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Muscle morphological and strength adaptations to endurance versus resistance training.

Running head: Morphological changes following endurance or resistance training.

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Muscle morphological and strength adaptations to endurance versus resistance training.

ABSTRACT

Less is known about changes in muscle strength and morphology after endurance training than after resistance training. The purpose of this study was to compare changes in lower extremity muscle strength, muscle cross sectional area and fascicle angle (FA) following 10 weeks of either progressive endurance training (END, n = 7) or resistance training (RT, n = 7) in young untrained men. Functional performance measures included Vo_2-max, maximal voluntary contraction (MVC) and dynamic strength. Muscle morphological measures included assessment of vastus lateralis (VL) FA obtained by ultrasonography, anatomical cross-sectional area (ACSA) obtained by magnetic resonance imaging of the thigh and physiological cross-sectional area (PCSA) deduced from biopsy samples from VL. No changes in FA, ACSA and PCSA were observed for the END group after training. In the RT group, increases were observed for FA by 23±8% ($p < 0.01$), ACSA for knee extensor muscle by 9±3% ($p = 0.001$), ACSA for total thigh muscle by 11±3% ($p < 0.001$) and in mean PCSA by 19±7% ($p < 0.05$). A significant positive correlation was observed between FA and PCSA ($r = 0.39$, $p < 0.05$). In the RT group knee extensor MVC increased by 19±5% ($p < 0.001$) and dynamic strength for knee extensor muscle increased by approximately 23±1% ($p < 0.05$) at all velocities (30, 90 and 180°/s), whereas no changes were observed for the END group. In conclusion muscle morphological changes related to strength improvements are induced by long-term resistance training, but virtually non-evident after long-term endurance training.

INTRODUCTION

Several morphological parameters have been shown to influence skeletal muscle performance (16). Among these are tendon stiffness, muscle anatomical and physiological cross-sectional area and fascicle angle (16, 32, 39). In recent years, fascicle angle has been devoted specific interest (8). Fascicle angle (FA) is defined as the angle between the longitudinal directions of the muscle fascicles and the aponeurosis, respectively (Figure 1). FA influences muscle force generating capacity and is adaptable to chronic loading (2, 8, 33, 37, 39). Since the force transmission to the aponeurosis is proportional to $\cos(FA)$ (33) an increase in FA alone would penalize the transmitted force. However, the increase in FA allows for more contractile tissue contained within a given anatomical cross-sectional area (ACSA)(33) or volume (39). Alexander and Vernon (2) suggested that the physiological cross-sectional area (PCSA) increases in proportion to $\sin(FA)$. Combining the penalizing effect of increased FA with the benefits from the increased PCSA suggests that an increase in FA up to 45° ($\cos(FA) \cdot \sin(FA) = (1/2) \cdot \sin(2\,FA)$) would increase the total contractile force on the aponeurosis (2).

Several studies on resitance training, have verified the adaptability of FA, ACSA and PCSA of different skeletal muscles (10, 20, 37, 39). Accordingly, Aagaard et al. (39) found a relative change of 35% in FA, 16% in PCSA and 10% in ACSA of m. vastus lateralis (VL) following 14 weeks of resistance training in young untrained men. The authors suggested that the discrepancy between the increases in PCSA and ACSA could be explained by the concomitant increase in FA. Others have found similar increases in FA following resistance training in VL in elderly, postoperative patients (37) and in elbow extensors in healthy, young men (20). The abovementioned studies have focused on resistance training and adaptations in FA and found significant increases in maximal voluntary contractions (MVC) which confirms the relationship between force generation capacity, FA and PCSA.

To our knowledge no longitudinal studies have investigated chronic adaptations of endurance training on FA. One cross-sectional study have compared distance runners with sprinters and controls and found greater FA

in VL in long-distance runners (1). Interestingly, FA of sprinters was similar to controls and thus significantly lower than distance runners. Neither ACSA nor PCSA were measured, but no difference in muscle thickness was observed between controls and distance runners (muscle thickness expressed as distance between the deep and superficial aponeurosis). The sprinters had significantly thicker VL than distance runners or controls. The measurement of muscle thickness is rather difficult to compare to measurements of PCSA and therefore it could be argued, that it cannot be firmly concluded from one study if the distance runners actually have a higher PCSA than the controls. Furthermore, the cross-sectional design of this specific study does not allow one to conclude whether the observed differences in FA were caused by different training regimens, long term exposure to running or genetic factors. Characteristic for endurance running are the high peak forces during continuous stretch-shortening cycles corresponding to almost two times bodyweight (13) and 10-20 times bodyweight during sprinting in elite sprinters (23). It could be speculated that the need to produce high forces during running could induce adaptations in PCSA leading to increased FA as reported by Abe et al. (1) in a group of distance runners. However, since peak forces during sprinting are 5-10 times higher than during distance running, one would expect larger PCSA and thus larger FA in sprinters. A longitudinal study investigating the effect of endurance running on PCSA did not find changes in PCSA following 12 weeks of high intensity endurance running (25). In the study by Abe et al. (1) the participants had competed in endurance events for a minimum of seven years. Therefore it could be speculated whether the years of training exposure could induce changes in PCSA leading to increases in FA.

