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**4TH INTERNATIONAL SCIENTIFIC
TENDINOPATHY SYMPOSIUM**
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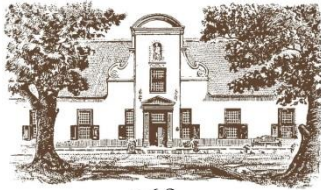
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O1: Persistence of tendon inflammation: a leading role for the stroma?

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Introduction: The mechanisms underpinning the development of chronic inflammation in tendinopathy are poorly understood. Recent work has highlighted the plasticity of macrophage activation signatures in human supraspinatus tendinopathy (1), however the contribution of resident stromal cells to the inflammatory response has not been investigated. Stromal activation is reported in Rheumatoid Arthritis in which resident stromal cells fail to switch off their inflammatory programme (2). In this study, we investigate the potential role of tendon stromal cells as an important population implicated in the development of chronic inflammation.

Methods: We studied stromal activation signatures in tendons obtained from patients before and after sub-acromial decompression treatment. Some patients became pain-free after treatment and in some pain persisted. Tendons were classified as early, intermediate or advanced stage disease. Healthy tendons were collected from patients undergoing shoulder stabilisation surgery or ACL repair. Markers of stromal activation (gp38/podoplanin, CD248 and VCAM) were investigated by flow cytometry and immunostaining of tendon tissues. Gene array, qPCR and flow cytometry were used to investigate stromal activation *in vitro*.

Results: Diseased tendon tissues showed increased gp38, CD248 and VCAM mRNA compared to healthy supraspinatus tendons ($p=0.01$). This stromal activation signature persisted in painful and pain-free patients 2-4 years after treatment. Diseased cells showed increased basal gp38, CD248 and VCAM mRNA compared to healthy cells ($p<0.05$). Treatment of healthy and diseased cells with IL-1 β or TNF α increased gp38 and VCAM mRNA and protein. IL-1 β treated diseased cells showed increased gp38 mRNA compared to IL-1 β treated healthy cells ($p=0.03$). Cytokine treatment did not induce expression of CD248. Treatment of diseased cells with LPS increased expression of NF κ B and Interferon inducible target genes compared to LPS treated healthy cells.

Discussion: We demonstrate inflamed tendon tissues show increased expression of stromal fibroblast activation markers compared to healthy tendon tissues and cells. Cytokine stimulation induced markers of activation including gp38 and VCAM but not CD248, suggesting distinct stromal responses in diseased tendons. Persistence of the stromal activation signature after treatment suggests tendon stromal cells may be continuously primed for inflammation. Stromal activation and memory may be an important mechanism for the development of chronic inflammation and recurrent tendon disease.

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O2: Role of immune cells on tendon disease: insights from an in vitro model

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Introduction: The cellular mechanisms of tendinopathy remain unclear particularly with respect to the importance of inflammation in both the pathogenesis of tendinopathy and the healing process. Recent immuno-histochemical studies have revealed the presence of immune-competent cells in both early and chronic human tendinopathy. The evidence that inflammation coexists with degenerative changes in tendon opens questions regarding potential crosstalk between immune-cells and tendon fibroblasts (TFs). To shed light on this process we investigated the effects of polarized macrophage interaction with tendon cell behavior in a novel in vitro model of tendinopathy.

Methods: Primary human TFs were seeded on nanofiber polymer mats that were structured to mimic the characteristic matrix of healthy vs diseased tendon. A TF/macrophage transwell system was used, allowing exchange of soluble factors without direct cell-cell contact. Briefly, THP-1 monocytes, differentiated to macrophages (M0), were chemically polarized to the pro-inflammatory M1-like phenotype or the anti-inflammatory M2-like phenotype validated using surface markers CD197 (M1) and CD206 (M2). TF response was assessed using lineage-specific markers tenomodulin, scleraxis and mohawk, collagen I/III and matrix remodelling capacity as indicated by matrix metalloproteinases (MMP).

Results: The extracellular matrix structure highly affected TF morphology, with models of healthy matrix yielding highly elongated cells and model tendinopathic matrices yielding more polygonal cells spread over multiple fibers. No notable changes in tendon marker expression were detected between substrate types. On the tendinopathic substrate models, TF stimulation by pro-inflammatory cytokines increased MMP 1, 3, and 13 expressions, compared to those on healthy matrix models.

Discussion: We established a combined in vitro system to study the effect of different factors on tendon cells displaying normal and pathological matrix phenotype. Using this approach, we explored the effect of immune cells and factors secreted by immune cells on human primary TFs and observed increased expression of genes related with ECM turnover in 'pathological' TFs exposed to pro-inflammatory cytokines. These results are consistent with previously published reports of 'molecular inflammation'. Our minimal tendinopathy model driven by tendinopathic matrix structure indicates existence of crosstalk between immune cells and TFs that are relevant to understanding of tendon disease.

O3: Altered structure integrity is associated with pain and dysfunction in volleyball players with patellar tendinopathy

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Introduction: Altered tendon integrity has been reported in athletes with Achilles tendinopathy. The present project aimed to compare tendon integrity between healthy and volleyball athletes with patellar tendinopathy; as well as to explore possible relationship between abnormal tendon integrity, pain and dysfunctions in those with patellar tendinopathy.

Methods: Twenty-two elite volleyball players (mean age =20.7 ±1.; BMI=32.0 ± 6.2; 8 male; mean training hour per week = 30) were recruited. Fourteen had patellar tendinopathy (5 had unilateral patellar tendinopathy and 9 had bilateral tendinopathy) determined by clinical assessments. Both

knees were scanned using the ultrasound tissue characterization system. Four echo types were discriminated and expressed as a percentage. Visual analogue scale (VAS) was used in quantifying the intensity of pain on palpation and during single-legged declined- squat test. The Victorian Institute of Sports Assessment Scale (VISA-p) was used in measurement dysfunctions.

Results: Significant increase in the proportion of echo type III+ VI was observed in athletes with patellar tendinopathy than control ($p < 0.05$). The proportion of III+IV was increased from 12.1% to 28.1%; and from 12.9% to 26.3% in the right and left knees, respectively. Correlations were found between the proportion of echo III+IV and the VISA-p scale. Specifically, a trend of negative correlation was detected between the proportion of echo III+IV and scores of question 4 of the visa-p scale (pain when doing a full weight-bearing lunge) ($\rho = -0.51$, $p = 0.078$) in 13 subjects with right patellar tendinopathy and the VISA-P scores ($\rho = 0.61$, $p = 0.059$) in 10 subjects with left tendinopathy.

Discussion: Increase in abnormal tendon structure was observed in elite volleyball players with patellar tendinopathy. These athletes lived in a training institute and trained 5 days a week and 6 hours a day. Most of the players with tendinopathic tendon still practiced with pain; and were able to squat fully without pain. During full-weight forward lunge, the activity-induced patellar pain had a trend of relationship with the abnormal tendon structure in subjects with right patellar tendinopathy. These findings suggest that more severe structural tendon changes show a trend towards a relationship with pain during tendon loading.

O4: Altered structure integrity is associated with pain and dysfunction in junior elite athletes with patellar tendinopathy

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Introduction: Altered tendon integrity has been reported in adult athletes. Whether similar phenomenon happens in junior elite athletes is unknown. Although an increase in abnormal tendon structure has been observed in subjects with tendinopathy, the relationship between tendon structure, pain and dysfunction is unclear. The present project aimed to compare tendon integrity between junior healthy and athletes with patellar tendinopathy and to explore possible relationships between abnormal tendon integrity, pain and dysfunctions in junior elite athletes with patellar tendinopathy.

Methods: Thirty-seven junior elite athletes (age ranged from 14-18 years; 26 male; mean training hour per week = 30; 19 volleyball players and 18 weight lifting athletes) were recruited. Fourteen of them had patellar tendinopathy (5 had unilateral right patellar tendinopathy and 9 had bilateral tendinopathy) determined by clinical assessments. The right knee was scanned using the ultrasound tissue characterization system. Four echo types were discriminated and expressed as a percentage. Visual analogue scale (VAS) was used in quantifying the intensity of pain on palpation and during single-legged declined-squat test. The Victorian Institute of Sports Assessment Scale (VISA-p) was used in measurement dysfunctions.

