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Review article

Tendinopathy in athletes

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ABSTRACT

Overuse related tendon pain is a significant problem in sport and can interfere with and, in some instances, end an athletic career. This article includes a consideration of the biology of tendon pain including a review of tendon anatomy and histopathology, risk factors for tendon pain, semantics of tendon pathology, and the pathogenesis of tendon pain. Evidence is presented to guide the physical therapist in clinical decision-making regarding the examination of and intervention strategies for athletes with tendon pain.

1. Introduction

Overuse related tendon pain is a very common condition in sport. Kannus (1997, p. 53) wrote that tendon pain is a "source of major concern in recreational and competitive athletes". Overuse injuries are more common than macrotraumatic injuries in athletics (Brukner & Bennell, 1997), and much of overuse injury is tendon related. Tendon pain is frequently reported in sports including volleyball, basketball, long distance running, and jumping events in track and field (Ferretti, Puddu, Mariani, & Neri, 1985; Jarvinen, Kannus, Maffulli, & Khan, 2005; Kujala, Sarna, & Kaprio, 2005; Lian, Engebretsen, & Bahr, 2005; Taunton et al., 2002). The tendons most commonly susceptible to overuse injuries are the patellar, Achilles, posterior tibialis, rotator cuff, long head of the biceps brachii, and the wrist extensors (Maffulli, Wong, & Almekinders, 2003; Rees, Wilson, & Wolman, 2006).

2. Tendon histology

The function of a tendon is to attach muscle to bone, thereby transferring force of the contracting muscle to bone and either inducing or controlling movement. The efficiency of this force transfer is maximized by minimal elasticity of the tendon. When muscular contraction occurs, the tendon is subjected to tensile loading. This results in the storage of elastic energy within the tendon. As human tendons demonstrate minimal hysteresis, most of the elastic energy stored in the tendon is released when the tensile load is removed (Maffulli, Renström, & Leadbetter, 2005). During the stretch-shortening cycle when an eccentric contraction is immediately followed by a concentric contraction, this stored energy is thought to contribute to the force generation and mechanical efficiency of the movement (Lieber, 2002, p. 168). This phenomenon has been supported is research in vivo on human Achilles tendons (Kubo, Kanehisa, & Fukunaga, 2005).

Each tendon has two junctional regions; the myotendinous junction (MTJ) at the muscle end of the tendon and the osseotendinous junction (OTJ) at the bone end. At the OTJ, or enthesis, there is a gradual transition of tissue from tendon to fibrocartilage to bone. Some tendons like the Achilles and the wrist extensors have the muscle insertion along the same line as the muscle belly and are referred to as traction tendons. In other tendons, like the posterior tibialis and the quadriceps, the tendon direction is not along the same line as the muscle belly, and these are referred to as gliding tendons. As gliding tendons encounter compressive and shear forces as they course around bony landmarks, the etiology of tendon pain in these tendons is likely different than that in traction tendons (Pufe, Petersen, Mentlein, & Tillmann, 2005).

The microanatomy of the tendon includes an extracellular matrix containing water, proteoglycans, glycoproteins, collagen, and elastin. The collagen is predominantly Type I in a normal tendon and the collagen fibers are arranged in a parallel fashion. The orientation of the fibers and the biomechanical properties of Type I collagen are designed for the force transmission from muscle to bone. The cells of the tendon tissue are tenoblasts and tenocytes, which are fibroblastic cells and active in the production and secretion of proteins (Maffulli et al., 2005). Tendon loading

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stimulates an acute increase in collagen synthesis, which peaks around 24 h after exercise and remains elevated for up to 72 h (Magnusson, Langberg, & Kjaer, 2010). However, the degradation of collagen is also increased after exercise, likely at a greater level than the increase in synthesis. Consequently, for the first 36 h after exercise, the collagen metabolic system is in a negative balance with degradation greater than synthesis (Fig. 1). This may explain that repeated exercise without sufficient rest can leave an athlete in a state of repeated collagen breakdown, and the development of overuse injury (Magnusson et al., 2010).

The collagen fibers are arranged into bundles, or fascicles, which are surrounded by the endotenon. The fascicles are bundled together by the outer epitenon layer which is intimately attached to the tendon. In some tendons, there is a layer external to the epitenon, the paratenon, which is lined with synovial cells and provides a resistance to friction over bony surfaces (Fig. 2). A painful tendon may have histopathological changes in the collagenaceous matrix or in the sheath, or both (Abate et al., 2009).

