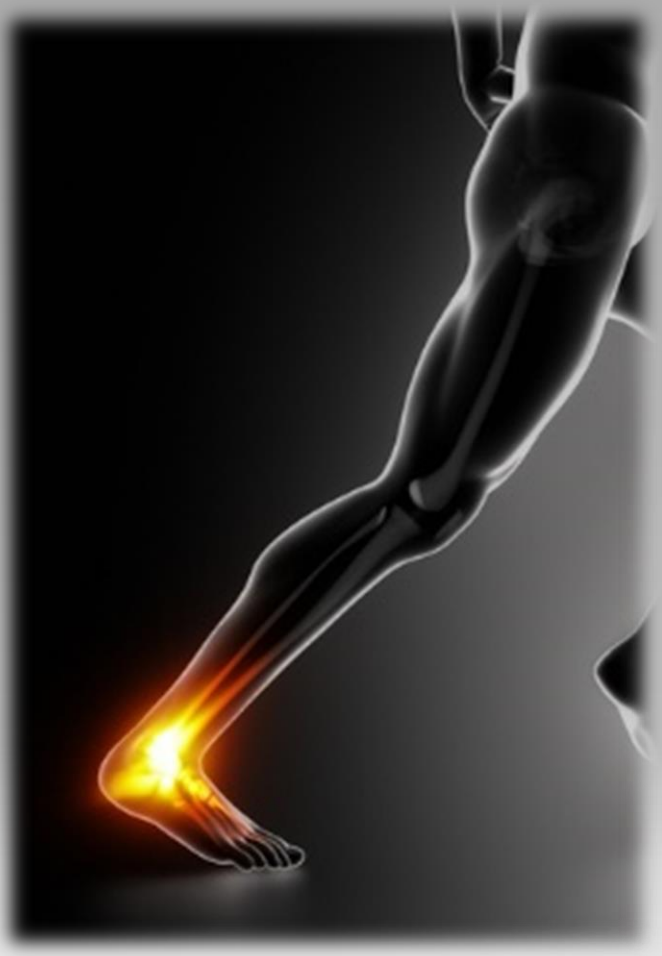

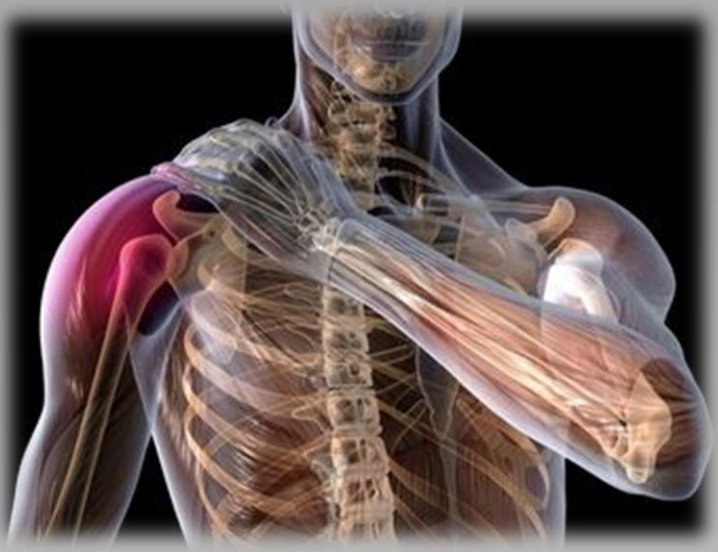

Tendinopathy

Tadhg Cronin, Eoghan Buckley, Victoria Mc Mahon, Cathal Noonan, Barry Noonan, William Ryan

Sports Short Course Booklet

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Regards,

William, Barry, Eoghan, Tadhg, Cathal & Victoria

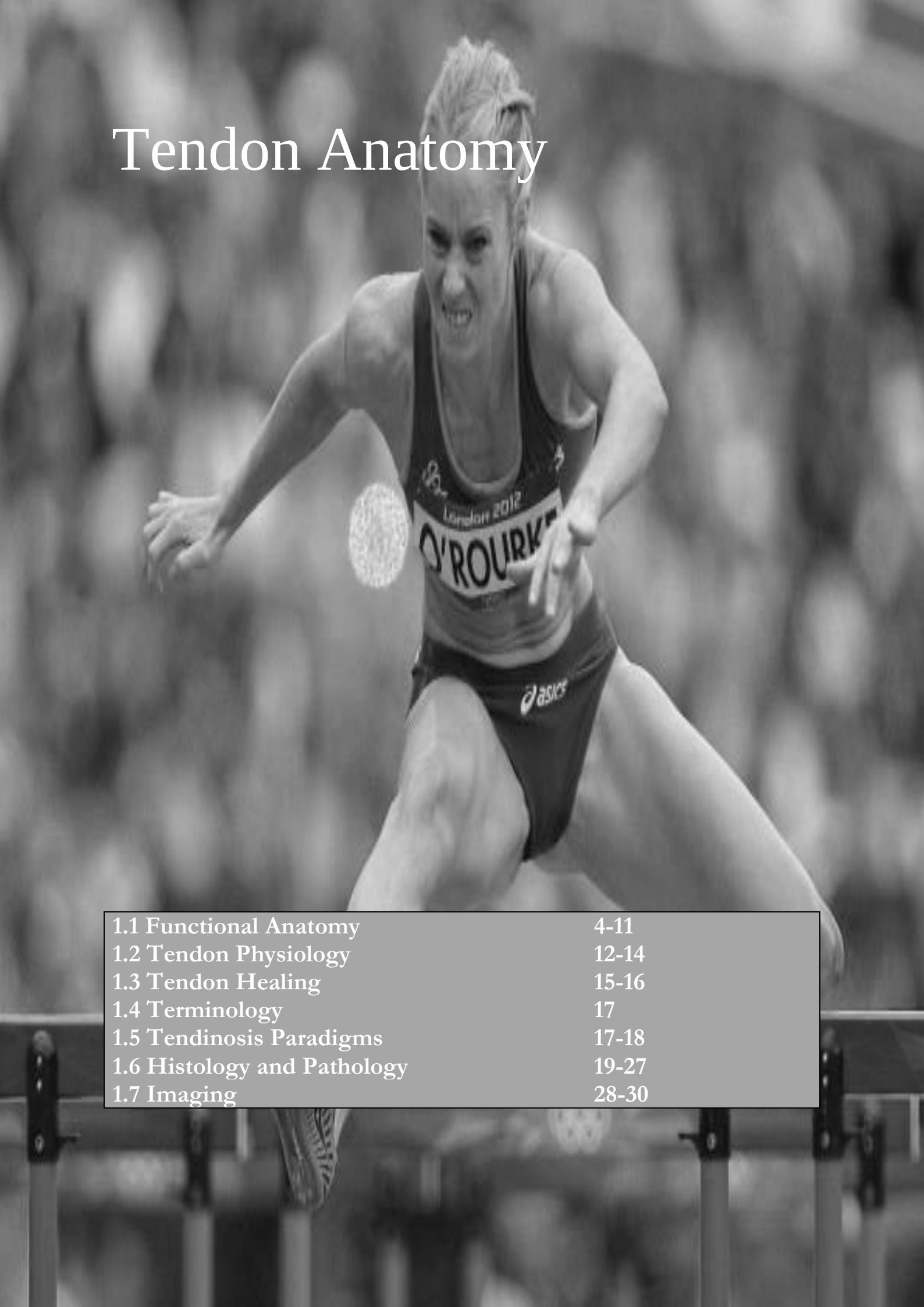
Introduction

Chronic tendon pathology, or tendinopathy, is a prevalent musculoskeletal condition which affects both athletes and non-athletes (Cook and Purdam 2013). It is an overuse injury characterized by localized tendon pain with loading and dysfunction (Malliaras *et al.* 2013). Currently, the most prevalent model in the research which outlines the stages of tendinopathy is the "Three Stage Continuum Model" put forward by Cook and Purdam (2009). This model incorporates three stages: reactive tendinopathy (tendon attempts to adapt to acute overload); tendon disrepair (tendon reaches this stage if persistent overload continues); and degenerative tendinopathy (unlikely that the tendon will be able to transition back to full repair).

The most commonly affected tendons are the achilles, rotator cuff, patellar, and elbow extensor tendons. Unfortunately, it would not be feasible to cover all tendons susceptible to tendinopathic changes in the scope of this short course. With this in mind it was decided to include the two most prevalent tendinopathies in this booklet, namely Achilles and rotator cuff tendinopathies. Regarding Achilles tendinopathy it has been reported that athletes are up to thirty times more at risk of developing this injury than their sedentary counterparts (Brukner and Khan 2007). In a general population, the incidence of rotator cuff tendinopathy ranges from 0.3% to 5.5% and has an annual prevalence from 0.5% to 7.4% (Littlewood *et al.* 2013). While there is a lack of data on the prevalence of rotator cuff tendinopathy in sporting populations, athletes engaged in sports that involve overhead motions are more susceptible to rotator cuff injury (Scott and Ashe 2006).

The management of tendinopathy can be challenging, and research in this area has failed to provide robust evidence for many of the commonly used interventions (Krogh et al 2012, Coombes et al 2010, Woodley et al 2007). However, since it is broadly agreed that there is much yet to be understood about the causes of tendinopathy (Riley 2008), it is perhaps unsurprising that clinical trials including patients with a range of degrees of tendinopathy report equivocal results (Bennell et al 2010). This short course will attempt to present an overview of the evidence and recommend the current best evidence based approach to management of tendinopathy in athletes. This will incorporate information on the risk factors that can predispose the injury, on the treatment protocols that have been appraised in the evidence and on the psychosocial factors that may need to be addressed in the rehabilitation process leading to a return to sport.

Tendon Anatomy



Tendon Anatomy

Tendons act as links between muscle and bone, enabling force to be transmitted and movement to occur at the joints. Each muscle contains two tendons, one proximal and one distal. The point at which the tendon joins the muscle is known as the musculotendinous junction, while the area where the tendon joins the bone is referred to as the osteotendinous junction (Jonsson 2009). On observation healthy tendons are a white colour with a fibro elastic texture. Tendons are covered by an epitenon which is, in turn, surrounded by a paratenon. The epitenon is a fine, loose connective-tissue sheath through which blood vessels, lymphatics and nerves are all contained (Jonsson 2009). The paratenon is described as being a loose areolar connective tissue covering mainly comprised of type 1 and 3 collagen fibrils, some elastic fibrils and an inner lining of synovial cells (Sharma and Maffuli 2005).

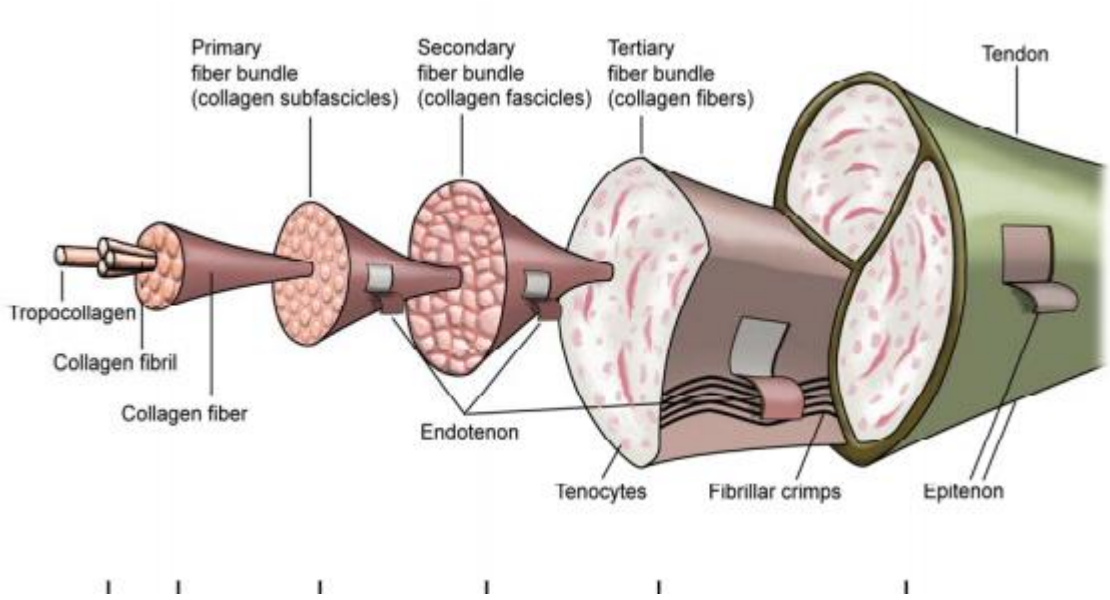


Fig 1.1: Diagrammatic representation of Tendon composition (Bjur 2010).

Tendons comprise of a number of basic elements, which include: collagen bundles, cells and ground substance. Tensile strength of tendons is attributed to the collagen formation within. This collagen formation may be primarily longitudinal, horizontal, or transverse in nature depending on the direction of load which the tendon commonly absorbs. The ground substance provides structural support to the collagen fibres (Sharma and Maffuli 2005). Normal tendons consist of dense, parallel and slightly wavy collagen bundles which display a reflective appearance under polarised light (Sharma and Maffuli 2005).

Tendons are composed, in the majority, by cells known as tenocytes. These cells are fibroblastic in nature; meaning they synthesise the formation of collagen and extracellular matrix (ECM) (Rio et al 2013). The collagen and ground substance referenced earlier is all encapsulated within the ECM and tenocytes lie end to end in channels between the collagen. Communication between these cells, in relation to environmental conditions, are regulated by gap junctions (Rio et al 2013). Calcium concentration, pH and mechanical load all influence the opening/closing patterns of gap junctions and, in turn, the activity of tenocytes.

Blood supply to the tendon is provided by a network of small arterioles which are orientated parallel to the collagen fibres within the endotenon (Cook et al 1998). Despite this blood supply tendons have been reported as displaying approximately 8 times lower O2 consumption when compared to skeletal muscle (Jonsson 2009). The benefit of this is that the tendon can remain under load and tension for longer without risk of ischaemia and injury. However this low O2 consumption also may play a role in impaired and slower healing after injury (Maffuli et al 2009).

Achilles Tendon Functional Anatomy

The Achilles tendon (TA) is the strongest and thickest tendon in the human body, and is formed at the confluence of the gastrocnemius and soleus muscles. The tendon is 15cm long and approximately 6 millimetres in thickness, in which between 80-95% of the collagen fibres are type 1, accounting for the mechanical strength of the tendon (Rompe et al, 2008; Pierre-Jerome et al, 2010). The two gastrocnemius heads and the soleus fuse approximately 5-6cm from its insertion into the calcaneus. The TA inserts into the posterior calcaneus, distal to the posterosuperior calcaneal tuberosity. Potentially the TA would be at risk of wear and tear due to friction between it and the calcaneus. However the calcaneal bursa, situated between the TA and the calcaneus, helps to cushion the area and reduce potential risk of friction damage (Ham et al 2015). Between fusing and inserting, the TA rotates 90 degrees (medial fibres rotate posteriorly and posterior fibres rotate laterally). This rotation is believed to support the elastic recoil of the TA, as the spiralisation of the fibres leads to an area of concentrated stress and can be seen as a mechanically advantageous feature (Pierre-Jerome et al, 2010; Doral et al, 2009). During stance phase of gait, 2.5 times the body weight is

transmitted through the TA and during running this figure increases to 6-8 times body weight through the TA (Pierre-Jerome et al, 2010).

Although the TA lacks a true paratendinous sheath, it is encapsulated by the paratenon; which is continuous with the perimysium and periosteum of the calf muscle and calcaneus (Brukner & Kahn, 2007). The paratenon is a richly vascularised structure which accounts for most of the blood supply to the TA. TA blood supply also originates from the musculotendinous and osteotendinous junctions (Rompe et al, 2008; Brukner & Kahn, 2007). The tendon is relatively avascular, particularly around the region of the confluence of gastrocnemius and soleus, 2-7cm from the insertion to the calcaneus and as such, this is the point at which it is most susceptible to injuries and ruptures (Pierre-Jerome et al, 2010; Brukner & Kahn, 2007). This 5cm portion of the TA also represents the thinnest cross sectional area of the tendon, ranging from .4-1.4cm^2 (Magnusson and Kjaer, 2003)

This avascularity has been identified through angiographic injection techniques (Paavola et al, 2002). The blood supply to the tendon is through branches of the peroneal and posterior tibial arteries. Blood supply to the proximal parts of the tendon is through vessels connecting from the gastrocnemius and soleus muscles (musculotendinous), and to the distal aspects from an arterial plexus at the posterior part of the calcaneal bone (osteotendinous). The distal supply starts at the insertion point and extends proximally for around 2cm (Bjur, 2010). As previously mentioned, due to the limited blood supply to this tendon, the oxygen consumption and metabolism are both low compared to muscles (Maffuli et al, 2011).

Nerve supply to the TA branches from the surrounding musculature and cutaneous nerves. These nerve endings usually terminate on the surface of the paratenon (Tan and Chan 2008). Myelinated nerve fibres are mechanoreceptors (detecting pressure and temperature), while the unmyelinated fibres are nociceptive (detecting pain) (Tan and Chan 2008).

The gastrocnemius muscle consists of 2 heads, with the medial head arising from the medial popliteal surface of the femur, posterior to the medial supracondylar line and the adductor tubercle (Doral et al, 2010). The lateral head originates from the posterolateral aspect of the femoral condyle, from a region extending from proximal and posterior to the lateral epicondyle to the most distal aspect of the linea aspera. The gastrocnemius muscles acts as

both a knee flexor and a plantarflexor of the ankle, and is activated when jumping and running (Doral et al, 2010). As such it contains predominantly type II muscle fibres for this explosive powerful performance (Bjur, 2010). The medial head of gastrocnemius supplies fibres to the posterior and lateral portions of the TA, whereas the fibres of the lateral head predominantly supply the anterior tendon layer. The remaining anteromedial portion of the TA is supplied by fibres from the soleus muscle (Wyndow et al, 2010). The soleus muscle is a large flat muscle that lies deep to the gastrocnemius. Together with the two heads of gastrocnemius, it forms the triceps surae, which acts to plantarflex the ankle. The soleus muscle originates from the medial border of the middle third of the tibia along the soleal line as well as from the proximal third of the posterior fibula, and it inserts into the posterior calcaneus through the TA (Doral et al, 2010). The soleus muscle has a stabilizing effect on the foot and is continuously active during erect standing (Gravare Sibernagel, 2006).

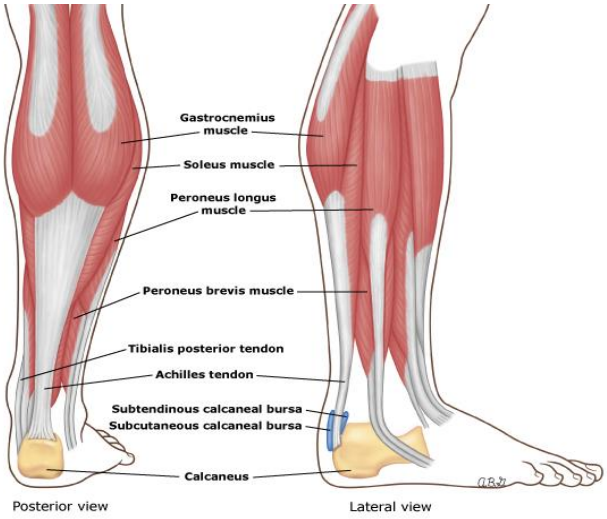


Fig 1.2: Diagrammatic representation of lower limb musculature and TA (Ham et al 2015).

The TA is composed mainly of collagen fibres (65-80%), which are the smallest unit of the tendon that are subject to mechanical strain (Kannus, 2000). In a healthy tendon, up to 95% of the collagen fibres are type I, though through the processes associated with AT and aging, some of these are replaced by type III fibres (Pierre-Jerome et al, 2010). These are predominantly longitudinally orientated fibres but they also run transversally and horizontally (Maffuli et al, 2011). The tight parallel orientation of the collagen fibres transfer great tensile forces from the muscles to the bone, but withstand shear forces less well, and provide little resistance to compressive forces (Freedman et al, 2014; Brukner & Kahn, 2007). Cross

linking takes place between each collagen fibre (Harries et al, 2000). The cross-linking feature of tendons contributes to the tendons ability to transmit great forces, and it also provides protection against enzymatic, mechanical or chemical breakdown of the collagen molecules.

In a healthy TA, the tendon transfers forces which are generated by the gastrocnemius and soleus to move the ankle joint. It also utilises elastic energy to minimise the energetic costs during walking (Wang et al, 2012). By utilising this elastic mechanism, the Achilles acts as an energy provider during locomotion, as it uses its natural elasticity and its spiral orientation to execute a stretch-recoil cycle to release energy during late stance phase. This increases performance and decreases the dependence on the gastrocnemius and soleus for energy production (van Ginkel et al, 2009). The overall elasticity of the tendon is quite low, and only stretches by between 3-8% of its length (Bjur, 2010). Elastin in the tendon is the substance responsible for the flexibility and elasticity of the tendon, and accounts for only 1-2% of its dry weight (Rompe et al, 2008).

