Effect of resistance training on skeletal muscle-specific force in elderly humans

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Reeves, N. D., M. V. Narici, and C. N. Maganaris. Effect of resistance training on skeletal muscle-specific force in elderly humans. J Appl Physiol 96: 885–892, 2004. First published October 24, 2003; 10.1152/japplphysiol.00688.2003.—This study assessed muscle-specific force in vivo following strength training in old age. Subjects were assigned to training (n = 9, age 74.3 ± 3.5 yr; mean ± SD) and control (n = 9, age 67.1 ± 2 yr) groups. Leg-extension and leg-press exercises (2 sets of 10 repetitions at 80% of the 5 repetition maximum) were performed three times/wk for 14 wk. Vastus lateralis (VL) muscle fascicle force was calculated from maximal isometric voluntary knee extensor torque with superimposed stimuli, accounting for the patella tendon moment arm length, ultrasound-based measurements of muscle architecture, and antagonist cocontraction estimated from electromyographic activity. Physiological cross-sectional area (PCSA) was calculated from the ratio of muscle volume to fascicle length. Specific force was calculated by dividing fascicle force by PCSA. Fascicle force increased by 11%, from 847.9 ± 365.3 N before to 939.3 ± 347.8 N after training (P < 0.05). Due to a relatively greater increase in fascicle length (11%) than muscle volume (6%), PCSA remained unchanged (pretraining: 30.4 ± 8.9 cm²; posttraining: 29.1 ± 8.4 cm²; P > 0.05). Activation capacity and VL muscle root mean square electromyographic activity increased by 5 and 40%, respectively, after training (P < 0.05), indicating increased agonist neural drive, whereas antagonist cocontraction remained unchanged (P > 0.05). The VL muscle-specific force increased by 19%, from 27 ± 6.3 N/cm² before to 32.1 ± 7.4 N/cm² after training (P < 0.01), highlighting the effectiveness of strength training for increasing the intrinsic force-producing capacity of skeletal muscle in old age.

Muscle architecture; strength training; ultrasound; physiological cross-sectional area

Muscle weakness in old age is a considerable health concern, as it may limit independent living and potentially contribute to falls, as well as to other forms of morbidity. Recent studies have shown that resistance training may enhance muscle strength, thus being an effective countermeasure against muscle weakness associated with aging (5, 11, 14, 18, 20, 42). These training-induced strength gains have been attributed to adaptations occurring in a number of components of the motor system. Although hypertrophy has been reported as one of the major factors contributing to strength gains with training, the increase in muscle size does not solely account for the increase in strength (11, 17, 19, 20, 46). This indicates that other factors, such as increased activation capacity of agonist muscles (20, 42), reduced activation of antagonist muscles (17, 18), and changes in tendon stiffness (40), might play a role in the strength gains observed with training in old age. It presently remains unknown whether the specific force of whole muscle would also increase in response to strength training in the elderly.

Specific force reflects the intrinsic force-producing capacity of a muscle (force per unit area; N/cm²). Two fundamental factors are required for accurate in vivo determination of specific force (30): 1) maximal isometric muscle force, that is to say, the maximum force developed in vivo at optimal fiber length under conditions of maximal neural activation, and 2) muscle physiological cross-sectional area (PCSA). The estimation of the former requires the tendon moment arm length and muscle pennation angle, whereas the latter requires the muscle volume and muscle fiber or fascicle length. Previous attempts to assess alterations in specific force with strength training in elderly humans have been inappropriate due to errors in assessing one or both of the previously mentioned factors necessary for specific force determination. For instance, instead of muscle force, joint torque (44, 46) or weight lifting capacity (46, 49) has previously been used, and a single anatomic cross-sectional area (ACSA) has typically been used to normalize these strength measurements to muscle size (44, 49). This approach seems inappropriate, because 1) net joint torque does not reflect only the agonist muscle forces (25), and 2) ACSA does not take into account muscle geometry and training-induced changes thereof (35), thus underestimating the PCSA of a pennate muscle (1, 16, 21). Furthermore, a single ACSA is typically assessed in the midregion of a muscle, which has been reported as the region displaying the greatest increase in ACSA following training (46) and would thus lead to overestimation of hypertrophy. To accurately estimate the effect of training on maximal muscle force and specific force, agonist-antagonist muscle activation must also be assessed. This is because strength training has been shown to increase voluntary activation capacity of agonist muscles by ~4% (20, 42). Also, cocontraction of antagonist muscles is reported to be higher in older individuals compared with young adults (26); however, this might be reduced by strength training (17, 18).

