Functional properties of human muscle fibers after short-term resistance exercise training

JEFFREY J. WIDRICK, JULIAN E. STELZER, TODD C. SHOEPPE, AND DENA P. GARNER
Department of Exercise and Sport Science, Oregon State University, Corvallis, Oregon 97331

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Address for reprint requests and other correspondence: J. J. Widrick, Dept. of Exercise and Sport Science, Oregon State Univ., Corvallis, OR 97331 (E-mail: jeff.widrick@orst.edu).

Skeletal muscle fibers have a remarkable ability to alter their phenotype in response to environmental stimuli or perturbations. An example of this capacity for adaptive change, or plasticity, is the cell hypertrophy that occurs after resistance exercise training. There is a general consensus that resistance training causes hypertrophy of all muscle fiber types, with fast fibers often showing a somewhat greater response than slow fibers (2, 11, 13, 18, 23, 31). There is also a growing body of knowledge detailing resistance exercise-induced changes in contractile protein isoform content, where the most significant alteration appears to be an upregulation of the type IIa myosin heavy chain (MHC) isoform coupled with a downregulation of the type IIx isoform (1, 31, 38).

Despite this progress in characterizing resistance training-induced changes in cell morphology and protein isoform content, relatively little information exists regarding the functional consequences of these adaptations. It is generally assumed that force is proportional to fiber cross-sectional area (CSA) and that the MHC isoform composition of a fiber, or its histochemically determined fiber type, is an accurate index of the fiber’s shortening velocity. However, the specific relationship between fiber CSA and force is dependent on fiber myofibrillar density, a variable that can vary between slow and fast fibers (33). Fiber maximal shortening velocity, while a function of cell MHC isoform content (25), is modulated by other sarcomeric proteins, such as the myosin light chains (3, 15, 32).

Chronic changes in the level of physical activity can alter the functional properties of individual muscle cells. For example, endurance training has little effect on fiber-specific force (force/fiber CSA), but it alters fiber myosin light chain isoform content and increases the unloaded shortening velocity (V₀) of slow fibers by ~20% (27, 37). In contrast, sprint training may decrease the specific force of fast fibers (20) without affecting slow or fast fiber shortening velocity (12).

Knowledge of the relationship between cell hypertrophy, protein content, and contractile function is therefore an important step in understanding the adaptive responses of skeletal muscle to resistance exercise training. However, no clear consensus has emerged as to the effects of resistance training on muscle fiber function. Rometowski et al. (26), studying 60- to 70-yr-old individuals, observed resistance training-induced reductions in the specific force of slow fibers and a decrease in the V₀ of fast fibers. Trappe et al. (34, 35) observed an increase in the V₀ of type I and IIA fibers from elderly males but not elderly females. However, unlike younger subjects, fast fibers obtained from the elderly participants in these studies often showed no evidence of hypertrophy (26, 34). Also, because aging affects both fiber-specific force and V₀ (9, 19), it is not clear whether the observed responses represent an effect of resistance training per se or an interaction between aging and training.

The purpose of the present study was to assess the relationships between fiber hypertrophy, protein isoform content, and contractile function following 12 wk of progressive resistance exercise training. The train-
ing program induced significant hypertrophy of all major groups of slow and fast muscle fibers. Measurements conducted on chemically skinned, maximally Ca\(^{2+}\)-activated muscle fibers indicated that resistance training altered quantitative aspects of fiber function, such as absolute peak force and absolute peak power, but did not affect qualitative or intrinsic mechanisms of contraction, such as specific force, \(V_o\), or peak power/fiber volume.

**METHODS**

Subjects. This study was approved by the Institutional Review Board at Oregon State University. Six men volunteered to serve as subjects after being informed of the nature of the study and after providing their consent in writing. Their mean (+SE) age, height, and body mass at the beginning of the study were 27 ± 2 yr, 175 ± 2 cm, and 82.3 ± 4.2 kg. Health questionnaire responses indicated that all subjects were nonsmokers, were free of any apparent signs or symptoms of neuromuscular disease, and were not taking medications or drugs, including anabolic steroids, that could affect their response to exercise training. None of the subjects had participated in a strength or endurance training program for a minimum of 1 yr preceding the study.