Collagen fibrils also play a role in elastic energy utilization as they are said to retain a crimp (Tan and Chan 2008). This is essentially a wavy formation of the fibrils (Fig. 1.1). This crimp, or wavy formation, continues to exist until a strain of greater than 2% is applied to the tendon. This represents the "Toe" portion of the stretch strain curve (Fig 1.3). The initial response to lengthening of the tendon is elongation and straightening of these fibres, as well as elastin fibres providing additional elasticity to the tendon (Paavola et al, 2002). After this point the stiffness of the tendon is increased and if too much strain is applied (>4%) then collagen fibre damage can occur. Ultimate strain of the tendon is approximately 8%, at which point rupture is likely to occur (Tan and Chan 2008).

Along its length the composition of the TA changes, with proximal tendon fibres also containing muscle fibres near the musculotendinous junction. The mid-portion of the tendon contains true tendon tissue, and towards its insertion into the calcaneus, fibres change from tendon tissue to a fibrocartilage mixture, and finally into lamellar bone (Gravare Sibernagel, 2006).

As the collagen accounts for approximately 80% of the tendons mass, and the elastin makes up 1-2%, the remainder of the tendon consists of a proteoglycan-water matrix, within which the collagen and elastin are embedded (Kannus, 2000). This proteoglycan complex is referred to as ground substance, and one of its functions is to help collagen fibres adhere to each other, while also providing lubrication to allow them to slide past each other (Harries et al, 2000). Within the tendon, collagen fibres are tightly packed, and these tightly packed fibres progressively aggregate into larger structures and bundles of fibres, called primary, secondary and tertiary fibre bundles, and amalgamation of these tertiary bundles form the tendon proper (Bjur, 2010). As already mentioned, the TA is surrounded by a peritendinous sheath (paratenon) to reduce friction, rather than a true synovial sheath (Kannus, 2000). This paratenon is a richly vascularised fibrillar elastic sleeve which permits free movement of the TA against surrounding tissues. The paratenon consists of a combination of type I & III collagen fibres, elastic fibrils and synovial cells for lubrication. Beneath the paratenon is a layer of epitenon, beneath which is the endotenon, whose function is to provide structure in the binding of primary, secondary and tertiary fibre bundles (Kannus, 2010).

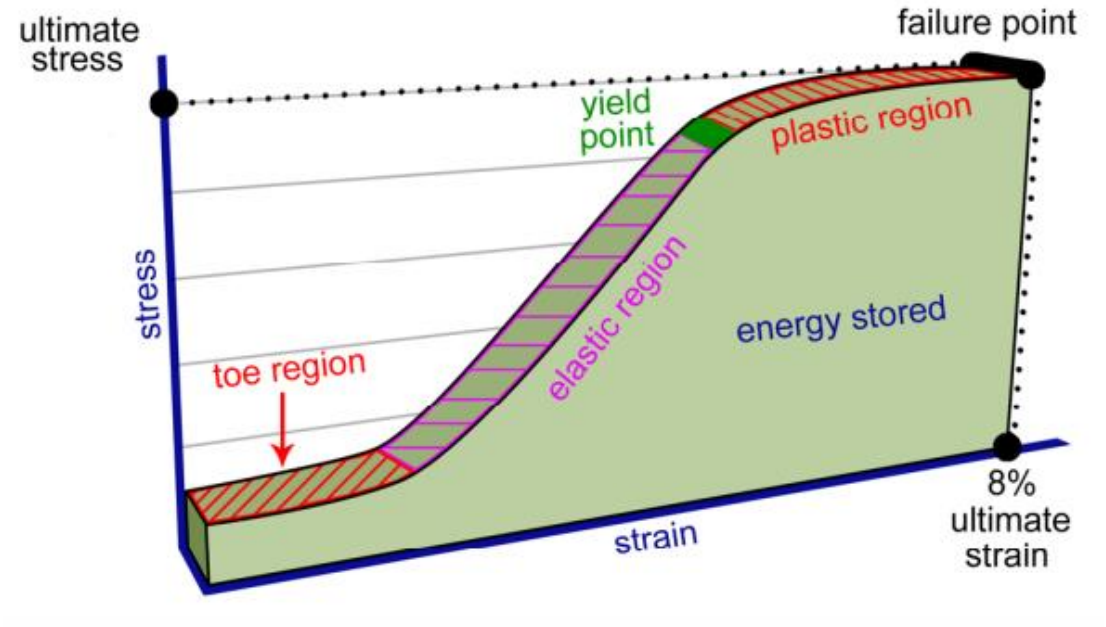


Fig 1.3: Stress- Strain curve associated with tendon loading (Bjur 2010)

Shoulder and Rotator Cuff Functional Anatomy

The shoulder complex is comprised of a set of articulations between the upper limb and the trunk. These articulations are the glenohumeral joint, sternoclavicular joint, acromioclavicular joint and the scapulothoracic articulation. Of all the joints, the shoulder has the greatest range of motion (Hermans *et al.* 2013). The glenohumeral joint involves the articulation between the humeral head and the glenoid fossa of the scapula, and provides the majority of shoulder mobility. As a result of the shallow glenoid fossa the joint relies on static and dynamic stability from the soft tissue structures surrounding it such as the glenohumeral ligaments and rotator cuff musculature (Figure 1.4) (Drake at al 2010).

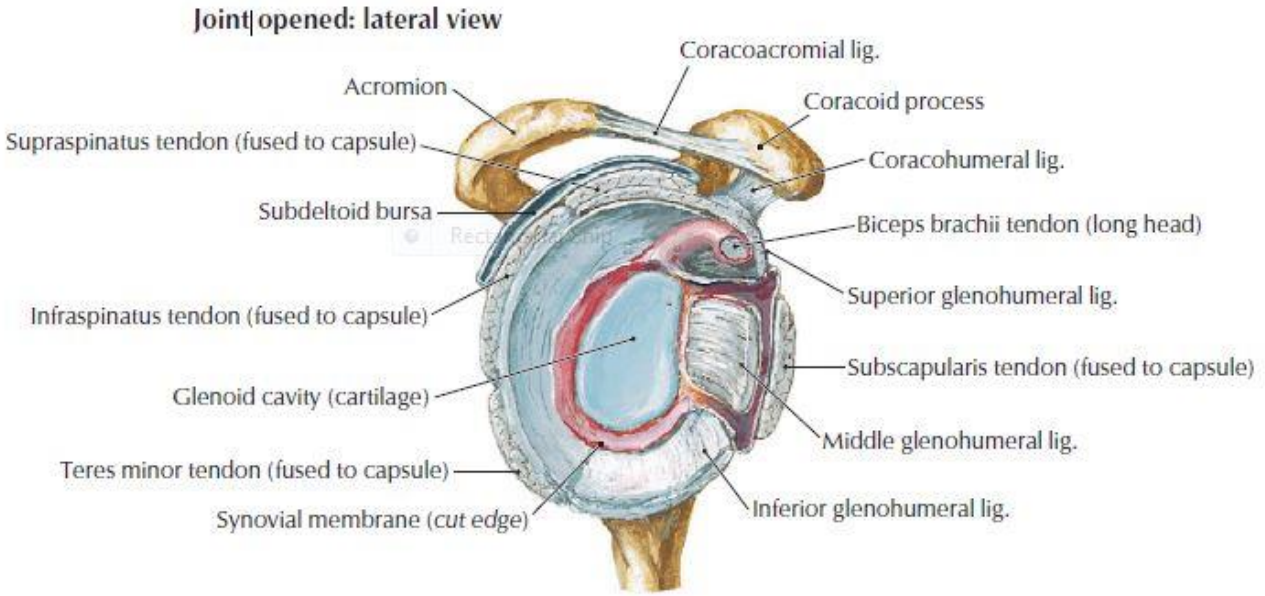


Fig 1.4: Cross Section of Glenoid Fossa (Hansen et al 2010)

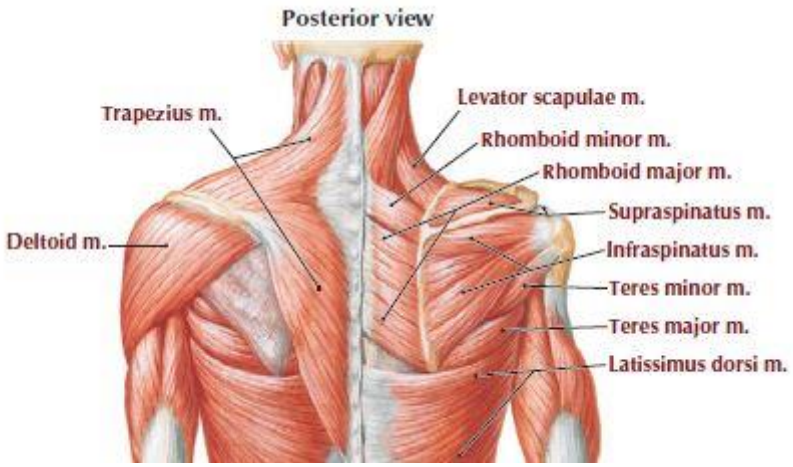
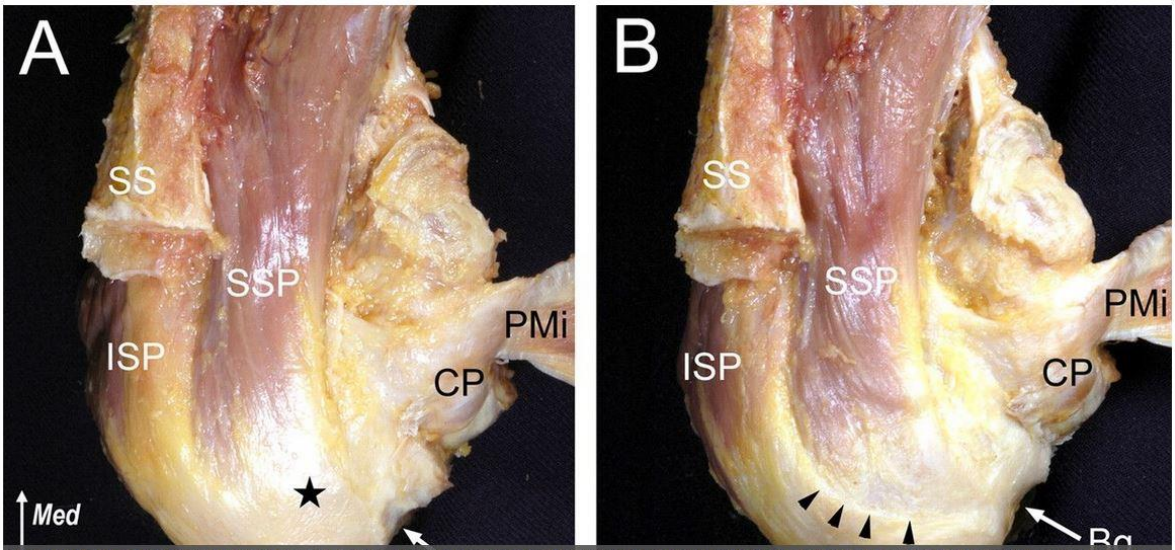


Figure 1.5: Showing rotator cuff muscles and surrounding structures.

The Rotator Cuff is comprised of four distinct muscles: supraspinatus, subscapularis, infraspinatus and terres minor (Figure 1.5). Combined they act as the primary dynamic stabilisers of the glenohumeral joint. In combination they oppose the superior pull of the deltoid muscle (Drake et al 2010).

The subscapulairs is a large and flat muscle arising from the infraspinatus fossa on the posterior surface of the scapula. Its primary function is to provide anterior stability of the glenohumeral joint and also assists in internal rotation (Longo *et al.* 2012). It inserts on the greater tuberosity of the humerus and it is closely associated with the teres minor muscle, arising on the axillary border of the scapula. Both muscles act as external rotators and provide posterior stability to the glenohumeral joint (Figure 1.4).

The supraspinatus muscle arises from the supraspinous fossa of the scapula tracking beneath the coracoacromial arch to insert onto the greater tuberosity. At their points of insertion is it not possible to distinguish the supraspinatus, infraspinatus and teres minor muscles. There is, however, a clear interval evident between supraspinatus and subscapularis. This interval is known as the rotator interval, which is occupied by the long head of the biceps tendon and the coracohumeral ligament (Mochizuki *et al.* 2009).



Photographs of the superior aspect of a right shoulder, after the acromion has been removed, showing the muscular and tendinous structures of the infraspinatus and the supraspinatus near their insertions onto the greater tuberosity. *A:* Connective tissues cover the supraspinatus and infraspinatus near the insertions (star). These tissues make it difficult to separate the infraspinatus from the supraspinatus. *B:* After removal of the overlying connective tissues, the anterior margin of the infraspinatus (arrowheads) is clearly observed. Bg = bicipital groove, CP = coracoid process, GT = greater tuberosity, ISP = infraspinatus, PMi = pectoralis minor, SS = scapular spine, SSP = supraspinatus, *Ant* = anterior, and *Med* = medial.

Fig 1.6: Diagram showing insertion of supraspinatus (SSP) and infraspinatus (ISP).

Despite common misconception the supraspinatus does not initiate abduction but rather all rotator cuff muscles act in unison to achieve arm elevation (Reed *et al.* 2013). The stabilizing versus torque producing roles of the rotator cuff is postulated to vary depending on arm position (Tardo *et al.* 2013). Infraspinatus and subscapularis have the potential to provide a significantly larger contribution in terms of force production, based on physiological cross-sectional area. In contrast the supraspinatus muscle represents only one fifth of the cross sectional area of all the rotator cuff muscles (Mathewson *et al.* 2014). Length-tension relationships appear to be critical to rotator cuff muscle function. The subscapularis exhibits high passive tension in positions of lateral rotation and abduction, whereas infraspinatus and supraspinatus had maximum passive tension in the neutral shoulder position (Ward *et al.* 2006). This would indicate that they have a function in stabilising the joint and not solely in force production. The supraspinatus muscle demonstrates structurally different anterior and posterior parts. Each of these parts has superficial, deep and middle subregions, with contrasting muscle fibre pennation angles (Kim *et al.* 2007). In a study of supraspinatus muscle fibre type by Kim et al (2013) the mean percentage of Type I (slow) fibres ranged from 56.73% to 63.97%. Results demonstrated significant variations in fibre type distribution. The middle part of the anterior region has a significantly greater percentage of Type I fibres compared to that of the posterior. The superficial part of the anterior region has a greater percentage of Type II (fast) fibers compared to the middle and deep parts. (Kim *et al.* 2013). The supraspinatus tendon is comprised of 6-9 independent collagen fascicles. The tendon is capable of compensating for changing joint angles through these structurally independent fascicles and can slide past one another. The tendon attachment exhibits a structure adapted to tensional load dispersion and resistance to compression (Fallon *et al.* 2002).

The insertion point on the greater tuberosity is covered with fibrocartilage. This fibrocartilage or ‘rotator cable’ is a thickened bundle of fibres that runs transversely across the supraspinatus and infraspinatus tendons. The crescent shaped area of the infraspinatus (See Figure 1.6 starred area) and supraspinatus at their distal attachment is termed the ‘rotator crescent’. This rotator cable has been argued to be vital in the retention of function despite the presence of series pathology or tears (Burkhart et al 1993).

Variations in the vascular supply and structure of the rotator cuff have been well documented (Hegedus 2010). Rothman and Parke (2006) described six arteries primarily responsible for supplying the rotator cuff musculature. The suprascapular, anterior circumflex humeral and posterior circumflex humeral arteries were present in 100% of cases studied. The thoracoacromial, suprahumeral and subscapular arteries were also present in other cases studied. The gross vascular supply of the rotator cuff has been best described by Chansky and Iannotti (1991). In their review of the vascularity of the rotator cuff they concluded that the anterior humeral circumflex artery and the suprascapular artery supply the anterior portion of the rotator cuff while the posterior humeral circumflex artery supplies the posterior portion of the rotator cuff. Although there is some agreement in the gross vascular supply and structure of the rotator cuff 60 years of research has debated the microvascularity. The debate has centred on the presence of a microvascular 'critical zone' that leads to tendon pathology. Hegedus (2010) concluded that recent in-vivo studies support the concept of viewing rotator cuff microvascularity on a continuum with no critical zone. This continuum describes an early stage tendinopathy displaying elements of hypovascualrity, hyperaemia with partial tearing and again, hypovascularity with full thickness tears. The results the review support the contention that there is some correlation between age, vascularity and rotator cuff degeneration. However, the relationship remains unclear and warrants further investigation.

Tendon Physiology

The immature form of a tenocyte is referred to as tenoblast and both are arranged in rows between the collagen fibres (Tan and Chan 2008). Together these cells comprise of 90-95% of the cellular composition of tendons. The remaining 5-10% of the cellular distribution is made up of chondrocytes (at the osteotendinous junctions), synovial cells (tendon sheath) and vascular cells which are smooth muscle cells of arterioles and endothelial cells of capillaries (Sharma and Maffuli 2005). Tenoblasts are spindle shaped and are highly metabolically active, as is reflected in the large presence of cytoplasmic organelles (Sharma and Maffulli 2005). As the tenoblast begins to mature it transforms into the elongated form of a tenocyte with a lower nucleus to cytoplasm ratio and a lower resultant metabolic activity. Tenocytes are involved in the synthesis of collagen and all components of the ECM. To provide the energy required to complete the above roles, tenocytes are involved in energy generation

through the krebs cycle, anaerobic glycolsis and the pentose phosphate shunt (Sharma and Maffuli 2005).

As outlined previously collagen fibrils unite to form fascicles ranging from primary to tertiary bundles. More specifically collagen fibrils are the smallest tendon component which can be tested mechanically and are visible under microscopy (Sharma and Maffuli 2005). However collagen fibrils can be dissected further into their smallest constituent, tropocollagen. A triple-helix peptide chain, tropocollagen is water soluble. Only when a number of tropocollagen join and crosslink do they become insoluble fibrils (Sharma and Maffuli 2005).

Surrounding and enveloping the collagen and tenocytes, within the extracellular matrix, is the ground substance. Aside from providing structural support to the collagen, a number of molecules within the ground substance hold important roles within the tendon structure. Proteoglycans (PG) for example are highly hydrophilic, thereby enabling rapid diffusion of water-soluble molecules and the migration of cells (Sharma and Maffuli 2005). A specific PG called decorin helps improve connection, tensile strength and viscoelasticity between the collagen fibrils (Lawerence 2014). Other molecules such as adhesive glycoproteins play a role in tendon repair and healing (Sharma and Maffuli 2005). Tenascin-C is a molecule found in abundance within the tendon. This molecule may play a role in collagen fibre orientation and alignment as the expression of this molecule is regulated by mechanical strain (Sharma and Maffuli 2005).

Tendons which are exposed to mechanical stress are often enveloped in a tendon sheath. Examples of this are the tendons of the hand and feet, where effective lubrication is required (Sharma and Maffuli 2005). Tendon sheaths are two layered with the deepest layer being known as the synovial sheath. This sheath functions to provide synovial fluid to improve lubrication of tendon and limit friction. The outer layer of the tendon sheath is called the fibrotic sheath helps as a fulcrum to improve tendon function. (Sharma and Maffuli 2005). The TA and PT, as mentioned earlier, lack this tendon sheath and instead have a dual layer paratenon.

Complex architecture is necessary at the myotendinous junction to ensure efficient transmission of force from the muscle, through the tendon, and to the bone. At this junction collagen fibrils of the tendon insert into grooves or recesses formed by the processes of long tubular myocytes. Proteins crucial to the integrity of this location include laminin, integrin, Vinculin, fibronectin and talin; they ensure that actin filaments which extend beyond the z-line interlock with collagen fibrils. This anchors the collagen and muscle fibres in place, enabling tension created by the contractile proteins of muscle fibres to be passed to the collagen fibrils (Sharma and Maffuli 2005). Even with this complex architecture in place, the myotendinous junction remains the weakest point of the muscle-tendon unit (Sharma and Maffulli 2005).

As the tendon joins with muscle, the tendon must also join with bone which occurs at the osteotendinous junction. The area can be subdivided into four zones: dense tendon zone, fibrocartilage, mineralized fibrocartilage and bone (Sharma and Maffulli 2005). These zones increase in stiffness from their preceding zone, with the transition from tenocytes to fibrochondorcytes and finally osteocytes. The unique structure of the osteotendinous junction limits collagen fibre bending, shearing and failure (Sharma and Maffuli 2005).

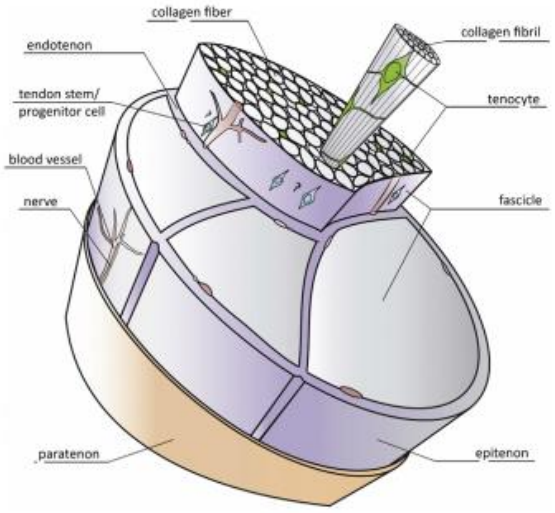


Fig 1.7: Schematic drawing to basic tendon structure (Docheva et al 2014).

