Single muscle fiber adaptations with marathon training

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Trappe, Scott, Matthew Harber, Andrew Creer, Philip Gallagher, Dustin Sivlka, Kiril Minchev, and David Whitsett. Single muscle fiber adaptations with marathon training. J Appl Physiol 101: 721–727, 2006. The purpose of this investigation was to characterize the effects of marathon training on single muscle fiber contractile function in a group of recreational runners. Muscle biopsy samples were obtained from the gastrocnemius muscle of seven individuals (22 ± 1 yr, 177 ± 3 cm, and 68 ± 2 kg) before, after 13 wk of training, and after 3 wk of taper. Slow-twitch myosin heavy chain (MHC I) and fast-twitch (MHC IIa) muscle fibers were analyzed for size, strength (Po), speed (Vo), and power. The run training program led to the successful completion of a marathon (range 3 h 56 min to 5 h 35 min). Oxygen uptake during submaximal running and citrate synthase activity were increased (P < 0.05) with the training program. Muscle fiber size declined (P < 0.05) by ~20% in both fiber types after training. Po was maintained in both fiber types with training and increased (P < 0.05) by 18% in the MHC IIa fibers after taper. This resulted in >60% increase (P < 0.05) in force per cross-sectional area in both fiber types. Fiber Vo increased (P < 0.05) by 28% in MHC I fibers with training and was unchanged in MHC IIa fibers. Peak power increased (P < 0.05) in MHC I and IIa fibers after training with a further increase (P < 0.05) in MHC IIa fiber power after taper. These data show that marathon training decreased slow-twitch and fast-twitch muscle fiber size but that it maintained or improved the functional profile of these fibers. A taper period before the marathon improved the functional profile of the muscle, which was targeted to the fast-twitch muscle fibers.

METHODS

Subjects

Seven subjects (4 men; 3 women) completed all phases of the testing protocols and the marathon. These subjects were 22 ± 1 yr, 177 ± 3 cm, and 68 ± 2 kg. All of the participants were recreationally active with limited running experience. None of the participants had ever attempted to run a marathon before this study. Before data collection and any training, the Institutional Review Board approved the study design and testing procedures. All subjects provided their written consent before participation.

Training Plan and Testing Schedule

All subjects were part of a university class that was designed to physically and mentally prepare them to train for and complete a 42.2-km (26.2 miles) marathon. The 16-wk run training plan is outlined in Table 1. The training program was divided into two phases: a 13-wk training period followed by a 3-wk taper period. Class participants had a gradual increase in running volume (~10% per week) over the 13-wk training period so that overall volume increased by ~140% compared with week 1. During the 3-wk taper, running volume was gradually decreased each week. Compared with the last week of training (week 13), running volume was reduced by 25% in week 14, 47% in week 15, and 80% the week before the marathon. This training program was designed to provide adequate fitness and recovery to minimize the likelihood of injury to ensure a successful completion of the marathon. This 4 days/wk program has

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be previously shown to be as effective as a 6 days/wk program for novice runners (12).

The runners were tested on three occasions: before the 16-wk training plan, after 13 wk of run training, and after 3 wk of taper and marathon. Single muscle fiber physiology experiments and oxidative enzyme activity were conducted at all three time points. Single muscle fiber MHC isoform experiments were conducted before the training program and after the taper. Treadmill testing for maximal oxygen uptake (VO_2 max) was conducted at the beginning of the training program and after the taper. No treadmill test was subsequently increased by 2% every 2 min until exhaustion. VO_2 max around 13 or more for a given stage, the grade on the treadmill was increased in 3-min stages to paces that were tolerable for each subject. Throughout these stages, the subjects were asked to rate their perception of effort using a Borg scale (2). When the subject’s rating was around 13 or more for a given stage, the grade on the treadmill was subsequently increased by 2% every 2 min until exhaustion. VO_2 max was confirmed by a leveling of oxygen uptake and a respiratory exchange ratio >1.10.