Resistance exercise training program. All subjects completed a resistance exercise training program consisting of 36 exercise sessions performed three times per week on nonconsecutive days. The training program used free-weight and machine-based exercises designed to overload the major lower (squats, knee extension, knee flexion, calf raises), upper (bench press, lat pull down, shoulder press, triceps press, biceps curl, seated row), and abdominal muscle groups.

During each training session, subjects completed three sets of 5–10 of the exercises listed above (divided approximately equally between those targeting the upper and lower body). Subjects performed 12 repetitions per set during the first 2 wk of the training program. Thereafter, one weekly session was performed at 10 repetitions per set, the second session at 8 repetitions per set, and the third weekly session at 6 repetitions per set. During all sessions, the training resistance was adjusted so that subjects were able to complete only the specified number of repetitions, plus or minus one repetition. This nonlinear periodized program was used to maximize training adaptations (17). All exercise sessions were supervised by one of the investigators or by a trained assistant.

Evaluation of voluntary strength and body composition. Subjects reported to the laboratory on two or three separate occasions before the start of the training program. These pretraining visits were used to obtain a pretraining muscle biopsy, to teach subjects proper exercise technique, and to assess body composition using an air displacement densitometry plethysmograph (Life Measurement Instruments, BOD POD, Concord, CA) and the Siri equation (29). Six-repetition maximum voluntary strength was reevaluated every 4 wk throughout the training program. Posttraining body composition was assessed in the week following the last training session.

Muscle biopsy. A pretraining muscle biopsy was obtained from the left vastus lateralis during the subjects’ initial visit to the laboratory. The biopsy was obtained before any other data collection, physiological testing, or training. To minimize the possibility of studying fibers that may have been damaged by the last bout of exercise, the posttraining biopsy was obtained 3–4 days following the final training session, after we had ensured that subjects were not experiencing any delayed muscle soreness. The posttraining sample was obtained from the right leg to eliminate the possibility of studying regenerating fibers at the pretraining biopsy site. All pre- and posttraining muscle samples were obtained from similar anatomic sites located mid-way between the greater trochanter and the patella.

Composition of the solutions for in vitro experiments. The composition of the relaxing and Ca\(^{2+}\)-activating solutions was determined using the computer program described by Fabiato (7) with apparent stability constants adjusted for temperature, pH, and ionic strength (6). Both solutions contained 7.0 mM EGTA, 20.0 mM imidazole, 1 mM free Mg\(^{2+}\), 4 mM MgATP, 14.5 mM creatine phosphate, and 15 U/ml creatine kinase. The free Ca\(^{2+}\) concentration of the relaxing and activating solutions was adjusted to pCa 9.0 and pCa 4.5, respectively (where pCa = −log [Ca\(^{2+}\)], using a 100-mM CaCl\(_2\) standard solution (Calcium Molarity Standard, Coring Incorporated, Corning, NY). In both solutions, pH was adjusted to 7.0 with KOH and total ionic strength to 180 mM with KCl. A dissection solution was made from relaxing solution and a protease inhibitor cocktail that was prepared according to the manufacturer (Complete EGTA-Free Protease Inhibitor, Boehringer Mannheim, Indianapolis, IN). The skinnin solution consisted of equal volumes of dissection solution and glycerol.

In vitro measurement of fiber contractile properties. Pre- and posttraining muscle biopsies were immediately placed in cold (4°C) dissection solution where they were longitudinally divided into small bundles of fibers. The fiber bundles were stored in skinnin solution maintained at 4°C for 24 h and then transferred to fresh skinnin solution and stored at −20°C.

