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Region specific patellar tendon hypertrophy in humans following resistance training

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Abstract

Aim: To examine if cross-sectional area (CSA) differs along the length of the human patellar tendon (PT), and if there is PT hypertrophy in response to resistance training.

Methods: Twelve healthy young men underwent baseline and post-training assessments. Maximal isometric knee extension strength (MVC) was determined unilaterally in both legs. PT CSA was measured at the proximal-, mid- and distal PT level and quadriceps muscle CSA was measured at mid-thigh level using magnetic resonance imaging. Mechanical properties of the patellar tendons were determined using ultrasonography. Subsequently, subjects performed 12 weeks of heavy resistance knee extension training with one leg (Heavy-leg), and light resistance knee extension training with the other leg (Light-leg).

Results: The MVC increased for heavy-leg ($15 \pm 4\%$, $P < 0.05$), but not for light-leg ($6 \pm 4\%$). Quadriceps CSA increased in heavy-legs ($6 \pm 1\%$, $P < 0.05$) while unchanged in light-legs. Proximal PT CSA (104 ± 4 mm^2) was smaller than the mid-tendon CSA (118 ± 3 mm^2), which again was smaller than distal tendon CSA (127 ± 2 mm^2, $P < 0.05$). Light-leg PT CSA increased by $7 \pm 3\%$ ($P < 0.05$) at the proximal tendon level, but was otherwise unchanged. Heavy-leg PT CSA increased at the proximal and distal tendon levels by $6 \pm 3\%$ and $4 \pm 2\%$ respectively ($P < 0.05$), but was unchanged at the mid tendon level. PT stiffness increased in heavy-legs ($P < 0.05$) but was unchanged in light-legs. Modulus remained unchanged in both legs.

Conclusions: To our knowledge, this study is the first to report tendon hypertrophy following resistance training. Further, the data show that the human PT CSA varies along the length of the tendon.

Keywords resistance training, tendinopathy, tendon cross-sectional area, tendon hypertrophy, tendon mechanical properties.

The human patellar tendon is commonly afflicted by overload injuries, especially patellar tendinopathy. The overall prevalence of patellar tendinopathy among sporting athletes has been estimated at ~15% with a prevalence in elite jumping athletes (volleyball players) as high as 50% (Ferretti *et al.* 1983, Ferretti 1986, Lian *et al.* 1996, 2005, Witvrouw *et al.* 2001, Morelli & Rowe 2004). In patellar tendinopathy, the proximal region (i.e. patella insertion) of the patellar tendon, particularly the posterior part, is more frequently

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affected (65%) than the distal region (i.e. tibial insertion) (Ferretti 1986, Johnson *et al.* 1996, Peace *et al.* 2006).

Some investigators believe that during knee flexion, the inferior apex of the patella has a propensity to impinge upon the deep surface of the tendon and thereby cause patellar tendinopathy (Johnson *et al.* 1996, Basso *et al.* 2001). However, this hypothesis was recently refuted by Schmid *et al.* (2002) who examined patella shape, tendon insertion location and patellar tendon angle using MRI in symptomatic and asymptomatic individuals. The anatomical configurations were similar in the two groups and the authors concluded that chronic strain-overload rather than impingement was the principal cause of patellar tendinopathy (Schmid *et al.* 2002). In fact, although there is no firm evidence, it is commonly accepted that the development of patellar tendinopathy is related to strain-overload and micro ruptures of the tendon fascicles (Jozsa *et al.* 1984, Ferretti 1986, Archambault *et al.* 1995, Kannus 1997, Khan *et al.* 1999). However, the suggested pathogenesis that repeated strain overloading of the tendon develops tendinopathy fails to explain the site specificity of the injury, i.e. its proximal and posterior prevalence.

The ability of tendinous tissue to respond to mechanical loading pattern of the muscle-tendon unit is of great interest, since it may relate to overloading injury (Kvist 1994, Archambault *et al.* 1995, Kannus *et al.* 1997). Tendon tissue has, until recently, generally been thought of as a sluggish structure with a slow metabolism and with an inability to structurally adapt to increase mechanical loading. However, novel *in vivo* studies have convincingly shown that human tendinous tissue is highly responsive to mechanical loading. In fact, collagen synthesis in the human Achilles and patellar tendon has been shown to increase acutely after a strenuous bout of endurance exercise and remain elevated up to 72 h post-exercise (Langberg *et al.* 2000, Miller *et al.* 2005). Additionally, the cross-sectional area (CSA) of the Achilles tendon of trained athletes has been shown to be greater than those in control subjects, which indirectly suggests that tendon hypertrophy may be an adaptive mechanism to loading (Rosager *et al.* 2002, Magnusson & Kjaer 2003, Kongsgaard *et al.* 2005). However, only a few longitudinal studies exist which have examined the effect of exercise training on the size of the human tendon (Hansen *et al.* 2003).