The vascular supply for most tendons comes from the OTJ and the MTJ, leaving a potential vascular compromise in the mid region between the OTJ and MTJ. Small vessels extend through the mesotenon, connecting the paratenon and the epitenon. In painful tendons, neovascularization occurs in which small, thin walled blood vessels proliferate, causing an increase in tendon blood flow (Alfredson & Ohberg, 2005; Alfredson, Ohberg, & Forsgren, 2003; Cook, Malliaras, De Luca, Ptasznik, & Morris, 2005; Ohberg, Lorentzon, & Alfredson, 2001; Pufe et al., 2005). The stimulation for this angiogenesis is upregulation of vascular endothelial growth factor (VEGF) in pathic tendons (Pufe et al., 2005; Scott, Lian, Bahr, Hart, & Duronio, 2008). Not only does VEGF stimulate the formation of the neovessels, but it also stimulates production of metalloproteinase enzymes, lytic to collagen, and downregulates the inhibitors of such enzymes (Pufe et al., 2005). The consequence of the VEGF action is decreased collagen strength and predisposition to additional microtears. VEGF and angiogenesis has been associated with painful tendons in several studies (Alfredson & Ohberg, 2005; Cook, Malliaras, et al., 2005; Ohberg et al., 2001; Pufe et al., 2005).

Nerve supply to and from tendons comes from surrounding muscle and vary by tendon and by regions of the tendon (Abate et al., 2009). For example, tendons of the finger flexors have greater innervation than the Achilles, and innervation tends to be greater toward the MTJ and the OTJ. The nerve endings in tendons include Ruffini corpuscle and Golgi tendon organ mechanoreceptors as well as free nerve endings that serve a nociceptive function. In a painful tendon, there is a marked increase in the ingrowth of these free nerve endings (Magnusson et al., 2010; Sanchis-Alfonso, Rosello-Sastre, & Subias-Lopez, 2001), and there is also evidence of higher concentrations of excitatory neurotransmitters in painful

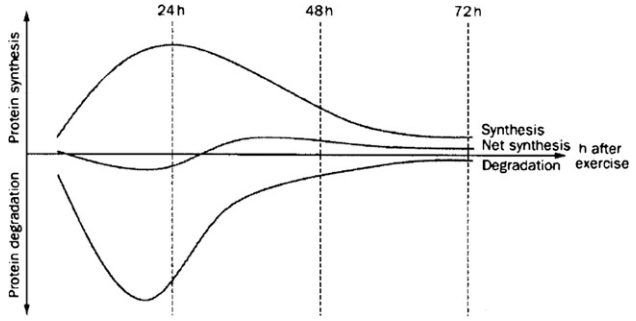

Fig. 1. Collagen synthesis and degradation following exercise (Taken from Magnusson et al., 2010).

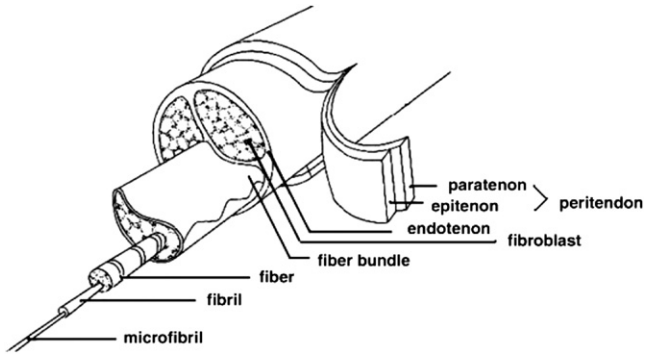

Fig. 2. Tendon structure (Taken from Kirkendall & Garrett, 1997).

tendons (Alfredson, 2005; Andersson, Danielson, Alfredson, & Forsgren, 2002; Bjur, Alfredson, & Forsgren, 2005; Lian et al., 2006; Schubert, Weidler, Lerch, Hofstadter, & Straub, 2005).

