Myocellular basis for tapering in competitive distance runners

Nicholas Luden, Erik Hayes, Andrew Galpin, Kiril Minchev, Bozena Jemiolo, Urika Raue, Todd A. Trappe, Matthew P. Harber, Ted Bowers, and Scott Trappe

Human Performance Laboratory, Ball State University, Muncie, Indiana

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Luden N, Hayes E, Galpin A, Minchev K, Jemiolo B, Raue U, Trappe TA, Harber MP, Bowers T, Trappe S. Myocellular basis for tapering in competitive distance runners. J Appl Physiol 108: 1501–1509, 2010. First published March 18, 2010; doi:10.1152/japplphysiol.00045.2010.—The purpose of this study was to examine the effects of a 3-wk taper on the physiology of competitive distance runners. We studied seven collegiate distance runners (20 ± 1 yr, 66 ± 1 kg) before and after a 3-wk taper. The primary measures included 8-km cross-country race performance, gastrocnemius single muscle fiber size and function (peak force, shortening velocity, and power), baseline and exercise-induced gene expression 4 h after a standardized 8-km run, citrate synthase activity, and maximal and submaximal cardiovascular physiology (oxygen consumption, ventilation, heart rate, and respiratory exchange ratio). Race performance improved by 3% following taper (P < 0.05). Myosin heavy chain (MHC) IIa fiber diameter (+7%, P < 0.05), peak force (+11%, P = 0.06), and absolute power (+9%, P < 0.05) increased following taper. In addition to the MHC IIa adaptations, taper elicited a distinct postexercise gene response. Specifically, the induction of MuRF-1 was attenuated following taper, whereas MRF4, HSP 72, and MT-2A displayed an exaggerated response (P < 0.05). No changes were observed in MHC I size or function, baseline gene expression, citrate synthase activity, or cardiovascular function. Our findings show that tapered training in competitive runners promoted MHC IIa fiber remodeling and an altered transcriptional response following the same exercise perturbation, with no adverse affects on aerobic fitness. Together, these results provide a myocellular basis for distance runners to taper in preparation for peak performance.

cross country performance; contractile properties; aerobic capacity; collegiate runners

TAPER is a strategy typically implemented by athletes in the days and weeks leading up to a competition of emphasis. Defined by a systematic reduction of training load (4), taper yields physiological and psychological benefits that together improve performance by approximately 2–8%. Evidence of this practice can be traced back to competitive swimming no later than the mid 1960s (7), but it was not until the 1980s that scientists began to examine the underlying physiology (4). Since then, a considerable amount of data has been gathered on swimmers (3, 4, 9, 26, 54, 59, 60), cyclists (25, 31, 35–38), and runners (17, 20–22, 32, 34, 51, 56). These studies have predominantly targeted cardiovascular, metabolic, and neuromuscular parameters in an attempt to identify the physiological factors supporting the ergogenic effect of taper. From these results, it does not appear that enhanced performance can be explained by alterations in cardiovascular or metabolic function, as they remain unchanged (17, 26, 32, 51, 60) or improve on a relatively small scale (9, 22, 25, 35–37). Conversely, augmented peak muscle power has emerged as a hallmark adaptation to tapered training (3, 4, 6, 25, 26, 44, 53, 54, 59, 65), which appears to be largely facilitated by myosin heavy chain (MHC) IIa muscle fiber adaptations (36, 54, 56). However, the majority of these findings have been documented in swimmers and cyclists with little known about the skeletal muscle response to taper in competitive runners.

The effect that taper has on skeletal muscle power (whole muscle or cellular) of competitive runners has been assessed on only two occasions. In the first, no modifications were detected in either the vertical jump or Margaria step test following a 3-wk intervention (20). Lack of specificity perhaps confounded these results, as improvements in sport-specific muscle power with taper may not necessarily translate into improvements in traditional whole muscle measures. In light of methodological challenges related to assessing running-specific muscle power, the single muscle fiber technique provides a distinct advantage by supplying information about myocellular function independent of motor learning/sport specificity. Our laboratory applied this technique during a 4-wk taper in competitive runners and observed decrements in gastrocnemius MHC I muscle fiber function with minimal changes in the MHC IIa fibers (17). However, the reduction of total training volume was notably accompanied by an increase in the amount of high-intensity training, which appears to have provided a unique training stimulus rather than the desired recovery.

