Single muscle fiber contractile properties of young competitive distance runners

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Harber M, Trappe S. Single muscle fiber contractile properties of young competitive distance runners. J Appl Physiol 105: 629–636, 2008. First published June 5, 2008; doi:10.1152/japplphysiol.00995.2007.—The purpose of this investigation was to characterize the contractile properties of individual slow- and fast-twitch myofibers from highly trained distance runners. Muscle biopsies were obtained from the gastrocnemius of eight competitive runners (Run) and eight recreationally active individuals (Rec). Slow-twitch [myosin heavy chain (MHC) I] and fast-twitch (MHC IIa) myofibers were isolated and analyzed for diameter (µm), peak force (Pp; mN), unloaded contraction velocity (Vc; fiber lengths/s), and power. Maximum oxygen uptake was higher (P < 0.05) in Run (71 ± 1 vs. 47 ± 2 ml·kg⁻¹·min⁻¹). Diameter of MHC I and MHC IIa fibers from Run subjects was ~20% greater (P < 0.05) than Rec. Peak force of the MHC IIa fibers was 31% higher (P < 0.05) in Run, whereas Pp of MHC I fibers was not different between groups. No differences for specific tension (Pp/cross-sectional area) were present between groups for either fiber type. Vc was higher (P < 0.05) in MHC I (+70%) and MHC IIa (+18%) fibers from Run subjects. In vitro peak absolute power (µN·s⁻¹) of both fiber types was greater (P < 0.05) in Run (131 and 85% for MHC I and MHC IIa, respectively). Additionally, normalized power (W/I) of the MHC I fibers was 64% higher in Run, whereas no differences were noted for normalized power of MHC IIa fibers. These data indicate that highly trained endurance runners have elevated contraction velocity in both slow- and fast-twitch myofibers. These characteristics of the fast-twitch muscle fibers have not been previously reported in competitive endurance athletes and may contribute to the high level of running performance in these athletes.

myosin heavy chain; exercise; endurance

SCIENTISTS HAVE LONG BEEN intrigued by the physiological attributes that allow elite athletes to achieve high levels of physical performance. Initial studies focused on describing the cardiovascular variables of champion athletes (1, 9, 23, 35). The introduction of the needle biopsy technique (2) led to an intense interest in the characterization of the skeletal muscle properties of exceptional athletes and how these variables are altered with endurance exercise (8, 12, 19, 37, 40). Technological advances have allowed further description of skeletal muscle’s adaptability to exercise training. Examining the chemically skinned isolated slow- and fast-twitch myofibers provides insights into the contractile functional properties of the muscle fiber independent of factors such as motor unit recruitment, muscle architecture, and protein heterogeneity that influence in vivo muscle function (4, 29, 53). Over the past decade, it has been shown that the contractile mechanics [i.e., contraction velocity, specific tension [peak force (Pp)/cross-sectional area (CSA)], power] at the myofiber level are sensitive to diverse alterations in physical activity patterns, including unloading (43, 47, 50) and various exercise-training interventions (20, 22, 28, 46, 51). From these studies, it appears that the pattern of adaptations in contractile function at the cellular level is specific to the mode of activity.

Resistance training is well characterized for its impact on muscle size and function. Adaptations at the cellular level reveal that the improvements in contractile function with resistance training can be primarily attributed to myofiber hypertrophy (39, 51). In contrast, endurance-type exercise training appears to alter the contractile mechanics intrinsic to the muscle tissue (i.e., contraction velocity, Pp/CSA normalized, power). Initial reports showed that endurance run training increased contraction velocity at the whole muscle and myofiber level in rodent soleus muscle (15, 38). Our laboratory has recently shown that marathon training improves peak and normalized myofiber power production of gastrocnemius muscle fibers in novice runners (46). This training induced increase in power production despite a decrease in myofiber size and was manifest through increases in myofiber contraction velocity and specific force. Therefore, unlike resistance training, it appears that endurance run training remodels the contractile mechanics of individual myofibers to optimize function Additionally, a 4-wk reduction in training volume decreases contraction velocity and peak power production of slow-twitch myofibers from the gastrocnemius of collegiate runners (20). Interestingly, a 28-day reduction in training volume in collegiate swimmers increases contraction velocity of both slow- and fast-twitch myofibers and improves peak power production of the fast-twitch myofibers from the deltoid muscle (41). These studies demonstrate that the contractile functional properties of isolated myofibers are pliable to alterations in training loads.