In light of the above considerations and limitations of previous studies, the present study aimed to assess the effect of strength training in old age on skeletal muscle-specific force in vivo. It was hypothesized that muscle-specific force would increase following training.

METHODS

Subjects. Eighteen subjects (10 women and 8 men) gave written, informed consent to participate in this study. All procedures con-
formed with the declaration of Helsinki and were approved by the Ethics Committee of the Institute for Biophysical and Clinical Research into Human Movement at the Manchester Metropolitan University. All subjects received medical clearance from their general practitioners before initiating the training. Exclusion criteria for participation in the study were the presence of known neurological, musculoskeletal, inflammatory, or metabolic disorders, uncontrolled hypertension or angina, or neoplastic disease. None of the subjects had previously taken part in any type of resistance training but were all physically active, participating in activities such as bowling, gardening, and walking. It was intended to have an equal number of men and women in each group in an attempt to avoid any bias of gender. Five women and four men (n = 9, age 74.3 ± 3.5 yr, body mass 69.7 ± 14.8 kg, and height 1.63 ± 0.09 m, means ± SD) were assigned randomly to the training group, with the remaining subjects assigned to the nontraining control group (n = 9, age 67.1 ± 2 yr, body mass 73.5 ± 14.9 kg, and height 1.68 ± 0.12 m).

Strength-training program. Strength training using isotonic resistance exercise machines (Technogym, Gambettola, Italy) was performed 3 days/wk for 14 wk. The training sessions began with a 5- to 10-min warm-up on a cycle ergometer at 60–65% of age-predicted maximum heart rate. Specific exercises performed were 1) the bilateral leg extension for the knee extensor muscle group and 2) the bilateral leg press for the hip extensor muscles and the quadriceps. All exercises were performed to provide the subjects with a whole body conditioning stimulus; these were the bilateral calf raise, chest press, seated row, abdominal crunch, and lower back extension. Subjects were familiarized with the exercises during the first 2 wk of training, during which the workload was gradually increased until they were able to perform a 5 repetition maximum (RM) for each exercise. It was considered that the 5 RM was more appropriate for elderly individuals unaccustomed to this type of strenuous exercise, as an alternative to the 1 RM. A similar approach of testing strength on the training devices using the 3–5 RM has been used previously for elderly individuals (42, 46, 49). The 5 RM was determined every 2 wk to assess any strength increases, with the purpose of maintaining the same relative training intensity. All exercises were performed with the concentric phase of the contraction lasting ~2 s, followed by a ~3-s eccentric phase. One specific warm-up set of 15 repetitions was performed for each exercise at an intensity of 45% of the 5 RM followed by two to three sets of static stretching for specific muscle groups. For each of the lower limb exercises, two training sets of ~10 repetitions were performed, initially at an intensity of ~60–70% of the 5 RM, progressing to 80% of the 5 RM within 1–3 wk, depending on the subject’s capabilities. According to the formula proposed by Brzycki (6) for young adults, this intensity (80% of the 5 RM) corresponds to ~70–75% of the predicted 1 RM. During the sessions, all subjects wore heart rate monitors, and the exercise was stopped if the heart rate rose to within ~15 beats/min of the age-predicted maximum heart rate. A recovery period of ~3 min was introduced between sets. Subjects were observed very closely during training to ensure performance of the correct technique, while verbal encouragement was given to maintain motivation. Compliance to training was very high; only ~7% of sessions were missed due to holidays, illness, etc.