Over the next 3 wk, bundles were transferred to relaxing solution where single muscle fiber segments were isolated using fine forceps. A single-fiber segment was mounted between an isometric force transducer (model 400, Aurora Scientific, Aurora, Ontario) and the arm of a high-speed motor (model 308B, Aurora Scientific) by securing the fiber ends in small stainless steel troughs using 4–0 monofilament posts and 10–0 suture (37). The motor was controlled by a high-speed servomechanism and circuit (model 300-FC1, Positron Development, Inglewood, CA) operating either in length (for slack tests) or force (for isotonic contractions) mode.

Once mounted, the fiber could be rapidly transferred between small wells milled into a stainless steel dip-plate. The plate was mounted to the stage of an inverted microscope where the fiber could be observed during data collection. Sarcomere length was adjusted to 2.5 μm using a calibrated ocular micrometer (×600). Fiber length (FL) was measured with a digital micrometer. Fiber width was read from the ocular micrometer while the fiber was briefly suspended in air (<5 s). Three separate measurements were obtained along the length of the fiber, with the fiber returned to relaxing solution between each measurement. Fiber CSA was calculated by assuming that the mounted fiber forms a cylinder when suspended in air (24, 37). The mean of the three CSA values was taken as the final fiber CSA. The temperature of the relaxing and activating solutions was continually monitored during data collection by a small thermocouple inserted into the solution bathing the fiber. Solution temperatures were maintained at 15°C during the experiments.

Output from the motor and force transducer was monitored during data collection by a small thermocouple inserted into the solution bathing the fiber. Solution temperatures were maintained at 15°C during the experiments.

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Austin, TX). Data reduction, analysis, and storage were performed by programs written in our laboratory (LabView, National Instruments).

Peak Ca\(^{2+}\)-activated force and \(V_c\) were evaluated using a slack test procedure. Fibers were activated, allowed to attain peak force, and then subjected to a rapid reduction in length (90% complete in \(-1\) ms). Force dropped to zero as the fiber shortened under no external load (Fig. 1A). When the fiber shortened to the point where it was no longer slack, there was a rapid redevelopment of tension. The fiber was returned to relaxing solution, reexerted to its original FL, and the entire procedure was repeated at a different slack step length. In this study, the longest slack step imposed on the fiber averaged 15.3 \(\pm\) 0.1% of FL and never exceeded 20% of FL. Peak Ca\(^{2+}\)-activated force was calculated as the difference between maximal force and the force baseline during unloaded shortening. Unloaded shortening velocity was taken as the slope of the relationship between the time required for tension redevelopment and slack step length (Fig. 1B).

Fiber force-velocity-power relationships were determined from a series of isotonic contractions (Fig. 1C). Contractions were performed in sets of three (each contraction 50–100 ms in duration) in which total shortening never exceeded 20% of FL. Shortening velocity was calculated from the motor position record obtained over the last 25–50 ms of each contraction. Fiber force was calculated over similar time points, using the force obtained during a slack step imposed immediately after the last isotonic contraction as a baseline. Data were fit by the Hill equation (14), \((P + a)(V + b) = (P_o + ab)^b\), where \(P_o\) is peak force and the constants \(a\) and \(b\) have dimensions of force and velocity, respectively (Fig. 1D). The parameters describing the relationship, \(V_{max}\) (the velocity-axis intercept), \(a/P_o\) (a unitless parameter describing the curvature of the relationship), and peak Ca\(^{2+}\)-activated force, were used to calculate fiber peak power (39).

Determination of fiber myosin isoform composition. After the physiological measurements, the fiber segment was removed from the transducer and motor, hydrolyzed in 30 \(\mu\)l of an SDS sample buffer (containing 62.5 mM Tris (pH 6.8), 2% SDS, 10% glycerol, 5% \(\beta\)-mercaptoethanol, 0.001% bromophenol blue], denatured for 4 min at 95°C, and stored at \(-80^\circ\)C. MHC isoforms in the fiber solute were loaded on a gel system consisting of a 7% polyacrylamide separating gel and a 3.5% stacking gel (8). Electrophoresis was carried out on Bio-Rad mini-Protean 3 electrophoresis cells running at 70 V for 22–24 h (4°C). Protein bands were visualized using the silver-staining procedure described by Shevchenko et al. (28) modified in that the silver nitrate incubation was carried out at room temperature instead of 4°C. MHC isoforms in the single-fiber segments were identified by comparison with human myosin standards that were run on one or more lanes of each gel. The myosin standards were made by extracting myosin from human vastus lateralis muscle biopsy samples. Figure 2 is an example of a gel illustrating separation of the three MHC isoforms present in adult skeletal muscle and the identification of the MHC isoform composition of single muscle fiber segments.

