Resistance training improves single muscle fiber contractile function in older women

SCOTT TRAPPE, MICHAEL GODARD, PHILIP GALLAGHER, CHAD CARROLL, GREG ROWDEN, AND DAVID PORTER. Resistance training improves single muscle fiber contractile function in older women. Am J Physiol Cell Physiol 281: C398–C406, 2001.—The purpose of this study was to 1) examine single cell contractile mechanics of skeletal muscle before and after 12 wk of progressive resistance training (PRT) in older women (n = 7; 74 ± 2 yr) and 2) to compare these results to our previously completed single cell PRT work with older men (n = 7; 74 ± 2 yr) (Trappe S, Williamson D, Godard M, Porter D, Rowden G, and Costill D. J Applied Physiol 89:143–152, 2000). Knee extensor PRT was performed 3 days/wk at 80% of one-repetition maximum. Muscle biopsies were obtained from the vastus lateralis before and after the PRT. Chemically skinned single muscle fibers (n = 313) were studied at 15°C for peak tension (P_o), unloaded shortening velocity (V_o), and power. Due to the low number of hybrid fibers identified post-PRT, direct comparisons were limited to MHC I and IIa fibers. Muscle fiber diameter increased 24% (90 ± 2 to 112 ± 6 μm; P < 0.05) in MHC I fibers with no change in MHC IIa fibers. P_o increased (P < 0.05) 33% in MHC I (0.76 ± 0.04 to 1.01 ± 0.09 mN) and 14% in MHC IIa (0.73 ± 0.04 to 0.83 ± 0.05 mN) fibers. Muscle fiber V_o was unaltered in both fiber types with PRT. MHC I and IIa fiber power increased (P < 0.05) 50% [11 ± 2 to 17 ± 2 μN-fiber length (FL)-s^{-1}] and 25% (40 ± 8 to 51 ± 6 μN-FL-s^{-1}), respectively. However, when peak power was normalized to cell size, no pre- to postimprovements were observed. These data indicate that PRT in elderly women increases muscle cell size, strength, and peak power in both slow and fast muscle fibers, which was similar to the older men. However, in contrast to the older men, no change in fiber V_o or normalized power was observed in the older women. These data suggest that older men and women respond differently at the muscle cell level to the same resistance-training stimulus.

aging; skeletal muscle; myofiber; myosin heavy chain

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same protocol and procedures that we used with the older men. Specifically our goals were twofold: 1) to examine single cell contractile properties before and after progressive resistance training (PRT) in older women and 2) to examine the influence of gender on single fiber contractile properties with PRT in older adults. We hypothesized that 1) the PRT will cause an increase in single fiber diameter, peak tension ($P_o$), maximum shortening velocity ($V_o$), and power of the slow MHC I and fast MHC IIa muscle fibers; 2) the MHC I muscle fibers will be more affected than the MHC IIa fibers in response to the PRT program (this is based on our findings with the older men); 3) similar improvements in whole muscle one repetition maximum (1-RM) strength will be observed among the older men and women; and 4) no gender-related differences will exist in the contractile properties of the MHC I and IIa muscle fibers before or after the PRT program. These hypotheses were proposed and initiated based on our work with older men (34) and before the publication of Frontera et al. (13).

**METHODS**

The methodologies, procedures, and instrumentation utilized in this investigation were identical to those previously used with the older men (34). All muscle tissue analysis for single cell contractile properties was performed by the same investigator using the single fiber physiology station that was used to study the skeletal muscle tissue from the older men.

**Subjects**

Seven older female volunteers participated in this investigation. Their age, height, and mass were $74 \pm 2$ yr, $162 \pm 3$ cm, and $71 \pm 4$ kg, respectively. Each participant underwent a thorough physical examination, which included a medical history, resting and exercise electrocardiogram, blood pressure, and orthopedic evaluation before initiation of the resistance training program. These volunteers were nonobese ($<28$ kg/m$^2$), normotensive, nonsmokers, nonmedicated (no hormone replacement therapy), and healthy as judged by physical examination. The volunteers were sedentary and had never performed resistance training before this study. The experimental protocol was approved by the Human Research Committees of Ball State University and Ball Memorial Hospital before data collection.

