Influence of concentric and eccentric resistance training on architectural adaptation in human quadriceps muscles

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Blazevich AJ, Cannavan D, Coleman DR, Horne S. Influence of concentric and eccentric resistance training on architectural adaptation in human quadriceps muscles. J Appl Physiol 103: 1565–1575, 2007. First published August 23, 2007; doi:10.1152/japplphysiol.00578.2007.—Studies using animal models have been unable to determine the mechanical stimuli that most influence muscle architectural adaptation. We examined the influence of contraction mode on muscle architectural change in humans, while also describing the time course of its adaptation through training and detraining. Twenty-one men and women performed slow-speed (30°/s) concentric-only (Con) or eccentric-only (Ecc) isokinetic knee extensor training for 10 wk before completing a 3-mo detraining period. Fascicle length of the vastus lateralis (VL), measured by ultrasonography, increased similarly in both groups after 5 wk (ΔCon = +6.3 ± 3.0%, ΔEcc = +3.1 ± 1.6%, mean = +4.7 ± 1.7%; P < 0.05). No further increase was found at 10 wk, although a small increase (mean ~2.5%; not significant) was evident after detraining. Fascicle angle increased in both groups at 5 wk (ΔCon = +11.1 ± 4.0%, ΔEcc = +11.9 ± 5.4%, mean = 11.5 ± 2.2%; P < 0.05) and 10 wk (ΔCon = +13.3 ± 3.0%, ΔEcc = +21.4 ± 6.9%, mean = 17.9 ± 3.7%; P < 0.01) in VL only and remained above baseline after detraining (mean = 13.2%); smaller changes in vastus medialis did not reach significance. The similar increase in fascicle length observed between the training groups mitigates against contraction mode being the predominant stimulus. Our data are also strongly indicative of (1) a direct association between VL fascicle length and shifts in the torque-angle relationship through training and detraining and (2) changes in fascicle angle being driven by space constraints in the hypertrophying muscle. Thus muscle architectural adaptations occur rapidly in response to resistance training but are strongly influenced by factors other than contraction mode.

A MUSCLE’S FORCE GENERATING properties are strongly influenced by the architectural arrangement of its fascicles. Therefore, given that muscle architecture is highly plastic, understanding the mechanical stimuli that influence it is of primary importance for those invested in optimizing muscle function and movement performance. In particular, fiber, or fascicle, length has a profound effect on muscle excursion range, maximum shortening velocity (and thus muscle power), and the force-length relationship. Despite the functional importance of fascicle length, the most important mechanical signal required to stimulate its adaptation has not been identified.

Examination of the effects of a range of mechanical stimuli has typically been performed with animal models. Researchers have reported increases in fiber length in muscles purported to have performed chronic eccentric work (16, 36, 37), whereas decreases (16) or a lack of change (36) is shown in those that worked concentrically. Also, rightward shifts in the force-length relationship of muscle have been shown after periods of eccentric muscle training (37), which has been suggested to be attributable to an increase in serial sarcomere number. These results are strongly indicative of contraction mode being a primary stimulus for fiber or fascicle length change, and there is some acceptance of the proposition that eccentric training increases muscle fiber length. Nonetheless, methodological difficulties involved with clearly establishing the contraction mode of the measured muscles [chiefly the rat vastus intermedius (VI)] limit the strength of this evidence (see e.g., Ref. 16). Furthermore, increases in fiber length (or sarcomere number, its surrogate when assuming a fixed sarcomere length) have been reported in muscles that were exercised through large excursions (34), which is suggestive of muscle excursion range (or absolute length) being a primary stimulus. Alternatively, movement velocity has been implicated. In humans, vastus lateralis (VL) fascicle length has been shown to be highly predictive of 100-m time in well-trained sprint runners (35), and increases in fascicle length were reported both in a group of athletes who removed heavy resistance training and increased their high-speed training (9) and in a group of resistance trainers who performed the concentric phase of their lifts with maximum velocity (3). Contraction velocity has also been demonstrated to be an influencing factor in smooth muscle cell growth (18). Nonetheless, the influence of muscle contraction velocity has yet to be studied in animals, and the elucidation of the most significant stimulus for fascicle length adaptation has not been possible with the use of animal models. Thus the possibility remains that a range of mechanical stimuli influence fascicle length.

In humans, the measurement of fascicle length in vivo with ultrasonography has revealed marked lengthening in both younger (3, 53) and older (47) populations after prolonged periods of resistance training, and fascicle shortening has been reported after gastrocnemius recession in spastic diplegia patients (56). The reliable measurement of fascicle length in humans offers the opportunity to use human models, which are associated with few of the methodological limitations inherent in animal models. Importantly, contraction mode, muscle excursion, and contraction velocity during a movement can be easily regulated by using isokinetic dynamometry to systematically examine the influence of each parameter, and the yet to be described temporal response of fascicle length change can...
be examined since repeated measurements are possible with noninvasive methods. In lieu of the above, the primary purpose of the present study was to test the hypothesis that fascicle length changes are unique to the training contraction mode. The hypothesis would be confirmed if increases in fascicle length result from eccentric training but a decrease or no change results from concentric training. Given that the time course of fascicle length change has yet to be described in humans, we measured, for the first time, fascicle length changes through periods of training (10 wk) and subsequent detraining (3 mo).

