Sprint training, in vitro and in vivo muscle function, and myosin heavy chain expression

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1Department of Physiology and Pharmacology, Karolinska Institute, Stockholm; 2Copenhagen Muscle Research Centre, Rigshospitalet, DK-2200 Copenhagen N, Denmark; 3Institute of Human Physiology, University of Pavia, I-27100 Pavia, Italy; and 4Department of Clinical Physiology, Huddinge Hospital, Karolinska Institute, S-114 86 Stockholm, Sweden

Harridge, S. D. R., R. Bottinelli, M. Canepari, M. Pellegrino, C. Reggiani, M. Esbjörnsson, P. D. Balsom, and B. Saltin. Sprint training, in vitro and in vivo muscle function, and myosin heavy chain expression. J. Appl. Physiol. 84(2): 442–449, 1998.—Sprint training represents the condition in which increases in muscle shortening speed, as well as in strength, might play a significant role in improving power generation. This study therefore aimed to determine the effects of sprint training on 1) the coupling between myosin heavy chain (MHC) isoform expression and function in single fibers, 2) the distribution of MHC isoforms across a whole muscle, and 3) in vivo muscle function. Seven young male subjects completed 6 wk of training (3-s sprints) on a cycle ergometer. Training was without effect on maximum shortening velocity in single fibers or in the relative distribution of MHC isoforms in either the soleus or the vastus lateralis muscles. Electrically evoked and voluntary isometric torque generation increased (P < 0.05) after training in both the plantar flexors (+8% at 50 Hz and +16% maximal voluntary contraction) and knee extensors (+8% at 50 Hz and +7% maximal voluntary contraction). With the shortening potential of the muscles apparently unchanged, the increased strength of the major lower limb muscles is likely to have contributed to the 7% increase (P < 0.05) in peak pedal frequency during cycling.

contraction; exercise

THE INCREASE IN MUSCLE POWER generation that results from training could be brought about by at least two different mechanisms. First, it can be brought about by an increase in the force-producing capability of the muscle, either through increased activation or through an increase in muscle size. Second, it can occur by increasing the speed at which a muscle can shorten against a given load.

A close coupling exists between the speed of muscle shortening and the expression of the different myosin heavy chain (MHC) isoforms. Muscle fibers that express the MHC-I isoform exhibit significantly slower maximal velocities of shortening (V₀) (24, 28) and power outputs (11) compared with fibers expressing the two fast MHC isoforms. Within the subgroups of fast isoforms, fibers that express the MHC-IIA isoform [which has recently been shown to be homologous to the MHC-IIA isoform found in small mammals (18, 34)] are significantly faster (24, 28) and can generate higher power outputs than fibers containing the MHC-IIB isoform (11, 36). Muscle speed of movement could be increased either by altering the relative expression of the fast and slow isoforms or by changing the coupling between isoform expression and function.

Skeletal muscle is a highly plastic tissue capable of altering its contractile proteins and its contractile properties with increased use, or with disuse (see Refs. 23 and 31 for reviews). The important role played by the activation pattern has been demonstrated by chronic electrical stimulation studies on animal muscle. Prolonged low-frequency electrical stimulation is capable of transforming fast contracting muscles into slow contracting muscles such that MHC isoform expression proceeds in the general direction MHC-IIB → MHC-IIA → MHC-I (31). Although there is some evidence that intermittent high-frequency stimulation may alter the properties of denervated soleus muscles to resemble those of a faster muscle (6, 22), an increase in the expression of MHC-IIA seems only possible with disuse (5). However, a change in activation pattern is only one determinant of the muscular adaptation to exercise. In general, changes in activation pattern are also accompanied by increases in mechanical load and by alterations of metabolic homeostasis (10). In human training studies, whether endurance or power based, the most common observation has been the decrease in the proportion of type II fibers, determined histochemically (4, 23) or, more recently, in MHC-IIA content determined electrophoretically (1). Sprint training seems to represent a notable exception because there is some evidence of a “bidirectional” transformation to MHC-IIA from both MHC-IIB and MHC-I (3, 19). From this point of view, sprint training might be similar to the above-mentioned condition of intermittent high-frequency stimulation (6, 22).