Measurement of maximal torque and voluntary activation capacity. Maximal isometric knee extension and flexion torque were assessed using an isokinetic dynamometer (Cybex NORM, Ronkonkoma, NY) at nine knee joint angles, ranging from 90 to 10°, in 10° increments (full extension = 0°), with the hip angle at 85° (supine position = 0°). All measurements were performed on the right leg. The center of rotation of the knee joint was visually aligned with the dynamometer axis of rotation, and straps were positioned at the hip, shoulders, and over the right thigh to prevent any extraneous movement. Subjects had previously visited the laboratory on at least one occasion to become familiarized with the procedures involved. Both maximal isometric knee extension and flexion torque were assessed at the tested knee joint angles in a randomized order, with 3-min rest between contractions. Subjects were instructed to perform a maximal isometric knee extension contraction by increasing their effort in a linear ramp fashion, so that maximal torque was reached after ~2 s and then maintained for ~3–4 s until a verbal signal was given to stop. When the voluntary torque peaked, a superimposed supramaximal double twitch with 50-μs pulse width and 50-ms interstimulus gap was applied. The doublet was generated by an electrical stimulator (model DS7, Digitimer stimulator, Welwyn, Garden City, UK) modified to give a maximum of 1,000-mA output and number delivered to the contact muscle group through two 7.5 × 12.5-cm self-adhesive electrodes (Versa-Stim, CONMED) placed on the distal and proximal regions of the thigh. Single twitches were applied at rest with increasing current intensity while maintaining a constant 400 V, to identify the stimulation intensity corresponding to maximal twitch torque. The point at which a further increase in current by 50 mA failed to increase the twitch torque was defined as the supramaximal stimulation intensity. The voluntary activation capacity of the quadriceps muscle group was calculated as maximum voluntary torque/ (maximum voluntary torque + superimposed stimulation torque), as previously reported (23, 24). It must be noted that performance of a ramp isometric contraction in the elderly may yield lower maximal torque values compared with the performance of a rapid isometric contraction (23, 24).

Notwithstanding potential differences in torque between slow and fast contractions, our protocol remained the same before and after training; hence, our measurements would be valid in assessing the effect of training.

Signals of torque, electrical stimuli application, and electromyographic (EMG) activity were displayed on the screen of a computer (Macintosh, G4, Apple Computer, Cupertino, CA), interfaced with an acquisition system (Acknowledge, Biopac Systems, Santa Barbara, CA) used for analog-to-digital conversion, at a sampling frequency of 2,000 Hz.

Measurement of EMG activity. EMG activity was assessed from the vastus lateralis (VL) muscle and the long head of the biceps femoris (BF) muscle. Two self-adhesive Ag-AgCl electrodes, 10 mm in diameter (Neuroline, Medicotest, Rugmarken Denmark), were placed in a bipolar configuration with a constant interelectrode distance of 20 mm at a site corresponding to the distal one-third of the muscle length (52). Reference electrodes were placed on the lateral tibial condyle. In an attempt to minimize cross talk from adjacent muscles, electrodes were placed along the midsagittal plane of each muscle, guided by axial plane ultrasound scanning. To estimate the common EMG signals between the VL and BF muscles, a cross-correlation analysis was performed (26, 51). At knee joint angles of 90, 60, and 30°, r values of 0.144, 0.063, and 0.085, respectively, were obtained, suggesting the presence of negligible cross talk (~2, <1, and <1%, respectively) between these two muscles (51). Shaving, skin abrasion, and cleaning with an alcohol-based solution always preceded electrode placement to reduce skin impedance <5,000 Ω. The location of all electrodes with respect to the muscle length and anatomic landmarks was recorded and also traced onto an acetate sheet to ensure identical placement on subsequent sessions. The raw EMG signal was preamplified and filtered using high- and low-pass filters set at 10 and 500 Hz, respectively. The root mean square (RMS) EMG activity of the BF and VL muscles was measured over a 50-ms time period corresponding to maximal isometric knee extension torque and normalized for a 1-s time period by multiplying the measured phase by a factor of 20. This measurement time phase (50 ms) yields an acceptable signal-to-noise ratio (43). To estimate the level of antagonistic cocontraction from the knee flexors, the BF muscle RMS EMG activity was measured during maximal isometric knee flexion per-
formed at the corresponding knee joint angle, over a 50-ms time period, corresponding to maximal torque. The antagonistic torque of the knee flexors during a maximal isometric knee extension was calculated, assuming a linear EMG-torque relation, from the EMG-torque relation of the BF muscle when acting as an agonist (22). The assumption of a linear EMG-torque relation was tested in a subsample \((n = 6)\) of elderly subjects at knee joint angles of 90, 60, and 30°. At each tested knee joint angle, subjects were instructed to maintain 25, 50, 75, and 100% of their maximal isometric knee flexion torque for \(3–4\) s. These tests confirmed a linear EMG-torque relation (Fig. 1).