The lack of information on muscle adaptations to taper in runners combined with anecdotal concerns about losing cardiovascular fitness has slowed the adoption of a structured run taper. A unique collaboration with a local collegiate cross-country team allowed us to thoroughly assess the effects of a structured 3-wk taper modeled after a program known to enhance both cycling and swimming performance (36, 54). In addition to measuring cross-country race performance we tested the hypotheses that taper would 1) improve MHC I and MHC IIa gastrocnemius muscle fiber function with more pronounced adaptations within the MHC IIa population and 2) sustain aerobic function at both the whole body and cellular level (citrate synthase activity). We were also interested in gaining insight into potential molecular alterations with taper by determining baseline mRNA levels of select genes implicated in proteolytic (atrogin-1, MuRF-1), myogenic (MRF4, myostatin), and protective (MT-2A, HSP 72) cellular processes. We further tested the possibility that these same gene markers would respond differently upon an identical 8-km training stimulus before and after taper.

METHODS

Subjects

Seven male runners recruited from Taylor University’s Cross-Country Team volunteered to take part in the study. The subjects were 20 ± 1 yr, 179 ± 2 cm, 66 ± 1 kg, and classified as competitive
runners based on 8-km lifetime best performances of 26:32 ± 0:32 (min:s). Before the investigation, the subjects had competitively participated in running for an average of 7 yr (range: 4.5–10.0 yr). Subjects were provided with written and oral information about the experimental procedures and potential risk before written consent. Support was granted by the coaching staff, and all procedures were approved by the Ball State University and Taylor University Institutional Review Boards before any testing.

### Experimental Design

Schematics of the study design are displayed in Fig. 1, A and B. Identical laboratory procedures were performed before and after the 3-wk taper, with each time point requiring two testing sessions (total sessions = 4). The subjects competed in an 8-km cross-country (XC) race 9 days before the onset of taper and immediately after the 3-wk taper. Two days following each race, baseline and postexercise muscle biopsies were obtained (before and after an 8-km standardized indoor track run). The biopsy samples were used for measures of single fiber size and contractile physiology, gene expression, and citrate synthase activity. Additionally, 3 days before and 10 days after taper, subjects reported to the laboratory for treadmill testing.

### Training

The coaching staff prescribed the training performed throughout the 11-wk season (8 wk of midseason training + 3 wk of taper) based on interactions and recommendations provided by our laboratory team. Training load was quantified with data acquired from Polar heart rate monitors and self-reported running logs. Weekly midseason training load (8 wk) was aggregated to represent one mean for the training conducted before taper. Average midseason weekly running volume (6 days/wk) was ~72 km. Weekly running volume was reduced throughout the 3-wk taper with the final week corresponding to 50% of midseason training. Worth noting is that the majority of the reduction in training volume was attributed to a decrease in moderate-intensity training. Training volume at three distinct training intensities are presented in Fig. 2.

### Cross-Country Race Performance

To maximize generalizability and minimize team interference, 8-km cross-country race performance was used as the primary performance criterion. As displayed in Fig. 1A, the pretaper performance took place 9 days before the start of taper amidst a period of heavy training, whereas the day before the posttaper performance (championship cross-country race) marked the end of the taper. Cross-country race performance was assessed on two different courses. Course details and corresponding weather conditions are presented in Table 1.

### Standardized 8-km Indoor Track Run

For the session before taper, subjects were instructed to run 8 km on a 200-m indoor track between 15.2 and 16.0 km/h (~45–48 s/lap) depending on talent level. This pace was chosen because it corresponds to a training velocity commonly performed by this group. Lap times were verbalized to the subjects throughout the run to ensure even pacing, and each lap split was manually recorded. For the session after taper, subjects were instructed to replicate their pretaper lap splits, which were again monitored by the investigative team.