3. Risk factors for tendon pain

There is evidence that some individuals may be more at risk for developing tendon pain than others. These risk factors are classified as intrinsic (within the body) or extrinsic (outside the body) and modifiable or non-modifiable. Intrinsic non-modifiable risk factors include increasing age (Astrom, 1998; Birch, Smith, Tasker, & Goodship, 2001; Riley, Goddard, & Hazleman, 2001; Sargon, Ozlu, & Oken, 2005; Tuite, Renstrom, & O'Brien, 1997), gender (Ashford, Cassella, McNamara, Stevens, & Turner, 2002; Astrom, 1998; Astrom & Rausing, 1995; Holmes & Lin, 2006; Knobloch et al., 2008; Lian et al., 2005; Ramos et al., 2009; Riley et al., 2001), and an individual's genetic makeup. Josza et al. (1989) described an increased frequency of tendon injury in persons with blood type O, and this was confirmed by Kujala et al. (1992). However, other studies have not found this same association between blood group and tendon pain (Leppilahti, Puranen, & Orava, 1996; Maffulli, Reaper, Waterston, & Ahya, 2000). New evidence suggests that one or more genes proximal to the ABO gene on chromosome 9 may be a causal factor in the development of tendon pain (Magra & Maffulli, 2007; Mokone et al., 2005; Mokone, Schwellnus, Noakes, & Collins, 2006).

Modifiable intrinsic risk factors for tendon pain that have been reported include abnormal kinematics (Abate et al., 2009; Azevedo, Lambert, Vaughan, O'Connor, & Schwellnus, 2009; Grau et al., 2008; Richards, Ajemian, Wiley, Brunet, & Zernicke, 2002; Richards, Ajemian, Wiley, & Zernicke, 1996; Ryan et al., 2009; Souza, Arya, Pollard, Salem, & Kulig, 2010), muscle inflexibility (Cook, Kiss, Khan, Purdam, & Webster, 2004; Witvrouw, Bellemans, Lysens, Danneels, & Cambier, 2001), obesity (Frey & Zamora, 2007; Holmes & Lin, 2006), and decreased eccentric muscle strength (Gaida, Cook, Bass, Austen, & Kiss, 2004; Grau et al., 2008). Modifiable extrinsic risk factors include sport (Ferretti, 1986; Kujala et al., 2005; Lian et al., 2005; Maffulli, Sharma, & Luscombe, 2004; Maffulli, Wong, et al., 2003; Sein, 2006) and training volume and surface (Ferretti, 1986).

4. Semantics of tendon pathology

One of the issues that have contributed to confusion in understanding tendon pain is the multiple terms associated with tendon pain. Bernstein (2006) wrote in an editorial on linguistic determinism in medicine that "the names of the diseases, one might argue, set the bounds on our thought, possibly to the detriment of patients." One example he used in that editorial was the rather

indiscriminate use of the term "tendinitis" for all tendon pain. Tendinitis describes a specific histopathological entity in which a predominant characteristic of the condition is inflammatory. In fact, multiple studies have suggested that a painful tendon may not have the presence of inflammatory cells or mediators (Abate et al., 2009; Hashimoto, Nobuhara, & Hamada, 2003; Khan, Cook, Bonar, Harcourt, & Astrom, 1999; Maffulli, Testa et al., 2004; Movin, Gad, Reinholt, & Rolf, 1997; Potter et al., 1995; Uhthoff & Sano, 1997). Bernstein (2006) argued that naming all tendon pathology "tendinitis" may lead a practitioner to an incorrect line of reasoning in designing a plan of care.

Tendinosis is used to describe a chronic degenerative state of a tendon where there is a conspicuous lack of inflammatory cells, disorganization and separation of the collagen bundles, increased proteoglycans in the extracellular matrix, hypercellularity, and neovascularization (Khan et al., 1999; Khan, Cook, Kannus, Maffulli, & Bonar, 2002; Maffulli, Wong, et al., 2003). Paratenonitis, also known as tenosynovitis, is an inflammatory condition of the paratenon and is characterized by the presence of inflammatory cells, hypervascularity of the sheath, and development of a fibrinous exudate in the sheath space causing tendon crepitation.

The terms tendinitis, tendinosis, and paratenonitis are all reserved for specific histopathologic conditions of the tendon, which, in fact, may not be known by the treating clinician. Several authors have suggested that tendinopathy is a better term for clinicians to use in describing tendon pain as it is histopathologic neutral (Cook, Khan, Maffulli, & Purdam, 2000; Jarvinen et al., 2005; Khan et al., 2002; Khan, Cook, Taunton, & Bonar, 2000; Maffulli, Khan, & Puddu, 1998; Stasinopoulos & Johnson, 2006).