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Tendon Healing

The natural healing process of tendons is a relatively poorly understood concept with a lack of detailed histological studies available. Some of the studies exploring this area have been obtained from animal models. On the whole there appears to be three main phases of tendon repair. These phases are not distinct and separate in nature as they overlap and their duration is relative to location and severity of injury (Docheva et al 2014).

Phase 1: Initial Inflammatory Stage

This begins with formation of a haematoma at the site of the injury along with attraction of inflammatory cells such as neutrophils and macrophages by pro-inflammatory cytokines (Docheva et al 2014). A vascular network is created at the site of injury through angiogenic secretions. This network acts as a blood, and primarily oxygen supply to ensure the survival of the immature fibrous tissue forming at the location of the injury (Docheva et al 2014). The initial components of the ECM, primarily the collagen type III fibres, are synthesized by fibroblasts (Docheva et al 2014).

Phase 2: The Proliferation Stage

A few days after the initial injury the synthesis of a number of components of the ECM, for example the proteoglycans and type III collagen, begins. Initially these components are laid down in a random formation (Docheva et al 2014).

Phase 3: The Remodelling Stage

This phase begins approximately 6-8 weeks post injury and includes two sub-stages:

- The consolidation stage – During this sub-stage there is a decrease in cellular permeability and tissue within the tendon becomes more fibrous and Type III collagen fibres being to be replaced by Type I fibres. Fibres, which were initially laid down randomly, start to re-organise along the longitudinal axis of the tendon, thereby improving the strength of the tendon. (Docheva et al 2014).
- The maturation stage: In this stage collagen fibrils begin to crosslink and the formation of mature collagen tissue begins.

In total this process can take 1-2 years in total depending on patient age and condition (Docheva et al 2014).

Tenocytes are involved in both the synthesis and degradation of components of the ECM, thereby ensuring the slow and continuous process that is tendon remodelling (Docheva et al 2014). It has been hypothesised that there are two cooperative mechanisms of tendon healing, referred to as extrinsic and intrinsic healing (Docheva et al 2014).

Extrinsic: it is proposed that fibroblasts and inflammatory cells migrate from the peripheral areas of the tendon and contribute to cell infiltration and adhesion formation (Docheva et al 2014).

Intrinsic: This occurs after the extrinsic phase and includes migration and proliferation of intrinsic endotenon cells to the injury site. These cells help reorganise the ECM and support the vascular network (Docheva et al 2014).

In most individuals, especially those who are older, the mechanical properties of the healed tendon is usually inferior to those of the uninjured tissue. This strength deficit arises due to the higher ratio of Type III collagen fibres to Type I, along with reduced integration of these fibres (Docheva et al 2014).

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Terminology

Terminology in tendon pathology

Tendinitis: A histopathological description of tendons which show inflammatory features however this term is sometimes misused to describe the clinical syndrome.

Tendinosis: A histopathological description of degeneration in all components of the tendon, including collagen fibres, tenocytes and extracellular matrix.

Tendinopathy: Umbrella term for all tendon overuse conditions, such as pain, swelling and impaired performance. (Padhiar et al 2010)

Tendinosis Paradigm (1990's)

Prior to the 1990's tendon pain was referred to tendinitis, which reflected the belief that inflammation was the primary driver of the pathological process (Rees et al 2013). This view was ingrained deeply in the medical literature of the time and, as a result of this belief, the primary treatment for this condition was non-steroidal anti-inflammatories and corticosteroids (Rees et al 2013).

In the 1990's there was a shift in the literature away from this inflammatory model of tendon disorder. This shift was in light of studies being published at the time which failed to demonstrate the presence of any acute inflammatory cells in the load bearing areas of tendons. Other studies began describing symptomatic tendons which displayed collagen thinning and separation without any inflammatory infiltrate (Rees et al 2013). With this evidence there was a strong push for the tendinosis model describing tendon pain arising completely without the presence of any inflammation.

Tendinosis Paradigm (2000's)

This shift towards a non-inflammatory based model of tendon disorder has continued into the 21st century with a new "Degenerative" model being established. This umbrella term includes the cumulative damage, vascular insufficiency modes and failed healing response; all of which are commonly associated with the modern thinking around tendinopathy. Ironically the tendinosis model has now become as deeply ingrained in the medical literature as tendinitis had been previously. This puts our understanding of the pathological process at risk of being oversimplified and result in potentially effective treatments being ignored (Rees et al 2013). Recent improvements in immunohistochemistry and gene expression analysis have enabled a more accurate understanding of the underlying pathophysiology at work within tendinopathy.

Histology and Pathology

Inflammation

As mentioned previously inflammation plays an initial role in normal tendon healing, however histological studies have failed to categorically identify the presence of the inflammatory cells (Neutrophils and macrophages) in tendinopathy. Recent advances in technology have enabled greater specificity and accuracy when detecting the presence of inflammatory cells within tendons. A number of studies have shown the presence of elements of an inflammatory reaction within established tendinopathy (Rees et al 2013).

Schubert et al (2005) for example analysed the composition of 10 TA samples, from patients with a history of tendinopathy. They demonstrated the presence of macrophages, along with T and B lymphocytes, was significantly higher in this sample. In conjunction with this, Schubert et al (2005) also examined 10 samples of spontaneously ruptured tendons. They identified the presence of a large numbers of granulocytes but did not see significant numbers of macrophages or T/B lymphocytes. Concurrently, samples of granulation tissue from patients with AT frequently contained haemosiderophages. This granulation tissue consisted of groupings of capillaries embedded in a fibroblast rich stroma with evidence of macrophage and B/T lymphocyte infiltration.

Hyperplasic and hypertrophied tenocytes have also been discovered in pathological tendons, which may provide indirect evidence of up-regulated inflammatory mediators (Scott et al 2007). Proliferation and increased metabolic activity of tenocytes is known to occur in the presence of cytokines and growth factors which are part of the inflammatory response (Rees et al 2013). A drawback to the study by Scott et al (2007) is that it was completed in an animal population and the results of this study cannot be extracted directly across to a human population.

However, studies by Kragsnaes et al (2014) and Dean et al (2014) have explored the presence of inflammatory cells, in human populations, in achilles and rotator cuff tendons respectively. Kragsnaes et al (2014) compared tissue samples between groups of non-ruptured chronic TA tendinopathy and a healthy population. All biopsies were analysed immunohistochemically for the presence of macrophages, hemosiderophages, T lymphocytes, B lymphocytes, natural

killer cells, schwann cells and endothelial cells. Significantly greater numbers of macrophages and endothelial cells were observed in the symptomatic population, versus the healthy population. Similarily Dean et al (2014) compared inflammation cell prevalence in healthy samples versus those presenting with rotator cuff tendinopathy. Findings of significantly higher presence of leucocytes and macrophages in the tendinopathy group were reported. The identifying and quantifying of the presence of these cells is not an admission of their activity however (Kragsnaes et al 2014).

While the area of inflammation in tendinopathy is poorly defined, over time our understanding of the pathological process may resemble the current understanding of the process of osteoarthritis; degeneration and mechanical overload being the key driver of pathology with elements being mediated through inflammatory response (Rees et al 2013).

It is currently unclear the exact role inflammation may or may not play in tendinopathy; either way with pathology the degenerative process soon supersedes this (Rees et al 2009). Disorganised healing, intra-tendinous degeneration, hyper cellularity and fibre disorientation are the primary histological findings in symptomatic tendons (Sharma and Maffuli 2005).

Matrix Changes

It is now understood that the swollen appearance of pathological tendons, namely achilles and patellar, is not as a result of inflammatory process as suggested initially. It is secondary to altered cell permeability and increased production of large proteoglycans (PGs) (Rio et al 2013). Large PGs, especially aggrecan, attract and bind H2O which results in causing the tendon to swell; thus this is completely devoid of an inflammatory response. It has been suggested that PGs may not only cause tendons to swell, but may also have a role in cell-matrix interference and tendon pain (Rios et al 2013).

For example, the swelling of the tendon will stimulate local c-fibres and increase hydrogen and potassium concentrations. This may influence ion channel activation and, in turn, stimulate nociceptors and be received as pain (Rio et al 2013). It also have been suggested that large PGs may disrupt communication between cells, resulting in a loss of gap junctions between parallel rows of tenocytes. The disruption of gap junctions could influence homeostasis sufficiently to bring about a nocioceptive response (Rios et al 2013). On the

other hand, impaired gap junction function could act to protect the tendon by isolating the area of damage and prevent toxic communication between cells (Rios et al 2013)

Tendon Appearance

Tendons which, usually appear white with a fibroelastic texture to the naked eye, appear grey/yellowish-brown and display a soft, fragile and oedematous texture (Kaux et al 2011). This is described as mucoid degeneration; commonly seen in TA, patellar tendinopathy and rotator cuff tendinopathies (Sharma and Maffuli 2005). The presence of large mucoid patches and vacuoles between collagen fibres is commonly associated with this form of degeneration. Lipoid degeneration, also seen in TA tendinopathy, refers to the abnormal presence of intratendinous lipid with a consequent disruption of collagen structure (Sharma and Maffuli 2005).

Under light microscopy tendinopathy also shows a number of histological changes:

- Disorganised and disrupted collagen with a loss in typical hierarchical structure (Riley 2008).
- Abnormally increased production of type III collagen, commonly associated with wound healing, by tenocytes local to the area of the tendinopathy (Cook et al 2002).
- Increased ground substance along with higher concentrations of proteins such as glycosaminoglycans and proteoglycans (Rees et al 2009). Increased turnover of proteoglycans results in alterations in tendon homeostasis and contribute to tissue dysfunction (Rees et al 2009).
- Cellularity changes with altered tenocyte concentration and appearance (Cook et al 2002).
- Loss of cellular homeostatic tension (Cook et al 2002) and increased apoptosis rates which may be related to oxidative stress (Millar et al 2009).
- Neovascularisation, as demonstrated on colour and power Doppler ultrasound (Alfredson et al 2006).

Cellular Appearance

Tenocytes react and respond to their environment, be that ionic, mechanical or osmotic. In tendinopathy, tenocytes begin to proliferate, become rounded in shape and have a greater proportion of protein-producing organelles. The changes appear to increase the production of receptors and substances involved in pain (Rios et al 2013); along with impacting the communication between cells though gap junctions as outlined previously.

Neovessels

Altered blood flow, dissimilar to that of a healthy tendon, is a common finding in Doppler ultrasound (Rees et al 2013). This change in blood flow is referred to as neovascularisation (Rees et al 2013). Angiogenesis, or the sprouting of new blood vessels, is a common histological finding in studies examining chronic tendinopathy (Rees et al 2013). It is hypothesized that this process of neovascularisation may be linked to tendon repair (Alfredson et al 2009) or the development of chronic pain (Knobloch 2008). Nerves and receptors, such as adrenoreceptors, which are found in vessel walls in tendinopathy are unlikely to be associated with pain; these are likely associated with angiogenesis (Rios et al 2013).

Along with angiogenesis, a body of literature also suggests neo-innervation accompanies the presence of neovessels. It is hypothesised that neural sprouting may be a cause or contributor to pain within the pathological tendon (Alfredson et al 2003). However there is currently limited evidence to support this hypothesis as presence of neural sprouting is not correlated with painful tendons (Rio et al 2013). This neural ingrowth comprises mainly of sympathetic nerves whose function is to regulate neovessels, these nerve fibres are not sensory in nature (Lawerence 2014)

A study by Sengkerij et al (2009) demonstrated the presence of neovascularization in the majority of the symptomatic TAs examined (N=33). However this study also reported that the degree of the presence of neovascularisation was not collated with the severity of the symptoms. Although neovascularisation has been associated with tendinopathy, not all painful tendons display increased vascularity (Cook et al 1998). It is also true that not all tendons with increased vascularity are painful (Cook et al 2004).

While it is fair to say that tendinopathy commonly displays features of neovascularisation and neural spouting, it is not accurate to correlate these changes with increased pain across all pathological presentations (Rios et al 2013).

Biochemical Changes

A myriad of biochemical changes occur in tendinopathy, however none of these would fully explain or account for tendon pain. It is likely that substances which have both pro- and anti-inflammatory effects play a role in mediating tendon pain (Rios et al 2013). This would include chemicals such as cytokines, interleukin and signalling molecules such as calcium (Rios et al 2013).

Cytokines [Tumour necrosis factor-alpha (TNK-a)] and interleukin [Interleukin-1-Beta (Il-1b)] for example are implicated in tendinopathy (Rios et al 2013). TNK-a is capable of matrix structure changes and inducing apoptosis. While Il-1b, along with inducing apoptosis, can cause cell proliferation (Rios et al 2013). Other cytokines, such as glial cells, have not been examined in tendinopathy but could impact communication of tendon pain as they are crucial in synaptic transmission (Rios et al 2013).

A neuropeptide known as substance-p (SP) is another chemical implicated in tendinopathy (Rios et al 2013). SP causes vasodilation and protein extravasion in the surrounding tissue in a process called peptidergic inflammation. This process is initiated by nocioceptor activation in the tissue which could be brought about by the increased tenocyte metabolism and proliferation caused by SP. Potentially this could result in a painful stimulus being received from the tendon.

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Degenerative Model

The most prevalent model in the research which outlines the stages of tendinopathy is the "Three Stage Continuum Model" put forward by Cook and Purdam (2009). These stages include some of the histological information outlined previously however, as part of the continuum model, it enables to structure when these changes occur. These stages of tendinopathy are outlined as follows:

Reactive Tendinopathy:

This occurs as the tendon attempts to adapt to acute overload. In this stage the tendon thickens due to increased cellular activity, proliferation and permeability along with the presence of aggrecan which binds to water. This response results in pushing of the collagen fibres apart and the overall swollen appearance of the tendon. Tenocyte shape distortion likely occurs due to surrounding pressure, impacting upon the function of the tendon cells. The tendon is attempting to buffer the overload by increasing stiffness while also attempting to reduce the stress by increasing volume. Importantly however during this stage fibre integrity is maintained, which influences the reversibility of this stage of tendinopathy. While this can be a painful stage of tendinopathy, full repair is achievable.

Dysrepair:

The tendon reaches this stage if persistent overload continues. Increased protein production continues however the tendon enters the phase of failed healing. Structural changes occur, such as the increased production of weaker Type III collagen, neovascularisation and neural ingrowth. As outlined previously, it is unlikely that neovessels and sprouting nerve fibres are involved in pain in tendinopathy. However they may provide a sign-post informing when the tendon has transitioned from reactive to late dysrepair. Clinically this stage often presents with ongoing intermittent symptoms however the structural changes which have occurred during this phase resulting in a limited capacity of full repair.

Degenerative:

This is the final stage of the tendinopathy continuum. Within this stage there are widespread structural changes. Type III collagen continues to replace Type I, while areas of reduced cellularity exist among a normally hyper cellular matrix. The thinner and irregular fibres reduce the tendons ability to absorb load, placing the tendon at risk of rupture. 97% of

spontaneous ruptures display degenerative changes which undoubtedly pre-date the rupture. The capacity for recovery from this stage is poor and it is very unlikely that the tendon will be able to transition backwards through the stages to full repair. However the tendon should still be able to adapt and tolerate functional loading.

A pictorial representation of this continuum can be seen in Fig ?

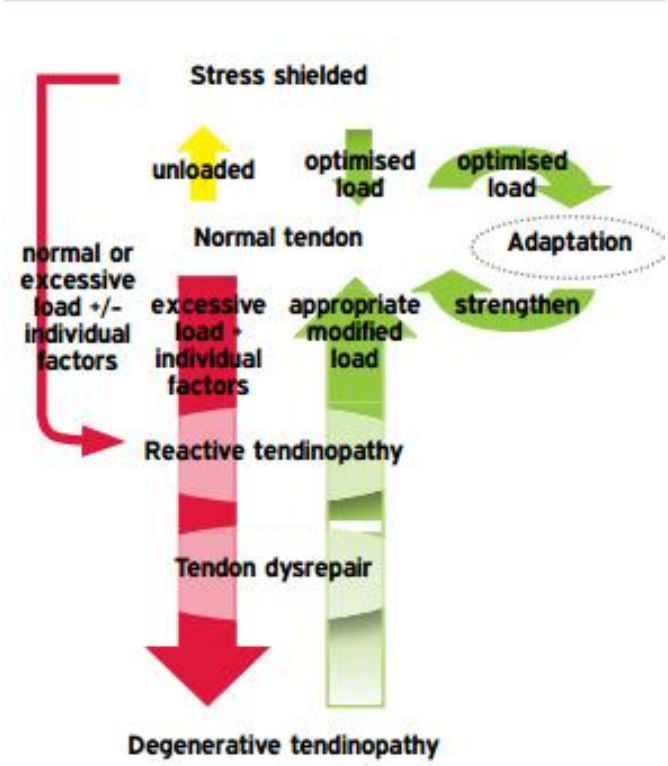

Fig 1.8: Pathology of load-induced tendinopathy (Cook and Purdam 2009)

The Iceberg Theory

Another theory put forward which attempts to rationalise the events occurring within the tendon and the occurrence of pain is the "Iceberg Theory", which was first proposed by Abate et al (2009).

To understand this theory one must first acknowledge the natural events that occur during loading within a physiological range deemed tolerable by the tendon. In this range, load does not damage the tendon but instead reinforces it as it stimulates the production of new collagen fibres (Abate et al 2009). Various forms of exercise which load tendons (e.g. eccentric, eccentric-concentric) have been shown to demonstrate, via microdialysis techniques, up-regulation of both the synthesis and degradation of collagen within the tendon. However, collagen synthesis not only outmatches collagen degradation; it also occurs for longer (Langberg et al 2007, Olesen et al 2007). The overall result of this synthesis and degradation is that the tissue within the tendon becomes stronger, more resistant to injury and demonstrates increased tensile strength and elastic stiffness (Corps et al 2008).

When tendons are pushed outside the normal physiological range of loading is when tendinopathy can occur (Abate et al 2009). Overload and repetitive strain results in collagen fibres sliding past each other, causing cross-link breaking and tissue denaturation (Abate et al 2009). Strenuous exercise also may result in high temperatures developing within the tendon itself. When poorly controlled this can result in cell death, namely fibroblasts which cannot sustain temperatures greater than 42.5^{0}C (Temperature of 43-45^{o}C have been demonstrated in tendons). In hypo-vascular areas of tendons, where homeostasis and temperature regulation can be limited, the tissue may be at greatest risk of degeneration (Li et al 2004).

Tendons, as mentioned previously, have low metabolic rates therefore optimal conditions for healing are: sufficient recovery time void of further overloading and with suitable blood supply (Abate et al 2009). Extrinsic factors, such as excessive sporting participation and training errors, along with intrinsic factors, such as osteoarticular pathologies and diseases impacting collagen metabolism, both may play a role in optimal healing conditions being achieved (Abate et al 2009). Considering these potential variables, healing potential may vary between individuals (Abate et al 2009).

The iceberg theory attempts to explain the differences between the extent of tissue degeneration and clinical presentation of pain. It propagates the idea that the events that occur within the tendon (i.e. collagen thinning etc.) lay hidden under the surface and only when a certain threshold of degeneration results in pain being registered (i.e. the tip of the iceberg). This theory acknowledges that there is two phases in tendon damage; the asymptomatic and symptomatic phases. It also acknowledges the potential relapse of symptoms which can be seen in some patients. Primarily, however, it explains how extensive tendon degeneration, enough to result in full rupture, can be painless prior to injury (Chester et al 2007).

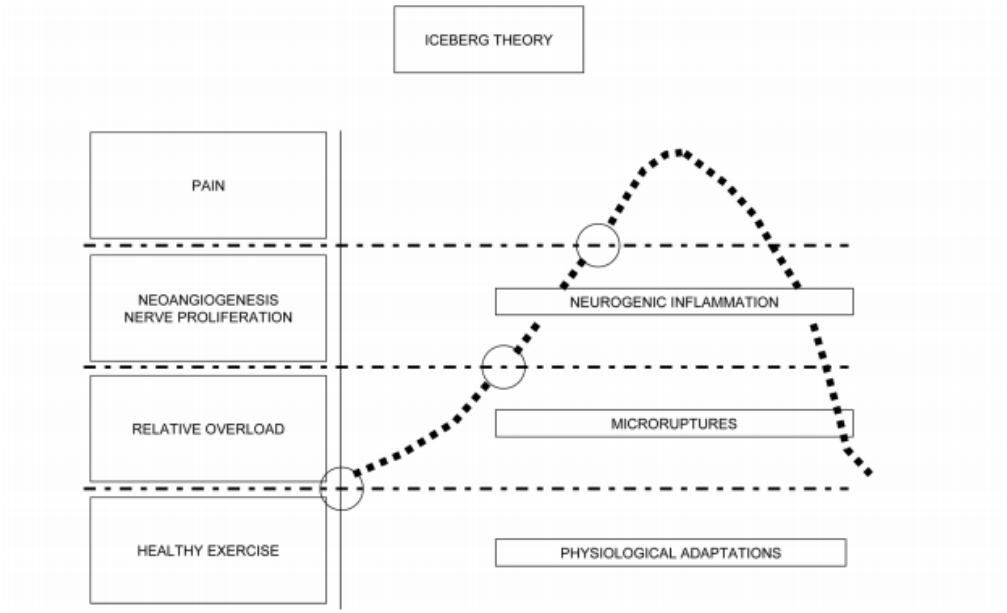

Fig 1.9: Diagrammatic representation of the Iceberg Theory (Abate et al 2009).