Statistical analysis. Data are presented as means \(\pm\) SE. Fiber segments were grouped according to their MHC isoform composition for analysis. To compare the morphological and functional properties of fibers differing in their MHC composition, pretrained fibers were analyzed with a twoway ANOVA (MHC isoform composition \(\times\) subject) and subsequent Tukey’s post hoc test. To investigate changes in fiber morphology or function as a result of resistance exercise, pre- and posttraining fibers were analyzed using a two-way ANOVA with main effects of subject and training status. In all analyses, each fiber was treated as a single observation. Pre- and posttraining characteristics of the subjects were compared with a repeated-measures ANOVA. Statistical significance was accepted at \(P < 0.05\).

RESULTS

General adaptations to training. Lean body mass rose 4% over the course of the training program (from 63.7 \(\pm\) 2.8 to 66.4 \(\pm\) 2.3 kg; \(P < 0.05\)), whereas total body mass was unchanged. Lower body neuromuscular strength, as assessed by the six-repetition maximum leg press exercise, rose from a pretraining value of 1,524 \(\pm\) 99 to 1,791 \(\pm\) 69 N at the 4th week (\(P > 0.05\) to 0.05).

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Fig. 1. Examples of methodology. A: superimposed position and force records of 2 slack tests conducted on a fiber segment. In record “a,” the fiber attained peak force and was subjected to a rapid slack length step. The fiber shortened under no load until it had taken up the slack, at which point there was a redevelopment of force. In contraction “b,” the fiber was subjected to a longer slack step resulting in a greater duration of unloaded shortening. Calibration bars represent 200 \(\mu\)m, 0.2 mN, and 100 ms. B: duration of unloaded shortening from 5 slack steps, including contractions “a” and “b” from A, plotted against the corresponding slack step length. The slope of the first-order least-squares regression (\(R^2 = 0.992\)) fit to the data points represents the fibers’ unloaded shortening velocity [0.47 fiber length (FL)/s]. C: series of 3 isotonic contractions terminated by a slack step. Isotonic force and the corresponding shortening velocity were determined over the last half of each contraction. Calibration bars represent 200 \(\mu\)m, 0.2 mN, and 100 ms. D: force and velocity data points from 9 contractions were plotted and fit by the Hill equation. Data points obtained from the records in C are indicated by \(\circ\). In this example, maximal shortening velocity was 1.70 FL/s, peak force was 0.96 mN or 129 kN/m\(^2\), and \(a/P_o\) was 0.050. Note that the fiber in A and B contained type I myosin heavy chain (MHC), whereas the fiber in C and D contained the Ila isoform.
RESISTANCE EXERCISE TRAINING

Table 1. CSA of fibers obtained before and after 12 wk of resistance training

<table>
<thead>
<tr>
<th>MHC Isoform</th>
<th>No. of Fibers</th>
<th>CSA, µm²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>I</td>
<td>86(42%)</td>
<td>69(42%)</td>
</tr>
<tr>
<td>I/Ia</td>
<td>8(4%)</td>
<td>0</td>
</tr>
<tr>
<td>Ia</td>
<td>62(30%)</td>
<td>90(55%)</td>
</tr>
<tr>
<td>Ia/Ix</td>
<td>45(22%)</td>
<td>4(3%)</td>
</tr>
<tr>
<td>Ix</td>
<td>3(2%)</td>
<td>0</td>
</tr>
</tbody>
</table>

Fiber cross-sectional area (CSA) values are means ± SE. No. of fibers indicates the number of fibers isolated from pre- and postraining muscle biopsies with the corresponding percentage of total pre- or postraining fibers in parentheses. *Significant difference (P < 0.05) between pre- and postraining fibers containing similar myosin heavy chain (MHC) isoforms. For the pretraining fibers, any 2 means with different superscript letters are significantly different (P < 0.05).