**Measurement of muscle architecture.** The VL muscle architecture was examined in vivo at the time of superimposed stimuli application using real-time B-mode ultrasonography (ATL-HDI 3000, Bothell), with a 40-mm, 7.5-MHz linear-array probe. Scans were taken in the midsagittal plane, at the level of 50% of the VL muscle length. With the use of measurements of muscle length and anatomic landmarks, this position was recorded and marked onto an acetate sheet to ensure that the same region of the muscle was scanned on subsequent sessions. The probe was coated with a water-soluble transmission gel to provide acoustic contact and was held in place using an external fixation device, secured by the experimenter. This device also served to disperse any pressure that might be caused by the probe on the dermal surface, thereby minimizing compression of the underlying structures. The probe was placed onto an echo-absorptive external marker fixed on the skin. The line cast on the ultrasound image by the external marker demonstrated if any movement of the probe occurred with respect to the scanned structure. If any movement of the probe did occur, this would result in scanning a different region of the muscle, and this trial would be omitted. Ultrasound was synchronized with all signals captured on the acquisition system using an external voltage trigger visible on both systems. Ultrasound scanning was maintained at \(\approx 25\) Hz and was recorded onto SVHS videotape for subsequent analysis. Ultrasound images were then acquired using frame-capture software (Adobe Premier version 5.1, Adobe Systems). Ultrasound scans corresponding to the maximal isometric knee extension torque were identified. The VL muscle fascicular paths were identified as the interspaces between the echoes arising from the perimysial tissue surrounding each fascicle. The ultrasound probe was oriented along the sagittal plane of the fascicular paths, both at rest and during maximal contraction, to allow the entire visible length of the fascicle to be clearly tracked. The VL muscle fascicles generally extended off the ultrasound scan window, and it was, therefore, necessary to estimate part of the fascicle length. With the use of digitizing software (NIH image version 1.61, National Institutes of Health, Bethesda, MD), the visible portion of the fascicle was measured, and the remaining part was estimated, assuming linear continuation of the fascicle and aponeurosis in the proximal direction. Similar approaches to estimating VL muscle fascicle lengths have previously been applied (12, 33), yielding errors of 2–7%. To visualize the entire fascicle length, the probe can be moved along the muscle during an isometric contraction, over a series of external markers fixed on the skin. It was not possible to apply this procedure throughout the testing, because of the position of both the stimulating and EMG electrodes. However, this procedure was applied in a subsample \((n = 6)\) of elderly subjects without the placement of any electrodes, to assess the error associated with estimating fascicle length. These tests showed that the associated error was 3.8, 4.2, and 3.9% of fascicle length during maximal contraction at knee joint angles of 90, 60, and 30°, respectively. Pennation angle was defined as the angle between the fascicular path and the deep aponeurosis of the VL muscle. When fascicles inserted curvilinearly into the aponeurosis, pennation angles were measured as the tangent at the insertion of the fascicle into the aponeurosis (31). When fascicle curvature was present, it occurred mainly at the insertion of the fascicles into the deep aponeurosis. A mean of four fascicle lengths and five pennation angles was assessed on each ultrasound image. The accuracy of muscle architecture measurements assessed using ultrasound has previously been validated against direct anatomic inspection on a cadaver (34).

**Measurement of tendon moment arm length.** Patella tendon moment arm lengths were assessed using a 0.2-T MRI scanner (E-Scan, Esato Biomedical, Genoa, Italy) to allow calculation of tendon forces. Sagittal plane scans were acquired using a T1-weighted spin-echo sequence with the following scanning parameters: time to echo, 26 ms; time of repetition, 850 ms; field of view, 180 \(\times\) 180 mm; matrix, 256 \(\times\) 192; 4-mm slice thickness; and 0.4-mm interslice gap. The patella tendon moment arm length was defined as the perpendicular distance from the midpoint of the patella tendon to the tibiofibemoral contact point (2) and was measured using digitizing software, as described above. Due to constraints in the size of the coil, it was only possible to image the knee joint in full extension. The previously reported ratio (2) of the patella tendon moment arm length between full extension and the specific knee joint angle studied was used to estimate the tendon moment arm length for each subject.

**Calculation of fascicle force.** The VL muscle was examined as representative of the entire quadriceps muscle group. The VL muscle fascicle force was calculated by following a series of steps, using previously applied methods (27, 28).

1) The sum of the maximal knee extension joint torque (maximal voluntary torque + superimposed stimulation torque) and the estimated antagonistic cocontraction torque from the knee flexors \((TQ_{coac})\) was divided by the MRI-based estimate of the patella tendon moment arm length \((MA_{PT})\) (Eq. 1)

\[
F_{PT} = (TQ_{KE} + TQ_{coac})/MA_{PT}
\]

where \(F_{PT}\) is the estimated patella tendon force and \(TQ_{KE}\) is the maximal isometric knee extension torque.

