Training-induced changes in structural and mechanical properties of the patellar tendon are related to muscle hypertrophy but not to strength gains


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The structure and size of the tendon determine its interaction with the associated muscle (10, 30), and the coordination of the changes occurring in both structures is critical to the preservation of muscle-tendon unit function. When tendon scaling is compared across species, or when positive allometry related to development is considered, muscle-tendon interaction seems invariably preserved through an optimum muscle-to-tendon area ratio (5, 14, 24). Although mature muscle-tendon units also undergo substantial structural changes with chronic overloading, the level of coordination between tendinous and muscular adaptations, and therefore the evolution of the interaction between these two structures, is not known.

Alongside typical increases in muscle mass and strength (39), it has been shown that resistance training induces remarkable changes in tendon mechanical and structural properties (26, 27, 43). In vivo data from most training studies indicate that human tendons become stiffer with chronic overloading without any change in tendon cross-sectional area (CSA), indicating changes in intrinsic material properties (27, 43). Evidence from animal models (7), from cross-sectional studies on humans athletes (34), and recently from individuals studied longitudinally (3, 26) indicates an increase in tendon CSA with chronic overloading. Using magnetic resonance imaging, Kongsgaard et al. (26) showed that resistance training resulted in hypertrophy near the insertion sites of the tendon. These findings bear an important implication, since hypothetical mechanisms underlying tendon hypertrophy may be drawn from the specificity of its anatomical distribution. For instance, if increases in CSA were restricted to the insertion sites only, the etiology of patellar tendon hypertrophy would be limited to these regions [e.g., tendon-bone compressive forces (26)]. However, the extent of regional hypertrophy along the entire tendon is not known, and a more detailed study with a greater number of scans is required to understand this phenomenon.

It is commonly accepted that training-induced tendon adaptations are driven by the stress resulting from muscular work. From a metabolic point of view, the elevated peritendon collagen synthesis typically observed with acute and chronic overloading (28, 29) seems to be coordinated with myofibrillar protein and muscle collagen synthesis following one bout of exercise (35). These synchronous responses in muscle and tendon suggest that increased loading would trigger mechanotransduction via the extracellular matrix of both in-series structures. However, elevated net protein synthesis alone does not denote protein incorporation, turnover rates of both tissues differ substantially (21, 35), and the comparative magnitude of morphological and functional adaptations in tendon and muscle is unknown. Although the preservation of the optimal muscle-tendon interaction would seemingly benefit from an increase in tendon stiffness proportional to gains in muscle strength and/or mass, the relationships between tendon and muscle structural and functional adaptations have never been tested experimentally.

To obtain a better understanding of the effect of chronic overloading on human tendon, we examined the adaptations that the human patellar tendon undergoes in response to a 9-wk resistance training program, and we investigated the relation-
ships between these adaptations and changes in muscle structure and function.

Since the actual work performed by the muscle results in the total stress transmitted to the tendon, we hypothesized that training-induced muscular adaptations would be related to changes in tendon. Namely, we expected that changes in patellar tendon structural and mechanical properties following training would be positively correlated to changes in physiological CSA (PCSA) and in maximal force of the quadriceps muscles. A secondary hypothesis was that the above relationships would be specific to tendon regions displaying larger increases in CSA.

METHODS

Subjects. Fifteen young men (age 20.4 ± 2.2 yr, height 1.77 ± 0.04 m, and mass 73.6 ± 6.3 kg) were recruited for this study. The present experiment is part of another study that investigated the effects of resistance training on muscle size and force (15, 16). All subjects were recruited from a university student population. The majority of them participated in recreational sporting activities (1–2 times/wk) but not at a competitive level. Exclusion criteria for participating in the experiments were the occurrence of lower limb fracture within the past 6 mo, the participation in a strength training programme within the past 12 mo, the intake of performance-enhancing dietary supplements, and a self-reported chronic health condition precluding resistance training. In accordance with the Declaration of Helsinki, each subject signed an informed consent, and the study was approved by the institutional Ethics Committee.

