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# Current trends in tendinopathy management

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A B S T R A C T

Tendinopathy (pain and dysfunction in a tendon) is a prevalent clinical musculoskeletal presentation across the age spectrum, mostly in active and sporting people. Excess load above the tendon's usual capacity is the primary cause of clinical presentation. The propensity towards chronicity and the extended times for recovery and optimal function and the challenge of managing tendinopathy in a sporting competition season make this a difficult condition to treat. Tendinopathy is a heterogeneous condition in terms of its pathology and clinical presentation. Despite ongoing research, there is no consensus on tendon pathoetiology and the complex relationship between tendon pathology, pain and function is incompletely understood. The diagnosis of tendinopathy is primarily clinical, with imaging only useful in special circumstances. There has been a surge of tendinopathy treatments, most of which are poorly supported and warrant further exploration. The evidence supports a slowly progressive loading program, rather than complete rest, with other treatment modalities used as adjuncts mainly targeted at achieving pain relief.



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## Introduction

Tendinopathy is the term used for the clinical presentation of pain in a tendon with resulting dysfunction. It is used in preference to pathological terms, such as tendinitis and tendinosis [1]. Tendinopathies are prevalent and persistent, impacting on the ability to exercise and work, resulting in a socio-economic burden.

The incidence of lateral epicondylitis in the general population is estimated to be 1–3% [3]. The incidence of tennis elbow or lateral elbow tendinopathy is 2–3.5 times higher in those over 40 years of age than those below 40 and higher in those playing tennis more than 2 h per day [4]. It is 5–9 times more common than medial elbow tendinopathy and up to 40% of tennis players experience lateral elbow tendinopathy [4].

The incidence and prevalence of lower limb tendinopathy have been shown to be 11.83 and 10.52 per 1000 person-years, respectively [2]. The prevalence of lower limb tendinopathy in the sporting population varies; 11.8%–14.4% of recreational volleyball and basketball players reported symptoms of patellar tendinopathy, rising to 32% and 45% in elite men's basketball and volleyball players, respectively [5]. The recurrence rate is high despite treatment at 49% and up to 50% retire from sport as a result of the condition [5]. Achilles tendinopathy has a prevalence of 6.2–9.5% in the athletic population, whilst a prevalence of 11.83 per 100 patient years in the non-athletic population [6].

This article focuses on our current understanding of tendinopathy including the pathoetiology, risk factors, diagnosis (including the role of diagnostic imaging) and management. Principles of management and current evidence-based approaches to treatment will be discussed using Achilles and rotator cuff tendinopathy as examples.

## Pathoetiology

There are several different structures in a tendon that can become pathological. Aside from pathology in the mid tendon, peritendinitis/tenosynovitis/tenovaginitis describe inflammatory pathology of the connective tissue layer that envelops the tendon. Enthesopathy is pathology that occurs at the tendon-bone junction where the tendon is subjected to compressive loading. Partial or complete tendon tears occur in a tendon with widespread degenerative pathology. Partial tears are often difficult to diagnose clinically, and there are no standardised criteria to distinguish partial tears from tendinosis on diagnostic imaging.

The pathogenesis of tendon pathology is not fully understood. Three conceptual models have been proposed to explain the pathogenesis of tendinopathy; 1) tendon cell response, 2) collagen disruption and, 3) inflammation. It is likely that all three models explain aspects of tendon pathology. The prevailing model is that tendon pathology occurs from a primary response of the tendon cells to overload, which results in tendon cell activation and proliferation, increases in proteoglycans that in turn results in collagen matrix disruption and increased vascularisation [7]. The tendon continuum describes a spectrum of tendinopathy based on clinical presentation, imaging findings and histopathological findings [8]. The stages are reactive, dysrepair and degenerative tendon pathology (see Fig. 1).

## Pain in tendinopathy

Pain is the key feature of tendinopathy, and clinically, it is initiated by excess tendon load. It occurs in the presence of tendon pathology, but the converse is not true; tendon pathology can exist without pain. Hence, tendon pathology itself cannot be the sole source of pain.

Pain in the stages of the tendon continuum appears to occur in the reactive stages (reactive and reactive on degenerative pathology), possibly when the tendon cells are most aggravated. The local nociceptive driver within the tendon pathology is unknown, but it is likely that pain pathways are modulated by a spinal, peripheral or central mechanisms. A non-nociceptive pathway influenced by a dysfunctional load detection system has also been postulated [9]. Tendon pain can also manifest as allodynia, a primary hyperalgesia response and mirroring in the opposite limb occurs occasionally but only in some tendons [9].

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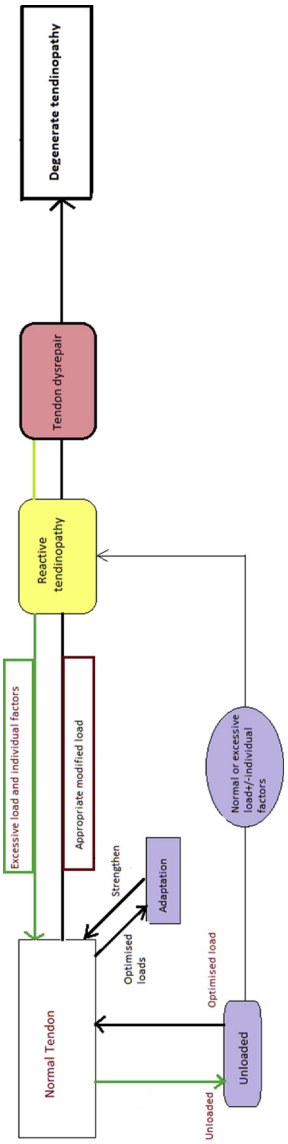


**Fig. 1.** The tendon continuum model.

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*Types of load and relation of pathology and pain*

Repetitive application of excess loads beyond the capacity of the tendon is thought to be a driver of both tendon pathology and pain. Tendons are physiologically adapted to tensile loads by nature of their fibrous structure; the most important tensile load is energy storage and release loads used in explosive activities, such as sprinting, hopping and change of direction.

