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*J Appl Physiol* 100:771-779, 2006. First published Dec 1, 2005; doi:10.1152/jappphysiol.01027.2005

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## Stretch-shortening cycle exercises: an effective training paradigm to enhance power output of human single muscle fibers

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Submitted 23 August 2005; accepted in final form 24 November 2005

**Malisoux, Laurent, Marc Francaux, Henri Nielens, and Daniel Theisen.** Stretch-shortening cycle exercises: an effective training paradigm to enhance power output of human single muscle fibers. *J Appl Physiol* 100: 771–779, 2006. First published December 1, 2005; doi:10.1152/jappphysiol.01027.2005.—Functional performance of lower limb muscles and contractile properties of chemically skinned single muscle fibers were evaluated before and after 8 wk of maximal effort stretch-shortening cycle (SSC) exercise training. Muscle biopsies were obtained from the vastus lateralis of eight men before and after the training period. Fibers were evaluated regarding their mechanical properties and subsequently classified according to their myosin heavy chain content (SDS-PAGE). After training, maximal leg extensor muscle force and vertical jump performance were improved 12% ( $P < 0.01$ ) and 13% ( $P < 0.001$ ), respectively. Single-fiber cross-sectional area increased 23% in type I ( $P < 0.01$ ), 22% in type IIa ( $P < 0.001$ ), and 30% in type IIa/IIx fibers ( $P < 0.001$ ). Peak force increased 19% in type I ( $P < 0.01$ ), 15% in type IIa ( $P < 0.001$ ), and 16% in type IIa/IIx fibers ( $P < 0.001$ ). When peak force was normalized with cross-sectional area, no changes were found for any fiber type. Maximal shortening velocity was increased 18, 29, and 22% in type I, IIa, and hybrid IIa/IIx fibers, respectively ( $P < 0.001$ ). Peak power was enhanced in all fiber types, and normalized peak power improved 9% in type IIa fibers ( $P < 0.05$ ). Fiber tension on passive stretch increased in IIa/IIx fibers only ( $P < 0.05$ ). In conclusion, short-term SSC exercise training enhanced single-fiber contraction performance via force and contraction velocity in type I, IIa, and IIa/IIx fibers. These results suggest that SSC exercises are an effective training approach to improve fiber force, contraction velocity, and therefore power.

skinned fibers; fiber hypertrophy; plyometry; passive stretch

SKELETAL MUSCLE IS A HIGHLY plastic tissue and very responsive to changing functional demands. Adaptations in whole muscle performance can be partly achieved by adjustments in the firing rate of motoneurons (7), the pattern of motor unit recruitment (29), muscle size (11), and muscle fiber-type expression (5, 13). Furthermore, some studies have demonstrated changes in diameter (1) and contractile properties (31) of single muscle fibers containing a specific myosin heavy chain (MHC) isoform (type I, IIa, or IIx). The latter observation suggests that different MHC isoforms present in skeletal muscle fibers do not account for all their functional heterogeneity (4). Independent of the mechanisms responsible for these adaptations, a training effect can be induced in muscle fibers expressing a single MHC isoform, suggesting alterations in the cross-bridge mechanics that result in associated changes of whole muscle function. Therefore, the assessment of the contractile charac-

teristics of single muscle fibers represents a powerful means to test the effects of different exercise paradigms and to optimize exercise-based interventions.

Recent studies have focused on the effects of different training programs on the contractile performance of single muscle fibers (22, 31, 32). Although a particular training regime may affect different fiber types in different ways and even lead to a shift in fiber-type expression, from a purely mechanical point of view it has generally been found that the induced adaptations are highly specific to the type of training. Endurance-trained master runners (43 yr of age) displayed higher fiber maximal shortening velocities ( $V_0$ ) than their sedentary peers, but only in type I fibers (32). Runners also displayed lower absolute peak  $\text{Ca}^{2+}$ -activated force ( $P_0$ ). Resistance training improves muscle function as a result of neuromotor control changes, especially in the early phase of intervention as well as muscle hypertrophy (16). The latter aspect has been demonstrated by the morphological and functional improvements in single fibers, with effects mainly on fiber force and cross-sectional area (CSA), especially in type IIa fibers (31). However, no changes were found in  $V_0$  for any fiber type (22, 31).

An important issue of exercise training interventions is to maximize the physiological effects while, at the same time, minimizing the exercise period. Based on the above-outlined findings, it can be speculated that the ideal intervention to enhance muscle function would need to encompass repetitive muscle contractions performed against relatively high loads and involving high contraction velocities. Especially the eccentric component seems to be critical in stimulating muscle growth (13) and performance adaptations (9). These contraction characteristics are inherent to stretch-shortening cycle (SSC) movements that involve a high-intensity eccentric component followed immediately by a rapid and powerful concentric contraction. Training based on maximal effort SSC exercises, also termed plyometric training, leads to performance enhancements, especially in activities requiring explosive muscle contractions. Typically, jumping performance is improved through a greater vertical velocity at takeoff (17) resulting from a higher contraction velocity and/or muscle force. The benefits from this type of training have been explained mainly on the basis of neuromuscular adaptations (6, 12, 29). SSC exercise training has been considered inappropriate to induce structural changes in muscle (12, 18), but to date no research has focused on the potential modifications induced in the contractile apparatus. Therefore, we tested the hypothesis that short-term SSC

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exercise training induces functional improvements in single human muscle fibers and may therefore represent an attractive exercise paradigm to improve both force and shortening velocity. More specifically, we hypothesized that 8 wk of SSC exercise training would increase fiber CSA,  $P_0$ ,  $V_0$ , and power in slow and fast muscle fibers.

The SSC is a natural form of muscle function occurring during activities such as walking, running, or hopping. Its particular characteristic is that muscle force and power generation are enhanced during the final (concentric) phase compared with a mere concentric contraction (15). This enhancement has been commonly attributed to the elastic energy stored in the tensile and contractile elements during the eccentric phase and released during the concentric contraction. Therefore, our secondary hypothesis was that passive tension developed by single fibers is modified by SSC exercise training.

## MATERIALS AND METHODS

### Subjects

Eight healthy men volunteered to participate in this study. Their mean ( $\pm$ SE) age, height, and body mass at the beginning of the study were  $23 \pm 1$  yr,  $177 \pm 2$  cm, and  $68 \pm 4$  kg, respectively. The participants had never followed a SSC exercise training program before this study and were at the time not implicated in sport activities involving high-impact jumping. They were all recreationally active, practicing activities such as soccer, hockey, swimming, cycling, judo, and gymnastics for  $3.1 \pm 0.4$  h/wk. They did not change their usual activity pattern during the study period. They were informed of any risks associated with participation in the study and provided their consent in writing. The protocol of this study was approved by the Faculty Ethical Review Committee and complied with the principles of the Declaration of Helsinki.

### SSC Exercise Training Program

All subjects completed the whole SSC exercise training program consisting of 24 sessions performed three times per week for a total of 5,228 jumps. All exercises were performed without overweight and included different types of jumps, presented here in order of increasing difficulty: static jump (vertical jump starting with knees flexed at  $90^\circ$  without prior countermovement), vertical countermovement jump, drop jump (height of 40 cm), double-leg triple jump, single-leg triple jump (starting alternatively with left and right leg, final landing on two legs), double-leg hurdle jump (1 repetition = 5 hurdles), and single-leg hurdle jump (similar leg, 1 repetition = 5 hurdles). The one-leg exercises were performed alternatively on each side to provide a similar workout for both legs. The subjects were instructed to perform all jumps at a maximal effort and to amplify the knee flexion during the landing phase so as to maximize the eccentric component imposed to the knee extensors. The number of jumps was progressively increased during the first 4 wk to minimize the risk of injury. The heavier exercises were introduced progressively so that the subjects were able to adapt to the specificity of the training. The sessions lasted for 20 min in the beginning to  $\sim 45$  min at the end of the training period. All training sessions were supervised by one of the investigators or by an experienced coworker.

