similarities with the distribution of myosin heavy chain (MyHC) isoforms and to investigate changes in these relations during adaptation to endurance and resistance training. Male Wistar strain rats were used in this study. One-dimensional electrophoresis was used for separation of MyHC and MyLC isoforms and two-dimensional electrophoresis was used for identification of MyLC different isoforms. A difference in the relative content of MyLC isoforms between FT muscles exists only in the case of MyLC 1_{slow} and 2_{slow} isoforms. Differences in the relative content of MyHC between FT muscles are considerably larger than differences in the MyLC isoforms. MyHC and MyLC isoforms both participate in the remodelling of contractile proteins during exercise training. In conclusion: The present study shows some discrepancy between the modulation of MyHC and MyLC isoforms in muscles with different oxidative potential during adaptation to endurance and resistance training. In ST muscles, there is full agreement between the increase in the relative content of MyHC IIA isoform and MyLC 2_{fast} and 3_{fast} isoforms during resistance training without significant changes during endurance training. The ratio of MyLC 3 and MyLC 2 isoforms increases during both types of exercise training, but it is two times higher during resistance training.

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Key words: MyLC – MyHC - Aerobic and anaerobic capacity in fast-twitch and slow-twitch muscles

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Introduction

Muscle myosin, accounting for 60% of the total myofibrillar protein, is composed of two heavy and two pairs of myosin light chains that are associated with the head region of the myosin heavy chain (MyHC) [25]. In adult skeletal muscle, different isoforms of myosin are synthesized according to the specialization of the muscle into fast-twitch (FT) or slow-twitch (ST). Although some of the differences may be related to post-translational events, the analysis of amino acid sequence indicated that different myosin isoforms are the products of different genes [17].

Great differences in the maximum velocity of shortening do not exist only between ST and FT muscles, but also among different FT muscles. Both MyHC content and the proportion of myosin light chain (MyLC), particularly alkali MyLC, are important determinants of unloaded shortening velocity [4,22]. Unfortunately, the role of MyLC isoforms in contractile machinery has not been fully determined. Even the distribution of MyLC isoforms between ST and FT muscles, as well as between FT muscles, still gives rise to serious discrepancies. It has been proposed that the functional role of MyLC isoforms is realized in combination with certain MyHC isoforms [21,26], but the distribution of MyHC and MyLC isoforms in different skeletal muscles has still not been determined with any certainty. It has been shown that exercise, depending on its character, induces differential expression of myosin protein isoforms in skeletal muscle [2]. Most exercise studies focus on the assessment of the composition of MyHC, because of its regulatory role in myosin ATPase activity and therefore in the velocity of muscle fiber shortening [5,9]. Only in a few studies has attention been paid to the simultaneous effect of exercise on the composition of MyHC and MyLC isoforms [23,27]. It is particularly unclear how exercise of different intensity and duration affects the changes in the relative content of MyLC isoforms and their distribution in different muscles. It has been shown that resistance training decreases the relative content of MyHC fastest isoform and increases the content of the slowest isoforms in FT muscles [16], and that changes in the pattern of MyHC isoforms during endurance training depend on the training volume used [16]. Still it is not known how deep adaptational changes appear between MyHC and MyLC isoforms in different muscles during exercise training with different intensity duration and changes in muscle fiber cross-sectional area, aerobic and anaerobic metabolic capacity.

The aim of the present study was: 1) to show the distribution of individual MyLC isoforms in FT and ST muscles and between FT muscles; 2) to find



quantitative differences between MyLC isoforms in these muscles, to identify similarities with the distribution of MyHC isoforms; 3) to study changes in myosin isoform with changes in aerobic and anaerobic metabolic capacities and fiber cross-section area during adaptation to endurance and resistance exercise. We hypothesized that histograms of the distribution of MyLC isoforms reveal structural and functional differences between ST and FT muscles. We also hypothesized that if there is similarity between the distribution of MyHC and MyLC fast and slow isoforms in FT muscles, adaptational changes due to exercise may show the physiological role of MyLC in the muscle contractile apparatus together with changes in muscle fiber cross-sectional area and with changes in aerobic and anaerobic metabolic capacity.

Materials and Methods

Laboratory animals were used in accordance with the European convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes and their use was monitored by the Committee of Laboratory Animal Science of the University of Tartu.

