Ultrasound characteristics of the patellar and quadriceps tendons among young elite athletes

H. Visnes1,2, A. Tegnander1,3, R. Bahr1

1Oslo Sports Trauma Research Center, Oslo, Norway, 2Kysthospitallet i Hagevik, Department of Orthopaedic Surgery, Haukeland University Hospital, Bergen, Norway, 3Teres Rosenborg, Trondheim, Norway

Corresponding author: Håvard Visnes, MD, PT, Oslo Sports Trauma Research Center, PO Box 4014 – Ullevål stadion, 0806 Oslo, Norway. Tel: +47 23 26 23 67, Fax: +47 23 26 23 07, E-mail: haavard.visnes@helse-bergen.no

Accepted for publication 13 January 2014

Tendons adapt in response to sports-specific loading, but sometimes develop tendinopathy. If the presence of ultrasound changes like hypoechoic areas and neovascularization in asymptomatic tendons precede (and predict) future tendon problems is unknown. The aim of this prospective cohort study was to investigate the relationship between the development of ultrasound changes in the patellar and quadriceps tendons and symptoms of jumper’s knee, as well to examine the medium-term effects of intensive training on tendon thickness among adolescent athletes. Elite junior volleyball athletes were followed with semi-annual ultrasound and clinical examinations (average follow-up: 1.7 years).

Of the 141 asymptomatic athletes included, 22 athletes (35 patellar tendons) developed jumper’s knee. In a multivariate logistic regression analysis, a baseline finding of a hypoechoic tendon area (odds ratio 3.3, 95% confidence interval 1.1 to 9.2) increased the risk of developing symptoms of jumper’s knee. Patellar tendon thickness among healthy athletes did not change (Wilk’s lambda, \( P = 0.07 \)) while quadriceps tendon thickness increased (\( P = 0.001 \)).

In conclusion, ultrasound changes at baseline were risk factors for developing symptoms of jumper’s knee. Also, among healthy athletes, we observed a 7–11% increase in quadriceps tendon thickness, while there was no increase in patellar tendon thickness.

Tendons adapt to mechanical loading, albeit not always adequately. The precise links between tendon metabolic response to increased loading and the changes observed at the macroscopic level remain elusive, but markers of collagen synthesis are increased in peritendinous tissue for 2–3 days after an acute training bout and remain elevated with prolonged training (Langberg et al., 1999, 2001). Increased tendon stiffness is observed in response to short-term resistance training (6–14 weeks) (Kongsgaard et al., 2007; Kubo et al., 2009, 2010; Seynnes et al., 2009; Malliaras et al., 2013). However, whether this increase is a result of tendon hypertrophy, as stiffness is directly dependent on tendon cross-sectional area (CSA), or of changes in the material properties (modulus), or a combination of these, is not fully understood (Heinemeier & Kjaer, 2011). Cross-sectional studies indicate that tendon CSA is larger in trained compared with sedentary tendons, (Kongsgaard et al., 2005; Couppe et al., 2008) while evidence from short-term training studies is conflicting (Kongsgaard et al., 2007; Kubo et al., 2009, 2010; Seynnes et al., 2009; Malliaras et al., 2013). Tendon structural changes from childhood to adulthood are not well investigated, and there is a need for prospective studies examining the response to intensive training during adolescence.

What is clear is that training volume and match exposure are major risk factors for developing symptoms of jumper’s knee, and that this condition is common in athletes from sports characterized by high demands on the tendon, such as volleyball, basketball and athletics (Lian et al., 2005; Visnes & Bahr, 2012). In many athletes with symptoms of jumper’s knee, ultrasound or magnetic resonance imaging (MRI) of the painful tendons will reveal morphological abnormalities, typically localized tendon thickening with hypoechoic areas and increased vascularity (Cook et al., 2004; Comin et al., 2013). These changes are usually seen in the deep proximal part. However, as early as in 1996, cross-sectional studies by Khan et al. (1996) and Lian et al. (1996) revealed that this is not always the case. They observed that not only were there symptomatic tendons with normal tendon morphology on ultrasound examination, but also that asymptomatic tendons with tendon thickening and hypoechoic areas were common findings which have been reproduced in subsequent cross-sectional studies (Cook et al., 1998, 2000a; Warden et al., 2007). However, from these studies it is not clear whether the presence of ultrasound changes in asymptomatic tendons precede (and predict) future tendon problems. Longitudinal studies are therefore needed, but...
have so far produced conflicting results (Cook et al., 2001; Fredberg & Bolvig, 2002; Gisslen & Alfredson, 2005; Gisslen et al., 2007; Fredberg et al., 2008; Comin et al., 2013; Giombini et al., 2013).

