Short-term high- vs. low-velocity isokinetic lengthening training results in greater hypertrophy of the elbow flexors in young men

Tim N. Shepstone, Jason E. Tang, Stephane Dallaire, Mark D. Schuenke, Robert S. Staron, and Stuart M. Phillips. Short-term high- vs. low-velocity isokinetic lengthening training results in greater hypertrophy of the elbow flexors in young men. J Appl Physiol 98: 1768–1776, 2005. First published January 7, 2005; doi:10.1152/japplphysiol.01027.2004.—We performed two studies to determine the effect of a resistive training program comprised of fast vs. slow isokinetic lengthening contractions on muscle fiber hypertrophy. In study I, we investigated the effect of fast (3.66 rad/s; Fast) or slow (0.35 rad/s; Slow) isokinetic high-resistance muscle lengthening contractions on muscle fiber and whole muscle cross-sectional area (CSA) of the elbow flexors was investigated in young men. Twelve subjects (23.8 ± 2.4 yr; means ± SD) performed maximal resistive lengthening isokinetic exercise with both arms for 8 wk (3 days/wk), during which they trained one arm at a Fast velocity while the contralateral arm performed an equivalent number of contractions at a Slow velocity. Before (Pre) and after (Post) the training, percutaneous muscle biopsies were taken from the middlebelly of the biceps brachii and analyzed for fiber type and CSA. Type I muscle fiber size increased Pre to Post (P < 0.05) in both Fast and Slow arms. Type IIA and IIX muscle fiber CSA increased in both arms, but the increases were greater in the Fast- vs. the Slow-trained arm (P < 0.05). Elbow flexor CSA increased in Fast and Slow arms, with the increase in the Fast arm showing a trend toward being greater (P = 0.06). Maximum torque-generating capacity also increased to a greater degree (P < 0.05) in the Fast arm, regardless of testing velocity. In study II, we attempted to provide some explanation of the greater hypertrophy observed in study I by examining an indicator of protein remodeling (Z-line streaming), which we hypothesized would be greater in the Fast condition. Nine men (21.7 ± 2.4 yr) performed an acute bout (n = 30, 3 sets × 10 repetitions/set) of maximal lengthening contractions at Fast and Slow velocities used in the training study. Biopsies revealed that Fast lengthening contractions resulted in more (185 ± 17%) Z-band streaming per millimeter squared muscle vs. the Slow arm. In conclusion, training using Fast (3.66 rad/s) lengthening contractions leads to greater hypertrophy and strength gains than Slow (0.35 rad/s) lengthening contractions. The greater hypertrophy seen in the Fast-trained arm (study I) may be related to a greater amount of protein remodeling (Z-band streaming; study II).

HYPERTROPHY OF SKELETAL MUSCLE as a result of resistance training is due to a chronic summation of periods of positive net protein balance. The positive net protein balance arises due to the synergistic stimulatory effect of both resistive exercise and feeding on muscle protein synthesis (6, 7, 48). The increase in protein synthesis is directed toward remodeling and addition of cellular protein structures, in particular myofibrillar proteins, and is critical in the process of skeletal muscle hypertrophy. Most forms of resistive exercise also induce disruption of the protein ultrastructure, commonly observed as Z-line streaming and myofibrillar disorder, that is greater with lengthening vs. shortening contractions (17, 18).

Both lengthening and shortening contractions [terms used according to arguments outlined by Faulkner (14) but often referred to as eccentric and concentric contractions, respectively] induce what has been labeled as muscle protein ultrastructural damage, which is greater with lengthening contractions (17, 18). With repeated exposure to lengthening contractions, a combination of factors bring about a reduction in damage: the so-called “repeated-bout” effect (for review, see Ref. 8). Where hypertrophy is concerned, various studies have shown that resistive muscle training using isokinetic (21, 26) and isokinetic (13, 23, 24, 27, 40) training protocols in the absence of lengthening contractions results in less hypertrophy, and smaller strength gains, than a corresponding condition consisting solely of shortening contractions or combinations of shortening and lengthening contractions. However, greater hypertrophy with isokinetic lengthening-only training programs is not always observed (9, 28, 31). Higher velocity lengthening contractions have been shown to increase muscular strength to a greater extent than slow contractions (13, 36); however, mechanism(s) responsible for this phenomenon remains unknown. Using B-mode ultrasound, Farthing and Chilibeck (13) showed that muscle thickness (i.e., hypertrophy) was greater with lengthening as opposed to shortening contractions, and that there was a tendency (~5% greater) for greater hypertrophy in fast (3.14 rad/s) vs. slow (0.52 rad/s) trained arms. Using their mixed design, these authors were likely underpowered to detect the difference between fast- and slow-trained arms (13). We hypothesized that the tendency for greater hypertrophy seen previously with faster vs. slower lengthening contractions (13) would be due to greater protein remodeling induced by fast contractions (51, 52). Additionally, high-velocity lengthening contractions, due to the nature of muscle mechanics on the lengthening portion of the force-velocity curve, generate greater muscle forces, which may also stimulate a greater protein synthetic response resulting in greater muscle hypertrophy.