With regard to the coupling between expression and function, we have recently demonstrated that single fibers containing a specific MHC isoform have the same V₀ irrespective of whether they originate from a fast or a slow contracting muscle (24). We expect that the same holds true for fibers containing the same MHC isoform but isolated from muscles exposed to different levels of stimulation or mechanical activity. The point is questionable because there is some evidence that the coupling between MHC isoform and contractile characteristics may be altered by a change in activity (20, 35, 36).

Human training may take many forms; sprint training might be considered to be the most likely condition in which increases in speed, either through a shift in fiber-type distribution or through an altered coupling between fiber type and speed of shortening, might contribute to increased power generation, in addition to
the increases in strength. In the present study our purpose was to test this possibility by determining whether high-intensity, short-duration sprint training could induce 1) an uncoupling between MHC expression and contractile properties of single fibers tested in vitro, 2) a change in the relative distribution of the different MHC isoforms within whole muscles, and 3) changes in the voluntary and electrically evoked contractile properties of whole muscles measured in vivo.

**METHODS**

Subjects. Seven male subjects (age 22 ± 2 yr, height 178 ± 7 cm, body mass 72 ± 8 kg) took part in the study. Another four male subjects (age 27 ± 6 yr, height 181 ± 2 cm, body mass 76 ± 7 kg) acted as control subjects. All subjects were recreationally active at the time of the study, but none were highly trained. All subjects gave written informed consent to participate in the study, which was approved by the ethics committee of the Karolinska Institute.

Muscle biopsy. Needle biopsies (8) were obtained from the midportion of the vastus lateralis and from the soleus muscle before and within 2–4 days of completion of the final training session. The biopsy was divided into three parts. The first, for histochemical analyses, was mounted in an embedding medium (tissue-tek) and frozen in isopentane precooled by liquid nitrogen. The two remaining parts were immediately frozen in liquid nitrogen and then homogenized (25) for electrophoretic determination of MHC composition or used for single-fiber dissection and determination of contractile properties.

Histochemical analysis. Serial transverse sections were cut in a cryostat at −20°C, mounted on slides, and stained for myofibrillar adenosinetriphosphatase (ATPase) (30). After preincubation at pH 10.3, 4.6, and 4.3, the fibers were classified as type I, type IIA, and type IIB (12). In each biopsy, 220–1,112 fibers were counted. Muscle fiber areas were measured by planimetry from NADH-tetrazolium reductase-stained sections (29) photographically reproduced at a known magnification. Twenty fibers of each type were subjectively rated as representative of the entire cross section. Three soleus biopsies, two from subjects in the training group and one from a control subject, suffered from artifacts of the freezing process, preventing the measurement of fiber cross-sectional area.

Electrophoresis. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis was performed by using 6% polyacrylamide slab gels as previously described (14, 15) to identify MHC isoforms. Gels were silver stained, and three bands were separated, corresponding to MHC-I, MHC-IIA, and MHC-IIB isoforms, the relative proportions of which were determined through densitometric analysis (GS300, Hoefer Scientific Instruments, San Francisco, CA). Examples of electrophoretic separation of MHC isoforms and of densitometric tracings are shown in Fig. 1.

Single-fiber contractile measurements. This method for determination of single-fiber contractile parameters has been previously described in detail (11, 24). Briefly, muscle biopsies were thawed and divided into small bundles of fibers while immersed in ice-cold skinning solution. Skinning, relaxing, and activating solutions were prepared as described previously (11). Single fibers were separated under a stereomicroscope (Wild, ×10–40 magnification) while immersed in skinning solution. Short segments of single fibers (1.5–2 mm long) were attached by aluminum foil clips to a force transducer (AE 801 Aksjeselskapet Mikroelektronik, Horten, Norway) at one end and to an electronic force stirrer (Wild, Ling Dynamic System, Royston, UK) driven by a feedback circuit at the other. The apparatus was mounted on the stage of an inverted microscope (Axiovert 10, Zeiss). Sarcomere length was measured optically at ×320 magnification as was fiber diameter, which was measured at three points along the length of the fiber. While the fiber segment was immersed in relaxing solution, sarcomere length was set at 2.3 and 2.5 µm by adjusting overall segment length. Fibers that could not be stretched to 2.3 µm without a large rise in resting tension were discarded. Segment length was measured by using a stereomicroscope fitted over the apparatus at ×40 magnification. The fiber was then activated by quickly moving it from relaxing to activating solution, and five instantaneous length changes (slacks) were performed, with an amplitude ranging from 5 to 14% of the resting length. The fiber was then removed and placed in solution (27) for subsequent electrophoretic determination of MHC content. V0 was obtained from the slope of the linear regression between the time taken to take up the slack (x-axis) and the amount of shortening imposed (y-axis) (17) and was expressed in fiber segment lengths per second (FL/s). Rates of tension redevelopment were measured by linear interpolation over the first 30 ms of tension rise after the beginning of tension redevelopment that followed a release of 10% segment length. These rates were normalized to the final level of isometric tension reached (P0). All measurements were made at 12°C. At least three fibers were measured from each biopsy. No fibers from the control subjects were analyzed for single-fiber contractile properties.

