Reduction in hybrid single muscle fiber proportions with resistance training in humans

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Received 19 April 2001; accepted in final form 22 June 2001

Williamson, D. L., P. M. Gallagher, C. C. Carroll, U. Raue, and S. W. Trappe. Reduction in hybrid single muscle fiber proportions with resistance training in humans. J Appl Physiol 91: 1955–1961, 2001.—The purpose of this investigation was to examine the effects of 12 wk of progressive resistance training (PRT) on single muscle fiber myosin heavy chain (MHC; I, I/IIa, I/IIa/IIx, IIa, IIa/IIx, Ix) isoform proportions in young individuals. Young, untrained men (YM; n = 6) and women (YW; n = 6) (age = 22 ± 1 and 25 ± 2 yr for YW and YM, respectively) received pre- and post-PRT muscle biopsies from the right vastus lateralis for single muscle fiber MHC distribution by electrophoretic analysis (192 ± 5 pre- and 183 ± 6 post-fibers/subject analyzed; 4,495 fibers total). Data are presented as percentages of the total fibers analyzed per subject. The PRT protocol elicited an increase in the pure MHC Ila (Δ = +24 and +27; YW and YM, respectively; P < 0.05) with no change in the pure MHC I distribution. The hybrid MHC distributions decreased I/IIa/Ix (Δ = −2; YM and YW; P < 0.05), Ia/Ix (Δ = −13 and −19 for YM and YW, respectively; P < 0.05), and total hybrid fiber proportion (I/IIa + Ia/Ix + Ia/Ix/IIa) decreased (Δ = −19 and −30 for YM and YW, respectively; P < 0.05) with the training, as did the MHC Ix distribution (Δ = −2; YM only; P < 0.05). Alterations in the predominance of MHC isoforms within hybrid fibers (decrease in MHC I-dominant I/IIa and nondominant MHC IIa/Ix, increase in MHC IIa-dominant IIa/Ix; P < 0.05) appeared to contribute to the increase in the MHC Ia proportion. Electrophoresis of muscle cross sections revealed an 7% increase (P < 0.05) in MHC IIa proportion in both groups, whereas the MHC Ix decrease by 7.5 and 11.6% post-PRT in YW and YM, respectively. MHC I proportions increase in YM by 4.8% (P < 0.05) post-PRT. These findings further support previous resistance training data in young adults with respect to the increase in the MHC Ia proportions but demonstrate that a majority of the change can be attributed to the decrease in single-fiber hybrid proportions.

PROGRESSIVE RESISTANCE TRAINING (PRT) has been shown to increase muscle strength and size in both young and old men and women (1, 11, 12, 20). In previous studies on younger adults (1, 7, 19, 20), alterations in fiber proportions have also been reported. The most consistent findings have been a decrease in the type IIb fiber distribution with a reciprocal increase in the type Ila fiber distribution in young adults. Conversely, resistance training does not appear to induce changes in fiber distribution among older adults (11, 12). These studies used either muscle cross sections (myosin ATPase histochemistry) or homogenates (SDS-PAGE) to identify skeletal muscle fiber types or myosin heavy chain (MHC) content.

In contrast to previous findings, using a similar knee extensor protocol (11, 12), we found that 12 wk of PRT significantly increased MHC I and decreased MHC hybrid fibers (MHC I/IIa, I/IIa/Ix, IIa/Ix) in untrained, older men (OM; 74 ± 2 yr). Moreover, single muscle fiber function (i.e., contractile strength, velocity, and power) of the MHC I fiber distributions increased to a greater magnitude than did the MHC IIa fibers (21, 22) in an older population of men and women. These studies demonstrated that skeletal muscle of OM have the capability for phenotypic adaptation (i.e., plasticity). The differences in fiber-type adaptation in our studies contrasted with others due, in part, to the technique used to evaluate fiber distribution (i.e., single muscle fiber MHC-based fiber type), as well as the age of the subjects (74 ± 25 yr) (21, 25).