When comparing endurance running to endurance cycling one of the differences is the peak forces, which are approximately five times higher during endurance running compared to endurance cycling (13, 15). Considering the difference in the mechanical stress on the trained muscles these two endurance modalities may induce different muscle architectural adaptations. In concordance, earlier studies have found no changes in PCSA following 12 weeks of endurance cycling (7).

Another study examined the acute effect of cycling on FA (11). The protocol consisted of an incremental cycle test with two minutes intervals starting at 25W and increasing with 25W in each interval until

exhaustion. This produced a significant increase in FA in VL five minutes following exhaustion (11). The acute increase in FA was speculated to be attributed to edema and/or increased vascular perfusion in VL however no test was performed to detect whether this change was chronic. Chronic FA changes are believed to be a consequence of hypertrophy (8, 18, 39). Hypertrophy is commonly observed with long term resistance training (4, 39), but has not been observed following six weeks of sprint cycling (3), 12 weeks of endurance cycling (7) or 12 weeks of endurance running (24, 25). Thus, while endurance cycling might cause acute changes in FA, the lack of hypertrophy after several weeks of training suggests that this change is not chronic, but this has not been thoroughly investigated.

The purpose of this study was therefore to compare muscle strength and morphological adaptations of long term endurance training to long-term resistance training, with primary focus on potentially chronic adaptations in FA and PCSA.

We hypothesized that long term resistance training would induce increases in muscle strength, PCSA with related increases in FA, constituting the explanatory link between changes in ACSA and PCSA. Furthermore, we hypothesized that endurance training would produce smaller increases in muscle strength, but without concurrent increases in FA and CSA.

METHODS

Experimental Approach to the Problem

The study was designed to compare adaptations in muscle morphology and muscle mechanics between long term endurance (END) and resistance training (RT). Eighteen healthy young men who had not engaged in structured endurance or resistance training for at least six months, were included. The subjects performed a maximal oxygen uptake test on a cycle ergometer to ensure that the $\dot{V}_{O_2 max}$ values corresponded to values witin range of untrained healthy young men (< 50 ml/min/kg). Inclusion was based on a combination of this test-result and a questionnaire to ensure against to high an activity level. The subjects were randomly distributed into one of two training groups, including either 10 weeks of endurance training on a cycle ergometers or progressive resistance training for the lower extremity muscle groups. Maximal oxygen uptake and maximal muscle strength were assessed with an incremental maximal oxygen up-take test and an isokinetic dynamometer, respectively. Muscle morphology was measured using ultrasonography, whole-muscle MRI scanning and muscle fiber analysis through histochemical procedures.

Subjects

Eighteen untrained, young, healthy, male subjects volunteered to participate in the training study (bodymass, 78.3±3.4 kg, height 1.81±0.02 m, age, 23.4±0.8 years; [mean±SEM]). Subjects were excluded if they had engaged in structured resistance or endurance training within the last six months before inclusion. Other exclusion criteria comprised any history of musculoskeletal injuries and current prescription medicine intake. All participants were informed of the purpose and the risks of the study and gave written, informed consent to participate. The study was approved by the ethics committee of Region Midtjylland (j. no. M-20080177) and conducted in accordance with the Declaration of Helsinki.

Experimental Design and Training Program

The RT group completed a training program consisting of three leg exercises each performed as 3-5 sets of 4-10 repetitions with repetitions corresponding to RM loading (Table 1). The recovery time between sets was

set to two minutes during the first 15 training sessions, increasing to three minutes for the remaining 15 training sessions. During the last 15 sessions subjects were instructed to perform the concentric part of the exercises as fast as possible. Prior to training subjects performed a light warm-up on a rowing ergometer (Concept 2 Model D, Concept 2, Morrisville, VT, USA) followed by two sub-maximal warm-up sets in the incline leg-press.

….Please insert Table 1 approximately here….

All endurance training was performed on stationary bicycles (Kettler Ergoracer GT, Kettler, Ense-parsit, Germany). Based on the $\dot{V}_{O_2max} - test$, target watt and heart rate for each training session was calculated, to aid maintenance of intended training intensity and to further aid this, the subjects were wearing a heart rate monitor throughout each training session. Each session began with a five-minute warm-up on the bike followed by one of three different weekly exercise sessions. At the end of all sessions the subjects performed five to ten minutes of light cycling. Each first weekly session consisted of continuous cycling of 30 to 45 minutes at 60-75% of watt-max. Each second weekly session consisted of two intervals of 20 minutes at 70-80% of watt-max interspaced by five minutes light cycling. Each third weekly session consisted of eight 4 minutes intervals at 80-90% of watt-max interspaced by one minute light cycling (Table 2). Two of the short interval sessions were replaced with mid-way maximal oxygen uptake tests to allow adjustments of relative intensity according to gradual training improvements.

….Please insert Table 2 approximately here….

All training sessions were supervised to ensure proper progression for both groups and all training sessions were separated by at least one day.

