ASSOCIATIONS BETWEEN PATELLAR TENDON STRUCTURE, MOVEMENT QUALITY, AND TRAINING CHARACTERISITICS IN MALE COLLEGIATE ATHLETES

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ABSTRACT

Kelsey M. Rankin: Associations between Patellar Tendon Structure, Movement Quality, and Training Characteristics in Male Collegiate Athletes
(Under the direction of Darin Padua)

Purpose: Ultrasound tissue characterization (UTC) is an imaging tool used to quantify tendon structural integrity. UTC has quantified patellar tendon response to load in athletes; PT structure between two conversely loaded high volume sports has yet to be compared. Methods: The JV Men's Basketball team (n=13) and Varsity Men's Swim team (n=13) at the University of North Carolina at Chapel Hill were imaged using the UTC device with a 10-MHz linear-array transducer mounted in a tracking device. UTC algorithms quantified the stability of pixel brightness over every 17 contiguous transverse images into 4 echo-types (I-IV). Participants completed a jump-landing task using the Electromagnetic Motion Capture System. The dominant limb of each participant was analyzed for patellar tendon structure and landing biomechanics. All participants completed a self-report training load questionnaire, including the validated PT assessment the VISA-P. Independent Samples T-tests, correlations and multiple linear regression model were used to assess association and correlation between tendon structure and measures of training intensity. Results: 26 subjects completes this study. There was significant difference in tendon structure between basketball and swim athletes ($p = 0.04$) with basketball athletes presenting with significantly less aligned tendon structure. There was moderate association between training intensity and tendon structure. Discussion: Training intensity and training load may negatively influence patellar tendon structural integrity.

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LIST OF ABBREVIATIONS

PT	Patellar Tendinopathy
PTA	Patellar Tendon Abnormalities
UTC	Ultrasound Tissue Characterization
AFS	Aligned Fibrillar Structure
DIS	Disorganized Tissue Structure
ROI	Region of Interest
CSA	Cross Sectional Area
KFD	Knee Flexion Displacement
vGRF	Vertical Ground Reaction Force
KEM	Knee Extension Moment
IGC	Initial Ground Contact
VAS	Visual Analog Scale
VISA-P	Victorian Institute of Sport Assessment- Patella
OSTRC	Oslo Sports Trauma Research Centre
SLDS	Single Leg Decline Squat

Chapter I: Introduction

Introduction

Anterior knee pain is a challenging condition to manage for healthcare providers. It's chronic and non-specific nature makes diagnosis and prognosis challenging. Patellar tendon pathology is gradual in onset and continuous if unmanaged.[1,2] Cook et al.[3] proposes that tendon pathology occurs along a continuum of three stages; reactive tendinopathy, tendon disrepair, and degenerative tendinopathy. Tendon structure is adaptable and can be driven along this continuum, dependent on what intrinsic and extrinsic factors are present. Load is the primary stimulus underlying the development of structural tendon pathology and symptoms.[3] Tendinopathy is a load-based pathology, commonly developing due to excessive load from high training and competition bouts.[4] While diagnosis of tendinopathy is traditionally based on location of self-reported pain, load-based pain reproduction, and/or structural pathology on diagnostic imaging, the literature demonstrates that the clinical presentation of tendinopathy can vary, with tendon-related symptoms and structural pathology not always occurring simultaneously. [5,6]

Patellar tendon structural abnormities (PTA) present as structural changes in tendon tissue commonly found at either tendon origin or insertion, inferior patellar pole and tibial tuberosity respectively, associated with or without pain, functional limitations, and change in load.[7] Cook et al.[8] reported that 22% of the athletes that had patellar tendon abnormalities (PTA) on ultrasound had no clinical symptoms. Rudavsky et al.[9] found that tendon pathology on imaging was reported in 24% of athletes to be asymptomatic, with male athletes having twice the prevalence as female athletes. It was theorized that males are at higher risk of developing PTA due to a larger knee extension moment compared to females. Often, athletes may choose to continue to participate in sport (practices and competitions) despite the presence of tendon-related pain; however, eventual reduction in sport participation and physical activity may result from the continued demand placed on this highly responsive tissue, leading to eventual reduction loading capacity, self-limiting physical activity, and reduced overall quality of life.[4,10]

Sports involving high volumes of jumping and landing, such as basketball and volleyball, experience the highest incidence of patellar tendinopathy (PT).[11,12] It is estimated that over 13% of athletes involved in basketball and volleyball will experience patellar tendon pain at some point in their competitive career.[11] The effect of tendinopathy on sport participation is difficult to measure, as it is rare that an individual will miss a training or competition bout due to this condition, especially early in its pathoetiologic process.[8] It has been shown that lower extremity loading in combination with jumping sports increases the risk for developing PT.[13] Sport-specific motions (squatting, lunging, cutting) are typically reported as painful by athletes with symptomatic patellar tendinopathy. External load, or the load exerted on the body, is a primary pathoetiologic component in PT; however, some sports known for their high training and competition volumes, such as competitive swimming, aren't represented in the literature.[3] Swimmers train an average of 5-6 hours per day through a competitive season that can last up to 12 months.[14] Their training is unique, as the majority takes place in an aquatic environment with un-weighted horizontal gravitational and rotational forces acting on the musculoskeletal system through their loading period.[14] Therefore, differences in loading parameters between basketball and swimming athletes likely influences the difference in incidence and prevalence of PT between sports.[1,3] However, associations between type of loading parameters and patellar tendon structure have not been systematically assessed in these specific athlete groups.

New ultrasound imaging techniques allow for improved visual analysis of tendon structure through ultrasound images.[15] Traditional ultrasonographic (US) technology typically quantifies and describes tendon structure based on grey-scale images; the most commonly reported outcomes to describe tendon structure in the literature include cross-sectional area (CSA), echogenicity, and neovascularization.[16–18] Grey-scale ultrasound imaging relies on more subjective interpretation of these tendon structural characteristics, and is not able to detect subtle changes in tendon structural integrity that are thought to represent positive or negative adaptation.[15] Ultrasound Tissue Characterization (UTC) is an novel ultrasonographic imaging technology that quantifies tendon structural integrity through advanced computer algorithms that categorize the characteristics, specifically the orientation and stability, of the collagen fibers within the tendon.[15,19,20] The ability of UTC to quantify tendon structure and assess response to training load makes it an ideal tool to assess differences existing tendon structure between dynamic and non-dynamic sport athletes.[19,21,22]

Lower extremity kinematics and kinetics in athletes with symptomatic PT may contribute to presentation of pathology. Rosen et al.[23] found that individuals with PT displayed different movement strategies compared to a healthy population during a double limb jump-landing task, including increased knee flexion displacement and increased peak internal knee extension moment. One key variable that influences tendon structure is the overall magnitude of mechanical load and the direction of mechanical load endured by the patellar tendon.[4,10] Changes in sagittal plane movement patterns in those with PT may be due to the associated alterations of quadriceps and hamstring activation in response to symptoms.[23] Given the patellar tendon's role in dissipating externally applied load and in generation of internal moment through the knee extensor mechanism, understanding movement characteristics of individuals at high-risk of developing patellar tendinopathy is important in order to prescribe and manage injury prevention and rehabilitation programs.

Clinical Significance

Current literature suggests that athletes with structural abnormalities of the patellar tendon have a higher risk of developing patellar tendinopathy than athletes without structural pathology.[6,20,24,25] However, it is unknown how type of training load (plyometric vs. non-plyometric) and lower extremity biomechanics influence tendon structure. The primary aim of this study is to determine the differences in patellar tendon structure between athletes participating in primarily land-based training (basketball) and aquatic-based training (swimming). This will be accomplished by examining the percentages of the four echo-types, all reflecting stages of tendon structural integrity, in the dominant limb's patellar tendon between male collegiate basketball athletes and male collegiate swimmers. The second aim will examine if specific landing kinematic & kinetic patterns influence patellar tendon structure of the dominant limb of male collegiate basketball players and swimming athletes. Finally, the third aim will examine the influence of both training load variables and biomechanical variables on tendon structure.

Research Aims and Hypotheses

<u>Aim 1:</u> Determine if there are differences in pre-season patellar tendon structural integrity of the dominant limb between male collegiate basketball and swimming athletes.

Hypothesis 1: We hypothesize that male collegiate basketball athletes will exhibit a significantly lower percentage of aligned fibrillar structure of the dominant limb patellar tendon than that of male collegiate swimmers.

Aim 2: Determine if landing kinematics & kinetics are associated with pre-season patellar tendon structural integrity of the dominant limb of male collegiate basketball athletes and male collegiate swimming athletes.

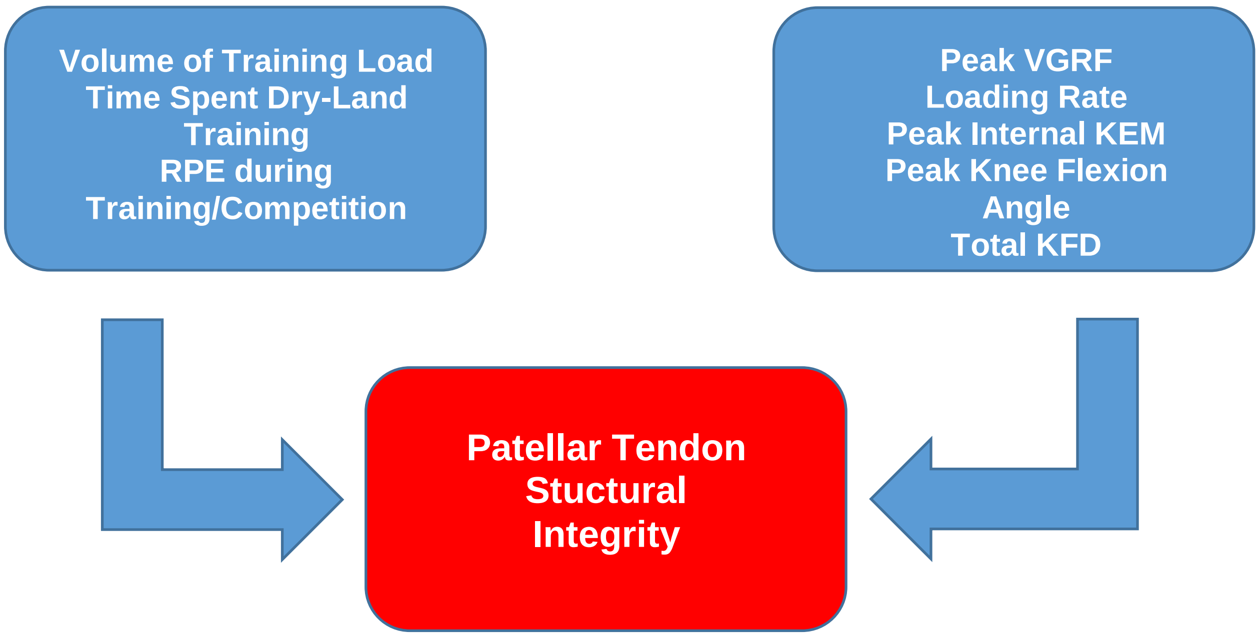
Hypothesis 1: We hypothesize that there will be a negative association between peak vertical ground reaction force and patellar tendon structural integrity, such that higher peak vertical ground reaction force will be associated with a lower percentage of aligned fibrillar structure of the dominant limb patellar tendon.

Hypothesis 2: We hypothesize that there will be a negative association between peak internal knee extension moment and patellar tendon structural integrity, such that higher peak internal knee extension moment will be associated with a lower percentage of aligned fibrillar structure of the dominant limb patellar tendon.

Hypothesis 3: We hypothesize that there will be a negative association between knee flexion motion displacement and patellar tendon structural integrity, such that greater knee flexion motion displacement will be associated with a lower percentage of aligned fibrillar structure of the dominant limb patellar tendon.

Hypothesis 4: We hypothesize that there will be a negative association between vertical ground reaction force loading rate and patellar tendon structural integrity, such that increased loading rate will be associated with a lower percentage of aligned fibrillar structure of the dominant limb patellar tendon.

Aim 3: Determine if patellar tendon structural integrity can be predicted based on a series of independent variables pertaining to training load characteristics and landing kinematics and kinetics in male collegiate basketball and swimming athletes.



Hypothesis 1: We hypothesize that there will be a significant predictive value between the following biomechanical variables and patellar tendon structural integrity, such that higher values of these variables will statistically significant predictor of patellar tendon structural integrity of the dominant limb patellar tendon:

- Peak Vertical Ground Reaction Force
- Loading Rate (Time between IGC and toe-off)
- Peak Internal Knee Extension Moment
- Peak Knee Flexion Angle
- Total Knee Flexion Displacement

Hypothesis 2: We hypothesize that there will be a positive predictive value between training load volume and patellar tendon structural integrity, such that increased training volume will statistically significant predicter of patellar tendon structural integrity of the dominant limb patellar tendon.

Hypothesis 3: We hypothesize that there will be a positive predictive value between dry land training and patellar tendon structural integrity, such that increased duration of dry land training will present as a statistically significant predicter of patellar tendon structural integrity of the dominant limb.

Hypothesis 4: We hypothesize that there will be a positive predictive value between rate of perceived exertion (RPE) during training/competition and patellar tendon structural integrity, such that increased RPE during training/competition will statistically significant predicter of patellar tendon structural integrity of the dominant limb.

Chapter II: Literature Review

Patellar tendon abnormalities are misunderstood musculoskeletal condition. Pathology is defined as "an overuse injury that involves pain at either the proximal or distal aspect of the patellar tendon, commonly associated with a decrease in functional ability."[26] Due to its chronic nature athletic participation may not be restricted, yet it's shown a significant impact on performance. Patellar tendon abnormalities are most prevalent in dynamic, high velocity sports, specifically volleyball and basketball.[11,27] Repetitive plyometric activity, such as jumping, leads to increased demand from lower extremity musculature causing abnormal stress through tendon tissue. [6,28] There is an estimated 20% incidence rate in athletic population.[26] Factors such as type of training, training load, and movement mechanics have all shown to effect the development of PTA.[5,29,30] This review will look into the role that each of these play in development of tendon tissue.

Etiology

PTA are thought to originate from an imbalance between intrinsic changes and external load.[3] Tendon has the ability to adapt to a mechanical load, with intensity, frequency and volume all influencing structural change.[31] In this paper, it will be referred to as "tendon load". Athletes experience a range of training load between their training seasons, leaving them more susceptible of developing pathology.[32]

Sports Specific

The highest prevalence of patellar tendon pathology is found in basketball and volleyball athletes.[2,11,33,34] De Vries et al.[11] looked at a 334 participant prospective cohort of elite volleyball and basketball athletes. They found that 13% of the participants developed symptomatic patellar tendinopathy over a three-year period. Combining physically demanding work (i.e. squatting, lifting, mentally tasking) with either sport increased risk for pathology.[13] Results suggested that while total training hours between participants did not differ, those who developed pathology reported increased volume of plyometric activities during training hours.[11] Biessling et al.[35] reported up to 45% of volleyball players will display pathological symptoms at some point throughout their athletic career. It's been hypothesized that early specialization in plyometric load could predispose an athlete to structural changes compared to a multi-faceted regimen.[10]

Low-impact, high volume sports such as swimming show a high prevalence for chronic conditions. Their imposed training philosophy transmits forces and loads differently due to the aquatic environment. Watanabe et al.[36] studied how a swimmer's horizontal posture through activity both transmits and resists force in water. Buoyancy torque, breathing techniques and drag resistance all contribute into a swimmer's ability to maintain position through motion. Kilani et al. compared the ground reaction force of three athletic positions: a swimmer starting off the block, a sprinter starting off the block, and a volleyball block jump. The volleyball vertical jump produced a greater GRF impulse than the other take-offs. Swimming starts get swimmers off the block slower than sprinters and volleyball players, but cover a greater horizontal distance covered. This is due to their constant linear momentum. Force absorbed through aquatic environments transmit through the body different than a land-based sport.