5. Pathogenesis of tendinopathy

The controversy pertaining to the pathogenesis of tendinopathy and the inflammatory pathway is one that has persisted. Fredberg and Stengaard-Pedersen (2008) challenged the tendinitis myth with recent evidence that pro-inflammatory agents are present in chronic tendinopathies, and the rapid response to corticosteroid intervention. These authors described a "tendinopathic iceberg" (Fig. 3) with a long period of time of asymptomatic changes in tendon with the exposed tip of the iceberg being a painful tendon. In a review paper, Abate et al. (2009) supported this iceberg theory and proposed a pathway of the pathogenesis of tendinopathy beginning with overuse leading to microdamage of the tendon, followed by inadequate tendon healing, a pathologic cascade of events including neovascularization, nerve ingrowth, tendon degeneration, and, ultimately, a painful tendon at the tip of the iceberg. In their conclusion, Abate et al. (2009) wrote that "it is conceivable that inflammation and degeneration are not mutually exclusive, but work together in the pathogenic cascade of tendinopathy."

Cook and Purdam (2009) have described a pathological model of the tendinopathy continuum with three stages: reactive tendinopathy, tendon disrepair, and degenerative tendinopathy (Fig. 4). The purpose of this model is to assist the clinician in determining appropriate intervention for the tendinopathy based on the pathology stage. These authors provide pharmacological and physical management guidelines based on whether the athlete is in the reactive tendinopathy/early tendon disrepair, or the late tendon disrepair/degeneration stage.

6. Clinical examination of the athlete with a painful tendon

In the clinical examination of an athlete with a painful tendon, taking a thorough history is crucial. Leadbetter (1992) emphasized the significance of the "principle of transitions" for athletes with overuse injury. This principle states that injury is most likely when an athlete has made some kind of training change including training routine (intensity, frequency, duration), training surface, equipment, or technique. Accordingly, taking the history must include questions that provide the examiner with information about potential transitions that may have led to the development of tendon pain. The typical presentation of tendinopathy is gradual onset of load-related tendon pain which, depending on the chronicity of the problem, may interfere with athletic performance or, in late stages, with activities of daily living. Staging of the overuse tendon condition is recommended using a grading system like that of Blazina, Kerlan, Jobe, Carter, and Carlson (1973) or Nirschl (1992) in order for the examiner to understand the level of restrictions of activities and sport performance resulting from the painful tendon. Additionally, there are tendon-specific outcomes assessment tools including the VISA scale for patellar (Visentini et al., 1998) or Achilles tendon pain (Robinson et al., 2001) which provide information on the effect of tendon pain on life activities.

Clinical examination of an athlete with tendon pain includes careful observation of skeletal alignment, muscle bulk, tendon size,

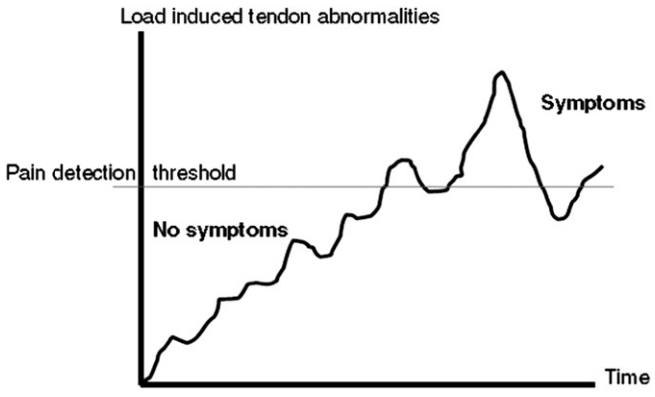

Fig. 3. The tendinopathic iceberg (Taken from Fredberg & Stengaard-Pedersen, 2008).

