
---

Dette er siste tekst-versjon av artikkelen, og den kan inneholde små forskjeller fra forlagets pdf-versjon. Forlagets pdf-versjon finner du på bjsm.bmj.com: [http://dx.doi.org/10.1136/bjsports-2013-092275](http://dx.doi.org/10.1136/bjsports-2013-092275)

---

This is the final text version of the article, and it may contain minor differences from the journal's pdf version. The original publication is available at bjsm.bmj.com: [http://dx.doi.org/10.1136/bjsports-2013-092275](http://dx.doi.org/10.1136/bjsports-2013-092275)
Mechanical properties of the patellar tendon in elite volleyball players with and without patellar tendinopathy


Norwegian School of Sport Sciences

Corresponding author:
Christian Helland
Department of Physical Performance & Oslo Sports Trauma Research Center, Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway

Address: Norwegian School of Sport Sciences, P.O Box 4014 – Ullevål stadion, 0806 Oslo, Norway
Fax: +47 23 26 23 07
Telephone: +47 90 11 34 31
E-mail: christian.k.helland@gmail.com

Co-authors:
Jens Bojsen-Møller: Department of Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway
Truls Raastad: Department of Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway
Olivier R Seynnes: Department of Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway
Marie M. Moltubakk: Department of Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway
Vidar Jakobsen: Department of Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway
Håvard Visnes: Oslo Sports Trauma Research Center, Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway
Roald Bahr: Oslo Sports Trauma Research Center, Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway

Key words: Patellar tendinopathy, jump performance, volleyball, tendon properties
ABSTRACT

Background/Aim: Although differences in mechanical properties between symptomatic and healthy tendons have been observed for the Achilles tendon, the impact of tendinopathy on patellar tendon mechanics is not fully documented. The aim of the present case-control study was to assess tendon mechanical properties and jump performance in elite athletes with and without patellar tendinopathy.

Methods: We identified 17 male volleyball players with patellar tendinopathy and 18 healthy matched controls from a 5-year prospective cohort study on junior elite volleyball players. Outcome variables included three measures of maximal vertical jump performance and ultrasound-based assessments of patellar tendon cross-sectional area, stiffness and Young’s modulus.

Results: The proximal cross-sectional area of the patellar tendon was significantly larger in the tendinopathic group (133 ± 11 vs. 112 ± 9 mm², respectively, p<0.001). Pathological tendons presented lower stiffness (2254 ± 280 vs. 2826 ± 603 N·mm⁻¹, respectively p=0.006) and Young’s modulus (0.99 ± 0.16 vs. 1.17 ± 0.25 GPa, respectively p=0.04) than healthy tendons. However, the difference between counter movement jump height and squat jump height (3.4 ± 2.2 vs. 1.2 ± 1.5 cm, p=0.005) were significantly higher in the tendinopathic group compared to the control group.

Conclusion: Patellar tendinopathy is associated with a decrease in tendon mechanical and material properties in elite athletes subjected to a high volume of jumping activity. However, tendinopathic volleyball players have a better ability to utilize the stretch shortening cycle when jumping compared to healthy counterparts.

INTRODUCTION

Patellar tendinopathy (jumper’s knee) is a common overuse injury in sports with great demands on rapid force development in the leg extensors. An estimated prevalence as high as 45% is reported in elite volleyball players,¹ and the condition is known to be a contributing factor to early retirement.² Several new treatment strategies exist, e.g. eccentric loading, heavy slow resistance training, sclerosing injections and injections of platelet-rich plasma.³⁻⁶ Nonetheless, patients rarely recover completely, and the understanding of the underlying mechanisms for efficacy of treatment is limited.³⁻⁶⁻⁷

Training and match exposure,⁸ competing at an elite level¹ compared to a non-elite level⁹ and sex (male being more prone to disorder)¹⁰ are significant risk factors for developing patellar tendinopathy. Incidentally, there has been shown in both a case-control study¹⁰ and a cross-sectional study¹¹ that jump performance is superior in athletes with patellar tendinopathy compared to healthy athletes (the jumper’s knee paradox). These findings have recently been corroborated in a 5-year prospective cohort study, where adolescent volleyball players who went on to develop tendinopathy jumped higher in a counter movement jump (CMJ) compared to players who remained healthy.¹² Yet, a higher jumping ability in individuals presenting a degenerative disorder in their tendon seems counterintuitive, in regard to the positive correlations between jump performance and the stiffness of healthy patellar tendons.¹³
Tendinopathy is related to structural tissue changes, such as increased type III collagen content, which may adversely influence tendon mechanical and material properties. In patients with Achilles tendinopathy, greater strain, lower stiffness and Young’s modulus, and a greater cross-sectional area (CSA) have been observed in comparison to their healthy counterparts. However, such differences were not detected in the patellar tendon of recreationally active patients or in badminton players with tendinopathy. To date, there is no data on tendon properties in patients involved in sports with a high volume of jumping activity (e.g. volleyball).

