

Young Investigator Section

Research article

ADAPTIVE CHANGES OF MYOSIN ISOFORMS IN RESPONSE TO LONG-TERM STRENGTH AND POWER TRAINING IN MIDDLE-AGED MEN

Raivo Puhke¹, Sirkka Aunola²✉, Pirjo Ailanto³, Karin Alev¹, Mika Venojärvi^{2,4},
Heikki Rusko^{5,6} and Teet Seene¹

¹ Institute of Exercise Biology and Physiotherapy, University of Tartu, Tartu, Estonia

² Laboratory for Population Research, Department of Health and Functional Capacity, National Public Health Institute, Turku, Finland

³ Department of Physiology, University of Kuopio, Kuopio, Finland

⁴ Medical Laboratory Technology, Turku University of Applied Sciences, Turku, Finland

⁵ KIHU - Research Institute for Olympic Sports, Jyväskylä, Finland

⁶ Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland

Received: 08 November 2005 / Accepted: 10 April 2006 / Published (online): 01 June 2006

ABSTRACT

The purpose of the study was to examine the adaptive changes in myosin heavy chain (MHC) and light chain (MLC) isoforms in human *vastus lateralis* muscle caused by long-term strength and power training (54 weeks, approximately 3 times a week) in untrained middle-aged men (16 in the training and 6 in the control group). Muscular MHC and MLC isoforms were determined by means of SDS-PAGE gel electrophoresis. During the training period, maximal anaerobic cycling power increased by 64 W ($p < 0.001$) and the maximal jumping height by 1.5 cm ($p < 0.05$) in the training group, but no significant changes were found in the control group. However, the group by time effect was not significant. In the training group, the increase of the maximal jumping height correlated with the number of strength and power training sessions ($r = 0.56$; $p < 0.05$). The change of the proportion of MHC IIa isoform from $52.6 \pm 12.2\%$ to $59.4 \pm 11.6\%$ did not reach statistical significance ($p = 0.070$ for group by time; within training group $p = 0.061$) and neither did the change of the proportion of MHC IIx isoform from $18.1 \pm 11.4\%$ to $11.1 \pm 9.1\%$ ($p = 0.104$ for group by time; within training group $p = 0.032$). The degree of change of MHC IIx isoform correlated with the amount of earlier recreational sports activity ($r = 0.61$; $p < 0.05$). In the training group, the changes of MLC1s isoform correlated negatively with the changes of MLC1f isoform ($r = -0.79$; $p < 0.05$) as well as with the changes in maximal anaerobic cycling power ($r = -0.81$; $p < 0.05$), and positively with those of MHC I isoform ($r = 0.81$; $p < 0.05$). In conclusion, the long-term strength and power training ~3 times a week seemed to have only slight effects on fast MHC isoforms in the *vastus lateralis* muscle of untrained middle-aged men; the proportion of MHC IIa tended to increase and that of MHC IIx tended to decrease. No changes in MLC isoform profile could be shown.

KEY WORDS: Anaerobic muscular power, contractile proteins, myosin heavy chain isoforms, myosin light chain isoforms, training, transformation.

INTRODUCTION

Myosin, the main contractile protein of muscle contraction, is composed of heavy chain (MHC) and light chain (MLC) isoforms. In adult human skeletal muscle MHC is expressed with slow (MHC I) and fast isoforms (MHC IIa and MHC IIx). Each MHC isoform has its distinct ATPase activity and properties of shortening velocity, wherefore the MHC profile is considered a feasible marker of fiber type diversity (Bottinelli, 2001; Pette and Staron, 2001).

Five distinct MLC isoforms are expressed in adult human skeletal muscle. The essential MLC isoforms include two fast (MLC1f and MLC3) and one slow (MLC1s) isoforms. The content of MLC3 isoform has shown to correlate with the shortening velocity of muscle fibers (Larsson and Moss, 1993). The regulatory part of MLC includes both slow and fast MLC isoforms (MLC2s, MLC2f). The expression of MLC2 isoforms has also been shown to be associated with the speed and velocity regulation of muscle contraction (Lowey et al., 1993).

Exercise training causes adaptive changes in myosin profile. In order to create significant changes on protein level, quite a long training period is necessary. A fast-to-slow transition in MHC isoform has been reported in previous studies, caused by specific types of resistance or weight training lasting 8–24 weeks (Adams et al., 1993; Campos et al., 2002; Harber et al., 2004; Sharman et al., 2001). A decrease of the proportion of MHC I isoform has been shown to occur after sprint or strength training (Andersen et al., 1994; Liu et al., 2003). We have not found any follow-up studies concerning MHC adaptation to long-term (≥ 1 year) exercise training in healthy people.

MLC adaptation to exercise training has been reported in several studies on laboratory animals (Ingalls et al., 1996; Wada et al., 2003; Wahrman et al., 2001). Few data exist about MLC adaptation to exercise training in human skeletal muscles. Trappe and co-workers (2000, 2001) examined MLC profile during 12 weeks of resistance training in elderly subjects but no significant changes in MLC profile were found.

