Invited Topical Review

Physiotherapy management of patellar tendinopathy (jumper’s knee)

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KEYWORDS
Patellar tendinopathy
treatment
physiotherapy

Introduction

Patellar tendinopathy (jumper’s knee) is a clinical diagnosis of pain and dysfunction in the patellar tendon. It most commonly affects jumping athletes from adolescence through to the fourth decade of life. This condition affects health and quality of life by limiting sports and activity participation for recreational athletes and can be career-ending for professional athletes. Once symptoms are aggravated, activities of daily living are affected, including stairs, squats, stand to sit, and prolonged sitting.

Patellar tendinopathy clinically presents as localised pain at the proximal tendon attachment to bone with high-level tendon loading, such as jumping and changing direction. Tendon pain at the superior patellar attachment (quadriceps tendinopathy) and at the tibial attachment occurs less frequently, but the diagnosis and management are similar to jumper’s knee. It is commonly clinically diagnosed in conjunction with imaging (ultrasound or magnetic resonance, often to exclude differential diagnoses such as patellofemoral pain), where structural disruptions on the scans represent areas of tendon pathology. Importantly, there is a disconnection between pathology on imaging and pain; it is common to have abnormal tendons on imaging in people with pain-free function.

Prevalence

Patellar tendinopathy is an overuse injury that typically has a gradual onset of pain. Athletes with mild to moderate symptoms frequently continue to train and compete. Determining the prevalence of overuse injuries such as patellar tendinopathy is difficult because overuse injuries are often not recorded when injuries are defined exclusively by time-loss from competitions and training. The time-loss model only records acute injuries and the most severe overuse injuries, making it difficult to gather an accurate estimate of the prevalence of patellar tendinopathy in the athletic population.

Studies that have specifically examined the prevalence of patellar tendinopathy showed that the type of sport performed affected the prevalence of tendinopathy. The highest prevalence in recreational athletes was in volleyball players (14.4%) and the lowest was in soccer players (2.5%); the prevalence was substantially higher in elite athletes. Tendon pathology on imaging in asymptomatic elite athletes was reported in 22% of athletes, male athletes had twice the prevalence as female athletes, and basketball players had the highest prevalence of pathology (36%) amongst the sports investigated: basketball, netball, cricket and Australian football. It is not only a condition that affects adults; the prevalence of patellar tendinopathy in young basketball players was reported as 7%, but 26% had tendon pathology on imaging without symptoms.

Patellar tendon rupture, however, is rare. The most extensive analysis of tendon rupture reported that only 6% of tendon ruptures across the body occurred in the patellar tendon. The majority of patellar tendon ruptures that do occur are in the older population (mean age 65 years). All those who had a patellar tendon rupture had pathology in the tendon. Because this is a relatively rare injury, it will not be discussed in this review.

Aetiology

The pathoetiology of tendinopathy is unknown and there are several models that attempt to describe the process. Of these,
the continuum model of tendinopathy has the most overt clinical correlation. The continuum model places tendon pathology in three somewhat interchangeable stages: reactive tendinopathy, tendon dysrepair and degenerative tendinopathy (Figure 1). Many patellar tendons have a combination of pathology state (reactive on degenerative pathology). A degenerative patellar tendon with a circumscribed degenerative area is thought to have insufficient structure to bear load resulting in overload in the normal area of the tendon, leading to a reactive tendinopathy in this area.

The capacity for tendon pathology to move forward and back along the continuum was demonstrated in the patellar tendons of basketball players. Players were imaged with ultrasound each month during the season and those with reactive tendinopathy and tendon dysrepair both progressed (to degenerative tendinopathy) and regressed (to normal tendon) through the season. Whilst it is known that pathology on imaging does not necessarily indicate painful patellar tendinopathy, certain changes (ie, the presence of large hypoechoic regions on ultrasound) may increase the risk of developing patellar tendinopathy.