Therefore, the aim of this study was to compare mechanical and material properties of the patellar tendon between male volleyball players with tendinopathy and a group of matched controls. To minimize recruitment bias, players were included from a 5-year prospective cohort study on young, elite volleyball players. We also wanted to investigate whether the greater jump height observed in CMJ in players with tendinopathy is present in a more sport-specific spike jump test. We hypothesized that, in spite of greater jumping ability, the patellar tendon of elite volleyball players with tendinopathy has lower stiffness and modulus than healthy controls.

MATERIAL AND METHODS

Recruitment and inclusion
The participants were former students at Toppvolley Norge, a boarding school which combines an elite volleyball development program with a 3-year baccalaureate degree. They were recruited from a 5-year prospective cohort study, where 22 of 69 male volleyball players developed jumper’s knee (figure 1). To be included, they also had to be competing at the national elite level at the time of inclusion for the present study. Among the 22 players with jumper’s knee, 20 were still playing volleyball and we matched them with 20 players from a group of 46 healthy males with respect to age, height, weight and year of inclusion into the prospective cohort study. In the jumper’s knee group, 3 players were excluded; 2 declined the invitation to participate and 1 no longer played at the elite level. In the control group, 2 no longer played at elite level. Thus, we included 17 players with a history of jumper’s knee and 18 healthy controls for further measurements. Due to the higher prevalence of patellar tendinopathy in males compared to females, we only recruited male players.

Ethics
The project was approved by the Regional Committee for Research Ethics and participants provided written informed consent.

Player groups
In the original cohort study, tendinopathy was diagnosed based on the following criteria: 1) tenderness on palpation of the proximal patellar tendon or the distal part of the quadriceps tendon insertion, 2) pain during volleyball training, consistent with the tender area and, 3) hypoechoic areas and increased tendon thickness corresponding to the painful area examined by ultrasonography. To be classified as jumper’s knee, symptoms had to have persisted for at least 12 weeks and represent a substantial problem to the player. Three of the originally healthy controls subsequently developed patellar tendinopathy, between the time of the cohort study and inclusion into the present study, and their results are presented separately as the control-patellar tendinopathy group. Of the 17 players with tendinopathy, 1 was completely free of symptoms at the time of inclusion into the present study. Thus, we
divided the participants into 4 groups based on their knee status at the time of the study (figure 1): 1) Participants with current patellar tendinopathy (n=13), 2) participants with current quadriceps tendinopathy (n=3), 3) healthy controls (n=15) and 4) controls with current patellar tendinopathy (n=3). Participant characteristics are shown in tables 1 and 2.

**Table 1** Participant characteristics for patellar tendinopathy group, quadriceps tendinopathy group, control group and control-patellar tendinopathy group.

<table>
<thead>
<tr>
<th></th>
<th>Patellar tendinopathy (n=13)</th>
<th>Control (n=15)</th>
<th>Quadriceps tendinopathy (n=3)</th>
<th>Control-patellar tendinopathy (n=3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>20 (19 to 21)</td>
<td>21 (18 to 22)</td>
<td>22 (20 to 24)</td>
<td>21 (20 to 22)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>189 (185 to 192)</td>
<td>190 (188 to 191)</td>
<td>194 (190 to 197)</td>
<td>194 (190 to 198)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>81 (78 to 85)</td>
<td>85 (81 to 89)</td>
<td>86 (84 to 88)</td>
<td>89 (82 to 95)</td>
</tr>
<tr>
<td>Patellar tendon length (mm)</td>
<td>53 (51 to 56)</td>
<td>52 (49 to 54)</td>
<td>52 (50 to 54)</td>
<td>56 (52 to 60)</td>
</tr>
<tr>
<td>VISAp score</td>
<td>76 (64 to 87)</td>
<td>98 (96 to 100)</td>
<td>86 (77 to 94)</td>
<td>73 (47 to 98)</td>
</tr>
</tbody>
</table>