Training Load

Training load is directly associated the development of chronic pathologies.[4] Rosengarten et al.[19] demonstrated that tendons produce responsive inflammation to micro-bouts of increased training. Tendon changes observed following a 4-day period of high volume training in Australian football players were attributed to cellular-driven response, increasing the volume of extra-cellular matrix. Increased inflammatory tendon response results following acute activity without compromising the integrity of tendon collagen structure.[19] Plyometrics are effective in increasing muscular power generation. High velocity muscle contractions rapidly stimulate mechanical loading signals, called mechano-transducers through the patellar tendon.[12] Quick, explosive movements such as jumping and squatting elicit muscular and tendon development. Muscle, tendon collagen and the extracellular matrix network are known to respond to altered levels of physical activity.[37] While both tissues respond to a mechanical stimulus, muscle adapts to increased load sooner than tendon. Following an acute bout of high training load, muscle will be able to create the same or greater force. Tendon tissue however is expected to sustain increases in muscular force to create motion. Tendon maladaptation will follow leading to abnormalities through the structure. [1,2,32] Whereas a single episode of exercise has little effect on the level of tendon adaptation, prolonged exercise or weeks of training can increase tendon collagen turnover and lead to a pathological state.[37]

Plyometric Loading

The term “eccentric strength” refers to the ability of a muscle to produce force as it is lengthening. The quadriceps function eccentrically when the knee is flexing. During the landing phase of a jump, rapid eccentric contraction from the quadriceps is required. This type of contraction places high tensile stress on the patellar tendon. High energy tasks such as a jump landing require strong eccentric control from the quadriceps. This creates an internal knee extension moment which can effect motor unit recruitment from antagonist musculature, coordinated contraction of the quadriceps, and firing frequency of muscle fibers from both structures.[1,20,38] Knee extension results in inferior movement of the patella, and knee flexion causes superior migration of the patellar. This superior migration partnered with a strong eccentric contraction increases the load on an already contracted tissue. With eccentric strength being a main component in plyometric exercise, it is important that the clinician understands the different loads and stresses that the tendon is experiencing at different phases of the jump-landing cycle. Individualizing athletes' training, monitoring changes in symptoms and adjusting training load is essential to maintain appropriate distribution of load through sport. Addressing this prior to any significant change in training load can increase muscle-tendon conditioning and enhance kinetic chain utilization.[32]

Continuum of Tendon Pathology

Several models have been developed to describe tendon pathology, each allowing for a better understanding of tendon pathology, treatment and prevention.[3] It's come into question if pathological tendon damage occurs along a continuum, and possibly even reversible. Cook et al.[3] believes there are three phases of tendon pathology: reactive tendinopathy, tendon disrepair, and degenerative tendinopathy. While each are presented as separate stages, there is continuity between the three.

Reactive Tendinopathy

Reactive tendinopathy is a result of a short-term adaptive thickening of a portion of tendon that acts to reduce stress and allow adaption to compressive loads.[3] This differs from typical tendon reaction to tensile load in that it increases cross sectional area as a whole, rather than just an increase in stiffness[3,20] It results from acute overload or a direct blow to the structure, such as occurs during the first days of practice or in three consecutive heavy training days. Tissue response is a homogeneous, non-inflammatory response that results in metaplastic changes in cellular proliferation. It is important to note that collagen integrity is maintained throughout this phase.

Tendon Disrepair

Tendon disrepair is the second stage of the continuum. During this phase, there is an attempt at tendon healing.[3] It is characterized by an overall increase in chondrocytes resulting in separation of collagen and disorganization of the cellular matrix. It may present with an increase in vascularity, thickness and neuronal ingrowth.[3] Imaging presents with discontinuity of collagen fibers and a "swollen" appearance through the structure. Potential for reversibility exists during this stage with appropriate load management and exercise to stimulate matrix restructuring. The frequency, volume or length of time over which load has been applied (ie, months or years of overload) determine the prognosis from this stage.[3]

Degenerative Tendinopathy

The final stage is considered degenerative tendinopathy. It presents with areas of cellularity, matric disorganization, neo-vessels, and little collagen regeneration. Imaging presents with islands of degenerative pathology interspersed between normal and degenerating tendon. With little chance of reversibility, if a degenerative tendon is placed under a high load rupture is likely. Research shows that 97% of tendons that rupture had reached degenerative state.[3,39] Ruptures represent end-stage of degeneration, supporting the non-reversible nature of the continuum[32]. It is important to note that reversibility is possible between reactive tendinopathy and tendon disrepair. Longitudinal studies show that between 10% and 30% of elite volleyball player's tendons reflected hypoechoic tissue when imaged at baseline and at the conclusion of their season. However at their follow-up screening, most returned to their normal state, reflecting that reversibility is possible through the early stages.[33,39]

It is not uncommon for tendon to progress to a degenerative state and not exhibit painful signs or symptoms. Cook et al.[27] found that 14% of adult basketball players and 24% of volleyball players presented with patellar tendon structural abnormalities without any complaint of knee pain. Diagnostic tools, such as Ultrasound Tissue Characterization (UTC), allow clinicians to determine is at risk of developing tendon pathology based on the structural integrity of the structure.[20] Pathology can exist for years without causing pain, leading researchers to question whether is it magnitude or response to load that is responsible. UTC allows quantification of tissue quality and can objectively measure a tendon response to acute load.[32,33] This can allow for better care and load management throughout competitive season.

Diagnosis

Accurate diagnosis of patellar tendinopathy is based upon thorough clinical examination. Intrinsically the presence of a hypoechoic region and/or increased cross-sectional area on sonographic ultrasound can be used diagnostically. [40,41] Identifying pain patterns, quantifying structural qualities, and assessing physiologic changes within tissue will provide an understanding of loads effect on tendon structure.[17,20,23] Diagnosis is difficult due to obscurity of symptom presentation. Presence of pain is not considered necessary for a tendon to be considered pathological.[17] Cook et al.[3] demonstrated that 2/3 of patellar tendons that were considered degenerated presented without pain.

Assessment

The most common finding with tissue injury is pain. Identifying and quantifying pain through subjective outcome measures allows clinicians to track any changes, progress or trends when a patellar tendon symptoms present[3,33] Self-report questionnaires such as the Victorian Institute of Sports Assessment (VISA-P) and Visual Analog Scale (VAS) are the gold standard for assessing a patient for patellar tendon abnormailities.[42,43] Both assessments are proven to be valid and reliable for assessing patients with patellar tendinopathy.[40,44] The questions of the VISA scale that test functional performance distinguish this measurement tool from others and provides the questionnaire with sensitivity necessary to reflect subtle changes in symptoms of jumper's knee.[43] Included with the VISA-P assessment is a single-limb (SL) decline squat, where the individual is instructed to perform a decline squat on a 45° slant board. The tendon is provocatively loaded through an eccentric quadriceps contraction to fully evaluate the perceived tendon response. The motion creates an eccentric quadriceps contraction, and targets to reproduce painful symptoms.[40] Pain is the clinical factor we seek to change through treatment and rehabilitation, and an improvement indicates treatment success.[3]

Tendon Morphology

Secondary to pain, tendon tissue quality is necessary to consider when assessing for PTA. Fiber type, tendon thickness, and the extracellular matrix determine the integrity of tendon structure. Abnormal tendon cells will produce signals that upregulate the production of protein and pain receptors. This results in an influx of inflammatory regulators, vascular vessels, and collagen producers. All signals acutely increase neural and pain sensitivity.[32]

Cross-Sectional Area

An increase in tendon thickness can also indicate internal mechanical stress to the structure. Mersmann et al.[2] found that during late adolescence, hypertrophy of the PT led to mechanical strengthening and increased stiffness of the tendon in relation to the morphological development of the quadriceps. They concluded that a larger cross-sectional area is a major mechanism leading to increased stiffness and thus tendon pathology. Typical tendon response to load is a short bout of stiffening to tensile loads, followed by eventual return to normal state. Short-term adaptive responses in pathological tendon include a homogeneous thickening of the portion of tendon under stress, increasing CSA and allowing adaption to compression.[3,32] Mann et al.[25] found that direction of an applied load can affect where the tendon is more susceptible to growth. They suggest that because histological adaptations occur as a result of increased tendon tension, direction of the load is more critical than magnitude in the development of patellar tendon abnormalities (PTA). The areas of tendon that experience the most tensile force will respond with the most cellular proliferation, and therefore a larger CSA comparably. Athletes are considered to have a PTA if a hypoechoic area is evident on scans both longitudinally and transversely.[8]

Neovascularization

Neovascularization is the formation of new blood vessels in tissue, typically indicative of tissue overload or damage.[45] Imaging techniques are able to show vascular changes that develop in the extracellular matrix. Alterations, such as neovessels, in the extracellular matrix occur according to loads leading to stiffer tendon structure.[44] Hiksrud et al.[16] demonstrated that the presence of neovessels in abnormal patellar tendons was associated with more pain than in abnormal tendons without. Neovascularization upregulates the release of proteins and signals that induce cellular proliferation, therefor decreasing tendon's tolerance to load. Cook et al.[39] in a cohort of symptomatic and asymptomatic volleyball players, reported significantly lower VISA-P scores in patients with abnormal tendons with neovascularization than in patients with abnormal tendons without neovascularization. This is believed to occur along a continuum where if cellular dysfunction begins to compromise matrix structural integrity, then tendon damage would be irreversible.[3,46] To prevent further matrix damage, appropriate progression of loads is essential to maintain integrity of the tendon matrix.[46]

Lower Extremity Biomechanics

Assessing lower extremity motion during functional tasks is crucial for evaluating restrictions. Observing movement through the lower extremity and trunk identifies muscular imbalances and restrictions that can increase risk of injury. Identifying lower limb impairments in athletes with patellar tendon tendinopathy is important to effectively treat the pathology.[47]

Lower Extremity Musculature

A relationship exists between the quadriceps and hamstring group to balance loads through the lower extremity. The patellar tendon is the most distal attachment of the quadriceps muscle group. It is the main structure that transmits force from the powerful muscle to bone. The hamstring muscle group has three musculotendinous attachments to evenly distribute muscular force and load. Haddas et al.[28] suggested that the quadriceps fatigue faster than the hamstrings due to decreased efficiency transferring loads through the knee. Thirty-two participants were instructed through a fatigue protocol utilizing free body-weight squats with 15% body weight until failure. Results showed fatigue produced a greater maximum knee-flexion moment, reflecting an alteration in the shock-absorbing mechanism at the knee joint. Increased knee flexion moments created high moments of compressive force through the PT, subjecting it to additional stress. The fatigue protocol effected the strength and control of the quadriceps more so than the hamstrings. A decrease in gross knee extension moment was experienced. [47–49] Similarly, Crossley et al.[33] found athletes with patellar tendinopathy present with decreased knee extension torque through an explosive jump. Their ability to efficiently activate their quadriceps to explode through the motion is attributed to muscular inhibition.[47] The compressive forces of the tendon through jump countermovement (knee flexion) inhibits the quadriceps to contract appropriately.

Muscle imbalances impact the musculotendinous junction.[50] During adolescence, muscle develops at a rate that is directly relates to bone growth. Tenocytes are the cells responsible for the metabolic, reproductive, and mechanical responses that occur in musculotendinous units. This includes the formation and turnover of proteins and growth factors within the extracellular matrix.[51] Mersmann et al.[2] found that dynamic changes in tenocytes through the quadriceps during adolescence occurs in response to maturation and mechanical loading. This creates an imbalance between tenocytes and muscle fibers. The mechanism of skeletal muscle adapting faster than its tendon is known as “Sports Specific Loading”.[2] The result is a muscle capable of the strength of a developed knee extensor with an adolescent, immature patellar tendon.

Lower Extremity Kinetics and Kinematics

Tendons have a unique ability to adapt through alterations in the extracellular matrix according to changes in mechanical loads.[44] Dynamic movements, such as an overhead squat or a jump landing task, are used in laboratory settings to identify movement patterns that put individuals at risk of lower extremity injury. Motion is evaluated using a tri-planar system.[52] Side-to-side (frontal plane), forward-backward (sagittal plane), and rotational (transverse) motion place load and stress on the lower extremity. If these stresses are not distributed appropriately, a higher risk of injury exists.[52] Mann et al.[44] utilized a cohort of 22 male basketball players to determine the risk factors through a drop-landing task that can predict the incidence of patellar tendon abnormalities. Landing with more hip extension creates greater sagittal plane motion relative to the base of support increasing tensile and compressive loads through the tendon. In addition, athletes presenting with increased knee flexion at initial contact presented with patellar tendon abnormalities. Landing with greater knee flexion increased compressive loading on the tendon. This is attributed to an increased quadriceps force ratio, with resultant tensile load through the lengthened tissue. Crossley et al.[33] found that individuals presenting with increased normalized knee extension torque were more at risk of presenting with patellar tendon abnormalities.[33] Similarly, Rosen et al.[23] indicated that maximum knee flexion displacement through a drop landing task had a large effect size, and pathological participants demonstrated an 8 degree decrease in total angular displacement compared with controls. This reflects that landing “stiff”, or with decreased knee flexion displacement, puts an individual `at risk for patellar tendon changes. Edwards et al.[53] suggested that individuals with PTA had increased knee flexion through landing compared to healthy controls. It was hypothesized that landing flexed places the tendon in a lengthened position, increasing tensile loads.

Landing Error Scoring System

Functional movement assessments are a vital clinical tool when determining who is at risk for injury due to movement patterns. There are a range of evaluation methods that clinicians can utilize in a sports medicine setting. The most common include a dynamic jump landing task, dynamic postural control exercises, Functional Movement Screening (FMS), and an overhead squat task. Each tool has its own set criteria to assess where an athlete may be weak, making it easy to provide feedback and identify problems in the musculoskeletal system. Movement quality is determined by a multitude of factors including poor neuromuscular control, balance deficiencies, and muscle imbalances[54–56]. The Landing Error Scoring System (LESS) assesses jump-landing biomechanics of an individual as they jump forward off a box, and scores any improper movement patterns as an error[56]. The subject is allowed three trials, each of which is assessed and scored for movement errors. The LESS has demonstrated good to excellent interrater and intrarater reliability, making it useful to implement as a screening tool to identify improper landing mechanics.[57,58] It has also been successful in evaluating changes in landing technique after an injury prevention program[59].

Understanding and utilizing movement quality screenings such as the LESS when treating PTA will allow clinicians to relate specific movement patterns to symptomatic tendons and assess how change in functional movements can elicit change in pathology. The LESS is a useful clinical assessment tool for identifying high-risk movement patterns through the lower extremity.[60] Red flag errors include increased trunk-flexion, hip flexion displacement, initial contact foot position, and knee flexion displacement. LESS screening demonstrates that movement quality is a predictive factor of ACL injury, with 86% sensitivity and 64% specificity.

Poor movement quality through the lower extremity as a whole increases ones injury risk susceptibility. [52,59,60] While the LESS is unable to capture 3-dimensional biomechanical movement patterns, it has still shown strong concurrent validity. Significant differences in movement quality between those with high or low LESS scores respectively demonstrate the tests ability to predict who is more at risk for lower extremity injury.[52] Research shows that a higher LESS score indicates poor movement quality and high risk of injury.[61] With a high predictive ability, this assessment test serves as a feasible tool for clinicians attempting to assess biomechanical errors in their patients.

Diagnostic Imaging

Traditional Ultrasound

Ultrasound has been shown to be a reliable and valid tendon examination method.[8] It has shown success identifying tendon abnormalities between similar groups of subjects. They've shown transmission techniques that have potential to provide non-invasive estimates of human tendon loading and response to load.[17] Ultrasound is a sensitive indicator of tendon injury, and can track the recovery of tendon properties that present to be abnormal.[17] It is not able to present the degree to which tendon structure has degenerated. It is able to show abnormality, but not severity.[8,16,17]

Ultrasound Tissue Characterization

Ultrasound Tissue Characterization (UTC) is an assessment tool used to quantify the structure of tendon based on the integrity of the echo pattern displayed through ultrasound analysis. Tendon fibrillar alignment, tendon volume, and cross-sectional area are all considered in the algorithmic enhanced ultrasound analysis.[31] The device uses an ultrasound beam allowing for 3-D imaging, semi-quantification of structure, and calculation of tendon dimensions.[20] UTC can be used as a diagnostic tool to determine when a tendon's structure is normal or abnormal progressing towards a pathological state. It is a diagnostic tool that is capable of objectively measuring tendon quality and determine if portions of tendon are on a spectrum between normal and pathological. UTC is often used as a technique to track tendon integrity through different rehabilitation modalities, potentially making it a great tool to further future research regarding tendinopathy.[19,20]

UTC quantifies tissue structure using four different stages. The device uses an algorithm to correlate pixel images that reflect uniformity of the tendon and expresses it as a color in the tendon. Green tendon represents healthy tissue, black represents pathological tissue, and blue/red identifies tissue that is at risk of becoming pathological[32]. Cook et al.[32] demonstrated that this approach is valid, reliable and capable of detecting tendon response to overload. Green, blue, red and black tissue representation reflect echo type I, II, III and IV, respectively[20]. Echotypes I and II correspond to high stability tendon with an aligned fibrillar structure. Types III and IV represent a disorganized, non-parallel fiber arrangement. These are considered pathological tissue cells[20]. Research shows that quantification of the UTC echo-types should be performed by the same investigator, and should remain blind to participant, clinical history and tendon classification throughout the interpretation of tendon echo-type to reduce risk of bias[20,46].

