# Can ultrasound imaging predict the development of Achilles and patellar tendinopathy? A systematic review and meta-analysis

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## ABSTRACT

**Background** Ultrasound (US) imaging is commonly used to visualise tendon structure. It is not clear whether the presence of structural abnormalities in asymptomatic tendons predicts the development of future tendon symptoms in the Achilles or patellar tendon.

**Aim** To perform a systematic review and meta-analysis investigating the ability of US imaging to predict future symptoms of patellar or Achilles tendinopathy.

**Methods** Prospective studies that performed US imaging of Achilles OR patellar tendon structure among asymptomatic patients at baseline and a clinical measure of pain and/or function at follow-up were included. Study quality was assessed using the Critical Appraisal Skills Programme tool by two independent reviewers, and predictive ability of US was assessed using meta-analyses.

**Results** The majority of participants in the review were from sporting populations. Meta-analysis revealed that tendon abnormalities on US are associated with future symptoms of both patellar and Achilles tendinopathy (RR=4.97, 95% CI 3.20 to 7.73). Subgroup analysis indicated that tendon abnormalities at baseline were associated with an increased risk of both Achilles (RR=7.33, 95% CI 2.95 to 18.24) and patellar (RR=4.35, 95% CI 2.62 to 7.23) tendinopathy.

**Conclusions** This systematic review and meta-analysis indicates that tendon abnormalities visualised using US in asymptomatic tendons are predictive of future tendinopathy and are associated with at least a fourfold increased risk.

**Implications** Identification of at-risk athletes using screening tools such as US may allow preventative programmes to be implemented. However, it is clear that other factors beyond tissue structure are involved in the development of lower limb tendinopathy.

## INTRODUCTION

Tendinopathy is an umbrella term commonly used to describe the clinical presentation of localised tendon pain with loading, tenderness to palpation and/or impaired function.[1] It is a highly prevalent musculoskeletal condition affecting both athletes and non-athletes, commonly affecting the Achilles, patellar and rotator cuff tendons.[2]

Ultrasound (US) and magnetic resonance imaging (MRI)[3] appear to be the most common methods for visualising tendon structure, and display similar levels of accuracy and sensitivity.[4] In particular, US has gained increasing popularity among musculoskeletal practitioners by offering advantages of being minimally invasive, as well as being quick and feasible to use in the community or even the sports field.[5]

Imaging of tendons with US has been used in the clinical setting to assist in the diagnosis of tendinopathy, monitor the efficacy of treatments and assess the risk of developing symptoms.[6–8] In many athletes with symptoms of tendinopathy, US imaging of the painful tendons will reveal structural abnormalities, typically localised tendon thickening with hypoechoic areas and altered vascularity.[9 10] In addition, structural abnormalities have previously been suggested to increase the risk of developing future symptoms of tendinopathy in various prospective studies.[11 12] Therefore, it has been proposed that if one were to identify those tendon abnormalities at baseline which may predispose an athlete to the development of future symptoms, one may be able to screen or identify asymptomatic athletes who are at high risk and introduce modifications to their training regimen and/or prophylactic interventions.[10] However, tendon 'abnormalities' have been identified in a large percentage of asymptomatic sporting populations, with hypoechoic areas, increased thickness and neovascularisation present in as many as 59% of asymptomatic individuals.[12–17] Consequently, the correlation between pain and structure in tendinopathy is not linear. This replicates research in other musculoskeletal disorders (eg, shoulder, hip, knee, low back pain (LBP)) where structural abnormalities are evident among asymptomatic individuals.[18–23]

The cross-sectional design of many studies investigating tendon structure means it is unclear whether structural abnormalities on imaging predict future symptoms, or whether they are simply a normal physiological response to specific sporting demands which does not indicate an increased risk of future symptoms. The lack of clarity in this area has led to considerable uncertainty in the management of sporting populations with a high prevalence of tendon-related injuries. Medical professionals face a dilemma on whether to modify an athlete's training and/or intervene using preventative strategies if they observe structural abnormalities on US imaging. Therefore, the aim of this review was to investigate the ability of US imaging to predict future symptoms of Achilles and patellar tendinopathy.

## METHODS

### Search strategy and study selection

This review has been registered in the PROSPERO database (CRD42015020664). The PRISMA

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**Box 1 Keywords for search strategy**

Tendin* OR Tendon* [abstract]
AND
knee* OR patella* OR 'jumpers knee' OR achilles OR heel OR 'tendo calcane*' OR tendocalcane* OR tendoachilles OR 'tendo achilles' [abstract]
AND
ultrasound* OR ultrason* OR imag* OR sonograph* OR "tissue character*" OR UTC [abstract]
AND
risk* OR predict* OR associat* OR relat* OR correlat* OR develop* OR prognos* OR prospect* OR longit* OR cohort* OR future [abstract]

statement for systematic reviews[24] was used to guide the format and reporting of this review. The following databases were searched in October 2015: Academic Search Complete, AMED, Biomedical Reference Collection, CINAHL, MEDLINE, SPORTDiscus, Web of Science and EMBASE. The strategy used a range of keywords in four categories which were combined: (1) tendinopathy (2) patellar and Achilles (3) US and (4) cohort/prospective studies. The keywords for the search strategy are outlined in box 1. Two independent reviewers (SMA and FC) assessed the potential studies retrieved from the databases, with any disagreements mediated by a third reviewer (KMC). Once duplicates were removed, titles and abstracts of the studies were screened for eligibility.