The purpose of the present study (study I) was to examine the early-phase adaptations in muscle fiber and whole muscle...
CSA of the elbow flexors, in young men after an isokinetic resistance training program with disparate lengthening contrac-
tile velocities [low velocity (Slow; 0.35 rad/s) and high velocity (Fast; 3.66 rad/s)]. We used naive untrained subjects to see a maximal response in terms of hypertrophy (2). Given the variability in the extent (32, 49) as well as time course (20) of resistance training-induced muscle hypertrophy and given that we only used a short-term study, and were trying to detect differences between two kinds of resistance training, we chose to use a within-subject design because this is a superior design compared with between-subject comparisons in terms of num-
ers of subjects needed to see a significant effect of the differing velocities. We also chose to use the biceps as the muscle for training because maximal lengthening actions during knee flexion would likely have resulted in subjects being able to exceed the maximal torque generating capacity of most dynamometers (405 N·m in our case), particularly after training. We hypothesized, in light of previous reports, that the Fast training would induce greater strength (12, 36) and hypertro-
phic (13) gains. The greater hypertrophy in the Fast trained arm would occur despite a substantially smaller torque-time inte-
gral vs. Slow training.

To examine the potential mechanisms underlying the differen-
tial effects of velocity on hypertrophy, we also conducted a separate study in which the acute changes in Z-line disruption were examined after Slow- and Fast-velocity lengthening con-
tractions. Biopsies were taken, from a separate group of sub-
jects from study I to examine the degree of Z-line disruption induced by a single acute bout of lengthening muscle contrac-
tions. Our hypothesis with this study was that Fast lengthening actions would induce greater Z-line disruption than Slow lengthening, which is indicative of protein remodeling, which may be a precursor to muscle hypertrophy (51, 52).

METHODS

Study I: Training Study

Subjects. Twelve healthy men (age 23.8 ± 3.4 yr, height 178.5 ±
9.6 cm, weight 82.5 ± 10.3 kg) who were recreationally active (i.e.,
no weight training) participated in the 8-wk training study. Subjects
were performing no more than 1–2 h/wk of structured physical
activity, and none was, or had been for the previous 6 mo, engaging
in resistance training. No subjects were taking protein or other
supplements or potential ergogenic aids of any kind. Subjects were
required to complete a routine medical screening questionnaire and
based on the questionnaire and physical examination were deemed
healthy. Subjects were advised of the purposes and risks associated
with the study, and they gave written, informed consent. The project
was approved by the Hamilton Health Sciences and McMaster Uni-
versity Research Ethics Board.

General Experimental Protocol

One week before the study start date, subjects attended a familiar-
ization session on the testing and training apparatus (Biodex-System
3, Biodex Medical Systems, Shirley, NY). On the study start date,subjects had a scan taken at the midpoint of their biceps brachii in
both arms by using high resolution peripheral quantitative computed
tomography (pQCT). Subjects also underwent a series of pretraining
(Pre) strength tests (see Strength measurements) on both arms inde-
pendently. Using this procedure, and given that the elbow flexors are
an easy muscle to recruit maximally with little to no training (33), it
is not surprising that we consistently observed interclass correlation
coefficients (ICC) of 0.95 and above for repeat testing of maximal
torque within (ICC = 0.97) and between (ICC = 0.95) testing
sessions. Five to seven days later, subjects reported back to the testing
center and muscle biopsy samples were taken from the belly of the biceps brachii muscle of each arm. Arms were then randomly assigned (counterbalanced for dominance based on strength) to be trained using either fast (Fast) or slow (Slow) lengthening contractions.

Subjects commenced training after 1 wk of rest. For the next 8 wk,
subjects reported to the testing center every Monday, Wednesday, and
Friday for exercise training. During the first week, one set (10 repeti-
tions/set) of maximal lengthening contractions was completed for
both Fast and Slow velocities, and every week subsequent an
additional set was added to a maximum of four, with 180 s of rest
between each set. For the remaining 4 wk, subjects continued to
complete four sets on every training day. All subjects completed
100% of the prescribed 24 training sessions.

After the 8-wk training protocol, subjects were again given 4 days
of rest before posttraining (Post) testing for maximal torque genera-
tion. Post muscle biopsies were then obtained from a position ~4–5
cm superior or inferior (randomized counterbalanced) to the Pre
biopsies.

Strength measurements. Strength tests were performed both Pre
and Post in a completely randomized order, with 4 min of rest in
between, at various contraction speeds (0.35, 1.05, 2.10, 3.14, and
3.66 rad/s) and types (lengthening, concentric, and isometric). Sub-
jects placed their elbow on a positioning pad so that the ulnar-humeral
joint was at the axis of rotation of the Biodex machine, and they could
comfortably grasp the lever arm handle, while their forearm was in a
supinated position. Restraining straps were placed so as to secure
the subject while seated in the dynamometer. Most importantly, a re-
straining strap was placed diagonally over the involved shoulder to
omit the involvement of any other muscle groups, other than the
elbow flexors, during performance of the maximal contractions.

