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Use of Ultrasound for Identification of Musculoskeletal Injury in Active Populations

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USE OF ULTRASOUND FOR IDENTIFICATION OF MUSCULOSKELETAL INJURY IN ACTIVE POPULATIONS

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ABSTRACT

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Lower extremity tendinopathies and fasciopathy conditions are prevalent amongst sedentary individuals,[1,2] athletes,[3,4] elite runners,[5,6]and military personnel[7] and can lead to chronic disability and reduction in quality of life. [8,9] Both tendinopathy and fasciopathy are thought to be mechanical in nature and caused by acute or chronic overloading leading to a disruption in the uniformity of the soft tissue structure. Ultrasound imaging has gained popularity in the last decade among many sports medicine clinicians to gain information regarding tissue structure for musculoskeletal injury (MKSI) management. Ultrasound imaging allows for the evaluation of in-vivo structures and at a relatively low cost with minimal assessment times.[10–12] However, the relationship between tendon structure and other signs and symptoms of overuse injuries such as these are not fully understood.

The overarching purpose of this dissertation was to investigate the role of musculoskeletal ultrasound imaging in identifying abnormal soft tissue changes and the relationship between abnormal structure and function. The first study evaluated whether those with plantar heel pain had significant differences in tendon structure than those without plantar heel pain. Results identified that those with plantar heel pain did have significant thickening of the patellar tendon via ultrasound evaluation; thus, providing evidence that ultrasound assessments may be useful in identifying structural changes in overuse pathologies.

The second study sought to describe the relationship between patellar tendon morphology and lower extremity function. The systematic review revealed a high amount of variability in methodological approaches within included studies; thus, identifying a need in future research to identify a uniform approach to evaluating patellar tendon structural changes of interest and outcomes of lower extremity function. Furthermore, it did reveal that there is a weak relationship between self-reported function and focal thickening or echogenicity changes. Additionally, there is low level evidence to support differences in landing patterns in those with patellar tendon abnormality (PTA) compared to controls; however, it may be task dependent.

Lastly, the third study primarily explored whether PTA was predictive of future patellar tendinopathy and lower extremity MSKI in military trainees. Those with PTA were found to not have an increased risk of lower extremity MSKI and the risk of patellar tendinopathy was unclear due to a lack of patellar tendinopathy cases. Secondarily, the study investigated the relationship between PTA and lower extremity function. In congruence with previous findings from the systematic review, there were no significant associations between self-reported or objective measures of function. Further research is still needed to assess the relationship between structure and function and the importance of abnormal structure to long term musculoskeletal health in broader populations and larger cohorts. Ultrasound evaluation may still provide useful information in the evaluation of MSKI but should be used in conjunction with other clinical tests and self-reported symptoms to guide diagnosis and management.

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NOMENCLATURE

BMI	Body Mass Index
CMJ	Counter Movement Jump
CSA	Cross-Sectional Area
DoD	Department of Defense
FAAM	Foot and Ankle Ability Measure
FPI	Foot posture Index
HPSG	Human Performance Support Group
IFMT	Intrinsic Foot Muscle Test
IFT	Initial Fitness Test
MDR	Military Health System Data Repository
MSKI	Musculoskeletal Injury
MVIC	Maximum Voluntary Contraction
OSTRC-OIQ	Oslo Sports Trauma Research Centre Overuse Injury Questionnaire
PHP	Plantar Heel Pain
PRO	Patient Reported Outcome
PTA	Patellar Tendon Abnormality
SWCC	Special Warfare Candidate Course
SWE	Shear Wave Elastography

SWTW	Special Warfare Training Wing
USAF	United States Air Force
UTC	Ultrasound Tissue Characterization
VAS	Visual Analog Scale
VISA-P	Victorian Institute of Sports Assessment-Patella

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CHAPTER 1

INTRODUCTION

1.1 Background

Ultrasound imaging has gained popularity in the last decade among many sports medicine clinicians. Ultrasound based assessments are of interest due to its broad availability, relatively low cost, and short assessment times.[10–12] Ultrasound assessment techniques allow for the evaluation of in-vivo structures in a non-invasive manner with minimal risk. Within sports medicine, ultrasound imaging has become an evaluation tool for guiding the management of musculoskeletal injury (MSKI).[13] Conventional ultrasound techniques such as b-mode and doppler ultrasonography can provide morphologic and structural information regarding musculoskeletal structures. Ultrasound assessments can provide quantitative and qualitative real-time assessment of tissue morphology with decreased risk and contraindications compared to traditional MRI imaging.

The first reported application of b-mode ultrasound imaging for MSKI was published in the 1970s.[14] Since that time, there have been thousands of publications assessing the use of ultrasound for diagnosis and managing MSKI. Additionally, it is now common for sports medicine clinicians to use ultrasound imaging techniques in various settings.

Ultrasound imaging for MSKI has gained traction due to its comparatively low cost and most importantly ease of accessibility that allows for real-time dynamic examination in the clinic or on the sports sideline. Advancement in ultrasound technology over the past five years has resulted in increased portability, increased clarity, and even allows for better quantification of tissue properties. Currently, portable machines with USB connected transducers that can be

linked to a tablet or phone are common in the market. Subscriptions associated with ultrasound machines also offer improved measuring and quantification capabilities making it easier for the clinician to analyze images.

Typically, clinicians rely on signs and symptoms to guide management and provide information about the extent of tissue damage. Patients that present with moderate tendinopathy, sprains, and strains are not typically prescribed additional imaging to confirm diagnosis. However, the tissue structure is important to managing these types of conditions and tissue remodeling is the basis for many rehabilitative strategies.[15] Ultrasound can be used as an extension of the physical examination to gain real time images of soft tissue to guide treatment plans or rule out differential diagnoses.[11] Information gained from ultrasound assessments may allow the clinicians to assess tendon structure during evaluation which otherwise would not have been available.

A majority of the recent published evidence for ultrasound imaging in the management of MSKI has focused on tendon and fascial pathologies.[13,16] Tendon and fascial pathologies, such as plantar fasciopathy and patellar tendinopathy, tend to be an ideal focus area for ultrasound imaging due to their high prevalence in active populations, relatively superficial location, and the need for additional information to guide the complex management of these pathologies. The assessment of tendon and fascial pathologies forms a major part of sports medicine. Ultrasound imaging may provide the most feasible method for providing specific information regarding tissue health when evaluating tendons and fascia.

Identifying markers of pathological change through ultrasound could improve clinical management through early diagnosis and correct staging of the pathology. Early diagnosis and intervention is an important factor in positive patient outcomes in degenerative pathologies such

as plantar fasciopathy and patellar tendinopathy which commonly lead to chronic pain.[15,17] Furthermore, having information that can identify and stage the severity of tendon degeneration can allow the clinician to provide an appropriate rehabilitation plan. Ultrasound imaging could be a useful tool to provide that otherwise missing pathophysiologic information that is needed to understand and improve management of these type of pathologies.

1.2 Statement of the Problem

Overuse injuries make up a majority of the MKSI injuries reported amongst physically active adults.[5,6,18] Overuse injuries such as tendinopathy and fasciopathy often lead to decreases in physical activity and quality of life that are recurrent throughout one's lifetime.[8,9] Information about the structural integrity of soft tissue changes may be able to guide rehabilitation or preventive strategies to prevent further symptoms. Further evidence is needed to evaluate the ability of ultrasound to identify abnormalities amongst symptomatic individuals and to identify possible subclinical indicators in diverse populations.

1.3 Purpose

Multiple purposes are contained in this dissertation to investigate the role of musculoskeletal ultrasound imaging in identifying abnormal soft tissue changes and the relationship between abnormal structure and function. The first purpose was to assess whether plantar fascia tissue changes can be identified through ultrasound assessment in those with reported heel pain. The second purpose was to systematically review the literature instigating the relationship between abnormal soft tissue changes in the patellar tendon and lower extremity function. The last purpose was to determine whether abnormal tissue changes in the patellar tendon place individuals at a high risk of patellar tendinopathy. There were multiple purposes

within this dissertation to thoroughly investigate the utilization of musculoskeletal ultrasound. Therefore, based on this premise, three projects with the following three aims were conducted.

1.4 Aims and Hypotheses:

Aim 1.1: To assess whether soft tissue changes are present in those with plantar heel pain compared to match healthy controls.

Hypotheses 1.1: Individuals with plantar heel pain will display greater anterior posterior thickness of the plantar fascia size of the decrease intrinsic foot muscle size via ultrasound assessment in comparison to healthy controls.

Aim 1.2: Investigate the aspects of the active and neural subsystems of the foot in those with and without plantar heel pain.

Hypothesis 1.2: Those with plantar heel pain will have decreases functional control and tactile sensation in comparison to healthy controls.

Aim 2: Synthesize existing evidence on the relationship between patellar tendon changes assessed by ultrasound imaging and subjective and objective function.

Hypothesis 2: Soft tissue abnormalities via ultrasound will be related to objective lower extremity function but will show only small to moderate association between self-reported measures based on the current symptoms of the population of interest.

Aim 3.1: Identify whether abnormal ultrasound imaging results place trainees at a higher risk of sustaining patellar tendinopathy or other lower extremity MSKI during a Special Warfare Training Wing (SWTW) candidate course.

Hypothesis 3.1: Those with abnormal tendon properties prior to a 6-week training course will be at a higher risk of sustaining patellar tendinopathy and other MSKI during course.

Aim 3.2: Assess the relationship between patellar tendon abnormality and lower extremity function.

Hypothesis 3.2: Patellar tendon abnormalities will be associated with measures of lower extremity function.

1.5 Operational Definitions

1. **Asymptomatic:** Having no signs or symptoms of condition of interest
2. **Symptomatic:** Having signs or symptoms of condition of interest

1.6 Assumptions

Chapter 3:

1. Participants were truthful and accurately disclosed their self-reported function.
2. Participants understood the directions provided during functional evaluations and thus their function was accurately assessed.

Chapter 4:

1. Extracted information in the included articles were accurate.
2. All studies that would meet the inclusion and exclusion criteria were identified.
3. VISA-P scores were calculated in a uniform manner and provided in a standardized manner.
4. Studies that identified those as showing no signs of patellar tendinopathy were evaluated in a standardized manner and were free of symptoms.

Chapter 5:

3. Participants were truthful and accurately disclosed their self-reported function.
4. All data extracted from the medical repository included all medical assessments and were free from error.

1.7 Limitations

Chapter 3:

1. Investigators performing the assessments were not blinded to group affiliation.

Chapter 4:

1. Our review was limited to available peer-reviewed literature.
2. Procedures for reporting the screening process for current signs and symptom were not well reported in some studies.

Chapter 5:

1. Lack of prevalence of patellar tendinopathy restricted ability to perform statistical analyses to address all aspects of primary aim.

CHAPTER 2

REVIEW OF THE LITERATURE

2.1 Epidemiology

Lower extremity tendinopathies and fasciopathy condition are prevalent amongst a diverse range of patient population including sedentary individuals,[1,2] athletes,[3,4] elite runners,[5,6]and military personnel.[7] It is typical for tendinopathy to be diagnosed in a wide age range from adolescent to geriatric populations and is known to be recurrent throughout ones lifespan.[19–21] Amongst a sample of the general population reporting to a general practitioners clinic over one year, plantar fasciitis had an incidence rate of 2.34 while lower extremity tendinopathy represented 3% of all musculoskeletal related symptoms within an incidence a rate of 10.2 per 1000 years.[18] Active populations, specifically those in high load environments with repetitive movements, are at an even higher risk of overuse conditions.

Tendinopathies and fasciopathy conditions make up a majority of overuse injuries reported in highly active populations.[6] Patellar tendinopathy for example, is most common in jumping athletes who continually place load on the knee extensor mechanism.[22] Patellar tendinopathy injury rates among NCAA athletes in jumping related sports, such as volleyball and basketball, have been reported between 19.16 to 49.41 compared to 16.10 per 1000 athlete exposures athletes in other sports.[22] Elite basketball and volleyball athletes seem to be at an even higher risk with reported prevalence rates as high as 50%.[23] Plantar fasciopathy, on the other hand, is most commonly identified in long distance runners and accounts for 11% of injuries amongst ultramarathon runners.[5,6]

Although, patellar tendinopathy and plantar fasciopathy seem to affect varying populations at different rates, one population that seems to have high rates of both are active duty

military members and military trainees.[7,24,25] Plantar fasciitis and patellar tendinopathy are common causes for medical visits amongst military members.[7,24,26] Prolonged as well sudden increases in load bearing intensity and duration are well-documented risk factors that may place active duty military members at an increased risk for overuse injury.[2,27]

2.2 Anatomy

Although tendinopathy and fasciopathy are considered overuse injuries their function with the musculoskeletal system and their structural components greatly differ. The primary function of the plantar fascia is to support the medial longitudinal arch and aid in propulsion during gait. The plantar fascia originates at the medial tubercle of the calcaneous and extends distally to the phalanges along the plantar aspects of the foot. This band of tissue in relation to the calcaneous, mid-tarsal joint, and metatarsals creates a truss that acts to support the foot. This mechanical model is called the windlass mechanism and was originally described by Hicks et al.[28] This truss-like mechanism, acts a tie rod that aids in dissipating forces along the medial longitudinal arch during mid-stance as well as shortens the foot during toe off. During, mid-stance the plantar fascia acts to control lengthening of the arch caused by ground reaction forces acting upon the forefoot and calcaneous. As the body transitions into the late stage of mid-stance the metatarsal phalangeal joints, primarily the first metatarsal phalangeal joint, starts to dorsiflex causing tension in the plantar fascia. This increase in tension leads to shortening of the distance between the metatarsal head and calcaneous creating an increase in arch height. This windlass mechanism is important to ensure proper locomotion. The controlled collapse of the medial longitudinal arch during mid-stance along with controlled pronation creates a lever that can be used to transition into supination of the foot to enact propulsion during toe off. Without the tension sustained by the plantar fascia, propulsion would be diminished and lead to abnormal

gait patterns. Intrinsic foot muscles have been shown to play a critical role in stabilizing the medial longitudinal arch. Induced fatigue of the intrinsic foot muscles, such as the abductor hallicus leads to increases in navicular drop.[29] Additionally, these muscles also increase in activity as postural demand increases indicating they may also be important during higher impact activity.[30] Thus intrinsic foot musculature may be able to modulate the strain placed on the plantar facia but further research is needed to investigate the relationship between patellar tendon strain and foot muscle activation.

Although the biomechanical function of the plantar fascia has been widely investigated there is little evidence on the structural properties of the plantar facia. The plantar fascia is made up on an inner core of dense and thick well-organized collagen fibers surrounded by a sheath comprised of a loose network of unorganized thin collagen fibers. The core of is primarily comprised of elongated fibrocytes within a uniform extracellular matrix of type I collagen fibers producing a network capable of sensing and adapting to changes in loading. Additionally, the medial, lateral, distal portions of the plantar fascia contain nerve endings as well as Pacinian and Ruffini corpuscles.[31,32] Ruffini endings detect stretch and deformation whereas Pacinian corpuscles detection transient pressure and high frequency vibration.[33] The presence of these suggest that the plantar fascia plays a role in delivering afferent mechanical information to elicit coordinated motor responses. There is little evidence on the cellular response to plantar fascia loading. One study found that the plantar fascia had metabolic reactions to mechanical load that differed based on the loading placed on the tissue.[34] Moderate loading induced gene expression that would induce type 1 collagen synthesis while strenuous increase cellular inflammatory responses.[34] Thus, loading may be key for turnover of collagen within the plantar fascia and

overloading may lead to changes within the fascia that lead to plantar fasciopathy but the etiology is still largely unknown.

Compared to fascia, tendinous structures connect contractile tissues to bone and play a crucial role in the production of movement by transferring contractile forces to the skeletal system. The patellar tendon, along with the proximal quadriceps tendon, aids in producing knee extension by transferring forces produced by the quadriceps to the anterior aspect of the tibia resulting in knee extension. Together the quadriceps, proximal quadriceps tendon, patellar and patellar tendon are known as the knee extensor mechanism. The patellar tendon is the distal portion of the quadriceps femoris tendon originating at the apex of the patella to the tibial tuberosity. It is sometimes referred to as the patellar ligament due to its connection between bony structures, however because of its functional purpose of movement protection it is considered tendinous. The attachment sites of the patellar tendon do influence its overall structure and the patellar tendon is stiffer than the proximal quadriceps tendon.[35] The quadriceps tendon is a more extensile and softer tissue to allow it to adapt and attenuate forces from pliable contractile forces proximally to stiff bone.[35] These structural differences may play a role in the higher prevalence of tendinopathy within the patellar tendon compared to the quadriceps tendon, which prevalence is estimated from 0.2% to 2% in athletes.[23,36]

The patellar tendon is characterized by hierarchal structure that is comprised mainly of type 1 collagen molecules. The collagen molecules are arranged in a uniform pattern making up a fibril. Groups of parallelly aligned collagen fibrils, fibroblasts and the extracellular matrix consisting of proteoglycans, glycoproteins, and glycosaminoglycans then form fascicle bundle. These fascicle bundles along with vascular and neural innervations comprise the tendon. Proteoglycans, glycoproteins, and glycosaminoglycans play a key role in the organization and

growth of the tendon. Additionally, fibroblasts cells are mainly responsible for production of collagen and are thought to stimulate protein synthesis as a result of mechanical loading.

In healthy tendons, mechanical stimuli induce tendon cell responses which trigger a cascade of signals to stimulate collagen expression and synthesis.[37] Increases in collagen expression is thought to be modulated by the strain placed on the fibroblast during deformation. Studies have shown that exercise leads to 2 to 3 fold increase in collagen production which is highest one day post exercise and stays elevated for up to 3 days.[38,39] Furthermore, consistent exposure to moderate loading through exercise training has been linked to tendon hypertrophy.[40] However, the ideal dosage for inducing tendon hypertrophy is unknown and need further investigation. Simultaneously, collagen within the tendon also goes through breakdown as a result of exercise and has been shown to be a negative gain of new collagen formation up 3 days following exercises.[41] Therefore, proper rest and recovery may be a key factor in maintaining homeostasis within the tendon.

2.3 Etiology and Histological Factors

Overuse injuries, such as tendinopathy and fasciopathy are characterized by insidious onset of self-limiting pain during loading and loss in function.[42] Not only are these pathologies detrimental to athletic performance, many patients report negative disruption to social activity, self-efficacy, overall-quality of life and general physical activity.[8,9] Both tendinopathy and fasciopathy are thought to be mechanical in nature and caused by acute or chronic overloading leading to a disruption in the uniformity of the soft tissue structure. However, there is a lack of consensus of relevant risk factors as well as what factors place an individual at risk of tendon overloading or the severity of histological changes.