### Inclusion criteria

- Prospective studies investigating the predictive value of patellar or Achilles tendon structure viewed at baseline using US.
- US measurements must have been accompanied with a clinical measure (pain, disability, function, time off sport/activity) to assess the risk of developing future clinical symptoms of Achilles or patellar tendinopathy.
- Duration of follow-up must have been at least 24 hours.
- Tendon structure may have been analysed qualitatively or quantitatively.
- Studies must have been in English, and published in the past 20 years.
- Studies could include participants of any age.
- Studies could include both insertional and mid-portion tendinopathy as well as participants with associated comorbidities.

### Exclusion criteria

- Studies looking only at development of changes in tissue structure, without accompanying clinical measures (as listed above).
- Studies which investigated tendons other than the patellar or Achilles tendon.
- Studies investigating tendon structure in animal populations.

### Assessment of methodological quality

In the absence of an optimum methodological quality rating tool for prospective studies of this nature, the Critical Appraisal Skills Programme (CASP)[25] checklist for cohort studies was used. This checklist contains 12 questions; the first 2 are screening questions, and the remaining 10 explore the results of the study and its validity and applicability to the local population. Questions two, seven, eight and nine explore similar areas and were consequently merged for the purpose of this review. Therefore, the included studies were appraised using seven guiding questions. Since the CASP has many considerations for each question, consistency is key when reviewing studies. Therefore, a list of criteria for each question to be considered when appraising the quality of the included studies was drawn up and agreed between the authors (see online supplementary file S1). Two authors (SMA and FC) scored the studies independently using the criteria outlined, with any disagreements in scoring mediated by a third reviewer (KMC). As a result of the CASP checklist being originally designed to be used as an educational tool as part of a workshop setting, no overall quality score was awarded to the included studies. Instead, the strengths and weaknesses of each study were noted based on these specific criteria.

### Data extraction

Data from the included studies were extracted by two reviewers (SMA and FC), including patient demographics, population sample, measures of tendon structure, the number of tendons that became symptomatic among those with normal or abnormal imaging at baseline, and the definition of tendon abnormality provided in each study. Tendon abnormalities were described as any deviation in tendon structure; for example, hypoechogenicity, increased thickness or increased vascularity as seen on power Doppler US. Similarities in the outcome measures used, the tendons involved, the participants included and the prediction of future symptoms allowed for pooled analysis of the data. Where data were not available, or the methods required clarification, the corresponding authors for the original studies were contacted. If data on measures of tendon structure and the number of tendons that became symptomatic among those with normal or abnormal imaging at baseline were not available after contacting the study authors, the results were not included in the meta-analysis and results were instead reported descriptively.

### Data analysis

Data analysis was performed by a statistician (HP). Data were entered into the Review Manager (RevMan) meta-analysis software, V.5.3 (Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014). Fixed-effects models using the Mantel-Haenszel method were selected to compute pooled unadjusted relative risks (RRs) with CIs where studies had low or moderate heterogeneity, with random-effects models selected otherwise. Heterogeneity between studies was assessed using the $I^2$ statistic, where an $I^2$ of 30% or less is considered to indicate low heterogeneity and the cut-offs of $I^2>30\%$ and $I^2>50\%$ are indicative of moderate and substantial heterogeneity, respectively.[26] $I^2$ describes the percentage of total variation across studies that is due to heterogeneity rather than chance and seeks to determine whether there are genuine differences underlying the results of the studies (heterogeneity), or whether the variation in findings is compatible with chance alone (homogeneity).[26] Summary RRs are presented using forest plots, stratified by tendon type. Differences between the tendon subgroups were tested for significance using the $\chi^2$ test. Small study bias and publication bias were assessed with funnel plot analysis. The 5% level of significance was used throughout the analyses.

## RESULTS

### Identification of studies

The electronic search yielded a total of 6449 potentially relevant studies. Twenty-four full-text studies were identified as potentially relevant after screening the title and abstract of each

study. Six additional studies were removed after screening the full texts of the identified studies.[27–32] Searching the reference lists of these full-text studies led to one additional study,[14] leaving 19 studies for final review. Following communication with one author of several eligible studies, two of the three shortlisted studies from his group were excluded, as there was overlapping data between the studies.[33–35] Consequently, the final number of studies for inclusion was 17.[10 14–16 35–46] The identification procedure is presented in figure 1.

