Different effects on human skeletal myosin heavy chain isoform expression: strength vs. combination training

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1Section of Sports and Rehabilitation Medicine, Department of Medicine II, University of Ulm, D-89070 Ulm; 2Institute of Sports Sciences, Department 1: Sport and Movement, Johann-Wolfgang-Goethe University, 60054 Frankfurt am Main; and 3Eden-Reha, Rehabilitation Clinic, 93093 Donaustauf, Germany

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Liu, Y., A. Schlumberger, K. Wirth, D. Schmidtbleicher, and J. M. Steinacker. Different effects on human skeletal myosin heavy chain isoform expression: strength vs. combination training. J Appl Physiol 94: 2282–2288, 2003; 10.1152/japplphysiol.00830.2002—Myosin heavy chain (MHC) isoform expression changes with physical training. This may be one of the mechanisms for muscular adaptation to exercise. We aimed to investigate the effects of different strength-training protocols on MHC isoform expression, bearing in mind that α-MHCslow (newly identified MHC isoform) mRNA may be up-regulated in response to training. Twelve volunteers performed a 6-wk strength training with maximum contractions (Max group), and another 12 of similar age performed combination training of maximum contractions and ballistic and stretch-shortening movements (Combi group). Muscle samples were taken from triceps brachii before and after training. MHC isoform composition was determined by SDS-PAGE silver staining, and mRNA levels of MHC isoforms were determined by RT-PCR. In Max group, there was an increase in MHC2A (49.4 to 66.7%, P < 0.01) and a decrease in MHC2X (33.4 to 19.5%, P < 0.01) after training, although there was no significant change in MHCslow. In Combi group, there was also an increase in MHC2A (47.7 to 62.7%, P < 0.05) and a decrease in MHCslow (18.2 to 9.2%, P < 0.05) but no significant change in MHC2X. An upregulation of α-MHCslow mRNA was, therefore, found in both groups as a result of training. The strength training with maximum contractions led to a shift in MHC isoform composition from 2X to 2A, whereas the combined strength training produced an MHC isoform composition shift from slow to 2A.

The skeletal muscle, one of the most important components responsible for physical performance and adaptation to exercise, is an extremely heterogeneous tissue in both structure and function (26). The alteration of myosin heavy chain (MHC) isoform composition in the muscle with exercise contributes greatly to this heterogeneity and serves as an important mechanism for muscle adaptation to exercise. The multigene families of MHC may be an important substrate to warrant this heterogeneity (32). For instance, four MHC isoforms, i.e., α-MHCslow, MHC2A, MHC2X, and MHC2B, can coexist in the hybrid muscle fibers of small animals (27). The MHC isoform equivalent to the MHC2B in small animals has not yet been identified in humans. Much effort has been made to characterize more MHC isoforms. In particular, the successful characterization of a new slow MHC isoform, termed α-MHCslow, has attracted considerable attention recently (12, 28, 30). α-MHCslow, an isoform of slow MHC, was thought to be expressed abundantly in the myocardium (3, 23) and some other special muscles like masseter (4–6, 37) and extraocular muscle (33). α-MHCslow is functionally distinct from the isoform β-MHCslow (9) and can be considered as an intermediate between MHC2A and β-MHCslow (28). It has been reported that α-MHCslow can be transiently expressed in rabbits during fast-to-slow transition and, therefore, may play an important role in the muscle adaptation to exercise or other stimuli (29).

A number of studies have shown that, with an increase in neuromuscular activities, the MHC isoform may shift from fast (2B or 2X) to slow (MHCslow), leading to a fast-to-slow muscle fiber transition (27). It would seem to be apparent that exercise training results in changes in neuromuscular activities, recruitment of different motor units, energy metabolism, and hormonal responses. This may lead to changes in MHC isoform composition, and exercise-induced change of MHC isoform composition seems to be dependent on the kinds of training, muscle groups, and energy metabolism.