While fascicle length impacts significantly on the length range and speed of active force production, a muscle’s fiber, or fascicle, angle is also of great functional importance. It affects force production by allowing a greater amount of contractile tissue to attach to a given area of tendon or aponeurosis, and by increasing the muscle’s architectural gearing ratio (AGR; ratio of longitudinal muscle strain to fascicle strain, $e/e_\ell$, during contraction) to allow fascicles the opportunity to produce forces at more optimum lengths and shortening speeds (5, 8, 28, 42, 61). Increases in fascicle angle have been reported after periods (14–16 wk) of heavy resistance training in humans (1, 30), although studies of shorter duration have been unable to agree on its temporal response (10, 53). It is also not clear how different muscles within a synergistic group respond to the same stimulus. With respect to the mechanisms that promote fascicle angle change, a common theory is that it varies from its “genetic set point” concomitantly with muscle size. The theory is given credence by the strong relationship between muscle thickness or cross-sectional area and fascicle angle (29, 30) and their concordant change with training (1, 30). Alternatively, alterations in AGR resulting from fascicle angle adaptations could influence force generation independently of hypertrophy, so mechanisms unrelated to hypertrophy should be considered. A second purpose of the present study, therefore, was to describe the time course of fascicle angle change in two synergist muscles [VL and vastus medialis (VM)] and to determine whether it is most strongly related to changes in muscle size rather than being an independent anatomic adaptation to mechanical loading.

**METHODS**

Subjects. Thirty-three recreationally active men and women volunteered for the study, with 12 men [age = 24.2 ± 5.7 yr (mean ± SD), height = 178.7 ± 5.9 cm, weight = 73.6 ± 6.2 kg] and 12 women (age = 21.3 ± 4.3 yr, height = 160.8 ± 4.3 cm, weight = 63.5 ± 10.3 kg) being assigned to one of two training groups and 4 men and 5 women being assigned to a nontraining control group. None of the subjects had a significant lower limb injury within the previous 5 yr, had inflammatory conditions or hypertension, had performed weight training, had manual lifting-oriented occupations, or performed vigorous exercise more than four times a week. All subjects gave written informed consent before the study, which was approved by the Human Research Ethics Committee within the School of Sport and Education at Brunel University and was conducted in accordance with the Declaration of Helsinki.

Study design. After the subjects were familiarized with the testing procedures, their concentric and eccentric strength was tested, as described below. Those allocated to the training groups were paired with respect to their sex and pretraining torque production (mean of peak concentric and eccentric torque measured at 30°/s) and then randomly allocated to either the concentric-only (Con) or the eccentric-only (Ecc) knee extension training group. The subjects were tested for strength, muscle size, and muscle architecture at weeks 0, 5, and 10 of the training period and again after 3 mo (14 wk) of detraining. Tests at 5 and 10 wk of training were conducted 4 (muscle architecture), 4–5 (strength testing), and 7 (muscle volume; week 10 only) days after the final training session of that training period; tests were performed at the same time of day as at week 0. Three subjects (2 men and 1 woman) withdrew from the training for reasons unrelated to the study; 10 and 11 subjects completed Con and Ecc training, respectively.

**Resistance training.** The subjects performed four (weeks 1–3), five (weeks 4–7), or six (weeks 8–10) sets of six maximal concentric (Con group) or eccentric (Ecc group) knee extension repetitions three times a week on an isokinetic dynamometer (Biodex system 3, Biodex Medical Systems); in the first three sessions the subjects exercised at 50%, 70%, and then 90% of their pretraining maximum to minimize muscle damage resulting from the unaccustomed heavy exercise. At least 1 day separated each session. The Con group trained by extending their knee “as fast and hard as possible,” although the dynamometer lever arm was set to move at 30°/s, from the maximum knee flexion angle allowed by the dynamometer-seat setup (≈100°) to maximum knee extension (≈10°) before returning their leg to the start position with a small knee flexion contraction. The Ecc group performed a small knee extension contraction (≈20 Nm) to move the leg to an extended (10–15°) position before maximally extending the knee to resist the subsequent downward movement of the lever arm of the dynamometer. The subjects were asked to “stop the dynamometer” by applying a maximum upward force, although the dynamometer lever arm continued to move at 30°/s throughout; the contractions were completed at a knee angle of ≈100°. The knee joint ranges of motion during the concentric and eccentric training were greater than normally encountered during walking (≈25°; Ref. 7), jogging (≈55°; Ref. 7), and countermovement jumping (≈80°; Ref. 11) but only slightly greater than stair climbing (≈88° for 25.5-cm stair; Ref. 4) and so can be considered as being a loading stimulus of a greater than normal range of motion. One minute of passive rest was allowed between sets for both training groups, and repetitions within a set were performed continuously. Knee angles were measured as the angle of a line joining the lateral epicondyle of the tibia to the lateral malleolus and a line joining the lateral condyle of the femur to the greater trochanter. The knee joint center was aligned to the center of rotation of the dynamometer, and the dynamometer lever arm was securely attached to the shank at ≈50 mm above the lateral malleolus. Straps were placed firmly across the chest and waist of the subjects to minimize extraneous movement. Individual positioning of the subject with respect to backrest inclination, seat height, and lever arm length was held constant between test sessions. Loud verbal encouragement was given for each repetition, and set-by-set feedback of results was provided to aid motivation. These results could be compared with personal best performances, which were known by the subjects. No strength training was performed during the 3-mo detraining period, although the subjects maintained their normal daily activities.

**Fascicle strain during training.** To determine the fascicle strain induced by the training contractions, maximum and minimum fascicle lengths were measured at midhigh in the VL in six subjects (3 men and 3 women) during concentric and eccentric contractions. An ultrasound transducer (45-mm linear array, 10 MHz; Megas GPX, Esaote, Italy) was fixed to a foam cast and secured over the midpoint of the VL by elastic strapping. Sufficient water-soluble gel was applied to the transducer to aid acoustic coupling and remove the need for dermal contact. The transducer was aligned in the direction of the muscle fascicles; appropriate orientation was achieved when muscle fascicles could be traced clearly across the ultrasound image. A series of 200 images (29-Hz sampling frequency) were taken during each of three maximal concentric and eccentric knee extension contractions for each subject. Sonographs containing images of the muscle fascicles at their shortest and longest lengths were used for analysis.
Muscle fascicle length was determined by the procedures described in Fascicle length measurement below. Strain was calculated as the percent change from the shortest to longest fascicle length.

