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# Low Range of Ankle Dorsiflexion Predisposes for Patellar Tendinopathy in Junior Elite Basketball Players

## A 1-Year Prospective Study

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**Background:** Patellar tendinopathy (PT) is one of the most common reasons for sport-induced pain of the knee. Low ankle dorsiflexion range might predispose for PT because of load-bearing compensation in the patellar tendon.

**Purpose:** The purpose of this 1-year prospective study was to analyze if a low ankle dorsiflexion range increases the risk of developing PT for basketball players.

**Study Design:** Cohort study (prognosis); Level of evidence, 2.

**Methods:** Ninety junior elite basketball players were examined for different characteristics and potential risk factors for PT, including ankle dorsiflexion range in the dominant and nondominant leg. Data were collected over a 1-year period and follow-up, including reexamination, was made at the end of the year.

**Results:** Seventy-five players met the inclusion criteria. At the follow-up, 12 players (16.0%) had developed unilateral PT. These players were found to have had a significantly lower mean ankle dorsiflexion range at baseline than the healthy players, with a mean difference of –4.7° ($P$ = .038) for the dominant limb and –5.1° ($P$ = .024) for the nondominant limb. Complementary statistical analysis showed that players with dorsiflexion range less than 36.5° had a risk of 18.5% to 29.4% of developing PT within a year, as compared with 1.8% to 2.1% for players with dorsiflexion range greater than 36.5°. Limbs with a history of 2 or more ankle sprains had a slightly less mean ankle dorsiflexion range compared to those with 0 or 1 sprain (mean difference, –1.5° to –2.5°), although this was only statistically significant for nondominant legs.

**Conclusion:** This study clearly shows that low ankle dorsiflexion range is a risk factor for developing PT in basketball players. In the studied material, an ankle dorsiflexion range of 36.5° was found to be the most appropriate cutoff point for prognostic screening. This might be useful information in identifying at-risk individuals in basketball teams and enabling preventive actions. A history of ankle sprains might contribute to reduced ankle dorsiflexion range.

**Keywords:** jumper's knee; tendinosis; ankle range of motion; ankle sprain

Patellar tendinopathy is one of the most common reasons for sport-induced pain of the knee, especially in sports involving explosive jumping maneuvers such as volleyball and basketball[8,11,33-35]; hence the layman's term for patellar tendinopathy, "jumper's knee."[5] The prevalence of patellar tendinopathy among basketball players is 7% for players aged 14 to 18 years and 32% for players aged 19 to 29 years.[11,35] As many as 53% of athletes afflicted by patellar tendinopathy have been reported to end their career because of the condition[26]; the symptoms include chronic pain in the patellar tendon, reduced function of the knee, and induced or enhanced pain when loading.[5,29] Although patellar tendinopathy was first described in the literature as early as 1973, it remains a poorly understood condition and the cause and pathogenesis leading to the chronic pain is still largely unknown.[14] The previous term for the condition, patellar "tendinitis," was based on the assumption of an underlying chronic inflammation. However, intratendinous microdialysis studies have shown no increase in prostaglandin $E_2$,[1] nor is there any evidence of inflammatory cells in the patellar tendon tissue in chronic patellar tendinopathy.[27] Studies instead point toward tissue pathologic changes of a more degenerative-like nature, called

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"tendinosis,"[28,36] with tissue features such as hypercellularity, vascular proliferation, and disorganization of collagen.[27]

Several theories for the pathogenesis of patellar tendinopathy have been presented over the years; hypotheses including both extrinsic and intrinsic factors.[3,4,13,19,37,44,49] Examples of extrinsic factors of importance are the amount and intensity of training, training surface, and the training material used, including shoes.[17,25] The significance of such factors for the development of patellar tendinopathy is well studied. Nevertheless, not all people exposed to extrinsic risk factors, such as intense and repetitive training, develop patellar tendinopathy, suggesting that one or more additional predisposing intrinsic factors play an important part in the pathogenesis. Strength, flexibility, hip circumference, muscular imbalance, patella alta, patellar hypermobility, jump and landing techniques, tendon elasticity, weight, height, gender, and muscular endurance are all intrinsic factors suggested to be of importance for patellar tendinopathy.[‡]