*Significantly different from control group, P<0.05

**Table 2** Number of participants in the volleyball-specific playing positions.

<table>
<thead>
<tr>
<th>Playing position</th>
<th>Patellar tendinopathy (n=13)</th>
<th>Quadriceps tendinopathy (n=3)</th>
<th>Control (n=15)</th>
<th>Control-patellar tendinopathy (n=3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outside hitter</td>
<td>5</td>
<td>2</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>Setter</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Middle blocker</td>
<td>4</td>
<td>-</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td>Libero</td>
<td>1</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
</tbody>
</table>

**Experimental procedures**
Participants underwent familiarization with all relevant tests at least 24 h prior to data collection. Data were collected in the same order as described below and all data collection was conducted by the same investigator throughout the study period (October 2011-June 2012), at the laboratory at Norwegian School of Sport Sciences and the laboratory at the Norwegian Olympic Sports Center (Olympiatoppen) in Oslo. Before data collection, participants did a standardized warm-up of 5-10 min sub-maximal treadmill running with increasing speed, starting at a moderate jogging speed individualized for each participant followed by 3 sub-maximal 40 m sprints separated by approximately 3 min of rest.

**Jump performance**
Participants performed squat jumps (SJ) and CMJ (sampling rate 2 kHz) with arms akimbo on a force platform (AMTI OR6-5-1, AMTI, Watertown, MA, USA). For SJ, participants were instructed to squat until their knee joint angle reached approximately 90°, while the trunk was maintained in an upright position. One to 2 seconds after reaching this position, the investigator (CH) gave the signal to perform a maximal vertical jump. The highest jump out of 3 to 5 attempts was used for further analysis. CMJ was conducted similarly to SJ, although hip, knee and ankle flexions were performed from an upright position to a self-determined depth, followed by an immediate maximal vertical jump. Thereafter, the participants conducted a series (3-5) of spike jumps under a custom-made pre-measured frame: participants were instructed to use arm swing and a classic, volleyball-specific, three-step
approach to jump vertically, touching an overhead target (a red plastic wire) with their dominant hand. The target was incrementally raised by 2 to 5 cm between jumps until maximum jump height was reached. Participants had a resting period of 1-3 minutes between each jump, and 3-5 minutes between each jump type. Finally, we measured standing reach height with a tape measure from the floor to the fingertip of the dominant hand. Participants had a rest period of approximately 15 minutes before proceeding with measurements of tendon properties and peak rate of force development.

Tendon properties and peak rate of force development
For measurements of tendon properties and maximal voluntary knee extensor contractions (MVC), the participants were secured on an isometric knee extension chair (Knee extension, Gym2000, Geithus, Norway) instrumented with a load cell (U2A, Hottinger Baldwin Messtechnik GmbH, Darmstadt, Germany). Since tendinopathy symptoms were observed on the right leg of a majority of players (except 3 cases), all tests were conducted on this side in the control group. Knee and hip angle were set at 90° (figure 2).

To assess maximal rate of force development, participants performed 3 MVCs of the knee extensor muscles, with the instruction to reach their maximum force as fast as possible. Data were stored on a separate computer with a sampling rate at 1000 Hz (LabVIEW 8.2, National Instruments Corporation, Austin, TE, USA). Contractions were separated by a 60 s resting period. Subsequently, 2 MVC knee flexions were performed, to enable assessment of hamstring co-activation. Tendon mechanical properties were assessed by measuring the elongation of the patellar tendon during isometric ramp contractions. An ultrasound linear array transducer (50 mm, 5-12 MHz HD11XE, Phillips, Bothell, WA, USA) was attached anteriorly to the patellar tendon with a custom-made device (figure 2), and the participants performed at least 3 maximal ramp contractions while ultrasound video sequences were recorded. A visual feedback displayed in front of the participants ensured that all ramp contractions were performed at a constant loading rate (100 N·s⁻¹). Trials were discarded when the force trace deviated from the required linear pattern upon visual inspection. To limit the influence of creep on tendon loading, 3 sub-maximal pre-conditioning contractions were performed a few seconds prior to each ramp contraction.