**Resistance Training Program**

All subjects performed a 12-wk PRT program designed to strengthen the vastus lateralis (6, 12, 34). This PRT program consisted of bilateral isometric leg extensions at 80% of their concentric 1-RM. Subjects raised and lowered the weight using a 2- to 3-s interval. The 1-RM was reevaluated every 2 wk, and the weight was adjusted accordingly to ensure that intensity was maintained at 80%. Subjects performed the PRT three times per week (for a total of 36 training sessions) with at least $24-48$ h between training sessions. Each training session consisted of 2 sets of 10 repetitions and a third set to failure. There was a 2-min rest period between each set. Each PRT session was preceded by a warm-up and cool-down period of 10 min of stationary cycling at low resistance (50 watts) and slow speed, as well as stretching of the muscle groups involved in the strength measurements.

**Muscle Biopsy**

A muscle biopsy sample ($\sim 100$ mg) was obtained from the vastus lateralis 1 wk before initiation of the PRT program (3). A second muscle biopsy was obtained after 12 wk of PRT from the vastus lateralis 3 days after the last resistance training session. Each muscle sample was sectioned longitudinally into several portions and placed in cold skinning solution and stored at $-20^\circ$C for later analysis of single muscle fiber physiology. After a single fiber experiment, the single fibers were analyzed for MHC and myosin light chain (MLC) protein expression as described in Gel Electrophoresis. After each muscle biopsy sample, all single fiber contractile measurements were completed on fresh tissue within a 4-wk period.

**Single Fiber Physiology Studies**

The cellular studies on the individual muscle fibers were carried out on a single fiber physiology station. On the day of an experiment, a 2- to 3-mm muscle fiber segment was isolated from a muscle bundle and transferred to an experimental chamber filled with relaxing solution where the ends were securely fastened between a force transducer (Cambridge Technology, Watertown, MA) and a DC torque motor (Cambridge Technology) as described by Moss (25). 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, Tokyo, Japan) so that the fiber could be viewed ($>800$) during an experiment.

Unamplified force and length signals were sent to a digital oscilloscope (Nicolet 310, Madison, WI) enabling fiber performance to be monitored throughout data collection. Analog force and position signals were amplified (Positron Development, Dual Differential Amplifier, 300-DIP2, Ingelwood, CA), converted to digital signals (National Instruments), and transferred to a computer [Micron Electronics, Millennium (Pentium processor), Nampa, IN] for analysis using customized software. Servo-motor arm and isotonic force clamps were controlled using a computer interfaced force-position controller (Positron Development, Force Controller, 300-FC1). The sarcomere spacing for each muscle fiber was adjusted to 2.5 $\mu$m using an eyepiece micrometer. A video camera (Sony CCD-IRIS, DXC-107A, 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 (4, 34, 38). Fiber width was determined at three points along the length of the captured computer image using public domain software (NIH Image v1.60). 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 ($<5$ s). All experiments were performed at $15^\circ$C.

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.

**Single Fiber Analysis**

The individual muscle fibers were analyzed for: 1) $P_o$, 2) maximal $V_o$, and 3) force-velocity-power parameters. Detailed descriptions and illustrations of these procedures have been presented in our previous work (34).
The within fiber test and/or retest of a single fiber in our lab for the measurements for P o, V o, and power are <1%. The coefficient of variation for the force transducer and servomechanical lever mechanism during the 3-yr period we have studied single cell function in older men and women is 1.0 and 1.2%, respectively. Thus we feel confident that the single fiber experiments were repeatable and that the instrumentation was reliable over the course of these studies.

Fiber P o. Resting tension was monitored with the fiber in relaxing solution (pCa 9.0). Subsequently, the fiber was then maximally activated in pCa 4.5 solution. P o was determined in each fiber by computer subtraction of the force baseline from the peak tension in the pCa 4.5 solution.

Fiber V o. Fiber V o was measured by the slack test technique (7). The fiber was fully activated in pCa 4.5 and then rapidly released to a shorter length. Four to six different activation and length steps (each ≥20% of FL) were used for each fiber with the slack distance plotted as a function of the duration of unloaded shortening. Fiber V o (FL/s) was calculated by dividing the slope of the fitted line by the segment length, and the data were normalized to a sarcomere length of 2.5 μm.