2) The relative contribution of the VL muscle to the patella tendon force calculated from Eq. 1 was estimated from the relative PCSA of the VL muscle with respect to the entire quadriceps muscle group, from the data of Narici et al. (36).

3) The VL muscle fascicle force was estimated by dividing VL muscle force by the cosine of the angle of pennation of fascicles, measured during maximal isometric contraction (Eq. 2)
where $F_{\text{Fasc}}$ is the estimated VL muscle fascicle force, $F_{\text{VL}}$ is the estimated force produced by the VL muscle, and $\cos \theta$ is the cosine of the angle of pennation of the VL muscle fascicles during maximal isometric contraction.

**Measurement of muscle ACSA.** B-mode ultrasonography using a 7.5-MHz linear-array probe was used to obtain axial-plane images of the VL muscle, using previously applied methods validated in the tibialis anterior muscle (10). All measurements were performed on the right leg after the subjects had been in the supine position for 20 min to allow fluid shifts to occur (4). During all measurements, the subjects were instructed to relax their leg muscles. The proximal and distal insertions and the midsagittal plane of the VL muscle were identified and marked on the skin. Axial sections were then clearly marked along the muscle at intervals of 30 mm from the proximal insertion. The distance of the last axial plane scan from the distal insertions was calculated by using the ACSAs measured with ultrasonography, in accordance with previously applied methods (10). In each section, the distance ($d$) between two muscle ACSAs ($a$ and $b$) was 30 mm, except for the most distal section, which varied between 5 and 30 mm. All sections were treated as truncated cones and calculated from the following equation:

$$V = \sqrt[3]{a \cdot (a + \sqrt{ab}) + b}$$

Volume of the entire VL muscle was calculated by summing up all of the inter-ACSA volumes.

**Calculation of muscle volume.** The VL muscle volume ($V$) was calculated by using the ACSAs measured with ultrasonography, in accordance with previously applied methods (10). In each section, the distance ($d$) between two muscle ACSAs ($a$ and $b$) was 30 mm, except for the most distal section, which varied between 5 and 30 mm. All sections were treated as truncated cones and calculated from the following equation:

$$V = \sqrt[3]{a \cdot (a + \sqrt{ab}) + b}$$

Volume of the entire VL muscle was calculated by summing up all of the inter-ACSA volumes.

**Calculation of muscle PCSA.** The VL muscle PCSA was calculated by dividing muscle volume by fascicle length (1, 21, 30). This calculation assumes a muscle model that is cylindrical in shape, with fibers of constant length. The assumption of a relatively constant fiber length throughout the muscle is supported by previous in vivo and in vitro findings in several human muscles (13, 29, 31, 34).

**Calculation of specific force.** Specific force of the VL muscle was calculated at the knee joint angle where maximal fascicle force peaked (calculated from the maximal voluntary torque + superimposed stimulation torque). This corresponded to the knee joint angle of 70° before and after training. Specific force was calculated by dividing fascicle force by PCSA (1, 15, 21, 30, 36, 39).

**Study design.** All subjects were tested at baseline. Subjects were restested after the 14-wk strength training program. The control group received no exercise training and was restested after 14 wk of continuing habitual daily activities.

**Reliability and validity of ACSA measurements.** The reliability of muscle ACSAs assessed from axial plane ultrasound scans was tested in a subsample ($n = 6$) of elderly subjects on two different occasions, separated by a mean of 3 days. The muscle ACSAs measured in scans 6–10 demonstrated good reliability (Fig. 2) with a mean intraclass correlation coefficient (ICC) of 0.99 and mean typical error of 0.3 cm$^2$. The validity of muscle ACSAs assessed using ultrasonography in scans 6–10 were tested against axial plane scans acquired using MRI (Fig. 2). The lower section of the right thigh was scanned using MRI after following the same procedures described previously to allow fluid shifts to occur. It was only possible to scan the lower portion of the thigh due to constraints in the size of the available coil. Axial plane images of the thigh were acquired using a T1-weighted spin-echo sequence with the following scanning parameters: time to echo, 28 ms; time of repetition, 850 ms; one acquisition; field of view, 200 × 200 mm; matrix, 256 × 192; 5-mm slice thickness; and 1-mm interslice gap. Oil-filled capsules used as external markers were secured onto the skin and used to identify the corresponding sections measured with ultrasonography. These tests indicated that the ultrasound method was valid, being associated with a mean typical error of 0.15 cm$^2$ and yielding a mean ICC of 0.99. To determine interexperimenter measurement reliability, six different experimenters measured VL muscle ACSAs at a given anatomic level in scans obtained with ultrasonography and MRI. The coefficient of variation values for ACSA measurements assessed by different experimenters were 2.1 and 0.8% for images obtained using ultrasonography and MRI, respectively.