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Imaging

While tendinopathy is often diagnosed using clinical sign and symptoms reported by the patient, imaging tools such as ultrasound (US) and magnetic resonance imaging (MRI) have been used to assess or confirm diagnosis of tendinopthy (Ham et al 2015). While these imaging techniques can be used to out rule other conditions, such as rupture or fracture, these methods can also outline degenerative changes that have occurred within the tendon (Ham et al 2015).

Ultrasound is increasingly used to assess changes within the tendon, for example: tendon thickening, neovessels, hypoechogenicity, disordered fibres and tissue gaps (Ham et al 2015). It is a quick, inexpensive and accessible, with the added benefit of providing dynamic images of the tendon (Ooi et al 2015). MRI on the other hand is more expensive, less accessible and is more advantageous in the differential diagnosis of cartilage/bone involvement (Ooi et al 2015). Ultrasound is deemed more applicable to tendinopathy secondary to its greater spatial resolution and ability to detect changes such as neovascularisation, however it is limited in that it only produce a 2D representation of a 3D structure (Antflick and Myers 2014).

While both colour Doppler ultrasound and MRI has been found to be accurate when diagnosing patellar tendinopathy in 83% and 70% of cases respectively (Warden et al 2007), the research investigating associations between imaging abnormalities and the clinical presence of AT have been conflicting. One study by Emerson et al (2010) compared detected changes on ultrasound and reported symptoms in 41 elite gymnasts and found that US overestimates the presence of tendinopathy in one third of cases. Similarly a small prospective study with a study population of 41 found that US (and MRI) showed only moderate correlation with a clinical assessment of TA (Khan et al 2003).

Technological advances in imaging however are beginning to yield more accurate results. Axial-strain sonoelastography (ASE) is a method of US which evaluates the mechanical properties of tendons. Specifically it looks at the amount of strain in tendons which is based on the idea that tissue responds to external compression thereby producing strain within the tissue; the lower the strain, the harder the tissue (Klauser et al 2010). 120 participants presenting with TA were included in a study by Ooi et al (2015), comparing ASE-US against b-mode and colour Doppler US. ASE-US was reported to demonstrated significantly greater

accuracy and reliability when compared against the other two imaging methods, along with excellent correlation with clinical findings (Ooi et al 2015).

Another developing method of US, known as Ultrasound Tissue Characterisation (UTC), appears to be promising method of imaging. This imaging technique provides detailed 3d views of the integrity of the tendon matrix. Different imaging colours represent various structural formations.

- Echo type 1 (Green) – Intact and aligned collagen fibres
- Echo type 2 (Blue) – Discontinuous collagen bundles
- Echo type 3 (Red) – Higher proportion of thinner collagen fibres
- Echo type 4 (Black) – Mainly cellular components and fluid in undefined tissue (Antflick and Myers 2014).

This method of imaging has potential to detect subtle changes in tendon structure not seen previously; however more research is required to validate fully the accuracy of UTC (Scott et al 2013).

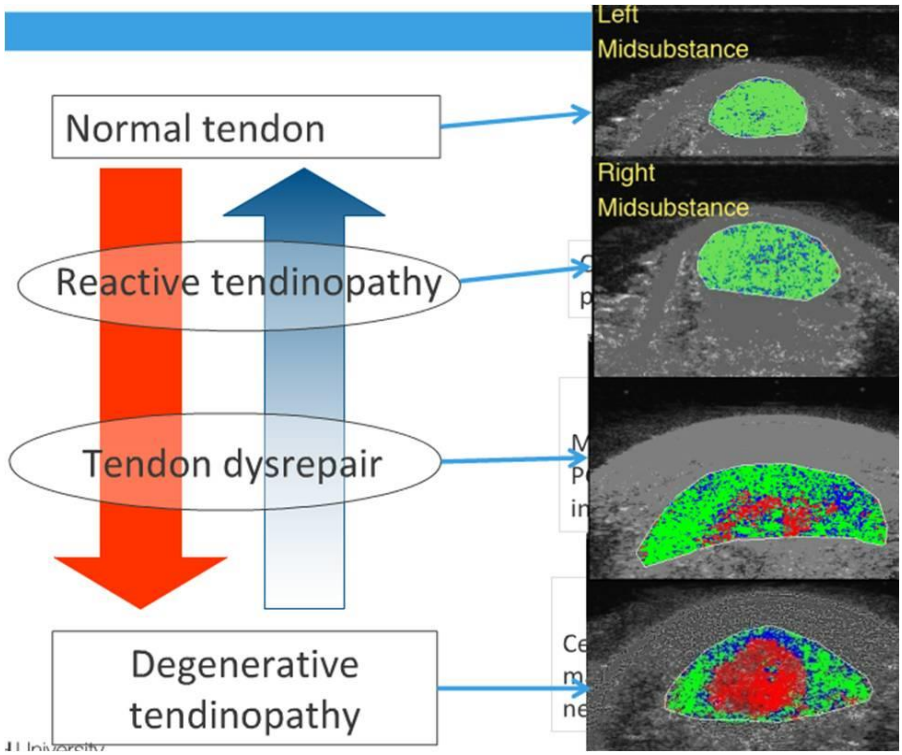
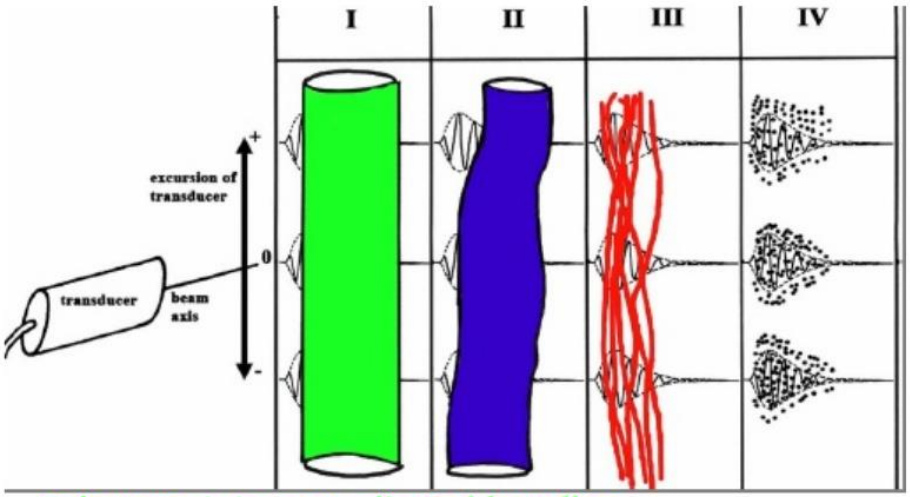


Fig 1.10: Comparison between stages of continuum model and UTC findings **(Docking 2014).**

Ultrasound tissue characterisation



Echotype I- Intact, aligned bundles

Echotype II- Increased waviness/separation of fibrils

Echotype III- Decreased fibrillar integrity

Echotype IV- Absence of fibrillar organisation

Fig 1.11: Overview of colour significance on UTC (Antflick and Myers 2014).

Achilles Tendinopathy

Prevalence

- Injury to the TA is very common among a variety of populations, from elite athletes to mostly sedentary individuals. As with many injuries of this aetiology, they present more commonly among the athletic population than in inactive people, and it has been reported that athletes are at up to thirty times greater risk of developing Achilles Tendinopathy (AT) than their sedentary counterparts (Brukner & Kahn, 2007).
- AT has been reported most commonly among individuals who participate in middle and long-distance running, tennis, badminton, volleyball, track and field, and soccer (Paavola et al, 2002).
- Siobhani et al (2013) investigated the prevalence of AT in ballet dancers, and found it to be up to 338.5/1000.
- Conflicting reports of prevalence have been reported in runners, both elite and amateur. It is accepted that incidences are increased in those who regularly run, though the numbers vary. Reports of between 25% and 50% are common in the literature (Siobhani et al, 2013; Abate et al, 2012; Gaida et al, 2010; Kraemer at, 2012; Fredburg & Stengaard-Pederson, 2008).
- Kraemer et al (2012) reported AT as the second most common overuse/chronic injury in rugby players, only behind chronic lateral ankle instability.
- Similar to that in ballet dancers, Joseph et al (2012) reported a lifetime prevalence of AT in male gymnasts at approximately 40%.
- In a study of elite professional footballers across Europe, Gajhede-Knudsen et al (2013) reported that 2.5% of all injuries sustained by soccer players were AT.
- The reported prevalence of AT in the sedentary population varies within a limited range throughout the literature, thus suggesting consistency of the findings. Authors claim that the prevalence of the condition is between 5.9%-10% (Fredburg & Stengaard-Pederson, 2008; de Jonge et al, 2011; Gaida et al, 2010; Abate et al, 2012).
- Another interesting finding in the literature is the presence of AT related changes in asymptomatic tendons. Gaida et al (2010) found that approximately 13% of asymptomatic runners had evident pathology under ultrasound investigation. Joseph et al (2012) described more modest findings in a non-athletic population, suggesting that 3.8% of people had sub-clinical degenerative changes in the TA. Contrary to these, other findings show much more significant incidences of asymptomatic degeneration

in tendons, with Joseph et al (2012) describing sub-clinical changes in up to 40% of male gymnasts. The investigations by Magnan et al (2014) suggested similar levels to this in runners, with 34% of people without pain showing ultrasonographic changes.

- The incidence of AT has been reported as high in middle to long distance runners, however in a study of former elite athletes in Finland, Kujala et al (2005) found that sprinters more commonly suffered from TA ruptures that tendinopathy.
- Across the literature it is reported that up to 30% of those who suffer AT have bilateral symptoms (Öhberg & Alfredson, 2004), and that over 40% of patients develop symptoms on the contralateral side, up to 8 years after the initial appearance of AT (Paavola et al., 2000).
- AT has been most commonly reported in individuals between the ages of 30-60 years, and in recent years there has been no distinction between the prevalence in men and women (Paavola et al, 2002; Gravare Sibernagel, 2006; Longo et al, 2009).
- As the presence of AT is a clinical diagnosis using subjective and objective assessment, the presence of ultrasonographic changes has been shown to have limited correlation with diagnosis. In a study by Comin et al (2013), the presence of sonographic abnormalities was common in ballet dancers, but only the presence of hypoechoic changes was predictive of future tendon pathology. Thus the presence of abnormalities detected using ultrasound imaging cannot constitute prevalence rates (Boesen et al, 2011).

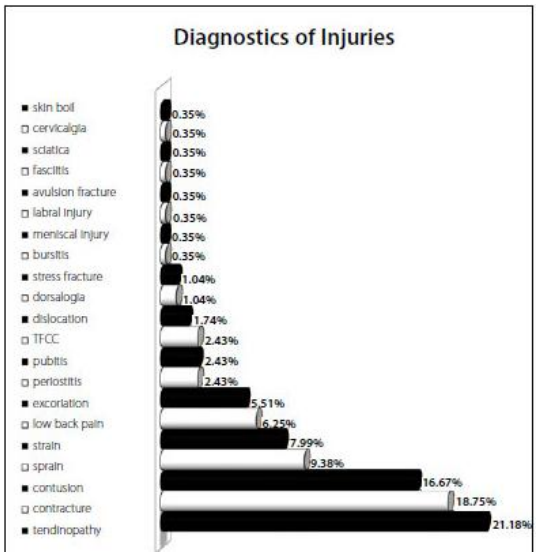


Fig 2.1: Diagnostics of the sports injuries(Silva et al, 2011)

Risk Factors

Dorsiflexion ROM & AT

Dorsiflexion range of motion (DF ROM) has been cited as a risk factor for the development of AT. Alterations in dorsiflexion range of motion have been associated with higher incidence and risk of AT (Carcia et al, 2010). A prospective study carried out by Mahieu et al (2006) measured DF ROM with knee flexed and extended, in order to isolate the significance of gastrocnemius and soleus individually. Their findings illustrated that increased passive dorsiflexion (<9°) in knee extension tended towards a significant association with the development of AT.

A study of similar design justified measuring dorsiflexion with an extended knee as the knee is flexed to approximately 40° in mid stance of running, when the ankle is in greatest dorsiflexion. Conversely to the findings in the previous study (Mahieu et al, 2006) the most significant finding in this group was that in the AT group ankle dorsiflexion with the knee flexed was limited (Rabin et al, 2014). Unit odds ratio indicated that for every 1° increase in DF ROM, the chances of developing AT were reduced by 0.23. It was also demonstrated that non-weight bearing range of dorsiflexion of less than 22° is a cut-off point for increased risk of developing AT (Rabin et al, 2014).

Somewhat supporting both studies, the findings of a 2-year prospective study of risk factors indicated that alterations in the DF ROM (increased or decreased) leads to increased susceptibility to AT (Kaufman et al, 1999). With knee extended, decreased DF ROM (<11.5°) makes a person 3.5 times more likely to develop AT. Alternately, increased dorsiflexion ROM (>15°) leads to 2.5 times greater risk of developing AT (Kaufman et al, 1999). These findings were supported by a systematic review which also finds reduced DF predisposes to AT (Manteanu & Barton, 2011).

The effect of limited dorsiflexion is theorised to contribute to the development of AT by increasing the pressure of shock absorption by the plantarflexors during running, and increasing the strain on the TA (Rabin et al, 2014). This has an effect of reducing the internal loading capacity of the TA (Manteanu & Barton, 2011). Another proposed mechanism for its effect is traction from the soleus muscle on the TA. As the deep calf muscle attached to the medial aspect of the TA, an area where 91% of US changes are found, it is hypothesised that

traction from an inflexible soleus muscle may lead to AT (de Vos et al, 2012). The interrater reliability of ankle dorsiflexion goniometric ROM assessment is reported to be up to 0.69, with excellent intrarater reliability of 0.90 (Martin & McPoil, 2005).

Adiposity and AT

Adiposity is believed to have a strong association with the development of AT. There are 2 proposed mechanisms of action for the influence of adiposity, which are mechanical and systemic processes (Gaida et al, 2009). The mechanical process suggests that higher levels of adiposity leads to increased body weight and higher forces travelling through the tendon. This sustained higher load puts increased strain on the tendon and leads to AT. This systemic effect explores the interference of bioactive peptides released by adipose tissue on the tendon structure. Increased adipose tissue increases the risk of cardiovascular disease, with increased low-level inflammation due to cytokines, and increase resistance to the flow of blood. This increased resistance is proposed to have negative influences on the healing capabilities of the tendon, with reduced oxygen supply and metabolism (Gaida et al, 2009). In an investigation of the effects of adiposity on the presence of ultrasonographic tendon changes Abate et al (2012) found that there was a significant prevalence of tendon changes in overweight runners. There were less sonographically visible changes in runners with lower body mass index (BMI), or sedentary individuals who were overweight (Abate et al, 2012). The authors also proposed an explanation for this, suggesting that fat distribution and adiposity may influence the function of the tendon components such as tenocytes and blood vessels. Their justification was that when the overweight runners caused micro trauma to the tendon, appropriate healing mechanisms were delayed by this influence, and degenerative changes occurred (Abate et al, 2012).

Gaida et al (2010) stated that men with a waist circumference of greater than 83cm were at greater risk of developing AT, with 33% of these individuals suffering from the condition. They also found that men had greater central fat distribution, and women predominantly had a peripheral distribution of fat, due to the influence of oestrogen on the prevention of central accumulation of fat (Bagge et al, 2011). Gaida et al (2010) found that 43% of the group with AT had significantly greater adiposity levels than the control group

Subtalar joint motion & pronation

Subtalar joint motion has been shown to have an association with the presence of AT. Abnormal subtalar joint range of motion is determined by increased range of motion in the planes of inversion and eversion (Munteanu & Barton, 2011). Both increased inversion and eversion have been shown to be present in sufferers of AT (Rabin et al, 2014; Kaufman et al, 1999). A possible mechanism for the influence of subtalar eversion is that limited dorsiflexion (DF) may lead to compensatory pronation of the foot, which in turn will cause internal rotation of the tibia. This internal rotation will be corrected to tibial external rotation when the knee is extended. This increased rotation of the tibia causes "wringing" of the TA, and forces the gastrocnemius and soleus to produce greater forces to plantarflex the foot during terminal stance of running (McCrory et al, 1999). Munteanu & Barton, 2011) The tightness of the TA & gastrocnemius-soleus complex has also been owed to increased pronation, as the reduced ROM of DF may be due to prolonged contraction of the triceps surae in an attempt to control pronation (O' Donoghue et al, 2008). Also, as 91% of US detected mid-portion disorders of the TA occur on the medial segment, there is believed to be an association between hyper-pronation of the foot leading to excessive posteromedial strain on the TA (de Vos et al, 2012).

In their systematic review Muntaenu & Burton (2011) established that athletes with AT displayed greater subtalar eversion ROM. This was supported by the prospective study carried out by Rabin et al (2014) on military recruits, with similar conclusions being drawn. Kaufman and colleagues (1999) demonstrated that increased ROM of subtalar inversion significantly increased the risk of AT, with those demonstrating more than 32.5° of inversion being 2.8 times more likely to display the condition. Limited ROM of inversion (<26°) was also shown to have a correlation with the condition in the same study (1.8 times more likely) (Kaufman et al, 1999).

There were variable results presented for goniometric subtalar joint ROM assessment. Scores of intrarater reliability were consistent for both inversion and eversion, with very good scores of 0.79 and 0.78 respectively (Elveru et al, 1988). Interrater reliability of the same sample presented rather poor results (0.32 & 0.17 respectively), which casts doubt upon the reliability of the measure when using different assessors. This result is consistent with other

literature in the area, which also finds poor reliability for the interrater measurement of subtalar range of motion (Smith-Oricchio & Harris, 1990).

Plantar flexion strength and AT

There has been an association between plantarflexion strength and AT, and this association may be either a consequence of the injury, or a risk factor for its development (Carcia et al, 2010). It has been shown through a cohort study that the plantarflexion strength measures of individuals suffering from AT are significantly lower than those of a healthy population (McCrory et al, 1999). However, in the AT group, strength was similar in both legs, suggesting that strength deficits may have been present before the clinical presentation of symptoms (McCrory et al, 1999).

A prospective study carried out by Mahieu et al (2006) found significant plantarflexion strength differences for almost all subjects affected by AT. Pre-training strength measurements on army recruits showed significantly less plantarflexion forces in the group that later developed symptoms of AT. They theorised that greater muscle strength in the triceps surae complex produces stronger tendons that are better able to resist strain and deal with the application of heavy loads (Mahieu et al, 2006). Through a battery of testing, Sibernagel et al (2006) were able to identify functional restrictions in the AT group. They showed impairments in strength through decreased performance of maximum concentric heel raise and maximum concentric-eccentric heel raise. They performed comparisons between the effected side and the non-effected side in the same individual and also demonstrated strength deficits between these, with consistently lower scores for the effected side (Sibernagel et al, 2006).

Thus from this we can conclude that decreased plantarflexion strength leads to an increased risk of the development of AT. We can also see that those with AT exhibit decreased - plantarflexion torque and strength on the effected side.

Age as a risk factor for AT

The association between age and AT has been mentioned frequently throughout the literature, with a belief that AT is most common in the fourth decade of life, and between the ages of thirty five and forty five (Wyndow et al, 2010). Kraemer and colleagues found a slight

correlation between age and AT with increased average age in the AT group of a matched pair analysis (Kraemer et al, 2012). A low quality prospective study carried out by Gajhede-Knudsen et al (2013) found similar results in a group of elite footballers competing in European club competition, with the average age of those suffering AT 27.2 years, compared to the symptoms free group with a mean age of 25.6 years. Contradicting these, Di Caprio et al (2010) in a prospective risk factor assessment argue that there was no association between AT and age in his cohort of recreational and competitive runners.

