

Functional Adaptability of Muscle Fibers to Long-Term Resistance Exercise

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ABSTRACT

SHOEPE, T. C., J. E. STELZER, D. P. GARNER, and J. J. WIDRICK. Functional Adaptability of Muscle Fibers to Long-Term Resistance Exercise. *Med. Sci. Sports Exerc.*, Vol. 35, No. 6, pp. 944–951, 2003. **Purpose:** We compared the functional properties of muscle fibers from two groups of subjects that differed widely in their training history to investigate whether long-term resistance exercise alters the intrinsic contractile properties of skeletal muscle fibers. **Methods:** Vastus lateralis muscle biopsies were obtained from six sedentary males (NT group, age = 23 ± 1 yr) and six males who had participated in regular resistance exercise training over the preceding 7.6 ± 1.6 yr (RT group, 22 ± 1 yr). Chemically skinned muscle fiber segments were activated with a saturating free $[Ca^{2+}]$ to quantify fiber peak Ca^{2+} -activated force (P_o), unloaded shortening velocity (V_o), and peak power. Fiber segment myosin heavy chain (MHC) isoform content was identified by gel electrophoresis. **Results:** Slow and fast fibers from the RT group were larger in CSA and produced greater absolute P_o and absolute peak power in comparison with fibers from the NT group. However, these differences were no longer evident after P_o and peak power were normalized to fiber CSA and fiber volume, respectively. V_o /fiber length was dependent on fiber MHC content but independent of training status. **Conclusion:** Fiber hypertrophy was sufficient to account for intergroup differences in P_o and peak power of slow and fast fibers. There was no evidence that the intrinsic contractility of slow or fast fibers, as evaluated by force, shortening velocity, and power normalized to the appropriate fiber dimensions, differed between RT and NT groups. **Key Words:** EXERCISE TRAINING, STRENGTH TRAINING, MUSCLE HYPERTROPHY, MYOSIN HEAVY CHAIN ISOFORMS

The functional adaptations that occur with resistance exercise training are typically interpreted within the context of a two-component model. This model consists of a central component that accounts for training-induced changes in motor unit recruitment (6) and a peripheral component that describes changes occurring at the level of the muscle tissue (14).

The most widely studied peripheral component of the training response is muscle hypertrophy. However, correlations between changes in muscle cross-sectional area (CSA) and muscular function are often poor (14), even when efforts are made to control or eliminate neural mechanisms modulating motor unit recruitment (13). Additionally, not all of the functional changes that occur with training are related to gross hypertrophy but appear to involve changes in qualitative or intrinsic contractile properties of the muscle (3,5,13). In addition to the hypertrophy of slow and fast muscle fibers (15,17,21–23,27), short-term resistance training has been reported to induce alterations in muscle fiber

architecture (1), fiber type distribution (28), and myosin heavy chain (MHC) isoform composition (2,34).

It has been proposed that adaptations to the functional properties of individual muscle cells may contribute to the peripheral training response (13). To investigate these adaptations, we recently studied chemically skinned single muscle fiber segments obtained from young adult male subjects before and after a 3-month period of progressive resistance-exercise training (32). Our results indicated that the functional adaptations of both slow and fast skeletal muscle cells to short-term resistance training included greater fiber absolute force and peak power but did not include changes to the intrinsic contractile properties of the cell, such as peak Ca^{2+} -activated force/fiber CSA (specific force), unloaded shortening velocity/fiber length (V_o), or peak power/fiber volume.

Although peripheral adaptations to resistance exercise are apparent after only a few months of training, there is evidence that muscle cells continue to adapt if the period of training is extended. For instance, subjects who have participated in resistance training programs spanning several years (4,15,16,27) typically show greater fiber hypertrophy than previously sedentary individuals who have trained for only 3–6 months (3,17,20,23). Furthermore, although short-term training is thought to affect central and peripheral mechanisms, improvements in performance during long-term training may be due primarily to peripheral adaptations (25). These characteristics of the training response raise the possibility that the 3-month training period used in our previous study may have been too brief in duration to induce

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Submitted for publication September 2002.

Accepted for publication January 2003.

0195-9131/03/3506-0944

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DOI: 10.1249/01.MSS.0000069756.17841.9E

the full array of functional adaptations at the single cell level.

The present study was undertaken to address this possibility. To maximize our chances of detecting resistance training-induced changes in Ca^{2+} -activated muscle fiber function, we studied fibers obtained from two groups of subjects differing widely in their habitual levels of exercise. One group was comprised of sedentary individuals with no recent history of resistance training. The other group was made up of subjects who had participated in progressive resistance-training programs for the preceding 6–10 yr.