**Reliability of measurements.** Apart from the reliability of the muscle ACSA measurements described above, further tests were performed to determine the reliability of the remaining measurements used in this study. These tests were performed on separate days by the same experimenter in a subsample ($n = 6$) of elderly individuals. The typical error and ICCs of variables assessed in these tests are displayed in Table 1.

**Statistical analysis.** Baseline differences between the training and control groups for the reported variables were tested using independent samples Student’s $t$-tests. Paired samples Student’s $t$-tests were used to test for differences in the 5 RM following training. A mixed design 2 × 2 factorial ANOVA was used to test for differences between group (training, control) and time (pretraining, posttraining) for all variables. Tests were followed up with a post hoc analysis using the Scheffé procedure where necessary. Normality of the data were checked and subsequently confirmed using the Kolmogorov-Smirnov test. The present sample size was selected based on a power analysis. Values presented are means ± SD; significance was accepted at the level $P < 0.05$.

The interday reliability of measurements was tested with an ICC using a one-way random-effects model. The validity of ultrasound
Table 1. Interday measurement reliability

<table>
<thead>
<tr>
<th>Knee Joint Angle, °</th>
<th>90</th>
<th>60</th>
<th>30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torque, N×m</td>
<td>1.7 (0.99)</td>
<td>2.8 (0.99)</td>
<td>3.6 (0.98)</td>
</tr>
<tr>
<td>VL RMS EMG, mVs</td>
<td>$3.38 \times 10^{-3}$ (0.99)</td>
<td>$7.77 \times 10^{-3}$ (0.94)</td>
<td>$7.39 \times 10^{-3}$ (0.99)</td>
</tr>
<tr>
<td>Activation capacity</td>
<td>0.01 (0.97)</td>
<td>0.01 (0.87)</td>
<td>0.01 (0.93)</td>
</tr>
<tr>
<td>Fascicle length, cm</td>
<td>0.1 (0.99)</td>
<td>0.1 (0.99)</td>
<td>0.1 (0.99)</td>
</tr>
<tr>
<td>Penetration angle, °</td>
<td>0.8 (0.97)</td>
<td>0.4 (0.99)</td>
<td>0.3 (0.99)</td>
</tr>
</tbody>
</table>

Values are typical errors, with intraclass correlation coefficients in parentheses. VL, vastus lateralis; RMS, root mean square; EMG, electromyography; BF, biceps femoris.

Table 2. Measured and estimated variables for the training and control groups

<table>
<thead>
<tr>
<th></th>
<th>Training Group</th>
<th>Control Group</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Leg extension 5 RM, kg</td>
<td>43.5±12</td>
<td>49.4±14.1†</td>
</tr>
<tr>
<td>Leg press 5 RM, kg</td>
<td>178.3±44.7</td>
<td>219.1±55.4*</td>
</tr>
<tr>
<td>Isometric torque, N×m</td>
<td>121.4±61</td>
<td>131.1±55.3†</td>
</tr>
<tr>
<td></td>
<td>0.95±0.06*</td>
<td>0.95±0.06*</td>
</tr>
<tr>
<td></td>
<td>7.8×10⁻²±5.7×10⁻²</td>
<td>1.1×1⁻⁹±9.6×1⁻⁸</td>
</tr>
<tr>
<td>Knee flexor cocontraction, %</td>
<td>49.7±32.8</td>
<td>50.3±33.9</td>
</tr>
<tr>
<td></td>
<td>8.4±0.8</td>
<td>9.3±1.3†</td>
</tr>
<tr>
<td></td>
<td>14.1±1.6</td>
<td>16.2±2.8†</td>
</tr>
<tr>
<td></td>
<td>847.9±365.3</td>
<td>939.3±347.8*</td>
</tr>
<tr>
<td></td>
<td>17.6±4.6</td>
<td>19.4±5.1</td>
</tr>
<tr>
<td></td>
<td>254.3±76</td>
<td>270.3±81.3†</td>
</tr>
<tr>
<td></td>
<td>30.4±8.9</td>
<td>29.1±8.4</td>
</tr>
<tr>
<td></td>
<td>27.0±6.3</td>
<td>32.1±7.4†</td>
</tr>
</tbody>
</table>

Values are means ± SD. 5 RM, 5 repetition maximum; ACSA, anatomic cross-sectional area; PCSA, physiological cross-sectional area. Significant difference between pre- and posttraining conditions: *P < 0.05 and †P < 0.01. ‡Significant difference from the training group at baseline, P < 0.05.