### Evaluation of Leg Strength and Power

Functional tests were conducted before and after the SSC training program to evaluate its effectiveness. One-repetition maximal force of leg extensors (two-leg press) and running speed (6 times 5-m shuttle run) were measured 1 wk before the first training session and 4 days after the last session. In addition, vertical jump height (static jump and

countermovement jump) was evaluated based on flight time, using a Bosco system device (Ergo-Jump, Elite).

### Muscle Biopsies

Two muscle samples were obtained from the vastus lateralis of the right leg using the needle biopsy technique (3) with suction. The pretraining muscle sample was obtained 3 days after the pretest session to minimize the possibility of studying damaged fibers. The posttraining muscle sample was obtained 3 days after the posttest session. Muscle samples were immediately placed in cold ( $0^\circ\text{C}$ ) skinning solutions and divided into small bundles of fibers. The bundles were stored in skinning solution at  $4^\circ\text{C}$  for 1 h, then transferred to fresh skinning solution, and stored at  $-20^\circ\text{C}$  for a minimum of 5 days before the first experiment.

*Skimming, activating, and relaxing solutions.* The skinning solution contained (in mM) 125 propionic acid, 2.0 EGTA, 1  $\text{MgCl}_2$ , 4.0 ATP, 20 imidazol (pH 7.0), and 50% (vol/vol) glycerol and protease inhibitors: 0.5 mM PMSF and 20  $\mu\text{g/ml}$  leupeptin. The composition of the relaxing and the activating solutions was based on calculations using an iterative computer program described by Fabiato and Fabiato (10) with apparent stability constants adjusted for temperature, pH, and ionic strength. Both solutions contained (in mM) 7.0 EGTA, 20 imidazol, 14.5 creatine phosphate, 1.0 free  $\text{Mg}^{2+}$ , and 4.0  $\text{MgATP}$ . The free  $\text{Ca}^{2+}$  concentration in the relaxing and activating solution was pCa 9.0 and pCa 4.5 (pCa =  $-\log[\text{Ca}^{2+}]$ ), respectively. In both solutions, pH was adjusted to 7.0 with KOH and total ionic strength to 180 mM with KCl.

### Single Muscle Fiber Mechanical Tests

Single muscle fibers were subjected to a series of mechanical tests administered in the same order as presented here, but not all tests were performed on all fibers (see RESULTS). All mechanical tests were completed within a time span of 5–6 wk following the muscle biopsies. On the day of an experiment, a bundle was transferred from the skinning solution to the relaxing solution on ice. Muscle fiber segments of 2.0–2.5 mm were isolated from the bundle. A single fiber was mounted between an isometric force transducer (model 400A, Aurora Scientific, Ontario, Canada) and the arm of a high-speed motor (model 312B, Aurora Scientific) by means of two connectors, as described by Moss (20). Briefly, fiber ends were fixed between a small stainless-steel cup (0.15-mm internal diameter) and a 4-0 monofilament post. Each side was secured with a loop of 10-0 nylon monofilament suture. The motor was operated either in length (slack tests and passive stretch tests) or in force mode (isotonic contractions) via a high-speed digital controller (model 600A, Aurora Scientific) consisting of an electronic interface, a 16-bit analog-to-digital converter, and custom software running under a real-time version of the Linux operating system. The controller also recorded the output signals from the motor and the force transducer (5-kHz sampling rate) and provided calibration and protocol programming procedures. Once mounted, the fiber could be rapidly transferred between the wells of a small Teflon plate containing either the relaxing or the activating solution. The setup was built over the stage of an inverted microscope (Axiovert 25C, Zeiss, Germany) so that the fiber could be viewed with a magnification of  $\times 400$ . The microscope stage was cooled using a bath/circulation thermostat (Ecoline RE 106, Lauda, Germany), and the temperature of the solutions was controlled by a thermocouple inserted into one of the wells. All experiments were performed at  $15^\circ\text{C}$ . Collected data were analyzed using custom-made software written in our laboratory (LabView, National Instruments).

### Single-Fiber Dimensions

Sarcomere length was adjusted to 2.5  $\mu\text{m}$  by use of a calibrated ocular micrometer ( $\times 400$ ). Subsequently, a digital camera (Camedia C3020 Z, Olympus) was connected to the microscope and interfaced

to a personal computer, allowing picture capture of the fiber while briefly suspended in the air ( $\pm 5$  s). Fiber width was determined on the calibrated picture and recorded as the average value measured at three points along the length of the sample. CSA was calculated assuming that the fiber takes a cylindrical form when it is suspended in the air. Fiber length (FL) was measured as the distance between the two fixation ends by using a calibrated digital picture recorded with a magnification of  $\times 50$ .

**Single-fiber  $P_0$ .** Fiber contraction was induced by rapidly transferring the fiber to a well-containing activating solution.  $P_0$  ( $\mu\text{N}$ ) was determined for each fiber as the highest force recorded while the fiber was in the activating solution (pCa 4.5). Peak specific tension ( $\text{kN}/\text{m}^2$ ) was defined as  $P_0/\text{CSA}$ .

**Single-fiber  $V_0$ .** The slack test method was used to measure  $V_0$ . The fiber was activated in the pCa 4.5 solution. When peak isometric force was reached, a step was imposed so that the force dropped rapidly to zero and then redeveloped after a time lapse proportional to the step length (Fig. 1). The fiber was then relaxed with the pCa 9.0 solution and slowly reextended to its original length. This operation was repeated with different step lengths so that a minimum of five different steps was applied on each fiber, with each step  $\leq 20\%$  of initial FL. For each fiber, the relationship between the time required for force redevelopment and the step length was fit with a first-order least squares regression. The slope corresponds to  $V_0$  of the fiber and was expressed in FL/s to account for differences in the number of sarcomeres in series between different fiber preparations.

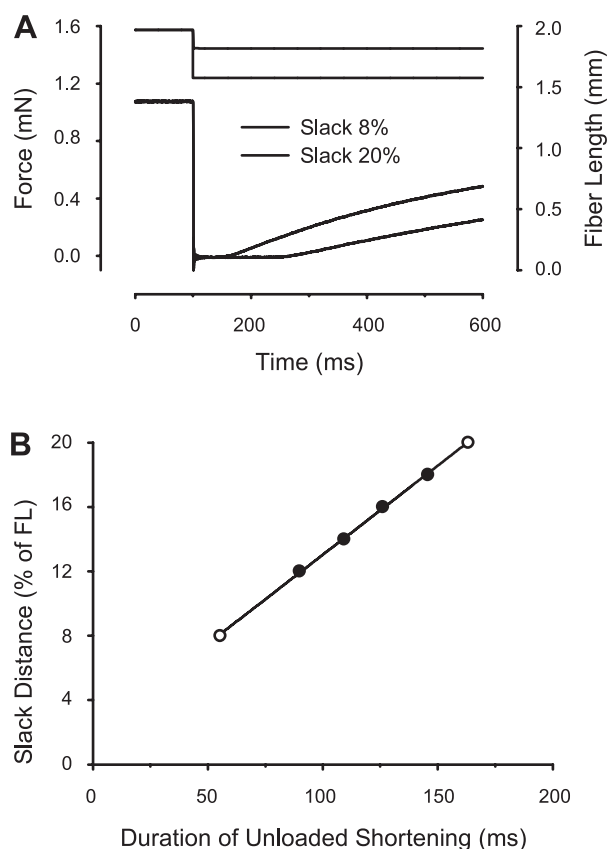


Fig. 1. Representative example of slack test experiments. *A*: superimposed position and force records of 2 slack tests performed on the same single fiber [myosin heavy chain (MHC) type I in this example]. When maximal peak force was reached, the fiber was subjected to a rapid shortening of various steps, in this case 8 and 20% of initial fiber length (FL). *B*: step lengths from 6 different slack tests plotted against the respective duration of unloaded shortening of the fiber.  $\circ$ , Data points drawn from the gross recordings in *A*. Fiber maximal shortening velocity was determined as the slope of the least squares linear regression and expressed as FL/s.