The purpose of the present study was to examine the adaptive changes of MHC and MLC isoforms in human *vastus lateralis* muscle in response to long-term strength and power training in previously untrained middle-aged men. Furthermore, we examined whether the changes in MHC and MLC isoforms would correlate with the changes in maximal anaerobic cycling power, vertical jumping height, and the total number of training sessions.

METHODS

Subjects

Seventy-five middle-aged healthy voluntary male subjects entered this study. Before the intervention, they participated in the health examinations and measurements included in the study protocol. Of the subjects, 56 volunteered for long-term exercise training and 19 for acting as a control subject. A total of 45 men in the training group and 18 in the control group completed the program; however, only 23 men in the training group trained to a sufficient extent (i.e. completed $\geq 2/3$ of the program). Of them the final training group consisted of the 16 men whose muscle biopsies were obtained both before and after the exercise intervention and whose tissue samples were large enough for the MHC and MLC analyses. Their age, height and weight were 43.8 ± 5.8 yrs, 1.79 ± 0.05 m, and 83.1 ± 12.8 kg (before) and 83.6 ± 13.3 kg (after), respectively.

Correspondingly, muscle biopsies were obtained from 6 persons belonging to the control group both before and after the 54-week intervention. Their age, height and weight were 40.0 ± 8.5 yrs, 1.77 ± 0.04 m, and 69.7 ± 9.7 kg (before) and 72.4 ± 9.2 kg (after), respectively. The characteristics of the both subject groups were quite similar with the voluntary subjects entered originally into the study ($n = 75$; their age, height and weight were 42.4 ± 6.7 yrs, 1.80 ± 0.06 m, and 80.4 ± 13.1 kg, respectively). All the participants were either previously untrained or engaged in recreational sports that some of them continued simultaneously with the study program.

All the subjects gave their written informed consent to participate in this study that was approved by the Ethical Committee of the Research and Development Centre of the Social Insurance Institution of Finland.

Training protocol

The subjects were advised to exercise 3 times a week according to a special strength and power training program for a period of 54 weeks (Appendix). Simultaneously, they continued 0–2 times a week their previous recreational sports activities (including walking, jogging, cycling, skiing, swimming, and ball games, such as volleyball, badminton, tennis, or soccer). The training group carried out $93.5 \pm 12.5\%$ (range 75–118%) of the planned sessions for special training in the gym and $134.6 \pm 35.2\%$ (range 76–210%) of the recommended amount of training (3 times a week) including both all type of exercise sessions in the planned program (see Appendix) and the subjects' spontaneous recreational sports activities. The strength and power training program was

individually adjusted to the muscular performance capacity of each subject.

The supervised program started with a lead-in strength-training period of 6 weeks, followed by a basic strength-training period of 4 weeks. The next phase consisted of progressive strength and power training [at 60–75% and 30–85% of 1-RM (repetition maximum), respectively] with stretching and elasticity exercises. For the first four weeks of this phase, the subjects exercised twice a week as consecutive training, together with power training sessions once a week as circuit training, after which they had one consecutive and two circuit training sessions a week during the next four weeks. Thereafter, the exercises were changed and the number of repetitions (varying from 4 to 12 rep.), and later on, also the number of sets, was increased (Appendix). At the beginning of the primary strength and power training phase, three circuits or sets with a pause of 2–3 min in between were performed; at the end of the training period the number of circuits was five. During the pauses the subjects performed warm-up (for muscles to be exercised next) and recovery stretching exercises (for muscles just used).

Power-type training and basic strength training were emphasized by turns, and the exercises focused mainly on the legs. The training group performed 7285 ± 1099 (range 5378–9868) femoral muscle exercises during the whole training period. In addition, various trunk and upper body exercises were performed. Special attention was paid to the velocity of muscle contraction in the exercises performed with small loads (power training) in order to induce training effects on the force and velocity characteristics of leg muscles, and especially on the fast-twitch fibers. After three months there was a two-week 'recovery phase' during which the subjects only carried out recreational outdoor sports 1–2 times per week. During the next 12 weeks (in the spring) the subjects continued the strength and power training program. In the summer time, the program included interval running, plyometric drills, shot put, discus, javelin, and circuit training.

Before the training intervention started, seven muscle groups were tested by using 30-second repetition tests. Individual training programs with 10 exercises were drawn up on the special training cards for each man in the training group. Later on, 10-second repetition tests or 1-RM were applied to determine the proper progression for training (additional weight and/or number of repetitions). New, individual training programs were given for each phase: the exercises were changed and loadings increased according to the program model.

Earlier recreational sports activity

Leisure-time physical activities after the school years were asked for and graded into five grades as follows: 0 = having no leisure-time physical activity during adult age, 1 = having 1–3 types of sports and being active < 5 years, 2 = having 1–3 types of sports and being active 5–10 years, 3 = having 1–3 types of sports and being active >10 years or having several types of sports and being active 5–10 years, 4 = having several types of sports and being active >10 years.