Animals: 46 adult male rats of the Wistar strain of the National Laboratory Animal Centre, Kuopio, Finland were randomly divided into a control group (n=26), and endurance (n=10) and resistance (n=10) training groups. All the animals were housed in identical environmental conditions in polycarbonate cages, at 21 °C, two per cage at 12/12 hrs light/dark period. They received diet (SDS-RM1 (C) 3/8, Witham, Essex, England) and water *ad libitum*. At the end of the experiment control rats weighed 296 ± 6.2 g, and had a muscle weight *Plantaris* (Pla) of 287 ± 7.3 mg, *Extensor Digitorum Longus* (EDL) 138 ± 4.2 mg, and *Soleus* (Sol) 120 ± 3.0 mg. Body weight in the endurance training group was 289 ± 7.0 g, Pla weight was 289 ± 7 mg, EDL weight was 139 ± 4.1 mg, and Sol weight was 121 ± 3.1 mg. Body weight in resistance training group was 312 ± 7.9 g, Pla weight was 312 ± 7.9 mg, EDL 152 ± 5.0 mg, and Sol weight 130 ± 3.4 mg.

The animals were anesthetized with intraperitoneal injection of ketamin (Calysol, Gedeon Richter A.O. Budapest, Hungary) and diazepam (Lab Renaudin, Franze) and sacrificed.

Exercise training: The endurance and resistance training group participated in a 6-week training program.

Endurance training: After a brief 5-day acclimatization that consisted of a treadmill run for 5-10 min at 35 m/min rats were made to run 5 days per week;



training volume was increased moderately during 6 weeks from 10 min to 60 min per day, as described by us earlier [18].

Resistance training: After a 5-day acclimatization that consisted of running without extra weights on a vertical treadmill at a speed 18 m/min. During the training period the rats carried extra weights on the flexible-rod vertical treadmill, as described by us earlier [16]. The training consisted of 2–5 runs per session for 6 days a week: Monday – 2, Tuesday – 3, Wednesday – 4, Thursday – 5, Friday – 4, Saturday – 3. Recovery time 1 min 30 sec between runs. Extra weight during the first week was $30 \pm 0.3\%$ of body weight (bw). During the six-week training period extra weight was increasing so that in the 6th week it constituted 110% of bw.

Muscle strenght measurement: The hindlimb grip strenght (N) was measured before and after training period with Grip Strenght Meter 0167–004L (Columbia Instruments).

Muscle dissection: Muscles: Pla, EDL, and Sol, from control animals and 24 hrs after the last training session from trained animals were dissected out, blotted, weighed and frozen in liquid nitrogen and kept at $-80\text{ }^{\circ}\text{C}$ until the separation of myofibrillar protein. Diaphragm (Dia) was used only to obtain characteristics of the pattern of MyLC and MyHC isoforms in control animals, since these isoforms are locating between the isoforms of ST and FT skeletal muscles (Figs. 2 and 3).

Separation of myofibrillar protein: Myofibrillar protein was separated as described previously [19] for further determination of MyHC and MyLC isoforms.

One-dimensional electrophoresis (SDS-polyacrylamide gel electrophoresis). The isoform composition of MyLC (Fig. 1C) and MyHC (Fig. 1A) was determined by SDS-PAGE. Separation of MyHC isoforms was achived by a previously described method [10] with 7.2% separating gel. Electrophoresis was run for 24 hrs at low temperature at 120 V (constant voltage). Gels were silver-stained by the method of Oakley *et al.* [14].

The MyLC isoforms were analyzed using 12.5% one-dimensional SDS-PAGE according to Laemmli [12], except that the glycerol content in the separating gel was 10%. Electrophoresis was performed at a constant current (30 mA) and stopped when the dye front reached the bottom of the gels, using a vertical slab gel system (Protean II Xi Bio-Rad). The gels were Coomassie Brilliant Blue R-250 stained.

Identification of MyLC and MyHC isoforms was based on the migration rate. MyLC and MyHC isoforms were quantified densitometrically by a computer-based image analysis system and software (Image Master 1D, program, Version 3.0, Amersham Pharmacia Biotech, Newcastle upon Tyne, England). As comparison of the results of one- and two-dimensional electrophoresis of MyLC isoforms did not



show significant differences, in the present study we used only the results of one-dimensional electrophoresis.