Aging skeletal muscle has a high amount of hybrid fibers (4, 15, 25, 16), which can be reduced with resistance training (21, 25). Resistance training studies in young individuals, examining fiber-type alterations, have typically utilized either multiple lower body exercises (e.g., knee extension, leg press, squat) and/or upper body exercises (e.g., bench press, arm curl, lateral pull-down) (1–3, 7, 19, 20). Whereas the majority of aging/resistance training studies have focused on the knee extensors (11, 12, 21, 25), the use of the same exercise protocol (used in our laboratory) in a group of young adults, as reported in many other aging/resistance training studies (11, 12, 25, 22), allowed for a

1For clarification purposes, human type IIb fibers have since been found to be homologous to the type IIx in the rat (18); thus MHC IIx will be used for classification in this investigation given the technique employed for fiber typing.

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comparison of skeletal muscle adaptation (i.e., an age effect).

Therefore, the purpose of this investigation was to examine the effects of PRT (12 wk) on single-fiber MHC isoform protein expression in young men and women. In addition, comparisons were made with our previous data in OM (25). We hypothesized that 1) hybrid fibers (i.e., I/IIa, I/IIa/IIx, and IIa/IIx) would decrease to the same degree as in OM (21, 25), 2) MHC I isoform expression would increase similarly to our previous findings in OM, and 3) whole muscle strength increases would be similar between the young adults and the OM, after the PRT.

METHODS

Subjects

Twelve healthy, young women (YW) and men (YM) (Table 1) were recruited from the community to serve as subjects in this investigation. The inclusion criteria for volunteers included untrained (aerobic, resistance, or other for 12 mo before the study), nonobese (body mass index <26 kg/m²), nonsmoking, normotensive, nonpregnant (women only) healthy men and women. All potential subjects underwent a telephone interview followed by a visit to the laboratory. To ensure that all subjects remained “untrained” except for the prescribed knee extensor training supervised by our investigative team, we assessed activities performed before each training session throughout the 12 wk. All potential subjects were informed of all procedures and risks associated with the screening, testing, and training. Informed consent was obtained from each volunteer, after the study was approved by the Internal Review Board of Ball State University and Ball Memorial Hospital.

Muscle Biopsy

Needle biopsies (~100–150 mg) were taken from the right vastus lateralis muscle (6). The muscle was immediately divided into longitudinal sections. A portion of the muscle was stored in a skinning solution (as described previously; Ref. 21) for single-fiber dissection and MHC analysis. Another section was vertically mounted in tragacanth gum (G-1128, Sigma Chemical, St. Louis, MO) and frozen in isopentane for muscle cross-sectional MHC-based fiber-type analysis. The biopsy was performed on the same leg both pre- and posttraining, with the posttraining biopsy being more proximal compared with the pretraining biopsy.

MHC Analysis

The MHC isoform profile for each fiber was determined by dissecting individual fibers under a microscope or from cross sections from a cryostat (described below) and then subjecting the fibers to SDS-PAGE, as described previously (21, 25). Briefly, ~200 fibers (~3–4 mm in length) from the pre- and the posttraining muscle bundles were dissected in relaxing solution (pCa2+ 9.0). After dissection, the fibers were solubilized in 80 μl of 1% SDS sample buffer and stored at −80°C until assayed. By using a 3.5% loading and a 5% separating gel for the MHC, at 4°C (SE 600 series, Hoefer, San Francisco, CA), the SDS-PAGE allowed us to evaluate the MHC (I, I/IIa, I/IIa/IIx, IIa, IIa/IIx, IIx) isoform in a single muscle fiber. The gels were silver stained, revealing the MHC isoform profile for the individual fiber, which corresponded to known molecular weights (Sigma Chemical) and a purified human standard from our laboratory containing the three MHC isoforms (I, I/IIa, IIx). Each subject’s MHC fiber-type distribution was determined from the ~200 fibers per biopsy pre- and the ~200 fibers posttraining. From these fibers, the percentage of the total distribution for each MHC-based fiber type was determined. The mean fiber-type percentage data were based upon the proportions of each MHC-based fiber type for each biopsy specimen (procedures previously described; Ref. 25).