METHODS

Subjects. This study was approved by the Institutional Review Board for the Protection of Human Subjects at Oregon State University. Twelve healthy adult male subjects volunteered to serve as subjects after being informed of the nature of the study and after providing their written consent.

Six subjects were recruited who had no history of resistance training. In addition, these individuals had not participated in any type of endurance training during the previous year. Except for occasional recreational activities, they were physically inactive. These subjects were assigned to the nontrained group (NT group).

Six additional subjects were recruited from an on-campus exercise facility. Recruitment was based on information about their current training program, their training history, and an evaluation of their current exercise performance. These individuals (the RT group) had 7.6 ± 1.6 yr of progressive resistance-training experience. Most RT subjects had started training while competing in high-school athletics and had continued training on a recreational basis during college. None of the RT subjects were currently competing in intercollegiate athletics, and none would be considered an elite powerlifter or bodybuilder.

The RT subjects were engaged in periodized progressive resistance-exercise programs that targeted all major upper and lower muscle groups. Their periodized programs required regular fluctuations in the training frequency, volume, and intensity. Over the previous year, training frequency was no less than $4 \text{ d}\cdot\text{wk}^{-1}$ in which training intensity varied from 67 to 100% of 1-repetition maximum. Per training session, 8–12 working sets were performed using larger muscle groups (quadriceps, pectoralis, etc.) and 6–8 working sets using smaller muscle group (biceps, calves, etc.), with each set done to failure. The training frequency of a particular large or small muscle group varied from one to three times per week. The RT subjects participated in no or minimal endurance exercise activities.

None of the NT or RT subjects were taking any type of medication at the time of the study and none reported a history of anabolic steroid use. Half of the RT subjects reported using creatine monohydrate at some point during the previous year.

Muscle biopsies. Percutaneous needle biopsies were taken from the mid left vastus lateralis of all subjects. For

the RT subjects, we wanted to avoid the possibility of studying fibers that may have been damaged by the last bout of resistance training. Therefore, the biopsies for the RT group were obtained approximately 5 d after the last workout involving the quadriceps muscle group. At the time of the biopsy, none of the RT subjects reported any symptoms of muscle soreness or tenderness that might have been indicative of activity-induced muscle damage. Hortobagyi et al. (12) reported that 14 d of detraining had a small effect on the CSA of Type II fibers (6% decline) but no effect on Type I fiber CSA or the voluntary concentric strength of power athletes. Thus, any detraining that may have occurred during a 5-d lay-off period would likely be minor, would have little impact on our overall conclusions, and would clearly be preferable to the possibility of studying damaged fibers.

Body composition. After the biopsy procedure, body density was estimated with an air displacement densitometry plethysmograph (Life Measurement Instruments, Concord, CA) as previously described (32).

Voluntary strength. Maximal voluntary strength of the left knee extensors was assessed on a dynamometer (Chattecx Corporation, KinCom III; Hixson, TN) 3 d after the biopsy. For all assessments, subjects were given sufficient time for warm-up and familiarization with the testing apparatus and the testing procedure. Subjects completed three maximum voluntary isometric contractions at 60° of knee flexion. Each contraction was 5 s in duration with a minimum of 1-min rest separating each trial. Subjects were verbally encouraged to contract the knee extensors maximally during each trial. The peak torque obtained from the three trials was used in analysis. Data were corrected for the effect of gravity on the dynamometer lever arm and the subjects' limb.

In vitro experiments. The computer program described by Fabiato (7) was used to determine the final concentration of metals, ligands, and metal-ligand complexes in the relaxing and activating solutions. The stability constants used in these calculations were adjusted for the experimental conditions of this project (8). The relaxing and activating solutions contained 20.0 mM imidazole, 7.0 mM EGTA, 4 mM Mg^{2+} -ATP, 1 mM free Mg^{2+} , 14.5 mM creatine phosphate, and $15 \text{ U}\cdot\text{mL}^{-1}$ 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 $\text{pCa} = -\log [\text{Ca}^{2+}]$), using a CaCl_2 molarity standard (Calcium Molarity Standard, Corning Inc., Corning, NY). The pH of both solutions was adjusted to 7.0 with KOH and total ionic strength to 180 mM with KCl. A dissection solution was made by dissolving protease inhibitors in an aliquot of relaxing solution (pCa 9.0) according to the directions of the manufacturer (Complete Mini EDTA-Free Protease Inhibitor Tablets, Boehringer Mannheim, Indianapolis, IN). The skinning solution consisted of 50% dissection solution and 50% glycerol.

Immediately after the biopsy procedure, muscle samples were removed from the biopsy needle and placed in cold dissection solution. Samples were dissected longitudinally

into smaller muscle bundles. The muscle bundles were stored in a skinning solution maintained at 4°C. After 24 h, samples were transferred to fresh skinning solution and stored at -20°C for up to 4 wk.