### Muscle Biopsy

A total of four muscle biopsies (~50–100 mg/each) were obtained from the lateral head of the gastrocnemius (1). The gastrocnemius was

![Fig. 1. A: general study design. Nos. are days relative to taper. R is championship cross-country (XC) race at the end of taper. Note: XC races were separated by 4 wk. B: muscle biopsy (Bx) design implemented before and after 3-wk taper.](#)
Table 1. Cross-country course profile and race conditions

<table>
<thead>
<tr>
<th>Time Point</th>
<th>Distance, km</th>
<th>Ascent, m</th>
<th>Descent, m</th>
<th>Temperature, °C</th>
<th>Wind, km/h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before taper</td>
<td>8.0</td>
<td>291</td>
<td>290</td>
<td>16.6</td>
<td>17.7</td>
</tr>
<tr>
<td>After taper</td>
<td>8.2</td>
<td>221</td>
<td>214</td>
<td>4.4</td>
<td>25.7</td>
</tr>
</tbody>
</table>

Ascent is the sum of all ascents recorded via Garmin Global Positioning System (GPS). Descent is the sum of all descents recorded via GPS. Temperature is that recorded at the start of the race. Wind is the average speed.

chosen based on its documented use during running (5) and the large amount of gastrocnemius research conducted in runners over the past 40 yr. Subjects reported to Taylor University 2 days (~48 h) following an 8-km cross-country race, a timeframe in which subjects were asked to refrain from physical activity. After 30 min of supine rest, a baseline muscle biopsy was obtained. Following the biopsy, a standardized 8-km run was performed on a 200-m indoor track (see above). Subjects then underwent 4 h of supine rest upon which a second biopsy was taken from the opposite leg. After a meal on the evening before testing (~6:00 PM), subjects fasted with ad libitum water intake until the 4-h postexercise biopsy. The 4-h postexercise biopsy time point was based on previous postexercise mRNA time course investigations from our laboratory (29, 63). Immediately following each biopsy, the sample was dried of excess blood, and any visible adipose and/or connective tissue was removed. The muscle following each biopsy, the sample was dried of excess blood, and any visible adipose and/or connective tissue was removed. The muscle tissue was processed as follows: 1) several longitudinal bundles from either biopsy (pre- or postexercise) were placed in cold skinnning solution (see below) and stored at ~20°C for later analysis of single muscle fiber physiology; 2) a ~15-mg piece from each biopsy was placed in 0.5 ml of RNALater (Ambion, Austin, TX) and stored at 4°C overnight, and subsequently stored at ~20°C until RNA extraction; and 3) a ~10-mg piece from either biopsy was quickly frozen in liquid nitrogen and stored at ~190°C for later analysis of citrate synthase activity.

Skinning, Relaxing, and Activating Solutions

The skinnning solution contained (in mM) 125 potassium propionate, 2.0 EGTA, 4.0 ATP, 1.0 MgCl2 and 20.0 imidazole (pH 7.0), and 50% (vol/vol) glycerol. The compositions of the relaxing and activating solutions were calculated using an interactive computer program described by Fabiato and Fabiato (11). These solutions were adjusted for temperature, pH, and ionic strength using stability constants in the calculations (15). Each solution contained (in mM) 7.0 EGTA, 20.0 imidazole, 14.5 creatine phosphate, 1.0 free Mg2+, 4.0 free MgATP, KCl and KOH to produce an ionic strength of 180 mM, and a pH of 7.0. The relaxing and activating solutions had a free [Ca2+] of pCa 9.0 and pCa 4.5, respectively (where pCa = −log [Ca2+]).

Single Muscle Fiber Experimental Set-Up

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 cold relaxing solution. The fiber ends were aligned in stainless steel troughs and securely fastened with 4.0 monofilament posts and 10.0 suture. The troughs were attached via titanium wires to a force transducer (model 400A; Cambridge Technology, Watertown, MA) and a DC torque motor (model 308B, Cambridge Technology) as described by Moss (33). 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) so that the fiber could be viewed (~800) during an experiment.

The single muscle fiber experiments were performed at 15°C, a temperature that has been used in our past human performance (54), aging (58), and unloading studies (57), and numerous other laboratories (27, 30, 61), as in vitro experiments with isolated skinned muscle fibers are more stable at 15°C degrees compared with physiological temperature (37°C) (2, 19). The temperature of the experimental chambers was monitored constantly (copper-constantan thermocouple, PP-T24S; Omega Engineering, Stamford, CT) during the experiments. To cool the relaxing and activating solutions in the experimental chambers, a bath (RTE 111; Neslab, Portsmouth, NH) constantly circulated water through a milled aluminum plate. A thermoelectric pump Pelier junction current source with a feedback circuit temperature controller (E5AX-VAA, sensitivity ± 0.3%; Omron) lowered the temperature of the experimental chambers to 15°C and maintained this temperature throughout the experiment.