Electromyographic activity of the biceps femoris muscle was recorded wirelessly (TeleMyo 2400 G2 Telemetry System, Noraxon Inc., Scottsdale, AZ, USA) during isometric knee extensions and flexions to estimate the co-activation level. The skin over the biceps femoris was shaved and rubbed with alcohol to reduce skin impedance. Self-adhesive surface electrodes (Ambu, Blue Sensor M, Ballerup, Denmark) were attached over the muscle belly with an inter-electrode distance of 2 cm. A reference electrode was placed on the patella. A wireless receiver (Mini-Receiver for TeleMyo G2, Noraxon Inc., Scottsdale, AZ, USA) synchronized force, EMG and ultrasound video.

Tendon and muscle morphology
Cross-sectional ultrasound images of the proximal, middle and distal parts of the tendon were recorded at rest and with 90° of knee flexion. Tendon CSA was assessed with video and image analysis software (ImageJ 1.45s, National Institute of Health, Austin, TE, USA). Tendon length was measured in the sagittal plane from ultrasound images, as the distance between the patellar apex and the tibial tuberosity.

Tendinopathy diagnostic
We palpated the patellar and quadriceps tendon insertions and obtained diagnostic ultrasound images of the patellar tendon, where hypoechoic areas and increased tendon thickness were noted. Body height was measured, and participants completed a Norwegian translation of the Victorian Institute of Sport Assessment questionnaire for patellar tendinopathy (VISAp), without any instructions from the investigator. The VISAp form is a commonly used questionnaire for measuring pain and function in the patellar or quadriceps tendons, where 100 represents a perfectly healthy knee, and 0 a completely non-functional, painful knee.

**Data Analysis**

Jump performance
SJ and CMJ results were stored and analyzed on a separate computer in a custom-made Matlab based software (Biojump 2.4, Biomekanikk AS, Oslo, Norway). Jump height was calculated from the time integration of the vertical force (Fz). The highest jumps of both SJ and CMJ were used for further analysis. The difference between CMJ and SJ was calculated, to look at the participants ability to utilize the stretch shortening cycle (SSC).

Spike jump height was calculated as the difference between standing reach height and jumping reach height.

Tendon properties
Tendon elongation was measured with video analysis software (Tracker Video Analysis and Modeling Tool 4.62, Open Source Physics, Aptos CA, USA). The coordinates of the patella apex and the edge of the tibial plateau were tracked during the ramp contractions and changes in the distance between these references were assimilated to changes in tendon length. Tendon force was calculated from the net force applied to the load cell (sampling rate 1500 Hz) corrected for antagonist co-activation, external moment arm and internal moment arm. Femur length was calculated from body height, and was used to estimate internal knee joint moment arm. All EMG signals were quantified by calculating the root mean square over a 50-ms time period. A linear relation between force and EMG amplitude was assumed to correct for the hamstrings co-activation. Hence, patellar tendon force \( F_t \) was calculated from the following equation:

\[
F_t = \frac{(F_q + F_h) \cdot M_e}{M_i},
\]

where \( F_q \) is force recorded at the load cell, \( F_h \) is estimated hamstring co-activation force, \( M_e \) and \( M_i \) are external and internal moment arm, respectively.

Force-elongation curves were fitted with a 2\(^{nd}\) or 3\(^{rd}\) order polynomial, with \( R^2 > 0.95 \), and stiffness was calculated in the final 10% of the linear part of the curve. Absolute stiffness, measured at maximal force level of each individual, and relative stiffness, measured at the highest common force level (4000 N) were calculated. We estimated Young’s modulus from both absolute and relative stiffness by multiplying stiffness with the ratio between tendon length and middle CSA. One participant was excluded from the analysis of relative stiffness and relative Young’s modulus because his maximal force did not reach 4000 N. Finally, stress was obtained by dividing maximal tendon force by middle CSA, and strain by dividing maximal tendon elongation by resting tendon length.

Rate of force development
Rate of force development data was smoothed with running average (20 ms) and peak rate of force development (\( N \cdot s^{-1} \)) calculations were derived between 10 ms (10\(^{th}\) ms – 1\(^{st}\) ms).
Statistical Analyses
We analyzed differences between-groups with a two tailed unpaired t-test, where an alpha level of 0.05 was set as a significant difference. We analyzed tendon CSA with a two-way ANOVA test, and with a Bonferoni post hoc test correction in case of significant interaction effects. All statistical analyses were conducted in Excel (Microsoft Office Excel 2007 Inc., Microsoft) or GraphPad Prism (GraphPad Prism 5.00, GraphPad Software, San Diego, CA, USA). Unless otherwise is noted, all analysis is done on the patellar tendinopathy group (n=13) and the control group (n=15). Data from the control-patellar tendinopathy group and the quadriceps tendinopathy group are presented separately to show the complete dataset. Data are presented as means with 95% confidence intervals (CI) unless otherwise noted.