Segments of single fibers (~4–5 mm in length) were isolated from the muscle bundles, and the segment ends were connected to an isometric force transducer (Aurora Scientific, Model 400; Aurora, Ontario) and a direct-current position motor (Aurora Scientific, Model 308B) as previously described in detail (32). The motor was controlled by a servomechanism and circuit (Positron Development, Model 300-FC1; Englewood, CA) operating in either position (slack tests) or force (isotonic contractions) mode. Force and position outputs were monitored on a digital oscilloscope (Integra 10, Nicolet Technologies, Madison, WI). Amplified outputs (Positron Development, Model 300-DIF2H) were digitized (5 kHz) and interfaced to a computer via a data acquisition board (Model AT-MIO-16E, National Instruments, Austin, TX). Display, analysis, and storage of data were performed using custom programs written in our laboratory (LabView, National Instruments).

The experimental apparatus was mounted on an inverted microscope (Olympus Optical Co., Model IX70; Melville, NY) so that the fiber could be viewed (600×) during data collection. Sarcomere length was adjusted to 2.5 μm using a calibrated eyepiece micrometer. Fiber length (FL) was measured using a digital micrometer. Fiber diameter was measured as the fiber was briefly suspended in air. Fiber CSA was calculated from the diameter measurement assum-

ing the fiber forms a cylinder in air (32). The mean of three measurements made along the length of the fiber, with the fiber returned to relaxing solution between each measurement, was taken as the final fiber CSA. Solution temperature was continually monitored by a small thermocouple. The temperature of the solutions was maintained at 15°C during the experiments.

Fiber V_o was determined using a slack test procedure. Fibers were activated and subjected to a rapid slack step that caused force to momentarily dropped to a zero force baseline (Fig. 1A). The fiber shortened under no load until it was no longer slack at which point there was a rapid redevelopment of tension. The fiber was transferred to relaxing solution, re-extended to its original FL, and the test repeated at a different slack step length. In this study, the longest slack step imposed on a fiber never exceeded 20% of FL and averaged $15.1 \pm 0.2\%$ of FL for the NT fibers ($N = 169$) and $15.4 \pm 0.2\%$ of FL for the RT fibers ($N = 170$). The duration of unloaded shortening was plotted against slack length and the data fit using least squares regression (Fig. 1B). The slope of the relationship was taken as V_o . Experiments were eliminated from analysis if the R^2 of the regression was less than 0.98, or if total compliance, defined as the intercept of the regression with the slack distance axis, exceeded 5% of FL. Compliance averaged $2.84 \pm 0.09\%$ of FL for the NT fibers ($N = 169$) and $2.85 \pm 0.10\%$ of FL for the RT fibers ($N = 170$). Peak force was determined as the difference between the peak force attained

