Neuromuscular Adaptations Associated with Knee Joint Angle-Specific Force Change

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ABSTRACT

NOORKÕIV, M., K. NOSAKA, and A. J. BLAZEVICH. Neuromuscular Adaptations Associated with Knee Joint Angle-Specific Force Change. *Med. Sci. Sports Exerc.*, Vol. 46, No. 8, pp. 1525–1537, 2014. **Purpose**: Neuromuscular adaptations to joint angle-specific force increases after isometric training have not yet been fully elucidated. This study examined angle-specific neuromuscular adaptations in response to isometric knee extension training at short (SL, joint angle 38.1° ± 3.7°) versus long (LL, 87.5° ± 6.0°) muscle lengths. **Methods**: Sixteen men trained three times a week for 6 wk either at SL (n = 8) or LL (n = 8). Voluntary maximal isometric knee extensor (MVC) force, doublet twitch force, EMG amplitudes (EMG/M_{max}), and voluntary activation during MVC force (VA%) were measured at eight knee joint angles (30°–100°) at weeks 0, 3, and 6. Muscle volume and cross-sectional area (CSA) were measured from magnetic resonance imaging scans, and fascicle length (L_f) was assessed using ultrasonography before and after training. **Results**: Clear joint angle specificity of force increase was seen in SL but not in LL. The 13.4% ± 9.7% (P = 0.01) force increase around the training angle in SL was related to changes in vastus lateralis and vastus medialis EMG/M_{max} around the training angle (r = 0.84–0.88, P < 0.05), without changes in the doublet twitch force–angle relation or muscle size. In LL, muscle volume and CSA increased and the changes in CSA at specific muscle regions were correlated with changes in MVC force. A 5.4% ± 4.9% (P = 0.001) increase in L_f found in both groups was not associated with angle-specific force changes. There were no angle-specific changes in VA%. **Conclusion**: The EMG/M_{max}, although not VA%, results suggest that neural adaptations underpinned training-related changes at short quadriceps lengths, but hypertrophic changes predominated after training at long lengths. The findings of this study should contribute to the development of more effective and evidence-based rehabilitation and strength training protocols. **Key Words:** MUSCLE LENGTH, HYPERTROPHY, FASCICLE LENGTH, CROSS-SECTIONAL AREA

BASIC SCIENCES

Increases in force production in response to resistance training typically occur at or around the joint angles adopted during training (26,45). Such specific increases in force are especially evident after joint angle-specific isometric training (26,32). However, despite the substantial functional importance of this neuromuscular property, the mechanisms underpinning it have not yet been fully elucidated. Most studies have proposed that angle-specific strength gains result from centrally mediated or reflex-mediated neural adaptations, as evidenced by increases in the EMG amplitudes of agonist muscles (12,26) and decreases in antagonist coactivation (12). However, several studies have reported a lack of association between changes in the level of muscle activation (i.e., EMG amplitudes) and increases in maximum force around the training angle (12,15). It has been also shown that muscle volume increases after training can occur with little or no change in EMG amplitude (32), which indicates that neural adaptations might not be the only candidate mechanisms influencing joint angle-specific torque changes.

It has been argued that if muscle hypertrophy was the major cause of the strength increase, then muscle strength should change uniformly across all joint angles rather than only at, or around, the training angle (45). Therefore, muscle hypertrophy is rarely considered an important mechanism influencing angle-specific changes. Nonetheless, for such an argument, it is assumed that a muscle's fascicle (or fiber) force–length relations are homogenous across the muscle and that a muscle (or muscle group) is activated uniformly along its length during contractions. However, these assumptions have been proven erroneous. For example, fascicle length (L_f) clearly varies across muscles such as the human quadriceps, and the length of muscle fascicles has been shown to vary along the length of individual muscles (6). Also, both intramuscular EMG (17) and magnetic resonance imaging (MRI) (1) evidence indicates that muscles are not uniformly activated along their lengths, and the hypertrophic response to dynamic resistance exercise has been clearly shown to be nonuniform both within and across

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TABLE 1. Characteristics of subjects (mean ± SD) for groups training at short (SL, $n = 8$) and long (LL, $n = 8$) muscle lengths.

	SL	LL	*P* Value
Age (y)	24.6 ± 4.9	22.8 ± 2.5	0.38
Height (m)	1.80 ± 0.10	1.80 ± 0.10	0.73
Mass (kg)	77.5 ± 13.7	78.2 ± 10.8	0.89
BMI ($kg \cdot m^{-2}$)	23.3 ± 2.8	23.9 ± 1.9	0.62
Force (N)	5112 ± 1008	4328 ± 430	0.19
Optimum angle (°)	64 ± 5	64 ± 5	1.00

"Force" is the knee extension force at week 0 at each subject's optimum joint angle. *P* values for between-group comparisons are shown in the right hand column. The greater mean force in SL resulted from a single subject producing a large muscle force, which is reflected in the greater SD.

muscles such as the quadriceps (5,36). Thus, region-specific hypertrophy could allow some compartments within muscles (which have specific force–length properties) to contribute more to joint torque and conceivably result in changes in angle-specific torque. However, the influence of joint angle-specific training on region-specific hypertrophy has yet to be explicitly studied after isometric training, and it is not known (and cannot be accurately modeled) whether such region-specific hypertrophy could exert an influence that is substantive enough to cause changes in the torque–angle relation.

Regardless, significant changes in muscle hypertrophy may take several weeks to develop, yet changes in angle-specific torque can occur very rapidly (42), so other adaptations also must influence the response. A primary, nonneural candidate is a changing L_f, which is thought to at least partly reflect a change in serial sarcomere number within the constituent fibers (27,40). It has been demonstrated in animal studies that sarcomere number is adjusted so as to achieve an optimum sarcomere length where a muscle experiences its highest levels of tension (8), and that addition and subtraction of sarcomeres will alter the force–length relation within the muscle (10). Therefore, muscle appears to adapt in sarcomere number to optimize the force–length relation (46). Greater increases in torque production at longer muscle lengths were found to be temporally associated with the increase in quadriceps L_f in humans (5), and these changes are known to occur within days/weeks of the commencement of a training program and even be complete within a few weeks (5). Also, retrospective modeling of previously published data has provided convincing evidence that changes in fiber/fascicle length could explain the observed changes in the torque–angle relation (27).

The above arguments are suggestive that, possibly in addition to neural adaptations, specific changes in muscle size and fascicle (or fiber) length may underpin both the rapid and subsequent changes in joint angle-specific force production. Thus, the aim of the present study was to test the effect of isometric knee extensor strength training on joint angle-specific neuromuscular and torque production adaptations and examine whether the training at short (knee extended) versus long (knee flexed) muscle lengths would induce different neuromuscular adaptation patterns. An isometric model was used to accurately control the joint angles used in training, although the data will also be of immediate practical interest to clinicians who use isometric training in their rehabilitation programs.

METHODS

Subjects

Sixteen healthy men (23.7 ± 4.0 yr) with no strength training experience or recent (>12 months) history of lower limb injury or musculoskeletal disorder volunteered for the study; the subjects' physical characteristics are given in Table 1. The effect size calculation for significant angle-specific force increase was based on a study of Kubo et al. (32) that gave a large effect size of 0.80% and 95% power at an alpha level of 0.05 (two tailed). Kubo et al. (32) used similar designs with nine subjects per group. The subjects were randomly assigned to one of two experimental groups: SL, which performed isometric training at a short quadriceps muscle length (i.e., extended knee joint position; $n = 8$), or LL, which performed isometric training at a long muscle length (flexed joint position; $n = 8$). The study was approved by the Edith Cowan University Human Research Ethics Committee and was done in accordance with the Declaration of Helsinki. The volunteers provided written informed consent before participating in the experiments.