Isometric torque. Subjects performed three repetitions of a maxi-
mum voluntary contraction at 1.05 rad (120°) of elbow flexion. Each
contraction was 5 s in duration with 30 s of rest between contrac-
tions. Maximum isometric torque was considered to be the highest peak
torque of the three contractions.

Shortening torque. Concentric torque was recorded as the highest
peak torque of three repetitions through 1.57 rad (90°) range of
motion. Every repetition began at 3.05 rad (175°) of elbow flexion and
concluded when the subject flexed his arm, and the Biodex lever arm,
with maximal force to 1.48 rad (85°) of elbow flexion. Concentric
 torque was evaluated at the five different velocities: 0.35, 1.05, 2.10,
3.14, and 3.66 rad/s.

Lengthening torque. Measurements of lengthening torque were
made at the five testing velocities in a similar manner as for concentric
torque. Lengthening torque was taken from the highest peak torque of three repetitions starting at 1.48 rad (85°) and concluding at 3.05 rad
(175°) of elbow flexion. Each lengthening contraction, completed at
five velocities (0.35, 1.05, 2.10, 3.14, and 3.66 rad/s), was completed
by the subject maximally resisting the lever arm, which was returning
to the resting position after the contraction.

pQCT scans. The midline of the belly of the biceps brachii was
determined by measuring halfway between midpoint of the antecubi-
tal and axilla areas. The arm was then inserted into a small cylinder
that functions as the measurement area of the pQCT densitometer
that excludes pixels that correspond to the density of intermuscular fat. In
pQCT scans.


to be trained using

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<1.33% (12 repeat scans). Interinvestigator variation in assessing muscle CSA was never >2.6%; hence, to minimize variability, all scans for one subject were analyzed by one investigator who was blinded to condition.

**Muscle biopsies.** Needle biopsy samples were obtained from each subject under local anesthesia (2% lidocaine) using manual suction. One biopsy was taken from the medial portion of the right and left biceps brachii Pre to establish a baseline measurement. Another biopsy sample was taken from each of the trained arms in the same manner Post. Samples were immediately dissected free of visible fat and connective tissue, and they were placed in optimum cutting temperature (OCT; Tissue Tech, Sakura Finetechical) embedding medium with the muscle fibers oriented perpendicular to the plane in which the muscle was to be cut. The samples were then quick frozen in isopentane cooled by liquid nitrogen, and then they were stored at –80°C until subsequent analysis.

**Histochemical analysis.** The frozen OCT-mounted muscle samples were serially cross-sectioned to 10 μm thick on a microtome cryostat (model HM500OM, MICROM International, Waldorf, Germany) for histochemical analysis. Myofibrillar adenosine triphosphatase (mATPase) histochemistry was performed using preincubation pH value 4.60 (50 mM potassium acetate and 17.5 mM calcium chloride) for 6.5 min to determine muscle fiber-type composition. Slides were then rinsed with distilled water and incubated in 3 mM ATP using an alkaline solution (75 mM glycine, 40.5 mM calcium chloride, 75 mM NaCl, and 67.5 mM NaOH, adjusted to pH 9.4) for 45 min at 37°C and agitated at regular intervals in a temperature-controlled incubator shaker. After the ATP incubation, a rinse with distilled water was done, and the samples were incubated in 1% CaCl2 for 3 min at room temperature. Slides were again rinsed with distilled water and incubated in 2% CoCl2 for 3 min at room temperature. Another rinse with distilled water and an incubation in 1% ammonium sulfide for 1 min at room temperature followed. Samples were rinsed with distilled water five times before being dehydrated by incubating for 2 min in each ethanol concentrations (70, 80, 90, 95, and 100%). Samples were then cleared using xylene. After the slides were dried, coverslips were mounted using Permunt (Fisher SP15) and allowed to dry overnight.

Sections were viewed under light microscope (Olympus BX-60, Olympus America, Melville, NY), and images were digitized using a SPOT camera (model SP401-115, SPOT Diagnostic Instruments, Sterling Heights, MD) and visualized using SPOT software (V3.2.4 for Windows, SPOT Diagnostic Instruments). Images were analyzed by using both Image-J software (National Institute of Mental Health, Bethesda, MD) and Image Pro Plus (V4.0 for Windows, Media Cybernetics, Silver Spring, MD). The number of images taken at ×200 magnification of each sample was between five and seven and was largely dependent on the quality of the serial sections. Each image contained ~30–50 fibers. Three fibers (type I, IIa, and IIx) were consistently distinguished using the Image-J software by setting cutoff limits resulting in the creation of optical density “bins” according to the darkest (type I), lightest (type IIa), and intermediate (type IIx) fibers, as previously described (44). The classification of the fiber type is thus dependent on the intensity of the staining by the mATPase histochemical protocol. At pH 4.60, the light, intermediate, and dark fibers correspond to fiber type IIA, type IIX, and type I, respectively.

