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Single Muscle Fiber Adaptations With Marathon Training

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ABSTRACT

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 biopsies were obtained from the gastrocnemius muscle of seven individuals (22±1 y; 177±3 cm and 68±2 kg) before, after 13 wks of run training and after 3 wks of taper. Slow-twitch (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 3h 56min to 5h 35min). Oxygen uptake during submaximal running and citrate synthase activity were improved ($P<0.05$) with the training program. Muscle fiber size declined ($P<0.05$) ~20% in both fiber types after training. Po was maintained in both fiber types with training and increased ($P<0.05$) 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 ($P<0.05$) 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 maintained or improved the functional profile of these fibers. A taper period before the marathon further improved the functional profile of the muscle, which was targeted to the fast-twitch muscle fibers.

INTRODUCTION

The marathon is a 42.2 km (26-mile, 385 yard) running event that challenges the participant to physiological extremes. Over the past half-century, scientists have studied numerous recreational and elite distance runners to better understand the physiological challenges and the physical qualities necessary for successful marathon running (cf. 25). Today, there are hundreds of marathons worldwide, with some of these events having more than 40,000 participants, the vast majority of which are recreational runners. We know from previous research that body size, aerobic conditioning, running efficiency, fiber type, nutrition and training regimen all contribute to marathon success (4-6, 8, 10, 11). We also know that runners of similar physiological abilities have been shown to have large differences in performance that cannot always be explained by these physiological variables (22). Rather, it is a combination of several physiological and psychological parameters that collectively determine distance-running success.

One physiological area that has received little attention with distance running is muscle function at the cellular level. Skeletal muscle is a heterogeneous tissue comprised of slow-twitch, fast-twitch and hybrid muscle fiber types (cf. 41). Each of these muscle fiber types has distinct contractile characteristics that contribute differently to whole muscle and athletic performance (3). How individual muscle fiber mechanics are altered with a marathon-training program is unknown. Given the high degree of skeletal muscle plasticity in humans with exercise, it is likely that the contractile function of slow-twitch and fast-twitch muscle fibers undergo differential alterations with distance run training. The current study resulted from an opportunity to interface a level of

scientific inquiry with a university class that was being conducted with the sole purpose of preparing recreationally active college students to complete their first marathon. In this manner, we were able to monitor the training program and select time points before, during and after training that included tests ranging from whole body measures of aerobic conditioning to muscle biopsies for the analysis of single muscle fiber contractile function. We hypothesized that the MHC I fibers would undergo quantitative and qualitative adaptations to the training and taper period while the MHC IIa fibers would have minor alterations in contractile behavior.

METHODS

Subjects

Seven subjects (4 male; 3 female) 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 prior to this study. Prior to data collection and any training, the Institutional Review Board approved the study design and testing procedures. All subjects provided their written consent prior to 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 to week 1. During the 3-wk taper, running volume was gradually decreased each week. Compared to the last week of training (week 13), running volume was reduced by 25% in week one, 47% in week two 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-d a week program has been previously shown to be as effective as a 6-d a week program for novice runners (12).

The runners were tested on three occasions: before the 16-wk training plan, after 13-wks of run training and after 3-wks of taper and marathon. Single muscle fiber physiology experiments and oxidative enzyme activity were conducted at all three time points. Single muscle fiber myosin heavy chain (MHC) isoform experiments were conducted before the training program and after the taper. Treadmill testing for maximal oxygen uptake was conducted at the beginning of the training program and after the 13-wk training period. No treadmill test was performed after the taper since we did not want to conduct a maximal test too close to marathon day. Further, we did not anticipate that any improvement in aerobic capacity would be observed as a result of the taper period (18).

Maximal Oxygen Consumption

Subjects completed an incremental treadmill test to voluntary exhaustion for the determination of maximal oxygen consumption (VO_2max). When tested, subjects initially walked on a level treadmill for 4 minutes. The speed was then 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. Maximal oxygen uptake was confirmed by a leveling of VO_2 and a respiratory exchange ratio (RER) greater than 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), 3L mixing chamber, and electronic O_2 and CO_2 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 pre 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 following the marathon. Thus, the final muscle biopsy included the taper phase and marathon. Collectively, this time-point is referred to as "post-taper" throughout the manuscript.