Tendon response to high-load types of exercise such as jumping, sprinting and heavy resistance training is poorly understood. Studies using animals models have shown tendon hypertrophy in response to high-load training in rats and horses (Sommer 1987, Birch *et al.* 1999). However in contrast, the few existing studies on humans have reported unchanged tendon CSA after periods (8–14 weeks) of heavy resistance training (Kubo *et al.* 2001a, 2002, Reeves *et al.* 2003). It is a well-established fact that heavy resistance training regimes can increase muscle CSA and muscle strength (Narici *et al.* 1989, Hakkinen *et al.* 1998a, Aagaard *et al.* 2001). When a muscle increases in size and strength, the tendon is subjected to greater stress unless it also amplifies its CSA. Theoretically, an increased tendon CSA may protect against overload injuries since it will reduce the tendon stress (force/area) which will reduce the magnitude of the tendon strain for a given external applied force. However, if human tendon hypertrophy in response to high-load types of exercise remains to be elucidated.

We have previously shown that the CSA of the Achilles tendon is non-uniformly distributed along the tendon length, and that the typically site of Achilles tendon injuries (3–6 cm proximal to the calcaneal insertion) has the smallest CSA (Rosager *et al.* 2002, Magnusson & Kjaer 2003, Kongsgaard *et al.* 2005), indicating that the magnitude of stress may be related to injury mechanisms. Thus, based on our previous findings, and on the basis of the strain-overload theory, we hypothesized that the high prevalence of patellar tendinopathy at the proximal tendon region could be related to a region specific smaller tendon CSA. The aims of the present study were to examine if the cross-sectional area differs along the length of the human patellar tendon, and if the human patellar tendon hypertrophy in response to heavy and light load resistance training.

Materials and methods

Subjects

Twelve healthy untrained young men were included. The mean (± SEM) age, body mass, height and body mass index (BMI) was 24.6 ± 1.0 years, 80.9 ± 3.9 kg, 183 ± 2 cm and 24.2 ± 1.0 kg m^{-2}. None of the subjects had participated in any systematic training or physical activity during the last 6 months. All participants were non-smokers and had no chronic illnesses or family history of diabetes. The study complied with the Declaration of Helsinki and was approved by the local ethics committee for medical research in Copenhagen (KF 01-171/04). All subjects gave their written informed consent to the conditions of the study.

Strength measurements

All subjects conducted a series of pre-trial strength tests (approx. 1-week before baseline testing) to familiarize

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themselves with the test procedure in order to reduce any learning effects.

Isometric knee extension peak torque of both legs was measured in a KinCom dynamometer (KinCom; Chattanooga group, Inc. Harrison, TN, USA). Subjects were seated in the rigid KinCom chair with a 10° recline backrest. They were firmly strapped at the hip and at the distal part of the thigh and had their arms held across their chest. The rotational axis of the KinCom lever arm was visually aligned with the rotational axis of the knee joint, and the lower leg was attached to the lever arm just above the medial malleolus. The individual positioning of the seat and dynamometer were recorded to ensure identical pre- to post-training settings. The isometric peak torque was corrected for the effect of gravity on the lower leg and foot (Aagaard *et al.* 1994, 1996). Unilateral maximal voluntary isometric knee extension strength ($MVC_{knee\backslash ext}$) was measured at an angle of 70° of knee flexion (0° = full extension) (Kues *et al.* 1992, Narici *et al.* 1992). The KinCom straingauge signal was sampled at 1000 HZ and during analysis lowpass filtered using a fourth order zero-log Butterworth filter with a 15 HZ cutoff frequency (Aagaard *et al.* 2001). The highest peak torque achieved during three maximal attempts with verbal encouragement and visual feedback was used for analysis.

Subjects had their one repetition maximum (1RM) knee extension strength determined in a unilateral leg extension machine (Technogym®; Super Executive Line, Gambottola, Italy). After a 5-min warm-up on a cycle ergometer, subjects were seated on the leg extension machine and firmly strapped at the hip and were allowed to grab the seat with their hands in order to remain in the seat. Thereafter, the 1RM was determined by gradually increasing the load until the subjects were unable to perform a full knee extension. The knee joint range of motion was from 100° to 30° and verbal encouragement was given during the trial. The 1RM was determined as the maximal resistance load lifted.

Muscle and tendon morphological measurements

The anatomical cross-sectional areas of m. quadriceps femoris (CSA_{quad}) of both legs were measured 20 cm proximal from the tibia plateau (mid-thigh level) by magnetic resonance imaging (MRI) [General Electric, Signa Horizon LX 1.5 Tesla, (GE Healthcare Diagnostic Imaging, Brøndby, Denmark) T1 weighted SE] using a lower extremity coil. The images were obtained using the following parameters: TR/TE = 500/14 ms, FOV 18, matrix 512 × 512 and slice thickness = 6 mm. Subsequently, the lean muscle mass of m. quadriceps (subcutaneous and intermuscular non-contractile tissue were not included in the measurement) was manually outlined using the software program WEB 1000 (AGFA®, Mortsel Belgium). The mean value of three measurements of the same image was used for analysis.