Experimental design and resistance training. The training and all testing procedures were performed on the right leg of all participants. Subjects attended a previst 1 wk before baseline measurements to be familiarized with involved equipment and procedures and to assess the maximal training load that they could lift once only (1RM) throughout the full range of knee extension. The range of movement spanned from 110° to 20° of knee flexion (0° = full knee extension). All measurements were performed at baseline and after 9 wk of heavy resistance training. Training was performed unilaterally, three times per week. Each session started with a warm-up set of 10 knee extensions (Technogym, Gambettola, Italy) at 40% of 1RM. The training part included four sets of 10 knee extensions at 80% of 1RM, with 2 min of rest between sets. Training load was adjusted weekly by reevaluating the 1RM at the start of the first session. Subjects were asked to maintain their habitual dietary intake and level of daily physical activity throughout the training duration.

Muscle strength and electromyography. Knee extension isometric torque was measured with an isokinetic dynamometer (Cybex NORM, New York, NY). The right leg was secured to the lever arm of the dynamometer, and the subjects performed at least two maximal knee extension voluntary contractions (MVC) at each of the following angles of knee flexion: 70°, 80°, and 90°. The highest of the peak torques obtained during the two attempts was retained as the maximal torque. The three angles were tested, in random order, 2 min apart, to identify the knee joint angle of maximal torque production. Because there is little variation in the patellar tendon moment arm in the knee flexion range of 70°–90° (52), the knee angle of peak torque was considered to correspond to maximal contractile force and optimal contractile length in the entire quadriceps muscle.

Subsequently, an additional contraction was performed at 90° of knee flexion, where maximal torque was reached by gradually increasing exertion over ~4 s. This ramp contraction, along with concomitant ultrasonographic recordings of patellar tendon deformation, was used to assess tendon mechanical properties (see Tendon mechanical and material properties). To estimate antagonist muscle co-activation, subjects performed a maximal knee flexion contraction at all angles. By assuming linearity between EMG activity and isometric torque production (2, 6), maximal antagonist EMG and torque recorded during knee flexion were combined to knee extension recordings to correct the extension torque for coactivation. Adhesive silver chloride EMG electrodes (20-mm interelectrode distance, Ambu, Ballerup, Denmark) were placed on the biceps femoris muscle to estimate antagonistic coactivation. Raw EMG signal was digitized (sampling frequency of 2 kHz), stored, and analyzed with commercially available software (Acqknowledge, Biopac System). To quantify EMG activity, root mean square calculations were performed over a 0.5-s period around the MVC peak torque for the knee flexion and knee extension trials.

Muscle PCSA. The following procedure to obtain muscle volume, fascicle length, and PCSA has been recently published (15). The quadriceps femoris muscle was scanned by using a magnetic resonance imaging (MRI) scanner (G-Scan, Esaote, Genoa, Italy), with the following protocol: Turbo 3D TI-weighted, matrix 256 × 256, repetition time (TR) of 40 ms, echo time (TE) of 16 ms, and slice thickness of 2.8 mm without inter-slice gap. Contiguous axial scans were performed perpendicular to the thigh, from the tibio-femoral joint to the iliac crest, and scans at a 3.08-cm interval along muscle length were retained for analysis. The anatomical CSA (ACSA) of each of the four heads of the quadriceps femoris was manually outlined (Osirix 2.7.5, Osiris Foundation, Geneva, Switzerland) and plotted against muscle length. Subsequently, a spline curve was fitted to the ACSA data points, and muscle volume was computed as the area under the curve. This technique has previously displayed strong test-retest reliability (49). Muscle architecture of each of the four muscles composing the quadriceps femoris was measured with ultrasonography (5 cm/10- to 15-MHz transducer, MyLab25, Esaote, Genoa, Italy). To calculate the PCSA for the whole quadriceps femoris, fascicle length measurements were made in each individual muscle head during MVC at the knee joint angle of peak torque (see Muscle strength and electromyography above). The ultrasound probe was positioned over the belly of each muscle head so that fascicles were aligned with the direction of the probe. Fascicle length was measured offline with image analysis software (ImageJ, Wayne Rasband, National Institutes of Health, Bethesda, MD) by manually outlining the fascicular path length between the superficial and deep aponeuroses. Out-of-frame portions of some examined fascicles (<30% of fascicular length) were extrapolated as straight lines (32, 37, 44). The validity and test-retest reliability of ultrasound-based measurement of fascicle lengths in human muscles have been established before (37, 49). Three investigators, blinded to the date at which scans were taken, independently analyzed either MRI, ultrasound muscle architecture scans, or ultrasound tendon scans. Total quadriceps femoris PCSA was obtained as the sum of individual PCSAs calculated in the vastus lateralis, vastus intermedius, vastus medialis, and rectus femoris muscles:

\[
P_{\text{CSA quad}} = \sum_{i} P_{\text{CSA muscle volume}} \times L_i
\]

where \( P_{\text{CSA quad}} \) is the total PCSA of the quadriceps femoris muscle, muscle volume, is the muscle volume in each one of the four heads of the quadriceps femoris muscle, and \( L_i \) is the fascicle length in each one of the four heads of the quadriceps femoris muscle.

Tendon morphology. Tendon dimensions were measured by using MRI with a Turbo 3D TI-weighted protocol (matrix 256 × 256, TR of 40 ms, TE of 16 ms, slice thickness of 3.1 mm without inter-slice gap). Contiguous axial scans were taken perpendicular to the patellar tendon direction from the distal edge of the tibial tuberosity to the lower part of the patella (Fig. 1). The contour of the patellar tendon CSA in each scan was manually outlined (Osirix 2.7.5, Osiris Foundation, Geneva, Switzerland) and plotted against tendon length, and a spline curve was fitted to the tendon CSA data points. The location of the proximal insertion of the tendon was set to the most proximal scan including the apex of the patella over its entire thickness (3.1 mm).
The distal insertion point was set at the proximal edge of the tibial tuberosity, where posterior fiber bundles insert (Fig. 1).

Tendon length at rest was defined as the length of the line running through the centre of the outlined areas, from one insertion to the other. This technique was chosen over the conventional measure of the distance between insertion points to account for the tendon slack. To measure and compare site-specific changes after training, tendon CSA was interpolated at each 10% interval of the tendon absolute length (Lt0 to Lt100) (3). Coefficient of variation and intraclass correlation coefficients for the novel tendon CSA and tendon length measurement approaches implemented in the present study were calculated in a separate group of seven subjects (Table 1). Mean tendon CSA was calculated as the average of interpolated CSAs.

**Tendon mechanical and material properties.** Tendon stiffness and Young’s modulus were obtained from ultrasound measurements. The ultrasound transducer (5-cm/10- to 15-MHz transducer, MyLab25, Esaote, Genoa, Italy) was positioned sagittally on the patellar tendon. An echo-absorptive marker was placed onto the skin to ascertain that the ultrasound probe did not move with respect to the tendon. Immediately before the ramp maximal isometric contraction, subjects performed a series of five submaximal isometric contractions to ensure preconditioning of the tendon (31). Computers used to record ultrasound, torque, and EMG data were synchronized. Elongation of the patellar tendon during a maximal ramp isometric contraction of the quadriceps femoris muscle was measured in each video frame as the displacement of the patellar insertion in the action line of the tendon. It should be acknowledged that the present technique might lead to an underestimation of the tendon elongation due to unmonitored tibial movements (19). Unfortunately, available ultrasound probes did not allow simultaneous scanning of both proximal and distal insertions of the tendon in all subjects. However, because the major portion of the tendon was scanned in an identical manner before and after training, this technical compromise does not invalidate the comparative outcome of our data. A good interday reliability of this technique of tendon elongation measurement has been demonstrated previously (43). Torque and EMG recordings were sampled down to 25 Hz to match the ultrasound video frequency. Patellar tendon force was calculated offline by dividing knee extension torque, corrected for antagonist coactivation, by the patellar tendon moment arm length. Patellar tendon moment arm length was estimated from MRI sagittal scans (Turbo 3D T1-weighted protocol, matrix 256 × 256, TR of 40 ms, TE of 16 ms, slice thickness of 3.1 mm without inter-slice gap) as the perpendicular distance from the patellar tendon to the midpoint of the distance between the tibio-femoral contact points in the lateral and medial femoral condyles (4, 51). Due to constraints in the knee coil and MRI scanner configuration, scans could only be performed with the knee fully extended. Therefore, the moment arm length at 90° of knee flexion was obtained from previously reported ratios of moment arm lengths at 90° over 0° (4, 43). From the analysis of all the scans recorded, individual force-elongation relationships were plotted and fitted with a second- or third-degree polynomial. For all participants, both pre- and posttraining tendon stiffness values were obtained over the highest 20% force interval in the subject, with the lowest maximum tendon force predetermined. This approach avoids the need for extrapolating some data points beyond the visible part of the force-elongation relationship, which would be required for the weaker participants if the force interval selected exceeded their force-generating potential (42). Tendon stress and strain were also calculated at the common highest force level. Tendon stress was obtained by dividing tendon maximal force by mean tendon CSA, and tendon strain was calculated as the percentage of tendon elongation to tendon length. Young’s modulus was obtained by multiplying tendon stiffness by the ratio of tendon length to mean tendon CSA.