Whole muscle contractile properties. The contractile properties of the knee extensor and the plantar flexor muscles were determined as described previously (24). Briefly, for the knee extensors the subjects sat upright in a rigid chair with the thigh horizontal and the knee flexed to 90°. A steel brace around the subjects’ lower leg transmitted force resulting from knee extension to a strain-gauge load cell. Stimulating electrodes were placed over the belly of the quadriceps femoris muscles. For the plantar flexors the subjects were seated in the upright position with the thigh horizontal, the knee flexed, and the ankle placed at an 85° angle. The foot rested on a plate with a strain gauge mounted 0.3 m from the axis of rotation of the ankle joint. Stimulating electrodes were placed over the belly of the soleus and the heads of the gastrocnemius. Contractions were evoked by using direct current square-wave pulses of 0.1 ms. Starting at subthreshold levels, voltage was increased in a stepwise manner until no further increases in torque were observed. Maximal twitch contractions (1 Hz) were evoked for measurement of torque, time to peak torque (TPT), and half relaxation time. Maximal tetanic responses were evoked during 2-s tetani at 10 (P0,10).
20 (P<sub>o20</sub>), 50 (P<sub>o50</sub>), and 100 Hz (P<sub>o100</sub>). The ratio of the maximal torques at 20 and 50 Hz (P<sub>o20</sub>:P<sub>o50</sub>) was determined, and at 50 Hz the maximal rate of torque development and maximal rate of relaxation were determined from the slope of the tangent of the steepest portion of each phase and were expressed as percent P<sub>o</sub> per millisecond. Maximum voluntary isometric torque was determined from three maximal voluntary efforts. A test of fatigue resistance was also performed, in which the muscle was stimulated for 300 ms (20 Hz for plantar flexors, 40 Hz for knee extensors) once per second for 2 min. A fatigue index (FI) was then calculated as the force generated on the last three contractions, expressed as a percentage of that generated on the first three.

Cycling performance testing. Sprint cycle performance was assessed by measuring the peak pedal frequency [crank – revolutions/min (rpm)] that could be achieved during a 3-s sprint performed from a standardized stationary start on a cycle ergometer fitted with toe clips and rounded handlebars (Wingate ergometer, Cardionics) (see Ref. 7) with a flywheel resistance of 4.5, 6.0, and 7.5% body weight, respectively. Crank rpm was measured by an on-line computer system, in which a photoelectric sensor mounted on the crank monitored 10 points/revolution. For testing, two sprints separated by 45 s of recovery were performed at each of the three above-mentioned flywheel resistances from a standardized stationary starting position. A 3-min rest period was allowed between each pair of sprints. The highest crank rpm obtained at each workload was used in the analysis. All subjects were habituated to this test procedure on two separate visits to the laboratory before the pretraining test and were verbally encouraged throughout. Each training and testing session was preceded by a standardized warmup.

Training. Training consisted of four sessions of sprint cycling per week for 6 wk on the same cycle ergometer. A prepared audiotaape was played during the session, indicating, through a series of auditory tones, the start and end of each sprint. Each session comprised three sets of 3-s maximal sprints starting from a stationary position, with a 30-s recovery. The following considerations were the basis of the choice of training protocol. Short intermittent high-frequency bursts of activity are the most likely to induce a slow-to-fast transition in MHC 

Type I and type II fibers combined) to increase after training (Fig. 5). The mean size of type I and type II fibers was not significantly different in post- compared with pretraining preparations from either muscle group. However, there was a tendency for mean fiber size (type I and type II fibers combined) to increase after training both in vastus lateralis (+7%, not significant) and soleus (+4%, not significant) muscles (Table 2).