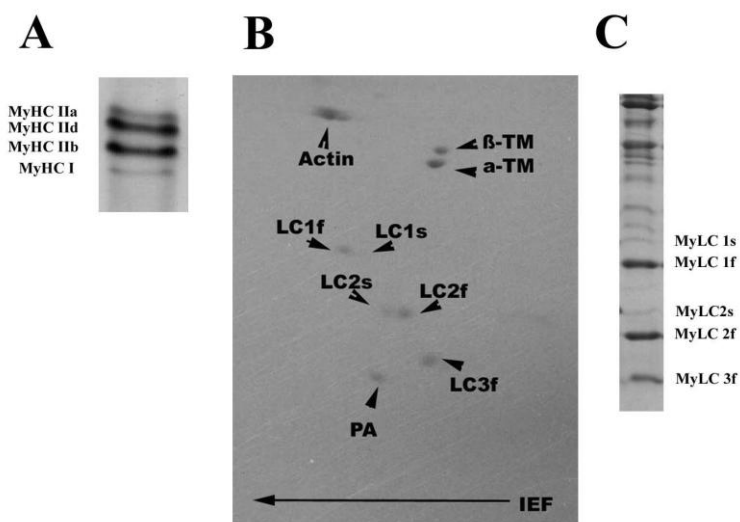


Fig. 1

One- and two-dimensional gel electrophoresis of MyHC and MyLC from FT muscle

A - MyHC isoforms; B - two-dimensional separation of MyLC isoforms; C - one-dimensional separation of MyLC isoforms

As comparison of results of one- and two-dimensional electrophoresis of MyLC isoforms did not show significant differences in the present study, we used only the results of one-dimensional electrophoresis. Two-dimensional electrophoresis was used for the identification of different MyLC isoforms

Two-dimensional electrophoresis: Two-dimensional electrophoresis was used for the identification of different MyLC isoforms. Proteins were separated by two-dimensional gel electrophoresis according to O'Farrell [15]. Electrophoresis in the first dimension was performed on glass capillaries by using 1.6% (pH 5.0–7.0) and 0.4% (pH 3.0–10.0) ampholines (Servalyt) in 4.2% polyacrylamide gel. Electrophoresis was first run for 30 min at 100 V, then 1 hr at 300 V, and 1 hr at 400 V. Separation in the second dimension was carried out at 70 mA with 1.5 mm



thick 15% separating gel and 3% stacking gel [26]. The gels were stained with Coomassie Brilliant Blue R 250. After destaining, the spots corresponding to MyLC isoforms (Fig. 1B) were excised, placed into tubes containing 1.5 ml 25% (vol/vol) pyridine, and incubated overnight. This allowed the elution of the bound dye in order to determine relative protein amounts. The eluted dye was measured spectrophotometrically at 605 nm [8].

Cytochromes aa₃ measurement: Cytochromes aa₃ were measured as described by as earlier [1].

Lactate dehydrogenase measurement: Lactate dehydrogenase activity was measured by the method of Shonk and Boxer [20].

Fiber size analyses: Muscles were fixed by Tissue Tek O.C.T. Compound 4583 (Miles Inc, USA), immersed in isopentane cooled and stored at -80 °C (with liquid nitrogen). Serial 10- μ m-thick tissue cross-sections were obtained from each muscle midbelly by cryostat microtome (Cryo Cut, American Optical Company).

Muscle fiber types were determined by the immunohistochemical procedures using myosin antibodies NCL-MyHC_s, NCL-MyHC_f (Novocastra Laboratories Ltd, Newcastle upon Tyne, UK).

This procedure allowed the delineation of the two major fiber types, I and II. Sections/samples were examined with an Olympus BX-40 light microscope (Tokyo, Japan) with digital camera Olympus DP-10, and measurements were taken using Olympus DP-soft software.

Protein assay: Myofibrillar protein was assayed by using the technique described by Bradford [6].

Statistics: Means and, standard error were calculated from individual values by standard procedures of Excel. The data were analyzed by statistical analysis system (SAS), using the analysis of variance (ANOVA), and the Pearson correlation coefficients and partial correlation coefficients were calculated. Differences were considered significant at $P < 0.05$.

Results

There was significant difference in LDH activity between EDL, Pla and Sol muscles (Fig. 2) as well as in cytochromes aa₃ concentration (Fig. 2). The relative content of MyLC isoforms in FT muscles is significantly different from that in ST muscles (Fig. 3). The difference in the relative content of MyLC isoforms between two FT muscles exists only for 1_{slow} and 2_{slow} MyLC isoforms (Fig. 4). Differences in the relative content of MyHC (Fig. 4) isoforms between two FT muscles are considerably larger than those of MyLC isoforms (Fig. 3). The comparison of the



relative content of MyLC and MyHC isoforms between ST and FT muscles revealed good agreement between MyLC and MyHC (Figs. 3 and 4). The relative content of MyLC isoforms between two FT muscles does not follow the logic of MyHC. There are many more similarities between the relative content of MyLC and MyHC isoforms when we divide both isoforms into fast and slow isoforms (Table 1). There are no significant changes between alkali and regulatory MyLC isoforms between Pla (subsequently 54.01%, 45.99%) and EDL (54.35%, 45.65%) muscles. The difference between Pla and EDL muscles in MyHC IIa isoform was 8.64% and in MyLC 1_{fast} isoforms 2.0%. Subsequent differences between these muscles in MyHC IIb and MyLC 3_{fast} isoforms were 21.28% and 0.90%. These differences between two FT muscles in MyLC and MyHC isoforms do not show good agreement. The relative content of MyHC IIb isoform in EDL muscle is approximately twice as high as that in Pla muscle, but there is no difference in the relative content of MyLC 3_{fast} isoform between these muscles. As shown in Fig. 3 the relative content of MyLC isoforms in Dia muscle remains between that of FT and ST muscles.