Compressive loads occur at the bone tendon junction and where tendons are compressed against bony prominences, which commonly occur in gluteus medius, hamstring, Achilles insertion and adductor tendinopathy [7]. Compressive loads induce pathological change at the point of compression proximal to the tendon-bone junction. The Achilles tendon and retrocalcaneal bursa are compressed at the superior calcaneal interface with repeated dorsiflexion of the ankle. External compression from footwear and taping can also affect the Achilles tendon, compression from nearby structures, such as the plantaris tendon can also compress the mid tendon. Tensile and compression loads can occur concurrently, and their additive effect is more damaging to the tendon [10].

Frictional or shear loads are induced by repetitive movements and can result in peri-tendon pathology - e.g. repeated dorsiflexion and plantar flexion movements of the ankle joint that cause friction between the tendon and sheath.

## Risk factors in tendinopathy

Associated factors have been identified in observational studies and have yet to be tested in prospective studies that account for other variables and mediators, and thus it cannot be stated that eliminating the associated factor will eliminate the risk of developing tendinopathy [4]. Risk factors in tendinopathy include intrinsic (systemic and biomechanical) and external risk factors. Intrinsic risks factors for tendinopathy include: older age, male sex (except in greater trochanteric pain syndrome), menopause, genetics, systemic conditions such as inflammatory and autoimmune conditions, diabetes, excess adiposity and hyperlipidaemia [11–13]. Adiposity is an established risk factor and diabetes (type 2) is strongly implicated in the development of tendinopathy [11,14]. Diabetes results in glycation end product deposition in the tendon. Similarly, hypercholesteremia results in the deposition of cholesterol in the tendon, and Gout can cause uric acid crystals deposition [11]. Enthesopathy is frequently encountered in spondyloarthropathies and rheumatoid arthritis and several other systemic conditions. Common medications implicated in tendinopathy include fluoroquinolone antibiotics, aromatase inhibitors and steroids [12] (see Table 1).

The intrinsic biomechanical risk factors vary with each tendon and region. There is conflicting evidence about the implications of foot posture, flexibility and neuromuscular capacity in lower limb tendinopathies. Factors implicated in Achilles tendinopathy include limited dorsiflexion range, increased lateral foot roll during running, plantar flexion weakness, a pronated or cavus foot [15]. Pathology on imaging increases the risk of developing tendinopathy fourfold and thus is considered an intrinsic risk factor [16].

**Table 1**
Systemic conditions and medications associated with enthesopathy.

| Rheumatological Conditions | Metabolic and Endocrine Related | Drug Induced |
|---|---|---|
| Spondyloarthropathies | Diabetes | Fluoroquinolones |
| Rheumatoid Arthritis | Chronic Renal Failure | Glucocorticoids |
| DISH | Familial hypercholesterolaemia | Retinoids |
| Chondrocalcinosis | Hypothyroidism | |
| SAPO | Hyperparathyroidism | |
| | Hypoparathyroidism | |
| | Ochronosis | |
| | Acromegaly | |
| | X-linked hypophosphatemia | |

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Extrinsic risk factors are those that are in the environment or external to the individual and are often the most amenable to modification. There is a clear association between load and the development of tendinopathy [15]. The onset of tendinopathy in response to load is modulated by tendon and musculoskeletal capacity and also modified by intrinsic factors. In the athlete, it is important to identify any spike in loads, any periods of deconditioning or biomechanical changes e.g. gait retraining or change in equipment.

## Diagnosis

The diagnosis of tendinopathy in sport and exercise-related populations is based on a thorough history and physical examination that clearly accounts for differential diagnoses.

### *History*

The diagnosis of tendinopathy is primarily a clinical one [17]. Tendon pain is localised, the patient can pinpoint the site of pain using only one or two fingers. More diffuse pain suggests a different diagnosis. For example, patellar tendon pain is felt at the inferior pole of the patella, pain that distributes down or across the tendon is likely to be patellofemoral pain [18]. Tendon pain has a positive correlation and temporal association with load, with the more compressive or tensile load placed on a tendon, the higher the pain level [18]. Tendon pain is limited to activity and subsides on removal of the offending loads [18]. In the early phases tendinopathic pain improves with activity and is worse in the cool down phase. Tendon pain does not spread or become increasingly worse with little or no load (signs of central pain sensitisation) [9]. There are hallmark signs of tendon pain that must be present for a diagnosis of tendinopathy. For example, Achilles tendinopathy is associated with morning stiffness; however, it is important to note that any stiffness more than 60 min is more likely to be associated with rheumatological causes [19].

Past history of injury, especially resulting in periods of decreased loading is a risk factor for tendinopathy on return to activity. It is also important to check risk factors specifics of the sport(s) played and any change in training as this is crucial to manage modifiable factors [15]. Careful questioning of personal and family medical history is required to assess the role of systemic diseases.

### *Examination*

Localised pain that increases as the load on the tendon increases suggests tendon pain. This can be detected by recording tendon pain and load management capacity in response to a progressive loading stimulus. For example, recording pain in a double leg heel raise, single leg heel raise, double leg hop and single leg hop is the progressive examination for the Achilles tendon [20]. Some people may not be able to complete progressive loading because of pain, fear of movement or poor function. Impaired function in the muscle tendon unit and the kinetic chain should be noted. Tendinopathy will affect function in the whole limb and weakness across the limb is common. Biomechanical abnormalities and tenderness on palpation are not particularly useful clinical findings for diagnosis and are not a reliable clinical finding in tendinopathy [21]. Tendon swelling may or may not be associated with tendon pain. Tendon crepitus may be evident in peri-tendon pathology.