During the test, oxygen uptake was measured at 30-s intervals using indirect calorimetry via an automated open-circuit system that incorporated a dry-gas meter (Rayfield Equipment, Waitsfield, VT), 3-liter mixing chamber, and electronic oxygen and carbon dioxide analyzers (Ametek S-3A/II and CD-3A, respectively, Applied Electrochemistry, AEI Technologies, Pittsburgh, PA), which were interfaced to a PC computer. Both analyzers were calibrated with standardized gases before each test.

**Muscle Biopsy**

A muscle biopsy (1) from the lateral gastrocnemius was obtained from each subject at all three time points. The pretraining muscle biopsies were obtained before training began, the second muscle biopsy (after 13 wk of training) was obtained in the first part of the week on a day with no run training, and the final muscle biopsy was obtained 2–3 days after the marathon.

The muscle biopsy was divided into 1) a muscle bundle for single muscle fiber physiology experiments that was placed in cold freezing solution (see below) and stored at −20°C until analysis, 2) a muscle bundle for the single fiber MHC experiments that was placed and stored in skinning solution, and 3) a muscle bundle for citrate synthase activity that was quickly frozen in liquid nitrogen and stored at −190°C until analysis.

**Oxidative Enzyme Activity**

Citrate synthase activity was measured from a ~10-mg portion of muscle through the reduction of 5,5’-dithiobis(2-nitrobenzoic acid) by the release of CoA-SH in the cleaving of acetyl-CoA (30).

**Skinning, Relaxing, and Activating Solutions**

The skinning solution contained (in mM) 125 K-propionate, 2.0 EGTA, 4.0 ATP, 1.0 MgCl_2, 20.0 imidazole (pH 7.0), and 50% (vol/vol) glycerol. The compositions of the relaxing and activating solutions were calculated as previously described (14). These solutions were adjusted for temperature, pH, and ionic strength using stability constants (17). Each solution contained (in mM) 7.0 EGTA, 20.0 imidazole, 14.5 creatine phosphate, 1.0 free Mg\(^{2+}\), 4.0 free MgATP, and KCl and KOH to produce and ionize strength of 180 mM and a pH of 7.0. The relaxing and activating solutions had a free Ca\(^{2+}\) concentration of 9.0 and pCa 4.5, respectively.

**Single Muscle Fiber Physiology Experiments**

On the day of an experiment, a 2- to 3-mm muscle fiber segment was isolated from a muscle bundle and transferred to an experimental chamber filled with relaxing solution where the ends were securely fastened between a force transducer (model 400A, Cambridge Technology, Watertown, MA) and a direct current torque motor (model 308B, Cambridge Technology) as described by Moss (26). The instrumentation was arranged so that the muscle fiber could be rapidly transferred back and forth between experimental chambers filled with relaxing or activating solutions. The apparatus was mounted on a microscope (model BH-2, Olympus, Tokyo, Japan) allowing the fiber to be viewed (×800) during an experiment. With the use of a calibrated eyepiece micrometer, sarcomeres along the isolated muscle segment length were adjusted to 2.5 μm. All single muscle fiber experiments were performed at 15°C.

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### Table 1. Run training plan

<table>
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<th>Wednesday</th>
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<th>Saturday</th>
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Values are distances in kilometers with miles in parentheses.
Unamplified Po and length signals were sent to a digital oscilloscope (model 310, Nicolet Madison, WI) enabling muscle fiber performance to be monitored throughout each experiment. Analog force and position signals were amplified (model 300-DIF2 dual-differential amplifier, Positron Development, Inglewood, CA), converted to digital signals (National Instruments) and transferred to a computer for analysis using customized software. Servomotor arm and isotonic force clamps were controlled using a computer-interfaced force-position controller (model 300-FC1 force controller, Positron Development).

For each single muscle fiber experiment, a fiber with a compliance [calculated as fiber length (FL) divided by y-intercept] >10% and/or a decrease in peak force (Po) of >10% was discarded and not used for analysis. The within-fiber test/retest of a single muscle fiber in our laboratory for the measurements of diameter, Po, contractile velocity [maximal unloaded shortening velocity (V\textsubscript{o}u)], and power were <1%. The coefficient of variation for the force transducer and servomechanical lever mechanism during the 16-wk period in which we unloaded shortening. Fiber analysis. The within-fiber test/retest of a single muscle fiber in our laboratory for the measurements of diameter, Po, contractile velocity [maximal unloaded shortening velocity (V\textsubscript{o}u)], and power were <1%. The coefficient of variation for the force transducer and servomechanical lever mechanism during the 16-wk period in which we examined single muscle fiber function was 0.3 and 0.5%, respectively.