It is also unknown at what age a patellar tendon is susceptible to pathology, but it does occur in young athletes. Studies have shown that tendon tissue is inert and does not renew after the age of 17, suggesting that once tendon is formed in puberty its structure is relatively stable. An early age of onset of patellar tendinopathy is supported by data that shows only two players developing it after the age of 16 in a school for talented volleyball players.

The aetiology of pain appears somewhat independent of underlying tendon pathology. Pain is frequently associated with pathological tendons, however tendon pain in apparently normal tendons has been demonstrated. Overload is reported as the key factor associated with pain onset. Overload is defined as activity above what the tendon has adapted to at that point in time, and can occur by a sudden and substantial increase in the volume of jumping or a return from injury/holiday without gradually ramping back into a regular schedule. The use of energy storage and release loads in jumping and change of direction is typically characteristic of overload causing patellar tendinopathy pain. Non-energy-storage loading or non-jumping activity (eg, cycling or swimming) and repetitive low loading (in runners) rarely aggravate the patellar tendon; other pathologies are generally suspected in these cases.

Risk and associated factors

Several studies have examined intrinsic and extrinsic risk and associated factors for both pathology and patellar tendinopathy (Table 1). Risk factors for pathology and risk factors for pain are likely to be different and will be distinguished in this section. Biomechanical studies of painful tendons will not be discussed, as altered mechanics may be an outcome of having a painful patellar tendon, however, they would certainly be considered as part of a management paradigm.

Extrinsic factors

An increase in training volume and frequency has been associated with the onset of patellar tendinopathy in several studies. Clinically, this is the most common factor that triggers patellar tendinopathy. Other factors, such as change in surface density and shock absorption, may have an effect as well. Although harder surfaces can increase patellar tendinopathy symptoms, they are less likely to be an issue nowadays as most indoor sport is now played on standard sprung wooden floors. Surface density and amount of shock absorption in both the shoes and the surface should still be considered, as athletes may be vulnerable when

<table>
<thead>
<tr>
<th>Study</th>
<th>Factor</th>
<th>Risk factor or associated factor</th>
<th>Patellar tendinopathy or tendon pathology</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visnes</td>
<td>Gender</td>
<td>Both</td>
<td>Both</td>
<td>Increased waist circumference associated with increased pathology Adolescents only</td>
</tr>
<tr>
<td>Cook</td>
<td></td>
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<td></td>
<td>Less extensible hamstrings associated with pathology</td>
</tr>
<tr>
<td>Malliaras</td>
<td>Waist circumference</td>
<td>Associated</td>
<td>Pathology</td>
<td>Less extensible hamstrings increase risk of patellar tendinopathy</td>
</tr>
<tr>
<td>Cook</td>
<td>Imaging abnormality</td>
<td>Risk</td>
<td>Pathology</td>
<td>Stiffer quadriceps increase risk of patellar tendinopathy</td>
</tr>
<tr>
<td>Witvrouw</td>
<td>Hamstring length</td>
<td>Associated</td>
<td>Pathology</td>
<td>Reduced dorsiflexion associated with increased pathology</td>
</tr>
<tr>
<td>Witvrouw</td>
<td>Quadriceps length</td>
<td>Risk</td>
<td>Pathology</td>
<td>Less knee bend at landing, altered hip strategies associated with pathology</td>
</tr>
<tr>
<td>Malliaras</td>
<td>Dorsiflexion</td>
<td>Associated</td>
<td>Pathology</td>
<td>Better jumping ability associated with patellar tendinopathy</td>
</tr>
<tr>
<td>Edwards</td>
<td>Altered landing strategies</td>
<td>Associated</td>
<td>Pathology</td>
<td>Increased fat pad size associated with patellar tendinopathy</td>
</tr>
<tr>
<td>Liard</td>
<td>Jumping ability</td>
<td>Both</td>
<td>Tendinopathy</td>
<td>Excess loading associated with patellar tendinopathy</td>
</tr>
</tbody>
</table>
training on hard floors, athletic tracks, or surfaces with high horizontal traction.