Results: Significant increase in the proportion of echo type III+ VI was observed in athletes with patellar tendinopathy than the control ($p < 0.05$). Echo III+IV was increased from 11% in the healthy to 24% in the tendinopathic tendons. Positive correlations were found between echo III+IV and intensity of pressure pain ($\rho = 0.81$, $p = 0.000$) and intensity of single-legged decline-squat test pain ($\rho = 0.83$, $p = 0.000$); as well as negatively correlated with VISA-p score ($\rho = -0.55$, $p = 0.053$).

Discussion: Altered tendon structure was observed in junior elite athletes with patellar tendinopathy. The athletes lived in a training institute and trained 5 days a week with same training intensity. Those with patellar tendinopathy had not modified their training schedule. However, abnormal tendon structure was associated with pain and dysfunctions in these athletes. Our findings suggest more severe changes in structure are related to pain and dysfunction in the junior elite athletes.

O5: Mechanical properties of the Plantaris and Achilles Tendons: a contributing factor to non-insertional Achilles Tendinopathy?

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Introduction: The plantaris tendon (PT) has been implicated in non-insertional Achilles tendinopathy (NIAT), but its precise role is not clearly understood. It is hypothesised to friction against the medial Achilles, potentially causing a neuro-inflammatory response. Different mechanical properties of the tendons could be a causative factor, resulting in elongation differences between the two tendons in response to loading. A prior study found PT is stiffer than Achilles Tendon(AT) however Young's Modulus was not reported, since testing equipment limited loading to 200N. Thus PT was only compared to sectioned parts of the AT, likely affecting its behaviour given its twisted structure. The purpose of this work was to define the mechanical properties of whole AT and PT taken from young specimens, with normal tendon morphology.

Methods: An ultrasound tissue characterisation scan (UTC) was performed on ten fresh frozen cadaveric ankles (mean age=32±7(M±SD) range:22-39; Female=6). PT and AT were dissected out and cross-sectional area recorded using a validated alginate mould and digital photography method. Tendons were individually tensile tested to failure at 1,000 mm/min, by gripping ends in freezing clamps, using an Instron machine (Instron Limited, UK). Sutures were positioned 50mm apart on the tendons, 10mm proximal and distal to the freezing clamp edge and tracked using a high speed video camera synchronised with the load values.

Results: Cross sectional area (CSA) of AT(68±14mm²(mean±SD)) was significantly greater than PT (3±1mm²)($P<0.01$). UTC scans showed a mean 89% organised tendon in AT 2-4cm from the calcaneal insertion. Stress and strain curves were plotted and Young's Modulus calculated. PT had a significantly higher modulus of elasticity (1.5GPa±0.3) than AT (0.9GPa±0.4)($P<0.001$). Fracture forces for the AT (3.6kN±0.5) were significantly higher than PT (0.3kN±0.1) due to CSA differences ($P<0.01$).

Conclusion: Accounting for CSA, young healthy PT were significantly stiffer than AT. This is the first time Young's modulus has been reported on same specimen, intact AT and PT, using high frequency sampling (1000Hz) and rapid tendon loading(1000 mm/min). Findings explain why clinically the AT may rupture and the PT remains intact, adding support to the premise of a friction phenomenon between AT and PT in a sub-population of patients with NIAT.

O6: Can imaging diagnose and predict the morbidity of Achilles and patellar tendinopathy in elite Australian football players?

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Introduction: The relevance of imaging in tendinopathy is widely debated due to the large proportion of asymptomatic pathology. Furthermore, there is controversy regarding whether normalisation of tendon structure is required for clinical improvements in pain. This study aimed to investigate whether

the presence of pathology, amount of disorganisation, or changes in tendon structure over time predict the presence, or magnitude of symptoms, in Achilles and patellar tendinopathy.

Methods: 474 elite male Australian football players were recruited. All players completed the OSTRC overuse questionnaire monthly during the pre- and competitive season to quantify the impact of Achilles and/or patellar tendinopathy on participation and performance in training and games. A subset of 193 participants underwent ultrasound tissue characterisation (UTC) scans of their Achilles and patellar tendons at baseline, monthly intervals throughout the three month pre-season, and end of season. The presence of pathology on grey-scale ultrasound (hypoechoic area and/or tendon thickening) was assessed, as well as the percentage of disorganised structure (DIS) quantified using UTC. Risk factor analysis was performed to identify any associations between the presence of pathology and symptoms. Linear regression was performed to identify any potential relationship between the percentage of DIS and OSTRC score.

Results: 13.3% and 16.5% of participants reported some level of Achilles or patellar tendon morbidity respectively. 66 players (40.5% of those scanned) exhibited a pathological patellar tendon on grey-scale US, yet 26 of these players (39.4%) were asymptomatic. Players with pathology on grey scale ultrasound at the start of the season were 7.3 (CI 3.6-14.7) times more likely to have symptoms. Based on UTC findings, participants with more than 5.8% of DIS structure in the patellar tendon were 5.1 (CI 1.2- 20.8) times more likely to have their participation and performance in training and games affected. No relationship was observed between percentage of DIS structure and the OSTRC overuse score ($p=0.977$, $r=0.011$).

Discussion: The presence of structural abnormalities was a risk factor for reduced participation and performance in training and games. In this population, imaging should not be used diagnostically due to the high proportion of asymptomatic tendon pathology and poor positive predictive value

O7: Quantifying tendon stiffness in Achilles and Patellar tendinopathy and healthy controls using shear wave elastography: A blinded, cross-sectional investigation.

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Introduction: Tendon injuries are characterized by changes in composition and structure, which may alter tendon mechanical properties. Shear wave elastography is a relatively new ultrasound-based, non-invasive technique, which can be used to quantitatively assess the shear modulus or stiffness of tendon. To date, there is limited evidence from separate studies suggesting increased and decreased stiffness in patellar and Achilles tendinopathy respectively.

Methods: A cross-sectional study was used to compare healthy individuals with those currently experiencing symptomatic (unilateral or bilateral) Achilles tendinopathy (AT) or patellar tendinopathy (PT). B-mode and elastography was performed using an Aixplorer ultrasound scanner (Supersonic Imagine version 8.2). An examiner blinded to the presence (and region) of tendinopathy performed bilateral measures of patellar and Achilles tendons, at both mid-tendon and insertional regions, in a randomised order. Elastography images were processed offline using customised Matlab scripts by a second researcher blinded to tendinopathy status. Repeated measures ANCOVA were used to examine the effect of condition (PT v C or AT v C) and region (mid v insertional) on each measure. Where significant interaction was found post-hoc testing was performed using generalised linear models. Age, sex and BMI were included in all models. Significance was set at 0.01.

Results: 67 participants (17 patellar tendinopathy, 22 Achilles tendinopathy, 28 controls) were recruited. Significant differences in age and BMI were found between groups, with AT group being

older and more overweight. Compared to controls, increased thickness (MD 1.7mm, 95%CI 0.5 to 2.9, $p=0.005$) and increased stiffness (MD 64.9KPa, 54.6 to 75.2, $p<0.001$) were found in the insertional (proximal) patellar tendon in patellar tendinopathy. Compared to controls, decreased stiffness (MD -27.2KPa, -43.1 to -11.3, $p<0.001$) was found at the Achilles insertion in Achilles tendinopathy, but not at the mid-tendon, where there was increased thickness (MD 1.9mm, 0.5 to 3.3, $p=0.01$). Achilles tendon stiffness was significantly negatively associated with age (B -0.5, 95% CI -0.9 to -0.1, $p=0.007$).

Conclusion: Regional changes in tendon thickness and stiffness are evident in Achilles and Patellar tendinopathy. Different mechanical demands and chronic tendon overload or underload may explain the increased stiffness in patellar and decreased stiffness in Achilles tendinopathy respectively.