Single Muscle Fiber Analysis

Individual muscle fibers were analyzed for diameter, peak force (Po), shortening velocity (Vo), and power characteristics as previously described (54, 55). Following an experiment, each single fiber was analyzed for MHC composition as described below. All single fiber contractile measurements were completed within a 4-wk period following the biopsies.

**Single fiber diameter.** The sarcomere spacing for each muscle fiber was adjusted to 2.5 μm with an eyepiece micrometer. A video camera (CD-IRIS, DXC-107A; Sony) connected to the microscope and interfaced to a computer allowed viewing on a computer monitor and storage of the digitized images of the muscle fibers during the experiment. Fiber diameter was determined from a captured computer image taken with the fiber briefly suspended in air (<3 s). Fiber width (diameter) was determined at three points along the length of the captured computer image using NIH public domain software (Scion Image, Release Beta 4.0.2, for Windows). Fiber cross-sectional area (CSA) was calculated from the mean width with the assumption that the fiber forms a cylindrical cross section when suspended in air.

**Force determination (Po and Po/CSA).** Force output and position transducers were amplified and sent to a microcomputer via a lab-PC+ 12-bit data-acquisition board (National Instruments). Resting force was monitored, followed by maximal activation in pCa 4.5 solution. Peak active force (Po) was determined for each fiber by computer subtraction of the baseline force from the peak force produced in the pCa 4.5 solution (62). Po was also expressed relative to fiber size (Po/CSA: specific tension).

**Unloaded shortening velocity (Vo).** Vo was measured by the slack test technique described by Edman (10). Fibers were transferred from relaxing solution (pCa 9.0) to activating solution (pCa 4.5) and brought to peak tension. The fiber was then rapidly shortened so that tension returned to baseline. The fiber shortened, taking up the induced slack, which was followed by redevelopment of tension. The fiber was then returned to relaxing solution and its original length. The time between the onset of slack and redevelopment of tension (i.e., period of unloaded shortening) was measured by computer analyses. Four different slack distances [each <15% of fiber length (FL)] were used for each fiber, and the slack length was plotted as a function of the duration of unloaded shortening. Velocity (FL/s) was calculated by dividing the slope of the fitted line by the fiber segment length, and the data were normalized to a sarcomere length of 2.5 μm.

**Single fiber power (peak power, normalized power, and Vmax).** Submaximal isometric 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. For the resultant force-velocity relationships, load was expressed as P/Po, where P is the force during load clamping and Po is the peak isometric force developed before the submaximal load clamps. Force and shortening velocity data points derived from the isotonic contractions were fit by the hyperbolic Hill equation (18). Only individual experiments in which r2 was ≥ 0.98 were accepted.

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Fiber peak power was calculated from the fitted force-velocity parameters \( (P_0, V_{\text{max}}, \text{and} a/P_0) \), where \( a \) is a force constant and \( V_{\text{max}} \) is the \( y \)-intercept. Absolute power \( (\mu \text{N} \cdot \text{FL/s}) \) was defined as the product of force \( (\mu \text{N}) \) and shortening velocity \( (\text{FL/s}) \). Normalized power \( (\mu \text{W}) \) was defined as the product of normalized force and shortening velocity.

**Single Muscle Fiber MHC Isoform Analysis**

The MHC composition of each fiber was determined with SDS-PAGE (SE 600 series, Hoefer, San Francisco, CA) analysis. Following each single fiber physiology experiment, the fibers were solubilized in 80 \( \mu \)l of 1% SDS sample buffer and stored at \(-20^\circ \text{C}\) until analyzed for MHC content. Samples were loaded on a 3.5% stacking gel and 5% separating gel and run 12 h at 4°C. The gels were silver stained (14), allowing for the MHC content \( (I, I/IIa, I/IIa/IIx, Ila, Ila/IIX, Ix) \) of each single muscle fiber to be determined.

**Total RNA Extraction and RNA Quality Check**

Our laboratory has previously detailed the methods for performing both RNA extraction, reverse transcription, and real-time qPCR (RT-qPCR) (63). Total RNA was extracted in TRI Reagent and 2 \( \mu \)l of PolyAcryl Carrier according to the manufacturer’s protocol (Molecular Research Center, Cincinnati, OH). The quantity \( (184.65 \pm 11.94 \text{ ng/\mu l}) \) and integrity \( (\text{RIN}=8.50 \pm 0.07) \) of total RNA extract was evaluated using the RNA 6000 Nano LabChip kit on an Agilent 2100 Bioanalyzer (Agilent Technologies, Palo Alto, CA).