In sports such as basketball, which involve an average of 70 jumps and landings per game, the patellar tendon is particularly exposed to strain, reaching up to 8 kN during landing after a jump—forces 6 to 8 times the player's weight.[29,52] The highest stress on the patellar tendon is performed during the eccentric phase during landing.[24,31,46] In studies by Richards et al[44] and Bisseling et al,[4] poor landing technique has been shown to be significantly linked to patellar tendinopathy. The degree of ankle dorsiflexion, as well as the strength of the muscles performing plantar flexion, are the most important components in the shock absorbance during landing.[42] Normally, the athlete lands on the forefoot, which then moves into dorsiflexion. This movement is in conjunction with an eccentric contraction of the triceps surae muscle.[15] In a study by Malliaras et al,[37] the strength of the muscles responsible for plantar flexion and the range of dorsiflexion were investigated in volleyball players with and without patellar tendinopathy in a cross-sectional study, showing that patients with the condition had significantly reduced dorsiflexion range as compared with controls.

Most studies investigating intrinsic factors, including the aforementioned study on dorsiflexion range, are of a cross-sectional nature, which makes it hard to know whether these factors contribute to the development of patellar tendinopathy or if they are a mere side effect of the condition. Nevertheless, there are 2 prospective studies[49,50] showing that increased tightness of the hamstrings and quadriceps femoris muscles are contributing factors for the development of patellar tendinopathy and anterior knee pain.

The existing knowledge on the complexity of the ankle joint, and its potential importance for the knee and for the development of patellar tendinopathy, is in general rather poor.[45] As evident by the arguments raised here, there are reasons to believe that a reduced ability to dorsiflex in the ankle joint reduces its contribution to absorption of force at landing after jumping activity, thereby increasing the loading on the patellar tendon and hypothetically the risk of developing patellar tendinopathy, which we know is related to frequent jumping and landing. This prospective study aims to test this hypothesis, examining the possible importance of reduced ankle dorsiflexion range for a person's risk of later developing symptomatic patellar tendinopathy. This was performed in a study on 90 junior elite basketball players with a 1-year follow up.

‡References 3, 4, 14, 19, 25, 37, 38, 44.

## MATERIALS AND METHODS

### Participants

Swedish junior basketball players on the national elite level, attending the annual training camp for ages 14 to 20 years, were recruited to the study. All 90 players (47 males, 43 females), that is, 180 knees, were examined and tested. General exclusion criteria for a knee were reconstructed anterior cruciate ligament, history of Osgood-Schlatter disease, femoropatellar cartilage injury, injections in or in close proximity to the knee, palpation tenderness in combination with anterior knee pain at baseline, or anterior knee pain during latest training session or match.

### Study Design

The general design of the study was to measure ankle dorsiflexion range in all players and, 1 year later, determine how many of the patellar tendons had developed tendinopathy, to see if the hypothesis was true, that lower ankle dorsiflexion range increases the risk of developing patellar tendinopathy.

### Questionnaire, Interview, and Clinical Assessment at Start-up/Baseline

At the starting point (baseline) of the study, each player individually completed a questionnaire, before measurements, regarding sport-specific exercise per week, non–sport-specific exercise per week (including running and weight lifting), history of ankle sprain, and dominant leg. This questionnaire was complemented by a short standardized interview, including questions regarding history of knee injuries/treatment, by the examiner. Following this was the clinical assessment (for identifying clinical exclusion criteria) and the measurements of ankle dorsiflexion range (see next section). The palpation of the patellar tendon—centrally, laterally and medially—was performed at the attachment to the patella by tilting the inferior pole of the patella anteriorly in a position with the knee in full extension, as with a flexed knee, the tenderness might be difficult to elicit.[7,12,29,33]

### Measurement of Ankle Dorsiflexion Range (Start-up/Baseline)

After a standardized warm-up protocol, ankle dorsiflexion range was measured according to the established weight-bearing lunge test previously used by others.[2,14] A line was drawn on the floor and continued up the wall. The foot was positioned on the line such that the heel and second toe were aligned to the line. The participant lunged forward to touch the line on the wall with the center of the patella, while the foot position was maintained with the heel in contact with the floor. At the maximum position, the gravity inclinometer was placed on the anterior border of tibia, 15 cm below the tibial tuberosity, and the angle was recorded. This method has previously shown high intrarater and interrater reliability.[2] The measurements were performed by the same physiotherapist (L.J.B.) in all cases.