O8: Evaluation of intratendinous displacement and strain in the Achilles tendon using quantitative high-frequency ultrasound imaging and an interactive clinician-friendly application.

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Introduction: Knowledge on in vivo mechanical tendon behavior is scarce. Previous research has provided insight in global as well as intratendinous tendon displacement, but information on in vivo intratendinous strain is absent. The objective of this study is to (a) investigate the reliability of a novel imaging technique using high-frequency ultrasound based speckle tracking, to establish a direct measure for local intratendinous displacement and strain. We hypothesize (b) non-uniform deformation with highest displacement in the deep layer, and highest strain in the superficial layer, when comparing superficial, middle and deep tendon layers.

Methods: A dynamic ultrasound of both Achilles tendons of 10 asymptomatic subjects was taken during 2 repetitions of a passive elongation and an isometric contraction. After segmentation and post-processing of the images, the intratendinous deformation was estimated by means of speckle tracking. Reliability of the method and non-uniform behavior (displacement and strain) between the three layers were evaluated. Intraclass correlation coefficients between two repetitions were evaluated separately for each leg, activation method (passive, isometric), response variable (displacement, strain) and layer (deep, middle, superficial).

Results: ICC's were overall acceptable, ranging from 0,57 to 0,94 for displacement in the superficial layer for both legs and activation methods. There was a statistically significant (alpha-level 0,05) non-uniform displacement when comparing the superficial, middle and deep layers, for passive as well as isometric activation ($p < 0,001$). Highest displacement was found in the deep layer. There was a statistically significant non-uniform strain, but only during passive activation, when comparing superficial, middle and deep layers (superficial versus middle: $p = 0,03$; superficial versus deep and middle versus deep: $p < 0,001$). Highest strain was found in the superficial layer.

Discussion: We have proven adequate reliability of this new technique and have confirmed previously found (1,2) non-uniform tendon behavior with highest displacement in the deep layer. Furthermore, we have shown a first insight in non-uniform strain behavior with highest strain in the superficial layer. This technique will improve the knowledge on etiology of tendinopathy, perhaps identify subjects at risk for developing pathology, and also provide options to optimize existing therapeutic loading programs.

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O9: The Epidemiology of Achilles tendinopathy in UK runners

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Introduction: Achilles tendinopathy affects 2% of the general population and 52% of ex-elite middle/long distance runners. Despite the high incidence levels reported in runners there have been few primary epidemiological studies and even fewer outside of elite sporting groups from various Scandinavian countries.

Aims

1. Determine the point and lifetime prevalence of Achilles tendinopathy within UK runners
2. Determine the associated factors for Achilles tendinopathy within UK runners

Methods: A self-reported historical cohort study was completed using a survey. The survey was developed based on a Delphi study of world tendon experts. The final survey was sent to 15,000 subscribers of a leading UK running magazine and was also run concurrently online. Binary logistic regression analysis was used to determine important variables.

Results: 1800 subjects responded to the study with 1475 subjects completing all necessary components. Lifetime prevalence of Achilles tendinopathy was 57%, point prevalence was 44%. Mid-portion tendinopathy was the most frequently reported (69%) with insertional (16%) and combined insertional and mid-portion (15%) accounting for the remaining %. The final logistic regression model comprised of statistically significant variables: number of years running, type of running, training surface, training terrain, weekly mileage, number of weekly runs, number of rest days, average running speed, Age, ankle ROM, calf stretching, previous calf pain, orthosis use, and lower limb strength training. This model had an accuracy of 65.1% with a sensitivity of 77.8% and a specificity of 47.7%.

Important extrinsic variables (Odds ratios and 95%CI) were: running on grass (1.63,0.16–16.76), training on flat terrain (1.49,0.27–8.27), regularly calf stretching (1.78,1.32–2.35), wearing prescribed (1.94,1.30–2.89) or off-the-shelf orthosis (1.56,1.18–2.07), and regularly strength training (1.44,1.17–1.77). Important intrinsic variables were: restricted ankle ROM (2.69,1.94–3.70) and regularly experiencing calf pain with running (1.80,1.43–2.25).

Discussion: Achilles tendinopathy affects large numbers of endurance runners. Extrinsic and intrinsic factors identified in this study should be used in further epidemiological studies, these variables may be important to consider in prevention studies. This study is the first to report previous calf pain, strength training and calf stretching as variables of interest in Achilles tendinopathy.

O10: Patellar tendon structure responds to load over a 7-week preseason in elite volleyball players.

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Introduction: Patellar tendinopathy is a common overuse injury in jumping sports, with higher incidence in volleyball players. Different risk factors are associated with patellar tendon problems, including training load (frequency, volume and intensity). Tendons show an adaptive or maladaptive response to load and the quality of its structure either improves or worsens. The aim of this study was

to investigate the relation between tendon load and the response of the patellar tendon structure of elite volleyball players.

Methods: Seventeen elite male volleyball players participated in this study. Volume (duration) and intensity (session Rating of Perceived Exertion (RPE)) were recorded every training and match by each player. Load was calculated by multiplying duration of the training and RPE. The cumulative load was defined by the sum of the weekly loads over 2 weeks before the UTC scan. Jump frequency was measured with accelerometers and with video notation analysis. To assess tendon structure changes, the Ultrasound Tissue Characterization (UTC) was used. The measures were assessed biweekly to quantify patellar tendon structure. Generalized estimating equation (GEE) models were used to test the relation between load parameters and echo pattern changes.

Results: On average, players spent 624 ± 215 minutes on training and matches per week with a RPE of 13.9 ± 2.1 indicating 'somewhat hard' intensity. Jump frequency was 278 ± 122 per week. Higher load parameters (volume, intensity and load) resulted all in a higher decrease of echo-type I ($p < 0.05$). Higher volume and load resulted in a higher decrease of echo-type II ($p < 0.05$). Higher cumulative weekly volume and cumulative weekly loads between the first and the last UTC were related to significant higher decreases in echo-type I ($p < 0.05$), but not related for echo-type II. No significant relations were found for jump frequency. None of the load parameters were related to echo-type III and IV.

Discussion: The present study showed that load was related to changes in echo-types over a 7 week preseason in elite volleyball players. A higher amount of load was related to greater echo pattern changes. Further research to the relation between load and echo pattern changes is needed.

O11: Immediate effects of altering sagittal plane trunk position during jump-landings in athletes with and without patellar tendinopathy

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Introduction: Pathomechanical models of tendinopathy emphasize overload as a key factor leading to the development of tendon pathology (1). Therefore, it seems reasonable that interventions aimed at reducing tendon load during sports-related tasks may help in rehabilitation and prevention of tendinopathy. Athletes with patellar tendon disorders use a stiffer landing pattern than asymptomatic controls (2). Sagittal plane trunk position during jump-landings might influence patellar tendon forces and symptoms in athletes with patellar tendinopathy. This study examined the effect of altering trunk position during jump-landings on lower limb biomechanics, patellar tendon force and pain in athletes with and without patellar tendinopathy.

Methods: Twenty-one elite and sub-elite male athletes were categorized into 3 groups, athletes with patellar tendinopathy (TG, $n=7$), asymptomatic athletes with patellar tendon abnormalities (AG, $n=7$) and asymptomatic athletes without tendon abnormalities (CG, $n=7$). Motion-analysis and force plate data were collected during drop-jump landings with a self-selected (SS), flexed (FLX) or extended trunk position (EXT). The latter two conditions were randomized. Sagittal plane peak kinematics, kinetics, patellar tendon force and pain during the landing tasks were calculated. Results were analysed with a 2-way repeated measures ANOVA.

Results: Peak patellar tendon force, knee extensor moment and knee pain decreased in the FLX landing compared to the SS landing, regardless of group. In addition, peak patellar tendon force, knee extensor moment and vertical ground reaction force were smaller in the FLX landing compared to the

EXT landing. The TG had smaller peak ankle dorsiflexion than the CG during jump-landings, regardless of trunk position.