Patellar tendon CSA and patellar tendon length was determined with the use of MRI and the lower extremity coil (General Electric, Signa Horizon LX 1.5 Tesla, T1 weighted SE). The patellar tendon CSA was determined by axial plane MR using the following parameters: TR/TE 400/14 ms, FOV 20, matrix 256 × 256, slice thickness 5.0 mm and spacing 0 mm. The axial scans were performed perpendicular to the patellar tendon. The tendon CSA was measured (1) just distal to the patellar insertion, (2) just proximal to the tibia insertion, and (3) midway between these two sites (Fig. 1). To examine if CSA differed along the length of the patellar tendon the pre-CSA values for the light-legs were selected for baseline analysis since there was no baseline differences between the light-leg and heavy-leg patellar tendon CSA. The patellar tendon length was determined from saggital plane MRI using the following parameters: TR 500, ET: 3 × (TE: 12.4 ms), FOV 16, Matrix 256 × 192, slice thickness 4.0 mm and no spacing. The patellar tendon length was obtained by measuring the distance from the dorsal insertion at the patella apex to the dorsal insertion on the tibia (Fig. 1). The patellar tendon CSA and patellar tendon length were manually outlined using the software program WEB 1000 (AGFA®). The mean value of three measurements of the same image was used for analysis. The MRI assessment investigator was blinded with regards to the intervention.

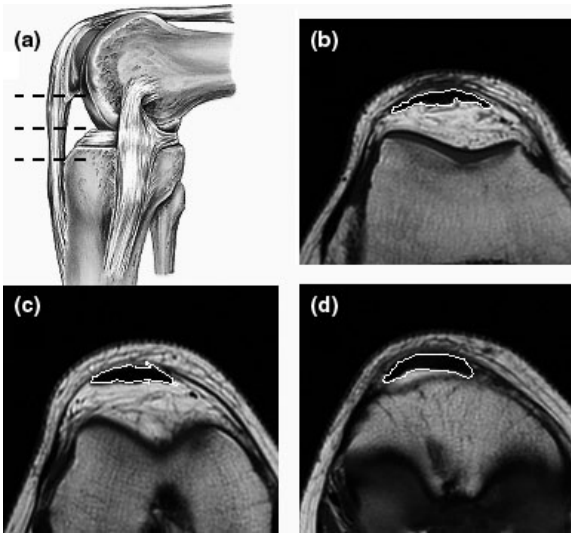

Figure 1 Schematic depiction of the axial tendon MRI slices. Axial MRI was obtained just distal to the patella, at mid tendon level and just proximal to the tibia insertion (a). Following MRI the CSA of the patellar tendon was manually outlined at the proximal- (b), Mid- (c) and distal tendon level (d). Notice that Hoffas fat pad form distinctive interdigitations with the proximal anterior tendon region (b).

Assessment of patellar tendon mechanical properties

The details of the measurement, including the reliability of the method, has been reported previously (Hansen *et al.* 2006). Subjects performed a 5-min warm-up on a stationary bike in order to secure proper preconditioning of the tendon prior to testing. Thereafter, the subjects were seated in a custom-made rigid chair with both hips and knees flexed to an angle of 90°. A leg cuff, connected to a strain gauge (Bofors KRG-4, Bofors, Sweden) through a rigid steel rod perpendicular to the lower leg, was mounted on the lower leg just above the medial malleolus. An ultrasound probe (7.5 MHz, linear array B-mode; Sonoline Sienna, Siemens, Erlangen, Germany) was fitted into a custom-made rigid cast that was secured to the skin above the patellar tendon in the saggital plane. The ultrasound probe and cast was positioned so that the patella, the patellar tendon and the tibia were all visible within the viewing field throughout the ramped contractions (Fig. 2).

After a careful preparation of the skin (shaving, abrasion and cleaning with alcohol) bipolar electromyography (EMG) surface electrodes (Medicotest; Type QN-10-A, Oelstykke, Denmark) were attached to the skin above the muscle bellies of the vastus lateralis, vastus medialis and biceps femoris with a 1.8 cm interelectrode distance. A reference electrode was placed over the proximal patella. To asses the magnitude of antagonist (hamstring) co-activation during the ramped knee extension efforts, the subjects performed two 5 s maximal isometric knee-flexion contractions separated by 1 min (sampled at 1000 Hz) (Bojsen-Moller *et al.* 2003). Assuming a linear relationship between EMG amplitude and muscle tension, the average of these data were used to correct for antagonist (hamstring) coactivation during the ramp trials that would otherwise underestimate the tendon force (Magnusson *et al.* 2001). During the hamstring contractions the strain-gauge force signal was filtered using a fourth order zero-log Butterworth filter with a 6 Hz cutoff frequency.