Experimental Design

To ensure that the muscle force applied during training was identical between the groups, isometric torque–angle relations were determined for each subject before study commencement and resolved to muscle force–angle relations by normalizing to the patellar tendon moment arm. Training was then performed at a hip angle of 80°, set with the goniometer (0° = full extension), and at a knee angle at which 80% of the maximum quadriceps muscle force (measured at the optimum joint angle) was elicited (the angle at which the force–angle curve fitted to the third-order polynomial crossed the 80% value on *y*-axis) (Fig. 1). The third-order polynomial R^2 value for force–angle curve fit of all subjects ranged from 0.85 to 0.99. Training angles were

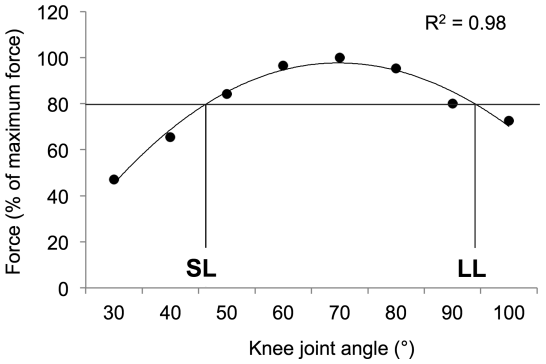

FIGURE 1—Calculation of the training angles for the groups training at short (SL) and long muscle lengths (LL). A third-order polynomial was fitted to the force–angle data, as shown in the figure. Force is expressed as the percentage of the force at the optimum angle.

reassessed by calculating the knee angle corresponding to 80% of the maximum force at the peak angle at week 3. The training angle of a subject was adjusted when the shift was ≥5° (positive shift = more extended, negative shift = more flexed). The training angle shifted ≥5° in four subjects (5°, 5°, 10°, and 15°) in SL and in five subjects (−5° in two subjects and 5° in three subjects) in LL; the mean shift was not significant in either group.

The subjects trained three times a week, with each session comprising five sets of five 5-s maximal voluntary isometric contractions (MVC) on a Biodex isokinetic dynamometer (Biodex Medical Systems, Shirley, NY) with a 5-s interrepetition rest and a 1-min interset rest. The subjects exerted their maximum force as fast and as hard as possible. Joint angle (or muscle length)-specific force changes occur very soon after the initiation of a training regimen (42). Thus, we reasoned that the causative mechanisms of this change must also occur in this short time frame. Vastus lateralis (VL) L_f and quadriceps muscle cross-sectional area (CSA) were measured before and after the training period, and both voluntary and electrically elicited torque–angle relations and levels of muscle activation (EMG and M-wave) were assessed after 0, 3, and 6 wk of training. The rest interval between the last training session and testing was a minimum of 3 d and a maximum of 4 d. Knee joint x-ray imaging was used to measure patellar moment arms before the study, which was assumed not to change over the 6-wk study period.

Patellar tendon moment arm was calculated using sagittal plane, low-radiation knee x-ray scans (Siemens Multix-MT 1384, model number 4803404) at eight knee angles (i.e., 30°, 40°, …, 100°). During scanning, the subjects were positioned supine with the knees flexed and heels placed against a custom-made wooden plate. The experimenter measured the knee angle with a goniometer. The subjects were asked to push against the wooden plate to activate the knee extensor muscles isometrically at approximately 60% of the perceived maximum. Pilot data on three subjects who were able to produce and maintain maximal contractions with this setup showed that patellar tendon moment arm change was minimal from approximately 60% to 100% of MVC, so the measurements were considered reflective of those obtained near MVC. From the x-ray images, patellar tendon moment arms were measured as the perpendicular distance between the patellar tendon action line and the instantaneous center of rotation using publicly available digitizing software (1.41, Wayne Rasband; National Institutes of Health, Bethesda, MD). The instantaneous center of rotation was determined using Reuleaux graphical analysis on images as described by Tsaopoulos et al. (43), and the tendon action line as the line of best fit through the tendon's midline. A second-order polynomial curve fit with $R^2 > 0.90$ was applied to the moment arm length–joint angle relation across the eight angles to obtain curves for each subject.

Isometric torque. Voluntary and electrically elicited (femoral nerve stimulation) knee extension forces were measured on Biodex system 3 isokinetic dynamometer (Biodex Medical Systems). The subjects were seated with their hip joint angle at 80°; the shoulders, hips, and the distal thigh were tightly strapped to the dynamometer; and the lever arm of the dynamometer was attached to the subjects' lower limbs just above the ankle joint. The lever arm length and mediolateral position and the backrest position were adjusted for accurate alignment of the knee axis with the axis of rotation of the dynamometer arm. During maximal contractions, the deformation of the Biodex system and soft tissues of the lower limb resulted in a small joint rotation, so the subjects performed a maximum knee extension contraction during the measurement of the reference angle to provide measurements that are more accurate. The subjects were required to initiate the maximal knee extension contractions as fast and as hard as possible and maintain them for 3 s. The study protocol was designed according to the previous authors who have used the interpolated twitch technique during an MVC (11). Real-time gravity-corrected torque was displayed on a computer screen, and the subjects were strongly verbally encouraged. A doublet supramaximal electrical stimulus was applied to the femoral nerve 2 s before and after the MVC as well as during the torque plateau of the MVC. The interday reliability (2-wk interval) of the torque measurements was determined in a pilot test on eight men, yielding a coefficient of variation (CV) of 6.2% ± 3.2% (mean ± SD) for voluntary torque and 5.0% ± 2.1% for electrically elicited torque.

Five seconds later, the subjects were asked to perform a 3-s maximal ramped isometric knee flexion contraction (reaching maximum gradually within 2 s), from which EMG signals were collected to allow calculations of coactivation levels. Each MVC test was performed twice at the same angle and then moved to the next angle, for the eight different angles (30°, 40°, 50°, 60°, 70°, 80, 90° and 100°; 0° = full knee extension). Joint angles were always tested in the order 30° to 100° to avoid muscle damage induced by the voluntary or electrically elicited contractions at long muscle lengths (21). A 1-min rest was imposed between tests at the same angle, and a 2-min rest was imposed between tests at different knee angles. Finally, the elicited and voluntary contractions were repeated at the first (30°) joint angle to determine whether the protocol had been completed without significant neuromuscular fatigue, where >5% torque decline was set as the criterion level (the CV of the two MVC trials of the same subject at the same knee angle at week 0 ranged from 0.7% to 5.0%).

Torque signals were collected using LabChart v.6.1.3. software (PowerLab System; ADInstruments, NSW, Australia) at a 1-kHz analog–digital sampling frequency and saved to a computer disk. MVC peak torque was taken as the maximum torque achieved before electrical stimulation at each joint angle, peak unpotentiated doublet torque was taken as the peak amplitude of the unpotentiated (i.e., pre-MVC) doublet, and peak potentiated doublet torque was taken as the peak amplitude of the potentiated (i.e., post-MVC)

doublet. Peak voluntary muscle force was subsequently calculated by dividing joint torque by patellar tendon moment arm, and peak isometric force is reported in this study. Peak force measured at each joint angle was also normalized to the peak force obtained at the optimal angle of peak force (i.e., normalized peak force). The training volume was calculated as the sum of 25 knee extension MVC force–time integrals (i.e., five sets of five MVC). The force–time integral was calculated as the area under the force trace from the beginning to the end of each 5-s MVC. Training volume was subsequently compared between groups. Because we expected that increases in force generating capacity would be accompanied by increases in tendon stiffness (44), we assumed the muscle length at each joint angle to be relatively consistent throughout the study as force production increased. Thus, the muscle force–joint angle relation was considered synonymous with the muscle's force–muscle length relation.

Muscle activation. ***Electromyography (EMG/M_{max} ratio).*** Muscle EMG records were obtained from VL, vastus medialis (VM), rectus femoris (RF), biceps femoris (BF), and semitendinosus (ST) muscles using a Bagnoli™ Desktop EMG System (Delsys Inc., Boston, MA) using parallel bar EMG sensors (single differential, DE-2.1) with a detection area of 10 mm^2 and intersensor distance of 10 mm. After the skin was prepared by shaving, abrading, and cleansing with alcohol, the electrodes were placed on the muscles in line with the predicted muscle fiber directions according to the SENIAM recommendations; a self-adhesive round (diameter = 5 cm) reference electrode was placed on the lateral malleolus. Surface EMG signals were collected at an analog–digital conversion rate of 1 kHz, amplified (100×), and stored synchronously with torque data on a computer disk. All EMG signals were band-pass filtered (20–450 Hz), and a root mean square (RMS) algorithm (200-ms averaging window) was applied to the data collected within 1.5 s before the mid-MVC electrical stimulation. The data average was taken as the peak EMG amplitude and was always situated on the plateau of the torque–time curve. The maximum M-wave amplitude (M_{max}) was calculated as the peak-to-peak amplitude of the filtered EMG record and used to normalize the EMG recorded during voluntary contractions (EMG/M_{max} ratio) (23). This ratio was considered a measure of central efferent neural drive (2).