Despite the previous studies demonstrating that alterations in training throughout a competitive season can influence myofiber contractile function in endurance athletes, no study has characterized contractile function of slow- and fast-twitch myofibers from young competitive runners in relation to a control group. Investigating highly trained competitive athletes would provide novel insights into the myocellular contractile profile of individual slow- and fast-twitch muscle fibers. Given that these athletes have been engaged in many years of training, the chronic training may result in adaptations in the muscle tissue that are not apparent with short-term training programs. Additionally, it is likely that competitive runners can tolerate higher training loads, which may also result in unique adaptations such as alterations in contraction velocity and peak power.
of fast-twitch fibers. Recent evidence suggest that examining populations that have been exposed to extreme training loads may reveal adaptations at the cellular level that are not observed using short-term (<13 wk) training interventions (10). Therefore, the intent of this investigation was to further characterize myofiber contractile function of individual muscle fibers from competitive distance runners. We hypothesized that myofibers from highly trained competitive runners with many years of run training experience will display a higher contraction velocity and produce greater peak power compared with age-matched recreationally active individuals.

METHODS

Subjects

Sixteen individuals volunteered for participation and were divided into two groups based on their distance running background and aptitude (Table 1). Eight subjects (n = 8 men) were classified as highly trained competitive runners (Run). These individuals were collegiate varsity cross-country athletes and were examined before the fall competitive season and ∼12 wk after the conclusion of the spring competition season. For the 8 wk before testing and sample collection, subjects averaged 95 ± 2 km/wk of total running with ∼5% of the total volume occurring above the anaerobic threshold (i.e., tempo runs). Data from a subset (n = 5) of these individuals have been presented previously (20). The other eight individuals (n = 8; 4 men, 4 women) were classified as recreationally active (Rec) with limited run training experience. Therefore, the intent of this investigation was to further characterize myofiber contractile function of individual muscle fibers from competitive distance runners. We hypothesized that myofibers from highly trained competitive runners with many years of run training experience will display a higher contraction velocity and produce greater peak power compared with age-matched recreationally active individuals.

Table 1. Subject characteristics for recreational and highly trained competitive runners

<table>
<thead>
<tr>
<th></th>
<th>Rec</th>
<th>Run</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Age, yr</td>
<td>22±1</td>
<td>21±1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>176±2.4</td>
<td>177±2.2</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72±4</td>
<td>65±3</td>
</tr>
<tr>
<td>VO2max, l/min</td>
<td>3.4±0.3</td>
<td>4.7±0.2*</td>
</tr>
<tr>
<td>VO2max, ml·kg⁻¹·min⁻¹</td>
<td>47±2</td>
<td>71±1*</td>
</tr>
<tr>
<td>Training history, yr</td>
<td>NA</td>
<td>8±1</td>
</tr>
<tr>
<td>Training volume, km/wk</td>
<td>&lt;25</td>
<td>95±2</td>
</tr>
<tr>
<td>Average training pace, km/h</td>
<td>NA</td>
<td>15.6±0.8</td>
</tr>
<tr>
<td>8-km run time, mins</td>
<td>NA</td>
<td>25:51</td>
</tr>
</tbody>
</table>

Values are mean ± SE; n, no. of subjects. Rec, recreational runners; Run, highly trained competitive runners; VO2max, maximal oxygen consumption; NA, not applicable. *P < 0.05 compared with Rec.
For each single muscle fiber experiment, a fiber with a compliance (calculated as fiber length (FL) divided by y-intercept) >10% and/or a decrease in $P_o$ of >10% was discarded and not used for analysis. The within-fiber test/retest of a single muscle fiber in our laboratory for the measurements of diameter, $P_o$, contractile velocity [maximal unloaded shortening velocity ($V_{o\max}$)], and power were <1%.