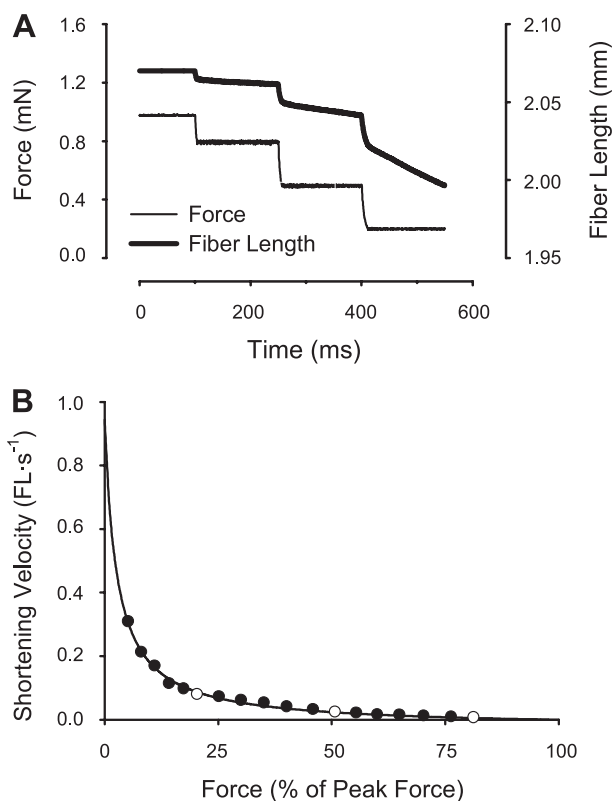


Fig. 2. Representative example of an isotonic contraction experiment. *A*: after maximal activation, the fiber was subjected to 3 successive load clamps of 150-ms duration. Velocity and force were evaluated over the last 50 ms of each step. *B*: data points of 6 isotonic contraction experiments from the same fiber (MHC I in this example) were fit with the Hill equation.  $\circ$ , Data drawn from the gross recording represented in *A*.

#### Single Fiber Force-Velocity Relationship

Fiber-shortening velocity was measured at different loads during isotonic contractions. After full activation with pCa 4.5 solution, the fiber was subjected to three successive isotonic load steps (Fig. 2). Each step was 150 ms in duration. Shortening velocity and force were measured over the final third of the step (50 ms) when the force was constant and the fiber-shortening velocity linear. The last isotonic clamp was followed by a step length to slacken the segment to 80% of initial FL. The fiber was then transferred back to the relaxing solution where it was slowly reextended to its original length. This procedure was repeated five to six times at different loads so that each fiber was submitted to a total of 15–18 isotonic contractions. All shortening velocities were normalized with respect to initial FL and expressed as FL/s. The data obtained on a single fiber were fit by the Hill equation (14) using an iterative nonlinear curve-fitting procedure (Marquardt-Levenberg algorithm), according to  $(P + a)(V + b) = (P_0 + a)b$ , where  $P$  is force,  $V$  is velocity, and the constants  $a$  and  $b$  have the dimension of force and velocity, respectively. Only individual experiments for which this relationship yielded a value for  $r^2$  of  $\geq 0.98$  were included for further analysis. Fiber power was calculated from the parameters of the fitted force-velocity relationship [ $P_0$ , maximal shortening velocity ( $V_{\max}$ ), and  $a/P_0$ , a unitless parameter describing the curvature of the force-velocity relationship]. Absolute power ( $\mu\text{N}\cdot\text{FL}/\text{s}$ ) was calculated as the product of force and contraction velocity, and normalized power ( $W/l$ ) was defined as the product of specific tension and contraction velocity.

#### Passive Tension Measurement

After the active tests, some fibers were subjected to a progressive stretch-release protocol while remaining in the pCa 9.0 solution (Fig.

3). Due to the time constraints inherent to this study, this test was performed only on approximately every second fiber. Starting from the initial FL, the fiber was slackened to 76% and then progressively stretched to 140% in successive steps of 8% of initial FL. Based on studied range of FL/s, the assumed range of sarcomere lengths was 1.90–3.50  $\mu\text{m}$ , representing a wide physiological range from below the slack sarcomere length to a value not exceeding the yield sarcomere length of skeletal muscle (21, 24). The stretch was established over a period of 10 s, after which FL was kept constant. Step duration was 3 min, during which passive force increased rapidly to a peak on stretching and then progressively declined toward a plateau during the constant length phase (stress relaxation). Following the step at 140% of initial FL, the fiber was progressively relaxed using similar time intervals for length changes and step durations. During the experiment, FL and passive force were continuously measured and recorded by the computer with a sampling rate of 10 Hz (Fig. 3A). Passive tension ( $\text{kN}/\text{m}^2$ ) was defined as the CSA-normalized force recorded at the end of each step, expressed as the change compared with the value recorded at 76% of initial fiber segment length. For each experiment, passive tension was expressed as a function of fiber strain, evaluated as the ratio of FL for a given stretch (mm) divided by FL at 76% of initial length (mm) minus one (Fig. 3B). The ascending limb of the passive tension-fiber strain relationship was analyzed using the equa-

tion  $y = E \cdot x^2$ , where  $E$  ( $\text{kN}/\text{m}^2$ ) represents complex Young's modulus reflecting the steepness of the curve and thus the stiffness of the fiber (24). Only individual experiments for which this relationship yielded an  $r^2$  value of  $\geq 0.98$  were included for further analyses. Hysteresis specific to each fiber ( $\text{kN}/\text{m}^2$ ) was defined as the area comprised between the ascending and descending limb of the passive tension-fiber strain relationship (Fig. 3B).

#### Fiber MHC Isoform Determination

MHC isoform content of single fibers was studied by SDS-PAGE, based on the method of Bamman et al. (2). After completion of the mechanical tests, the muscle fiber was removed from the ergometer for biochemical analysis. The fiber was first dissolved in 25  $\mu\text{l}$  of SDS sample buffer [0.160 M tris(hydroxymethyl)aminomethane, pH 6.8, 4% SDS, 2 mg/ml bromophenol blue, 20% glycerol, 30  $\mu\text{g}/\text{ml}$  leupeptin, and 1%  $\beta$ -mercaptoethanol] and stored at  $-20^\circ\text{C}$  before further processing. Later, the aliquot was heated at  $95^\circ\text{C}$  for 3 min, and 5  $\mu\text{l}$  of this extract were applied to a mini-protean 3 cell system (Bio-Rad, Hercules, CA). Total acrylamide concentrations were 4 and 8% in the stacking and the separating gel, respectively (acrylamide:bis-acrylamide, 50:1). Gels ( $10 \times 8 \times 0.075$  cm) were run at a constant voltage of 140 V for 17 h at  $4^\circ\text{C}$  and subsequently stained using a Silver Stain Plus kit (Bio-Rad). A sample of homogenized human skeletal muscle was used as the standard for the identification of MHC isoform bands, appearing as a triplet with type I, IIa, and IIx MHCs running as the lower, middle, and upper bands, respectively. The migration order (MHC I > MHC IIa > MHC IIx) had previously been confirmed by Western blotting using specific immunoreactivities of monoclonal antibodies. MHC expression was determined on each fiber segment used for the mechanical properties tests plus on  $\sim 120$  fibers per biopsy to determine the MHC profile on a representative sample of the muscle in each subject. The latter samples were stained with 0.04% (wt/vol) Coomassie blue G 250.