Measurement Procedures

Maximal oxygen uptake test. The $\dot{V}_{O_2max}$ test was performed on all subjects pre and post training. To determine $\dot{V}_{O_2peak}$ during bicycling, all subjects performed an incremental exercise test on a Monark Ergomedic 834E bicycle ergometer (Monark ergometic 894 E, Monark, Varberg, Sweden). The test was conducted at 70 revolutions per minute (RPM) with 35 W increments every minute. Rate of oxygen uptake and carbon dioxide release were determined every 10 seconds by an online respiratory gas exchange analyzer (AMIS 2001, Innovision, Odense, Denmark). The protocol was a standardized, stepwise progressive test to exhaustion. $\dot{V}_{O_2max}$ was defined as the highest mean of three consecutive measurements of ten seconds intervals including the $\dot{V}_{O_2peak}$. Watt-max was estimated to principles of Andersen LB (5).The heart rate was monitored continuously with a heart rate monitor (Polar, Oulu, Finland).

Isokinetic measurements. Subsequent to a standardized warm-up consisting of 5 minutes light aerobic exercise (100 W) on a stationary bicycle (Monark, Varberg, Sweden), the subjects were seated in an isokinetic dynamometer (Humac Norm, CSMI, Stoughton, USA) with 90° hip flexion and restraining straps crossing the torso and right leg. The transverse axis of the subject's knee was aligned with the axis of the dynamometer. The right leg was placed behind a stabilization bar while the left leg was attached to the dynamometer arm. Subjects were instructed to hold on to the chair handles. The dynamometer was adjusted individually so the contact point between the subjects' leg and the dynamometer arm was 3 cm proximal to the malleolus medialis. When contraction type or angular velocity was changed a few trials were given to allow familiarization. The protocol consisted of three maximal concentric and eccentric knee extensor contractions at three different angular velocities: 30, 90 and 180°/s with standardized verbal encouragement given during the contractions. This was followed by three maximal isometric knee extensor contractions at 70° knee flexion (0° equals full extension) and three maximal isometric knee flexor contractions at 20° knee flexion. All contractions were interspaced with one minute recovery time. All trials were sampled at 100Hz. Peak torque from the best of the three trials was used for further analysis.

Whole-Muscle Cross-Sectional Area Analysis—Magnetic Resonance Imaging. All imaging was performed with a 1.5-T scanner (Philips Achieva, Best, the Netherlands). The subjects were placed in supine position with the feet entering the scanner first. The MRI scans were performed on the left leg using a cardiac coil. After an initial frontal scout scan, 20 transversal slices were acquired, of which only one was used for the present study. The first slice was 215 mm proximally to the fossa intercondylaris and the following images were acquired distally from this point. A T1-weighted, fast spin echo sequence with the following parameters was used: scan matrix = 288x282, field of view = 230x230 mm^2, number of slices = 20, slice thickness = 7.5 mm, slice gap = 1 mm, repetition time = 2 seconds, echo time = 5.3 milliseconds, echo train length = 18, number of signal averages = 3, and scan time = 3:12 minutes. Whole-muscle CSA was obtained from the most proximal slice at a position corresponding to one-half of the femur length. Whole-muscle CSA of three muscle compartments was calculated using custom made software. The first compartment was the knee extensors (mm. vastus lateralis, vastus medialis, vastus intermedius and rectus femoris), the second was the knee flexors (mm. semitendinosus, semimembranosus, and biceps femoris—caput longum and caput breve), and the third was the hip adductors (mm. adductor magnus, adductor longus and gracilis). Furthermore, the total thigh muscle CSA was calculated as the sum of the three compartments. Two investigators performed blinded area measurements on all images and intraclass correlation coefficients (ICC) were calculated. The final CSA for the three compartments used for later analysis was taken as the mean of the results from the two investigators (ICC = 0.95).

Muscle Fiber Analysis—ATPase Histochemistry. Biopsies were obtained from the middle section of the VL muscle by applying the Bergström needle technique. The muscle samples were dissected free of visible fat and connective tissue and a part of the biopsy was immediately mounted with Tissue-Tek, frozen in isopentane cooled with liquid nitrogen, and stored at -80°C until further analysis. Serial sections (10 μm) of the muscle biopsy samples were cut in a cryostat (-20°C), and cross-sections from pre- and post-training biopsies from the same subject were placed on the same slide and processed simultaneously for ATPase histochemistry. ATPase histochemistry analysis was performed after preincubation at pH of 4.37, 4.60, and 10.30 as described previously (12), to enable determination of fiber type specific CSA and fiber type

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distribution. The serial sections were visualized and analyzed as described in detail by Andersen and

Aagaard (4). The serial sections were visualized and analyzed using a Leica DM2000 microscope (Leica,

Stockholm, Sweden) and a Leica Hi-resolution Color DFC camera (Leica, Stockholm, Sweden) combined

with image-analysis software (Leica Qwin ver. 3, Leica, Stockholm, Sweden) as described by Dalgas et al.

(14). The investigator was blinded to pre/post samples and subject information.