Electrical stimulation procedures. A self-adhesive cathode (diameter = 1 cm) was placed over the femoral nerve, and the anode (diameter = 1 cm) was placed 2 cm lateral to it. Doublet square-wave pulses (pulse width = 200 μs, frequency = 100 Hz) were delivered to the femoral nerve using a constant current stimulator (Digimeter, model DS7AH, Welwyn Garden City, UK). The subjects were seated with their knees at an optimal angle (60°), and the optimal position for the cathode was determined as that eliciting the largest VL M-wave response at a low stimulation intensity. The stimulation intensity was then gradually increased until the M-wave plateau (M_{max}) was obtained, and subsequent stimulations at all knee angles were applied using a current equivalent to 140% of M_{max}. During the procedure of finding the electrical stimulation intensity for the maximal M-wave response (M_{max}), a twitch torque window was simultaneously displayed next to the EMG signal window, so checking that both M_{max} and twitch torque reached a plateau was made (20). Doublet stimulations were applied 2 s before, during, and 2 s after the MVC performed at each knee angle. The voluntary activation percentages (VA%) measured during MVC were quantified by comparing the amplitudes of the superimposed doublet forces with the resting doublet force evoked by electrical stimulations, using the following formula (18,39):

$$VA\% = \left(1 - \left(\frac{\tau_{doublet,MVC}}{\tau_{doublet,pot}}\right)\right) \times 100$$

where $\tau_{doublet,MVC}$ is doublet force elicited during MVC and $\tau_{doublet,pot}$ is the potentiated doublet force (elicited after the MVC).

Antagonist coactivation. To estimate the level of antagonist coactivation, BF and ST EMG activity was measured during ramped maximal isometric knee flexion contractions performed at each joint angle. After the application of a 50-ms RMS filter, the relative activity of the muscle during knee extension contractions was expressed relative to this maximum. In addition, the antagonist torque of the knee flexors during maximal isometric knee extension contractions was calculated assuming that the knee flexion moment was equally distributed across BF and ST muscles; thus, second-order polynomial equations describing the torque–EMG relations were calculated and provided the EMG amplitude per half of the total joint torque. The resulting coefficients were used to estimate the knee flexion moment from the EMG values obtained during the knee extensions (25).

Quadriceps muscle CSA and volume. Muscle CSA was measured (Siemens 1.5TMagnetomEspree Open Bore MRI scanner; Siemens, Sweden) contiguously (5-mm-slice thickness) from the proximal end of the greater trochanter to the medial femoral condyle in both legs using an axial (transverse) T_1-weighted spin echo acquisition sequence (400 × 300 mm field of view, resolution 512 × 192, TR = 450 ms, TE = 12 ms, slice width = 5 mm, flip angle = 70°, bandwidth 130 Hz·Px^{-1}). Also, T_2-weighted images (8 mm slice thickness) were taken from the middistance between the proximal end of the greater trochanter and the medial femoral condyle. The subjects rested supine with their legs together, and a strap was placed around the knees to allow them to relax without lateral hip rotation occurring. A phased array spine coil was used posteriorly, and two phased array body surface coils were placed anteriorly along the thigh. Four separate slice groups were scanned at isocenter to minimize any distortion effects of the magnetic field. The subjects were required to avoid physical activity for 48 h and sit quietly for 20 min before scanning. The reliability of CSA and volume measurements using MRI has been shown to be high using procedures similar to those adopted presently (35). Muscle volume and CSA were digitized using

image-processing software (OsiriXv.4.0; Pixmeo, Switzerland). All MRI scans were digitized twice for RF, VL, VM, and vastus intermedius (VI), with care being taken to avoid major blood vessels and fat infiltrations. Digitizing CV for all quadriceps heads ranged 0.8%–0.9%. CSA was then selected at 10% intervals along the single muscle length for CSA analysis (i.e., CSA of the 5-mm slice). Muscle volumes were subsequently calculated using an OsiriX software algorithm. The image density (sum of gray scale values of all pixels in the region of interest divided by the total number of pixels) of T_2 MRI images were analyzed (OsiriXv.4.0, Pixmeo) by tracing the whole quadriceps muscles on images to ensure that prior exercise did not influence the cross-sectional area measures (5).

Fascicle length (L_f). B-mode axial-plane ultrasound (AlokaSSD-a10, software number 6.1.0; Aloka, Tokyo, Japan) images of VL were taken at 33%, 50%, and 66% (i.e., proximal region, midregion, and distal region) of the distance from the greater trochanter to the lateral epicondyle and of RF at 56% (i.e., midregion) from the anterior superior iliac spine to the superior border of patella using a 10-MHz linear-array probe (60-mm width) in an extended field-of-view mode (sampling frequency = 90 frames per second). The subjects rested supine with knee and hip joint angles at 0° (i.e., full extension). Two scans were acquired during each session. The measurement reliability by the experimenter using the extended field-of-view methodology has been described previously (38); the average difference in VL L_f measured when markings were removed between scans (i.e., assessing "intersession" reliability) was 3.0 ± 2.5 mm corresponding to 3.8% ± 3.2% with an intraclass correlation coefficient (ICC) of 0.95 (95% confidence interval, 0.80–0.99); the absolute intersession measurement error was 1.8 mm. Because of the complex architecture of RF, it was possible to obtain clear images from only seven subjects in both groups.

Statistical analysis. Data distribution was tested with the Shapiro–Wilk test. For intra- (i.e., comparison of changes in dependent variables over time at eight angles) and intergroup (i.e., comparison of changes in dependent variables between the two groups) comparisons, two-way and three-way ANOVA, respectively, with repeated measures were used. An independent *t*-test was used as a *post hoc* test when comparing SL and LL. Paired *t*-tests were used to compare within-group changes across the training period (i.e., after weeks 0 and 3, weeks 3 and 6, and weeks 0 and 6). Shifts in T_2 MRI image density from pre- to posttraining and the group–time interaction were tested with repeated-measures ANOVA. Pearson correlation coefficients were computed to examine the (linear) relations between the training-induced changes in the measured variables. The use of multiple correlations was important to test the specific hypothesis that hypertrophy in specific muscle regions would be associated with increases in force production at specific muscle lengths. Because of the increased likelihood of type 1 error, the overall trends of the correlations, rather than their specific magnitudes, are considered most important in this analysis. Antagonist coactivation (i.e., calculated knee flexion torque/knee extension torque ratio) and VA% were compared between joint angles, between groups, and over time using the nonparametric Friedman ANOVA because a Shapiro–Wilk test indicated nonnormal data distribution. CV (SD/mean) was calculated to describe experimenter reliability, and ICC was calculated to describe interday (i.e., subjects plus experimenter) reliability. During all statistical tests, the α-level was set to 0.05. Values are presented as mean ± SD in the text and tables and as mean ± SE in the figures. Effect sizes (the mean change score divided by the pooled mean SD) were calculated on near-significant results ($P < 0.1$), where an effect size of 0.2 was considered as small, 0.5 as medium, and 0.8 as large. SPSS Statistics software version 20.0.0 (IBM Corp., New York) was used for all statistical analyses.