Discussion: Landing with greater sagittal trunk flexion decreased patellar tendon force in elite jumping athletes. An immediate decrease in knee pain was also observed in symptomatic athletes with a more flexed trunk position during landing. Increasing trunk flexion during landing might be an important strategy to reduce tendon overload in jumping athletes. Increasing ankle dorsiflexion during landing might also be important in athletes with patellar tendinopathy.

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O12: Comparison of the mechanical properties between healthy tendon and tendon pathology using ultrasound elastography: A systematic review.

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Introduction: Tendon injuries are characterized by substantial changes in composition and structure which lead to altered tendon mechanical properties. Recent work suggests that direct or indirect estimation of tendon mechanical properties using ultrasound elastography may be useful in evaluation of tendon pathology, such as tendinopathy or tears. However large differences in technology, methodology and populations studied is evident between studies. A systematic review was performed to synthesise evidence and provide consensus regarding methodology, terminology and outcomes for future research.

Methods: Five databases were searched using the terms elastography or sonoelastography and tendon (Feb 2016). Studies published in English using ultrasound elastography to compare mechanical properties of tendon between people with or without tendon pathology (tendinopathy or tear), or between affected and contralateral unaffected tendons were included. Reviews, abstracts or case studies were excluded. Two independent reviewers determined eligibility, assessed methodological quality and extracted data.

Results: Abstracts and titles of 195 studies were identified for initial review. 16 studies (n=1010 individuals) met inclusion criteria, 13 of these provided comparison with a healthy control population. Achilles tendon pathology was most commonly studied (7 studies, n=468), followed by lateral epicondylalgia (4 studies, n=198), rotator cuff (3 studies n=276) and patellar tendinopathy (2 studies, n=68). The majority of studies used compression or strain elastography (13 studies, n=822) where static compression imposed by the operator induces strain within the tissue. Studies reported a qualitative grading of tendon softness/hardness or a relative strain ratio between the tendon and surrounding tissue. Most studies concluded that tendinopathy was associated with tendon softening, although 1 study of Achilles tendinopathy and 2 studies of post-surgical Achilles repair described affected tendons as hard. Only two studies used shear-wave elastography, where measurement of the speed of shear wave propagation through tendon enables quantitative estimation of tendon stiffness. These studies reported increased tendon stiffness for patellar tendinopathy while reduced stiffness for Achilles tendinopathy or tear.

Conclusion: Evidence suggests altered mechanical properties by tendinopathy may be detected using ultrasound elastography. This review provides recommendations to improve consensus regarding terminology and standardized application of qualitative and quantitative scoring methods.

O13: Do symptomatic Achilles tendons contain sufficient aligned fibrillar structure?

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Introduction: Ultrasound Tissue Characterisation (UTC) provides a detailed quantification of the tendon matrix, recently pathological tendons were shown to have sufficient aligned fibrillar structure (AFS) (1). However this study did not isolate symptomatic from the pathological tendons. It is possible that symptomatic tendons present a different pattern of structural integrity.

Aim

To assess the aligned and disorganised fibrillar structure (DFS) of symptomatic Achilles tendons compared to non-symptomatic tendons.

Methods: Participants with and without Achilles tendinopathy were recruited. 29 symptomatic tendons and 29 healthy tendons were included. Using UTC the area of greatest tendon echogenicity was identified and most affected 10mm section contoured. Contours were laid at 2mm intervals giving 50 contiguous transverse images which were automatically interpolated to form a tendon volume. Analysis was completed using window size 9. The mean cross-sectional area (mCSA) of AFS was quantified using UTC algorithms.

Results: The mCSA of AFS was 76.8 mm² (SD 28.5mm²) in the symptomatic tendons and 69.7mm² (14.2mm²) in the non-symptomatic tendons. The mCSA of the DFS was 40.9mm² (30.7mm²) and 4.7mm² (2.4mm²) respectively. mCSA of AFS was not significantly different ($p=0.237$) between the groups. mCSA of DFS was significantly different between the groups ($p<0.001$). A scatter plot of the mCSA of the symptomatic tendons plotted with the interquartile range (IQR) of the healthy tendons showed 21% (n=6) falling below the first quartile, 31% (n=9) falling within the middle two quarters and 48% (n=14) falling above the fourth quartile.

Discussion: Our study demonstrates large changes in DFS in symptomatic tendons compared with non-symptomatic tendons. Our study supports the previous findings of sufficient AFS in the majority of subjects (79%) but highlights a subgroup (21% of symptomatic tendons) that appear to have insufficient AFS. This is the first identification of this subgroup. The higher proportion with low AFS in our study compared with previous work may be due to analysing a smaller section around the lesion rather than the whole tendon, also our study included symptomatic rather than pathological tendons.

The group with low mCSA of AFS may be at risk of tendon rupture, further work is needed to address these findings.

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O14: Statin treatment is associated with trigger finger

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Introduction: Statin medication is known to have relatively few side-effects, but recent research have suggested potential side effects on tendons, with tendinopathy or ruptures as the final consequence (1,2). But firm evidence for an association is lacking. The aim of the present study was to determine whether statin treatment is associated with a specific tendon disorder, trigger finger.

Methods: Two population-based large Swedish cohorts, the Swedish mammography cohort (SMC; n = 52 220 women) and the cohort of Swedish men (COSM; n=40 713) were used for this study. A time-dependent Cox regression analysis was applied to determine the association between statin treatment and trigger finger. The multivariable analysis was adjusted for age, BMI, smoking, energy intake, corticosteroid use, quinolone use, diabetes mellitus and renal insufficiency.

Results: Statin use was common both among women (37%) and men (44%). The most common statin used was simvastatin (67%) followed by atorvastatin (24%). Women who had used statins had a higher incidence of trigger finger than those who had never used statins, with a crude HR of 1.44, and after multivariable-adjustment 1.21 (95% CI: 1.03-1.43). The risk among men was higher, HR 1.63 and after adjustment 1.42 (95% CI: 1.15-1.75). The risk seemed to be dose-independent. The highest risk was seen among users of rosuvastatin (HR among men 2.19, 95% CI: 1.33-3.62) and the lowest risk was associated with simvastatin (HR among men 1.33, 95% CI: 1.06-1.68). The increased risk was only seen with current use of statin (adjusted HR 1.5 among men, 95% CI: 1.21-1.85) but not among former users (adjusted HR 0.71, 95% CI: 0.38-1.31).

Discussion: Statin use increases the risk of trigger finger, especially among men. The highest risk was associated with the use of rosuvastatin. The risk appears to be linked to current use of statins and disappears after the treatment has been stopped.

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O15: Development and validation of the VISA-A(Sedentary) questionnaire: A modified version of the VISA-A for nonathletic patients with Achilles tendinopathy.

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Introduction: The VISA-A is a condition-specific outcome measure for Achilles tendinopathy. It was developed primarily for use in athletic individuals and may be less sensitive to clinical change in non-active individuals with Achilles tendinopathy.

Aim

To developed and validate a modified version of the VISA-A suitable for non-athletic individuals.

Methods: Limitations of VISA-A were identified through participant focus groups, and consultation with tendinopathy clinicians. The preliminary version of VISA-A(sedentary) ready for pilot testing was developed and refined until consensus was achieved among the authors.

Twenty-two non-athletic individuals with Achilles tendinopathy, treated with routine care at the Leicester Royal Infirmary, are included in this preliminary report. Participants completed both the VISA-A and VISA-A(sedentary) at baseline, at day 3, and at discharge. At discharge participants completed the global rating of change scale (GROC) to evaluate treatment success.

Results: Reliability of VISA-A(sedentary) was high (ICC 0.992, n=22). At discharge, VISA-A had improved by 7 points (95%CI 5.2 to 8.5, n=13) whilst the VISA-A(sedentary) improved by 14 (95%CI = 9.8 to 19.0). VISA-A(sedentary) correlated with GROC ($r=0.621$, $p=0.024$) whereas VISA-A did not ($r=0.412$, $p=0.162$).

Discussion: VISA-A(sedentary) shows excellent test-retest reliability, and appears to be more sensitive to change in a non-athletic population. The correlation with patient perception of treatment success suggests that this tool is a valid method of measure pain and functional limitation in non-athletic individuals with Achilles tendinopathy.