Maximal anaerobic cycling power

The maximal anaerobic power of leg muscles was measured using a bicycle ergometry modification of maximal anaerobic power test on a treadmill (Rusko et al., 1993). In this intermittently progressive test, the loading consisted of 5–10 successive 20-s cycling periods with a pause of 100 s between the periods. The test was continued until the subject could not keep the correct predetermined pace, which was 90 revolutions/min for men younger than 40 years and 86 revolutions/min for men older than 40 years. The increment in work rate for each consecutive cycling period was adjusted to increase the oxygen demand by $5\text{--}6 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$.

Vertical jump

The explosive force of leg muscles was measured as vertical jumping height. The jump was performed from a static leg position with the knee angle of 100° and without a countermovement. The hands were held on the hips. A contact mat and reaction time equipment (Newtest powertimer®, Finland) were used to measure the off-the-ground time from which the jumping height was calculated (Bosco et al., 1983).

Muscle biopsies

Muscle biopsies of 50–100 mg were taken under local anesthesia from the left vastus lateralis muscle, using the percutaneous conchotome technique, at the beginning and at the end of the training period. The muscle sample was frozen in liquid nitrogen and stored at -70°C for further analysis of heavy and light chain myosin isoforms.

MHC and MLC determination

Muscle samples were pulverized in liquid nitrogen and homogenized in phosphate buffer according to Sugiura and Murakami (1990). The protein concentration of the homogenates was determined by using the Bio-Rad Protein Assay Kit (Bio-Rad Laboratories, USA). Aliquots of protein were loaded on the gel after incubation of 10 min at 65°C in

Table 1. Effects of a 54-week strength and power training program on the maximal anaerobic cycling power and maximal jumping height and corresponding work. Values are means (\pm SD).

	Maximal anaerobic cycling		Maximal vertical jumping			
	power (W)		height (cm)		work during jump (J)	
	Before	After	Before	After	Before	After
Training group (n=16)	538 (78)	602 (87)***	32.8 (4.3)	34.3 (5.2)*	267 (52)	281 (63) **
Control group (n=6)	474 (138)	512 (142)	30.3 (5.7)	28.7 (4.9)	213 (59)	204 (41)

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ in comparison with pre-training period.

lysis buffer, containing 10% (v/v) glycerol, 5% (v/v) 2-mercaptoethanol, 2.3% SDS, 0.05% bromphenole blue, and 62.5 mM TRIS-HCl pH 6.8.

The MHC isoforms were separated by 5–8% gradient SDS-PAGE gel using 0.75 mm thick gradient separating gel and 3.5% stacking gel (Bär and Pette, 1988). Electrophoresis was performed using vertical slab gel system (Protean II Xi Bio-Rad). 1 μ g myofibrillar protein sample was loaded on gel. Electrophoresis took 24 h at 120V. The gels were silver stained by using the Bio-Rad Silver-Stain Plus Kit according to the manufacturer's instructions.

The MLC isoforms were separated by 12.5% one-dimensional SDS PAGE gel system according to Laemmli (1970), except that the glycerol content in the separating gel was 10%. 10 μ g myofibrillar protein sample was loaded on 1mm thick gel per well. Electrophoresis was carried out at a constant current (30mA) using mini-Protean II Bio-Rad Electrophoresis Cell. The gels were Coomassie blue R-250 stained. The positions of MLC isoforms on the gel were identified by their apparent molecular masses, in comparison with protein mobility of the kaleidoscope pre-stained standard marker proteins (Bio-Rad) and by reports in the literature. Staining reactions of MHC and MLC were quantified by a computer-based image analysis system and software (Image Master 1D, Amersham Pharmacia Biotech).

Statistical analysis

All results are presented as means \pm SD. Differences

between the variable means of the training and control groups were assessed using Kruskal-Wallis test, and changes within groups using Wilcoxon's matched-pairs signed-ranks test. Pearson's correlation coefficients or Spearman's rank correlation coefficients were calculated to evaluate the associations between the variables and their changes. If necessary for Pearson's correlation coefficients, logarithmic transformations were applied to correct skewed data distributions. Differences were considered significant at $p < 0.05$. Logical correlation coefficients are presented for evaluating reliability and accuracy of electrophoretic MLC analyses.

RESULTS

The mean values of maximal anaerobic cycling power and maximal jumping height at baseline and after the intervention are presented in Table 1. On the average, the control group was 3.8 years younger (ns) and weighed 13.4 kg less ($p < 0.05$) than the training group. At baseline, the groups also differed for the myosin heavy chain distribution: the proportion of MHC IIa was 17.6 %-units higher and MHC IIx was 11.3 %-units lower in the control group ($p < 0.05$ for both; Table 2). During the training period, maximal anaerobic cycling power increased by 64 W ($p < 0.001$) and the maximal jumping height by 1.5 cm ($p < 0.05$) in the training group, but no significant changes were found in the control group (Table 1). However, the group by time

Table 2. Proportion of myosin heavy chain (MHC) isoforms (%) before and after a 54-week strength and power training program in the training and control groups. Values are means (\pm SD).