The hybrid fibers MHC I/IIa, I/IIa/IIx, or IIa/IIx (per SDS-PAGE/silver stain) were further analyzed by using a computer imaging system (ChemImager 4000, Alpha Innotech, San Leandro, CA) (see Fig. 1). The relative proportions of the hybrid fiber were determined. Dominance of a specific MHC

### Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>YW (n = 6)</th>
<th>YM (n = 6)</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>21.7 ± 1.2</td>
<td>25.3 ± 1.9</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>71.0 ± 3.0</td>
<td>73.7 ± 4.2</td>
</tr>
<tr>
<td>Height, cm</td>
<td>169.4 ± 3.6</td>
<td>179.1 ± 4.4*</td>
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</table>

Values are means ± SE. YW, young women; YM, young men. *Significant difference between YW and YM (P < 0.05).
isoform in a hybrid fiber was established if 66.6% or more of the total MHC was of a specific type (4). For example, if you were to calculate the IIa-dominant MHC I/IIa distribution from the fiber count \((n = 30)\), \((30 \text{ fibers observed}/1,074 \text{ total fiber distribution for YM-post}) \times 100 = 2.8\% \) (the IIa-dominant MHC I/IIa of the total fiber distribution YM-post/5.2\% total YM-post MHC I/IIa fiber distribution) = 54\% (relative distribution of that dominant isoform within the hybrid fiber or 54\% of the MHC I/IIa distribution is dominant IIa). There were few MHC I/IIa/IIx \((n = 37)\) and no MHC I/IIx ("jump"; Ref. 4) fibers reported in this investigation, and therefore they were not analyzed for predominance of MHC isoform.

Five to ten transverse sections \((10 \mu m)\) were also cut at \(-20^\circ\text{C}\) in a cryostat (Tissue-Tek II, Miles Laboratory, Elkhart, IN) for MHC-based fiber-type analysis. The sections were then solubilized in 80 \(\mu l\) of 1% SDS buffer (described above), boiled for 5 min, then loaded 2 \(\mu l\) onto a 5% polyacrylamide gel for SDS-PAGE/silver stain analysis (described above). The gels revealed a pattern of three MHC bands in each lane for each sample (I, IIa, and IIx), which was then analyzed for relative proportion to the total MHC by a computer imaging system (ChemImager 4000, Alpha Innotech).

**Training Program**

All training was performed under supervision at the Human Performance Laboratory. Before the training bout, each subject warmed up on a cycle ergometer (Met 100, Cybex, New York, NY) for \(\approx 5\) min at a low level of intensity \((25-50\ W)\). After the warm-up, each subject was seated on the bilateral isotonic knee extensor device (Cybex Eagle). The training involved a concentric and an eccentric component.

Each subject was required to lift the weight to full knee extension (concentric) and lower the weight to the starting position (eccentric). Each component (concentric and eccentric) was \(\approx 2-3\ s\) in duration. The subjects performed three sets, the first two sets of 10 repetitions and the last set to volitional exhaustion \((2-3\ min\ rest\ between\ sets)\). All PRT exercise sessions were performed at 80\% of one-repetition maximum (1 RM) and reassessed every 2 wk to maintain the intensity. The 1 RM was determined by increasing the weight one-half \((2.88\ kg)\) or one full plate \((5.75\ kg)\) (Cybex Eagle) with each full bilateral knee extension. The test continued until the subject was not able to maintain proper form and/or fully extend the legs at the given weight. The training was conducted three times per week, with a minimum of 48 h between sessions (i.e., no back-to-back sessions), lasting 12 wk in duration. The exercise protocol employed in this investigation is identical to the protocol previously utilized at our laboratory in older women and men (21, 22, 25).