Ultrasonography measurements. Sagittal ultrasonography (US) images of the VL muscle were recorded with

a Siemens real-time scanner with a 7.5-MHz linear array transducer. Images were obtained in a seated

position with 90° hip flexion and 70° knee flexion in the isokinetic dynamometer to ensure correct and

reproducible joint angles (representative image is depicted in Figure 1). The US probe was placed at 50% of

femur length over the midbelly of the VL muscle. Images were saved and later analyzed for FA by using

Scion Image (Scion Image, Scion Corporation, MD, USA). The VL FA was measured as the angle between

VL muscle fiber fascicles and the deep aponeurosis at the insertion (39). To avoid influence from other tests

the US test was performed on a separate day. All images were analyzed twice by two investigators to ensure

reliability. Mean values were used for further analysis.

….Please insert Figure 1 approximately here….

Statistical Analysis

After passing a test for normality and equal variance of distribution, data were expressed as mean ± SEM. A

two way repeated measures ANOVA was used to analyze time and/or group interactions and a Tukey post

hoc analysis, when a significant overall time or group effects was observed, was used to analyze for

individual, pair wise differences. Furthermore, a Pearson product moment correlation coefficient was used to

test the correlation between PCSA and FA. Significance was set at an alpha level < 0.05. All statistical

analysis where performed using SigmaPlot (SigmaPlot v 11.0, Systat Software Inc., Chicago, IL, USA).

RESULTS

Two exercise-unrelated dropouts occurred in each training group. Thus, all presented data are from the remaining 14 subjects (n=7 in each training group).

Baseline values

No difference between RT and END groups were observed prior to commencing training in any of the reported variables.

Changes in VL muscle fascicle angle

Results for FA are shown in Figure 2. In the RT group VL muscle FA increased from ~10±1° before training to ~13±1° after training ($p < 0.01$) corresponding to a relative increase of ~23±8%. No changes in FA were observed in the END group.

….Please insert Figure 2 approximately here….

Change in physiological CSA (PCSA)

Results for PCSA are shown in Figure 3. In the RT group overall fiber size increased from ~5207±266 μm^2 before training to ~6125±342 μm^2 after training ($p < 0.05$), corresponding to a relative increase of ~19±7%. No changes in PCSA were observed in the END group.

….Please insert Figure 3 approximately here….

Changes in anatomical CSA (ACSA)

Results for ACSA are shown in Figure 4. Total thigh ACSA in the RT group increased from ~137±5 cm^2 before training to ~153±5 cm^2 after training ($p < 0.001$), corresponding to a relative increase of ~11±3%. ACSA for the knee extensor compartment in the RT group increased from ~77±3 cm^2 before training to 84±2

cm^2 after training (p = 0.001), corresponding to a relative increase of ~9±3%. ACSA for the knee flexor compartment in the RT group increased from ~33±2 cm^2 before training to ~38±2 cm^2 after training (p < 0.001), corresponding to a relative increase of ~14±2%. No changes were observed for the adductor compartment in the RT group. In the END group no changes were observed in any of the muscle compartments and thus neither in total thigh ACSA.

….Please insert Figure 4 approximately here….

Correlation between FA and PCSA

A significant positive correlation (r = 0.39, p < 0.05) was found between the VL FA and VL PCSA.

Body-mass

The RT group increased their body-mass pre to post from 76.5±4 to 78.8±4 kg (p < 0.05), whereas no changes were observed for the END group (p = 0.633).

Changes in maximal oxygen uptake

The END group increased from ~45±2 ml/min/kg before training to ~50±2 ml/min/kg after training (p < 0.001), corresponding to a relative increase of ~10±2%. No changes were observed in the RT group. The values for the RT group pre and post were 46.9±3 ml/min/kg and 46.9±3 ml/min/kg (p = 0.958), respectively.

Changes in maximal muscle strength

Results for MVC and dynamic strength are shown in Figure 5. The RT group increased knee flexor MVC from ~151±14 Nm before training to ~176±7 Nm after training (p < 0.001) and knee extensor MVC from ~238±17 Nm before training to ~280±21 Nm after training (p < 0.001), corresponding to relative increases of ~28±6% and ~20±5%, respectively. Knee flexor strength in the RT group was significantly greater than in

the END group after training (p < 0.05). For the dynamic contractions the RT group increased torque at all angular velocities by an average of ~23±1 % for the knee extensors and ~22±2% for the knee flexors. No changes in torque at any angular velocity were observed in the END group. At all dynamic contractions the RT group was significantly stronger than the END group at post-training (p < 0.05).

….Please insert Figure 5 approximately here….

DISCUSSION

The main finding of the present study is that long term endurance training, unlike long term resistance training, is not a powerful inducer of chronic changes in muscle morphology. Accordingly, in the present study long term endurance training did not produce any changes in ACSA, PCSA and FA. This was in direct contrast to our observations on the RT group, where we found significant increases in FA (+23%), ACSA (+9%) and PCSA (+19%). These training-specific morphological adaptations are supported by the results from strength measurements in which the RT group increased strength at all conditions whereas no virtually changes were observed for the END group.