The etiology of plantar fasciitis although postulated to be linked to mechanisms of overloading are not well defined.[42–44] Fasciopathy is postulated to be caused by overloading of the fascia resulting in microtrauma through high loads or moderate repetitive loads placed on the medial longitudinal arch which exceeds the capability of normal repair process either due to magnitude of strain or continued repetitive strain of the plantar fascia.[43] Although normative repair processes stimulates the inflammatory repair process, this process may be stalled by continued repetitive trauma leading to a halt in the inflammation process and chronic degeneration. Inflammation markers within the plantar fascia seems to be absent in those with chronic tendinopathy.[45] Those with prolonged symptoms from plantar fasciitis show signs of degenerative changes within the plantar fascia.[31,46] Biopsy from the plantar fascia in those with chronic plantar heel pain confirms the presence of collagen breakdown, matrix degradation, and vascular changes.[31] These histological changes are evident through uniformity changes within the fascial tissue as well as focal thickening which could be viewed in a clinical setting through ultrasound imaging. Due to a lack of prospective studies from early onset of fascial microtrauma to chronic fasciopathy little is known about the metabolic processes that lead to degenerative fasciopathy. It does appear that those with plantar fasciopathy may have similar structural changes to tendons with chronic tendinopathy.

Factors related to plantar fasciitis are multifactorial. BMI and level of activity have been consistently associated with plantar fasciitis whereas as internal factors such as ankle foot biomechanics and foot posture are still inconclusive.[42–44] McKeon et al. suggests addressing heel pain by evaluating all aspect of the proposed foot core system. This system consists of three subsystems including active, passive and neural subsystems. Previous research has consistently focused on the passive subsystem through evaluation of different aspects of foot posture and has

rarely explored the influence of intrinsic foot muscle function or neural involvement in plantar fasciopathy risk. There is a large need to identify additional factors related to plantar fascia overloading and pain to mitigate future injury. Identification of risk factors for plantar fasciitis may also provide and give insight into the etiology of other overuse pathologies like tendinopathy.

Patellar tendinopathy, similar to plantar fasciopathy, can lead to degenerative soft tissue changes. The exact processes by which patellar tendinopathy develops or progresses is still unclear. However, consistent levels of tendon loading seem to be important to maintaining tendon health.[47,48] Despite the patellar tendon's ability to adapt to mechanical loading, repetitive or acute overloading can result in tendinopathy. Lack of sufficient time for the tendon to return to a proper balance between collagen breakdown and production may place the tendon at risk of injury.[49] When loading causes excessive and repetitive microdamage, collagen synthesis and matrix production must increase. If a tendon is unable to maintain homeostasis between collagen and matrix repair this could create a catalyst for degenerative processes. Tendon changes in those with chronic tendinopathy are characterized by disorganization and softening of fibril bundles, rounded shape of fibroblasts, expansion of the extracellular tissue matrix, hypercellularity and increased vascularity.[50–54] Increased cellular activity associated with cell proliferation indicates that tendinopathy is likely an ongoing process of tissue remodeling.[55] These observed changes also appear to be less expansive in those with early symptoms of tendinopathy compared to those with chronic tendinopathy suggesting that these changes occur progressively along with the continuation of symptoms.[56] Furthermore, this supports the hypothesis that tendinopathy may resolve and progress along a continuum.

Cook et al. hypothesized that the symptoms of tendinopathy, including structural changes, progress and heal along a continuum.[15] Tendons at the beginning of the continuum are described as a reactive tendinopathy characterized by poor function and load capacity but with small structural changes that have a high likelihood of returning to normal. Tendons can then either negatively progress possibly through the inability to reach a state of tendon repair and become painful or incur further structural damage or can heal and respond to clinical interventions. If tendons continue to progress along the continuum, it is suggested that they may reach a stage where the ability of the tendon to remodel is limited. Understanding the structural integrity as well as reported symptoms could help guide tailored treatment strategies. However, further research is still needed to identify factors that place one at risk for the onset of tendon overloading as well as the factors that leading to progression of the disease and non-responders to treatment.

Much like other overuse injuries there is poor agreement among study conclusions regarding risk factors for patellar tendinopathy. Extrinsic risk factors such as participating in jumping related sports, being of the male sex, and of older age have been consistently reported reported.[18,57] However, research findings for intrinsic factors have been mostly inconclusive. A recent systematic review focused on reviewing modifiable risk factors, reported a lack of strong evidence of any potential modifiable risk factors for patellar tendinopathy. Although, there was limited evidence that those with decreased lower extremity mobility, greater countermovement jump height distance and increased training volume was associated with injury. The lack of identified modifiable risk factor makes it difficult for clinicians to create prevention programs for the mitigation of injury. Mitigation of tendinopathy injuries is especially important because of the complexities that surround treatment strategies for patellar tendinopathy.

2.4 Rehabilitative Strategies

Treatment for overuse injuries with an overload mechanism generally focuses on reducing pain through a reduction loading to the affected structure followed by focal strengthening and a gradual return to loading. Many strategies focus on healing of the affected structures and working on realignment of the fibers through focused loading strategies. Although typical rehabilitation is based on realignment, self-reported pain is the main outcome used to monitor progress. Increasing self-report function and pain are important primary outcomes however it may also be important to monitor structural changes to guide treatment strategies. Past evidence indicated that new strategies for the treatment of overuse injuries are of need due to high reoccurrence rates and moderate to poor long-term outcomes in tendinopathy and fasciopathy rehabilitation.

Plantar fasciitis often focuses on reducing strain to the plantar fascia and measures effectiveness through self-reported pain and function. Evidence has shown that current conservative rehabilitation strategies are inconsistent in providing positive outcomes in patients with plantar fasciitis.[58–60] Although traditional rehabilitation may improve pain and self-reported function in this population, the majority of individuals do not return to prior levels of pain-free function.[59–61] Failure to improve with conservative management often results in more costly, higher risk procedures such as plantar fascial release surgery and extracorporeal shock wave therapy.[58,62] These expensive and high-risk treatments also vary in effectiveness and often do not allow the patient to return to pre-injury levels of function. For example, a recent study found that 43% of active duty military members that were treated with extracorporeal shock wave therapy were unable to return to running and 18% left the military due to foot related pain.[62] The

variance in patient outcomes with plantar fasciitis may be due to the lack of clinical objective measures to direct intervention strategies and monitor patient progress.

Rehabilitation techniques for patellar tendinopathy also seem to fall short based on the high chronicity and reoccurrence rate reported within those with previous patellar tendinopathy. Common interventions for patellar tendinopathy include anti-inflammatory medications, high-volume image-guided injections, image guided platelet-rich plasma injections and strengthening protocols for example eccentric strengthening. Although implementation of some of these interventions seem to have moderate short-term success in improving symptoms, long term follow-up has identified that patients continue to have lingering or a return of symptoms.

Poor long-term outcomes may indicate that not all the underlying causes of the pathology are being addressed due to failure to identify the correct treatment plan for the patients' specific symptoms. For those with plantar fasciitis this may involve the treatment of other deficiencies within the foot core system such as intrinsic foot muscle activations or tactile sensation excitability in addition to structural changes. Based on the tendon continuum hypothesis addressed previously, understanding the structural changes within the symptomatic tendon are important to providing treatment that will move a patient's tendon along the correct pathway. For example, degenerative tendons may not have the ability to adapt the non-uniformity that has developed within the tendon and therefore may need a treatment strategy focused on pain relief and strengthening fibers of the tendon around the affected collagen bundles. Ultrasound imaging technology can provide a convenient and non-invasive way to view structural changes to identify in vivo structural changes and monitor soft tissue healing.

2.5 Diagnostics

Early identification of structural changes can provide crucial information to identify pathology and guide rehabilitation strategies. Early identification in overuse injuries is important to identifying pathology and guide return to activity decisions. For example, in a patient with patellar tendinopathy information regarding structural abnormalities could help determine whether an individual should return to full sport participation or whether further monitoring is needed. Additionally, it may be able to identify subclinical changes not apparent through a physical assessment or reported symptoms.

Diagnostic ultrasound has become a useful tool for sports medicine clinicians to identify in vivo soft tissue changes. Advances in technologies over the last five years has made ultrasound technology feasible due to increases in portability, image clarity, live quantifiable measurements, and decreased cost.[10–12] Previous studies have shown that ultrasound has been able to identify tendon and fascial changes between symptomatic and asymptomatic.[63–65] Ultrasound imaging has shown high diagnostic accuracy in detecting those with and without patellar tendinopathy.[66,67] For example, one study found that ultrasound imaging had a sensitivity of 81.3% and specificity of 95.6%. when using the presence of pathological tendon thickening to diagnosis those with chronic tendinopathy in comparison to controls.[68] Studies have also identified that ultrasound had higher sensitivity in detecting abnormalities than MRI suggesting that it may have more value than more expensive imaging techniques.[69] Furthermore, a systematic review of the literature suggests that there is moderate evidence to support the use of ultrasound for predicting patellar tendon injuries through the identification of structural abnormalities within the tendon.[70]

Advancements in ultrasound technology have also brought the expansion of evaluation techniques available to clinicians. Grey scale or B mode is the primary mode of ultrasound evaluation. Grey scale imaging provides a two-dimensional image of musculoskeletal structures using ultrasound waves. The tissues of interest are depicted in variable brightness based on the return of sound waves emitted by the transducer. Grey scale ultrasound evaluation allows clinicians to observe musculoskeletal architecture and identify musculoskeletal tissue changes. Additionally, measurements such a tissue CSA or thickness can be taken on the device or using post analysis software. In the evaluation of tendinopathy and fascial structures, focal thickening, CSA, and hypoechoic areas are commonly measured to identify pathology.[70] Power doppler is another evaluation technique that allows clinician to view vascular activity within the tissue of interest. Typically, vascularity activity is viewed as a color map that allows clinicians to assess the direction as well as the magnitude of vascular flow. Quantitative evaluation is also available on newer systems which allows clinicians to gain to objectively measure vascularity rather than qualitative assessment.

The newest advancements in ultrasound evaluation are shear wave elastography (SWE) and ultrasound tissue characterization (UTC). These techniques allow clinicians to gain information regarding the biomechanical properties of musculoskeletal tissue. SWE uses a mechanical signal through the tissue area under the probe to estimate the elasticity of the tissue of interest.[71,72] SWE is comprised of three sequential steps: excitation application, tissue response measurement, and mechanical parameters estimation.[71] Shear waves images are applied over the b-mode ultrasound image and provides quantitative color electrograms which depict area of greater and lesser tissue elasticity within the area of interest. SWE allows for real-time quantification of tissue stiffness, and, by this, the assessment of the mechanical properties. UTC

uses an array transducer combined with a tracking device to take sequential images of along the length of a tendon to construct a three-dimensional data. This technique allows for the calculation of echo types across the tendon that describe the integrity and organization of fibrillar structure.[73] Both SWE and UTC are fairly new evaluation techniques with a high price point and therefore are not as readily accessible to clinicians as grey scale or power doppler evaluation techniques.

Despite moderate evidence to support the use of ultrasound to identify and manage degenerative pathologies, the relationship between structural changes viewed through imaging and patients' symptoms are not fully understood. Multiple studies have identified tendon abnormalities in those without current symptoms.[74] Asymptomatic abnormalities appear to be more common in physically active individuals, especially those most at risk such as jumping athletes, in comparison to the general population suggesting structural changes may not be the primary contributor to pain.[74] Therefore, it is important to further investigate the predictive value of ultrasound to identify subclinical changes that could mitigate injury. Additionally, more research is needed to understand the relationship between structural changes, function, and pain.

CHAPTER 3

THE EVALUATION OF THE FOOT CORE SYSTEM IN THOSE WITH PLANTAR HEEL PAIN[63]

3.1 Introduction

Plantar heel pain (PHP) accounts for nearly 8% of all injuries in those who participate in running related activities and is associated with annual healthcare costs of nearly $400 million in the United States.[75,76] Patients with PHP report pain and palpable tenderness in the area of the medial tubercle of the calcaneus, pain that is increased when taking the first few steps in the morning, and at the beginning of exercise.[77] Most importantly, those with PHP have reported a significant decrease in health related quality of life signifying this condition creates activity limitations and participation restrictions.[9] Although there has been consistent evidence regarding the symptoms of PHP, there has been inconsistent evidence to explain the etiology of this condition which limits the ability to create novel rehabilitation strategies.

An abundance of research has been conducted to identify risk factors for PHP.[27,78–81] Risk factors can typically be classified as either intrinsic or extrinsic. Intrinsic risk factors are commonly related to biological and anatomical factors such as weight, age, gender, and foot posture whereas extrinsic risk factors are related to training, shoe wear, and training surfaces. A recent systematic review conducted by Van Leeuwen and colleagues (2016) evaluated all risk factors that have been investigated in athletic and non-athletic populations and determined that the majority of evidence related to risk factors was inconclusive or inconsistent. However, there was consistent evidence that non-active individuals with PHP displayed a higher BMI. Studies that have measured static foot posture are inconclusive in identifying differences in foot posture in the non-active population with little evidence regarding active individuals.[44,82] The lack of

other identifiable risk factors indicates measures of static foot posture alone may not provide all of the information necessary to investigate the foot's ability to attenuate forces and stabilize the foot. Additional evaluation into other aspects of foot function may be needed to fully understand the underlying impairments associated with PHP.

Recently, a new model for investigating foot function has been proposed which suggests foot function is composed of three subsystems (active, passive, and neural) that interact to stabilize the foot and sustain sensorimotor function.[83] Together the active, passive, and neural subsystems form the foot core system which allows dynamic foot control. A majority of the research related to identifying intrinsic risk factors and treating PHP have focused on the passive subsystem using measures such as navicular drop, rear-foot angle, and the FPI-6.[44] Thickness of the plantar fascia has also been identified as potential method to discriminate between those with and without PHP.[84] However, there has been limited investigation of the active (intrinsic and extrinsic foot muscles) or neural (musculotendinous, ligamentous, and plantar cutaneous receptors) subsystems as they relate to PHP. Examining aspects of all three subsystems may advance our understanding of PHP and lead to novel directions for rehabilitation.

In regards to the active subsystem, the intrinsic foot muscles play a key component of force attenuation, midfoot stabilization, and preparation for propulsion.[85,86] Intrinsic foot muscle activation has a direct relationship with medial longitudinal arch loading suggesting these muscles contribute to the buttress like stabilization provided by the plantar aponeurosis.[85] Cadaveric research has determined medial longitudinal arch height can be manipulated by releasing or tensioning the abductor hallucis muscle alone.[87] Intrinsic foot muscle atrophy has been identified in those with PHP using volumetric MRI measurements which suggests these muscles may be linked to the development or progression of this condition.[88] Further

investigating alterations in intrinsic foot muscle involvement in individuals with PHP may provide insight into deficiencies in force attenuation and midfoot stabilization which are associated with this condition.

The neural subsystem of the foot core is responsible for sensory function and plays a key role in initiating motor output. The medial and lateral plantar branches of the tibial nerve innervate the intrinsic foot muscles as well as the skin on the plantar surface of the foot. Both nerves course through the tarsal tunnel and intrinsic foot muscles, making them susceptible to impingement; particularly in the presence of altered foot posture and active inflammation.[89] Individuals with other lower extremity injuries, such as anterior cruciate ligament injury and chronic ankle instability, have exhibited decreased sensory function in the foot which is thought to be related to sensory-reweighting following damage to ligamentous receptors.[90,91] Currently, no research has specifically investigated the neural subsystem of the foot in those with PHP; however, diminished plantar cutaneous sensation could diminish sensory feedback related to the interaction between the foot and environment.

Examining each subsystem and viewing foot function as a multifunctional system may identify different avenues for treatment of foot dysfunction.[92] Therefore, the aim of this study was to compare aspects of the passive, active, and neural subsystems of the foot in those with and without PHP. Our hypothesis was that individuals with PHP will more commonly demonstrate a pronated foot posture, increased plantar fascia thickness, decreased intrinsic foot muscle function, and diminished plantar sensation in comparison to matched healthy individuals.

3.2 Methods

Design

This cross-sectional study was designed to compare aspects of the passive, active, and neural subsystems of the foot in physically active adults with and without PHP. All participants were recruited through advertisements and word of mouth from the general population and surrounding community of a large public university in the United States from March 2017 to November 2018**.** All participants signed an informed consent approved by the institutional review board. Participants reported to the research laboratory on the campus of a large urban public university for a single testing session. The dependent variables included the FPI-6, intrinsic foot muscle test, cross-sectional area (CSA) and thickness of the abductor hallucis muscle, plantar fascia thickness, and light touch plantar cutaneous sensation. All assessments were performed by a licensed athletic trainer with over three years of experience in utilizing the measurements in this study, excluding the FPI-6 which was also collected by another athletic trainer with over one year of experience utilizing foot posture measures.

Participants

Sixteen participants with PHP (females=13, males=3) and sixteen matched participants with no history of PHP (females=13, males=3) were included in the study. To be included in either group, participants had to report a score of ≥24 on the Godin Leisure-Time Exercise Questionnaire, no history of lower extremity injury (besides heel pain) in the last three months, lower extremity surgery, or any other disorder that could affect foot function (e.g. diabetes, neuropathy, rheumatoid arthritis).[93] PHP participants also had to report heel pain for ≥1 month, pain upon palpation of the medial calcaneal tuberosity, and a history of early morning plantar heel pain within the past month which decreased after walking, and/or increased pain after exercise or prolonged periods of standing. Healthy participants reported no history of heel pain. Healthy participants were matched to a healthy participant based on age, height, weight, and

limb. Subjects in both groups completed the Foot and Ankle Ability Measure (FAAM) Activities of Daily Living and Sports subscales to assess self-reported function. For each scale the item score total was divided by the highest potential score, multiplied by 100, and expressed as a percentage. Lower percentages indicated a lower level of function. In the case of participants with bi-lateral heel pain, the limb with the lower self-reported level of function, as measured by the lower FAAM subscales, was selected. All participant demographics are reported in Table 1.

Instrumentation

Intrinsic foot muscle function was assessed using the Intrinsic Foot Muscle Test (IFMT). CSA of the abductor hallucis and plantar fascia thickness was captured using diagnostic ultrasound with a 17-5 MHz linear array transducer probe (Sonosite M-Turbo; FUJIFILM SonoSite Incorporated, Bothell, WA) and measured using Image J software (National Institute for Health, Bethesda, MD). Sensation was tested using Semmes-Weinstein Monofilaments (Touch-Test Sensory Evaluator; North Coast Medical, Gilroy, CA).

Static Foot Posture

Foot posture was assessed using the FPI-6. The FPI-6 uses a multi-factorial approach to assessing foot posture that has demonstrated excellent intra-rater reliability (ICC=0.90).[94] During assessment the participants stood in a relaxed bi-pedal stance. The participant's foot posture was graded individually based on six criteria: talar head palpation, supra and infra lateral malleolar curvature, calcaneal frontal plane position, prominence in the region of the talonavicular joint, congruence of the medial longitudinal arch, and abduction/adduction of the forefoot on the rearfoot. Each criterion was graded based on a 5-point likert-type scale ranging from -2 to 2 and then were summed for a total score ranging between 12 and -12 as described in previous methods. Total scores are typically categorized into 5 categories: normal (0 to +5), pronated (+6

to +9), highly pronated (+10 and above), supinated (−1 to−4), and highly supinated (−5 to −12).[95] Total score was utilized for statistical analysis.