Strength training is frequently used to improve the development of force and velocity. Subsequently, various motor units with different recruitment thresholds will be activated (8). It is also well known that strength training has significant impact on MHC isoform expression at the protein level (2). In strength training with maximum contractions, the fast motor units with a high threshold will be activated, leading to a fast-to-slow shift in MHC isoform profile (17). A recent observation demonstrated that a combination of different training methods may improve the effects of strength training on force development (13, 39). The strength

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1By definition, MHC denotes the myosin heavy chain at the protein level, whereas MHC isoforms at the mRNA level are especially denoted by mRNA.

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training combined with high speed and power, for example, provides a superior stimulus for enhancing intra- and intermuscular coordination (10).

Although there have been a number of studies on the relation between training and MHC isoform in leg muscles, there are relatively fewer data available on MHC isoforms derived from arms and, to our knowledge, no reports on the effects of strength training combined with very fast ballistic and stretching-shortening movement on MHC isoform expression. We have, therefore, established a new method for strength training that combines the traditional strength training with ballistic movement at lower workload (30%) and stretch-shortening movement. We hypothesized that this new training approach would have different effects on the physical performance and MHC isoform compared with the traditional strength training. Thus, in this study, we aimed to investigate the effects of the new combined method for strength training on physical performance and MHC isoform expression at both the protein and mRNA level, particularly $\alpha$-MHC$_{\text{slow}}$ mRNA in the triceps brachii.

**METHODS**

**Subjects.** Twenty-four male physical education students with experience in strength training were enrolled in this study. They all had had 3 mo to 5 yr of regular strength training for the arms. They were randomized into two groups: the Max group received the traditional strength training with maximum contractions, and the Combi group received the strength training combined with ballistic and stretch-shortening contractions. Age, height, and body mass were recorded (Table 1). This study was approved by the Ethics Committee of the Medical Faculty of the University of Ulm (Germany), and written consent was obtained from all participants.

**Training protocol.** The training was performed on Monday, Wednesday, and Friday each week for 6 wk. In the Max group, the same training was carried out on the 3 training days: bench press with maximum contractions [3-repetitions maximum (RM) load, determined by 1 RM and stretch-shortening movements]. The subjects were instructed to apply force as quickly as possible and throw the bar with maximum velocity. Each subject was given three trials, interspersed by a 30-s interval. The velocity measurements were made by means of an optical reflective sensor system (Fichte, University of Frankfurt, Germany) with an accuracy of 4 mm and 1/5,000 s. The highest values were taken for statistical analyses.

To ensure sufficient familiarization with the protocol, maximum strength and maximum velocity testing was carried out twice before training. Posttraining testing was performed 3 and 11 days after the completion of the protocol. Again, the highest values were taken for statistical analysis. The correlation coefficients of the two pretraining sessions were 0.98 and 0.94 (both $P < 0.01$) for 1 RM and maximum movement velocity, respectively. Coefficient of variation for 1 RM was 2.8% for 1 RM and 2.2% for maximum movement velocity.

**Muscle biopsy.** Muscle biopsy was carried out at rest on a nontraining day, 3 days before and 7 days after the training protocols. Muscle samples were taken from the long head of $m$. triceps brachii of the dominant side of the subjects by using the fine-needle biopsy technique (20, 21). After routine disinfection of the skin, a 13-gauge biopsy needle (Peter Pfluegbeil Medizinische Instrumente, Zorneding, Germany) was punctured 1 cm into the muscle belly, and biopsy gauge was shot three times to attain $\approx$3 mg of muscle tissue. The sample was immediately frozen in liquid nitrogen and then stored at $-80^\circ$C.