**Single Muscle Fiber Data Analysis**

Individual muscle fibers were analyzed for diameter, Po, Vo, and power characteristics. Detailed descriptions and illustrations of these procedures have been presented in our laboratory’s previous work (32, 35).

*Single fiber Po.* A video camera (model DXC-107A, Sony CCD-IRIS, Tokyo, Japan) connected to the microscope and interfaced to a computer allowed viewing on a computer monitor. Fiber diameter was determined from a captured computer image taken with the fiber briefly suspended in air (<5 s). Fiber width (diameter) was determined at three points along the segment length of the image using public domain software (NIH Image version 1.61) and averaged to provide a mean diameter measurement.

*Single fiber Po.* The outputs of the force and position transducers were amplified and sent to a computer via a Lab-PC+ 12-bit data-acquisition board (National Instruments). Resting force was monitored and then the fiber was maximally activated in pCa 4.5 solution. Po was determined in each fiber by subtraction of baseline force from Po.

*Single fiber Vo.* Fiber Vo was measured by the slack test technique (13). Each fiber was maximally activated and then rapidly released to a shorter length, such that force fell to baseline. The fiber shortened, taking up the slack, after which force redeveloped. The fiber was then placed in relaxing solution and returned to its original length. Four to six different length steps (each ±15% of FL) were used for each fiber with the slack distance plotted as a function of the duration of unloaded shortening. Fiber Vo (FL/s) was calculated by dividing the slope of the fitted line and normalized to FL and a sarcomere spacing of 2.5 μm.

*Single fiber power.* Submaximal isotonic load clamps were performed on each fiber for determination of force-power parameters. Each fiber segment was fully activated and then subjected to a series of isotonic load steps. This procedure was performed at various loads with a total of 15–18 isotonic contractions. Po and Vo data points derived from the isotonic contractions were fit using the Hill equation (20). Individual experiments in which r\textsuperscript{2} was ≥0.98 were accepted. Fiber power (μN·FL/s) was defined as the product of force (μN) and shortening velocity (FL/s). Normalized power (watts generated per unit fiber volume; W/l) was defined as the product of normalized force (fiber force per cross-sectional area) and shortening velocity.

**Single Muscle Fiber MHC Isoform Analysis**

The MHC isoform profile for each fiber was determined by isolating individual fibers under a microscope and performing SDS-PAGE (SE 600 series, Hoefer, San Francisco, CA) analysis (40). The fibers were dissected in relaxing solution and then solubilized in 80 μl of 1% SDS sample buffer [10% SDS, 6 mg/ml EDTA, 0.06 M Tris (pH 6.8), 2 mg/ml bromphenol blue, 15% glycerol, and 5% β-mercaptoethanol]. These samples were stored at −80°C until analyzed for MHC content. Samples were loaded on a 3.5% loading and a 5% separating gel, and run 12 h at 4°C. The gels were silver stained (16), allowing for the MHC isoform profile (I, I/IIa, I/IIa/IIx, IIa, IIa/IIx, IIx) for each individual fiber to be determined. This same procedure was used to determine the MHC profile for a single muscle fiber following the physiology experiments.

**Statistical Analysis**

A comparison of changes in Vo\textsubscript{o} max and MHC profile was performed using a paired t-test. Citrate synthase activity was analyzed using a repeated-measures ANOVA. Single muscle fiber physiology parameters were analyzed using a univariate ANOVA with nested means. For this analysis, the number of fibers studied for a particular individual were nested to represent a mean for fiber diameter, Po, per cross-sectional area, Vo, peak power, and normalized power of both MHC I and MHC IIa fibers. Significance was set at P < 0.05, and a Student-Newman-Keuls post hoc test was performed when significance was noted. Data are presented as means ± SE.