**Statistics.** Differences in CSA along the length of the tendon were tested with a one-way ANOVA, followed with Newman-Keuls post hoc test. Posttraining changes in MVC force, quadriceps volume and PCSA, muscle fascicle length, tendon stiffness, and modulus were examined using a paired Student’s t-test. Changes in tendon CSA were analyzed using a two-way ANOVA with a Bonferroni posttest, where factors are the training effect and the location at which CSA was measured along the tendon length. Relationships between changes in tendon-dependant variables and changes in muscle-dependant variables of interest were tested with a Pearson’s product moment correlation. The coefficient of correlation (r), coefficient of determination (R²), and the 95% confidence interval (CI) are reported for each significant relationship. Level of significance was set at P < 0.05. All data are presented as means ± SD.

**RESULTS**

**Muscle strength.** After 9 wk of heavy resistance training, MVC torque increased by 32 ± 9% (P < 0.0001; Table 2).

**Table 1. Reliability of tendon CSA and tendon length measurements**

<table>
<thead>
<tr>
<th>Variables</th>
<th>CV, %</th>
<th>ICC</th>
<th>Lower CL</th>
<th>Upper CL</th>
</tr>
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<tr>
<td>Lt0 CSA</td>
<td>0.16</td>
<td>0.96</td>
<td>0.65</td>
<td>1.00</td>
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<tr>
<td>Lt10 CSA</td>
<td>1.01</td>
<td>0.99</td>
<td>0.93</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt20 CSA</td>
<td>1.11</td>
<td>0.91</td>
<td>0.39</td>
<td>0.99</td>
</tr>
<tr>
<td>Lt30 CSA</td>
<td>1.29</td>
<td>0.99</td>
<td>0.91</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt40 CSA</td>
<td>0.06</td>
<td>0.96</td>
<td>0.64</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt50 CSA</td>
<td>0.60</td>
<td>0.99</td>
<td>0.98</td>
<td>0.86</td>
</tr>
<tr>
<td>Lt60 CSA</td>
<td>0.59</td>
<td>0.98</td>
<td>0.86</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt70 CSA</td>
<td>0.32</td>
<td>0.99</td>
<td>0.92</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt80 CSA</td>
<td>0.46</td>
<td>0.97</td>
<td>0.77</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt90 CSA</td>
<td>0.47</td>
<td>0.96</td>
<td>0.67</td>
<td>1.00</td>
</tr>
<tr>
<td>Lt100 CSA</td>
<td>0.09</td>
<td>0.98</td>
<td>0.86</td>
<td>1.00</td>
</tr>
<tr>
<td>PT length</td>
<td>0.04</td>
<td>0.99</td>
<td>0.91</td>
<td>1.00</td>
</tr>
</tbody>
</table>

CSA, cross-sectional area; CV, coefficient of variation; ICC, intraclass correlation; CL, 95% confidence limit; Lt x, tendon site located % of tendon length distal to the patellar insertion; PT, Patellar tendon. N = 7.
Neither mean optimum angle (80°) nor antagonistic co-activation changed after training. Quadriceps maximal force corrected for antagonistic co-action increased by 31 ± 12% (P < 0.0001; Table 2).