Thus, the aims of this prospective cohort study were to investigate the relationship between the development of ultrasound changes in the patellar and quadriceps tendons and symptoms of jumper’s knee, as well to examine the medium-term effects of intensive loading on tendon thickness among adolescent athletes.

Methods
Subjects and setting
We recruited participants for this prospective cohort study among players entering the Toppvolley Norge (TVN) program. TVN is located in Sand, Norway and combines an elite volleyball training program with a 3-year senior high school boarding school program. The recruitment process is shown in Fig. 1. Students enrolled at the age of 15–16 years, and they were expected to complete 3 years for a college-entry baccalaureate degree. Some students entered the program in the second or third year (Fig. 2). TVN aimed to recruit the most talented junior volleyball players in Norway, and the athletes represented the school and their home clubs in the Norwegian national leagues at various levels.

We began the recruitment process when school started with an information meeting every autumn. Potential participants were also informed in writing before their written consent was obtained, and also that of their parents if the athlete was younger than 18 years old. Athletes were eligible for participation in the study as long as they attended TVN. The study was approved by the Regional Committee for Medical and Health Research Ethics and the Norwegian Social Science Data Services.

Risk factors
Ultrasound examination
Every school year started with an ultrasound examination within the first weeks and then again every 6 months for as long as students remained at TVN. An experienced orthopedic surgeon with extensive musculoskeletal ultrasound training (AT), who was blinded to the clinical history, carried out all ultrasound examinations during the 4-year study period. Two ultrasound machines with similar specifications were used for all examinations over the study period (GE Logiq e, 12L-RS probe and GE Logic Book XP, 12L-RS probe, GE Vingmed ultrasound AS, Horten, Norway). Recent studies have shown that intratendinous flow can be increased during exercise (Malliaras et al., 2008; Koenig et al., 2010), and none of the athletes came straight from training, but we had no information about their activity during the previous 24 h. Ultrasound examination of both the patellar and quadriceps tendon was carried out with the patient supine, with the knee in slight flexion (20°). The quadriceps muscles were relaxed to avoid underestimating color Doppler flow (Koenig et al., 2007). Tendon thickness was measured in the proximal, mid and distal part of the tendon. Any pathologic changes in the tendons were registered on a standard form. All clearly defined hypoechoic areas seen in both longitudinal and transverse ultrasound scans were registered as pathologic, regardless of the size of the area. An example with ultrasound scans of the same tendon at inclusion, at the time of diagnosis (after symptom debut) and the final examination before leaving school is shown in Fig. 2. Color Doppler was used to diagnose increased blood flow within the tendon, which has been termed neovascularization (Ohberg et al., 2001). The color Doppler gain was set just below the level that produced random noise in the image (Boesen et al., 2012), and the Doppler settings were the same for all examinations. To our knowledge, no technology existed to reliably quantify flow in the tendon when the study started. Therefore, we used the same semiquantitative method that has been used in previous studies (no flow = 0; flow outside the tendon = 1; one or two vessels inside the tendon = 2; and multiple vessels inside the tendon = 3) (Hoksrud et al., 2006; Gisslen et al., 2007). For subsequent data analyses, we defined neovascularization as the presence of vessels inside the tendon (2 and 3).

Training volume
Training volume was recorded prospectively on a weekly basis. We organized the registration on an individual basis through a web-based weekly training diary. Data were only collected during the 10-month school year, and the method has been described in detail previously (Visnes & Bahr, 2012).