Fiber force-velocity and power. Submaximal isotonic load clamps were performed on each fiber for determination of force-velocity parameters. Each fiber segment was fully activated in pCa 4.5 and then subjected to three isotonic load steps. This procedure was performed five to six times at various loads so that each fiber was subjected to a total of 15–18 isotonic contractions.

For the force-velocity relationships, load was expressed as P/P o, where P is the force during load clamping and P o is the peak isometric force developed before the submaximal load clamps. Force and shortening velocity data points derived from the isotonic contractions were fit using the hyperbolic Hill equation (17). Only individual experiments in which r² ≥ 0.98 were included for analysis.

Fiber power was calculated from the fitted force-velocity parameters [P o, maximum velocity (V max), and a/P o]. Absolute power (μN·FL·s⁻¹) was defined as the product of force (in μN) and shortening velocity (in FL/s). Normalized power (kN·m⁻²·FL·s⁻¹) was defined as the product of normalized force, i.e., fiber tension or force and/or fiber CSA and shortening velocity.

Skinning, Relaxing, and Activating Solutions

The skinning solution contained (in mM): 125 K-propionate, 2.0 EGTA, 4.0 ATP, 1.0 MgCl₂, 20.0 imidazole (pH 7.0), and 50% (vol/vol) glycerol. Fibers were kept in this solution for a minimum of 1 day but not longer than 4 wk (14, 18).

The compositions of the relaxing and activating solutions were calculated using an iterative computer program (8). These solutions were adjusted for temperature, pH, and ionic strength using stability constants in the calculations (16). Each solution contained (in mM) 7.0 EGTA, 20.0 imidazole, 14.5 creatine phosphate, 1.0 free Mg²⁺, 4 free MgATP, KCl, and KOH to produce and ionic strength of 180 mM and a pH of 7.0. The relaxing and activating solutions had a free [Ca²⁺] of pCa 9.0 and pCa 4.5, respectively (where pCa = −log Ca²⁺ concentration).

Gel Electrophoresis

After the single muscle fiber physiology measurements, each fiber was solubilized in 80 μL of 1% SDS sample buffer and stored at −80°C until assayed (15). To determine the MHC and MLC 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% (MHC) or 12% (MLC) separating gel at 4°C. After the gel electrophoresis, the gels were silver-stained as described by Guilain et al. (15). A computer-based image analysis system and software (Alpha Innotech, ChemImager 4000, San Leandro, CA) were used to quantify the relative density of each MLC band.

Statistical Analysis

For a given fiber type (MHC) and variable (diameter, P o, V o, power), data were averaged and pooled by subject. Statistical significance of the means for a given variable was assessed using a two-tailed Student t-test. Gender by time effects was analyzed by using a repeated measures ANOVA. A probability of <5% (P < 0.05) was considered to be significant. All data are means ± SE.

Given the small number of hybrid (I/IIa and IIX/IIX) and IIx fibers studied in these experiments (Table 1), they were not included in the analysis. Thus the pre- to postcomparisons of single muscle fiber contractile function are limited to the MHC I and IIa fiber types.

With the muscle samples from the older women, several of the fibers appeared stringy and atrophic under the microscope. These fibers were fragile and often failed or did not meet the inclusion criteria (as outlined in METHODS) when subjected to maximal tension (a total of 19 pre- and 16 post-MHC IIa fibers were excluded from analysis). Gel electrophoresis experiments on these fibers indicated them to express the MHC IIa and IIX/IIX protein (Trappe, unpublished observations). Thus we were not able to do as many successful physiological experiments on the MHC IIa fibers from the older women as we were from the older men. As a result, these data should be interpreted with some caution, as we were not able to study a large population of MHC IIa fibers from the elderly women.