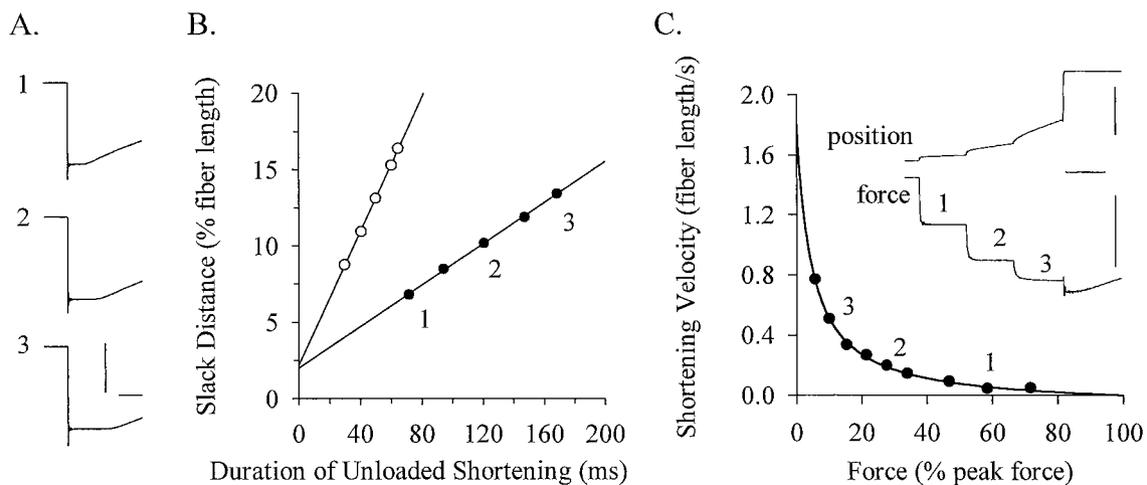


FIGURE 1—Examples of the methodology used to measure contractile properties of Ca^{2+} -activated muscle fiber segments. **A.** Force records of three slack steps performed on a fiber segment. The records consist of the final 100 ms of a fixed-end contraction at peak Ca^{2+} -activated force, the rapid drop in force as the fiber is slacked, a period of unloaded shortening while the fiber takes up the imposed slack and the redevelopment of tension at the point where the fiber is no longer slack. Slack tests 1, 2, and 3 were performed at increasing longer slack steps causing a progressive prolongation in the duration of unloaded shortening. Peak Ca^{2+} -activated force is determined as the difference between the force preceding the slack step and the force baseline. Calibration bars indicate 0.5 mN and 100 ms. **B.** The filled symbols represent the duration of unloaded shortening from the three slack steps in panel A (indicated by the corresponding numbers) plotted against the corresponding slack lengths. Also included are data from two additional slack steps performed on the same fiber. The slope of the relationship is unloaded shortening velocity or V_o . The filled symbols represent a fiber subsequently found to contain Type I MHC ($V_o = 0.67$ fiber lengths $\cdot\text{s}^{-1}$). For comparison, the open symbols represent data obtained from a fiber containing Type IIa MHC ($V_o = 2.18$ fiber lengths $\cdot\text{s}^{-1}$). **C.** The inset shows a force record beginning at peak Ca^{2+} -activated force and followed by three periods of isotonic shortening. The final period of isotonic shortening is terminated with a slack step that is used to establish a force baseline. Aligned above the force record is the corresponding change in motor position or fiber length. Calibration bars represent 200 μm, 50 ms, and 0.5 mN. Force and shortening velocity for the three isotonic steps (indicated by the numbers) have been plotted in the graph along with data from two other series of contractions. The data were fit with the Hill equation (see text). In this example, maximal shortening velocity was 1.81 fiber lengths $\cdot\text{s}^{-1}$, a/P_o was 0.046, and peak force was 0.80 mN. The fiber was found to contain Type IIa MHC.

during Ca^{2+} activation and the force baseline during unloaded shortening.

The force-velocity-power characteristics of the fiber were derived from data collected during a series of isotonic contractions. Three isotonic contractions (50–100 ms in duration) were administered per activation with force and velocity (slope of the position record) determined over the last half of each contraction (inset of Fig. 1C). A slack step was imposed on the fiber immediately after the third contraction in order to obtain a force baseline. Total shortening across all three isotonic contractions never exceeded 20% of FL. Data were plotted (Fig. 1C) and fit by the Hill equation (11). Fibers were eliminated from analysis if $R^2 < 0.97$. The three parameters describing the relationship, V_{\max} (the velocity axis intercept), P_o , and a/P_o (a unitless parameter describing the shape of the relationship) were used to calculate fiber power (35). For graphical comparisons, force-velocity-power relationships were constructed using the average parameters describing each group of fibers.

Fiber MHC analysis. After the functional experiments, the fiber segment was removed from the transducer and motor and placed in 30 μL of a sodium dodecyl sulfate (SDS) sample buffer. The fiber was denatured for 4 min at 95°C and stored at -80°C . Details of the gel system and the silver staining procedure are presented in Widrick et al. (32). Briefly, a 7% separating gel and a 3.5% stacking gel were used to separate the MHC isoforms present in each fiber segment. Gels were silver stained and each fiber segment characterized as expressing Type I, IIa, IIx, or multiple MHC isoforms by comparing protein band migration to a human myosin extract run in one or more lanes of the gel. The extract was prepared from human vastus lateralis muscle samples according to the procedure of Laframboise et al. (19). An example of a silver stained 7% gel and MHC isoform identification is shown in Figure 2.

Statistical analysis. An ANOVA, with main effects of training status and subjects nested within training status, was used to evaluate inter-group differences in fiber contractile properties. Separate analyses were conducted on each fiber type. ANOVA was used to compare the descriptive characteristics and voluntary isometric strength of the NT and RT groups. Statistical significance was accepted at $P < 0.05$. All statistics were performed using SAS version 8.0 (SAS Institute, Inc., Cary, NC). Data are presented as mean \pm SE.

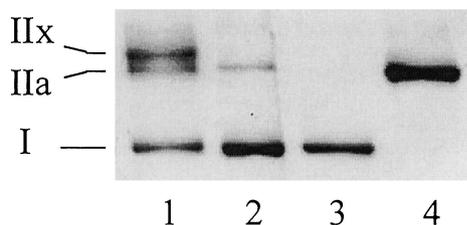


FIGURE 2—Representative silver-stained polyacrylamide gel illustrating fiber MHC isoform identification. A myosin extract, prepared from human vastus lateralis muscle, was loaded in lane 1 to show the migration of the MHC isoforms present in adult skeletal muscle. Lanes 2–4 each contain a single fiber segment run after functional analysis. Note the fiber in lane 2 expresses two MHC isoforms.