**RESULTS**

**Running Performance**

All subjects completed the marathon distance. The average time for the runners to complete the marathon distance was 4 h 54 min (range = 3 h 56 min to 5 h 35 min).

**Treadmill Data**

Body weight was similar at each treadmill testing session (68 ± 2 vs. 67 ± 3 kg). There was a trend for an increase in absolute Vo\textsubscript{o} max from before to after run training (3.37 vs. 3.50 l/min; P = 0.09), whereas relative Vo\textsubscript{o} max (49.5 vs. 52.0 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) was unchanged. Maximal ventilation (93.7 vs. 97.0 l/min) and maximal heart rate (197 vs. 198 beats/min) were similar before to after run training.

Absolute (2.43 vs. 2.28 l/min) and relative (36.0 vs. 33.6 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) oxygen consumption at a submaximal running speed of 9.65 km/h was lower (P < 0.05) after the training program. No changes in heart rate (162 vs. 161 beats/min) or ventilation (48.4 vs. 46.5 l/min) were observed during submaximal running.

**Oxidative Enzyme Activity**

Citrate synthase activity increased by 37% (19.2 ± 1.4 vs. 26.3 ± 2.246 mol·kg\textsuperscript{-1}·min\textsuperscript{-1}) oxygen consumption at a submaximal running speed of 9.65 km/h was lower (P < 0.05) after the training program. No changes in citrate synthase activity was observed at the end of training compared with after the taper.

**Single Muscle Fiber MHC Profile**

MHC experiments were performed on 856 single fibers (122 ± 1 per subject) before training and 837 single fibers (120 ± 2 per subject) after taper. A summary of the MHC changes from before to after run training is shown in Fig. 1. The change in MHC composition was an 8% increase (P < 0.05) in MHC I fibers (48 ± 6 to 56 ± 6%), a 5% decrease (P < 0.05) in MHC I/IIa hybrid fibers (7 ± 1 to 2 ± 1%), and a decrease (P < 0.05) in total MHC hybrids (24 ± 7 to 13 ± 4%). No change in the MHC IIa fiber profile (30 ± 5 to 30 ±
power increased \((P < 0.05)\) 16% with training and an additional 26% with taper (overall a 47% increase).

Peak normalized power increased \((P < 0.05)\) by 100% in the MHC I fibers with training and taper. MHC IIa peak normalized power increased \((P < 0.05)\) by 70% with training and by an additional 14% with taper.

Normalized power distribution is shown in Fig. 3. Before any training, 83% of the MHC I fibers had a normalized power below 1.5 W/l compared with 9% after training and <1% after taper. For the MHC IIa fibers, 49% had a normalized power below 5 W/l before training compared with only 9% after taper. In addition, 7% of the MHC IIa fibers had a normalized power above 10 W/l before training compared with 29% after training and 62% after taper.

DISCUSSION

The unique aspect of this project was the study of single muscle fiber contractile function at distinct phases of training in a group of runners preparing for their first marathon. This research highlights the high degree of plasticity among MHC I and IIa muscle fibers with modest improvements in the aerobic potential of these marathon runners. The main findings from this study were that 13 wk of run training led to a decrease in MHC I and IIa muscle fiber diameter but maintained or improved the functional profile of these fibers. Second, with a 3-wk reduced training phase (taper), alterations in the functional profile of the muscle were targeted to the MHC IIa fibers.

Aerobic Adaptations

\(\dot{V}_O_2\) \text{max} was not improved as would be anticipated with marathon training, although there was a trend \((+130 \text{ ml}; \ P = 0.09)\) for an increase. For these novice runners, the improvement in running economy \((-150 \text{ ml})\) appears to be one of the key assets of the training program, which is considered one of the more critical factors for running success in recreational and elite runners (6). The lower submaximal oxygen uptake combined with the small increase in \(\dot{V}_O_2\) \text{max} resulted in a decline in fractional utilization (73% before training compared with 66% after training) at 9.6 km/h, which was similar to the training pace and subsequent marathon run pace for these individuals. The similarity in heart rate with a decline in oxygen uptake
during submaximal running was surprising and suggests that stroke volume was reduced during this effort. The reasons for this are unclear, but they could be related to hydration status or variation in submaximal heart rate (range 151–173 beats/min).

