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Time kinetics of acute changes in muscle architecture in response to resistance exercise

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Abstract

Objectives: The study aimed to assess acute changes in muscle architecture and its recovery after exhaustive exercise. We hypothesised that repetitive leg press exercise would decrease vastus lateralis fascicle length, while increasing both muscle thickness and pennation angles. By investigating the time kinetics of recovery of these parameters, we wished to gain insight into the mechanisms responsible for muscle architectural changes during exercise. *Design*: Muscle architecture was assessed in 41 male volunteers (25.2 ± 3.7 yrs; 1.78 ± 0.06 m; 76.4 ± 11.7 kg) before and directly after, as well as 5, 10, 15, and 30 min after induction of fatigue by leg press exercise. *Method*: Vastus lateralis muscle thickness, pennation angles and fascicle lengths were measured at rest by ultrasonography. Muscular fatigue was induced by an exhaustive series of maximum power, single leg press repetitions. *Results*: Following leg press exercise vastus lateralis muscle thickness and pennation angles were increased by approximately 7 and 10%, whereas fascicle lengths decreased by 2%. Different recovery times (muscle thickness: 30 min; pennation angles: 15 min; fascicle lengths: 5 min) were observed. *Conclusions*: The differential time courses of recovery suggest that changes in muscle thickness, pennation angles, and fascicle lengths are driven by different exercise-related stimuli. Increased muscle perfusion and tendon creep are likely candidates accounting for short-term changes in muscle architecture.

Keywords: Pennation angle; Fascicle length; Muscle thickness; Vastus lateralis; Leg press

1. Introduction

The arrangement of muscle fascicles with respect to the muscle's axis of force generation, generally referred to as muscle architecture, is one of the factors most strongly affecting a skeletal muscle's contractile properties.[1,2] Since the rise of modern imaging techniques, which allow for its non-invasive examination in vivo, muscle architecture has received increased attention, with an ever growing number of studies documenting the effects of chronically increased use, disuse or aging on fascicle arrangement.[3]

In spite of the increased interest in the functional relevance of fascicle geometry, few data exist on acute changes in muscle architecture. Several earlier works documented the immediate effects of different knee extension tasks,[4] cycling exercise,[5] or repetitive jumps[6] on the arrangement of muscle fascicles. The results reported in these studies, however, were solely descriptive in nature, incomplete, or inconsistent. For example, fascicle lengths (Lf) were reported to increase following an exhaustive series of jumps (soleus muscle),[6] but to decrease during a sustained isometric contraction (gastrocnemius medialis muscle),[7] and remained unaccounted for in other studies.[4,5] Differential reactions of muscle morphology to an acute bout of resistance exercise have also been observed in recent MRI-based studies. Here, muscle oedema, as reflected by increased signal intensities in T2-weighted images, were found to peak as early as 10 min after concentric knee extension exercise,[8] or to persist for several days after eccentric only knee extensions.[9]

The discrepancies in these findings indicate that acute changes in muscle morphology: (a) are specific to the muscle

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examined and the exercise performed and (b) may be triggered by distinct physiological mechanisms. Data on acute changes in muscle architecture might allow for insights into these mechanisms, deepen our understanding of the intramuscular processes involved in muscular fatigue, and provide a rationale for long-term training adaptations. To elucidate the muscular reactions to an exhaustive series of leg press repetitions, the present study therefore aimed to assess acute changes and the time kinetics of recovery of vastus lateralis (VL) muscle architecture. In agreement with previously published results,[10] we hypothesised that repetitive knee extension exercise would increase the quadriceps femoris muscle perfusion, resulting in augmented VL muscle thickness (MT). Furthermore, tendons and aponeuroses of the VL have been shown to exhibit creep in response to fatiguing knee extension protocols.[4] As increased tendon elasticity is known to allow for greater sarcomere shortening,[11] and fascicles pivot about their origin while shortening,[12] we further hypothesised that the exercise intervention would lead to a transient decrease in VL Lf and a concomitant increase in VL fascicle pennation angle (θ).