The muscle biopsy was divided into 1) a muscle bundle for single muscle fiber physiology experiments was placed in cold skinning solution (see below) and stored at -20°C until analysis, 2) a muscle bundle for the single fiber MHC experiments was

placed and stored in skinning solution and 3) a muscle bundle for citrate synthase activity 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 (mM): 125 K propionate, 2.0 EGTA, 4.0 ATP, 1.0 $MgCl_2$, 20.0 imidazole (pH 7.0), and 50% (v/v) 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 (mM) 7.0 EGTA, 20.0 imidazole, 14.5 creatine phosphate, 1.0 free Mg^{2+}, 4.0 free MgATP, KCl and KOH to produce and ionic strength of 180 mM and a pH of 7.0. The relaxing and activating solutions had a free [Ca^{2+}] of pCa 9.0 and pCa 4.5, respectively.

Single Muscle Fiber Physiology Experiments

On the day of an experiment, a 2-3 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 DC 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 (Olympus BH-2, Japan) allowing the fiber to be viewed (800x) during an experiment. Using 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.

Unamplified force and length signals were sent to a digital oscilloscope (Nicolet 310, Madison, WI) enabling muscle fiber performance to be monitored throughout each experiment. Analog force and position signals were amplified (Positron Development, Dual Differential Amplifier, 300-DIF2, Ingelwood, CA), converted to digital signals (National Instruments, Inc.) and transferred to a computer for analysis using customized software. Servo-motor arm and isotonic force clamps were controlled using a computer interfaced force-position controller (Positron Development, Force Controller, 300-FC1, Ingelwood, CA).

For each single muscle fiber experiment, a fiber with a compliance (calculated as fiber length divided by y-intercept) greater than 10%, and/or a decrease in peak force of more than 10% was discarded and not used for analysis. The within fiber test/re-test of a single muscle fiber in our lab for the measurements of diameter, peak force, contractile velocity and power were less than 1%. The coefficient of variation for the force transducer and servo-mechanical lever mechanism during the 16-wk period 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, peak force (Po), maximal unloaded shortening velocity (V_O), and power characteristics. Detailed descriptions and illustrations of these procedures have been presented in our previous work (32, 35).

Single Fiber Diameter. A video camera (Sony CCD-IRIS, DXC-107A, 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 v1.61) and averaged to provide a mean diameter measurement.

Single Fiber Strength (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, Inc.). Resting force was monitored and then the fiber was maximally activated in pCa 4.5 solution. Peak active force (Po) was determined in each fiber by subtraction of baseline force from peak force.

Single Fiber Contractile Velocity (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 fiber length (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 fiber length 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. Force and shortening velocity data points derived from the isotonic contractions were fit using the Hill equation (20). Individual experiments in which r^2 was greater than or equal to 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; Watts/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% b-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 hours 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_2max 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, 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 ± S.E.

RESULTS

Running Performance

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

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_2max pre to post run training (3.37 vs. 3.50 l/min; $P=0.09$), while relative VO_2max (49.5 vs. 52.0 ml/kg/min) was unchanged. Maximal ventilation (93.7 vs. 97.0 l/min) and maximal heart rate (197 vs. 198 beats/min) were similar pre to post run training.

Absolute (2.43 vs. 2.28 l/min) and relative (36.0 vs. 33.6 ml/kg/min) oxygen consumption at a submaximal running speed of 9.65 kmh 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 37% (19.2±1.4 to 26.3±1.2 μmol•g^{-1}•min^{-1}) after 13-wks of training ($P<0.05$). No change in citrate synthase activity was observed at the end of training compared to 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 pre to post is shown in Figure 1. The delta 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±4%) or the hybrid population of MHC IIa/IIx and I/IIx fibers was observed.

Single Muscle Fiber Physiology Experiments

A total of 403 fibers from the gastrocnemius were studied as part of the single fiber physiology experiments. Approximately 20 muscle fibers were studied from each subject at each time point. Due to the small number of hybrid fibers they were not included for analysis. No pure MHC IIx fibers were observed at any time point from these subjects.