One primary focus of our investigation was on training-specific adaptability in FA. Though acute increases in FA may occur following exhaustive cycling (11), our results suggest that no chronic change in FA takes place following long term endurance cycling. Brancaccio et al. (11) suggested edema and/or perfusion as the reason for the acute increase in FA. To avoid any acute effect on FA the ultrasound measurements in our study were performed including several days interspacing these measurements from days including other test or exercise procedures.

VL FA increased in the RT group after ten weeks of training. Both the absolute values pre and post and the relative change correspond well with the literature (9, 37, 39). Absolute values for PCSA at pre-training are higher than observed by Aagaard et al. (39) but are in accordance with the studies by Staron et al. (36) and Kraemer et al. (25). However, the relative change of 19% for the PCSA in the RT group is also in accordance with Aagaard et al. (39). The absolute values before and after training for the ACSA as well as the relative improvement in the RT group are also in accordance with previous studies (29, 38, 39). For the RT group our data from PCSA (+19%), ACSA (+9%) and FA (+23%) confirms the previously suggested explanation for discrepancy between increases in PCSA and ACSA by Aagaard et al. (39). The results from the END group suggest that when no changes are observed in PCSA no changes are seen in FA.

Previously it has been suggested that a chronic increase in FA following resistance training may be caused by the increase in PCSA (8, 39). This hypothesis has been supported by studies reporting a significant correlation between muscle thickness/limb length and FA (18, 19). We found a significant positive correlation between PCSA and FA confirming these previous results. However, this correlation should still be interpreted with some caution, since the Pearson product moment assumes a linear relation between the variables, which in our case is based on an assumption. Also, a significant correlation does not necessarily reflect a causal relationship between PCSA and FA; although, results from the RT group indicate that they are strongly connected.

From a mechanical point of view, an increase in hypertrophy of the muscle increases the passive stiffness (26). This point of view is supported by the cross-sectional study by Ryan et al. (34) who reported significant correlations between the ACSA and passive stiffness of the plantar flexors. Furthermore, Klinge et al. (21) observed an ~15% increase in stiffness of the hamstrings after 13 weeks of isometric resistance training. If no change occurs in the aponeurosis or tendon stiffness, the increased passive stiffness of the muscle fibers could theoretically stretch the aponeurosis and/or tendon and increase the FA. Thus, it can be speculated that increased muscle stiffness caused by hypertrophy provides an explanation as a mechanism for changes in FA. On the other hand, tendon stiffness increases following resistance training (22, 32) can therefore be argued to counteract the increased muscle stiffness from hypertrophy and render it insufficient to increase FA. Structural proteins such as titin and desmin are strongly connected to passive stiffness (27, 35). An increases in desmin protein concentration following resistance training (but not endurance cycling) has previously been observed by Parcell et al. (31) could serve as an explanation to further increase the passive stiffness of the muscle tissue. We speculate that this could possibly overcome the increased stiffness of the tendinous tissue and ultimately increase the FA. To explore such a question, further studies are needed to investigate possible relations between FA and structural proteins such as desmin, titin or functionally related proteins.

With respect to changes in strength measures, the observed increases in isometric MVC in the RT group were as expected and similar to those reported in earlier studies (29, 39, 40). Unlike following resistance training, endurance training did not improve isometric MVC for knee extension and flexion. Likewise, endurance training induced no changes in the dynamic strength measurements. An earlier study by McCarthy et al. (28) with young untrained males showed a similar response to continuous endurance cycling. Contrary to this, another comparative study by Nelson et al. (30) found increases in dynamic knee extension at 180°/s following endurance training, which were similar to the increases following resistance training. In the latter study, no strength increases were observed at 30 and 60°/s in the endurance group, similar to our results. Furthermore, in the study by Nelson et al. (30) dynamic measurements were normalized to body-mass which seemed to decrease following training and therefore may have influenced the dynamic strength results. In the present study, the RT group improved strength during both eccentric and concentric contractions at all angular velocities. These results are in accordance with previous studies testing force-velocity in isokinetic dynamometers (6, 29) and during loaded squat jumps (17). When comparing increases in knee extensor MVC with increases in VL PCSA, Aagaard et al. (39) found an almost 1:1 relation between the parameters. Similarly, our results from the RT group exhibit a relative increase in PCSA of 19% and a relative increase in knee extensor MVC of 20%.

In conclusion, long term endurance training by cycling, was unable to produce changes in PCSA, ACSA and FA. This is in direct contrast to changes in and interdependence of muscle morphological parameters observed in response to long term resistance training.

PRACTICAL APPLICATION

The present data demonstrate the specific adaptations to various exercise forms. When conducting cycling-based endurance training one should not expect permanent muscle morphological changes, i.e. changes in fascicle angle or physiological cross-sectional area. The lack of changes in muscle morphology is further reflected in the lack of changes in both isometric and isokinetic measurements. On the contrary, when conducting high intensity resistance training adaptations in morphology and therefore also in isometric and isokinetic measurements takes places and seems to be highly interrelated. Depending on which qualities the practitioner wishes to improve, this knowledge can be useful to ensure effective training improvements.