O16: Increased upper trapezius muscle stiffness is associated with less reduction of the subacromial space in athletes with rotator cuff tendinopathy

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Introduction: Reduction of subacromial space (SAS) during arm elevation may contribute to rotator cuff tendinopathy. Excessive tension of the upper trapezius (UT) was suggested as either a compensatory strategy used to preserve the SAS or aggravating factor to rotator cuff tendinopathy. This study aimed to determine whether the UT muscle stiffness is altered in athletes with rotator cuff tendinopathy compared to asymptomatic athletes and to investigate the relationship between the change in UT muscle stiffness and the SAS during shoulder abduction in athletes with rotator cuff tendinopathy.

Methods: Forty-three male volleyball players (17 asymptomatic and 26 with rotator cuff tendinopathy, mean age = 22.9 ± 3.5 years) participated in the study. The SAS was measured using B-mode ultrasound and UT shear modulus (used as an index of muscle stiffness) was quantified using Supersonic Shear Imaging with arm positioned at 0° , 30° and 60° of shoulder abduction.¹ The percentage change in SAS and UT shear modulus were calculated.

Results: Athletes with rotator cuff tendinopathy exhibited a higher UT shear modulus than the asymptomatic athletes (all $p < 0.007$). Significant negative correlation was found between the change in UT shear modulus and SAS during shoulder abduction from 0° to 60° in athletes with rotator cuff tendinopathy ($r = -0.487$, $p = 0.018$). Hence, stiffer UT was associated with less reduction of the SAS during shoulder abduction from 0° to 60° .

Discussion: Taking advantage of elastography to assess the UT muscle stiffness, the present study showed that athletes with rotator cuff tendinopathy exhibited higher UT shear modulus than the asymptomatic athletes. An increase in UT shear modulus was associated with less reduction of the SAS during shoulder abduction in those with rotator cuff tendinopathy. Our findings may suggest that an increase in UT muscle stiffness could be a compensatory strategy used by athletes with rotator cuff tendinopathy to preserve the SAS and to reduce impingement during shoulder elevation.

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O17: No effect of COX-2 inhibition on the composition of inflammatory cell populations during early and mid-time tendon healing

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Introduction: The regenerating tissue during early tendon healing is to a large part composed of leukocytes (CD45⁺ cells). In a rat Achilles tendon model, the inflammation resolves between 5 and 10 days. In the same model, NSAIDs impair healing if given during the first 5 days, but after that it has a positive effect ¹. We therefore tested the hypothesis that an NSAID would exert its effects through influencing the composition or size of the inflammatory cell population.

Methods: 24 rats underwent Achilles tendon transection. Rats were randomized to parecoxib treatment (6.4 mg/kg subcutaneously) or saline control. Rats received injections day 0-2 and were euthanized day 3 or received daily injections day 6-9 and were euthanized day 10. The regenerating tissue was digested using collagenase prior to cell isolation. Cells were then stained with antibodies (CD45, CD11b, CD68, CCR7, CD206, CD163, CD3, CD4, CD8a, CD25, Foxp3) to characterize different sub-populations of macrophages and T cells. Data was acquired using flow cytometry and two-way ANOVA was used to test the hypothesis, using CCR7/CD206 ratio as dependent variable, and time and drug treatment as independent.

Results: There was a strong and significant effect of time on virtually all inflammatory cell subpopulations (CD45, CD11b, CD68, CCR7, CD163, CD206, CD3, CD4) ($p < 0.001$), but no significant effect of parecoxib at either time point. The CCR7/CD206 ratio was not affected by the drug treatment. The pattern of cell composition appeared quite similar between the parecoxib and control groups at each time point.

Discussion: NSAIDs strongly influence tendon healing ¹. Our results suggest that this effect might not be mediated via changes in leukocyte composition, but possibly via changes in leukocyte behavior or via direct effects on mesenchymal-derived cells. An important effect of NSAIDs is to reduce levels of PGE₂, which is known to influence many mesenchyme-derived cell types through the EP₂ receptor, thereby e.g. impairing fracture healing ². It seems that the term “non-inflammatory” covers only a small part of the effects of NSAIDs in tendon healing.

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O18: What does normal tendon structure look like? New insights into tissue characterisation in the Achilles tendon.

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Introduction: Recently Ultrasound Tissue Characterisation (UTC) was introduced as a reliable method for quantification of tendon structure. Despite increasing publications on the use of UTC, it is striking that there is a lack of normative data in active adolescents. Therefore, the aim of this study was to provide normative values of the Achilles tendon as quantified by UTC.

Methods: Seventy physiotherapy students (26 male and 44 female) with no history of Achilles tendon injuries were recruited. The Achilles tendons were scanned with UTC to characterise tendon structure.

Results: This study demonstrated that Achilles tendons of active, healthy adolescents contained 54.6% echo-type I, 42.8% echo-type II, 2.2% echo-type III, and 0.3% echo-type IV at midportion. The comparison between insertion and midportion of the tendon showed more echo-type II at insertion ($p < 0.001$). Furthermore, female tendons contained significantly more echo-type II, in both insertion and midportion compared to male tendons ($p = 0.004$ and $p = 0.003$, respectively).

Discussion: The results of this study, with respect to the MDC (minimum detectable change), highlight differences in the UTC echopattern in the normal population (sex and regional location), which are important considerations for future studies.

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O19: The Sympathetic Nervous System is a Factor in Chronic Achilles Tendinopathy: An *In Vivo* Study

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Introduction: Tendinopathy is a common condition, which has been linked to surrogate measures of sympathetic nervous system (SNS) activity and insulin resistance. This study aimed to compare *in vivo* measures of the SNS and insulin resistance between individuals with and without Achilles tendinopathy.

Methods: This case-control study compared Achilles tendinopathy sufferers to healthy controls. SNS activity was quantified using *in vivo* muscle sympathetic nerve activity (MSNA), which gave 4 measures of MSNA; burst frequency, burst incidence, units per minute and units per 100 heartbeats. Insulin resistance was assessed via the Matsuda Index calculated from a modified oral glucose tolerance test. Metabolic status was further documented with a fasting lipid panel. Ultrasound tissue characterisation quantified tendon structure.

Results: Resting MSNA did not differ between the 15 cases and 20 controls. Tendon pain duration in tendinopathy patients was correlated with MSNA burst frequency ($R^2 = 0.324$, $p = 0.027$) and burst incidence ($R^2 = 0.408$, $p = 0.010$). After excluding an unexplained outlier, analysis by group revealed an association between reduced tendon structure and increased MSNA activity on all measures ($R^2 = 0.144 - 0.170$, $p = 0.024 - 0.039$). Fasting glucose was slightly higher in cases than controls and weakly correlated with pain severity ($R^2 = 0.140$, $p = 0.027$), but no other metabolic measures were associated with tendon pain/structure.

Conclusion: This study suggests that increased SNS activity is associated with tendon pain duration; building on previous data indicating the SNS is involved in recalcitrant tendinopathy. Metabolic parameters had little relationship with Achilles tendinopathy in this metabolically homogenous sample. Prospective studies are required to uncover the precise temporal relationship between SNS activity, insulin resistance and tendon pain/structure.

O20: Decrease in tendon strain is associated with intensity of pain in jumping athletes with patellar tendinopathy

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Introduction: Changes in tendon mechanical properties is one of the proposed pathogenesis associated with tendinopathy.¹ The aim of this study was to explore the inter-relationships among tendon strain, intensity of self-perceived pain and activity-related pain in jumping athletes with patellar tendinopathy.

Methods: Thirty-five male basketball, volleyball and handball players (mean age=22.4±4.0 years, 16 had unilateral symptoms) with patellar tendinopathy for more than 3 months (mean 31.9±25.4 months) participated in the study. Tendon strain of the patellar tendon was examined by ultrasonography and dynamometry. The intensities of self-perceived pain (maximal pain on past 7 days and pain during single-legged declined-squat test) were enquired using Visual Analogue Scale.