RESULTS

Training Angle, Joint Moment Arms, and Total Training Volume

SL and LL trained at angles ranging from 30° to 50° (peak force at the training angle at week 0 = 3984 ± 1198 N) and 75° to 100° (peak force at training angle at week 0 = 3397 ± 693 N), respectively, but despite some individual changes being made after 3 wk of training, there was no significant change in the training angles over the 6-wk period (average training angle ± SD was 43.1° ± 4.6° at weeks 0 and 38.1° ± 3.7° at week 6 in SL, and 86.9° ± 6.5° at weeks 0 and 87.5° ± 6.0° at week 6 in LL). Moment arms ranged from 42.1 to 63.4 mm, with the largest occurring at extended knee positions. There were no differences in moment arm magnitudes between the groups (the average moment arm was 53.8 ± 5.3 mm). The values of ICC for MVC measured at 30° at the beginning and end of each testing session were 0.95, 0.95, and 0.96 at weeks 0, 3, and 6, respectively. The change in the training volume (i.e., force–time integral) within the first training session (week 1, session 1) differed between the SL and LL; changes in the torque–time integral of a single MVC contraction (i.e., effect of fatigue) were 1.2% ± 10.8% in SL but 19.4% ± 12.6% in LL ($P = 0.03$). However, the between-group difference did not continue through the training period. The change in training volume within the last training session (week 6, session 18) was not different between the groups (the change in the torque–time integral per MVC was 4.9% ± 11.8% in SL and 1.4% ± 18.1% in LL, $P = 0.4$), and there was no difference between the groups over the training period. There were no between-group differences in the changes in the total training volume (i.e., torque–time integral) with an average change in training volume from week 0 to week 6 of 9.1% ± 6.3% [$P = 0.06$, effect size (ES) = 0.6] and 8.5% ± 8.1% ($P = 0.07$, ES = 1.2) in SL and LL, respectively.

Maximum Voluntary Isometric Contraction (MVC) Force

The ANOVA results revealed a significant angle–time interaction in SL ($P = 0.004$). Further analyses showed that peak quadriceps force increased at the 40° joint angle after 6 wk of training (13.4% ± 9.7%, $P = 0.01$; Table 2). The peak force normalized to the peak obtained at the optimum angle (i.e., normalized force) increased after 3 wk at 30° (5.9% ± 8.6%, $P = 0.02$), 40° (7.4% ± 9.1%, $P = 0.001$), and 50° (6.9% ± 7.2%, $P = 0.04$), and also after 6 wk at 30° (9.0% ± 7.0%, $P = 0.02$), 40° (10.7% ± 4.5%, $P = 0.001$), and 50° (8.1% ± 7.3%, $P = 0.05$).

In LL, peak quadriceps force did not change significantly with training at any joint angle (Table 2). The individual force–angle relations showed distinct adaptation patterns as shown in Figure 2.

Doublet Force (Evoked before MVC)

The absolute values of doublet force are given in Table 2. ANOVA showed significant group–angle–time interaction effect for doublet force ($P = 0.02$). Further analyses revealed that there was no significant increase in doublet force in SL. Doublet force changed in LL at 90° at weeks 3 (11.7% ± 15.3%, $P = 0.03$) and 6 (6.0% ± 9.7%, $P = 0.04$).

Muscle Activation

EMG amplitude normalized to M_{max} (EMG/M_{max}). The EMG/M_{max} data are presented on Figure 3. The ANOVA results revealed a significant group–angle–time interaction effects for VL, VM, and RF EMG/M_{max} values ($P = 0.02$, $P = 0.03$, and $P = 0.03$, respectively). In SL, the VL EMG/M_{max} ratio increased by week 3 at 40° (26.4% ± 44.0%, $P = 0.04$)

TABLE 2. Absolute values of study variables measured at weeks 0, 3, and 6 in SL (group that trained at short muscle length) and LL (group that trained at long muscle length).

Variable	Week 0	SL Week 3	Week 6	Week 0	LL Week 3	Week 6
MVC force (N)						
30°	3379 ± 1011	3595 ± 1027	3752 ± 716*	2778 ± 463	2748 ± 532	3119 ± 690
40°	3984 ± 1197	4304 ± 1250*	4549 ± 860**	3518 ± 469	3571 ± 716	3824 ± 852
50°	4610 ± 1496	4874 ± 1167*	5009 ± 709*	4006 ± 489	4098 ± 931	4325 ± 845
60°	5112 ± 1426	5206 ± 1059	5271 ± 776	4328 ± 609	4565 ± 996	4633 ± 900
70°	4993 ± 1266	5000 ± 789	5089 ± 933	4316 ± 572	4478 ± 1118	4408 ± 860
80°	4715 ± 1130	4468 ± 746	4644 ± 899	3943 ± 628	3966 ± 989	4037 ± 1050
90°	4281 ± 1170	4131 ± 794	4200 ± 806	3943 ± 693	3966 ± 747	4037 ± 804
100°	4194 ± 1042	4009 ± 825	4002 ± 928	3321 ± 760	3480 ± 736	3706 ± 637
Doublet force (N)						
30°	67.5 ± 8.7	67.5 ± 14.1	64.8 ± 18.2	59.5 ± 11.4	59.6 ± 5.6	70.9 ±14.2
40°	84.2 ± 8.3	78.9 ± 12.6	82.6 ± 18.0	73.1 ± 16.5	78.3 ± 6.1	74.3 ± 14.0
50°	90.2 ± 8.3	90.2 ± 14.5	87.3 ± 16.2	86.0 ± 19.6	87.3 ± 10.6	83.6 ± 19.6
60°	96.1 ± 4.5	94.6 ± 5.4	89.4 ± 18.8	87.8 ± 20.5	87.0 ± 12.3	82.5 ± 18.0
70°	92.3 ± 8.9	93.1 ± 5.9	87.6 ± 14.4	90.7 ± 8.5	89.4 ± 10.8	87.9 ± 16.6
80°	89.3 ± 9.5	84.3 ± 13.6	82.4 ± 10.0	77.7 ± 18.2	86.2 ± 7.2	81.7 ± 20.0
90°	86.5 ± 13.0	82.1 ± 13.8	79.4 ± 13.9	76.7 ± 18.0	86.1 ± 10.6*	79.1 ± 20.4#
100°	94.2 ± 10.8	83.0 ± 20.7	88.2 ± 12.7	82.4 ± 23.2	92.5 ± 8.6	86.7 ± 22.9
Voluntary activation (VA%)						
30°	94.8 ± 7.4	95.9 ± 5.0	92.5 ± 8.2	95.1 ± 5.0	93.1 ± 8.7	97.5 ± 4.6
40°	91.9 ± 10.3	96.1 ± 5.4	94.4 ± 7.1	90.8 ± 8.0	91.5 ± 11.8	93.2 ± 14.4
50°	91.0 ± 10.3	94.1 ± 9.5	97.2 ± 4.1	90.1 ± 9.3	90.1 ± 12.0	95.1 ± 9.5*
60°	96.6 ± 4.7	96.7 ± 7.2	97.0 ± 3.8	92.8 ± 7.6	93.9 ± 5.2	98.6 ± 2.6*
70°	97.9 ±3.9	95.9 ± 7.8	98.6 ± 4.3	97.5 ± 3.2	97.4 ± 2.5	93.5 ± 16.5
80°	99.1 ± 2.5	97.1 ± 7.5	100.0 ± 0.0	99.1 ± 1.4	98.8 ± 2.6	99.0 ± 2.7
90°	99.1 ± 1.9	100.0 ± 0.0	100.0 ± 0.0	99.3 ± 1.0	100.0 ± 0.0	99.1 ± 2.4
100°	98.3 ± 3.5	100.0 ± 0.0	96.0 ± 8.4	97.7 ± 4.1	100.0 ± 0.0	99.4 ± 1.7
Antagonist cocontraction (agonist torque/antagonist torque)						
30°	23.9 ± 8.9	8.2 ± 7.8	22.9 ± 10.9	9.5 ± 8.1	13.6 ± 4.9	9.2 ± 11.3
40°	14.0 ± 9.8	10.4 ± 7.4	18.0 ± 7.1	5.0 ± 3.3	10.7 ± 5.4	8.4 ± 8.9
50°	16.1 ± 9.9	5.5 ± 4.7	15.4 ± 6.1	5.3 ± 4.5	11.1 ± 5.1	5.6 ± 7.5
60°	13.6 ± 7.3	7.5 ± 5.5	13.3 ± 6.5	4.1 ± 7.1	10.3 ± 5.8	6.6 ± 7.3
70°	18.7 ± 9.7	8.1 ± 5.6	12.4 ± 7.8	5.0 ± 9.0	11.2 ± 7.2	5.0 ± 5.4
80°	16.8 ± 12.5	6.4 ± 7.2	12.3 ± 9.1	3.6 ± 5.4	11.6 ± 6.3	4.7 ± 4.6
90°	14.7 ± 11.7	5.2 ± 6.8	10.5 ± 8.3	5.0 ± 3.3	12.2 ± 6.8	3.5 ± 4.1
100°	11.6 ± 10.9	5.9 ± 4.3	9.0 ± 8.2	3.9 ± 3.9	4.1 ± 2.0	3.8 ± 3.9
	Pre (week 0)	**Post (week 6)**	**Δ%**	**Pre (week 0)**	**Post (week 6)**	**Δ%**
Quadriceps muscle volume (cm^3)						
VL	680.7 ± 144.4	674.9 ± 152.2	−1.2 ± 1.1	634.7 ± 142.5	677.5 ± 162.8	6.3 ± 1.4**
VM	502.2 ± 89.6	497.7 ± 96.0	−1.2 ± 1.3	468.5 ± 160.2	491.1 ± 166.4	4.8 ± 0.9**
VI	570.4 ± 134.1	573.7 ± 132.9	0.5 ± 1.8	532.1 ± 197.9	548.7 ± 200.9	3.1 ± 1.8
RF	294.8 ± 67.4	294.2 ± 63.0	0.04 ± 1.1	267.2 ± 64.4	290.7 ± 67.4	8.2 ± 1.5**
Quadriceps	2048.2 ± 391.1	2040.6 ± 395.6	−0.5 ± 1.1	1902.5 ± 530.1	2008.0 ± 567.6	5.2 ± 1.0**
Fascicle length (L_f, mm)						
VL_{prox}	88.2 ± 10.2	90.2 ± 11.7	1.9 ± 6.1	86.6 ± 11.2	89.5 ± 8.9	1.3 ± 5.9
VL_{mid}	82.8 ± 7.6	87.8 ± 8.3	5.6 ± 3.7**	83.0 ± 10.8	86.2 ± 7.9	3.8 ± 7.2
VL_{dist}	84.4 ± 14.9	83.8 ± 16.2	1.1 ± 7.2	77.9 ± 15.1	83.1 ± 16.8	5.8 ± 6.4*
RF_{mid}	119.0 ± 11.2	119.4 ± 13.3	0.1 ± 4.2	97.7 ± 20.9	98.5 ± 17.8	2.2 ± 13.8