**MHC analysis.** The muscle sample was homogenized in 50 ml extraction buffer (40) containing 100 mmol/l Na$_2$P$_2$O$_7$·10H$_2$O, 5 mmol/l EGTA, 5 mmol/l MgCl$_2$·6H$_2$O, 0.3 mmol/l KCl, and 1 mmol/l dithiothreitol with an ultrasonic homogenizer (Bandelin Sonopuls Homogenisator HD2070, Berlin, Germany). The muscle homogenates were stirred on ice for 20 min and centrifuged at 4°C and 16,000 g for 10 min. The supernatant was collected and mixed with an equal volume of glycerol. The total protein concentration was determined according to Lowry et al. (22). The protein solution was prepared by the method reported by Peuker and Pette (31). The primer sequences used and the corresponding RT-PCR products for each of the MHC isoforms are summarized in Table 2. The reaction conditions and proce-
imens were described in detail elsewhere (30, 31). In brief, the total reaction volume for each sample was 25 μl and contained 2 μl cDNA solution, 25 pmol of each primer, 100 μM of each 2-deoxynucleotide 5'-triphosphate, 2 mM MgCl₂, and 0.5 unit of Taq polymerase. Thirty cycles of 45 s at 94°C, 60 s at 56°C, and 30 s at 72°C were performed in an automatic incubation system (Crocodile III, Appligene, F-67402, Illkirch, France). The RT-PCR products were densitometrically measured on 3% agarose gel containing ethidium bromide-stained agarose gel for analyses of MHC isoform composition (38). 

For the MHC proteins, the amount of each isoform was calculated as an integral value from each RT-PCR product as a percentage. The mRNA levels of MHC isoforms was estimated in three steps: 1) an integral value from each RT-PCR product as a percentage. The increase in maximum movement velocity was significantly greater in the Combi group than in the Max group (0.1 vs. 0.07 m/s, P < 0.05; Table 3). All muscle samples were successfully analyzed. In the Max group, there was a significant increase in MHC2A (49.4 to 66.7%, P < 0.01) and a decrease in MHC2X (33.4 to 19.5%, P < 0.01), whereas the change in MHCslow (17.2 to 13.8%) was not statistically significant (Fig. 2A). In the Combi group, there was also an increase in MHC2a (47.7 to 62.7%, P < 0.05), no change in MHC2x (34.1 to 28.1%), but a significant decrease in MHCslow (18.2 to 9.2%, P < 0.01; Fig. 2B). The mRNA levels of β-MHCslow, MHC2A, and MHC2X did not change (Fig. 3), whereas α-MHCslow mRNA increased significantly in both groups. The increase in α-MHCslow mRNA in the Max group (from 100 to 308%,

Table 2. Primer sequences and products of RT-PCR performed in the study by Peuker and Pette (31)

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Sense</th>
<th>Antisense</th>
<th>Product Size, bp</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-Actin</td>
<td>CgCgACATCACAgAgAaAgCT</td>
<td>GggCgATgATCCATCTTC</td>
<td>367</td>
</tr>
<tr>
<td>α-MHCslow</td>
<td>CggCTACAgAgCCCTgAgAACA</td>
<td>ATTACAggTGgCAAgAgTgAg</td>
<td>265</td>
</tr>
<tr>
<td>β-MHCslow</td>
<td>ACAAgAgCgATcAaAgAgTC</td>
<td>TCAAgAgTgAgCAgAgCCTAC</td>
<td>219</td>
</tr>
<tr>
<td>MHC2A</td>
<td>AggCTCCAaAATTgAaAgTA</td>
<td>TTTCCCTTGCAAgAggTAGA</td>
<td>325</td>
</tr>
<tr>
<td>MHC2X</td>
<td>AAgAgCgAgCggAggTTCAaCAGC</td>
<td>TTATCTCAAAAGCATAAgTACA</td>
<td>132</td>
</tr>
</tbody>
</table>

MHC, myosin heavy chain.
DISCUSSION

The alteration of MHC isoform composition serves as an important mechanism for skeletal muscle adaptation to exercise. Changes in neuromuscular activities secondary to exercise may lead to such alternations. In the present study, we investigated the MHC isoform expression at protein and mRNA levels in response to strength training and found that, after 6-wk training, MHC2A increased significantly, along with a decrease in MHC 2X in maximum contraction training and a decrease in MHC slow at the protein level in combination training. On the other hand, both training strategies improved the maximum strength (1 RM) significantly (Table 3). However, the improvement in the maximum movement velocity was statistically significant only in the Combi group.