Groups	MHC isoforms (%)					
	Before			After		
	MHC I	MHC IIa	MHC IIx	MHC I	MHC IIa	MHC IIx
Training group (n=16)	29.1 (13.3)	52.6 (12.2) [#]	18.1 (11.4) [#]	29.4 (10.9)	59.4 (11.6)	11.1 (9.1)*
Control group (n=6)	22.8 (14.9)	70.2 (17.2)	6.8 (10.9)	25.8 (21.0)	67.3 (20.6)	7.0 (13.0)

* $p < 0.05$ in comparison with pre-training period, [#] $p < 0.05$ in comparison with control group.

Table 3. Proportion of myosin light chain (MLC) isoforms¹ in the *vastus lateralis* muscle before and after a 54-week strength and power training program in training group subjects (n = 8). Values are means (\pm SD).

	MLC1s	MLC1f	MLC2s	MLC2f	MLC3
Before	32.0 (5.4)	23.9 (3.8)	13.6 (3.8)	17.8 (5.0)	12.7 (5.2)
After	30.6 (4.4)	25.5 (2.0)	13.7 (2.9)	18.8 (5.4)	11.3 (4.6)

¹s = slow type, f = fast type

effect was not significant. In the training group, the increase of the maximal jumping height correlated with the number of strength and power training sessions ($r = 0.56$; $p < 0.05$).

After 54 weeks of strength and power training the proportion of MHC I isoform remained the same (Table 2). The change of the proportion of MHC IIa isoform from $52.6 \pm 12.2\%$ to $59.4 \pm 11.6\%$ did not reach statistical significance ($p = 0.070$ for group by time; within training group $p = 0.061$) and neither did the change of the proportion of MHC IIx isoform from $18.1 \pm 11.4\%$ to $11.1 \pm 9.1\%$ ($p = 0.104$ for group by time; within training group $p = 0.032$) (Table 2). The degree of change of MHC IIx isoform correlated with the earlier recreational sports activity during adult age ($r = 0.61$; $p < 0.05$). No changes in MHC isoforms were found in the control group.

During the training period, the proportion of MLC1f changed from $23.9 \pm 3.8\%$ to $25.5 \pm 2.0\%$ and the proportion of MLC2f changed from $17.8 \pm 5.0\%$ to $18.8 \pm 5.4\%$ in the training group. None of the changes in MLC isoforms reached statistical significance (Table 3). In the training group, the changes of MLC1f isoform correlated negatively with those of MLC1s isoform ($r = -0.79$; $p < 0.05$), and the changes of MLC1s isoform correlated positively with changes of MHC I isoform ($r = 0.81$; $p < 0.05$) and negatively with changes in maximal anaerobic cycling power ($r = -0.81$; $p < 0.05$).

DISCUSSION

Long-term strength and power training improved the maximal anaerobic cycling power in all subjects in the training group, and the mean maximal jumping height in the training group. These changes were in line with a tendency of increased proportion of MHC IIa and a decreased proportion of MHC IIx isoforms. Similar adaptive responses of MHC IIx to regular exercise training have been shown in previous studies (e.g., Campos et al., 2002; Sharman et al., 2001). Both endurance and strength and power types of physical activity decrease the proportion of the fastest human MHC isoform (Andersen et al., 1994; Carroll et al., 1998; Klitgaard et al., 1990; Staron et al., 1994; Tajsharghi et al., 2004). We found no studies in previous literature where exercise training would have caused an increase in

the proportion of MHC IIx isoform. The proportion of MHC IIx isoform only increases after declined neuromuscular activity, such as detraining (Andersen and Aagaard, 2000), or during inactivity, such as immobilization (Talmadge et al., 2002) or long-term bed rest (Andersen et al., 1999; Hostler et al., 2001; Trappe et al., 2004).

In the present study, the training lasted for 54 weeks and decreased the proportion of MHC IIx by 7.0 %-units. Similar decreases of MHC IIx have been reported in other studies with shorter exercise interventions (Campos et al., 2002; Putman et al., 2004; Sharman et al., 2001). In the study with young adults, only six weeks of exercise training (combined strength and endurance training) resulted in a significant decrease in MHC IIx content (Putman et al., 2004).

The increase of MHC IIa and decrease of MHC IIx isoforms have been suggested to reflect a transition from the fast to the slower type of MHC; so also in the present study. A similar MHC transition has been found after heavy resistance training in young (Adams et al., 1993; Campos et al., 2002; Liu et al., 2003) and old subjects (Sharman et al., 2001). Alternative, bi-directional MHC transitions from MHC I and MHC IIx to MHC IIa have previously been demonstrated after sprint training (Andersen et al., 1994). In the present study, the mean percentage of MHC I remained the same during the training period, which was comparable with the results of previous studies with resistance or strength training interventions (Campos et al., 2002; Hostler et al., 2001).

MHC profile analysis and histochemical fiber type analysis are not fully comparable. It has been shown that MHC profile responds faster to exercise stimulus than mATPase fiber profile does (Staron et al., 1994) and that, for example, part of histochemically assessed IIB fibers contain MHC IIa isoforms (Sant'ana Pereira et al., 1995). Thus, the MHC profile analyses are more sensitive measures to show changes in functional properties of muscle fibers as a result of exercise training than the mATPase fiber profile analyses do.