RESULTS

Subject characteristics. The RT and NT subjects were similar in age (22 ± 1 vs 23 ± 1 yr, respectively, $P > 0.05$) and stature (180 ± 4 vs 179 ± 2 cm, respectively, $P > 0.05$). The RT subjects had a greater body mass (95.4 ± 2.9 vs 80.9 ± 5.4 kg, $P < 0.05$) and lean body mass (83.7 ± 1.6 vs 63.2 ± 3.2 kg, $P < 0.05$) compared with the NT group.

Voluntary strength. Maximal voluntary knee extension isometric torque was significantly greater for the RT group versus the NT group (360 ± 19 vs 266 ± 20 N·m, $P < 0.05$). However, maximal voluntary isometric torque per kilogram lean body mass was similar for RT and NT subjects (4.30 ± 0.22 vs 4.19 ± 0.18 N·m·kg $^{-1}$ lean body mass, $P > 0.05$).

Skinned fiber analysis. Peak Ca^{2+} -activated force was measured in a total of 220 NT and 213 RT fibers with the majority of these fibers (~90%) containing either Type I MHC, IIa MHC, or both the Type IIa and IIx isoforms. On an absolute basis, peak Ca^{2+} -activated force was significantly greater for the Type I (+54%), IIa (+46%), and IIa/IIx (+37%) fibers obtained from the RT group (Table 1). These differences in force could be directly attributed to the greater average CSA of the Type I (+53%), IIa (+48%), and IIa/IIx (+37%) fibers from the RT subjects since fiber specific force did not differ between NT and RT groups (Table 1).

The remaining fibers contained either Type I/IIa MHC (11 from the NT group; 3 from the RT group) or Type IIx MHC (8 from the NT group; 1 from the RT group). These sample sizes were too small for statistical analysis, but in both cases, fibers from the RT group appeared larger in CSA (Type I/IIa: $6,344 \pm 348$ vs $10,934 \pm 1483$ μm^2 ; Type IIx: $5,621 \pm 673$ vs $11,468$ μm^2) but produced approximately the same specific force (Type I/IIa: 139 ± 8 vs 128 ± 15 kN·m $^{-2}$; Type IIx: 148 ± 7 vs 144 kN·m $^{-2}$) as corresponding fibers from the NT group.

The average V_o of fibers containing Type I, IIa, or IIa/IIx MHC are presented in Table 2. The V_o of these fibers was highly dependent on MHC isoform expression. When fibers were grouped according to their MHC isoform content, no differences in V_o were observed between fibers from the NT and RT subjects.

Similar to the slack test results, fiber V_{\max} was not different between NT and RT groups for Type I (0.68 ± 0.03 vs 0.70 ± 0.03 FL·s $^{-1}$), IIa (1.85 ± 0.09 vs 1.79 ± 0.08 FL·s $^{-1}$), or IIa/IIx (1.94 ± 0.11 vs 2.28 ± 0.14 FL·s $^{-1}$) fibers (Fig. 3, see Table 3 for number of fibers per mean). The shape or curvature of the force-velocity relationships were also similar between NT and RT groups because no intergroup differences in the parameter a/P_o were noted for fibers expressing Type I (0.030 ± 0.001 vs 0.026 ± 0.001), IIa (0.056 ± 0.003 vs 0.053 ± 0.002), or IIa/IIx (0.081 ± 0.010 vs 0.061 ± 0.004) MHC. However, Type I, IIa, and IIa/IIx fibers obtained from the RT group produced 40%, 35%, and 38% greater absolute power, respectively, than corresponding fibers from the NT group (Fig. 3; Table 3).

TABLE 1. Fiber cross-sectional area and peak Ca²⁺-activated force.

	Fiber MHC Isoform Expression		
	I	Ila	Ila/Iix
CSA (μm^2)			
NT	4927 \pm 141 (97)	6790 \pm 193 (67)	6469 \pm 173 (37)
RT	7552 \pm 221 (77)*	10,074 \pm 208 (110)*	8873 \pm 490 (22)*
Peak force (mN)			
NT	0.56 \pm 0.02	0.93 \pm 0.03	0.95 \pm 0.03
RT	0.86 \pm 0.02*	1.36 \pm 0.03*	1.30 \pm 0.07*
Peak force ($\text{kN}\cdot\text{m}^{-2}$)			
NT	115 \pm 2	139 \pm 3	147 \pm 3
RT	115 \pm 2	136 \pm 2	148 \pm 4

Values are mean \pm SE with number of fibers in parentheses. * Significant difference between NT and RT means ($P < 0.05$).

