

Sports and Exercise-Related Tendinopathies

A Review of Selected Topical Issues by Participants of the Second International Scientific Tendinopathy Symposium (ISTS) Vancouver 2012

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Br J Sports Med. 2013;47(9):536-544.

Abstract and Introduction

Abstract

In September 2010, the first International Scientific Tendinopathy Symposium (ISTS) was held in Umeå, Sweden, to establish a forum for original scientific and clinical insights in this growing field of clinical research and practice. The second ISTS was organised by the same group and held in Vancouver, Canada, in September 2012. This symposium was preceded by a round-table meeting in which the participants engaged in focused discussions, resulting in the following overview of tendinopathy clinical and research issues. This paper is a narrative review and summary developed during and after the second ISTS. The document is designed to highlight some key issues raised at ISTS 2012, and to integrate them into a shared conceptual framework. It should be considered an update and a signposting document rather than a comprehensive review. The document is developed for use by physiotherapists, physicians, athletic trainers, massage therapists and other health professionals as well as team coaches and strength/conditioning managers involved in care of sportspeople or workers with tendinopathy.

Introduction

As mechanical loading plays such a key role in the development, and rehabilitation, of many cases of sports-related tendinopathy, the distinct structural and functional adaptations and loading environments of tendons at different anatomical locations are important.^[1] Lower extremity tendons, such as the Achilles, store and release substantial amounts of tensile energy,^[2,3] whereas gliding tendons such as those at the wrist demonstrate specific adaptations to resist primarily frictional loading, such as retinaculae or synovial sheaths, whose function can be affected by injury.^[4] Thus, tendons are distinct and varied in their loading requirements.

The structure and function of tendons have been well described elsewhere.^[5-7] We alert the reader to recent findings regarding the scleraxis gene.^[8] Tendon cells (tenocytes) are characterised by their expression of scleraxis, both in developing tendon and ligament, as well as in adult human tenocytes. As expected, scleraxis expression is mechanically regulated, showing a reduction following tendon transection^[9] and exhibiting a dose-response with increasing strains or repetitions of movement.^[10] Scleraxis expression is increased during the repair and remodelling stages of tissue healing, as the tendon attempts to restore its phenotype—this attempt to restore normal tendon phenotype following injury is frequently imperfect, leading to metaplastic or fibrotic change in injured tendon.

Dynamic tissues like tendon shift their anabolic/catabolic balance according to their mechanical loading history.^[11] Emerging evidence suggests that local production of classically neuronal modulators, like neuropeptides, by tenocytes in response to load may regulate local tissue remodelling,^[12-14] in addition to their role in nociception. The tendon's surroundings are richly innervated by mechanoreceptors, including Ruffini corpuscles, Pacinian corpuscles and free nerve endings, all of which may contribute both to proprioception and to nociception.^[15] The nerve supply of tendon also includes many autonomic fibres, likely involved in regulating tendon blood flow as well as local tenocyte metabolism and pain signalling.^[16]

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Pain and Pathogenesis: International Scientific Tendinopathy Symposium 2012 Update

The past decade has seen impressive strides in our knowledge of pathological processes which underpin the development of chronic tendon pain. The pathology of tendinosis—in which the structural degeneration of the load-bearing matrix is a key feature, with an absence or minimal presence of inflammatory cells—has been confirmed in several recent studies. The opening session of the International Scientific Tendinopathy Symposium (ISTS) 2012 focused on advances in knowledge of chronic tendon pain.

In tendinopathies, tenocytes produce nociceptive as well as inflammatory/catabolic substances.^[18 19] These are induced through repetitive mechanical loading as seen both *in vitro* and *in vivo*.^[13 20] Whether the release of these substances is associated with the perception of pain is unknown. Nerve fascicles containing sensory afferents are present in tendons and especially in the peritendinous tissue; these nerves express receptors for the nociceptive substances which could thereby sensitise the nerves and augment pain signalling.^[18]

Relatively little is known about the pathogenesis of tendon pain in the early stages; tendinopathic changes in tendon are typically progressive, yet frequently asymptomatic. Many patients present themselves to physicians and physiotherapists when they are symptomatic; this presentation may be precipitated by a temporary increase in tendon loading. In a proportion of patients, the symptoms will inevitably settle spontaneously, only to recur later; thus, a cyclical pattern of symptoms and remission is not uncommon. This natural history of the condition needs to be factored into randomised clinical trials and laboratory studies as otherwise there might be a bias in the interpretation of findings (eg, if the trials require a change in usual activity levels, particularly a reduction of load).

In the following section, we briefly summarise recent findings regarding the pathogenesis of chronic tendinopathy at various anatomic locations.

Achilles

Sweden's Professor Alfredson presented data suggesting that unilateral treatment of patients with bilateral Achilles tendinopathy (using a minimally invasive scraping procedure) can lead to reduction or resolution of symptoms both on the operated and non-operated side.^[21] This is in keeping with the results cited below for other tendons, highlighting a potentially underappreciated role of the central nervous system in generating tendon pain and, potentially, tissue pathology and abnormal movement patterns. Alternately, it may be that when one tendon is treated, both tendons undergo a period of relative rest during the recovery period leading to bilateral improvement.

Patellar

In an observational study of athletes with patellar tendinopathy, van Wilgen *et al*^[22] reported the occurrence of reduced mechanical pain thresholds and pinprick allodynia in patellar tendinopathy patients, which was interpreted as reflecting the involvement of central sensitisation of myelinated (Ad-fibre) input. In a separate study, the Victorian Institute of Sport Assessment (VISA)-P score correlated inversely with the presence of sulfated glycosaminoglycans, demonstrating that, although the nervous system plays a modulatory role, tissue pathology is clearly related to the pain and functional deficits of tendinopathy (ISTS abstract, *in press*).