Sample images were converted to 8-bit, 256-gray-scale images, which linearly scales each pixel and assigns a value from between 0 (black) to 255 (white). By setting lower and upper threshold values, optical density bins were created that were as follows: 0–95 for dark areas, 100–175 for intermediate areas, 180–255 for light areas. Using these cutoffs, the three fiber types were more objectively classified. A similar procedure was employed using the Image Pro Plus software. In comparing the ability of Image J and Image Pro Plus to classify the same images (n = 26 sample sets Pre and Post), we obtained an ICC of 0.985 (P < 0.001), and we also found that the ICC for repeated image analysis was 1.0 for both programs.

Direct tracings using the Image Pro Plus software determined fiber CSA, which were expressed in micrometers squared. Fiber-type percent area (defined as total fiber area for one fiber type divided by summed total fiber area for all fiber types multiplied by 100), using the same cutoff thresholds and fiber-type distribution (number of one fiber type as a percentage of the total number of fibers examined) measurements were also calculated automatically using Image Pro.

**MHC protocol.** Mixed-muscle myosin heavy chain (MHC) analysis was carried out as described previously (16). Briefly, four to six serial sections from the OCT-embedded muscle sample were cut (20 μm) and placed into microfuge tubes containing 250 μL of lysing buffer (10% wt/vol glycerol, 5% vol/vol 2-mercaptoethanol, and 2.3% wt/vol SDS in 62.5 mM Tris pH 6.8) and were heated for 10 min at 60°C. Approximately 4–6 μL of the lysed muscle extract were loaded into a 20-cm × 20-cm 1.5-mm SDS-polyacrylamide gel, with Pre and Post samples adjacent to one another. The gel was poured and set in such a way that the top 25% of the gel was a 4% stacking gel, whereas the remaining 75% of the gel was a 4–8% acrylamide gradient. Samples were run overnight (19–21 h) at 120 V and subsequently stained with Coomasie blue. Three separate and distinct MHC isoforms (I, IIa, and IIx) were visually identified according to their masses (as compared with known standards). The gels were then scanned using a laser densitometer, the relative staining intensity (i.e., number of arbitrary densitometric units) of each band was calculated, and the intensity was expressed as a percentage of the total staining intensity (i.e., the summed arbitrary units of all three bands).

**Statistical analysis (study I).** Muscle fiber size, fiber type, pQCT, and MHC data were analyzed by using a two-factor repeated-measures ANOVA, with time (Pre vs. Post), and condition (Fast vs. Slow) as the factors. Strength data were analyzed by using a four-factor repeated-measures ANOVA, with time (Pre vs. Post), condition (Fast vs. Slow), contraction (eccentric vs. concentric), and velocity (5 levels: 0.35, 1.05, 2.10, 3.14, and 3.66 rad/s) as the factors. All analyses were performed using SPSS (version 11.5, Chicago, IL). Statistical significance for all analyses was accepted as P < 0.05. Significant main effects and interactions, where seen in the ANOVA, were further analyzed by using Tukey’s post hoc test. Values presented are means (SD).

**Study II: Acute Study**

**Subjects.** Nine men (age 23.2 ± 2.4 yr, height 181.9 ± 6.1 cm, weight 81.1 ± 5.6 kg) who were recreationally active (i.e., no weight training and no more than 1 structured exercise bout per week) participated in the acute study protocol. Subjects were required to complete a routine medical screening questionnaire and based on the questionnaire and physical examination were deemed healthy. Subjects were advised of the purposes and risks associated with the study, and they gave written, informed consent. The project was approved by the Hamilton Health Sciences and McMaster University Research Ethics Board.

**Experimental protocol.** For 1 wk before reporting to the testing center, subjects were instructed to refrain from any form of strenuous upper body activity. On the first study day, subjects had a muscle biopsy removed from the belly of biceps brachii of both right and left arms. Subjects also received instruction on the exercise protocol and use of the Biodex apparatus used for testing. For the week after the first biopsy, subjects were again asked to refrain from strenuous upper body activity. The next day, subjects returned to the Ivor Wynne Centre to repeat the contraction protocol and subsequently had the second biopsy sample from each arm taken. Subjects again returned to repeat the contraction protocol 24 and 72 h after the second biopsy session.

**Exercise protocol.** The exercise protocol was completed on the Biodex dynamometer. Subjects placed their elbow on the Biodex positioning pad so that their forearm was in a supinated position, the ulnar-humeral joint was at the axis of rotation of the Biodex lever arm,
that sections identified as having Z-band streaming were shown also to have disrupted Z-bands by using electron microscopy (5, 45, 46). We also confirmed, using electron microscopy (×3,500–5,000 magnification), in 30 randomly selected blocks (6 at rest, 12 at 24 h after the Fast exercise, and 12 others at 24 h after the Slow exercise) that Z-line streaming as identified using high-magnification light microscopy were in fact areas of disruption as estimated. Damage estimated by light microscopy and by electron microscopy were highly correlated (r = 0.98, P < 0.0001).