Diagnosis
As the aim of the study was to correlate ultrasound changes in the patellar/quadriceps tendons to symptoms of jumper’s knee, we defined jumper’s knee as a clinical diagnosis based on the following diagnostic criteria: (a) A history of pain in the quadriceps or patellar tendons at their patellar insertions in connection with training or competition, and (b) tenderness to palpation corresponding to the painful area (Lian et al., 2005; Visnes & Bahr, 2012; Visnes et al., 2013). In addition, all symptomatic athletes underwent a standard knee examination to exclude other diagnoses. Symptoms had to have been present for a minimum of 12 weeks. As symptom onset often is gradual and slow, the athlete had to consider that the symptoms were sufficient to represent a substantial problem. The school physician and physiotherapist recorded cases prospectively, and the principal investigator (HV) visited TVN at least twice a year (August/September and March/April) to examine all athletes.

Prognosis
The clinical outcome measured over the study period was knee function using VISA score, designed specifically to quantify knee function in subjects with patellar tendinopathy and has been shown to be a reliable and valid measure (Cook et al., 1998; Visentini et al., 1998; Khan et al., 1999). The patients self-recorded VISA score for each knee separately immediately after the first ultrasound examination, and semi-annually after that.

Calculations and statistical methods
We used SPSS (version 20.0, SPSS Inc., Chicago, Illinois, USA) to perform the analyses. Baseline data and results are reported as the average ± SD unless otherwise noted. Baseline data for height, weight, age, tendon thickness and previous training volume were analyzed with an independent sample t-test for healthy athletes, new cases and for athletes with pre-existing jumper’s knee. Changes in tendon thickness per site were analyzed with an independent sample t-test for the hypothesis that all changes in the tendon variables were equal to zero simultaneously. This test creates a vector of changes per tendon where change in measurements from proximal, mid and distal were three components in the vector account the within-person correlation of the components in the vector. This was done by the Wilk’s lambda
Fig. 1. Flowchart depicting the inclusion and follow-up of subjects during the 4-year study period.
method. Analyses were adjusted for gender. Similarly, total changes in tendon thickness were also tested against zero using the same method. The multivariable gender differences in changes (of tendon variables) were tested by Hotelling’s T-squared statistics. Associations between training volume and changes in tendon thickness were assessed by multiple linear regression analyses. Odds ratios for hypoechoic areas and neovascularization at baseline were analyzed with a logistic regression analysis model adjusted for gender. P-values of $< 0.05$ were considered statistically significant.

Results
Recruitment and athlete characteristics

During the 4-year study period, all student-athletes at TVN were included and 158 students (84 females, 74 males) were followed for 268 student-seasons. Students were enrolled at TVN for an average of 1.7 years (SD 0.7); a total of 72 students were followed for 1 year, 63 students for 2 years and 24 for 3 years.

Seventeen (six females, 11 males) of 158 students had pre-existing jumper’s knee when enroling at TVN. In most of these cases ($n = 14$, 19 tendons), their symptoms stemmed from the proximal patellar tendon, while in three cases (one female, two males, four tendons) symptoms were from the distal quadriceps tendon.

Of the 141 asymptomatic athletes included, 22 developed jumper’s knee during their time at TVN, 18 boys and four girls (Fig. 2), on average 0.9 years (SD 0.6) after inclusion, and all with symptoms from the proximal patellar tendon. Of the 22 athletes with new problems, 13 (three females, 10 males) developed bilateral symptoms; therefore, a total of 35 tendons developed jumper’s knee.

As shown in Table 1, there were no differences in height, weight or age between asymptomatic athletes, athletes with pre-existing jumper’s knee and new cases, except for women with pre-existing jumper’s knee, who had higher body mass than the new cases.

Tendon thickness

Data from the 119 athletes who remained asymptomatic are presented in Table 2, while data from 22 athletes who developed jumper’s knee are shown in Table 3. At baseline, males had thicker quadriceps and patellar tendons compared with females (global multivariable linear model, $P = 0.001$). Boys who went on to develop jumper’s knee had thicker tendons at baseline (proximal:
Table 1. Baseline characteristics

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Healthy n=45</td>
<td>New cases n=18</td>
<td>Pre-existing jumper’s knee n=11</td>
<td>Healthy n=74</td>
</tr>
<tr>
<td>Age (year)</td>
<td>17.0 ± 0.9</td>
<td>16.8 ± 0.6</td>
<td>16.7 ± 0.6</td>
<td>16.7 ± 0.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>186 ± 6</td>
<td>185 ± 8</td>
<td>181 ± 9</td>
<td>171 ± 6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75 ± 8</td>
<td>72 ± 7</td>
<td>71 ± 10</td>
<td>65 ± 8</td>
</tr>
</tbody>
</table>