**Single Muscle Fiber Analysis**

Individual muscle fibers were analyzed for diameter, $P_o$, $V_o$, and power characteristics. Detailed descriptions and illustrations of these procedures have been presented in our laboratory’s previous work (44, 48).

**Single fiber diameter.** A video camera (model DXC-107A, Sony CCD-IRIS, Tokyo, Japan) connected to the microscope and interfaced to a computer allowed viewing on a computer monitor 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 ($<5$ s). Fiber width (diameter) was determined at three points along the length of the captured computer image using public domain software (NIH Image version 1.61) and averaged to provide a mean diameter measurement. For the fiber size-dependent variables (i.e., $P_o$/CSA and normalized power), CSA was determined with the assumption that the fiber forms a circular shape while suspended in air.

**$P_o$, determination.** The output of the force 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 and then the fiber was maximally activated in pCa 4.5 solution. $P_o$ was determined in each fiber by computer subtraction of the force baseline from the peak in the pCa 4.5 solution. $V_o$, determination. Fiber $V_o$ was measured by the slack test technique as described by Edman (11). Fibers were transferred from resting state (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 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 2.50 μm.

**Power, determination.** Submaximal isotonic load clamps were performed on each fiber for determination of force-power parameters. Each fiber segment was fully activated and subjected to a series of three isotonic load steps. The procedure was performed at various loads so that each fiber was subjected to a total of 15–18 isotonic contractions. $P_o$ and $V_o$ data points derived from the isotonic contractions were fit using the Hill equation (24). Only individual experiments with $r^2 \geq 0.98$ were included for analysis. Fiber power was calculated from the fitted force-power velocity parameters [$P_o$, shortening velocity ($V_{o\max}$), and $a/P_o$ where $a$ is a force constant]. Absolute peak power ($\mu\text{N}\cdot\text{FL}\cdot\text{s}^{-1}$) was defined as the product of force (in μN) and $V_{o\max}$ (in FL/s) while normalized power (W/I) was defined as the product of normalized force, (i.e., $P_o$/CSA) and $V_{o\max}$ (in FL/s).

**Myosin Heavy Chain Isoform Composition**

Following the single muscle fiber physiological measurements, each fiber was solubilized in 80 μl of 10% SDS sample buffer and stored at −20°C until assayed. To determine the myosin heavy chain (MHC) composition, fibers were run on a Hoefer SE 600 gel electrophoresis system that consisted of a 3.5% (wt/vol) acrylamide stacking gel with 5% separating gel at 4°C. Follow ing gel electrophoresis, gels were silver stained as described by Giulian et al. (17). MHC isoforms were identified according to final relative migration position from the SDS-PAGE/silver staining. The MHC were categorized as MHC I, Ila, IIX, I/Ila, I/Ila/IIX, I/IIX, and Ia/IIX.

**Single Fiber Composite Values**

To account for differences in MHC profile between the two groups, we implemented a weighted single fiber value system based on the composition of the fibers studied for each subject because our laboratory and others have previously performed (30, 42, 47). The resulting value for a given variable represents a composite value that theoretically reflects the average for the entire muscle (in this case the gastrocnemius). We recognize that these calculations are theoretical and make the assumption that the MHC profile of the fibers we randomly selected is representative of the whole muscle. To support this assumption, the MHC profile of the fibers selected for physiological experiments is in close agreement with a more comprehensive MHC analysis performed in our laboratory in similar populations (20, 21, 46). An average of 20 ± 1 and 22 ± 1 fibers per subject for Rec and Run, respectively, were used to determine composite values.