Rotator Cuff

A recent systematic review summarised the main findings from both human studies and animal models of rotator cuff overuse.^[23] The paper concluded that 'intrinsic, extrinsic and environmental factors all have an important role to play in the disordered tendon homeostasis of rotator cuff disease, which can lead to progressive mechanical failure'. The paper described a number of degenerative mechanisms, some related to classic inflammatory pathways (eg, interleukin-1, substance P (SP)) and others related to altered loading conditions (eg, impingement) or systemic influences (eg, ageing). The paper noted a disconnection between the amount of degenerative change and the extent of symptoms.

Elbow

Vicenzino presented data from a series of sensory motor system studies that implicated the role of the central nervous system in lateral epicondylalgia (LE). Our knowledge of LE pathology has advanced little since the seminal work by Kraushaar and Nirschl;^[24] however, Coombes *et al*^[25] have presented an

integrated model in which tissue pathology interacts with the nervous system to cause widespread mechanical hyperalgesia and motor control deficits. The model is proposed to identify subsets of patients who may have more substantial pain or motor system deficits, as these patients may respond differently to clinical intervention.

Diagnosis and Imaging

Tendinopathy is a syndrome of tendon pain and thickening—the diagnosis is based primarily on patient history and physical examination. However, Fredberg *et al* [26] reported a high level of misdiagnosis for Achilles and patellar tendinopathy, despite receiving referrals from orthopaedic departments. The role of clinical tests (eg, palpation tenderness for patellar tendinopathy) has been questioned as palpation is highly sensitive at reproducing symptoms, yet not specific in accurately determining the painful/pathological structure.^[27] Given the new findings reported above, mechanical hyperalgesia may be reducing the presumed specificity of many commonly used clinical tests. Ultrasound (US) and MRI are used in the clinical setting to confirm the presence and location of tendon thickening or other structural change and associated findings.

The choice of US or MRI has traditionally been determined by the clinician based on personal preference and experience rather than on evidence-based guidelines. A number of studies have evaluated the accuracy and sensitivity of US (0.63–0.83 and 0.68–0.87, respectively)^[28–32] and MRI (0.68–0.70 and 0.50–0.57, respectively)^[29 31 33] in detecting tendinopathy in different tendons. Direct comparison between the two modalities has shown US, in trained hands, to be more accurate than MRI due to the superior spatial resolution of US imaging.^[29 31 34 35] These studies used clinical diagnosis as the yardstick in determining the accuracy and sensitivity. Paavola *et al* [36] reported that US imaging had correctly diagnosed 83% of surgically confirmed Achilles peritendinitis/tendinopathy cases. US with Doppler also provide the advantage of being able to visualise the areas of increased blood flow.

At ISTS 2012, the value of US imaging in achieving a more specific diagnosis was apparent in a retrospective study of 143 US scans (ISTS 2012 abstract, in press). Although the proximal pole of the patella was, as expected, the most common location of pathology (present in 71% of tendons), pathology was also frequently observed at the distal pole (38%), sometimes in conjunction with other locations, but more commonly, as the sole location. Mid-substance pathology always occurred in association with involvement of either the proximal or the distal insertion. This level of anatomic specificity in diagnosis is clearly of value when tendon-directed treatments (eg, injections or exercise) are being considered.

However, the phenomenon that imaging abnormalities do not necessarily signal the presence of clinically significant symptoms has been well established. Cook *et al* [37] reported that 22% of elite athletes demonstrated pathological lesions within the patellar tendon, despite the absence of anterior knee pain. Similar cross-sectional studies using MRI in rotator cuff injury have shown structural abnormalities at similar frequencies in symptomatic and age-matched controls.^[38] Importantly, the presence of asymptomatic structural abnormalities within tendons identified using imaging has been shown to increase the risk of developing pain.^[39 40]

With regard to the rotator cuff, early studies indicated a progressive course of rotator cuff tendinopathy.^[41 42] However, a recent study reported a low risk for tear progression in small, symptomatic supraspinatus tears (25% in 3.5 years).^[43] The progression of rotator cuff tendinopathy is associated with increased symptom manifestation.^[44 45]

Despite their clinical utility, both US and MRI are subject to artefacts and typically yield poor interobserver and intraobserver reliability.^[28 34] Investigators are developing more reliable and sensitive methods of quantitative tendon imaging, but these methods are, at this point in time, more relevant for research than for clinical practice. At ISTS 2012, two emerging methods for analysing and quantifying grey-scale US images were presented. De-Groot-Ferrando and colleagues (ISTS 2012 abstract, in press) described a reliable method to quantify morphometric parameters as well as textural features such as contrast and entropy. Van Schie *et al* (ISTS 2012 abstract, in press) reviewed Ultrasound Tissue Characterisation (UTC), which captures contiguous transverse images over the length of the tendon. These methods may have the potential to detect subtle changes in tendon structure not seen previously; however, further research is required. Sonoelastography is another developing technique which needs further evaluation

within the field of tendinopathy.

For a full discussion of outcome measures see Measuring outcomes section. The use of imaging as a primary outcome measure in assessing the efficacy of treatments of tendinopathy is not recommended. This is due in part to the issues outlined above with regard to not only the limited psychometrics of US and MRI but also a lack of imaging improvement (despite clinical improvement or resolution) which has been reported with a variety of treatments including sclerosing injections, eccentric exercise, tenotomy and platelet-rich plasma (PRP) injections.^[46–50] Imaging may serve as a useful secondary outcome measure, particularly at longer time points. Ohberg *et al*^[51] reported an improvement in the fibrillar architecture on US in chronic Achilles tendinopathy patients after an eccentric loading programme (average follow-up of 3.8 years), and Sunding *et al* (ISTS 2012 abstract) demonstrated a reduced anterior-posterior diameter of the patellar tendon treated by minimally invasive arthroscopic shaving 3–5 years earlier.