### *Tendon imaging*

As there is poor correlation between abnormal tendon structure and pain, imaging has almost no role in diagnosis and prognosis in tendinopathy and cannot be used as an outcome measure [22,23]. Imaging may help with differential diagnosis (identifying peri-tendon pathology), confirming tendon rupture and staging tendinopathy in the continuum. Ultrasound (including sonoelastography and ultrasound tissue characterisation) and magnetic resonance imaging (MRI) are the preferred imaging investigations (see Figs. 2 and 3). All imaging findings should be interpreted with respect to the clinical presentation [23].

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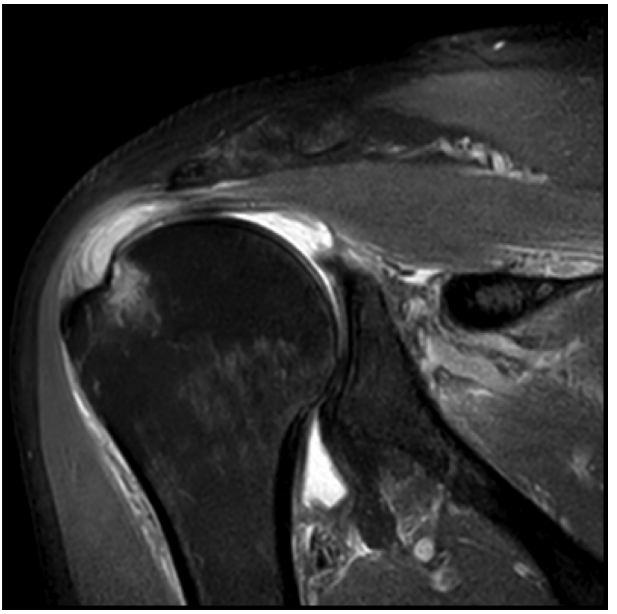

**Fig. 2.** MRI depicting supraspinatus tendinopathy with glenohumeral joint effusion and oedema in seronegative arthritis.

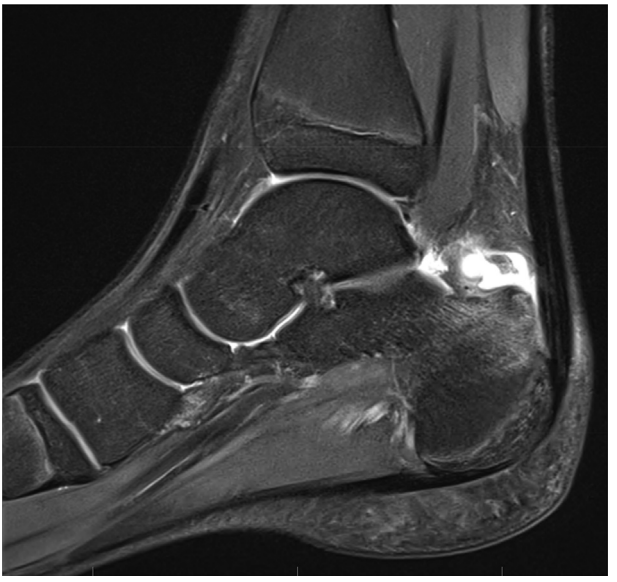

**Fig. 3.** MRI depicting inflammatory arthropathy, Achilles enthesopathy and retrocalcaneal bursitis.

*Differential diagnosis*

Neural pain and referral from adjacent joints can mimic tendon pain. In the Achilles, posterior ankle joint impingement, sural nerve irritation and pain from the medial ankle tendons can mimic Achilles

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tendon pain. Red flags include pain without loading, extended stiffness in the morning (seronegative arthropathy), night pain and poor function (undiagnosed rupture) [19,20].

The differential diagnosis of rotator cuff tendinopathy (aside from the sub-acromial bursa) is vast and includes biceps tendinopathy, acromioclavicular joint osteoarthritis or laxity, glenohumeral osteoarthritis, adhesive capsulitis, cervical radiculopathy, inflammatory arthropathy, tumours, thoracic outlet syndrome [24]. Ischaemic cardiac pain and referral of pain from the abdominal organs to the shoulder must also be considered.

## Management of tendinopathy

Tendon pain can be managed through a rehabilitation program, where the person ceases provocative activities and exercises to restore function over a longer time frame. The principles of management of tendinopathy revolve around education, load monitoring and management, an exercise-based rehabilitation program and adjunct interventions for pain.

Educating patients has several benefits including managing expectations of a quick fix, relieving anxiety about imaging findings and facilitating adherence in an exercise-based rehabilitation program. Patients should be made aware that some discomfort may be noted during the rehabilitation program, adherence is important as recovery may take months. Discussing evidence-based treatment options will help eliminate the confusion created by the plethora of treatment options available, many of which are not well supported by the evidence [25].

Exercise-based therapy forms the cornerstone of treatment over the past three decades [26]. Progressively increasing strength, endurance and functional programs incorporating the affected muscle-tendon unit and the kinetic chain are required for the affected and unaffected limb. It is important to establish goals at the beginning of the rehabilitation that are unique to the patient to ensure that the starting point of the program is appropriate, and the functional needs of the patient are met at the completion of the rehabilitation program. Progressions and protocols for each tendon and stage of tendinopathy to achieve good clinical outcomes vary between individuals. Referral to a health care professional with expertise in the area of tendinopathy who understand the needs and biomechanics of the particular sport is key.

An exercise-based rehabilitation program should form the core of therapy supplemented by interventions as required for pain relief. A conservative program should always be trialled first, and a surgical opinion reserved for cases that fail quality conservative management as outcomes after surgery for overuse tendinopathy are disappointing [27]. Exceptions where an earlier surgical opinion may be warranted are tendon ruptures and a lengthened tibialis posterior tendon.

Complete rest is contraindicated in tendinopathy management and is shown to decrease muscle power, decrease the mechanical properties of the tendon, affect the kinetic chain and lead to changes in motor cortex drive (hyper excitability and hyper inhibition) [28]. Rest will result in an immediate decrease in pain but return to loading will increase the pain because of the negative effect of unloading on the tendon, muscle, kinetic chain and brain.