The ultrasound S-VHS video images obtained during the ramp trials were sampled at 50 Hz using frame-by-frame capturing software (Matrox Marvel G400-TV, Dorval, Canada). Force and integrated EMG data (rectified, time constant of 200 ms) were synchronously sampled on two separate PC's at 50 Hz via a 12-bit A/D converter (dt 2810A; Data Translation, Marlboro; MA, USA). The two computers were inter-connected to permit synchronous sampling of all data using a custom-built trigger device (Bojsen-Moller *et al.* 2003). The subjects performed 4–5 slow force isometric ramps by applying gradually increasing force on the cuff over a 10 s period, while patellar tendon displacement, knee extension force and electromyography were measured simultaneously. Each ramp was separated by a 2 min rest. All measurements were performed on both legs. During the ramp contractions, force was sampled

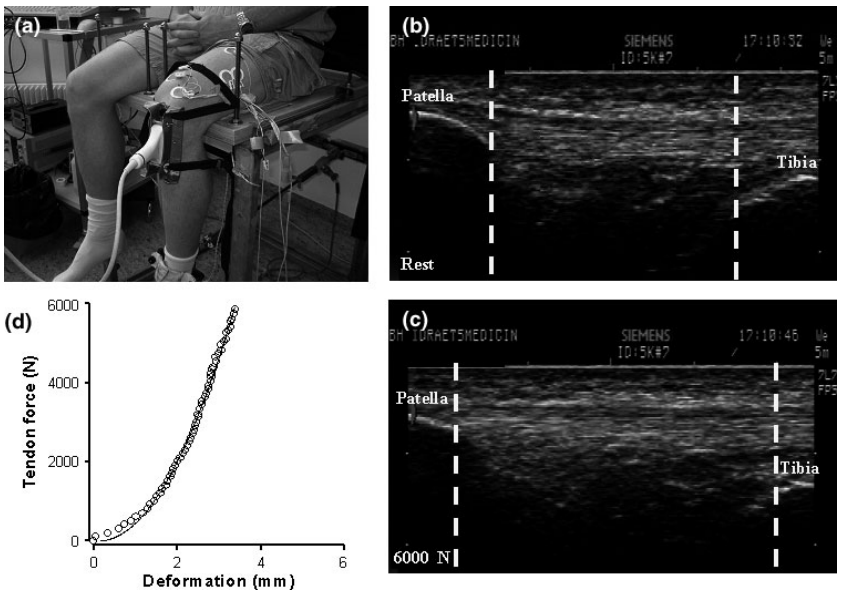

Figure 2 Measurement of patellar tendon mechanical properties. Subjects were seated in a custom-made rigid chair with hips and knees flexed to 90 °, with a leg-cuff mounted to their lower leg and with the ultrasound-probe positioned in a way so that the patella and the tibia were both visual (a). Ultrasound video images were captured in order to measure displacement of the patella and tibia during force exertion (b & c). Synchronized values of patellar tendon force and tendon elongation/deformation was used to construct force-deformation curves from which the mechanical properties were subsequently calculated based on polynomial fitting (d).

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at 50 Hz and filtered at a 1.0 Hz cutoff frequency using a fourth order zero-log Butterworth filter. The two ramp contractions that yielded the greatest force were used for further analysis. The trials (both pre- and post-trials) were subsequently analysed to the greatest common force for each individual subject.

Patellar tendon force was calculated by dividing the estimated total knee extension moment (corrected for hamstring co-activation) by the internal moment arm, which was estimated from individually measured femur lengths (Visser *et al.* 1990). Patellar tendon stress was calculated by dividing tendon force with the proximal-tendon CSA determined from the MRI. The proximal-tendon CSA was chosen because this region of the tendon was found to have the smallest CSA and therefore was subjected to the highest stress values. Patellar tendon deformation was defined as the change in linear distance between the patellar apex and the tibia (Magnusson *et al.* 2003, Hansen *et al.* 2006). Tendon strain was calculated as the change in length related to the original length ($\Delta L/L_0$) and expressed as a percentage. Each force-deformation curve was fitted to a second or third order polynomial fit, which in all cases exceeded $R^2 = 0.95$. Tendon stiffness ($\Delta F/\Delta L$) and modulus (stress/strain) were calculated in the final 10% of the force-deformation and stress–strain curves respectively (Magnusson *et al.* 2001, 2003). Again, the investigator of the tendon mechanical properties was blinded to the intervention of the different legs.