**Intrinsic factors**

Several studies have attempted to identify specific anthropometric characteristics that may increase the risk of patellar tendinopathy symptoms. These characteristics include: height, weight, lower limb joint range of motion, leg length, body composition, lower limb alignment, and the length and strength of the hamstring and quadriceps. Thigh muscle length (shorter or less extensible quadriceps and hamstrings) has been associated with patellar tendinopathy, whilst greater strength has been associated with reduced pain and improved function. Conversely, better knee extensor strength and jumping ability has been reported in athletes with patellar tendinopathy, especially in jumps involving energy storage. Young women, but not young men, with tendon pathology have been found to have a better vertical jump performance than those without pathology. Clinical observation aligns with patellar tendinopathy being more prevalent among athletes with better jumping ability.

Different lower limb kinematics and muscle recruitment in horizontal landing phase have been associated with tendon pathology. Edwards et al demonstrated the horizontal braking force to place the highest load on the patellar tendon. They suggested that the compression through the patellofemoral joint and the patellar tendon and the tensile loading with the knee flexed all contribute to pathology in those with asymptomatic tendon pathology.

Lower foot arch height, reduced ankle dorsiflexion, greater leg length discrepancy, and patella alta in men have each been associated with patellar tendinopathy. Boys and men are two to four times more likely to develop patellar tendinopathy than girls.

Increased waist circumference in men is associated with greater prevalence of pathology on ultrasound. It has been reported that men with a waist circumference greater than 83 cm are more likely to have abnormal changes on imaging (74% versus 15% in those with less than 83 cm). One study found that athletes with patellar tendinopathy were generally younger, taller and weighed more than those without patellar tendinopathy.

Infrapatellar fat pad size was significantly larger in those with tendinopathy than in controls.

**Assessment**

**History**

There are few papers providing evidence on assessment procedures, therefore this section is based on expert opinion. As with all musculoskeletal conditions, a detailed history is very important and must first identify if the tendon is the likely source of pain. This is determined initially in the history by asking the person to indicate where they feel their pain during a patellar tendon-loading task (such as jumping and changing direction). They should point with one finger to the tendon attachment to the patella; more widely distributed pain should raise the possibility of a different diagnosis. Second, a history should identify the reason that the tendon has become painful; this is classically due to tendon overload. Two common overload scenarios are seen: a large increase in overall load from a stable base (eg, beginning plyometric training or participation in a high-volume tournament) or returning to usual training after a significant period of downtime (eg, return to training after 4 to 6 weeks time off for an ankle sprain or holidays). Elite athletes can have repeated loading/unloading periods due to injuries and season breaks over several years, which gradually reduces the capacity of the tendon to tolerate load and leaves it vulnerable to overload with small changes in training. No identifiable change in load or pain induced from a load that should not induce patellar tendinopathy (such as cycling) should suggest alternative diagnosis.

Pain behaviour also has a classic presentation: the tendon may be sore to start activity, respond variably to warm-up (from completely relieving symptoms to not at all) and will then be worse the next day, which can persist for several days. The athlete will rarely complain of night pain and morning stiffness (unless symptoms are severe), but will complain of pain with prolonged sitting, especially in a car. Pain with sitting can be a good reassessment sign as the condition improves. Pain during daily activity is also common; stairs and squatting are provocative.

Most athletes who present clinically with patellar tendinopathy are good power athletes; they will describe being good at jumping and being quick, especially in change of direction. They will complain that their tendon pain affects their performance, reducing the attributes that allow them to excel at sport.

When taking a history, it is critical to document all previous treatment that the patient has explored, including all types of interventions and rehabilitation strategies, descriptions of the successful and unsuccessful interventions, and details of all exercises including number of repetitions, sets, weights and frequency. Many people will consult a variety of physiotherapy, orthopaedic and sports medicine professionals; inconsistency of care may prolong the rehabilitation process. The history should document all the known risk factors for tendinopathy, such as diabetes, high cholesterol, seronegative arthropathies and the use of fluoroquinolones. These are known to contribute to other tendinopathies, but their role in the patellar tendon is unknown. Finally, the examiner should ask about past injury and medical history, including previous injuries that have necessitated unloading or time off from sports activity or that may have altered the manner in which the athlete absorbs energy in athletic manoeuvres.