Results: Partial correlation test was conducted with sports and side of affected knee as control variables. Significant negative correlation between the patellar tendon strain and the maximal intensity of self-perceived pain ($r=-0.49$, $p=0.005$), and pain during single-legged declined-squat test ($r=-0.36$, $p=0.045$) were detected. Body weight, age, nor duration of symptom was associated with the intensity of pain (all $p>0.05$). There is a trend of significant difference on tendon strain between subjects with unilateral and bilateral symptoms ($9.0\pm3.7\%$ & $11.6\pm4.1\%$ respectively, $p=0.058$).

Discussion: Patellar tendon strain and intensity of pain are related. A higher tendon strain is associated with less intensity of self-perceived pain in jumping athletes with patellar tendinopathy. Side of affected knee may affect the mechanical loading on the patellar tendon. Our findings show that tendon mechanical property is associated with the intensity of tendon-related pain in athletes with patellar tendinopathy.

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O21: Evaluating somatosensory and psychological profiles of participants with patellar and Achilles tendinopathy: a single-blind case-control study design

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Introduction: Tendinopathy is characterized by pain and disability, which frequently persists. Although persistent pain is frequently associated with sensitization within the central nervous system, there is a lack of evidence in lower limb tendinopathy. The aim was to evaluate somatosensory and psychological profiles of participants with patellar tendinopathy (PT) and Achilles tendinopathy (AT) compared to separate control groups.

Methods: We compared PT (mean age 29.5 years, n=19, 26% female) to healthy controls (26.7 years, n=15, 53% female), and AT (45.7 years, n=30, 43% female) to healthy controls (41.0 years, n=11, 45% female). Quantitative Sensory Testing (QST) consisting of 4 pain threshold tests and 3 sensory threshold tests were performed according to the German Research Network on Neuropathic pain guidelines.¹ An assessor blinded to participants' condition applied standardized QST to the infra patellar pole for PT, mid-Achilles tendon for AT and the lateral epicondyle (remote site) for both groups. Participants completed the VISA-P/VISA-A, Health-related Quality of Life, the Hospital Anxiety and Depression Scale, the Active Australia Questionnaire and the Mental Toughness Questionnaire. An ANCOVA (Mann Whitney U test for ordinal data) was performed to compare groups, adjusting for sex, age and BMI.

Results: Compared to controls, participants with PT displayed significantly lower pressure pain threshold over the affected patellar tendon (mean difference: -192.55 kPa; 95%CI -340.37, -44.72; p=0.012). No differences were found related to the remote site and for any other QST in PT and AT. Compared to controls, PT and AT had significantly higher BMI (p=0.008, p=0.001 respectively) and lower quality of life (both p<0.001), while higher mental toughness scores were observed in PT compared to controls. The AT group was significantly older, with higher BMI, lower quality of life and higher depression scores than the PT group.

Discussion: PT is characterised by localised mechanical hyperalgesia and greater mental toughness. The evidence of central sensitization in PT and AT differs from that in the upper limb, suggesting a need to consider different mechanisms underlying the reported pain and disability, and likely different approaches to management. Caution is required in drawing inferences due to small participant numbers, necessitating confirmatory replication studies.

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O22: Immediate effects of one session of extracorporeal shock wave therapy on the elasticity of tendon in athletes with patellar tendinopathy

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Introduction: Patellar tendinopathy (PT) is one of the most common sport injuries in jumping athletes. Changes in the tendon elasticity stiffness have been detected in athletes with PT. Extracorporeal shockwave therapy (ESWT) was used to reduce pain among athletes with PT. However, it is not known whether the reduction in pain induced by ESWT is associated with the modulation of the tendon elasticity.

Purposes: To examine the immediate effects of ESWT on the elasticity of the patellar tendon; and to explore the possible relationships between the changes in tendon resilience mechanical properties induced by 1-session of ESWT and the intensity of activity-related pain.

Methods: A single-blinded randomized controlled trial Methods: Thirty-six male athletes aged between 18 and 32 with PT for more than 3 months were recruited from local basketball and volleyball teams. The subjects were randomized into ESWT and sham groups. ESWT was delivered at an intensity of maximum tolerable pain in the ESWT group. in the ESWT group received one session of ESWT at their maximum tolerable pain Supersonic Shearwave Imaging (SSI) was used to measure tendon elasticity shear modulus. Intensity of pain during single-legged declined-squat were acquired at pre- and immediately post-intervention

Results: Significantly greater reduction in the tendon shear elastic modulus was detected in the ESWT group compared with the sham group (by 24.7% and 8.0% in the ESWT and sham groups, respectively; $p < 0.05$). The patellar tendon shear elastic modulus was significantly reduced by 24.7% and 8.0 % in the ESWT and sham groups, respectively. In the ESWT group, the change in the tendon shear elastic modulus was related to the change in the intensity of squatting pain and the composite change in the knee range and squatting pain ($r = 0.52$, and 0.59 , respectively; all $p < 0.05$).

Conclusions: These findings suggest that possible association between the changes in the change of tendon elasticity mechanical properties and activity-related pain induced by 1-session of ESWT in jumping athletes with PT. Changes in tendon mechanical properties may be one of the treatment mechanisms induced by ESWT in reducing the pain associated with PT athletes with PT.

O23: Pressure pain thresholds? Don't do what we did!

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Introduction: Pain-pressure thresholds (PPT) are used to study peripheral and central pain processing among individuals with tendinopathy. However, pathological tendon changes may exist without pain. There are no studies that separate people with asymptomatic tendon pathology, those with a history of tendon pain and controls (no history of pain and no tendon pathology on imaging.)

Methods: This observational study was part of a larger study. In this component we compared PPT between individuals with and without asymptomatic tendon pathology, and between individuals with and without a past history of tendon pain. The patellar, Achilles and supraspinatus tendons of 130 participants were deemed structurally normal or abnormal with ultrasound. PPT was determined using a digital algometer at 40kPa/sec at the patellar tendon, quadriceps muscle, L3 spinous process and deltoid insertion.

Results: Two people were excluded for reporting pain history that was not related to tendon therefore 128 people were included in this study. Original data analysis was conducted that (incorrectly) included asymptomatic pathology in the control group and compared with those with a history of tendon pain. When these groups were separated, it was found that asymptomatic tendon pathology was associated with increased PPT ($p=0.000$, $n=7$ pathology, $n=92$ controls) compared with controls. This analysis was also conducted with matched pairs due to the different group numbers (matched for BMI, age and sex but blinded to PPT data) and found to remain significant ($p<0.004$, $n=7$). Those with a history of patellar tendon pain appeared to have decreased PPT until the asymptomatic pathology were removed from analysis, that is, the asymptomatic group artificially inflated the PPTs of the control group leading to the incorrect conclusion that those with a history of patellar tendon pain remained peripheral sensitized. In fact, there was no difference between controls and those with a past history of tendon pain.

Discussion: PPTs in the literature show large ranges for normal populations - pathology and pain appear to influence PPT results. Future research should consider the individual effects of pathology and pain and build on this pilot study. These findings point toward central nervous system adaptations (but perhaps not in the expected direction). Identifying factors that might protect people from experiencing pain despite the presence of pathology, and the features of people who have successfully recovered from tendon pain advances our understanding of this painful condition.

O24: Interrogating the role of angiogenesis genes on musculoskeletal soft tissue injury risk

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Introduction: Angiogenesis is a fundamental component of the matrix remodelling pathway with a fundamental role in the healing and adaptive response. To date, variants within the genes encoding vascular endothelial growth factor (*VEGFA*) and kinase insert-domain receptor (*KDR*) have been implicated with risk of anterior cruciate ligament (ACL) ruptures [1]. The aim of this study was to investigate genetic variants in angiogenesis genes with risk of Achilles tendinopathy (TEN) in two independent populations (South African (SA) and British (UK)).