It is important to identify the load volumes and types that initiate pain. These are often unaccustomed energy storage loads and/or compressive loads. These loads should be reduced, in the athletic setting, this frequently involves a change in the athlete's training sessions to avoid aggravating loads, cross training and involves a discussion with the medical team and coaching staff. Managing loads within the season always presents a challenge to the professional athlete. Daily monitoring through numeric pain scales to a loading test (hop for Achilles, single leg bridge in hamstring tendinopathy) can be used to gauge a response 24 h after activity.

Rehabilitation loads are then gradually increased to increase tendon capacity as dictated by tendon response to load. The Victorian Institute (VISA) questionnaire that has been validated for Achilles, hamstring, gluteal and patellar tendinopathy is more suited being performed monthly [13]. Aside from decreasing provocative load, pain modulation can be achieved using isometrics, which have shown to have superior efficacy over isotonic contractions in achieving immediate pain relief in patellar tendinopathy [29].

Prevention of recurrent tendon pain is a key and in active athletes requires monitoring loads, performing rehabilitation exercises in the off season, correcting biomechanical abnormalities and

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engaging the sound limb in the rehabilitation program. Metabolic control through exercise, dietary means and pharmacological intervention is also advisable in prevention. Controlling risks factors leading to metabolic conditions may also be useful means to prevent the occurrence of symptoms [11].

*Exercise based rehabilitation programs for lower limb tendinopathy*

Exercise-based tendon rehabilitation is the most evidence-based intervention in the management of tendinopathy [30,31]. Eccentric exercise dominated rehabilitation protocols for two decades following Alfredson et al.'s highly cited eccentric exercise protocol for Achilles tendinopathy [32]. However, a meta-analysis conducted in 2013 and Beyer et al.'s randomised controlled trial (RCT) in 2015 that compared eccentrics to other progressive loading programs showed that there was limited evidence to support eccentrics as a standalone treatment [32]. Eccentrics, however, are integrated with concentric exercises throughout most loading programs.

Heavy slow resistance (HSR) exercises aim to induce collagen synthesis and can enhance the mechanical stiffness of the tendon [33] and were shown to have similar good efficacy compared to eccentric exercises for Achilles tendinopathy in a randomised trial by Kongsgaard et al. that was sustained at 12 and 52 weeks [34]. Heavy slow resistance exercises have similar efficacy for treating patellar tendinopathy. In a RCT comparing HSR, corticosteroid injection (CSI) and eccentric squat over a 12-week period, HSR provided the greatest patient satisfaction at 6 months [33].

Both isotonic and isometric exercises have been shown to induce immediate pain relief following a single bout of exercise but only isometric exercises were shown to sustain the analgesia effect after 45 min in patellar tendinopathy [29]. In a follow up RCT, a greater improvement in pain scores was noted for isometrics at 4 weeks for the treatment of patellar tendinopathy over isotonic exercise [35], and both forms of exercise were shown to reduce pain levels in patellar tendinopathy in−season [31]. A systematic review conducted by Lim et al., in 2018 investigating the effects of isometrics, eccentric or slow resistance exercises on pain and function in individuals with patellar tendinopathy concluded that isometrics offered more benefit in terms of offering short-term pain relief especially useful in athletic competition, whilst heavy slow resistance or eccentric exercises demonstrated long-term pain relief and improvement in knee function [36].

The stages of exercise-based rehabilitation outlined below can be adapted to all lower limb tendinopathies and tailored to suit the person's current and required function. The program involves progressive loading and will decreases the overall load on the tendon and consequently reduces pain.

*Stage 1 isometric exercise*

Isometric exercise relieves tendon pain and can be used before provocative activities. These exercises also relieve cortical inhibition, and are useful before strength work to allow maximal muscle strength. To achieve pain reduction a sustained isometric muscle contraction in mid to inner range of movement (to minimise compressive loads) should be held for 30−45 s and repeated five times. A 2-min rest is necessary between each isometric contraction to allow muscle and brain recovery. For many people with high levels of pain and low levels of function, isometric exercises can be performed using body weight resistance as a home-based exercise program and can be repeated more than once a day. Isometric loads can be progressed from double leg, to single leg to using free or machine-based weights.

When pain is controlled, progression to the next stage is recommended, usually within a week. Isometrics can be continued throughout the rest of the rehabilitation program, performed prior to isotonic exercise or in-season prior to training in an athlete to manage tendon pain.

*Stage 2 isotonic and heavy slow resistance exercises*

Isotonic exercises improve muscle strength and tendon stiffness. These exercises do not reduce cortical inhibition; the use of isometrics prior to the isotonic session may improve strength gains [29]. Isotonic exercises should be completed slowly; targeting the affected muscle tendon unit in isolation (e.g. calf raises and leg extensions). These exercises should be done on alternate days and involve 4 sets of 6−8 single leg repetitions [37].

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Muscle tendon units below the affected tendon must also be loaded in this stage of rehabilitation, as calf muscle function (strength and endurance) is critical for all lower limb tendinopathies. It is important to address all weak components of the kinetic chain and the other limb and should be based on the assessment findings.

This stage of exercise should be pain free for the tendon, although some muscle soreness is possible. Once a strength base is established and depending on the ultimate goals of the individual, additional endurance components can be added.

The motor cortex may be affected by increasing the eccentric to concentric ratio (slightly longer eccentric phase) as this is shown to increase neuroplasticity [38]. Self-paced exercises with external cues (visual or auditory) can also positively affect the motor cortex. Strengthening the other limb for cross-education effects is also recommended (loading one muscle results in strength gains on the contra lateral side).

Progression to the next stage is recommended when adequate strength and endurance are achieved in the affected muscle and kinetic chain deficits improve; a process that can take up to 12 weeks [37].

*Stage 3 increase in speed and energy storage exercises*

A gradual increase in faster functional movements 2–3 times a week should be commenced whilst maintaining a base of isometrics and strength and endurance exercises on alternate days. These exercises should be done with body weight only and involve whole kinetic chain movements.