#### Statistical Analyses

Results are presented as means  $\pm$  SE. The effect of SSC exercise training on functional results and MHC profiles was examined using a one-way ANOVA for repeated measurements. All fiber segments were grouped according to their MHC isoform composition to evaluate the changes in single fibers. Differences between fiber types (according to MHC isoforms) in pretraining samples were assessed using a two-way ANOVA, with subject and fiber type as main factors. Pre- and posttraining results were analyzed with a two-way ANOVA, with subject and training status as main factors. Tukey's post hoc tests were used to identify individual differences. Because of lack of data for type I/IIa and IIx fibers, the pattern of empty cells did not allow the use of a two-way ANOVA, which makes it impossible to evaluate the training effect in these fibers. Statistical significance was accepted at  $P < 0.05$ .

## RESULTS

### Functional Performance

Compliance to the 8-wk SSC exercise training program was 100% for the eight participants, leading to significant improvements in all functional performances (Table 1). Vertical jump performances were increased for the static jump and the countermovement jump by 9% ( $P < 0.01$ ) and 13% ( $P < 0.001$ ), respectively, as a result of SSC exercise training. Shuttle run time was decreased by 4% ( $P < 0.05$ ) after training, and one-repetition maximal strength developed at the leg press was improved by 12% ( $P < 0.01$ ).

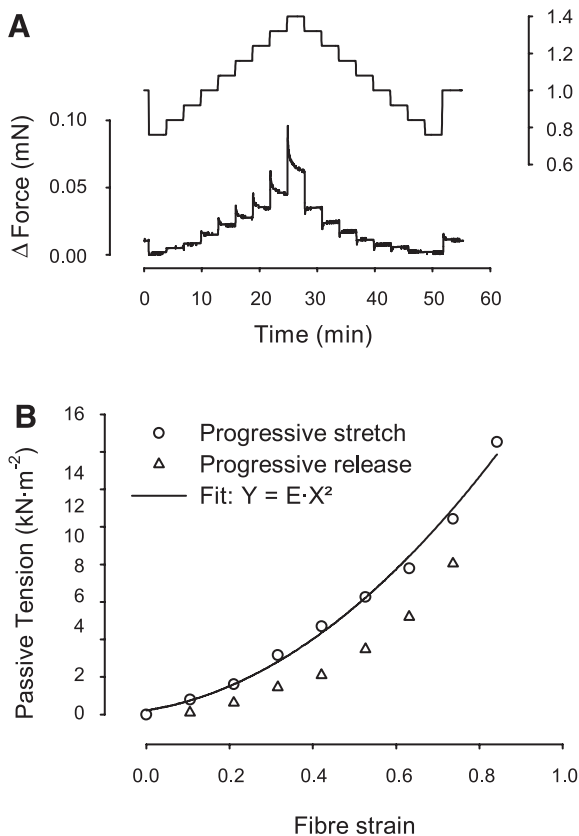


Fig. 3. Progressive stretch experiment. A: fiber was progressively stretched from 76 to 140% of initial FL (sarcomere length of 2.5  $\mu\text{m}$ ) and then released again to 76% using a step duration of 3 min.  $\Delta$ Force, change of passive force with respect to the value recorded at 76% of initial fiber length. B: passive tension was taken as the force recorded at the end of each step divided by fiber cross-sectional area and expressed as the change compared with the value at 76% of initial FL. Fiber strain was defined as any given FL divided by FL at 76% of initial length minus one. The ascending limb of the passive tension-fiber strain relationship ( $\circ$ ) was used to determine complex Young's modulus ( $E$ ) according to  $y = E \cdot x^2$  (solid line). The data displayed were acquired from a fiber containing MHC I, and the complex Young's modulus value was 20.44  $\text{kN}/\text{m}^2$ .

Table 1. Functional performance before and after 8 wk of SSC exercise training

Functional Tests	Pre	Post
SJ, m	0.40±0.01	0.43±0.01†
CMJ, m	0.41±0.01	0.47±0.01‡
Shuttle run, s	9.98±0.17	9.62±0.09*
Leg press, kg	223±15	248±12†

Value are means ± SE. SSC, stretch-shortening cycling; SJ, static jump; CMJ, countermovement jump; Pre, before SSC training; Post, after SSC training. Significant differences between Pre and Post tests: \* $P < 0.05$ ; † $P < 0.01$ ; ‡ $P < 0.001$ .

### Myosin Isoform Composition of Pre- and Posttraining Fibers

Myosin isoform composition was determined on a total of 1,115 pre- and 1,002 posttraining fibers from the vastus lateralis muscle of the eight participants (Table 2). The majority of muscle fibers contained MHC I, MHC IIa, or MHC IIa/IIx in pre- and posttraining samples (90 and 92% of all fibers analyzed, respectively, before and after training). There was a tendency for an increased proportion of type IIa fibers (from 33 to 41%;  $P = 0.081$ ) and a decreased percentage of type IIx fibers (from 7 to 3%;  $P = 0.059$ ), but not all subjects responded in the same way to training. The total number of hybrid fibers (I/IIa, IIa/IIx, and I/IIa/IIx) was not altered (30 and 28%, respectively, before and after the training), and the only significant change was found in the proportion of type I/IIa fibers, which rose from 2 to 5% ( $P < 0.05$ ).

### Single Muscle Fiber CSA, $P_0$ , and $P_0/CSA$

Before training, fiber CSA was significantly smaller in type I fibers compared with type IIa fibers (Table 3). However, type I fibers also produced significantly less peak force than type IIa fibers, thus yielding similar values for specific tension in both fiber types. Hybrid fibers containing IIa/IIx MHC produced significantly greater normalized force than type IIa fibers because of their lower CSA and similar  $P_0$ . The data of type I/IIa and IIx fibers must be interpreted with caution due to the limited number of cases. After SSC exercise training, mean CSA of type I, IIa, and IIa/IIx fibers were significantly increased by 23, 22, and 30%, respectively. These hypertrophies were coupled with respective increases of 19, 15, and 16% in  $P_0$  of these fibers. Consequently, no training-induced changes were observed for these fiber types when  $P_0$  was normalized by CSA. The results from type I/IIa and IIx fibers cannot be interpreted due to the limited data available.

### Unloaded $V_0$

Before training, type I fibers contracted three times slower than type IIa fibers and 4.4 times slower than type IIx fibers during the slack tests (Table 4). Hybrid fibers containing type I/IIa or IIa/IIx MHC had shortening velocities that were intermediate to those of fibers containing one or the other single MHC isoform. The explosive muscle contractions inherent to the SSC exercise training induced a significant improvement of  $V_0$  in all fiber types after 8 wk of practice ( $P < 0.001$ ). Type II fibers showed the greatest increase (+29% for type IIa fibers and +22% for type IIa/IIx fibers), whereas type I fibers displayed a somewhat smaller improvement (+18%).

### Force-Velocity-Power Relationships

Force-velocity-power experiments were applied on a slightly smaller number of fibers than the slack tests (Table 5). Before training, peak power was lower in slow fibers than in each fiber group containing fast MHC isoforms ( $P < 0.05$ ). When normalized by CSA, peak power in type IIa fibers was still significantly greater than in type I fibers but lower compared with type IIa/IIx fibers ( $P < 0.05$ ). Since SSC exercise training had an effect on  $V_{max}$  and peak force in several fiber groups, peak power was enhanced in type I (25%;  $P < 0.01$ ), type IIa (34%;  $P < 0.001$ ), and type IIa/IIx fibers (49%;  $P < 0.001$ ). Normalized peak power was increased 9% ( $P < 0.05$ ) following training in type IIa fibers only, with no significant modifications in type I and IIa/IIx fibers.