*P = 0.05 vs new cases

5.0 ± 0.2 mm (SE), mid-portion: 4.6 ± 0.2 mm] compared with males that remained healthy (proximal: 4.5 ± 0.1 mm, mid-portion: 4.3 ± 0.1 mm) (independent t-test: proximal P = 0.02, mid-portion; P = 0.02). There was no baseline difference in distal patellar tendon thickness, quadriceps tendon thickness or among females.

Table 2 describes all details of thickness changes in athletes who did not develop any symptoms. We found an overall increase in quadriceps tendon thickness (Wilk’s lambda, all subjects: P = 0.001, women: P = 0.001, men: P = 0.14), while no increase was detected in the patellar tendon (all subjects: P = 0.07, women: P = 0.14, men: P = 0.36).

As shown in Table 3, in males the patellar tendon thickness increased from inclusion to the first ultrasound examination after diagnosis in the 28 tendons affected by jumper’s knee among males, especially in the proximal tendon. Proximal tendon thickness continued to increase in these tendons until the players left TVN (paired t-test, all subjects: P = 0.02, women: P = 0.24, men: P = 0.01). The increase in quadriceps tendon thickness among athletes developing jumper’s knee in their patellar tendon was similar in magnitude to that observed in asymptomatic athletes, but not significantly different from baseline (Wilk’s lambda, all subjects: P = 0.20, women: P = 0.74, men: P = 0.55).

Training volume

The 22 athletes developing jumper’s knee did more volleyball training [new cases: 14.0 ± 0.5 (SE) h/week, healthy athletes: 12.0 ± 0.3 h/week; independent t-test, P = 0.003], but not more strength training (new cases: 2.6 ± 0.3 h/week, healthy athletes: 2.5 ± 0.2 h/week; P = 0.90) compared with the 119 asymptomatic athletes, while we did not detect a difference in the volume of volleyball training (new cases: 9.5 ± 1.0 h/week, healthy athletes: 8.1 ± 0.4 h/week; independent t-test, P = 0.14) or strength training (new cases: 1.2 ± 0.3 h/week, healthy athletes: 1.4 ± 0.2 h/week; independent t-test, P = 0.57) during the year before they were included in the cohort. There was a weak correlation between the volume of volleyball training and change in patellar tendon thickness (linear multiple regression analysis: r² = 0.054, P = 0.04), but not for quadriceps tendon thickness change (r² = 0.002, P = 0.46).

Hypoechoic areas and neovascularization

The frequency of ultrasound changes is described in Table 4. The average hypoechoic CSA observed in the patellar tendon was 43 ± 36 mm² at inclusion and 65 ± 50 mm² at the last ultrasound examination, while the smallest hypoechoic area registered was 6 mm². About half of the asymptomatic athletes who went on to develop jumper’s knee had ultrasound changes when they started at TVN. In a multivariate logistic regression analysis, a baseline finding of a hypoechoic tendon area [odds ratio (OR) 3.3, 95% confidence interval (CI) 1.1 to 9.2] and neovascularization (OR 2.7, 95% CI 1.1 to 6.5) increased the risk of developing jumper’s knee. In 24 of the 35 cases (69%), the final ultrasound examination before diagnosis revealed hypoechoic areas and in 22 of these 24 cases there was also neovascularization. At the time when the clinical diagnosis of jumper’s knee had been made, 83% of the tendons revealed hypoechoic areas and 74% neovascularization. The prevalence of ultrasound changes among the athletes who developed jumper’s knee remained the same from the time of diagnosis until they left school (Table 4). For athletes who remained asymptomatic, the prevalence of ultrasound changes was similar from inclusion through the end of the study period (Table 4).

Prognosis

A total of 39 athletes (22 new cases of jumper’s knee and 17 with pre-existing jumper’s knee) were followed for an average of 1.0 (SD 0.7) year and with 2.0 (SD 1.4) ultrasound examinations after diagnosis. They reported a VISA score of 72 (SD 23) when first diagnosed, which did not change during subsequent examinations and was 73 (SD 21) when they left TVN. As shown in Table 4, the majority had also persistent ultrasound changes.