Monitoring of load response is a key at this stage as speed adds significant load on the tendon. The response of the tendon to a load (e.g. hopping) or a hallmark sign (morning pain and stiffness) the day after faster loads will clearly demonstrate if the tendon is tolerating the load. An increase in pain or stiffness indicates that the load has been too much and must be adjusted for the next loading session. Progression to the next stage is dependent on the capacity of the tendon to tolerate faster loads.

*Stage 4 energy storage and release or sport specific exercises*

Restoring the elastic properties of the kinetic chain is dependent on the functional and sporting needs of the person. Sports specific drills should be slowly introduced and progressed until return to sport specific activities are achieved.

Energy storage and release or sport specific exercises can replace stage 3 exercise, but stage 1 and 2 exercises should be maintained twice a week. As an example, rehabilitation can follow a 3-day cycle with energy storage and release exercises on day 1, cross training on day 2 and isotonic exercise on day 3 [13].

## Management of Achilles tendinopathy

Achilles tendinopathy is the most prevalent of all lower limb tendinopathies and can occur in the elite athletic population (lifetime occurrence of 50%) and the sedentary population (5.9% of lifetime cumulative incidence) [39]. The Achilles tendon succumbs to entheseal, mid tendon and peritendon pathology [40], in addition a plantaris tendon, seen in approximately 90% of the population, can influence Achilles tendon biomechanics and pathoetiology.

### *Aetiology of achilles tendinopathy*

The relative thickness and strength of this tendon compared to other tendons makes it well-suited to withstand the loads of a weight bearing and locomotive tendon that is subjected to high energy storage loads like sprinting and jumping [15]. Achilles tendinopathy is predominantly an overuse condition; the loads that precipitate pathology and pain are energy storage (jumping and sprinting), compression (hill running) or friction loads (high volumes of repetitive ankle movements).

### *Clinical presentation of achilles tendinopathy*

Mid Achilles tendinopathy pain is located to a spot between 2 and 7 cm proximal to the insertion, whereas enthesopathy occurs just proximal to the tendon insertion onto the calcaneus [6]. Midportion and insertional Achilles tendinopathy differ in clinical presentation and the treatment modalities.

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Peritendon pathology can extend over the tendon. Atypical pain should prompt questioning and examination for inflammatory conditions.

*Examination of achilles tendinopathy*

Key clinical features include localised pain, increased by energy storage and/or compressive loads. Decreased calf strength and endurance are common. A stiff cavus foot and ankle, crepitus in peritendonitis and the presence of a Haglund's morphology may be apparent. Achilles tendon rupture must be considered in an acute presentation. Differential diagnoses are superficial calcaneal bursitis, sural nerve related pain, accessory soleus, posterior impingement and flexor hallucis longus and tibialis posterior tendinopathy. Investigations are reserved for atypical presentations in a non-responsive tendon.

*Management of achilles tendinopathy*

The principles of rehabilitation apply to the management of Achilles tendinopathy; the four-stage program outlined above is an effective approach. There are several additional clinical management points to consider. Insertional Achilles tendinopathy will respond best to the removal of compression with substantial heel lifts (3–4 cm) for a minimum of 6–8 weeks. Check the patient's footwear to ensure that compression at the back of the heel and avoiding a high heel counter. Peritendon pain and pathology can be achieved through topical application of heparinoid and non-steroidal anti-inflammatory creams. A systematic review and meta-analysis reviewed the effects of loading protocols in mid Achilles tendinopathy including heavy eccentric calf training, concentric training, eccentric overload training and heavy slow resistance training demonstrated improvements in the VISA-A scores at 2-weeks since introduction of the program with a peak in 12 weeks but the mechanism of this change warrants further research [6].

## Management of rotator cuff tendinopathy

Shoulder disorders are a leading cause of pain and disability in society with 1 in 3 people experiencing shoulder pain at some stage in their life [41]. Recurrence is common, and symptoms are often persistent with 40%–50% of patients reporting on-going morbidity after 6–12 months [41]. Subacromial pain syndrome (SPS) refers to a complex clinical presentation of shoulder pain and dysfunction with symptoms considered to arise from a number of shoulder pathologies associated with the soft tissues occupying the subacromial space, particularly the rotator cuff tendons and subacromial bursa [42]. The syndrome has been described by a number of terms, more recently, 'subacromial pain syndrome' or 'rotator cuff related shoulder pain' [43]. These changes in terminology reflect historical debate regarding the mechanisms and tissues involved in rotator cuff tendinopathy and SPS, as well as evolving scientific opinion about the pathogenesis of tendon disorders [44,45]. SPS has been shown to be the most prevalent upper extremity disorder seen in working populations [46], associated with work related physical demands, particularly those involving repetitive and forceful postures [47]. Estimates of prevalence and incidence vary widely; a recent systematic review [48] reported overall incidence ranging from 0.3% to 5.5% and annual prevalence from 0.5% to 7.4%. Prevalence is correlated with increasing age and reported to occur in approximately 15% of those aged over 70 years [49].

*Anatomy, biomechanics and aetiology of rotator cuff tendinopathy*

The rotator cuff tendons lie within the confines of the subacromial arch, a potential source of compression. Reduced rotator cuff muscle activation and rotator cuff fatiguing protocols [50] have been associated with superior migration of the humeral head in healthy people [51]. Superior migration of the humeral head narrows the subacromial space creating the potential for tendon compression [51] on the at-risk, articular side of the rotator cuff tendons.

Many extrinsic and intrinsic factors may contribute to the development of rotator cuff tendinopathy [45]. Extrinsically, the rotator cuff tendons and/or bursa are subjected to repetitive compression and

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abrasion from anatomical structures within the subacromial space, causing progressive pathological change [52]. Intrinsically, rotator cuff tendinopathy may be associated with individual risk factors and tendon response to overuse [45]. Intrinsic mechanisms may alter mechanical loading properties in turn altering shoulder kinematics with consequent muscle strength/length deficits.