Single Fiber Diameter

Single muscle fiber diameter is shown in Figure 2. MHC I fibers decreased ($P<0.05$) 21% in diameter with training. The MHC IIa fibers decreased ($P<0.05$) 23% with training. No further change in muscle fiber diameter occurred with the taper period.

Single Fiber Peak Force (Po)

Single muscle fiber peak force is shown in Table 2. There was no significant change in the MHC I fiber Po over the three measured time points. MHC IIa fiber Po increased ($P<0.05$) 18% with taper. Given the decreases in fiber diameter with no change or an increase in fiber Po, there was an increase in fiber force per cross-sectional area for both fiber types (Table 2). The MHC I and IIa muscle fibers had a ~60% increase ($P<0.05$) in specific force with training.

Single Fiber Shortening Velocity (Vo)

Fiber Vo is shown in Table 2. The MHC I fibers had a 28% increase ($P<0.05$) in Vo after training, with no further change after taper. MHC IIa fiber Vo was similar at all three time points.

Single Fiber Power

Peak power and peak normalized power is shown in Table 3. MHC I peak power increased ($P<0.05$) 56% after training with no further change after taper. MHC IIa peak power increased ($P<0.05$) 16% with training and an additional 26% with taper (overall a 47% increase).

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

Normalized power distribution is shown in Figure 3. Prior to any training, 83% of the MHC I fibers had a normalized power below 1.5 W/L compared to 9% after training and less than 1% after taper. For the MHC IIa fibers, 49% had a normalized power below 5 W/L prior to training compared to only 9% after taper. In addition, 7% of the MHC IIa fibers had a normalized power above 10 W/L before training compared to 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-weeks 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. Secondly, with a 3-week reduced training phase (taper), alterations in the functional profile of the muscle were targeted to the MHC IIa fibers.

Aerobic Adaptations

Maximal oxygen consumption was not improved as would be anticipated with marathon training, although there was a trend (+130 ml; p=0.09) for an increase. For these novice runners, the improvement in running economy (-150 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 VO_2 combined with the small increase in VO_2max resulted in a decline in fractional utilization (73% before training compared to 66% after training) at 9.6 kmh, which was similar to the training pace and subsequent marathon run pace for these individuals. The similarity in heart rate with a decline in VO_2 during submaximal running was surprising and suggests that stroke volume was reduced during this effort. The reasons

for this are unclear, but could be related to hydration status or variation in submaximal heart rate (range 151 to 173 b/min).

The pretest VO_2max values were more in-line with trained recreational runners (19) as compared to 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 as compared to cardiovascular system.

Single Fiber Adaptations To Training

Prior to the run training program, single muscle fiber diameter was in the range that has been previously reported for untrained individuals (7, 32, 38), and smaller than typically observed in more talented runners (7, 8, 18). Fiber diameter decreased ~20% in MHC I and IIa muscle fibers with 13-wks 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 years 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 force. The mechanism for this uncoupling is unknown, although several have been postulated (9, 24, 29).

The elevated contractile velocity in MHC I fibers coincides with a previous study on masters runners showing MHC I fiber Vo was ~20% higher compared to matched sedentary adults (38). In contrast, we recently found that MHC I fiber Vo declined among college runners during a cross-country season (18). Prior to being tested before 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 pre-testing MHC I fiber Vo's from these athletes (1.66 FL/s) was on the upper end of the range typically observed for human slow-twitch muscle fibers (15). Following 8-wks of intense running, MHC I fiber Vo declined 21% (to 1.27 FL/s). Thus, it could be argued that the weeks of consistent submaximal aerobic training prior to the start of the cross-country season led to an increase fiber Vo and when interval training was incorporated, fiber Vo declined. Taken together, these studies support the notion that distance running can alter shortening velocity 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 current study is that the power profile of human skeletal muscle was improved with distance running. Since normalized power accounts for muscle cell diameter,

strength and contractile speed, it provides a functional overview of muscle cell performance. In the current study, normalized power increased >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-wks of distance running. This continuum shift (as highlighted in Figure 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-week 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 5-6 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 8k performance was not improved at the championship competition following 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-d 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.