2. Methods

Forty-one male volunteers (age: 25.2 ± 3.7 yrs; height: 1.78 ± 0.06 m; mass: 76.4 ± 11.7 kg) were recruited via personal contact and advertisements on the website of the Viennese sports students association. All study members were free of internal or orthopaedic disease and gave written informed consent. The study was conducted in agreement with the declaration of Helsinki and approved by the ethics committee of the Medical University of Vienna.

All tests and measurements were performed on the dominant leg only, defined as the preferential kicking leg. This was the right leg in 39 participants (95%).

First, VL muscle architecture was assessed at rest (pre-exercise values) by real-time B-mode ultrasonography with a 5 cm, 7.5 MHz linear array ultrasound probe (AU5 Harmonic, Esaote Biomedica, Genoa, Italy). Then, following a standardised warm-up program consisting of a 6-min treadmill run (average velocity 8–10 km/h), all participants performed a fully exhaustive series of single leg press repetitions. To assess acute changes and the time course of recovery, follow-up measurements of muscle architecture were performed directly after (post-exercise values), as well as 5, 10, 15, and 30 min after the induction of muscular fatigue.

For measurements of muscle architecture, participants lay in a supine position on an examination bed with their knees in the anatomical position (0°). Vastus lateralis MT, Lf and θ were measured in the centre of the muscle belly, halfway between the lateral epicondyle of the knee and the greater trochanter, using sagittal-plane ultrasound scans. This site was clearly marked on the skin to provide a standardised measurement site and ensure that assessments at all measurement points were taken from the same external site. Muscle thickness, defined as the perpendicular distance between the superficial and deep aponeurosis, was measured in the proximal, mid, and distal region in each ultrasonic image and the average of the three values was used for further analyses. Pennation angles were measured as the angle of insertion of the muscle fascicles into the deep aponeurosis. Again, for a given ultrasonic image, θ was measured in three different locations and the average was calculated for further analyses. Fascicle length was defined as the length of the fascicular path between the deep and superficial aponeurosis. The VL fascicles were generally longer than the width of probe and therefore not entirely visible in the ultrasonic images. The visible portion of Lf was measured accounting for fascicle curvature and the remaining part was estimated by linear extrapolation. This approach assumes linearity of fascicles and aponeuroses in the non-visible regions and may therefore slightly underestimate the true Lf.[13] Furthermore, possible variation in ultrasound probe tilt or differences in the orientation of fascicles relative to the skin between measurements obtained pre- and post-exercise might have introduced bias.[14] Yet, the validity of this ultrasound technique for measurements of human muscle architecture is generally accepted.[15] To assess test-retest reliability, two ultrasound scans were recorded at each measurement point. Intraclass correlation coefficients ranged from 0.97 to 0.99 for MT, 0.92 to 0.99 for Lf, and 0.96 to 0.99 for θ. In addition, the typical errors (standard errors) of measurement were calculated using the equation $SD_{diff}/\sqrt{2}$, where SD_{diff} is the standard deviation of the difference scores between the two measurements. The typical errors ranged from 0.04 to 0.07 cm for MT, 0.06 to 0.13 cm for Lf and 0.41 to 0.79° for θ. All ultrasound images were analysed with publicly available imaging software (ImageJ 1.43b, NIH, Bethesda, MD, USA).