Table 2. Peak Ca²⁺-activated force of fibers obtained before and after 12 wk of resistance training

<table>
<thead>
<tr>
<th>MHC Isoform</th>
<th>Fiber Force, mN</th>
<th>Fiber Force, kN/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>I</td>
<td>0.60 ± 0.02ᵇ</td>
<td>0.84 ± 0.03ᵃ</td>
</tr>
<tr>
<td>Ia</td>
<td>0.93 ± 0.06ᵇ</td>
<td>1.20 ± 0.03ᵇ</td>
</tr>
<tr>
<td>Ia/Ix</td>
<td>0.95 ± 0.06ᵇ</td>
<td>1.27 ± 0.06ᵇ</td>
</tr>
<tr>
<td>Ix</td>
<td>1.11 ± 0.13ᵇ</td>
<td>1.50 ± 0.18ᵇ,c</td>
</tr>
</tbody>
</table>

Values are means ± SE. Number of fibers per mean same as in Table 1. *Significant difference (P < 0.05) between pre- and postraining fibers containing similar MHC isoforms. For the pretraining fibers, any 2 means with no common superscript letters are significantly different (P < 0.05).
IIa fibers. After training, these ranges were reduced by ~50%, to 115–128 and 131–146 kN/m², respectively.

Unloaded shortening velocity. Before training, fibers containing type IIa MHC shortened fivefold faster than fibers containing type I MHC, whereas fibers containing type IIx fibers shortened 1.6 times faster than the type IIa fibers (Table 3). Fibers containing two MHC isoforms had shortening velocities that were intermediate to those of fibers containing one or the other of the isoforms. Resistance training had no effect on the

Table 3. Unloaded shortening velocity of fibers obtained before and after 12 wk of resistance training

<table>
<thead>
<tr>
<th>MHC Isoform</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0.58 ± 0.02^a</td>
<td>0.57 ± 0.02</td>
</tr>
<tr>
<td>IIa</td>
<td>1.61 ± 0.36^b</td>
<td>2.77 ± 0.11</td>
</tr>
<tr>
<td>IIa/IIx</td>
<td>3.78 ± 0.20^c</td>
<td>3.49 ± 0.60</td>
</tr>
<tr>
<td>IIx</td>
<td>4.72 ± 0.79^d</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE fiber lengths/s. Unloaded shortening velocity was determined by the slack test procedure. Number of fibers per mean same as in Table 1. For the pretraining fibers, any 2 means with different superscript letters are significantly different (P < 0.05).
mean unloaded shortening velocity of fibers containing type I, type IIa, or type IIa/IIX MHC.

**Force-velocity-power relationships.** Figure 5 shows composite force-velocity-power relationships of groups of type I, IIa, and IIa/IIX fibers. Fibers used in the force-velocity-power experiments represented a subset of the fibers subjected to the slack test procedure (see Table 4 for the number of fibers per mean). As can be observed from Fig. 5, the mean peak Ca\(^{2+}\)-activated force of the pre- and posttraining fibers was almost identical to the values obtained during the slack test procedure. In agreement with the slack test results, pretraining \(V_{\text{max}}\) (determined by extrapolation of the