*Pathology of rotator cuff tendinopathy*

The histopathology in the rotator cuff is similar to other tendons. The consequences of tendon thickening, however, may be more poorly tolerated given the anatomical constraints of the tendon within the sub acromial space. Slowed return to baseline dimensions of the thickened tendon compared to normal tendon has been shown with loading to fatigue [53].

Ossification of the attachment of the coracoacromial ligament is seen in patients with rotator cuff related shoulder pain. It is not clear whether impingement of the thickened tendon on the overlying coracoacromial ligament at its attachment to the acromion has any role in the development of this ossification. The reverse consideration, whether coracoacromial ligament ossification and impingement on the tendon or intrinsic tendon disease is causative for tendinopathy remains contentious. The literature supports multifactorial and variable etiologic factors in rotator cuff tendinopathy. Additionally, it is not possible to say which anatomical structure is responsible for pain when it is present as rotator cuff tendinopathy and tears are observed in a large number of asymptomatic shoulders [54].

*Examination of rotator cuff tendinopathy*

Clinical diagnosis of SPS is based on patient history and a physical examination, supported by imaging. Tests purported to evaluate SPS are classified as pain provocation and rotator cuff integrity tests with some authors suggesting that such tests can be used to rule SPS in or out [55]. Traditionally the Neer's, Hawkins-Kennedy and painful arc tests were advocated as diagnostic of SPS [42], though no single test shown to be sufficiently sensitive or specific to definitively determine the presence of this disorder [55,56]. Using test results in combination is thought to improve accuracy of diagnosis in SPS [56]. A combination of the Hawkins-Kennedy, the painful arc sign, and the resisted external rotation test has been considered to yield the best post-test probability (95%) for any degree of SPS, that is, a patient who tested positive on all of the above three tests had a 95% chance of having some degree of SPS. Conversely, if all three tests were negative, the likelihood of having SPS was less than 24% [56].

*Imaging of rotator cuff tendinopathy*

Imaging for rotator cuff tears has been shown to be poorly correlated with pain, many asymptomatic individuals demonstrate rotator cuff tendon pathology [43]. Imaging has been shown to be sensitive mainly for full-thickness rotator cuff tears [57]. Routine diagnostic imaging for musculoskeletal conditions yields little to no benefit and shoulder pain presentations are over-investigated [43]. Imaging for SPS is indicated only if there is suspicion of a red flag condition, fracture, dislocation or inflammatory arthropathy [58].

*Management of rotator cuff tendinopathy*

Current management options for SPS involve conservative management (physiotherapy/exercise), injection therapy and surgery (open or arthroscopic subacromial decompression). Clinical effectiveness of subacromial decompression surgery shows no benefits compared to placebo surgery [59] or simple bursectomy [60], likely because of the important role of intrinsic mechanisms of this disorder [45]. Rotator cuff tendon failure and individual risk factors are not altered by surgery; neither are the recognised biomechanical risk factors including aberrant scapular and glenohumeral kinematics and muscle imbalances or postural deficits [45].

Exercise has been shown to be as effective as surgery at 1 and 10 year follow up [61]. Further, Holmgren et al., in 2012 in their RCT of 102 patients with chronic SPS, on a waiting list for surgery, reported that a structured exercise program reduced the need for surgery in 80% of their cohort [61].

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There is conflicting evidence on the value of manual therapy and exercise for managing rotator cuff tendinopathy [62]. A tendon loading program that involves progressively restoring tensile strength can lead to improvements in tendon function and pain. Intuitively, programs that aim to normalise movement patterns with the goal of optimising the loading environment of the tendon are likely to have better outcomes, particularly in SPS where the complex interplay of the scapulo-thoracic and glenohumeral articulations is frequently interrupted [63]. Although eccentric exercises have been advocated for the treatment of tendinopathy, there is conflicting evidence to show superior outcomes when compared with other loading programs [64]. Isometrics are gaining traction for use in the management of SPS [41] and findings from a small pilot study suggest that low-load isometric exercises may positively influence pain and tendon thickness of the rotator cuff [65].

CSI, prolotherapy injection therapies, plasma-rich platelet (PRP) and mesenchymal stem cell therapies have all been described in the treatment of SPS. CSIs are widely used, and a survey practitioner found that 96% believe CSIs are efficacious in managing SPS [66] and that these injections may facilitate engagement with physiotherapy [67]. A recent meta-analysis investigating the effects of CSI on SPS identified no difference between CSI and placebo after 12 weeks [68] and no benefit from multiple injections. The use of PRP in the management of rotator cuff pain is also common, although there is no evidence of its effectiveness over placebo injection in rotator cuff tendinopathy [69] and some evidence that these injections might also negatively impact rotator cuff tendon tissue [70].

## Adjuncts to exercise therapy

Although exercise therapy is best supported in literature, adjuncts may be required as supportive treatment and pain-relieving measures and in cases of unsatisfactory clinical progression. The adjuncts have been categorised as electrophysical agents, medications, injectable therapies, passive treatments, experimental treatments and surgery.

Each adjunct as mentioned below has a proposed mechanism of action in tendinopathy, preferred criteria for use and risk profile. Consider using the most evidence-based treatments with a low risk profile that are suitable for the person and tendon, avoiding experimental procedures where the risk profile is unknown.

### *Electrophysical agents*

#### *Extracorporeal shock wave therapy*

The mechanism of action of extracorporeal shock wave therapy (ESWT) is not fully understood although a few theories have been postulated for its action including regeneration of tendon tissue and an analgesic effect when applied at a low dose. The efficacy correlates with the frequency used and has a dose dependent response with the number of treatment sessions [71,72].