In the present study, strength and power training lasting 54 weeks tended to increase the proportion of MLC1f in the *vastus lateralis* muscle in the training group. In addition, the negative correlation between the changes of MLC1f and

MLC1s proportions ($r = -0.79$; $p < 0.05$) shows that these alterations were in the same direction in most of the subjects (6/8). Trappe and co-workers (2000; 2001) studied the effects of a 12-week resistance training intervention on MLC isoform proportions in subjects aged approximately 74 years and found no significant changes in single muscle fibers of *m. vastus lateralis*. Their exercise program, however, differed from our program: the exercises were performed with heavy loads (80% of 1 RM) and with slow speed which resulted in increased cell size, strength and contractile velocity in both slow- and fast MHC muscle fibers ('more pronounced in MHC I fibers') in men (Trappe et al., 2000) and only in MHC I muscle fibers in women (Trappe et al., 2001). In contrast, we used varying (30–85% of 1 RM) but mainly lower loads and high exercise tempo or high contractile velocity (power-type resistance training). Furthermore, the subjects in the study by Trappe et al. (2001) were older, and the training period was only a quarter of that in our study. Given that the duration of the training period in our study was much longer, that the subjects had rather high proportion of fast MHC muscle fibers in their *vastus lateralis* muscles, and that the training focused on the fast muscle fibers, the tentative changes in MLC isoforms can be assumed to be real. The small number of subjects and the fact that MHC and MLC fiber proportions were assessed using the muscle tissue homogenate instead of the single fiber method could be reasons for insufficient power of our study. Although the changes in the MLC isoforms during the long-term strength and power training were not statistically significant, they correlated with the changes in MHC profiles. The changes in MLC isoforms may possibly be associated with the transition of MHC isoforms as a result of the strength and power training and thus indicate improved muscle contraction. This, however, remains to be investigated in the future.

In previous studies, the shortest exercise period shown to affect the MHC isoform profile (results from the *triceps brachii* muscle) has been six weeks of strength training with maximum contractions and training 3 times a week (+17.3% in MHC IIa and -13.9% in MHC IIx isoforms) and with a combination of three types of training (once a week per type): strength training with maximum contractions, ballistic exercises, and stretch-shortening movements (+15% in MHC IIa and -9% in MHC I isoforms) (Liu et al., 2003). Training effects are perhaps detected earlier in the upper arm muscles than in the postural muscles, such as the *vastus lateralis* muscle.

Recently, Kyröläinen and co-workers (2005) demonstrated that, during a 15-week power training

period, drop jump increased in young, recreationally active men but there was no significant change in the MHC isoforms and muscle fiber proportions. This may be due to the high initial training status and the fact that the subjects continued their previous endurance-type sport activities (cycling, walking and ball games) for 6 hours a week on the average. In the present study, the strength and power training lasted for 54 weeks, but we only observed an increase of 6.8 %-units in MHC IIa and a decrease of 7.0 %-units in MHC IIx isoforms. In both studies, the subjects exercised 3 times a week, as targeted in our study. Strength training with maximum contraction causes a stronger stress for skeletal muscles, leading to muscle hypertrophy, than the varying exercises with varying relative loads carried out in our training program. The training focused more on developing the speed of muscle contraction than on increasing the maximal strength of muscles. It is also possible that the adaptive changes in skeletal muscle structure are not as extensive in middle-aged participants as in young participants, e.g., as a result from a smaller margin for MHC IIx to change due to lower baseline proportions (Short et al., 2005) and perhaps also a lower synthesis rate of MHC in the older people (Hasten et al., 2000). An additional reason for low training effects can be that the power type strength training was not carried out equally successfully by all the subjects. This suggestion was supported by the correlation coefficient of $r = 0.61$ between the amount of earlier recreational sports activity and the reduction of MHC IIx isoform reflecting the idea that the experienced subjects were more skilled and could perform exercises more effectively. However, in the both studies jumping performance increased significantly, perhaps due to neural adaptations (Häkkinen et al., 1985; Häkkinen and Häkkinen, 1995; Moritani and De Vries, 1980), but changes in MHC profiles did not reach statistical significance. That might be interpreted so that the type and/or amount of training was not proper for subjects and training included to many varying elements influencing on MHC profiles with opposite manner.

The present study raised a number of interesting questions that still remain open. In the future, studies should be designed with larger populations and with more homogeneous study groups. Furthermore, to get more reliable knowledge about training-induced adaptations in muscle tissue, to make correct interpretations, and to draw appropriate conclusions from the results, it is important to estimate the protein intake during the intervention. Similarly, it is critical to evaluate the effects of the current training status, the exercise background and the age of the study group on the

variables studied already before fixing the study design. Otherwise, it is very difficult to confirm any changes reliably. In addition, it is essential to have knowledge of the extent of simultaneous physical activities of the subjects so as to ensure a positive nitrogen balance and sufficient recovery time for the muscles to develop their structures and energy capacity during the intervention period.