<table>
<thead>
<tr>
<th>MHC Isoform</th>
<th>Peak Power, (\mu\text{N} \cdot \text{FL}^{-1} \cdot \text{s}^{-1}) Pre</th>
<th>Peak Power, (\mu\text{N} \cdot \text{FL}^{-1} \cdot \text{s}^{-1}) Post</th>
<th>Peak Power, (\text{W} / \text{F} \cdot \text{L}^{-1}) Pre</th>
<th>Peak Power, (\text{W} / \text{F} \cdot \text{L}^{-1}) Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>8.3 ± 0.3(^\text{a})</td>
<td>10.8 ± 0.4(^\text{a})</td>
<td>1.53 ± 0.04(^\text{a})</td>
<td>1.55 ± 0.03</td>
</tr>
<tr>
<td>I/IIa</td>
<td>37.7 ± 6.4(^\text{b})</td>
<td>37.7 ± 4.4(^\text{a})</td>
<td>6.10 ± 0.89(^\text{b})</td>
<td>6.10 ± 0.89</td>
</tr>
<tr>
<td>IIa</td>
<td>49.1 ± 1.7(^\text{b})</td>
<td>69.9 ± 2.2(^\text{b})</td>
<td>7.50 ± 0.25(^\text{b})</td>
<td>8.02 ± 0.22</td>
</tr>
<tr>
<td>IIa/IIX</td>
<td>72.6 ± 4.0(^\text{c})</td>
<td>82.4 ± 14.3(^\text{c})</td>
<td>11.37 ± 0.63(^\text{c})</td>
<td>9.83 ± 1.06</td>
</tr>
<tr>
<td>IIx</td>
<td>115.2 ± 19.5(^\text{d})</td>
<td>15.5 ± 2.72(^\text{d})</td>
<td>15.5 ± 2.72(^\text{d})</td>
<td>15.5 ± 2.72</td>
</tr>
</tbody>
</table>

Values are means ± SE. Number of fibers (pre, post): type I (78, 64), type I/IIa (7, 0), type IIa (54, 80), type IIa/IIX (37, 5), type IIx (3, 0). *Significant difference \((P < 0.05)\) between pre- and posttraining fibers containing similar MHC isoforms. For the pretraining fibers, any 2 means with different superscript letters are significantly different \((P < 0.05)\). FL, fiber length.

The parameter \(a/P_o\) was significantly less \((P < 0.05)\) for pretraining type I fibers (0.033 ± 0.001) than for pretraining type I/IIa (0.045 ± 0.008), IIa (0.054 ± 0.003), IIa/IIX (0.084 ± 0.008), or IIx (0.091 ± 0.015) fibers, an indication that faster fibers had force-velocity relationships with progressively less curvature. Resistance exercise altered \(a/P_o\) for the type IIa fibers only, reducing this parameter by 0.007 \((P < 0.05)\). Because resistance training had relatively small effects on \(a/P_o\), there was no pre- to posttraining difference \((P > 0.05)\) in the fractional force or velocity that elicited peak power for the type I (0.149 ± 0.002 vs. 0.143 ± 0.002), IIa (0.181 ± 0.004 vs. 0.172 ± 0.002), or IIa/IIX (0.208 ± 0.007 vs. 0.199 ± 0.008) fibers.

Because of their greater \(P_o\), \(V_{\text{max}}\), and \(a/P_o\), peak power was 4- to 14-fold greater for type II vs. type I fibers (Table 4). Resistance training increased the absolute peak power of the type I and type IIa fibers by 30 and 42%, respectively. Absolute peak power of the type IIa/IIX fibers rose almost 14% after training, but this change was not statistically significant. Resistance training had no effect on peak power per unit fiber volume for any group of fibers.

**DISCUSSION**

The CSA of vastus lateralis muscle fibers containing type I, IIa, or IIa/IIX MHC increased by an average of 30% after 36 resistance exercise training sessions. These data are consistent with the resistance training-induced increases in slow- and fast-fiber CSA reported in the histochemical literature (13, 18, 21, 23). Note that direct comparisons between the present data and histochemical results must take into account the 20% swelling in fiber diameter that occurs during the chemical skinning process (10). Once adjusted by a factor of