**Reverse Transcription and Quantitative Real-Time RT PCR**

Oligo(dT) primed first-strand cDNA were synthesized using SuperScript II RT (Invitrogen, Carlsbad, CA) according to the manufacturer’s protocol. Quantification of mRNA content for each sample and all genes was performed in duplicate using a 72-well Rotor-Gene 3000 Centrifugal Real-Time Cycler (Corbett Research, Mortlake, NSW, Australia). Housekeeping gene GAPDH was validated and used for a reference gene as we have previously reported (24). All primers used in this study were mRNA-specific (in different exons and crossing over an intron) and designed (Vector NTI Advance 9 software, Invitrogen) for RT qPCR analysis using SYBR Green chemistry. Primer sequences for atrogin-1, MuRF-1, MRF4, and CACCATGCGCTC-5' GAGC-3', HSP 72 reverse 3'-CAAGAGCACCGGCAA-5', MT-2A reverse 3'-CGCCTCCTGCAACCATGCGCTC-5', MT-2A forward 5'-CCGCCGTTGACTCTGTGACCC-3', MT-2A reverse 3'-GCCCGAGGGGCAGACGGAGGC-5'. Details about qPCR parameters, dilution curve, as well as amplicon melting curve analysis have been reported previously (29, 63).

The amplification calculated by Rotor-Gene software for each run was specific and highly efficient \( (1.05 \pm 0.02, R^2 = 0.99 \pm 0.00; \text{slope} = 3.24 \pm 0.07) \).

**Relative qPCR Data Analysis**

The influence of tapered training on baseline gene expression \( (\text{pretaperexpression level before taper compared with preexpression level after taper}) \) was analyzed using \( 2^{-\Delta C_t} \) method \( \Delta C_t = (C_t \text{target gene} - C_t \text{reference gene})_{\text{base}} \), expressed in arbitrary units \( (\text{AU}) \) (28). Exercise-induced gene expression before taper \( (\text{pre- vs. postexercise level}) \) and after taper \( (\text{pre- vs. postexercise level}) \) was analyzed using \( 2^{-\Delta C_{\Delta C_t}} \) method \( \Delta C_{\Delta C_t} = (C_t \text{target gene} - C_t \text{reference gene})_{\text{base}} \times (C_t \text{target gene} - C_t \text{reference gene})_{\text{preexpression level}} \), expressed as fold change of post to pre level \( (28, 45, 63) \).

**Citrate Synthase Activity**

Citrate synthase activity was measured from an \( \sim 10\text{-mg} \) 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 (52).

**Treadmill Testing**

Subjects completed an incremental treadmill test to volitional exhaustion for the determination of both submaximal and maximum oxygen consumption \( (\dot{V}O_{2\text{max}}) \) 3 days before the onset of taper and 10 days following the end of taper. The subjects commenced the treadmill test with 3 min of level walking. The speed was then progressively increased to 12 km/h \( (4\text{ min}) \), 13.7 km/h \( (4\text{ min}) \), and 16.1 km/h \( (3\text{ min}) \) at which submaximal data are reported. After 3 min at 16.1 km/h, the treadmill velocity was clamped and the grade increased to 4%. Each subsequent 2-min stage was accompanied by a 2% increase in treadmill grade until exhaustion. \( \dot{V}O_{2\text{max}} \) was confirmed with an oxygen consumption plateau 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, Waukesha, 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.

**Statistics**

The effects of taper on 1) cross-country race performance, 2) single fiber size and contractile physiology, 3) preexercise (baseline) gene expression before and after taper, and exercise-induced (pre- vs. post-8-km run) gene expression before and after taper, 4) citrate synthase activity, and 5) cardiovascular physiology were analyzed with a paired \( t \)-test. A two-tailed \( t \)-test was utilized for all variables. Based on our directional hypotheses that MHC Ila single fiber size would increase and function would improve \( (36, 54, 56) \), we also applied a one-tailed \( t \)-test to the MHC Ila fibers \( (2\text{-tailed} P \text{ values are reported (see Table 3). For the single muscle fiber physiology parameters, all fibers studied within each individual were aggregated to represent a mean value for both MHC I and MHC Ila fibers. Because of the minimal number of hybrid and pure MHC Ila studied, analyses were restricted to MHC I and Ila fibers. Significance was set at \( P < 0.05 \). All data are presented as means \( \pm \text{SE} \).