**Statistical Analysis**

Pre- vs. posttraining respective MHC isoform (I, I/IIa, I/IIa/IIx, I/IIa, IIa, IIa/IIx, IIx, total hybrid fiber proportions) and 1-RM results were analyzed using a general linear model, repeated-measures design (Bonferroni post hoc analysis) (SPSS v. 10.0.5), comparing YW and YM, pre- and post-PRT. YW, YM, and OM comparisons were made for change in pure MHC I, IIa, IIx, and total hybrid fiber distributions and 1 RM. Significance was set at the \(P < 0.05\) level. All data are represented as means \(\pm\ SE\), determined from fiber percent data for each subject biopsy specimen.

<table>
<thead>
<tr>
<th>Fiber Types</th>
<th>Total Hybrid Fibers</th>
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<tbody>
<tr>
<td>I</td>
<td>I/IIa</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>37.3 ± 4.0*</td>
</tr>
<tr>
<td>I/IIa</td>
<td>3.5 ± 1.2</td>
</tr>
<tr>
<td>I/IIa/IIx</td>
<td>52.7 ± 4.7†</td>
</tr>
<tr>
<td>Post</td>
<td>35.1 ± 6.5</td>
</tr>
<tr>
<td>I/IIa</td>
<td>0.4 ± 0.2</td>
</tr>
<tr>
<td>I/IIa/IIx</td>
<td></td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>26.9 ± 1.6</td>
</tr>
<tr>
<td>I/IIa</td>
<td>5.7 ± 1.4</td>
</tr>
<tr>
<td>I/IIa/IIx</td>
<td>59.4 ± 3.4†</td>
</tr>
<tr>
<td>Post</td>
<td>29.4 ± 3.5</td>
</tr>
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</table>

Values are means \(\pm\ SE\), expressed as a percentage, for a given myosin heavy chain (MHC)-based fiber type. Hybrid fibers were further analyzed for the predominant MHC isoform within a hybrid fiber (see Methods). PRT, progressive resistance training. Bold type (e.g., I/IIx) denotes dominance (>66.6\%) of the respective isoform in that hybrid fiber, whereas a hybrid fiber with no dominance (<66.6\%) is represented in plain text (e.g., I/IIx). *Significant difference \((P < 0.05)\) between the YW and YM. †Significant difference \((P < 0.05)\) pretraining (Pre) to posttraining (Post).
RESULTS

Single Muscle Fiber MHC

Pretraining. MHC-based single-fiber proportions are presented in Table 2. These data are derived from a sample of 190 ± 5 (1,140 total) and 194 ± 3 single fibers analyzed per subject (1,165 total) for YW and YM, respectively. Before the training program, YW displayed a greater (P < 0.05) distribution of MHC I (Δ = −10) and MHC IIx (Δ = +3) and lower (P < 0.05) total hybrid fibers proportions (MHC I/IIa + I/IIa/IIx + IIa/Ix) compared with YM pretraining (Δ = −10) respectively.