The control group was small and heterogeneous for age, maximal anaerobic cycling power, and proportion of MHC IIx. Unfortunately, only few of the control subjects were willing to give samples of their muscle tissue. These matters and the size difference between the training and control groups reduce the reliability of comparisons between the groups to some extent.

CONCLUSION

The present study showed that 54 weeks of strength and power training caused minor adaptive changes of myosin isoforms in the *vastus lateralis* muscle in untrained middle-aged men. Our training program improved the maximal jumping height and the anaerobic cycling power of leg muscles probably due to neural changes. Adaptive changes in the muscle tissue took place in fast MHC isoforms; the proportion of MHC IIx tended to decrease and the proportion of MHC IIa tended to increase. Our results are in accordance with previous studies where more intensive, short-term strength training programs have been shown to cause MHC transformations. The possible connections between the decrease in the proportion of MHC IIx isoforms and the changes in MLC isoforms should be profoundly investigated in future.

ACKNOWLEDGMENTS

The authors wish to thank Ms Arja Kylliäinen for the statistical assistance and the Estonian Scientific Foundation for grants 1065 and 6501.

REFERENCES

- Adams, G.R., Hather, B.M., Baldwin, K.M. and Dudley, G.A. (1993) Skeletal muscle myosin heavy chain composition and resistance training. *Journal of Applied Physiology* **74**, 911–915.
- Andersen, J.L. and Aagaard, P. (2000) Myosin heavy chain IIx overshoot in human skeletal muscle. *Muscle & Nerve* **23**, 1095–1104.
- Andersen, J.L., Gruschy-Knudsen, T., Sandri, C., Larsson, L. and Schiaffino, S. (1999) Bed rest increases the amount of mismatched fibers in human skeletal muscle. *Journal of Applied Physiology* **86**, 455–460.
- Andersen, J.L., Klitgaard, H. and Saltin, B. (1994) Myosin heavy chain isoforms in single fibres from m. vastus lateralis of sprinters: influence of training. *Acta Physiologica Scandinavica* **151**, 135–142.
- Bär, A. and Pette, D. (1988) Three fast myosin heavy chains in adult rat skeletal muscle. *FEBS Letters* **235**, 153–155.
- Bosco, C., Luhtanen, P. and Komi, P.V. (1983) A simple method for measurement of mechanical power in jumping. *European Journal of Applied Physiology and Occupational Physiology* **50**, 273–282.
- Bottinelli, R. (2001) Functional heterogeneity of mammalian single muscle fibres: do myosin isoforms tell the whole story? *Pflügers Archiv: European Journal of Physiology* **443**, 6–17.
- Campos, G.E.R., Luecke, T.J., Wendel, H.K., Toma, K., Hagerman, F.C., Murray, T.F., Ragg, K.E., Ratamess, N.A., Kraemer, W.J. and Staron, R.S. (2002) Muscular adaptations in response to three different resistance-training regimens: specificity of repetition maximum training zones. *European Journal of Applied Physiology* **88**, 50–60.
- Carroll, T.J., Abernethy, P.J., Logan, P.A., Barber, M. and McEniery, M.T. (1998) Resistance training frequency: strength and myosin heavy chain responses to two and three bouts per week. *European Journal of Applied Physiology and Occupational Physiology* **78**, 270–275.
- Harber, M.P., Fry, A.C., Rubin, M.R., Smith, J.C. and Weiss, L.W. (2004) Skeletal muscle and hormonal adaptations to circuit weight training in untrained men. *Scandinavian Journal of Medicine & Science in Sports* **14**, 176–185.
- Hasten, D.L., Pak-Loduca, J., Obert, K.A. and Yarasheski, K.E. (2000) Resistance exercise acutely increases MHC and mixed muscle protein synthesis rates in 78–84 and 23–32 yr olds. *American Journal of Physiology, Endocrinology and Metabolism* **278**, E620–E626.
- Hostler, D., Schwirian, C., Campos, G., Toma, K., Crill, M.T., Hagerman, G.R., Hagerman, F.C. and Staron, R.S. (2001) Skeletal muscle adaptations in elastic resistance-trained young men and women. *European Journal of Applied Physiology* **86**, 112–118.
- Häkkinen, K. and Häkkinen, A. (1995) Neuromuscular adaptations during intensive strength training in middle-aged and elderly males and females. *Electromyography and Clinical Neurophysiology* **35**, 137–147.
- Häkkinen, K., Komi, P.V. and Alen, M. (1985) Effect of explosive type strength training on isometric force- and relaxation time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiologica Scandinavica* **125**, 587–600.
- Ingalls, C.P., Barnes, W.S. and Smith, S.B. (1996) Interaction between clenbuterol and run training: effects on exercise performance and MLC isoform content. *Journal of Applied Physiology* **80**, 795–801.