**RESULTS**

**Running Performance**

Cross-country race performance improved \( (P < 0.05) \) \( 3 \pm 1\% \) at the championship meet \( (26:52 \pm 0.27) \) compared with pretaper \( (27:42 \pm 0.21) \). These results are strengthened by the fact that subjects ran faster on the posttaper course despite the course being 200 m longer \( (\text{pre 8.0 km vs. post 8.2 km}) \), a difference that is theoretically worth an additional 40 s at a 27:00 8-km pace. Mean running velocity was calculated to account for the different lengths and mean velocity was 6 \( \pm 1\% \) faster \( (P < 0.05) \) at the championship meet compared with pretaper \( (289 \pm 4 \text{ vs. 306} \pm 5 \text{ m/min}) \) (Fig. 3).

**Single Fiber Physiology**

**Single fiber count.** A total of 348 single fibers were successfully studied before taper \( (n = 169, \text{MHC I} = 82, \text{MHC I/IIa} = 5, \text{MHC Ila} = 74, \text{MHC Ila/IIX} = 8, \text{MHC IIX} = 0) \) and after taper \( (n = 179, \text{MHC I} = 84, \text{MHC I/IIa} = 3, \text{MHC Ila} = 79, \text{MHC Ila/IIX} = 13, \text{MHC IIX} = 0) \). This corresponded to \( \sim 12 \text{ MHC I fibers/subject and 11 MHC Ila fibers/subject at each time point} \). A total of 20 fibers were discarded due to deterioration of contractile function during experimentation \( (\text{before} = 17 \% \text{ (9\% of attempted fibers), after} = 3 \%) \).
Exercise-Induced Gene Expression

The intent of this project was to examine how the physiology of competitive runners is influenced by a structured taper program. We addressed this query by performing measures of single muscle fiber size and contractile function, baseline and exercise-induced gene expression, citrate synthase activity, and maximal and submaximal cardiovascular physiology (oxygen consumption, ventilation, HR, and respiratory exchange ratio), before and after taper. Coinciding with a 3% improvement in 8-km cross-country race performance, the primary physiological effects of taper were 1) enhanced single muscle fiber size and function that targeted MHC IIa muscle fibers, 2) an altered

### Table 2. Single muscle fiber size and contractile summary for MHC I fibers before and after taper

<table>
<thead>
<tr>
<th>Time</th>
<th>Diameter, μm</th>
<th>CSA, μm²</th>
<th>Po, mN</th>
<th>Po/CSA, kN/m²</th>
<th>Vo, FL/s</th>
<th>Vmax, FL/s</th>
<th>Power, μN·FL/s</th>
<th>Normalized Power, W/t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>80.2 ± 3.8</td>
<td>5,121 ± 451</td>
<td>0.56 ± 0.05</td>
<td>109.0 ± 2.8</td>
<td>1.05 ± 0.07</td>
<td>0.81 ± 0.05</td>
<td>9.1 ± 1.0</td>
<td>1.77 ± 0.09</td>
</tr>
<tr>
<td>After</td>
<td>82.8 ± 3.4</td>
<td>5,437 ± 431</td>
<td>0.57 ± 0.04</td>
<td>105.8 ± 3.9</td>
<td>0.98 ± 0.03</td>
<td>0.81 ± 0.02</td>
<td>8.2 ± 0.6</td>
<td>1.52 ± 0.07</td>
</tr>
<tr>
<td>P Value</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SE. Before, before taper; After, after taper; MHC, myosin heavy chain; CSA, fiber cross-sectional area; Po, peak force; Po/CSA, specific tension; Vo, unloaded shortening velocity; Vmax, maximal shortening velocity; FL, fiber length; NS, not significant.
running velocity was also considered, and the subjects ran an average of 6% faster following taper (7 of 7 runners), further substantiating the performance benefit of taper.