Treatment

Rehabilitation

Rehabilitation Planning. The development of a rehabilitation plan for an individual presenting with confirmed symptomatic tendinopathy requires complex clinical reasoning, with reference to the pathoanatomical diagnosis. Tendon pathology and subsequent rehabilitation will vary considerably depending on the site of pathology; stage of the tendinopathy; functional assessment; activity status of the person; contributing issues throughout the kinetic chain; comorbidities; and concurrent presentations.

While the critical assessment of rehabilitation programmes in the literature^[52–55] is welcomed and advances knowledge in the field, the application of the evidence base is likely to be enhanced by the practitioner's clinical reasoning relating to the individual presentation. While a number of clinical trials have investigated the efficacy of unimodal rehabilitation interventions, generic rehabilitation prescriptions based solely on evidence-based medicine are unlikely to be optimal in the rehabilitation of tendinopathy, particularly in athletes.

Recent literature concerning the rehabilitation of tendinopathy confirms that the most important treatment modality is appropriate loading.^[56] The continuum model of tendinopathy^[57] provides a reasoned basis for considering targeted rehabilitation dependent on current clinical presentation.

Influence of Rehabilitation on Pathology and Pain. Each component of the rehabilitation programme, in particular loading, must be manipulated in relation to the nature, speed and magnitude of the forces applied to the muscle/tendon/bone unit in order to achieve the goals of the particular management phase without causing an exacerbation of the pathological state or pain. Exercise prescription can target matrix reorganisation and collagen synthesis,^[11 58] reduce tenocyte activity,^[57] effect tendon compliance^[59 60] or have an analgesic effect.^[61]

While matrix reorganisation and improved collagen integrity are sometimes considered goals of rehabilitation, measurable structural change does not necessarily correlate with therapeutic outcome.^[62] There is reasonable evidence to refute observable structural change as an explanation for the benefit of eccentric exercise in tendinopathy.^[63 64] Exercise prescription may exert positive therapeutic effects through other mechanisms, such as change in compliance, functional strength, innervation, vascularity or perception of pain.

There is recent evidence to support an increased spinal hyperexcitability in patients with chronically painful tendinopathy^[65] and there are a number of studies illustrating central or spinal involvement in the aetiology of tendinopathy.^[66–69] Consequently when planning the rehabilitation programme, a clinician should assess the potential contribution of spinal function and central sensitisation. In addition, targeted intervention to the contralateral limb can be considered. This may have the dual benefit of limiting the progression of contralateral pathology, while positively influencing the symptomatic limb through a crossover effect.^[70]

The analgesic effects of exercise (in addition to manual therapy and medical interventions) on the central pain mechanisms contributing to tendon pathology may have an important role; however, there is also some evidence to suggest that it is appropriate to perform specific eccentric exercises into pain for

maximal efficacy^[53 71] in the later rehabilitation of certain tendinopathic changes (eg, in chronic patellar or Achilles tendinopathy in middle aged athletes).

Exercise-based Treatment of Tendinopathy. It is important to address the re-education of muscle function, as opposed to considering the tendon in isolation, when planning the rehabilitation strategy. While early stimulus of the muscle tendon unit is typically focused on isometric muscle activation, which may include muscle stimulation, most programmes advocate the progression to higher loads as guided by symptom presentation.

Many tendinopathies have concomitant muscle atrophy which may require a prolonged stimulus at moderate loads, generally repeated for 3–4 sessions per week for optimal muscle hypertrophy.^[72 73] In more marked cases of atrophy, exercise performance in a state of vascular occlusion has been demonstrated to have positive hypertrophic effects.^[74–76]

Progression beyond the early isolated strength and hypertrophy loading requires functional conditioning of the muscle–tendon unit, graduating tendon load through more explosive concentric work, prior to starting eccentric skill specific re-education such as landings, before finally introducing sports specific challenges such as sprinting and cutting.

In summary, the key considerations when formulating a graduated tendinopathy rehabilitation programme are attention to detail, functional loading progression and specific preparation of both the muscle and tendon components to meet the demands of the sport.

In order to measure the response to rehabilitation and guide progression, suitable outcome measures, such as VISA scores or functional tests should be used frequently throughout the process. These will facilitate and guide a progression throughout the rehabilitative process based on functional criteria, as opposed to less sensitive, time-related markers of progression, thus ensuring an individualised approach to management.

The development of UTC shows promise in providing a more objective means of imaging changes in tendinopathic presentation in comparison to more conventional US imaging.^[77] While further research is needed in this field, early prospective studies by Rosengarten *et al* (ref from ISTS abstracts), conducted on a population of elite Australian Rules Football players, indicate that UTC can be useful in monitoring an individual's short-term response to load.

Compliance With Rehabilitation. As with all rehabilitation programmes, it is critical to ensure that patients have a clear understanding and realistic expectations in order to achieve adherence to the programme and increase the likelihood of attaining successful outcomes.

From the outset, it is important to explain the nature of the pathology, including the slow progression of tendon healing,^[78] while communicating what should be expected in relation to pain behaviour during, and in response to, loading stimulus and exercise performance. Furthermore, expectations should be managed throughout the rehabilitation process in relation to the ongoing participation in certain training and competition, which is unlikely to be recommended in the early stages of a significant tendinopathy.