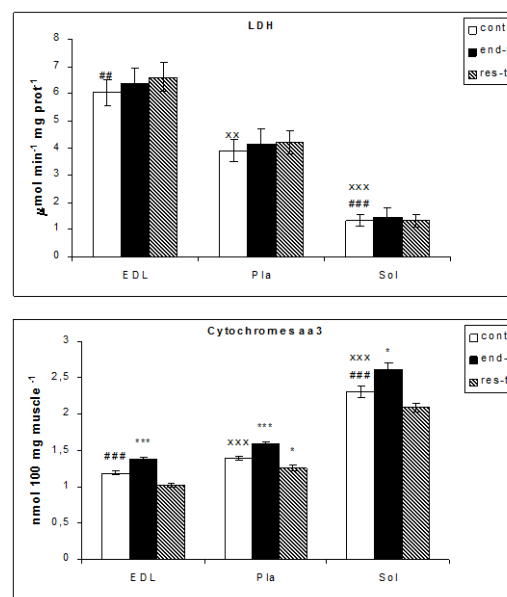


Fig. 2

Changes in skeletal muscle anaerobic and aerobic metabolic capacities during endurance and resistance training

LDH - lactate dehydrogenase LDH activity was used as indicator of anaerobic metabolic capacity of muscle; Cytochromes aa₃ was used as indicator of aerobic metabolic capacity of muscle; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Sol - *Soleus* muscle; contr - control group (n=10); end-tr - endurance trained group (n=10); res-tr - resistance trained group (n=10)

*P<0.05 in comparison with subsequent control group; ***P<0.001 in comparison with subsequent control group;

xxP<0.01 in comparison with EDL muscle; xxxP<0.001 in comparison with EDL muscle; ###-p<0.001 in comparison with Pla muscle

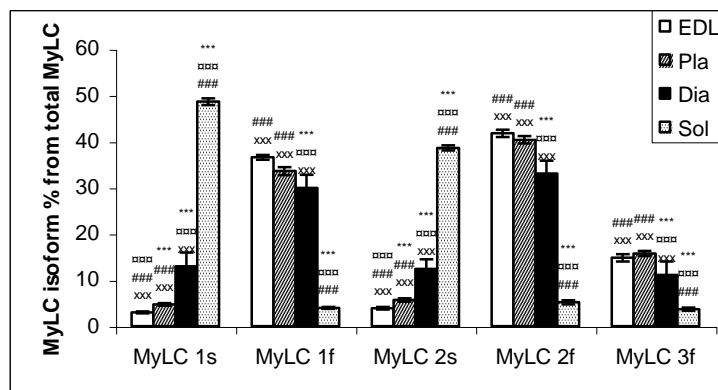


Fig. 3

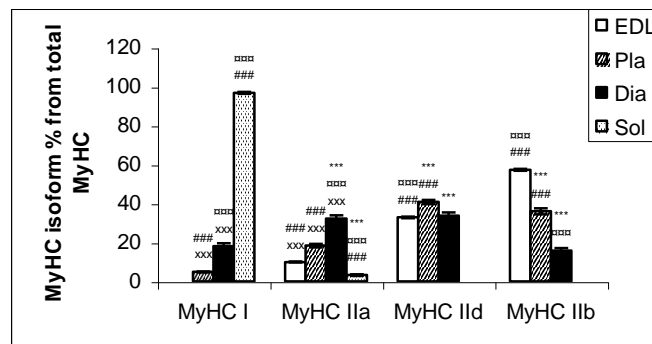
Relative content MyLC isoforms' in different muscles

Values are means \pm standard errors; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Dia - Diaphragm; Sol - *Soleus* muscle

*P<0.05 } in comparison with subsequent EDL isoform

***P<0.001 }
 □□□-P<0.001 in comparison with subsequent Pla isoform; ###P<0.001 in comparison with subsequent Diaphragm isoform; xxxP<0.001 in comparison with subsequent Sol isoform; n=26