### Clinical Assessment at 1-Year Follow-up: Diagnosing Patellar Tendinopathy

The follow-up was performed 1 year later at the following annual training camp for the national elite junior basketball players. A short interview of the participants was followed by a clinical investigation by the physiotherapist. Players who developed symptoms from the knee during the 1-year period between the camps were investigated and diagnosed at the time of the symptoms, and thereby included in the 1-year incidence data even if the symptoms had improved by the time of follow-up.

The following criteria were used for diagnosing patellar tendinopathy: history of activity-related anterior knee pain and reduced function of the knee, distinct palpation tenderness corresponding to the painful area, and knee pain provoked by a previously described single-legged decline squat test, designed to optimize the loading on the patellar tendon.[32] The interview and clinical investigation were used as the instrument of diagnosis, as ultrasound and MRI have shown low predictive value in diagnosing patellar tendinopathy.[10,30,33]

Activity-related pain at the insertion of the patellar tendon to the patella is an established characteristic of patellar tendinopathy.[11,29] Palpation in the patellar tendon is a key component of the clinical diagnosis.[12] However, mild tenderness at palpation of the patellar tendon insertion in asymptomatic individuals, with no tendon-loading pain, should be considered as a normal finding.[12,33]

### Statistical Analysis

Data were statistically analyzed using PASW Statistics 18 software (18.0.0, SPSS Inc, Chicago, Illinois). Statistical tests used (independent and paired-samples $t$ tests, Pearson $\chi^2$, and Fisher exact tests) are accounted for in the Results section, when the test in question is applied.

Differences between dominant and nondominant legs regarding ankle dorsiflexion range were hypothesized, due to different use in basketball,[40] and such changes were confirmed in a paired-samples $t$ test (see Results section), which takes into account that the legs belong to the same individual. In response to this, data were analyzed separately for the respective legs.

To determine whether it is possible to find a clinically relevant cutoff point for ankle dorsiflexion range that can be used for pointing out individuals at high risk of developing tendinopathy, receiver operating characteristic (ROC) curves were calculated. The area under the ROC curve represents the probability that a selection based on the risk factor for a randomly chosen positive case will exceed the result for a randomly chosen negative case. The area under the curve can range from 0.5 (no accuracy) to 1.0 (perfect accuracy). If it is found to be statistically significant, it means that using the risk factor as a determinant is better than guessing. Since the ROC curve plots sensitivity against 1 minus specificity, the coordinates of the curve can be considered possible cutoff points, and the most suitable cutoff can be chosen.

Significance was predetermined at $P < .05$.

TABLE 1
Descriptive Data at Baseline for the Participants Included (N = 75)[a]

| | Mean ± SD | Range |
|---|---|---|
| Age, y | 17.8 ± 1.6 | 14.4-20.6 |
| Weight, kg | 79.6 ± 11.8 | 57.0-110.0 |
| Height, cm | 188.0 ± 10.4 | 163.0-214.0 |
| BMI | 22.4 ± 1.6 | 17.6-27.1 |
| Sport-specific training, h/wk | 12.4 ± 3.8 | 4.0-25.0 |
| Non–sport-specific training, h/wk | 2.7 ± 1.5 | 0.0-10.0 |
| All exercise, h/wk | 15.1 ± 4.2 | 5.0-29.0 |

[a]SD, standard deviation; BMI, body mass index.