Muscle physiological cross-sectional area. Quadriceps femoris muscle volume increased by 6.4 ± 3.4% (P < 0.001; Table 2). The architecture of the rectus femoris muscle could not be reliably assessed in two subjects; therefore, PCSA data were calculated for 13 subjects. Fascicle length as measured during MVC at the optimum joint angle did not change significantly in any of the four heads of the quadriceps after training (Table 2). Quadriceps PCSA increased by 7.0 ± 7.0% (P < 0.01; Table 2). Among individual changes in PCSA, one value (+22%) was three times higher than the average increase of the group. Such an increase in PCSA of the quadriceps femoris seems unlikely given the 9-wk duration of the present training protocol. For these reasons, correlation analyses including quadriceps PCSA are presented here for 13 subjects and for 12 subjects.

Tendon morphology. Although tendon CSA followed an incremental pattern from the proximal to the distal insertion, statistically significant differences were only observed at Lt90 and Lt100. At these two distant regions, CSA was larger [by 10–17% at Lt90 (P < 0.001) and by 22–28% at Lt100 (P < 0.001)] than at any other site proximally along the tendon length. Finally, tendon CSA was 13% larger at Lt100 compared with Lt90 (P < 0.001) (Fig. 2).

Tendon length did not change significantly after training (52 ± 7 vs. 52 ± 6 mm; P = 0.79). Mean tendon CSA increased by 3.7 ± 2.2% (P < 0.001). However, region-specific analyses revealed that tendon CSA only increased significantly at five sites along the tendon length: at Lt20 (5.2 ± 5.3%; P < 0.05), Lt30 (5.3 ± 7.2%; P < 0.05), Lt60 (5.0 ± 5.3%; P < 0.05), Lt90 (4.9 ± 6.3%; P < 0.05), and Lt100 (5.7 ± 4.7%; P < 0.001) (Figs. 2 and 3).

We did not observe any significant relationship between the changes in tendon CSA and in quadriceps PCSA, with the exceptions of the changes in mean tendon CSA and in tendon CSA at Lt90, displaying negative correlations with the changes in muscle PCSA (in both cases, r = −0.64, R² = 0.41, 95% CI = −0.88 to −0.14, P < 0.05; Fig. 4). However, when correlation analyses were run without the possible outlier (22% increase in PCSA), this relationship lost significance for the changes in mean tendon CSA and was weaker for tendon CSA changes at Lt90 (r = −0.62, R² = 0.38, 95% CI = −0.88 to −0.07, P < 0.05, n = 12).

Tendon mechanical properties. Tendon stiffness increased by 24.1 ± 16.1% (P < 0.001; Table 2), and Young’s modulus increased by 19.7 ± 16.1% (P < 0.01; Table 2).

Posttraining changes in tendon stiffness were positively correlated with changes in muscle PCSA (r = 0.68, R² = 0.46, 95% CI = 0.21–0.90, P < 0.01; Fig. 5), and a similar, stronger relationship was observed between changes in Young’s modulus and changes in muscle PCSA (r = 0.75, R² = 0.57, 95% CI = 0.35–0.92, P < 0.01; Fig. 5). These relationships were improved when the suspected outlier was removed (r = 0.73, R² = 0.53, 95% CI = 0.27–0.92, P < 0.01, and r = 0.77, R² = 0.60, 95% CI = 0.36–0.93, P < 0.01 for changes in stiffness and modulus, respectively). There was no significant relationship between changes in tendon mechanical properties and maximal force.