Table 1. Single fiber MHC composition of vastus lateralis fibers from older women before and after resistance training

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
</tr>
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<tbody>
<tr>
<td>n</td>
<td>Percent</td>
<td>Percent</td>
</tr>
<tr>
<td>MHC isoform</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>135</td>
<td>123</td>
</tr>
<tr>
<td>I/IIa</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>IIa</td>
<td>18</td>
<td>21</td>
</tr>
<tr>
<td>IIX/IIX</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>IIx</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Hybrid + IIX</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Total fibers</td>
<td>160</td>
<td>153</td>
</tr>
</tbody>
</table>

n, Fiber count; hybrid, a fiber coexpressing myosin heavy chain (MHC) isoforms (I/IIa and IIX/IIX); Pre, before resistance training; Post, after resistance training.
Table 2. Single fiber MLC composition of vastus lateralis fibers from older women before and after resistance training

<table>
<thead>
<tr>
<th>MLC Isoform</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>MLC1f</td>
<td>NE</td>
<td>NE</td>
</tr>
<tr>
<td>MLC1s</td>
<td>0.48±0.01</td>
<td>0.48±0.02</td>
</tr>
<tr>
<td>MLC2s</td>
<td>0.44±0.02</td>
<td>0.44±0.01</td>
</tr>
<tr>
<td>MLC2f</td>
<td>NE</td>
<td>NE</td>
</tr>
<tr>
<td>MLC3s</td>
<td>0.08±0.01</td>
<td>0.08±0.01</td>
</tr>
<tr>
<td>MLC3f</td>
<td>0.48±0.01</td>
<td>0.48±0.01</td>
</tr>
</tbody>
</table>

Table 2 shows the results of the MLC analysis. There was no difference in the relative content of MLC isoforms in the MHC I and IIa fibers pre- to posttraining. As noted in our previous research (34, 39), the MHC I fibers did not express the MLC1f and MLC2f, while the MHC IIa fibers did not express the MLC1s and MLC2s isoforms. Furthermore, there were no pre-to posttraining differences in the MLC 3:2 ratio. The MLC 3:2 ratio for the MHC I fibers was 0.178±0.018 pretraining and 0.176±0.014 posttraining. For the MHC IIa fibers, the MLC 3:2 ratio was 0.332±0.020 and 0.311±0.022 pre- and posttraining, respectively.

Table 3. Single muscle fiber diameter, peak force, and specific tension of vastus lateralis fibers from older women before and after resistance training

<table>
<thead>
<tr>
<th>Fiber Type</th>
<th>Diameter, μm</th>
<th>P_o, mN</th>
<th>P_o/CSA, kN/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
</tr>
<tr>
<td>MHCI</td>
<td>90±2†</td>
<td>112±6†</td>
<td>0.76±0.04</td>
</tr>
<tr>
<td>MHCIIa</td>
<td>83±2</td>
<td>88±3</td>
<td>0.73±0.04</td>
</tr>
</tbody>
</table>

Values are means ± SE. P_o/CSA, peak force per fiber cross-sectional area. *P < 0.05, pre- to postresistance training; †P < 0.05, MHCI vs. MHCIIa.
Isotonic Contractile Properties

All three parameters describing the force-velocity relationship ($V_{\text{max}}, P_o,$ and $a/P_o$) are shown in Table 3 ($P_o$) and Table 5 ($V_{\text{max}}$ and $a/P_o$). Similar to the fiber $V_o$ findings, there was no change in fiber $V_{\text{max}}$ in either the MHC I or IIa fibers after the training. In general, $V_{\text{max}}$ values were slightly lower than the $V_o$ values. However, the same result (i.e., no change) could be concluded from both variables. As with the $V_o$, the MHC IIa fibers contracted 3.5- to 4-fold faster ($P < 0.05$) than the MHC I fibers.

Composite power curves from MHC I and IIa fibers are illustrated in Figs. 1 and 2. Figure 1 shows the average absolute peak power ($\mu \text{N} \cdot \text{FL} \cdot \text{s}^{-1}$) developed before and after the training. The MHC I fibers increased ($P < 0.05$) their peak power output 5.6 $\mu \text{N} \cdot \text{FL} \cdot \text{s}^{-1}$ (50%), while the MHC IIa fibers increased ($P < 0.05$) their peak power output 10.3 $\mu \text{N} \cdot \text{FL} \cdot \text{s}^{-1}$ (25%). On average, the MHC IIa fibers had peak power values about fivefold greater ($P < 0.05$) than the MHC I fibers.