**Statistical Analysis**

Descriptive characteristics and composite values were analyzed with an unpaired Student’s t-test. Single muscle fiber physiological variables (diameter, $P_o$, $P_o$/CSA, $V_o$, $V_{o\max}$, absolute power, normalized power) were analyzed using an ANOVA with nested means. The number of studied fibers for a particular individual were nested to represent a mean for MHC I and MHC Ila fibers for each subject. This mean was then used to calculate the group mean and SE. Because the minimal number of hybrid and pure MHC IIX present in these subjects, analyses were restricted to MHC I and Ila fibers. Significance was set at $P < 0.05$, and a Student-Newman-Keuls post hoc test was used when significance was noted. All data are presented as means ± SE.

**RESULTS**

**Subjects**

Subject characteristics for Run and Rec groups are presented in Table 1. Both absolute (l/min) and relative (ml·kg⁻¹·min⁻¹) maximal oxygen consumption were higher in the Run group.

**Single Fiber Physiological Experiments and MHC Composition**

A total of 333 fibers from the gastrocnemius were studied as part of the single muscle fiber physiology experiments (Table 2). On average, 20 and 22 fibers per subject were analyzed for Rec and Run, respectively. Of the fibers examined for Run, 65% were identified as MHC I and 32% were MHC Ila. For Rec, 50% of the fibers examined were MHC I, 38% were MHC Ila, and 12% were identified as hybrids. No MHC IIX fibers were identified according to final relative migration position from the SDS-PAGE/silver staining. The MHC were categorized as MHC I, Ila, IIX, I/Ila, I/Ila/IIX, I/IIX, and Ia/IIX.

<table>
<thead>
<tr>
<th>MHC I</th>
<th>MHC Ila</th>
<th>MHC IIX</th>
<th>I/Ila</th>
<th>I/Ila/IIX</th>
<th>I/IIX</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Rec</td>
<td>81</td>
<td>50</td>
<td>11</td>
<td>7</td>
<td>61</td>
<td>38</td>
</tr>
<tr>
<td>Run</td>
<td>112</td>
<td>65</td>
<td>0</td>
<td>0</td>
<td>55</td>
<td>32</td>
</tr>
</tbody>
</table>

MHC, myosin heavy chain; n, no. of fibers studied; %, percentage of the total fibers consisting of that fiber type.
fibers were studied, which is representative of these populations (20, 21, 46).

**Single Fiber Diameter**

Single muscle fiber diameter for MHC I and MHC IIa for Rec and Run are presented in Fig. 1. Diameter of both MHC I and MHC IIa fibers was ~20% greater ($P < 0.05$) in Run compared with Rec.

**Single Fiber Power**

Force-power curves for both MHC I and MHC IIa fibers from both groups are presented in Fig. 2. Peak absolute power of the MHC I and MHC IIa fibers was 131 and 85% greater ($P < 0.05$), respectively, in Run. Peak normalized power of the MHC I fibers was 64% greater ($P < 0.05$) for Run, and MHC IIa peak normalized power was not different between groups (Fig. 3).

**Single Fiber $P_o$ and $P_o$/CSA**

Single muscle fiber $P_o$ and $P_o$/CSA are presented in Table 3. No differences for MHC I $P_o$ were noted between groups. $P_o$/CSA of the MHC I fibers showed a trend ($P = 0.07$) toward being higher in Rec. MHC IIa fibers from Run generated greater ($P < 0.05$) $P_o$ than Rec. The greater $P_o$ of the MHC IIa fibers in Run was attributable to the greater fiber diameter because $P_o$/CSA of the MHC IIa fibers was not different between the groups.

**Single Fiber $V_o$**

Single muscle fiber $V_o$ is presented in Table 3. Contraction velocity of both MHC I and MHC IIa fibers was higher ($P < 0.05$) for Run compared with Rec. Specifically, MHC I and MHC IIa fibers contracted 70 and 18%, respectively, faster in Run.