Plantar Cutaneous Sensation Thresholds

Sensation was assessed using Semmes-Weinstein Monofilaments at the head of the first metatarsal and 50% of the medial longitudinal arch.[96] Participants were asked to lay prone on the table while wearing noise reducing head phones and verbally indicate as quickly as possible when they perceived a monofilament was applied. The monofilaments were applied using a previously described 4-2-1 stepping algorithm which has demonstrated acceptable intrarater (ICC=0.61-0.85) and interrater reliability (ICC=0.62– 0.92) for identifying light touch detection thresholds.[97] The lightest weight monofilament which was detected during at least 50% of the applications was identified as the detection threshold. Detection thresholds were evaluated once at each site and used for analysis.

Intrinsic Foot Muscle Function

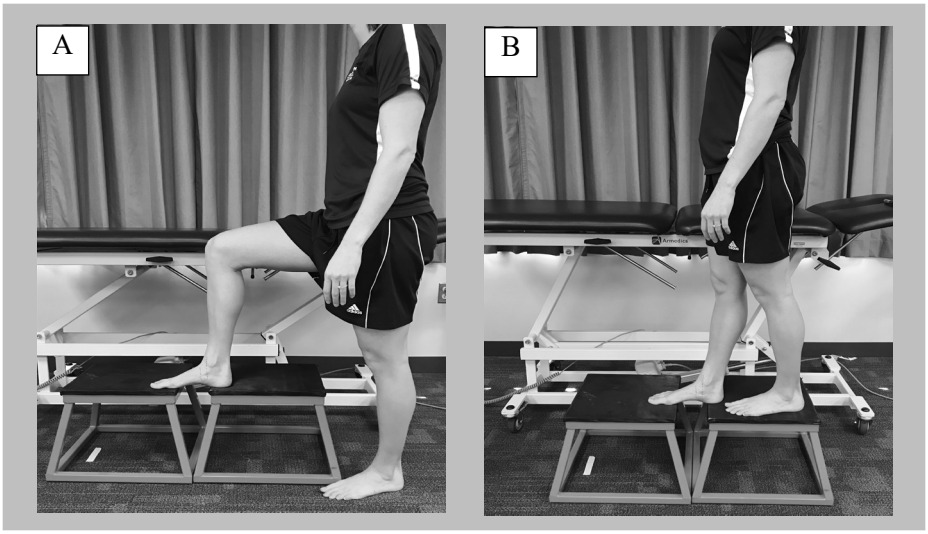
Function of the intrinsic foot muscles was assessed through the intrinsic foot muscle test. Participants viewed a video of the test being instructed and performed using the technique described by Jam (2006) in order to provide a clear and consistent explanation of the IFMT.[98] Participants were asked to stand barefoot in front of a wall with their feet shoulder width apart, raise their toes off the floor while consistently maintaining their foot arch, and then lower their toes while maintaining arch height. Participants performed one practice trial for 30 seconds in which they were given verbal instructions related to the test positioning and the goal of the task but no feedback on their performance. After the practice trial, participants performed a 30 second trial in which the investigator assessed whether the participant was able to continuously maintain navicular height without extending toes off the ground, curling the toes, or changes in navicular

height. The investigator graded the task as satisfactory, fair, or poor based on whether they were able to maintain navicular height without displaying compensatory extrinsic muscle activity throughout the trial consistently, inconsistently, or not at all.[99] The grade was recorded and used for analysis.

Diagnostic Ultrasound

Diagnostic ultrasound was used to assess CSA and thickness of the abductor hallucis. Prior to capturing abductor hallucis measurements the assessment area was defined by two parallel lines that were drawn anterior to the medial malleolus and posterior to the navicular tuberosity. Abductor hallucis CSA and thickness were measured first during a resting state followed by an active state (Figure 3.1). During the resting state, the participant stood comfortably on the non-test limb with the test limb resting on a box. Active measurements were collected with the participant standing on the step with equal weight dispersed between both limbs. The participant also performed the same task they did during the IFMT task but in a tandem stance instead of single leg. CSA and thickness measures were captured as they performed the task. The test-retest reliability of abductor hallucis CSA (ICC=0.81) and thickness (ICC= 0.93) measurements are excellent during both resting and active conditions (Fraser, Mangum, & Hertel, 2018).

Figure 3.1 Participant positioning during the resting state (A) and active state (B) measurements of abductor hallucis with the diagnostic ultrasound.

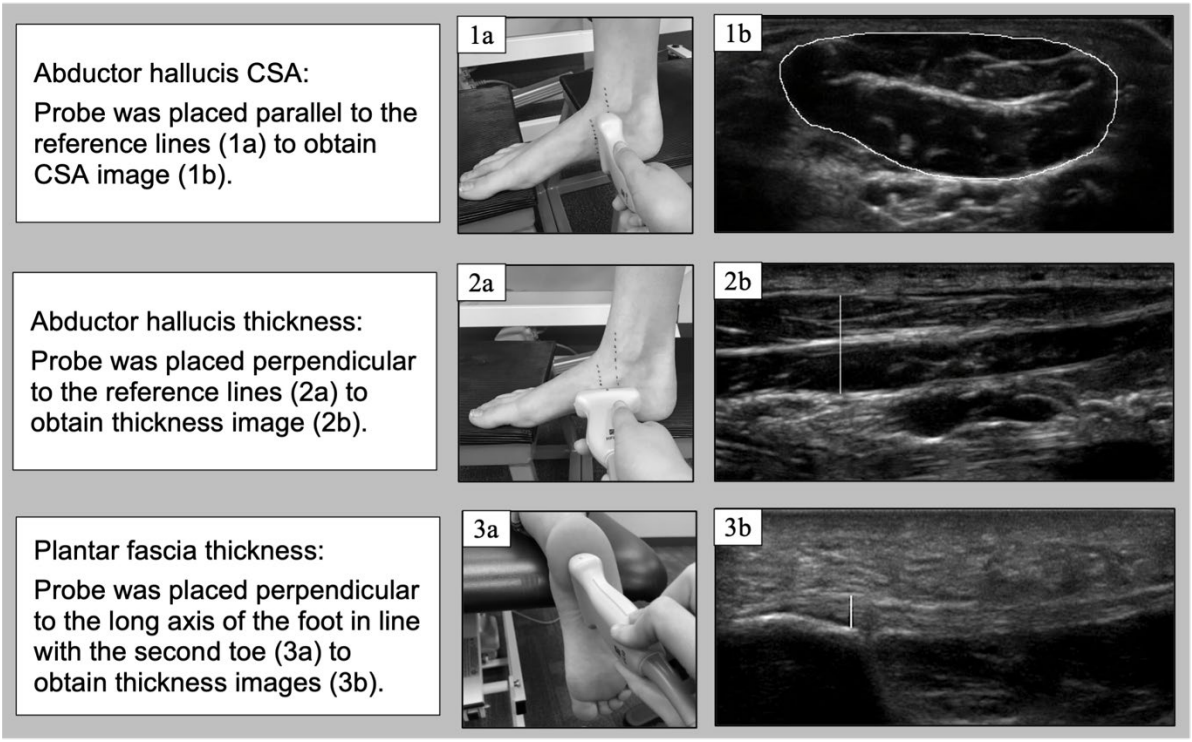


After measurements of the abductor hallucis were completed, plantar fascia thickness was also assessed using diagnostic ultrasound. For this assessment, the participant laid prone on a treatment table with their feet off the edge of a plinth allowing their ankle joint to rest in a neutral position. Plantar fascia thickness was measured at three different sites along a longitudinal line between the medial calcaneal tubercle and the second toe based on previous methods which displayed excellent interrater reliability (ICC 0.94-0.98).[100] The first segment was captured at the origin of the plantar fascia on the medial calcaneal tubercle. The second segment was scanned at the plantar aspect of the navicular tubercle and the last segment was captured slightly proximal to the second metatarsal head.

Two separate images were taken at each site with removal of the probe in between each image. Following collection, all images were exported and loaded to a desktop computer for

analysis using Image J software (National Institute for Health, Bethesda, MD). The image software was calibrated to 100 pixels/cm which was based on a 0.5 cm caliper placed on the ultrasound image. CSA images of the abductor hallucis were measured by tracing along the inner aspect of the fascial border. Thickness images were measured using a vertical caliper. Abductor hallucis thickness was measured from the inner aspect of the superficial fascial border to the inner aspect of the deep fascial border at the thickest aspect of the muscle. Plantar fascia thickness was measured from the superior to inferior border of the fascia at the thickest aspect of the fascia for each of the three different segments. The average of the two measurements at each site were averaged and used for analysis. The active measurements for the abductor hallucis were normalized to the resting measurements and reported as an activation ratio.[101] Activation ratios >1 indicate an increase in muscle size during the active condition whereas ratios <1 indicate a decrease in muscle size during the active condition.[101] Probe placement and corresponding images can be in in Figure 3.2.

Figure 3.2 Probe placement and images for abductor hallucis and plantar fascia measurements.



Statistical Analysis

Descriptive statistics were calculated for group characteristics and included all dependent variables. The Shapiro-Wilk test was used to evaluate the normality of the data for each dependent variable in order determine the most appropriate test to examine group differences. Independent t-tests were used to assess group differences for abductor hallucis morphology and plantar fascia thickness. Mann-Whitney U tests examined differences between groups for the FPI-6 and sensation thresholds. Hedge's g effect sizes were also calculated for all measures.[102] Effect sizes (ESs) were interpreted as large (≥0.8), medium (≥0.5), and small (≥0.2) as suggested by (Cohen, 2013).[103] Finally, a Chi-square test assessed group differences in IFMT ratings. Due

to the small number of participants who obtained a satisfactory rating, the satisfactory and fair categories were combined to meet minimum statistical requirements. The threshold for statistical significance was set a priori at $p<0.05$.

3.3 Results

There were no significant differences in age, height, weight, or physical activity level between groups. Those with PHP had significantly lower scores on the FAAM and FAAM-Sport indicating poorer self-reported function (Table 3.1). Individuals with PHP exhibited a more pronated foot posture (Hedge's g=1.07, 95% CI[0.33-1.81], p=0.02), decreased sensation at the base of the first metatarsal (Hedge's g=0.92, 95% CI[0.19-1.65], p=0.01,), and greater plantar fascia thickness at the proximal site (Hedge's g=0.88, 95% CI[0.15-1.61], p=0.02) compared to healthy matched controls. There were no significant differences in abductor hallucis CSA or thickness at rest, active ratio for the CSA or thickness, or plantar fascia thickness at the midfoot or distal regions. Additionally, there were no significant differences in IFMT ratings (p=0.08). Descriptive statistics for all dependent variables can be found in Tables 3.2 to 3.4.

Table 3.1 Participant demographics.

Variable	Healthy (Mean±SD)	PHP (Mean±SD)	P-value
Age (years)	26.06±1.73	25±2.20	0.70
Height (m)	1.67±0.02	1.65±0.02	0.55
Weight (kg)	67.14±2.79	66.71±2.76	0.91
GLTEQ	64.94±4.50	72.69±7.57	0.39
FAAM-ADL(%)†	100.00±0.00	82.12±22.02	<0.01*
FAAM-S (%)†	100.00±0.00	79.69±29.68	<0.01*
Heel Pain (mo)†	0.00±0.00	30.00±55.80	<0.01*

SD=Standard Deviation, PHP=Plantar Heel Pain, FAAM=Foot and Ankle Ability Measure, FAAM-S=Foot and Ankle Ability Measure Sport Subscale, GLS-Godin Leisure-Time Exercise Questionnaire
*Denotes significant group difference ($p<0.05$). † Denotes that the median and interquartile range are being displayed as well as Mann Whitney U test.

Table 3.2. Comparison of values for clinical measures between healthy and PHP participants.

	Healthy Median (IQR)	PHP Median (IQR)	P-Value	Hedge's g (95% CI)
FPI-6	3.50 (5.5)	6.50 (7.0)	0.02	1.07 (0.33-1.81)
Sensation Threshold 1st Met	3.22 (0.78)	3.61 (0.80)	0.01*	0.92 (0.19-1.65)
Sensation Threshold Arch	3.61 (0.74)	3.84 (0.47)	0.06	0.73 (0.02-1.45)

PHP=Plantar Heel Pain, IQR=Interquartile Range, CI=Confidence Interval
*Denotes significant difference between healthy and plantar heel pain groups (≤0.05)

Table 3.3 Comparison of values for ultrasound clinical measures between healthy and PHP participants.

	Healthy (Mean±SD)	PHP (Mean±SD)	P-value	Hedge's g (95% CI)
Abductor Hallucis CSA Resting (cm^2)	1.87±0.47	2.0±0.52	0.45	0.28 (-0.42-0.97)
Abductor Hallucis Thickness Resting (cm)	1.10±0.24	1.16±0.23	0.46	0.25 (-0.44-0.95)
Abductor Hallucis CSA Active Ratio	1.06±0.89	1.08±0.08	0.52	0.21 (-0.49-0.90)
Abductor Hallucis Thickness Active Ratio	1.03±0.11	1.01±0.12	0.57	-0.19 (-0.89-0.50)
Proximal Plantar Fascia Thickness (cm)	0.25±0.04	0.32±0.10	0.02*	0.88 (0.15-1.61)
Midfoot Plantar Fascia Thickness (cm)	0.13±0.02	0.13±0.03	0.81	0.08 (-0.62-0.77)
Distal Plantar Fascia Thickness (cm)	0.08±0.01	0.09±0.02	0.33	0.36 (-0.34-1.06)

PHP=Plantar Heel Pain, CSA=Cross sectional area, SD=Standard Deviation, CI=Confidence Interval
*Denotes significant difference between healthy and plantar heel pain groups (≤0.05)

Table 3.4. Frequency of IFMT Rating.

	Healthy N=16	PHP N=16
Poor	5	10
Fair	9	6
Excellent	2	0

IFMT= Intrinsic Foot Muscle Test, PHP=Plantar Heel Pain

3.4 Discussion

The main findings of this study were those with PHP presented with increased static foot pronation, heightened plantar cutaneous sensation thresholds, and proximal plantar fascia thickening compared to healthy controls. However, there were no significant differences in abductor hallucis size, activation ratios, or IFMT performance between groups. Overall, these findings indicate that those with PHP displayed distinct differences within the passive and neural subsystems of the foot core.

Individuals with PHP exhibited a more pronated foot posture and increased plantar fascia thickness compared to those without a history of PHP. This study provides evidence that there may be an association between foot posture and PHP in active individuals. However, previous research in non-active individuals with PHP has found mixed results regarding static foot posture.[78,82] Irving et al. (2007) identified a more pronated foot posture in those with PHP compared to healthy controls but other studies that also utilized the FPI-6 have found foot posture values consistent with a more neutral foot posture in those with PHP.[59,82,104] Therefore, more evidence may be needed to understand the link between static foot posture and plantar fascia strain in an active population. Within this study, those with PHP also displayed increased plantar fascia thickness near the calcaneal region in comparison to healthy matched controls. This finding is in line with previous research that has identified plantar fascia thickening at its origin on the calcaneus in this population.[84,105] Overall, these results support the idea that alterations in the passive system of the foot core contribute to PHP.

Along with tissue changes in the plantar fascia, significant deficits in cutaneous sensation were also found in the PHP group. Sensation deficits were most prominent at the head of the first metatarsal; however, a large positive effect size was also identified at the medial longitudinal

arch. These findings suggest that patients with PHP; similar to other lower extremity pathologies such as chronic ankle instability and ACL reconstruction, may obtain less tactile information from the environment to direct their movements.[90,91] There have not been any previous studies that have explored neural involvement with PHP; however, future studies should determine if this impairment is linked to aberrant movement patterns or force absorption characteristics in these patients. Therefore, the findings of the current study suggest the neural subsystem of the foot core may be compromised in patients with plantar heel pain and should be further investigated as an underlying contributor to movement dysfunction in this population.

We hypothesized that individuals with PHP would demonstrate alterations in the morphology or activation ratio of the abductor hallucis. Results from two previous studies found differences in intrinsic foot muscle volume through the use of MRI.[88,106] Although both previous studies identified decreased intrinsic foot muscle volume in those with PHP compared to healthy controls, they were inconsistent in the location of the deficits. One study observed decreases only in the forefoot volume, whereas the other study found differences only in the rearfoot volume.[88,106] Total intrinsic muscle volume for the plantar foot was not significantly diminished in either study. The assessment in this study was unique in that it also utilized active and resting states of the muscle to evaluate activation ratios. The activation ratios were derived from previous research in which they were used to investigate intrinsic foot muscle activation following a training regimen.[101] These findings indicate that PHP participants could activate the abductor hallucis during the contracted conditions, similar to healthy controls. This is supported by the non-significant difference in performance ratings on the IFMT between groups. This evidence suggests that those with PHP do not have atrophy of the abductor hallucis or decreased motor function during standing tasks. This study may differ from previous studies since it only

included the abductor hallucis and not all of the intrinsic foot muscles as in previous MRI studies. The abductor hallucis was collected as a representation of the intrinsic foot muscle group due to evidence on its contribution to supporting the medial longitudinal arch as well as the accessibility to the muscle using diagnostic ultrasound.[30] Additionally, the task used to initiate activation may not have represented the muscle function in everyday use as activation may be linked to the lengthening of the foot under load rather than cued activation. Based on the data collected in this study, no evidence was identified that strongly linked involvement of the active system to plantar heel pain participants; although examining foot control with the IFMT may warrant further investigation.

Limitations

There are limitations to this study. Individuals that participated in the study were excluded if they reported a lower extremity injury within the past three months. However, participants in either group could have had other past injuries that may have affected their outcomes. The investigators performing the assessments were not blinded to group affiliation. The study also included a small sample size.

Despite sample size constraints, meaningful differences were detected within the passive and active subsystems of the foot core system when comparing individuals with and without PHP. However, a larger sample may have permitted detection of smaller group differences in variables such as the IFMT.

Additional efforts to quantify intrinsic foot muscle function as well as the other subsystems of the foot are necessary to understanding the mechanisms of PHP. There is currently no gold standard to assess intrinsic foot muscle function and therefore, further investigation is needed to assess active foot performance in this population.[107] The IFMT used to assess intrinsic

foot muscle function has slight to moderate intra-rater reliability (ICC= 0.17-0.44).[101] However, the IFMT may still be able to provide useful information regarding foot function performance.[98] Also, the present study focused on the abductor hallucis muscle and may not represent the entire active subsystem. The current study also evaluated the participant's ability to contract the abductor hallucis during a static task and may not represent the coordination of these muscles during dynamic activities such as gait. Further research should be done to evaluate additional intrinsic foot musculature during dynamic activity when all subsystems are working collectively.

Furthermore, this research investigation sought to examine characteristics of those with PHP compared to matched healthy controls in an active population. The participants with PHP within this study reported decreased foot function per the FAAM scales. However, on average they reported lower levels of disability than individuals with PHP in more general populations.[108] Additionally, the individuals in this study reported relatively high levels of physical activity indicating they were able to participate in activity despite their condition. Therefore, the findings of this study may not be generalizable to the sedentary population with PHP.

3.5 Conclusion

These findings provide evidence that PHP is associated with both mechanical and sensory deficits in the foot within the active population. These findings also suggest that ultrasound imaging may provide clinical utility in the identification of plantar fasciopathy. Future research should determine how impairments in the passive and neural subsystems contribute to the development or progression of PHP by exploring their connection to self-reported function and dynamic activities.