**Strength testing.** After warm-up, maximum concentric and eccentric knee extension torque was measured at 30°/s with the setup described above. The subjects performed three consecutive contractions with 2 min of rest separating the concentric and eccentric tests. Torque and position data were calibrated and sampled at a 2,000-Hz analog-digital conversion rate and stored on a computer running Spike2 software (Cambridge Electronic Design, Cambridge, UK). The data were subsequently filtered with a digital fourth-order, zero-lag Butterworth filter with a 14-Hz cutoff frequency (62); peak torque produced during each set was taken as the criterion measure.

**Torque-angle relationship.** To assess changes in the knee extension torque-angle relationship after training, torque was recorded for each 10° of the range of motion for the best concentric and eccentric trials. Changes in both absolute and normalized torque (i.e., expressed relative to the peak torque recorded at any angle) were determined at each angle. This approach, similar to that used by Butterfield and Herzog (15) and Lynn et al. (37), was in place of a measurement of the change in angle of peak torque since the torque-angle curves generated by the subjects fluctuated slightly throughout the range of motion, and particularly during the flatter portion of the curve nearing peak torque. These small fluctuations substantially affected the reliability of the measure of angle of peak torque. The average shift in the torque-angle relationship for each subject was also calculated by averaging the normalized concentric and eccentric torque values at each angle and then calculating their change from the pretraining values; increases at 80 and 90° and decreases at 30, 40, 50, 60, and 70° were taken to contribute to positive torque-angle shifts, so the proportional change was calculated as:

\[
\sum (\text{CE}_{40,90° \text{ mid-detrain}} - \text{CE}_{80,90° \text{ pre}}) - \sum (\text{CE}_{30–70° \text{ mid-detrain}} - \text{CE}_{50–70° \text{ mid-detrain}})
\]

where \( \text{CE} \) is the average of the normalized concentric and eccentric torques at a specific movement speed and subscripts 30–90° refer to the joint angles at which the normalized values were calculated; the subscript pre indicates that the measure was taken at pretraining (i.e., 0 wk), whereas the subscript mid-detrain indicates that measures were taken at midtraining, posttraining, and detraining (i.e., 5 wk, 10 wk and at detraining). The average shift was then recorded for each subject and plotted (see Fig. 10 for example).

**Muscle architecture.** In vivo muscle architecture was examined with two-dimensional (2D), B-mode ultrasonography (Megas GPX, Esato) with a 45-mm linear array transducer (10 MHz). To obtain the images, subjects lay supine with their legs fully extended and their muscles relaxed. A water-soluble gel was applied to the transducer to aid acoustic coupling and remove the need to contact the skin; this eliminated deformations of the muscle that can occur when pressure is directly applied to the skin and underlying muscle. Scans were performed on the right leg with the transducer oriented parallel to the muscle fascicles and perpendicular to the skin. Thus the transducer’s orientation relative to the longitudinal axis of the thigh was different between subjects. Transducer alignment was considered appropriate when several fascicles could be easily delineated without interruption across the image. Sonographs were taken in the middle of the VL and the VM at 50% of the distance from the central palpable point of the greater trochanter to the lateral condyle of the femur. At week 0, scanning locations were mapped onto a malleable plastic sheet, along with other distinguishing landmarks (e.g., border of patella, freckles, scars, etc.) to ensure that repeated scans were taken from the same site. One difficulty with repeated scanning is that small changes in the orientation of the transducer between test occasions can result in significant variation in the muscle architecture recorded on the image. To minimize this, we compared on-screen images at the 5 wk, 10 wk, and detraining testing sessions to those obtained at week 0. Echoes from interspaces among the fascicles and heterogeneities in the subcutaneous adipose tissue are unique to the transducer orientation (shown in Fig. 1), and these heterogeneities were easily visualized after the training and detraining periods in >80% of the subjects. Thus the transducer could be oriented so that these markings were the same in both sets of images, which provided confidence that transducer orientation discrepancies were small and were unlikely to account for measured pre-/posttraining architectural differences. All measurements were carried out by the same experienced sonographer.

**Fascicle length measurement.** Fascicle length was measured only in the VL, as the complex architecture of the VM ensured that the reliability of length measurements was unacceptably low (8, 10). Fascicle length was defined as the length of the fascicular path between the superficial and deep aponeurosis. In most cases, the fascicles extended off the acquired image. The length of the missing portion was estimated by linear extrapolation. This was done by measuring the linear distance from the identifiable end of a fascicle to the intersection of a line drawn from the fascicle and a line drawn from the superficial aponeurosis (see, e.g., Refs. 38, 51). The error involved with this technique has been shown to be reasonably low (−2.3%) in contracted tibialis anterior (46), where fascicle curvature is significant. Given that fascicles in the relaxed VL are relatively straight (see Fig. 1), we estimate that our error would be somewhat smaller. Repeated measurements yielded a coefficient of variation (CV) of 1.7% (±1.4 mm) and were performed by a single researcher.

**Fascicle angle measurement.** Images were analyzed with publicly available imaging software (ImageJ 1.36b, National Institutes of Health; free to download from http://rsb.info.nih.gov/ij/). Landmarks corresponding to the muscle fascicles and aponeurosis were marked on the image; a slight increase in muscle thickness is apparent between 0 wk and 10 wk for this subject. Adipose and connective tissue marks as well as visible blood vessels are circled on the 10 wk image (right). Visibility of these markings is strongly dependent on the transducer angle and can be used to ensure repeatability of transducer placement across time points.