**Force-$V_{max}$ Parameters**

MHC I $V_{max}$ was higher ($P < 0.05$) in Run (0.94 ± 0.06 FL/s) compared with Rec (0.51 ± 0.09 FL/s), whereas no differences were noted in $V_{max}$ of MHC IIa fibers (2.70 ± 0.32 and 2.40 ± 0.22 FL/s for Run and Rec, respectively). $aP_o/V_o$, which describes the curvature of the force-$V_{max}$ curve, was not different between groups for MHC I (0.041 ± 0.003 and 0.049 ± 0.007 for Run and Rec, respectively) or MHC IIa (0.048 ± 0.006 and 0.042 ± 0.006 for Run and Rec, respectively) fibers. M, defined as the percentage of $P_o$ at which peak power occurs, for MHC I fibers was 0.16 ± 0.01 and 0.17 ± 0.01 for Run and Rec respectively. M values for MHC IIa fibers were 0.17 ± 0.01 and 0.16 ± 0.01 for Run and Rec, respectively. No statistical differences were noted for M.
and peak power were improved in the slow-twitch fibers only.

Specific tension; $V_o$, contractile velocity ($V_o$), absolute power, and peak normalized power (Nrm Power) in MHC I and MHC IIa myofibers. *$P < 0.05$, Run vs. Rec.

DISCUSSION

To gain further insights into the modulation of skeletal muscle fiber contractility by physical activity, we compared the contractile mechanics of individual chemically skinned slow- and fast-twitch myofibers from highly trained competitive distance runners to age-matched recreationally active individuals. The primary finding from this investigation was that $V_o$ was higher in both fiber types of the competitive runners. The higher contraction velocity coupled with a larger fiber diameter resulted in greater peak absolute power production of both slow- and fast-twitch muscle fibers in the distance runners. Furthermore, after correction for fiber size, peak power of the slow-twitch myofibers remained higher in the competitive runners (Fig. 3). These findings provide a more comprehensive single fiber contractile profile of endurance athletes that have been chronically exposed to high-intensity training.

A novel aspect of the present data is the significant enhancement in contractile mechanics of both slow- and fast-twitch myofibers of the highly trained competitive runners. In our laboratory’s recent investigation examining the effects of marathon training on single muscle fiber contractile function, $V_o$, and peak power were improved in the slow-twitch fibers only (46). Studies examining the influence of variations in training volume on myofiber contractile function in competitive swimmers have demonstrated the plasticity of the fast-twitch fiber population. A short-term (10-day) period of intense swim training reduced fast-twitch myofiber size and contraction velocity (14). In contrast, a planned reduction in training volume and intensity designed to elicit optimal performance, robustly improved fast-twitch myofiber size, $V_o$, and peak power production in highly trained swimmers (41). These studies, combined with the current data, indicate that the fast-twitch myofibers are responsive to endurance-type activity and play a crucial role in modulating muscle performance in highly trained competitive endurance athletes.

A novel finding from the present investigation was that $V_o$ of the fast-twitch myofibers was elevated in highly trained competitive runners because previous reports have concluded that run training only alters $V_o$ of slow-twitch fibers (37, 44, 51). Malisoux et al. (28) recently reported that plyometric training increased shortening velocity of both slow- and fast-twitch myofibers, and to our knowledge, this is the only investigation to report alterations in contraction velocity of both fiber types with a training intervention. An increase in contraction velocity with endurance training is somewhat paradoxical because endurance training is associated with a shift toward a slower myofiber phenotype (46). Indeed, the highly trained runners in the present study had a greater proportion of slow-twitch myofibers compared with the recreational runners (20, 21, 46).

When examining the composite value for contraction velocity, which takes into account the muscle fiber type distribution to derive a theoretical contraction velocity for the whole muscle (47), the highly trained runners had a 16% greater contraction velocity. The higher $V_o$ with endurance training was one of the key determinants of the elevated power production at the myofiber level, particularly in the slow-twitch myofibers. These results lend support to the notion that endurance training, unlike resistance training, alters $V_o$ at the myofiber level.