## RESULTS

### Characteristics of Participants Included

Of the 90 players examined, 75 players (38 males, 37 females)—in total 148 tendons—met the inclusion criteria. Descriptive data for potential risk factors on an individual level noted at baseline are shown in Table 1. Of the excluded players, 3 had a history of Osgood-Schlatter disease, 8 had palpation tenderness and activity-related anterior knee pain during latest training or match at start-up, 1 had arthroscopically verified femoropatellar cartilage injury, 1 could not determine the dominant foot, and 2 did not participate at follow-up. In addition, 2 individuals had 1 of their tendons excluded due to a history of unilateral Osgood-Schlatter disease in that leg.

As for limb-specific risk factors (ie, range of ankle joint dorsiflexion and number of ankle sprains), the data for dominant legs (n = 75) and nondominant legs (n = 73), as determined by the questionnaire, were investigated separately since the mean ankle dorsiflexion range in the dominant leg (38.7°; standard deviation [SD] ±5.4°; range, 26.0°-51.0°) was found to be significantly lower than in the nondominant leg (40.0° ± 5.3°; range, 23.0°-50.0°), as seen with paired-samples $t$ test (mean difference, –1.3°; 95% confidence interval [CI], 0.3-2.3; $P$ = .015).

### Outcome of Potential Risk Factors on Tendon Health

At the 1-year follow-up, unilateral patellar tendinopathy was diagnosed in 12 players (8 males, 4 females) of the

TABLE 2
Comparison Between Factors at Baseline of Participants Later Developing Tendinopathy With Those of Healthy Players at Follow-up[a]

| | Tendinopathy (n = 12), Mean ± SD | Normal (n = 63), Mean ± SD | Mean Difference (95% CI) | *P* Value |
|---|---|---|---|---|
| Age, y | 17.4 ± 1.3 | 17.9 ± 1.7 | −0.5 (–1.4, 0.3) | .214 |
| Weight, kg | 84.1 ± 17.1 | 78.7 ± 10.5 | 5.4 (–5.7, 16.5) | .310 |
| Height, cm | 194.2 ± 13.3 | 186.8 ± 9.5 | 7.4 (–1.3, 16.1) | .090 |
| BMI | 22.1 ± 2.0 | 22.5 ± 1.5 | −0.4 (–1.4, 0.6) | .430 |
| Sport-specific training, h/wk | 13.5 ± 4.5 | 12.2 ± 3.7 | 1.3 (–1.1, 3.7) | .270 |
| Non–sport-specific training, h/wk | 2.5 ± 1.3 | 2.7 ± 1.6 | −0.2 (–1.2, 0.7) | .635 |
| All exercise, h/wk | 16.0 ± 5.2 | 14.9 ± 4.0 | 1.1 (–1.5, 3.8) | .407 |

[a]SD, standard deviation; CI, confidence interval; BMI, body mass index.

TABLE 3
Comparison Between Mean Ankle Dorsiflexion Range at Baseline for Diagnosis Groups at Follow-up[a]

| | Tendinopathy, Mean ± SD | Normal, Mean ± SD | Mean Difference (95% CI) | *P* Value |
|---|---|---|---|---|
| Ankle dorsiflexion range in dominant leg, deg | 34.3 ± 4.0 (n = 6) | 39.1 ± 5.3 (n = 69) | −4.7 (–9.2, –0.3) | .038[b] |
| Ankle dorsiflexion range in nondominant leg, deg | 35.3 ± 4.5 (n = 6) | 40.4 ± 5.2 (n = 67) | −5.1 (–9.5, –0.7) | .024[b] |

[a]SD, standard deviation; CI, confidence interval.
[b]Significant at $P < .05$ (independent-samples $t$ test).

75 players included, giving a 1-year incidence of 16.0%. No bilateral patellar tendinopathy was diagnosed. Tendinopathy incidence was almost twice as common in men (21.1%) as in women (10.8%), although this difference was not significant ($P$ = .226, Pearson $\chi^2$). There was no statistically significant difference in incidence of patellar tendinopathy in tendons of dominant legs (6 of 75 [8.0%]) as compared with nondominant legs (6 of 73 [8.2%]).