A review of literature done by Van Leeuwen in 2008 found that the ESWT treatment is generally safe and showed promise in the treatment of elbow tendinopathy, rotator cuff tendinopathy and plantar fasciitis. However, the small number of studies that were evaluated differed in protocol, methods and quality, thus recommendations on a suitable protocol could not be established [71]. A systematic review done by Schmitz et al., in 2015 reported that an optimal protocol would be 2000 impulses per session spread over 3 sessions a week apart at the highest patient tolerated flux density [73]. Neither radial of focused therapy was reported to be superior in this systematic review, although it does show superior outcomes in calcific tendinopathy rather than non-calcific tendinopathy [72]. Further high quality studies are required to determine an efficacious treatment protocol and currently evidence states that ESWT is generally safe and has better efficacy in combination with an exercise program [74].

#### *Ultrasound, transcutaneous electrical nerve stimulation, pulsed electromagnetic field and extracorporeal shock wave therapy*

Dingesmase et al., 2012 investigated the efficacy of other electrophysical modalities (i.e., ultrasound, transcutaneous electrical nerve stimulation, low-level laser therapy, pulsed electromagnetic field therapy and extracorporeal shock wave therapy) [75]. This systematic review explored and evaluated 2

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reviews and 20 RCTS. Of the modalities considered, only laser and pulsed ultrasound demonstrated short-term pain relief and improved function [75].The need for higher quality studies is essential to determine if electrophysical agents have a role in managing tendinopathy.

*Medications*

*Non-steroidal anti-inflammatory medications*

Inflammation is not a strong feature of tendon pathology, however non-steroidal anti-inflammatories (NSAIDs) may have a non-inflammatory mechanism of action [76] and may provide a short-term (7–14 days) analgesic effect [77]. Gastrointestinal and cardiovascular risk factors will determine safety of non-selective and selective NSAIDs. Commonly used NSAIDs other than Ibuprofen may adversely affect tendon healing at the bone-tendon junction (rat patellar tendon) thus their use post injury or surgical repair should be limited [78]. The short-term use of topical NSAIDs through gel formulation or patch application can minimise systemic side effects, efficacy is comparable to oral NSAIDs. Minimal erythema at the site of application can occur.

*Triple therapy*

The combination of ibuprofen, doxycycline and the addition of green tea or omega 3 fatty acids as a 'poly pill' approach has been suggested [76]. Doxycycline inhibits metalloproteinases, prevents collagen and matrix breakdown and may be potentiated by ibuprofen. The addition of green tea and omega fatty acids could maximise the effect by TNF alpha inhibition. The regimen includes 400 mg of Ibuprofen thrice daily, 100 mg of doxycycline once a day for a period of 14–28 days [76]. The common side effects of doxycycline are gastrointestinal disturbances side effects and photosensitivity [79].

*Glycerol trinitrate patches*

Glycerol trinitrate patches releases nitric oxide, which may promote collagen synthesis [80]. Randomised double-blinded placebo-controlled trials have demonstrated these patches improved clinical outcomes in elbow, non-insertional Achilles and rotator cuff tendinopathy [81]. Conversely, these patches showed no benefit over placebo or an eccentric exercise programme in patellar tendinopathy [82,83]. The treatment appears to be beneficial in chronic tendinopathies over acute and subacute tendinopathies [84]. The dosage used in these intervention studies was 1.25 mg/24 h (i.e. 1/4th of a 5 mg/24-h patch) applied over the most tender area, and the period of use has varied from 3 days to 24 weeks [83,84]. The most common side effects of this therapy are headache and contact dermatitis, and the treatment is contraindicated in patients with ischaemic heart disease or using sildenafil citrate [83].

*Injectable therapies*

Injectable therapies include CSI, PRP, autologous blood injections, autologous cell injections, sclerotherapy and prolotherapy. There is strong evidence to support the use of radiological guidance during these injections as it improves accuracy but does not prevent distribution to other tissues [85,86].

*Corticosteroid injections*

CSIs have been used as a short-term pain-relieving medication, however they have shown to have detrimental effects on tendon pain and function in the following 6–12 months period [86,87]. A systematic review of 50 investigations involving both animal and human participants concluded that local CSI produced significant negative effects on tendon cells in vitro, including reduced cell viability, cell proliferation, and collagen synthesis [88]. Increased collagen disorganization and necrosis as well as reduced mechanical properties were also identified.

Steroids commonly injected around a tendon include dexamethasone, triamcinolone, methylprednisolone and hydrocortisone, and vary in crystalline properties, dosage and onset and duration of action. Intratendinous CSI are not recommended due to the potential for tendon rupture [83,89]. Steroid injections have also been used frequently for bursitis and tenosynovitis. It is common practice

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to mix local anaesthetic with the steroid preparation and the complications of the anaesthetic have also to be considered. The upper limit of injections per year has not been studied.

Patients have traditionally been advised to rest from vigorous activities from a range of 3 days to 3 weeks and this time is largely anecdotal. Education that CSI provide short-term pain relief to achieve stable pain levels to commence an exercise rehabilitation program is essential. Risks of use include skin flush and discolouration, infection, tendon rupture, nerve and vessel trauma, tissue atrophy and systemic side effects.

Ultrasound-guided saline and CSI in the pre-Achilles fascia are proposed to strip the nerves and vessels. A mixture of 10 ml of 0.5% bupivacaine, 25 mg of hydrocortisone acetate and up to 40 ml of normal saline resulted in pain reduction, improvement in function and VISA-A scores at the 30 week and a 12-month mark post injection [89]. However, a recent study in Achilles tendinopathy showed similar good clinical outcomes at 10 weeks through use of a high-volume saline injection without steroid [90].

*Platelet rich plasma and autologous blood injections*

Autologous blood injections into an affected tendon (with or without imaging guidance) are aimed at increasing the concentration of growth factors to promote tendon healing. To date, there is limited evidence to support its use [91]. The side effect of a pain flare thought to be due to the presence of red blood cells; infection and the need for multiple injections are also considered drawbacks. The use of repeated injections is also common practice but there is no evidence that this practice increases efficacy.