Statistical analysis (study II). Z-line streaming was analyzed as a paired t-test, because no disrupted areas were seen in the baseline biopsies; hence, values obtained were essentially a difference from baseline (i.e., only a single value for Slow and Fast). All analyses were performed using SPSS (version 11.5). Statistical significance for all analyses was accepted as P < 0.05. Values presented are means (SD).

RESULTS

Study I: Training Study

Strength. Training resulted in subjects being able to generate higher maximal torques during lengthening contractions, regardless of velocity, compared with shortening contractions (P < 0.05). Training also resulted in a main effect for training mode (Fast > Slow); hence, subjects were able to generate higher maximal torques after Fast training, regardless of velocity, compared with shortening contractions (P = 0.023). Fast training increased maximum torques across all velocities by an average of 11.3 ± 10.4 N·m, whereas Slow training only increased strength by 6.3 ± 12.0 N·m (P = 0.041; Fig. 1).

pQCT. Values for whole muscle CSA Pre and Post training are shown in Fig. 2. All changes are significantly different from zero (P < 0.01), with no significant difference between Fast and Slow (P = 0.060 for a time × condition interaction) and with a significant difference (P = 0.024) between Post Fast vs. Post Slow by post hoc test.

Muscle fiber CSA. Training resulted in a significant increase in the mean CSA of all fiber types (P = 0.016). Type I muscle fibers increased in size (P = 0.042; Fig. 3) by an average of 9.3 ± 5.0% (range Slow = 3–560 μm², Fast = 94–1,300 μm²) with no significant differences between Fast or Slow (P = 0.19). The change in fiber area after training for only the type

Fig. 1. Peak torque before (Pre) and after (Post) 8 wk (24 sessions) of Fast (3.66 rad/s) and Slow (0.35 rad/s) training (study I). A: Slow arm. B: Fast arm. Values are means (SD) for 12 subjects. *Significant main effect for time, P < 0.05. †Significant time × condition interaction (Fast > Slow) at all testing velocities Post, P < 0.05.

and they could comfortably grasp the lever arm handle. A restraining strap was placed diagonally over the involved shoulder to omit involvement from other muscle groups. Each subject completed three sets (10 repetitions/set) of maximal lengthening contractions through 1.57 rad (90°) of arm flexion with 180 s of rest between sets. One arm was exercised at the Fast velocity (3.66 rad/s) while the other was exercised at the Slow velocity (0.35 rad/s).

Muscle sampling. Needle biopsy samples were obtained from each subject under local anesthesia (2% lidocaine) using manual suction. One biopsy was taken from the medial portion of the right and left biceps brachii to establish a baseline measurement. Another biopsy was exercised at the Fast velocity (3.66 rad/s) and Slow (0.35 rad/s) training (3.66 rad/s) and Slow) at all testing velocities. Values are means (SD) for 12 subjects. *Significant main effect for time, P < 0.05. †Significant time × condition interaction (Fast > Slow) at all testing velocities Post, P < 0.05.

Light microscopy. After initial fixation, the tissue samples were postfixed in osmium tetroxide, dehydrated in graded baths of ethanol, and embedded in an epoxy resin (Spurr’s) with the fibers oriented longitudinally. Each block was then sectioned (0.5 μm) and stained with toluidine blue as described previously (5, 45, 46).

Individual fibers from each longitudinal muscle section were studied under ×1,000 magnification and examined for moderate (3–10 continuous and/or adjacent Z bands) and extreme (10 or more continuous and/or adjacent Z bands) Z-band streaming as described previously (5, 45, 46). Sample areas were calculated, and the amount of Z-band streaming was expressed per millimeter squared of muscle. All muscle sections were scored and viewed blind to the investigator as to the treatment (Fast or Slow) and subject. ICCs for repeated sample analysis with one investigator were consistently >0.96 on 10 separate samples. In addition, interinvestigator estimates of Z-line streaming using this method were also consistently >0.94, as reported previously (5, 45, 46). To reduce the variability, one investigator performed all of the analyses. Using this method, it has been shown

Fig. 2. Whole muscle peripheral computed tomography pQCT scan for muscle cross-sectional area (CSA) before and after 8 wk of Fast and Slow training. Values are means (SD) for 12 subjects. *Significant main effect for time, P < 0.05. Time × condition interaction, P = 0.06.
II fiber subtypes was greater in the Fast-trained (IIa: range 279–1,548 μm²; IIx range 963–1,730 μm²) vs. the Slow-trained arm (IIa: range 57–966 μm²; IIx range 205–1,124 μm²; all \( P < 0.007 \); Fig. 3).