The means for each potential risk factor noted at baseline for the individuals included were compared between diagnosis groups (tendinopathy vs normal at follow-up) using the independent-samples $t$ test (Table 2). None of the factors were shown to be significantly associated with tendinopathy development at the individual level.

As stated above, data for limb-specific risk factors were investigated separately for the dominant legs and the nondominant legs (as determined by the questionnaire), since the legs differed significantly concerning range of dorsiflexion. For each leg, the mean range of ankle joint dorsiflexion at baseline for the tendons included was compared between diagnosis groups (tendinopathy vs normal at follow-up), using the independent-samples $t$ test. It was found that players who had developed tendinopathy at follow-up had had a significantly reduced mean ankle dorsiflexion range at baseline compared with those who were healthy at follow-up, the mean difference being 4.7° to 5.1° depending on the leg. The differences were statistically significant for both the dominant ($P$ = .038) and the nondominant leg ($P$ = .024). Details are provided in Table 3.

## Possible Importance of History of Ankle Sprains on Dorsiflexion Range and Tendinopathy Development

To determine whether the history of ankle sprain seemed to influence the ankle joint dorsiflexion range, the mean range of dorsiflexion at baseline was compared between ankles of limbs that had undergone 2 or more ankle sprains and those of limbs with a history of 0 or 1 ankle sprain. It was found that the former group had a slightly lower mean range of dorsiflexion compared with the latter group (mean difference 1.5°-2.5° depending on the leg), and that this difference was statistically significant, as seen with the independent-samples $t$ test, for the nondominant leg ($P$ = .048) but not for the dominant leg ($P$ = .233). Details are provided in Table 4.

The incidence of patellar tendinopathy in tendons of legs with 2 or more ankle sprains (10.5%) was higher as compared with those with 0 or 1 ankle sprain (6.6%), although this difference was not significant ($P$ = .537, Fisher exact test).

## Determining Diagnostic Cutoff for Ankle Dorsiflexion Range With High Risk of Developing Tendinopathy

The area under the ROC curves was 0.78 for the dominant leg (Figure 1) and 0.81 for the nondominant leg (Figure 2), both being statistically significant ($P$ = .024 and $P$ = .013, respectively). Using the coordinates of the curves, the angle of dorsiflexion range that most

TABLE 4
Comparison Between Mean Ankle Dorsiflexion Range at Baseline for Groups Based on the History of Ankle Sprains Prior to Start-up[a]

| | ≥2 Ankle Sprains, Mean ± SD | ≤1 Ankle Sprains, Mean ± SD | Mean Difference (95% CI) | *P* Value |
|---|---|---|---|---|
| Ankle dorsiflexion range in dominant leg, deg | 37.7 ± 5.7 (n = 28) | 39.3 ± 5.2 (n = 47) | −1.5 (–4.1, 1.0) | .233 |
| Ankle dorsiflexion range in nondominant leg, deg | 38.5 ± 5.9 (n = 29) | 41.0 ± 4.7 (n = 44) | −2.5 (–5.0, 0.0) | .048[b] |

[a]SD, standard deviation; CI, confidence interval.
[b]Significant at $P < .05$ (independent-samples $t$ test).