**DISCUSSION**

To our knowledge, this is the first report on the morphological changes along the entire length of the human patellar tendon after resistance training assessed in vivo. The analysis of the relationships between training-induced muscular and tendinous adaptations revealed that quadriceps hypertrophy but not an increase in maximal force correlates significantly with the increases in patellar tendon stiffness and modulus (R² = 0.46 and 0.57, respectively). Contrary to our hypothesis, tendon hypertrophy did not correlate positively with the increase in muscle PCSA. These data suggest that changes in tendon mechanical and material properties to short-term overloading are closely related to the loading history (i.e., combined intensity, duration, and number of cycles over the training duration) and that tendon hypertrophy is driven by other mechanisms than those eliciting the increase in tendon stiffness.

**Table 2. Muscular and tendinous adaptations**

<table>
<thead>
<tr>
<th>Muscle property</th>
<th>Baseline</th>
<th>Posttraining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee extension MVC, Nm</td>
<td>260.3 ± 47.4</td>
<td>341.6 ± 49.8*</td>
</tr>
<tr>
<td>Quadriceps force, N</td>
<td>6.82 ± 1.055</td>
<td>8.84 ± 0.931*</td>
</tr>
<tr>
<td>Quadriceps volume, ml</td>
<td>2.057 ± 0.199</td>
<td>2.189 ± 0.232*</td>
</tr>
<tr>
<td>VL optimal Lf, mm</td>
<td>91 ± 13</td>
<td>87 ± 14</td>
</tr>
<tr>
<td>VL optimal Lf, mm</td>
<td>102 ± 13</td>
<td>103 ± 11</td>
</tr>
<tr>
<td>RF optimal Lf, mm</td>
<td>68 ± 12</td>
<td>72 ± 20</td>
</tr>
<tr>
<td>VM optimal Lf, mm</td>
<td>102 ± 13</td>
<td>104 ± 15</td>
</tr>
<tr>
<td>Quadriceps PCSA, cm²</td>
<td>231.4 ± 25.5</td>
<td>248.1 ± 37.1*</td>
</tr>
<tr>
<td>Tendon mean CSA, mm²</td>
<td>103 ± 2</td>
<td>107 ± 1*</td>
</tr>
<tr>
<td>Tendon stress, MPa</td>
<td>44.0 ± 2.7</td>
<td>42.4 ± 2.6*</td>
</tr>
<tr>
<td>Tendon elongation, mm</td>
<td>4.7 ± 1.0</td>
<td>3.9 ± 1.0</td>
</tr>
<tr>
<td>Tendon strain, %</td>
<td>8.9 ± 1.9</td>
<td>7.2 ± 1.5**</td>
</tr>
<tr>
<td>Tendon stiffness, N/mm</td>
<td>1.864 ± 0.468</td>
<td>2.288 ± 0.546*</td>
</tr>
<tr>
<td>Tendon Young’s modulus, GPa</td>
<td>0.98 ± 0.30</td>
<td>1.16 ± 0.31*</td>
</tr>
</tbody>
</table>

Values are means ± SE. Knee extension MVC, maximal voluntary contraction torque at optimal knee joint angle; quadriceps force, net force of the quadriceps muscle at optimal knee joint angle, corrected for co-activation; VL, vastus lateralis; VI, vastus intermedius; RF, rectus femoris; VM, vastus medialis; Lf, fascicle length; PCSA, physiological CSA. *Significantly different from baseline (P < 0.01).
Muscle strength and size. The 32% increase in MVC torque observed in the present study is either similar (18, 48) or greater than that reported in other studies of similar duration [15–20% (38, 39)]. Differences in training volume and testing modalities (e.g., optimal joint angle) likely explain these discrepancies. The 7% increase in quadriceps PCSA is comparable to the level of muscle size increase typically reported in training studies [5–10% (38, 39)].

Tendon mechanical properties, modulus, and CSA. The stiffness of the patellar tendon increased by 24% after 9 wk of resistance training. This result is in line with the increase in tendon stiffness observed following resistance training in both

Fig. 3. MR scan showing patellar tendon CSA at the site Lt100 before (A) and after (B) training.