Muscle fiber type. A significant decrease was observed in the percent distribution of type IIx fibers (\( P = 0.031 \); Table 1), regardless of training. Although statistical significance was not reached, a trend was also observed toward a decrease in the percent area that type IIx fibers occupy (\( P = 0.056 \); Table 1). There was, however, a significant difference in the percent area of type Ila fibers from Pre to Post but only in the Fast-trained arm (\( P = 0.05 \); Table 1). The percent area occupied by Ila fibers was also significantly greater in the Fast vs. the Slow arm (\( P = 0.041 \); Table 1).

MHC content. The percentage of MHC isoforms expressed showed a significant decrease in the percentage of type IIX isoform expressed in both Fast and Slow arms after training (\( P = 0.033 \); Table 1). Similarly, there was an increase in the percentage of type Ila isoforms present after training that was greater in the Fast vs. the Slow arm. The percentage of MHC isoforms expressed according to gel electrophoresis correlated well with the percent area for each fiber type as analyzed by mATPase histochemistry (\( r = 0.95, P = 0.0006 \); Fig. 4).

Study II: Acute Study

Z-line streaming. The extent of moderate Z-band disruption (expressed per mm² of muscle) seen in all muscle samples from the postexercise time point was greater (\( P < 0.001 \)) than baseline samples, in which no damage could be identified. Z-band disruption was greater (185 ± 17%) after the Fast exercise protocol than that seen after the Slow exercise protocol (\( P = 0.002 \); Fig. 5). The extent of extreme, compared with moderate, Z-band streaming (defined as encompassing >10 serially or longitudinally adjacent sarcomeres with damage) (17, 18) was not significantly different (\( P = 0.096 \)) between Fast- and Slow-exercised arms (Fast = 0.10 ± 0.15 and Slow = 0.04 ± 0.11 areas of extreme damage per mm² of muscle), but it was observed in six of nine Fast samples and only two of nine Slow samples.

Table 1. Percentage muscle fiber composition and muscle fiber area by histochemistry, as well as percentage myosin heavy chain from biopsies taken before and after training

<table>
<thead>
<tr>
<th></th>
<th>Type I (%)</th>
<th>Type Ila (%)</th>
<th>Type IIx (%)</th>
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<tbody>
<tr>
<td>Fast</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>46.9 (15.6)</td>
<td>46.0 (12.8)</td>
<td>9.1 (8.7)</td>
</tr>
<tr>
<td>Post</td>
<td>42.3 (10.0)</td>
<td>50.9 (9.0)</td>
<td>6.8 (6.6)*</td>
</tr>
<tr>
<td>Slow</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>45.2 (12.8)</td>
<td>43.5 (12.5)</td>
<td>11.3 (9.7)</td>
</tr>
<tr>
<td>Post</td>
<td>49.8 (3.3)</td>
<td>42.8 (10.4)</td>
<td>7.4 (5.9)*</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th></th>
<th>Fiber, %</th>
<th>Area, %</th>
<th>MHC, %</th>
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<tr>
<td>Fast</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Pre</td>
<td>43.3 (11.8)</td>
<td>58.2 (13.2)</td>
<td>8.5 (7.6)</td>
</tr>
<tr>
<td>Post</td>
<td>27.8 (7.6)</td>
<td>66.6 (9.0)*</td>
<td>5.6 (6.2)</td>
</tr>
<tr>
<td>Slow</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>32.5 (11.8)</td>
<td>58.1 (12.8)</td>
<td>9.3 (6.9)</td>
</tr>
<tr>
<td>Post</td>
<td>34.1 (8.7)</td>
<td>59.8 (8.7)</td>
<td>6.1 (4.8)</td>
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</table>

Values are means (SD) for 12 subjects. Fast, fast trained arm (3.66 rad/s); Slow, slow trained arm (0.35 rad/s); Pre, before training; Post, after training; MHC, myosin heavy chain. *Significantly different from Pre, \( P < 0.05 \). †Significantly different from Slow arm at the same time, \( P < 0.01 \).
shortening (3.14 and 0.52 rad/s) muscle training promoted a greater degree of hypertrophy. In addition, the difference in hypertrophy between the degree of hypertrophy induced by training with fast and slow (0.52 rad/s) lengthening conditions, whereas not statistically different, was 5% (13); their mixed-factor design likely lacked the statistical power to detect this difference. In our study, we attempted to address this shortcoming by using a completely within-subject design and by using two lengthening conditions that were more disparate in velocity: 10-fold vs. only a 6-fold used by Farthing and Chilibeck, thinking that a greater difference in velocity may play a role in emphasizing differences in protein remodeling and thus hypertrophy, according to our hypothesis.