The proposed method of effect of age on AT has also been investigated, with authors presenting their view on different mechanisms. Decreased arterial blood flow, local hypoxia, decreased nutrition, impaired metabolism and the presence of free radicals were cited by one study as the negative effects of aging on the tendon's ability to carry load and recover from micro-trauma (Fredburg & Stengaard-Pederson, 2008). Carcia et al (2010) offers other alternative explanations of the effects of aging on the tendon's susceptibility to injury. It is believed in this review that the tendon undergoes morphological and biomechanical changes similar to other body tissues, associated with aging. It is proposed that aging decreases collagen diameter & density, as well as the glycosaminoglycan and water content of the tendon. The biomechanical effects of aging on the tendon may include decreased tensile strength, reduced linear stiffness and a compromised ultimate load (Carcia et al, 2010). Also associated with aging are a decreased rate of collagen synthesis, and an accumulation of obstructive macromolecules in the tendon matrix. All of these proposed mechanisms are more common in individuals over the age of 35 (Carcia et al, 2010).

Genetics & AT

As with many other conditions, there is believed to be a genetic/hereditary component in the development of AT. This is believed to be influenced by the effect of genes on the inflammatory pathway in the pathogenesis of AT. It has been suggested that inflammatory gene expression profiles of tenocytes are modulated in response to mechanical loading, and that cytokines trigger tenocyte apoptosis and pathological extracellular matrix degeneration (September et al, 2011). This becomes pathological when the rate of degeneration exceeds the healing response, and this is believed to be the case with this genetic predisposition. However, only two gene variations have been shown to have an association with AT, therefore the genetic influence on the development of AT is relatively rare (September et al,

2008). The association between genes and AT has been shown to be significant; however there has not been an association between genetics and TA rupture (Magra & Maffuli, 2007).

Fluoroquinolones & AT

The development of AT has been associated with a history of treatment with quinolone antibiotics. These are often used in the treatment of community acquired respiratory infections (Khaliq & Zhanel, 2003). Fluoroquinolone induced AT has been reported in approximately 6% of cases after taking the antibiotic (Carcia et al, 2010). The presentation of symptoms may occur within 2 hours of commencing treatment or up to 6 months after discontinuing antibiotic therapy, though up to 85% present within one month of commencement (Lewis & Cook, 2014). Symptoms usually present quickly with sudden onset of pain and morning stiffness in the tendon, and 95% of cases are reported in the TA (Lewis & Cook, 2014). Presentation of fluoroquinolone induced tendinopathy is more common in middle aged men, and can be influenced by comorbidities such as corticosteroid use (32.7% of AT patients), renal transplants (12.2%-15.6%), diabetes mellitus and rheumatic disease. It has also been shown increased prevalence in very physically active patients (Khaliq & Zhanel, 2003). Fluoroquinolone use should be discontinued immediately when symptoms of AT appear (Lewis & Cook, 2014). Treatment of fluoroquinolone induced AT differs slightly from traditional treatment with the first phase of treatment involving supporting the chemically damaged TA with a brace to allow for tendon recovery before later beginning a progressive loading program (Lewis & Cook, 2014).

Comorbidities associated with AT

The comorbidities associated with AT include conditions associated with reducing the blood flow to the region. These conditions can affect the blood supply or quality of blood to the region. This causes damage as a mechanism for tendinopathy is a failed healing response in the tendon. If the blood supply to the region is reduced, or the quality of nutrients received from the blood is affected, the tendon may not have the metabolites available to boost the healing process. Medical conditions that have been repeatedly associated with AT include obesity, hypertension, increased cholesterol and diabetes (Carcia et al, 2010). In all of these conditions the quality of blood supply is affected. Kraemer et al (2012) discussed similar comorbidities, with links between familial hypercholesterolemia, arterial hypertension, diabetes (in men under the age of 44) and AT. Interestingly, it was found that there were

significantly less people with cardiovascular disease and smokers in the group with AT than in the control group (Kraemer et al, 2012). The possible explanation offered for this was that 70% of people who suffer from AT regularly participate in physical activity, and physical activity is a method of preventing cardiovascular disease. The reduced number of smokers was explained by examining the lifestyle habits of people who regularly exercise, and hypothesising that the culture and lifestyle of those who regularly exercise may suggest that less of the physically active cohort smoke, as well as the association between running and smoking cessation (Oestergaard Neilsen et al, 2012).

Intrinsic Factors	**Extrinsic Factors**
Lateral Ankle Instability	Sand Running
Free Radical Damage (re-perfusion after ischemia)	Local or systemic steroid exposure
Hypoxia	Shod Running
Hyperthermia	Years of Activity
Impaired tenocyte apoptosis	Spiked/shock absorbing running shoes
Gender-rate of collagen remodelling in response to exercise and fascicle strength is lower in females.	Lateral heel strike with pronation
Increased Q-angle	Muscle Imbalances
Arterosclerosis	
History of previous overuse injury	

Table 2.1: Additional risk factors discussed in the literature (van Ginkel et al, 2009; Taunton et al, 2002; Carcia et al, 2010).

Ankle Dorsiflexion Range of Motion	
ICF category	Measurement of impairment of body function, mobility of a single joint
Description	Passive non-weight-bearing goniometric measure of dorsiflexion with the knee extended to 0° and flexed to 45°. Measures with the knee extended are intended to be descriptive of gastrocnemius flexibility, while those with the knee flexed are thought to reveal soleus flexibility.
Measurement method	The patient assumes a supine position on examination table with ankle and foot suspended over the end of the table for taking the goniometric measure of ankle dorsiflexion with subtalar joint in neutral. The stationary arm of the goniometer is aligned with fibular head. The axis of the goniometer placed just distal to the lateral malleolus and the moveable arm of the goniometer aligned parallel with the plantar aspect of the calcaneus and fifth metatarsal.
Subtalar Joint Range of Motion	
ICF category	Measurement of impairment of body function, mobility of a single joint.
Description	Passive non-weight-bearing goniometric measure of rearfoot inversion and eversion range of motion.
Measurement method	The stationary arm of the goniometer is held over a bisection of the distal one-third of the tibia and fibula. The axis is placed over the subtalar joint, while the moveable arm is placed over a bisection of the posterior aspect of the calcaneus.
Plantar Flexion Strength	
ICF category	Measurement of impairment of body function, power of isolated muscles and muscle groups.
Description	Assessment of plantar flexion force production at a controlled speed.
Measurement method	Plantar flexion torque (both average and peak torque) were assessed with an isokinetic dynamometer in 2 positions (sitting with knee flexed to 90° and supine with knee extended to 0°) at 30°/s and 180°/s, using both concentric and eccentric contractions.

Table 2.2: Assessment of risk factors (Carcia et al, 2010)

Training Characteristics and AT

Training characteristics and loading of the TA are believed to be a large determinant of the development of tendinopathy. Some of the cited training errors include increase in mileage, increase in intensity and returning from a 'lay-off' period of inactivity (Carcia et al, 2010). A systematic review of running related injuries investigated the effects of training volume, distance, time, frequency, intensity and pace on the development of running related injuries in novice, recreational and elite runners (Oestergaard Neilsen et al, 2012). In a review of the aetiology of running injuries, Hreljac (2003) concluded that overuse running related injuries were preventable as the causes of these injuries were training errors.

Volume/Distance:

In a systematic review of the above mentioned running related injury factors, volume in the form of distance covered per week was assessed as a risk factor for the development of injuries (Oestergaard Nielsen et al, 2012). 4 prospective cohort studies showed an increased relative risk of running related injury in runners who cover various distances per week. Running greater than 20 miles per week doubled the risk of injury in men, and triple this risk in women. It was shown that running greater than 40 miles per week significantly increased the risk of injury, with men and women up to 3 times more likely to develop running related injuries, including AT, when consistently (for greater than 3 months) covering these distances. Contradicting these findings, 2 other prospective studies mentioned in this systematic review found no significant association between miles run per week and the development of running related injuries.

The finding of several retrospective studies in this systematic review supported the assumption that greater miles per week increase the risk of running related injuries. One of these studies found that the proportion of women reporting injury was highest from those covering 40-49 miles per week, and the proportion of men reporting injury was highest among those running 30-39 miles per week. 2 further studies reported that the average weekly mileages of runners sustaining injuries were higher than uninjured runners [26.3km (injured) vs 22.0km (uninjured); 47.5mls (injured) vs 29.6mls (uninjured)]. It was also found that the relative risk of injury in runners who's longest weekly run exceeds 5 miles was higher than those who do not run this distance (men 2.49, women 1.78).

Duration

The duration of running has frequently been reported in the units of minutes per week, and the prevalence of injury is reported as 'running related injury per thousand hours of running' (RRI/1000hrs). The prevalence of injuries varies greatly for experienced runners and marathon runners to novice runners.

RRI prevalence per session running length was investigated by one study, concluding that 15 minutes, 30 minutes and 45 minutes duration of running for novice runners lead to differences in the number of injuries (22%, 24% & 54% respectively). Novice runners are reported to have a very high risk of injury, with up to 33 RRI/1000hrs being reported in novice runners who spend 52-59mins/wk running in this systematic review. Prevalence of RRI/1000hrs is much lower in experienced marathon runners with reported varying between 6.9-12.1 RRI/1000hrs in athletes running between 162 and 240 minutes per week. This association was acknowledged by the authors of this systematic review, with their reasoning that the risk of injury per mile of training decreases with greater total mileage for experienced and marathon runners. They believe that experienced runners may have a greater knowledge of their own injury thresholds compared to novice runners, and may make efforts not to exceed these thresholds, thereby leading to decreased injury rates per miles and minutes of running every week.

Intensity

Running speed/intensity may not have a decisive bearing on the development of AT. In this systematic review, 2 studies found that running intensity may have an influence, with findings that less than 8 minutes per mile may lead to more injuries than more than 8 minutes per mile. The other study found a similar association with greater or less than 15 minutes per mile. No other studies review showed a significant association between the intensity and the development of Achilles injuries.

Frequency

There was no significant association between the frequency of running and the prevalence of AT in this systematic review. The only association between RRIs and frequency were found for shin pain and anterior thigh muscular injuries.

Running Terrain

Running surfaces have been associated in the past with AT, with more compliant surfaces, uneven ground and changing terrains cited as factors that may predispose individuals to the condition (Pierre-Jerome et al, 2010; Cadez-Schmidt et al, 2014). However, much of the evidence for this is anecdotal and low quality studies. A prospective study found no influence of the running surface on the development of injury, but the authors did feel that it was more difficult to assess this due to limitations in quantifying the intensity of running and time spent on each of the different surfaces (Taunton et al, 2002).

Changes in training characteristics

It has been proposed that overuse injuries including AT occur more frequently during periods of changing and increasing training intensity, duration, or frequency (Schepsis et al, 2002; Carcia et al, 2010; Pierre-Jerome et al, 2010). However, though this association may be explained biomechanically through the increased loading strain on the tendon through increased mileage/intensity of training, there is a very limited background evidence to support the association. The link is drawn on assumptions and retrospective analysis rather than prospective research (Clement et al, 1981). One randomized control trial found no link between graded increase in training intensity (10% per week) and standard training increase (24% increase per week) over a thirteen week period. The incidence of running related injuries was 20.8% in the graded training group compared to 20.3% in the standard training program group (Buist et al, 2008). Though the large body of retrospective and case study evidence would suggest that rapid increase in training intensity is a direct risk factor for TA injuries, the findings presented in the present RCT encourages scepticism in the interpretation of these studies. It was suggested that more rapid increases in training intensity of up to 40% may be more effective in highlighting the issue, though there are ethical issues to be considered before undertaking such a project (Carcia et al, 2010).

The findings of the above RCT should not be interpreted as a justification for increasing training intensity by up to 24% weekly, rather that more high quality research with different populations may need to be carried out before recommendations can be made on the subject.

Clinical Implications

Due to age related physiological changes, the process associated with tendinopathy is most common in the 4th decade of life, with an increased risk of the condition in people over the age of 35. Those with abnormal sub-talar and dorsiflexion ranges are predisposed to the condition, with increases and decreases in the available range of both movements being strongly associated with the condition. Higher levels of fat have been associated with the condition in both men and women, with a central distribution of fat being seen in men, and women presenting with more peripherally distributed fat. There is a tentative link between genetic predisposition and AT development, as well as a correlation between certain comorbidities such as obesity, diabetes and hypercholesterolemia, which it is believed affects the peripheral arterial blood supply. Though little significant research evidence exists for its affect, the link between training characteristics and symptom development in AT in an athletic population cannot be understated. Drawing from clinician experience, the association between an increased intensity of training or sudden change in training characteristics was stressed to us as a crucial factor in the development of this condition, and risk factor assessment should certainly contain an aspect of training characteristic profiling.

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Diagnosis of AT

Though several attempts have been made, no classification system has been commonly accepted for use in AT (Puffer & Zachazewski, 1988). Thus, the diagnosis of AT relies on a thorough subjective examination in combination with a physical assessment including palpation and functional movements. A study by Hutchinson et al (2013) featured an assessment of 10 tests for AT (Figure 2.2). These included; subjective self-reported pain, subjective reporting of morning stiffness, tendon thickening, crepitus, palpation, The Royal London test, the arc sign, the stretch of passive dorsiflexion test, single heel raise and hop test. These included 2 subjective measures, 5 palpation tests and 3 tendon loading tests. With ultrasound used as the diagnostic standard in this study, Hutchinson and colleagues (2013) assessed the clinical utility and reproducibility of these test in the diagnosis of AT. The review of clinical tests found that only 2 of the tests were sufficiently valid and reliable for use in a clinical setting, and these were the location of self-reported pain and pain on palpation (Hutchinson et al, 2013). Another study found good overall level of sensitivity for the Royal London Hospital test and the palpation of the painful arc sign (Figure 2.3) (Maffuli et al, 2003). These authors concluded that in a painful tendon where the area of tenderness presents with swelling that moves with the tendon, and whose tenderness decreases in dorsiflexion, a diagnosis of tendinopathy may be appropriate (Maffuli et al, 2003).

Definition of each of the ten clinical tests.

	Test	Definition of test	Positive result for a tendinopathy
Subjective evaluation			
S1	Subjective self reported pain[a]	Tested by the clinician asking the patient "Can you point out where you get pain"	Pain located 2–6 cm above the insertion of the Achilles tendon to the calcaneum pointed out by patient
S2	Subjective reporting of morning stiffness[a]	The clinician asks the patient "How are your symptoms first thing in the morning when you get out of bed?"	Pain is usually worse for the first few steps in the morning
Palpation tests			
P1	Tendon thickening[a]	The clinician palpates the Achilles tendon in a distal to proximal direction, between 2 and 6 cm above the insertion into the calcaneum gently squeezing the tendon between the index finger and the thumb feeling for localised thickening of the tendon	A subjective opinion of tendon thickening
P2	Crepitus[a]	The clinician palpates the Achilles tendon between 2 and 6 cm above the insertion into the calcaneum, gently squeezing the tendon between the index finger and the thumb feeling for crepitation ("wet leather" sign) in the Achilles tendon with passive ankle motion	A subjective opinion of crepitation by the clinician
P3	Palpation [6]	Performed by the clinician gently palpating the whole length of the tendon in a proximal to distal direction, gently squeezing the tendon between the thumb and the index finger	The patients subjective reporting of pain
P4	The Royal London test [6,10,11]	Performed by the clinician palpating the tendon for any local tenderness with the ankle either in neutral position or in slight plantar flexion. The ankle was then actively dorsiflexed and plantarflexed. With the ankle in maximum dorsiflexion, the portion of the tendon found to be tender was palpated again	Tenderness on palpation decreases significantly or disappears completely with maximum dorsiflexion
P5	The Arc sign [6]	Performed by the clinician identifying the intratendinous swelling in the tendon and asking the patient to actively dorsi flex and plantar flex the ankle joint observing the movement of the swelling between the malleoli	The intratendinous swelling moves relative to the malleoli with the tendon during the ankle movement
Tendon loading tests			
L1	The stretch of passive dorsi flexion with knee joint in flexion[a]	This was performed by the patient placing the affected leg forwards (the toes must face forwards) and lean onto it until they can feel a stretch in the tendon. The patient's heel must not lift off the ground	Patient reporting pain at the extreme range of the movement
L2	Single leg heel raise[a]	This test was performed by the patient rising up onto tip toes and lowering back to the floor, on the affected leg	Patient complaining of pain on either the up or downward movement
L3	Hop test[a]	This test was performed by the patient hopping forward over a line marked on the floor	Patient complaining of pain in the mid Achilles tendon during the exercise

Fig 2.2: Overview of tests for AT (Hutchinson et al, 2013)

Achilles Tendon Palpation Test	
ICF category	Measurement of impairment of body function, pain in body part
Description	The patient is positioned prone on an examination table with the ankles hanging just over the edge of the table. Gentle palpation of the entire Achilles tendon is performed by squeezing the tendon between thumb and index fingers.
Measurement method	The patient is asked to indicate whether pain was present or absent with palpation.
Arc Sign	
ICF category	Measurement of impairment of body function, pain in body part
Description	The patient is positioned prone on an examination table with the ankles hanging relaxed just over the edge of the table. The patient is asked to actively plantar flex and dorsiflex the ankles.
Measurement method	Examiners are instructed to determine if the area of maximal localized swelling moves proximal and distal with the tendon during active range of motion or remains static. If the identified area moves proximal and distal, the result is classified as "tendinopathy present." Conversely, if the area remains static, the result is classified as "tendinopathy absent."
Royal London Test	
ICF category	Measurement of impairment of body function, pain in body part
Description	The patient is positioned prone on an examination table, with the ankles hanging relaxed just over the edge of the table. In this position, the examiner identifies the portion of the Achilles tendon which is maximally tender to palpation. The patient is then asked to actively dorsiflex the ankle. The examiner once again palpates the part of the tendon that was identified as maximally tender, however, this time in maximal dorsiflexion. Patients with Achilles tendinopathy often report a substantial decrease or absence of pain when the palpation technique is repeated in dorsiflexion.
Measurement method	With the ankle in maximal active dorsiflexion, the examiner classifies the identified area with palpation as "tenderness present" or "tenderness absent."

Fig 2.3: Overview of most clinically relevant Achilles tests (Carcia et al, 2010).

Other subjective symptoms that have been reported as clinical determinants of a diagnosis of tendinopathy include intermittent activity related pain, stiffness upon immobility and increased pain upon recurrence of activity, and stiffness/pain at the commencement of exercise that decreases during exercise only to return shortly after exercise (Carcia et al, 2010). These symptoms are reported across the literature, though there has been very little high quality research published to support their association with AT.

A further systematic review of the diagnosis and assessment of AT found interesting results (Reiman et al, 2014). This review found that some measures that have been used for assessment may be more applicable when used for screening and vice versa. The report found strong specificity for the use of crepitus, the arc sign, the Royal London Hospital (RHL) test, single-legged heel raise and tendon thickening in the assessment of AT. This review also found excellent intra-rater reliability for the RHL test, self-report of pain and morning stiffness, palpation and the arc sign (Reiman et al, 2014). Similar tests showed sufficient to excellent levels of inter-rater reliability, with the exception of the RHL test, which displayed

moderate inter-rater reliability. The findings of this systematic review indicated that self-report of pain and morning stiffness are more applicable in the screening process than for diagnosis. Conversely, it was displayed that TA thickening, crepitus and all tendon loading measures are better when used in the diagnostic process than for screening (Reiman et al, 2014).

In order to add to the body of literature and assessment of AT, Sibernagel and colleagues developed a test battery to evaluate if AT caused functional deficits on the injured side compared to the non/less injured side in patients (Sibernagel et al, 2006). The test battery consist of 6 tests, three jump tests (counter movement jumps, drop counter movement jump & hopping), two strength tests (concentric toe-raises & concentric-eccentric toe-raises) as well as toe-raises for endurance. The study found that AT not only presented with pain and AT related symptoms, but also with impairments in lower limb function. The battery was reliable and was able to differentiate between the "most" symptomatic and "least" symptomatic leg in patients with bilateral AT. The tests in the battery were shown to have an excellent level of interrater reliability (ICC 0.76-0.94), with the concentric toe-raise test have fair interrater reliability (0.73). Very good results were also shown for the sensitivity of the tests, with individual sensitivities ranging from 33-48%, but the whole test battery having a sensitivity of 88% (Figure 2.4) (Sibernagel et al, 2006).