Leg press exercise was performed on a custom-made leg press device. The participants were seated on the leg press with the seat and backrest reclined at 45°. The foot of the tested leg was positioned at the centre of the footboard, which was hydraulically adjusted in height to set the knee angle at 90°. The second leg was slightly abducted, with the calf resting on a lateral foothold, to minimize possible transmission of force provided by the non-tested leg. Additionally, the participants were instructed to grab handles mounted on the sides of the seat to help stabilize the pelvis and further reduce erroneous movement. Following a familiarization trial consisting of 5 repetitions at 50% of the individual one-repetition maximum (1-RM), the study members were instructed to perform as many repetitions as possible with that load (lopt) that allowed the participants to generate the greatest power (Pmax). To determine lopt, the leg press load-power relationship was determined in each participant by use of a velocity sensor (FITRODyne Basic LCD, FiTRONIC s.r.o., Bratislava, Slovakia) attached to the weight stack at the backside of the leg press device. Both tests of load-power relationships and 1-RM, similarly preceded by a 6-min treadmill run, were performed on a separate occasion, following standard procedures.[16,17]

Table 1
Vastus lateralis muscle architecture before and after exhaustive leg press exercise.

	MT (cm)	θ (°)	Lf (cm)
Pre-exercise	2.5 ± 0.4	22.3 ± 3.6	6.7 ± 1.0
Post-exercise	2.6 ± 0.4*	24.6 ± 3.8*	6.6 ± 1.0*
5 min	2.6 ± 0.4*	23.6 ± 3.7*	6.7 ± 1.0
10 min	2.6 ± 0.4*	23.2 ± 3.8*	6.7 ± 1.0
15 min	2.5 ± 0.4*	22.8 ± 3.8	6.7 ± 1.0
30 min	2.5 ± 0.3	22.3 ± 3.7	6.7 ± 1.0

Note: Lf: fascicle length, MT: muscle thickness, θ: pennation angle.
* Values differ significantly ($p < 0.05$) from the corresponding measurement at rest (pre-exercise).

Differences between the measurements obtained at different times were tested with one-way repeated measures ANOVAs and paired sample *t*-tests were used for further analyses. Pearson's correlation coefficients were calculated for correlational analyses. The statistical level of significance was set at $p < 0.05$ and values are reported as means ± SD.

3. Results

The results achieved during leg press tests were as follows: Pmax: 398.8 ± 101.9 W; lopt: 91.4 ± 19.2 kg; 1-RM: 134.8 ± 34.5 kg. The measurement values of VL MT, θ, and Lf before and after the exhaustive leg press test series are shown in Table 1. Repeated measures ANOVAs revealed that all parameters of VL muscle architecture were significantly affected by the exhaustive leg press exercise stimulus; i.e. the post-exercise measurements differed statistically from the corresponding baseline values. Post hoc analyses of MT ($F(3.41, 136.45) = 46.03$; $p = 0.000$) showed that the measurement of MT at rest (pre-exercise) differed from the values obtained at all other time points, except from those measured after 30 min. This suggests that MT increased significantly in response to exhaustive leg press exercise, and normalised only after approximately 30 min. Values of θ ($F(2.84, 113.71) = 54.11$; $p = 0.000$), by contrast, remained significantly elevated for 10 min only, with the measurement results obtained after 15 and 30 min not differing significantly from the values obtained at rest. Lf ($F(3.42, 140.04) = 5.48$; $p = 0.001$) decreased from 6.7 cm (pre-exercise) to 6.6 cm (post-exercise). Follow-up measurements showed that Lf swiftly regained (normalization to pre-exercise values after 5 min) pre-exercise levels after cessation of the exercise. The time course of the relative changes of all parameters assessed is shown in Fig. 1.

The magnitude of exercise-related changes in MT and θ, calculated as the difference between post-exercise and pre-exercise value, was not correlated with the corresponding baseline values. However, the degree of fascicle shortening invoked by the exercise intervention was positively correlated ($r = 0.41$; $p = 0.039$) to the Lf baseline value. This suggests that the decrease in Lf provoked by resistance exercise is more distinct in those fascicles that are longer at rest.

4. Discussion

The purpose of the present study was to investigate instantaneous changes in muscle architecture and to document its recovery after an acute bout of resistance exercise. Our findings show that VL MT and θ increase by approximately 7 and 10%, whereas VL Lf decreases (−2%) in response to a fully exhaustive series of leg press exercises. In addition, the parameters of muscle architecture were found to recover at distinctive rates, suggesting that MT, θ, and Lf may be affected by differential physiological mechanisms, triggered by exhaustive resistance exercise.