Figure 2 represents peak power normalized ($\text{kN} \cdot \text{m}^{-2} \cdot \text{FL} \cdot \text{s}^{-1}$) for cell size. When peak power was corrected for cell size, there was no difference pre- to posttraining in power output. Normalized peak power for the MHC I fibers was 1.74 ± 0.21 and 1.68 ± 0.10 kN·m⁻²·FL⁻¹·s⁻¹ pre- to postresistance training, respectively. For the MHC IIa fibers, normalized peak power was 7.35 ± 1.33 and 8.87 ± 1.44 kN·m⁻²·FL⁻¹·s⁻¹ pre- to postresistance training, respectively. Thus the increase in peak power can be attributed to the increase in $P_o$.

Comparison of Older Men and Women

The percent change pre- to posttraining for the major variables (diameter, $P_o$, or $P_o$/CSA, $V_o$, absolute power, and normalized power) studied as part of this investigation and with older men (34) are shown for the MHC I and IIa fibers in Figs. 3 and 4, respectively. For the MHC I fibers, there was no difference among men and women.

### Table 4. Single fiber unloaded shortening velocity of vastus lateralis fibers from older women before and after resistance training

<table>
<thead>
<tr>
<th>Fiber Type</th>
<th>$V_o$, FL/s</th>
<th>%Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHCI</td>
<td>1.17 ± 0.08</td>
<td>1.16 ± 0.08</td>
</tr>
<tr>
<td>MHCIiia</td>
<td>3.71 ± 0.58</td>
<td>3.56 ± 0.35</td>
</tr>
</tbody>
</table>

Values are means ± SE. FL, fiber length; $V_o$, unloaded shortening velocity. *$P < 0.05$, MHC I vs. MHC IIa.

### Table 5. Single fiber contractile properties of vastus lateralis fibers from older women before and after resistance training

<table>
<thead>
<tr>
<th>Fiber Type</th>
<th>$V_{\text{max}}$, FL/s</th>
<th>$a/P_o$</th>
<th>Absolute Peak Power, $\mu \text{N} \cdot \text{FL} \cdot \text{s}^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>MHCI</td>
<td>0.83 ± 0.12</td>
<td>0.025 ± 0.005</td>
<td>11.0 ± 1.6</td>
</tr>
<tr>
<td>MHCIiia</td>
<td>0.96 ± 0.09</td>
<td>0.027 ± 0.001</td>
<td>16.6 ± 2.0†</td>
</tr>
<tr>
<td>MHCIiia</td>
<td>3.70 ± 0.80†</td>
<td>0.035 ± 0.006</td>
<td>40.3 ± 8.0†</td>
</tr>
<tr>
<td>MHCIiia</td>
<td>3.41 ± 0.60†</td>
<td>0.037 ± 0.001</td>
<td>50.6 ± 5.8†</td>
</tr>
</tbody>
</table>

Values are means ± SE. $a$, Force constant; $V_{\text{max}}$, maximal shortening velocity. *$P < 0.05$, pre- to postresistance training; †$P < 0.05$, MHC I vs. MHC IIa.
women in the percent change in fiber size, $P_o$, or $P_o$/CSA. However, the older men had a larger ($P < 0.05$) change in fiber $V_o$, absolute power, and normalized power compared with the older women. Similarly, the MHC IIa fibers of the men and women responded to the same degree except for fiber $V_o$ and absolute power, which increased more ($P < 0.05$) in the older men as a result of the resistance training.

Before the resistance training (baseline), there were significant gender differences in the single muscle fiber contractile properties. The MHC I fibers from the older women were 8% larger ($P < 0.05$), 31% stronger ($P_o$), 38% faster ($V_o$), and 42% more powerful ($P < 0.05$; all listed variables) compared with the older men. For the MHC IIa fibers, no differences were found in cell size or $P_o$ among the sexes. However, the women’s IIa fibers contracted 69% faster ($V_o$; $P < 0.05$) and produced 58% more peak power ($P < 0.05$) compared with the men. No differences in specific tension ($P_o$/CSA) were noted between the men and women.

After the resistance training, several changes occurred in single cell function occurred; however, selected single fiber characteristics were different among the sexes. The women’s MHC I fibers remained larger and stronger ($P_o$) (~12% for both; $P < 0.05$) compared with the older men. In contrast to the baseline profile, the women’s MHC I fibers contracted 22% slower ($P < 0.05$) compared with the men. No differences between the men and women were noted in peak power ($\mu$N·FL·s$^{-1}$) after the PRT program. For the MHC IIa fibers, $V_o$ was unchanged in the women with PRT but still contracted 12% faster ($P < 0.05$) compared with the men. No post-PRT differences in specific tension ($P_o$/CSA) were noted between the men and women.