Skeletal muscle power has emerged as a key physiological element of taper adaptation in swimmers, cyclists, and rowers (3, 4, 25, 26, 53, 54, 59). The increase in MHC IIa fiber size and power coupled with the 3% improvement in performance in the present study suggests that improvements in muscle power with taper may also be important for competitive runners. Gains in muscle power have been shown to significantly improve middle-long distance race performance (39). Moreover, our laboratory recently reported that MHC I and MHC IIa gastrocnemius muscle fibers of competitive runners display a greater capacity to generate power compared with muscle fibers obtained from recreational runners (16), and that MHC I and MHC IIa fiber power increased when these same recreational runners completed 13 wk of marathon run training (56). Although we do not know to what extent, it is reasonable to assume that a more powerful MHC IIa myocellular profile is beneficial for whole muscle and running performance, especially considering the large prevalence of MHC IIa fibers (44%) among those studied for single fiber physiology (see RESULTS).

The heightened sensitivity of the MHC IIa fibers to taper, compared with MHC I, is intriguing but not surprising as similar fiber type-specific adaptations have been observed in swimmers (54), recreational runners (56), and cyclists (36). The concept that MHC IIa fiber remodeling with taper can help fine-tune athletic performance over a range of athletic populations, as our laboratory has previously suggested (56), can now

<table>
<thead>
<tr>
<th>Time</th>
<th>Diameter, μm</th>
<th>CSA, μm²</th>
<th>Po, mN</th>
<th>PoCSA, kN/m²</th>
<th>Vo, FL/s</th>
<th>Vmax, FL/s</th>
<th>Power, μN·FL/s</th>
<th>Normalized Power, W/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>85.6 ± 4.2</td>
<td>5,841 ± 527</td>
<td>0.78 ± 0.07</td>
<td>133.4 ± 2.6</td>
<td>3.24 ± 0.11</td>
<td>3.05 ± 0.11</td>
<td>58.7 ± 5.4</td>
<td>10.09 ± 0.21</td>
</tr>
<tr>
<td>After</td>
<td>91.4 ± 4.3</td>
<td>6,649 ± 632</td>
<td>0.87 ± 0.09</td>
<td>130.2 ± 4.0</td>
<td>3.37 ± 0.11</td>
<td>3.20 ± 0.10</td>
<td>63.8 ± 6.2</td>
<td>9.66 ± 0.41</td>
</tr>
<tr>
<td>%Change</td>
<td>7 ± 3</td>
<td>15 ± 6</td>
<td>11 ± 6</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>P Value</td>
<td>P = 0.038</td>
<td>P = 0.050</td>
<td>P = 0.120</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>P = 0.089</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SE. Before, before taper; After, after taper.

The concept that MHC IIa fiber remodeling with taper can help fine-tune athletic performance over a range of athletic populations, as our laboratory has previously suggested (56), can now
be extended to include competitive runners. The mechanism(s) behind the fiber type-specific response is unknown, but it is worth noting that a suboptimal taper program may interfere with this process. For example, a similar cohort of collegiate runners diminished their total training volume for 4 wk while simultaneously increasing the quantity of interval training (17). The runners experienced a decrement in MHC I fiber size and function, no adaptations among the MHC IIA fibers, and no significant improvement in race performance, outcomes that are likely related to the added interval training. Intensity should therefore be closely monitored to ensure that training conducted during taper allows adequate recovery to promote gains in myocardial power. The 15% increase in MHC IIA fiber cross-sectional area with the 3-wk taper in the present study translates to a growth rate of 5% per week. This is slower than the 14% and 8% per week the 3-wk taper in the present study translates to a growth rate.

Table 4. Maximal and submaximal cardiovascular physiology before and after taper

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<th>Stage</th>
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<td>3.68 ± 0.05</td>
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In summary, we performed whole body and myocellular physiological analyses to examine the effects of a structured taper program in college-aged competitive distance runners. The gastrocnemius was advantageously remodeled with taper, evidenced by an increase in MHC IIA fiber size and power along with a distinct transcriptional response to exercise. These alterations were accompanied by improved race performance and a static aerobic profile. Because of the absence of a nontaper group it may not be fair to conclude with certainty that our findings were the direct result of taper. However, similar research revealed that myocellular characteristics and cycling performance improved following taper with no changes occurring in the nontaper group (continued training) (36). Although other factors such as race conditions, neural modifications, and psychology cannot altogether be ignored, the increase in MHC IIA single fiber power likely contributed to the 3% improvement in running performance. Overall this investigation provides a strong myocellular basis for distance runners, and athletes in general, to taper their training volume in preparation for peak performance.

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DISCLOSURES
No conflicts of interest (financial or otherwise) are declared by the authors.

REFERENCES