Clinical Significance

A thorough evaluation is necessary when identifying a patient with patellar tendinopathy. Inconsistency of interaction between symptoms, function and structure makes diagnosis difficult for clinicians.[11,20,23] Tendinopathy is a load induced pathology that responds well to activity modification. A mismanagement of load results in maladaptation of tendon structure, and potential pathology.[32]

The literature concludes that a multitude of factors contribute to patellar tendon structural change. Dynamic sports, such as basketball and volleyball, are predisposed to tendon maladaptation due to repetitive high velocity eccentric moments that are mimicked through sport.[27,62] Volume and frequency, typically quantified by number of training sessions and hours spent, has proven to be critical in the development of abnormal tendon tissue.[13] Research has shown that specific movement patterns such as decreased sagittal plane motion through landing and decreased knee extension torque may be lead to development of pathology.[32,33] What's not clear in the literature is how lower extremity biomechanics translate to pre-existing tendon abnormalities. Assessing baseline lower extremity mechanics and comparing them to structural changes over the course of a basketball season will allow clinicians to create a focused approach to better treat tendon maladaptation.

Chapter III: Methods

Study Design

A cross-sectional study design was used to assess the relationships between patellar tendon structure, movement quality and training load in male collegiate basketball and swimming athletes. All members of the University of North Carolina at Chapel Hill Junior Varsity Men's Basketball Team and Varsity Men's Swimming Team were invited to participate. Data was collected approximately 1-2 weeks prior to the initiation of each team's formal practice and competition schedule. All data collection sessions were completed in the Sports Medicine Research Laboratory on the campus of the University of North Carolina at Chapel Hill. All participants were required to read and sign an informed consent document approved by the University's Institutional Review Board (IRB #16-1932) prior to data collection.

Participants

Twenty-six male Junior Varsity Men's Basketball (n=13) and Varsity Men's Swimming athletes (n=13) from the University of North Carolina at Chapel Hill were recruited and screened to participate in the research study. Equal numbers of participants were enrolled from the Junior Varsity (JV) Men's Basketball team (n=15) and the and Varsity Men's Swimming team (n=15). All eligible participants were between the ages of 18-28. Participants were excluded if they have a history of lower extremity surgery or have sustained a lower extremity injury in the last 6 months that prevents them from completing any of the assessments. Participants were asked to complete a series of questionnaires addressing exclusion criteria (Table 1). Anthropometric data (height and weight) was collected by one clinician. Height was measured in centimeters (cm) using a stadiometer (Detecto Model 758C, Cardinal Scale Manufacturing, Webb City, MO, USA),and weight measured in kilograms (kg) using a digital scale (Perspective Enterprises, Portage, MI, USA).

Self-Reported Function & Training Load Questionnaires

All participants reported to the laboratory for a single data collection session. The purpose and methods of the study were reviewed; participants were aware that participation in this study is strictly voluntary, and that participation in the study had no influence on their eligibility for participation in their sport. Upon voluntary agreement to enroll, participants were asked to sign an informed consent form. Following consent, subjects were asked to complete a series of questionnaires. To determine training history, questions were specific to address training characteristics, sports specific activity and musculoskeletal injury history within the past calendar year. (Appendix 1). Subjects completed the Victorian Institute of Sport Assessment-Patella (VISA-P) overuse injury questionnaire. This is a validated measure for assessing anterior knee pain (Appendix 2).[6,42,43] A single leg decline squat (SLDS) was performed after completion of paper-based questionnaire responses. The single-leg decline squat on a decline board of ⩾15° results in a 40% higher knee extension moment, thus increasing patellar tendon force, compared to the same exercise on a flat floor.[11]Subjects used a 25˚ decline board to perform a unilateral SLDS to approximately 60˚ of knee flexion, or until the onset of symptoms, whichever occurs first. Pain was quantified and recorded using a visual analogue scale (VAS) and a pain-mapping diagram that allows participants to locate the site(s) of any pain during the SLDS (Appendix 3). SLDS was performed on subject's dominant and non-dominant limb. Responses were gathered and divided into basketball athletes and swim athletes. Descriptive statistics were computed to assess differences in loading history, injury history, and loading volume between basketball and aquatic athletes. Results were used for analysis to assess the influence training load has on patellar tendon structural integrity. All questionnaires were completed via hard-copy, using pen and paper provided by investigators.

Biomechanical Data Collection

Instrumentation

The electromagnetic motion capture system (MotionStar, Ascension Technology Corporation, Burlington, VT) with corresponding Ascension Flock of Birds electromagnetic trackers, interfaced to a nonconductive force plate (Type 4060-08; Bertec Corporation, Columbus, Ohio) was used to measure lower body kinematics and kinetics, respectively. Sampling frequency for kinematic data was set at 140 Hz, and set 1400 Hz for kinetic data. Biomechanical variables were quantified using the Motion Monitor for Research v8.0 (Innovative Sports Training, Chicago, Illinois) motion analysis software package.

The subjects were asked to stand on the testing platform in a neutral position, with each foot in the center of the respective force plate. Electromagnetic sensors were positioned unilaterally on the subject's dominant leg as follows:

- Anterior shaft of the third Metatarsal
- Midshaft of the Medial Tibia
- Lateral aspect of the of the Femur
- Midline of the Sacrum

Sensors were secured with double-sided tape, pre-wrap, and white athletic tape. Sensor leads were then gathered in a Velco-Belt wrapped around the subject's waistline and gathered to the lateral side of the subject to ensure they do not impede natural movement during the testing procedures.

A right-hand coordinate system was used to establish world and segment axis system in which the positive direction for the x- axis is anterior, for the y-axis is medial, and for the z-axis is superior. Lower extremity joints (ankle, knee, hip) were defined based on digitization of bony landmarks to calculate the midpoint of each joint. The landmarks utilized were as followed:

- Right and Left ASIS
- Bilateral Medial and Lateral Epicondyles
- Bilateral Medial and Lateral Malleoli
- Bilateral Proximal Phalanx of the Anterior Aspect of the Second Metatarsal

The ankle joint center was defined as the midpoint between the medial and lateral malleoli, knee joint center as the midpoint between the medial and lateral femoral epicondyles, with the hip joint center estimated from the right and left anterior superior iliac spines using the Bell method.[64]

Jump-Landing Task

All subjects performed 5 trials of a standardized jump-landing task. The task required the individual to jump forward off a 30-cm-high box, set at a distance of 50% of their height away from the front edge of the force plates. Participants were instructed to land with one foot in the center of each respective force plates, and immediately jump straight up for maximal vertical height. The investigator ensured that subjects started the jump in a neutral position (i.e., feet shoulder-width apart and toes pointing forward at the edge of the box) and jumping as high as they could after their initial landing from the box. No other verbal instructions were provided so as not to bias the participants' natural movement pattern. A successful jump was characterized by both feet simultaneously leaving the box, jumping forward off the box, reaching the target landing area below, and completing the task in a fluid motion (no pause in movement of body's center of mass after making contact with the ground until takeoff for subsequent jump). If a jump was unsuccessful, the subject was asked to repeat the trial. This was repeated until each subject completes 5 successful jump-landing trials.

Phase Identification

Biomechanical variables for the subject's dominant limb were evaluated across the entire stance phase. The stance phase was defined as the time from initial ground contact (IC), or the time point when the vertical ground reaction force (vGRF) exceeds 10N, until toe-off (vGRF <10 N).[65] The stance phase can be further divided into the *loading* phase (IC through peak knee flexion position = the first 50% of the stance phase) and the *propulsive* phase (peak knee flexion position to toe-off = the second 50% of the stance phase). Peak knee flexion position was defined as the time point when the knee reached its maximum flexion angle during the stance phase.

Data Processing

We estimated the 3-dimensional coordinates of the lower extremity bony landmarks using the Motion Monitor for Research v8.0 (Innovative Sports Training, Chicago, Illinois) motion analysis software package. All kinematic data was smoothed using a 14 Hz fourth-order low-pass Butterworth filter. All kinetic data was interpolated and synchronized with the raw 1400 Hz ground reaction force data. Knee joint motion was defined as the motion of the shank segment relative to the thigh segment using a Cardan angle rotation sequence of Y ((+) flexion/(−) extension), X′ ((+) varus (or tibial adduction)/(−) valgus (or tibial abduction)), Z″ ((+) internal rotation/(−) external rotation). Hip joint motion was defined as motion of the thigh segment relative to the pelvis segment using a Cardan angle sequence of Y ((+) extension/(−) flexion), X′ ((+) adduction/(−)), Z″ ((+) internal rotation/(−) external. The following kinematic variables during the loading phase of the jump landing task were assessed: knee flexion angle at initial ground contact (IGC), maximum knee flexion angle during loading phase, knee flexion displacement, peak internal knee extension moment during first 50% of the loading phase, hip flexion angle at IGC, and maximum hip flexion angle.

Dependent Variable Calculation

The middle three trials from the five jump-landing trials were averaged and utilized for analysis. Four biomechanical variables were assessed using the Flock of Birds Motion Capture System. Knee flexion displacement was calculated as the difference between the knee joint angle (°) at initial ground contact and maximum knee joint angle during the loading phase (enter formula here). The peak internal knee extension moment was defined as the peak internal knee extension moment during the loading phase. This was represented as a (+) extension // (-) flexion moment around the knee. It is dependent not only upon the size of the perpendicular force exerted by the quadriceps but also upon its distance from the knee joint center. Peak vertical ground reaction force (Peak VGRF) was calculated as largest value of VGRF during the stance phase (time from initial contact until toe-off). The loading rate of the vertical ground reaction force (LR VGRF) as defined as the peak VGRF value divided by time from initial ground contact to peak value (N/s^2). Joint moments were normalized to the product of body mass (kg) and height (cm). VGRF data was normalized to body mass (kg). All moments were reported as a positive value. Means, standard deviations, 95% confidence intervals, and ranges for each dependent variable were calculated and reported.

Ultrasound Tissue Characterization

Ultrasound Tissue Characterization (UTC) is a valid and reliable clinical tool that quantifies the structural integrity of tendon tissue.[15,20,21,66] A UTC ultrasound scan of the patellar tendon was taken prior to each participant's biomechanical assessment. UTC scans were performed by a single researcher with previous training using the device. Subjects lied supine on a treatment plinth with their knee bent to approximately 90° of passive flexion to place adequate tension through the patellar tendon for image quality.[19–21] A 10 MHz (focus: 1.3 cm, depth: 3 cm) linear-array transducer (Smartprobe 10L5; Terason 2000, Burlington, Maine, USA) was mounted in the tracking device with a motor-drive and built-in acoustic stand-off pad (UTC Tracker, UTC Imagine, Stein, The Netherlands). The tracking device ensures a consistent position of the transducer through the scan, as the transducer is not able to tilt or rotate once secured. The investigator marked the inferior pole of the patella as the origin of the scan and the tibial tuberosity as the end. Ultrasound coupling gel was applied to the transducer and the participant's anterior knee.

To take the scan, the investigator placed transducer perpendicular to the long axis of the patellar tendon (Appendix 4). Once the inferior patellar pole was visualized on the laptop screen and the transducer centered in both the sagittal and transverse views, the scan was initiated by the investigator pushing "start". The tracking device automatically movedthe transducer along the long axis of the tendon, from the inferior patellar pole to the tibial tuberosity, capturing contiguous transverse grey-scale images at intervals of 0.2mm. One UTC scan was collected bilaterally for each participant. All scans were saved to a secure laptop computer using the participant's de-identified study identification number.

Each scan was acquisitioned to generate a 3-dimensional data block using UTC software (UTC 2011, UTC Imaging).[66] UTC algorithms automatically quantify the stability of pixel brightness over transverse images of the tendon into four echo-types (I-IV): (I) intact and aligned tendon structure, (II) less waving tendon structure, (III) mainly fibrillar tissue, and (IV) mainly amorphous matric with loose fibrils and fluid.[66] The patellar tendon image was analyzed from the inferior pole of the patella to 20 mm distally with percentages of aforementioned echo types in this region of interest (ROI) being calculated. Van Ark et al.[31] found that this ROI consistently presents with localized pain and structural change following acute load.

All scans were de-identified at the time of acquisition to ensure the image processing can be completed with the investigator blinded to participant and day of the scan. The patellar tendon image was manually contoured by a trained investigator. Tendon borders were contoured manually at every 20 frames across the ROI. Once completed, contours were automatically interpolated between the ROIs by the UTC software, generating average percentages of each echo-type for the total ROI and at each contour. Based on their stability/degree of tendon structure, four echo types will be discriminated.[20,66] Percentages of type I, II, III, and IV were classified for each scan. For this study, six values were used to assess patellar tendon structural integrity:

- % Type I
- % Type II
- % Type III
- % Type IV
- Aligned Fibrillar Structure (AFS, echo types I and II)
- Disorganized Tissue Structure (DIS, echo types III and IV)

The cross-sectional areas (CSA) of AFS and DIS were calculated for each transverse scan by manually multiplying the total CSA by the proportion of echo type I + II or echo type III + IV to determine total tendon volume. All percentages represented dependent variables representing tendon structural integrity utilized during statistical analysis. Our study focused on tendon volume percentages of echo types I and II ; short-term structural changes in these echo types have been shown in previous studies examining acute structural change in response to load.[19,31]

Statistical Analysis

All data was analyzed using SPSS v21.0 for Macintosh (IBM, Armonk, New York). Descriptive statistics (means, standard deviations, medians, interquartile ranges, and 95%

confidence intervals) were calculated for all participant demographic information and questionnaire data, kinematic and kinetic dependent variables, and UTC variables. Statistical significance for all tests were set at an *a priori* alpha level of $\alpha = 0.05$.

The following statistical analyses were completed for each study aim:

- Aim 1: We used an independent samples *t*-test to assess for differences in patellar tendon structural integrity between male collegiate basketball players and male collegiate swimmers. Mean % each specific echo type (Type I- Type IV), mean % of AFS and mean % DIS for each cohort were assessed for a statistically significant difference.
- **Aim 2**: We used a Pearson r correlation to measure the relationship between patellar tendon structure and landing kinematic and kinetic variables. Values of % AFS for all subjects were associated with values for each of the following biomechanical dependent variables:
    - Peak VGRF
    - Loading Rate VGRF
    - Initial Internal KEM
    - Peak Internal KEM
    - Initial Knee Flexion Angle
    - Total Knee Flexion Displacement

If association existed between patellar tendon structure and any of the dependent variables, we used partial correlations to control specifically for group to assess where an association exists.

- Aim 3: We used a multiple linear regression model to evaluate the influence of sport, loading history, and biomechanics on tendon structure. Tendon structure was our continuous variable. Independent variables consisted of:
    - Peak VGRF
    - Loading Rate VGRF
    - Initial Internal KEM
    - Peak Internal KEM
    - Initial Knee Flexion Angle
    - Total Knee Flexion Displacement
    - Training Volume per week
    - Time Spent Training on Dry Land
    - RPE during training/ competition

We assessed to see which, if any, of our independent variables can predict patellar tendon structural integrity. Partial correlations were used to control for group (basketball vs. aquatic) if any positive predictive value is found between groups for those variables. A partial correlation determined if the positive predictive value exists specifically in the basketball cohort or the swim cohort.

Chapter IV: Results

Twenty-six participants were included in the final analysis. Two participants, one from each cohort, were excluded from analysis due to lower extremity injury that occurring within six weeks prior to data collection. Participant demographics and training histories are outlined in Table I. Height, weight, and descriptive statistics are represented for basketball and swim cohorts.