In a sporting environment, the coach should be involved throughout the rehabilitation process. This time provides an opportunity to educate the athlete and coach about the provocative nature of repetitive or sudden exposure to high tendon loads. Furthermore, the multidisciplinary approach to programme planning should be continued upon return to sport, to encourage appropriate load management strategies and to reduce the risk of recurrence.

Adjunct Treatments. The close interaction between therapist and physician in the progression of rehabilitation is important to ensure that medical interventions complement the rehabilitation process, specifically in relation to the loading strategy. The timing and choice of non-steroidal anti-inflammatory drugs (NSAIDs) must be considered in relation to the potential influence on tenocyte activity and glycosaminoglycan synthesis.^[79–81]

While NSAIDs may be employed to good therapeutic effect in a reactive tendon, the tendon response to load may be affected, with potential inhibition of collagen synthesis,^[82] while certain NSAIDs may contribute a deleterious effect on muscular adaption.^[83 84] Medical intervention should, therefore, be

determined in conjunction with and in support of the current goals of the rehabilitation plan.

Long-term Management of the At-risk Tendon. The recent literature on tendon turnover further illustrates the importance of the off-season management for the future prevention of tendinopathy in athletes.^[85] The maxim that 'tendons don't like rest or change' (Jill Cook, ISTS 2012 workshop) should be instilled in athletes with a propensity for tendinopathy and be communicated to coaches who plan preseason training sessions.

Tendinopathy quite commonly presents as the result of a focused overload of a particular tendon within the kinetic chain. To this end, a failure to consider the energy absorption capabilities of related structures (including joint range and muscular function), as well as the consideration of the contralateral limb, may predispose the athlete to a recurrence.

The off-season training phase for athletes with a history of tendinopathy should include a continued tendon loading programme in order to prevent the reduction in tendon integrity and stiffness. Subsequent return to training phases should include appropriately spaced, graduated increases in load. In the absence of such strategies, the athlete will be predisposed to active tendinopathy upon resumption of full training.

While evidence-based support of this approach to long-term management may be limited, the principles suggested in this rehabilitation approach should decrease the likelihood of recurrent presentations of tendinopathy.

Injection

The relative accessibility of tendons and their insertions or associated structures (bursae), as well as the highly localised nature of many tendinopathies, make the local injection of medical therapies an attractive and logical therapeutic opportunity. In current practice, glucocorticoids remain the most widely used injectable therapy for a variety of tendinopathies. They are often given in conjunction with local anaesthetic, although other treatments including prolotherapy, sclerotherapy, protease inhibitor injections and, recently, biologics such as autologous whole blood and PRP have also been employed as injectable medical treatments. US guidance has also increased the confidence of physicians injecting intratendinously, peritendinously or into bursae. A key distinction made by round-table discussants was that the treatments may be divided into those directed *into* the tendon (with the potential for causing needle damage to tendon tissue) versus those which are targeted *outside* the tendon.

The role of glucocorticoid injection therapy has long been debated with the balance of benefit versus harm of such treatment a potentially serious concern for clinicians. Potential mechanisms of action in tendinopathy include a reduction of extrinsic or intrinsic inflammation, reduction of tenocyte proliferation or cellular activity, antiangiogenic activity, inhibition of scarring/adhesion, antinociceptive action or some combination thereof. The efficacy of glucocorticoid injections for rotator cuff tendinopathy, Achilles tendinopathy, patellar tendinopathy and tennis elbow has been investigated at length, with the sum of evidence suggesting that the majority of patients may experience short-term improvement in pain and/or function, but experience a higher risk of relapse in the medium to long term.^[71 86-89] Clinical trials have been limited by the heterogeneity of the patient cohorts under investigation and the timing and type of outcome measures used in the studies. A recent meta-analysis demonstrated worse results from glucocorticoid use when compared with other treatments and placebo in the intermediate and long-term follow-up of treatment for tennis elbow.^[90]

Prolotherapy and sclerotherapy represent treatments quite opposed in their proposed mechanisms of action. The original aim of prolotherapy was to promote a local inflammation-repair response. Conversely, sclerotherapy employs colour Doppler US to target peritendinous areas of increased signal which have been shown to be richly vascularised and innervated. There is also some suggestion that agents used for prolotherapy may themselves have vascular sclerosing properties. A systematic review by Coombes *et al*^[90] concluded that "ultrasonography-guided injection of lauromacrogol (polidocanol) and prolotherapy injection of hypertonic glucose and local anaesthetic are potential therapeutic techniques, based on moderate evidence of improvements in the intermediate term for patellar tendinopathy and lateral epicondylalgia, respectively."

For midportion Achilles tendinopathy, the US-guided injection of polidocanol into peritendinous regions

with high blood flow and nerves has also been reported, in a small randomised controlled trial, to lead to pain relief and improved patient satisfaction.^[91] At 2-year follow-up after successful polidocanol injections, the Achilles tendon demonstrated a decreased thickness and improved tendon structure, demonstrating the potential influence of these soft tissues on the tendon tissue proper.^[92] Further prospective clinical trials may determine whether there are subgroups of Achilles midportion tendinopathy patients (sedentary/active, male/female) that may respond differently to polidocanol injection.^[93 94]

Lesser studied injectable agents include sodium hyaluronate, botulinum toxin and protease inhibitor solutions. The mechanism of action for each of these treatments is unclear. There is little if any evidence to demonstrate the efficacy of these treatments in reducing pain or restoring function. Tennis elbow has been suggested as the most likely type of tendinopathy to respond to treatment with sodium hyaluronate or botulinum toxin.^[90 95] The use of protease inhibitor agents has been questioned due to the high rate of systemic allergic reactions to aprotinin, the most commonly used agent.^[96–98]

Biological therapies, such as autologous whole blood and PRP, have gathered popularity and media interest as treatments for tendinopathy in recent years. They aim to deliver a 'blunderbuss' of bioactive substances to the site of pathology and thereby stimulate a healing process. These treatments build on the theoretical principles of prolotherapy, although with the instigation of tendon healing via normal physiological pathways rather than via an inflammatory response to a noxious substance.