The pretest \( V_{\text{O}_2\text{max}} \) values were more in line with trained recreational runners (19) compared with typical college-age individuals (41), suggesting that their cardiovascular system was reasonably well conditioned at the beginning of the training program. Conversely, the oxidative potential of the muscle (citrate synthase, increase in MHC I fibers) was significantly increased with the training program, suggesting enhanced aerobic potential. These data suggest that the run training program appears to have provided more improvement in the oxidative profile of the muscle compared with cardiovascular system.

**Single Fiber Adaptations To Training**

Before the run training program, single muscle fiber diameter was in the range that has been previously reported for untrained individuals (7, 32, 38), and it was smaller than typically observed in more talented runners (7, 8, 18). Fiber diameter decreased by \( \sim 20\% \) in MHC I and IIa muscle fibers with 13 wk of running, suggesting that fiber diameter was similarly affected in both fiber types. A decrease in muscle fiber diameter would, in theory, allow for a shorter diffusion distance of oxygen and substrates during running. Fiber diameter decreases with endurance run training have been previously reported in collegiate runners during a cross-country season (18) and tend to be smaller after 20 yr of running (36). Despite the reduction in MHC I and IIa fiber diameter, the functional characteristics were unaffected or improved with run training. Typically, alterations in muscle cell strength are proportional to changes in muscle cell size (31, 33, 35, 37). More recently, however, extreme perturbations such as chronic distance running (18), long-term bed rest (23, 28, 34), and immobilization (9) have been shown to uncouple fiber size and \( P_o \). The mechanism for this uncoupling is unknown, although several have been postulated (9, 24, 29).

The elevated \( V_o \) in MHC I fibers coincides with a previous study on masters runners showing that MHC I fiber \( V_o \) was \( \sim 20\% \) higher compared with matched sedentary adults (38). In contrast, our laboratory recently found that MHC I fiber \( V_o \) declined among college runners during a cross-country season (18). Before being tested at the start of the cross-country season, these college runners had been training for several years and performed several weeks of aerobic base running before the study. The pretesting MHC I fiber \( V_o \) values from these athletes (1.66 FL/s) was on the upper end of the range typically observed for human slow-twitch muscle fibers (15). After 8 wk of intense running, MHC I fiber \( V_o \) declined by 21% (to 1.27 FL/s). Thus it could be argued that the weeks of consistent submaximal aerobic training before the start of the cross-country season led to an increase fiber \( V_o \), and when interval training was incorporated, fiber \( V_o \) declined. Taken together, these studies support the notion that distance running

**Table 2. Single muscle fiber \( P_o, P_o/CSA, \) and \( V_o \) of MHC I and IIa muscle fibers**

<table>
<thead>
<tr>
<th></th>
<th>( P_o ), mN</th>
<th>( P_o/CSA ), kN/m²</th>
<th>( V_o ), FL/s</th>
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<td>MHC I</td>
<td></td>
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<tr>
<td>Pre</td>
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<td>87 ± 7</td>
<td>0.99 ± 0.04</td>
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<td>0.55 ± 0.04</td>
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<td>1.27 ± 0.11*</td>
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<tr>
<td>Tapered</td>
<td>0.56 ± 0.04</td>
<td>130 ± 5*</td>
<td>1.22 ± 0.08*</td>
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<td>MHC IIa</td>
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<td>0.61 ± 0.04</td>
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<td>0.72 ± 0.04†</td>
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Values are means ± SE. Pre, pre training; Trained, after 13 wk of run training; Tapered, after 3 wk of taper; MHC, myosin heavy chain; \( P_o \), peak force; CSA, cross-sectional area; \( V_o \), maximal unloaded shortening velocity; FL, fiber lengths. * \( P < 0.05 \) from Pre. † \( P < 0.05 \) from Pre and Trained (both fiber types).