Resistance training interventions

Following the baseline measurements one leg was randomly assigned to heavy resistance knee extension training (Heavy-leg), and the other leg was assigned to light resistance knee extension training (Light-leg). Both training interventions included 12 weeks of resistance training for the quadriceps femoris muscle in a leg extension machine (Technogym®; Super Executive Line, Gambottola, Italy) with three exercise sessions per week. A total of 36 training sessions were prescribed and the lowest accepted training frequency was 2.5 sessions per week. In each training session the heavy-legs performed ten sets of eight repetitions with a load corresponding to 70% of 1RM with 3 min of rest between each set. The light-legs performed a total of 10 sets of 36 repetitions with a load equaling the amount of work to the work performed by the heavy leg. For the light-legs each set was separated by 30 s. of rest. Thus, both legs always performed an equal absolute amount of work. Before the tenth, twentieth and thirtieth training session, a new 1RM was determined for each leg as previously described and the training loads were adjusted accordingly. A training session lasted approximately 35 min. All training sessions were supervised.

Nutrient supply

All subjects received a 100 mL nutrient drink (Komplet Näring, Semper; Novartis Healthcare, Copenhagen, Denmark) immediately after each training session. The energy value of the 100 mL drink was 120 kcal (5 g protein, 16 g Carbohydrates and 4 g fat). Furthermore, all subjects were encouraged to eat as soon as possible after the completion of each training session.

Statistical analysis

All data are presented as mean ± standard error of the mean (SEM). All data were analysed with the use of non-parametric statistics since the distribution of the data did not follow a Gaussian distribution. Differences in patellar tendon CSA along the length of the tendon, differences in patellar tendon mechanical properties, differences in muscle CSA and differences in strength values were analysed using the Friedman's two-way analysis of variance with Dunn's multiple comparison test as post-test. Changes in patellar tendon CSA and anthropometrical data were analysed using the Wilcoxon matched pairs test. Differences in relative delta-values were analysed using the Mann–Whitney test. All tests were carried out as two-tailed with a chosen level of significance of 0.05. The statistical analyses were performed using the statistical software package GRAPHPAD PRISM® (San Diego, CA, USA) Version 4.01 (2004).

Results

Strength, muscle CSA and anthropometry

Subject body mass did not change during the intervention period (80.9 ± 3.9 kg vs. 81.0 ± 3.8 kg). There were no significant differences at baseline between the heavy-legs and the light-legs in any of the strength parameters or muscle CSA (Table 1). In the heavy-legs the CSA of m.quadriceps increased significantly from 7917 ± 296 mm^2 to 8412 ± 320 mm^2 (+6.3 ± 1.3%, $P < 0.05$). In the light-legs the CSA of m. quadriceps did not change (Table 1). The relative increase in quadriceps CSA in heavy-legs was significantly greater than in light-legs (6.3 ± 1.3% vs. 1.1 ± 0.8%, $P < 0.01$). 1RM knee extension strength increased significantly in both legs (Light leg: $P < 0.05$, Heavy-leg: $P < 0.01$). However, the 1RM strength gain was significantly greater in the heavy-legs compared with light-legs (35.3 ± 4.4% vs. 19.5 ± 2.3%, $P < 0.05$) (Table 1). MVC increased significantly from 253 ± 13 Nm to 290 ± 17 Nm in the heavy-legs ($P < 0.05$) but did not increase significantly in the light-legs. Thus, the relative increase in MVC was significantly greater in heavy-legs compared to light-legs (14.6 ± 4.3% vs. 6.1 ± 4.0%, $P < 0.05$) (Table 1).

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Table 1 Knee extension strength and muscle CSA

	Light-leg (*n*)			Heavy-leg		
	Pre	Post	Change (%)	Pre	Post	Change (%)
CSA_{quad} (mm^2)	8038 ± 305	8121 ± 309	1.1 ± 0.8	7917 ± 296	8412 ± 320*	6.3 ± 1.4‡
1RM (kg)	82.5 ± 5.0	97.9 ± 5.4*	19.5 ± 2.3	78.3 ± 4.8	104.1 ± 4.3†	35.3 ± 4.4‡
MVC (Nm)	276.7 ± 17.2	291.7 ± 17.8	6.1 ± 4.0	253.5 ± 13.1	290.0 ± 17.1*	14.6 ± 4.3‡

Values are mean ± SEM.
CSA_{quad}, m. quadriceps cross-sectional area; 1RM, one repetition maximum; MVC, maximal isometric knee extension strength.
*Significantly greater than pre ($P < 0.05$).
†Significantly greater than pre ($P < 0.01$).
‡Significantly greater than light-leg ($P < 0.05$).