Methods: A genetic-association study was conducted on a total of 120 SA & 130 UK asymptomatic controls (CON) and 108 SA & 87 UK participants with Achilles tendinopathy (TEN). All participants were genotyped for five functional polymorphisms in *VEGFA* (rs699947, rs1570360, rs2010963) and *KDR* (rs1870377 and rs2071559). Genotype frequency distributions were compared between the groups and haplotypes were also inferred. In order to refine the previously implicated genomic regions in *VEGFA* and *KDR*, whole exome sequencing (WES) was performed on 10 cases and 10 controls, representative of extreme phenotypes, using the Illumina HiSeq 2000 platform (Agilent V5 +UTR).

Results: The main finding of this study was the association of the *VEGFA* A-G-G inferred haplotype with increased risk of TEN in the SA group (15% CON vs 20% TEN, $p=0.048$) and the combined SA+UK group (14% CON vs 20% TEN, $p=0.009$). Additionally, the *VEGFA* rs699947 CC genotype was independently associated with reduced TEN risk in the SA group (32% CON vs. 17% TEN, $p=0.019$, OR: 2.30, 95% CI: 1.14-4.64). Preliminary analysis of the WES data highlighted twenty-three

genetic variants between the *VEGFA* and *KDR* genes, of which 4 variants show a greater than 25% allele frequency difference between groups.

Discussion: These novel findings implicating the *VEGFA* gene with TEN risk provide preliminary evidence highlighting the potential biological significance of the angiogenesis signalling pathway in the aetiology of Achilles tendinopathy. Additional investigation of the WES data is currently underway to investigate the novel variants further.

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O25: Microvascular volume in symptomatic Achilles tendons is associated with VISA-A score, but does not predict ESW-induced intrinsic tendon tenderness

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Introduction: In the continuum model of tendon pathology, the role of neovascularisation for explaining pain and function is still poorly understood. A recent study suggests that standard Power Doppler Ultrasound (PDU) has insufficient sensitivity to reliably assess the level of neovascularisation in tendons¹. In accordance, the aim of this study was to study the association between contrast-enhanced ultrasound (CEU) based microvascular volume (MV), VISA-A scores and intrinsic Achilles tendon (AT) tenderness as assessed by a novel non-invasive intrinsic dolorimetry methodology.

Methods: After completing a VISA-A questionnaire, real-time harmonic CEU measurements (Aplio 500, Toshiba Medical Systems, Australia) of the MV of the AT mid-portion using perflutren lipid microspheres (DEFINITY®, Lantheus Medical Imaging, Australia) contrast agent were taken in 20 patients (13 men/7 women, age: 44±8 yrs, BMI:24.4±3.3 kg.m⁻²) with clinical symptoms (duration 54±90 months) of uni- or bilateral Achilles tendinopathy. Intrinsic tendon tenderness was assessed by applying ultralow doses (0.01-0.3 mJ.mm⁻² at 1 Hz) of single focused extracorporeal shock waves (ESW, DuoLith SD1, F-SW probe, Storz Medical AG, Switzerland) to the skin overlying the dorsal side of the Achilles tendon. ESW detection threshold (ESW-DT) was determined for a total of seven 1-cm AT sections in disto-proximal direction from its insertion. Linear Mixed Model (LMM) analysis using 'R'² was used to determine the association between MV, VISA-A and ESW-DT for both symptomatic and asymptomatic ATs (n=39).

Results: LMM analysis shows a significant association between VISA-A and MV (B=-50.1, 95%CI=[-79.9;-23.0], p=0.0015) as well as symptom duration (B=-24.0, 95%CI=[-36.1;-11.2], p=0.001). No significant association was found between MV and ESW-DT in the mid-portion of the AT.

Discussion: The variation in intrinsic AT tenderness as assessed by ESW cannot be explained by the variation in Achilles tendinopathy associated hypervascularity. Nevertheless, in contrast to previous high-quality PDU-studies^{3,4}, our results indicate that hypervascularity of the AT mid-portion as assessed by CEU is moderately associated with increased AT pain and poorer AT function as assessed by VISA-A. In accordance, CEU-based microvascularity measurements have potential clinical application to objectively assess and monitor changes in tendinopathy-related pain and function throughout the course of a rehabilitation program or following a therapeutic intervention.

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O26: Plantaris excision and Achilles tendon scraping is associated with reduction in pain and improvement in tendon structure in patients with mid-portion Achilles tendinopathy

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Introduction: The plantaris tendon has recently been described as a possible important factor in midportion Achilles tendinopathy. Clinical¹, histopathological² and surgical studies^{3,4} lend support to this relationship. The aim of this study was to prospectively examine a larger number of surgical cases of mid-portion Achilles with suspected plantaris involvement.

Methods: A prospective design was implemented using UTC to examine structure and Visa-A to study clinical results. All subjects had suspected plantaris involvement and had failed non-operative treatment. Plantaris excision and ventral Achilles scraping were performed on all subjects. Twenty-three tendons from 18 subjects (12 men and 6 woman) with a mean age of 39 years (range 26-56 years) were included. Duration of symptoms ranged from 2 to 120 months. All patients were physically active consisting of 14 runners, 1 cricket player, 1 rugby player and 1 recreational walker. There were three elite athletes.

Results: At 6 months follow up, Wilcoxon Signed Ranks test demonstrated a significant increase in mean Visa A score ($Z = -3.726$ $p < 0.001$) from 58.1 (range 30-86) to 92.6 (range 69-100). In addition, there was a significant increase in mean aligned fibrillar structure of the tendon on UTC ($Z = -4.0470$ $p < 0.001$) from 83.7% to 90.6%. However, Kendall's tau test revealed no correlation between improvements in Visa A and aligned fibrillar structure ($r = -0.209$ $p = 0.237$). At 6 months, all subjects were satisfied with the procedure and 16 out of 18 subjects had returned to pre-injury levels.

Discussion: Plantaris excision and scraping of the ventromedial Achilles tendon seems to have potential to reduce pain and improve tendon structure in a larger cohort of subjects. However, there is no direct correlation between reduction in pain and improvement in structure. Studies on a larger group of patients with longer follow up are required to confirm these findings on pain and structure.

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O27: Fat pad adjacent to tendinopathy: coincidental or causal?

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Introduction: Is it a merely a curiosity that fat pads are found adjacent to the area of tendon affected by tendinopathy? We propose that fat pads share an intimate anatomical and functional relationship with their adjacent tendons, and may therefore be an important contributor to the pathogenesis of tendinopathy.

Methods: Key papers addressing this topic were identified through a structured search of PubMed, CINAHL and Medline and Web of Science. The results were synthesised as a narrative review.

Results: Large fat pads located adjacent to tendons correspond to sites commonly affected by tendinopathy. For example, Kager's fat pad is directly adjacent to the commonly injured area of the Achilles tendon, while Hoffa's fat pad is directly adjacent to the commonly injured patellar tendon. Direct neurovascular and fibrous connections between fat pads and tendons are consistently identified in anatomical studies. There is also evidence associating enlarged fat pads and fat pad-mediated angiogenesis with tendinopathy pathogenesis. Finally, fat pads located adjacent to pathologic tendons exhibit elevated cytokine levels. These cytokines may influence the tendon via the pathways involved in meta-inflammation.

Discussion: Fat pads are commonly overlooked in clinical research due to an inadequate understanding of their functional and biological significance in disease. The intimate anatomical and functional relationship between fat pads and tendons supports our hypothesis that fat pads may be a contributor to the pathogenesis of tendinopathy. The relationship is likely to be complex, and we hope this review will focus attention and stimulate research to improve our understanding of this important structure and its relationship with tendinopathy.

O28: Botulinum toxin A blocks the release of acetylcholine from tendon cells – A novel role for botox in treating tendinopathy?

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Introduction: Botulinum toxin is considered the most potent biotoxin known to man. The toxin acts by blocking the release of acetylcholine (ACh) causing paralysis of muscles. This mechanism has been used for more than two decades, in order to treat symptoms in conditions such as spastic paraesthesia, in products such as Botox. BoNT/A binds to the SV2a receptor and is internalised into efferent neurons. Following internalisation, it cleaves the membrane bound protein SNAP-25. When SNAP-25 is cleaved, vesicles containing ACh are unable to fuse with the cell membrane and thus cannot release ACh into the synaptic cleft. Studies with varying results have been directed towards the muscles of tendons involved in tendinopathy. Our group has shown that tendon cells produce and release ACh, and this production is upregulated in tendinopathy. This study aimed to evaluate whether BoNT/A could be directed towards the tendon – and *not* the muscle – in order to treat tendinopathy by blocking the endogenously produced ACh from tendon cells.