Fig. 1. Sonographs of vastus lateralis (VL) at 0 wk and 10 wk. Lines delineating the aponeurosis and visible fascicle shown on the 0 wk image (left) are used to measure fascicle length and angle (θ). Arrows on the edges of the image show the muscle thickness (MT; mean of distance between superficial and deep aponeurosis measured at each edge of the sonograph); a slight increase in muscle thickness is apparent between 0 wk and 10 wk for this subject. Adipose and connective tissue marks as well as visible blood vessels are circled on the 10 wk image (right). Visibility of these markings is strongly dependent on the transducer angle and can be used to ensure repeatability of transducer placement across time points.
digitized (Fig. 1). Two points on each fascicle were digitized, one ~3 mm from the deep aponeurosis and the second at 50% of the distance from the deep to superficial aponeurosis. This allowed accurate delineation of the fascicles without incorporating the slightly greater fascicle curvature that can occur at the insertion point of the fascicles on the deep aponeurosis (58). Repeated measurements yielded a CV of 1.6% (~0.32%).

**Muscle volume and anatomic and physiological cross-sectional area.** At weeks 0 and 10, axial T2-weighted MRI scans of the thigh were obtained at 16-mm intervals from the superior border of the patella to the greater trochanter [3.0 T Magnetom, Siemens, Berlin, Germany; repetition time (TR) 4,260 ms, echo time (TE) 95 ms, averages: 3, field of view 200 × 200, slice thickness 4 mm, slice separation 14 mm, center-to-center slice distance 16 mm] from the training subjects. Technical difficulties with the scanner at 10 wk precluded the acquisition of images from the control subjects with sufficient contrast for analysis, although a high stability in MRI-based cross-sectional area measurements has previously been demonstrated over a 10-wk period [sum of 7 levels of cross section: pre = 323.7 ± 52.8 (SD), post = 320.9 ± 53.0 cm²] in subjects who do not change their exercise patterns (25). For training subjects, the scans were taken with a standard body coil, with the subjects lying supine with the legs fully extended; example scans are shown in Fig. 2. From these scans, anatomic muscle cross-sectional area was measured for each slice by manually tracing the perimeter of each muscle with Scion Image for Windows (free to download at http://scioncorp.com/frames/fr_download_now.htm). Care was taken to exclude adipose and connective tissue incursions. In proximal images, clear delineation of the vastii muscles was not always possible because of a lack of observable intermuscular septum. In these cases, a line was drawn from the end of the observable septum to a landmark on the muscle’s perimeter where the septum had intersected in distal images. Each slice was measured three times, with the median cross-sectional area being taken as representative; repeated measurements were very reliable (CV_{VL} = 1.5%, CV_{VM} = 1.9%). Whole quadriceps cross-sectional area was calculated as the sum of each of the four muscle cross sections, and individual muscle and whole quadriceps volumes were calculated by summing the product of cross-sectional area and slice thickness of all images. For VL only, physiological cross-sectional area (PCSA) was also calculated with the equation:

$$PCSA = \frac{Vol}{FL \times \cos \theta}$$

where Vol is the muscle volume, FL is its fascicle length, and \( \theta \) is the fascicle angle.

Intense exercise performed within several days of the MRI scan can influence muscle volume via osmotically driven intracellular fluid shifts (38, 43); such shifts have been shown to result both from damage to the contractile apparatus as a result of intense exercise (23, 49, 54) and from activation-dependent myocellular metabolism (2, 31, 55). This shift can be easily quantified by examining changes in the image density of T2-weighted MRI images. Although subjects were asked not to exercise before the testing, we examined the image density (sum of gray scale values of all pixels in the region of interest divided by total number of pixels) of MRI images by tracing the whole quadriceps muscles on images taken at 50% of the muscle length (i.e., at the same point as muscle architecture measurements) to ensure that prior exercise and testing did not influence the cross-sectional area measures. One subject showed a 12.8% shift in image density and was omitted from further analysis; the average shift for the remaining subjects was ~0.49 ± 0.8% (SE), so fluid shifts are unlikely to have influenced the results.

**Muscle thickness.** In addition to the MRI measurements, muscle thickness was also measured at weeks 0, 5, and 10 and after the detraining period from the obtained 2D ultrasound images. Muscle thickness was defined as the mean of the distances between superficial and deep aponeurosis measured at the ends of each 45-mm wide sonograph. The method and its reliability have been reported previously (8, 10).

**Statistical analysis.** Analyses were performed with SPSS Version 13.0 (SPSS, Chicago, IL). Temporal changes in strength, muscle architecture, and torque-angle variables were examined simultaneously by multivariate analysis of variance with repeated measures. Significant main and interaction effects were further examined with paired (for resolving time-related changes) or independent (for resolving group-related differences) samples t-tests. Differences in the change in cross-sectional area measured by MRI at different muscle locations were tested with paired-samples t-tests. Greenhouse-Geisser adjustment was applied on occasions when the assumption of sphericity was violated (Mauchly’s test of sphericity, \( P < 0.05 \)). Unless otherwise stated, all parameters are reported as means ± SE. Statistical significance was set at \( P < 0.05 \), although the results of statistical tests associated with an \( \alpha \)-level of <0.1 are discussed.

**RESULTS**

**Strength changes.** Although there was no change in the control group (\( \Delta_{\text{Con}} = +0.1 \pm 2.3 \) Nm (0.5 ± 1.8%); \( \Delta_{\text{Ecc}} = +7.5 \pm 5.5 \) Nm (3.0 ± 3.2%)), peak torque recorded in concentric and eccentric contractions increased significantly after 5 and 10 wk of training in both training groups, as shown in Fig. 3. The increase in concentric torque was greater in the Con group (24.1 ± 4.2%) than in the Ecc group (16.4 ± 5.1%) (\( P < 0.05 \)), but there was no difference in the increase in
Fig. 3. Strength increases (both groups combined) after 10 wk of knee extension training and 14 wk of detraining. Both concentric and eccentric torque increased; the increase in concentric strength was greater in the concentric-only (Con) group than the eccentric-only (Ecc) group (P < 0.05; data not shown). Torque production decreased significantly in eccentric contractions after the detraining period (dashed line) only (P < 0.05); torque produced after detraining was significantly greater than at 0 wk (P < 0.01). Significant difference from 0 wk to 10 wk: **P < 0.01, ***P < 0.001.

eccentric strength between Con and Ecc groups (Δ_con = 35.9 ± 12.7%; Δ_ecc = 38.9 ± 14.2%).