Results

At baseline, knee flexor cocontraction was significantly different between the training and control groups (Table 2), and this difference remained following training. No significant differences existed between the two groups for any other measured or estimated variables. Body mass was not significantly altered in either the training group (pretraining: 69.7 ± 14.8 kg; posttraining: 67.2 ± 15.2 kg) or the control group (precontrol: 73.5 ± 14.9 kg; postcontrol: 75.1 ± 15.4 kg).

Weight lifting capacity. For the bilateral leg-extension exercise, the 5 RM increased by 14% after the 14-wk training period (P < 0.01; Table 2). For the bilateral leg-press exercise, the 5 RM increased by 23% following training (P < 0.01; Table 2). Weight lifting capacity was not assessed in the control group.

Muscle size. Following training, ACSA increased by 3–10% over all measured sections of the VL muscle (Fig. 3A), reaching significance (P < 0.01) at scans 4 (7% increase), 5 (8% increase), 6 (9% increase), 7 (10% increase), and 9 (8% increase). Maximum VL muscle ACSA was observed in the mid-thigh region, 15 cm from the proximal insertion, at scan 5 for both the training (pretraining: 17.6 ± 4.6 cm²; posttraining: 19.3 ± 5.3 cm²; P < 0.01; Fig. 3A) and control (precontrol: 19.4 ± 5.1 cm²; postcontrol: 19.4 ± 5.1 cm²; Fig. 3B) groups. The largest absolute increase in muscle ACSA occurred in the mid-thigh region, from scans 4–7. In contrast, no significant changes in ACSA were observed at any level throughout the VL muscle in the control group (Fig. 3B). The VL muscle volume increased by 6% after training (P < 0.01), but was not significantly altered in the control group (Table 2). The VL muscle PCSA did not change significantly in either the training or control groups (Table 2).

Muscle architecture. The VL muscle fascicle length during maximal contraction increased by 11% after training (P < 0.01; Table 2). Penetration angle during maximal contraction increased by 13% after training (P < 0.01; Table 2). In contrast, no significant changes in fascicle length or penetration angle occurred in the control group (Table 2).

Agonist-antagonist muscle activation. Activation capacity of the quadriceps muscle group assessed by superimposing electrical stimuli increased by 5% after training (Table 2). The VL muscle RMS EMG activity increased by 40% following training (P < 0.01; Table 2). No significant changes in activation capacity or VL muscle RMS EMG activity occurred in the control group (Table 2). The level of antagonistic cocontraction from the knee flexors remained unchanged in the training and control groups (Table 2).

Torque, fascicle force, and muscle-specific force. Isometric knee extension torque increased significantly by 8% following training (Table 2) and decreased by 11% in the control group, possibly due to lower voluntary efforts (see RMS EMG activity and activation capacity values in Table 2). The VL muscle fascicle force increased significantly by 11% after training (Table 2). In contrast, no significant change in fascicle force occurred in the control group (Table 2). Training increased specific force of the VL muscle by 19% after training (P <
0.01; Table 2). In contrast, no significant change in muscle-specific force occurred in the control group (Table 2).

**DISCUSSION**

The present study investigated the effect of strength training in old age on skeletal muscle-specific force in vivo. Specific force was found to increase by 19% following strength training.

Despite the training-induced increase in muscle-specific force, in contrast to a previous report following strength training in the elderly (35), muscle PCSA remained unaltered (Table 2), because training increased fascicle length more than muscle volume (11 and 6% increase, respectively). It should be stressed that an underestimation or overestimation of fascicle length would lead to muscle PCSA being overestimated and underestimated, respectively (see METHODS), potentially masking training-induced adaptations. Order-of-magnitude calculations were performed to assess the error introduced in the PCSA estimate in the “worst-case” scenario, where fascicle lengths are underestimated before and overestimated after training, respectively. The effect of an indicative error in fascicular length similar in magnitude to that introduced by approximating the fascicular continuation as being linear (~5%) was assessed. This analysis showed that PCSA would be 28.9 and 30.7 cm² before and after training, respectively. It should also be stressed that errors may have been introduced if the assumptions made in the calculation of PCSA that all fascicles have similar lengths and pennation angles are reduced in old age (37); thus, based on data previously obtained in young adults, the present findings might suggest a training-induced reversal of these aging and disuse effects. The present finding of altered architectural features highlights the unique muscular adaptations occurring in response to training. Such adaptations would affect fascicle force transmission to the tendon and the active range of excursion (16), factors neglected by measurement of ACSA alone.