RESULTS

Whole muscle performance. Crank rpm measured during a 3-s sprint significantly increased at each of the three frictional resistances tested (+5.9% at a resistance of 4.5% body wt, +7.1% at 6.0% body wt, +9.3% at 7.5% body wt; Fig. 2A). No changes were observed in the control subjects (Fig. 2B). This demonstrates that sprint training was effective in improving power generation during cycling.

With regard to whole muscle isometric strength, the knee extensor muscles showed significant increases in electrically evoked torque at 20 (+10%), 50 (+8%), and 100 Hz (+14%) and in maximum voluntary torque production (7%) (Fig. 3A). TPT was significantly longer after training (Table 1), but there were no other significant changes in the non-torque-dependent contractile parameters (half relaxation time, P<sub>o20</sub>:P<sub>o50</sub>, maximal rate of torque development at 50 Hz, maximal relaxation rate at 50 Hz, FI) in either trained or nontrained subjects. In the plantar flexors (Fig. 3C), isometric torque also was significantly increased after training by 11% at 20, 50, and 100 Hz and by 16% in maximal voluntary contractions. There were no changes in the non-torque-dependent contractile properties in the plantar flexors (Table 1). No changes in any whole muscle contractile property were observed in the control subjects (Fig. 3B and D).

Muscle composition. In terms of the distribution of MHC isoforms determined by using gel electrophoresis, the soleus was found to be dominated by MHC-I with no detectable MHC-IIB (Figs. 1 and 4). The vastus lateralis muscle was composed of a more even mix of MHC-I and MHC-II, with four of the seven trained subjects possessing MHC-IIB (Figs. 1 and 4). In three of these subjects MHC-IIB composition decreased, but it increased in the fourth subject. Statistical analysis showed that there were no changes in the distribution of the different isoforms in the soleus, or in the vastus lateralis in either trained or nontrained subjects. This was also the case when fiber composition was determined on the basis of ATPase histochemistry and fiber counting (Fig. 5). The mean size of type I and type II fibers was not significantly different in post- compared with pretraining preparations from either muscle group. However, there was a tendency for mean fiber size (type I and type II fibers combined) to increase after training both in vastus lateralis (+7%, not significant) and soleus (+4%, not significant) muscles (Table 2).
have recently reported (24), no difference is present in \( V_o \) between fibers expressing the same MHC and those isolated from different muscles. The fibers from soleus and vastus lateralis muscles were therefore pooled. This increased the numbers of fibers used for statistical comparison. Single fibers expressing the MHC-I isoform were found to have values for \( V_o \) significantly (not indicated in Table 3) lower than those expressing MHC-IIA or MHC-IIA together with MHC-IIB isoforms. After training, \( V_o \) of fibers expressing only MHC-I or MHC-IIA isoforms was unchanged. A few fibers contained MHC-I and MHC-IIA and generally exhibited \( V_o \) values between those of the pure MHC-I or MHC-IIA fibers. These hybrid fibers appeared to decrease in \( V_o \) after training. However, this can be explained by differences in the relative content of MHC-IIA in these fibers. In pretraining fibers the amount of MHC-IIA averaged 45 ± 14% of the fiber, whereas in the posttraining fibers it only averaged 17 ± 1%. Only fibers from the vastus lateralis were found to express both MHC-IIA and MHC-IIB. These fibers exhibited higher \( V_o \) values, higher than the fibers just expressing MHC-IIA, but were unchanged after training. No fibers were found to exhibit only MHC-IIB, either pre- or posttraining.

Faster rates of tension development were observed in fibers expressing MHC-IIA or MHC-IIA/MHC-IIB isoforms when compared with MHC-I fibers, but no
changes were observed after the training regimen, except in the few fibers that expressed MHC-I and MHC-IIA, a finding that again reflects a difference in the relative occurrence of the two isoforms. No changes in single-fiber cross-sectional area (data not shown) were observed after training. In terms of specific tension, no effect of the training was seen in the MHC-I fibers or the MHC-IIA fibers, although there was a nonsignificant tendency for it to increase in MHC-IIA fibers after training.