After 3 mo of detraining, there was a nonsignificant decrease in concentric torque of 5.6 ± 2.7% (P = 0.084) from that recorded at 10 wk for the training subjects (pooled data) and a statistically significant decrease in eccentric torque of 11.4 ± 2.3% (P < 0.05). There was no difference between the training groups. After detraining, concentric and eccentric torque remained above pretraining levels [Δ_con = 13.8 ± 3.5% (P < 0.01); Δ_ecc = 17.7 ± 7.1% (P < 0.05)].

Torque-angle relationship. At 0 wk, greater torque was produced in eccentric, compared with concentric, actions by the training subjects (Figs. 3 and 4). Both peak concentric and eccentric torque were produced at a knee angle of ~70° (note: data were taken at each 10° of range of motion, so the optimum knee angle was not specifically determined; see METHODS), with less torque being produced at angles further from optimum. Both absolutely and proportionally, peak eccentric torques exceeded concentric torques at larger knee angles, although normalized torque at smaller angles was the same between contraction modes, so there was a small difference in the stimulus applied to the muscles of the subjects during training.

Despite this, there were no between-group differences in either absolute or normalized (to peak) torque-angle relations at any time point in either concentric or eccentric actions. Pooled analyses revealed both an increase in torque production, largely around the angle of peak torque (Fig. 4), and a shift in the normalized torque curve (Fig. 5), which occurred largely within the first 5 wk of training. There was a small and nonsignificant trend toward a reverse shift from 5 to 10 wk. The shift in the torque-angle curve was most prominent in eccentric contractions where greater absolute changes were found. Interestingly, there was a further shift in the torque-angle curve to more acute knee angles (i.e., longer muscle lengths) with detraining (see Fig. 6). These shifts were only statistically significant for eccentric contractions. There were no shifts in the torque-angle relationship for the control subjects.

Muscle size. Whole quadriceps volume increased from 2,342.9 ± 111.2 to 2,586 ± 114.8 cm³ (10.2%; P < 0.001) after 10 wk of training, with no difference between training groups. VL [825.6 ± 40.9 to 916.9 ± 43.2 cm³ (11.1%; P < 0.001) and VM [486.2 ± 26.8 to 558.3 ± 27.7 cm³ (14.8%); P < 0.001] volume also increased significantly. These increases in volume were not related to pretraining muscle volume. As a result of both volume and architectural changes, the PCSA of VL increased from 115.1 ± 5.3 to 124.2 ± 4.9 cm² (7.9%; P < 0.01).

Although the focus of this study was on VL and VM, we took the opportunity to examine region-specific changes in all of the quadriceps muscles by comparing anatomic cross-sectional changes measured at proximal (mean of the 3 slices nearest to 25% of the distance from the proximal end point of the muscle) to distal (75% from proximal end point of the muscle) locations. Whereas increases in cross section of VL and rectus femoris were relatively consistent along their lengths, as shown in Table 1, there was a trend (P = 0.08) toward a greater increase distally in VM (Δ_VMprox = 11.1 ± 0.4 cm², Δ_VMdist = +3.0 ± 0.9 cm²). There was also a trend toward a greater increase distally in VI when calculated as an absolute change (Δ_VIdist = 0.0 ± 0.5, Δ_VIdist = +2.2 ± 0.8 cm²; P = 0.055), and a significantly greater increase distally when calculated as a relative change (Δ_VIdist = +1.0 ± 2.1%, Δ_VMdist = +13.6 ± 4.8%; P < 0.05). While individual variation...
in the hypertrophic response precluded the greater distal increase in whole quadriceps cross-sectional area from reaching statistical significance ($\Delta_{\text{prox}} = 7.3\%$, $\Delta_{\text{dist}} = 12.6\%$), increases were greater in the distal compared with proximal region (3-fold) in 75% of subjects.

To more closely examine the temporal hypertrophic response, muscle thickness measures were also obtained. Whereas no change in muscle thickness was found for control subjects ($\Delta_{\text{VL}} = +0.56 \pm 0.96$ mm (2.6 ± 3.6%); $\Delta_{\text{VM}} = +0.33 \pm 0.57$ mm (2.5 ± 3.8%)), there was a statistically significant increase in muscle thickness of VL at 5 and 10 wk ($P < 0.05$) and of VM at 10 wk ($P < 0.05$) (Fig. 7) in the training subjects (both groups combined). Thus the temporal response of the muscles was not the same. The slight decrease in both muscles after detraining was not significant; however, the detraining values were also not different from values at 0 wk. There was no statistical difference in the changes in muscle thickness between Con and Ecc groups ($\Delta_{\text{Con}} = +25.4 \pm 12.6$ mm (11.8%); $\Delta_{\text{Ecc}} = +18.4 \pm 10.4$ mm (8.3%)).

Muscle architecture. The mechanical strain of VL fascicles measured at mid thigh in three men and three women before training was $105.1 \pm 9.0$% (62.4 ± 7.5 to 128.3 ± 15.5 mm) and $90.1 \pm 12.3$% (60.0 ± 4.4 to 114.5 ± 12.0 mm) for concentric and eccentric contractions, respectively. Thus there was a tendency for subjects to produce tension through a slightly longer fascicle length range during concentric contractions, which largely resulted from their VL fascicles being moderately longer (~14 mm) at maximum knee flexion. Relative to the overall length change, however, this difference was small and was not statistically significant ($P = 0.30$) for the six subjects tested.

