Myocellular basis for tapering in competitive distance runners

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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 (17, 20–22, 32, 34, 51, 56).

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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...
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 muscle 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 tissue was processed as follows: 1) several longitudinal bundles from either biopsy (pre- or postexercise) were placed in cold skinnng 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, PT-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 Pelletier 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 muscle 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 (CCD-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 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. 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 $r^2$ was ≥ 0.98 were accepted.
Fiber peak power was calculated from the fitted force-velocity parameters ($P_0$, $V_{max}$, and $a/P_0$, where $a$ is a force constant and $V_{max}$ is the $y$-intercept). Absolute power ($\mu$N·FL/s) was defined as the product of force ($\mu$N) and shortening velocity (FL/s). Normalized power (W/I) 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 µl of 1% SDS sample buffer and stored at −20°C until analyzed for MHC content. Samples were loaded on a 3.5% stacking 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, IIa, IIa/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 µl of PolyAcryl Carrier according to the manufacturer’s protocol (Molecular Research Center, Cincinnati, OH). The quantity (184.65 ± 11.94 ng/µl) and integrity (RIN-8.50 ± 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 atrogain-1, MuRF-1, MRF4, and standard 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.

**RESULTS**

Running Performance

Cross-country race performance improved ($P < 0.05$) 3 ± 1% at the championship meet (26:52 ± 0.27) compared with pretaper (27:42 ± 0.21). These results are strengthened by the fact that subjects ran faster on the posttaper course despite the course being 200 m longer (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 ± 1% faster ($P < 0.05$) at the championship meet compared with pretaper (289 ± 4 vs. 306 ± 5 m/min) (Fig. 3).

**Single Fiber Physiology**

**Single fiber count.** A total of 348 single fibers were successfully studied before taper (total $n = 169$, MHC I = 82, MHC I/IIa = 5, MHC IIa = 74, MHC IIa/IIx = 8, MHC IIx = 0) and after taper (total $n = 179$, MHC I = 84, MHC I/IIa = 3, MHC IIa = 79, MHC IIa/IIx = 13, MHC IIx = 0). This corresponded to ≈12 MHC I fibers/subject and 11 MHC IIa fibers/subject at each time point. A total of 20 fibers were discarded due to deterioration of contractile function during experimentation [before =17 (9% of attempted fibers), after = 3 (2%)].
Diameter/CSA. MHC I fiber size did not change with taper (Table 2). MHC IIa fibers were larger ($P < 0.05$) following taper as evidenced by a $7 \pm 3\%$ increase in diameter and a $15 \pm 6\%$ increase in CSA (Table 3).

Po and Po/CSA. MHC Ila fiber force increased ($11 \pm 6\%$, $P = 0.06$), with no change in specific tension (Table 3). No changes were observed in peak force or specific tension in MHC I muscle fibers with taper (Table 2).

Peak absolute and normalized power. MHC Ila fibers increased peak absolute power by $9 \pm 4\%$ with taper ($P < 0.05$) (Fig. 4 and Table 3). In contrast, MHC I fiber peak absolute power was similar before and after taper (Fig. 4 and Table 2). Peak power normalized to cell size did not change in either fiber type (Tables 2 and 3).

Baseline Gene Expression

Taper did not influence gastrocnemius baseline mRNA expression of any of the genes of interest. Baseline mRNA levels (arbitrary units) for each gene of interest before and after taper, respectively, were as follows: atrogin-1 (12.6 $\pm$ 1.3 vs. 14.4 $\pm$ 2.8), MuRF-1 (1.7 $\pm$ 0.2 vs. 2.1 $\pm$ 0.2), MRF4 (5.7 $\pm$ 0.5 vs. 4.8 $\pm$ 0.7), myostatin (0.12 $\pm$ 0.02 vs. 0.13 $\pm$ 0.03), HSP 72 (0.46 $\pm$ 0.03 vs. 0.38 $\pm$ 0.05), and MT-2A (2.3 $\pm$ 0.3 vs. 2.1 $\pm$ 0.2).

Exercise-Induced Gene Expression

Standardized 8-km indoor track run. The subjects completed the 8-km run in 30:18 $\pm$ 0:30 (min:s) and 30:20 $\pm$ 0:32 for pre- and posttaper, respectively. This corresponded to 89 $\pm$ 1 and 88 $\pm$ 1% HRmax before and after taper.

Proteolytic gene expression. Proteolytic gene expression with exercise is displayed in Fig. 5. MuRF-1 mRNA was elevated ($P < 0.05$) following exercise before and after taper, with an attenuated ($P < 0.05$) response after taper (2.3-fold vs. 1.7-fold). In contrast, atrogin-1 mRNA expression was not altered with exercise before or after taper.

Myogenic gene expression. Myogenic gene expression with exercise is displayed in Fig. 5. The mRNA expression of MRF4 in response to exercise was different before taper compared with after taper ($P < 0.05$). Specifically, MRF4 did not change with exercise before taper (0.9-fold), whereas MRF4 expression was elevated ($P < 0.05$) 4 h postexercise following taper (1.4-fold). The myostatin mRNA exercise response was not influenced by taper despite a postexercise decrease in myostatin before taper (0.4-fold, $P < 0.05$) and no change in myostatin after taper (0.6-fold).