Discussion

Our findings from this prospective study indicate that the presence of hypoechoic areas and neovascularization...
Table 2. Patellar and quadriceps tendon thickness (mm) at inclusion and change (Δ) of tendon thickness from inclusion until they left Toppvolley Norge in the 119 athletes (45 males, 74 females) who did not develop symptoms of jumper’s knee

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Proximal tendon</td>
<td>Mid-tendon</td>
</tr>
<tr>
<td></td>
<td>Mean (± SD) Δ (95% CI)</td>
<td>Mean (± SD) Δ (95% CI)</td>
</tr>
<tr>
<td>Patellar tendon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>4.5 ± 0.8</td>
<td>0.2 (−0.2, 0.5)</td>
</tr>
<tr>
<td>Left</td>
<td>4.6 ± 0.8</td>
<td>0.0 (−0.1, 0.1)</td>
</tr>
<tr>
<td>Quadriceps tendon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>7.0 ± 1.4</td>
<td>0.5 (0.1, 0.9)</td>
</tr>
<tr>
<td>Left</td>
<td>7.1 ± 1.3</td>
<td>0.6 (0.2, 1.0)</td>
</tr>
</tbody>
</table>

Significant changes are denoted in bold.

Table 3. Patellar tendon thickness (mm) at inclusion and change (Δ, from inclusion until they developed symptoms) in tendon thickness in the 22 athletes who developed symptoms of jumper’s knee from their patellar tendon

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Proximal tendon</td>
<td>Mid-tendon</td>
</tr>
<tr>
<td></td>
<td>Mean (± SD) Δ (95% CI)</td>
<td>Mean (± SD) Δ (95% CI)</td>
</tr>
<tr>
<td>Symptomatic tendon (n = 35)</td>
<td>5.0 ± 1.2</td>
<td>1.6 (0.9, 2.3)</td>
</tr>
<tr>
<td>Contralateral asymptomatic tendon (n = 9)</td>
<td>5.0 ± 1.0</td>
<td>0.8 (−0.1, 1.8)</td>
</tr>
</tbody>
</table>

Significant changes are denoted in bold.*Only one female tendon without contralateral symptoms.
among asymptomatic athletes at baseline represented a risk factor for developing tendon pain. Data on tendon thickness revealed a differential effect between the patellar and quadriceps tendons; in asymptomatic athletes, patellar tendon thickness did not change despite the high volume of training, while the quadriceps tendon hypertrophied.

Relationship between symptoms and structural changes
The relationship between structural tendon changes and pain remains to be fully elucidated. We observed structural changes at baseline in more than half of the tendons who later went on to develop symptoms of jumper’s knee and at the time of diagnosis 83% of painful tendons revealed such changes. This could be interpreted to mean that structural changes precede symptoms. However, this also means that 17% of symptomatic tendons looked normal on ultrasound examination. Moreover, only 17 of 46 tendons (36%) with hypoechoic areas at baseline developed symptoms during a 1.7-year observation period with intensive volleyball training. However, it should be noted that 72 of the 158 students were only followed for 1 year, and we do not know how many would have developed symptoms had they completed the 3-year school program. Nevertheless, as suggested by the early cross-sectional studies (Khan et al., 1996; Lian et al., 1996), our prospective data show that there is clearly not a one-to-one relationship between pain and structural changes.