Table I: Descriptive Statistics of Study Participants

	Mean	Std. Deviation	Minimum	Maximum	95 % Confidence Interval Lower	95 % Confidence Interval Upper
Basketball (n=13)						
Height (cm)	195.92	12.98	175.00	220.00	188.08	203.77
Mass (kg)	92.74	7.46	80.20	104.30	88.22	97.24
Competitions/week	0.92	1.38	0.00	4.00	0.09	1.76
Training Sessions/week	4.31	1.75	2.00	8.00	3.25	5.37
Strength Training/week	0.62	1.19	0.00	3.00	-0.03	0.5
RPE Competition	4.23	0.93	3.00	6.00	3.67	4.79
RPE Training	5.38	0.77	4.00	7.00	4.92	5.85
Yrs Participated in Court Sport	7.69	2.02	5.00	12.00	6.47	8.91
VISA-P Score (out of 100)	90.69	15.35	54.00	100.00	81.41	99.97
Swim (n=13)						
Height (cm)	178.38	6.68	165.00	190.00	174.35	182.42
Mass (kg)	85.73	6.46	76.00	99.30	81.81	89.63
Competitions/week	0.62	0.51	0.00	1.00	0.31	0.92
Training Sessions/week	9.15	2.34	2.00	11.00	7.74	10.57
Strength Training/week	2.85	0.38	2.00	3.00	2.62	3.07
RPE Comp	0.46	1.66	0.00	6.00	-0.54	1.47
RPE Training	7.15	1.14	5.00	9.00	6.46	7.84

Yrs Participated in Court Sport	2.54	2.40	0.00	7.00	1.09	3.99
VISA-P Score (out of 100)	98.54	2.99	90.00	100.00	96.73	100.34

UTC Characterization

The primary objective for Aim 1 was to explore differences in patellar tendon structure, quantified by Ultrasound Tissue Characterization (UTC) between basketball and swimming athletes. The variables of interest were as follows: % Echo Type I-IV, % AFS (or the combination of percentage of Type I & II), % DFS (or the combination of the percentage of Type III & IV). A significant difference was found between athlete groups in % of Type I fibrillar structure (basketball: 51.6 % ± 9.47%; swim:58.62% ± 6.58%, p= 0.04), as well as % of Type III fibrillar structure (basketball: 4.9 ± 3.49%, swim: 2.52 ± 1.59%, p = 0.04). A significant difference was also found between percentage of AFS (basketball: 94.15 ± 4.38%, swimming: 96.9 ± 2.19%, p = 0.04) and percentage of DFS (basketball: 5.85 ± 4.39%, swimming: 3.07 ± .16%, p=0.03), with basketball athletes presenting with a lower percentage of aligned fibrillar structure and a higher percentage of degenerative fibrillar structure compared to swimmers. No significance was found between groups for the percentage of Type II (p=0.13) and Type IV (p=0.23) alone. Results for Aim 1 analysis support our hypothesis that the basketball cohort would exhibit a significantly lower percentage of aligned fibrillar structure in their dominant leg compared to the swim cohort. Results from the comparison of UTC echo-types between sport can be found in Table II.

Table II: Descriptive Statistics comparing UTC Echo Types (%)

	Basketball		**Swim**			
UTC Percentages	Mean	S.D.	Mean	S.D.	P-Value	Effect Size
Type I	51.60	9.47	58.62	6.58	0.04*	-0.87
Type II	42.54	7.86	38.27	5.65	0.13	0.63
Type III	4.90	3.49	2.52	1.59	0.04*	0.94
Type IV	0.95	0.97	0.55	0.60	0.23	0.51
AFS	94.15	4.38	96.90	2.19	0.04	-0.84
DFS	5.85	4.38	3.07	0.16	0.03*	1.22

*Indicates statistical significant at $p < 0.05$.
AFS = aligned fibrillar structure, DFS = disorganized fibrillar structure

Biomechanics

Kinematic and kinetic variables are outlined in Table III. The primary kinematic variables of interest were peak knee flexion angle and knee flexion displacement across the stance phase. The mean knee flexion displacement for the basketball cohort was less than that of the swimming cohort (67.34 ± 15.4 vs. 80.57 ± 17.04, respectively; p=0.049). Additionally, there was a statistically significant difference in peak knee flexion angle between cohorts (basketball: 88.87 ± 12.04 degrees, swimming: 98.69 ±18.5 degrees; p=0.01), indicating that swim athletes reached a greater maximum knee flexion angle during the stance phase of the jump landing task than basketball athletes. Further analysis found statistically significant difference in hip abduction displacement (basketball: -2.28 ± 3.18, swimming: -6.13 ±5.36, p = 0.033) and hip external rotation displacement (basketball: -9.71 ± 4.10, swimming: -5.03 ±3.44, p=0.004) during jump landing task, with the basketball cohort demonstrating lesser hip abduction displacement, and greater hip external rotation displacement than swimmers. Additionally, the basketball cohort presented with lesser knee varum displacement (basketball: 2.28 ± 2.21, swimming: 7.70 ± 6.07, *p*=0.005), and lesser knee internal rotation (basketball: 14.2 ± 7.9, swimming: 21.09 ± 8.29, *p*=0.04) during the jump landing task than the swimming cohort.

Table III: Descriptive Statistics comparing Biomechanical Variables

	Basketball		**Swim**			
	Mean	S.D.	Mean	S.D.	P-Value	Effect Size
Hip Flexion DSP	-45.39	20.50	-60.27	19.45	0.07	0.74
Hip Adduction DSP	5.03	3.36	5.05	4.06	0.99	-0.01
Hip Abduction DSP	-2.28	3.18	-6.13	5.36	0.03*	0.90
Hip Internal Rotation DSP	7.51	7.97	12.32	6.30	0.10	-0.67
Hip External Rotation DSP	-9.71	4.10	-5.03	3.44	0.00*	-1.24
Max Knee Flexion Angle	88.87	12.04	98.69	18.50	0.01*	-0.64
Knee Flexion DSP	67.34	15.40	80.57	17.04	0.05*	-0.82
Knee Valgus DSP	-8.84	3.74	-5.90	4.10	0.07	-0.75
Knee Varus DSP	2.28	2.21	7.70	6.07	0.01*	-1.31
Knee Internal Rotation DSP	14.20	7.90	21.09	8.29	0.04*	-0.85
Knee External Rotation DSP	-3.51	3.80	-5.24	5.38	0.35	0.38
VGRF (BW)	2.44	0.82	2.23	0.73	0.52	0.27
APGRF (BW)	0.09	0.07	0.06	0.08	0.26	0.40
MLGRF (BW)	0.28	0.09	0.22	0.04	0.05*	0.92
Peak Knee Flexion Moment	40.07	19.11	61.43	63.01	0.25	-0.52
Peak Knee Extension Moment	-42.38	16.24	-34.75	13.38	0.43	-0.44
Peak Knee Varus Moment	37.80	25.40	45.00	40.25	0.59	-0.22

Peak Knee Valgus Moment	-52.36	19.89	-87.96	80.53	0.14	0.71
Peak Knee Int Rot Moment	27.72	14.02	32.58	37.41	0.67	-0.19
Peak Knee Ext Rot Moment	-45.50	22.13	-42.81	29.38	0.80	-0.10

*Indicates statistical significance at $p < 0.05$

The primary kinetic variables of interest were as follows: VGRF, APGRF, MLGRF, and 3-dimensional knee angular moments. The mean VGRF for the basketball cohort was nearly equal to that of the swimming cohort (2.44 BW ± 0.82 BW vs. 2.23 BW ± 0.73 BW, *p*=.052.) No significant difference was found between groups. However, a significant difference was found in the medial-lateral ground reaction force with the basketball cohort displaying greater MLGRF compared to the swimming cohort (0.28 BW ± 0.09 BW vs. 0.22 BW ± 0.04 BW, *p*=0.04). Peak knee extension moment (pKEM) was the primary internal knee moment of interest, and no significance difference was found in pKEM between basketball cohort and swimming cohort (-42.38 BW ± 16.24 BW vs. -34.75 BW ± 13.38 BW, *p*=0.43). All kinetic variables assessed are outlined in Table III.

The aforementioned biomechanical analysis provides insight into movement characteristics for each cohort prior to analyzing tendon structure between groups (Aim 1) and assessing for correlation between structure and function (Aim 2). Our results indicate that during a double-limb landing task, swim athletes undergo greater sagittal plane excursion. Prior research has demonstrated this result to have varying effect on patellar tendon structure. Rosen et al.[23] showed less knee flexion displacement increases the strain on the patellar tendon. Mann et al.[44] conversely presented greater sagittal plane motion increases the shear and compressive force acting upon the tendon, leading to patellar tendon structural abnormality. No prior research has addressed the impact of hip and knee rotation on patellar tendon structure. Our basketball cohort presented with greater hip external rotation and lesser knee internal rotation, utilizing a lateral and posterior chain dominant landing strategy that could impact force through the tendon.

Correlational Analysis

The primary objective of Aim 2 explored the correlation between jump-landing biomechanics and associated patellar tendon structural integrity. The variables assessed for correlation were % of each UTC echo-type, % AFS, % DFS and the following biomechanical variables: Peak VGRF, Loading Rate VGRF, Initial Internal KEM, Peak Internal KEM, Initial Knee Flexion Angle, and Total Knee Flexion Displacement. No statistically significant correlations existed between any of the biomechanical variables and any of the UTC echo-types, %AFS or %DFS. Knee valgus displacement demonstrated statistically significant correlation within groups with the percentage of Type I and Type II tendon structure, indicating that as knee valgus displacement increases, tendon structural integrity decreases (Type 1 $r=0.402$ $p=0.042$: Type II $r= -.439$, $p= 0.025$). Further analysis showed no other significant correlation between patellar tendon structure and biomechanical variables in this cohort. Results from the correlation analysis are outlined in Table IV.

Table IV: Correlation Analysis between Biomechanics and UTC Echo Types (%)

		Type I	Type II	Type III	Type IV	% AFS	% DFS
Knee Flexion DSP Avg	Pearson Correlation (*r*)	0.011	0.044	-0.126	-0.042	0.111	-0.11
	p-value	0.957	0.831	0.54	0.839	0.589	0.594
Knee Varus DSP Avg	Pearson Correlation	0.272	-0.228	-0.24	-0.093	0.211	-0.21
	Sig. (2-tailed)	0.179	0.263	0.238	0.651	0.3	0.298
Knee Valgus DSP Avg	Pearson Correlation	.402	-.439	-0.153	0.037	0.116	-0.11
	Sig. (2-tailed)	**0.042***	**0.025***	0.456	0.856	0.572	0.581
Knee IR DSP Avg	Pearson Correlation	0.006	-0.055	0.066	0.152	-0.09	0.087
	Sig. (2-tailed)	0.976	0.789	0.748	0.457	0.658	0.673
Knee ER DSP Avg	Pearson Correlation	0.129	-0.296	0.235	0.314	-0.26	0.257
	Sig. (2-tailed)	0.528	0.142	0.248	0.119	0.2	0.204
vGRF Peak Avg	Pearson Correlation	0.09	-0.096	-0.021	-0.071	0.03	-0.03
	Sig. (2-tailed)	0.661	0.64	0.918	0.731	0.884	0.874
APGRF Peak Avg	Pearson Correlation	-0.14	0.222	-0.062	-0.153	0.083	-0.08
	Sig. (2-tailed)	0.483	0.276	0.765	0.455	0.688	0.685
MLGRF Peak Avg	Pearson Correlation	-0.29	0.305	0.127	-0.011	-0.1	0.099
	Sig. (2-tailed)	0.152	0.129	0.536	0.957	0.62	0.631
Peak Knee Extension Moment Avg	Pearson Correlation	0.019	-0.034	0.022	0.015	-0.02	0.021
	Sig. (2-tailed)	0.926	0.869	0.914	0.941	0.924	0.918

*Indicates statistical significant at $p < 0.05$.
DSP= Displacement, Avg= Average, IR=Internal Rotation, ER= External Rotation, vGRF=Vertical Ground Reaction Force, APGRF= Anterior-Posterior Ground Reaction Force, MLGRF= Medial-Lateral Ground Reaction Force

We hypothesized that a negative association would exist between kinetic and kinematic variables and patellar tendon structural integrity, such that larger sagittal plane knee motion and higher lower extremity loading during a jump-landing task would associate with a lower percentage of aligned fibrillar structure of the dominant limb patellar tendon. Our results did not support this hypothesis. Prior research shows a negative relationship exists between tendon structural integrity and quadriceps dominant movement quality, represented by motions in the sagittal plane such as knee flexion displacement and knee extension moment. [28,53,67] It's possible our findings are a result of a small sample size, or a sample that is not representative of the collegiate athletic population of basketball athletes or swimmers that we are interested in. It is also a possibility that given the time-point of our data collection, factors such as cumulative fatigue or cumulative tissue overload that may occur during the in-season period were not present due to the pre-season timing of data collection, accounting for a lack of association. Further research could utilize meaningful bench marks, including pre-season, mid-season, and post-season analysis, to examine the effect of training load on both movement quality and tendon structural integrity.

Multiple Linear Regression Model

Aim 3 explored our biomechanical variables, self-reported measures of function and training history, interpolating if predictive value and influence on patellar tendons structure exists. The variables of interest were: Peak VGRF, Loading Rate VGRF, Initial Internal KEM, Peak Internal KEM, Initial Knee Flexion Angle, Total Knee Flexion Displacement, VISA-P Score, Single Leg Decline Squat, Strength/Plyometric Training. Despite the lack of support for correlation found in Aim 2, a multiple linear regression model was used to evaluate the influence of these variables on patellar tendon structure within groups. Regression analysis showed none of the chosen variables to be significant predictors of patellar tendon structure (% AFS and % DFS). Results can be found in Table V.

Table V: Effect of Movement Quality and Training to predict AFS on UTC Images

	Unstandardized Coefficients		Standardized Coefficents		
	B	Standard Error	Beta	t	*p-value*
Knee Flexion Displacement Avg	0.03	0.207	0.143	0.146	0.886

Max Knee Flexion Angle	-0.017	0.228	-0.076	-0.076	0.941
Peak KEM Average	-0.685	0.083	0.543	-0.321	0.207
Loading Rate VGRF	-2.822	57.915	-0.016	-0.049	0.962
Peak VGRF Average	0.051	1.532	0.011	0.034	0.974
Competitions (per week)	0.526	1.397	0.148	0.377	0.712
Training Sessions (per week)	-0.683	0.836	-0.594	-0.817	0.429
LE Strength Sessions (per week)	0.672	1.184	0.261	0.567	0.58
Plyometric Sessions (per week)	-0.972	1.385	-0.283	-0.702	0.495
RPE Competition	-0.741	1.328	-0.47	-0.558	0.586
RPE Training	-0.211	1.384	-0.075	-0.153	0.881
Yrs. Participated Court Sport	-0.246	0.434	-0.229	-0.567	0.58
VISA-P Score	-0.025	0.111	-0.08	-0.23	0.822

a. Dependent Variable: % AFS

*Indicates statistical significant at $p < 0.05$.

Trends in the statistical analysis lead to further analysis of training characteristics and self-reported load between cohorts. All results are outlined in Table VI for between group comparison of training characteristics. Measures of training volume were to be estimated as average per week over their previous six months of training. A statistically significant difference between groups was found within the following variables: competitions per week (basketball: 0.92 ± 1.38, swimming 0.62 ±0.51, *p*0=0.03), lower extremity strength sessions per week (basketball: 0.62 ± 1.19, swimming 2.85 ± 0.38, *p*=0.002), plyometric sessions per week (p=.001), years spent participating in a court-based sport (basketball 7.69 ±2.02, swimming 2.54 ± 2.40, *p*=0.011), and VISA-P Score (basketball 90.69 ± 5.35, swimming 98.54 ± 2.99, *p*=0.001). Basketball presented with significantly greater competitions per week, a greater volume of plyometric activity per week, longer time spent participating in court-based sport, and a greater VISA-P score than the swim cohort. Swimmers experienced significantly greater lower extremity strength training sessions than the basketball cohort. This can be attributed to the structured nature of the Varsity Swim Team, with mandatory bi-weekly weight training sessions with a university hired strength coach.

Table VI: Comparison of Training Characteristics between Group

	t	*p-value*	95% Confidence Interval of the Difference	
			Lower	Upper
Competitions (per week)	0.754	0.003♠	-0.535	1.15
Training Sessions (per week)	-5.98	0.955	-6.519	-3.173
LE Strength Sessions (per week)	-6.431	0.002♣	-2.947	-1.515
Plyometric Sessions (per week)	2.93	0.005♠	0.318	1.836
RPE Competitions	7.135	0.8	2.679	4.86
RPE Training Sessions	-4.631	0.404	-2.558	-0.981
Years Spent participating in court based sport	5.926	0.011♠	3.359	6.949
VISA-P Score	-1.809	0.001♠	-16.8	1.108

*Indicates statistical significant at $p < 0.05$.
♠Indicates greater difference in basketball athletes
♣Indicates greater difference in swim athletes

Overall, our results did not identify with a majority of what's been represented in previous literature regarding movement quality and patellar tendon structure. There is evidence to support that frontal and transverse plane motion can influence tendon structure. Our results did support the finding that volume and quality of load can alter fibrillar alignment of the tendon. Despite the volume that aquatic athletes endure, their lack of plyometric activity may lead to an overall healthier tendon structure compared to court-based sports such as basketball. Our results demonstrate that training history and athlete type have a greater influence on tendon structure than movement biomechanics.