The extrapolation of the force-velocity relationship was used as a second method to evaluate  $V_{max}$ .  $V_{max}$  confirmed the slack test results in pretraining fibers and showed that type IIa fibers ( $1.64 \pm 0.07$  FL/s) contracted significantly faster ( $P < 0.05$ ) than type I fibers ( $0.66 \pm 0.03$  FL/s) and slower than type IIa/IIx fibers ( $1.93 \pm 0.10$  FL/s) or type IIx fibers ( $2.19 \pm 0.26$  FL/s). In agreement with  $V_0$ ,  $V_{max}$  increased significantly in type IIa fibers to  $1.94 \pm 0.06$  FL/s ( $P < 0.01$ ) and in type IIa/IIx fibers to  $2.31 \pm 0.13$  FL/s ( $P < 0.05$ ) after 8 wk of SSC exercise training. However,  $V_{max}$  did not change in type I fibers ( $0.73 \pm 0.04$  FL/s posttraining;  $P = 0.20$ ).

Pre- and posttraining force-velocity and force-power curves of type I, IIa, and IIa/IIx fibers were calculated based on the parameters of the fitted force-velocity relationship. Average curves of these fiber types in the two conditions are illustrated in Fig. 4. The parameter expressing the shape of the curve,  $a/P_0$ , did not change for type I fibers ( $0.039 \pm 0.002$  and  $0.036 \pm 0.002$  pre- and posttraining, respectively), type IIa fibers ( $0.048 \pm 0.003$  and  $0.042 \pm 0.002$  pre- and posttraining, respectively), and type IIa/IIx fibers ( $0.038 \pm 0.002$  and  $0.040 \pm 0.002$  pre- and posttraining, respectively). SSC exercise training had an effect on the velocity that produced peak power for type IIa fibers (increases from  $0.27 \pm 0.01$  to  $0.31 \pm 0.01$  FL/s;  $P < 0.001$ ) and type IIa/IIx fibers (increases from  $0.30 \pm 0.01$  to  $0.36 \pm 0.02$  FL/s;  $P < 0.05$ ), but not for type I fibers (increases from  $0.100 \pm 0.003$  to  $0.109 \pm 0.003$ ;  $P = 0.11$ ). Similarly, the absolute force at which peak power was reached increased in type IIa (from  $0.20 \pm 0.01$  to  $0.23 \pm 0.01$  mN;  $P < 0.001$ ) and type IIa/IIx fibers (from  $0.18 \pm 0.01$  to  $0.21 \pm 0.01$  mN;  $P < 0.001$ ), but not in type I fibers (increase from  $0.13 \pm 0.01$  to  $0.15 \pm 0.01$  mN;  $P = 0.09$ ). No effect on the normalized force at the peak power was observed for any fiber type.

Table 2. MHC isoform profiles before and after 8 wk of SSC exercise training

MHC Isoforms, %	Pre	Post	P Value
I	30.0±4.9	29.2±4.1	0.774
I/IIa	1.9±0.5	5.0±1.4	0.048*
IIa	33.4±5.2	40.6±4.2	0.081
IIa/IIx	26.9±1.9	22.2±4.3	0.235
IIx	7.0±3.0	2.6±1.9	0.059
I/IIa/IIx	0.8±0.4	0.4±0.2	0.406

Values are means ± SE relative percentages of fibers found in 8 subjects. MHC, myosin heavy chain. \*Significant difference between Pre and Post ( $P < 0.05$ ).

Table 3. CSA of fibers,  $P_0$ , and  $P_0/CSA$  before and after 8 wk of SSC exercise training

MHC Isoform	No. of Fibers		CSA, $\mu\text{m}^2$		$P_0$ , mN		$P_0/CSA$ , $\text{kN/m}^2$	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
I	61	50	4,782 ± 200 <sup>a</sup>	5,862 ± 264 <sup>c</sup>	0.83 ± 0.03 <sup>a</sup>	0.99 ± 0.03 <sup>c</sup>	181 ± 6 <sup>a,b</sup>	173 ± 4
I/IIa	3	6	4,800 ± 896 <sup>a,b</sup>	8,716 ± 1,583	1.12 ± 0.08 <sup>a,b</sup>	1.23 ± 0.17	249 ± 46 <sup>a,b</sup>	147 ± 10
IIa	80	72	6,511 ± 177 <sup>b</sup>	7,918 ± 213 <sup>d</sup>	1.19 ± 0.03 <sup>b</sup>	1.37 ± 0.03 <sup>d</sup>	183 ± 3 <sup>a</sup>	176 ± 3
IIa/IIx	49	28	5,518 ± 243 <sup>a</sup>	7,167 ± 273 <sup>d</sup>	1.11 ± 0.05 <sup>b</sup>	1.29 ± 0.04 <sup>d</sup>	205 ± 5 <sup>b</sup>	182 ± 5
IIx	14	7	6,043 ± 492 <sup>a,b</sup>	6,571 ± 463	1.22 ± 0.08 <sup>b</sup>	1.27 ± 0.07	205 ± 8 <sup>a,b</sup>	197 ± 12

Values are means ± SE. CSA, cross-sectional area;  $P_0$ , peak  $\text{Ca}^{2+}$ -activated force. <sup>a,b</sup>Significant differences between fiber types in Pre samples are shown with different superscript letters. Significant differences between Pre and Post fibers containing similar MHC isoforms: <sup>c</sup> $P < 0.01$ ; <sup>d</sup> $P < 0.001$ . Due to lack of data, no statistical analysis was performed to evaluate the training effect in type I/IIa and type IIx fibers.

### Passive Tension

The tension developed by the single fibers on passive stretching and releasing was measured on a total of 74 pretraining and 71 posttraining fibers, which turned out to express type I, IIa, or IIa/IIx MHC. The results were largely affected by the MHC content of the fibers (Fig. 5). In pretraining conditions, fibers expressing type I MHC had significantly lower values for complex Young's modulus ( $E = 23.2 \pm 1.8 \text{ kN/m}^2$ ;  $n = 22$ ) than type IIa fibers ( $E = 42.5 \pm 1.7 \text{ kN/m}^2$ ;  $n = 35$ ), which were themselves more compliant than type IIa/IIx fibers ( $E = 54.3 \pm 3.2 \text{ kN/m}^2$ ;  $n = 17$ ;  $P < 0.05$ ). After training, the stiffness of type IIa/IIx fibers was increased ( $E = 66.1 \pm 4.6 \text{ kN/m}^2$ ;  $n = 12$ ;  $P < 0.05$ ), whereas no changes were noted in type I ( $n = 20$ ) and IIa fibers ( $n = 39$ ).

Hysteresis was also highly dependent on the MHC isoforms expressed, with values of  $1.67 \pm 0.15$ ,  $3.59 \pm 0.16$ , and  $5.18 \pm 0.31 \text{ kN/m}^2$  for type I, IIa, and IIa/IIx pretraining fibers, respectively. After SSC exercise training, hysteresis was higher in single fibers containing IIa ( $4.09 \pm 0.13 \text{ kN/m}^2$ ;  $P < 0.01$ ) and IIa/IIx MHC ( $6.44 \pm 0.47 \text{ kN/m}^2$ ;  $P < 0.05$ ), with no changes in type I fibers ( $1.54 \pm 0.74 \text{ kN/m}^2$ ).