Patellar tendon CSA

There was a significant difference in the CSA of the patellar tendon along its length. The proximal patellar tendon CSA (104 ± 4 mm^2) was significantly smaller than the middle tendon CSA (118 ± 3 mm^2), which again was significantly smaller than the distal tendon CSA (127 ± 2 mm^2) ($P < 0.05$, Fig. 3). There were no significant differences at baseline in patellar tendon CSA along the length of the tendon between the heavy-legs and the light-legs (Fig. 4). In the light-legs the patellar tendon CSA increased by 7 ± 3% ($P < 0.05$) at the proximal tendon level following training, but remained unchanged at the mid- and distal-tendon levels (Fig. 4). In the heavy-legs the patellar tendon CSA increased by 6 ± 3% at the proximal-tendon level ($P < 0.05$) and by 4 ± 2% at the distal tendon level ($P < 0.05$), but was unchanged at the mid-tendon level (Fig. 4).

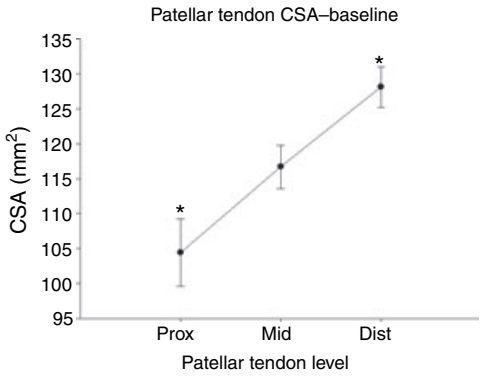

Figure 3 Baseline values of the patellar tendon cross-sectional area (CSA) distribution. CSA were measured at the proximal- (prox), mid- (mid) and distal tendon level (dist). *Significantly different from mid-tendon value, $P < 0.05$. Data are expressed as mean ± SEM.

Patellar tendon mechanical properties

Patellar tendon mechanical properties are reported in Table 2. No differences were found between the light-legs and the heavy-legs at baseline for any of the measured mechanical properties (Table 2). Additionally, no changes in peak moment, peak tendon force, deformation or stress were found in either of the groups after the intervention period (Table 2). Strain decreased from 6.3 ± 0.6% before the intervention period to

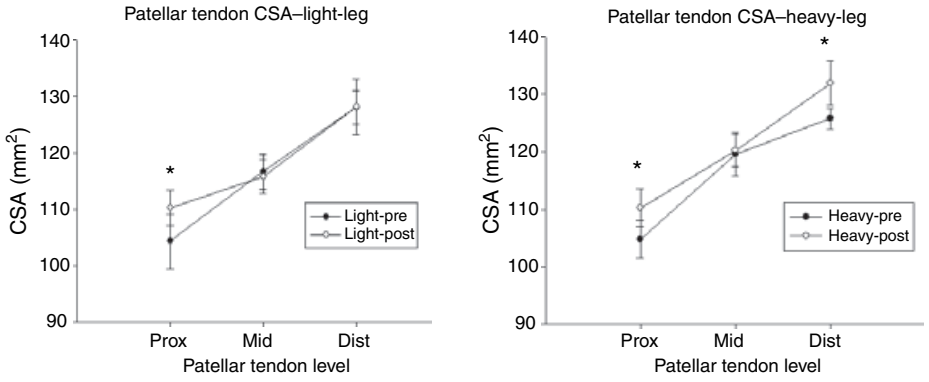

Figure 4 Patellar tendon cross-sectional area (CSA) distribution at baseline and after the intervention period for the light-legs and heavy-legs respectively. *Significantly different from pre-values, $P < 0.05$. Data are expressed as mean ± SEM.

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Table 2 Patellar tendon mechanical properties

	Light-leg		Heavy-leg	
	Pre	Post	Pre	Post
Peak moment (Nm)	167 ± 14	165 ± 13	164 ± 11	172 ± 13
Peak tendon force (N)	5676 ± 403	5658 ± 462	5619 ± 377	5650 ± 364
Deformation (mm)	2.8 ± 0.2	2.8 ± 0.2	2.7 ± 0.2	2.6 ± 0.3
Stress (MPa)	44.7 ± 3.9	42.0 ± 4.0	44.9 ± ± 3.8	43.0 ± 3.6
Strain (%)	6.3 ± 0.4	6.5 ± 0.4	6.3 ± 0.6	5.8 ± 0.6
Stiffness (N mm^{-1})	3716 ± 452	3375 ± 361	3676 ± 377	4213 ± 405*
Modulus (Gpa)	1.42 ± 0.21	1.36 ± 0.19	1.47 ± 0.17	1.65 ± 0.16

Tendon mechanics calculated on proximal tendon CSA and common force. Values are means ± SEM. Average common force: 4725 ± 374 N.
*Significantly higher than pre ($P < 0.05$).