There was no change in VL fascicle length in control subjects [$\Delta = -0.3 \pm 0.7$ mm (−0.3 ± 0.9%) over the 10-wk testing period. A pooled analysis of the training groups revealed a significant increase after 5 wk in the training subjects (4.7 ± 1.7%; $P < 0.05$), but no further change to 10 wk ($\Delta_{\text{pre-post}} = +4.2 \pm 1.4$%; see Fig. 8). However, there was no difference in the change in fascicle length between the training groups ($\Delta_{\text{Con}} = +6.3 \pm 3.0$% ($P = 0.039$); $\Delta_{\text{Ecc}} = +3.1 \pm 1.6$% ($P = 0.056$)). The small mean increase in fascicle length after detraining ($\Delta_{\text{post-detraining}} = +2.5 \pm 2.2$%) did not reach statistical significance and was not different between training groups. VM fascicle length was not estimated because its complex architecture rendered the estimates unreliable.

There was no change in VL or VM fascicle angle over 10 wk in the control subjects ($\Delta_{\text{VL}} = -0.24 \pm 0.52$° (0.4 ± 3.6%); $\Delta_{\text{VM}} = +0.40 \pm 0.31$° (2.5 ± 2.1%)). A pooled analysis revealed statistically significant VL fascicle angle increases of 11.5 ± 3.2% ($P < 0.05$) and 17.9 ± 3.7% ($P < 0.01$) after 5 and 10 wk, respectively (mean data are shown in Fig. 9). There was a nonsignificant reduction after detraining such that fascicle angle remained greater after detraining than at pretraining (13.2 ± 4.2%). Smaller changes in VM fascicle angle of 4.8 ± 2.5% and 8.2 ± 3.4% after 5 and 10 wk were more variable by comparison with VL and did not reach statistical significance. There were also no statistically significant training groups.
dependent differences in either VL or VM fascicle angle change over the training period. For VL, fascicle angle of the Ecc group increased by 21.4 ± 7.3% (P = 0.03), whereas changes in the Con group did not reach statistical significance (13.3 ± 3.0%; P = 0.06) after 10 wk. The smaller changes in VM fascicle angle for Con (7.0 ± 4.9%) and Ecc (10.2 ± 5.1%) groups over the same period were not significant.

**DISCUSSION**

We have examined the relative contribution of the training contraction mode to muscle architectural adaptations in human VL and VM. While there were no changes in the strength of a control group measured across a 10-wk nontraining period, the training induced significant increases in concentric and eccentric strength of both concentric-only training (Con) and eccentric-only training (Ecc) subjects. The knee extensor strength responses to high-resistance training reported in the literature are inconsistent, with a set of 16 “responders” (i.e., 75% of the sample) showing the greatest response. Given that different regions of the quadriceps muscles are architecturally disparate and the muscles would encounter varying stresses during contraction, it is not unexpected that hypertrophy would vary according to the training contraction mode specifically.
sarcomere number adaptations to 10 days of either downhill or uphill running training (it was not possible to measure VI length change). They confirmed that VL actively lengthens during downhill running and largely shortens during uphill running but found a similar decrease in sarcomere number in the VL of both training groups [downhill group = −4.0%, not significant (NS); uphill group = −5.3%, $P < 0.05$]. Interestingly, their Figures 1 and 2 (Ref. 16) showed that VL is active at short lengths during the period of EMG activity in both uphill and downhill running, which might explain the similar loss of sarcomeres in both training groups. In VI, sarcomere number increased in the downhill-trained rats and decreased in the uphill-trained rats, but there is no evidence (in their study or other studies) that VI operates similarly to VL. Thus some caution must be exercised in interpreting these data as evidence for contraction mode-related alterations in sarcomere number. In another study by the same research group (32), lengthening contractions of the tibialis anterior and extensor digitorum longus of rabbits did not result in significant increases in sarcomere number. To further confirm the finding that the training range of motion, rather than contraction mode or velocity, has the greater effect on fascicle length change, examination of fascicle length changes to training using a less than normal contraction length range is required. Furthermore, our data are unable to determine whether the maximum length achieved by the muscle or the total range of lengthening it experiences is the primary stimulus for range of motion-dependent fascicle lengthening, since the fascicle strains encountered during the training were not appreciably different.

**Stimulus for fascicle length adaptation.** A main purpose of the present research was to test the hypothesis that muscle contraction mode strongly influences the magnitude of fascicle length adaptation. Our finding that Con and Ecc groups showed similar increases (rather than opposite responses) is not supportive of it, so factors other than contraction mode must influence fascicle length. Using our study design, we were able to gain some insight into the possible factors underpinning fascicle length change. We were able to examine the possible influence of the training range of motion (or muscle excursion) by having the subjects train through a knee joint range of motion of 95–100°, which is substantially greater than that normally encountered during walking (~15°; Ref. 4), jogging (~55°; Ref. 7), and countermovement jumping (~80°; Ref. 11), although only slightly greater than stair climbing (~88° for 25.5-cm stair; Ref. 4), and so can be considered as being a loading stimulus of greater than normal range of motion. The finding of a similar increase in fascicle length between the groups is supportive of the training range of motion (or muscle excursion range) being the dominant stimulus for fascicle length adaptation. It is unlikely that shortening velocity had a strong influence because we would have expected that slow training velocities would cause a reduction (or no change) in fascicle length in both training groups, since muscle loading at higher shortening speeds has been associated with fascicle length increases in skeletal muscle (3, 9).