**Table 3. Single muscle fiber peak power and peak normalized power for MHC I and IIa muscle fibers**

<table>
<thead>
<tr>
<th></th>
<th>Peak Power, ( \mu\text{N} \cdot \text{FL} \cdot \text{s}^{-1} )</th>
<th>Normalized Power, W/ℓ</th>
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<td>MHC IIa</td>
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<td>6.2 ± 0.9</td>
<td>32.2 ± 3.0</td>
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<td>Trained</td>
<td>9.7 ± 1.2*</td>
<td>37.5 ± 4.2</td>
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<tr>
<td>Tapered</td>
<td>9.9 ± 0.9*</td>
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Fig. 3. Normalized power distribution of gastrocnemius muscle fibers before training (Pre), after 13 wk of run training (Trained), and after 3 wk of taper (Tapered). A: MHC I muscle fibers. B: MHC IIa muscle fibers.
can alter $V_o$ of MHC I muscle fibers with little effect on MHC IIa muscle fiber contractile speed.

Changes in human single fiber muscle power and normalized power in response to exercise training have been previously shown (18, 31, 35, 37, 39). What is unique about the present study is that the power profile of human skeletal muscle was improved with distance running. Because normalized power accounts for muscle cell diameter, strength, and contractile speed, it provides a functional overview of muscle cell performance. In the present study, normalized power increased by >70% in both MHC I and IIa fibers with 13 wk of running. Independent of fiber type, normalized power ranged from 0.30 to 12.71 W/l before training and from 0.71 to 17.99 W/l after 13 wk of distance running. This continuum shift (as highlighted in Fig. 3) to produce more power for a given fiber size (and fiber type) provides additional insight into muscle alterations that most likely translate to whole muscle performance. These data provide novel information demonstrating that both MHC I and IIa muscle fibers increase their power output with long slow distance running.

**Single Fiber Adaptations To Taper**

After the 3-wk taper period, there was an increase in strength and power of the MHC IIa muscle fibers, with no change in MHC I muscle fiber performance. The improvement in MHC IIa muscle contractile function with tapering is counterintuitive given that long slow distance running is primarily aerobic and would rely on MHC I muscle fibers during this type of physical activity. The functional profile of all fiber types shows that MHC IIa muscle fibers span a much greater functional range and are five to six times more powerful than slow-twitch muscle fibers (3, 32). As a result, minor modifications in MHC IIa muscle fiber mechanics would, in theory, have a much greater impact on whole muscle performance than large changes in MHC I muscle fiber mechanics.

The fiber-specific changes with tapering parallel mechanical improvements in MHC IIa muscle fibers among tapered collegiate swimmers (31) and cyclists (27). Conversely, a similar length taper period in collegiate cross-country runners reduced MHC I muscle fiber diameter, strength, speed, and power, with no alterations in MHC IIa muscle fibers (18). Although these college-level runners reduced their overall running volume, the amount of interval training was increased during the taper. This increased interval training during the taper may explain the decline in single muscle fiber function. Also worth noting, the collegiate runners 8-km performance was not improved at the championship competition after the taper, whereas swimmers who increased fast-twitch muscle fiber contractile function with taper had a 4% improvement in performance. A properly conducted taper ranging from 7 to 21 days typically yields a 2–4% performance gain in swimmers and runners (21, 31). Collectively, these studies highlight the importance that MHC IIa muscle fibers have in fine tuning athletic performance in events ranging from more power-oriented sports such as swimming to more aerobic-based sports such as distance running. These data also support the concept that adequate reductions in training volume and intensity are required for improved muscle and athletic performance.

In summary, the decrease in oxygen consumption during submaximal running, increased citrate synthase activity and increased proportion of MHC I fibers are all indicative of changes that would aid in distance running performance (5, 8). In addition, this study illustrates a high degree of muscle plasticity with long slow distance running in recreational individuals. In the course of the 13 wk of training the muscle fibers decreased in diameter (MHC I and IIa) while maintaining their force-generating capacity (MHC I and IIa) and increasing $V_o$ and power (MHC I only). During the taper, the MHC I fibers were not significantly altered, whereas the MHC IIa fibers increased in P0 and power. These data show that both fiber types were susceptible to run training-induced alterations. From a performance perspective the changes in MHC I fiber mechanics may be beneficial for distance running by the end of the training period whereas the beneficial changes in MHC IIa fibers may not be realized until after a period of taper.

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