In context of the available literature, the greater fiber diameters observed in our Run group is not surprising. It has been suggested that prolonged endurance exercise training results in selective hypertrophy of the slow-twitch myofibers in young competitive runners (36). The present data show that both slow- and fast-twitch muscle fiber diameter from Run was ~20% greater than Rec. Our findings are supported by previous investigations of elite and highly trained competitive runners (7, 8). Longitudinal studies examining the influence of

Table 3. Peak force, specific tension, and unloaded shortening velocity, for MHC I and IIa myofibers

<table>
<thead>
<tr>
<th>Value</th>
<th>Rec</th>
<th>Run</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_o$, mN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MHC I</td>
<td>$0.48 \pm 0.04$</td>
<td>$0.58 \pm 0.06$</td>
<td>0.14</td>
</tr>
<tr>
<td>MHC IIa</td>
<td>$0.58 \pm 0.04$</td>
<td>$0.76 \pm 0.05$</td>
<td>0.01</td>
</tr>
<tr>
<td>$P_o$/CSA, kN/m²</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MHC I</td>
<td>91±7</td>
<td>75±3</td>
<td>0.07</td>
</tr>
<tr>
<td>MHC IIa</td>
<td>102±11</td>
<td>89±5</td>
<td>0.29</td>
</tr>
<tr>
<td>$V_o$, FL/s</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MHC I</td>
<td>$0.97 \pm 0.04$</td>
<td>$1.65 \pm 0.08$</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>MHC IIa</td>
<td>$3.16 \pm 0.14$</td>
<td>$3.72 \pm 0.24$</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Values are means ± SE. $P_o$, peak force; CSA, cross-sectional area; $P_o$/CSA, specific tension; $V_o$, unloaded shortening velocity; FL, fiber lengths.
Table 4. Single muscle fiber composite values for Rec and Run

<table>
<thead>
<tr>
<th></th>
<th>Diameter, μm</th>
<th>P0, mN</th>
<th>P0/CSA, kN·m²</th>
<th>V0, FL/s</th>
<th>Power, μN·FL·s⁻¹</th>
<th>Nrm Power, W/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rec</td>
<td>86±4</td>
<td>0.53±0.04</td>
<td>95±8</td>
<td>2.01±0.13</td>
<td>18.3±1.9</td>
<td>3.2±0.4</td>
</tr>
<tr>
<td>Run</td>
<td>101±5</td>
<td>0.64±0.04</td>
<td>80±4</td>
<td>2.34±0.08</td>
<td>28.9±2.7</td>
<td>3.5±0.4</td>
</tr>
<tr>
<td>%Δ</td>
<td>+17</td>
<td>+21</td>
<td>−16</td>
<td>+16</td>
<td>+58</td>
<td>+9</td>
</tr>
<tr>
<td>P Value</td>
<td>0.02</td>
<td>0.07</td>
<td>0.13</td>
<td>0.04</td>
<td>0.01</td>
<td>0.56</td>
</tr>
</tbody>
</table>

Composite value represents a theoretical value based on the MHC composition for each subject that reflects the average muscle fiber in gastrocnemius muscle. Power, peak absolute power; Nrm Power, normalized power; %Δ, percent difference (Run relative to Rec).

endurance training on muscle fiber size have produced equivocal findings (18, 25, 46). Our laboratory recently reported that a run training program designed to prepare novice runners to complete the marathon distance resulted in significant reductions in size of both fiber types (46). Presumably, reductions in fiber size should facilitate the transport of oxygen and metabolites into the muscle cell. The functional importance of larger muscle fibers in highly trained competitive runners is unclear. Furthermore, because of the cross-sectional nature of the present study, it cannot be determined whether the greater fiber diameter observed in the trained runners is attributed to several years of high volume and intense training, a genetic component that predisposed these individuals to success in endurance running, or a combination of these factors. Coggan et al. (5) reported that a long-duration (9–12 mo) training program increased CSA of both slow- and fast-twitch muscle fibers in older (64 yr) men and women. Therefore, the reduction in myofiber diameter with marathon training that our laboratory previously reported may represent a transient adaptation that precedes an eventual induction of muscle hypertrophy. Widrick et al. (52) reported that myofibers from former elite distance runners (age 44 yr) were smaller than age-matched sedentary control subjects. Interestingly, these same athletes had larger slow- and fast-twitch muscle fibers when they were younger (age 26 yr) and training competitively at elite levels (8). Therefore, the myofiber atrophy reported by Widrick et al. over a 25-yr period may be related to the aging process and/or reductions in training volume [139 ± 11 (1970) vs. 69 ± 7 km/wk (1993)] and intensity over time.