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 low long slow distance running in recreational individuals. In the course of the 13-wks of training the muscle fibers decreased in diameter (MHC I and IIa) while maintaining their force generating capacity

(MHC I and IIa) and increasing contractile speed and power (MHC I only). During the taper, the MHC I fibers were not significantly altered, while the MHC IIa fibers increased in strength 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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List of Figures

Table 1. Run training plan. All distances shown are in kilometers (miles).

Week	Mon	Tues	Wed	Thurs	Fri	Sat	Sun	Total
Training Schedule								
1	4.9 (3)	6.5 (4)	off	4.9 (3)	off	8.1 (5)	off	24.3 (15)
2	4.9 (3)	6.5 (4)	off	4.9 (3)	off	9.7 (6)	off	25.9 (16)
3	4.9 (3)	6.5 (4)	off	4.9 (3)	off	11.3 (7)	off	27.5 (17)
4	4.9 (3)	8.1 (5)	off	4.9 (3)	off	13.0 (8)	off	30.8 (19)
5	4.9 (3)	8.1 (5)	off	4.9 (3)	off	16.2 (10)	off	34.0 (21)
6	6.5 (4)	8.1 (5)	off	6.5 (4)	off	17.8 (11)	off	38.9 (24)
7	6.5 (4)	9.7 (6)	off	6.5 (4)	off	19.4 (12)	off	42.1 (26)
8	6.5 (4)	9.7 (6)	off	6.5 (4)	off	22.7 (14)	off	45.4 (28)
9	6.5 (4)	11.3 (7)	off	6.5 (4)	off	25.9 (16)	off	50.2 (31)
10	8.1 (5)	13.0 (8)	off	8.1 (5)	off	25.9 (16)	off	55.1 (34)
11	8.1 (5)	13.0 (8)	off	8.1 (5)	off	25.9 (16)	off	55.1 (34)
12	8.1 (5)	13.0 (8)	off	8.1 (5)	off	29.1 (18)	off	58.3 (36)
13	8.1 (5)	13.0 (8)	off	8.1 (5)	off	29.1 (18)	off	58.3 (36)
Taper Schedule								
14	8.1 (5)	13.0 (8)	off	8.1 (5)	off	14.6 (9)	off	43.7 (27)
15	4.9 (3)	8.1 (5)	off	4.9 (3)	off	13.0 (8)	off	30.8 (19)
16	4.9 (3)	4.9 (3)	off	Walk 3	off	42.2 (26.2)		57.0 (35.2)

Table 2. Single muscle fiber Po, Po/CSA and Vo of MHC I and IIa muscle fibers.

	Po (mN)		Po/CSA (kN•m^2)		Vo (FL•s^{-1})	
	MHC I	MHC IIa	MHC I	MHC IIa	MHC I	MHC IIa
Pre	0.49±0.04	0.60±0.04	87±7	98±12	0.99±0.04	3.18±0.15
Trained	0.55±0.04	0.61±0.04	141±5*	157±12*	1.27±0.11*	3.38±0.27
Tapered	0.56±0.04	0.72±0.04†	130±5*	160±9*§	1.22±0.08*	3.34±0.34

*p<0.05 from Pre
†p<0.05 from Pre and Trained (both fiber types)
§p<0.05 from MHC I fibers

Pre = pre training
Trained = after 13-wks of run training
Tapered = after 3-wks of taper

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

	Peak Power (μN•FL/s)		Normalized Power (W/L)	
	MHC I	MHC IIa	MHC I	MHC IIa
Pre	6.2±0.9	32.2±3.0	1.07±0.12	5.48±0.99
Trained	9.7±1.2*	37.5±4.2	2.43±0.17*	9.32±1.06*
Tapered	9.9±0.9*	47.2±2.8†	2.27±0.16*	10.63±1.01†

*p<0.05 from Pre
†p<0.05 from Pre and Trained

Pre = pre training
Trained = after 13-wks of run training
Tapered = after 3-wks of taper

FL/s = fiber length per second
W/L = watt per liter