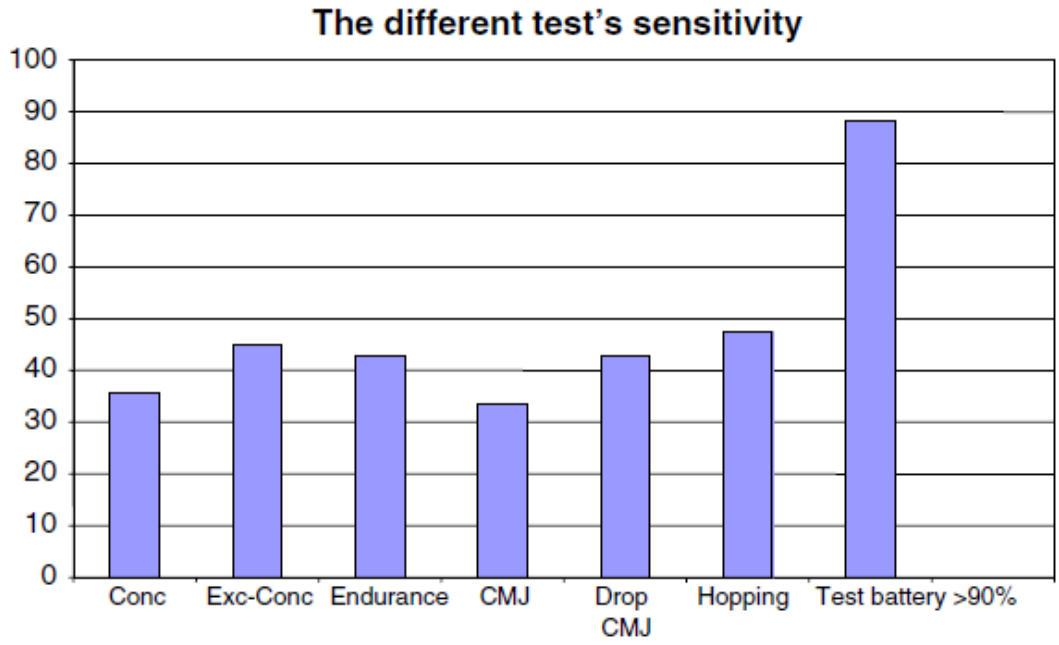

Fig 2.4: Sensitivity of Sibernagel functional assessment tests (Sibernagel et al, 2006)

Outcome Measures

The Victorian Institute of Sport Assessment self-administered Achilles questionnaire (VISA-A) is a robust tool used at the initial assessment and following treatment of AT (Carcia et al, 2010). It has been shown to be quick to administer (less than 5 minutes), and responsive to clinical changes, thereby highlighting its potential use as a means of detecting progress (Robinson et al, 2001). The VISA-A measures functional aspects such as the patient's perceived ability to walk, descend stairs, perform uni-lateral heel raises, single-legged hop and participate in their specific recreational activities (Carcia et al, 2010). Imaging does not provide a yardstick for tendon disorders, therefore diagnosis and progress need to be tracked by clinicians using assessment and screening tools (Robinson et al, 2001).

The VISA-A is an index of the severity of a clinically diagnosed Achilles pathology, and should not be used for diagnosis, as other pathologies in the region may affect the score (Robinson et al, 2001). The VISA-A is an assessment tool that scores from 0-100 with higher scores reflective of a healthy or less painful tendon. Scores from 24-100 have been reported for TA patients and healthy control participants, with the belief that a score of less than 24 may indicate severe pathology such as a tendon rupture, with a huge effect on the ability of the person to carry out their activities of daily living (Vestergaard Iverson et al, 2012). The validity of the score has been shown to be high, as control participants without tendon pathology consistently score greater than 95 out of 100 on the questionnaire. This means that the questionnaire may be useful in tracking the progress of a patient in a clinical setting, as there is a very wide interval for improvement of symptoms (24-96). This systematic review proposed the figure of 90 as the threshold for symptom recovery from AT, and the minimal clinically important change score has been proposed between 12 and 20 by some authors (Vestergaard Iverson et al, 2012). It has been difficult to quantify these scores in the literature though, as there have not been enough high powered longitudinal studies carried out on the subject (Vestergaard Iverson et al, 2012).

In an athlete specific context, the VISA-A can be very useful as a determinant of symptom recovery when returning to sport. It has been proposed that a VISA-A score of greater than 90 be attained by an athlete before returning fully to sport (Vestergaard Iverson et al, 2012). This was chosen as full symptomatic recovery is proposed at a score of 90, and this would

need to be attained by an athlete in order to return to sport without any Achilles related functional limitation.

The VISA-A is the only Achilles specific outcome measure that can be found. The Foot and Ankle ability measure (FAAM) and the American Academy of Orthopaedic Surgeons developed Lower Limb Outcomes Assessment Instruments Foot and Ankle Module are lower limb assessments, but neither have been shown to be valid or reliable for use in AT (Martin & Irrgang, 2007). Therefore, the use of the VISA-A is recommended for patients with AT due to its validity, reliability and ease of use.

Clinical Recommendations

From a review of the literature, it is possible to identify the tests that are most appropriate for the assessment of AT. These tests can be sub-divided into categories for diagnosis and screening based on their properties. The most pertinent tests for the diagnosis of AT are palpation for TA thickening, crepitus, and all tendon loading tests, as well as pain on palpation and the Royal London Hospital test. The VISA-A and the self-report of pain and morning stiffness measures should be carried out after diagnostic tests have indicated the presence of AT, and these can be used to monitor the progress of symptoms and as an indication of the functional limitations presented due to AT pain.

		Diagnosis	Progress/Screening
Self-reported	Pain		✓
	morning stiffness		✓
Palpation	Tendon thickening	✓	
	Crepitus	✓	
	Pain	✓	
	The Royal London Hospital test	✓	
	The arc sign	✓	
Loading	Soleus stretch	✓	
	Single leg heel raise	✓	
	Hop test	✓	
Outcome Measure	VISA-A		✓

Table 2.3: Screening versus diagnosis. Clinical tests for AT

Imaging

Ultrasound & AT

AT is a degenerative condition that may lead to rupture asymptomatically (Joseph et al, 2012). Degenerative changes associated with AT on ultrasound include hypoechoic regions, increased tendon diameter, fibre disorganisation and neovascularisation (Joseph et al, 2012). In the literature, there have been tentative links between the presence of ultrasonographic abnormalities listed above and the symptomatic diagnosis of AT, with authors arguing that the association should not lead to diagnosis, and diagnosis only be made through clinical examination of pain, swelling and inability to perform strenuous activity (Boesen et al, 2011). Links have been drawn between the occurrence of neovascularisation and pain in AT, with biopsies revealing the ingrowth of new nerve endings and blood vessels in degenerative tissues (Boesen et al, 2011). The normally poorly vascularised tendon, through the process of degeneration, becomes hypervascularised. This occurs along with degradation of the extracellular matrix, which is required for the sprouting and invasion of new blood vessels (Magnan et al, 2014).

Recently there has been a transition towards the belief that increased vascularisation may not be degenerative in nature, and may simply be a response to loading (Comin et al, 2012). Several studies have found that there is no correlation between sonographic abnormalities and the development of symptoms (Comin et al, 2012; Boesen et al, 2011; de Vos et al, 2012). Cassel et al (2014) stated that whether the presence of intratendinous (IT) changes and vascularisation can be considered pathological or a physiological adaptation to loading has to be interpreted with clinical symptoms in mind. A systematic review found no association between decreased pain, increased function & treatment satisfaction (clinical improvement) and neovascularisation, structural changes and tendon diameter (US degeneration) (Drew et al, 2014). The reviewers suggested that improvements in clinical outcomes were not directly associated with IT changes on ultrasound (Drew et al, 2014).

A prospective observational study found that a significant increase in VISA-A scores, depicting decreased pain and improved function in AT, had no correlation with echogenicity on US imaging (de Vos et al, 2012). At 6 month follow up, there was no increase in organised tendon structure after eccentric exercise, indicating that tendon structure is not related to symptom severity and cannot be used as a predictor of clinical outcome (de Vos et

al, 2012). The outcome of this prospective study suggested that even though there was a statistically significant improvement in symptoms and function within 6 months, clinically meaningful change in US tendon structures may take more time (de Vos et al, 2012).

Boesen et al (2011) offer an explanation for the presence of vascular changes in athletes load bearing tendons, with a belief that vascularisation is a natural response to loading. In the examination of 14 badminton players at a tournament, the majority of players exhibited increased IT flow at baseline, which increased stepwise in all tendons in relation to physical activity when loading was repeated. The number of tendons affected, and the amount of IT flow increased after each match (Boesen et al, 2011). This supports the association between increased IT flow and non-pathological loading (Boesen et al, 2011).

It is possible that the severity of IT flow, as detected by US imaging, can be used for the identification of increased risk of development of AT, rather than the diagnosis of the condition (Boesen et al, 2011). It is believed that increased IT flow is mainly a physiological vascular response to increased load, rather than a diagnostic criteria for AT (Cassel et al, 2014; de Vos et al, 2012; Comin et al, 2012).

MRI & AT

MRI has been shown to have a good diagnostic value in AT, with areas of altered collagen fibre structure and increased interfibrillar ground substance showing increased signal intensity of MRI (Alfredson & Cook, 2007). However, AT pathology is also found in tendons which appear normal on MRI (Alfredson & Cook, 2007). Due to its three dimensional picturing and its ability to accurately display soft tissues, MRI has been shown to have high sensitivity and specificity in the diagnosis of AT (Carcia et al, 2010). Similarly high levels were demonstrated using US imaging. Due to the expense of MRI imaging it is not often used in the diagnosis of AT, and clinical assessments are most often used in the diagnostic process (Carcia et al, 2010).

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Differential Diagnoses

TA Rupture

Acute TA ruptures occur in between 7 & 13 per 100000 every year, with a recent increase in the prevalence of the injury due to increased participation in recreational sports. The incidence is highest in males between the ages of 30 & 39 years, with up to 75% of injuries occurring during sporting activity (Nandra et al, 2011; Maffuli et al, 2014). Ruptures usually occur due to biomechanical influences and degenerative changes in the TA in the form of AT, which may be clinical or subclinical, depending on the presence of symptoms preceding rupture (Alfredson et al, 2009). If Achilles rupture is suspected, there are tests that may be carried out to assess this. The American Academy of Orthopaedic Surgeons produced guidelines for the diagnosis and treatment of TA rupture, based on a systematic review of the literature. The guidelines found that the Thomson (squeeze) test, decreased ankle plantarflexion strength, presence of a palpable gap and increased passive ankle dorsiflexion range of motion were the most accurate clinical tests for the diagnosis of TA rupture (AAOS, 2009). Other clinical tests to out-rule a rupture of the TA are the Matle's test, which involves placing the patient in prone and flexing both knees to 90° and placing the ankle joint into neutral. If the effected side displays more dorsiflexion at perceived neutral, the result is positive for TA rupture (Reiman et al, 2014). Matle's test displayed high sensitivity and specificity for the detection of an TA rupture. This systematic review found that the calf squeeze test (Thomson's test) and palpation for TA gap were the most sensitive and specific tests for TA rupture, followed by Matle's test (Reiman et al, 2014).

Management of the TA rupture is controversial. In the sedentary population (25%) of ruptures conservative management may be favoured, though this may lead to weakened muscle, and up to 13% re-rupture (Maffuli et al, 2014). Surgical management is invasive and risks of complications are up to 20 times that of conservative management (Maffuli et al, 2014). Surgical management should be approached with caution in the following groups; diabetics, those with peripheral neuropathies or peripheral vascular disease, those above the age of 65, those with immunocompromised states, sedentary lifestyles or obese (AAOS, 2009). The most common complications are infection and re-rupture.

Retrocalcaneal bursitis

The function of the retrocalcaneal bursa is to allow transmission of force from the TA to the calcaneus without creating excessive tissue tension. It does this by allowing the separation of the tendon from the bone during plantarflexion, as the angle of the tendon's path along the bone increases (Canoso et al, 1988). It is often difficult to differentiate the source of pain in this region between the bursa and the TA. Pain is usually caused by strong or repetitive triceps surae contractions, often involving extremes of range, such as hopping, jumping, running and uphill walking (Frey et al, 1992). Diagnosis may be performed following thorough subjective and objective examinations to distinguish it from AT, and may be confirmed using ultrasonography, MRI or CT scans (Frey et al, 1992). Physiotherapy intervention may require biomechanical correction, anti-inflammatory advice, stretching and the use of heel wedges, however failing conservative management, surgical options are available. Surgical management involves resection of the calcaneus, though on 6 year follow up, only 50% success rate was demonstrated (Angermann et al, 1990).

Posterior impingement syndrome

Posterior impingement syndrome constitutes a group of pathologies that result from repeated plantar flexion of the foot, leading to compression and trapping of soft tissues between the posterior aspect of the distal tibia and the superior surface of the calcaneus (Sainani et al, 2011). Posterior ankle pain sometimes occurs post calcaneal fractures, and usually takes the form of posterior impingement syndrome or Haglund's deformity (Lui, 2008). This condition most often effects athletes and dancers, and seldom presents in an inactive population, with ballet dancers, horizontal jump athletes and soccer players predominantly implicated (Ribbans et al, 2015; Rogers et al, 2010). It is most often caused by os trigonium, posterior

tenosynovitis, or Shephard's fracture of the posterolateral talus (Ribbans et al, 2015). This may present as a dull ache in the heel area or a sharp pain on activities in plantar flexion, such as 'en pointe' work in dancing, or hopping/jumping (Vali et al, 2014). Subjective and objective assessments are required for diagnosis of posterior impingement syndrome. Objective assessment should include palpating the talus and calcaneus of point tenderness. Radiographic imaging may be necessary to detect the presence of body or soft tissue abnormalities in the region that may be causing the pain (Vali et al, 2014).

Dislocation of peroneal tendons

Peroneal tendon injuries are one of the most common complications of calcaneal fractures (Lui, 2008). They are often mistaken as lateral ankle ligament injuries, and usually occur in sports involving rapid direction changes such as hockey and football. Dislocation and subluxation of the peroneal tendons occur when the peroneal muscles are forcefully contracted while the foot is in a dorsiflexed and inverted position (Roth et al, 2009). Clinical tests include palpation of the retrofibular region for pain or tenderness & active range of motion testing to produce a click in forced end of range plantarflexion and eversion (Roth et al, 2009). Active dorsiflexion and eversion of the foot from a position of inversion and plantarflexion will determine if active contraction of the peroneal muscles reproduces the pain (Roth et al, 2009).

Stress fracture

Stress fractures are among the top three overuse injuries reported in athletic populations, with particularly high prevalence among those who participate in soccer, running, gymnastics and dance (Siobhani et al, 2013). Repetitive activity and increased training load appear to be risk factors for the development of stress fractures (Chen et al, 2006). Stress fractures can appear in the navicular bone (Mann, 2009), metatarsal bones (Watson, 2013), the cuboid (Beaman et al, 1993) or in the calcaneus (Aldridge, 2004) in athletes and diagnosis is usually through radiographic imaging.

Sural nerve irritation or neuroma

The sural nerve descends between the two heads of gastrocnemius and gives cutaneous branches to the posterior and lateral aspects of the lower third of the leg. It also supplies the lateral calcaneus, heel, ankle and along the lateral border of the foot to the 5th terminal

metacarpophalangeal joint (Pringle et al, 1974). The sural nerve can become irritated in the posterior compartment of the lower limb, along its course through the posterolateral ankle (Pringle et al, 1974). It can also become injured due to direct trauma to the posterior leg, usually in the area of its course through the heads of gastrocnemius. It may present with numbness or pain to the lateral ankle and heel (Babwah, 2012).

Sever's disease

Also known as calcaneal apophysitis, Sever's disease is posterior heel pain that presents mostly in physically active children aged between 8 and 14 years old. This pain accounts for up to 15% of musculoskeletal injuries in children this age (Howard, 2014). Sever's disease is believed to be caused by overuse and microtrauma, leading to repeated microavulsions at the junction of the bone and cartilage, with no time for the bone to heal (Howard, 2014). Diagnosis is usually through comprehensive subjective history, and a positive squeeze test of the medial and lateral heel, reproducing pain. The condition is usually self-limiting, with limited evidence to support the use of orthoses and heel raises (James et al, 2013).

Os trigonium

The os trigonium is a small bone at the posterior aspect of the talus that fails to fuse with the talus in approximately 3-15% of people (Jones et al, 1999). The os trigonium injury is characterised by pain and swelling in the posterolateral region of the ankle, and some of the mechanisms of injury described include avulsion by the posterior talofibular ligament, direct trauma to the posterior aspect of the ankle, repeated trauma form repetitive hyperplantarflexion, or an acute episode of hyperplantarflexion (Jones et al, 1999). Diagnosis can be difficult as it often presents similar to other conditions of the posterior foot, including flexor hallicus injuries and is often concurrent with posterior impingement syndrome. Therefore, imaging is most often used in the diagnosis of this condition (Jones et al, 1999).

Tarsal tunnel syndrome

Tarsal tunnel syndrome is an entrapment neuropathy involving compression of the posterior tibial nerve beneath the flexor retinaculum in the posterior compartment of the ankle, caused by either trauma or repetitive injury (Plyler et al, 2012). Other causes identified include hypertrophic retinaculum, osteophytes or lipoma (Ahmed et al, 2012). The predominant symptom is pain over the tarsal tunnel behind the medial malleolus with radiation to the

longitudinal arch and plantar aspect of the foot, including the heel, with secondary symptoms of tightness, burning, tingling and numbness over the same distribution (Ahmed et al, 2012). Symptoms are exacerbated by walking, running or standing. Tinel's sign and pain provocation tests such as combined dorsiflexion and eversion can be used for diagnosis. Conservative management may include anti-inflammatory medication, activity modification and mobilisations. Limited evidence has emerged supporting the use of night splints and orthotic insoles, though this is inconclusive (Ahmed et al, 2012).

Tenosynovitis

Achilles, peroneal and tibialis anterior tenosynovitis involve inflammation of the tendons outer sheath. In the TA this is often called the paratenonitis. Paratenonitis presents in approximately 15% of Achilles tendinopathies (Pierre-Jerome et al, 2010). It can occur acutely in runners, and is believed to be caused by increased extrinsic pressure, causing friction between the TA and its overlying sheath (Pierre-Jerome et al, 2010). Very little evidence exists supporting the conservative management of paratenonitis, and surgical management has been described with some favourable results (Kvist & Kvist, 1980).

Tibialis posterior tendon dysfunction/tendinopathy

Rupture of the tibialis posterior tendon is associated with acquired adult flatfoot, as this is a muscle which supports the arch. Rupture of the tendon can occur with ongoing dysfunction of the tendon preceding the injury (George & Davis, 2008). Injury may occur in endurance athletes, and occurs most often in runners and triathletes (Howitt et al, 2009). Tendon dysfunction presents with swelling and pain posterior to the medial malleolus and pain weight bearing (Howitt et al, 2009). Stage 1 dysfunction presents with mild swelling, medial ankle pain and normal but possibly painful heel raise. Stage 2 dysfunction presents with progressive flattening of the medial arch of the foot, with secondary abduction of the mid-foot and an extreme difficulty maintaining heel raise. Stage 3 presents with similar to stage 2, however there may be a fixed hindfoot deformity (Kulig et al, 2009). A randomised control trial of non-surgical interventions for tibialis posterior tendon dysfunction found that the condition responded well to the provision of orthoses and stretching and found much lower pain scores and increased perceived function score in groups that participated in progressive resisted concentric and eccentric exercises (Kulig et al, 2009).

Arthritic condition

Arthritis at the ankle is most often of post-traumatic aetiology, and those who present with the condition usually have a history of fracture (Thomas & Daniels, 2003). Ankle conditions can also commonly present in patients with Rheumatoid arthritis (RA), with up to 90% of people suffering from RA affected by foot and ankle problems (Waller et al, 2012). Ankle arthritis presents with pain and swelling across the joint line, as well as limited painful range of motion. A detailed subjective assessment is important in distinguishing this condition, and radiographic imaging can be used to confirm the diagnosis (Thomas & Daniels, 2003).

Plantar fasciitis

Plantar fasciitis presents in up to 10% of the population and is particularly prevalent in endurance athletes and dancers (Anderson & Stanek, 2013). The condition effects the aponeurosis extending from the medial tuberosity of the calcaneus to the heads of the metatarsal bones, and presents with sharp heel pain that is worst in the morning when taking the first steps of the day, and can become irritated by standing or walking for a prolonged period of time (Serbest et al, 2013). Pain is usually localised to the medial calcaneal tubercle, and can be reproduced by stretching the plantar fascia or by palpation of the heel and proximal aspects of the fascia (Serbest et al, 2013). Treatment of plantar fasciitis is multi-factorial with limited evidence to support gastrocnemius & soleus stretching (Garrett & Nerbert, 2013; Almubarak, 2012), foot orthoses (Anderson & Stanek, 2013), dorsiflexion night splints (Lee et al, 2012), extracorporeal shockwave therapy or steroid injection (Serbest et al, 2013) in isolation.

Haglund's Deformity

Haglund's Deformity is the enlargement of the posterosuperior prominence of the calcaneus, which is sometimes associated with insertional AT (Kang et al, 2012). It is believed to be caused by repeated pressure between the calcaneus and the TA, leading to inflammation and swelling at the superior aspect of the posterior calcaneus, and can often present with an inflamed retrocalcaneal bursa (Bulstra et al, 2014). Clinically, it may be possible to observe a lateral calcaneal protuberance, with regional inflammatory changes. Radiographic imaging can be used to distinguish Haglund's deformity from isolated retrocalcaneal bursitis, superficial TA bursitis or insertional tendinopathy (Sofka et al, 2006). Conservative

management for this condition can include re-evaluation of footwear, heel inserts and oral anti-inflammatories (Sofka et al, 2006). Surgical options may include retrocalcaneal decompression, calcaneal ostectomy or osteotomy. These have varying results and re-occurrence of symptoms happens frequently if inadequate bone is resected. Other surgical complications may include excessive scar tissue formation, nerve entrapment, weakening or rupture of the TA, or non-union of the calcaneal osteotomy (Brunner et al, 2005).