- Klitgaard, H., Zhou, M. and Richter, E.A. (1990) Myosin heavy chain composition of single fibres from m. biceps brachii of male body builders. *Acta Physiologica Scandinavica* **140**, 175–180.
- Kyröläinen, H., Avela, J., McBride, J.M., Koskinen, S., Andersen, J.L., Sipilä, S., Takala, T.E.S. and Komi, P.V. (2005) Effect of power training on muscle structure and neuromuscular performance. *Scandinavian Journal of Medicine & Science in Sports* **15**, 58–64.
- Laemmli, U. (1970) Cleavage of structural proteins during the assembly of the head of bacteriophage T4. *Nature* **227**, 680–686.
- Larsson, L. and Moss, R.L. (1993) Maximum velocity of shortening in relation to myosin isoform composition in single fibres from human skeletal muscles. *Journal of Physiology (London)* **472**, 595–614.
- Liu, Y., Schlumberger, A., Wirth, K., Schmidtbleicher, D. and Steinacker, J.M. (2003) Different effects on human skeletal myosin heavy chain isoform expression: strength vs. combination training. *Journal of Applied Physiology* **94**, 2282–2288.
- Lowey, S., Waller, G.S. and Trybun, K.M. (1993) Skeletal muscle myosin light chains are essential for physiological speed of shortening. *Nature* **365**, 454–456.
- Moritani, T. and De Vries, H. (1980) Potential for gross muscle hypertrophy in older men. *Journal of Gerontology* **35**, 672–682.
- Pette, D. and Staron, R.S. (2001) Transitions of muscle fibre phenotypic profiles. *Histochemistry and Cell Biology* **115**, 359–372.
- Putman, C.T., Xu, X., Gillies, E., MacLeon, I.M. and Bell, G.J. (2004) Effects of strength, endurance and combined training on myosin heavy chain content and fibre-type distribution in humans. *European Journal of Applied Physiology* **92**, 376–384.
- Rusko, H., Nummela, A. and Mero, A. (1993) A new method for the evaluation of anaerobic running power in athletes. *European Journal of Applied Physiology and Occupational Physiology* **66**, 97–101.
- Sant'ana Pereira, J.A.A., Wessels, A., Nijtmans, L., Moorman, A.F.M. and Sargeant, A.J. (1995) New method for the accurate characterization of single human muscle fibres demonstrated a relation between mATPase and MyHC expression in pure and hybrid fibre type. *Journal of Muscle Research and Cell Motility* **16**, 21–34.
- Sharman, M.J., Newton, R.U., Triplett-McBride, T., McGuigan, M.R.M., McBride, J.M., Häkkinen, A., Häkkinen, K. and Kraemer, W.J. (2001) Changes in myosin heavy chain composition with heavy resistance training in 60- to 75-years-old men and women. *European Journal of Applied Physiology* **84**, 127–132.
- Short, K.R., Vittone, J.L., Bigelow, M.L., Proctor, D.N., Coenen-Schimke, J.M., Rys, P. and Nair, K.S. (2005) Changes in myosin heavy chain mRNA and protein expression in human skeletal muscle with age and endurance exercise training. *Journal of Applied Physiology* **99**, 95–102.
- Staron, R.S., Karapondo, D.L., Kraemer, W.J., Fry, A.C., Gordon, S.E., Falkel, J.E., Hagerman, F.C. and Hikida, R.S. (1994) Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. *Journal of Applied Physiology* **76**, 1247–1255.
- Sugiura, T. and Murakami, N. (1990) Separation of myosin heavy chain isoforms in rat skeletal muscle by gradient sodium dodecyl sulfate-polyacrylamide gel electrophoresis. *Biomedical Research* **11**, 87–91.
- Tajsharghi, H., Stibrant Sunnerhagen, K., Darin, N., Kyllerman, M. and Oldfors, A. (2004) Induced shift in myosin heavy chain expression in myosin myopathy by endurance training. *Journal of Neurology* **251**, 179–183.
- Talmadge, R., Castro, M., Apple, D. and Dudley, G. (2002) Phenotypic adaptations in human muscle fibers 6 and 24 wk after spinal cord injury. *Journal of Applied Physiology* **92**, 147–154.
- Trappe, S., Godard, M., Gallagher, P., Carroll, C., Rowden, G. and Porter, D. (2001) Resistance training improves single muscle fiber contractile function in older women. *American Journal of Physiology, Cell Physiology* **281**, C398–C406.
- Trappe, S., Trappe, T., Gallagher, P., Harber, M., Alkner, B. and Tesch, P. (2004) Human single muscle fibre function with 84 day bed-rest and resistance exercise. *Journal of Physiology* **557 (Pt2)**, 501–513.
- Trappe, S., Williamson, D., Godard, M., Porter, D., Rowden, G. and Costill, D. (2000) Effect of resistance training on single muscle fiber contractile function in older men. *Journal of Applied Physiology* **89**, 143–152.
- Wada, M., Inashima, S., Yamada, T. and Matsunaga, S. (2003) Endurance training-induced changes in alkali light chain patterns in type IIB fibers of the rat. *Journal of Applied Physiology* **94**, 923–929.
- Wahrman, J.P., Winand, R. and Rieu, M. (2001) Plasticity of skeletal myosin in endurance-trained rats (I). A quantitative study. *European Journal of Applied Physiology* **84**, 367–372.