Fig. 4. Relationships between changes in quadriceps physiological CSA (PCSA) and changes in patellar tendon mean CSA (A) and CSA at Lt90 (B). For both relationships, $r = -0.64$, $R^2 = 0.41$, and $P < 0.05$.

Fig. 5. Relationships between changes in muscle PCSA and changes in tendon stiffness (A) and Young’s modulus (B). Changes in muscle PCSA were positively correlated to changes in patellar tendon stiffness ($r = 0.68$, $R^2 = 0.46$, $P < 0.01$) and modulus ($r = 0.75$, $R^2 = 0.57$, $P < 0.01$).
young (26, 27) and elderly (43) subjects and is imputable to an increase in Young’s modulus and/or tendon hypertrophy.

In the present study, modulus increased by 20%, suggesting that a large part of the changes in mechanical properties could be ascribed to changes in the material properties of the tendon. Likewise, other authors found that the relative increase in patellar tendon stiffness observed after resistance training mirrored the increase in modulus (27, 43). In contrast, Kongsgaard et al. (26) measured a 19% increase in patellar tendon stiffness after 12 wk of resistance training in young subjects, whereas the concomitant 12% increase in modulus did not reach significance. However, Kongsgaard et al. calculated the modulus by using a single CSA value from a scan on the proximal region of the tendon rather than the mean CSA along the entire tendon, and the discrepant results found by these authors may well be imputable to different calculation methods.

Following resistance training, mean patellar tendon CSA increased by 3.7%. Tendon hypertrophy was heterogeneous along the tendon length: increases in CSA superior to 3% were observed proximally at Lt20 and Lt30 (beneath the patellar insertion) and distally from Lt60 to Lt100 (Fig. 2). However, our analysis revealed that tendon hypertrophy was only significant at Lt20 (5.2%), Lt30 (5.3%), Lt60 (5.0%), Lt90 (4.9%), and Lt100 (5.7%). The pattern of tendon hypertrophy observed in the present study indicates why this phenomenon may have gone undetected in some previous training studies (27, 43), where tendon CSA was calculated as an average of three measurements from regions with little or no hypertrophy.

Similarly to our findings, recent studies have reported loading-induced increases in CSA of the human patellar tendon near the osteotendinous junctions (OTJ) after short- (26) and long-term (12) duration of increased loading. The present results extend previous observations, since CSA increases were also observed between OTJ’s and the tendon mid-length at sites Lt30 (~15 mm distal to the patella apex) and Lt60 (~30 mm proximal to the tibial insertion). Our findings could partly be consistent with the theory of higher tendon-to-bone compressive forces stimulating an increase in extracellular matrix protein synthesis (26). At near-extension knee joint angles, the passive mechanical forces acting across the patellar tendon may be augmented in both muscle and tendon, although in different proportions, in rats subjected to increased loading (21, 22). Allegedly, interindividual variability in the expression of these growth factors could in turn drive variable remodeling in connective tissue and in muscle tendon unit. The identification of such factors is key to the understanding of these relationships and requires further investigation.

On the contrary, there was no positive relationship between the changes in quadriceps PCSA and tendon hypertrophy or the changes in tendon mechanical properties of the tendon. For instance, insulin-like growth factor-I, mechanical growth factor, or transforming growth factor-β1 have been augmented in both muscle and tendon, although in different proportions, in rats subjected to increased loading (21, 22). Allegedly, interindividual variability in the expression of these growth factors could in turn drive variable remodeling in connective tissue and in muscle tendon unit. The identification of such factors is key to the understanding of these relationships and requires further investigation.
stresses would be sustained by primarily incorporating collagen toward changes in the internal structure of the tendon (e.g., collagen fiber packing) rather than toward molecular arrangements leading to an increase of overall tendon CSA. It is interesting to note that both collagen degradation and synthesis are elevated for at least 4 wk of physical training (28, 29) but that a net increase in collagen synthesis is observed after 11 wk (28). Based on the biphasic response of collagen metabolism with training and in line with the above hypothesis, Kjaer (25) recently suggested that the early tendon adaptations were directed toward intrinsic structural changes and that an increase in tendon CSA might occur with the onset of net collagen synthesis. This hypothesis contrasts with cross-sectional studies showing that an increase in tendon stiffness in response to long-term changes in habitual function and loading are accomplished by tendon hypertrophy and not by changes in the tendon’s material (24, 33, 40, 41). Regardless of the exact mechanisms underlying this association, the present results show that tendon hypertrophy has a different relationship to muscle hypertrophy and loading history than that observed between the increase in tendon stiffness and modulus during short-term resistance training. In that, they suggest that tendon hypertrophy may not be directly driven by the functional requirement of muscle-tendon interaction.