Muscle volume and L_f (fascicle length) were measured at weeks 0 and 6; the percentage change from week 0 to week 6 is given. Significant change from baseline (week 0) is marked with * for $P < 0.05$ and ** for $P < 0.01$. Significant change from 3 to 6 is marked with # ($P < 0.05$).

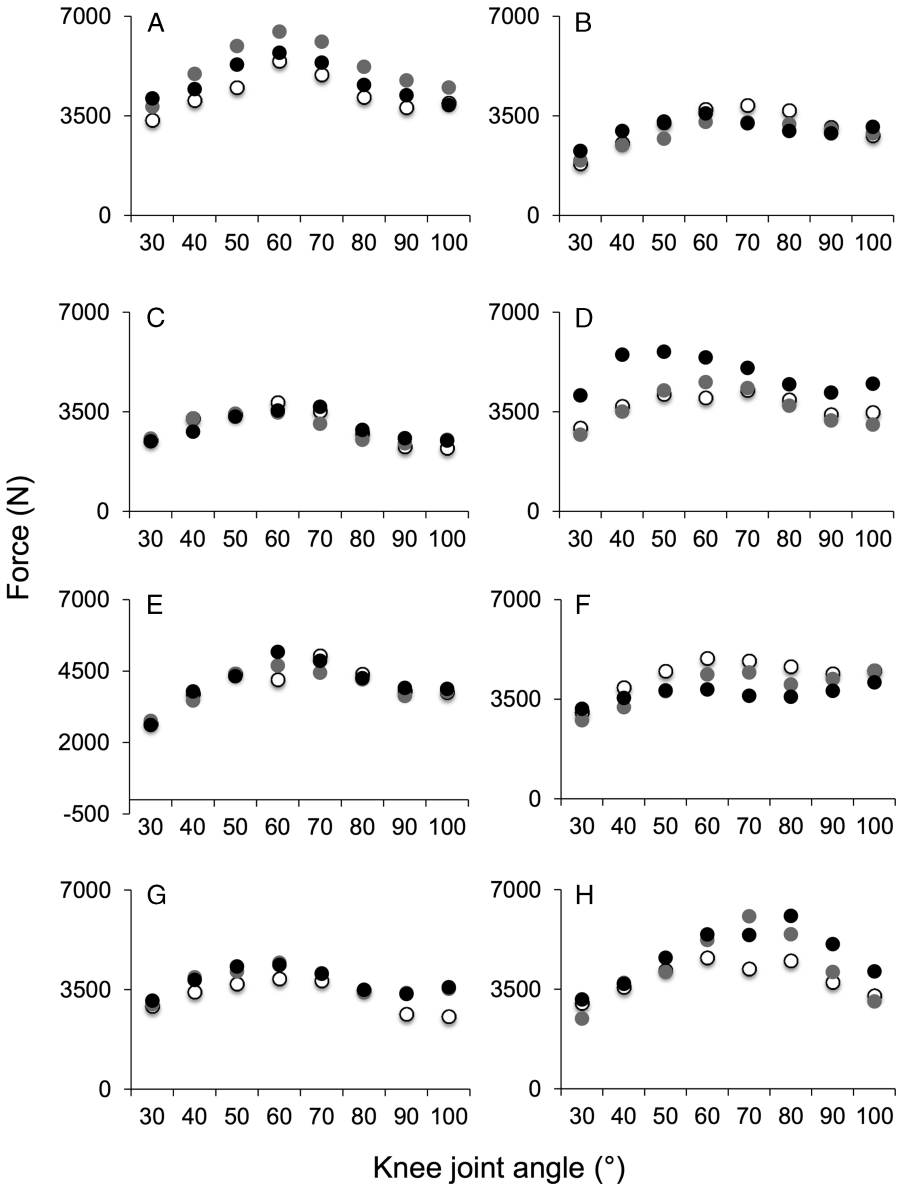

FIGURE 2—Force–angle relations for each subject (A–H) in the group training at long muscle length, demonstrating high variability in the angle-specific response to training. White circles = force at week 0, gray circles = force at week 3, and black circles = force at week 6. The training angle did not differ between the LL subgroups (P = 1.00).

and 50° (22.5% ± 42.1%, P = 0.04). The change in peak force at 40° at week 6 correlated significantly with the changes in VL EMG/M_{max} at 40° (r =0.84, P = 0.02). VM EMG/M_{max} changed across all joint angles (4.7%–29.8%, P < 0.04) after 6 wk of training. The change in peak force at 30° and 40° correlated significantly with the changes in VM EMG/M_{max} at 30° (r = 0.87, P = 0.03) and 40° (r = 0.88, P = 0.04) after 6 wk. Thus, increases in EMG/M_{max} tended to occur around the training angle. There were no changes in RF EMG/M_{max}. In LL, the VL EMG/M_{max} ratio increased across all angles by week 3 (22.1%–59.5%, P < 0.05) and increased further by week 6 at 100° only (32.3%, P = 0.04). The RF EMG/M_{max} ratio increased from weeks 3–6 at 50° to 80° (30.4%–40.0%, P < 0.02). There were no changes in VM EMG/M_{max}.

Voluntary activation percentage (VA%). Absolute values for VA% are given in Table 2. ANOVA showed significant group–angle–time interaction effect (P = 0.001). Further analyses revealed that there were no significant changes in SL. In LL, VA% increased from weeks 3–6 at 50° (5.5% ± 5.5%, P = 0.03) and 60° (4.8% ± 4.7%, P = 0.03). On the basis of two-way ANOVA, VA% increased with increasing joint angle from knee 30° to 100° of flexion similarly in both groups at week 0 and week 3; at week 6, there was no effect of angle on VA% in either group.