Previous longitudinal studies on the patellar tendon have yielded conflicting evidence. In 2000, Cook et al. (2000b) followed 26 young asymptomatic basketball players (14–18 years) for 16 months. At baseline, 10 of 52 patellar tendons had hypoechoic areas and three of them went on to develop jumper’s knee compared with three of the 42 normal tendons. The authors concluded that a hypoechoic area represented a risk factor for developing jumper’s knee, but also that structural changes were not necessarily associated with symptoms. However, the same research group also followed 23 asymptomatic football, basketball and cricket players (age: 30 years) for 4 years (Cook et al., 2001). In this small prospective study on adult athletes, ultrasound changes did not predict future tendon problems. At baseline, 18 of 46 tendons had hypoechoic changes. During the 4-year study period, six tendons developed clinical symptoms; of these, four had hypoechoic changes at baseline and two were normal. Fredberg & Bolvig (2002) then followed 54 adult professional soccer players for one season. They concluded that hypoechoic regions were associated with development of symptoms, even if only three of 18 with hypoechoic areas at baseline became symptomatic. Notably, nine tendons were still hypoechoic but asymptomatic and six tendons normalized. Also, six normal tendons developed hypoechoic areas but no symptoms after 11 months. Gisslen et al. (2007) monitored a population comparable with ours, 22 adolescent volleyball players (16 years at inclusion), for 3 years. Of the 36 tendons which were asymptomatic at baseline, three of nine with structural changes did develop symptoms, while only two of the 27 structurally normal tendons later developed jumper’s knee. Fredberg et al. (2008) randomized 209 professional soccer players to either a control group or to an intervention group who was given a prophylactic eccentric training program for the Achilles and patellar tendons during one soccer season. At inclusion, 28% had “severely abnormal” patellar

Table 4. Frequency of ultrasound changes (number of tendons, %) observed at the time of inclusion, time of diagnosis and at last examination before leaving school in players who remained healthy (n = 119 athletes), in asymptomatic players who went on to develop jumper’s knee (n = 22 athletes) and in players with pre-existing jumper’s knee (n = 17 athletes, total sample: n = 158 players, 316 tendons)

<table>
<thead>
<tr>
<th></th>
<th>Inclusion</th>
<th>Time of diagnosis</th>
<th>Last examination</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hypoechoic areas</td>
<td>Neovascularization</td>
<td>Hypoechoic areas</td>
</tr>
<tr>
<td>Patellar tendon</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic athletes (n = 238)</td>
<td>24 (10%)</td>
<td>9 (4%)</td>
<td>27 (11%)</td>
</tr>
<tr>
<td>New cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Symptomatic tendon (n = 35)</td>
<td>17 (55%)</td>
<td>15 (48%)</td>
<td>29 (83%)</td>
</tr>
<tr>
<td>- Contralateral asymptomatic tendon (n = 9)</td>
<td>5 (56%)</td>
<td>3 (33%)</td>
<td>5 (56%)</td>
</tr>
<tr>
<td>Pre-existing jumper’s knee</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Symptomatic tendon (n = 19)</td>
<td>14 (74%)</td>
<td>16 (84%)</td>
<td>12 (67%)*</td>
</tr>
<tr>
<td>- Contralateral asymptomatic tendon (n = 15)</td>
<td>3 (20%)</td>
<td>3 (20%)</td>
<td>6 (40%)</td>
</tr>
<tr>
<td>Quadriceps tendon</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic tendon (n = 312)</td>
<td>22 (7%)</td>
<td>12 (4%)</td>
<td>14 (4%)</td>
</tr>
<tr>
<td>Pre-existing jumper’s knee (n = 4)</td>
<td>3 (75%)</td>
<td>0</td>
<td>2 (50%)</td>
</tr>
</tbody>
</table>

*18 of 19 tendons were tested more than one time.
tendons. After the intervention period, the frequency of ultrasonographic abnormalities in the patellar tendons was lower in the training group than in the control group (10% vs 20%), and this was also true in players with abnormal patellar tendons at inclusion (61% vs 79%). They concluded that players had a relative risk of 2.2 ($P = .09$) for developing symptoms during the season if severe ultrasonographic abnormalities in the patellar tendons were detected before the start of the season. Two more recent studies have also identified ultrasound changes as a risk factor (Comin et al., 2013; Giombini et al., 2013). Comin et al. (2013) surveyed 79 professional ballet dancers in a 24-month longitudinal study. Of 158 patellar tendons, seven developed disabling symptoms, but only three of these were among the 19 who had hypoechoic changes at baseline. Still, of 139 with normal tendons only four developed symptoms. Giombini et al. (2013) followed 37 elite fencers training for the Olympics (age: 27 years) for 36 months. As two of eight tendons with abnormal ultrasound findings at baseline developed symptoms and none of 66 normal tendons did, they concluded that ultrasound imaging may be predictive for development of future symptoms in patients with patellar tendinopathy.