Methods: Tendon cells cultured from the Achilles tendon of healthy controls and patients diagnosed with tendinopathy were exposed to BoNT/A (Dysport[®]) or NaCl (control) for 24 hours. Culture media was collected and an assay directed towards ACh was used to measure the levels of excreted ACh.

Immunohistochemistry towards SV2a and SNAP25 were performed on the same cells.

Results: Cultured tendon cells expressed SV2a and SNAP25 as visualised by immunohistochemistry. Treating the cells with BoNT/A for 24h decreased the immunoreactivity of SNAP25. The ACh-assay showed a significant, dose-dependent, decrease in the concentration of ACh in the culture media following BoNT/A-treatment for 24 hours, as compared to NaCl-treated controls.

Discussion: This study shows the expression of the machinery involved in ACh release, and the receptors required for BoNT/A action. Treating tendon cells with BoNT/A decreased the expression of SNAP25, which implies that it has been cleaved and inactivated. BoNT/A-treated cells excreted less ACh in the culture media. As production of ACh in tendon cells has been shown to be up regulated in tendinopathy, BoNT/A-treatment may be a tool in affecting the intrinsic pathways hypothesised by Danielson and others, in the biochemical model of tendinopathy. By directing BoNT/A injections towards the tendon instead of into the muscle – as has been the paradigm in earlier studies – improved results may be achieved.

O29: Collagen Genes and Risk of Carpal Tunnel Syndrome

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Introduction: Carpal tunnel syndrome (CTS) is one of the most common disorders treated by hand surgeons¹. Although the exact mechanism is poorly understood, tendon pathology has been suggested to play a role in the aetiology of this injury. Previously, variants in genes encoding structural and functional tendon proteins, such as type V collagen, have been associated with CTS². The aim of this study was to determine whether variants within several other collagen genes are associated with CTS in a Coloured South African population.

Methods: Participants with carpal tunnel syndrome (CTS, n=103) as well as matched asymptomatic control participants (CON, n=150) were genotyped for various collagen gene variants including *COL1A1* rs1800012 (G/T), *COL11A1* rs3753841 (T/C), *COL11A1* rs1676486 (C/T) and *COL11A2* rs1799907 (T/A).

Results: The TT genotype of *COL11A1* rs3753841 was significantly over-represented in the CTS group (21.4%) compared to CTS CON group (7.9%) (p=0.004). Furthermore, a trend for the T minor allele of *COL1A1* rs1800012 to be over-represented in the CTS group (p=0.055) with a significant association in female participants (p=0.036) was observed. Constructed inferred pseudo-haplotypes also suggest various gene-gene interactions between the investigated variants.

Discussion: These findings provide further information about the role of genetic risk factors as well as the role of collagen fibril variation, and the result thereof, in the aetiology of CTS. These risk factors could potentially aid in the development of risk models aimed at identifying individuals at risk for developing this injury and strategies that target modifiable risk factors to mitigate the effect of non-modifiable risk factors, such as the genetic risk, could potentially be developed to reduce incidence and morbidity of CTS.

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O30: Disruption of TGF β Signaling in the Scleraxis Cell Lineage – a Genetic Model of Tendon Degeneration

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We previously reported that disruption of TGF β signaling in limb mesenchyme resulted in failure of tendon differentiation. To study additional roles of TGF β signaling in tendon development and maturation we chose to disrupt TGF β signaling in tenocytes after they assumed the tendon cell fate by using the tendon deleter *ScxCre* to target the type II TGF β receptor. Mutant pups (CKO; *Tgfb2*^{-/-}; *ScxCre*) appeared normal at birth but exhibited movement difficulties and splayed limbs by P3. Examination of the tendon reporter *ScxGFP* signal revealed that tendon formation was not affected in CKO embryos, but in post-natal stages, some tendons that appeared intact at birth were abruptly eliminated and other tendons retained structural integrity with a mosaic loss of tendon gene expression, including the *ScxGFP* signal, in the majority of the tenocytes. Lineage tracing revealed that these cells were derived from earlier *Scx*-expressing cells, suggesting that the tendon cell fate was disrupted in CKO tendons. Interestingly, we found some indications for tenocyte dedifferentiation but no evidence for trans-differentiation of these mutant tenocytes. In addition, CKO tendons also revealed varying degrees of tendon degradation. This mutant phenotype thus highlights an unexpected fragility of the tendon cell fate that may play a role in the etiology of tendinopathy. Analysis of this phenotype may therefore be instrumental for identifying the molecular and cellular requirements for maintenance of the tendon cell fate.

Lineage tracing also revealed that the cells that expressed *ScxGFP* in CKO tendons were not descendants of the original tenocytes but rather, cells that were newly recruited into the tendon concurrent with the onset of the degenerative processes described above. Consistent with a stem/progenitor origin of these cells, we found in them sporadic expression of stem/progenitor markers such as Nucleostamin and Sox9. Interestingly, these cells adopted an aberrant morphology and a very large volume resulting in tendon matrix disruption in their vicinity. This is the first demonstration of active cell recruitment into a non-injured tendon that may be used to identify the origin and activation mechanisms for tendon stem/progenitor cells, but may also point to molecular and cellular processes that underlie the progression of tendinopathy.

O31: A cross sectional study correlating Ultrasound Tissue Characterisation and Shear wave elastography in normal and tendinopathic Achilles tendons

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Background: The often poor correlation between clinical symptoms, function, imaging features and histopathology of tendinopathy remains a considerable challenge in the scientific understanding and clinical management of the condition. Novel ultrasound (US) imaging modalities may help bridge this gap.

Aim: This cross-sectional study of symptomatic mid-portion Achilles tendinopathy (AT) and asymptomatic Achilles tendons aimed to investigate the relationship between tendon structural integrity as determined by Ultrasound Tissue Characterisation (UTC, *UTC imaging, The Netherlands*), stiffness as determined by Shear Wave Ultrasound Elastography (SWUE, *Acuson Siemens*), and clinical symptoms and function as determined by the VISA-A questionnaire.

Methods: Subjects from a university and affiliated clinic were invited to participate following ethical approval. All participants completed the VISA-A questionnaire and underwent both UTC and SWUE measurement of their mid-portion Achilles tendons by two experienced technicians. The AT group was confirmed by an experienced clinician. Comparison of means and correlation statistics were calculated using SPSS v22, with statistical significance taken as P<0.05.

Results: 34 participants (21 males, 13 females; mean age 43, SD 16) were recruited, offering 68 tendons. 18 participants with mid-portion Achilles tendinopathy (AT group) had a mean VISA-A score of 74 (95% confidence interval 69-80) and 16 participants with no history of tendinopathy (Norm group) had a mean VISA-A score of 99 (CI 98-100). There was a significant difference between UTC and SWUE measurements between the AT group (echotype one 55%, CI 49-60; echotype two 35%, CI 31-38; SWE velocity 7.9m/s, CI 7.3-8.4) and Norm group (echotype one 71%, CI 68-73; echotype two 29%, CI 26-31; SWE velocity 9.1.m/s, CI 8.6-9.6). There was good correlation between SWUE and VISA-A score ($r=0.6$) and fair correlation between UTC echotype 1+2 and VISA-A score ($r=0.39$) across all participants. Correlation between SWUE and UTC was poor ($r<0.4$) when the entire group was considered and when the Norm and AT groups were separated.

Conclusion: Both SWUE and UTC appear able to distinguish symptomatic mid-portion Achilles tendinopathy from normal tendons. SWUE showed stronger correlation with VISA-A score than UTC, potentially indicating greater relevance of estimating mechanical properties of tendons in facilitating clinical management of Achilles tendinopathy.