DISCUSSION

The results obtained in this study show that 6 wk of sprint training produced a significant increase in whole muscle performance (peak crank rpm and isometric

Table 1. Whole muscle contractile properties

<table>
<thead>
<tr>
<th>Group</th>
<th>TPT, ms</th>
<th>RT ‰, ms</th>
<th>TCT, ms</th>
<th>P20/50</th>
<th>dP50,%Pois</th>
<th>dP50,%Pois</th>
<th>FI</th>
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<tbody>
<tr>
<td>Knee extensors</td>
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<tr>
<td>Trained subjects</td>
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<tr>
<td>Pre</td>
<td>81 ± 4</td>
<td>67 ± 4</td>
<td>148 ± 7</td>
<td>0.80 ± 0.02</td>
<td>0.99 ± 0.08</td>
<td>1.35 ± 0.11</td>
<td>0.53 ± 0.03</td>
</tr>
<tr>
<td>Post</td>
<td>85 ± 3*</td>
<td>61 ± 3</td>
<td>146 ± 5</td>
<td>0.81 ± 0.03</td>
<td>0.94 ± 0.05</td>
<td>1.48 ± 0.05</td>
<td>0.55 ± 0.03</td>
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<tr>
<td>Control subjects</td>
<td></td>
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<tr>
<td>Pre</td>
<td>79 ± 4</td>
<td>76 ± 6</td>
<td>155 ± 6</td>
<td>0.79 ± 0.04</td>
<td>1.07 ± 0.07</td>
<td>1.40 ± 0.04</td>
<td>0.60 ± 0.07</td>
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<tr>
<td>Post</td>
<td>80 ± 4</td>
<td>71 ± 4</td>
<td>151 ± 6</td>
<td>0.83 ± 0.02</td>
<td>1.04 ± 0.07</td>
<td>1.41 ± 0.11</td>
<td>0.63 ± 0.06</td>
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<tr>
<td>Plantar flexors</td>
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<tr>
<td>Trained subjects</td>
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<tr>
<td>Pre</td>
<td>127 ± 4</td>
<td>105 ± 5</td>
<td>232 ± 8</td>
<td>0.82 ± 0.02</td>
<td>0.37 ± 0.02</td>
<td>1.23 ± 0.07</td>
<td>0.89 ± 0.02</td>
</tr>
<tr>
<td>Post</td>
<td>127 ± 4</td>
<td>99 ± 5</td>
<td>226 ± 3</td>
<td>0.82 ± 0.03</td>
<td>0.39 ± 0.01</td>
<td>1.17 ± 0.04</td>
<td>0.91 ± 0.02</td>
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<tr>
<td>Control subjects</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Pre</td>
<td>116 ± 6</td>
<td>93 ± 8</td>
<td>192 ± 11</td>
<td>0.85 ± 0.01</td>
<td>0.38 ± 0.02</td>
<td>1.20 ± 0.05</td>
<td>0.84 ± 0.06</td>
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<tr>
<td>Post</td>
<td>117 ± 7</td>
<td>94 ± 5</td>
<td>187 ± 5</td>
<td>0.83 ± 0.01</td>
<td>0.39 ± 0.02</td>
<td>1.18 ± 0.06</td>
<td>0.86 ± 0.03</td>
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</tbody>
</table>

Values are means ± SE; n = 7 sprint-trained subjects and 4 control subjects. Pre and Post, before and after training, respectively; TPT, time to peak torque; RT ‰, half relaxation time; TCT, total contraction time; P20/50, ratio of torque at 20 (P20) and 50 Hz (P50); dP50, maximal rate of torque development during tetanus at 50 Hz; dP50, maximal rate of relaxation from a 50-Hz tetanus; FI, fatigue index. Significantly different from pretraining. *P < 0.05.

Fig. 4. Relative distribution of MHC isoforms Pre (open bars) and Post (solid bars) 6 wk of sprint training. A and B: VL in trained and control subjects, respectively. C and D: S in trained and control subjects, respectively.
torque development) without any significant variation in MHC isof orm or fiber-type distribution or change in single-fiber contractile characteristics. Specifically, there was no shift toward a greater expression of fast MHC isoforms and there was no increase in muscle fiber \( V_o \). This indicates that the improvement in cycling performance resulted principally from an increase in strength.