## Characteristics of the included studies

A detailed description of the selected studies is outlined in table 1. The mean ages of the participants were similar across the included studies with participant's ages ranging from 15 to 66 years. In relation to the participant characteristics, all but 1 of the 17 included studies were carried out within sporting populations. Specifically, five involved volleyball,[35 38 39 41 44] two involved basketball,[16 36] three involved soccer players,[14 42 45] one involved fencing,[12] one involved badminton,[46] one involved ballet dancers,[10] two involved running[15 43] and one involved various sporting populations.[37] The remaining study[16] was performed on participants from the general population referred to a sports medicine centre. Twelve of the included studies included both male and female participants; four used male participants only,[14 37 42 45] while the remaining study[16] used female participants only. The length of the follow-up period ranged from five days to four years. The majority of studies used US as the modality of choice to investigate tendon structure, with only one study using Ultrasound Tissue Characterisation (UTC)[41] In relation to the tendon structures investigated over the prospective period, nine studies investigated hypoechogenicity, thickness and vascularity,[10 12 15 38–40 42–44] six investigated only hypoechogenicity and thickness,[14 16 35–37 45] while the remaining two studies investigated only vascularity[46] and hypoechogenicity,[41] respectively. There was a large variation in the clinical measures of pain and/or function used among the included studies ranging from subjective reporting of pain, measures of function (eg, VISA scale), time absent from sport, performance measures (single leg squat and plyometric movements), as well as clinical measures such as tenderness on palpation.

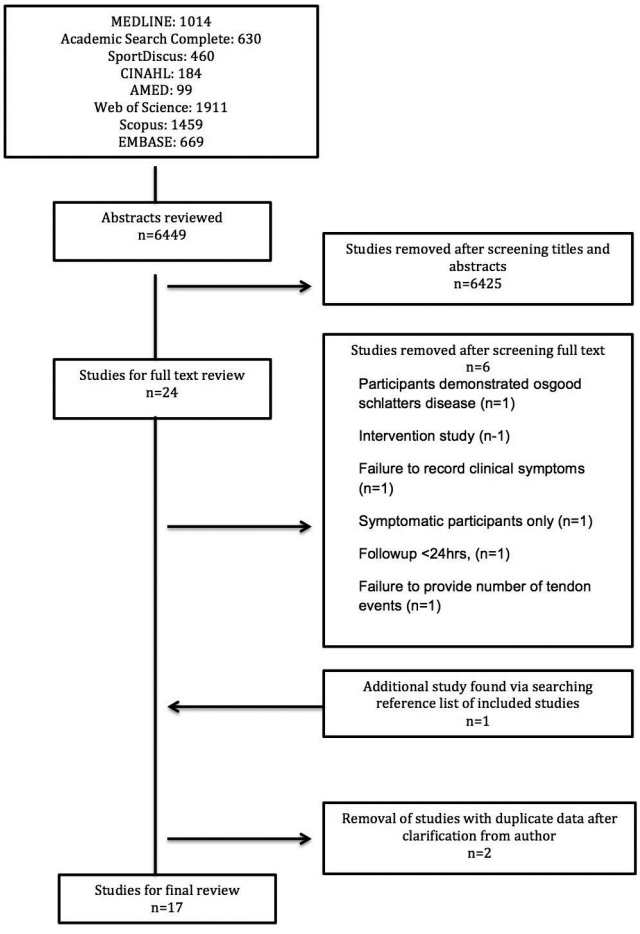


**Figure 1** Flow chart of included studies.

## Methodological study quality

The critical appraisal of included studies using the CASP checklist is shown in table 2. Overall, most studies satisfied the criteria outlined. All of the included studies used a representative study population, and recruited participants using predefined appropriate inclusion criteria. One of the main weaknesses of the included studies[10 14 42] involved the control of confounding factors, such as the use of blinding and/or control of training load. In relation to appropriate follow-up of the included participants, there was a variation in the methodological quality. Six of the included studies reported dropouts between baseline and follow-up.[12 15 39 42 44 46] However, only one of these studies[46] described the characteristics of the participants who dropped out. A final methodological concern is the aforementioned large variation in how pain and/or function were measured. Since some studies did not meet some of the criteria, the completeness, interpretation and generalisability of the results may each have been affected. However, in general, in all of the included studies, all had clear aims, used sound methodological quality and used appropriate study designs.

## Meta-analysis

Twelve of the 17 studies were eligible for meta-analysis due to similarities in study characteristics.[12 14 16 35–43] The remaining five studies could not be included in the meta-analysis due to insufficient data provided on the number of tendons that became symptomatic at follow-up.

## Does US predict lower limb tendinopathy (Achilles and patellar combined)

Tendon abnormalities on US are predictive of the development of future symptoms of patellar or Achilles tendinopathy (RR=4.97, 95% CI 3.20 to 7.73). In relation to the combined overall number of tendons that developed patellar or Achilles tendinopathy, figure 2 demonstrates that 21% (49/229) of asymptomatic abnormal tendons developed symptoms versus 3% (19/635) of asymptomatic structurally normal tendons. In addition, observed $I^2$ values of 14% indicated low levels of heterogeneity.[47] Data analysis revealed no evidence of systematic publication bias, which is highlighted in the funnel plot in figure 3.