5.8 ± 0.6% after the intervention in the heavy-legs whereas strain increased from 6.3 ± 0.4 to 6.5 ± 0.4% in the light-legs; however, none of these changes were statistically significant. Tendon stiffness remained unchanged in the light-legs. In contrast, the tendon stiffness for the heavy-legs increased significantly from 3676 ± 377 N mm^{-1} to 4213 ± 405 N mm^{-1} ($P < 0.05$) after training. Tendon modulus did not change significantly in either of the legs despite that the tendon modulus tendon to increase from 1.47 ± 0.17 GPa to 1.65 ± 0.16 GPa in the heavy legs (Table 2).

Discussion

The main findings of the present study were that the human patellar tendon has a non-uniform CSA distribution along its length with increasing CSA from the proximal to distal tendon region, and that the human patellar tendon displayed a region specific hypertrophy in response to resistance training. To the best of our knowledge, these were previously unrecognized observations.

Quadriceps strength and morphology

The baseline anatomical quadriceps muscle CSA in the present study were well in agreement with previously reported values for young untrained men (Narici *et al.* 1988, Aagaard *et al.* 2001). The increase in muscle anatomical CSA in the heavy-legs (6.3%) corresponds to previously reported changes of 5–12% in anatomical CSA for young men after 10–15 weeks of heavy resistance training (Narici *et al.* 1989, Rutherford & Jones 1992, Hakkinen *et al.* 1998b, Aagaard *et al.* 2001). The ~15% increase in maximal isometric quadriceps strength for the heavy-legs was in agreement with previous reports of changes in quadriceps muscle strength following heavy resistance training (Narici *et al.* 1989, Colliander & Tesch 1990, Aagaard *et al.* 1996, 2001). The increased 1RM and the lack of change in either muscle CSA or isometric strength in the light-legs might be a learning effect, as the same knee extension machine was used for training and for 1RM testing.

Patellar tendon mechanical properties

The baseline tendon stiffness (~3700 N mm^{-1}) and modulus (~1.45 GPa) of the present study correspond to that reported for the patellar tendon in young males (Hansen *et al.* 2006), elderly subjects (Reeves *et al.* 2003), and from *in vitro* mechanical testing of patellar tendon specimens (Butler *et al.* 1984). The baseline strain at MVC (6.3%) correspond to that reported in human patellar tendons (Reeves *et al.* 2003, Hansen *et al.* 2006, Maganaris *et al.* 2006). Additionally, the baseline stress at MVC (~45 MPa) is in agreement with that of Reeves *et al.* (2003), but are larger than that reported by Hansen *et al.* (2006) (~30 Mpa) and Maganaris *et al.* (2006) (~24 Mpa). The difference in stress is likely to be related to the between study variation in patellar tendon CSA. Patellar tendon stiffness remained unchanged in the light-legs but increased significantly in the heavy-legs (Table 2), which is somewhat lower than the 65% increase in patellar tendon stiffness in elderly after 14 weeks of heavy resistance training (Reeves *et al.* 2003). In addition to the obvious improvement in force development (Ker *et al.* 1988), an increased tendon stiffness may have some important clinical consequences (see below).

The modulus of the patellar tendons in the light-legs did not change while the modulus of the patellar tendon in the heavy-legs tended to increase. Thus, the lack of significant increases in modulus after heavy resistance training in the present study is somewhat in opposition to the vast increase in tendon modulus in response to heavy resistance training in elderly subjects reported by Reeves *et al.* (2003). Albeit speculative, the discrepancy

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may be related to differences in intervention protocol and/or subject age.

Patellar tendon CSA distribution

The CSA of the patellar tendon was less at the proximal compared to the mid- and distal-tendon region (Table 1). A scrutiny of the axial MRI scans revealed that the smaller tendon CSA at the proximal region was associated with discrete interdigitations of Hoffas fat pad into the anterior part of the proximal regions (Fig. 2b), which has also been described by others (Toumi *et al.* 2006).

Contrary to our findings, others have been unable to show a difference in CSA along the length of the patellar tendon (Maganaris *et al.* 2006), however, the accuracy of ultrasonography to measure tendon dimensions has been questioned (Roberts *et al.* 1999). The larger distal patellar CSA is similar to that in the Achilles tendon (Rosager *et al.* 2002, Magnusson & Kjaer 2003, Kongsgaard *et al.* 2005), and may be related to compressive loads of the tendons against the bones, i.e. tibia and calcaneous, respectively, during movement. Compressive loads have been shown to stimulate the synthesis of extracellular matrix proteins (Evanko & Vogel 1993, Robbins *et al.* 1997, Malaviya *et al.* 2000).