Stress-related gene expression. Stress-related gene expression with exercise is displayed in Fig. 5. HSP 72 gene expression increased ($P < 0.05$) with exercise before and after taper. Notably, a more robust response to exercise ($P < 0.05$) was elicited following taper (1.8-fold vs. 2.2-fold). A similar pattern was detected with MT-2A gene expression—an increase ($P < 0.05$) following exercise before and taper, with an augmented response following taper (1.3-fold vs. 1.9-fold, $P < 0.05$).

Oxidative Enzyme Activity

Gastrocnemius citrate synthase activity ($\mu$mol.g$^{-1}$.min$^{-1}$) did not change with the 3-wk taper (36.8 $\pm$ 3.0 vs. 34.7 $\pm$ 3.9).

Treadmill Testing

Treadmill results are presented in Table 4. Both absolute $\dot{V}O_{2max}$ (l/min) and relative $\dot{V}O_{2max}$ (ml/kg/min) were similar before and after taper. Similarly, neither maximum ventilation nor maximum heart rate changed with taper. At a submaximal speed of 16.09 km/h (6:00 min/mile), no changes were observed in oxygen consumption, ventilation, or heart rate.

DISCUSSION

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 Ila muscle fibers, 2) an altered...
running velocity was also considered, and the subjects ran different courses. Notwithstanding these potentially confounding factors, the race conditions were similar enough to allow for a comparison of performance times (Table 1). The 3% performance improvement in the present study is in line with the range of 2–8% that has been reported with taper, across different modes of activity (4, 22, 54, 59). When the present results are combined with the running literature (17, 20–22, 32, 34, 51), characteristics of an effective run taper include a substantial reduction in training volume (>40% decrease from pretaper) over a 1- to 3-wk period, along with minimal changes in training intensity (20, 22, 51). A strength of this project is that cross-country race performance was used as the performance criterion. However, as a consequence, the races were conducted outside of a controlled laboratory setting and on different courses. Notwithstanding these potentially confounding factors, the race conditions were similar enough to allow for a comparison of performance times (Table 1). The most striking difference between races was the 200-m discrepancy in course length (pre 8.0 km vs. post 8.2 km). Because of the obvious consequences this has on finishing times, mean 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, 65). 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 include a substantial reduction in training volume (40%) among those studied for single fiber physiology (see MATERIALS AND METHODS).

### Table 3. Single muscle fiber size and contractile summary for MHC IIa 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/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.

![Fig. 4. Myosin heavy chain (MHC) I and MHC IIa fiber absolute power curves pre- and posttaper. *P < 0.05, pre- vs. posttaper.](http://example.com/fig4)

**Fig. 4.** Myosin heavy chain (MHC) I and MHC IIa fiber absolute power curves pre- and posttaper. *P < 0.05, pre- vs. posttaper.

![Fig. 5. Exercise-induced (standardized 8-km run) changes in the expression of proteolytic, myogenic, and protective genes, before and after taper. Each bar depicts the fold change of postexercise (4 h) mRNA levels compared with preexercise mRNA levels. Values are reported as means ± SE. *P < 0.05 represents significant difference of postexercise gene expression compared with preexercise expression for each standardized 8-km run exercise. †P < 0.05 represents significant difference of postexercise levels before taper compared with after taper.](http://example.com/fig5)

**Fig. 5.** Exercise-induced (standardized 8-km run) changes in the expression of proteolytic, myogenic, and protective genes, before and after taper. Each bar depicts the fold change of postexercise (4 h) mRNA levels compared with preexercise mRNA levels. Values are reported as means ± SE. *P < 0.05 represents significant difference of postexercise gene expression compared with preexercise expression for each standardized 8-km run exercise. †P < 0.05 represents significant difference of postexercise levels before taper compared with after taper.
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 myocellular 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 that has previously been reported with taper (36, 54) but still more rapid than the <3% elicited by traditional resistance training interventions (23, 46, 61). Superior rates of fiber growth following tapered or reduced training challenge the traditional “overload-induced adaptation” principle. The molecular events required to achieve this magnitude of growth with taper are unknown. Trained muscle typically displays higher rates of protein synthesis at rest (41, 42), suggesting that the translational machinery necessary for sustained periods of elevated protein synthesis have been established. It is therefore possible that heavy phases of training suppress the rate of muscle protein synthesis, as has been shown in rats (50), and that when the training overload is removed in the form of taper, the existing machinery proceeds at high rates. On the other hand, overtraining can also elevate rates of protein catabolism (50). Assuming that the rate of protein breakdown returns to “baseline” with the removal of heavy training, altered protein balance due to changes in both synthesis and breakdown may explain the marked MHC IIA fiber growth with taper. Further research is necessary to test this hypothesis as well as to provide a mechanistic explanation for why these adaptations are exclusive to MHC IIA fibers.