Chapter V: Manuscript

Introduction

Tendinopathy is a load-based pathology, commonly developing in athletic populations due to excessive load from high training and competition bouts.[1,24,27,46]Despite its prevalence in sports medicine, a gold standard treatment and management plan is yet to be discovered. Its diagnosis proves difficult as it's diagnosis is traditionally based on location of self-reported pain, load-based pain reproduction, and/or structural pathology on diagnostic imaging. The literature demonstrates that the clinical presentation of tendinopathy can vary, with tendon-related symptoms and structural pathology not always occurring simultaneously.[15,25–27,34] Cook et al.[32] proposes that tendon pathology occurs along a continuum of three stages: reactive tendinopathy, tendon disrepair, and degenerative tendinopathy. All stages correlate to a level of tendon structural abnormality, occurring with or without pain or functional limitation. [7]

Sports involving high volumes of jumping and landing, such as basketball and volleyball, experience the highest incidence of patellar tendinopathy (PT).[11,12] It is estimated that over 13% of athletes involved in basketball and volleyball will experience patellar tendon pain at some point in their competitive career.[11] The effect of tendinopathy on sport participation is difficult to measure, as it is rare that an individual will miss a training or competition bout due to this condition, especially early in its pathoetiologic process. It has been shown that lower extremity strength training in combination with plyometric activity increases the risk for developing PT.[13] Sport-specific motions that apply high eccentric loads to the knee extensor mechanism (i.e. squatting, lunging, cutting) are typically reported as painful by athletes with symptomatic patellar tendinopathy. External load, or the load exerted on the body, is a primary pathoetiologic component in PT; however, some sports known for their high training and competition volumes, such as competitive swimming, aren't represented in the literature.[3]

Swimmers train an average of 5-6 hours per day through a competitive season that can last up to 12 months.[14] Their training is unique, as the majority takes place in an aquatic environment with un-weighted horizontal gravitational and rotational forces acting on the musculoskeletal system.[14] Therefore, differences in loading parameters between basketball and swimming athletes likely influence the difference in incidence and prevalence of PT between sports.[1,3] However, associations between training characteristic parameters and patellar tendon structure have not been systematically assessed in these specific athlete groups.

Ultrasound imaging has been a key technique used to evaluate tendon structural integrity when diagnosing tendinopathy. Advances in ultrasound imaging techniques allow for enhanced evaluation of tendon structural integrity.[15] Ultrasound Tissue Characterization (UTC) is a novel ultrasonographic imaging technology that quantifies tendon structural integrity through advanced computer algorithms that categorize the characteristics, specifically the orientation and stability, of the collagen fibers within the tendon.[15,19,20] Prior research has utilized the technology to assess the structural changes of tendon in plyometric based sports such as volleyball and basketball, assessing how factors such as load and time impact tendon structure.[19,22,23]The ability of UTC to quantify tendon structure and assess response to training load makes it an ideal tool to assess differences existing tendon structure between land vs. aquatic sport athletes.[19,21,22]

Lower extremity kinematics and kinetics may contribute the development and progression of patellar tendinopathy in athletic populations. Rosen et al.[23] found that individuals with PT displayed different movement strategies compared to a healthy population during a double limb jump-landing task, including increased knee flexion displacement and increased peak internal knee extension moment. One key variable that influences tendon structure is the overall magnitude of mechanical load and the direction of mechanical load endured by the patellar tendon.[4,10] Changes in sagittal plane movement patterns in those with PT may be due to the associated alterations of quadriceps and hamstring activation in response to symptoms.[23] Given the patellar tendon's role in dissipating externally applied load and in generation of internal moment through the knee extensor mechanism, understanding movement characteristics of individuals at high-risk of developing patellar tendinopathy is important to prescribe and manage injury prevention and rehabilitation programs.

Current literature suggests that athletes with structural abnormalities of the patellar tendon have a higher risk of developing patellar tendinopathy than athletes without structural pathology.[6,20,24,25] However, it is unknown how type of training load (i.e. primarily plyometric vs. non-plyometric) and lower extremity biomechanics influence tendon structural integrity. The primary aim of this study was to determine if there are differences in patellar tendon structure between athletes participating in primarily land-based training (basketball) and aquatic-based training (swimming). The second aim examined if specific landing kinematic & kinetic patterns influence patellar tendon structural integrity of the dominant limb of male collegiate basketball players and swimming athletes. Finally, the third aim examined the influence of both training load variables and biomechanical variables on tendon structure.

Methods

Participants

Twenty-Six Junior Varsity (JV) Men's Basketball and Varsity Men's Swimming athletes from the University of North Carolina at Chapel Hill were recruited and screened to participate in the research study. Equal numbers of participants were enrolled from the Junior Varsity (JV)

Men's Basketball team (n=13) and the and Varsity Men's Swimming team (n=13). All eligible participants were between the ages of 18-22. Participants were excluded if they have a history of lower extremity surgery or have sustained a lower extremity injury in the last 6 months that prevents them from completing any of the assessments. Two participants, one basketball athlete and one swim athlete, were excluded due to thiscriteria. Participants were asked to complete a series of questionnaires addressing exclusion criteria (Table 1). Anthropometric data (height and weight) was collected by one clinician. Height was measured in centimeters (cm) using a stadiometer (Detecto Model 758C, Cardinal Scale Manufacturing, Webb City, MO, USA), and weight measured in kilograms (kg) using a digital scale (Perspective Enterprises, Portage, MI, USA). Informed consent was obtained from all participants prior to study initiation in accordance with the University of North Carolina at Chapel Hill's Institutional Review Board.

Self-Reported Function & Training Load Questionnaires

Following informed consent, subjects were asked to complete a series of self-reported questionnaires. To determine training history, questions were specific to address training characteristics, sports specific activity and musculoskeletal injury history within the past calendar year. (Appendix 1). Subjects completed the Victorian Institute of Sport Assessment-Patella (VISA-P) overuse injury questionnaire. This is a validated measure for assessing anterior knee pain (Appendix 2).[6,42,43] A single leg decline squat (SLDS) was performed after completion of paper-based questionnaire responses. The single-leg decline squat on a decline board of ⩾15° results in a 40% higher knee extension moment, thus increasing patellar tendon loading, compared to the same exercise on a flat floor.[11] Subjects used a 25˚ decline board to perform a unilateral SLDS to approximately 60˚ of knee flexion, or until the onset of symptoms, whichever occured first. Pain was quantified and recorded using a visual analogue scale (VAS) and a pain-mapping diagram that allowed participants to locate the site(s) of any pain during the SLDS

(Appendix 3). SLDS was performed on both subject's dominant and non-dominant limb. All questionnaires were completed via hard-copy.

Biomechanical Data Collection

Instrumentation

The electromagnetic motion capture system (MotionStar, Ascension Technology Corporation, Burlington, VT) with corresponding Ascension Flock of Birds electromagnetic trackers, interfaced to a nonconductive force plate (Type 4060-08; Bertec Corporation, Columbus, Ohio) was used to measure lower extremity kinematics and kinetics, respectively. Sampling frequency was set at 140 Hz for kinematic data and 1400 Hz for kinetic data. Biomechanical variables were quantified using the Motion Monitor for Research v8.0 (Innovative Sports Training, Chicago, Illinois) motion analysis software package.

The subject was asked to stand on the testing platform in a neutral position, with each foot in the center of the respective force plate. Electromagnetic sensors were positioned unilaterally on the subject's self-reported dominant leg as follows: anterior shaft of the third metatarsal, midshaft of the medial tibia, lateral aspect of the of the femur, midline of the sacrum. Sensors were secured with double-sided tape, pre-wrap, and white athletic tape. Sensor leads were then gathered in a Velco-Belt wrapped around the subject's waistline and gathered to the lateral side of the subject to ensure they did not impede natural movement during the testing procedures.

A right-hand coordinate system was used to establish world and segment axis system in which the positive direction for the x- axis is anterior, for the y-axis is medial, and for the z-axis is superior. Lower extremity joints (ankle, knee, hip) were defined based on digitization of bony landmarks to calculate the midpoint of each joint. The landmarks utilized were followed: right and left ASIS, bilateral medial and lateral epicondyles, bilateral medial and lateral malleoli, bilateral proximal phalanx of the anterior aspect of the second metatarsal. The ankle joint center was defined as the midpoint between the medial and lateral malleoli, knee joint center as the midpoint between the medial and lateral femoral epicondyles, with the hip joint center estimated from the right and left anterior superior iliac spines using the Bell method.[64]

Jump-Landing Task

All subjects performed 5 trials of a standardized double-limb jump-landing task. The task required the individual to jump forward off a 30-cm-high box, set at a distance of 50% of the subject's height away from the front edge of the force plates. Subjects were instructed to land with one foot in the center of each respective force plate, and immediately jump straight up for maximal vertical height. The investigator ensured that subjects started the jump in a neutral position (i.e., feet shoulder-width apart and toes pointing forward at the edge of the box). No other verbal instructions were provided so as not to bias the subjects' natural movement pattern. A successful jump was characterized by both feet simultaneously leaving the box, jumping forward off the box, reaching the target landing area on the force plates, and completing the task in a fluid motion (no pause in movement of body's center of mass after making contact with the ground until takeoff for subsequent jump). If a jump was unsuccessful, the subject was asked to repeat the trial until each subject completed 5 successful jump-landing trials.

Biomechanical Data Reduction

The middle three trials from the five jump-landing trials were averaged and utilized for analysis. If one of these three middle trials was faulty, one of the other two trials (first or fifth) was utilized for analysis. Initial ground contact (IC) for variable reduction was defined as the time point at which the vertical ground reaction force (vGRF) exceeded 10N. Similarly, toe-off was defined as the time frame where vGRF was less than 10N. The stance phase was defined as the period of time between IC and toe-off. Five biomechanical variables were assessed. Knee flexion angle (°) at initial contact and maximum knee flexion angle (°) through the stance phase were collected for all trials. Knee flexion displacement was calculated as the difference between the knee joint angle (°) at initial ground contact and maximum knee joint angle during the stance phase. The peak internal knee extension moment was defined as the peak internal knee extension moment during the stance phase. This was represented as (-) extension / (+) flexion moment around the knee based on the right-hand rule system of joint rotations. Peak vertical ground reaction force (Peak VGRF) was calculated as largest value of VGRF during the stance phase (time from initial contact until toe-off). Joint moments were normalized to the product of body mass (kg) and height (cm). VGRF data was normalized to body mass (kg). All moments were reported as a positive value. Means, standard deviations, 95% confidence intervals, and ranges (minimums and maximums) for each dependent variable were calculated and reported. (Table II)

Ultrasound Tissue Characterization

Ultrasound Tissue Characterization (UTC) is a valid and reliable clinical tool that quantifies the structural integrity of tendon tissue.[15,20,21,66] UTC scans were performed by a single researcher (L.S.) with previous training using the device. Subjects lay supine on a treatment plinth with their knee bent to approximately 90-100° of passive flexion to place adequate tension through the patellar tendon for image quality.[19–21] A 10 MHz (focus: 1.3 cm, depth: 3 cm) linear-array transducer (Smartprobe 10L5; Terason 2000, Burlington, Maine, USA) was mounted in the tracking device with a motor-drive and built-in acoustic stand-off pad (UTC Tracker, UTC Imagine, Stein, The Netherlands). The tracking device ensures a consistent position of the transducer through the scan, as the transducer is not able to tilt or rotate once secured. The investigator marked the inferior pole of the patella as the origin of the scan and the tibial tuberosity as the end. Ultrasound coupling gel was applied to the transducer and the subject's anterior knee.

To take the scan, the investigator placed transducer perpendicular to the long axis of the patellar tendon (Appendix 4). Once the inferior patellar pole was visualized on the laptop screen and the transducer centered in both the sagittal and transverse views, the scan was initiated by the investigator pushing "start". The tracking device automatically moved the transducer along the long axis of the tendon, from the inferior patellar pole to the tibial tuberosity, capturing contiguous transverse grey-scale images at intervals of 0.2mm. One UTC scan was collected on each limb for each subject, though only the scan for the self-reported dominant limb was utilized for analysis. All scans were saved to a secure laptop computer using the subject's de-identified study identification number.

Each scan was acquisitioned to generate a 3-dimensional data block using UTC software (UTC 2011, UTC Imaging).[66] UTC algorithms automatically quantified the stability of pixel brightness over transverse images of the tendon into four echo-types (I-IV): (I) intact and aligned tendon structure, (II) less waving tendon structure, (III) mainly fibrillar tissue, and (IV) mainly amorphous matric with loose fibrils and fluid.[66] The patellar tendon image was analyzed from the inferior pole of the patella to 20 mm distally with percentages of aforementioned echo types in this region of interest (ROI) being calculated. Van Ark et al.[31] found that this ROI consistently presents with localized pain and structural change following acute load.

All scans were de-identified at the time of acquisition to ensure the image processing can be completed with the investigator blinded to participant and day of the scan. The patellar tendon image was manually contoured by a trained investigator (K.R) ($ICC_{2,1}$ = 0.75-0.86). Tendon borders were contoured manually at every 20 frames across the ROI. Once completed, contours were automatically interpolated between the ROIs by the UTC software, generating average percentages of each echo-type for the total ROI and at each contour. Based on their stability/degree of tendon structure, four echo types were discriminated.[20,66] Percentages of type I, II, III, and IV were classified for each scan. For this study, six values have been used to assess patellar tendon structural integrity: % Type I, % Type II, % Type III, % Type IV, Aligned Fibrillar Structure (AFS, echo types I and II), Disorganized Tissue Structure (DIS, echo types III and IV). All percentages represent dependent variables of levels of tendon structural integrity utilized during statistical analysis. Our study focused tendon echo-type percentages on echo types I and II; short-term structural changes in these echo types have been shown in previous studies examining acute structural change in response to load.[19,31]

Statistical Analysis

All data was analyzed using SPSS v21.0 for Macintosh (IBM, Armonk, New York). Descriptive statistics (means, standard deviations, medians, interquartile ranges, and 95% confidence intervals) will be calculated for all participant demographic information and questionnaire data, kinematic and kinetic dependent variables, and UTC variables. Statistical significance for all tests were set at an *a priori* alpha level of $\alpha = 0.05$.

The following statistical analyses were completed for each study aim:

- Aim 1: We used independent samples *t*-test to assess for differences in patellar tendon structural integrity between male collegiate basketball players and male collegiate swimmers. Mean % of each specific echo type (Type I- Type IV), mean % of AFS and mean % DFS for each cohort were assessed for a statistically significant difference.
- Aim 2: We used a Pearson r correlation to measure the relationship between patellar tendon structure and landing kinematic and kinetic variables. Values of % AFS for all

subjects were associated with values for each of the following biomechanical dependent variables:

  - Peak VGRF
  - Loading Rate VGRF
  - Initial Internal KEM
  - Peak Internal KEM
  - Initial Knee Flexion Angle
  - Total Knee Flexion Displacement

If association existed between patellar tendon structure and any of the dependent variables, we used partial correlations to control specifically for group to assess where an association exists.

- Aim 3: We used a multiple linear regression model to evaluate the influence of sport, loading history, and biomechanics on tendon structure. Tendon structure was our continuous variable. Independent variables consisted of:
  - Peak VGRF
  - Loading Rate VGRF
  - Initial Internal KEM
  - Peak Internal KEM
  - Initial Knee Flexion Angle
  - Total Knee Flexion Displacement
  - Training Volume per week
  - Time Spent Training on Dry Land
  - RPE during training/ competition

We assessed which, if any, of our independent variables can predict patellar tendon structural integrity. Partial correlations were used to control for group (basketball vs. aquatic) if any positive predictive value was found between groups for those variables. A partial correlation determined if the positive predictive value existed exclusively in the basketball cohort or the swim cohort.

Results

Twenty-six participants were included in the final analysis. Two participants, one from each cohort, were excluded from analysis due to lower extremity injury that occurred within six weeks prior to data collection. Participant demographics and training histories are outlined in Table I. Height, weight, and descriptive statistics are represented for basketball and swim cohorts.