Assessory soleus muscle injury

The soleus muscle is most often injured when the knee is in flexion, unlike the gastrocnemius muscle which is injured in knee extension. As it is a deep, multi-pennate muscle with good vascularity, ultrasound imaging for the diagnosis of soleus injury is unreliable (Balius et al, 2014). MRI imaging may be used by top level athletes to confirm diagnosis, but this injury usually resolves very well, and can be assessed clinically with pain in dorsiflexion with knee flexed. Therefore MRI is seldom required and conservative management is sufficient (Balius et al, 2014).

Treatment Management of AT

Conservative treatment is the preferred choice for treating ATP, especially for a period of 3-6 months (Alfredon & Cook 2007; Longo et al 2009; Ro we et al 2012).

Why are eccentric exercises proposed to be effective?

Eccentric exercise can be defined as an overall lengthening of a muscle as it develops tension and contracts to control motion (Camargo *et al* 2014). This type of training differs from a normal conventional training regimen as the tension generated in muscle fibres when lengthening is proposed to be considerably greater then when muscle fibres are shortening (Camargo *et al* 2014). These large forces produced eccentrically are hypothesised to induce remodelling response when applied chronically and progressively (LaStayo *et al* 2003).

However, controversy around this remains as studies have shown that peak tendon forces in eccentric loading are of an equal magnitude to concentric loading, thereby suggesting that the tendon force magnitude alone is not responsible for the therapeutic effects seen with eccentric loading (Rees *et al* 2008). Furthermore, this study by Rees *et al* (2008) found that tendons are subjected to continual loading and unloading in a sinusoidal-type pattern during eccentric exercises with high-frequency oscillations in tendon force. This was noted to be largely absent with concentric exercises. They suggest that this pattern of loading and unloading with its force fluctuations may provide an important stimulus for tendon remodelling and may be in fact responsible for the therapeutic benefit associated with eccentric loading (Rees *et al* 2008). They compare this phenomenon to increases in bone density when bone responds to high-frequency loading.

Another possible mechanism is the effect of eccentric exercises in collagen synthesis. Langberg *et al* (2007) carried out a small study investigating the effect of eccentric exercises on collagen synthesis. All 12 subjects (6 with AT, and 6 healthy ATs) were elite male soccer players, and all 12 performed 12 weeks of heavy resistance eccentric training. Those with AT were found to have increased Type I collagen synthesis after the training while the healthy subjects were unchanged. Furthermore, there was no corresponding decrease in collagen degradation markers, while there was a corresponding decrease in pain levels and return to sport.

Another mechanism which has been proposed is 'pain habituation' due to completion of several weeks of pain-provoking eccentric exercises. In essence, the person decreases or ceases to respond to pain or pain-related stimuli due to the eccentric exercises (Murtaugh and Ihm 2013). Also, neuromuscular benefits through central adaptation of both agonist and antagonist muscles has been hypothesised (Rees *et al* 2008). It is clear several possible mechanisms exist, however, exact mechanisms as to why eccentric training appears to optimize the rehabilitation of a painful tendon is of yet not fully known.

Eccentric Exercise:

The use of eccentric exercise in AT has received considerable attention in the literature (Altman et al 2012). In 1984, Curwin and Stanish pioneered what they deemed 'eccentric training' for tendon injuries. Their programme consisted of 3 sets of 10 repetitions of eccentric loading which was progressed weekly according to the pain levels experienced between repetitions 20 and 30. It was reported that out of the 75 participants, 95% of them experienced symptom resolution within 6-8 weeks. Alfredson et al (1998) then made three essential modifications to this protocol. Firstly, they considered pain as part of the normal recovery process and advised patients to continue with the exercises even as pain worsened. If the patient could complete the exercises pain free, additional load was added until pain was provoked. The second moderation involved the exercise itself with no concentric component included in the heel drop which meant the unaffected contralateral limb returns the ankle to the starting position (Altman et al 2012). Additionally, two types of heel drops were included in the program, one with the knee flexed and one with it extended. The final alteration was the number of exercises carried out, Alfredson and colleagues (1998) advised on 180 heel drops a day (3x15 sets twice a day, once with knee flexed and once with knee extended). The control group were awaiting surgery. This 12 week programme resulted in all 15 eccentric exercise participants returning to full activity levels with a significant decrease in pain during activity and a significant improvement in calf muscle strength of the affected side for the intervention group. Consequently, the majority of studies investigating eccentric exercise for AT base it on the Alfredson model.

Sussmilch-Leitch et al (2012) completed a systematic review and meta-analysis of the literature for AT in line with the Preferred Reporting of Systematic Reviews and Meta-Analyses (PRISMA) statement. The review included randomized control trials evaluating the effect of at least one non-surgical, non-pharmalogical intervention on pain and/or altered function in AT. No restrictions were placed on the duration of participant symptoms or the length of treatment. Most studies used participants with chronic tendinopathy (3 months +) with the minimum intervention being 6 weeks ranging up to 12 months. A modified version of the PEDro scale was used to assess the methodological quality of the included articles which was shown to have good inter-rater reliability for systematic reviews (Bisset et al 2005). 19 studies were included for review after quality assessment scores and risk of bias were analysed. Criteria which were not met by the least number of studies were blinding of therapists and reporting of reliability and validity of outcome measures. The most commonly used outcome measures were the Visual Analogue Scale (79%) and the Victorian Institute of Sport Assessment –Achilles (VISA-A) questionnaire (37%).

Eccentric exercise was the most frequently investigated intervention (17/19 studies) in this systematic review. Effect sizes from a number of RCTs showed eccentric exercise to be effective (Sussmilch-Leitch et al 2012). Nine studies investigated eccentric exercise as a primary interest while the remaining eight studies used eccentric exercise as a control or adjunct intervention. One study (Rompe et al 2008) included participants with insertional tendinopathy and so the results are not applicable to this discussion. Modified PEDro scores ranged from 4/14 to 12/14. Rompe et al (2007) was the only study to compare eccentric exercise to a wait-and-see protocol with the results largely favouring a 12-week eccentric exercise programme with significant improvements in the VISA-A post-intervention (11/14 on modified PEDro scale). Knobloch et al (2007) similarly found significant improvements in pain (VAS) and paratendinous capillary blood flow for eccentric exercise when compared with cryotherapy alone for 12 weeks (7/14).

Silbernagel et al (2001) (6/14) compared two interventions for patients with chronic AT. The control group completed similar exercises to the intervention group including calf stretching and bilateral eccentric/concentric heel raises progressed onto unilateral heel raises except for the additional eccentric overload completed by the intervention group which involved eccentric heel drops over step. There was an overall better result for the intervention group

who had significant improvements in pain, were satisfied with their activity level and considered themselves 'fully recvoered'. Similarly, Herrington and McCulloch (2007) (8/14) assessed the benefit of adding eccentric exercise to a multimodal approach of deep friction massage, ultrasound and calf stretching. VISA-A scores showed that the eccentric exercise group experienced a significantly greater improvement in pain and function after 12 weeks compared to the control group. Mafi (2001) and Niesen-Vertommen (1992) compared eccentric exercise to concentric exercise. Mafi (2001) did not complete a between group comparison of pain however significant improvements in VAS scores were seen for both forms of loading, but this was only in the participants who were satisfied with treatment (8/14). Niesen-Vertommen (1992) found participants in the eccentric exercise groups had a significantly greater reduction in pain (4/14). Costa et al (2005) (12/14) and Chester et al (2008) (7/14) are discussed below as these studies compared eccentric exercise to shockwave therapy and therapeutic ultrasound respectively. In summary, Sussmitch-Leitch et al (2012) state that eccentric exercise should be an integral component in the management of AT.

The results from this systematic review correspond with the clinical practice guidelines for AT recommended by Carcia et al (2010) who found 'strong' evidence (a preponderance of level 1 and/or level 2 studies supporting the intervention with at least one level 1 study) for eccentric loading for AT.

As mentioned, the majority of studies on eccentric exercise for AT base it on the Alfredson model. Although significant improvements in pain and planter flexor strength were seen in this study, the protocol lacked scientific basis as it was primarily based on clinical experience (Woodley et al 2012). Consequently, different training programmes have originated and the optimum dosage for eccentric exercise remains unclear (Habets and van Cingel 2014).

Training Parameters for Eccentric Exercise:

Habets and van Cingel (2014) completed a systematic review on 13 randomized and clinical control trials evaluating the effect of eccentric exercise for chronic mid-portion AT. The aim was to investigate which training parameters were most effective for pain and patient reported function. The systematic review was performed in accordance with the PRISMA guidelines. To assess the methodological value of the included studies the author's used the PEDro scoring tool which has been shown to be reliable for use in systematic reviews (Maher

et al 2003). PEDro scores ranged from 8/10 to 1/10. The authors' state that none of the studies met the criteria regarding the blinding of subjects and therapists', however blinding therapists or participants to a specific form of exercise is difficult to accomplish. The participants included ranged from 32.5 years to 53.5 years and were both athletes and non-athletes. Only one study reported deterioration in functional activities following an eccentric exercise intervention (Chester et al 2008). This study used a similar protocol to Alfredson et al (1998) but included a 10 second static hold in end range dorsiflexion before returning to the starting position (Chester et al 2008). However, this should be interpreted with caution as the study had a small sample size and limited methodological quality (5/10 Pedro).

6 out of 13 studies used the Alfredson protocol but only one study explicitly mentioned the weight that was used and information on the speed at which the exercise was performed is lacking in all studies. Alfredon (1998) stated that exercises were performed slowly but failed to provide further details. All studies which used the Alfredson protocol found significant improvements for both pain and function. However other protocols did achieve similar results. Rompe et al (2007) asked patients to perform 1x10 repetitions on the first day and progressed to 3x15 repetitions once daily on the seventh day. From weeks 2-12 they continued according to the Alfredson programme. There was a 49% improvement at 16 weeks follow-up according to the Victorian Institute of Sport Assessment- Achilles (VISA-A). Similarly, Roos et al (2004) gradually progressed exercises beginning with 1x15 for the first two days and progressing to 2x15 for days 3-4 and 3x15 on days 5-7. Exercises were performed with the knee extended for the first week and the original Alfredson protocol was introduced from weeks 2-12. Significant results were reported but as the authors used a different outcome measure (Foot and Ankle Outcome Score) to other studies which followed the Alfredson procedure vigorously, a comparison to these studies is difficult.

A few studies used different eccentric training protocols. Petersen et al (2007) prescribed the Alfredson eccentric exercises thrice daily and reported significant improvements in pain and function as measured with the visual analogue scale (VAS) and the American Orthopaedic Foot and Ankle Society (AOFAS) score. However, as this was the only study which used that AOFAS and also had some methodological shortcomings (5/10) it is difficult to interpret the results. Knobloch et al (2007) used 3x15 once a day and 2 second speed for the eccentric phase. Pain reduction was 50% but the study was underpowered with only 15 participants. In

their other study Knobloch et al (2008) performed 3x15 exercises twice a day and found a 33% reduction in pain but had a more adequate sample size of 54. Both studies were of moderate methodological quality (6/10). Habets and van Clingel (2014) conclude that that there is no consensus regarding the effectiveness of different training parameters for eccentric exercise programmes. This is due to the heterogeneity in study populations and outcome measures. A lack of data on training compliance also prevents the magnitude of the intervention from being calculated. Habets and van Clingel (2014) acknowledge that no studies have directly compared different eccentric exercise programmes with different training parameters or compared eccentric exercise with other exercise regimens such as concentric-eccentric exercise training, isometric training or heavy-slow resistance training, identifying a gap for future research.

Is eccentric training the only loading option?

Isolated eccentric muscle training has become the dominant conservative management for AT. Sussmitch-Leitch et al (2012) commented on this prominence in their systematic review as only one of the included studies did not use an eccentric exercise programme as their primary intervention, as a comparison intervention or as a component of a multi-model approach. But in some studies, up to 45% of sedentary participants have not responded to eccentric strengthening (a less than 10 point improvement in the VISA-A) (Sayana and Maffulli 2007). In Maffulli et al (2008) only 60% of participants in an athletic population responded to an intensive heavy load eccentric heel drop regimen alone. Both these studies indicate that eccentric loading may not be effective for all patients with tendinopathy. A systematic review by Malliaras et al (2013) compared clinical outcomes and identified potential mechanisms for effectiveness in Achilles and patellar tendinopathy loading programmes. The four most common loading programmes in articles included in this review can be seen in Table 2.4; eccentric, Silbernagel-combined, Curwin and Stanish and heavy slow resistance (HSR). The Silbernagel-combined programme involves progression from eccentric-concentric to eccentric load and finally faster eccentric-concentric and plyometrics. Stanish and Curwin involves both eccentric and concentric contractions while HSR involves slow double leg isotonic eccentric-concentric contractions with added weight according to the weight you can lift for a defined number of repetitions. Table 2.4 also lists the number of articles which focused on a specific tendon according to the loading programme used.

Programmes	Type of Exercise	Sets, Reps	Frequency	Progression	Pain	Tendon
Alfredson	Eccentric	3 x 15	Twice daily	Via loading	Enough load to achieve up to moderate pain	16 Achilles 10 patellar
Curwin and Stanish	Eccentric-concentric, power	3 x 10-20	Daily	Initially increase speed, then load	Enough load to be painful in the third set	1 Achilles 2 patellar
Silbernagel	Eccentric-concentric, eccentric, faster eccentric-concentric, balance exercise, plyometrics	Various	Daily	Increase volume and type of exercise	Acceptable if less than 5/10 on VAS* (10 worse pain imaginable) and subsided by the day	4 Achilles
HSR	Eccentric-concentric. 3 seconds for heel drop, 3 seconds for raising up	4 sets by 15-6 rep max.	3x/week	Increase load/ reps according to repetition maximum	Acceptable if was not worse after.	2 patellar

Table 2.4: Characteristics of Alfredson, Curwin and Stanish, Silbernagel and HSR programmes. Adapted from Malliaras et al (2013). *VAS= Visual Analogue Scale

Are isolated eccentric contractions justified clinically?

Rowe et al (2012) completed a mixed methods study incorporating a systematic review of the literature along with clinical reasoning from experts for the conservative management of mid-portion AT. It was found that eccentric loading exercises have the strongest supporting evidence of all the conservative modalities (Rowe et al 2012). Incorporating the second pillar of evidence based practice-clinical expertise, it was also found that clinicians nearly always used eccentric loading exercises for mid-portion AT patients. However, clinicians reported using complex clinical reasoning to adapt the protocols used in research for their individual patients. For example, the need to change regimented eccentric protocols due to patient's pain and incorporate mixed concentric/eccentric or isometric loading initially (Rowe et al 2012).

Malliaras et al (2013) suggest that clinical improvement is not dependent on isolated eccentric loading in AT but do state that other mechanisms of eccentric muscle contraction which do not relate to load potential were not examined in this review. For example two studies (Henriksen et al 2009; Rees et al 2008) found that there are tendon force fluctuations (8-12Hz) with eccentric contraction that are not present during a concentric contraction. It is not known whether this influences muscle tendon adaption or is simply related to motor control differences.

The highest level of evidence supported eccentric and Silbernagel-combined loading in the Achilles (moderate evidence) and HSR loading in the patellar (strong evidence). There was limited evidence for Stanish and Curwin and isokinetic loading in the Achilles. Although eccentric loading is frequently associated with greater muscle-tendon unit load and adaption, Malliaras *et al* (2013) did not identify any evidence of this among tendinopathy patients. A systematic review by Roig *et al* (2009) showed that eccentric training resulted in greater muscle strength gains and hypertrophy than concentric loading in healthy patients. However, this did not remain true when the load was equalized suggesting that load intensity rather than contraction type is the stimulus for greater muscle tendon load.

In terms of muscle tendon unit compliance and length-tension relationship of the tendon, Malliaras et al (2013) state that clarifying whether contraction type, load intensity or loading range of motion influence outcomes such as ankle range of motion (dorsi and planter flexion) and reduced resistance to passive stretch (muscle-tendon unit compliance) relates to clinical

outcomes in tendinopathy may improve effectiveness in rehabilitation. For example, a loading programme which achieves increased muscle-tendon unit compliance may lead to better clinical outcomes in patients with AT who have reduced muscle tendon unit compliance.

Pain

Alfredson et al (1998) hypothesized that painful eccentric calf drops have a direct effect on neurovascular ingrowth which may influence pain. In Sussmitch-Leitch et al (2012), 89% of the studies included in their review adopted the Alfredson approach of allowing pain during exercise. Malliaras et al (2013) did not identify any evidence that a change in glutamate may explain clinical outcomes in tendinopathy. They did state that the popular approach of exposing tendons to progressive eccentric loading whilst monitoring tendon irritability is likely to have some effect on pain perception (Malliaras et al 2013). Pain during exercise may influence patient compliance and Sussmitch-Leitch et al (2012) advise on the use of diaries for monitoring this in future studies so as to determine its effect on patient outcomes.

Practice Point: Clinicians should consider eccentric-concentric loading alongside or instead of eccentric loading in ATP. The gradual progression from eccentric-concentric to eccentric followed by faster loading used in the Silbernagel combined loading programme may benefit patients who are unable to start with an Alfredson eccentric programme due to pain or calf weakness (Malliaras et al 2013). Heavy slow resistance training may be more likely to achieve tendon adaption and may be better suited to patient subgroups such as those with less irritable symptoms or athletes who have high-load demands (Malliaras et al 2013). However, caution is needed in interpreting findings as only two studies, both in the patellar tendon, investigated this mechanism of loading.

Maintenance Programmes

Some studies reported neuromuscular and jump performance deficits at 12 months and 5 years, questioning the length of current loading programmes and suggesting that maintenance programmes may be required on return to sport (Malliaras et al 2013). Continued sport, as long as symptoms allow, appears to have a specific positive effect on calf-power and jump performance which is not gained with the Silbernagel loading programme alone even though it includes faster calf loading and stretch-shorten cycle rehabilitation (Malliaras et al 2013). Continued sports may lead to specific gains such as improved jump performance (Silbernagel

et al 2007a), however this should be implemented carefully alongside a pain monitoring system (Malliaras et al 2013). Pain was acceptable in rehabilitation programmes in most studies included in this systematic review but the clinician must educate patients about acceptable loading-related pain (Malliaras et al 2013).

Future Research:

- Should focus on variables within the eccentric training programme such as speed of exercises, duration, rate of progression and loading, chronicity and severity of condition (Rowe et al 2012).
- Future studies need to investigate how load intensity, time under tension, speed, contraction type and other factors influence clinical and mechanistic outcomes rather than accepting isolated eccentric loading as the gold standard for tendinopathy rehabilitation (Malliaras et al 2013).

Concentric Exercise

Moderate evidence from Mafi et al 2001 and Niesen-Vertommen et al 1992 suggests that concentric calf muscle training is not as effective as an eccentric training programme. These studies have been discussed in more detail under the '*Eccentric Exercise'* heading as both studies compared concentric and eccentric programmes and found significantly better reductions in pain for the eccentric group. However, in both studies the concentric group did register some improvement and clinicans frequently use combined eccentric-concentric exercises for patients who cannot tolerate eccentric exercises due to pain or weakness (Rowe et al 2012). This is similar to the work of Silbernagel et al (2007) where patients progressed from combined eccentric-concentric to eccentric exercises.

Future research:

- Guidance on when to introduce combined concentric-eccentric exercises.

Practice Point:

If patient is too sore to complete eccentric loading exercises, consider concentric or isometric loading.

Shock-wave Therapy

A meta-analysis revealed that shockwave therapy (SWT) and eccentric exercise have similar effects (Sussmitch-Leitch et al 2012). Five studies evaluated the effects SWT on AT using the VISA-A with a mean methodological quality of 11.2 +/- 0.4 out of 14 according to the modified PEDro scale. One study compared SWT to eccentric exercise alone and there were no significant effects for outcome on pain or function at 16 weeks (Rompe et al 2007). In addition, Rompe et al (2007) also had a wait & see group allowing comparisons between control and SWT groups with the results significantly favouring SWT at 16 weeks. Rompe and colleagues (2009) also analysed the combined effects of SWT and eccentric exercise compared to eccentric exercise alone after 16 weeks with moderate significant effects favouring the combined approach. This suggests that utilising SWT in combination with eccentric exercise is likely to have better patient outcomes than SWT or eccentric exercise alone. However, at 12 months follow-up there was no difference in outcomes between the groups. This corresponds with the findings of two high quality double-blind placebo controlled trials (Costa et al 2005, 12/14; Ramussen et al 2008, 11/14) which found that when sham SWT was compared to standard SWT there were no significant differences, suggesting a placebo effect. Furthermore, there are practical considerations regarding the use of SWT. Considering the need for specialised equipment and practitioner training, SWT is an intervention which is not as easy to use or apply when compared to eccentric exercise. Sussmitch- Leitch et al (2012) suggest the use of SWT for patients who cannot or will not use eccentric exercise.