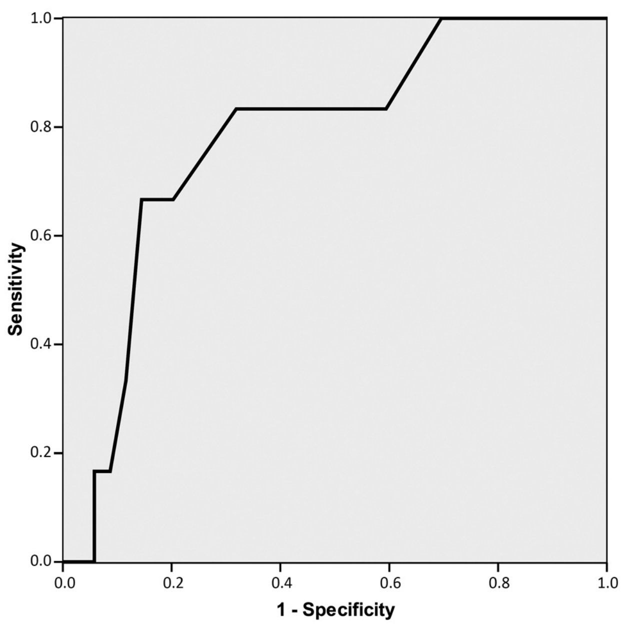


**Figure 1.** Receiver operating characteristic curve for the ankle dorsiflexion range in the dominant leg, as a risk factor for developing tendinopathy in the patellar tendon on that side (ipsilaterally). The area under the curve is 0.78 (*P* = .024); the coordinates represent possible cutoff point in ankle dorsiflexion range (optimal cutoff point determined to be 36.5°).

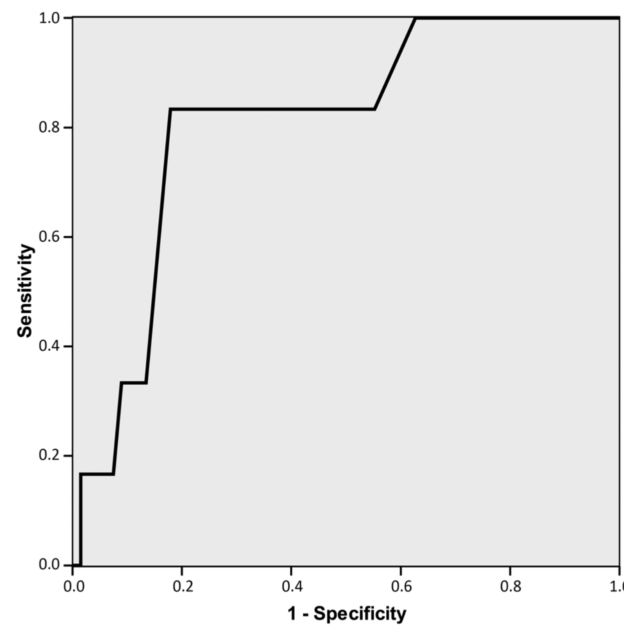


**Figure 2.** Receiver operating characteristic curve for the ankle dorsiflexion range in the nondominant leg, as a risk factor for developing tendinopathy in the patellar tendon on that side (ipsilaterally). The area under the curve is 0.81 (*P* = .013); the coordinates represent possible cutoff point in ankle dorsiflexion range (optimal cutoff point determined to be 36.5°).

accurately identified individuals at risk of developing tendinopathy was determined to be 36.5° (for both the dominant and nondominant leg).

When applying this cutoff point to our data, players who were identified as belonging to the higher-risk group at baseline (ie, who had an ankle dorsiflexion range of <36.5°) had a significantly higher post-test probability of developing tendinopathy within a year, as compared with players identified as belonging to the lower-risk group at baseline (ie, who had an ankle dorsiflexion range of >36.5°). The post-test probability in the higher-risk group was 18.5% for the dominant leg and 29.4% for the nondominant leg, whereas the post-test probability in the lower-risk group was 2.1% for the dominant leg and 1.8% for the nondominant leg. The differences between the risk groups were statistically significant for both the dominant (*P* = .021) and the nondominant (*P* = .002) leg. The characteristics of the test of measuring ankle dorsiflexion range with a cutoff point of 36.5° as a prognostic screening factor for risk of developing tendinopathy are shown in Table 5.

## DISCUSSION

This study clearly shows that a low ankle dorsiflexion range is a risk factor for developing patellar tendinopathy in young basketball athletes. In the sample studied, an ankle dorsiflexion range of 36.5° was found to be the most appropriate cutoff point for prognostic screening of individuals at high risk of developing patellar tendinopathy; the risk of doing so within a year for players with a lower dorsiflexion range than 36.5° was 18.5% to 29.4% as compared with 1.8%