**Fig. 4**

Relative content MyHC isoforms' in different muscles

Values are means \pm standard errors; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Dia - Diaphragm; Sol - *Soleus* muscle

*** $P < 0.001$ in comparison with subsequent EDL isoform; □□□ $P < 0.001$ in comparison with subsequent Pla isoform; #### $P < 0.001$ in comparison with subsequent Diaphragm isoform; xxx $P < 0.001$ in comparison with subsequent Sol isoform; $n = 26$

LDH activity in different skeletal muscle had negative correlation with MyHC I isoform content ($r = -0.92$; $P < 0.001$) and positive correlation with MyHC IIa, IIc and IIb subsequently ($r = 0.55$; 0.77 and 0.99 ; $P < 0.001$). Concentration of cytochromes aa_3 in different skeletal muscles had positive correlation with MyHC I isoform content ($r = 0.94$; $P < 0.001$) and negative correlation with the content of MyHC IIa, IIc and IIb subsequently $r = -0.68$; -0.85 and -0.94 ; $P < 0.001$). MyHC turnover rate had positive correlation with oxidative potential in muscle ($r = 0.61$; $P < 0.001$).

As seen in Fig. 5, the histogram of the MyLC isoforms in Sol muscle shows that the distribution of MyLC slow isoforms is significantly wider than that of fast isoforms. This is not typical of the FT muscles.

Hindlimb grip strength in control group was 5.63 ± 0.30 N/100 g bw). Endurance training did not change hindlimb strength significantly (5.97 ± 0.31 N/100 g bw) but resistance training increased this indicator until 6.70 ± 0.34 N/100 g bw ($P < 0.05$ in comparison with control group). LDH activity did not change significantly during exercise-training, but cytochromes aa_3 concentration increased in all studied

muscles during endurance training (Fig. 2). Resistance training increased FT fibers diameter in EDL and Pla muscles (Fig. 6).

Table 1

Comparison of MyHC and MyLC fast and slow isoforms relative content in different muscles

Myosin isoforms	Muscles			
	Sol	Dia	Pla	EDL
MyHC fast isoform % (IIa+IId+IIb) from total MyHC	3.23±0.46 *** □□□ ###	81.90±1.46 *** □□□ xxx	95.10±0.26 *** xxx ###	100.00±1.91 □□□ xxx ###
MyLC fast isoform % (1f+2f+3f) from total MyLC	12.78±0.55 *** □□□ ###	74.02±2.84 *** □□□ xxx	89.63±0.52 ** xxx ###	93.13±0.37 □□□ xxx ###
MyHC slow isoform % (I) from total MyHC	96.77±0.46 □□□ ###	18.10±1.44 □□□ xxx	4.90±0.26 xxx ###	—
MyLC slow isoform % (1s+2s) from total MyLC	87.22±0.55 *** □□□ ###	25.39±2.49 *** □□□ xxx	10.39±0.52 *** xxx ###	6.87±0.37 □□□ xxx ###

Values are means ± standard errors; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Sol - *Soleus* muscle; Dia – Diaphragm

MyHC - myosin heavy chain

I - MyHC isoform; Iia - MyHC isoform; Iid - MyHC isoform; Iib - MyHC isoform

MyLC - myosin light chain

1s - 1 slow MyLC; 2s - 2 slow MyLC; 1f - 1 fast MyLC; 2f - 2 fast MyLC; 3f - 3 fast MyLC



**P<0.01 }
} in comparison with subsequent EDL isoforms
***P<0.001 }
○○○P<0.001 in comparison with subsequent Pla isoforms
xxxP<0.001 in comparison with subsequent Sol isoforms
###P<0.001 in comparison with subsequent Dia isoforms; n=26

Endurance training caused a significant decrease in the relative content of MyLC 2_{slow} isoform in EDL muscle and MyLC 1_{slow} isoform in Pla muscle (Fig. 7). The relative content of MyLC 3_{fast} isoforms increased in both FT muscles during endurance training. In Sol muscle, resistance training led to an increase in the relative content of MyLC 2_{fast} and 3_{fast} isoforms (Fig. 7). Resistance training decreased the relative content of MyLC 1_{slow} and 2_{slow} isoforms in both FT muscles and increased that of MyLC 3_{fast} in EDL muscle. MyLC 3/MyLC 2 ratio increased during both training regimes in FT and ST muscles. In EDL muscle the ratio increased 34.3% (P<0.001) during endurance training and 29.2% (P<0.001) during resistance training. In Pla muscle, by 33.2% (P<0.001) during endurance training and 16.6% (P<0.001) during resistance training. In Sol muscle subsequently by 12.25 and 23.1% (P<0.001). In both FT muscles, the relative content of MyHC IIb isoform decreased and that of MyHC IIc isoform increased during endurance as well as during resistance training (Fig. 8). The relative content of MyHC IIa isoform increased during endurance training in Pla muscle. Resistance training caused a decrease in MyHC IIa isoform in EDL muscle and an increase in Pla muscle (Fig. 8). The relative content of MyHC I isoform increased during both training regimes in Pla muscle and did not change significantly in Sol muscle (Fig. 8). In Sol muscle the relative content of MyHC IIa decreased during endurance training and increased during resistance training (Fig. 8). In Sol muscle, an increase in training volume had a negative correlation with the relative content of MyHC IIa isoforms ($r=-0.742$), and an increase in training intensity was positively correlated with the relative content of MyHC IIa isoform ($r=0.722$).