## Does US predict patellar tendinopathy?

Thirteen studies investigated the predictive role of US imaging in the development of future patellar tendinopathy. Nine of these studies were included in the meta-analysis.[12 14 16 35–39 41] Patellar tendon abnormalities at baseline were found to increase the risk of development of future patellar tendinopathy (RR=4.35, 95% CI 2.62 to 7.23). In addition, observed $I^2$

**Table 1** Characteristics of included studies

| Author | Participant Demographics | Population | Tendon | Parameter investigated | Tendon abnormality definition |
|---|---|---|---|---|---|
| Boesen *et al*[46] | n=86 (56M/30F)<br>Mean age 21.7 | Semiprofessional badminton players | Achilles and patellar | Vascularity | *Abnormal:* Presence of increased vascularity (≥grade 1) according to a defined six-point scale (grade 0–5). |
| Comin *et al*[10] | n=79 (35M/44F)<br>Mean age 27.4 | Professional ballet dancers | Achilles and patellar | Thickness<br>Hypoechogenicity<br>Vascularity<br>Tendon clefts<br>Intratendinous calcifications | *Abnormal:* Presence of (1) hypoechogenicity, or (2) increased thickness, or (3) vascularity, or (4) intratendinous calcifications (all undefined) |
| Cook *et al*[36] | n=26 (8M/18F)<br>Age range 14–18 | Elite junior basketball players | Patellar | Thickness<br>Hypoechogenicity | *Abnormal:* Presence of (1) thickness, or (2) hypoechogenicity (all undefined). |
| Cook *et al*[37] | n=24 (24M)<br>Mean age 27.5 | Athletes from various sports: basketball, cricket, netball and Australian rules football. | Patellar | Thickness<br>Hypoechogenicity | *Abnormal:* Presence of (1) thickness, or (2) hypoechogenicity (all undefined). |
| Fredberg and Bolvig[14] | n=54 (54M)<br>Age range 18–35 | Professional soccer players | Achilles and patellar | Thickness<br>Hypoechogenicity | *Abnormal:* Presence of (1) thickening >1 mm, or (2) hypoechogenicity >1 mm. |
| Fredberg *et al*[45] | n=207 (207M)<br>Mean age 25 | Professional soccer players | Achilles and patellar | Thickness<br>Hypoechogenicity | *Abnormal:* Presence of (1) thickness >0.5 mm in the Achilles and patellar tendon, or (2) hypoechogenicity >0.5 mm in the Achilles tendon and >1 mm in the patellar tendon. |
| Giombini *et al*[12] | n=37 (15M/22F)<br>Mean age 27.2 | Elite fencers | Achilles and patellar | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness (undefined), or (2) hypoechogenicity (undefined), or (3) increased vascularity (≥stage 2) as defined by Gisslen *et al*[39] |
| Gisslén *et al*[38] | n=22 (11M/11F)<br>Mean age 16.25 | Elite junior volleyball players | Patellar | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness (undefined), or (2) hypoechogenicity (undefined), or (3) vascularity (≥stage 2) to a defined four-point scale (grade 0–3). |
| Gisslén *et al*[39] | n=60 (29M/31F)<br>Mean age 17.6 | Junior volleyball players | Patellar | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness (undefined), or (2) hypoechogenicity (undefined), or (3) vascularity (≥stage 2) according to a defined four-point scale (grade 0–3). |
| Hirschmuller *et al*[15] | n=634 (425M/209F)<br>Mean age 41.2 | Long-distance runners | Achilles | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness (undefined), or (2) hypoechogenicity (undefined), or (3) presence of vascularity according to a defined five-point scale. |
| Jhingan *et al*[42] | n=18 (18M)<br>Mean age 23.5 | Elite soccer players | Achilles | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness > 1 mm, or (2) hypoechogenicity >1 mm, or (3) paratenon blurring, or (4) vascularity (undefined). |
| Khan *et al*[40] | n=45 (27M/18F)<br>Mean age 42 | Patients from a university sports medicine centre | Achilles | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness >6 mm, or (2) hypoechogenicity (undefined)<br>Presence of the above features were graded according to a defined three-point scale. |
| Khan *et al*[16] | n=30 (30F)<br>Mean age 24.05 | Basketball players | Patellar | Thickness<br>Hypoechogenicity | *Abnormal:* Presence of (1) increased thickness (undefined), or (2) hypoechogenicity (undefined) |
| Malliaras *et al*[34] | n=58 (36M/22F)<br>Mean age 26.1 | Elite and recreational volleyball players | Patellar | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased thickness (undefined), or (2) hypoechogenicity (undefined), or (3) vascularity of at least one vessel in the sagittal plane >1 mm in length. |
| Ooi *et al*[43] | n=41 (25M/16F)<br>Mean age 37.25 | Marathon runners | Achilles | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) increased tendon thickness (undefined), or (2) hypoechogenicity (≥grade 2) according to a defined three-point scale (grade 1–3), or (3) vascularity (≥grade 2) according to a defined three-point scale (grade 1–3). |
| van Ark *et al*[41] | n=41 (30M/11F)<br>Mean age 17.2 | Elite junior volleyball players | Patellar | Hypoechogenicity | *Abnormal:* Presence of hypoechogenicity (undefined). |
| Visnes *et al*[44] | n=158 (84M/74F)<br>Mean age 17 | Elite junior volleyball players | Patellar | Thickness<br>Hypoechogenicity<br>Vascularity | *Abnormal:* Presence of (1) hypoechogenicity (undefined), or (2) increased vascularity (≥stage 2) as defined by Gisslen *et al* (2007) |