The finding that kinetic parameters such as \( V_o \) and rate of tension rise in single fibers were unaffected by the training regimen is of interest because it supports the suggestion of a strict coupling between MHC content and contractile properties (24, 28). However, there is other evidence to suggest that this might not always be the case. In an earlier study Fitts et al. (20) reported that \( V_o \) values of MHC-I fibers from the deltoid were higher in swimmers after 10 wk of swim training (1.03 vs. 0.86 FL/s at 15°C) when compared with control subjects, although no further increase was observed after a further more intensive 10 days of training (1.10 FL/s). These values are considerably higher than reported here or elsewhere (24, 28, 35) for \( V_o \) of MHC-I fibers from human muscle. Furthermore, in a more recent cross-sectional study Widrick et al. (35) also reported that \( V_o \) values of MHC-I fibers were significantly faster (0.51 vs. 0.43 FL/s) in endurance-trained master athletes aged 42 yr when compared with nonactive controls. Interestingly, the force-velocity properties of these fibers and maximal velocity as extrapolated from Hill’s equation were not different (36). In contrast to MHC-I fibers, MHC-II fibers from the deltoid showed a reduction in \( V_o \) with the 10 days of further intensive swim training. In the above-mentioned study, however, MHC-IIA and MHC-IIB were not separated. MHC-IIB, which is more highly expressed by muscle fibers in the upper, rather than lower, limb appears to be particularly sensitive, seemingly “switching” to MHC-IIA with increased usage (1, 3, 5). A loss of MHC-IIB would have a marked effect on \( V_o \), as evidenced in the present study by the higher \( V_o \) values observed in those fibers that coexpressed MHC-IIB and MHC-IIA. Indeed, Larsson and Moss (28) observed that \( V_o \) increased as an exponential function of MHC-IIB content in these hybrid fibers, and thus any reduction in MHC-IIB content that occurs as a result of increased usage will likely have a slowing effect on \( V_o \).

Of the many studies that have examined fiber composition changes with different types of training by using the histochemical determination of myosin ATPase, the most common observation is a reduction in the relative number of type IIB fibers (see Ref. 23 for review). However, in the present study no significant change in the proportion of type IIB fibers was observed. Histo-

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Table 2. Mean fiber area

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre</th>
<th>Post</th>
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<tbody>
<tr>
<td>Vastus lateralis</td>
<td></td>
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<tr>
<td>Trained subjects</td>
<td>5,144 ± 375</td>
<td>5,489 ± 284</td>
</tr>
<tr>
<td>Control subjects</td>
<td>5,050 ± 367</td>
<td>5,185 ± 275</td>
</tr>
<tr>
<td>Soleus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trained subjects</td>
<td>6,296 ± 418</td>
<td>6,531 ± 576</td>
</tr>
<tr>
<td>Control subjects</td>
<td>7,071 ± 299</td>
<td>6,119 ± 292</td>
</tr>
</tbody>
</table>

Values are means ± SE in µm²; n = 5 trained subjects and 3 control subjects for soleus.
chemically determined type IIB fibers have been shown to be primarily hybrid fibers, composed of MHC-I and MHC-IIB isoforms (33). In agreement with the previously published histochemical data, Adams et al. (1) reported a reduction in MHC-IIB content after 3 mo of strength training, as determined by using gel electrophoresis. Similar findings were observed after general sprint training (3), as expressed by a decrease in the number of individual fibers that expressed MHC-IIB.

Indeed, it has been suggested that MHC-IIB is the "default" gene (21), the expression of which is increased with disuse and "switches" to MHC-IIB when activity is increased (5). In the present study MHC-IIB was detected in the vastus lateralis in only four of the subjects, an observation that possibly reflects a relatively high level of habitual physical activity in our subjects, although none was sprint trained or strength trained. In three of these subjects MHC-IIB decreased after training but increased in the fourth subject, leading to no overall statistically significant decrease.

There are a number of possible explanations for MHC-IIB not decreasing in all of the subjects. One possibility might be that the 6 wk of training, or even the 3-s exercise bouts, were of insufficient duration. Alternatively, it is possible that there was poor recruitment of the high-threshold type IIB motor units during this type of exercise. This seems unlikely because the threshold for recruitment falls with increasing speed of movement (16). A final possibility may be that resistance or mechanical load is more important than rate of motor unit firing or velocity of contraction for inducing myosin switching, hence the decline in MHC-IIB reported by others with short-duration, but high-resistance, contractions (1).