Posttraining. MHC-based fiber-type analysis of 186 ± 4 (1,116 total) and 179 ± 9 single fibers/subject (1,074 total) was performed for YW and YM, respectively. After the PRT protocol (Table 2), both YW and YM decreased their total hybrid proportion (Δ = −19 and −29, respectively; P < 0.05). There was a decrease in the MHC I/IIa/Ix (Δ = −2 and −2; P < 0.05) and MHC I/IIa/Ix (Δ = −13 and −19; P < 0.05) fiber-type percentages for both YW and YM, respectively, and a decrease (Δ = −11; P < 0.05) in the MHC I/IIa for YM only. The MHC I/IIa/Ix fibers were analyzed for relative dominance (see Fig. 1) but did not appear to provide a clear trend in the data, possibly because of the low number of fibers pre- and posttraining (n = 54; 1.2% of the total fiber distribution). Fiber-type proportions of the pure MHC IIa increased (Δ = +24 and +27; P < 0.05), with no alteration in the pure MHC I fibers, for YW and YM, respectively (Table 2). Decreases in the MHC I-dominant MHC I/IIa and nondominant I/IIa/Ix fibers (Table 3; P < 0.05), an increase in the MHC IIa-dominant I/IIa/Ix (P < 0.05), and no change in the MHC IIa-dominant I/IIa/Ix, MHC IIx-dominant I/IIa/Ix and the nondominant I/IIa/Ix MHC contributed to the fiber alterations with the PRT. YW decreased (Δ = −2.5; P < 0.05) the pure MHC IIx proportion, unlike YM, who did not exhibit any pure IIx fibers in the entire study (Table 2). When we sum the percentages for fibers containing the MHC-based IIx fiber type (i.e., I/IIa/Ix + IIa/Ix + IIx), pre- to post-PRT, we observe significant decreases in both YW (Δ = −17.0 ± 7.0) and YM (Δ = −20.7 ± 5.5). This represents 71.4 and 76.1% of the increase in the MHC IIa fiber-type percentage in YW and YM, respectively. However, when the sum of the decrease in MHC hybrid proportions (i.e., I/IIa + I/IIa/Ix + IIa/Ix) is compared with the increase in MHC IIa, this accounts for 80.1 and 108.9% of the change in YW and YM, respectively.

Cross-section Analysis for MHC

Pretraining. Muscle cross-section/SDS-PAGE analysis of MHC proportions revealed no differences between groups (MHC I = 35.6 ± 1.8 and 34.9 ± 2.6, IIa = 41.4 ± 2.3 and 47.5 ± 0.9, IIX = 23.0 ± 2.8 and 17.6 ± 2.9%, YW and YM, respectively). There was also a significant decrease in MHC IIX proportions in both groups (23.0 ± 2.8 to 15.5 ± 2.2 and 17.6 ± 2.9 to 6.0 ± 2.5%; YW and YM, respectively). However, YM did increase (P < 0.05) their MHC I pre- to post-PRT (34.9 ± 2.6 to 39.7 ± 1.4%), whereas YW did not change over time (35.6 ± 1.8 to 35.9 ± 1.5%). When we compared MHC I, IIa, and IIx proportions of pure muscle fiber data to the cross-section data, the correlations (range = 0.01–0.31) were not significant (P > 0.05).

Whole Muscle Strength

YM were stronger than YW pre-PRT (49%; P < 0.05). Both YW and YM increased (P < 0.05), 55 and 34%, respectively, in 1-RM strength after the 12-wk training protocol (Table 4).

DISCUSSION

The present investigation was undertaken to replicate a protocol used in previous aging-resistance train-
ing studies, as well as in our own laboratory (11, 12, 21, 22, 25), and applied to young adults (18–30 yr). This would allow us to determine whether young and old skeletal muscle display similar adaptations to the same resistance training protocol. Furthermore, utilizing single muscle fiber MHC analysis, we have added further insight to fiber-type adaptations with resistance training in young and old individuals.

The primary findings from this investigation were an increase \( P < 0.05 \) in the pure MHC IIa fiber proportion (single muscle fibers) in both YM and YW (in contrast to our hypothesis and previous data using the same protocol), with a concomitant decrease \( P < 0.05 \) in the total MHC hybrid fiber proportions (MHC I/IIa + I/IIa/IIx + I/IIa/Ix), the MHC I/IIa/Ix, the MHC I/IIa distribution (YM only) and the MHC IIa/Ix distribution, after 12 wk of progressive knee extensor training. These data were further supported by an increase \( P < 0.05 \) in the MHC IIa proportion and a reciprocal decrease in MHC IIx after training in both YW and YM, when using cross-section/SDS-PAGE analysis. The analysis of the hybrid fibers for relative MHC dominance by densitometry revealed that the MHC I-dominant MHC I/IIa fiber distributions decreased \( P < 0.05 \), with no change in the nondominant I/IIa and MHC IIa-dominant MHC I/IIa fibers. Likewise contributing to the increase in MHC IIa fiber distribution, the MHC IIa-dominant Ia/IIa increased \( P < 0.05 \) and the nondominant Ia/IIa MHC decreased \( P < 0.05 \), with no alteration in the MHC IIx-dominant MHC Ia/Ix fiber distributions. Contrary to our previous data in OM (21, 25), using the same protocol, YM and YW did not show an increase in the MHC I fiber distribution (see Fig. 2 and Table 2).

