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 histochromically 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. Romatowski 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, 176 ± 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 (squat, knee extension, knee flexion, calf raises), upper (bench press, lat pull down, shoulder press, triceps press, bicep 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, to determine their six-repetition maximum voluntary strength for leg press and bench press exercise, 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, Corning 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 skinning 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 skinning solution maintained at 4°C for 24 h and then transferred to fresh skinning 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 pins 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 on a digital oscilloscope, amplified (model 300-DIF2H, Positron Development), digitized (5 kHz), and interfaced to a personal computer (AT-MIO-15E-1, National Instruments, Inc.).
Austin, TX). Data reduction, analysis, and storage were performed by programs written in our laboratory (LabView, National Instruments).

Peak Ca\(^{2+}\)-activated force and \(V_{\text{max}}\) 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, reextended 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 ± 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 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 \((P + a)(V + b) = (P_o + a)b\), 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, \(V_{\text{max}}\) (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 μl of an SDS sample buffer (containing 62.5 mM Tris (pH 6.8), 2% SDS, 10% glycerol, 5% β-mercaptoethanol, 0.001% bromophenol blue), denatured for 4 min at 95°C, and stored at −80°C. Later, a portion of the fiber solute was 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 ± 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, pretraining fibers were analyzed with a two-way ANOVA (MHC isoform composition × 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 ± 2.8 to 66.4 ± 2.3 kg; \(P < 0.05\)), whereas total body mass was unchanged. Lower body neuromuscular strength, as assessed by the six-repetition maximum for leg press exercise, rose from a pretraining value of 1,524 ± 99 to 1,791 ± 69 N at the 4th week (\(P > 0.05\)).
Fig. 2. Portion of a silver-stained polyacrylamide gel illustrating the identification of MHC isoforms in single muscle fiber segments. Lane 2 was loaded with a human myosin standard. Portions of single-fiber segments were loaded in the remaining lanes (1 segment/lane). Fibers loaded in lanes 1, 3, and 4 contained type IIa, type I, and type IIa/IIx MHC, respectively. Overall, 94% of the pre- and 90% of the posttraining fibers were classified as containing either type I, IIa, or IIa/IIx MHC.

vs. pretraining), 2,241 ± 117 N at the 8th week (P < 0.05 vs. pretraining), and 2,532 ± 115 N at the 12th week (P < 0.05 vs. pretraining) of training. Over the course of the training program, leg press six-repetition maximum strength increased 62% relative to total body mass (from 18.5 ± 0.8 to 30.0 ± 1.5 N/kg body mass; P < 0.05) or 61% relative to lean body mass (from 23.8 ± 0.7 to 38.3 ± 2.1 N/kg lean body mass; P < 0.05).

Myosin isoform composition of pre- and posttraining fibers. Functional properties were determined on 204 pre- and 163 posttraining vastus lateralis muscle fibers. As seen in Table 1, the relative number of fibers containing type I MHC was similar before and after training. However, the relative number of fibers containing type IIa MHC increased from 30% before training to 55% after training, whereas the relative number of single fibers containing type Ila and type IIx MHC fell from 22 to 3%. Posttraining fibers containing type IIx or type I/IIa MHC were relatively rare. Consequently, 94% of the pre- and 100% of the posttraining fibers studied contained either type I, type IIa, or type IIa/IIx MHC.

Fiber CSA and peak Ca2⁺-activated force. Before training, fibers containing type II MHC, either exclusively or in combination with another isoform (i.e., I/IIa, IIa, IIa/IIx, or IIx), were significantly larger in CSA than fibers containing the type I MHC exclusively (Table 1). The mean CSA of the type I, IIa, and IIa/IIx fibers all increased with resistance training. On an absolute basis, hypertrophy of the type IIa (+1,989 μm²) and IIa/IIx (+2,014 μm²) fibers exceeded that of the type I (+1,596 μm²) fibers by 25%. The relative increase in CSA averaged 30% for all three groups of fibers.

Pretraining fibers containing fast MHC isoforms produced significantly greater force than fibers containing type I MHC (Table 2). This was due to the greater CSA of the fast fibers coupled with their significantly greater specific force. Resistance training resulted in significant increases in the absolute peak Ca2⁺-activated force of fibers containing type I (+40%), IIa (+35%), and IIa/IIx (+34%) MHC. On average, training-induced increases in fiber CSA and Ca2⁺-activated force were proportional, because the mean specific force of the type I, IIa, and IIa/IIx fibers was unchanged over the course of the study (Table 2). Figure 3 clearly shows a shift in the frequency distributions of CSA and peak Ca2⁺-activated force of all three groups of fibers toward greater values after training. To examine the relationship between fiber CSA and peak Ca2⁺-activated force, the individual fibers compiled in the histograms were plotted in a scattergram (Fig. 3), and reference lines were drawn that represented the average pooled specific force of the type I (117 kN/m²; Fig. 3A), type IIa (136 kN/m²; Fig. 3B), and type IIa/IIx (146 kN/m²; Fig. 3C) fibers. Pre- and posttraining type I, IIa, and IIa/IIx fibers all clustered about their respective reference lines, showing little deviation from the mean specific force across a wide range of fiber CSA and peak Ca2⁺-activated force.