CHAPTER 4

RELATIONSHIP BETWEEN PATELLAR TENDON ABNORMALITIES AND LOWER EXTREMITY FUNCTION: A SYSTEMATIC REVIEW

4.1 Introduction

Patellar tendinopathy is common among physically active individuals from adolescent age to late adulthood and can led to a disruption of daily life through negative impacts on social life, physical activity levels, and self-efficacy.[8] Patellar tendon injuries can develop throughout an individual's lifespan as well as an acute phase of training in an active individual. Elite athletes that participate in jumping type sports seem to have been the most at risk over other populations.[23]

Tendinopathy is used as an overarching diagnosis for individuals that present clinically with tendon pain during loading of the knee extensors and decreased reported function. Development of the disease is attributed to repetitive stress over time or a significant ramp up in loading placed on the extensor mechanism. Continuous acute overloading of the tendon, which does not allow the tissue to heal and adapt, can lead to pathological changes within the tendon resulting in pain and dysfunction.[54] If continuous overloading is not identified and managed appropriately, studies suggest that patellar tendinopathy can lead to long term pain and disability.[21]

Patellar tendinopathy has a high incidence of chronicity and recurrence which could be attributed to the poor long term outcomes reported following popular treatment approaches. Current treatment strategies such as active rest, eccentric strengthing,[109–112] anti-inflammatory medications, high-volume image-guided injections,[113] image guided platelet-rich plasma injections,[114] extracorporeal shockwave therapy,[115] and surgical interventions[116] have poor long

term impact despite short term pain reduction.[117] Adolescents diagnosed with patellar tendinopathy, in a prospective study, reported continued pain and dysfunction into adulthood.[21] Moreover, many adults diagnosed with patellar tendinopathy report reoccurring pain continuing after rehabilitation programs. The tendon continuum model proposed by Cook et al.[15] hypothesizes that tendon health falls on a continuum with three main stages of pathology: reactive tendinopathy, tendon disrepair and degenerative tendinopathy. The model identifies the importance of early identification and proper management of care as it is unknown whether a tendon can fully repair once it has reached the degenerative stage; this is a possible reason for poor prognoses for individuals with chronic tendinopathy.[15] Improving early diagnosis before a tendon is in the degenerative stage may improve long term outcomes.

Diagnosis and management of patellar tendinopathy has traditionally relied on detailed subjective information and a number of special tests that focus on eliciting pain through tendon loading. Although these tests are very sensitive for identifying patients with patellar tendon pain, they are unable to provide specific information relating tendon structure. Histopathological changes such as deformed tendon fibers, separated and loss of hyalinization, non-uniform alignment of fibers and random blood vessel formation within the tendon have been confirmed in those with tendinopathy through biopsied tissue following surgical intervention.[54]

Assessing tendon structure is important to properly staging tendon health along the continuum model of tendon pathology. Those with suspected late-stage degeneration will need an entirely different management strategy and loading program than individuals with early-stage tendon response. Current special tests may not be to provide enough information to correctly stratify individuals along the continuum or identify sub clinical tendinopathy. Identifying tendon structure markers indicative of degeneration could aid in early management and mitigate further

tendon damage. Management techniques for patellar tendinopathy focus on pain reduction, protection through low loading, and principles related to tissue repair, such as realigning new tendon fibers through graded activity. However, imaging of tendon structure is not typically used to verify tendon disrepair and monitor the remodeling process during rehabilitation. A better understanding of the underlying pathophysiology driving patellar tendon pain and dysfunction may aid in improving outcomes in patellar tendinopathy patients.

Ultrasound imaging has been shown to be an accurate tool identifying pathologic changes such thickening, paratenon irregularly, intra-tendinous calcifications, and increased focal echogenicity in those with patellar tendinopathy versus healthy controls.[118] Ultrasound imaging has a high diagnostic accuracy in detecting abnormal findings in anterior knee pain patients and specifically patellar tendinopathy.[66,67] For example, Nishida et al.[68] assessed thickness measurements in those with and without the condition and ultrasonography had sensitivity of 81.3% and specificity of 95.6% for patellar tendinopathy. A few studies also identified that ultrasound had higher sensitivity in detecting abnormalities than MRI suggesting that it may have more value than more expensive imaging techniques.[69]

Although, ultrasound imaging has shown promise in identifying abnormalities in those with reported symptoms, there have been several studies that have identified patellar matrix changes in seemingly asymptomatic individuals, especially in highly active populations. Several rationales have been described to explain these asymptomatic changes. Some researchers have noted that the presence of tissue changes in asymptomatic individuals suggests that structural changes identified via ultrasound may not be clinically relevant while other research suggests that these changes may actually be indicative of early sub-clinical changes that can lead to tendon degenration.[15,119,120]

A systematic review with meta-analysis detailed that ultrasound imaging was able to identify tissue characteristics associated with a higher risk of patellar tendinopathy symptoms.[120] Being able to identify sub clinical factors associated with future injury may allow clinicians to intervene early and mitigate future injury. Although these abnormal findings identified through ultrasound seem to have diagnostic and predictive utility, there is little consensus on the relationship between abnormal tendon structure findings and perceived and clinical measures of lower extremity function. An array of studies have reported on the relationship between ultrasound findings and function but there lacks a thorough assessment of the findings from the current literature.[64,121] Further assessment of studies that have sought to understand the relationship between patellar matrix changes and function, clinical and self-reported, is warranted. Information gained from this review would help gauge the clinical utility of ultrasound to guide management for those with patellar tendinopathy. The purpose of this review was to synthesize existing evidence on the relationship between patellar tendon changes assessed by ultrasound imaging and subjective and objective function.

4.2 Methods

Search Strategy

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses were used to guide the methods of this review. Searches were conducted in MEDLINE, CINHAL, SPORTDiscus, and the Cochrane Library from inception to 30 February 2024 using the following terms: (patellar tend* OR patella tend*) AND (ultrasonograph* OR ultrasound OR sonograph*). Eligibility criteria were developed to narrow down literature based on population, ultrasound evaluation technique, and functional outcomes. Reference lists for identified studies were screened to identify any eligible studies not found in the initial search.

Eligibility Criteria

Articles identified through search terms on all databases were merged to identify and remove all duplicate articles. Articles were further eliminated by screening the title and abstracts of studies based on inclusion and exclusion criteria. The full text of all remaining articles were reviewed to verify they met eligibility criteria.

Peer reviewed articles were included if they assessed patellar tendon morphology using ultrasound imaging and either perceived or objective measures of lower extremity function. Furthermore, they must have reported on the relationship between ultrasound parameters and lower extremity function or reported group differences between those with and without patellar tendon morphology abnormalities. Only the results from baseline assessments were included for studies that included an intervention component to decrease an interventional effect on the results. Studies were excluded if they did not meet the inclusion criteria or if they included populations with current knee conditions unrelated to patellar tendinopathy such as ligamentous tears or repair, Osgood-Schlatter disease, patellofemoral pain; and participants with disorders of the motor system impacting coordination and muscular control. The process of including and excluding articles is illustrated in Figure 4.1

Data Extraction

Once the eligible articles were identified studies were grouped based the ultrasound parameters used for assessment as well as the reported functional outcomes. Functional outcomes were separated and described based whether it was an objective or subjective measure. Study characteristics were extracted from all studies to include: author, year, study design, sample size, participant demographics, ultrasound imaging parameters and type of functional assessment.

Quality evaluation

The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement (checklist for cohort, case–control and cross-sectional studies) was used to critically appraise the quality of all included studies. The STROBE statement is a checklist consisting of 22 items that are regarded as critical for reliable reporting of observational studies.[122] All included articles were appraised individually by two authors. Discrepancies in quality scores between the two raters were discussed to reach a consensus. Each item scored a maximum of 1 point if full reporting criteria were met and 0 point if the criteria was not met, for a total possible score of 22 points.

4.3 Results

Data Synthesis

The initial database search identified 3,151 results which resulted in 23 included articles after the implementation of the search strategy (Figure 4.1).[65,121,123–139] Results of the quality evaluation are depicted in (Table 4.2). Due to the high heterogeneity of the methods differences and included outcomes among the included studies a meta-analysis was not warranted.

Subjective measures of lower extremity function and ultrasound imaging results are reported in 19 of the included studies (Table 4.3). [65,121,123–139] All articles that reported objective function used the VISA-P to record participants self-reported function except one study which used the Oslo Sports Trauma Research Centre Overuse Injury Questionnaire (OSTRC-OIQ). Descriptive statistics of the VISA-P scores are depicted in Table 4.3. In addition to administering self-reported pain questionnaires, a few studies also assessed pain during function using a visual analog scale (VAS).[125,131,136]

Objective measures of lower extremity function along with ultrasound results were reported in nine of the included studies (Table 4.4).[121,130,132,134,136,140–143] The type of lower extremity functional test varied largely across the identified studies and included lower extremity mobility, kinetics and biomechanics during jumping tasks and maximum voluntary contraction (MVIC) assessments of thigh musculature. Results from studies that investigated similar patellar tendon morphology characteristics and functional tests were compared to provide a qualitative results summary.

Figure 4.1 Literature search flow chart.

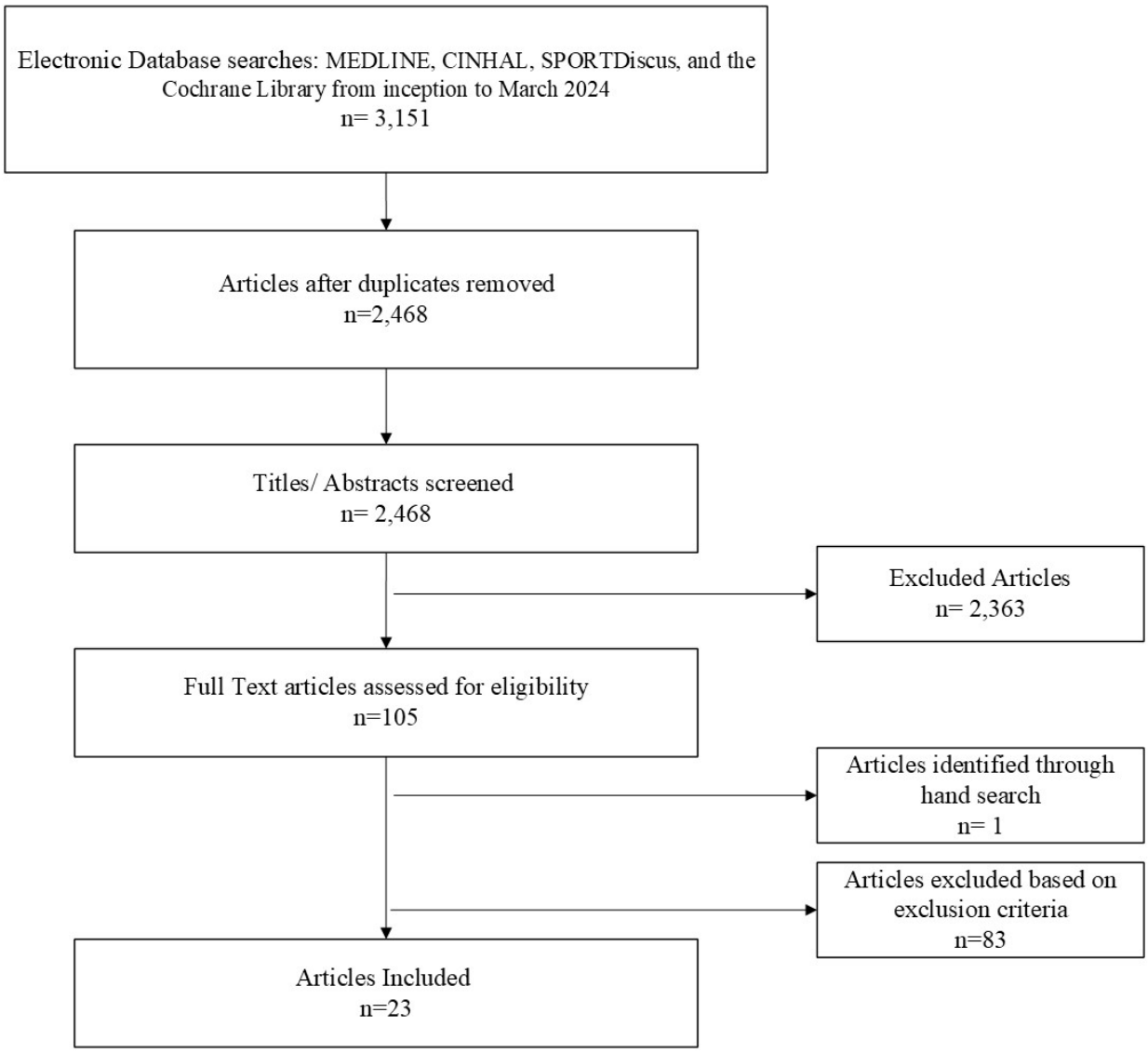


Table 4.1 Characteristics of included studies.

Study	Study Design	Participants Characteristics	Population	US Classification/ Measurement	Functional Outcome(s)
Arias-Buría et al. 2020[123]	Cross-Sectional	40 Total 20 PT(F:0; 45.0 ±4.5yrs) 20 Control(F:0; 42.5 ±5.5yrs)	Athletes with unilateral painful patellar tendinopathy and controls	Thickness(mm); Width(mm); CSA(mm^2) *Vascularity:* (0): no vessels visible; (1) 1 to 2 vessels; (2) 3 to 5 vessels; (3) vessels in up to 30%;(4) vessels in 30–50% of ROI; (5) vessels in >50% of ROI	VISA-P
Benítez-Martínez et al. 2020[124]	Observational	73 Total (F:0; 26.8±4.8yrs) No PTA: 155 tendons PTA: 91 tendons	Professional basketball players	*Abnormal Thickening:* thickened tendon that altered the tendon symmetry in the transverse section *Hypoechogenicity:* zone with focal changes in echogenicity *Neovascularization*: vessel greater than 1 mm in length within the tendon on Doppler assessment	VISA-P
Cook et al. 2000[65]	Prospective cohort	26 Total (F:18; 14-18 yrs*) 10 PTA 42 Control	Asymptomatic elite junior basketball players'	*Normal; Abnormal:* presence of hypo-echoic region, or fusiform swelling Maximum length (mm); Width (mm); Height of hypoechoic regions (mm)	VISA-P
Cook et al. 2004a[140]	Cross-sectional	135 Total (F:64; 14-18yrs*)	Junior basketball players	*Normal; Abnormal:* abnormality in the fibre structure of the tendon such as a hypoechoic region or fusiform swelling.	Agility run; Endurance; Maximum vertical jump; Speed;
Cook et al. 2004b[125]	Comparative design	111 Total (F:42; NR yrs) 27 PTA without vascularity 19 PTA with vascularity	Elite and non-elite volleyball players	*Grey-Scale PTA:* (1)Normal; (2) Abnormal: presence of discrete hypoechoic area or separation of fibers with increased AP diameter Hypoechoic CSA (mm^2) *Vascular PTA:* (1)Normal; (2) Abnormal: vessel within the tendon estimated >1mm in length	VISA-P; SLDS VAS

Table 4.1 Continued.

Study	Study Design	Participants Characteristics	Population	US Classification/ Measurement	Functional Outcome(s)
Dirrichs et al. 2018[126]	Prospective longitudinal	35 Total (F:15; 46 ± 18) 47 Symptomatic tendons (Achilles:17 ; Patellar:15; humeral-epicondylar:15)	Individuals with chronic pain of the Achilles, patellar, or humeral radial/ulnar epicondylar tendon	*Grey-scale:* (1)Normal; (2)Abnormal: Inhomogeneous texture, hypoechogenic tendon swelling or thickening, partial tear, tendon calcification, or fluid *Vascularity:* (1)Normal; (2)Abnormal: presence of vessels within the tendon on power doppler *Tendon Stiffness:* (1)Normal: SWE values of above 70 kPa (4.83 m/s); (2) Abnormal: SWE values of below 70 kPa (4.83 m/s)	VISA-P
Docking et al. 2020[127]	Prospective cohort	175 Total 149 Achilles (F:0; 189.9±7.8 yrs) 152 Patellar (F:0; 189.7±8.1 yrs)	Elite male football players with and without tendinopathy	*Grey-scale:* (1)Normal;(2)Abnormal: presence of hypoechoic region or fusiform swelling *Tissue Characterization:*(1) aligned tendon bundles; (2) increased separation and/or waving fasciculi; (3) decreased fibrillar integrity; (4) absence of fibrillar organization Tendon diameter (mm); Percentage of echo types (%); Mean CSA of echo types (mm^2); Total tendon mean CSA (mm^2)	OSTRC-OQ
Durcan et al. 2014[128]	Cross-sectional	83 Total (F:0; 20.4±1.5yrs) 30 PTA (F:0; 20.4±1.4 yrs) 53 Normal (F:0; 20.4±1.5 yrs)	Members of the rugby academies in Ireland	*Normal(1); Abnormal*: (2) small areas of cystic degenerative change and hypoechoic regions < 3mm; (3) Macrocalculi over 3 mm or hypoechoic areas <8 mm; (4) Thickened tendon, areas of degenerative change >8mm	VISA-P
Edwards et al. 2010[141]	Cohort	14 Total 7 PTA (F:0; 25.2±4.7 yrs) 7 Controls(F:0; 22.3±2.4 yrs)	Asymptomatic athletes from sports involving repetitive landing	*Normal; Abnormal*: presence of hypo-echoic region, or fusiform swelling	Stop-jump landing (kinetic, kinematics)

Table 4.1 Continued.

Study	Study Design	Participants Characteristics	Population	US Classification/ Measurement	Functional Outcome(s)
Fazekas et al. 2018[129]	Cross-Sectional	31 Total (F:18; 21.4 ± 1.3 yrs)	Asymptomatic elite jumping athletes	*Normal; Abnormal:* presence of micro-calculi/calculi and/or hypoechoic regions	VISA-P
Gaida et al. 2004[130]	Cohort	39 Total 8 Unilateral PTA: (F:8; 20±2 yrs) 7 Bilateral PTA (F:8; 21±3 yrs) 24 No PTA (F:8; 21±3yrs)	Basketball athletes	*Normal; Abnormal*: presence of hypoechoic region	VISA-P; Vertical jump; Sit and reach; Isokinetic quadriceps strength
Hoksrud et al. 2008[131]	Cohort	63 Total 10 No PTA (F:NR; 23.8±5.6 yrs) 16 Grey-scale PTA (F:NR; 25.9 ±5.2 yrs) 37 Grey-scale PTA& Neovascularization (F:5; 24.6±6.0 yrs)	Elite athletes with clinically confirmed jumpers knee	*Grey-scale:* (1)Normal; (2)Abnormal: localized widening, including irregular structure and hypoechoic areas *Neovascularization*: 1 or several vessels inside the area with structural changes	VISA-P; SLDS with VAS
Kulig et al. 2015[142]	Cross-Sectional	18 Total 9 PTA (F:0; 25.9 ± 6.2 yrs) 9 Control (F:0; 23.1 ± 7.3 yrs)	Elite volleyball athletes with patellar tendinopathy and controls	*Normal; Abnormal:* presence of increased tendon thickness, hypoechoic regions, and/or neovascularization	Spike jump landing (kinetics, kinematics)
Mann et al. 2013[132]	Cohort	20 Total (F:0; 17.7±1.5 yrs) 10 Healthy 10 PTA	Asymptomatic junior basketball athletes	*Normal*; *Abnormal:* presence of hypo-echoic region, or fusiform swelling *Severity:* Echogenicity CSA (mm^2)	VISA-P; Stop jump landing (Kinetics, Kinematics); Lower Extremity Flexibility; Maximum Vertical Jump

Table 4.1 Continued.