Training increased voluntary activation capacity of the quadriceps muscle group by 5%, in line with previous findings (20, 42). Consistent with the increase in activation capacity was the increased RMS EMG activity of the VL muscle following training. The ACSA increased by 3–10% along the muscle length, with a mean increase of 6% (Fig. 3A). These findings are in line with previous reports of increases in muscle ACSA from 6 to 10% following strength training in elderly individuals (11, 14, 19, 20, 49). Increases in ACSA occurred predominantly in the midregion of the muscle, in the area of maximal ACSA, consistent with previous findings after strength training in the elderly (41, 46). The present findings indicate that measuring a single ACSA in the midregion of the muscle, at the area of maximal girth, as previous studies have typically done (5, 11, 14, 17), would overestimate the increase in ACSA. In agreement with previous findings, training caused significant alterations in muscle architecture (35). Fascicle lengths and pennation angles increased, suggesting the addition of sarcomeres in series and in parallel, respectively (32, 50). Muscle fascicle lengths and pennation angles are reduced in old age (37); thus, based on data previously obtained in young adults, the present findings might suggest a training-induced reversal of these aging and disuse effects. The present finding of altered architectural features highlights the unique muscular adaptations occurring in response to training. Such adaptations would affect fascicle force transmission to the tendon and the active range of excursion (16), factors neglected by measurement of ACSA alone.

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**Table 3. Relative increase in the ACSAs of the knee extensor muscles assessed from axial-plane MRI scans**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Increase in ACSA, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vastus lateralis</td>
<td>6.3 ± 3.2</td>
</tr>
<tr>
<td>Vastus intermedius</td>
<td>5.2 ± 2.6</td>
</tr>
<tr>
<td>Vastus medialis</td>
<td>5.7 ± 3.3</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>5.2 ± 4.7</td>
</tr>
</tbody>
</table>

Values are means ± SD of percent increase measured from scans taken at 3 anatomic levels: the rectus femoris myotendinous junction (reference point) and scans located 30 and 60 mm proximal to the reference point.
training (Table 2). Taken together, these results indicate an increased neural drive of the agonist muscles, most likely caused by a greater number of active motor units or increased firing frequency of motor units (38). In contrast to some previous reports (17, 18), training did not cause any reduction in the level of cocontraction from antagonist muscles. The level of cocontraction varied considerably between subjects, a point reflected in the significant difference between the groups at baseline. However, the assessment of EMG from the cocontracting BF muscle demonstrated good reproducibility (Table 1). The effect of intersubject variability in the level of cocontraction would not invalidate the calculation of fascicle force, as this was determined individually for each subject. The level of cocontraction was in fact very high (~43%) compared with that reported (~15%) in young adults (7). This might reflect a greater requirement for the knee flexors to assist the anterior cruciate ligament in reducing anterior tibial translation at the knee joint in old age (3). Taken together, our agonist-antagonist activation data highlight that specific force and the changes with training would be considerably underestimated without accounting for these factors.

Although previous studies in elderly humans have shown no increase in the specific tension of single fibers following strength training (47, 48), our in vivo results show that the specific force of whole muscle does increase. Training resulted in a 19% increase in specific force, from 27 to 32 N/cm². Specific force values obtained in the present study are in line with previous reports of 15–38 N/cm² obtained in human muscle (21, 30, 36) and 15–29 N/cm² obtained in animal muscle (9, 39, 45). It should be stressed that, in contrast to single-fiber experiments where activation remains at a given level (47, 48), changes in activation level might occur when contractions are performed with voluntary effort, and this should be accounted for in the estimation of specific force. If specific force had been calculated from the voluntary torque only, the posttraining increase observed would partly reflect differences in the activation level found. However, this problem was largely avoided in the present study because, in both pre- and posttraining conditions, the calculation of specific force encompassed parameters measured at the time of supramaximal electrical stimuli superimposed on the voluntary effort. Artificial maximal activation of the entire muscle would entirely eliminate this problem, but this would require supramaximal tetanic stimulation through the femoral nerve, a procedure that may cause considerable discomfort, raising ethical considerations for its use with elderly populations.

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GRANTS

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REFERENCES