MHC, myosin heavy chain; CSA, cross-sectional area; NT, nontrained group; RT, resistance-trained group.

These differences between the NT and RT groups were no longer evident once peak power was normalized to fiber volume (Table 3).

DISCUSSION

As expected, slow and fast vastus lateralis muscle fibers obtained from the RT subjects were larger in CSA than corresponding fibers obtained from the NT subjects. The novel findings of this study concern the Ca²⁺-activated contractile properties of these hypertrophied fibers. These results can be summarized as follows: 1) slow and fast muscle fibers obtained from the RT group produced 37–54% greater peak Ca²⁺-activated force and 35–40% greater peak power than corresponding fibers obtained from the NT subjects; 2) these differences were no longer evident when force and power were normalized to fiber CSA and fiber volume, respectively; and 3) the relationship between fiber MHC isoform content and shortening velocity did not differ between NT and RT groups.

In their review of the literature, Jones et al. (14) suggested that peripheral adaptations to resistance exercise training might include changes to the contractile properties of individual muscle cells. We examined this possibility in a previous 3-month longitudinal training study (32). The results of that study indicated that several aspects of cross-bridge mechanisms of contraction, such as absolute force or power, increased in direct proportion to fiber hypertrophy. In contrast, the intrinsic contractile properties of the fibers, such as force/fiber CSA, shortening velocity/fiber length, and power/fiber volume, did not respond to the short-term resistance-training program.

The rationale for undertaking the present study was to examine whether more prolonged resistance training would induce additional adaptations in fiber contractility beyond

TABLE 2. Fiber unloaded shortening velocity.

	Fiber MHC Isoform Expression		
	I	Ila	Ila/Iix
NT	0.58 \pm 0.01 (86)	2.88 \pm 0.14 (51)	3.59 \pm 0.22 (32)
RT	0.57 \pm 0.02 (68)	2.99 \pm 0.10 (81)	4.27 \pm 0.26 (21)

Values are mean \pm SE fiber lengths \cdot s⁻¹ with number of fibers in parentheses. There was no statistical difference for NT vs RT comparisons within each MHC isoform group ($P > 0.05$).

MHC, myosin heavy chain; NT, nontrained group; RT, resistance-trained group.

those observed during our short-term longitudinal training study (32). The similarities between the present results and our previous work suggests that the responses noted after 3 months of training hold even if the duration of training is extended to several years. Thus, the major conclusion of the present work is that despite years of training, a generalized resistance exercise program is effective in increasing absolute peak Ca²⁺-activated force and power of skeletal muscle cells but does not alter the intrinsic ability of fibers of similar MHC isoform content to produce force or power or to shorten.

There are few data available concerning the functional properties of human skeletal muscle fibers after long-term resistance-exercise training. However, it is possible to compare our fiber CSA data to the histochemical literature provided the present values are adjusted for the 20% increase in fiber diameter (44% increase in fiber CSA) that occurs during the chemical skinning process (10). After making this adjustment, the CSA of the slow and fast fibers obtained from our NT group are in good agreement with histochemical studies that have examined sedentary populations (3,15,22,23,28). Although there is variation from study to study, the slow and fast fibers from the RT group are generally larger in CSA than fibers obtained from subjects who have trained for 3–6 months (17,22,23). Finally, fast fibers from the present RT subjects are considerably smaller in CSA than fast fibers obtained from elite or competitive powerlifters or bodybuilders (15,16,21,27), whereas the slow fibers obtained from the RT subjects appear similar (15,21,27) or smaller (16) in CSA than fibers from elite competitors.

In terms of hypertrophy, fibers from the present RT subjects appear to fall somewhere between fibers obtained from individuals participating in short-term training programs and fibers from elite resistance-trained athletes. This seems reasonable as we selected habitual, but nonelite, trainers. It is unknown how fibers may adapt to more specialized training programs that are designed to induce very specific functional or morphological adaptations. Therefore, the generalizability of the present results and conclusions to other types of high-intensity resistance training is unknown.