n= sample size; M= Male; F= Female; UTC= Ultrasound tissue characterisation.

**Table 2** Critical appraisal of included studies using the CASP cohort checklist

| Study | Q1 | Q2 | Q3 | Q4 | Q5 | Q6 | Q7 |
|---|---|---|---|---|---|---|---|
| Boesen *et al*[46] | ✓ | ✓ | ✓ | ✓ | × | ✓ | ✓ |
| Comin *et al*[10] | ✓ | ✓ | × | ✓ | ✓ | ✓ | ✓ |
| Cook *et al*[37] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Cook *et al*[36] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Fredberg *et al*[45] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Fredberg and Bolvig[14] | ✓ | ✓ | × | ✓ | ✓ | ✓ | ✓ |
| Giombini *et al*[12] | ✓ | ✓ | ✓ | × | ✓ | ✓ | ✓ |
| Gisslén *et al*[38] | ✓ | ✓ | ✓ | × | ✓ | ✓ | ✓ |
| Gisslén *et al*[39] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Hirschmuller *et al*[15] | ✓ | ✓ | ✓ | × | ✓ | ✓ | ✓ |
| Jhingan *et al*[42] | ✓ | ✓ | × | × | ✓ | ✓ | ✓ |
| Kahn *et al*[16] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Khan *et al*[40] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Malliaras [34] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Ooi *et al*[43] | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| van Ark *et al*[41] | ✓ | ✓ | ✓ | ✓ | × | ✓ | ✓ |
| Visnes *et al*[44] | ✓ | ✓ | ✓ | × | ✓ | ✓ | ✓ |

CASP, Critical Appraisal Skills Programme.

values of 1% indicated low levels of heterogeneity.[47] These figures are displayed in figure 2.

The remaining four patellar tendon studies included in this review could not be included in the meta-analysis, and instead are reported descriptively.[10 44–46] Fredberg *et al*[45] reported that the RR for developing patellar tendinopathy was 2.2 in those with structural abnormalities at baseline; however, this did not reach statistical significance (95% CI 0.90 to 5.7; p>0.05). Visnes *et al*[44] reported that a baseline finding of a hypoechoic area increased the risk (odds ratio (OR) 3.3, 95% CI 1.10 to 9.2, p<0.05) of developing patellar tendinopathy, while Comin *et al*[10] also concluded that there was an association between the presence of hypoechogenicity at baseline and an increased incidence of patellar tendinopathy (OR 6.3, 95% CI 1.30 to 30.8, p<0.05). The remaining study by Boesen *et al*[46] investigated the predictive value of vascularity in the development of future patellar tendinopathy, and found that vascularity did not predict symptomatic outcome during an 8-month-long badminton season.

In relation to the overall number of events, 21% (33/159) of abnormal asymptomatic patellar tendons became symptomatic versus 4% (16/431) of structurally normal asymptomatic patellar tendons.

### Does US predict Achilles tendinopathy?

Nine studies investigated the predictive role of US imaging in the development of Achilles tendinopathy. Five of these studies were included in a meta-analysis.[12 14 40 42 43] Achilles tendon abnormalities at baseline were associated with an increased risk of development of future Achilles tendinopathy (RR=7.33, 95% CI 2.95 to 18.24). In addition, observed $I^2$ values of 27% again indicated low levels of heterogeneity.[47] These figures are displayed in figure 2.

The remaining four Achilles tendon studies[10 15 45 46] could not be included in the meta-analysis. A relationship between the presence of Achilles tendon abnormalities and the development of future Achilles tendinopathy was reported by both Comin *et al*[10] (OR=6.3, 95% CI 1.30 to 30.8, p<0.05) and Fredberg *et al*[45] (RR of 2.8, 95% CI 1.60 to 4.9; p<0.05). Hirschmuller *et al*[15] found that the presence of neovascularisation was associated with a sevenfold (OR=6.9, 95% CI 2.60 to 18.8, p<0.05) increased risk of developing Achilles tendinopathy, while the predictive value of thickening was found to be just outside the values needed for statistical significance (p>0.05).