To summarize, previous prospective studies document that there is clearly not a one-to-one relationship between pain and structural changes. Hypoechoic changes are observed in 10–30% of asymptomatic tendons, and hypoechoic areas can resolve, remain unchanged or expand (Lian et al., 1996; Khan et al., 1997; Cook et al., 2000a; Fredberg & Bolvig, 2002; Gisslen et al., 2007; Fredberg et al., 2008). At the same time, the presence of hypoechoic areas in the tendon seems to increase the risk of developing tendon pain. This effect may be somewhat more pronounced in younger athletes than among adults. The results from the previous longitudinal studies indicate that $16–33\%$ of tendons with abnormalities go on to develop jumper’s knee (Fredberg & Bolvig, 2002; Gisslen et al., 2007; Comin et al., 2013; Giombini et al., 2013) and our findings, which is one of the largest to date on the patellar tendon, correspond to this (36%).

Whether color Doppler may contribute additional information to grey-scale ultrasound is unknown. Boesen et al. (2012) used color Doppler only in their study among semi-professional badminton players. They concluded that intratendinous blood flow did not predict injury and that the level of intratendinous blood flow more likely represents a physiological response. Previous studies have also shown that there is some degree of color Doppler activity in the tendon after exercise (Boesen et al., 2006; Malliaras et al., 2008; Koenig et al., 2010). In our study, we had no control of the activity level of the athletes even though they did not come directly from training. It remains an important research question to distinguish between possible physiological intratendinous flow and possible pathological activity (Koenig et al., 2010).

Increased proximal patellar tendon thickness is often used as diagnostic criteria of patellar tendinopathy (Coupp et al., 2013). The current paradigm of tendinopathy describes a continuum of pathological changes, with diffuse thickening thought to represent a “prepathological” state (Cook & Purdam, 2009). Our findings correspond to this. The development of tendinopathy has been suggested to be a “systemic” process (Cook & Purdam, 2009), and even if our players only developed unilateral jumper’s knee, their asymptomatic contralateral side also increased in thickness. Thus, care should be taken when using this as reference.

**Tendon adaptation to training**

Our study is the first to examine whether tendons hypertrophy in response to intensive training during adolescence. Among athletes without signs of tendinopathy, we observed an 7–11% increase in quadriceps tendon thickness, while there was no increase in patellar tendon thickness. The quadriceps tendon was also approximately 50% thicker than the patellar tendon. The results were somewhat surprising. We would have expected an adaptive increase in thickness in both tendons considering that the students did 12–14 h of volleyball training and 2–3 h of strength training each week for 1.7 years (i.e. two full school seasons) on average. However, it should be noted that 72 of the 158 students were only followed for 1 year.

The only longitudinal studies available to date on the patellar tendon are on mature athletes and with a much shorter observation period (Kongsgaard et al., 2007; Kubo et al., 2009, 2010; Seynnes et al., 2009; Malliaras et al., 2013). Kongsgaard et al. (2007) enrolled 12 untrained 25-year-old men in 12 weeks of resistance training three times per week, comparing one leg training with heavy loads (10 sets of eight repetitions at 70% of one repetition maximum) in a leg extension machine to the contralateral leg training with light loads (10 sets of 36 repetitions with a load equaling the amount of work to the work performed by the heavy leg). Using MRI, they concluded that proximal tendon CSA increased in both groups. Seynnes et al. (2009) recruited 15 20-year-old university students, who at baseline were involved in recreational training 1–2 times/week. After 9 weeks of heavy resistance training, patellar tendon CSA increased by 3.7% as assessed by MRI. In contrast to these findings, Kubo et al. (2009, 2010) have published two studies where they did not find any change of CSA using MRI. First, they followed a cohort of 10 healthy males prospectively for 12 weeks (Kubo et al., 2009). The program was isometric knee extension training (four times per week, at 70% of maximal voluntary strength with 10
badminton players, participating at the elite level for consistently subjected to higher loads. Seven fencers and of the lower extremities, e.g. where the leading leg is tion is to study athletes with asymmetric loading patterns stiffness and modulus compared with the control group. All measurements were performed on the same leg. In any group; however, the high load eccentric group had significantly greater increases in force, stiffness and modulus compared with the control group.