Adams et al. (1) recently showed that training using contractions that were isometric, lengthening, or shortening resulted in an equivalent hypertrophy in rats. The torque integrals for these three training modes were obviously quite different from each other. Despite similar degrees of hypertrophy, increases in mRNA expression for mecano-growth factor and insulin-like growth factor I (IGF-I), two proteins that have been implicated as playing a role in hypertrophic muscle growth (19), were increased only in the isometric and shortening contraction conditions (1). In humans, a single bout of lengthening resistive exercise resulted in a greater increase in IGF-I mRNA expression vs. a bout of shortening exercise (4). We observed, despite a >10-fold lower mean torque-time integral (Fig. 6), a greater degree of hypertrophy (Fig. 3) with a training protocol that involved only high velocity lengthening contractions. However, why we observed greater hypertrophy with Fast as opposed to Slow lengthening contractions when Adams et al. (1) observed quite similar hypertrophy with different contraction types and vastly dissimilar force-time integrals is not readily apparent. The difference in the model utilized in our study as opposed to that of Adams et al. may explain some of the differences: rats vs. humans, imposed vs. voluntary contractions, and the number of training bouts were greater in our study (24 vs. only 10 used by Adams et al.).
Fast-trained arms. Of course, we have no direct measurements provides a basis for why we saw greater hypertrophy in the series of events outlined by Phillips (37), then this were to result in greater protein accretion over time, according to previous reports (24, 25, 27), however, strength gains are typically dependent on training modes. Hence, when training with lengthening-only muscle contractions, it is expected that lengthening strength would increase more then concentric strength. There is also the possibility of cross-education, that is, an effect of one training protocol impacting on the contralateral arm (10, 41, 47). However, when both limbs are training simultaneously, it is impossible to estimate how the strength gains in one limb would transfer to the other limb. In other studies we examined, strength was variably altered in an untrained limb (41) or remained unchanged (47); hence, in our design where both limbs are training, it appears that strength gains that transfer from one limb to other would be minimal, compared with the transfer of strength to a nontrained limb. We acknowledge that training mode may have been transferred to the contralateral limb, but evidence for this phenomenon in simultaneously training limbs indicates similar results to what we observed, that is an overall generally greater increase in strength with lengthening muscle actions with no specificity effect for velocity (12, 36). Additionally, because the gains in maximal torque were greater in the Fast-trained arm, then any cross-education in terms of strength gains would be to increase strength gains in the Slow-trained arm in which we observed “inferior” strength gains (Fig. 1). Hence, the fact that we saw a difference between the Fast and Slow arms in terms of strength gains is even more impressive and likely due to our use of a unilateral all-within-subject design.

Previously, our laboratory had labeled the ultrastructural observation of smeared or disrupted Z lines as muscle damage (5, 46), in accordance with others (15, 18, 39). Recently, it was reported that an acute bout of lengthening muscle actions, which would have resulted in severe muscle damage, resulted in no detectable disturbances of the myofiber protein structure (11). In fact, what we observed and labeled as Z-line streaming, and have previously called muscle damage (5, 46), has recently been reported not to be muscle damage but instead myofibrillar remodeling (11, 51, 52). This conclusion was based on an elegant and detailed immunohistochemical and electron microscopic examination of the protein composition of amorphous or smeared Z lines (11). These authors showed that the smeared Z lines from damaged muscle were quite dissimilar from their normal protein composition, containing instead higher than normal quantities of actin and desmin, which the authors concluded was an indication of fiber protein remodeling and not damage (51, 52). With this interpretation in mind (51, 52), then our data indicate that, in the Fast training condition, which as our acute study showed was accompanied by greater “Z-line streaming” (Fig. 5), there would have been greater fiber remodeling. Our laboratory has recently observed that lengthening contractions, as opposed to shortening contractions, are associated with a more rapid rise in myofibrillar protein synthesis (34). If a greater protein synthetic response were to result in greater protein accretion over time, according to the series of events outlined by Phillips (37), then this provides a basis for why we saw greater hypertrophy in the Fast-trained arms. Of course, we have no direct measurements of protein turnover, synthesis or breakdown, in this study, so this theory remains speculative.

Training increased muscle strength at all tested velocities, regardless of training mode; however, the overall gain in maximal torque generation was greater in the Fast arm at all velocities (i.e., a main effect of training mode; Fig. 1). This result is most likely at least in part a consequence of the greater hypertrophy seen within the Fast-trained arm (Fig. 3). According to previous reports (24, 25, 27), however, strength gains are typically dependent on training modes. Hence, when training with lengthening-only muscle contractions, it is expected that lengthening strength would increase more then concentric strength. There is also the possibility of cross-education, that is, an effect of one training protocol impacting on the contralateral arm (10, 41, 47). However, when both limbs are training simultaneously, it is impossible to estimate how the strength gains in one limb would transfer to the other limb. In other studies we examined, strength was variably altered in an untrained limb (41) or remained unchanged (47); hence, in our design where both limbs are training, it appears that strength gains that transfer from one limb to other would be minimal, compared with the transfer of strength to a nontrained limb. We acknowledge that training mode may have been transferred to the contralateral limb, but evidence for this phenomenon in simultaneously training limbs indicates similar results to what we observed, that is an overall generally greater increase in strength with lengthening muscle actions with no specificity effect for velocity (12, 36). Additionally, because the gains in maximal torque were greater in the Fast-trained arm, then any cross-education in terms of strength gains would be to increase strength gains in the Slow-trained arm in which we observed “inferior” strength gains (Fig. 1). Hence, the fact that we saw a difference between the Fast and Slow arms in terms of strength gains is even more impressive and likely due to our use of a unilateral all-within-subject design.