Study	Study Design	Participants Characteristics	Population	US Classification/ Measurement	Functional Outcome(s)
Mendonça et al. 2016a[133]	Cross-sectional	43 Total (F:5; 24.8 ± 6.7 yrs) 15 PTA 32 Normal	Volleyball, soccer, and running athletes	*Normal*; *Abnormal*: Presence of hypoechoic areas	VISA-P SLDS
Mendonça et al. 2016b[134]	Cross-sectional	31 Total (F:0; 25.26±6.60 yrs) PTA: 8 No PTA: 23	Active male basketball and volleyball athletes	*Normal*; *Abnormal:* A hypoechoic region evident on both the longitudinal and transverse scans	VISA-P; Lower extremity ROM, alignment and strength
Ooi et al. 2016[135]	Cohort	35 Total (F:20; 22.2 ±3.1 yrs) 47 PTA 42 Soft	Volleyball athletes	*Greyscale:* (1)Normal; (2)Abnormal: presence of hypo-echoic region >2mm, or fusiform swelling *Elastography:*(1)Hard (Elastic): blue and/or green colour strain map (2)Soft (Less Elastic): yellow and/or red colour strain map	VISA-P
Pietrosimone et al. 2021[143]	Cohort	43 Total 15 Asymptomatic PTA (F:0; 21.13±1.88 yrs) 13 Symptomatic PTA (F:0; 19.62±1.61 yrs) 15 Control (F:0; 19.60±1.55 yrs)	Physically active males	*Normal; Abnormal:* A hypoechoic region >2 mm evident on both the longitudinal and transverse scans	Jump landing (kinetic, kinematics)
Scattone et al. 2017[136]	Cross-Sectional	21 Total 7 Symptomatic PTA (F:0; 18.0±1.15 yrs) 7 Asymptomatic PTA (F:0; 21.0±5.16 yrs) 7 Control (16.29±1.38)	Volleyball basketball athletes	*Normal; Abnormal:* presence of hypo-echoic region	VISA-P; VAS; Drop landing (kinetic, kinematics)

Table 4.1 Continued.

Study	Study Design	Participants Characteristics	Population	US Classification/ Measurement	Functional Outcome(s)
Sprague et al. 2022[121]	Cross-sectional	41 Total (F:15; 37[13.0]** yrs)	Individuals with patellar tendinopathy	Maximum Thickness (mm); Average CSA (mm); Shear Modulus (kPa)	CJM; Quadriceps MVIC;VISA-P
Wearing et al. 2015[137]	Cross-Sectional	19 Total 9 PT(F:NR; 17.8 ±0.8 yrs) 10 Control (F:NR; 18.2 ±0.7)	Volleyball players with patellar tendinopathy and controls	*Qualitative Vascularity* (0) no vessels present within the tendon (1-4) one to four vessels present within the tendon, respectively *Quantitative Vascularity* Power Doppler area	VISA-P
Zhang at al. 2020[138]	Cross-Sectional	43 Total(F:24; 16-26 yrs*) 31 PT 12 Control	Volleyball athletes with patellar tendinopathy and healthy matched controls	Thickness (mm); CSA (mm); Stiffness*:* (kPa; Young's modulus up to a maximum of 600 kPa)	VISA-P
Zhang et al. 2014[139]	Cross-Sectional	33 Total 13 PT (F:0; 22.9±4.6 yrs) 20 Control (F:0; 24.9±4.4 yrs)	Volleyball, basketball athletes with patellar tendinopathy and controls	Thickness ratio: (mm); CSA ratio: (mm^2); Stiffness ratio: (ratio between painful and non-painful sides, Young's modulus [kPa])	VISA-P

PT= Patellar Tendinopathy; PTA= Patellar Tendon Abnormality VAS= Visual Analog Scale; ROM= Range of motion; CSA= Cross-sectional area; CMJ= Counter Movement Jump; SLDS
*Range Reported; **Median and Interquartile Range Reported

Table 4.2 Study quality evaluation scores per the STROBE.

Study	Title & Abstract	Background	Objectives	Study Design	Setting	Participants	Variables	Data Sources	Bias	Study size	Quant Variables	Statistics	Participants	Descriptive Data	Outcome Data	Main Results	Other Analyses	Key Results	Limitations	Interpretation	Generalizability	Funding	Total
Arias-Buría et al. 2019[123]	1	1	1	0	0	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1
Benítez-Martínez et al. 2020[124]	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Cook et al.2000[65]	1	1	1	1	1	1	1	1	0	0	1	0	1	0	1	1	1	1	0	1	1	1	1
Cook et al 2004a[140]	1	1	1	0	1	0	1	1	1	0	1	1	1	1	1	1	1	1	0	1	1	0	1
Cook et al. 2004b[125]	1	1	1	1	1	0	1	1	1	0	1	1	1	0	1	1	1	1	0	1	1	0	1
Dirrichs et al. 2018[126]	1	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	0	2
Docking et al. 2020[127]	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	2
Durcan et al.2014[128]	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	2
Edwards et al.2010[141]	1	1	1	0	0	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1
Fazekas et al.2018[129]	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Gaida et al. 2004[130]	1	1	1	0	1	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	0	1
Hoksrud et al. 2008[131]	1	1	1	1	1	1	1	1	0	0	1	1	1	1	1	1	1	1	0	1	1	0	1
Kulig et al. 2015[142]	1	1	1	0	0	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	0	1
Mann et al. 2013[132]	1	1	1	0	0	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1
Mendonça et al. 2016a[133]	1	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	0	2
Mendonça et al.2016b[134]	1	1	1	1	0	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1
Ooi et al. 2016[135]	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Pietrosimone et al. 2021[143]	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Scattone et al. 2017[136]	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Sprague et al. 2022[121]	1	1	1	1	0	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1
Wearing et al. 2015[137]	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
Zhang at al. 2020[138]	1	1	1	0	0	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	0	1
Zhang et al. 2014[139]	1	1	1	1	0	1	1	1	0	0	1	1	1	1	1	1	1	1	1	1	1	1	1

Table 4.3 Summary of subjective outcome findings for included studies.

Study	Outcome Type	VISA-P Score, mean±SD			Group Classification Or Measurement	Findings
		Cohort	No PTA	PTA		
Arias-Buría et al. 2020[123]	VISA-P	NR	NR	42.1 ± 4.3	Thickness (mm); Width (mm); CSA (mm^2); Vascularity (1-5)	No significant linear association between ultrasound measures of thickness (rho=-0.145, p=0.543), width (rho=0.009, p=0.970) or CSA(rho=-0.411,p=0.873) and VISA-P, respectively. No significant linear association between neovascularization and VISA-P scores (rho=NR).
Benítez-Martínez et al. 2020[124]	VISA-P	93.6±10.4	94.6 ± 9.1	Hypoechogenicity: 96.4 ± 7.7 Thickening: 93.5 ± 10.5 Grey-scale & vascular PTA: 82.9 ± 14.3	Abnormal Thickening; Hypoechogenicity; Neovascularization	Significant differences in VISA-P scores (p=0.001) between tendons with different patterns of abnormalities. VISA-P mean scores for tendons with focal thickness, hypoechogenicity, and neovascularization were significantly lower than tendons with only focal hypoechogenicity or hypoechogenicity and thickening. Presence of neovascularization was the only abnormality significantly associated with VISA-P scores (p <0.01)
Cook et al. 2000[65]	VISA-P	NR	93.1 ± 5.8	94.5 ±6.0	Normal; PTA Unilateral; PTA Bilateral	VISA-P score at baseline for those with a PTA had a mean difference of 1.4 (95%CI -2.72,5.52) compared to controls.
Cook et al. 2004b[125]	VISA-P SLDS Test with VAS	NR	100 (82.5-100)*	Grey-scale: 87 (76-100)* Grey-scale & vascular PTA: 78 (62-96)*	Normal; Grey-scale PTA; Neovascularization Hypoechoic CSA (mm^2)	Those with tendons with neovascularization had significantly lower VISA-P scores (p = 0.045) and significantly more pain during the decline squat than abnormal tendons without neovascularization (p =0.048).CSA of hypoechoic area in abnormal tendons without neovascularization was not significantly correlated with pain on the decline squat. (rho=0.222; p= 0.07)
Dirrichs et al. 2018[126]	VISA-P	38 ± 15	NR	NR	Grey Scale (Normal/Abnormal) Neovascularization (Normal/Abnormal) Tendon Stiffness (Normal/Abnormal)	Clinical VISA-P, VISA-P, and DASH scores and had a weak association (r=0.24) with abnormalities identified through gray-scale US and correlated moderately with power doppler US findings(r=0.59). Clinical scores were strongly correlated with tendon stiffness via shear wave elastography (r=0.80).

Table 4.3 Continued.

Study	Outcome Type	VISA-P Score, mean±SD			Group Classification Or Measurement	Findings
		Cohort	No PTA	PTA		
Docking et al. 2020[127]	OSTRC-OQ	NA	NA	NA	Greyscale: (Normal / Abnormal): Tissue Characterization: (I-IV) Tendon diameter (mm); Percentage of echo types (%); CSA of echo types (mm^2); Total CSA (mm^2)	No significant differences were found for OSTRC scores at baseline between those with a mean CSA of aligned fibrillar structure above, within, or below the IQR of structurally normal tendons (p=0.347).
Durcan et al. 2014[128]	VISA-P	NR	94.8 ± 11.9	87.4 ± 17.5	Normal; PTA	Participants with a normal US reported higher function via VISA-P scores in comparison to those with PTA. (p=0.046)
Fazekas et al. 2018[129]	VISA-P	NR	93.5 ± 6.4	80.8 ± 8.6	Normal; PTA	Statistically lower VISA-P values were observed in those that had PTA with the right knee (p=0.003, ES= 1.67) but not in the left knee (p=0.250, ES=0.512) although both with moderate to large effect sizes. Presence of the hypoechoic areas among the asymptomatic population was 19.4% (right knee) and 29.0% (left knee).
Gaida et al.2004[130]	VISA-P	NR	90 ± 10	Unilateral: 83 ± 18 Bilateral: 72 ± 23	Normal; Unilateral PTA; Bilateral PTA	Participants with a bilateral PTA had significant lower VISA-P scores than healthy controls but not those with unilateral PTA.
Hoksrud et al. 2008[131]	VISA-P SLDST VAS	57 ± 15	NR	NR	Normal; Grey-scale PTA; Grey-scale PTA & Neovascularization	Participants with Grey-scale changes and neovascularization reported lower VISA-P scores and higher pain levels during the SLDST at baseline than those with Grey-scale tendon changes alone or normal tendons (ANOVA, p = 0.001; ANOVA, P = 0.001, respectively). No significant difference in VISA-P or VAS after SLDST between those with only grey-scale PTA and controls.

Table 4.3 Continued.

Study	Outcome Type	VISA-P Score, mean±SD			Group Classification Or Measurement	Findings
		Cohort	No PTA	PTA		
Mann et al. 2013[132]	VISA-P	NR	95.3 ± 4.9	90.8 ± 13.3	Normal; PTA Severity: Echogenicity CSA(mm^2)	Multiple regression model did not indicate that VISA-P scores were substantial predictors of PTA. VISA-P scores (R^2 = 0.392) and hip ROM (R^2 = 0.124; standard error 12.33) were significant predictors of PTA severity.
Mendonça et al. 2016b[134]	VISA-P	NR	91.90 ± 13.00	77.00 ± 21.50	Normal; PTA	VISA-P significantly lower in those with tendon abnormalities (p=0.011)
Mendonça et al. 2016a[133]	VISA-P	NR	92.5 ± 12.7	77.3 ± 23.5	Normal; PTA	VISA-P questionnaire for athletes with PTA was significantly lower than those without PTA (p = .008; mean difference,–15.24; 95% CI: –26.37, –4.10). The CART analysis selected only the SLDS, tendon pain history, and VISA-P scores as predictors of PTA.(p= .001, 95% CI: 66, 94).
Ooi et al. 2016[135]	VISA-P	NR	Greyscale: 87 ± 11.9 Hard: 89 ± 13.6	Grey scale: 79 ± 18.3 Soft: 77 ± 17.3	Grey Scale US (Normal/Abnormal) Tendon Stiffness (Normal/Abnormal)	No significant difference in VISA-P scores between those with and without grey scale US abnormalities (p=0.098). Patellar tendons categorized as abnormally soft had statistically significantly lower VISA-P scores (p=0.004) than those categorized as normal.
Scattone et al. 2017[136]	VISA-P VAS	NR	96.43 ± 6.13	Asymptomatic: 94.29 ± 7.72 Symptomatic: 69.86 ± 10.79	Normal; Abnormal	Those with clinically confirmed tendinopathy and PTA had significantly lower VISA-P scores and higher reported VAS than those with asymptomatic PTA and controls (p=0.001).
Sprague et al. 2022[121]	VISA-P	60.0 ± 24.0	NR	NR	Thickness (mm); Average CSA (mm); Stiffness (kPa)	No significant relationship was found between VISA-P scores and tendon morphology (p=0.81, R^2 change = 0.012) or mechanical properties (p=0.19, R^2=0.099) after adjusting for age, sex, and BMI.

Table 4.3 Continued.

Study	Outcome Type	VISA-P Score, mean±SD			Group Classification Or Measurement	Findings
		Cohort	No PTA	PTA		
Wearing et al. 2015[137]	VISA-P	70 ± 11	NR	70 ± 11	Qualitative Vascularity: (0-4) Quantitative Vascularity: Power Doppler area	Power doppler area moderately correlated with VISA-P score (r=0.58, p=0.05).
Zhang at al. 2020[138]	VISA-P	NR	NR	NR	Thickness (mm); CSA (mm^2); Stiffness: (kPa)	Small yet significant negative correlation between thickness and VISA-P scores (r=0.307, p = 0.034) and CSA and VISA-P scores (r=0.382, p=0.007). Strong negative correlation between the elastic modulus in the patellar tendon and VISA-P scores at baseline r=-0.784, p<0.001)
Zhang et al. 2014[139]	VISA-P	*NR*	*NR*	*≤ 80 per inclusion*	Thickness: (mm); CSA: (mm^2); Stiffness: (kPa)	No significant relationships were detected between thickness and CSA and VISA-P scores. Significant negative association between elastic ratio and VISA-P total score (rho=-0.61, p=0.026) .

VISA-P=, NR= Not reported
*Reported Median (Interquartile Range)

Table 4.4 Summary of objective functional outcome findings for included studies.

Study	Functional Outcome Type	Group Classification or Measurement	Findings
Cook et al. 2004a[140]	Agility run; 20m shuttle; Maximum vertical jump; 10m Sprint; Sit & reach	Normal; PTA	No significant group differences for agility, shuttle, or sprint test results ($p>0.05$). Females with PTA had higher vertical jump distances than those with normal tendons ($p<0.039$) but no differences were found between groups in male participants ($t=0.148$, $p=0.883$). Females with unilateral PTA had significantly lower scores on the sit and reach than females without abnormalities ($p=0.01$) and females with bilateral PTA ($p=0.004$). Males with bilateral PTA had significantly reduced sit and reach scores compared to males with no tendon abnormalizes ($p=0.03$).
Edwards et al. 2010[141]	Static dorsiflexion, Stop jump landing (Kinetics, Kinematics)	PTA, Control	No significant differences in patellar tendon loading or ground reaction force variables during vertical landing. PTA group had significantly lower vertical ground reaction force loading rate than controls ($p=0.03$) during vertical landing phase. No significant differences in patellar tendon loading or ground reaction force variables during horizontal landing. Those with PTA landed with a significantly different kinematic strategy than controls during the horizontal landing phase displaying greater knee flexion at initial contact and less hip flexion then continuing to extend hip joint throughout landing.
Gaida et al. 2004[130]	Max CMJ; Sit & reach; Isokinetic quadriceps; strength	Normal; Unilateral PTA; Bilateral PTA	No significant differences between any of the groups in any of the measured functional outcome variables.
Kulig et al. 2015[142]	Spike jump landing (kinetics, kinematics)	Symptomatic PTA; Control	Significantly higher lower extremity contact angle in controls compared to PTA group (p-0.04, ES=1.06). Significantly lower peak ankle dorsiflexion coefficient of variation in PTA group compared to controls (p-0.01). No significant group differences in any other ankle, knee or hip kinematics variables. No significant group differences were found for peak vertical or breaking ground reaction force and vertical or braking impulse during early or late stages of landing. Higher breaking impulses were strongly correlated with lower values of lower extremity contact angle during early landing phase and moderate correlated during the late stages of landing ($r=-0.890$, $r=-0.660$).
Mann et al. 2013[132]	Stop jump landing (Kinetics, Kinematics); Lower Extremity Flexibility; Maximum Vertical Jump	Normal; PTA Severity: Echogenicity CSA(mm^2)	Multiple regression model indicated that negative hip joint ROM ($R^2=0.474$), greater knee flexion at IC ($R^2=0.112$), and reduced quadriceps flexibility ($R2 = 0.090$) were significant predictors of PTA. Significant predictors of PTA severity were VISA-p scores ($R^2=0.392$) and negative hip ROM ($R^2=0.124$).

Table 4.4 Continued.