TABLE 5
Sensitivity, Specificity, and Predictive Values for the Set Cutoff Point at 36.5°[a]

| | Positive Predictive Value (95% CI) | Negative Predictive Value (95% CI) | Sensitivity (95% CI) | Specificity (95% CI) |
|---|---|---|---|---|
| Dominant leg | 18.5% (7.0-38.7) | 97.9% (87.5-99.9) | 83.3% (36.5-99.1) | 68.1% (55.7-78.5) |
| Nondominant leg | 29.4% (11.4-56.0) | 98.2% (89.2-99.9) | 83.3% (36.5-99.1) | 82.1% (70.4-90.0) |

[a]CI, confidence interval.

to 2.1% in players with a dorsiflexion range greater than 36.5°. This might be useful information in identifying individuals at risk on basketball teams, thus enabling extra preventive actions in those individuals. The prospective design of the study adds to the existing knowledge within the field, since this shows that low ankle dorsiflexion range precedes patellar tendinopathy development, and is not just a mere side effect of the latter.

## Sample

In this prospective study, the 1-year incidence of developing patellar tendinopathy for junior elite basketball players was 16.0%. Another study reports a 2-year incidence of patellar tendinopathy of 13.8%, although this was seen for an athletic population of various sports, not just basketball.[49] It is known that the prevalence of patellar tendinopathy in athletes is the highest in volleyball and basketball,[35] making the 1-year incidence of the present sample reasonable.

The incidence for male athletes in the present study was 21.1% and for female athletes, 10.8%; this difference, however, was not statistically significant. Nevertheless, patellar tendinopathy is previously reported to be twice as common in males as in females.[9,35]

Height, weight, or body mass index at baseline did not differ significantly between those who developed tendinopathy and those who stayed healthy in this study, although there was a tendency for players developing tendinopathy to be taller than the others (mean difference, 7.4 cm; $P = .090$). The fact that height and weight does not seem to be correlated to patellar tendinopathy is confirmed by previous studies.[13,49] Nevertheless, Gaida et al[19] reported that female basketball players who developed unilateral tendinopathy had a significantly higher waist-to-hip ratio compared with controls.

It is well known that the degree of training/load is associated with development of patellar tendinopathy.[17,20] Feretti[17] showed that those who play volleyball more than 4 times a week are at greater risk of developing patellar tendinopathy than those who play less. In the sample of the present study, however, no statistically significant differences could be seen in the amount of training between players developing tendinopathy and those who did not. This was the case for both sport-specific exercise (basketball) and non–sport-specific exercise, as well as for the total amount of exercise. It should be noted that training regimens for junior basketball players on elite-level national teams, such as in this study, are rather homogeneous.

There was a statistically significant difference between the mean ankle dorsiflexion range of the dominant foot and that of the nondominant foot in the sample we studied, although the difference was small (1.3° lower mean range in the dominant limb). This is an interesting fact to reflect upon. It is known that the take-off leg in lay-up (1-legged jump in basketball) is the opposite of the hand used for throwing the ball.[40] For that reason, the nondominant leg is mostly used at take-off, which might explain the slightly higher dorsiflexion range of this leg. The difference seen between the dominant and the nondominant limb, albeit small, justified the separate analysis of the limbs.

## Possible Explanations for Reduced Ankle Dorsiflexion Range Leading to Tendinopathy

It has been shown that the dorsiflexion movement with the greatest moment of force (108 N·m) measured in volleyball players occurs at the landing; other moments measured at dorsiflexion were clearly less.[44] Furthermore, the absorbance of kinetic energy at the impact and throughout the ankle dorsiflexion range after landing from a jump is 37% to 50% of the total kinetic energy absorbed by the involved muscular system.[15] The possible reduced capacity of energy absorption at the ankle joint due to reduced dorsiflexion range might have to be compensated for in the knee joint, which means higher load on the patellar tendon and risk for tendon injury and patellar tendinopathy.

It should furthermore not be disregarded that the maximal degree of ankle dorsiflexion (ie, the instant when the ankle is the most dorsiflexed) occurs at the initiation of a take-off, when the knee is in flexion. A reduced ankle dorsiflexion in this phase can therefore affect the biomechanics in the jumping, which might result in increased loading on the patellar tendon.