To the best of our knowledge, only two studies[4,5] have previously reported acute adaptations in quadriceps muscle architecture following exhaustive exercise. Brancaccio et al.[5] assessed rectus femoris and vastus intermedius MT and VL θ prior and 12–15 min after the end of an incremental cycle ergometer test to exhaustion. After cessation of the exercise, the authors reported MT and VL θ to be increased by 8.7 and 12.5%, respectively. Similarly, Kubo et al.[4] reported VL MT and θ to be greater by 5 and 11%, respectively, after an exhaustive series of isometric knee extensions. These findings agree well with our results, as also in our study VL MT and θ were found to remain elevated well after cessation of the exercise.

Brancaccio et al.[5] attributed the increases in MT to increased vascular perfusion and to inflammatory reactions coming along with exercise-induced muscle damage. In fact, it is well known that the increased need for oxygen and energy sources triggers an increase in cardiac output and a redistribution of blood flow to the working musculature[18] and that the resulting increase in skeletal muscle perfusion occurs rapidly at the onset of exercise.[19] Increased vascular perfusion would presumably induce an increase in muscle volume and, consequently, in MT.

Early muscle swelling might also be caused by an inflammatory reaction. Indeed, some markers that reflect an acute inflammation, such as leukocytes, IL-6, and C-reactive protein, have been shown to be elevated immediately post-exercise.[20] Yet, serum creatine kinase levels reportedly increase at a much slower rate, to peak around the second day post-exercise,[20,21] coinciding with the delayed onset of muscle soreness.[22] Consistent with these observations, several MRI-based studies have recently shown that the peak in T2 relaxation times, which are indicative of muscle oedema that accompany the exercise-induced muscle damage, tends to occur about 24 h after cessation of eccentric exercise.[23] In our study, by contrast, MT was highest directly after the exercise intervention and started to decrease right after. Pre-exercise levels were reached within 30 min. Similar findings were also reported by Hayashi et al.[8] who found the increases in thigh circumference observed after repetitive isokinetic knee extension exercise to normalize within less than 40 min. These observations mitigate against the hypothesis that early increases in MT may be caused by intramuscular inflammatory processes. Rather, it seems likely that

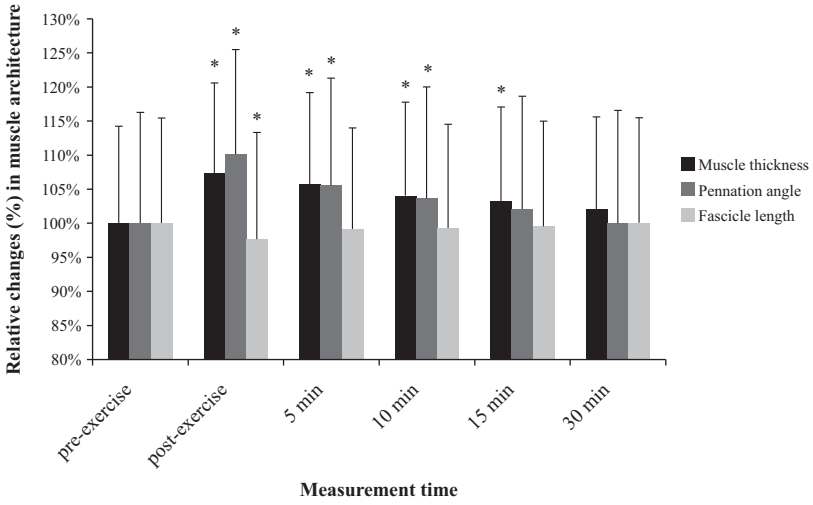

Fig. 1. Time course of changes in muscle architecture. *Note*: Lf: fascicle length, MT: muscle thickness, θ: pennation angle. * Values differ significantly ($p < 0.05$) from the corresponding measurement at rest (pre-exercise).

the changes in MT observed after exhaustive leg press exercise are mostly due to augmented muscle perfusion, whereas noticeable inflammation-induced muscle swelling presumably occurs at a later time. In favour of this hypothesis, an earlier work also described the exercise-induced swelling of arm flexor muscles as biphasic.[24] Future studies might test the correlation between exercise-induced increases in MT and blood supply using near-infrared spectroscopy, which allows for direct measurements of muscle perfusion.