Antagonist coactivation (knee flexion τ/knee extension τ). Antagonist coactivation did not change in either group and did not differ between joint angles (Table 2). The average coactivation ratios in SL were 12.5% ± 11.8%,

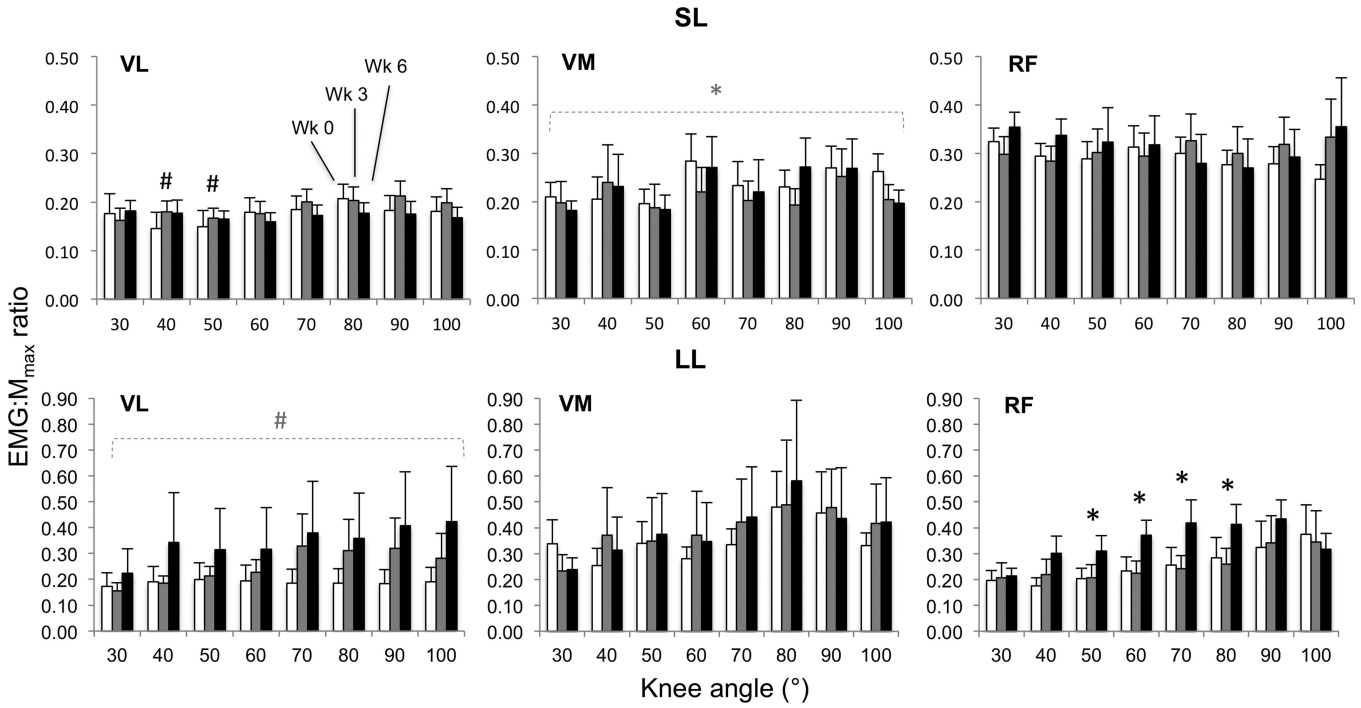

FIGURE 3—EMG/M_{max} at eight different knee angles in SL (A) and in LL (B) groups. Significant change from week 0 to 3 ($P < 0.05$) = #, from week 3 to 6 = *, across all joint angles from week 0 to 3 = #, across all joint angles from week 0 to 6 = *. White columns show EMG/M_{max} at week 0, gray columns at week 3, and black columns at week 6.

8.3% ± 6.5%, and 11.2% ± 10.7% at weeks 0, 3, and 6, respectively, and in LL were 5.2% ± 5.6%, 10.6% ± 15.3%, and 5.9% ± 6.6% at weeks 0, 3, and 6.

Quadriceps Muscle CSA and Volume

Repeated-measures ANOVA showed no changes in quadriceps muscle T_2 MRI image density over time (P = 0.798) or group–time interaction effects (P = 0.718), suggesting that fluid retention could not have been a factor influencing the different between-group changes in CSA. Quadriceps muscle CSA and volume changed after training only in LL. The CSA values before and after training and percentage changes at the nine sites along RF, VL, VM, and VI are shown in Figure 4, and the volume data are presented in Table 2. There were no differences in the changes in muscle volume between RF, VL, and VM, and no change in volume and CSA was detected in VI. The change in force at each angle was correlated with the change in CSA at different muscle regions in LL after 6 wk of training, and

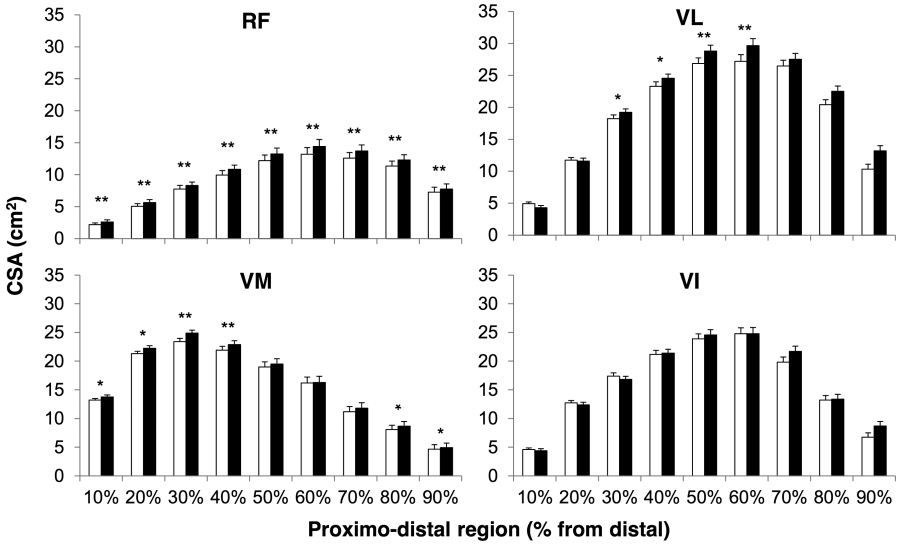

FIGURE 4—Pretraining (white columns) and posttraining (black columns) values for RF, VL, VM, and VI anatomical cross-sectional areas (at 10% intervals from origin to insertion) in the group training at long muscle length. Significant changes in CSA are marked with *($P < 0.05$), **($P < 0.01$), and *($P < 0.001$).**

interestingly, the absolute changes in force production (i.e., week 6 MVC force – week 0 MVC force) at some knee angles were significantly correlated with changes in CSA at specific regions of the quadriceps muscle: the change in peak force at 40° was correlated with the change in CSA in the distal (10%, 30%, and 40%) regions of RF (Fig. 4C; r = 0.89, P = 0.01; r = 0.73, P = 0.04; r = 0.77, P = 0.03, for 10%, 30%, and 40% region, respectively). Changes in peak force at 60°–90° knee angles were correlated with increases in CSA at 60% (i.e., midmuscle) in VL (r = 0.81, P = 0.02; r = 0.82, P = 0.01; r = 0.86, P = 0.01; r = 0.79, P = 0.02, for 60°–90° knee angles, respectively), and the changes in peak force at 30° and 40° were correlated with changes in CSA at 80% (r = 0.85, P = 0.01; r = 0.80, P = 0.02) and 90% (r = 0.80, P = 0.02; r = 0.92, P = 0.001 for 30° and 40° knee angles, respectively) of VL (i.e., proximal). The change in peak force at 30° and 40° was correlated with the changes in VM CSA at the 10% region (i.e., distal; r = 0.79, P = 0.02, and r = 0.76, P = 0.03, for 30° and 40° knee angles, respectively).