Significance of the correlation results. Correlation does not necessarily presume causation, and significant correlations should be considered light of objective parameters (17). A few criteria support the strongest relationships observed in the present study between changes in PCSA and in stiffness/modulus. First of all, these relationships are physiologically coherent: overloading is a common stimulus to increases in both tendon stiffness and muscle size. Statistically, the correlations are robust: the two-tailed analyses reached a significance level of \( P < 0.01 \), and the removal of one possible outlier did not decrease the level of correlation. Note that a larger sample size would probably improve the relatively large confidence interval. Finally, this relationship is in line with another well-established relationship between muscle size and tendon size (and therefore tendon mechanical properties) in the context of allometry related to growth or scaling across species (see Ref. 5 for review). On the other hand, a number of factors suggest that the relationship between muscle PCSA and tendon CSA should be considered with caution. Admittedly, this correlation does not seem physiologically coherent when the theory of tendon scaling and the other relationships found in the present experiment are considered. In addition, the correlation was only observed in one region of the tendon (Lt90) out of the 11 regions investigated. Statistically, the relationship is weaker (\( P < 0.05 \)) than with tendon stiffness/modulus; the removal of one probable outlier cancelled the association between changes in PCSA and mean tendon CSA and weakened the correlation with changes in tendon CSA at Lt90. Consequently, further investigation is required to ascertain the validity of this particular relationship.

Clinical relevance and hypothetical mechanisms. The exact causes of the tendon hypertrophy remain ambiguous. Yet, if one considers that tendon injury results from a failure of adaptation to increased tensile stress, hypothetical mechanisms may be found in the etiology of patellar tendinopathy (34, 45). Kongsgaard et al. (26) highlighted the concurrence of CSA increase and injury near the OTJs and advocated that the stress dissipation resulting from tendon hypertrophy could be seen as a protective adaptation during resistance training. In fact, MRI-based studies indicate that tendinopathies are not restricted to OTJs and that pathological areas cover 10 to >20 mm of the tendon length in proximal tendinopathies (23) and 15 mm in distal tendinopathies (47). The fact that the load-induced hypertrophy observed in the present study took place at a similar anatomical localization as tendinopathy is compatible with the hypothesis of tendon hypertrophy as a protective mechanism against increased stress levels. In addition to load-induced tibial displacement (52), the patella shifts laterally and rotates and tilts during knee movements (36, 46). As a result, changes in orientation of the patellar tendon (1) may cause changes in stress direction and concentration in specific tendon areas or fascicles. Differential axial stresses (13, 20) or internal shear stresses similar to that observed in other tendons (8, 9) could in turn challenge the tendon microstructure, inducing micro-tears. Hence, hypertrophy could result from the tendon repair and/or remodeling processes and, more than dissipating high stresses, could serve the purpose of shielding weaker tendon regions.

Conclusion. This study has shown that patellar tendon hypertrophy is prominent near but not limited to OTJs. The positive relationship between increases in tendon stiffness and modulus and muscle PCSA, but not with maximal force, suggests that changes in tendon mechanical and material properties to overloading are closely related to the loading history rather than to the increase in the maximal stress that can be exerted on the tendon. The possible inverse correlations between changes in tendon mean CSA and in muscle PCSA, or lack thereof, implies that the mechanisms underlying tendon hypertrophy during short-term chronic loading are different to those determining tendon allometry related to development.

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REFERENCES