### DISCUSSION

#### Effect of SSC Exercise Training on the Contractile Apparatus

Over the last years, a series of studies have documented the functional properties of human single muscle fibers in different populations, and the mechanical changes induced on specific fiber types by various training regimes. Currently, an optimal exercise program to enhance muscle function via both contrac-

tion velocity and force has not been elucidated. The results of this study demonstrate that short-term SSC exercise training enhances functional performance of all muscle fiber types through improvements of both shortening velocity and force. After only 8 wk of SSC exercise training with three weekly sessions,  $P_0$  was increased in type I, type IIa, and hybrid type IIa/IIx fibers, mainly as a result of greater fiber CSA. These force enhancements were concomitant to increases of  $V_0$  in all fiber types. As a consequence, absolute peak power of single fibers was enhanced by 25, 34, and 49% in type I, type IIa, and hybrid type IIa/IIx fibers, respectively, thus confirming our major hypothesis. Additionally, a new and rather uncommon finding was that normalized peak power was higher in type IIa fibers following the training period. This observation can be essentially explained by the large effect on contraction velocity and, more specifically, the velocity at which peak power was reached. Favorable changes at the single fiber level were associated with significant improvements in all functional variables related to whole muscle performance. This is the first study to show significant improvements simultaneously in characteristics related to fiber size, force, contraction velocity, and power in already moderately active healthy young men. Therefore, we propose that SSC exercise training should be considered as an interesting additional or alternative approach to existing exercise programs to enhance overall muscle function.

#### Muscular and Neuromuscular Origin of Improved Performance

SSC exercise training is based on explosive movements such as jumping and bounding to enhance the ability of muscle to generate power. This kind of training utilizes the SSC in lower limb muscles, involving a rapid eccentric contraction, immediately followed by a powerful concentric contraction phase. SSC exercise training has been shown to improve jumping ability and other high-power movements (17, 23). Critical to performance enhancements is the velocity of the eccentric phase just preceding the concentric contraction (23). Although the benefits of this type of training are well recognized, the mechanisms involved in the effects on whole muscle performance have been insufficiently explored. Training effects are mostly attributed to neuromuscular adaptations, i.e., muscle activity patterns of agonists and antagonists (6), as well as motor unit recruitment strategies (12, 18, 29). Although short-term SSC exercise training did not induce substantial modifications of the MHC isoform composition, the present study provides new data that demonstrates increases in muscle fiber

Table 4. Maximum unloaded shortening velocity (FL/s) before and after 8 wk of SSC exercise training

MHC Isoform	$V_0$ , FL/s	
	Pre	Post
I	0.94 ± 0.03 <sup>a</sup>	1.11 ± 0.03 <sup>c</sup>
I/IIa	2.18 ± 0.49 <sup>b</sup>	1.79 ± 0.26
IIa	2.94 ± 0.06 <sup>b</sup>	3.78 ± 0.07 <sup>c</sup>
IIa/IIx	3.58 ± 0.10 <sup>c</sup>	4.37 ± 0.14 <sup>c</sup>
IIx	4.13 ± 0.19 <sup>d</sup>	5.25 ± 0.14

Values are means ± SE, representing the same number of fibers as in Table 3. FL, fiber length. <sup>a-d</sup>Significant differences between fiber types in Pre samples are symbolized by different superscript letters. Significant differences between Pre and Post fibers containing similar MHC isoforms: <sup>c</sup> $P < 0.001$ . Due to lack of data, no statistical analysis was performed to evaluate the training effect in type I/IIa and type IIx fibers.

Table 5. Peak power of fibers evaluated before and after 8 wk of SSC exercise training

MHC Isoform	No. of Fibers		Absolute Peak Power, $\mu\text{N}\cdot\text{FL}\cdot\text{s}^{-1}$		Normalized Peak Power, W/l	
	Pre	Post	Pre	Post	Pre	Post
I	60	51	12.9 $\pm$ 0.5 <sup>a</sup>	16.1 $\pm$ 0.7 <sup>e</sup>	2.85 $\pm$ 0.11 <sup>a</sup>	2.84 $\pm$ 0.08
I/IIa	3	6	40.9 $\pm$ 7.2 <sup>b</sup>	37.0 $\pm$ 9.9	8.62 $\pm$ 0.57 <sup>b,c</sup>	4.14 $\pm$ 0.69
IIa	78	72	51.2 $\pm$ 1.5 <sup>b</sup>	68.7 $\pm$ 1.7 <sup>f</sup>	8.11 $\pm$ 0.22 <sup>c</sup>	8.87 $\pm$ 0.20 <sup>d</sup>
IIa/IIx	48	28	49.7 $\pm$ 2.8 <sup>b</sup>	74.2 $\pm$ 2.8 <sup>f</sup>	9.45 $\pm$ 0.49 <sup>b</sup>	10.74 $\pm$ 0.58
IIx	14	7	59.4 $\pm$ 6.0 <sup>b</sup>	104.1 $\pm$ 7.1	10.53 $\pm$ 1.24 <sup>b</sup>	16.54 $\pm$ 1.88

Values are means  $\pm$  SE. <sup>a-c</sup> Significant differences between fiber types in Pre samples are symbolized by different superscript letters. Significant differences between Pre and Post fibers containing similar MHC isoforms: <sup>d</sup> $P < 0.05$ ; <sup>e</sup> $P < 0.01$ ; <sup>f</sup> $P < 0.001$ . Due to lack of data, no statistical analysis was performed to evaluate the training effect in type I/IIa and type IIx fibers.

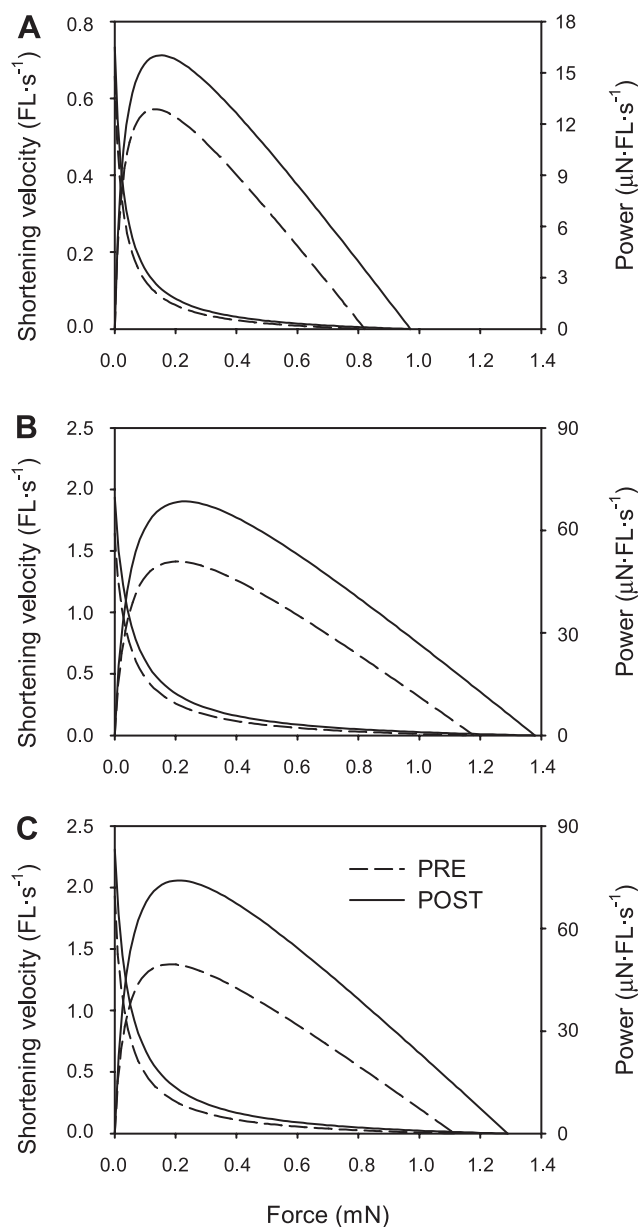


Fig. 4. Shortening velocity and power as a function of fiber force in samples expressing type I (A), type IIa (B), or type IIa/IIx fibers (C). Data represent the average curves of all fibers before (Pre) and after (Post) 8 wk of stretch-shortening cycle exercise training. The specific curve of each fiber was determined by the parameters of the fitted force-velocity relationship.

size and enhancements in functional properties of the contractile apparatus. The characteristics inherent to this type of training were reflected in the improvements of single-fiber contraction performance.  $V_0$  was increased in type I and even more so in type II fibers, probably due to the rapid executions, and single-fiber force was enhanced as a result of the high-intensity contractions recruiting all fiber types.