The overall gains seen in maximal torque generation were quite moderate, and hence differences would have been more difficult to detect were they present. We suspect that the moderate strength gains may have been due to some residual fatigue from the training itself, despite several days of rest after the subjects’ last training bout before strength assessment. In fact, Fig. 7 shows the peak torque values for training sessions (study I) averaged by week throughout the 8-wk training protocol. Values are means (SD) for 12 subjects. *Significantly different between Fast and Slow arms at the specified week, $P < 0.05$. $\dagger$ Different from week 1, $P < 0.05$. $\ddagger$ Different from weeks 1–7, $P < 0.05$. $\triangle$ Different from week 7, $P < 0.05$. **Fig. 7.** Mean peak torque values for training sessions (study I) averaged by week throughout the 8-wk training protocol. Values are means (SD) for 12 subjects. *Significantly different between Fast and Slow arms at the specified week, $P < 0.05$. $\dagger$ Different from week 1, $P < 0.05$. $\ddagger$ Different from weeks 1–7, $P < 0.05$. $\triangle$ Different from week 7, $P < 0.05$.
mean peak torque on a week-to-week basis throughout the course of the study and shows that, during training, the weekly peak torque was actually depressed, likely as a result of muscle protein remodeling (see Ref. 50 for a review), for some time before recovering and eventually exceeding pretraining values only in the last week. This pattern of depressed force and yet substantial fiber remodeling, ultimately resulting in hypertrophy, is similar to that seen with chronic low-frequency stimulation in animals. In this model of extreme overload, force is persistently depressed, and yet fiber adaptations still occur while substantial fiber remodeling is still present (22). In two other studies in which differing lengthening velocities were used for training (12, 36), the gains in maximal torque were not large. For example, Paddon-Jones et al. (36) found no significant gains in maximal torque generation in a slow-trained arm performing lengthening contractions at 0.52 rad/s and a 5- to 10-N m increase in the fast-trained (3.14 rad/s) arm. Farthing and Chilibeck (12) also reported the increments in maximal torque were in the range of 11–14 N m.

Muscle fiber-type transitions that occur with resistance training have usually demonstrated a shift from IIX to IIA fibers (25, 42, 43). However, a recent observation of fiber-type distribution after fast lengthening training showed an increase in the percentage of type IIX fibers and a decrease in type I fibers found within muscle samples (36); on completion of the present study, no such changes were observed. In fact, a decrease was seen in type IIX fibers after both Fast and Slow lengthening training according to both mATPase histochemical and MHC gel electrophoretic analysis. Although the hypothesized concomitant increase in IIX fiber distribution was not seen, we did observe a significant increase in the total area occupied by type IIA fibers but in the Fast-trained arm only. A shift from IIX to IIA fibers was to be expected according to Andersen et al. (3), who hypothesized that the type IIX isoform is the “default” MHC that is expressed frequently in untrained subjects. However, when a resistance training protocol is implemented, the MHC isoforms shift toward an increase in type IIA fiber percent (25, 42, 43). Type IIA fibers, although they may have a lower maximal torque-generating capacity (29), do have a greater oxidative capacity, which confers on them a greater resistance to fatigue. Moreover, the increase in fiber CSA of both the IIA and IIX fibers is more than compensates for any small shift away from IIX to IIA.

To place our findings in some context with other resistance training protocols, it is interesting to note that the degree of hypertrophy we observed compared with other studies in which the elbow flexors have been trained show similar increases in muscle CSA (13), greater increases in CSA and individual muscle fiber area with longer training protocols (35, 38), or no change in fiber size-trained subjects who superimposed elbow flexor training on top of their normal routines (2). The increases in muscle CSA and fiber area that our laboratory and others have observed (13, 35, 38) tend to be larger than that seen in muscles such as the vastus lateralis (42, 43). Hence, our findings must be viewed in the context of a short-term training protocol of elbow flexor training and could differ if different subjects (i.e., trained) or muscles (i.e., lower body locomotor muscles) had been studied.

We observed that higher velocity (3.66 rad/s) isokinetic lengthening contractions are associated with greater muscular hypertrophy than slower (0.35 rad/s) velocity lengthening contractions. This finding occurred despite a >10-fold greater time under tension in the Fast- vs. the Slow-trained arms. We observed that acutely an isolated set of Fast contractions resulted in greater disruption to the muscle ultrastructure, which could be indicative of greater protein remodeling and potentially faster onset (34) and potentially a greater duration of protein synthesis. Over time, the greater protein synthesis may result in greater hypertrophy.

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REFERENCES

VELOCITY OF LENGTHENING CONTRACTIONS AND HYPERTROPHY