**Fig. 5.** Force-velocity and force-power relationships of pre- and posttraining fibers. Dashed lines indicate pretraining relationships; solid lines represent posttraining relationships. A: force-velocity-power relationships for fibers containing type I MHC. B: force-velocity-power relationships for fibers containing type IIa MHC. C: force-velocity-power relationships for fibers containing type IIa/IIX MHC. D: force-power relationships for fibers containing type I, IIa, or IIa/IIX MHC. The relationship of the pretraining type IIa fibers has been included for comparison.
The adaptations to resistance training are complex, involving both neural (5) and peripheral mechanisms (16). It is therefore difficult to state with any degree of confidence to what extent the changes in $Ca^{2+}$-activated muscle fiber function reported here affect neuromuscular performance. A reasonable interpretation of the present data, in regards to its effect on muscular function, is that short-term resistance training alters the potential of muscle fibers to produce torque and power in the direction and magnitude reported here.

On the basis of this argument, it seems likely that the increased neuromuscular power observed after strength training is due, at least in part, to the greater potential of individual muscle fibers to produce power. The contribution of the type II fibers would be particularly important in this regard as they produce sixfold greater power than the type I fibers. As with previous studies (1, 38), we found an overall reduction in type IIx MHC isoform content as indicated by 1) the absence of posttraining fibers containing the type IIx MHC exclusively, 2) a reduction in the relative number of fibers containing both type IIx and type Ila MHC, and 3) a corresponding increase in fibers containing type Ila MHC exclusively. A loss in “pure” type IIx fibers would be expected to reduce overall power, although the magnitude of the reduction is unclear because of the relatively rare occurrence of these fibers. Data in Fig. 5 and Table 4 show that the training-induced shift from hybrid Ila/IIx fibers toward Ila fibers may have a minor impact on muscular power potential as the peak power of these groups of fibers is identical ($P = 0.90$). In this case, fiber hypertrophy was sufficient to compensate for a loss in fiber power that would likely have occurred due to a training-induced shift in MHC content toward a slower isoform.

Finally, we studied the responses of relatively young, previously sedentary subjects to training to maximize the generalizability of our results. Previous studies examining contractile properties of skinned muscle fibers following short-term resistance training reached different conclusions regarding the effects of resistance training on muscle fiber function. For instance, one group found that 12 wk of resistance exercise training had no effect on $V_o$ of fibers from female subjects (34) but elevated the $V_o$ of type I and Ila fibers from male subjects by 75 and 45%, respectively (35). Others reported reductions in slow fiber-specific force with resistance training (26). It is noteworthy that all of these studies were conducted on fibers obtained from subjects averaging 74 yr of age in one case (34, 35) and 60–70 yr in the other (26). Slow and fast skeletal muscle fibers from elderly subjects have lower specific force and substantially reduced unloaded shortening velocities compared with fibers obtained from young-to-middle-aged subjects (9, 19). Because there was no young or middle-aged control group in these previous training studies, it is not clear whether the reported changes in fiber-specific force and $V_o$ represent the effect of resistance training per se, an interaction between aging and resistance training, or a generalized effect of increased physical activity on muscle fiber.
function of the elderly. Along similar lines, hindlimb suspension reduces the specific force of rat type I soleus fibers, but resistance exercise performed during hindlimb suspension is effective in returning this variable to weight-bearing levels (36). Taken together, a reasonable interpretation is that short-term resistance training has no effect on specific force or $V_o$ of slow or fast fibers, unless these functional properties have been altered as a result of other interventions or processes. 

Summary and conclusions. Twelve weeks of progressive resistance exercise training, sufficient to increase neuromuscular strength by $>60\%$, resulted in significant hypertrophy of fibers containing type I, IIA, or IIA/IIX MHC. Peak Ca$^{2+}$-activated force and absolute peak power rose in direct proportion with the increase in fiber CSA, whereas unloaded shortening velocity and power per fiber volume were unaffected by training. Short-term strength training altered the functional properties of slow and fast vastus lateralis muscle fibers obtained from previously sedentary young male subjects in a quantitative manner, i.e., related directly to an increase in the number of cross bridges, without affecting the density of cross bridges or their intrinsic contractile properties.

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