## Possible Importance of History of Ankle Sprains

This study found that limbs that had undergone 2 or more ankle sprains had a slightly less mean range of dorsiflexion compared with limbs with a history of 0 or 1 ankle sprain, although this was only significant for the nondominant leg. Furthermore, there was a trend of a higher incidence of patellar tendinopathy in tendons of legs with 2 or more ankle sprains as compared with those with 0 or 1 ankle sprain. This would indicate that ankle sprains might predispose for a lower range of ankle dorsiflexion, which seems logical since repeated trauma might lead to reduced range of motion. This is certainly a fact in the acute phase,[22] but might also be the case in the chronic stages.[23,43]

After ankle sprain, the postural steadiness is reduced in the sprained ankle and evidence also suggests a bilateral impaired balance after unilateral sprain.[21,48] This loss of function increases the risk of resprains. Proper rehabilitation, including balance training, substantially reduces the risk of sustaining ankle sprains; this effect is seen especially in those with a history of previous ankle sprains.[39] The players examined in this study lacked proper rehabilitation of ankle sprains and, as a side note, this was particularly seen for those players who had a history of 2 sprains or more. The lack of rehabilitation obviously increases the risk of resprains. Bearing that in mind and the fact that ankle sprains seem to lead to reduced ankle dorsiflexion, it is not far-fetched to assume that this is an important factor to deal with in the prevention of patellar tendinopathy.

### Possible Clinical Implications and Conclusion

Based on the results of this study, it seems imperative for basketball players with an ankle dorsiflexion range less than 36.5° to improve the range of dorsiflexion to prevent the development of patellar tendinopathy.

A cutoff point for prognostic screening of individuals at high risk of developing patellar tendinopathy set at an ankle dorsiflexion range of 36.5° seems to be appropriate for junior elite basketball players, but is not necessarily applicable to other sports or age groups. A cross-sectional study of 113 volleyball players set the cutoff point at 45°.[37] However, that study was not prospective and the ankle dorsiflexion range was measured at the time of diagnosis.

The results of the present study suggest that if the cutoff point of 36.5° was applied to a group of athletes with similar characteristics to those we studied (junior elite-level basketball players), 83.3% of the athletes who will develop tendinopathy within a year would be identified as being in the higher-risk group (ie, with an ankle dorsiflexion range <36.5° [sensitivity]), while 17.9% to 31.9% of the players who will not develop tendinopathy would also be identified as belonging to the higher-risk group. These would seem to be acceptable numbers, given that the potential actions taken upon this information would be a fairly mild and noninvasive treatment for increased mobility, which might be beneficial for all players. In addition, the present study only provides data of 1-year incidence and the risk of later developing patellar tendinopathy is thereby unknown. Considering known high prevalence numbers for patellar tendinopathy in basketball players,[35] it would be expected that additional players develop tendinopathy in time. Also, tendinopathy in general is known to be a bilateral disease, with many patients having tendon changes in the contralateral, asymptomatic tendon, and in some cases later developing symptoms there as well.[41]

Finally, as many as 97.9% to 98.2% of the players in the lower-risk group (ankle dorsiflexion range >36.5°) did not develop tendinopathy within a year (negative predictive value), which is a very good result that makes it possible for an examiner to rule out risk of developing tendinopathy within a year with a good degree of certainty.

Reduced ankle dorsiflexion range might occur due to tightness in the calf muscles,[51] inherent ankle joint stiffness, and/or as a result of ankle sprains.[23,43] It is shown that the range of dorsiflexion can be improved with calf stretching[16,51] and joint mobilization,[18,22] meaning that either of these reasons for reduced motion can be treated successfully. Joint mobilization using Mulligan's mobilization with movement (MWM) technique has been shown to significantly improve the dorsiflexion in a subacute phase after ankle sprain.[6] It is also shown that the MWM technique, both weightbearing and non-weightbearing, results in significant improvement of posterior talar glide and weightbearing dorsiflexion as compared with controls.[47] If all players with ankle dorsiflexion lower than 36.5° improve their motion, the development of patellar tendinopathy might be reduced.

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