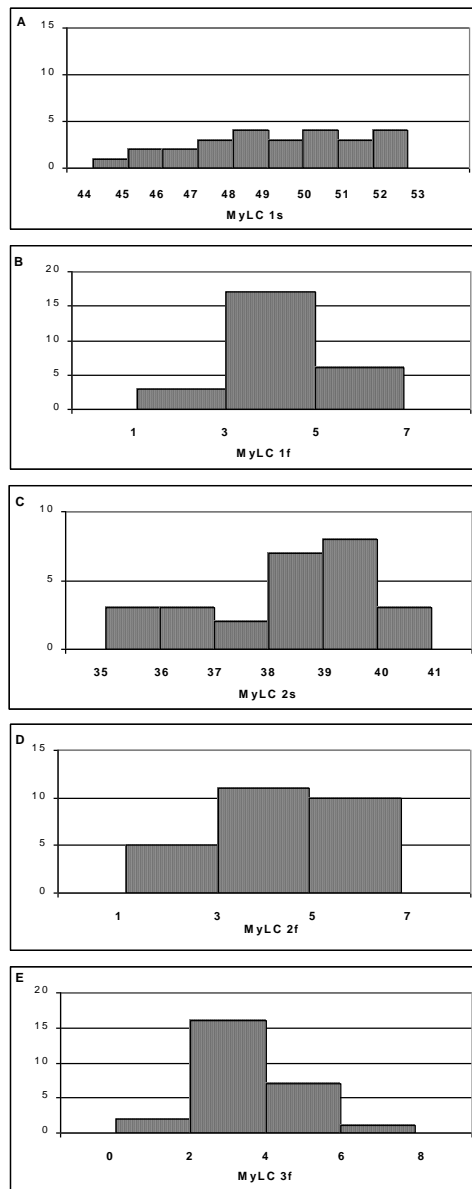
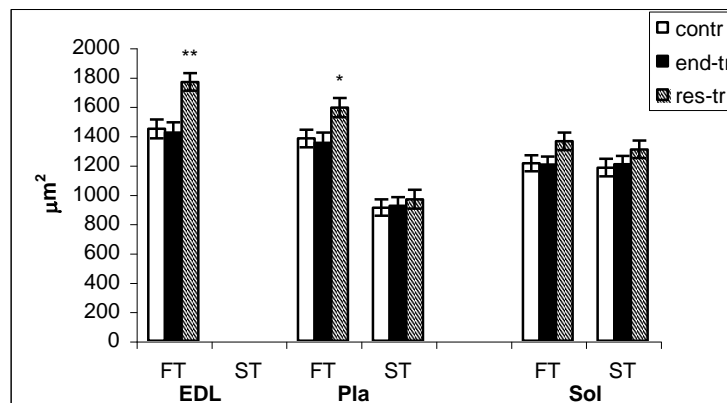


Fig. 5
Histogram of MyLC isoforms in *Soleus* muscle
Ordinate axis represent the number of cases per interval; Abscise axis represents the interval of MyLC subsequent isoforms % from total MyLC; n=26



**Fig. 6**

Changes in muscle fiber's cross-sectional area during endurance and resistance training

FT - fast-twitch fibers; ST - slow-twitch fibers; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Sol - *Soleus* muscle; contr - control group (n=26); End - tr—endurance trained group (n=10); res-tr - resistance trained group (n=10)