Although it makes sense to believe that the fast muscle contractions inherent to our training protocol could be accountable for the increase in fiber contraction velocity, we have no evidence to support a causal relationship. Furthermore, the present data do not provide an explanation about the mechanisms involved in the functional improvements of fiber mechanics, especially  $V_0$ . It has been commonly observed that fibers containing the same MHC isoform have variable  $V_0$  and power and that changes can be induced by physiological and pathological conditions. Some possible causes involve differential myosin light chain isoform expression, myosin concentration in single muscle fibers, and the influence of other sarcomeric proteins on the properties of actomyosin interaction (4). The pattern of myosin light chain expression does not seem a likely candidate to explain the changes in  $V_0$  observed in the present study, since previous work on human muscle fibers failed to correlate the two characteristics (26, 30, 32). If the myosin concentration was a determining factor in our obser-

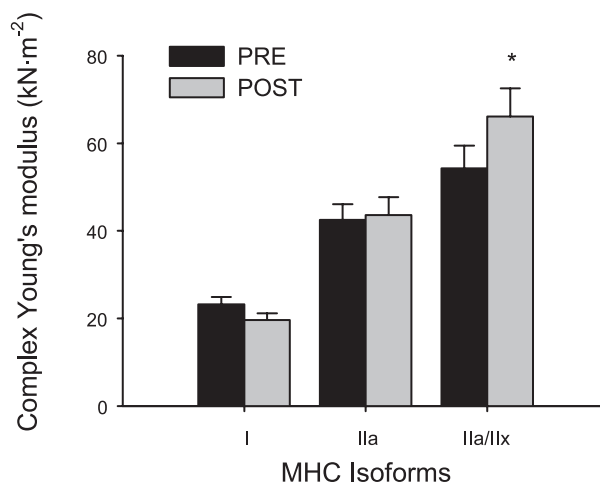


Fig. 5. Complex Young's modulus in type I, IIa, and IIa/IIx fibers before (Pre;  $n = 22, 35,$  and  $17,$  respectively) and after (Post;  $n = 20, 39,$  and  $12,$  respectively) 8 wk of stretch-shortening cycle exercise training. \*Significant differences between pre- and posttraining fibers containing similar MHC isoforms ( $P < 0.05$ ). Due to lack of data, fiber types I/IIa and IIx could not be analyzed.

vations, it is likely that specific tension would have been altered following training. Despite some variability observed in  $P_0/CSA$ , no significant changes could be revealed here. The influence of other sarcomeric proteins probably represents the most promising direction of study to explain the functional heterogeneity of single muscle fibers. Clearly, further research is required to elucidate this phenomenon.

#### *SSC vs. Resistance Exercise Training*

The results in single-fiber mechanics found in the present study compare well with those described by Widrick et al. (31) following a 12-wk resistance exercise training. They reported increases of 30 and 42% in absolute peak power of type I and IIa fibers, respectively, compared with our increases of 25 and 34%, respectively. However, fiber power increases following resistance training (31) were not due to an improvement of  $V_0$  but exclusively to fiber hypertrophy and enhanced absolute forces of 40 and 35% for type I and IIa fibers, respectively. Similarly, Shoeppe et al. (22) found that single-fiber power was higher in individuals who had been involved in regular resistance training for several years, compared with sedentary control subjects. Again, the authors put forward the fiber hypertrophy as the main factor explaining the higher absolute peak power. SSC exercise training seems to differentiate from resistance training on account of the  $V_0$  improvement observed in all fiber types, in compensation for the smaller enhancements in fiber force. Consequently, absolute power was increased in type I, IIa, and IIa/IIx fibers, and even normalized peak power was improved in type IIa fibers. This latter aspect is new, in the sense that none of the above-mentioned studies found any effect for this variable. This observation suggests that SSC exercise training is effective in improving the cross-bridge mechanics of single fibers.

Although resistance training has been associated with an increase in type IIa fibers coupled with a decrease in type IIa/IIx fibers (31), SSC exercise training induced only minor changes in type IIa fibers (+7%) and type IIa/IIx fibers (-5%). It is possible that a longer training period could have induced significant fiber-type transformations similar to those observed following longer-term resistance training. It should be noted here that our subjects had a rather low percentage of type I fibers compared with values reported for the general population (1, 31).

The changes in single-fiber mechanics following resistance training have also been studied in elderly populations, with the general aim to evaluate its effectiveness in the treatment of age-induced loss of muscle mass (sarcopenia) and deterioration of contractile performance. Older men displayed positive effects in  $P_0$ , but also in  $V_0$ , with quite large increases of 75 and 45% for type I and IIa fibers, respectively (26). The latter observation could have resulted from an age-related slowing of contraction velocity observed in men (19). On the other hand, aging has been associated with a lower specific tension developed by single fibers (19). This observation may partially explain the significant increases in normalized peak power found in type I and IIa fibers of elderly men (26), a result not commonly observed in training studies, as discussed above. In the present study, SSC training improved the cross-bridge mechanics in muscle fibers of our active young individuals,

thus revealing itself as an efficient method to improve contractile performance in this population.

#### *SSC Exercise Training as a Potential Countermeasure to Prevent Muscle Wasting*

Although the loss in size and function of human skeletal muscle with aging or unloading at the whole muscle and cellular level is well documented (8, 19), an ideal intervention for preventing deterioration of contractile function has not been determined. Although high-intensity, low-volume resistance exercise training during an 84-day bed rest in six participants maintained whole muscle size and peak force (25), this intervention did not prevent subtle changes at the single-fiber level. The authors noted a decrease of absolute and normalized power in type I muscle fibers of 20 and 30%, respectively. In type II fibers,  $V_0$  was increased and normalized power declined, but these results were not statistically significant. A shift in MHC profile from slower muscle fibers to faster, more powerful fibers and the preservation of type II fiber functional properties contributed to the maintenance of whole muscle strength and size during long-term bed rest. However, the authors underlined the inability of resistance training to maintain the functional characteristics of slow-twitch fibers. In light of this observation, SSC exercise training could be an interesting complement to exercise countermeasure programs used in unloading conditions, such as spaceflights. Furthermore, dynamic, high-impact SSC exercises are likely to produce large rates of deformation of the bone matrix and thus provide an appropriate mechanical stimulus to protect the individual against bone mass loss (28). However, these speculations need to be verified by future research.

#### *Passive Fiber Properties*

To the authors' knowledge, this is the first study to characterize the passive properties of single human muscle fibers according to their MHC isoform expression. However, since the total number of fibers analyzed is low, these results should be confirmed by further research. In pretraining samples, complex Young's modulus as well as hysteresis were significantly different between type I, IIa, and hybrid IIa/IIx fibers, the two latter displaying higher values for both variables. SSC exercise training induced increases in fiber stiffness, but only in IIa/IIx fibers, whereas hysteresis was greater in type IIa and IIa/IIx fibers. Type I fibers were not affected by training. These results indicate that whole muscle stiffness could be modulated via differential fiber-type expression or via changes in the passive characteristics of single muscle fibers, especially type II fibers.