**Examination**

The VISA-P (Victorian Institute of Sports Assessment for the Patellar tendon) should be completed as a baseline measure to allow monitoring of pain and function. The VISA-P is a brief questionnaire that assesses symptoms, simple tests of function and ability to participate in sports. Six of the eight questions are on a visual analogue scale (VAS) from 0 to 10, with 10 representing optimal health. The maximal score for an asymptomatic, fully functioning athlete is 100 points, the lowest theoretical score is 0 and less than 80 points corresponds with dysfunction. It has high impedance, so it is best repeated monthly and the minimal clinically significant change is 13 points. Tenderness on palpation is a poor diagnostic technique and should never be used as an outcome measure, however, pain pressure threshold, as measured by algometry, has been found to be significantly lower in athletes with patellar tendinopathy (threshold of 36.8 N) when compared to healthy athletes.

Observation will nearly always reveal wasting of the quadriceps and calf muscles (especially gastrocnemius) compared to the contralateral side; the degree of atrophy is dependent on the length of symptoms. Athletes who continue to train and play, even at an elite level, are not immune to strength and bulk losses, as they are forced to unload because of pain.

**Clinical tests**

A key test is the single-leg decline squat. While standing on the affected leg on a 25 deg decline board, the patient is asked to maintain an upright trunk and squat up to 90 deg if possible (Figure 2). The test is also done standing on the unaffected leg. For each leg, the maximum angle of knee flexion achieved is recorded, at which point pain is recorded on a visual analogue scale. Diagnostically the pain should remain isolated to the tendon/Patellar tendon. This test is an excellent self-assessment to isolate and monitor the tendon’s response to load on a daily basis.

Kinetic chain function is always affected, the leg ‘spring’ has poor function, and is commonly stiff at the knee and
soft at the ankle and hip. The quality of movement can be assessed
with various single-leg hop tests and specific change of direction
tasks. Record pain (VAS) and function at take off and landing,33 and
note if more load induces more pain. If possible, measurement of
angles and individual joint moments through video/biomechanical
analysis can help with more elite athletes. Hop tests for height and
distance can also be used to assess kinetic chain quality, as well as
providing an objective means of monitoring progress.
Muscle strength, assessed through clinical and functional
measures (repeated calf raise and decline squats), is useful to
assess the level of unloading in the essential muscles. Dorsiflexion
range of movement is a critical assessment, as the ankle and calf
absorb much of the landing energy.34 Stiff talocrural joint
dorsiflexion,26 global foot stiffness and/or hallux rigidus all
contribute to increased load on the musculotendinous complexes
of the leg.

Imaging

Imaging with traditional ultrasound and magnetic resonance
can identify the presence of pathology in the tendon. Ultrasound
tissue characterisation, a novel form of ultrasound, can quantify
the degree of disorganisation within a tendon and may enhance
clinical information from imaging (Figures 3 and 4).35 Imaging will
nearly always demonstrate tendon pathology, regardless of the
imaging modality used. The presence of imaging abnormality does
not mean that the pathology is the source of the pain so clinical
confirmation, as described above, is essential. More importantly,
the pathology is commonly degenerative, often circumscribed and
does not change over time, so imaging the tendon as an outcome
measure is unhelpful, as pain can improve without positive
changes in tendon structure on imaging.35 In elite jumping sports,
such as volleyball, patellar tendon changes are nearly the norm, which needs to be considered when interpreting clinical and imaging findings.

**Differential diagnosis**

The history and examination are crucial to distinguish patellar tendinopathy from other diagnoses including: patellofemoral pain; pathology of the plica or fat pad; patellar subluxation or a patellar tracking problem; and Osgood-Schlatter disease.\(^{36}\)

**Physiotherapy management**

While pathology in a patellar tendon may not ever completely resolve, symptoms of patellar tendinopathy can generally be managed conservatively. This section will draw from the literature on therapeutic management of patellar tendinopathy, as well as clinical expertise and emerging areas of research.