Finally, similar to the findings for patellar tendinopathy, one study by Boesen *et al*[46] found that intratendinous flow did not predict the development of Achilles tendinopathy during an 8-month-long badminton season. In relation to the overall number of events, 23% (16/70) of abnormal asymptomatic tendons became symptomatic versus 2% (3/204) of structurally normal asymptomatic tendons.

## DISCUSSION

### Main findings

The results of this review demonstrated a consistent pattern across the included studies of an increased risk of developing Achilles and patellar tendinopathy if tendons displayed abnormalities on US at baseline. The pooled RR statistics estimated tendons with an abnormal US to have a nearly fivefold increased risk of becoming symptomatic compared to tendons with a normal US.

### The burden of tendinopathy

The prevalence of Achilles tendinopathy is as high as 30% in runners while patellar tendinopathy is common particularly in jumping sports.[48] Despite the relatively high prevalence of lower limb tendinopathy, particularly in sporting populations, rehabilitation continues to be lengthy, with mixed outcomes often reported.[49–51] Consequently, this leads to frustration from the athlete's perspective and health practitioners' perspective. Furthermore, lengthy and sometimes unsuccessful rehabilitation in amateur and professional sporting domains lead to an increased financial burden on individuals and sporting organisations.

### The role of imaging in predicting tendinopathy: the case for

Given the substantial impact of Achilles and patellar tendinopathy, identification of 'at risk' athletes is a priority to try and prevent the negative consequences of tendinopathy on sporting participation and quality of life. MRI and US technology are imaging modalities of choice when visualising tendon dimensions.[52] In particular, US has gained increasing popularity among musculoskeletal practitioners and recent technical advancements have made US more affordable and accessible, particularly in the area of sports medicine and tendon disorders.[53] Results of this meta-analysis demonstrated a consistent trend that structural abnormalities at baseline are associated with the development of future tendinopathy. Thus, it is possible that structural changes in asymptomatic populations may represent markers of early presymptomatic pathology, which is later characterised by episodes of pain and/or reduced function. The strength and consistency of these findings have potentially important implications for the clinical management and prevention of patellar and Achilles tendon disorders. Visualising tendon abnormalities using US may identify potential at-risk athletes and may allow appropriate intervention through the implementation of preventative strategies, such as adapting training load[54] or appropriate tendon loading programmes.[45] However, evidence to support such strategies are lacking at this time, with future studies warranted. Similar to results in this review, a meta-analysis by Steffens *et al*[55] demonstrated that the presence of structural abnormalities, in particular the combined

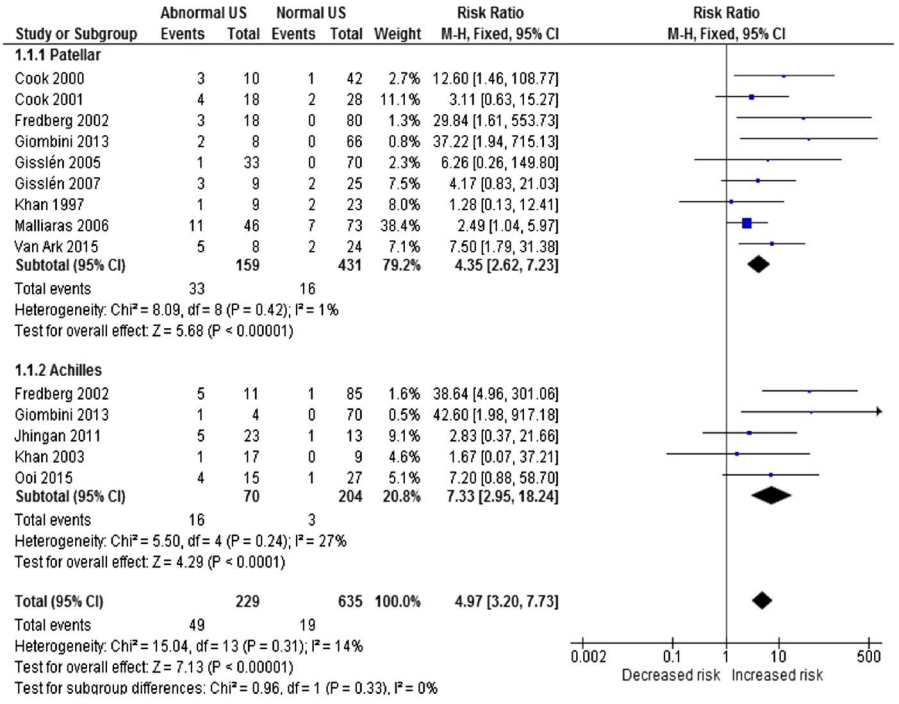


**Figure 2** Meta-analysis results for studies investigating the prediction of patellar and Achilles tendinopathy (combined and individual subgroup analysis).