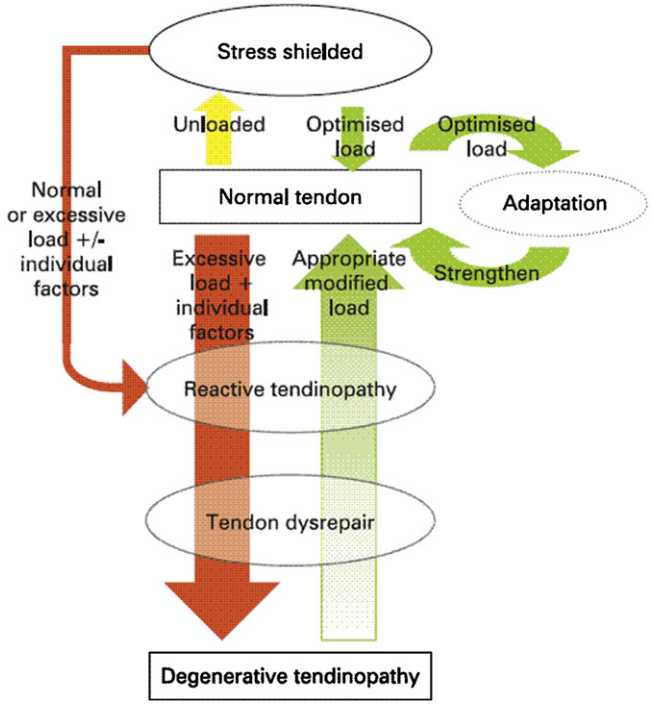

Fig. 4. Three-stage tendinopathy model (Taken from Cook & Purdam, 2009).

and swelling. Joint mobility and muscle length are assessed with notation of joint end feels. Standard muscle performance testing is completed which involves loading of the involved tendon, and the examiner should record the presence and location of pain and/or weakness. In some cases, specific tendon loading tests are advocated for testing of the painful tendon such as the decline squat for patellar tendinopathy (Purdam, Cook, Hopper, & Khan, 2003), or the heel raises for Achilles tendinopathy (Alfredson & Cook, 2007; Silbernagel, Gustavsson, Thomee, & Karlsson, 2006).

Based on the evidence that abnormal kinematics may be risk factors for the development of tendon pain (Abate et al., 2009; Azevedo et al., 2009; Grau et al., 2008; Richards et al., 2002; Richards et al., 1996; Ryan et al., 2009; Souza et al., 2010; Van Ginckel et al., 2009), movement analysis is a critical part of the clinical examination of an athlete with tendon pain. Examples of abnormal kinematic risk factors include excessive foot pronation for patellar (Grau et al., 2008) and Achilles (Donoghue, Harrison, Laxton, & Jones, 2008; McCrory et al., 1999; Ryan et al., 2009) tendinopathy and abnormal scapular position and movement for rotator cuff tendinopathy (Ludewig & Reynolds, 2009). Souza et al. (2010) reported that significant differences existed in hopping kinematics between a group of athletes with patellar tendinopathy and a control group with the tendinopathy group showing decreased demand on the knee joint. What is unknown from these data, however, is whether the kinematic differences led to the patellar tendon pain, or were a result of the patellar tendon pain.

Palpation of the involved tendon is routinely done to assess pain, tissue quality, and the presence of crepitation. Studies pertaining to the clinical utility of palpation suggest that moderate tenderness of a tendon is a normal finding in athletes and does not predict pathologic changes within the tendon (Cook, Khan, Kiss, Purdam, & Griffiths, 2001; Maffulli, Kenward et al., 2003; Ramos et al., 2009).

Imaging of the tendon may provide additional information regarding intra-tendinous changes. Generally, plain-film radiography is of little diagnostic value unless there is calcification within the tendon. Both gray-scale ultrasonography and magnetic resonance imaging can reveal structural changes in tendon including tendon swelling and collagen disorganization. However, such structural changes are not well correlated to symptoms as athletes can have painful tendons without structural change and structural change without pain (Cook, Khan, Kiss, Coleman, & Griffiths, 2001; Cook, Khan, Kiss, Purdam et al., 2001; Cook, Khan, Kiss, Purdam, & Griffiths, 2000; Gold, Seeger, & Yao, 1993; Khan et al., 1997). Color Doppler sonography allows for quantification of tendon neovascularity in a painful tendon (Cook, Ptazsnik et al., 2005; Gisslen & Alfredson, 2005).

7. Intervention for the painful tendon

The intervention plan for an athlete with tendon pain should be based on an integration of the clinician's clinical judgment, the patient's values, and the best available evidence (Sackett, 2000). Although tendinopathy is a common condition in athletes, there are few randomized controlled trials that have investigated the interventions recommended for athletes with tendon pain. Consequently, clinical reasoning is crucial in linking restrictions in daily and sport activities with impairments in body structure and function. The focus of this section of the paper is on those interventions typically used by physical therapists.