**Active interventions**

Intervention is aimed at initially addressing pain reduction, followed by a progressive resistive exercise program to target strength deficits, power exercises to improve the capacity in the stretch-shorten cycle, and finally functional return-to-sport training (Table 2). Daily pain monitoring using the single-leg decline squat provides the best information about tendon response to load; consistent or improving scores suggest that the tendon is coping with the loading environment.

**Pain reduction**

Reducing an athlete’s symptoms requires load management, although it is important to avoid complete cessation of tendon loading activities, as that will further reduce the load capacity of the tendon.\(^{33}\) Removing high-load drills from training, reducing frequency of training (twice a week is tolerable for many tendons) and decreasing volume (reducing time of training) are all useful means of reducing load on the tendon without resorting to complete rest.

Sustained isometric contractions have been shown to be analgesic.\(^{37}\) In painful patellar tendinopathy (usually a reactive or active degenerative pathology), pain relief can be obtained for 2 to 8 hours with heavy sustained isometric contractions. Voluntary contractions at 70% of maximum, held for 45 to 60 seconds and repeated four times is one loading strategy that has been shown to have a large hypoalgesic effect. This loading can be done before a game or training, and can be done several times a day.\(^{38}\) If the tendon is highly irritative, bilateral exercise, shorter holding time and fewer repetitions are recommended.\(^{38}\) Additionally, medication may help to augment pain reduction and/or pathological change in a reactive tendon,\(^{39}\) so consultation with a physician is advised.

**Strengthening**

Eccentric, heavy slow resistance, isotonic and isometric exercises have all been investigated in patellar tendinopathy. Eccentric exercises have generally been shown to have good short-term and long-term effects on symptoms and VISA-P scores. There are several different types of eccentric patellar tendon loading exercises; however, there is no difference in the results of a 12-week eccentric training program between the bilateral weighted squat (Bromsman device) twice a week and the unilateral decline squat daily.\(^{40}\)

Several interventions have used the 25 deg single-leg decline squat, which has been shown to have better outcomes than a single-leg flat squat.\(^{41}\) Two investigations have shown that angles above 15 deg are equivocal\(^{42,43}\) and that the decline board is effective by increasing the moment arm of the knee.\(^{44}\)

Two studies have investigated the effect of eccentric exercise in the competitive season. Visnes et al reported no overall effect and a short-term worsening with decline squat training on function in symptomatic athletes continuing a regular training program, compared to a regular training program only.\(^{45}\) Fredberg et al showed an increased risk of injury for asymptomatic athletes with pathology on ultrasound who completed a prophylactic eccentric decline squat training program.\(^{46}\) This suggests that the addition of eccentric exercise while an athlete is in a high-load environment is detrimental to the tendon. When comparing an eccentric decline squat protocol to a patellar tenotomy, there was no difference in the outcomes and both showed improvement.\(^{47}\) Surgical intervention is not recommended over an exercise rehabilitation program in the first instance.

Heavy slow resistance exercises were investigated by Kongsgaard and colleagues,\(^{48}\) who compared the effects of a

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**Table 2**

Suggested rehabilitation progression for patellar tendinopathy.