In addition to a reduction in MHC-IIB, an increase in the number of single fibers expressing MHC-I and histochemically determined type IIA fibers (19) has been shown to occur in response to "sprint" training, and this seemed to be at the expense not only of MHC-IIB isoforms and type IIB fibers but also of MHC-I and type I fibers. It has been suggested that sprint training may induce a bidirectional change toward MHC-I/A at the expense of both MHC-I and MHC-IIB (4, 20). However, no statistically significant changes in MHC-I and MHC-I/A content and in the relative number of type I and IIA fibers were observed in the present study.

In the knee extensors whole muscle twitch, TPT, a marker of contractile speed, was significantly increased after training, albeit not by a great amount (+6%). A similar prolongation has been observed previously in the elbow extensors of elderly individuals undergoing strength training (32), but these data contrast with a decrease in TPT reported in young individuals undergoing isometric strength training of the plantar flexors (2). With no significant changes in myosin expression, it is possible that the small increase in TPT is a reflection of changes in excitation-contraction coupling, specifically the kinetics of Ca²⁺ release and reuptake from the sarcoplasmic reticulum (26). The results of chronic stimulation experiments on animal muscle suggest that the excitation-contraction coupling mechanism is the first to adapt to increased activity and precedes changes in MHC expression and shortening properties (31). Thus the observed prolongation of TPT would suggest an initial shift from faster toward slower fiber types.

With no change in single-fiber kinetics or in MHC distribution occurring in either muscle, the increased pedal frequency observed during cycling cannot be attributable to muscles that have faster shortening properties. The higher pedal frequencies are thus more likely to be attributable both to neural adaptations to the cycling process itself and to stronger leg muscles. Both the plantar flexor and the knee extensor muscles increased their isometric torque production. This was the case when the muscle was activated both voluntarily and involuntarily through direct electrical stimulation. This may be explained, at least in part, by a tendency toward both an increased mean fiber area (7% in the vastus lateralis and 4% in the soleus) and an increase in the specific tension of the MHC-I fibers. The latter finding must be treated with some caution, however, given the relatively small number of these MHC-IIA fibers and the fact that the absolute values, ~40 kN/m², are still weak relative to other reported values of 60 (11) and 200 kN/m² (28). In the latter study, however, adjustments were made to correct for fiber swelling because of the skinning procedure, and measurements were carried out at a higher temperature, resulting in values closer to the specific tensions of mammalian muscle in physiological conditions (13).

In conclusion, the present study is the first to determine single-fiber and electrically evoked whole muscle contractile properties in response to sprint training in humans. The results showed that no increase in shortening speed of single fibers and no shift toward fast MHC isoforms occurred. Those muscle fibers that expressed the same MHC possessed similar values for \( V_o \) before and after training. The training was also without effect on MHC distribution in either the vastus

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### Table 3. Single-fiber contractile properties

<table>
<thead>
<tr>
<th>Fiber Type</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHC-I</td>
<td>0.28±0.02</td>
<td>0.27±0.02</td>
</tr>
<tr>
<td>MHC-I/IIA</td>
<td>0.87±0.26</td>
<td>0.41±0.09</td>
</tr>
<tr>
<td>MHC-IIA</td>
<td>1.19±0.12</td>
<td>1.28±0.20</td>
</tr>
<tr>
<td>MHC-IIA/IIB</td>
<td>1.91±0.10</td>
<td>1.52±0.41</td>
</tr>
</tbody>
</table>

Values are means ± SE of 46 and 43 myosin heavy chain (MHC)-I, 4 and 4 MHC-I/IIA, 14 and 8 MHC-IIA, 8 and 4 MHC-IIA/IIB pre- and posttraining fibers, respectively. Values for maximum shortening velocity (\( V_o \)), rate of tension redevelopment (\( dp/dt \)), and specific tension in chemically skinned and maximally activated fibers at 12°C are shown. Fibers from soleus and vastus lateralis muscles were pooled.
lateralis or soleus muscles. However, the maximum pedal frequency at three different flywheel resistances increased significantly. The increase in the torque-producing ability of both the plantar flexor and knee extensor muscles was the most likely determinant of the improvement in cycling performance with this sprint training protocol.

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