Despite the high incidence of patellar tendinopathy, the aetiology remains elusive. It is generally accepted that injury is related to tensile load and strain of the tendon fascicles and fibrils (Jozsa *et al.* 1984, Ferretti 1986, Archambault *et al.* 1995, Khan *et al.* 1999). The present data show that for a given load the patellar tendon stress varies considerably along its length, and that it is the greatest at the proximal tendon, which may in part explain the frequency of patellar tendinopathy at the proximal tendon region (Ferretti *et al.* 1983, Ferretti 1986). We have previously shown that the CSA of the human Achilles tendon is the smallest ~3–6 cm proximal to the insertion into calcaneus, which corresponds to the site where Achilles tendinopathy and ruptures frequently occur (Magnusson & Kjaer 2003, Kongsgaard *et al.* 2005). However, it remains unknown why the distal portion of the patellar tendon is more frequently affected by tendinopathy than the mid-region of the patellar tendon (Ferretti *et al.* 1983, Ferretti 1986). If force is distributed homogenously throughout the patellar tendon, our data indicates that stress in the mid-region is somewhat greater than that of the distal portion of the tendon, and thus tendon stress is unlikely to be the sole factor in the aetiology of patellar tendinopathy. Previous *in vitro* study on the human patellar tendon have indicated that the tissue of the osteo tendinous junction (OTJ) display inferior modulus and maximum stress compared to mid-tendon tissue (Butler *et al.* 1984).

Patellar tendon hypertrophy

Mid-tendon CSA was unchanged, while both low- and heavy load resistance training yielded increased proximal tendon CSA. For the light-legs, the increased proximal tendon CSA was accompanied by unchanged stiffness and modulus, indicating an increased non-collagenous content.

The increased tendon CSA, increased stiffness and unaltered modulus in the heavy-legs suggest real tendon hypertrophy (i.e. net formation of collagen tissue) without a change in the material properties. Although several studies, using animal models, have demonstrated tendon hypertrophy in response to high-load training (Sommer 1987, Banes *et al.* 1999, Birch *et al.* 1999, Olesen *et al.* 2006), this is to the best of our knowledge, the first human intervention study to report tendon hypertrophy. Further, these data extend previous observations that volleyball players have larger normalized Achilles tendons compared to non-jumping athletes (Kongsgaard *et al.* 2005). However, others (Kubo *et al.* 2001b, 2002, Reeves *et al.* 2003) have been unable to show tendon hypertrophy following heavy resistance training as measured with ultrasonograhpy and low resolution MRI. Both tendon collagen and muscle protein synthesis is augmented after exercise indicating a coupling between muscle and tendon (Miller *et al.* 2005). Langberg *et al.* has reported that eccentric resistance training increases peritendinous type 1 collagen synthesis (Langberg *et al.* 2006), and it has been shown that when the patellar tendon is used as an ACL graft it increase in CSA by almost 50% by 6 months post-surgery, demonstrating that the human patellar tendon tissue has the ability to grow in size (Shimizu *et al.* 2006). Although the mechanism for increased CSA at the insertional regions remains unclear, it appears that the tendon region near the OTJ display some specific intrinsic properties making these regions more susceptible to collagen incorporation and tendon growth. It is possible that the compressive loads in these regions contribute to the hypertrophy since it stimulate the synthesis of extracellular matrix proteins in tendinous tissue (Evanko & Vogel 1993, Robbins *et al.* 1997, Malaviya *et al.* 2000).

Clinical relevance

Increased tendon CSA and tendon stiffness will decrease tendon stress and strain for any given magnitude of tensile loading, and may therefore reduce the risk of overload injuries (Ker *et al.* 1988). The general strain-overload theory is based on the assumption that repeated straining of the tendon above some given threshold fatigue damage accumulates whereupon injury occurs (Jozsa *et al.* 1984, Ferretti 1986, Archa-

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mbault *et al.* 1995, Kannus 1997, Khan *et al.* 1999, Ker *et al.* 2000). Several *in vitro* studies have reported that changes in tendon CSA, tendon stiffness and tendon modulus are accompanied by equivalent changes in tensile strength and ultimate stress (Woo *et al.* 1982, Nakagawa *et al.* 1994, 1996, Yamamoto *et al.* 1999, Yasuda & Hayashi 1999, Matsumoto *et al.* 2003). Strengthening exercise regimes, particularly regimes focusing on eccentric strengthening, have already been advocated as treatment of tendon overuse conditions, such as Achilles and patellar tendinopathies during the past 20 years (Clement *et al.* 1984, Alfredson *et al.* 1998, Holmich *et al.* 1999, Cannell *et al.* 2001, Purdam *et al.* 2004, Young *et al.* 2005). Thus, the present data provide a mechanism by which resistance training may reduce the stress and thereby the risk of injury.

In summary, the present study is to the best of our knowledge the first human study to report tendon hypertrophy following heavy resistance training. Further, the data show that tendon hypertrophy to heavy-resistance training in the patellar tendon was related to the proximal and distal region, but not to the mid-region of the tendon. Finally, the present study found that the CSA of the human patellar tendon is non-evenly distributed along the length of the tendon but increases in a proximal to distal direction.

Conflict of interest

There are no conflicts of interest in the present study.

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