RESULTS
Mechanical and material properties
Absolute and relative patellar tendon stiffness and Young’s modulus was significantly lower in the patellar tendinopathy group compared to controls (32%, 20%, 29% and 15%, respectively) (figure 3). Stiffness and Young’s modulus for formerly healthy participants with current patellar tendinopathy were similar to symptomatic participants for both relative and absolute values. Participants with quadriceps tendinopathy had similar values compared to participants in the control group for both relative and absolute stiffness and Young’s modulus. Force, stiffness, stress, strain, elongation and Young’s modulus values for the patellar tendon for the patellar tendinopathy and control group are shown in table 3.

Table 3 Mechanical and material properties for the patellar tendinopathy group and control groups.

<table>
<thead>
<tr>
<th></th>
<th>Patellar tendinopathy (n=13)</th>
<th>Control (n=15)</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak force (N)</td>
<td>5298 (4593 to 6002)</td>
<td>5809(5195 to 6423)</td>
<td>511.4 (-464.2 to 1487)</td>
<td>0.29</td>
</tr>
<tr>
<td>Normalized peak force (N/BW0.67)</td>
<td>278 (244 to 313)</td>
<td>295 (268 to 322)</td>
<td>16.8 (-29 to 62)</td>
<td>0.45</td>
</tr>
<tr>
<td>Stiffness-absolute (N-mm⁻¹)</td>
<td>2504 (2209 to 2800)</td>
<td>3684 (3082 to 4287)</td>
<td>1180 (441 to 1919)</td>
<td>0.003</td>
</tr>
<tr>
<td>Stiffness-relative (N-mm⁻¹)</td>
<td>2254 (2096 to 2412)</td>
<td>2826 (2521 to 3131)</td>
<td>571.6 (182 to 961)</td>
<td>0.006</td>
</tr>
<tr>
<td>Stress (Mpa)</td>
<td>43 (37 to 50)</td>
<td>47 (43 to 51)</td>
<td>3.4 (-5 to 11)</td>
<td>0.39</td>
</tr>
<tr>
<td>Strain (%)</td>
<td>7 (6 to 7)</td>
<td>7 (6 to 8)</td>
<td>0.2 (-0.73 to 1.07)</td>
<td>0.70</td>
</tr>
<tr>
<td>Elongation (mm)</td>
<td>4 (3 to 4)</td>
<td>4 (3 to 4)</td>
<td>0.04 (-0.48 to 0.57)</td>
<td>0.87</td>
</tr>
<tr>
<td>Young’s modulus-absolute (Gpa)</td>
<td>1.09 (0.93 to 1.25)</td>
<td>1.53 (1.28 to 1.77)</td>
<td>0.4 (0.11 to 0.75)</td>
<td>0.01</td>
</tr>
<tr>
<td>Young’s modulus-relative (Gpa)</td>
<td>0.99 (0.89 to 1.08)</td>
<td>1.17 (1.04 to 1.30)</td>
<td>0.2 (0.01 to 0.36)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Stiffness-absolute and Young’s modulus-absolute are absolute values calculated from peak force. Stiffness-relative and Young’s modulus-relative are values calculated from the greatest common force level at 4000 N (n=12).

Tendon proximal CSA was significantly 19% larger in the patellar tendinopathy group compared to controls (figure 4).
Jump performance
The patellar tendinopathy group had a significantly greater (183%) CMJ-SJ difference compared to the controls (figure 5). There were no differences in spike jump, SJ and CMJ performance between the two groups (table 4); however, when data from the quadriceps tendinopathy group (n=3) were combined with those of the patellar tendinopathy group (n=16), there was a significant difference in spike jump height (7 cm, 0.07 to 14) and spike jump reach (6 cm, 0.5 to 11) compared to controls.

Table 4 Jump performance for the patellar tendinopathy and control groups.