**DISCUSSION**

The intent of this investigation was to extend our single muscle fiber experiments on older men to include older women. Using the skinned fiber preparation, we were able to examine the contractile properties of single muscle fibers in older women before and after 12 wk of resistance training to determine if gender-related differences existed. The primary finding from this investigation was that older women increased single cell size (MHC I only), $P_o$, and absolute power (MHC I and IIa) after the PRT program. However, in contrast to the older men, there was no increase in fiber $V_o$ or normalized power in the MHC I and IIa fibers.
The findings from this resistance training investigation are interesting, because gender differences were observed in single fiber function but not whole muscle function. The older women improved 56% in whole muscle knee extensor strength compared with 50% for the men (P > 0.05; Ref. 34). Whole muscle size (computed tomography) also increased ~6% in both men and women (S. Trappe, unpublished observations). Furthermore, the 50% increase in muscle strength and 6% increase in muscle size are in agreement with other investigations on older adults using a similar resistance training protocol (1, 5, 6, 9, 12). Thus it can be argued that this protocol was effective in inducing whole muscle strength and size gains with no apparent gender difference. However, the single muscle fiber data clearly show that men and women do not adapt the same to resistance training at the cell level. This suggests that the older men and women have different mechanisms for functional improvement in single cell physiology that are not detected using whole muscle measurements.

Part of the difference in single muscle fiber adaptation to resistance training between the older men and women might be related to single fiber contractile behavior before training (baseline). Before resistance training, the profile of the women’s single fibers showed them to have a functional advantage (especially the MHC I fibers) compared with the men. One possible explanation for these differences may be related to the physical activity and/or lifestyle of the women compared with the men. Although both sexes in our studies were classified as sedentary, it is possible that the women’s daily routine over the past several years (or decades) had shifted to a much slower movement pattern and had become more reliant on recruiting slow contracting fibers to perform tasks. As a result, the slow fibers were used more and improved their functional behavior, whereas the fast fibers were used less thereby decreasing their contractile function. Support for this theory, at least in part, is based on the evidence that neuromuscular interactions (i.e., electrical activity) are important for regulating changes in muscle proteins (27), gene regulation (30), and contractile function (23). Thus the decreased recruitment of fast-twitch fibers may have caused them to gradually lose some of their functional ability and become atrophic. This might also help explain, in part, why a portion the women’s fast fibers were fragile and failed during experimentation.

Only one other study has directly compared single cell function among older men and women. Frontera et al. (13) found that single fibers from inactive older men (74 yr) were stronger (P<sub>s</sub>) compared with inactive older women (72 yr), even after correcting for fiber size. They did not, however, have measures of V<sub>0</sub> or power for comparison. In contrast, the current study did not find a difference in single fiber P<sub>0</sub> before resistance training (baseline) when adjusted for cell size (P<sub>0</sub>/CSA; specific tension). In addition, no differences in specific tension were found pre- to post-PRT in the current investigation. This is in agreement with our findings pre- to post-PRT with older men (34), data on swimmers with reduced training (33), and bed rest studies (37). However, differences in single fiber muscle quality (i.e., specific tension) have been reported between young and old males (13, 21) and after 17 days of space flight (36) and 6 wk of bed rest (20). Collectively, these data suggest that differences in muscle quality are possible but not universal.

Several factors influence muscle quality and muscle adaptations that were most likely variable among the human volunteers used among these studies. Factors such as physical activity, environmental conditions, nutritional status (10), protein synthesis (35, 40), and hormone status (11, 28) are known to influence muscle plasticity and development. Perhaps the most obvious candidate for influencing the gender-related differences in muscle cell function observed in our work may have been hormone fluctuations in the years before these experiments and during the 12 wk of training. It has been shown that postmenopausal women are more susceptible to advanced muscle weakness, which can be partially attenuated by hormone replacement therapy (28). None of the women in this investigation had ever participated in hormone replacement therapy programs, suggesting that they may have had an advanced degree of sarcopenia compared with the men. The fragile nature of the women’s fast-twitch fibers during the physiological experiments supports the notion of advanced sarcopenia and indicates that fiber specific sarcopenia at the cell level may exist among older men and women. Additionally, insulin-like growth factor has been linked to muscle remodeling and may be an important component regulating fiber-specific adaptations (11). Whether such hormones (or lack of) modulate the changes in contractile properties of individual muscle fibers with resistance training is unknown at this time.