Although we did not assess protein turnover in the present study, we performed targeted mRNA analyses to provide preliminary information about molecular queues that may be involved in muscle remodeling. The transcriptional response elicited by an identical 8- km indoor track run differed in magnitude [4 of 6 genes of interest (GOI) between pre- and posttaper]. For example, exercise-induced MuRF-1 was dampened following taper. Because the induction of MuRF-1 mRNA is associated with proteolysis of myofibrillar proteins (47), our data suggest that tapered muscle is less susceptible to postexercise myofibrillar protein breakdown. Conversely, growth-related MRF4 displayed an exaggerated exercise response following taper. We recognize the limitations of formulating conclusions based on the behavior of only two genes at a single time point, but the combined MuRF-1 and MRF4 response is capable with muscle fiber hypertrophy and provides preliminary support for the speculation outlined above. An explanation for fiber type-specific hypertrophy within this model is not available. However, different fiber types can exhibit distinct patterns of mRNA expression (24, 64) and it is possible that the current gene data are the sole result of changes within the MHC IIA fiber population.

HSP 72 and MT-2A, two genes that are likely part of a concerted protective effort during/following exercise (12, 40, 43), were more responsive to the posttaper exercise perturbation. Considering that race performance improved along with MHC IIA gastrocnemius fiber size and function, we suspect that the mRNA response observed with taper represents a positive adjustment to the postexercise molecular environment. Interestingly, trained skeletal muscle is reportedly less transcriptionally sensitive to exercise (13, 49). In contrast, we found that an arguably “better-conditioned” muscle following taper displayed an exaggerated postexercise gene response (3 GOI were more responsive than pre taper). It may be that the relative transcriptional insensitivity of trained muscle is exacerbated by heavy periods of training, thus becoming even less sensitive, and that this effect is reversed with taper. Regardless, these gene data are the first gathered on endurance-trained vs. tapered muscle in competitive athletes, and further research is required to better understand the implications of our findings.

Maximal and submaximal cardiovascular physiology and muscle enzyme activity (citrate synthase) were evaluated before and 10 days after taper. The observation that aerobic capacity (V\textsubscript{O\textsubscript{2max}}) was maintained in the present study concurs with a large panel of comparable investigations, all reporting that V\textsubscript{O\textsubscript{2max}} either remains the same (17, 20-22, 48, 51, 56, 60) or improves (25, 35, 37) with taper. From these data, it is clear that the training performed during taper is sufficient to prevent the decline in V\textsubscript{O\textsubscript{2max}} symptomatic of detraining (8). Independent of V\textsubscript{O\textsubscript{2max}}, taper has been shown to improve running economy (decreased O\textsubscript{2} consumption at a set running velocity) (20, 22). However, in agreement with others (17, 21, 32), we found that running economy is maintained with taper. There are no obvious explanations for the varied findings, but they are perhaps related to disparities in talent level, previous training, the speed at which running economy was assessed, the characteristics of the taper regimen itself, or the 10-day delay between the end of taper and the treadmill testing in the present study. Similar to the whole body measures, there was no change in citrate synthase, indicating that the aerobic potential of the muscle was also maintained with taper. Collectively these data strongly support that overall aerobic function was at least maintained during the 3-wk taper.

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

<table>
<thead>
<tr>
<th>Stage</th>
<th>Time</th>
<th>VO\textsubscript{2}, ml·kg\textsuperscript{-1}·min\textsuperscript{-1}</th>
<th>VO\textsubscript{2}, l/min</th>
<th>V\textsubscript{E}, l/min</th>
<th>RER</th>
<th>HR, Beats/min</th>
<th>%V\textsubscript{O\textsubscript{2max}}</th>
</tr>
</thead>
<tbody>
<tr>
<td>16.1 km/h</td>
<td>Before</td>
<td>55.6 ± 0.1</td>
<td>3.68 ± 0.05</td>
<td>82.0 ± 0.2</td>
<td>0.96 ± 0.01</td>
<td>175 ± 0</td>
<td>80 ± 1%</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>56.0 ± 0.1</td>
<td>3.66 ± 0.05</td>
<td>81.4 ± 0.5</td>
<td>0.98 ± 0.02</td>
<td>172 ± 0</td>
<td>81 ± 1%</td>
</tr>
<tr>
<td>Maximal</td>
<td>Before</td>
<td>69.7 ± 0.1</td>
<td>4.61 ± 0.07</td>
<td>137.1 ± 0.3</td>
<td>1.17 ± 0.02</td>
<td>193 ± 0</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>After</td>
<td>69.1 ± 0.1</td>
<td>4.53 ± 0.08</td>
<td>135.7 ± 0.5</td>
<td>1.18 ± 0.02</td>
<td>192 ± 0</td>
<td>—</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SE. VO\textsubscript{2}, oxygen consumption; V\textsubscript{E}, ventilation; RER, respiratory exchange ratio; HR, heart rate; V\textsubscript{O\textsubscript{2max}}, maximal VO\textsubscript{2}; Before, before taper; After, after taper.
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