The primary objective for Aim 1 was to explore differences in patellar tendon structure, quantified by Ultrasound Tissue Characterization (UTC) between basketball and swimming athletes. The variables of interest were as follows: % Echo Type I-IV, % AFS (or the combination of percentage of Type I & II), % DFS (or the combination of the percentage of Type III & IV). A significant difference was found between athlete groups in % of Type I fibrillar structure (basketball: 51.6 % ± 9.47%; swim:58.62% ± 6.58%, *p*=0 .04), as well as % of Type III fibrillar structure (basketball: 4.9 ± 3.49%, swim: 2.52 ± 1.59%, *p*0= .04). A significant difference was also found between percentage of AFS (basketball: 94.15 ± 4.38%, swimming: 96.9 ± 2.19%, *p*0=.04) and percentage of DFS (basketball: 5.85 ± 4.39%, swimming: 3.07 ± .16%, *p*=0.03), with basketball athletes presenting with a lower percentage of aligned fibrillar structure and a higher percentage of degenerative fibrillar structure compared to swimmers. No significance difference was found between groups for the percentage of Type II and Type IV alone. Results for Aim 1 analysis support our hypothesis that the basketball cohort would exhibit a significantly lower percentage of aligned fibrillar structure in their dominant leg than the swim cohort. Results from the comparison of UTC echo-types between sport can be found in Table III.

Kinematic and kinetic variables are outlined in Table II. The primary kinematic variables of interest were peak knee flexion angle and knee flexion displacement across the stance phase. The mean knee flexion displacement for the basketball cohort was less than that of the swimming cohort (67.34 ± 15.4 vs. 80.57 ± 17.04, respectively; p=0.049). Additionally, there was a statistically significant difference in peak knee flexion angle between cohorts (basketball: 88.87 ± 12.04 degrees, swimming: 98.69 ±18.5 degrees; p=0.01), indicating that swim athletes reached a greater maximum knee flexion angle during the stance phase of the jump landing task than basketball athletes. Further analysis found a statistically significant difference in hip abduction displacement (basketball: -2.28 ± 3.18, swimming: -6.13 ±5.36, p = 0.033) and hip external rotation displacement (basketball: -9.71 ± 4.10, swimming: -5.03 ±3.44, p=0.004) during jump landing task, with the basketball cohort demonstrating lesser hip abduction displacement, and greater hip external rotation displacement than swimmers. Additionally, the basketball cohort presented with lesser knee varum displacement (basketball: 2.28 ± 2.21, swimming: 7.70 ± 6.07, p=0.005), and lesser knee internal rotation (basketball: 14.2 ± 7.9, swimming: 21.09 ± 8.29, p=0.04) during the jump landing task than the swimming cohort.

The primary kinetic variables of interest were as follows: VGRF, APGRF, MLGRF, and 3-dimensional knee angular moments. The mean VGRF for the basketball cohort was nearly equal to that of the swimming cohort (2.44 BW ± 0.82 BW vs. 2.23 BW ± 0.73 BW, p=.052.) No significant difference was found between groups. However, a significant difference was found in the medial-lateral ground reaction force with the basketball cohort displaying greater MLGRF compared to the swimming cohort (0.28 BW ± 0.09 BW vs. 0.22 BW ± 0.04 BW, *p*=0.04). Peak knee extension moment (pKEM) was the primary internal knee moment of interest, and no significance difference was found in pKEM between basketball cohort and swimming cohort (-42.38 BW ± 16.24 BW vs. -34.75 BW ± 13.38 BW, *p*=0.43). All kinetic variables assessed are outlined in Table II.

The aforementioned biomechanical analysis provided insight into movement characteristics for each cohort prior to analyzing tendon structure between groups (Aim 1) and assessing for correlation between structure and function (Aim 2). Our results indicate that during a double-limb landing task, swim athletes undergo greater sagittal plane excursion. Prior research has demonstrated this result to have varying effect on patellar tendon structure. Rosen et al.[23] showed less knee flexion displacement increases the strain on the patellar tendon. Mann et al.[44] conversely presented greater sagittal plane motion increases the shear and compressive force acting upon the tendon, leading to patellar tendon structural abnormality. No prior research has addressed the impact of hip and knee rotation on patellar tendon structure. Our basketball cohort presented with greater hip external rotation and lesser knee internal rotation, utilizing a lateral and posterior chain dominant landing strategy that could impact force through the tendon.

The primary objective of Aim 2 explored the correlation between jump-landing biomechanics and associated patellar tendon structural integrity. The variables assessed for correlation were % of each UTC echo-type, % AFS, % DFS and the following biomechanical variables: Peak VGRF, Loading Rate VGRF, Initial Internal KEM, Peak Internal KEM, Initial Knee Flexion Angle, and Total Knee Flexion Displacement. No statistically significant correlations existed between any of the biomechanical variables and any of the UTC echo-types, %AFS or %DFS. Knee valgus displacement demonstrated statistically significant correlation within groups with the percentage of Type I and Type II tendon structure, indicating that as knee valgus displacement increases, tendon structural integrity decreases (Type 1 r=0.402 p=0.042: Type II r= -.439, p= 0.025). Further analysis showed no other significant correlation between patellar tendon structure and biomechanical variables in this cohort. Results from the correlation analysis are outlined in Table IV.

We hypothesized that a negative association would exist between kinetic and kinematic variables and patellar tendon structural integrity, such that larger sagittal plane knee motion and higher lower extremity loading during a jump-landing task would associate with a lower percentage of aligned fibrillar structure of the dominant limb patellar tendon. Our results did not support this hypothesis. Prior research shows a negative relationship exists between tendon structural integrity and quadriceps dominant movement quality, represented by motions in the sagittal plane such as knee flexion displacement and knee extension moment.[28,53,67] It is possible our findings are a result of a small sample size, or a sample that is not representative of the collegiate athletic population of basketball athletes or swimmers that we are interested in. It is also a possibility that given the time-point of our data collection, factors such as cumulative fatigue or cumulative tissue overload that may occur during the in-season period were not present due to the pre-season timing of data collection, accounting for a lack of association. Further research could utilize meaningful bench marks, including pre-season, mid-season, and post-season analysis, to examine the effect of training load on both movement quality and tendon structural integrity.

Aim 3 explored our biomechanical variables, self-reported measures of function and training history, interpolating if predictive value and influence on patellar tendons structure exists. The variables of interest were: Peak VGRF, Loading Rate VGRF, Initial Internal KEM, Peak Internal KEM, Initial Knee Flexion Angle, Total Knee Flexion Displacement , VISA-P

Score, Single Leg Decline Squat, Strength/Plyometric Training. Despite the lack of support for correlation found in Aim 2, a multiple linear regression model was used to evaluate the influence of these variables on patellar tendon structure within groups. Regression analysis showed none of the chosen variables to be significant predictors of patellar tendon structure (% AFS and % DFS). Results can be found in Table V.

Trends in the statistical analysis lead to further analysis of training characteristics and self-reported load between cohorts. All results are outlined in Table VI for between group comparison of training characteristics. Measures of training volume were to be estimated as average per week over their previous six months of training. A statistically significant difference between groups was found within the following variables: competitions per week (basketball: 0.92 ± 1.38, swimming 0.62 ±0.51, *p*0=0.03), lower extremity strength sessions per week (basketball: 0.62 ± 1.19, swimming 2.85 ± 0.38, *p*=0.002), plyometric sessions per week (p=.001), years spent participating in a court-based sport (basketball 7.69 ±2.02, swimming 2.54 ± 2.40, *p*=0.011), and VISA-P Score (basketball 90.69 ± 5.35, swimming 98.54 ± 2.99, *p*=0.001). Basketball presented with significantly greater competitions per week, a greater volume of plyometric activity per week, longer time spent participating in court-based sport, and a greater VISA-P score than the swim cohort. Swimmers experienced significantly greater lower extremity strength training sessions than the basketball cohort. This can be attributed to the structured nature of the Varsity Swim Team, with mandatory bi-weekly weight training sessions with a university hired strength coach.

Overall, our results did not identify with a majority of what's been represented in previous literature regarding movement quality and patellar tendon structure. Thre is evidence to support that frontal and transverse plane motion can influence tendon structure. Our results did support the finding that volume and quality of load can alter fibrillar alignment of the tendon. Despite the volume that aquatic athletes endure, their lack of plyometric activity may lead to an overall healthier tendon structure compared to court-based sports such as basketball. Our results demonstrate that training history and athlete type have a greater influence on tendon structure than movement biomechanics.

Discussion

The present study examined the effect that training load, sport, and lower extremity biomechanics on patellar tendon structure. Our findings indicate that basketball athletes have reduced PT quality compared to swimmers as evidenced by decreased Type 1 and AFS values and increased Type 3 and DFS values. There was a significant difference in tendon fibrillar structure between basketball athletes and swimmers, with basketball athletes presenting with approximately 2% greater percentage of Type III echo-type (degenerative), and approximately 9% lesser percentage of Type I echo-type (healthy and aligned) compared to swim athletes. Similarly, significance was found in percentage of aligned fibrillar structure (AFS) between groups and percentage of disorganized tissue structure (DIS) between groups, with basketball athletes presenting with significantly more DIS and less AFS compared to swim athletes. Our measure of tendon structural integrity is innovative, as the ultrasound tissue characterization device is a new and innovative technology with little previous research. We also noted that in addition to having reduced PT quality, basketball athletes also demonstrated reduced VISA P scores, thus indicating basketball players have more severe symptoms. To our knowledge, this study is the first to examine the difference of patellar tendon structure between a court-based sport and an aquatic sport.

We were interested in further investigating whether the observed differences of reduced PT quality and increased PT severity were influenced by lower extremity biomechanics and/or prior loading history. We believed this was important to investigate as it may provide insight into future intervention targets (improving biomechanics or load management) in those with symptomatic PT. To investigate the role of biomechanics and load history we compared key biomechanical and load history variables between the basketball athletes (poor PT quality and higher symptom severity) to swimmers (good PT quality and little to no symptom severity). Our findings demonstrated that basketball athletes did not demonstrate biomechanical differences believed to facilitate worse PT quality. Specifically, basketball athletes demonstrated reduced knee flexion motion and there were no differences in peak knee extension moment and vertical ground reaction force values. Thus, differences in biomechanics do not appear to influence differences in PT quality between basketball athletes and swimmers.

Kinetic and kinematic variables of interest had all been represented in previous research as potentially associated with the development of tendinopathy.[44,52,53] Knee flexion displacement, peak knee extension moment, and peak vertical ground reaction force have all been shown to associate with patellar tendon structure, such that greater values correlate to hyperechoic tendon regions.[13,35,44,48,68,69] Previous research has demonstrated the relationship between knee flexion displacement and landing stiffness.[35] We found no correlation between knee flexion displacement and peak vertical ground reaction force in our cohort. Swimmers demonstrated a significantly greater knee flexion displacement through jump landing than basketball athletes, utilizing what is may possibly be considered a more provocative landing strategy for patellar tendon pathology. Mann et al.[44] found landing with greater knee flexion increases compressive loading on the tendon. This is attributed to an increased demand on the extensor mechanism to control the internal knee extension moment via the quadriceps, with higher tensile and compressive loads through the patellar tendon tissue. Our results showed no correlation between knee flexion displacement and tendon structure. There was no significance found in peak knee extension moment between group, nor its effect on tendon structure. Given the extensor mechanism and the relationship between quadriceps contraction and shear force elicited at the patellar tendon, this finding may be due to our small sample size. It is possible a larger sample size or a more demanding single-limb task would have resulted in an association between lower extremity loading and patellar tendon structure. There is also possibility that because we only chose to analyze the biomechanics of one limb during a double-limb task that we didn't account for weight shifts or limb symmetry during the landing task. These movement qualities could be potential compensations to avoid loading a pathological tendon.

We did note large differences in the participation history of jumping and landing activities, which are known to facilitate high loads on the PT. Basketball athletes reported much greater amounts of landing and cutting activities compared to swimmers. We hypothesize that the loading history profile, not biomechanics profile, that has resulted in reduced PT quality and VISA P scores in basketball athletes.

Conventional ultrasound imaging techniques have been used to evaluate tendon structure through assessment of cross-sectional area and echo-geneity, both commonly found in pathological tendon status.[8,20] However, measuring tendon structure via a 2-D grey-scale US image does not allow quantification of the 3-D qualities of collagen arrangement. Therefore, advancements, such as UTC imaging, are valuable to continue to better understand the relationship between tendon structure and application of load.[15,20] The appropriate modification of load to monitor pathological tendon has yet to be identified. Our results compare two high volume sports which by default are influenced by the effect that gravity, ground reaction force, and plyometric load have on load-bearing soft tissues across the lower extremity. There were no significant differences between swim and basketball athletes regarding their landing biomechanics, volume of training sessions/competitions, or perceived exertion of their training, all of which are qualities that can be used to explain volume and intensity of sports training. However, a significant difference was found in the composition of training sessions, specifically lower extremity strength training, plyometric training, and time spent participating in a court-based sports. Basketball athletes presented with greater time spent under gravitational load, reflecting greater load on the extensor mechanism of the knee via the patellar tendon. There are limitations to our data, given its cross-sectional nature collected only at the pre-season baseline time-point. Therefore, we are unable to assume that the self-reported training at this time-point is reflective of the load endured across a competitive season. Future research should employ a longitudinal design with in-season benchmarks to potentially better understand if changes in training load characteristics influence patellar tendon structure.

The load reducing effect of water due to buoyancy is a main advantage of aquatic exercise compared to activity on land in terms of avoiding excessive stress placed on load-bearing soft tissue, such as the patellar tendon.[70] However swimmers do not go completely without loading their knee extensor mechanism. Starts off the block, flip turns, and drag force all act to load soft tissue structures through the knee joint, however the load experienced is not truly equitable to the vertical load of land-based athletes. When compared to the similar activities on land, knee joint forces were reduced by 36–55% in water with absolute reductions being greater than 100% body weight during weight-bearing and dynamic activities.[71] Ferretti et al.[47] found PTA to be more common in volleyball players who train more than 4 times per week. Similarly, Witvrouw et al.[26] found out of 189 court-based athletes, patellar tendon pathology affected 13.8% of the sample. There was significance found in the role that extrinsic parameters played in the development of the condition, specifically volume of load.[26] Most previous research compares court-based athletes to health controls, where one cohort endures moderate to severe load and the other activities of daily living. A unique and meaningful feature of the present study is that collegiate swimmers undergo equal, if not more, volume and training load than basketball athletes. Our results demonstrated no difference in the perceived training load between groups, but did demonstrate a difference in tendon structure between groups. We are able to hypothesize from these results that it is potentially the application and type of load that may influence tendon structure the most, rather than overall volume of training.

Limitations

Sample size for this project was dictated by the number of athletes available on the men's JV basketball team and men's Varsity swim team. Additionly, because this was a cross-sectional design, we were not able to standardize the time point at which each subject was collected. Because of this, there was some variability in self-reported measures of load and RPE. There was also variability in the time point at which both team's data was collected. Swim athletes were tested one week prior to the start of their competitive season, whereas due to time constraints basketball athletes were testing 2-3 days prior to the initiation of their season.

Conclusion

The continuum model of tendon pathology advocates for a multi-modal approach to diagnosis and management, including patient-reported outcome measures, clinical examination findings, and imaging technologies.[3] UTC provides an imaging option for measuring tendon structural integrity and detecting change over time, which can both be used to monitor asymptomatic athletes and appropriate tendon response to load. This can enhance a clinician's ability to modify tendon exposure and tendon load, using an evidence-based return to sport program with athletes.[1,22] Future research using UTC should utilize a longitudinal design. This would enable the tracking of changes in tendon structure over the course of a competitive season in a high vertically loaded compared to aquatically-based athletes. It would also create the ability to track biomechanical changes over the course of a season, factoring in compensations or changes due to seasonal fatigue into assessment of movement quality. Future research is warranted to further investigate the effects of loading history on PT quality and symptom severity.

Appendix 1: Training Load Questionnaire

Subject ID:____________

The following questionnaire will address the details of your sport, injury history, training history, and the impact of that pain and injury have on your sport participation and ability to train and perform. Please answer to the best of your ability. Responses should include any and all participation in sport from the past calendar year.