In the model described by Cook and Purdam (2009), an athlete with reactive tendinopathy/early tendon disrepair should have an imposed period of relative rest in which tendon load is reduced to minimize progression of pathology. This period should not be complete cessation of the offending activity, but a decrease in the overall training volume of the activity. Complete rest through immobilization of a joint has a negative effect on tendon strength and should not be practiced (Kannus, Jozsa, Natri, & Jarvinen, 1997). The training volume parameters – intensity, frequency, or duration – are evaluated and adjusted based on the individual athlete and the circumstances of the case. To maintain cardiovascular and pulmonary fitness, cross training activities with reduced tendon loads are appropriate.

Although physical agents including cryotherapy, ultrasound (US), phonophoresis, and iontophoresis are commonly employed clinically in the treatment of athletes with tendinopathy, there is a paucity of evidence to support their use. Cryotherapy is often used to decrease painful tendinopathic symptoms; Rivenburgh (1992) described the tissue effects of cold including decreasing the movement of protein from capillaries and may decrease blood flow. However, there is no higher level evidence that supports or refutes the use of cold for tendon pain.

Therapeutic US is also a commonly utilized clinical modality for tendon pain. Like cryotherapy, there is no compelling evidence to support or refute its use in patellar tendon pain. Therapeutic US has been shown to have positive effects on collagen production in vitro (Ramirez, Schwane, McFarland, & Starcher, 1997). It has also been shown to have a significant thermal effect when using a 3 MHz treatment at 1.0 W/cm^2 (Chan, Myrer, Measom, & Draper, 1998), but whether this is desirable for healing of tendon pain is not known. One study on the effect of low-intensity pulsed ultrasound (LIPUS) on patellar tendinopathy (Warden et al., 2008) concluded that this modality had no additional benefit when compared to placebo ultrasound on patellar tendon symptoms.

Phonophoresis is a technique in which US is used to drive a pharmaceutical agent through the skin into a painful region. Klaiman et al. (1998) compared the effect of US and phonophoresis using fluocinonide on various musculoskeletal conditions including tendon pain. They found that US alone decreased pain and increased pressure tolerance, but the addition of fluocinonide did not augment the effect. Penderghest, Kimura, and Gulick (1998) examined the effect of the addition of phonophoresis to a stretching and strengthening program for patients with tendon pain. Their results were consistent with those of Klaiman et al. (1998). These studies do not support the use of phonophoresis for patients with tendon pain.

Iontophoresis is another modality used to treat tendon pain with a similar rationale as phonophoresis in driving a pharmaceutical across the skin. There is animal research evidence that iontophoresis is effective in driving dexamethasone into patellar tendon tissue (Nowicki, Hummer, Heidt, & Colosimo, 2002). Pellechia, Hamel, and Behnke (1994) showed that iontophoresis with dexamethasone and lidocaine was more effective than modalities combined with transverse friction massage (TFM) for decreasing pain and increasing function in patients with patellar tendon pain. However, the clinical decision to use iontophoresis or phonophoresis with an anti-inflammatory pharmaceutical for tendon pain may be flawed as it presupposes that an inflammatory process is the primary pain generator in tendon pain (Abate et al., 2009; Andres & Murrell, 2008; Cook, Khan, Maffulli, et al., 2000; Cook & Purdam, 2009; Khan et al., 2002; Khan et al., 2000).

Another intervention that has been used to treat tendon pain is low-lever laser therapy (LLLT). This modality is relatively new to the United States but has been used in Europe and Canada for many years. A systematic review with meta-analysis on the use of LLLT for tendinopathy was conducted, and 25 controlled clinical trials met the standards to be included in the review (Tumilty et al., 2010). The finding of the review were mixed, with 12 studies supporting the use of LLLT and 13 studies were either not conclusive or did not demonstrate an effect. However, the authors identified that one

finding of their review was that the 12 positive studies shared the use of dosages consistent with current dosage recommendations for the treatment of tendon pain (Tumilty et al., 2010).

Transverse friction massage, advocated by Cyriax and Coldham (1984) for tendon pain, is purported to reduce adhesions within the tendon and encourage normal realignment of collagen fibers. There is basic science evidence from animal studies that soft tissue mobilization can increase fibroblastic activity (Davidson et al., 1997; Gehlsen, Ganion, & Helfst, 1999) but there are no studies of the tissue effect of TFM on tendinopathy in humans. The aforecited work by Pellecchia, Hamel, and Behnke (1994) showed a positive effect of the combined TFM/modality intervention, but whether the effect was from the TFM, modalities, or combination is unknown. In a recent systematic review on the use of TFM for treatment of tendonitis, the authors concluded that there was no evidence to support the use of deep TFM (Brosseau et al., 2002).