Several small case series which have investigated the autologous whole blood injection for lateral and medial epicondylalgia and patellar tendinopathy report improvements in pain and function in the short-to-medium term although the lack of control group and small numbers limits the applicability of these studies.^[99]

The use of PRP as a treatment option has increased dramatically over the last decade with a large number of published articles investigating its value in the treatment of an array of musculoskeletal problems, including tendinopathy. Despite such activity in the literature, high quality randomised controlled trials into the use of PRP for specific tendinopathies are, at the time of publication, still limited in number. Existing research suggests an improvement in clinical symptoms of tennis elbow following PRP injection compared with either glucocorticoids or autologous whole blood injections.^[100] Some positive results have been noted in its use for patellar and Achilles tendinopathy; however, these outcomes must be weighed against high quality studies suggesting that PRP offers no benefit in the treatment of Achilles tendinopathy.^[101 102] The focus of PRP use in rotator cuff tendinopathy has been as an adjunct during rotator cuff repair surgery; there is insufficient evidence to support its efficacy to date.^[100 103]

High volume injections for the Achilles and patellar tendons, in which large volumes (up to 40 ml) of normal saline (along with bupivacaine and hydrocortisone acetate) are injected peritendinously, have been reported in case series, but not yet subjected to a randomised controlled trial.^[104]

Surgery

The focus of this section is the chronically painful, but not ruptured, tendinopathic tendon. There are few studies with optimal scientific design, and few studies with mid-term or long-term follow-ups. As is commonly noted in systematic reviews, studies with a poor scientific design show good results, and studies with a good scientific design show poor results.^[105]

Achilles. For the chronically painful Achilles midportion and proximal patellar tendon, surgical treatment has been the gold standard when conservative treatments have failed. However, it is well known that the majority of traditional surgical techniques (eg, excision of macroscopically abnormal tendon tissue through a longitudinal split of the tendon) require long-postoperative rehabilitation periods (4–12 months), and the clinical short-term results are variable. An alternate method consisting of multiple longitudinal incisions inside the tendon, followed by a relatively quick rehabilitation period, has also been described,^[106] but that method appears to have been more or less abandoned. Studies with longer follow-up results or new prospective studies using that technique are absent from the literature.

Recent studies on basic tendon biology and imaging led to the development of a new surgical treatment method for midportion Achilles tendinopathy—the US and Doppler-guided mini surgical scraping technique.^[107] The surgery is directly related to the US and Doppler findings; the 'scraping' being performed in specific regions outside the tendon which demonstrate increased colour Doppler signal in

association with structural grey-scale findings. This minimally invasive surgery allows for a quick rehabilitation. Patients begin ambulation after the first postoperative day, and achieve full tendon loading activity after 2–6 weeks. The preliminary clinical results using this method are promising and with very few complications,^[107] but are observational in nature.

Patellar. A well-designed randomised study on patients with Jumper's knee/proximal patellar tendinopathy, comparing the traditional surgical treatment method with eccentric training treatment, showed no significant differences between the groups, with approximately a 50% rate of clinical success after surgery.^[108] The traditional surgical method, in which macroscopically abnormal tendon tissue is excised through a longitudinal split of the tendon, was questioned in this study.

For the proximal patellar tendon, a recently developed method is based on the same basic biological rationale (extensive sensory innervation located peritendinously) and principles as the 'scraping' technique for the Achilles tendon (see previous section), namely US and Doppler-guided arthroscopic shaving.^[109] This arthroscopic method (often performed under local anaesthesia, via 2 minor key holes) allows for a quick rehabilitation during which the patients begin ambulation on the first postoperative day and can achieve full tendon loading activity after 4–8 weeks. The preliminary clinical (observational) results using this method are also very promising, with good results in patients with different activity levels (sedentary to elite level activities) and very few complications.^[109]

Further studies on these methods are required, including larger, randomised studies reported in accordance with the CONSORT statement,^[110] with longer follow-up periods, and using validated outcome measures (detailed below).

Rotator Cuff. Rotator cuff tendinopathy encompasses several characteristic changes of the rotator cuff tendons from tendinopathy without macroscopic tendon discontinuity, through partial-thickness and full-thickness tears, to rotator cuff arthropathy.

Surgical treatment of rotator cuff tendinopathy is applied after failed conservative treatment except in the case of a traumatic tendon tear where early surgical intervention is advocated. The interventions range from subacromial decompression in the case of therapy-resistant impingement and partial rotator cuff tears involving less than 50% of the tendon thickness, to tendon repair in the case of significant partial tears and rotator cuff tears, or tendon transfers when facing an irreparable tear; reverse shoulder prosthesis with or without tendon transfer may be applied in the case of rotator cuff arthropathy. In addition, the biceps tendon needs to be addressed with tenodesis or tenotomy when the biceps and its pulley system are affected.

Rotator cuff repair is a clinically very successful procedure, but healing occurs through scar tissue and often is incomplete. Despite the introduction of several innovative surgical techniques (eg, single row with several suture modifications, single row with triple loaded suture anchors, double row, triple row, suture bridge, transosseous and transosseous equivalent repair) aiming to optimise the mechanical construct and improve healing of rotator cuff tears, failure rates remain high, especially among large and massive tears.^[111–114] Recent literature does not support an advantage by the use of one specific technique based on clinical outcome scores or healing rate analysed by CT arthrogram and MRI.^[115–121] The tendon remains the weak link of the repair. Whereas some advocate the use of single row repair constructs in small rotator cuff tears and double-row repair in medium and large tears, others are concerned about the reports on medial failure of the repair construct following double-row techniques and increased costs associated with longer surgical time and higher implant costs (^[114 116 119]{Lorbach, 2012, p.1778}^[122–124]). Chronic rotator cuff tendon tears are accompanied by significant, irreversible deterioration of muscle quality which is decisive for the prognosis indicating timing of surgery to be essential.