Along with the changes in MT, we also observed significant increases in VL θ, which again well agrees with results previously reported.[4,5] Theoretically, the greater pennation angles measured post-exercise might reflect an elevated muscle tonic activity. Although conclusions about muscle tone from EMG data may be complicated, no increases in resting EMG activity were found in acutely fatigued muscles.[25] Therefore, it seems unlikely that increases in fascicle pennation would result from exercise-induced hypertonicity. A more probable explanation for the increase in VL θ involves changes in tendinous tissue compliance. As has been shown previously,[4,26] repeated muscular contractions may induce tendon creep. With increased compliance of tendons and aponeuroses, sarcomeres and, thus, fascicles will shorten more at any given level of muscle force.[11] As fascicles pivot about their origin during contraction, fascicle shortening is associated with increases in fascicle pennation angles.[12] Therefore, it seems likely that exercise-induced tendon creep would induce increases in VL θ. In support of this hypothesis, greater tendon elasticity has been shown to indeed coincide with greater pennation angles,[4,27] as measured at rest.

Confirming the proposed tendon creep and in agreement with the increased post-exercise value of VL θ, the VL Lf measured directly after the exercise intervention was significantly shorter than the corresponding baseline value. Given the decrease in Lf, albeit small in dimension, and the concomitant increase in MT, geometrical constraints might have further necessitated increases in VL θ. Interestingly, we found the exercise-induced fascicle shortening to be more distinct in those fascicles that are longer at rest. The reasons for this correlation between pre-exercise fascicle length and amount of fascicle shortening are unclear. Possibly, a given decrease in tendon stiffness would allow each sarcomere to shorten by a given percentage. As longer fascicles consist of a greater number of in-series sarcomeres, it may be speculated that the fascicle shortening caused by tendon creep would be greater in longer fascicles. During recovery, Lf swiftly regained pre-exercise levels, suggesting that the original stiffness of the tendinous tissue is restored rapidly after cessation of exercise. However, the differences in Lf observed between measurement points were throughout small and we acknowledge that bias might have been introduced as fascicles were not entirely visible in the ultrasound scans so that parts of the fascicular path had to be estimated by linear extrapolation. Changes in fascicle curvature could therefore not be fully accounted for.

5. Conclusion

Vastus lateralis muscle thickness and pennation angles are significantly elevated following an exhaustive series of single leg press repetitions, whereas fascicle lengths decreased in response to the exercise intervention. Here, the amount of exercise-induced fascicle shortening was weakly positively correlated to the fascicle length baseline value, suggesting that longer fascicles shorten relatively more in response to exhausting leg press exercise. The differential time courses of recovery suggest that the changes in the parameters of muscle architecture are driven by different exercise-related stimuli. Increased muscle perfusion and tendon creep are likely candidates accounting for short-term changes in muscle architecture. Future research should directly relate the

time course of changes in tendon stiffness and muscle perfusion to the time course of changes in muscle architecture.

Practical implications

- Measurements of muscle architecture are affected by previously performed bouts of exercise. Sufficient recovery times are essential to assess baseline values and avoid activity-related bias.
- Parameters of muscle architecture recover at distinctive rates. Multiple factors may therefore account for acute changes in muscle architecture. In the future, better understanding of these underlying mechanisms may allow practitioners to evaluate the exercise-induced reactions of the muscle-tendon unit by measurements of muscle architecture alone.

Disclosures

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