Our results might be somewhat surprising given the data suggestive of contraction mode being an important mediating factor for VI sarcomere addition when muscle loading in rats is provided by downhill running training (16, 36, 37). However, in those studies the excursion range of VI was not explicitly confirmed. Butterfield et al. (16) analyzed the operating length range of the VL in rats by sonomicrometry before measuring sarcomere number adaptations to 10 days of either downhill or uphill running but found a similar decrease in sarcomere number in the VL of both training groups [downhill group = −4.0%, not significant (NS); uphill group = −5.3%, $P < 0.05$]. Interestingly, their Figures 1 and 2 (Ref. 16) showed that VL is active at short lengths during the period of EMG activity in both uphill and downhill running, which might explain the similar loss of sarcomeres in both training groups. In VI, sarcomere number increased in the downhill-trained rats and decreased in the uphill-trained rats, but there is no evidence (in their study or other studies) that VI operates similarly to VL. Thus some caution must be exercised in interpreting these data as evidence for contraction mode-related alterations in sarcomere number. In another study by the same research group (32), lengthening contractions of the tibialis anterior and extensor digitorum longus of rabbits did not result in significant increases in sarcomere number. To further confirm the finding that the training range of motion, rather than contraction mode or velocity, has the greater effect on fascicle length change, examination of fascicle length changes to training using a less than normal contraction length range is required. Furthermore, our data are unable to determine whether the maximum length achieved by the muscle or the total range of lengthening it experiences is the primary stimulus for range of motion-dependent fascicle lengthening, since the fascicle strains encountered during the training were not appreciably different.

**Time course of fascicle length adaptation.** We also sought to describe the temporal response of fascicle length change for the first time in humans. An important finding was that fascicle length adapted within the first weeks of training, during which period neural adaptations have traditionally been thought to dominate. Our results confirm the rapid adaptation in fascicle length reported by Seyennes et al. (53) and by others using animal models (16, 36, 37). We found increases of ~4.7% compared with increases of 9.9% reported by Seyennes et al. (53). The lesser increase found in the present study can probably be explained by our measurement of fascicle length with the knee fully extended, and the muscle shortened, compared with Seyennes et al. (53), who measured fascicle length with 80° of knee flexion. Also, their subjects trained three times a week, with testing being conducted at short intervals (10, 20, and 35 days). Thus training and testing were possibly separated by only a few days, and osmotic fluid shifts resulting from the training stress might have had some impact on the measured change. We took sonographs at least 4 days after the last training session to minimize the impact of this phenomenon, yet our results confirm the rapid changes they reported, although ours were smaller in magnitude. We have shown that these adaptations do not continue past ~5 wk in humans, and they therefore have a different temporal response to the fascicle angle adaptations (see below). The implications of this are that only relatively short periods of training are required to induce the necessary adaptation, so alterations in muscle function imposed by the fascicle length changes should also occur rapidly.

One such functional alteration associated with fascicle length change is the shift in the knee extension torque-angle relationship. Significant shifts toward longer muscle lengths (i.e., rightward shift) have been shown previously after periods...
of knee extension training (17, 47, 50), although in one study an increase in tendon stiffness after training in older subjects was suggested to have prevented a shift in the torque-angle relationship despite there being a clear shift in the muscle length-force relation (47). The idea that architectural adaptations might underpin changes in torque-angle relationships has gained momentum since there has been a lack of association of neural and torque-angle adaptations, with some authors showing a concomitant change (57) and others not (47, 59, 60). Other authors have also examined relationships between voluntary and electrically elicited contractions and found little similarity (32). This has been taken as good evidence that mechanical, rather than neural, factors underpin the torque-angle relationship. Given that increases in fiber length resulting from the addition of sarcomeres are typically associated with the rightward shift of the torque-angle curve in animals (14, 15), we expected to see a relationship between fascicle length change and the torque-angle shift, which would therefore be the same in both training groups. As shown in Figs. 4 and 5, the torque-angle relationship shifted toward the right in the first 5 wk of training but did not shift (in fact, there was a small trend toward a negative shift) from 5 to 10 wk of training; these changes were not group dependent. Unexpectedly, we found a further small shift to the right in detraining (Fig. 6). The magnitude of increase (2.5%; NS) was more than half of the increase attributable to the training, although there was some interindividual variability in this response. The reasons for this increase are not clear, although evidence for an increase in fascicle length in VL after the removal of heavy resistance training has been previously documented (9); interestingly, the changes reported in that study also occurred within 5 wk. We take this as evidence that learning responses cannot be solely responsible for the shifts because we would not then have seen a change in the detraining period. Regardless, the temporal response of the torque-angle curve matched the fascicle length response very closely, even to the point of them having a similar response to detraining (Fig. 10). The finding also of a constant ratio of adaptation, such that a 1% increase in fascicle length was associated with approximately a 1% shift in the normalized torque-angle relationship, can also be considered as strong evidence that fascicle length adaptations directly influence force-length relations of humans muscles, and that the range of motion of the training exercises is the strongest influencing factor for fascicle length adaptations. It also confirms the association between fascicle length changes and the force-length relation shown rarely in humans (e.g., Ref. 47) but consistently shown between sarcomere addition (i.e., fiber lengthening) and changes in force-length relation in animals (14, 15).

Fascicle angle adaptation. To test the hypothesis that fascicle angle adaptations would be strongly related to muscle size, we measured muscle thickness of both VL and VM during training and detraining and then compared its temporal response to that of fascicle angle. In both muscles, there was a tendency for muscle thickness and fascicle angle to increase with training and then decrease slightly with detraining. As shown in Fig. 11, the responses in VL, for which we obtained measures of the highest reliability, are almost identical. We found a constant ratio of adaptation such that a 1% increase in fascicle angle was associated with a ~0.5% increase in muscle thickness. These data are indicative of a strong relationship between muscle size and fascicle angle, as has been previously reported (29, 30). As expected, the changes in fascicle angle, which were measured in a single plane, and those of the 2D (cross-sectional area) and 3D (volume) measures of muscle size were not as well related. Our alternative hypothesis was that fascicle angle increases were a separate anatomic adaptation to the stress imposed on the muscle during training and might be unrelated to muscle hypertrophy. Our finding of a similar response in the Con group, who produced lesser torque in training, and the Ecc group, who produced greater torque in training, mitigates against this. The slightly greater increase in fascicle angle obtained by Ecc subjects was influenced by a strong “responder,” and therefore was associated with a greater SE. Therefore, we are not confident that the greater training load imposed on Ecc subjects can account for the small (and nonsignificant) between-group difference. Thus our data are more suggestive that fascicle angle adapts in response to muscle hypertrophy, and might therefore be driven by space constraints in the hypertrophying muscle.