Previous studies employing a resistance training stimulus on young subjects (18–30 yr) have reported decreases in MHC IIx (range = 4–12%) with concomitant increases in MHC IIa or MHC Ia/Ix (1, 2, 7, 19, 20) proportions, with no change in the MHC I proportions. Although these data are consistent with the present investigation (increase in the single-fiber and cross-section MHC IIa proportion), the aforementioned studies used ATPase histochemistry, muscle cross sections/SDS-PAGE/silver staining, and/or muscle homogenates/SDS-PAGE/silver staining to assess fiber-type alterations. Furthermore, the alteration in the MHC IIx fiber proportion reported in these studies \( P < 0.05 \) appeared dramatic in nature (supported by cross-section data reported in this investigation); however, this is not the case for the present investigation when using the single-fiber technique. This may be due to a possible misclassification of Ia/Ia/Ix, Ia/Ix, and/or Ix fiber types, resulting in an overestimation of the percentage and/or change in the Ia and/or Ix proportion (3). The significant decline in the fibers containing the MHC IIx isoform (Ia/Ia/Ix + Ia/Ix + Ix) appears to contribute to the increase in the MHC IIa percentage, further supporting previous research (1, 2, 7, 19, 20). The decrement in hybrid fiber proportions appears to contribute more to the increase in MHC Ia with PRT than the fibers containing MHC IIx isoforms. The changes in hybrids are effected by a slight decrease in YW MHC I and IIx (2.2 and 2.5, respectively) and a minimal increase in YM MHC I (2.5) after the PRT, but the decrease in hybrids accounts for most of the change in the MHC Ia fibers. Regardless, there appears to be a small amount of the pure MHC IIx fiber type in biopsy samples from human mixed muscle (vastus lateralis), which has been reported by other investigators (3, 4, 15, 16, 24–26) employing the single-fiber technique.

The decrease in MHC hybrid fibers appeared to contribute to the MHC IIa increase in this investigation (see Table 3). We saw a decline in the MHC I-dominant MHC Ia/Ia and the MHC IIa nondominant MHC Ia/Ix fibers and an increase in the MHC IIa-dominant MHC Ia/Ix fiber distribution. Even though we did not observe single-fiber changes in MHC I and MHC IIx fiber distribution with PRT, a decrease in the total hybrid fiber proportions and the alterations within these hybrid fibers (MHC I/Ia and Ia/Ix; i.e., relative dominance) appear to have contributed to the increase in

\[ \text{Fig. 2. MHC isoform adaptations [pre- to post-progressive resistance training (PRT)] after 12 wk of PRT in older men (OM; } n = 7; \text{ Ref. 25) and younger men (YM; } n = 6) \text{ and women (YW; } n = 6). \]
MHC IIa. Although not all relative contributions to the hybrid fibers were significant, there were trends with an increase in MHC IIa and a decrease in MHC IIx-dominant fibers in the MHC I/IIa and IIa/IIx fibers ($P = 0.070$ and $0.073$), respectively. The reduction in the hybrid fibers with the PRT may limit the ability to accurately assess changes in MHC isoform dominance and may become more evident with a higher sample size.