An advantage of the present design is that we were able to study subjects after much longer periods of training than would be practical using a longitudinal design. However, a limitation of this approach is that we had no control over extraneous factors that may have impacted the training response. One of these factors, which is a concern in longitudinal as well as cross-sectional studies, is the influence of genetics on muscle plasticity. In untrained subjects, genetic factors have been reported to contribute greatly to intra-individual differences in muscular strength and muscle CSA (30). If the RT subjects self-selected to participate in prolonged resistance training based on some characteristic(s) of their pretraining genotype, and if genotype interacts with resistance training, the responses of the present RT group may not be generalizable to other populations. In a 10-wk resistance training study, Thomis et al. (30) concluded that genotype influenced the training adaptations in

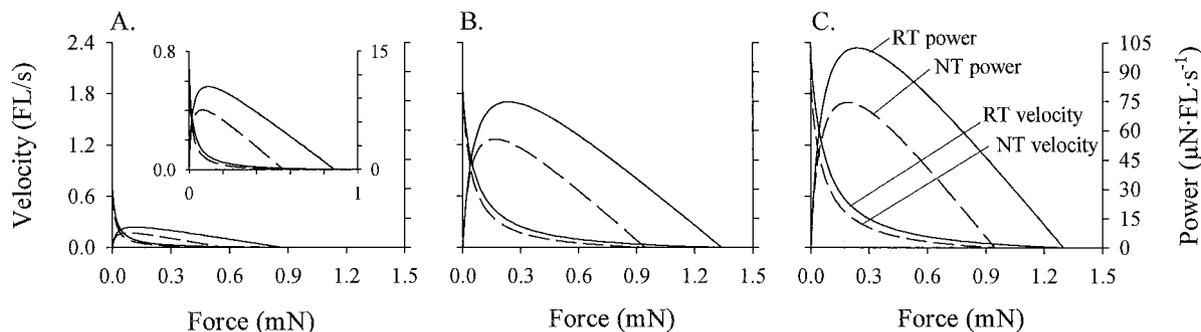


FIGURE 3—Force-velocity-power relationships for fibers obtained from the nontrained (NT) and resistance-trained (RT) groups. Relationships for the NT group are indicated by the *dashed lines*. Relationships for the RT group are indicated by the *solid lines*. The relationships were constructed using the average parameters describing the force-velocity relationships of the Type I, IIa, and IIa/IIx fibers from the two groups of subjects (see Methods). To facilitate visual comparisons, the x-axis and both y-axes (velocity and power) are scaled identically across all three main panels. **A.** Relationships for fibers containing Type I MHC. The inset is an enlarged view of the relationships illustrated in the main panel. **B.** Relationships for fibers containing Type IIa MHC. **C.** Relationships for fibers containing Type IIa/IIx MHC. See Table 3 for the number of fibers per relationship.

1-repetition maximum strength and voluntary isometric strength, but not muscle CSA or most measures of dynamic strength (concentric and eccentric contractions). These authors also concluded that genotype accounted for approximately 20% of the intrasubject variation in 1-repetition maximum strength and voluntary isometric strength, which is substantially less than the genetic contribution to endurance-training adaptations. We are unaware of any similar studies examining prolonged resistance exercise training, and it is difficult to extrapolate the findings of short-term training studies to the long-term training conducted by the present subjects. For these reasons, care must be exercised when generalizing the present results to other subject populations.

A second factor that should be considered when interpreting the present data is the nutritional status of the subjects. In this regard, half of the RT subjects reported using creatine monohydrate supplements at some point during the year leading up to the biopsy. Creatine supplementation may result in muscle enlargement as a result of water retention (29). However, any osmotically induced increase in muscle size secondary to creatine use would have no bearing on the present results since the chemical skinning processes removes the normal osmotic barrier between the extra- and intra-cellular compartments and the skinned fiber segments are then studied under consistent conditions of ionic strength and phosphocreatine concentration.

TABLE 3. Fiber peak power.

	Fiber MHC Isoform Expression		
	I	IIa	IIa/IIx
$\mu\text{N}\cdot\text{FL}\cdot\text{s}^{-1}$			
NT	7.5 \pm 0.3 (81)	55.3 \pm 2.2 (56)	74.3 \pm 4.1 (24)
RT	10.5 \pm 0.4 (65)*	74.5 \pm 2.9 (83)*	102.3 \pm 7.4 (20)*
$\text{W}\cdot\text{L}^{-1}$			
NT	1.53 \pm 0.03	8.34 \pm 0.31	11.95 \pm 0.72
RT	1.41 \pm 0.04	7.58 \pm 0.27	11.97 \pm 0.96

Values are mean \pm SE with number of fibers in parentheses. * Significant difference between NT and RT means ($P < 0.05$). Fiber power has been expressed in absolute terms ($\mu\text{N}\cdot\text{FL}\cdot\text{s}^{-1}$) and normalized to fiber volume ($\text{W}\cdot\text{L}^{-1}$ of fiber).

MHC, myosin heavy chain; NT, nontrained group; RT, resistance-trained group; FL, fiber length.