<u>DETAILS OF LEVEL OF COMPETITION/</u>

1. In what competition do you participate in sport?
☐ Collegiate Basketball ☐ Collegiate Swimming

2. If BASKETBALL, what is your primary position?
☐ Point Guard ☐ Shooting Guard ☐ Forward ☐ Center

3. A. If SWIMMING, what is your primary swim stroke?
☐ Breaststroke ☐ Freestlye ☐ Butterfly ☐Back Stroke

B. What is your primary training group?
☐ Sprint ☐ Mid-Distance ☐ Distance

<u>PAST MEDICAL HISTORY</u>

4. Do you have diabetes?
☐ No ☐ Yes – Type 1 diabetes ☐ Yes – Type 2 diabetes

5. Any Inflammatory conditions?
☐ None ☐ Rheumatoid arthritis ☐ Psoriatic arthritis
☐ Ankolysing spondylarthropathy ☐ Gout / pseudogout

6. Do you have high cholesterol?
☐ No ☐ Yes

7. What medications are you currently taking?

8. Have you used fluroquinolones or corticosteroids in the past 12 months?
☐ No ☐ Yes

<u>HISTORY OF INJURIES</u>

Please Circle (Yes or No) regarding your situation.

Yes	No	Have you had an injury to either leg (other than patellar tendinopathy) that has altered you function in the past 6 months?
Yes	No	Have you had a surgery to either leg (knee, ankle, hip) in the past 1 year?
Yes	No	Have you had an injection (corticosteroids, plasma-rich-protein, etc.) to the patellar tendon in the last 3 months?
Yes	No	Do you have any knee ligaments that have not been reconstructed?
Yes	No	Do you have any nerve injuries in your legs or lower back?
Yes	No	Do you have any known muscular abnormalities?
Yes	No	Do you have a heart condition that would stop you from exercising?
Yes	No	Have you ever been diagnosed with cancer over your knee or thigh?
Yes	No	Do you currently have an infection over your thigh or in your knee?
Yes	No	Do you know of a hypersensitivity to electrical stimulation?

9. **Have you ever sustained any major <u>lower limb injury(s)</u> (ankle, knee or hip) that required medical attention or disturbed your normal activities for more than one week?**
☐ Yes ☐ No (If yes, please specify what injuries in the table below)

Lower Limb Injury(s)	**In the previous 12 months**	
	Right	***Left***
Describe the injury		
Sport or Activity Occurred		
Time missed due to injuries		

10. **Have you ever sustained any major <u>upper limb injury(s)</u> (shoulder, elbow or wrist) that required medical attention or disturbed your normal activities for more than one week? (If yes, what injuries)**
☐ Yes ☐ No (If yes, please specify what injuries in the table below)

	In the previous 12 months

Upper Limb Injury(s)	***Right***	***Left***
Describe the injury		
Sport or Activity Occurred		
Time missed due to injuries		

11. Have you ever sustained any major back injury(s) that required medical attention or disturbed your normal activities for more than one week? (If yes, what injuries)

☐ Yes ☐ No (If yes, please specify what injuries in the table below)

Back Injury(s)	**In the previous 12 months**	
	Right	***Left***
Describe the injury		
Sport or Activity Occurred		
Time missed due to injuries		

<u>LOADING HISTORY</u>

Answer the following questions based your activity level for the past calender year (12 months). *Competitons* are considered formal and organized pariticipation in sport with game officials (games, scrimmage, meets). *Training sessions* are considered any sports specific activity organized by your sport (Practice, weights, dryland, conditioning)

12. **How many competitions do you participate in each week?**
☐ None ☐ 1 ☐ 2 ☐ 3 ☐ 4
☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10

13. **How many training sessions do you perform each week?**
☐ No sessions ☐ 1 session ☐ 2 sessions ☐ 3 sessions ☐ 4 sessions
☐ 5 sessions ☐ 6 sessions ☐ 7 sessions ☐ 8 sessions ☐ 9 sessions
☐ More than 10 sessions

14. **On average, how many minutes are you active during competition?**
☐ None ☐ 0-10 minutes ☐ 10-20 minutes ☐ 20-30 minutes
☐ 30-40 minutes ☐ 40-50 minutes ☐ More than 50 minutes

15. **On average, how long is each training session?**
☐ No training ☐ 0-30 minutes ☐ 30-60 minutes ☐ 60-90 minutes
☐ More than 90 minutes

1 - 10 Borg Rating of Perceived Exertion Scale	
0	Rest
1	Really Easy
2	Easy
3	Moderate
4	Sort of Hard
5	Hard
6	
7	Really Hard
8	
9	Really, Really, Hard
10	Maximal: Just like my hardest race

16. **Based on the scale above, how hard was this week of competition?**
Rest Maximal
☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10

1 - 10 Borg Rating of Perceived Exertion Scale	
0	Rest
1	Really Easy
2	Easy
3	Moderate
4	Sort of Hard
5	Hard
6	
7	Really Hard
8	
9	Really, Really, Hard
10	Maximal: Just like my hardest race

17. Based on the scale above, how hard was this week of training sessions?

Rest Maximal

☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10

18. Do you do any other type of training specifically for your sport other than your training (e.g. weights, plyometrics, running, dryland, etc)? Please describe below in detail. (duration, activity type)

☐ Yes ☐ No

19. Do you participate in any other sports?

☐ Yes ☐ No

If yes please specify: ____.

List any other physical activity(s) that you are currently involved in on a regular basis (more than once per week)?

OTHER INJURIES

20. Have you had ANY INJURIES in the past month that have limited your ability to participate in training or games?

☐ No *Skip to Question 19*
☐ Yes

21. How many days have you been unable to participate in all aspects of training in the past month?

____

22. To what extent have you reduced your training volume in the last month compared to a normal month? *

Mark only one option.

☐ No reduction
☐ To a minor extent
☐ To a moderate extent
☐ To a major extent
☐ Cannot participate at all

TENDINOPATHY OVERUSE QUESTIONNAIRE

23. Have you EVER had PATELLAR TENDON pain?

☐ No
☐ Yes – Right knee
☐ Yes – Left knee
☐ Yes – both knees

24. What AGGRAVATES your PATELLAR TENDON pain? Select 1 or more options that apply to you.

☐ Jumping / landing
☐ Changing direction
☐ Straight line running
☐ Stretching
☐ Riding a bicycle
☐ Walking
☐ I have no pain with any activities
☐ Other:______________________

25. Did you miss any competitions/ training sessions last season due to PATELLAR TENDON pain?

☐ No ☐ Yes

26. If yes, how many competitions/training sessions did you miss last season?

☐ Games:___________ ☐ Training sessions:___________

Appendix 2. Victorian Institute of Sport of Sport Assessment- Patellar Tendon (VISA-P)

V I C T O R I A N   I N S T I T U T E   O F   S P O R T

1. For how many minutes can you sit pain free?

0 mins ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 100 mins Points ☐

0 1 2 3 4 5 6 7 8 9 10

2. Do you have pain walking downstairs with a normal gait cycle?

strong severe pain ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

3. Do you have pain at the knee with full active non-weightbearing knee extension?

strong severe pain ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain when doing a full weight bearing lunge?

strong severe pain ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

5. Do you have problems squatting?

Unable ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ no problems Points ☐

0 1 2 3 4 5 6 7 8 9 10

6. Do you have pain during or immediately after doing 10 single leg hops?

strong severe pain/unable ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

7. Are you currently undertaking sport or other physical activity?

0 ❒ Not at all

4 ❒ Modified training ± modified competition

7 ❒ Full training ± competition but not at same level as when symptoms began

10 ❒ Competing at the same or higher level as when symptoms began

8. Please complete **EITHER A, B or C** in this question.

• If you have **no pain** while undertaking sport please complete **Q8a only**.

• If you have **pain while undertaking sport but it does not stop you** from completing the activity, please complete **Q8b only**.

• If you have **pain that stops you from completing sporting activities**, please complete **Q8c only**.

8a. If you have **no pain** while undertaking sport, for how long can you train/practise?

NIL	1-5 mins	6-10 mins	7-15 mins	>15 mins	
❐	❐	❐	❐	❐	Points ☐
0	7	14	21	30	

OR

8b. If you have some pain while undertaking sport, but it does not stop you from completing your training/practice for how long can you train/practise?

NIL	1-5 mins	6-10 mins	7-15 mins	>15 mins	
❐	❐	❐	❐	❐	
0	4	10	14	20	Points ☐

OR

8c. If you have **pain which stops you** from completing your training/practice for how long can you train/practise?

NIL	1-5 mins	6-10 mins	7-15 mins	>15 mins	
❐	❐	❐	❐	❐	
0	2	5	7	10	Points ☐

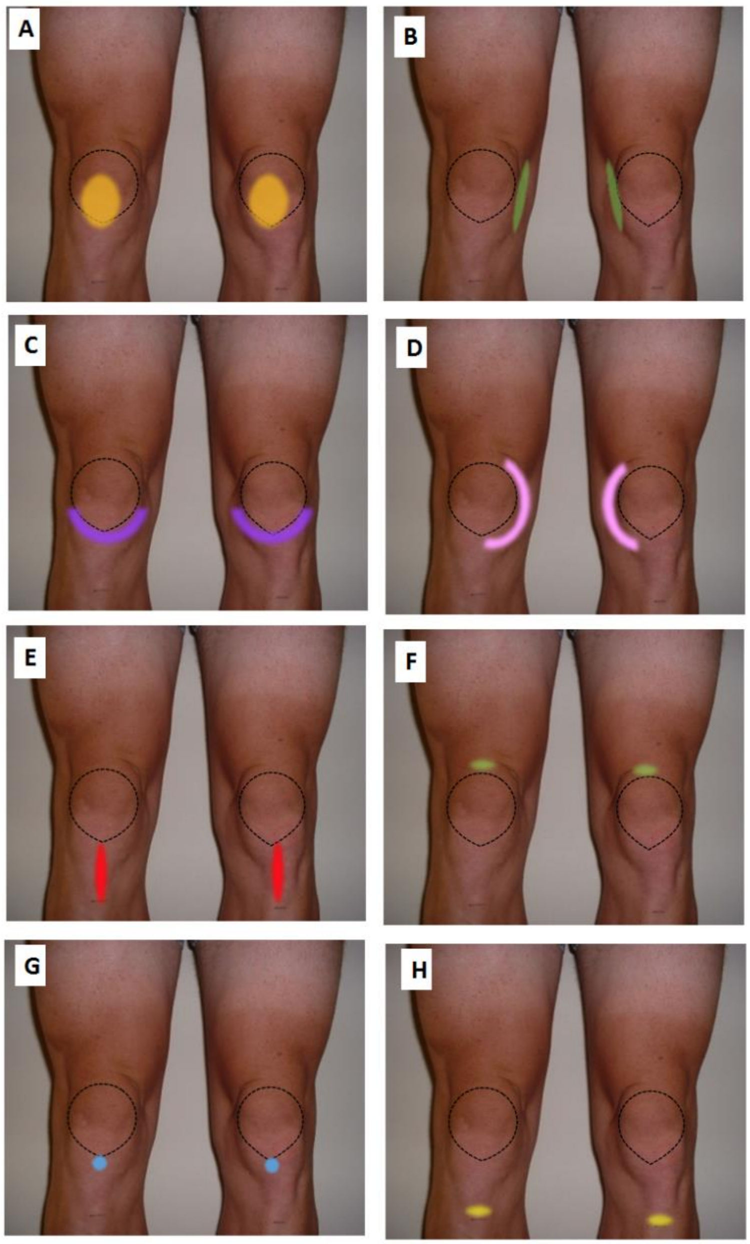
TOTAL VISA SCORE ❐

Appendix 3. Pain map for SLDS

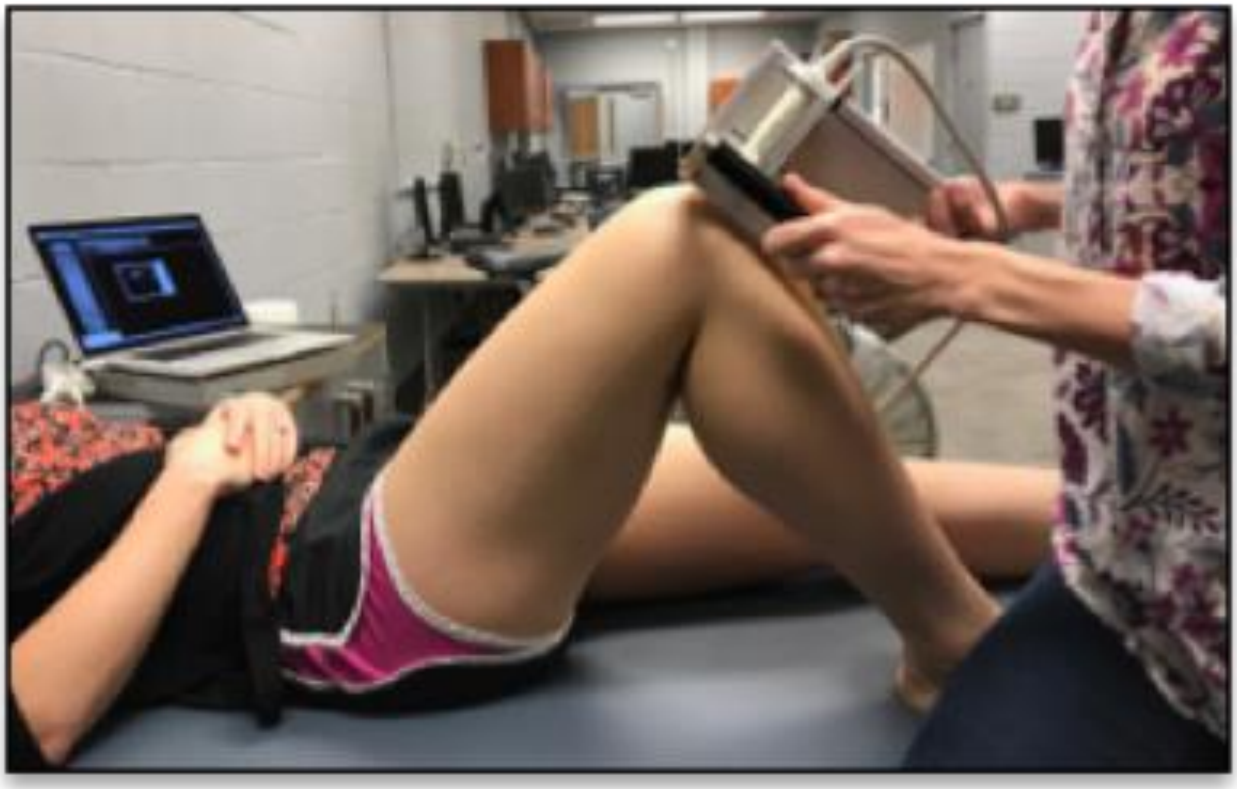
Use the below scale to rate you pain on a scale of 0-10 following completion of SLDS. If a response >0 is chosen, indicate on the pain map following your location of pain.

☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10

Please note the black line is an approximation of the kneecap.



Appendix 4. Subject set-up for UTC patellar tendon scan.



REFERENCES

1. Crossley KM, Thancanamootoo K, Metcalf BR, Cook JL, Purdam CR, Warden SJ. Clinical features of patellar tendinopathy and their implications for rehabilitation. *J Orthop Res*. 2007;25(9):1164-1175. doi:10.1002/jor.20415.

2. Mersmann F, Bohm S, Schroll A, Boeth H, Duda GN, Arampatzis A. Muscle and tendon adaptation in adolescent athletes: A longitudinal study. *Scand J Med Sci Sport*. 2015:1-8. doi:10.1111/sms.12631.

3. Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *Br J Sports Med*. 2009;43:409-416. doi:10.1136/bjsm.2008.051193.

4. Visnes H, Bahr R. Training volume and body composition as risk factors for developing jumper's knee among young elite volleyball players. *Scand J Med Sci Sports*. 2012;23(5):n/a-n/a. doi:10.1111/j.1600-0838.2011.01430.x.

5. Van Der H, Harjo De Poel W, Diercks RL, Van De Akker-Scheek I, Submitted JZ. Is the jumper's knee a lander's knee? A systematic review of the relation between take-off and landing biomechanics and patellar tendinopathy.

6. Mann KJ, Edwards S, Drinkwater EJ, Bird SP. A lower limb assessment tool for athletes at risk of developing patellar tendinopathy. *Med Sci Sports Exerc*. 2013;45(3):527-533. doi:10.1249/MSS.0b013e318275e0f2.

7. Schwartz A, Watson JN, Hutchinson MR. Patellar Tendinopathy. *Sports Health*. 2015;7(5):415-420. doi:10.1177/1941738114568775.

8. Cook JL, Kiss ZS, Khan KM, Purdam CR, Webster KE. Anthropometry, physical performance, and ultrasound patellar tendon abnormality in elite junior basketball players: a cross-sectional study. *Br J Sports Med*. 2004;38(2):206-209. doi:10.1136/BJSM.2003.004747.