A common intervention used for patients with tendon pain is counterforce bracing. These braces are applied in circumferential manner to impose a compressive force on a tendon, with the intent of "unloading" the tensile force at the tendon insertion to bone. Studies on the use of counterforce bracing for wrist extensor tendinopathy have shown no change in the force production of the muscle but an increase in the patients' pain threshold to muscle stretch (Chan & Ng, 2003; Ng & Chan, 2004). In spite of the very common use of this intervention for patients with tendinopathy, evidence to support its use for patellar or Achilles tendon pain is lacking.

As tendon pathology has been historically associated with tendinitis, it is not surprising that anti-inflammatory pharmaceuticals are suggested for patients with tendon pain. Although the prescription of pharmaceuticals is the purview of the physicians, patients seen by rehabilitation professionals may be using over-the-counter non-steroidal anti-inflammatory drugs (NSAIDs) by their own choice. In a systematic review of the literature on treatment of tendinitis, Almekinders and Temple (1998) reported that the use of oral NSAIDs may result in pain relief but the long term effect on the tendon is not known. Similarly, the use of injected corticosteroids may result in pain relief, but there is concern regarding the effect of corticosteroid on tendon strength (Paavola et al., 2002; Shrier, Matheson, & Kohl, 1996). In a randomized, double-blind, placebo-controlled study (Fredberg et al., 2004) of steroid injection in patients with patellar and Achilles tendinopathy, a significant reduction in pain and tendon thickness was observed in the steroid groups for both Achilles and patellar tendon pain. Fredberg et al. (2004) argued that the results of this study suggest that the tendinitis–tendinosis question remains unresolved.

Exercise intervention for tendon pain is common, and decision-making regarding exercise selection should be based on the impairments identified in the examination. Muscle tightness has been proposed as a risk factor for patellar tendon pain (Cook et al., 2004; Witvrouw et al., 2001) and Achilles tendon pain (Kaufman, Brodine, Shaffer, Johnson, & Cullison, 1999). In one study, 46 patients with Achilles tendon pain were randomly assigned to a 12-week eccentric Achilles training program or a calf-stretching program (Norregaard, Larsen, Bieler, & Langberg, 2007). Patients were reevaluated at 3, 6, 9, 12, and 52 weeks for tendon pain, tendon thickness measure by ultrasonography, and the patients' self-reported outcome. Both groups showed improvement but there was no significant difference between groups (Norregaard et al., 2007). No other study was identified that studied the effect of stretching only on tendon pain.

One area of emerging evidence for the treatment of tendon pain is eccentric exercise. The seminal work on the use of eccentric exercise in tendon pain was done by Curwin and Stanish (1984). They advocated eccentric exercise for patellar tendinopathy to maximally stress the tendon to increase tendon strength. Their program involved six weeks of drop squat training (3×10 repetitions/day), progressing in the first week from a slow speed to faster speeds, and then adding resistance in weeks two through six. In a retrospective review of 66 patients treated with the eccentric program for patellar tendon pain, the authors reported complete relief of pain in 20 patients, marked decrease in symptoms in 42 patients, and four patients reported worsening of symptoms (Stanish, Curwin, & Mandel, 2000).

High-load eccentric training has been used successfully to treat non-insertional Achilles tendinopathy (Alfredson, Pietila, Jonsson, & Lorentzon, 1998). One feature of this eccentric training is the significance of pain during the eccentric exercise. Alfredson et al. (1998) proposed that the eccentric exercise should be dosed at 3×15 repetitions twice daily, seven days/week for 12 weeks and should be painful to perform. These authors recommended that when a patient reaches the point that the exercise is no longer painful; the load should be increased to the point that it becomes painful again. A systematic review on the appropriate dosage for eccentric exercise in non-insertional Achilles tendinopathy concluded that there is insufficient evidence at this time to establish a set dosage, but the effect of eccentric exercise on Achilles tendon pain was positive (Meyer, Tumilty, & Baxter, 2009).