The temporal response of fascicle angle to training has not been previously described. We found that in VL fascicle angle increased significantly (11.0%; Fig. 9) after 5 wk and continued to increase throughout the 10-wk training period (17.9%). Previously, Seyennes et al. (53) reported a significant (7.7%) increase in VL fascicle angle after 5 wk. The smaller magni-

![Fig. 10. Temporal patterns of normalized fascicle length (FL) and torque-angle relationship change (pooled data). Fascicle length change was calculated as a proportion of pretraining values. The shift in the torque-angle relationship was calculated by averaging the normalized concentric and eccentric torques at each angle for each subject and then calculating the change from pretraining values; increases at 80 and 90° and decreases at 30, 40, 50, 60, and 70° contributed to positive torque-angle relationship shifts. The average shift was then recorded for each subject and plotted. There was a similar temporal response of fascicle length and the torque-angle relationship through both training and detraining.](http://jap.physiology.org/)

![Fig. 11. Temporal patterns of normalized VL fascicle angle (FA) and muscle thickness (MT) change (pooled data). There was a similar response of fascicle angle and muscle thickness through both training and detraining.](http://jap.physiology.org/)
tude of increase shown in that study might have resulted from their testing of muscle architecture with the knee flexed to 80°, where fascicle angle would be smaller and changes possibly harder to detect. We have also shown for the first time that fascicle angle continues to increase as training progresses, at least to the 10th week, where our measurements were completed. Previous studies have reported significant increases after 14- and 16-wk training periods (1, 30), but the pattern of adaptation had not been examined. Together, our data and those of others strongly suggest that fascicle angle increases continually, at least for the first few months of training. Given that increases in fascicle angle allow a greater amount of contractile tissue to attach to the tendon and aponeurosis and that it promotes an increase in the AGR, which allows the rotating fibers the opportunity to work closer to their optimum (from both force-velocity and force-length perspectives), this increase in fascicle angle most probably contributes to the continuing increase in strength over the first months of resistance training.

Interestingly, changes in VM were typically of lesser magnitude (8.2%) and more variable. The VM has a complex architecture (8, 51) and is probably activated differently from VL, and its activation adaptation progresses differently with training (21, 22, 44, 45). Our data show that the adaptations in fascicle angle are not always consistent between the two muscles and that the response of VM has more interindividual variability. This is the first study examining changes in synergist muscles with training in humans, so it is not clear whether such intermuscular differences occur routinely. Nonetheless, we have also found that the fascicle angle adaptations in both VM (nonsignificant) and VL are largely retained after a 3-mo detraining period. To our knowledge, no studies have examined the response of fascicle angle to a period of detraining after a previous program of training in humans or other animals. The lack of significant decrease in fascicle angle in detraining has important consequences for the retention of strength after detraining (note that knee extension torque did not return to baseline after the detraining period, either) and is suggestive of the possibility that advantageous adaptations to training can be largely retained after lengthy periods of injury or illness in previously untrained subjects who complete a long-enough period of resistance exercise.

Given that the likely primary stimuli for fascicle length and angle changes are not the same, and the time courses of adaptation for fascicle length and angle are obviously different, it is clear that there are separate genetic and/or signaling bases underpinning fascicle angle and length changes. Recent advances in the muscle molecular and genetic sciences have revealed a complex system of mechanotransduction in skeletal muscle and a mesh of transcription and translation events leading to muscle hypertrophy [see reviews by Rennie et al. (48), Chin (19), and Burkholder (13)]. Muscle stretch, or simultaneous stretch and activation, is commonly used to study these processes. It is perhaps not surprising that the specific sequence of events leading to fiber hypertrophy vs. lengthening have not been elucidated since simultaneous stress and strain seem to result in both of these adaptations. Models used to explore the genetic and/or signaling phenomena that are unique to either muscle hypertrophy or fiber lengthening should perhaps incorporate a stimulus in which stress is applied at shorter vs. longer muscle lengths.

In summary, we have confirmed previous findings of rapid muscle architectural adaptation in humans and show for the first time that the temporal responses of fascicle angle and length are not the same: changes in fascicle angle continue relatively linearly for the first few months of training, whereas fascicle length adapts rapidly and does not continue past the first few weeks. We have also shown that adaptations in fascicle length are not influenced by contraction mode, but are most likely influenced by the training range of motion (i.e., fascicle strain); contraction velocity probably has little effect. In contrast, fascicle angle seems to be inextricably linked to muscle hypertrophy. Interestingly, 3 mo of detraining was not sufficient for muscle thickness, fascicle angle, or fascicle length to return to baseline levels, and in fact there was a small increase (not significant) in fascicle length in detraining. Interestingly, the changes in fascicle length were closely associated with shifts in the torque-angle curve, and because the increases were essentially the same in both training groups it is likely that the training range of motion was the most significant influencing factor. Whether the length range of force production or the absolute muscle length is the greater stimulus cannot be determined from our data; however, we can confirm that the genetic and/or signaling basis for fascicle length adaptations is different from that for muscle hypertrophy/fascicle angle adaptation in humans.

REFERENCES