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Figure legends

Figure 1: Representative sagital ultrasound image from the vastus lateralis muscle. Fascicle Angle, FA, is determined as the angle between the fascicles and the deep aponeurosis.

Figure 2: Fascicle Angle (FA). Values for m. vastus lateralis FA (°) ± SEM pre and post ten weeks of either endurance training (END) or resistance training (RT). The results from two-way ANOVA are shown in the upper right corner. * denotes significant difference pre versus post training for RT ($p < 0.01$).
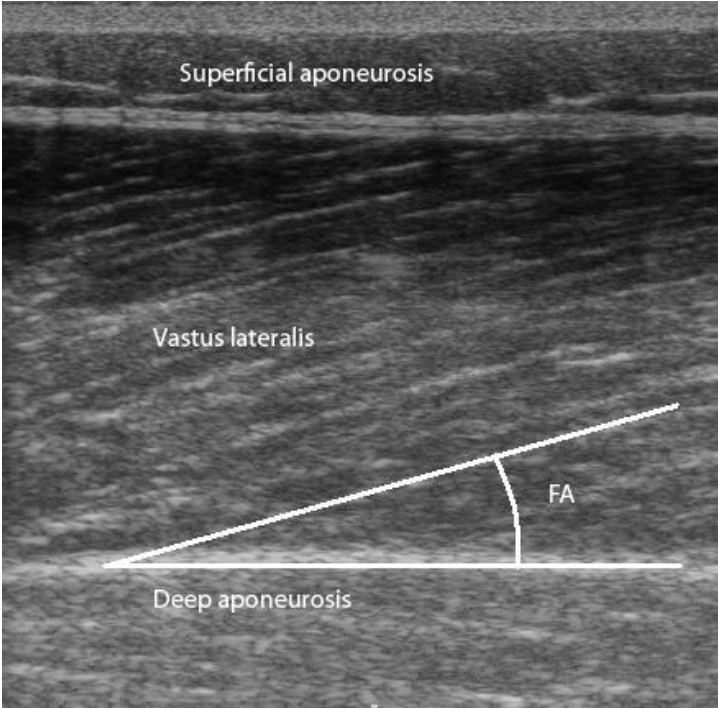
Figure 3: Physiological Cross-Sectional Area (PCSA). Values for m. vastus lateralis PCSA (μm^2) ± SEM pre and post ten weeks of either endurance training (END) or resistance training (RT). The results from two-way ANOVA are shown in the upper right corner. * denotes significant difference pre versus post training for RT ($p < 0.05$).

Figure 4: Anatomical Cross-Sectional Area (ACSA). Values for knee extensor and total thigh ACSA (cm^2) ± SEM pre and post ten weeks of either endurance training (END) or resistance training (RT). The results from two-way ANOVA are shown in the upper right corner. * denotes significant difference pre versus post training for RT ($p < 0.01$).

Figure 5: Quadriceps Torque-Angular velocity. Values for knee extensor torque (Nm) ± SEM pre and post of either endurance training (END) or resistance training (RT). Positive angular velocity equals concentric contractions, while negative angular velocity equals eccentric contractions. * denotes significant difference pre versus post training for RT ($p < 0.05$). # denotes significant group differences post training ($p < 0.05$).
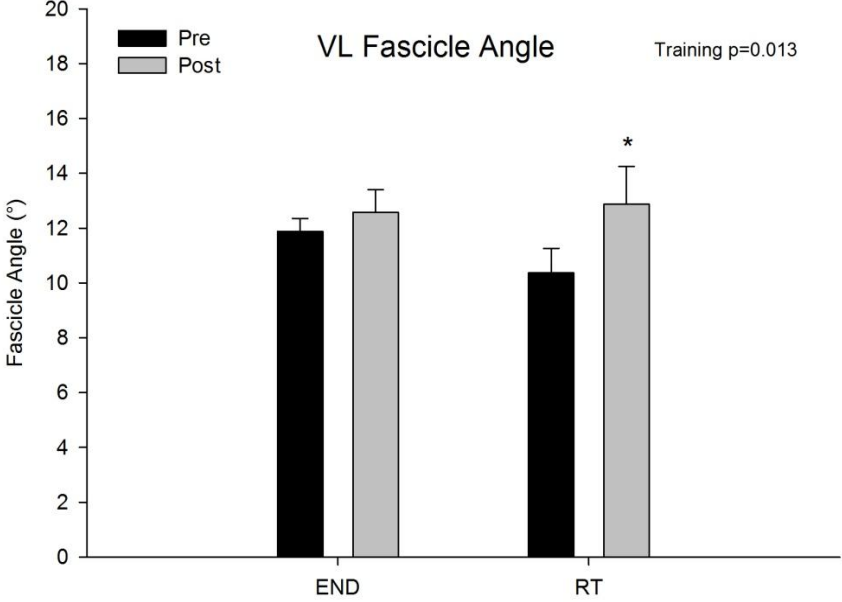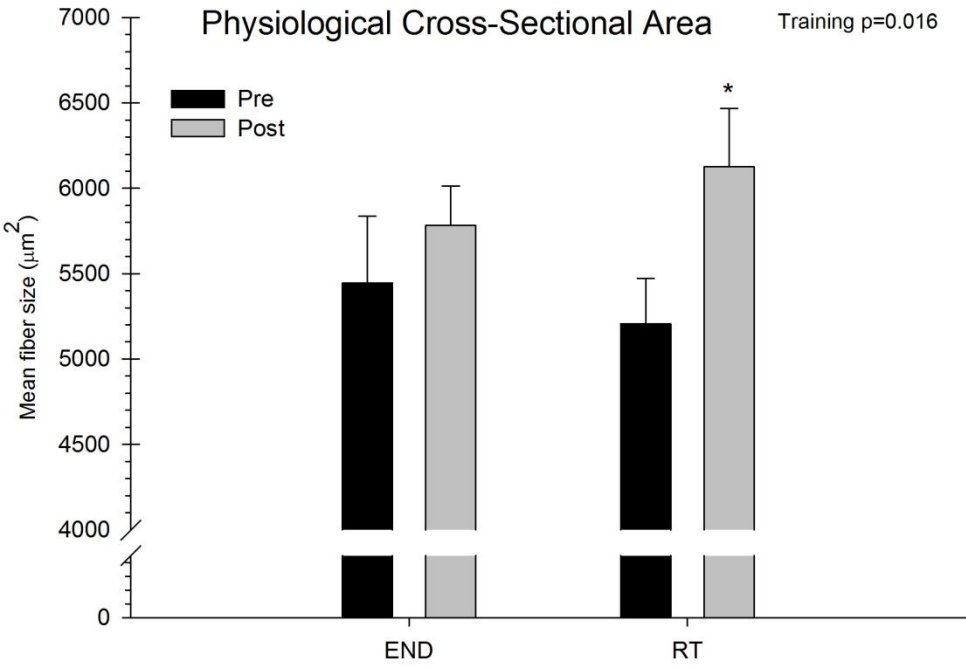
Figures