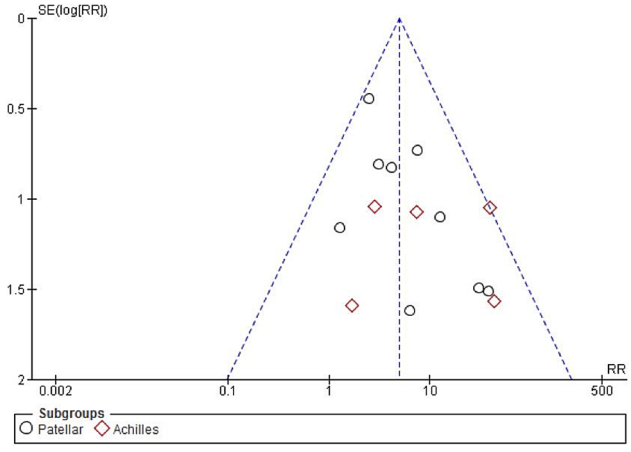


**Figure 3** Funnel plot analysis of study and publication bias.

number of abnormalities, was moderately predictive of future symptoms of LBP using MRI. Furthermore, in other health conditions such as cardiovascular disease, structural pathology in the form of atherosclerosis has been shown to be a predictor in the aetiology of the disorder.[56]

## The role of imaging in predicting tendinopathy: the case against

Intervening in all cases of structurally abnormal tendons identified using US imaging may not be warranted given the overall poor correlation between structure and pain in tendinopathy. Tendon structural abnormalities have been reported in a large percentage of asymptomatic individuals[57] with studies suggesting that tendon abnormalities are present in many as 59% of asymptomatic populations,[57] particularly increasing with age and sporting involvement. Structural abnormalities in asymptomatic populations are not limited to tendons, with a wide variety of studies demonstrating structural changes in asymptomatic sporting populations throughout the body. Papavasiliou *et al*[18] investigated structural changes of the hip in asymptomatic gymnasts using MRI, indicating that up to 63% of the sample group displayed signs of hip 'impingement' on imaging. These findings are replicated through numerous regions of the body among sporting populations with structural abnormalities present in as many as 89% of asymptomatic athletes across the shoulder, knee, hip and spine.[20–22 58]

The Cochrane guidelines for interpreting RR ratios advise that the clinical importance of a given RR ratio cannot be assessed without knowledge of the typical risk of events without treatment. This review indicates that the overall number of normal tendons that developed symptoms was small (19/635=3%), while the meta-analysis estimates that the overall risk of abnormal tendons developing symptoms is 4.97 times higher. Consequently, the actual risk of asymptomatic athletes with a structural abnormality in their tendon developing tendinopathy is only ~15%. Thus, theoretically in a sample of 100 athletes, as many as 59[57] of these will have asymptomatic structure on US, and as few as 9 of these 59 athletes may only go on to develop future symptoms. Consequently, although there is a clear statistical relationship between tendon abnormalities and development of future symptoms, it is only a moderate one. One may therefore argue that routine imaging in athletic populations may be inappropriate, time-consuming and costly to perform in every athlete. Perhaps tendon abnormalities in asymptomatic populations may simply indicate necessary structural adaptations to the sporting demands and loads placed on them.

There is also evidence to suggest that intervening with unnecessary imaging can actually result in adverse and harmful effects on patient's beliefs and behaviours.[59] By way of illustration, one study involving low-risk patients with LBP demonstrated worse overall outcomes in relation to pain and overall health in those who underwent imaging compared to those who did not receive any imaging.[60] Unfortunately, there is no comparable research until now in relation to tendon disorders, and making direct comparisons between LBP and tendon disorders

should be carried out with caution. The growing popularity and potential advantages of modalities such as UTC or elastography offer the potential to better visualise tendon structure. However with advanced visalisation comes the need to weigh up the potential negative consequences of making an athlete feel that their body is vulnerable based on what may be normal physiological responses to the loading demands. Perhaps this indicates the fundamental role of a clinician's communication strategies when relaying imaging findings to athletic populations. This reflects the complex nature, and our limited understanding, of tendinopathy.[57]

### Clinical implications: to image or not to image

While structural abnormalities are predictive of the development of future tendinopathy, how to interpret this relationship in the clinical setting is difficult given the high percentage of abnormalities in asymptomatic populations. Therefore, it may be the case that structural abnormalities should be considered as just one of several risk factors used to predict the development of tendinopathy as opposed to a sole predictor. Numerous other intrinsic and extrinsic risk factors have been identified for the development of tendinopathy. Training volume and frequency,[61] higher impacts caused by faster training,[62] as well as a change in surface density and shock absorption,[63] have all been proposed as extrinsic risk factors. In relation to intrinsic risk factors, altered foot function,[63] reduced ankle dorsiflexion,[64] sex,[36] diabetes,[65] adiposity,[66] muscle weakness[67] and genetic factors[68] have all been proposed. Unfortunately, the majority of studies investigating these factors have been cross-sectional, leading to an inability to clarify a direct cause and effect relationship. As a result, despite advancements in our understanding of lower limb tendinopathy, how these factors might combine to predict the development of tendinopathy remains unclear.