Elbow. Well-conducted studies have failed to demonstrate a consistently successful operative treatment for epicondylalgia. The Cochrane database has not identified any surgical treatment as being effective. Current surgical treatments focus on one of two objectives: (1) removing the tendinosis tissue and/or repairing the origin^[125] or (2) releasing the origin completely.^[126] Controlled resisted stress during the rehabilitation period is advocated.

Measuring Outcomes

Systematic reviews of clinical trials for the management of tendinopathies commonly report on the inconsistency of outcome measures which limits data synthesis and meta-analysis.^[71 127–131] This lack of consistency of outcome measures is also a barrier identified by clinicians in the translation of research into clinical practice (see Knowledge translation, below).

There are few outcome measures specifically designed for tendinopathies, and those that do exist have not achieved widespread implementation. A case in point is the VISA-P which was originally developed in the late 1990s for patellar tendinopathy and later modified for use in other regions, such as the Achilles tendon (VISA-A).^[132 133] It was developed through a review of the literature, consultation with experts and trialling on patients, as well as having undergone clinimetric testing (eg, reliability, construct validity, discrimination and sensitivity to change).^[132 133] At ISTS 2012, the correlation of VISA with extent of tissue pathology (as gauged by glycosaminoglycan content) was also reported (ISTS abstract, in press). The VISA incorporates elements of symptom ratings in various loaded states, amount of activity possible and ratings of participation. Tendinopathy presents clinically as an activity-related pain state that limits participation and as such the VISA scale would appear to be an appropriate outcome measure specific for tendinopathy. Despite the intervening decade since the inception of the VISA-A, it has only been used in 37% of the recently reviewed studies (census date September 2011) reporting the management of Achilles tendinopathy.^[129] Research is required to evaluate why the VISA scales have not been widely adopted in clinical trials, as they seem to address the spectrum of pain, disability and participation. It appears that outcome measures for all tendinopathies require development and further evaluation in terms of their clinimetric properties. This might be facilitated by reference to the COSMIN checklist.^[134]

A group of experts from the fields of general medical practice, rheumatology, public health, physiotherapy, occupational therapy, podiatry, health services research, chiropractic, clinical trial methods and designs, biostatistics and health economics produced a set of recommendations for research into the non-pharmacological management for common musculoskeletal conditions.^[135] This group identified as the fifth most agreeable (81% agreement) recommendation the development of core outcome sets, which would enable comparisons between trials and synthesis of their data.^[136] Currently, there are no agreed upon core outcome sets for the common tendinopathies. It was affirmed at the ISTS 2012 that core outcome sets should be developed and implemented for the different types of tendinopathy.

The notion of a standardised set of outcomes across a wide range of health matters was central to the formation of the Core Outcome Measurement in Effectiveness/Efficacy Trials initiative (<http://www.comet-initiative.org/>), which was launched in 2010 in order to bring together those interested in the development and application of core outcome sets. The COMET initiative would appear as an appropriate vehicle with which to engage in the development and implementation of core outcome sets for tendinopathy. An exemplar of core outcome domains that might be included in a core outcome set can be gleaned from the Initiative on Methods, Measurements and Pain Assessment in Clinical Trials (IMMPACT, <http://www.immpact.org/>), which determined that chronic pain clinical trials should assess pain, physical functioning, emotional functioning, participant ratings of improvement and satisfaction with treatment, symptoms and adverse events and participant disposition, such as treatment adherence.^[137] Functioning and well-being have been recently found to be the areas to assess which are important to patients.^[138]

A recent mixed methods study that integrated a systematic review with clinical reasoning for the management of Achilles tendinopathy reported that physiotherapists found it difficult to reconcile their use of evidence informed painful eccentric exercises with findings that the patients' primary concern is pain reduction.^[130] The overwhelming use of measures of pain severity in clinical trials (eg, 79–89% in recent systematic reviews of Achilles tendinopathy^[138 139]) add to these difficulties. Given that tendinopathies are in the main primarily activity-related pain states, it would seem reasonable that research also focuses on outcomes that measure physical activities and participation (including sports) that are known to produce pain and disability in the different types of tendinopathy (also known as tendon and/or sport-specific functional performance tests). The incorporation of activities and participation is consistent with the International Classification of Functioning, Disability and Health, which is the WHO's framework for measuring health and disability (<http://www.who.int/classifications/icf/en/>). These functional performance tests might constitute part of a core outcome set.

It is our observation that few clinical trials utilise patient global rating of change scales (GROC), which essentially records the patient's determination of the global change in their condition after treatment.^[140]

GROCs implicitly allow the patient to weight whatever is important to them in coming to this determination. The inclusion of such patient centric outcomes was one of the recommendations for trials of non-pharmacological management of common musculoskeletal problems^[136] and should be considered in further developing the outcome measures for tendinopathy.

Research is required into core outcome sets for the different tendinopathies, ensuring that as well as pain, activity and participation are also captured along with GROCs. While there might be an expected level of similarity in what constitutes these core outcome sets, it will be important to ensure that they are specific to the type of tendinopathy (eg, Achilles, gluteal, lateral elbow, biceps femoris, etc) and incorporate all users (eg, patients, payers, healthcare professions, researchers and service delivery) in their formation and ultimately implementation.