<table>
<thead>
<tr>
<th></th>
<th>Patellar tendinopathy (n=13)</th>
<th>Control (n=15)</th>
<th>Difference</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standing reach (cm)</td>
<td>249 (244 to 254)</td>
<td>249 (246 to 252)</td>
<td>0.3 (-5.9 to 6.5)</td>
<td>0.92</td>
</tr>
<tr>
<td>Spike jump reach (cm)</td>
<td>329 (323 to 334)</td>
<td>323 (319 to 327)</td>
<td>5.8 (-1.5 to 13.1)</td>
<td>0.11</td>
</tr>
<tr>
<td>Spike jump height (cm)</td>
<td>80 (75 to 84)</td>
<td>74 (71 to 78)</td>
<td>5.5 (-0.3 to 11.3)</td>
<td>0.06</td>
</tr>
<tr>
<td>SJ (cm)</td>
<td>37 (35 to 40)</td>
<td>38 (36 to 39)</td>
<td>-0.1 (-3.5 to 3.3)</td>
<td>0.94</td>
</tr>
<tr>
<td>CMJ (cm)</td>
<td>41 (37 to 44)</td>
<td>39 (37 to 41)</td>
<td>2.1 (-1.9 to 6.0)</td>
<td>0.29</td>
</tr>
<tr>
<td>CMJ-SJ difference (cm)</td>
<td>3.4 (2 to 5)</td>
<td>1.2 (0.5 to 2.0)</td>
<td>2.2 (0.7 to 3.6)</td>
<td>0.004</td>
</tr>
</tbody>
</table>

SJ: Squat jump, CMJ: Counter movement jump

Isometric rate of force development
There were no significant differences in the peak rate of force development between the patellar tendinopathy and control group (figure 6).

DISCUSSION
This is the first report of lower stiffness and Young’s modulus in symptomatic patellar tendons. In line with previous findings, the present findings confirm the larger proximal cross sectional area of the patellar tendon with tendinopathy. These findings indicate that chronic tendinopathy is associated with altered morphological, mechanical and material properties.

Our findings support previous observations on the Achilles tendon with respect to CSA, stiffness and Young’s modulus, but are in contrast to previous findings on the patellar tendon. Yet, a trend towards lower stiffness (8%) had been reported when comparing a tendinopathic group (n=8) to a age and activity matched group of healthy (n=9) recreational athletes. It should also be noted that the present protocol differs from previous studies on patellar tendinopathies, where the age range of patients was as large as 18-50 yrs, and where comparisons were made within individuals, between tendinopathic and healthy tendons. Age and training type have previously been shown to potentially influence the properties of the patellar tendon. These parameters could explain the differences between the present findings and earlier studies, where they may have induced a larger variability. In contrast, we examined a group of elite athletes practicing the same physical activity, within a more homogenous age group, and compared these injured individuals to a healthy control group.

Although we found a lower stiffness in symptomatic tendons, tendon proximal CSA was larger in these tendons compared to controls, in accordance with previous findings. Theoretically, a greater CSA would have led to a larger stiffness if the tendon composition were the same. However, ultrasound examination revealed that the thickened area was
structurally abnormal, with substantial collagen disarray and hypoechoic areas reflecting increased ground substance. Therefore, the present data suggest that the tissue degeneration impacts both mechanical and material properties of the patellar tendon.

One strength of this study lies in the fact that all participants were recruited from a 5-year cohort study, following a population of adolescent athletes prospectively to include 17 out of 19 patients and 18 out of 18 controls among those eligible (still competing at a high level), unlike other studies where bias may have occurred in the selection process. However, tendon properties were not measured at baseline. Therefore, we can only speculate as to whether the differences in mechanical and material properties observed are the cause, or the consequence of chronic tendinopathy.

In line with previous findings, symptomatic players had an overall better jump performance compared to matched controls, as evidenced by a greater difference between CMJ and SJ height and by the sport-specific spike jump. The difference between CMJ and SJ has previously been suggested to reflect an athletes ability to utilize the SSC. In the concentric SJ however, no differences between the two groups was observed. Consequently, players with patellar tendinopathy seem to have an increased capacity to take advantage of the elastic components of the extensor apparatus by utilizing their SSC more efficiently, compared to healthy players.