Although creatine supplementation has been associated with improved performance during repeated bouts of short-duration, high-intensity exercise, there is little evidence that creatine supplementation *per se* improves maximal isometric strength (29). Enhanced muscle fiber hypertrophy has been reported when creatine supplementation is coupled with several weeks of resistance training; however, this is thought to be related to the ability of supplemented subjects to attain a greater training intensity or volume (31). Although this serves to accelerate peripheral adaptations over the short term, any potential benefit derived from accelerating fiber hypertrophy may be minimal in the present study as the RT subjects had accumulated a high volume of exercise spread over many years. To examine this possibility, the RT subjects were broken down into subgroups based on their prior creatine use. ANOVA (main effects of creatine use and subjects nested within creatine use) indicated no nonuser vs creatine user differences ($P > 0.05$) in the CSA, peak Ca^{2+} -activated force (mN), or specific force for fibers containing Type I, IIa, or IIa/IIx MHC. Thus, we could detect no difference in fibers obtained from subjects who reported using creatine at some point during the previous 12 months versus those who reported no creatine use during this same time period.

We are unaware of any previous studies that have examined the functional properties of muscle fibers from individuals participating in prolonged resistance-training programs. However, comparable data exist for middle-aged men who regularly participated in endurance training (running) over a 20- to 25-yr period. Type I and IIa gastrocnemius fibers from these master runners had specific forces that were similar to the values observed for fibers obtained from their sedentary peers (33). This is similar to the present results and suggests that the specific force of Type I and IIa fibers is insensitive to chronic endurance- or resistance-exercise training. The upper limit to fiber specific force is likely determined by the volume of the cell that can be devoted to myofibrils (24). Our short-term (32) and long-term (present study) resistance training data suggest that myofibrillar volume increases in direct proportion with

exercise-induced fiber hypertrophy, a conclusion that is consistent with morphological studies (3,22). Note that these conclusions regarding muscle fiber specific force and physical activity may only apply to muscle fibers from young, healthy, ambulatory individuals.

In contrast to fiber specific force, fiber shortening velocity appears to adapt differently to long-term endurance exercise versus long-term resistance exercise. Although there was no evidence in the present study that prolonged resistance training had any effect on the relationships between fiber MHC isoform content and fiber V_o , Type I gastrocnemius fibers obtained from master runners showed a 19% faster V_o /fiber length than Type I fibers obtained from their sedentary peers (33). It has been proposed that the increase in Type I fiber V_o after prolonged endurance training is related to training-induced shift in myosin light chain isoform content (26,33). This subtle change in fiber V_o may serve to fine-tune the functional properties of the fiber to optimize power and efficiency, an adaptation that may be beneficial for activities in which the muscle contracts repeatedly and where performance may be limited by substrate supply (e.g., endurance running). The absence of this functional adaptation with resistance training may have little effect on the performance of activities requiring a relatively limited number of muscle contractions.

Finally, it may seem surprising that the relative intergroup differences in fiber peak Ca^{2+} -activated force exceeded the relative difference in voluntary strength between the two groups. However, for the reasons detailed in the introduction, the interpretation of voluntary strength is complex, involving not only the variables examined in the present study but also fiber type composition, fiber architecture, and the recruitment patterns of both agonistic and antagonistic muscle groups. In addition, the skinned fibers studied in the present study are a simplified model of muscle cells *in vivo*. Our preparation bypasses mechanisms of excitation-contraction coupling, yet a number of functional and morpho-

logical changes that occur with resistance training have been interpreted as indicative of changes in these processes (3–5). The relaxing and activating solutions used in this study were designed to replicate the concentrations of important ions and substrates present in mammalian muscle cells (7). As such, they provide a highly standardized and rigorously controlled method of studying cell function and conducting comparisons between fibers from the NT and RT groups. However, a limitation of this approach is that it does not account for training-induced changes in the intracellular milieu that may occur *in vivo*. Nor do the present solutions contain the levels of inorganic phosphate that are present in mammalian cells at rest (18) and that are sufficient to substantially depress Ca^{2+} -activated fiber force (9). For all of these reasons, it is difficult to make direct quantitative comparisons between the measurements of voluntary strength and the fiber P_o data of this study.

To summarize, we compared the contractile properties of Ca^{2+} -activated skinned fibers obtained from individuals who had participated in resistance exercise training for the previous 6–10 yr with fibers obtained from their sedentary peers. Intergroup differences in absolute measurements of fiber peak Ca^{2+} -activated force and peak power were attributed to differences in fiber CSA. In contrast, normalized or intrinsic contractile properties were similar between groups. These results demonstrate that changes in peak Ca^{2+} -activated force/fiber CSA, shortening velocity/fiber length, and peak power/fiber volume are not obligatory in slow or fast muscle fibers that have undergone substantial hypertrophy as a result of several years of resistance training.

The authors extend their gratitude to the subjects participating in this project and to Jeffrey Mull, M.D. for his assistance.

This work was supported by National Institute of Arthritis and Musculoskeletal and Skin Diseases grant AR46392.

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