Given the high proportion of structural abnormalities in asymptomatic populations, one may argue that routine imaging may be costly and time-consuming. However, given the financial burden and the potential career-threatening impact of tendinopathy in athletic populations,[69] any modality which facilitates the identification of athletes at higher risk of injury may outweigh the financial or time-consuming nature of routine imaging.

Increasing recognition of the limitations of the biomedical structure-pain model has led to calls to investigate sports injury and injury prevention from a biopsychosocial viewpoint, where aspects other than local tissue damage, such as sociodemographic, psychological, lifestyle and social factors, are considered.[70] Psychosocial issues such as sleep,[71] disturbed breaks,[72] fatigue[72] and anxiety,[73] all have been demonstrated as risk factors for the development of injury in sporting populations. However, despite the importance of intrinsic psychosocial factors in the development of injury, there has been a reluctance to combine these factors with other traditional biological risk factors such as biomechanics, strength or training load in injury prediction studies. Prospective studies monitoring traditional risk factors such as tendon imaging in conjunction with some of the psychosocial factors outlined above may provide some much needed clarity and knowledge in the difficult area of tendinopathy, or perhaps even sports injury prevention in general.

### Limitations

One of the most frequently cited criticisms in relation to US imaging is in relation to its reliability. In comparison to MRI, US is perceived to have a higher risk of error or variance when evaluating tendon dimensions, due to factors such as operator inexperience, non-standardised imaging protocols and variations in transducer positioning.[74] Although there was a large variation in the operator experience in the included studies, a recent systematic review[75] demonstrated that US displays good to excellent levels of inter-rater and intra-rater reliability in measuring tendon thickness and cross-sectional area. Furthermore, Smith *et al*[76] concluded that US imaging was reliable in diagnosing partial and full thickness tears of the rotator cuff. Emerging imaging modalities such as UTC or sonoelastography are gaining popularity in the management of tendon disorders and may provide new insights into tendon structure by providing higher resolution and quantifying tendon structure more objectively compared to traditional US.[57] Furthermore, while MRI is frequently used to visualise tendon structure, this review looked only at the role of US imaging.

Another potential limitation of this review centres on the variability of terminology used in defining what is accepted as a structurally 'abnormal' tendon when viewed using US. For example, Comin *et al*[10] used the presence (mild, moderate, severe) or absence of hypoechoicity to define abnormality. Three studies defined tendon thickening >1 mm as abnormal.[14 42 45] One study required thickening >3 mm to classify a tendon as abnormal,[15] while another study required thickening of >6 mm to classify a tendon as abnormal.[40] Thus, the large variability on what constitutes an abnormal tendon across the included studies raises the possibility of overestimation or underestimation of the relationship between structure and future symptoms. Another limitation is in relation to the population investigated in the included studies; the majority of the included studies investigated athletic populations. Given the incidence of Achilles and patellar tendinopathy in non-athletic populations, caution is advised when extrapolating findings to non-athletic populations.

A further potential issue is the lack of gold standard tests for diagnosing tendinopathy. There is a lack of consensus within the research and clinical fields as regards the most appropriate clinical diagnostic test for tendinopathy. The large variation in diagnostic tests used in the studies in this review, as well as the lack of a widely accepted gold standard test, contributes to the difficulty in assessing the diagnostic value of US findings. Finally, a number of studies in the review investigated the predictive value of tendon vascularity using Doppler US. Temperature is an important confounding factor when using Doppler settings.[77] However, none of the included studies using Doppler US indicated controlling for this confounding factor. Nevertheless, only one study relied on vascularity as the only measure, such that this concern is unlikely to have affected the findings.

## CONCLUSIONS

The results of this systematic review and meta-analysis found that tendon abnormalities are predictive of the development of future Achilles or patellar tendinopathy. This may have potentially important clinical implications in the prevention and management of tendon disorders. However, this association between tendon abnormalities and the development of future symptoms was only moderate. Furthermore, given the high percentage of tendon abnormalities in asymptomatic tendons, imaging findings should only be considered as one component of the clinical prediction of tendinopathy.

## What are the findings?

- Asymptomatic athletes with tendon abnormalities at baseline are more likely to develop Achilles or patellar tendinopathy compared to those with structurally normal tendons.
- The likelihood of developing Achilles or patellar tendinopathy was nearly five times greater in those with tendon abnormalities (RR 4.97).

## How might it impact on clinical practice in the future?

- Since the relationship between structural abnormalities and future symptoms was only moderate, structural abnormalities should not be considered as a sole predictor of the development of tendinopathy.
- Combining the monitoring of tendon imaging with other factors linked to the development of pain, such as training load and psychosocial health, may enhance the management regarding the prediction of Achilles or patellar tendinopathy.

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