At first sight, our results are similar to those of previous studies (7, 13, 35). Harris et al. (13) demonstrated that a combined strength training was superior to the strength training with high force or high power alone with regard to the improvement of a wide variety of athletic performance variables requiring strength, power, and speed. Newton et al. (24) showed that the ballistic training, in addition to resistance training, in well-trained athletes could improve the explosive force (vertical jump). The effects of training on muscles have been documented at muscle fiber-type transition and MHC isoform at the protein level. In general, strength training leads to fast-to-slow muscle fiber transition (Ib → IId/x → Ia → I) and to distribution shift of MHC isoform composition at the protein level from 2B → 2X → 2A → slow (27). However, the effects on MHC isoform composition may vary with different training methods and muscles studied. In this study, the training with maximum contractions led to an increase in MHC2A and a decrease in MHC2X at the protein level, indicating an MHC isoform composition shift: 2X → 2A. This is indeed similar to the findings by Allemeier et al. (1) and Jürimäe et al. (17). The combined strength training in this study produced an increase in MHC2A and a decrease in MHCslow at the protein level, suggesting an MHC isoform composition shift: slow → 2A. This is of novelty, as there has not been similar findings showing an exercise-induced slow-to-fast shift in MHC isoform composition at the protein level in the literature to date, although Jansson et al. (15) demonstrated a 4- to 6-wk sprint training-induced slow-to-fast muscle fiber transition (slow → Ila) in vastus lateralis using myofibrillar ATPase stain.

Table 3. Training effects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>1 RM, kg</th>
<th>Vmax, m/s</th>
<th>ΔVmax, m/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before training</td>
<td>83.8 ± 19.3</td>
<td>2.89 ± 0.32</td>
<td>0.07 ± 0.09</td>
</tr>
<tr>
<td>After training</td>
<td>89.4 ± 22.7</td>
<td>2.96 ± 0.35</td>
<td>0.10 ± 0.11</td>
</tr>
<tr>
<td>Combi group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before training</td>
<td>83.6 ± 20.1</td>
<td>2.97 ± 0.31</td>
<td></td>
</tr>
<tr>
<td>After training</td>
<td>88.6 ± 19.4</td>
<td>3.07 ± 0.26</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. 1 RM, one repetition maximum; Vmax, maximum movement velocity; ΔVmax, difference in Vmax before vs. after training. Significant difference vs. before training in the same group: *P < 0.05, †P < 0.01.

P < 0.01; Fig. 3A) was significantly greater than that in the Combi group (from 100 to 160%, P < 0.01; Fig. 3B).
The strength training-induced increase in MHC2A was of improvement in performance as a result of training. In the present study, the maximum movement velocity in the Max group did not decrease as a result of the 2X strength, which was documented in both groups. In the Max group, the lack of a significant increase in maximum velocity may be attributed to the reduced MHC2X composition, which then attenuated the effect of the slow → 2A shift in MHC isoform composition. However, the maximum movement velocity in the Max group did not decrease as a result of the 2X → 2A shift. This was probably a compensatory response as the decrease in MHCslow was not significant. In the Combi group, MHC2X was better preserved, which, together with the slow → 2A shift in isoform composition, has possibly led to a significant improvement in maximum movement velocity.

We are not absolutely certain why the two training protocols produced different MHC isoform compositions. One, however, should consider the following. First, it is known that the neural adaptation plays an important role in the strength training (34). The neural impact on muscle response to training may exert at the level of motor units with different recruitment thresholds and at the level of firing rates (8). The two training protocols may differ in the activation of neural control, the recruitment of motor units, and the firing models. Second, mechanical factors may contribute to the differences between the two groups. It is apparent that the ballistic and stretch-shortening movements in the Combi group have different mechanical impacts on muscle compared with the maximum contractions.

The mRNA of all MHC isoforms except α-MHCslow did not change after the 6-wk strength training, which differed from the results of MHC isoform composition at the protein level. This discrepancy between protein and mRNA level may be mainly attributed to their different kinetics (16). Jashinski and colleagues (16) determined the time-dependent decay of MHC2B mRNA in low-frequency-stimulated rat extensor digitorum longus muscle at both the protein and mRNA level. The decay of MHC2B mRNA was much faster than that of MHC2B protein (half-time = 62 h vs. 11 days). This discrepancy, on the other hand, suggests that the post-transcriptional mechanisms play an important role in muscular adaptation to exercise.