Figure 1

Figure 2

Figure 3

Physiological Cross-Sectional Area

Training p=0.016

Mean fiber size (μm^2)
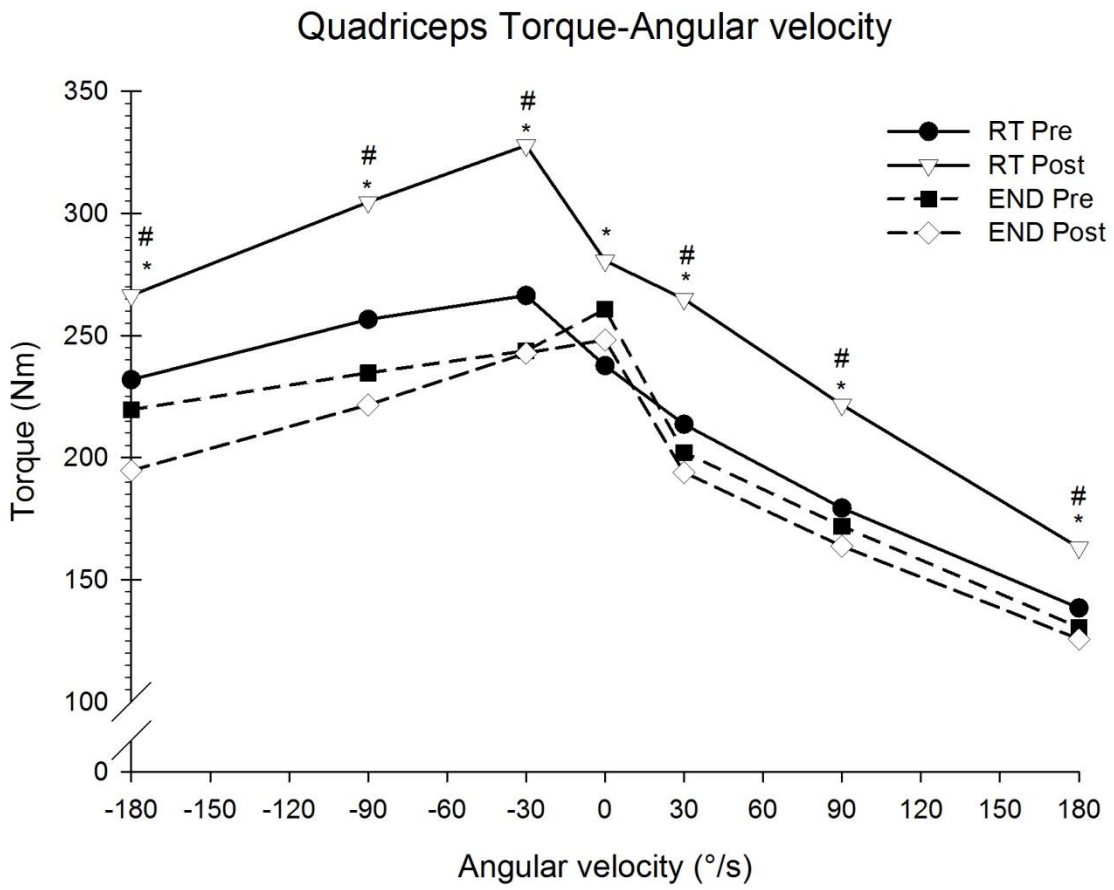
Pre
Post

END   RT

Figure 4

Anatomical Cross-Sectional Area

Figure 5

Quadriceps Torque-Angular velocity
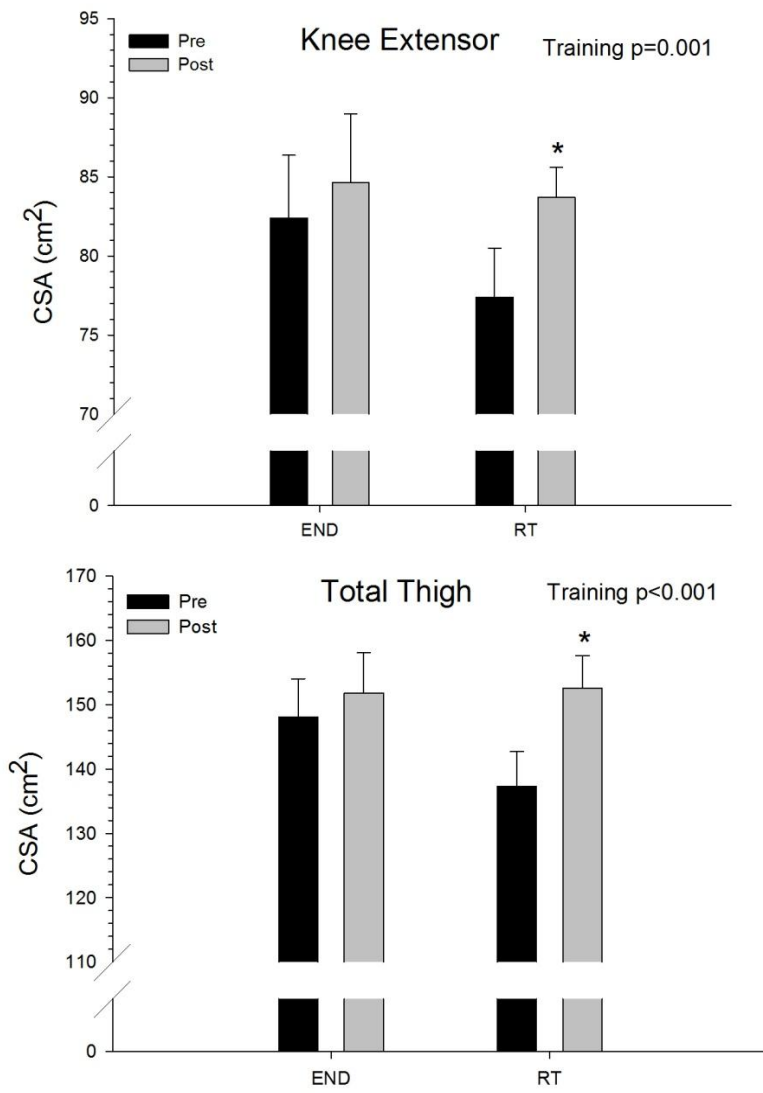
Tables

Session	1-4	5-10	11-15	16-20	21-25	26-29
Leg-press						
Sets	4	4	5	5	5	5
Reps	10,10,10,10	10,10,10,8	8,8,8,6,6	8,8,6,6,6	8,6,6,4,4	6,6,4,4,4
Knee extension						
Sets	5	5	5	5	5	5
Reps	10,10,10,8,8	10,8,8,8,6	8,8,8,6,6	8,8,6,6,4	8,6,6,4,4	6,6,4,4,4
Hamstring curl						
Sets	4	4	5	5	5	5
Reps	10,10,10,10	10,10,10,8	8,8,8,6,6	8,8,6,6,6	8,6,6,4,4	6,6,4,4,4

Table 1: Training protocol for resistance training. 2-3 minutes recovery was allowed between each set. Repetitions (reps) correspond to RM loading.

	Endurance group		
Week	**Session 1**	**Session 2**	**Session 3**
1	30-40min at 60%		2x20min at 70%
2	30-40min at 60%	2x20min at 60-70%	8x4min at 70-80%
3	45min at 60-65%	2x20min at 60-70%	*Maximal oxygen-uptake test*
4	45min at 65-70%	2x20min at 70-75%	8x4min at 80-85%
5	45min at 70%	2x20min at 70-75%	8x4min at 80-85%
6	45min at 75%	2x20min at 80-85%	*Maximal oxygen-uptake test*
7	45min at 70%	2x20min at 70-75%	8x4min at 80-85%
8	45min at 70%	2x20min at 75-80%	8x4min at 85-90%
9	45min at 70-75%	2x20min at 75-80%	8x4min at 85-90%
10	45min at 75%	2x20min at 80%	8x4min at 90%

Table 2: Repetitions x Duration of work at intensity of wattmax. Pause between 2x20min was 5min. and 1

min between 8x4min intervals.