<table>
<thead>
<tr>
<th>Phase of rehabilitation</th>
<th>Aim of treatment</th>
<th>Intervention</th>
<th>Example exercises</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain management</td>
<td>Reduce pain</td>
<td>Isometric exercises in mid-range as tolerated.</td>
<td>Sustained holds on leg extension; 45 s, 4 repetitions, 2 times/day.</td>
</tr>
<tr>
<td></td>
<td>Improve strength</td>
<td>Heavy slow resistance as tolerated (isotonic)</td>
<td>Leg extension/press, 4 sets of 6-8 repetitions, 3-5 times/wk</td>
</tr>
<tr>
<td>Strength progression</td>
<td></td>
<td>Progressive resistance exercise program, functional tasks, address movement patterns, kinetic chain and endurance training as required</td>
<td>Walk lunge with body weight or extra weight, stair walking</td>
</tr>
<tr>
<td>Functional strengthening</td>
<td>Increase power</td>
<td>Increase speed of muscle contraction, lower the number of repetitions</td>
<td>Split squats, faster stairs, skipping exercises</td>
</tr>
<tr>
<td></td>
<td>Decrease symptoms</td>
<td>Plyometric exercises, graduated gradually</td>
<td>Jumping, deceleration and change of direction tasks</td>
</tr>
<tr>
<td></td>
<td>and prevention of flare ups</td>
<td>Drills specific to sport including endurance training</td>
<td>Sports specific drills at set intensity and duration</td>
</tr>
<tr>
<td>Maintenance</td>
<td></td>
<td>Education, continue strength training and manage loading as tolerated</td>
<td>Continue leg extension strength or Spanish squat exercise while training and playing</td>
</tr>
</tbody>
</table>
peritendinous corticosteroid injection to the proximal patellar tendon to a decline squat eccentric exercise protocol and a heavy slow resistance protocol in people with patellar tendinopathy. All three groups showed improvements at 12 weeks; however, at 6 months only the groups using the eccentric exercises and the heavy slow resistance exercises still showed improved VISA-P and VAS scores. The heavy slow resistance group showed improved tissue normalisation of the collagen and also demonstrated better clinical presentations than the eccentric group within the 12-week follow-up.

Combined exercises with eccentrics, concentrics and plyometric training for the Achilles tendon were studied by Silbernagel and colleagues. Athletes were allowed to continue training in their sports during the first 6 weeks of rehabilitation, as long as their pain did not go over 5/10 on the VAS during activity and returned to normal by the next morning. While this study was investigating Achilles tendinopathy, this combined approach is often used clinically with patellar tendinopathy and should be considered as a treatment option.

Functional strengthening and return to sports

Functional strengthening must address high-load tendon capacity as well as kinetic chain deficits and movement patterns. Once these patterns have improved, the athlete should begin sports-specific training. Faster contractions can progress loads towards the stretch-shorten cycle that forms the basis for return to sports. Early drills should include: skipping, jumping and hopping, progressing to agility tasks, direction changes, sprinting and bounding movements. It is important to quantify these tasks and use a high-low-medium-load day approach in early reintroduction of high-load activities and return to sports. Also, include training specificity when returning an athlete back to their sport, including movement assessment for optimal kinetic chain loading.

Passive interventions

Other techniques may be useful in augmenting an exercise program; however, there is little evidence for effect of passive treatments for patellar tendinopathy. Exercise, pulsed ultrasound and transverse friction massage have been compared, and exercise had the best effects in the short and long term. Manual therapy techniques, including myofascial manipulation of the knee extensor muscle group, have had a positive effect on reducing pain in patellar tendinopathy patients in short-term and long-term follow-up. Other passive therapies, including braces and taping techniques, are often used clinically to help unload the patellar tendon, however, no evidence supports their efficacy. Passive therapies are best used to reduce symptoms in season so the athlete can continue to participate in rehabilitation and sport.

Other interventions

Extracorporeal shockwave therapy, corticosteroid injections, platelet-rich plasma and other injections are interventions frequently used in the clinical setting, yet have limited evidence supporting their use in patellar tendinopathy. There was no benefit of extracorporeal shockwave therapy compared to placebo for in-season athletes with chronic patellar tendinopathy. A direct comparison between platelet-rich plasma and extracorporeal shockwave therapy showed significantly better outcomes in the platelet-rich plasma group at 6-month and 12-month follow-up, compared to the extracorporeal shockwave therapy group; however, both groups showed similar and significant improvements at the 2-month follow-up. Peritendinous corticosteroid injection, oral steroid medication, or iontophoresis may be useful and effective at quickly reducing cell response and pain in a reactive tendon, however, the long-term outcomes are worse than those obtained with exercise. Corticosteroid injection, however, is not indicated in degenerative tendinopathy. Analgesic injections may alter an athlete’s perception of pain and ability to moderate activity, this absence of symptoms has been associated with poorer outcomes and is not advised in season.