In vivo skeletal muscle power production is often considered the most relevant physiological variable of contractile function because it incorporates force generation and V0 and is a strong predictor of functional mobility and performance (16, 32, 33). In the present study, slow- and fast-twitch fibers from the competitive runners demonstrated a greater absolute peak power production compared with the recreational runners. When normalized to fiber size, peak power remained higher in the slow-twitch fibers of the competitive runners. Several investigations have reported alterations in single muscle fiber power output with modifications in physical activity patterns (20, 28, 39, 41, 45, 46, 48, 51). It is apparent from these studies that the mechanisms underling the modulations in myofiber power production are dependent on the mode of exercise training. The resistance training-induced improvements in power production are directly related to myofiber hypertrophy, which results in an enhanced force production without altering P0/CSA or V0 (39, 45, 51). In contrast, endurance-type activity can impact muscle function by altering the intrinsic contractile mechanics chemically skinned individual muscle fibers, independent of fiber size. Our laboratory has previously shown that a planned reduction in training load after a period of intense training (i.e., taper phase) in collegiate swimmers improves both peak absolute and normalized power production of the fast-twitch myofibers through increases in myofiber diameter and an elevation in V0 (41). Additionally, periods of increased training load (i.e., increasing intensity while maintaining volume) in collegiate runners reduced power production of the slow-twitch myofibers through reductions in myofiber size and contraction velocity (20). Recently, our laboratory examined the influence of a 13-wk marathon training program on single muscle fiber contractility in a group of novice runners (46). Despite reductions in fiber diameter, peak absolute and normalized power of the slow-twitch myofibers were enhanced in response to the training. Furthermore, peak absolute and normalized power production of the fast-twitch myofibers were improved after a 3-wk taper phase. Thus the present results, in combination with previous studies, suggest that improving power production at the cellular level is an adaptation that has been optimized to the improvement of distance running performance.

In the present investigation, we examined the contractile properties of isolated chemically skinned muscle fibers to further characterize skeletal muscle function in competitive runners. A strength of this approach is the assessment of fiber type-specific contractile mechanics of the muscle tissue independent of factors such as motor unit recruitment, muscle architecture, and protein heterogeneity that influence in vivo muscle function (4, 29, 53). Although alterations in contractile function at the whole muscle level are reflective of myofiber contractile behavior (41, 45, 48, 51), it is difficult to directly compare values obtained in isolated muscle fibers with values obtained in vivo. In addition to the factors previously mentioned, isolated myofibers are examined in vitro at 15°C, which alters peak tension, V0, and power production (4, 27, 34). This approach provides information about the contractile mechanics intrinsic to the myofiber in a fiber type-specific manner that provides insights into the cellular factors that may influence whole muscle performance.

In summary, considerable interest has been paid to deciphering the physiological attributes that define elite athletes. This is the first study to characterize contractile function at the cellular level in young highly trained competitive runners. In addition to the greater cardiovascular capacity (+51%), we also observed an enhanced performance of the contractile properties (size, speed, and power) of the slow- and fast-twitch muscle fibers from the highly trained competitive runners (Fig. 3). These findings are interesting in that endurance athletes do not typically exhibit high levels of whole muscle power production (6, 49). The disconnect between myofiber and whole muscle contractile function is likely related to differences in muscle
fibertype composition because endurance athletes typically display high amounts of slow-twitch muscle fibers, which produce drastically less peak power than fast-twitch fibers (Fig. 2). Our findings reveal that young competitive distance runners demonstrate unique myofiber contractile functional properties, specifically a higher \( V_o \) of the fast-twitch fibers, compared with recreational runners that have not been reported previously and are not evident after 13-wk of marathon training (46). Although the relationship between contractile properties at the single fiber and whole muscle level are not directly quantifiable in the present study, these contractile characteristics may contribute to the high level of running performance in these endurance athletes.

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