*P<0.05 in comparison with subsequent control group

**P<0.001 in comparison with subsequent control group



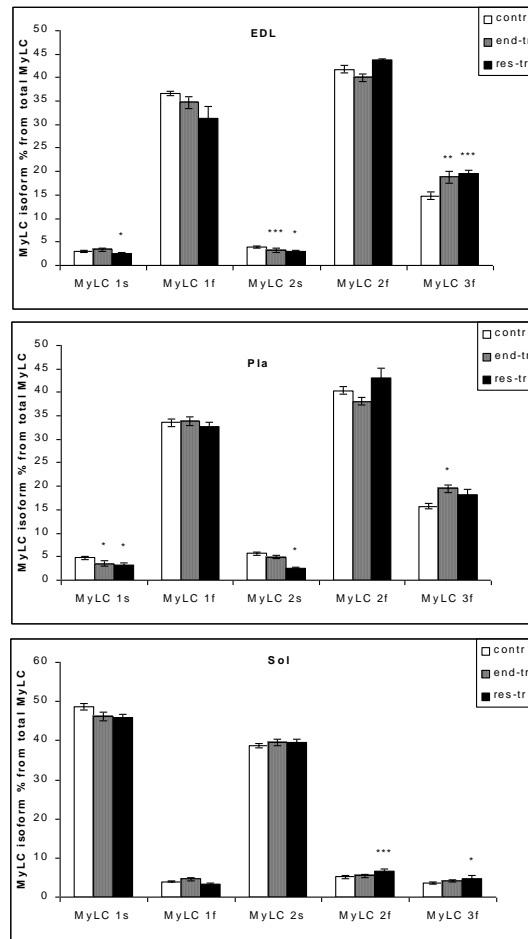


Fig. 7
 The effect of endurance and resistance training on the relative content of MyLC isoforms' in FT and ST muscles

Values are means \pm standard errors; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Sol - *Soleus* muscle; contr - control group (n=26); end-tr - endurance trained group (n=10); res-tr - resistance trained group (n=10)

*P<0.05 }
 **P<0.01 } in comparison with subsequent MyHC isoform of
 ***P<0.001 } control group



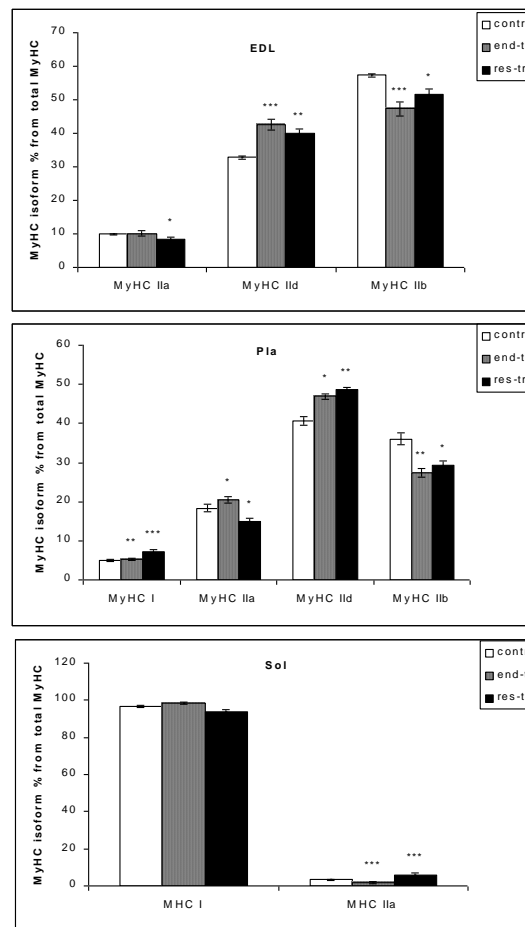


Fig. 8

The effect of endurance and resistance training on the relative content of MyHC isoforms in FT and ST muscles

Values are means \pm standard errors; EDL - *Extensor Digitorum Longus* muscle; Pla - *Plantaris* muscle; Sol - *Soleus* muscle; contr - control group (n=26); end-tr - endurance training group (n=10); res-tr - resistance training group (n=10)

*P<0.05 }
 **P<0.01 } in comparison with subsequent MyHC isoform of
 ***P<0.001 } control group



Discussion

During the last two decades it has been shown that among multiple isoforms of muscle proteins MyHC and MyLC isoforms play an important role in muscle function. Wahrman, Winand and Rieu [27] quantified changes in MyLC isoforms in different muscles during exercise. They claim that, in order to better understand the function of MyLC in skeletal muscle it is necessary to study changes in MyLC in parallel with the quantification of MyHC under the same conditions. The above mentioned standpoint is supported by studies where, during muscle atrophy, an increase in MyLC 1_{fast} and 2_{fast} isoforms was found in parallel with the increase in the relative content of MyHC IIb isoform [7].

Hayashibara and Miyanishi [11] showed that the decrease in the relative content of MyLC 1_{fast} isoform is the indicator of the slowing of muscle contraction. Our study supports this standpoint, since the relative content of MyLC 1_{fast} isoform is decreasing in muscles in the following direction: EDL→Pla→Dia→Sol.