Study	Functional Outcome Type	Group Classification or Measurement	Findings
Mendonça et al. 2016b[134]	Lower extremity ROM, alignment and strength	Normal; PTA	Frontal plane patellar medial rotation, shank foot alignment, and iliotibial band flexibility were significantly related to the presence of PTA (p=0.046, p=0.013; p=0.006, respectively). Hip strength, ankle dorsiflexion ROM, McConnell angle, frontal plane knee projection angle and passive internal ROM were not significantly related to the presence of PTA.
Pietrosimone et al. 2021[143]	Jump landing (kinetic, kinematics)	Symptomatic PTA; Asymptomatic PTA; Control	Those with asymptomatic PTA exhibited smaller knee-flexion angles than the control group throughout most of the stance phase during the landing task (Cohen d=1.14, Cohen d=0.99). The asymptomatic PTA group also demonstrated smaller knee-flexion angles than the control group during the early and late landing stages of the stance phase (Cohen d=0.96). There were no significant group differences in sagittal-plane knee angle between those with symptomatic PTA and asymptomatic PTA. Symptomatic PTA group exhibited significant decreased in knee extensor moment, patellar tendon force, and knee power during various phases of the landing task compared to other groups. Those with asymptomatic PTA did not display significant differences in kinetic variables in comparison.
Scattone et al. 2017[136]	Drop landing (kinetic, kinematics)	Symptomatic PTA; Asymptomatic PTA; Control	Symptomatic PTA group exhibited smaller peak knee extensor moment compared to asymptomatic PTA group (p=0.0029, ES=1.77). Those with symptomatic PTA produced lower patellar tendon force than those with asymptomatic PTA (p-0.045, ES-0.98). Those with symptomatic PTA had on average less peak dorsiflexion compared to the control group (p-0.055, ES=1.21).
Sprague et al. 2022[121]	Max CMJ; Quadriceps MVIC	Maximum Thickness (mm); Average CSA (mm); Shear Modulus (kPa)	A significant positive relationship between CMJ height and tendon thickness ($p < 0.001$, $\beta = 0.718$) and a significant negative relationship between CMJ height and CSA ($p = 0.001$, $\beta = -0.538$) was identified after adjusting for age, sex, BMI, and pain levels

PT= Patellar Tendinopathy; PTA= Patellar Tendon Abnormality VAS= Visual Analog Scale; ROM= Range of motion; CSA= Cross-sectional area; CMJ= Counter Movement Jump

Subjective Outcomes of Self-Reported Function

A majority of the included studies that sought to understand the relationship between tendon morphology and function categorized participants as normal or abnormal based on certain ultrasound parameters. Primarily, PTA was defined by abnormal findings via four different ultrasound evaluation types such as greyscale, power doppler, shear wave elastography (SWE), and ultrasound tissue characterization (UTC). The most prominently used evaluation mode was grey scale ultrasound[123,125,126,132,133] followed by power doppler,[123–126,131,137,138,142] SWE,[121,126,135,138,139] and UTC.[127] The parameters used to define PTA are described in Table 5.1. Grey scale ultrasound was primarily used to identify focal echogenicity or focal thickening measured through grey scale (B-mode) ultrasound, tendon neovascularization was primarily measured via power doppler and decreases in tendon stiffness were assessed by shear wave elastography.

Of the 22 included studies, 19 evaluated self-reported function.[65,121,123–139] The VISA-P was used to evaluate self-reported function in all studies except one which used the OSTRC-OIQ. Additionally, three studies assessed pain using a Visual Analog Scale in addition to the VISA-P. Scattone et al. administered a VAS to participants instructing them to describe their level of pain from the previous week whereas two other studies instructed patients to report their pain using a VAS following a single leg decline squat test.[136]

Although a majority of the studies were consistent in the use of VISA-P to report self-reported function, the methods for measuring or categorizing tendon abnormality and characteristics of included participants were inconsistent. Included studies had differences in inclusion and exclusion criteria for participants. Some studies included only symptomatic participants with a clinical diagnosis of patellar tendinopathy while others included those

asymptomatic with PTA. Additionally, the statistical approach to describe the impact of PTA on lower extremity function differed among the included studies. Eight of the 19 included studies assessed the relationship between abnormalities and pain related function through correlational analyses whereas the others only assessed group differences.[121,123,125,126,132,133,137–139]

Focal Hypoechogenicity and Thickening

Studies that assessed gross measurements of tendon thickness, CSA, or width found that these measurements were not strongly associated with VISA-P Scores.[121,123,138,139] Conversely, Zhang et al. (2020) reported thicker tendons and those with larger CSA are associated with lower VISA-P scores ($r=0.307$, $p = 0.034$; $r=0.382$, $p=0.007$, respectively).[138] However, both correlations were of small magnitude, thus indicating a weak relationship between grey-scale morphology and self-reported pain with function. Additionally, a majority of the included studies that evaluated the relationship between the presence of PTA (abnormal or normal) and VISA-P scores found that there was no meaningful relationship between the two.[124,126,132] Only one study found that VISA-P scores were a significant predictor of PTA, but ultimately in application of the model found that the inclusion of VISA-P scores did not provide any significant clinical implications.[134] The size of the PTA also seems to have little relationship to pain related function. Cook et al. found no significant correlation between the CSA of the focal hypoechoic area in abnormal tendons and pain reported on through a VAS following a single leg decline squat test.[125] Overall, it seems that there may be little correlational relationship between greyscale abnormalities and measures self-reported function, but other ultrasound assessment parameters may be able to identify factors related to pain with function.

Included studies are not in agreement regarding whether group differences exist between those with patellar tendon and PTA identified through grey-scale ultrasound. As anticipated

participants with clinically confirmed symptoms of patellar tendinopathy and PTA had significantly lower VISA-P scores than those with seemingly asymptomatic PTA and controls.[131,143] To thoroughly understand whether those with PTA reported lower values of self-reported function, studies that did not include participants based on previously reported signs and symptoms were reviewed. Studies that screened cohorts of athletes for PTA reported inconsistent results regarding group differences in self-reported function between those with PTA and normal tendons.[125,128,130,133–135] In studies that identified significant group differences in VISA-P scores, the PTA group reported mean scores greater than 70 which would still be deemed subclinical.[128,130,133,134] Furthermore, cohort studies such as Fazekas et al. identified the presence of grey-scale PTA in roughly 30% of jumping athletes with no reported clinical symptoms suggesting that thickening and hypoechoic areas of the tendon may not be the primary driver of pain during functional activity.[129]

Neovascularization

Neovascularization within the patellar tendon may have a stronger relationship with pain during function than other structural changes observed through grey-scale ultrasound assessment. A moderate relationship between the presence of neovascularization and VISA-P scores was observed within two of the included studies.[126,137] However another study found conflicting results suggesting there was little to no relationship.[123] All three studies used the appearance of vessels within then tendon to define a tendon as abnormal or normal, however differences could be attributed to different patient populations as well as the devices and device settings used. When those with neovascularization present within the tendon were compared to individuals with only focal tendon and hypoechoic changes, those with neovascularization tended to have higher reported lower extremity function.[124,125,131] Therefore, the presence of multiple

sonographic abnormalities may be more likely to have lower rating of function. All studies that investigated group differences in self-reported pain between those with neovascularity as opposed to only thickening or echogenicity changes found that those with neovascularization present within the tendon had significantly higher measures of self -reported pain of the VISA-P.[124,125,131] Additionally, those with tendon neovascularization had significantly more pain following a decline squat than abnormal tendons without neovascularization.[125,131] Although studies were able to identify group difference between those with and without neovascularization, correlational analyses from other studies indicate these identifiers may not be able to describe the severity of pain with function but highlight differences between wider populations. It may be that there are other unfound factors that affect individual patterns of pain and function.

Tendon Stiffness& Tissue Characterization

There is moderate consistent evidence to suggest that tendon stiffness is related to self-reported function. In total there were four studies that investigated the relationship between tendon stiffness via shear wave elastography on ultrasound and VISA-P scores. Three of the four were in agreement that individuals with softer tendons, thus less elastic, tended to have significantly lower VISA-P scores. Thus, these results indicate that the stiffness of the patellar tendon may relate to pain during functional activity.

The relationship between tissue characterization and self-reported outcomes were only investigated in one of the included studies.[127] Docking et al identified that those with patellar tendon abnormalities did not have significantly lower mean CSA of aligned fibular structure in comparison to healthy tendons. Additionally, abnormal tendons with varying levels of aligned

fibular structures did not have significantly different levels of self-reported function and pain via the Oslo Sports Trauma Research Center Overuse Injury Questionnaire (OSTRC-OIQ).

Objective Functional Outcomes

Nine articles were identified that met inclusion criteria and investigated the relationship between patellar tendon abnormality via ultrasound imaging and objective lower extremity function.[121,130,132,134,136,140–143] Included articles assessed a number of functional outcome measures but displayed little uniformity in the type of outcome and the methodology.

Maximum Vertical Jump

There is limited and inconclusive evidence to support a relationship between patellar tendon morphology and maximum vertical jump.[121,130,132,140] Four of the included studies assessed the relationship between maximal vertical jump and tendon morphology. Two studies reported significant findings but were inconsistent across sex and in the direction of the relationship. Greater jump distances were reported in females with patellar tendon abnormalities but not males in comparison to those with healthy tendons.[140] Additionally, Sprague et al.[121] reported there were significant relationships between tendon thickness and CSA and vertical jump height, respectively. However, CSA and tendon thickness displayed opposite relationships with jump height suggesting those with decreased tendon thickness and increased CSA displayed lower vertical jump heights.[121] The two remaining studies refuted the previous findings by not finding any significant relationship between tendon abnormalities assessed via grey scale ultrasound and maximum vertical jump.[130,132]

Jump Landing Kinematic and Kinetics

Limited evidence is available to assess the relationship between patellar tendon abnormality and landing mechanics. Five studies were identified in the literature review that

evaluated kinetic and kinematic variables during a landing task in those with patellar tendon abnormality. The studies used a variety of different landing tasks, variables of interest, and analyses methods to evaluate landing strategies in this population. Additionally, there are key differences in the characteristics of the populations recruited for the included studies due to the inclusion and exclusion criteria implemented. Four articles included individuals with asymptomatic patellar tendon abnormality and three included individuals with clinical symptoms of patellar tendinopathy along with patellar tendon abnormality.[132,136,141–143]

There is moderate consensus amongst the results to suggest that individuals with asymptomatic PTA display similar landing pattens during vertical landing tasks in comparison to healthy controls. Two studies found significant differences in sagittal plane kinematics but they were not consistent with one reporting smaller knee flexion angles during the early and late phases of landing in comparison to controls and another reporting significantly higher velocities of hip flexion at peak vertical ground reaction force.[141,143] Furthermore, Scattone at al. [136] found no significant differences in hip, knee, or ankle angles at initial contact or peak range of motion. None of the included studies identified any significant differences in kinetic variables between those with asymptomatic PTA vs controls or symptomatic PTA during vertical landing tasks.[132,136,143] Therefore, those with asymptomatic PTA do not seem to exhibit any significant differences in loading during vertical landing that may predispose these individuals to patellar tendinopathy.

Despite similar landing patterns for vertical tasks, there is limited but consistent evidence that implies that those with asymptomatic PTA may use different kinematic strategies than healthy controls during horizontal landing tasks.[132,141] Edwards et al.[141] and Mann et al.[132] found that those with asymptomatic PTA exhibited greater knee flexion and less hip flexion at initial

contact followed by increasing hip extension through throughout the horizontal landing portion of during a stop-jump landing task. Although these studies found significant differences in sagittal plane kinematics during landing, they did not find significant difference in loading. Hence, those with asymptomatic PTA may be using different strategies to attenuate force during horizontal loading than healthy controls.

Two of the three studies investigating landing patterns in those with symptomatic PTA found significant differences in kinetic variables during landing tasks between those with symptomatic PTA versus controls or those with asymptomatic PTA.[136,143] Pietrosimone et al.[143] and Scattone et al.[136] reported that those with symptomatic PTA produced lower patellar tendon forces and knee extensor moments during landing tasks. Both studies concluded that this is consistent with a load avoidance strategy to limit strain on the patellar tendon. However, another study investigating lower extremity kinematic and kinetic patterns during a spike jump maneuver in volleyball athletes did not identify any significant differences in peak vertical or breaking ground reaction force and vertical or braking impulse during early or late stages of landing.[142]

Although there was some agreement regarding the loading strategy exhibited by those with symptomatic PTA, differences in assessment technique and included kinematic variables make it difficult to synthesize evidence whether a different kinematic strategy exists among those with symptomatic PTA. All studies identified decreases in sagittal plane flexion in those with symptomatic PTA versus controls however they differed in terms of which joint these differences were observed.[136,142,143] An assessment of sagittal knee motion during a jump landing task identified smaller knee flexion angles throughout the stance phase, thus less knee flexion excursion, in those with symptomatic PTA compared to controls (Cohen d =1.14).[143] Conversely, during a drop landing, Scattone and colleagues[136] found no differences in initial contact or peak

knee flexion angle between symptomatic or controls but did find significant difference in peak ankle dorsiflexion in those with symptomatic PTA.[136] Additionally, another study using a volleyball spike jump maneuver found so significant differences in joint angles at initial or peak joint angles between symptomatic participants and controls.[142] In summary there was little consensus on kinematic or kinetic strategies in those with confirmed symptomatic PTA.

Lower Extremity Mobility

Five studies were identified that assessed lower limb flexibility in those with PTA.[130,132,134,140,141] The most common outcomes reported were sit and reach and static dorsiflexion range of motion. A study amongst junior basketball players found inconsistent results regarding sit and reach scores amongst bilateral and unilateral groups with PTA versus controls.[140] Gaida et al.[130] did not identify any differences in sit and reach scores amongst those with and without PTA. Static dorsiflexion differences also were not identified between those with PTA and controls in any of the studies in which it was investigated.[132,134,141] Finally, quadriceps flexibility was only investigated in one study in which it was found to be a significant predictor of the presence PTA but not severity in junior basketball players. There is little existing evidence explaining the relationship between lower extremity mobility and the presence of PTA.

Additional Outcomes

There were a handful of lower extremity functional outcomes that were only investigated in one of the included studies and thus has limited supporting evidence. Quadriceps strength was not a significant predictor of the presence of PTA or severity amongst those with patellar tendinopathy. Additionally, Mendonca et al.[134] identified that patellar alignment, shank foot alignment and iliotibial band flexibility were related to the presence of PTA in volleyball athletes.

4.4 Discussion

Numerous studies have been published which have evaluated the relationship between painful symptoms, function, and patellar tendinopathy. Based on the current review of available literature there is conflicting evidence as to whether a relationship exists between objective and subjective measures of lower extremity function and PTA. The inconsistent results observed amongst the studies may be due to high heterogeneity of methodology and patient characteristics.

Although there is some evidence to support the relationship between certain intratendinous changes and function many of the studies used varying techniques and outcomes making synthesis difficult. Not surprisingly, those participants with clinically diagnosed patellar tendinopathy reported higher VISA scores than those with asymptomatic PTA controls.[131,143] Studies that screened participants for PTA, irrespective of clinical diagnosis, found contradictory findings regarding group differences in self-reported function between those with and without tendon abnormality. Thus, all individuals with PTA may not exhibit signs and symptoms of patellar tendinopathy. It may be that these individuals are exhibiting subclinical features of patellar tendinopathy. A few studies in the current review found significant differences in VISA-P scores between those with tendon abnormalities without clinically diagnosed patellar tendinopathy versus controls, despite the mean scores for the abnormal group being above the clinical threshold of 75 that is typically deemed as symptomatic. These results may be indicating that these individuals fall along the early stages of the continuum described by Cook et al.[15] Although not all tendons with tendon abnormalities progress to a degenerative state, tendon abnormalities have been shown to be an indicator of increased tendinopathy risk.[70] Recent pooled findings show that individuals with patellar tendon abnormality may be six time more

likely to be diagnosed with patellar tendinopathy in the future.[70] The ultrasound parameters used to define whether a patellar tendon was abnormal may account for variations in findings.

Different types of intratendinous changes seem to have varying relationships with self - reported function. Focal tendon thickening and echogenicity seem to have a weaker relationship with self-reported pain and function than other parameters such as vascularity, tendon stiffness, and tissue characterization.[124–126,131,135,138,139] Two studies identified significant relationships between neovascularization within the patellar tendon and self-reported pain.[126,137] Additionally, those who presented with tendon neovascularization tended to report more pain and functional impairments than those with focal thickening and/or focal echogenicity.[124,125,131] Previous research has also found that the pooled presence of neovascularization in asymptomatic individuals is much lower compared to the presence of thickening and or focal echogenicity.[74] It is still unclear as to whether vascular changes are a sign of the progression of the degenerative changes of the tendon or a key source of pain. Studies which have identified vascularity changes in individuals without pain suggest that the new blood vessels are not the primary source of pain.[123] Advances in power doppler technologies and the ability to quantify vascular changes may be able to increase our understanding of its role in pain and function in time with future studies. Ultrasound technology advancement have also brought new ways to characterize tendon changes.

Limited evidence is available to determine whether measurements of tendon stiffness or tissue characterization plays a key role in pain and function. The current review has found moderate evidence to suggest that those with soft, less elastic tendons tended to have decreased self-reported function than those with hard, more elastic, tendons. Furthermore, Dirrichs et al.[126] reported that 81.3% of tendons that had significantly improved per participant scores on the

VISA-P following treatment returned to normal tendon stiffness. Thus, tendon stiffness seems to provide useful information to guide clinical care and monitor progress and coincides with pain free function. Further studies using quantitative measures of tendon stiffness are needed to confirm this relationship in broader populations. Further evidence is also needed to access the relationship between tissue characterization. Docking et al.[127] found that the mean CSA of aligned fibrillar structure within the tendon did not have an influence on self-reported function in those with abnormal tendons. Interestingly, those with abnormal tendons did not have any significant difference in the mean CSA of aligned fibrillar structures despite having increases in total mean CSA. This supports previous hypotheses about the tendon continuum which suggest tendons can adapt to overloading by creating new tissues but that the damaged fibrillar structures may be unable to adapt after reaching late degenerative stages.[15] Due to the limited evidence on the relationship between ultrasound tissue characterization and self-reported function, the findings described should be considered with caution. Further research with consistent patient reported outcomes and quantitative assessments within various population will be needed to understand the relationship between structure and function.

Overall, the synthesis of results from the included studies confirms that not all individuals with the presence of PTA will report decreases in function. This is supported by a previous systematic review which evaluated the likelihood of patellar tendon abnormalities in participants that did not report knee pain.[74] The systematic review concluded the rate of patellar tendon abnormalities, identified via ultrasound, among participants ranged from 0 to 50%. Additionally, the rate of asymptomatic tendon abnormality was found to be higher among highly active individuals, especially those in sports with frequent jumping activity.[74] The high rate of tendon

abnormalities in individuals without pain shows that there must be additional factors that are driving self-reported pain and functional impairments in those with patellar tendinopathy.

However, despite limited evidence to suggest PTA is related to current signs and symptoms, a number of studies have identified the presence of PTA a predictor of future injury.[64,65,144] Therefore, there may be some link to structural change and long-term function. The tendon continuum hypothesis may provide a possible explanation for the relationship. The tendon continuum suggests that tendon change happened along a continuum and can adapt based on the adaptability of the tendon as well as the loading placed upon the tendon.[15]Those with asymptomatic PTA may have a tendon that is characterized as early stage reactive and may be able to adapt with rest and decreased loading back to a normal state; however, if no rest or continual or increasing load is sustained the tendon can become degenerative and may eventually be unable to adapt and return to uniform pattern. Thus, individuals in this stage exhibit chronic pain and may need a different form of treatment.

Results from this review indicate there is a lack of literature pertaining to the relationship between patellar tendon abnormalities and objective measures of function. Only nine articles were identified that met inclusion criteria. Additionally, there was little consistency in the objective assessments employed in the included studies which made it difficult for synthesis of the results and ultimate conclusions. Maximum vertical jump, landing biomechanics, lower extremity mobility were the most common assessments. Additionally, the majority of studies defined tendon abnormality by the presence of focal thickening and/or focal hypoechogenicity evaluated through grey scale ultrasound.

Based on the current review, there seems to be little to no evidence to support differences in maximum vertical jump height or lower extremity mobility between those patellar tendon

abnormalities and controls. Additionally, static dorsiflexion was not significant different between those with tendon abnormality and controls and any of the included studies. Mann et al.[132] found quadriceps flexibility to be significant predictor of the presence of tendon abnormality, although it was not evaluated in any other study, and therefore should be reviewed with caution. Future studies may want to include other clinical assessments to assess lower extremity function to see if deficits are a result of the experimental task or if there is no connection.