The changes in α-MHCslow mRNA aroused much interest. α-MHCslow is considered to be an intermediate between β-MHCslow and MHC2A in functional characteristics (9, 14). The upregulated expression of α-MHCslow thus suggests an active transition procedure in MHC isoforms (28, 29). However, there have not been similar observations on α-MHCslow mRNA in humans. In our present study, we have investigated the expression of α-MHCslow at the mRNA level, and the results show that the level of α-MHCslow mRNA was significantly elevated in both groups, whereas the mRNA level of the other MHC isoforms remained unchanged. This elevated level of α-MHCslow mRNA may suggest that the alteration in MHC isoform composition induced by strength training was not accomplished over the study period. Another possibility is that satellite cells were activated through exercise (36). It has been reported that, in animals with compensatory muscular hypertrophy, satellite cells are activated with increased muscle activities (36). Because α-MHCslow is considered to be an embryonic form of MHC, the activation of satellite cells may well be responsible for an upregulation in α-MHCslow mRNA observed in the present study. This would, therefore, explain why there was a persistent high level of α-MHCslow mRNA. Unfortunately, the separation of the two isoforms of MHCslow at the protein level, i.e., α-MHCslow and β-MHCslow, was technically by no

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The changes in MHC isoform composition observed in the present study shed some light on the mechanism of improvement in performance as a result of training. The strength training-induced increase in MHC2A was probably responsible for the improvement in maximum strength, which was documented in both groups. In the Max group, the lack of a significant increase in maximum velocity may be attributed to the reduced MHC2X composition, which then attenuated the effect of the slow → 2A shift in MHC isoform composition. However, the maximum movement velocity in the Max group did not decrease as a result of the 2X → 2A shift. This was probably a compensatory response as the decrease in MHCslow was not significant. In the Combi group, MHC2X was better preserved, which, together with the slow → 2A shift in isoform composition, has possibly led to a significant improvement in maximum movement velocity.

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means simple and thus not performed in this study. It, therefore, remains unknown whether the expression of α-MHC slow has also changed at the protein level. In the present study, MHC slow at the protein level consisted of α-MHC slow and β-MHC slow. If α-MHC slow were a small part of MHC slow, the change in MHC slow at the protein level would be mainly that of β-MHC slow. This is supported by our present results, a significant increase in MHC slow at the protein level, along with a significant increase in α-MHC slow mRNA in the Combi group, but not in the Max group, although the increase in α-MHC slow mRNA posttraining was even greater than that in the Max group (Figs. 2 and 3). It has been demonstrated in animal study that α-MHC slow mRNA was upregulated during a fast-to-slow muscle fiber transition shown by single-fiber analysis (30), but it is not clear whether α-MHC slow mRNA can also be upregulated during a slow-to-fast muscle fiber transition. Because α-MHC slow is regarded as an intermediate between β-MHC slow and MHC2A, it is possible that α-MHC slow mRNA can be upregulated by the events of slow-to-fast muscle fiber or MHC isoform transition. In our present study, there is a shift in MHC isoform composition at the protein level from slow to 2A (i.e., slow to fast) in both groups, although it was not statistically significant in the Max group. This was accompanied by a clear upregulation of α-MHC slow mRNA. It is, therefore, possible that α-MHC slow mRNA can be upregulated during a shift in MHC isoform composition from slow to 2A.

In conclusion, a 6-wk strength training produces significant changes in isoform composition in the muscle of triceps brachii. Strength training with maximum contractions leads to an increase in MHC2A and a decrease in MHC2X, indicating a 2X → 2A shift in MHC isoform composition at the protein level, whereas strength training combined with maximum contraction and ballistic and stretch–shortening movement induces an increase in MHC2A and a decrease in MHC slow, indicating slow → 2A shift of MHC isoform composition. The strength training can produce a distinct up-regulation of α-MHC slow mRNA, the physiological significance of which deserves further investigations.

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