The relation between the relative content of MyHC fastest isoform and MyLC 3_{fast} isoform and muscle contraction speed has also been shown to exist [4]. In some studies it has been shown that there is a positive correlation between muscle contraction speed and MyLC 3_{fast}/MyLC 1_{fast} isoforms ratio [5,22], but some researchers have denied this [13]. Our study shows that the above-mentioned relation exists only between FT and ST muscles, but not between two FT muscles, EDL and Pla. It has been shown that higher apparent affinities exist between the MyLC 3_{fast} isoform and MyHC IIb and IId isoforms [21,26]. Our study shows that there are correlations between the relative content of MyHC IIb, IId and MyLC 3_{fast} isoforms in ST and FT muscles. We did not find any correlation between MyLC 1_{fast}/MyLC 3_{fast} isoforms ratio and the relative content of MyHC IIb and IId isoforms in skeletal muscle. In muscles where MyHC fastest isoform is dominating, MyLC 1_{fast}/MyLC 3_{fast} isoforms ratio has been shown to be lower than in muscles where MyHC IId isoform is dominating [26].

The low affinity of MyLC 3_{fast} and the higher affinity of MyLC 1_{fast} isoform with MyHC IId isoform shows differences between myosin isoforms in FT muscles [24]. Our study does not support the standpoint that, in FT muscles, the relative content of alkali MyLC isoforms is higher than in ST [3]. The present study shows that there are certain relations between MyHC and MyLC isoforms in FT and ST muscles. The histogram of the distribution of MyLC isoforms in different muscles shows that, in ST muscle, the distribution of dominating slow isoforms is wider than that of fast isoforms. It is first shown in this study wide distribution of dominating isoforms show the physiological role and adaptational capacity of



MyLC isoforms in skeletal muscle to everyday activity, since ST muscles are participating in the process of slow movements and keeping static positions. Wide distribution of MyLC slow isoforms in Sol muscle show the adaptational peculiarities of the muscle *via* these isoforms.

Partial correlation in different skeletal muscles show, that oxidative potential plays an important role in the regulation of MyHC and MyLC isoforms content.

Adaptation to different duration and intensity exercise training revealed that there are some similarities in the changes in the relative content of MyHC isoforms in FT muscles during both types of exercise. Thus, endurance as well as resistance training causes a decrease of MyHC IId isoform, although changes during endurance training are greater than during resistance training in FT muscles. The relative content of MyHC IId isoforms also increases with both training regimes in FT muscles. At the same time the relative content of MyLC 3_{fast} isoforms is increasing or has a tendency to increase in these muscles during both training regimes. There is a discrepancy between changes in the relative content of MyHC fastest isoform and MyLC 3_{fast} isoform during the adaptation to exercise training. As the total content of MyLC fast isoforms did at the same time not change, the relative content of MyHC IId isoform increased, and MyLC 3_{fast} isoforms also have an affinity to bind with MyHC IId isoform, we do not see a contradiction from the standpoint of the stoichiometry of the myosin molecule. In ST muscle there is full agreement between the increase in the relative content of MyHC IId isoform and MyLC 2_{fast} and 3_{fast} isoforms during resistance training. Endurance training leads to the decrease in the relative content of MyHC IId isoform in Sol muscle but there are no significant changes in MyLC fast isoforms. At the same time the ratio of MyLC 3 and MyLC 2 isoforms show an increase during both types of exercise training. It is interesting to mention that the above-mentioned ratio is two times higher during resistance training. It was shown that MyLC 3/MyLC 2 ratio increased during unloading and during the increase in muscle shortening velocity [28]. Increase in MyLC 3/MyLC 2 ratio in both FT and ST muscles during endurance and resistance training disagrees with the results of Williamson *et al.* [29], but shows that mechanical loading leads to changes in MyLC isoforms in skeletal muscle

Conclusion

The present study revealed some discrepancy between the modulation of MyHC and MyLC isoforms in different muscles during the adaptation to long-lasting low intensity and short-lasting vigorous exercise. Gravity of change in MyHC and



MyLC isoforms in FT and ST muscles in response to exercise training do not give reason to presume that MyLC isoforms have a lesser role in skeletal muscle function. As the function of myofibrillar proteins depends on the contractile, regulatory and minor proteins, the role of MyLC isoforms in ST and FT muscles needs further investigation in the context of changes in other myofibrillar proteins. Although the exact role of MyLC isoforms in skeletal muscle during the adaptation to the exercise training is not fully known. Changes in the relative content of MyLC isoforms is related to the character of training.

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