The relationship between biomechanical variables and patellar tendon abnormality may differ based on the experimental task as well as the presence of pain. Synthesis of the results proved problematic due to the differences in selected participants and experimental tasks. Two of the three included studies that specifically included participants with symptomatic patellar tendon abnormality found these individuals produced lower patellar tendon forces and knee extensor moments during vertical landing tasks.[136,143] The studies which identified these changes concluded that these movement patterns were consistent with a pain avoidance strategy. The identified loading pattern is consistent with results from other studies that evaluated landing strategies of participants with painful patellar tendinopathy but unconfirmed patellar tendon abnormality.[145] Sorenson et al.[145]identified volleyball athletes with a history of patellar tendinopathy exhibited 29% less energy absorption during a jump landing task compared to healthy controls. The load avoidance seems to be driven by pain since this loading pattern during landing was not identified in participants with asymptomatic PTA.

No key biomechanical variable was consistently found in those with asymptomatic PTA compared to either controls or those with symptomatic PTA. Inconsistent findings across studies were most likely due to key differences in the experimental task. Two studies which assessed a similar stop jump task found that asymptomatic individuals had a significantly different loading

strategy than controls.[132,141] These participants landed with increased knee flexion and decreased hip flexion at initial contact followed by an increase in hip extension throughout the horizontal landing.[132,141] The replication of results across these studies provide evidence to suggest that experimental tasks with a horizontal loading phases may be able to induce different landing strategies in those with asymptomatic PTA versus tasks with just a vertical landing component. Horizontal landings have been shown to place a higher load on the patellar tendon than vertical landing tasks.[146] Thus future studies may want to use experimental tasks with a horizontal component that is relevant to the cohorts typical movement patterns.

This systematic review has a number of limitations. An extensive search was conducted in databases that the authors believed would incorporate the largest amount of studies that captured our desired outcomes and population. Even though a systematic search was conducted along with hand searches of includes studies there still may be relevant articles that were not located. Additionally, due to the methodological differences between the included studies we were unable to conduct a meta-analysis. Finally, we refer to participants from included studies as asymptomatic or symptomatic based information regarding clinical evaluations reported within the studies. However, the clinical diagnosis parameters differed between studies, and thus participants described as symptomatic could be different in the clinical presentation. Due to the limited evidence and consistency across included studies we recommend that future research is conducted to explore the relationship between tendon characteristics and subjective and objective function.

4.5 Conclusion

This study will provide researchers and clinicians a thorough review of current evidence regarding patellar tendon structure and lower extremity function. Despite the many articles

describing how tendon characteristics relate to subjective function, especially in comparison to objective measures of function, the lack of methodological uniformity between studies makes it is difficult to come to strong conclusions. There is limited evidence to support a significant relationship between objective measures of lower extremity function and tendon abnormalities. Additionally, there is limited evidence of a change in biomechanical patterns during horizontal landing assessments, but further investigation will be needed. The inconsistencies in defining patellar tendon abnormalities and the varying outcomes used to assess lower extremity function in those with patellar tendon abnormalities highlights a need for consistent evaluation techniques and functional outcome measurements within future studies.

CHAPTER 5

PREDICTIBILITY OF PATELLAR TENDON ABNORMALITIES FOR FUTURE SYMTPOMS OF PATELLAR TENDONOPATHY IN U.S. MILITARY TRAINEES

5.1 Introduction

Musculoskeletal injuries (MSKIs) induced by chronic overload or a rapid increase in physical activity, such as tendinopathies, are common in military trainees and place a large burden on US Military operations and the Military Health Care System.[24,147] MSKI is the primary reason for lost duty and training time amongst active duty military members.[147] During a two year observation period in Air Force basic trainee recruits, the MSKI incidence rate was 3.39 per 1000 training days and placed a financial burden of $43 million in medical and operational costs on the Department of Defense (DoD).[24] Special Warfare trainees are at an even higher risk of MSKI during technical training compared to other career fields.[25,148,149] Between 2010 and 2020, 49% of United States Air Force (USAF) trainees attending the Tactical Air Control Party Apprentice Course sustained an MSKI.[25] The most frequent region of MSKI was lower extremity and the most commonly reported type was non-specific overuse injuries including non-specific pain and tendinopathy.[25] The high incidence rate among military trainees is attributed to the high load regiments among individuals in these population.

MSKI is a major reason of attrition among military training pipelines such as those conducted within the USAF Special Warfare Training Wing (SWTW). The SWTW houses the Special Warfare Human Performance Support Group (HPSG), which was established to optimize "physical and mental performance, reduce injury and speed rehabilitation to create more capable and resilient ground operators."[150] To improve performance and reduce injuries, there must first be a thorough baseline understanding of factors concerning the SWTW training environment.

Thus, identifying factors that attribute to potentially chronic conditions such as patellar tendinopathy should be of great interest.

Abnormal tendon structure may be a marker for future tendon injury such as patellar tendinopathy. A recent systematic review reported that those with patellar tendon abnormality, measured by ultrasound, have a six times greater risk of developing future symptoms of patellar tendinopathy.[70] The majority of studies included in the systematic review were collegiate and professional athletes. Further research is needed in this area to see if it is also an indicator of injury in other populations such as military trainees. Identifying risk factors associated with lower extremity injury is a high priority among military training installations.

Establishing key factors of MSKI, especially tendinopathy, can provide a starting point from which to identify candidates that may need further progressive loading prior to starting a rigorous technical training course within the USAF SWTW. Pain is usually the first indication of patellar tendinopathy and is typically the key indicator used for management following diagnosis.[151] Early identification may allow clinicians to implement load management practices before further tissue damage and pain regulation becomes necessary, thus mitigating extensive medical delays in training.

Additionally, measurements of patellar tendon health in trainees could aid in measuring the success of programs aimed at improving training efficiency, reducing attrition, illness and injury prevention, and other factors related to human performance in SWTW trainees. The primary aim of the study was to identify whether abnormal ultrasound findings place trainees at a higher risk of sustaining patellar tendinopathy or other lower extremity musculoskeletal injury during the SWTW candidate course. The secondary aim was to assess the relationship between ultrasound imaging parameters and lower extremity function. We hypothesized that those with

abnormal tendon properties prior to a 6-week training course will be at a higher risk of sustaining patellar tendinopathy and other MSKI during course.

5.2 Methods

Study Design

This study used a prospective cohort study design.

Population

All individuals were enrolled in a single class of the Special Warfare Candidate Course (SWCC) conducted by the SWTW. All enlisted, non-prior and prior service, airmen seeking to enter an SWTW pipeline must first complete the SWCC. All candidates that entered course, had available data, and did not have a previous diagnosed history of patellar tendinopathy in the previous 90 days were included. There were 71 individuals that were included in the study.

Procedures

This was an exempt study approved by the Old Dominion University Institutional Review Board. Information for this study was extracted from data that was collected by HPSG staff during a single SWCC, which was conducted at Lackland Air Force Base in San Antonio, Texas. The course is roughly 35 days and seeks to prepare students for the mental and physical rigors of the courses within the SWTW. Data was extracted from regular fitness tests, body composition assessments, and medical screenings completed the week prior to start of course. Ultrasound imaging findings of the patella tendon, countermovement jump heights and perceived knee function scores during these baseline assessments were analyzed. Additionally, cardiovascular aspects of the IFT were analyzed. The perceived knee function questionnaire, ultrasound assessment and countermovement jump assessments were completed intermittently throughout three days based on scheduling. The IFT was completed last and was done on the same day for

all candidates. Injury surveillance information 90 days prior to course to 7 days after course was obtained from local HPSG medical documentation as well as data extraction from military electronic medical records.

Ultrasound Evaluation

Ultrasound imaging was captured on candidates during the week prior to the start of the SWCC. Imaging was collected by medical personnel with a minimum of 10 hours of training and at least 3 years of experience. Ultrasound imaging of the patella tendon focused on tendon thickness, echogenicity, and vascularity. These three parameters were chosen based on a recent meta-analysis that identified that a three parameter approach was the most predictive of patellar tendinopathy.[120] Additionally, the ordinal criteria for each parameter is a modified version of a categorial model that aligns with the continuum of care model by Cook et al.[15]. The original categorical model was shown to have substantial intra-rater and inter-reliability in staging Achilles tendon abnormalities on ultrasound.[152]

Imaging was taken in the morning before any strenuous physical activity. Bilateral examination of patellar tendons was performed with a high-resolution portable ultrasound machine (Butterfy Model: iQ+, Butterfly Network Inc) in B-mode and Power Doppler mode. For each mode the MSK default setting was selected. The Butterfly iQ software did not allow for modification of any of the ultrasound settings except for gain and depth. The values of the pre-set MSK parameters are not disclosed by the manufacturer. Gain and depth adjustments were made, if needed, to improve the quality of the images. For the assessments, the individual was positioned supine with their knees flexed to 30 degrees using a bolster. The probe was positioned entirely on the patellar tendon and the full length of the tendon was assessed for areas of focal hypoechoic areas or thickening in both the longitudinal and axial views. Images of focal

hypoechoic areas and thickening were captured to determine echogenicity and thickening staging. Additionally, images were taken, of all the tendon at the proximal and distal attachment of the tendon in the longitudinal view to obtain thickness measurements during post analysis. To assess tendon vascularity, a dynamic evaluation using Power Doppler was performed. Dynamic videos and images were taken for post assessment grading of vascular activity.

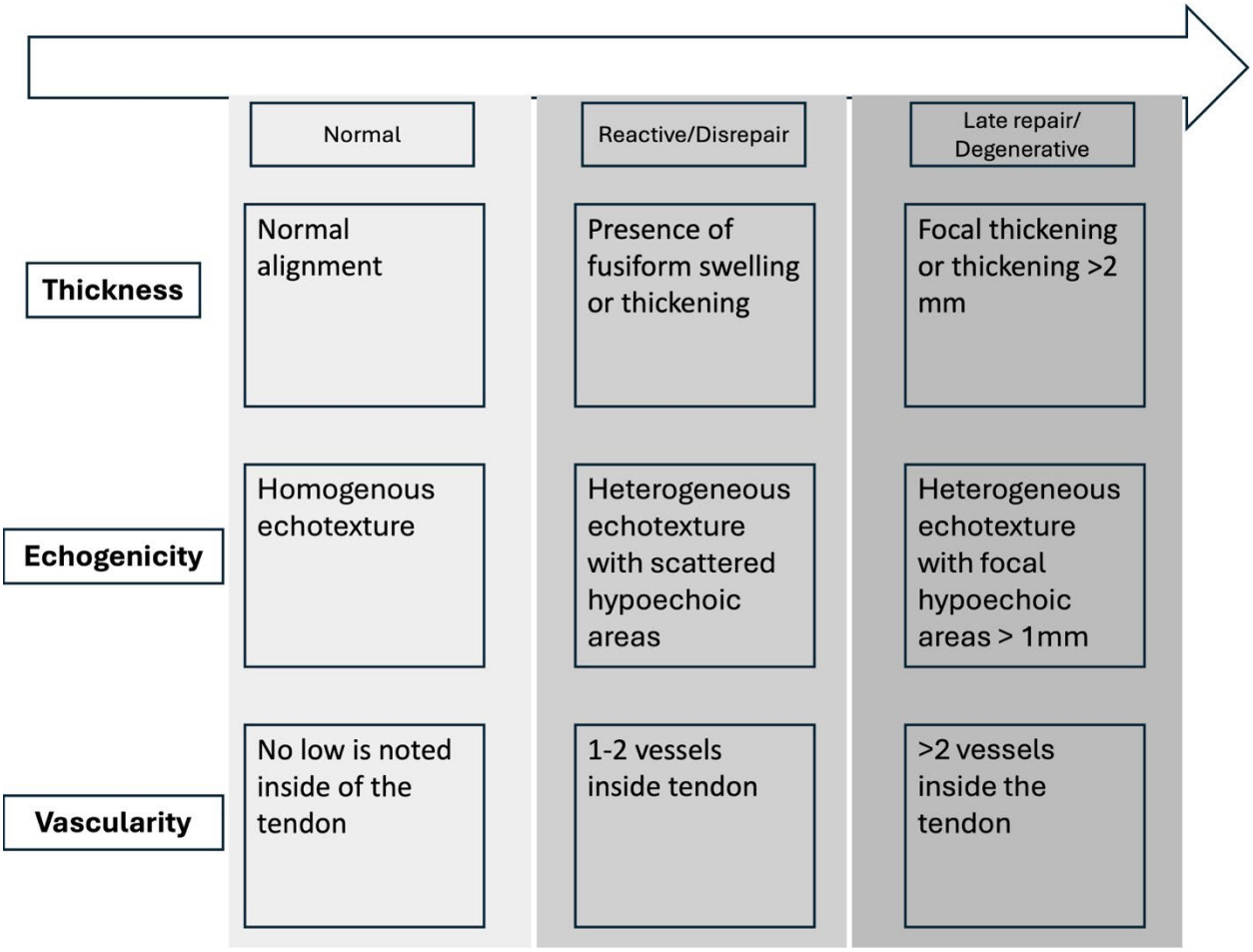
All measurements and staging of the tendon were performed during post analysis using images taken during the evaluation. Image J software (National Institute for Health, Bethesda, MD) was used to obtain all measurements. An ordinal scale was used to stage each parameter according to previous methods[119,144] with staging based on the continuum of care model.[15,152] Staging and ordinal scale is depicted in Figure 5.1. Thickness was measured from longitudinal images at the proximal and distal attachment of the patellar tendon to determine staging. If focal thickening was observed, the tendon thickness was measured where at the maximal area of fusiform swelling. If no visual changes were apparent then images were taken at 6mm from distal patellar attachment per previous methods.[119] Echogenicity staging was determined through measuring any focal hypoechoic regions in the longitudinal or axial images as well as a qualitative assessment of the structural appearance of the tendon fascicles in the longitudinal images. Heterogenous echotexture was determined by the appearance of diffuse hypoechoic areas between fibrillar echoes or the appearance of discontinuity in collagen fibrillar echoes. Vascularity will be staged by evaluating the images from the Power Doppler evaluation for vascular activity per the specified criteria in Figure 5.1.[144]

Perceived function

Symptoms and perceived function of trainees were evaluated through the individual scores on the VISA-P. The survey consists of eight items. Six items rate pain level during daily

and functional activities on a numerical pain scale from 0-10. Two items rate pain level during daily activities. The maximum possible score is 100 points, which represents full pain free function. The VISA-P score is the most widely reported PRO to measure changes in individuals with patellar tendon and has been established as a valid and reliable with minimal detectable change of 13 points. [153,154]

Figure 5.1 Ultrasound imaging staging criteria based on a modified method.



Clinical assessment of lower extremity function

To be eligible to start the SWCC, airmen must first complete an initial fitness test prior to entry. These scores were taken within one to seven days of the ultrasound assessment and other measuresf of perceived and objective function. Scores from their initial fitness test were obtained to describe the relationship between lower extremity function and ultrasound imaging outcome. Components of the fitness test include:1.5-mile run, 500-meter fin swim, push-ups (within 2 min), sit-ups (within 2 min), and pull-ups (within 2 min). The 1.5-mile run, and 500-meter fin swim times were used for analysis. The 1.5-mile run was performed on a quarter mile track with the candidates completing 6 laps. The 500-meter swim was performed in an Olympic size pool. The events are all done consecutively in the same order within the same day to assess candidates' overall fitness.

Data from a counter movement jump test was also assessed to obtain a clinically relevant measure of lower extremity function during the first week of course prior to the start of physical training. Trainees were asked to stand on a single limb with their hands behind their back. They were then instructed to quickly bend their standing leg and jump as high as they can and land on the same jumping leg. Trainees performed this three times on each limb. Jumps were performed on a Just Jump mat (Probotics Inc, Huntsville, Alabama) and the maximum recorded vertical height was used for analysis.

Injury history and surveillance

All cases of prior and future MSKI were collected by extracting medical data from the Military Health System Data Repository. The Military Health System Data Repository is a central repository for all beneficiary healthcare related data for more than 260 health care facilities and from non-DoD data sources. A MSKI taxonomy framework, previously used within

the study population, was used to classify injury type and region.[25] All cases of lower extremity injury and, specifically patellar tendinopathy, were cases of interest. Additionally, all MSKI cases of the regional knee defined as non-specific in the MSKI matrix were further evaluated using locally collected injury trackers to verify if a more specific diagnosis was available.

Prior history of injury was defined as a reported medical diagnosis within 90 days prior to the SWCC start date. Future injury will be defined by as a reported medical diagnosis that occurred during course or within 7 days after course end date or last status date within the course for those that did not complete course. The addition of seven days post course was to allow for injuries that may have been reported after course end. All individuals will be categorized as binary "yes/no" for having a previous lower extremity injury. They will also be categorized as a binary "yes/no" for or future lower extremity injury or patellar tendinopathy, respectively.

Statistical Analysis

A chi-square test was performed to assess the association of tendon abnormality with the occurrence of a lower extremity MKSI during course. The univariable cumulative incidence function was used to assess the time to lower extremity injury in the presence of the competing risk of dropping out of course early. The Gray's test was employed for statistical comparisons of the cumulative incidence probability of lower extremity MSKI, at 6-weeks from starting course, between groups including; (1) whether imaging results were abnormal versus normal, and (2) whether candidates had a history of prior MSKI within 90-days of starting course (yes versus no). Candidates who graduated from course without lower extremity MSKI were censored from the cumulative incidence function.

Univariate and multivariable logistic regression analysis were conducted to examine variables associated with tendon abnormality parameters. Univariate statistical tests were chosen

based on the type of variable and whether the data met parametric criteria. Univariate logistic regression analyses were conducted to determine the influence of the following variables on presence of PTA: demographic variables, maximal counter-movement jump, fitness scores, VISA-P scores, and previous MSKI. Variables with a significant relationship ($p<0.05$) to tendon abnormality parameters on univariate analysis were included in the multivariable model. An alpha level of 0.05 was the threshold for statistical significance in all analyses. To remain in the multivariable model, variables needed to have a p-value of less than 0.05. To measure the goodness-of-fit of the final model, the receiver operating characteristic (ROC) curve was analyzed. Statistical analysis was performed using SAS (SAS Institute, Cary, NC) to compute the cumulative incidence function and SPSS (IBM Corp, Armonk, N.Y) was used to perform all other analyses.

5.3 Results

Of the 71 participants, 51 individuals had at least one patellar tendon identified as normal, and 20 participants had at least one that was categorized as reactive or degenerative stage tendon abnormality. Descriptive statistics for demographic variables are depicted in Table 5.1. There were no significant differences among the normal tendon and abnormal tendon groups for demographic variables of age, height, weight, body fat percentage, or days in the course (Table 5.1). Descriptive statistics for perceived knee function and measures of objective lower extremity fitness are depicted in Table 5.2. Among the 20 participants that were categorized as abnormal, 9 (45%) went on to report a lower extremity MSKI and 3 (15%) reported patellar tendinopathy. There were 51 participants that were categorized as healthy; yet 25 (49%) of these participants reported a lower extremity MSKI, one of which was diagnosed with patellar tendinopathy.