Studies of the efficacy of platelet-rich plasma injections as a treatment for tendinopathy show little effect. A literature review in 2011 showed positive outcomes for several injection-based studies with small sample sizes; further research is needed. Surgical interventions including arthroscopic shaving and sclerosing injections are improving in their ability to reduce pain and amount of time out of sports. When considering surgery, it is important to factor in stage of tendinopathy and treat it as part of a well-rounded rehabilitation program involving kinetic chain exercises, education in proper landing technique and management of load and return to sports.

Education

It is important for the athlete to have realistic expectations of the rehabilitation process and to understand that management of their symptoms is required throughout their sports career, whether recreational or professional. The athlete must know how to monitor symptoms and adjust participation and loading appropriately throughout the rehabilitation process and in return to sport, and should always maintain strength exercises twice weekly throughout their sporting careers.

Tendons generally have a delayed response to load and will cause minimal pain during activity, but flare 24 hours later. Regular pain monitoring will help guide and progress the exercise program and should be maintained after return to sport. The best monitoring is the single-leg decline squat, which an athlete can use to self-assess symptoms in order to determine response to rehabilitation and participation in their sport. A journal of symptoms and pain on decline squat will help the athlete to identify triggers, monitor loading response and learn to manage symptoms independently.

Factors affecting prognosis

Return to sport can be slow and is often dependent on severity of the pain and dysfunction, the quality of rehabilitation, and intrinsic and extrinsic factors. Gemignani et al associated mild pathology in the tendon to 20 days of rehabilitation before return to sports, and more severe pathology with approximately 90 days until return to sport. However, these imaging-based guidelines may underestimate return-to-sport time, considering that other factors affect prognosis. The athlete who presents with a high level of kinetic chain dysfunction, regardless of pain level, will take considerable time (6 to 12 months) to recover both muscle and tendon capacity. This is complicated if the athlete aspires to return to a high level of performance, for example an elite high jumper will require much more rehabilitation than a recreational football player, as the jumping demands differ greatly. Even within elite sport there are levels of loading for the patellar tendon, a volleyball player will jump and land much more than a basketball player and will also require greater rehabilitation time. Regardless, impatience with rehabilitation creates a poorer prognosis; time, proper rehabilitation and appropriate graded return to sports are an effective treatment.

Factors affecting response to therapy

Pain in tendinopathies is poorly understood, however, there is emerging evidence in support of an element of central sensitisation or pathophysiological up-regulation of the central nervous system. A small study has demonstrated that athletes with patellar tendinopathy have a lower mechanical pain threshold and greater sensitivity to vibration disappearance than non-injured athletes. Local pathology, such as neovascularisation, lacks evidence as the primary pain driver, which is yet to be determined.
Avenues for further research

More research is required to fully understand how a tendon fails in adaptive capacity and pathology develops, and what causes the pain in the tendons that is so specific to loading. Intervention studies to clarify an optimal loading program, as well as the eventual development of a prevention program would also be beneficial.

Conclusions

Research has increased our understanding of patellar tendonopathy and pathology but there is still more to discover. Currently, the most important factors in managing athletes with patellar tendonopathy are to educate them about how to modify loading according to symptoms, to ensure that they understand how to increase or decrease loading appropriately, and to assess and modify intrinsic and extrinsic factors that may be contributing to overload.

Ethics approval: Nil

Competing interests: Nil

Source(s) of support: Professor Cook is supported by the Australian Centre for Research into Sports Injury and its Preven-
tion, which is one of the International Research Centres for Prevention of Injury and Protection of Athlete Health supported by the International Olympic Committee (IOC). Prof. Cook is supported by a NHMRC practitioner fellowship (1058493).

Acknowledgements: We thank SI Docking for the supply of the tendon ultrasound figures.

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