Prevention

Tendinopathies are common and often cause long-lasting symptoms of pain and dysfunction which negatively impact on working and/or sporting capacity. Thus, preventive strategies for tendinopathy seem warranted. However, little research on the prevention of tendinopathy has been published. In 1992, van Mechelen *et al*^[141] introduced the sports injury prevention research model. This model will be used as a framework for describing what is known already, and what should be targeted in future research projects.

Step 1: Magnitude of the Problem

Several studies have been published on the frequency of sport-related and exercise-related tendinopathies.^[142-147] However, comparison and interpretation of results is difficult due to a lack of consistent case definitions and inappropriate time-loss-based injury registration methods. Most studies are performed in selected populations such as elite athletes, or in participants of one specific sport. The definition of tendon problems is often unclear since tendon pain, US tendon abnormalities and sport-specific diagnoses such as jumper's knee or tennis elbow are used interchangeably. Many people continue their work or sporting activities despite their chronic overuse injury and/or tendon pain. This means that in many epidemiological studies, despite their prevalence tendinopathies are not included in the incidence rate as new injuries that cause time loss from work or sports.

Bahr^[148] recommended the quantification of overuse injuries in a standardised way using prevalence (not incidence) in prospective studies with continuous or serial measurements of symptoms. Valid and sensitive scoring instruments that measure pain and functional level should be used, and severity should be determined in relation to this rather than from time lost from sport/work (as many athletes continue to compete despite pain). The VISA-A and VISA-P are examples of valid, sensitive and cross-culturally adapted questionnaires which measure pain, function and sports participation in athletes with Achilles or patellar tendon problems (discussed above). Recently, the aforementioned recommendations led to the development of a new method, including a questionnaire, for the registration of overuse problems.^[149]

Step 2: Establishing Aetiology and Mechanisms of Injury

Since overuse injuries, including tendinopathies, have a multifactorial aetiology, establishing the mechanism of injury is not an easy task. Both intrinsic and extrinsic factors have been described in the literature, but there is little robust and often even conflicting evidence for these. A link between the genetic profile and tendinopathy has been reported and some people may be more susceptible to tendinopathy than others.^[150] Genetic screening to identify people at risk of developing tendinopathy might play an important role in future prevention strategy. However, among all potential risk factors, the load to the tendon is considered the most important factor in the aetiology of sport-related and exercise-related tendon pathology and pain.^[151] Moreover, load is a factor that can be modified by prevention strategies.

A promising development is UTC. Preliminary data show that this non-invasive, operator-independent imaging technique can visualise load-dependent changes in a very early stage, at least for the large lower extremity tendons.^[77]

Another potential option is to monitor the volume of loading to which a tendon is exposed (eg, number of jumps, hours of training, distance run, etc); however, there is an inherent inaccuracy in any such measurement utilised to date. Visnes and Bahr^[152] showed a significant difference between healthy and volleyball players with jumpers knee in hours dedicated to volleyball, jump training and number of sets

played. In a sample population of Australian football players, no correlation was observed between the distance covered during the game and the magnitude of change in tendon structure. (ISTS 2012 abstract, in press) The major limitation of global positioning system data is that it quantifies distance and speed travelled, where the impact of load on tendon structure may be more complex (ie, jumps performed in each session, changes in surfaces, etc).

Clarification of the complex relationship between load (including an individual's own unique biomechanics) and tendon changes together with other prospective studies investigating the various (modifiable) risk factors and their influence on the tendon would certainly aid in establishing more appropriate and effective preventive measures. Currently, in most situations, the clinician is not in a position to provide guidance in terms of how much load an individual patient can safely engage in to prevent the development of tendinopathy. However, cautioning against sudden changes to tendon loading may be prudent.

Steps 3 and 4: Developing, Introducing and Evaluating a Training Programme

Developing and introducing training programmes that teach coaches and athletes how to load tendons in the most appropriate way, and how to change their strength, flexibility and proprioception in the most efficient way could be important preventive measures to reduce the risk of developing tendinopathy.

So far, most studies of injury prevention in sporting populations have focused on reducing the incidence of acute injuries such as ACL rupture or ankle sprain.^[153 154] To our knowledge, only a few studies have targeted the prevention of tendinopathy. A prospective study in elite female soccer players demonstrated that soccer-specific balance training can reduce the incidence of patellar and Achilles tendinopathy.^[155] A dose–effect relationship between the duration of balance training and injury incidence was found.^[155] Fredberg *et al*^[156] reported on a randomised controlled trial (RCT) in which elite soccer players were followed over 12 months with use of ultrasonography and injury registration. Half the teams were randomised to an intervention group with prophylactic eccentric training and stretching of the Achilles and patellar tendons during the season. This study demonstrated that the prophylactic training reduced the risk of developing US abnormalities in the patellar tendons, but had no positive effects on the risk of injury. On the contrary, in asymptomatic players with ultrasonographically abnormal patellar tendons, prophylactic eccentric training and stretching increased the injury risk.^[156]

Only programmes that can and will be adopted by athletes, coaches and sporting associations will be successful in preventing injuries.^[157] Hence, implementation strategies and effects of research are necessary to evaluate if preventive methods really are being adopted by the athletes. Finally, the costs and effectiveness of the introduced preventive measures should be evaluated by repeating step 1, or preferably by conducting an RCT.