9. Rudavsky A, Cook J. Physiotherapy management of patellar tendinopathy (jumper's knee). *J Physiother*. 2014;60(3):122-129. doi:10.1016/j.jphys.2014.06.022.

10. Visnes H, Aandahl HÅ, Bahr R. Jumper's knee paradox--jumping ability is a risk factor for developing jumper's knee: a 5-year prospective study. *Br J Sports Med*. 2013;47(8):503-507. doi:10.1136/bjsports-2012-091385.

11. de Vries AJ, van der Worp H, Diercks RL, van den Akker-Scheek I, Zwerver J. Risk factors for patellar tendinopathy in volleyball and basketball players: A survey-based prospective cohort study. *Scand J Med Sci Sports*. 2015;25(5):678-684. doi:10.1111/sms.12294.

12. van der Worp H, Zwerver J, Kuijer PPFM, Frings-Dresen MHW, van den Akker-Scheek I. The impact of physically demanding work of basketball and volleyball players on the risk for patellar tendinopathy and on work limitations. *J Back Musculoskelet Rehabil*. 2011;24(1):49-55. doi:10.3233/BMR-2011-0274.

13. van der Worp H, van Ark M, Roerink S, Pepping G-J, van den Akker-Scheek I, Zwerver J. Risk factors for patellar tendinopathy: a systematic review of the literature. *Br J Sports Med*. 2011;45(5):446-452. doi:10.1136/bjsm.2011.084079.

14. Khodaee M, Edelman GT, Spittler J, et al. Medical Care for Swimmers. *Sport Med - open*. 2015;2:27. doi:10.1186/s40798-016-0051-2.

15. Wezenbeek E, Mahieu N, Willems T, et al. What does normal tendon structure look like? New insights into tissue characterization in the Achilles tendon. *Scand J Med Sci Sports*. 2016:1-8. doi:10.1111/sms.12706.

16. Hoksrud A, Öhberg L, Alfredson H, Bahr R, Md L. Color Doppler Ultrasound Findings in Patellar Tendinopathy (Jumper's Knee). doi:10.1177/0363546508319897.

17. Wearing SC, Hooper SL, Smeathers JE, Pourcelot P, Crevier-Denoix N, Brauner T. Tendinopathy alters ultrasound transmission in the patellar tendon during squatting. *Scand J Med Sci Sport*. 2015:1-8. doi:10.1111/sms.12602.

18. Wezenbeek E, Mahieu N, Willems T, et al. What does normal tendon structure look like? New insights into tissue characterization in the Achilles tendon. *Scand J Med Sci Sports*. June 2016. doi:10.1111/sms.12706.

19. Rosengarten SD, Cook JL, Bryant AL, Cordy JT, Daffy J, Docking SI. Australian football players' Achilles tendons respond to game loads within 2 days: an ultrasound tissue characterisation (UTC) study. *Br J Sports Med*. 2014:1-6. doi:10.1136/bjsports-2013-092713.

20. Docking SI, Cook J. Pathological tendons maintain sufficient aligned fibrillar structure on ultrasound tissue characterization (UTC). *Scand J Med Sci Sports*. 2016;26(6):675-683. doi:10.1111/sms.12491.

21. Docking SI, Rosengarten SD, Cook J. Achilles tendon structure improves on UTC imaging over a 5-month pre-season in elite Australian football players. *Scand J Med Sci Sport*. 2016;26(5):557-563. doi:10.1111/sms.12469.

22. Stanley LE, Lucero A, Mauntel TC, et al. *Achilles Tendon Adaptation in Cross-Country Runners across a Competitive Season*.; 2017. doi:10.1111/sms.12903.

23. Rosen AB, Ko J, Simpson KJ, Kim S-H, Brown CN. Lower Extremity Kinematics During a Drop Jump in Individuals With Patellar Tendinopathy. doi:10.1177/2325967115576100.

24. Malliaras P, Cook J, Purdam C, Rio E. Patellar Tendinopathy: Clinical Diagnosis, Load Management, and Advice for Challenging Case Presentations. *J Orthop Sport Phys Ther*. 2015;45(11):887-898. doi:10.2519/jospt.2015.5987.

25. Mendonça LD, Verhagen E, Bittencourt NFN, Gonçalves GGP, Ocarino JM, Fonseca ST. Factors associated with the presence of patellar tendon abnormalities in male athletes. *J Sci Med Sport*. 2016;19(5):389-394. doi:10.1016/j.jsams.2015.05.011.

26. Witvrouw E, Bellemans J, Lysens R, Danneels L, Cambier D. Intrinsic Risk Factors for the Development of Patellar Tendinitis in an Athletic Population A Two-Year Prospective Study*.

27. Cook JL, Khan KM, Kiss ZS, Griffiths L. Patellar tendinopathy in junior basketball players: a controlled clinical and ultrasonographic study of 268 patellar tendons in players aged 14-18 years. *Scand J Med Sci Sports*. 2000;10(4):216-220. doi:10.1034/j.1600-0838.2000.010004216.x.

28. Haddas R, James CR, Hooper TL. Lower extremity fatigue, sex, and landing performance in a population with recurrent low back pain. *J Athl Train*. 2015;50(4):378-384. doi:10.4085/1062-6050-49.3.61.

29. Mersmann F, Bohm S, Schroll A, Marzilger R. Athletic Training Affects the Uniformity of Muscle and Tendon Adaptation during Adolescence. *J Appl Physiol*. 2016. doi:10.220.32.247.

30. Burgess KE, Connick MJ, Graham-Smith P, Pearson SJ. Plyometric vs. Isometric Training Influences on Tendon Properties and Muscle Output. *J Strength Cond Res*. 2007;21(3):986. doi:10.1519/R-20235.1.

31. van Ark M, Docking SI, van den Akker-Scheek I, et al. Does the adolescent patellar tendon respond to 5 days of cumulative load during a volleyball tournament? *Scand J Med Sci Sports*. 2016;26(2):189-196. doi:10.1111/sms.12426.

32. Cook JL, Purdam CR. The challenge of managing tendinopathy in competing athletes. *Br J Sports Med*. 2014;48(7):506-509. doi:10.1136/bjsports-2012-092078.

33. Crossley KM, Thancanamootoo K, Metcalf BR, Cook JL, Purdam CR, Warden SJ. Clinical features of patellar tendinopathy and their implications for rehabilitation. *J Orthop Res*. 2007;25(9):1164-1175. doi:10.1002/jor.20415.

34. Malliaras P, Cook JL, Kent P. Reduced ankle dorsiflexion range may increase the risk of patellar tendon injury among volleyball players. *J Sci Med Sport*. 2006;9(4):304-309. doi:10.1016/j.jsams.2006.03.015.

35. Bisseling RW, Hof AL, Bredeweg SW, Zwerver J, Mulder T. Relationship between landing strategy and patellar tendinopathy in volleyball. *Br J Sports Med*. 2007;41(7):e8. doi:10.1136/bjsm.2006.032565.

36. Watanabe Y, Wakayoshi K, Nomura T. New evaluation index for the retainability of a swimmer's horizontal posture. *PLoS One*. 2017;12(5):e0177368. doi:10.1371/journal.pone.0177368.

37. Kjaer M, Langberg H, Miller BF, et al. Metabolic activity and collagen turnover in human tendon in response to physical activity. *J Musculoskelet Neuronal Interact*. 2005;5(1):41-52.

38. Cormie P, McGuigan MR, Newton RU. Developing Maximal Neuromuscular Power. *Sport Med*. 2011;41(1):17-38. doi:10.2165/11537690-000000000-00000.

39. J.L Cook. Asymptomatic hypoechoic regions on patellar tendon ultrasound: A 4-year cli...: EBSCOhost. http://web.b.ebscohost.com/ehost/pdfviewer/pdfviewer?sid=36280c82-6137-4962-a575-b531e75c8555%40sessionmgr105&vid=7&hid=123.

40. Jonsson P, Alfredson H. Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomised study. *Br J Sports Med*. 2005;39(11):847. doi:10.1136/bjsm.2005.018630.

41. Ooi CC, Richards PJ, Maffulli N, et al. A soft patellar tendon on ultrasound elastography is associated with pain and functional deficit in volleyball players. *J Sci Med Sport*. 2016;19:373-378. doi:10.1016/j.jsams.2015.06.003.

42. Mendonça L de M, Ocarino JM, Bittencourt NFN, Fernandes LMO, Verhagen E, Fonseca ST. The Accuracy of the VISA-P Questionnaire, Single-Leg Decline Squat, and Tendon Pain History to Identify Patellar Tendon Abnormalities in Adult Athletes. *J Orthop Sport Phys Ther*. 2016;46(8):673-680. doi:10.2519/jospt.2016.6192.

43. Hernandez-Sanchez S, Abat F, Hidalgo MD, et al. Confirmatory factor analysis of VISA-P scale and measurement invariance across sexes in athletes with patellar tendinopathy. *J Sport Heal Sci*. 2016. doi:10.1016/j.jshs.2016.01.020.

44. MANN KJ, EDWARDS S, DRINKWATER EJ, BIRD SP. A Lower Limb Assessment Tool for Athletes at Risk of Developing Patellar Tendinopathy. *Med Sci Sport Exerc*. 2013;45(3):527-533. doi:10.1249/MSS.0b013e318275e0f2.

45. Gisslén K, Alfredson H. Neovascularisation and pain in jumper's knee: a prospective clinical and sonographic study in elite junior volleyball players. *Br J Sports Med*. 2005;39(7):423-8-8. doi:10.1136/bjsm.2004.013342.

46. Cook JL, Rio E, Purdam CR, Docking SI. Revisiting the continuum model of tendon pathology: what is its merit in clinical practice and research? *Br J Sports Med*. 2016;50(19):1187-1191. doi:10.1136/bjsports-2015-095422.

47. Scattone Silva R, Nakagawa TH, Ferreira ALG, Garcia LC, Santos JEM, Serrão F V. Lower limb strength and flexibility in athletes with and without patellar tendinopathy. *Phys Ther Sport*. 2016;20:19-25. doi:10.1016/j.ptsp.2015.12.001.

48. van Ark M, Cook JL, Docking SI, et al. Do isometric and isotonic exercise programs reduce pain in athletes with patellar tendinopathy in-season? A randomised clinical trial. *J Sci Med Sport*. 2016;19(9):702-706. doi:10.1016/j.jsams.2015.11.006.

49. Richards DP, Ajemian S V., Wiley JP, Zernicke RF. Knee Joint Dynamics Predict Patellar Tendinitis in Elite Volleyball Players. *Am J Sports Med*. 1996;24(5):676-683. doi:10.1177/036354659602400520.

50. Bohm S, Mersmann F, Arampatzis A. Human tendon adaptation in response to mechanical loading : a systematic review and meta-analysis of exercise intervention studies on healthy adults. 2015. doi:10.1186/s40798-015-0009-9.

51. Milz S, Ockert B, Putz R. [Tenocytes and the extracellular matrix : a reciprocal relationship]. *Orthopade*. 2009;38(11):1071-1079. doi:10.1007/s00132-009-1490-y.

52. Padua DA, DiStefano LJ, Marshall SW, Beutler AI, de la Motte SJ, DiStefano MJ. Retention of movement pattern changes after a lower extremity injury prevention program is affected by program duration. *Am J Sports Med*. 2012;40(2):300-306. doi:10.1177/0363546511425474.

53. Edwards S, Steele JR, McGhee DE. Does a drop landing represent a whole skill landing and is this moderated by fatigue? *Scand J Med Sci Sports*. 2009;20(3):516-523. doi:10.1111/j.1600-0838.2009.00964.x.

54. Theiss JL, Gerber JP, Cameron KL, et al. Jump-Landing Differences Between Varsity, Club, and Intramural Athletes. *J Strength Cond Res*. 2014;28(4):1164-1171. doi:10.1519/JSC.0b013e3182a1fdcd.

55. Stickley C, Ronald Hetzler Kaori Tamura Nathan Murata Scott Lozanoff C. BIOMECHANICAL RISK FACTORS OF LOWER EXTREMITY OVERUSE INJURY RELATED TO FATIGUE.

56. Stobierski LM, Fayson SD, Minthorn LM, Valovich McLeod TC, Welch CE. Reliability of clinician scoring of the functional movement screen to assess movement patterns. *J Sport Rehabil*. 2015;24(2):219-222. doi:10.1123/jsr.2013-0139.

57. Padua DA, DiStefano LJ, Beutler AI, de la Motte SJ, DiStefano MJ, Marshall SW. The Landing Error Scoring System as a Screening Tool for an Anterior Cruciate Ligament Injury–Prevention Program in Elite-Youth Soccer Athletes. *J Athl Train*. 2015;50(6):589-595. doi:10.4085/1062-6050-50.1.10.

58. Padua D a, Boling MC, Distefano LJ, Onate J a, Beutler AI, Marshall SW. Reliability of the landing error scoring system-real time, a clinical assessment tool of jump-landing biomechanics. *J Sport Rehabil*. 2011;20(2):145-156.

59. DiStefano LJ, Padua DA, DiStefano MJ, Marshall SW. Influence of age, sex, technique, and exercise program on movement patterns after an anterior cruciate ligament injury prevention program in youth soccer players. *Am J Sports Med*. 2009;37(3):495-505. doi:10.1177/0363546508327542.

60. Padua DA, DiStefano LJ, Beutler AI, de la Motte SJ, DiStefano MJ, Marshall SW. The Landing Error Scoring System as a Screening Tool for an Anterior Cruciate Ligament Injury-Prevention Program in Elite-Youth Soccer Athletes. *J Athl Train*. 2015;50(6):589-595. doi:10.4085/1062-6050-50.1.10.

61. DiStefano LJ, Marshall SW, Padua DA, et al. The Effects of an Injury Prevention Program on Landing Biomechanics Over Time. *Am J Sports Med*. 2016;44(3):767-776. doi:10.1177/0363546515621270.

62. Bisseling RW, Hof AL, Bredeweg SW, Zwerver J, Mulder T. Are the take-off and landing phase dynamics of the volleyball spike jump related to patellar tendinopathy? *Br J Sports Med*. 2008;42(6):483-489. doi:10.1136/bjsm.2007.044057.

63. Clarsen B, Myklebust G, Bahr R. Development and validation of a new method for the registration of overuse injuries in sports injury epidemiology: the Oslo Sports Trauma Research Centre (OSTRC) Overuse Injury Questionnaire. *Br J Sport Med*. 2013;47:495-502. http://dx.doi.org/. Accessed July 19, 2017.

64. Frank BS, Gilsdorf CM, Goerger BM, Prentice WE, Padua DA. Neuromuscular fatigue alters postural control and sagittal plane hip biomechanics in active females with anterior cruciate ligament reconstruction. *Sports Health*. 2014;6(4):301-308. doi:10.1177/1941738114530950.

65. Goerger BM, Marshall SW, Beutler AI, Blackburn JT, Wilckens JH, Padua DA. Anterior cruciate ligament injury alters preinjury lower extremity biomechanics in the injured and uninjured leg: the JUMP-ACL study. *Br J Sports Med*. 2015;49(3):188-195. doi:10.1136/bjsports-2013-092982.

66. van Schie 2001_horse tendon UTC validity.pdf.

67. Lian O, Engebretsen L, Ovrebø R V, Bahr R. Characteristics of the leg extensors in male volleyball players with jumper's knee. *Am J Sports Med*. 24(3):380-385. http://www.ncbi.nlm.nih.gov/pubmed/8734892. Accessed November 8, 2016.

68. Edwards S, Steele JR, McGhee DE, Beattie S, Purdam C, Cook JL. Landing strategies of athletes with an asymptomatic patellar tendon abnormality. *Med Sci Sports Exerc*. 2010;42(11):2072-2080. doi:10.1249/MSS.0b013e3181e0550b.

69. Mendonça LD, Verhagen E, Bittencourt NF, Gonçalves GGP, Ocarino JM, Fonseca ST. Factors associated with the presence of patellar tendon abnormalities in male athletes. *J Sci Med Sport*. 2015;19(5):1-6. doi:10.1016/j.jsams.2015.05.011.

70. Alberton CL, Santana Pinto S, Amélia N, et al. Kinesiological Analysis of Stationary Running Performed in Aquatic and Dry Land Environments. *J Hum Kinet*. 2015;49(49):5-14. doi:10.1515/hukin-2015-0103.

71. Kutzner I, Richter A, Gordt K, et al. Does aquatic exercise reduce hip and knee joint loading? In vivo load measurements with instrumented implants. *PLoS One*. 2017;12(3):e0171972. doi:10.1371/journal.pone.0171972.