Fascicle Length (L_f)

Absolute L_f values are presented in Table 2. Mid-VL L_f increased in SL by 5.6% ± 3.7% (5.0 ± 3.5 mm, P = 0.01); however, the trend toward an increase in LL was not significant (3.8% ± 7.2%, 3.2 ± 6.3 mm; ES = 0.5, P = 0.20). When SL and LL data were pooled, L_f at week 0 was found to correlate significantly with both the absolute (r = −0.50, P = 0.05) and relative (r = −0.54, P = 0.03) change in L_f, indicating that, regardless of group, subjects who had a shorter L_f before training showed the greatest increase in L_f. Therefore, the data were reanalyzed using pretraining L_f as a covariate. The analysis revealed that there were no between-group differences in the change in L_f despite there being an increase in L_f overall (i.e., both groups combined, P = 0.001). Thus, the L_f increase in the VL midregion was considered identical between the groups, having a mean of 5.4% ± 4.9% (4.8 ± 4.4 mm, P = 0.001).

One-way ANOVA (weeks 0 and 6) showed significant group (SL and LL) effect for distal VL L_f (P = 0.01). Distal and proximal VL L_f did not change in SL. L_f increased 5.8% ± 6.4% (0.5 ± 0.5 mm, P = 0.02) at the distal region of VL in LL and did not change in the proximal region. RF L_f did not change in either group. The change in L_f did not correlate with the change in force in either group.

DISCUSSION

Specific neuromuscular adaptations in response to isometric knee extension training at short (SL, knee extended) versus long (LL, knee flexed) muscle lengths were examined. The subjects in both groups trained at knee angles that corresponded to 80% of their maximum voluntary quadriceps muscle force, so they produced the same forces over the 6-wk training period. A major finding was that clear muscle length (i.e., joint angle) specificity was only seen in SL, whereas adaptations in LL were broader and varied considerably between subjects. Importantly, the force increase at the training angle in SL appeared to be underpinned by changes in agonist muscle activation, as indicated by increases in the VL EMG/M_{max} ratio around the training angle (40° = 53.5% and 50° = 55.6%). These changes occurred without a shift in the electrically elicited force–angle relation or detectable changes in muscle CSA or volume. In LL, however, muscle volume and CSA increased significantly and the change in CSA of specific muscle regions within the quadriceps correlated well with muscle force produced at joint angles of 30° (i.e., extension) and 100° (i.e., flexion). This occurred with increases in RF EMG/M_{max} amplitudes and increased voluntary activation (VA%) measured at angles different from those adopted during training (i.e., 50° and 60°) and without a change in antagonist cocontraction, and these changes were not correlated with changes in angle-specific MVC force. The results suggest that selective regional muscle hypertrophy was most clearly associated with the variable changes in the muscle's force–length relation in LL. Interestingly, a small (5.4% ± 4.9%) and similar increase in L_f was found in both training groups. Although some evidence of an angle-specific adaptation was indicated by the finding that L_f in the distal region of VL increased significantly only in LL (5.8% ± 6.4%), the changes in L_f were not correlated with the changes in force in either group (or when all subjects were considered together). Thus, changes in L_f did not appear to be related to the change in angle-specific force. These results highlight the complex adaptive processes underpinning changes in the force–length (and torque–angle) relation in composite muscle groups such as the quadriceps femoris and show that the mechanisms underpinning joint angle-specific changes (especially muscle hypertrophy) can be different when training is performed at different muscle lengths.

Angle-Specific Changes in MVC Force

The angle-specific changes in MVC force found presently are in line with findings by others; it has been shown that the increase in strength is specific to the training angle after training at short muscle lengths (26,32), whereas changes appear more variable after training at longer muscle lengths (26,32). However, the present data further show that there was a substantial interindividual variability in the response in LL even though the change in MVC force did not reach significance. Importantly, the MVC force increase at short lengths in LL could not be explained by fatigue influencing MVC force produced at the end of the testing sessions because a high ICC (0.95–0.96) was obtained between the MVC values measured at 30° at the beginning and end of the testing sessions. It is also unlikely that a selective increase in tendon stiffness (i.e., influencing force production at specific muscle lengths) could explain the finding because several

studies have found a limited effect of strength training on tendon stiffness with <6 wk of training (30,44). Thus, any changes in tendon stiffness in the present study would be expected to be small, and changes in tendon properties are expected to have a smaller effect on the length-dependent increase in torque than neural and muscular hypertrophic adaptations (31). In addition, Kubo et al. (32) found an increased patellar tendon stiffness, which should shift the muscle's length–force relation toward shorter lengths, only in a group that performed isometric training at long muscle lengths. These findings of Kubo et al. cannot be reconciled with the present findings, where SL subjects clearly improved their MVC force production at shorter muscle lengths. Rather, the finding that the individual MVC force increases were correlated with changes in region-specific muscle hypertrophy suggests that the individual MVC force increases in LL might have resulted from muscular adaptations, as discussed below. These observations of variable training adaptations in LL and the finding that muscle hypertrophy did not translate into consistent and significant increases in isometric muscle force are important and need subsequent examination with a larger sample size.

Changes in Muscle Activation

Previous studies have indicated that neural adaptations primarily underpin angle-specific force increases (12,26), and force increases that occur at a specific joint angle only (13,28) after isometric training. In the present study, neural adaptations were quantified by three measures: 1) changes in the EMG signal amplitude measured during maximal voluntary contractions, normalized to the M-wave elicited by supramaximal femoral nerve stimulation (i.e., EMG: M_{max}); 2) changes in voluntary activation (VA%) using the twitch interpolation technique; and 3) changes in antagonist coactivation.

Normalized EMG was considered to be reflective of central drive because changes in muscle membrane properties, and other influencing factors distal to the CNS, are expected to be largely removed by the M-wave normalization process (2) and increase the reliability of the measurement (23). It is known that several anatomical (i.e., thickness of subcutaneous tissue, tissue inhomogeneities, size and distribution of motor unit territories in the muscle, length of fibers) and physiological factors (i.e., fiber membrane, fiber type and motor unit properties), which might have been influenced by resistance training in the present study, affect the surface EMG signal (14,24). Factors such as electrode location and the detection system used to obtain the recordings might also have affected the present results with the repeated-measures design (14); however, these potential effects were minimized by strictly following the rules for EMG electrode placement and methodology throughout, and the M-wave normalization process.

The finding that VL EMG/M_{max} increased at short muscle lengths (40° and 50°) after just 3 wk of training in SL is thus considered indicative of a joint angle (muscle length)-specific increase in efferent neural drive (22). Notably, changes in both VL and VM EMG/M_{max} at short muscle lengths (30° and 40°) were significantly correlated with the angle-specific MVC force increase at 30°–40°, providing further support for the hypothesis that the changes in MVC force production in SL were dependent upon neural adaptations. In contrast, changes in EMG/M_{max} were not muscle length-specific in LL but occurred more around the optimal (i.e., nontraining) angle; VL EMG/M_{max} increased at all angles by week 3 and increased further by week 6 only at 100°, whereas RF EMG/M_{max} increased by week 6 from 50° to 80°. These changes in EMG/M_{max} are difficult to interpret because of the highly variable pattern of individual changes in MVC force in LL, and because of the lack of significant relation between the changes in EMG/M_{max} and other study variables. In addition, muscle morphological changes (e.g., increase in muscle region-specific CSA and L_f or changes in muscle fiber type) might have had some effect on the EMG recordings in the present study (14). Thus, although the role of neural adaptations is not clear in LL in this study, these data suggest that neural adaptations were not a major factor underpinning the individual MVC force adaptations in LL.

The interpolated twitch technique (VA%) was used as an additional test of muscle activation, despite the assessment of muscle activation using the interpolated twitch technique also being influenced by the muscle's contractile characteristics (34) and being a less sensitive measure of motor neuronal excitation at near-maximal forces (18); the VA% calculation also has a lesser validity in muscles with greater series elasticity, as would be likely in longer muscles and/or tendons such as the quadriceps femoris (33). In the present study, there was no significant change in VA% in subjects who trained at a short muscle length, and VA% increased only at nontraining angles (50° and 60°) in LL. Moreover, these increases in VA% were not correlated with changes in muscle MVC force. It may be important that VA% measured at shorter muscle lengths was significantly less than at longer muscle lengths before training in all subjects, and that the between-angle differences in VA% after 6 wk of training disappeared in both groups. An impaired voluntary activation at short muscle lengths has been previously shown for both the elbow flexor (39) and the quadriceps (28) muscles, and such activation differences have been associated with length-dependent differences in muscle doublet characteristics (39). It is known that electrically elicited muscle doublets are smaller and of shorter duration at shorter muscle lengths, and higher frequencies are required to produce tetanic forces in stimulated contractions (4). These findings suggest that a greater efferent neural drive would be required to maximally activate muscles at shorter lengths. It has been proposed that a functional reserve of motor units exists that is not readily available for use during maximal contractions, and that resistance training may allow the subject to learn to fully activate all motor units available (13). That is, the participants "learned" to maximally recruit available muscle

fibers or "disinhibit" motor units with training at short muscle lengths, which might explain the lack of changes in force at angles other than those close to the training angle in SL. This idea has been adopted as an explanation for angle specificity after isometric training previously (3). If this hypothesis is true, then the training intensity in SL might have been very slightly less relative to LL, at least in the early stages of the 6-wk training program.