AUTHORS BIOGRAPHY

Raivo PUHKE

Employment

Researcher at the Institute of Exercise Biology and Physiotherapy, University of Tartu, Tartu, EST

Degree

MSc, PhD student

Research interests

Contractile proteins, muscle adaptation, exercise training

E-mail: raivo.puhke@ut.ee

Sirkka AUNOLA

Employment

Senior Researcher at the Laboratory for Population Research, Department of Health and Functional Capacity, National Public Health Institute, Turku, FIN

Degree

PhD

Research interests

Muscle metabolism, strength and power training, prevention of metabolic syndrome, anaerobic threshold

E-mail: sirkka.aunola@ktl.fi

Pirjo AILANTO

Employment

Instructor and Planner at the Finnish Back Association, Turku, FIN

Degree

MSc, PhD student in the Department of Physiology, University of Kuopio, Kuopio, FIN

Research interests

Strength and power training, physical functioning

E-mail: pailanto@saunalahti.fi

Karin ALEV

Employment

Researcher at the Institute of Exercise Biology and Physiotherapy, University of Tartu, Tartu, EST

Degree

PhD

Research interests

Contractile proteins, muscle adaptation, exercise training

E-mail: karin.alev@ut.ee

Mika VENOJÄRVI

Employment

Senior Lecturer in the Medical Laboratory Technology, Turku University of Applied Sciences, Turku, FIN

Degree

MSc, PhD student in the Department of Physiology, University of Kuopio, Kuopio, FIN

Research interests

Exercise nutrition and glucose uptake, muscle metabolism and oxidative stress

E-mail: mika.venojarvi@turkuamk.fi

Heikki RUSKO

Employment

Professor at the Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, FIN

Degree

PhD

Research interests

Altitude and endurance training, heart rate variability related to stress–overtraining–burnout, determinants of endurance performance

E-mail: heikki.rusko@sport.jyu.fi

Teet SEENE

Employment

Professor emeritus at the Institute of Exercise Biology and Physiotherapy, University of Tartu, Tartu, EST

Degree

PhD, MD

Research interests

Muscle morphology, structure of skeletal muscle, contractile proteins

E-mail: teet.seene@ut.ee

KEY POINTS

- A long-term strength and power training program seemed to decrease the proportion of MHC IIx isoform in previously untrained middle-aged men.
- The degree of change of MHC IIx isoform correlated with the amount of earlier recreational sports activity.
- The changes of MLC isoforms were associated with the transition of MHC isoforms. Whether this means improved speed and coordination of muscle contraction remains to be investigated in the future.

✉ Sirkka Aunola

National Public Health Institute, Peltolantie 3, FI-20720, Turku, Finland

APPENDIX

Strength and power training program, duration 54 weeks.

Period	Length of period (weeks)	Type of training	Sessions / week	Amount of repetitions /session (sets / reps / % 1RM / recovery time)	Number of exercises / session
I	6	basic strength / circuit training	3	3 x 10–20 reps at ~60% of 1RM, 5 min between circuits ^s	10
II	4	basic strength / consecutive training	3	4 x 4–15 / 70% / 1–2 min / 5 min between sets	7
III and IV	4	(a) power training / circuit training	1 x a, 2 x b	3 (III) / 4 (IV) sets x 10 s at ~50% / 5 min between circuits	6–7
		(b) basic strength / consecutive training	2 x a, 1 x b	5–3–X*–3 reps at 70% / 1–2 min / 5 min between sets	6
V	2	outdoors sports	1–2	(free program ~ recovery phase)	–
VI and VII	6	(a) power training / circuit training	1 x a, 2 x b	as phase I, but leg press at 70% of 1RM	7–8
		(b) basic strength	2 x a, 1 x b	6–4–X*–4 reps at 70% / 2–3 min / 5 min between sets	7–8
VIII a	4	circuit training	1	4 x 15–30 reps / 5 min between sets	8
		training for elasticity	2	swing of the shot or medicine ball forward with two hands, standing broad-jump, multi-jump / jumping over low hurdles, spurts	6–8
VIII b	2–3	basic strength	2	as phase VI b	7
		running exercise	2	10 x about 100 m	1
VIII c	4	circuit training	1	as phase VIII a	8
		training for elasticity	2	as phase VIII a	6–8
IX	3	power training	2	5 circuits, as phase VI a	7
		running exercise	1	as phase VIII b	1
X	2	power training	1	as phase IX	7
		basic strength	2	as phase VI	6
XI	4	power training / circuit training	2	6 reps at 70% – 6 reps at 75% – 4 reps at 85% – 6 reps at 79% / 2–3 min / 5 min between circuits	6
		basic strength	1	4 x 8 at ~70%	7
XII	2	power training	1	as phase XI	6
		self-selected sports	1	free program / recovery phase	–

^s stretching exercises during the recovery time

* X = maximal number of repetitions (reps), but not over 20 reps