Previous investigations with humans and rodents have shown that fiber contractile velocity can be reduced by as much as 40% with aging (21, 32). In our previous study with older men, we found that resistance training elevated fiber V<sub>0</sub> by 75 and 45% in the MHC I and IIa fibers, respectively, thus reversing the decline in single fiber V<sub>0</sub> often observed with aging. However, no change in fiber V<sub>0</sub> was found with the older women. This is perhaps the most significant finding from our resistance training studies with older adults. This clearly points to different mechanisms that are regulating the cellular adaptations with PRT among older men and women.

The mechanism behind the difference in fiber V<sub>0</sub> response to resistance training among the older men and women is unknown. The MHC isoform composition (2, 26) and MLC content (24, 26) have been implicated for modulating speed of muscle shortening. There were no significant changes in MHC phenotype in the single fibers studied as part of the physiology experiments. Although changes in MHC content cannot be ruled out as undetected MHC, phenotypes have been implicated for possibly playing a role in regulating V<sub>0</sub> (29). Moreover, we were unable to detect changes in MLC com-
position that could account for the increased $V_o$ in the fibers of the older men (34, 39). Though we did not observe any change in fiber $V_o$ from the single fibers of the women, the MLC data were unchanged and paralleled the findings from the older men. Thus it appears that the relative amount of MLCs are not playing a key role in regulating the change or lack of change in fiber $V_o$ in aging men and women who perform resistance training.

Of particular interest were the similarities in single muscle fiber peak power output after the training from the men and women, despite differences in baseline values. Fiber power is dependent on the interaction of $P_o$ and $V_o$. Despite pre- to post-PRT differences in $P_o$ and $V_o$ among the sexes and fiber type, these variables were regulated in such a way to produce comparable peak power after PRT. The comparable single fiber power output after PRT may help explain why similar improvements are found in whole muscle measures among men and women with resistance training. It also points to the fact that single muscle cell function is not solely dependent on a single strategy to reach a common endpoint, such as power production. This is an important finding because most human movements are isotonic in nature and largely depend on power output for human locomotion.

Although the resistance-training program employed was identical for the men and the women used in these investigations, we observed that single MHC I and IIa muscle fibers adapt differently to this stimulus. It appears that some of the differences can be explained, in part, by the difference in contractile function among the men and women before the resistance training program. However, these pretraining differences cannot account for all of the variance in single cell function among these men and women. Other aspects of excitation-contraction mechanics that were not measured as part of this investigation must be considered, in particular, cell proteins and their geometrical configuration, calcium kinetics, protein kinetics, force per crossbridge, energy consumption, and transcription and translation factors.

In conclusion, we have completed a 12-wk resistance training investigation in older women and men examining whole muscle and single muscle fiber function. To our knowledge, this is the first investigation to directly compare single fiber function in older men and women before and after resistance training. From the whole muscle data, it would appear that older men and women respond similarly to resistance training. However, the adaptation to resistance training in single muscle fiber contractile function was considerably different among older men and women. Most notably, no change in fiber $V_o$ was observed in the older women compared with the large elevation in fiber $V_o$ in older men. Collectively, the present study in older women and our previous study in older men (34) demonstrated a gender-related difference in the contractile behavior before and after resistance training in older adults. One common aspect of these studies was that the resistance training protocol used appeared to be more effective for improving MHC I fiber contractile properties in both men and women. These data indicate that our understanding of muscle behavior is incomplete and that more research is needed to understand the basic mechanisms modulating muscle performance with aging, gender, and physical activity.

The authors would like to thank David Costill, Bill Fink, David Williamson, and Gary Lee and for technical assistance with this project.

This investigation was supported by National Institute on Aging Grant AG-15486 to S. Trappe.

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