Eccentric exercise has been used successfully to treat patellar tendon pain (Bahr, Fossan, Loken, & Engebretsen, 2006; Cannell, Taunton, Clement, Smith, & Khan, 2001; Frohm, Saartok, Halvorsen, & Renstrom, 2007; Jonsson & Alfredson, 2005; Purdam et al., 2004; Stasinopoulos & Stasinopoulos, 2004; Young, Cook, Purdam, Kiss, & Alfredson, 2005), supraspinatus tendinopathy (Eriksson, 2006; Jonsson, Wahlstrom, Ohberg, & Alfredson, 2006), wrist extensor tendinopathy (Croisier, Foidart-Dessalle, Tinant, Crielaard, & Forthomme, 2007; Martinez-Silvestrini et al., 2005; Svernlov & Adolfsson, 2001), and posterior tibialis tendinopathy (Kulig, Lederhaus, Reischl, Arya, & Bashford, 2009). Rees, Wolman, and Wilson (2009) summarized the existing evidence on the effect of eccentric exercise on tendinopathy and identified emerging evidence including increase collagen synthesis, decrease in neovascularization, and a pattern of oscillatory loading not present in concentric exercise.

There is evidence, however, that eccentric exercise may not always be successful in the treatment of tendinopathy. Visnes, Hoksrud, Cook, and Bahr (2005) found that a group of volleyball athletes with patellar tendinopathy did not respond to 12 weeks of eccentric training during the competitive season. As the athletes continued to participate in volleyball training and competition during the eccentric training period, it is not known whether the athletes would have responded to the eccentric training if they were not participating in volleyball activities. Sayana and Maffulli (2007) studied the use of eccentric exercise in a group of 34 sedentary, non-athletic patients. They found that only 56% of patients responded favorably to the eccentric training program, with the other 44% requiring tendon injection or surgery, A follow-up study using high-load eccentric training for Achilles tendinopathy in athletic patients found a similar percent of patients (60%) responded to the eccentric exercise treatment as in the study of non-athletic patients (Maffulli, Walley, Sayana, Longo, & Denaro, 2008).

As described earlier, there is evidence that the development of tendon pain may be related to abnormal kinematics during functional activities. These findings would suggest that a potential intervention for tendinopathy would be interventions to address these abnormal movement patterns. For Achilles tendon pain, a recent published clinical guideline (Carcia, Martin, Houck, & Wukich, 2010) indicated that there was C level evidence (weak)

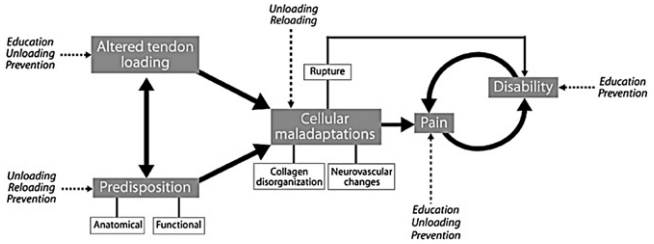

Fig. 5. The EdUReP model (Taken from Davenport et al., 2005).

to support the use of foot orthotics to "reduce pain and alter ankle and foot kinematics while running". Otherwise, evidence is lacking to support interventions designed to address abnormal kinematics in athletes with tendinopathy.

Davenport, Kulig, Matharu, and Blanco (2005) have proposed a non-surgical treatment model for tendinopathy that they referred to as the EdUReP model (Fig. 5). In this model, the approach to tendon pain is educational intervention (Ed), unloading of the tendon (U), gradual reloading of the tendon (Re), and prevention of tendon pain recurrence (P). Although the authors did not subject this model to testing, they proposed it as "an evidence-based treatment construct that aims to reduce functional limitation and disability through amelioration of tissue pathology."

Other interventions for tendon pain include those that are physician driven including extracorporeal shock wave therapy, nitric acid patches, injections with substances such as normal saline, anesthetics, corticosteroids, polidocanol, aprotinin, or platelet rich plasma, and surgery. Review of these interventions are outside the scope of this paper, but several excellent evidence-based expert reviews of such interventions have been published (Maffulli, Longo, & Denaro, 2010; Rees, Maffulli, & Cook, 2009; Rees et al., 2006; Riley, 2008).

8. Conclusion

Tendon pain is a common condition seen by sports medicine health care professionals. At this time, the evidence suggests that while tendon pain may have an early inflammatory stage, much of what is seen is not a primary inflammatory problem but one characterized by tendon disrepair and degeneration. The treatment of tendon pain is multifactorial and thereby challenging and requires careful consideration of the chronicity of the problem and the athlete-specific impairments associated with the tendon pain. At present, most evidence supports an approach to tendon pain using eccentric exercise, but non-responders must be identified and considered for treatment with other interventions as directed by a physician. There is a pressing need for ongoing research to better identify risk factors and appropriate prevention and intervention strategies for tendon pain.

Conflict of interest

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