The chi-square test identified that there was no association between the presence of PTA and lower extremity MSKI during course (χ^2=0.093, p=0.80). Furthermore, the analysis of cumulative incidence showed there was no significant increase in lower extremity injury risk for those with present PTA compared to those with normal tendon structure (p=0.87). Cumulative incidence probability of lower extremity MSKI at six weeks for individuals with normal tendon structure was 0.49 (95% CI: 0.36-0.62) and 0.45 (95% CI: 0.22-0.65) for those with PTA. Additionally, those with a previous history of MSKI did not have a higher cumulative incidence probability of lower extremity MSKI compared to individuals without a previous history (p=0.77). Cumulative incidence at six weeks for those without a previous MSKI was 0.51 (95% CI: 0.36-0.64) compared to those with a previous MSKI 0.55 (95% CI: 0.31-0.73) as depicted in Figure 5.2. The presence of patellar tendon abnormality was not significantly associated with any of the included variables besides previous MSKI in univariate analyses (Table 5.3). A binary logistic regression revealed that those with previous MSKI were more likely to have an abnormal patellar tendon upon assessment (OR 6.15, 95% CI: 1.99-19.05, p=0.02).

Table 5.1 Participant demographics (mean±SD) for those with and without patellar tendon abnormality.

Variable	Normal (N=51)	PTA (N=20)	P-value
Age (years)	26.06±1.73	23.10±4.38	0.18
Height (m)	69.75±2.86	69.95±2.49	0.78
Weight (kg)	79.04±8.26	79.63±11.10	0.81
Days in Training	40.78±9.92	36.05±10.45	0.08

SD=Standard Deviation

Table 5.2 Descriptive statistics (mean±SD) for measures of objective and subjective function.

Variable	Normal (N=51)	PTA (N=20)
VISA-P	100 (2)*	100 (3)*
Dominant CMJ (in)	9.77±1.52	9.50±1.74
Non-Dominant CMJ (in)	9.68±1.73	9.70±2.08
500 Meter Swim (s)	660.49±80.56	682.90±99.94
1.5 Mile Run (s)	581.24±22.59	581.80±22.97

SD=Standard Deviation, VISA-P=Victorian Institute of Sports (Australia) Scale, CMJ= Counter movement jump
*Denotes reporting of median (Interquartile Range).

Figure 5.2 Cumulative incidence probability of MSKI during course for those with and without history of prior MSKI.

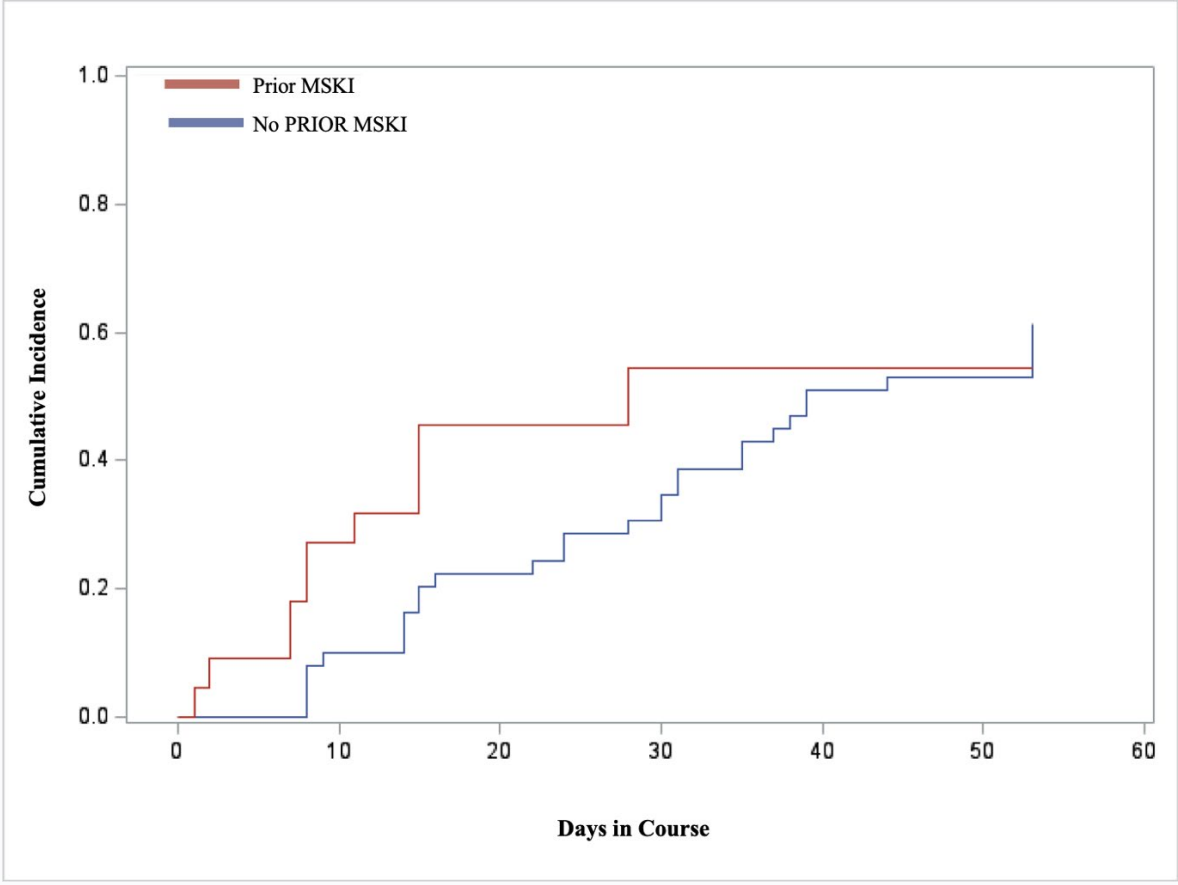


Table 5.3 Univariate testing results for factors associated with the probability of PTA.

Factor	Values	P-value	Odds Ratio (95% CI)
Prior MSKI	Yes / No	0.02*	6.15 (1.99-19.05)
VISA-P	Total score	0.987	1.00 (0.87-1.15)
Height	Inches	0.778	1.03 (0.85-1.24)
Weight	Kilogram	0.803	1.00 (0.95-1.07)
Dominant CMJ Height	Inches	0.481	0.89 (0.63-1.2)
Non-Dominant CMJ Height	Inches	0.972	1.00 (0.76-1.34)
500 Meter Swim	Seconds	0.325	1.00 (0.99-1.0)
1.5 Mile Run	Seconds	0.924	1.00 (0.98-1.03)

LE= Lower Extremity, CMJ= Counter movement jump, VISA-P= Victorian Institute of Sports Scale
*Denotes significant p-value (>0.05)

5.4 Discussion

MSKI Risk

The results from the study do not support the author's hypothesis that those with patellar tendon abnormality would be at an increased risk for patellar tendinopathy and lower extremity MSKI compared to individuals with no intratendinous abnormalities. Although the presence of PTA was identified in three of the four individuals that went on to have a patellar tendinopathy, this was not a large enough sample size to evaluate the significance of this findings. A systematic review has found that individuals may be up to six times more likely to develop patellar tendinopathy when PTA is present.[70]

Six weeks of training may not have been a long enough exposure time to fully evaluate the predictive value of PTA. Previous studies that evaluated the predictive value of PTA for future patellar tendinopathy had follow up times that ranged from one week to four years.[70]

Furthermore, these studies did not consistently report on the time that an individual with PTA developed patellar tendinopathy; therefore, it is unknown how long it may take individuals with PTA to report symptoms. Previous research on the pathophysiology of patellar tendinopathy has found that structural changes such as hypercellularity and collagen disruption in those with early onset of patellar tendinopathy are less apparent than in those with chronic tendinopathy symptoms.[56] Therefore, these structural changes may be difficult to identify using greyscale ultrasound prior to the onset of symptoms. Finally, a majority of the studies which found PTA was highly predictive of patellar tendinopathy were in athletes participating in sports with a large abundance of jumping activity.[70] The current population of students do not perform the same type of training activity, and, therefore, may have differences in patellar tendon loading than jumping athletes. Future research should confirm if the same relationship exists in military trainees that are also at as high risk of patellar tendinopathy. Further research is also warranted to explore the relationship between tendon abnormality and the risk of patellar tendinopathy in military trainees.

Those with PTA were not at a significantly higher risk of lower extremity MSKI than those with tendons categorized as normal. Intratendinous changes may not place a large enough burden on lower extremity function to induce injury. The mechanics that lead to PTA are not well known and may not lead to other lower extremity abnormalities in structure or function. Studies that have evaluated landing biomechanics in those with PTA have shown some evidence that these individuals have a different kinematic strategy, which may place them at higher risk of injury.[132,146] Those with symptomatic PTA have exhibited decreases in sagittal plane knee flexion along with increased hip extension during landing in horizontal landing tasks.[132,141] However, despite kinematic differences individuals with PTA did not exhibit differences in

loading during the landing tasks. [132,141] Therefore, these differences in kinematic strategy may not be indicative of high risk behavior and could possibly be an adaption to the presence of PTA.

The lack of increased risk is partially supported by the non-significant relationship found between function and structure in those with asymptomatic PTA within this study. The lack of relationship between PTA and MSKI risk could also reflect that tendons with PTA may have been undergoing a short disruption in tendon structure and were able to return to a state of normal tendon structure. Since follow up examinations were not assessed, it is unknown whether the PTA changes observed at baseline worsened or resolved throughout the training course. A majority of the tendon abnormalities observed in this study were categorized as reactive. According to the tendon continuum, tendons within this state may be more likely to be able to return to a normal state as opposed to tendons with more significant tendon structure disruption.[15] This is also partially supported by studies that have found that tendons undergo a collagen breakdown followed by a proliferation phase following training indicating that tendons may be able to return to a normal state following disruption.[40] Furthermore, a prospective study has shown that tendons with abnormalities can return to a normal state without reporting pain or injury.[155] Unfortunately, due to a low prevalence of tendons categorized as degenerative, we were unable to evaluate whether those at different stages on the continuum had varying risk of lower extremity MSKI. This is one of the first studies to assess PTA using a scale based on the continuum model. Therefore, future studies are needed to assess how the stage of PTA influences the probability of injury risk.

With this cohort, 48% of trainees sustained a lower extremity MSKI. This high prevalence of lower extremity MSKI has also been identified in other courses within the training pipeline in the SWTW. Previous research found 47.3% of individuals sustained an injury during

course.[148] Previous research in this population has found that previous injury was a significant factor in MSKI risk.[148] This is not supported by the current study where there was no increased risk of lower extremity injury in those with a previous history of MSKI. The lack of consistent findings may be due to differences in individual characteristics, training course differences, and the type of previous lower extremity MSKI incurred between study cohorts. For example, individuals within this study were participating in an entry level course that includes different demands than courses investigated in previous research. Although, the type of previous MSKI in this cohort was predominately lower extremity non-specific injury, similar to previous findings in this population, there may be differences in the type of non-specific injury between cohorts. Non-specific injuries such as tendinopathy in other lower extremity joints. Finally, there may be individual characteristics between cohorts such as training history that may be playing a role in the susceptibility to injury that were not investigated in this study.

Association between structure and function

Patellar tendon abnormalities evaluated through grey scale ultrasound assessment were not significantly associated with measures of lower extremity function. Participants within the study did not display VISA-P scores below 75, indicating clinically meaningful reduction in self-reported knee function or knee pain with activity.[153] Asymptomatic PTA has been identified in other highly active cohorts.[74] This study provides initial evidence that asymptomatic PTA also exists in military trainees. The lack of significant relationship between CMJ maximal height and PTA is also supported through previous research.[130] However, there may be some evidence to support that vertical landings may not be as sensitive to changes in landing patterns as horizontal landings.[132,141] Biomechanical analyses using 3D motion capture during horizontal tasks have been able to identify difference in landing patterns in those with asymptomatic PTA than those

with normal tendons.[132,141] Therefore, future research should consider incorporating clinical jumping assessments with a horizontal component just as the single leg crossover hop test for distance. Finally, broad measures of performance were not influenced by PTA. Since we did not evaluate knee extensor strength specifically, it is unknown whether these individuals had decreased extensor strength that may influence or lead to adaptive strategies during broad performance evaluations. Overall, PTA was not associated with any of the lower extremity functional tests and, therefore, this study supports previous research that has been unable to identify a connection between patellar tendon structure and function.

There was a significant associated between the presence of tendon abnormalities and previous MSKI. Although none of the individuals had diagnoses of patellar tendinopathy in the last 90 days, 30% of students had a previous MSKI. A majority of the previous injuries were lower extremity injuries. Therefore, it is plausible that these injuries may have led to increases in general lower extremity strain thus resulting in patellar tendon changes. Without knowing the specific timeframe in which PTA developed within these individual students, it is difficult to conclude whether the previous injury played a role in the formation of PTA. Additionally, previous MSKI was not at a higher risk of future injury during course. Therefore, although previous MSKI was a significant predictor of PTA prior to course it did not impact overall lower extremity injury risk.

Limitations

The largest limitation of the current study is the sample size included. Due to the small sample size certain statistical analysis were not indicated. For example, within this cohort, there were only four individuals that were diagnosed with patellar tendinopathy. Therefore, statistical analysis on the predictive utility of PTA findings for future patellar tendinopathy was not

assessed. Additionally, due to the small number of individuals with tendons categorized as degenerative, those with degenerative and reactive tendons were combined for analyses. Finally, a multivariate analysis to assess the relationship between PTA and demographic, previous injury, and lower extremity function variables was not indicated due to the sample size.

Furthermore, the type of ultrasound device and evaluation technique used may have led to differences in findings from previous studies. The ultrasound probe used in the current study was portable and may not have the same image quality as conventional cart-based ultrasound units. A recent study found that the results from portable ultrasound units were in agreement with those from conventional ultrasound equipment in 76% of evaluations for tendon abnormality.[156] The typical disagreements were in terms of severity versus the presence or absence of abnormality.[156] Thus, studies that use conventional ultrasound equipment may find varying findings from the current study. The type of parameters assessed through ultrasound may also influence congruent results with other studies. The current study focused on fibrillar uniformity and vascular changes with the tendon through grey-scale and power doppler assessments. Other studies have found that the stiffness of the tendon, evaluated through shear wave ultrasonography, may be more sensitive at finding structural changes related to function. Future studies are needed to determine if results from shear wave elastography evaluations are predictive of patellar tendinopathy or other lower extremity MSKI.

The methods implemented for the included assessments and MSKI surveillance also have limitations. The ultrasound, CMJ, and perceived function evaluations were not done in a systematic order for all individuals. This could have led to order effects between individuals. Additionally, previous and future injury was determined by the presence of a diagnosed injury in the individual's medical record. Therefore, the results of this study depend on individuals

accurately reporting their injuries to medical staff. Finally, the specific cohort that was recruited for this study does not allow for these results to be generalized to broader populations such as inactive individuals or highly active athletes.

Clinical Implications

These findings may help clinicians identify how to interpret and manage individuals with patellar tendon abnormalities. The current study found that intratendinous abnormalities exist in military trainees during technical training. However, these changes may not be predictive of future MSKI. Therefore, it may not be necessary to restrict these individuals from activity or start any type of interventional loading program. Information on intratendinous changes may be more useful when evaluating an individual with other clinical signs and symptoms of patellar tendinopathy. Information about intratendinous changes in those with clinical symptoms of patellar tendinopathy could provide useful information, when used in conjunction with other clinical tests, regarding where tendon falls along the tendon continuum to help guide treatments plans in those with patellar tendinopathy.

5.5 Conclusion

This study was one of the first to evaluate patellar tendinopathy using a grading system based on the tendon continuum model. Due to a limited number of patellar tendinopathy cases, this study was unable to assess the predictive utility of PTA for patellar tendinopathy in a cohort of military trainees. Additionally, this study did not find the presence of PTA to be predictive of lower extremity injury during course. Furthermore, the presence of PTA was not associated with subjective or objective function, although it was associated with previous MSKI injury. Future research is needed to further evaluate the predictive utility of PTA in military trainees and its relationship with other clinically relevant functional assessments.

CHAPTER 6

SUMMARY

The overarching purpose of this dissertation is to investigate the role of musculoskeletal ultrasound imaging in identifying abnormal soft tissue changes and the relationship between abnormal structure and function. Multiple aims guided the design of three independent studies to address the overarching purpose. The first aim was to assess whether plantar fascia tissue changes can be identified through ultrasound assessment in those with reported heel pain. Results from this study found that those with PHP exhibited significantly thicker tendons than those without plantar heel pain. Additionally, those with PHP exhibited decreased tactile sensation but no differences in abductor hallicus morphology or dynamic foot core stabilization. Overall, the study highlighted the importance of the evaluation of all subsystems of the foot core, to include structural changes within the plantar fascia.

Although ultrasound was shown to have the capability to depict structural changes in a common musculoskeletal injury, there lacked a broader understanding of the relationship between structural changes and function. The second study sought to provide information on the relationship between structural changes and function through a systematic review of the current literature on patellar tendon abnormality and lower extremity function. The review revealed a large amount of heterogeneity among the included studies. The different techniques to grade and assess lower extremity function in those at risk for patellar tendinopathy highlighted the need for future studies to develop consistent outcomes. Due to the methodological differences amongst the studies, it was difficult to make strong conclusions about the relationship between tendon abnormality and the included outcomes. However, the review did find that there was little to no meaningful relationship found between patellar tendon abnormality and self-reported function

among the majority of studies. Furthermore, there may be some evidence to suggest that those with asymptomatic PTA exhibited differences in landing patterns but were only evident in landing tasks with a horizontal landing phase.

To further examine the importance of PTA found through ultrasound a third study was conducted. The primary aim of the third study was to determine whether abnormal tissue changes in the patellar tendon place individuals at a higher risk of patellar tendinopathy or other lower extremity MSKI. Results indicated that those with PTA were not at a greater risk of sustaining a lower extremity injury than those whose tendons were categorized as normal. Due to a number of trainees being diagnosed with patellar tendinopathy, we were unable to assess the risk probability between those with and without PTA. The second aim of the study was to evaluate the relationship between PTA and lower extremity function. PTA was not significant associated with subjective or objective measures of lower extremity function.

Overall, ultrasound imaging may be useful in identifying pathological differences in those with symptomatic musculoskeletal conditions; however, the relationship between function and structure still needs future investigations. Findings from this dissertation imply that information from ultrasound evaluation may provide useful information during evaluation but should not be used as the sole determinant for diagnosis and should be used in conjunction with other clinical assessment. Information from ultrasound evaluation could still provide clinicians with helpful information to help understand the structural integrity of tendinopathy or fasciopathy and guide treatment strategies.

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PUBLICATIONS

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1. Mata JD. Patrick AL. Tchandja JN. Haydu LE. Hogan KK. Kasper KB. Trigg SD. Butler CR. (2024) Anthropomorphic and Physical Fitness Characteristics of United States Air Force Basic Military Training: Special Warfare Versus Nonspecial Warfare Recruits, Fiscal Year 2019-2023. Cureus 16(6).
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