Yet the higher jumping capability and the lower tendon stiffness of the present tendinopathic patients contrasts with the positive correlation found previously between these variables in male cyclists and volleyball players. Such relations between jump performance (SJ, CMJ and spike jump) and tendon stiffness or pathology (VISAp) were not observed in the current study, for neither the control nor the patellar tendonopathy group. The reasons for this discrepancy may include methodological differences e.g. different tendon testing method and the different sample of participants (volleyball players and cyclists vs. volleyball players only). Nevertheless, in light of these previous findings a causal link between the patellar tendon properties and the higher jumping performance of tendinopathic volleyball players would seem highly speculative. Instead, based on previous literature suggesting that tendinopathy may result from inappropriate or excessive tendon stress, or different jump/landing strategies, players who jump higher during practice and match may be more susceptible to develop patellar tendinopathy. This is supported by the 5-year cohort study from which the present participants were recruited from, in which individuals with a greater jump height developed tendinopathy.

**CONCLUSION**

This study provides evidence of differences in tendon properties between symptomatic and asymptomatic elite volleyball players. Stiffness and Young’s modulus were lower in players with patellar tendinopathy, in spite of a greater difference between CMJ and SJ height.

**What are the new findings:**

- Despite a larger proximal CSA, the patellar tendon of volleyball players with tendinopathy displayed lower stiffness and Young’s modulus than healthy controls.
- Players with patellar tendinopathy have a better jumping ability in tasks implying storage and release of elastic energy.
- Symptomatic players (patellar and quadriceps tendinopathy) have a greater spike jump height than healthy players.
How might it impact on clinical practice in the near future:

- This research shows that there are substantial differences in tendon and muscle properties between players with patellar tendinopathy and healthy players, suggesting that it may be possible to use these differences to identify players at risk, and consequently to adapt their training programs to prevent the development of tendinopathy.

Acknowledgement
The Oslo Sports Trauma Research Center has been established at the Norwegian School of Sport Sciences through generous grants from the Royal Norwegian Ministry of Culture, the South-Eastern Norway Regional Health Authority, the International Olympic Committee, the Norwegian Olympic Committee & Confederation of Sport, and Norsk Tipping AS. We thank the Norwegian Olympic Training Center and all the volleyball players participated in this study.

Competing interests
None.

Funding
This research received no specific funding.

Contributorship statement:
Christian Helland: manuscript writer, concept and design, data acquisition, data analysis and interpretation.

Jens Bojsen-Møller: manuscript review/revision, concept and design, data analysis and interpretation.

Truls Raastad: manuscript review/revision, concept and design, data analysis and interpretation.

Olivier R. Seynnes: manuscript review/revision, concept and design, data analysis and interpretation.

Marie M. Moltubakk: manuscript review/revision, concept and design, and data acquisition.

Vidar Jakobsen: manuscript review/revision, concept and design, and data acquisition.

Håvard Visnes: manuscript review/revision, concept and design, and data interpretation.

Roald Bahr: manuscript review/revision, concept and design, data analysis and interpretation.
REFERENCES


Figure 1 Flowchart depicting the recruitment and inclusion of participants into the study.
Figure 2 Measurements of tendon elongation and tendon force in an isometric knee extension chair, with a fixed hip and knee angle at 90°. The leg cuff is attached to the lower leg and the ultrasound probe firmly attached to the patellar tendon.

Figure 3 Stiffness (A and B) and Young’s modulus (C and D) for the patellar tendinopathy group (n=12 and n=13 for the relative and absolute values, respectively), control group (n=15), formerly healthy participants with current patellar tendinopathy (n=3) and the quadriceps tendinopathy group (n=3). Values are presented in both relative (A and C) and absolute (B and D) values. The horizontal lines represent the group means.
Figure 4 Tendon cross-sectional area for the patellar tendinopathy group (open figures; n=13) and control group (closed figures; n=15 (distal CSA, n=13)) at the proximal, middle and distal part of the tendon. Mean ± SD. *Significantly different from control, P<0.001. When comparing middle CSA with the same controls used for distal CSA (n=13) to patellar tendinopathy group (n=13), no significant difference was found (p=0.92).

Figure 5 SJ height (A), CMJ height (B), CMJ-SJ difference (C) and spike jump height (D) for the patellar tendinopathy group (n=13), control group (n=15), formerly healthy participants with current patellar tendinopathy (n=3) and quadriceps tendinopathy group (n=3). Horizontal lines represent the group means.
Figure 6 Isometric knee extension peak rate of force development (RFDpeak) for the patellar tendinopathy group (n=13), control group (n=15), formerly healthy participants with current patellar tendinopathy (n=3) and quadriceps tendinopathy group (n=3). Horizontal lines represent group means.