Another research approach used to address this question is to study athletes with asymmetric loading patterns of the lower extremities, e.g. where the leading leg is consistently subjected to higher loads. Seven fencers and badminton players, participating at the elite level for > 5 years, displayed a side-to-side isometric knee extensor strength difference of more than 15%, in favor of their lead extremity (Couppé et al., 2008). Tendon CSA in the lead extremity was also significantly greater (20–28%) proximally and distally. Furthermore, the lead extremity displayed greater tendon stiffness with no difference in modulus, suggesting that the change in mechanical properties was largely the result of the increase observed in CSA.

Tendon adaptation to load in adolescents has not been investigated previously. We had a medium-term follow-up for an average of 1.7 years. The observed 7–11% increase in quadriceps tendon thickness suggests that the sample size and accuracy of the thickness measurements is more than sufficient, yet there was no sign of hypertrophy of the patellar tendon. At baseline, the quadriceps tendon was 50% thicker compared with patellar tendon, and we do not know why quadriceps and patellar tendons seem to respond differently to loading. However, it may be speculated that this difference can provide clues as to why the patellar tendon is more susceptible to tendinopathy than the quadriceps tendon (Lian et al., 2005).

One question is whether MRI, which has been used in some studies (Kongsgaard et al., 2007; Kubo et al., 2009, 2010; Seynnes et al., 2009), would have been a better tool than ultrasound to detect tendon hypertrophy, and whether this was the reason why we did not detect any change in patellar tendon thickness. We do not believe this to be the case. As can be seen in Table 2, there was no trend toward an increase in patellar tendon thickness in healthy subjects. Table 2 also shows how a change in quadriceps tendon thickness of 0.5–0.7 mm (approximately 10%) consistently was detected using ultrasound. All measurements were made by the same, experienced musculoskeletal ultrasonographer throughout the entire study period and we can see no reason why the accuracy would differ between the quadriceps and patellar tendons. We therefore conclude that tendon thickness did increase in the quadriceps tendon, but not in the patellar tendon. Still, as we have no non-athletic control group with which to compare, we do not know if the increase in quadriceps tendon thickness is the result of intensive training or a consequence of growth and maturation.

Clinical implications
As shown in this and previous studies, evidently there is not a one-to-one relationship between pain and structural changes. Therefore, the interpretation of findings form diagnostic ultrasound examinations must be done with caution. In our study, 17% of those with a clinical diagnosis had no ultrasound changes at the time of diagnosis and at any given time 11% of asymptomatic tendons displayed ultrasound changes. Yet, two-thirds of patients with hypoechoic areas on their baseline ultrasound examination did not develop symptoms during our 1.7-year observation period with intensive training. For the same reason, we cannot recommend instituting routine ultrasound screening programs to prevent jumper’s knee. Based on data from the same cohort, we have identified gender, training volume, match exposure and jumping ability as significant risk factors for jumper’s knee (Visnes & Bahr, 2012; Visnes et al., 2013). Broadly speaking, the most talented boys are at particular risk, partly because of a more intensive training and competition schedule and partly because of higher tendon loads in each jump. We do not know if these athletes could potentially benefit from regular ultrasound monitoring of their tendons, nor what the optimal scanning frequency would be. More advanced ultrasound modalities are emerging, such as ultrasonographic tissue characterization (van Schie et al., 2010), but future studies are needed to determine if such monitoring programs could help manage load in exposed athlete populations.

In conclusion, hypoechoic areas and neovascularization at baseline were risk factors for developing jumper’s knee in this prospective study among elite adolescent volleyball athletes. We observed a 7–11% increase in quadriceps tendon thickness among healthy athletes, while there was no increase in patellar tendon thickness.

Key words: Patellar tendinopathy, jumper’s knee, ultrasound, overuse injury, risk factors, tendinosis, cohort, prevention.
Acknowledgements

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. The authors thank Ingar Holme for statistical advice. We also thank Øyvind Marvik, the other coaches and the players at Toppvolley Norge for helping us organize this study. Kysthospitalet & Hagevik, Department of Orthopaedic Surgery, Haukeland University Hospital, has partially funded the principal investigator.

References


Koenig MJ, Torp-Pedersen ST, Christensen R, Boesen MI, Terslev L, Hartkopp A, Bliddal H. Effect of
Ultrasound: patellar and quadriceps tendon