Previous data from our laboratory (25) demonstrated that PRT significantly reduced hybrid fiber distributions (MHC I/IIa, I/IIa/IIx, Ia/IIx) and significantly increased the pure MHC I fiber distribution in OM, using the same PRT protocol as in the present investigation (Fig. 2). This was further supported by changes in single muscle fiber function of OM increasing in strength, contractile velocity, and power in both the MHC I and IIa fibers, but to a greater magnitude in the MHC I fibers (21). Since then, we have reported that MHC-based fiber-type proportions do not differ between healthy, untrained young (18–30 yr) and old (65–85 yr) men and women (24). This indicates that skeletal muscle MHC-based fiber-type proportions appears to be independent of age in an untrained state (24) but age dependent in a trained state (resistance exercise), as demonstrated collectively in the previous (25) and present investigations (Fig. 2). Moreover, other single muscle fiber MHC-based fiber typing investigations of moderately old and very old adult skeletal muscle (4, 16) support the notion that the pure type I fiber proportion may be lower than previously thought. Figure 2 also shows the difference in adaptation between the young and old to the same 12-wk protocol (80% 1 RM). YW and YM significantly increased the pure MHC IIa and no change in the pure MHC I proportion. Conversely, the OM significantly increased the pure MHC I fiber, with minimal change in the pure MHC IIa proportion. Furthermore, all three groups significantly decreased the total hybrid fiber proportions (MHC I/IIa + I/IIa/IIx + IIa/IIx) and increased 1-RM strength by the same magnitude (YW = 55%, YM = 34%, OM = 52%), after the training. Both the young and the old reported little change in the MHC IIX distributions after the PRT (single-fiber data), further supporting the misclassification of the MHC IIX distribution by ATPase and/or muscle homogenerate techniques in human skeletal muscle.

The muscle biopsy procedure represents a possible confounding variable in this investigation. The post-training biopsy was performed proximal to the pre-training biopsy (same leg) so as to avoid possible scar tissue or damage from the previous biopsy site; biopsies were taken at approximately the same depth each time. It has been previously reported that increasing the number of samples reduces the error in the techniques (10) and also that there is a predominance of type II fibers superficially and type I fibers in deep regions of the whole muscle cross section (vastus lateralis) in younger age groups (17). Although this is a concern, we feel that the variation within our single-fiber measurements is too low to account for more than ~0–2% of the change in proportion. In addition, all subjects in the investigation did display the same trends in single-fiber MHC alterations (i.e., MHC hybrid decrease, MHC IIa increase, and little to no change in the MHC I and IIX), further supporting the findings.

Reasons for the age-related adaptation in MHC-based fiber-type proportion, after 12 wk of PRT, are unknown. Possible explanations for the greater magnitude of change in the young MHC IIa may lie downstream of the nerve (vs. the significant increase in MHC I in the OM); i.e., pretranslational or postranslational events. Alterations in the expression of proteins involved in calcium transport (9, 14), signaling proteins (e.g., calcineurin, calmodulin kinase, ras), transcription factors (e.g., NFAT, MEF2) (8), and rate and/or efficiency of gene transcription (i.e., MHC isoform-specific mRNA) (5, 23) have been reported to differ in aging muscle. Age has also been associated with a decrease in the eukaryotic initiation factor 2 (i.e., translation into posttranslation), subsequently reducing the amount of protein synthesis (13). Any combination of these factors may affect MHC protein transcription and/or synthesis and remains unclear in human muscle.

In conclusion, the present investigation has provided evidence that skeletal muscle of young individuals adapted differently compared with the OM (from our previous investigation) when subjected to an identical training stimulus. More specifically, both the young and the old adults reduced their MHC-based hybrid fiber-type proportions to the same degree with PRT. The young adults increased the pure MHC IIa percentages, whereas the OM increased the pure MHC I fiber (Fig. 2). These data support our understanding of the skeletal muscle fiber-type adaptations to resistance training in young compared with old adults is incomplete, and more emphasis must be placed upon the controlling mechanisms of fiber-type transformations and their effect(s) on the function of skeletal muscle.

We thank Jason Witter and Brian Terpstra for assistance. This investigation was supported by National Institute on Aging Grant AG-154876 (to S. W. Trappe).

REFERENCES