Platelet-rich plasma (PRP) injections (leucocyte rich or poor PRP) [92] were designed to concentrate platelets believed to contain important growth factors and cytokines to promote tendon healing. There is no high-quality evidence to support its use [93,94]. There is some evidence to suggest that PRP may have a detrimental effect on animal [95] as well as human [70] tendon tissue. The addition of corticosteroid and anaesthetics alone or in combination to PRP was shown to decrease tendon viability and proliferation in vitro studies [96].

*Prolotherapy and sclerotherapy*

Prolotherapy involves the injection of an irritant e.g. hyperosmolar dextrose and saline to stimulate a tendon inflammatory response. Sclerotherapy involves the injection of Polidocanol to target neovascularity in the tendon. A systematic review and meta-analysis showed some promise for both sclerotherapy and prolotherapy as safe and effective treatment modality in Achilles tendinopathy; however, methodological limitations have been acknowledged [97]. Prolotherapy was not superior to a physiotherapy program or in combination in lateral epicondylitis [98]. The side effects of sclerotherapy in Achilles tendinopathy include embolia cutis medicamentosa, and triceps surae lesions [97].

*Iontophoresis*

Iontophoresis involves the delivery of corticosteroid *trans*-dermally by means of a lower level electric current to enhance absorption with a dosage up to 5 mA and a session lasting up to 30 min, with dexamethasone being the most frequently used corticosteroid, sometimes combined with Lidocaine [99]. The therapy is best suited for superficial tendons and associated bursitis and is aimed at inducing pain relief. The therapy requires multiple sessions and has a low side effect profile of mid tingling or discomfort during the procedure and skin irritation. A systematic review by McKivigan et al. concluded that there was insufficient evidence to support its use in the treatment of epicondylitis, although should not be ruled out in treatment as an alternative to oral therapy due to the dose response effect [99]. The authors recommend that it may be used as an adjunct due to its analgesic effect and low side effect profile, in combination with an exercise program but not as stand-alone therapy.

*Passive treatments*

Treatments like ice, stretching, acupuncture, deep frictional massage, have been shown to have little efficacy [17,28,77]. Stretching is now believed to worsen compression and deep frictional massage can

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provoke pain with no gains in tendon function and should thus be avoided [28].The use of heel raises can be beneficial in mid and insertional Achilles tendinopathy by reducing compression.

*Experimental treatments*

Hyaluronic acid, autologous tenocyte/fibroblast implantation and other cell-based therapy, growth factors, stem cells and botulinum toxin have unknown effects [100–103].

*Surgery*

The indications for tendon surgery involve the exhaustion of non-surgical options and involve procedures, such as stripping, debridement of the devitalised tissue, decompression, tenodesis and tendon transfers. Surgical outcomes are variable depending on the procedure being performed and the tendon region involved [104].

A critical review of the outcomes of surgery for chronic Achilles tendinopathy found an inverse correlation between surgical success rates and the methodological quality of the study [104]. The authors recommend the need for higher quality studies to understand the role of surgery in managing tendon pain. Specific methodological principals that should be addressed include; prospective design, investigators independent to surgeons, definite patient selection, documentation of post-operative rehabilitation programs, operative histology and imaging, and use of pain and return to sport outcomes [104].

Plantaris tendon may have a role in Achilles tendinopathy; a prospective consecutive case series in 32 elite athletes with mid Achilles tendinopathy had their Plantaris tendon excised along with a ventral Achilles tendon debridement, which resulted in symptomatic improvement and function and early return to high level sport [105].

In patellar tendinopathy, a systematic review reports a lower side effect profile and quicker return to sport with arthroscopy over open surgery [106] although this can mean a time frame of around 6–12 months and there may be deficits in full function on return to sport [107].

## Prevention

The pathogenesis of tendinopathy is multifactorial and complex. Although potential risk factors for tendinopathy have been identified, few such as excessive load, systemic conditions like metabolic disease have a strong association. Majority of risks factors have a limited support base and are often not amenable to modification. It appears thus that the most efficacious means to prevent tendinopathy is careful load management during activity and sport. A neuromuscular soccer specific program was tested in a RCT involving professional soccer player and was found to be efficacious in reducing the risk of Achilles and patellar tendinopathy; however, the results were not replicated when an eccentric program was tested in the same sport [15].It seems logical that control of systemic disease would prevent the onset of tendinopathy. Further research is required to determine if modifying these systemic risk factors could prevent tendinopathy [11].

## Summary

Current research demonstrates that tendinopathy pathoetiology is complex. Clinical diagnosis is a key with imaging showing poor correlation to pain and function. Complete rest is detrimental to tendon and a supervised gradual loading program is the most evidence-based approach to managing tendon pathology. There has been a surge in the number of adjunct treatments available, most of which have inadequate evidence to back their use. The interplay between pathology and pain and why some pathological tendons develop pain or rupture is unknown. The intricate pathway between local nociceptive and centrally mediated pathways needs to be studied further. At this stage, an optimal loading program for any stage of tendinopathy has not been developed.

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**Practice points**

- A detailed history of change in loads, technique or biomechanics is essential to addressing contributing factors in tendinopathy
- Be aware that tendinopathy can be a manifestation of systemic disease
- Imaging findings often poorly correlate with the clinical picture and should be reserved for circumstances when a differential diagnosis is suspected or poor response to treatment
- Exercise-based rehabilitation is the key for long-term relief of pain
- Educating the athlete is key for buy in, especially considering the lengthy times to rehabilitation

**Research agenda**

- Larger double blinded placebo controlled RCTs are required to test therapeutic interventions in tendinopathy management
- A gold standard test for the diagnosis of tendinopathy or diagnostic criteria and definitions of tendinopathy need to be established with a consensus view of experts in the area
- The newer imaging modalities require validation for improving diagnosis and prognosis
- The pathophysiology of tendon pain and the role of local and central pathways are essential to explore as pain is the presenting and persistent clinical presentation
- The optimal loading program and protocol for a particular tendon and stage needs to be determined by examining the efficacy of further randomised trials of exercise interventions and measure local tendon, muscle and neural changes that contribute to the improvement in pain and function

## Conflict of interest

There are no conflicts of interest to declare.

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