The most likely candidate to confer elastic properties in single fibers is titin, an ~3 MDa giant structural protein that spans a half sarcomere from the M-line to the Z-disk (27). Two isoforms have been identified in skeletal muscle (21), but their influence on passive tensile properties of human muscle fibers has not yet been investigated. It has been speculated that differential expression of titin isoforms could regulate single-fiber stiffness (21), but further research is required to study this relationship.

In conclusion, 8 wk of training involving repetitive maximal SSC exercises increased single-fiber diameter, peak force, and shortening velocity, leading to enhanced fiber power. These beneficial effects were found in type I, IIa, and IIa/IIx fibers. In

addition, normalized peak power was improved in type IIa fibers. Fiber tension on passive stretch was increased in IIa/IIx fibers only. The favorable changes noted in all fiber types suggest that SSC exercises represent an interesting training paradigm to improve single-fiber force, contraction velocity, and power.

#### GRANTS

This study was supported by a grant (to D. Theisen) from the Fonds Spéciaux de Recherche, Université Catholique de Louvain.

#### REFERENCES

- Bamman MM, Clarke MS, Feeback DL, Talmadge RJ, Stevens BR, Lieberman SA, and Greenisen MC. Impact of resistance exercise during bed rest on skeletal muscle sarcopenia and myosin isoform distribution. *J Appl Physiol* 84: 157–163, 1998.
- Bamman MM, Clarke MS, Talmadge RJ, and Feeback DL. Enhanced protein electrophoresis technique for separating human skeletal muscle myosin heavy chain isoforms. *Electrophoresis* 20: 466–468, 1999.
- Bergstrom J. Muscle electrolytes in man. *Scand J Clin Lab Invest* 14, Suppl 68: 61–110, 1962.
- Bottinelli R. Functional heterogeneity of mammalian single muscle fibres: do myosin isoforms tell the whole story? *Pflügers Arch* 443: 6–17, 2001.
- Canepari M, Rossi R, Pellegrino MA, Orrell RW, Cobbold M, Hartridge S, and Bottinelli R. Effects of resistance-training on myosin function studied using the in vitro motility assay in young and older men. *J Appl Physiol* 98: 2390–2395, 2005.
- Chimera NJ, Swanik KA, Swanik CB, and Straub SJ. Effects of plyometric training on muscle-activation strategies and performance in female athletes. *J Athl Train* 39: 24–31, 2004.
- Connelly DM, Rice CL, Roos MR, and Vandervoort AA. Motor unit firing rates and contractile properties in tibialis anterior of young and old men. *J Appl Physiol* 87: 843–852, 1999.
- D'Antona G, Pellegrino MA, Adami R, Rossi R, Carlizzi CN, Canepari M, Saltin B, and Bottinelli R. The effect of ageing and immobilization on structure and function of human skeletal muscle fibres. *J Physiol* 552: 499–511, 2003.
- Dudley GA, Tesch PA, Miller BJ, and Buchanan P. Importance of eccentric actions in performance adaptations to resistance training. *Aviat Space Environ Med* 62: 543–550, 1991.
- Fabiato A and Fabiato F. Calculator programs for computing the composition of the solutions containing multiple metals and ligands used for experiments in skinned muscle cells. *J Physiol* 75: 463–505, 1979.
- Goldberg AL, Etlinger JD, Goldspink DF, and Jablecki C. Mechanism of work-induced hypertrophy of skeletal muscle. *Med Sci Sports Exerc* 7: 185–198, 1975.
- Hakkinen K, Pakarinen A, Kyrolainen H, Cheng S, Kim DH, and Komi PV. Neuromuscular adaptations and serum hormones in females during prolonged power training. *Int J Sports Med* 11: 91–98, 1990.
- Hather BM, Tesch PA, Buchanan P, and Dudley GA. Influence of eccentric actions on skeletal muscle adaptations to resistance training. *Acta Physiol Scand* 143: 177–185, 1991.
- Hill AV. The heat of shortening and the dynamic constants of muscle. *Proc R Soc B* 126: 136–195, 1938.
- Komi PV. Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. *J Biomech* 33: 1197–1206, 2000.
- Kraemer WJ, Fleck SJ, and Evans WJ. Strength and power training: physiological mechanisms of adaptation. *Exerc Sport Sci Rev* 24: 363–397, 1996.
- Kyrolainen H, Avela J, McBride JM, Koskinen S, Andersen JL, Sipila S, Takala TE, and Komi PV. Effects of power training on mechanical efficiency in jumping. *Eur J Appl Physiol* 91: 155–159, 2004.
- Kyrolainen H, Avela J, McBride JM, Koskinen S, Andersen JL, Sipila S, Takala TE, and Komi PV. Effects of power training on muscle structure and neuromuscular performance. *Scand J Med Sci Sports* 15: 58–64, 2005.
- Larsson L, Li X, and Frontera WR. Effects of aging on shortening velocity and myosin isoform composition in single human skeletal muscle cells. *Am J Physiol Cell Physiol* 272: C638–C649, 1997.
- Moss RL. Sarcomere length-tension relations of frog skinned muscle fibres during calcium activation at short lengths. *J Physiol* 292: 177–192, 1979.
- Neagoe C, Opitz CA, Makarenko I, and Linke WA. Gigantic variety: expression patterns of titin isoforms in striated muscles and consequences for myofibrillar passive stiffness. *J Muscle Res Cell Motil* 24: 175–189, 2003.
- Shoepe TC, Stelzer JE, Garner DP, and Widrick JJ. Functional adaptability of muscle fibers to long-term resistance exercise. *Med Sci Sports Exerc* 35: 944–951, 2003.
- Toumi H, Best TM, Martin A, F'Guyer S, and Poumarat G. Effects of eccentric phase velocity of plyometric training on the vertical jump. *Int J Sports Med* 25: 391–398, 2004.
- Toursel T, Stevens L, Granzier H, and Mounier Y. Passive tension of rat skeletal soleus muscle fibers: effects of unloading conditions. *J Appl Physiol* 92: 1465–1472, 2002.
- Trappe S, Trappe T, Gallagher P, Harber M, Alkner B, and Tesch P. Human single muscle fibre function with 84 day bed-rest and resistance exercise. *J Physiol* 557: 501–513, 2004.
- Trappe S, Williamson D, Godard M, Porter D, Rowden G, and Costill D. Effect of resistance training on single muscle fiber contractile function in older men. *J Appl Physiol* 89: 143–152, 2000.
- Tskhovrebova L and Trinick J. Titin: properties and family relationships. *Nat Rev Mol Cell Biol* 4: 679–689, 2003.
- Turner CH and Robling AG. Designing exercise regimens to increase bone strength. *Exerc Sport Sci Rev* 31: 45–50, 2003.
- Van Cutsem M, Duchateau J, and Hainaut K. Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. *J Physiol* 513: 295–305, 1998.
- Widrick JJ, Knuth ST, Norenberg KM, Romatowski JG, Bain JL, Riley DA, Karhanek M, Trappe SW, Trappe TA, Costill DL, and Fitts RH. Effect of a 17 day spaceflight on contractile properties of human soleus muscle fibres. *J Physiol* 516: 915–930, 1999.
- Widrick JJ, Stelzer JE, Shoepe TC, and Garner DP. Functional properties of human muscle fibers after short-term resistance exercise training. *Am J Physiol Regul Integr Comp Physiol* 283: R408–R416, 2002.
- Widrick JJ, Trappe SW, Blaser CA, Costill DL, and Fitts RH. Isometric force and maximal shortening velocity of single muscle fibers from elite master runners. *Am J Physiol Cell Physiol* 271: C666–C675, 1996.