Antagonist cocontraction modification with training has been shown to affect the total torque generated about a joint (12). In this study, antagonist cocontraction did not change significantly with training. This finding is in agreement with other studies showing that antagonist joint torque contributions are generally small and could not account for large changes in angle-specific force (7).

Changes in Muscle CSA and Volume

An important finding of the present study was that muscle hypertrophy occurred only in LL, despite both groups training at the same muscle force level. Four possible reasons might explain this finding. First, mechanosensitive signaling mechanisms may have only been critically upregulated when the muscle was activated at the longer length (41). Second, Ca^{2+}-dependent signaling cascades might only have been augmented when the muscle was stretched and accumulation of intracellular calcium could occur through stretch-activated ion channel activation (9). Third, the metabolic stress may have been greater during training in the lengthened muscle position, which is thought to promote protein synthesis (41); in fact, Kooistra et al. (29) have found an approximately 20% lower steady-state muscle oxygen consumption at a 30° compared with a 90° knee angle during maximal voluntary and electrically induced isometric contractions, which was suggested to be due to a lower maximal activation as detailed above (a lesser VA% at short muscle lengths before training was also found in the present study). Nonetheless, a training-dependent difference in the release of auto/paracrine growth factors such as IGF-1Ec (i.e., mechano growth factor), which are secreted in response to mechanical loading in muscle (16), is probably not a major factor because the muscle force produced and training volume accrued were identical between the groups. Potentially, 6 wk might not have been a sufficient time for muscle hypertrophy in SL in the present study; Kubo et al. (32) trained participants for 12 wk and found muscle hypertrophy to occur in both short (knee angle = 50°) and long (knee angle = 100°) training groups. Regardless, the finding that significant hypertrophy occurred only in LL is of substantial practical importance and needs to be more explicitly studied in the future.

There can be a concern in exercise training experiments that residual edema might inflate muscle CSA during posttraining testing. In the present study, the participants were scanned 3–4 d after the last training session and were well accustomed to the exercise training, so we expect that changes in muscle size resulting from osmotic fluid shifts are likely to be negligible. However, there is a possibility that the LL group may have shown a greater fluid retention (contributing to their CSA increase) by quantifying changes in the T_2 MRI signal between pre- and posttraining scans, i.e., by examining changes in the image density of the T_2-weighted MRI images (5). The finding of a lack of change in image density over time and the lack of group–time interaction effect indicates that fluid retention could not have been a factor influencing the different between-group changes in CSA.

The increases in RF (8.1%), VL (6.3%), and VM (4.6%) volume in LL subjects were of similar magnitude, which is not completely consistent with previous reports (31,32) because increased volume has been found also in VI in these previous studies. Nonetheless, a novel observation in the present study was that hypertrophy at specific proximodistal muscle regions correlated well with the individual (angle-specific) increases in peak force. These data lend support to the hypothesis that selective hypertrophy of specific regions within the quadriceps might be sufficient to result in joint angle-specific force increases (37,45), although this has not been found after dynamic training (39). In particular, RF hypertrophy might have been a significant contributor to the peak force increase at short muscle lengths in some of the subjects (Fig. 2) because it has been shown that RF exerts its maximum force at shorter lengths than the other quadriceps components (19). A specific examination of training at a long muscle length using a larger samples size could be implemented to directly test this hypothesis.

Changes in L_f

A surprising finding was that L_f in the VL midregion increased by 5.6% (approximately 5 mm) in SL, which was statistically similar to the 3.8% (approximately 3.2 mm) increase in LL. This magnitude of change is in agreement with the results in the study of Blazevich et al. (5) who found a similar (4.7% ± 1.7%) increase in VL L_f after 10 wk of strength training. Other authors (42) have reported greater increases in VL L_f after 35 d of resistance training (9.9% ± 1.2%); however, L_f was measured with the knee flexed, which could have placed a greater longitudinal strain on the fascicles and increased the measured change. Given that the muscles were exercised isometrically at short muscle lengths in SL, the result has the important implication that the provision of muscular stress at long muscle lengths is not a requirement for L_f adaptation. It appears, then, that other mechanosensitive mechanisms must be involved. An alternative explanation that should be considered, however, is that changes in L_f reflect adaptations other than adjustments in serial sarcomere number within the constituent muscle fibers, such as changes in resting muscle compliance or end point tension (i.e., an increased longitudinal tension force, perhaps resulting from an increase in tendon stiffness) that result in a greater fascicle resting length.

It should be pointed out that distal region of VL L_f increased (by 5.8%) in LL only, which might be considered as some indication that a muscle length-specific adaptation in L_f occurred in response to the training. Thus, region-specific adaptations should be closely monitored in future studies to determine whether this is a repeatable finding. Regardless, the data indicate that there is little difference in the L_f response to isometric training at short and long muscle lengths in the quadriceps, which mitigates against absolute muscle length being a major factor influencing L_f adaptations. No data have been presented in other muscles in response to such training, so it is not yet known whether this is a common finding in human muscles.

Study Limitations

An important limitation of this research was that there was disagreement between the two methods of measuring neural adaptation in SL. The EMG/M_{max} results showed clear joint angle-specific changes in SL; however, VA% did not change significantly. As discussed previously, both EMG/M_{max} and VA% methods have limitations, although we believe in this instance that the limitations of VA% quantification may be more profound, and the data require cautious interpretation. The suggestion that neural changes underpinned angle-specific force change in SL in the present study is based on clear training angle-specific adaptations in VL that correlated with the increases in force in SL. However, it must be acknowledged that, first, the lack of change in VA% in SL and variable changes in EMG/M_{max} in LL (Fig. 3) indicate that the neural adaptations were not completely clear and more detailed examinations are required in future. Second, the relative sensitivity and validity of measuring neural adaptations at different muscle lengths with EMG/M_{max} as compared with VA% methodology requires further investigation.

Another limitation of this study is the reliance on correlational data. We found significant correlations between the individual change in muscle force and change in muscle size despite there being no statistical change in force in LL. It can be argued that the change in force did not reach significance because force did not change at a specific joint angle as in SL. However, significant relation between the force and muscle hypertrophy in LL is a novel and important finding of the present study, and as discussed, it is important to examine the interindividual variability in the response to training at long muscle lengths and its relation to hypertrophy in a larger sample size.

CONCLUSIONS

Distinct adaptive mechanisms were found to underpin muscle length (joint angle)-specific force production after training performed at short versus long quadriceps muscle lengths. After training at shorter muscle lengths, clear angle-specific adaptations were observed that were most clearly related to changes in agonist (but not antagonist) muscle activation, as indicated by the angle-specific increase in EMG/M_{max} ratio. Also, there was no hypertrophy observed and the change in L_f at the VL midregion was not correlated with the change in MVC force. However, after training at a longer muscle length, the individual change in MVC force was variable and without a significant angle-specific response, despite there being significant muscle hypertrophy. The individual changes in MVC force across knee angles seemed to be associated with the magnitude of hypertrophy in specific regions of the quadriceps, and this finding should be further tested in studies with a larger sample size. Nonetheless, there were no significant associations with muscle length-specific changes in agonist or antagonist muscle activation in the group that trained at long muscle lengths. Interestingly, fascicle length changes were similar between the groups and were not related to length-specific changes in force production. Thus, either the influence of fascicle length changes was small when compared with other adaptations or the changes in fascicle length were not reflective of changes in serial sarcomere number within the constituent fibers.

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