All subjects experienced hypertrophy of both type I and IIa fibers, and in general, peak Ca2⁺-activated force of type I and IIa fibers rose in direct proportion to the increase in fiber CSA (Fig. 4). However, there was some intersubject variability in this response with fibers from some subjects showing small increases and others small decreases in specific force. Closer examination reveals that these changes were in directions that reduced the variability of the population as a whole. For example, pretraining average specific force for the six subjects ranged from 107 to 134 kN/m² for the type I fibers and from 119 to 153 kN/m² for the type 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 unloaded shortening velocity of fibers obtained before and after 12 wk of resistance training.

**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 ± 0.02a</td>
<td>0.57 ± 0.02</td>
</tr>
<tr>
<td>I/IIa</td>
<td>1.61 ± 0.36b</td>
<td>2.77 ± 0.11</td>
</tr>
<tr>
<td>IIa</td>
<td>2.95 ± 0.17c</td>
<td>3.49 ± 0.60</td>
</tr>
<tr>
<td>IIa/IIx</td>
<td>3.78 ± 0.20d</td>
<td>4.72 ± 0.79d</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 force-velocity relationship) was significantly greater \((P < 0.05)\) in fibers containing type I (1.65 ± 0.25 FL/s), IIa (1.77 ± 0.09 FL/s), IIa/IIx (1.82 ± 0.09 FL/s), and IIx (2.15 ± 0.38 FL/s) MHC than in fibers containing the type I (0.64 ± 0.02 FL/s) MHC isoform. However, \(V_{\text{max}}\) did not differentiate between subgroups of type II fibers or fibers containing multiple MHC isoforms. Resistance training had no significant effect on the \(V_{\text{max}}\) of the type I, IIa, or IIa/IIx fibers.

The parameter \(a/P_o\) was significantly less \((P < 0.05)\) for pretraining type I fibers (0.033 ± 0.001) than for pretraining type 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

### Table 4. Peak power of fibers obtained before and after 12 wk of resistance training

<table>
<thead>
<tr>
<th>MHC Isoform</th>
<th>Peak Power, µN-FL(^{-1})s(^{-1})</th>
<th>Peak Power, W/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>8.3 ± 0.3^a</td>
<td>1.53 ± 0.04^a</td>
</tr>
<tr>
<td>I/IIa</td>
<td>37.7 ± 4.4^b</td>
<td>6.10 ± 0.89^a</td>
</tr>
<tr>
<td>IIa</td>
<td>49.1 ± 1.7^b</td>
<td>7.50 ± 0.25^b</td>
</tr>
<tr>
<td>IIa/IIx</td>
<td>72.6 ± 4.0^c</td>
<td>11.37 ± 0.63^c</td>
</tr>
<tr>
<td>IIx</td>
<td>115.2 ± 19.5^d</td>
<td>15.51 ± 2.72^a</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.
1.44, the absolute CSAs of the slow and fast fibers reported here fall well within the range of values determined by enzyme histochemistry for sedentary and resistance-trained young male subjects (13, 18, 21, 23, 30).

The novel findings of the present study concern the physiological properties of the posttraining, hypertrophied muscle fibers. We found that peak Ca$^{2+}$-activated force rose in direct proportion with muscle fiber hypertrophy because 1) the mean specific forces of pre- and posttraining fibers were similar and 2) the relationships between fiber CSA and peak Ca$^{2+}$-activated force did not appear to be altered by the training protocol. Thus the short-term training program used in this investigation had no effect on the average specific force of fibers containing type I, IIa, or IIA/IIX MHC. Because the single-fiber segments were maximally activated with Ca$^{2+}$, their force is proportional to the number of cross bridges in parallel. Because force increased proportionally with fiber hypertrophy, the most direct conclusion drawn from these results is that cross bridge density was unaltered by the training protocol. Our physiological results therefore support ultrastructural studies showing that the myofilament density (4) and the percentage of cell volume occupied by myofibrils (2, 22) are not altered in skeletal muscle fibers of young men by progressive resistance exercise training.

We did observe small changes in the average specific force of fibers from some subjects. Importantly, these changes were always in a direction that reduced the overall variability of the population at large. Thus, for previously sedentary male subjects, the specific force of both slow and fast fibers appears to be more tightly regulated after a period of resistance exercise training.

The unloaded shortening velocities of the type I, IIa, and IIA/IIX fibers were not altered by resistance training. These results are similar to those of Harridge et al. (12) who reported no change in fiber shortening velocity after 6 wk of training involving very brief (3 s), high-intensity cycle sprint bouts. In contrast, endurance training has been found to increase the $V_o$ of type I fibers by 20%, an adaptation that appears to be related to alterations in fiber myosin light chain isoform content (27, 37). Taken together, fibers containing type I MHC seem to respond differently to high- vs. low-intensity exercise, but whether this is due to the exercise intensity per se, i.e., the force of the muscular contractions, or to other factors related to the specific exercise protocols is not known.

In the absence of resistance training-induced changes in unloaded or maximal shortening velocity, or in the shape of the force-velocity relationships, the peak power of the type I and IIa fibers increased roughly in proportion to changes in their peak Ca$^{2+}$-activated force. Normalization of power to fiber volume, which negates the effects of the training-induced change in fiber CSA, confirms this interpretation. The elevated power of the posttraining fibers was therefore due to their ability to attain greater force. Thus fiber hypertrophy was directly and solely responsible for the increased peak power of the posttraining slow and fast fibers.

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 IIA MHC, and 3) a corresponding increase in fibers containing type IIA 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 IIA/IIX fibers toward IIA 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 IIA 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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