Knowledge Translation: Uptake of Research in the Clinical Setting

Achieving evidence-based treatment of tendinopathies is a significant challenge. A compelling example is the persistent use of corticosteroid injections for lateral elbow tendinopathy. In general, it takes approximately 17 years to get 14% of research findings adopted into practice.^[158] Moreover, only 30–50% of patients receive recommended care, 20–30% receive care that is not needed or that is potentially harmful^[159 160] and 96% may receive care with the absence of evidence of effectiveness.^[161] Many factors have been shown to influence the current practice patterns. The use of evidence, particularly that from primary research, ranks lower in terms of influence on practice than experience, continuing education, practice patterns of colleagues and entry level training.^[162] It is therefore imperative that a concerted effort is undertaken to ensure that research is pertinent to, meaningful to and feasible for easy uptake into the clinical setting.

Common barriers to achieving evidence-based practice include: not enough time to search for the evidence; too much evidence being available; lack of relevant applicable evidence; the evidence being scattered through a number of databases/journals, that is, not collated into a single location; inadequate access to the evidence (limited availability of free text); limited training in how to access the evidence; poor presentation of the evidence; limited competence and confidence in appraising the quality of the evidence; lack of applicability of the evidence to a heterogeneous patient population; publication bias; lack of autonomy; lack of incentives; clinicians' personal attitudes/intrinsic motivation; and patients'

expectations.^[163–165] An interesting survey and analysis of practice patterns for subacromial pain among general practitioners (GPs) and physiotherapists found that there was only a weak association between treatments which were trusted, and treatments which had been shown to be effective for that condition. For example, GPs and physiotherapists stated that they trusted US therapy, despite the conclusion of systematic reviews that this therapy is ineffective.^[166]

The struggle to successfully bridge the 'knowledge to action gap' is being informed by the efforts of 'implementation research' defined as 'the scientific study of methods to promote the systematic uptake of clinical research findings and other evidence-based practices into routine practice, and hence to improve the quality (effectiveness, reliability, safety, appropriateness, equity and efficiency) of healthcare.'^[167] Many strategies to enhance uptake of evidence into practice have been investigated including use of didactic education sessions, audit and feedback, reminders, opinion leaders, educational materials, patient mediated interventions, financial incentives and interventions tailored to identified barriers—the median improvements of these strategies is approximately 10%.^[168–172] Recently, the use of multiple rather than single interventions, particularly those which are specifically tailored to identified barriers, has been associated with approximately double the improvement than those of single interventions.^[173–174]

There is other evidence in the medical behaviour change literature which can inform strategies to enhance adoption of evidence into practice. Green *et al*^[175] identified four general practice style of the clinician: (1) seekers—those who consider evidence and data which are systematically gathered more important in directing choice of intervention than that of personal experience; (2) receptives—although evidence oriented these individuals tend to rely more on the judgment of respected others; (3) traditionalists—regard clinical experience and authority of respected others as most important and (4) pragmatists—weigh the competing demands of day-to-day practice more heavily than the validity of the evidence. Accordingly, in order to more successfully impact practice change, one first needs to identify the relative representation of each of these practice styles. The traditional methods of knowledge translation have targeted the 'seeker'—one who hunts for the best evidence. This is easily appreciated when one considers that there have been 13 new systematic reviews pertaining to the treatment of tendinopathies published since 2012 alone (PubMed search, February 2013). However, given that seekers make up the smallest percentage of clinicians (~3%)^[175] and pragmatists the greatest (~60%),^[176] there is a need to address to greater degree the needs of the pragmatist. Provision of didactic education or educational materials (provision of knowledge which validates the effectiveness of the intervention) is not enough. Indeed, the prediction of a change in behaviour triggered by provision of knowledge alone is poorly substantiated.^[177] Instead, there is an increased focus on shifting support to facilitate practice change to 'packaging the evidence' into evidence-based practice resources, for example, tools/toolkits which support the practical *application* of the knowledge, for example, simple algorithms and/or videos demonstrating efficient use of the intervention. This strategy is more aligned to assisting the pragmatist to change clinical practice. This approach has been employed recently with the development of the Achilles tendinopathy toolkit, which is now freely available online at the Physiopedia website.

In conclusion, the current literature dedicated to the facilitation of evidence-informed clinical practice provides some important messages for the tendinopathy research community. (1) Research should result from partnerships between researchers and clinicians. (2) Barriers to the application of evidence should be identified and specifically targeted using implementation strategies that are based on behavioural theories.^[177–179] (3) Health research funders should be encouraged to create the conditions for effective knowledge translation.^[180] (4) The effectiveness of these strategies should be evaluated rigorously.^[181–182]

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Acknowledgments

We would like to thank all the round-table participants for the lively and fruitful discussion. The third ISTS 2014 will be held at the University of Oxford, UK, 5–6 September.

Collaborators

ISTS 2012 round-table study group members—H Al Tunaiji, G Alvarez Rey, G Andersson, L Backman, H Behzad, J Bovard, P Brasher, M Collins, J Cornish, A de Groot Ferrando, F Valera Garrido, P Dijkstra, TF Fernandez, G Fong, J Gaida, J Gillies, N Grewal, D Hart, K Hildebrand, E Huisman, KM Khan, H Langberg, A Lawson, D Martinez Silvan, R Mousavizadeh, F Minaya Munoz, D Musson, P Nassab, E O'Brien, J Robinson, S Rosengarten, T Samiric, E Scase, A September, A Sharma, M Smith, RK Smith, D Solomon, K Sunding, G Thornton and H van Schie.

Funding

ISTS 2012 received funding from a Canadian Institutes of Health Research Meetings Plannings and Dissemination grant (grant number MCO-117690). Additional sponsorship was obtained from Sonosite, Astym, TOBI (the Orthobiologic Institute), the Physiotherapy Association of British Columbia, and the British Journal of Sports Medicine..

Provenance and peer review

Not commissioned; internally peer reviewed.

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Br J Sports Med. 2013;47(9):536-544. © 2013 BMJ Publishing Group Ltd & British Association of Sport and Exercise Medicine