Practice point:

Implementing SWT may be considered an inappropriate addition to the treatment of ATP with eccentric exercises being as effective in the long run. But, it may be desirable for athletic patients requiring a quicker recovery and return to sport (Rowe et al 2012).

Laser

According to Rowe et al (2012) findings, clinicians do not use laser therapy in the treatment of AT as it is perceived as more applicable to acute inflammation and as an expensive device with no good quality evidence to support its use. However, Rowe et al (2012) found moderate evidence for the addition of laser therapy to an eccentric exercise programme for

AT. Stergioulas et al (2008) (8/10 on PEDro) compared eccentric exercise and laser therapy to eccentric exercise plus sham laser therapy over a period of 8 weeks. There was a significant difference in pain according to the VAS at 4, 8 and 12 weeks for the combined group and not the sham group.

Tumilty et al (2008) also compared laser therapy with sham laser therapy in addition to an eccentric exercise programme. Sussmilch-Leitch et al (2012) completed a meta-analysis of the data from Tumilty et al (2008) (10/10 on PEDro) and Sterigoulas et al (2008) which did not support the use of laser therapy in conjunction with eccentric exercise at 4 weeks but did find significant effects favouring the use of laser therapy at 12 weeks. More recent evidence from Tumilty et al (2012) (10/10 on PEDro) found no evidence for the use of laser therapy in conjunction with eccentric exercise according to the VISA-A and VAS, even at 12 weeks follow-up.

Practice point:

Further research is warranted for the use of laser therapy in conjunction with eccentric exercise as the evidence is conflicting.

Ultrasound

Chester et al (2008) completed a pilot RCT comparing eccentric exercise and ultrasound to eccentric exercises alone for the treatment of AT. At twelve weeks there were no significant differences or clear trends between groups for pain or function according to the VAS and functional index of the leg and lower limb. This was attributed to the small sample size (n=16) and the results for pain and function were deemed inconclusive (Rowe et al 2012).

Future research

- Further high-quality research is required to confirm the effectiveness of ultrasound for patients with AT.

Braces/Splints

Sussmitch-Leitch et al (2012) also conducted a meta-analysis on the results of two studies (McAleenan et al 2010; de Jong et al 2010) which investigated the effects of a night splint to an eccentric exercise programme and found no significant results in terms of function or pain at 12 weeks. There was also no evidence found for the addition of a heel brace to a 12 week eccentric exercise programme (Knobloch et al 2007). However, Rowe et al (2012) found that clinicians would consider the use of splinting or bracing for patients with failed healing or late-stage tendinopathies.

Practice Point:

There is currently no evidence to support the use of splinting or bracing for ATP. However, clinicians have used it as a last resort (Rowe et al 2012)

Stretching

Stretching has anecdotally been recommended as an intervention for patients with AT and there is little evidence to support its use for preventing or managing AT (Altman et al 2010). One study by Norregaard et al (2006) compared an eccentric exercise programme to a calf-stretching programme for twelve weeks in 45 patients with chronic AT. Although subjects were randomly assigned, there was a lack of methodological quality as the reliability for the outcome measrues was not stated and it was unclear how many subjects were ultimately in each group. Follow-up was completed at 3,6,9,12 and 52 weeks. Both groups gradually improved according to the parameters (tendon, tenderness, tendon thickness, self-report symptoms and patient's global assessment) but it is unclear if this was due to the passage of time, the intervention or a combination of both as there was no control group (Altman et al 2010).

Stretching has been used as an adjunct to some interventions identified in the literature but it has not been investigated as a separate modality (Rowe et al 2012). Range of movement was not considered an important element for AT unless specific joint or muscle restriction were identified by the clinicans in Rowe et al (2012). Some clinicians reasoned for not including stretches so as to avoid compression of the tendon, a loss of strengthening and exacerbating pain (Rowe et al 2012).

Practice Point: No evidence currently exists to support the use of stretching for ATP but equally there is no evidence to confirm that this 'compresses' the tendon or that you 'run the risk' of making the tendon weaker by doing so. Common sense suggests stretching exercises can be used to reduce pain and improve function in patients who have limited dorsiflexion range of motion in ATP.

Prognosis

The long-term prognosis of AT is generally good when conservative management is used. 4-6 months of conservative management is recommended for before exploring the possibility of surgical management (Carcia et al, 2010). Conservative management generally consists of a 12 week program of heavy-load eccentric exercises, to which up to 90% of individuals are satisfied with results and capable of returning to previous tendon-loading activities (Alfredson & Cook, 2007). A long term follow up (3.8 years mean) post 12 week Alfredson eccentric exercise protocol found reduced tendon thickness and more normal ultrasound imaging , with decreased tendon volume on magnetic resonance imaging (MRI) and 23% decrease in signal intensity (Alfredson & Cook, 2007).

Similar results were found in other studies where long-term follow up were applied. After 4.2 years, Gardin et al (2010) found that from using 3 months eccentric exercise program, 95% of patients reported decreased pain, with 65% of these experiencing none or very mild pain on strenuous activity. At follow-up, 85% had improved performance, and 60% had normal performance. Pain and functional measures were significantly better at long-term follow up than before commencement of treatment and immediately after the 3 month exercise program (Gardin et al, 2010). In the same study, 4 patients who did not follow the eccentric exercise protocol showed no improvements in pain or function at 17 month follow-up. This suggests good long-term prognosis following eccentric training and conservative management (Gardin et al, 2010).

Since the popularisation of the eccentric strengthening protocol for AT, conservative management has become a more effective means of treating the condition. Alfredson & Cook (2007) reported that when eccentric strengthening was used 82% of patients return to their

previous level of physical activity, whereas when concentric strengthening was chosen, only 36% of patients report satisfaction and return to full previous levels of physical activity. This is evident when the management from the 1990s and early 2000 is brought into question. When patients were treated with relative rest and activity modification, as well as stretching of the triceps surae and peritendinous corticosteroid injections 24%-49% of AT patients were treated surgically (Paavola et al, 2000). Within their own population 29% required surgical management between onset of symptoms and 8 year follow up due to failure of conservative management (Paavola et al, 2000). At a similar time, when a 12 week eccentric exercise protocol was adhered to, just 7% of patients required surgical intervention at 2-year follow up (Alfredson & Lorentzen, 2000). This is comparable to results found 7 years later using the same protocol, when 90% of individuals were satisfied with their treatment results (Alfredson & Cook, 2007).

Surgical management is an invasive procedure which involves the removal of fibrotic adhesions and degenerative nodules to restore vascularity (Carcia et al, 2010). Surgery is normally a percutaneous tenotomy, or an open procedure, which is designed to irritate the tendon and initiate a chemically mediated healing response (Alfredson & Cook, 2007). Percutaneous tenotomy leads to good/excellent results at 18 month follow up, with 67% of individuals returning to full physical activity within 7 months. It has been suggested that the results of surgical intervention are dependent on the extensive rehabilitation post-surgery, and that the results may not be as favourable for those who do not perform adequate rehabilitation to gain full strength and functional capacity (Alfredson & Cook, 2007).

Future Directions

Though evidence strongly supports eccentric based exercise interventions, other loading protocols have been introduced, with potentially promising results. The implementation of Heavy Slow Resistance (HSR) has begun in the treatment of both patellar and the Achilles tendinopathies. The research surrounding the use of HSR is in its infancy, though promising results have been shown in the patella (Kongsgaard et al, 2009; Kongsgaard et al, 2010). A systematic review by Malliaras et al (2013) identified a strong potential for the use of HSR in patellar tendinopathy, though suggested that research evidence was currently lacking for its application in AT due to no RCTs being carried out on this. Further research focussing on

HSR as a treatment modality for AT is currently underway, and the results of this study are anticipated.

Though little research evidence exists for its clinical application, some clinicians believe that isometric contractions have an analgesic effect on tendon pain associated with AT. Their use has been identified to load the tendon without increasing pain in reactive and painful tendinopathy, and to continue loading a tendon which has become increasingly painful following eccentric exercises for the treatment of AT. Information gathered around this was from clinical expertise, though research into the subject area and clinical utility of this contraction type is currently underway.

Key References
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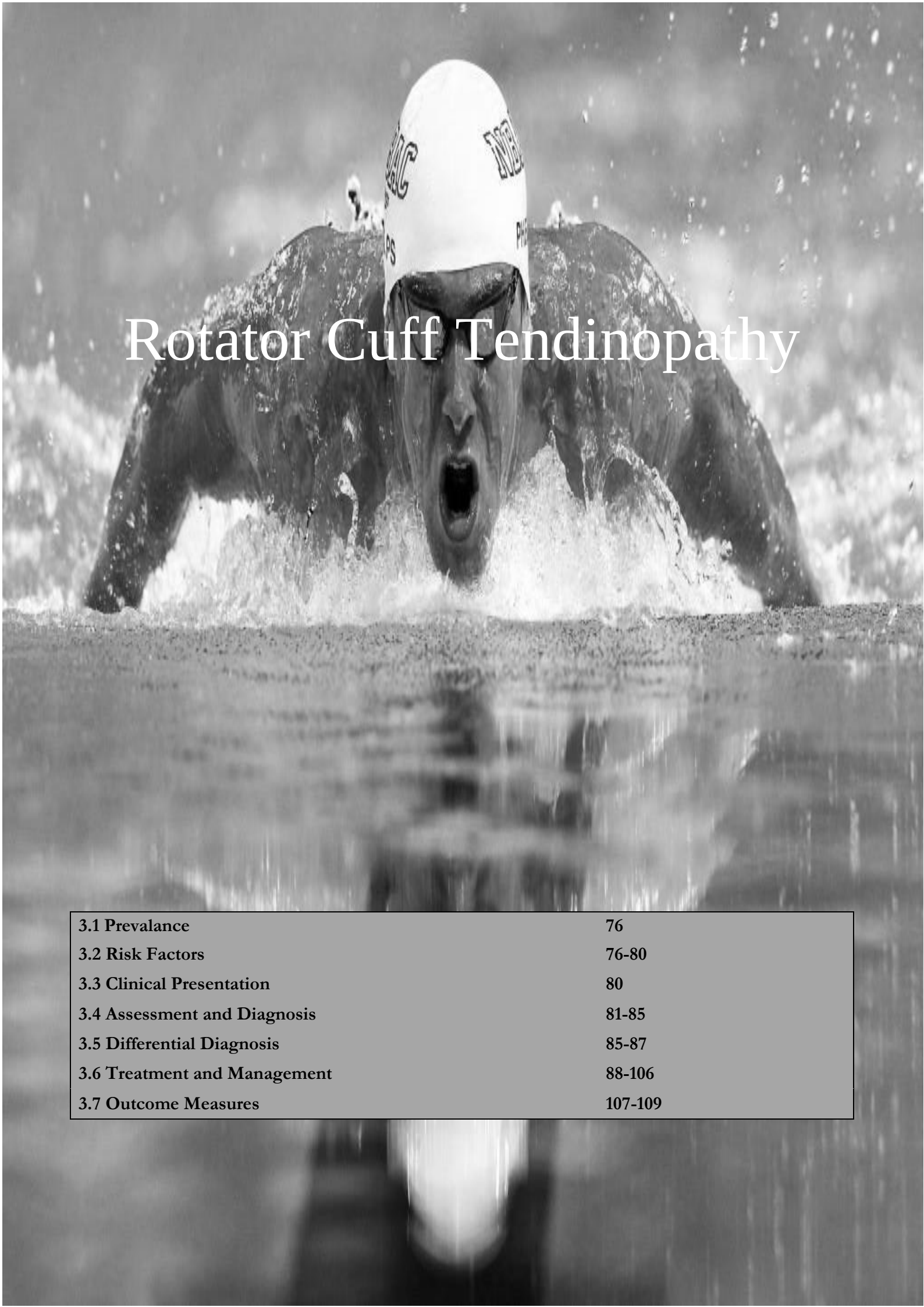
• Rowe, V., Hemmings, S., Barton, C., Malliaras, P., Maffulli, N. and Morrissey, D. (2012) 'Conservative management of midportion Achilles tendinopathy; A mixed methods study, integrating systematic review and clinical reasoning' *Sports Medicine,* 42(11), 941-967.



Rotator Cuff Tendinopathy

Prevalence of Rotator Cuff Tendinopathy

Shoulder pain is a very common clinical presentation ranked the 3rd most common musculoskeletal reason for GP consultation annually (Hanratty *et al* 2012). Specifically 85% of disorders of the shoulder can then be further related to rotator cuff tendinopathy (Ostor *et al* 2005).

In a general population, the incidence of rotator cuff tendinopathy ranges from 0.3% to 5.5% and annual prevalence from 0.5% to 7.4% (Littlewood *et al* 2013). Interestingly, the shoulder is the most common site of pain reported in the wheelchair population ranging from 31% to 73% (Finley and Rodgers 2004).

There is a lack of data on the prevalence of rotator cuff tendinopathy in sporting populations However, athletes engaged in sports that involve overhead motions such as throwing athletes (javelin, baseball and American football), tennis, or swimming are more susceptible to rotator cuff injury (Scott and Ashe 2006). It is found that a combination of swimming more than 15 hours/week or 35 km/week predict the appearance of rotator cuff tendinopathy in 71% of elite swimmers.

Risk Factors: Rotator Cuff Tendinopathy

Little is understood about the pathogenesis of tendon pain in the early stages. Tendinopathic changes are frequently asymptomatic. Many patients present themselves to physicians and physiotherapists when they are symptomatic. This presentation may be precipitated by an increase in tendon loading (Scott *et al.* 2013).

A recent systematic review summarised the main histological and molecular findings in human and animal models of rotator cuff overuse (Dean *et al.* 2012). The paper concluded that intrinsic, extrinsic and environmental factors in combination or isolation have a role in the disordered tendon homeostasis of rotator cuff disease. The paper outlines a number of degenerative mechanisms, some related to inflammatory pathways (interleukin-1, substance P) and others related to altered loading conditions or systemic influences (aging). The paper noted the disparity of the level of degenerative change and the extent of symptoms.

The mechanisms of the development of rotator ruff tendinopathy are multi factorial. Outlined in Figure 3.1 are the extrinsic and intrinsic mechanisms of rotator cuff Tendinopathy as proposed by Seitz et al (2011). This review attempted to identify subgroups of patients potentially affected by rotator cuff tendinopathy based on these mechanisms. By developing a clearer understanding of the mechanism involved in the aetiology of rotator cuff tendinopathy it is postulated that a better suite of targeted interventions can be used by therapists and clinicians. Lewis (2010) used the continuum model of tendinopathy to describe how rotator cuff pathology can oscillate between the three stages of reactive, disrepair and degenerative tendinopathy (Lewis 2010).

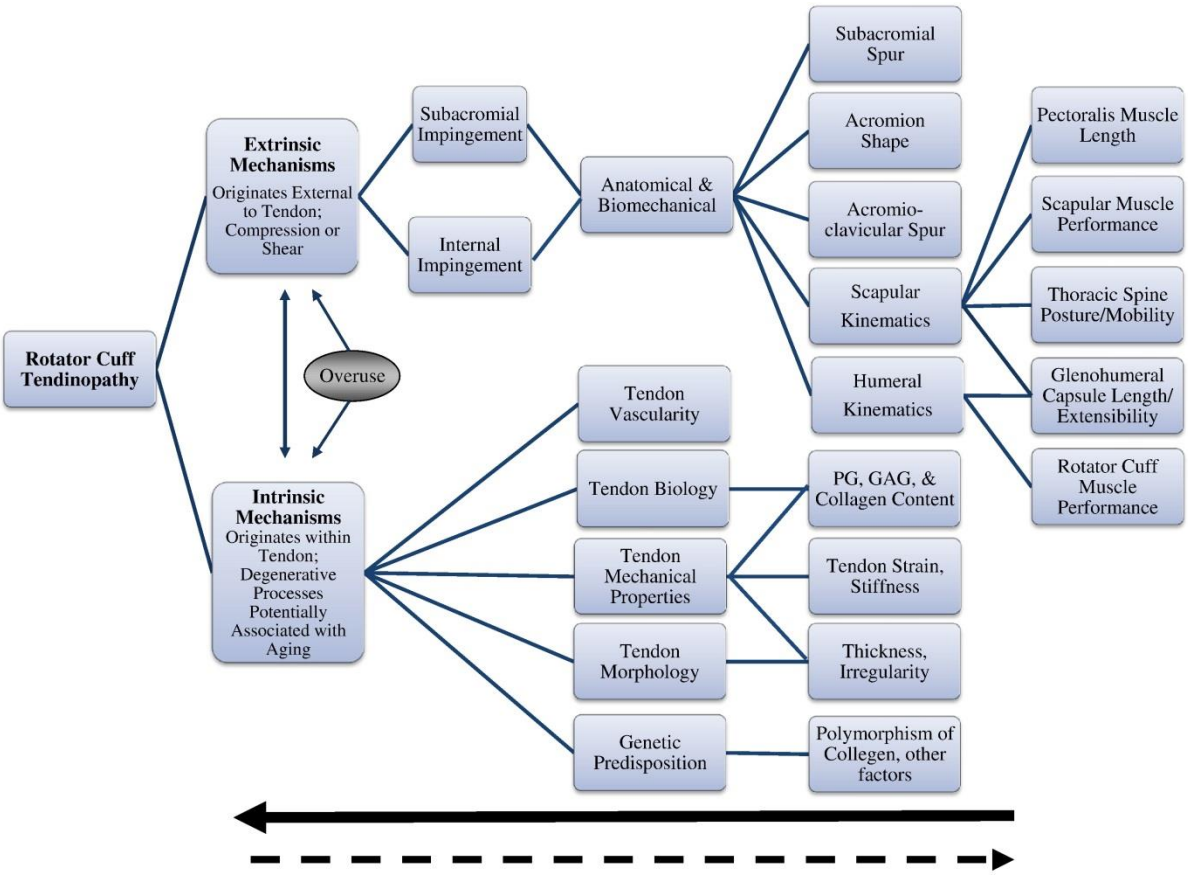


Fig 3.1: Extrinsic and intrinsic mechanisms of Rotator Cuff Tendinopathy. Lines indicate non directional evidence of these relationships (Seitz et al. 2011).

Extrinsic Factors

Extrinsic mechanisms of rotator cuff tendinopathy are those relating to the external compression of the tendons due to anatomical or biomechanical factors. Neer proposed an extrinsic mechanism to the development of Rotator Cuff tendinopathy with compression of the RC tendons and associated tissues within the subacromial space (Lewis *et al.* 2001). This compressive load may not describe all rotator cuff tendinopathies accurately. A novel extrinsic mechanism, internal impingement, has been described in overhead athletes (Burkhart *et al.* 2003). Internal impingement occurs due to compression of the articular side rather than the bursal side of the rotator cuff tendons, between the posterior superior glenoid rim and humerus when the arm is in full external rotation, abduction and extension. The rotator cuff tendons are thought to become impinged between the posterior glenoid rim and the greater tuberosity of the humeral head, leading to tendon degeneration and labral damage (Manske *et al.* 2004).

The evidence in relation to the association between acromial shape and rotator cuff tendinopathy is conflicting. Whilst some studies suggest linkages between the presence of a hooked (Type III) or sloped acromion and rotator cuff tears, other studies suggest that the prevalence of altered acromial shape is similar among those with and without rotator cuff tendon pathology (Hirano *et al.* 2002). There is evidence from a number of studies that altered acromial shape is resultant of the development of spurs on the coraco-acromial arch, as a secondary consequence of rotator cuff tendinopathy or stresses on the coracroacromial ligament (Chambler *et al.* 2003).

While anatomical differences or degenerative bony change of the coraco-acromial arch have been proposed as a static source of subacromial impingement, kinematic abnormalities of scapular and shoulder girdle motion have been suggested as a possible source of dynamic reduction of the subacromial space during shoulder overhead motion (Ludewig and Cook 2000). A recent systematic review exploring the relationship between scapular orientation and subacromial impingement demonstrated that studies that suggest causation are confounded by very small sample sizes and the use of healthy shoulders in young people as comparisons (Ratcliffe *et al.* 2014).

A potential dynamic mechanism of subacromial space reduction is superior migration of the humeral head, which has been extensively described to contribute to rotator cuff dysfunction (Keener *et al.* 2009) . The mechanism of this upward humeral head displacement is not clearly understood, lack of inferior stabilisation by the rotator cuff, relative to a normal or increased upward pull of the deltoid muscle, is an explanation that has been postulated (Kim *et al.* 2010). Electromyographic studies of rotator cuff activation in people with rotator cuff tendinopathy have reported reduced activation levels in supraspinatus and infraspinatus muscles (Bandholm *et al.* 2008).

Intrinsic Factors

Intrinsic mechanisms of rotator cuff tendinopathy describe factors which influence tendon health and quality, causing degeneration that exceeds the tendon's capacity to heal and repair, and includes factors such as overload of tendons capacity, ageing, genetics, vascular changes and adiposity (Seitz *et al.* 2011).

Evidence would suggest that the onset of rotator tendinopathy increase with advanced age. Both symptomatic and asymptomatic prevalence rates of degenerative rotator cuff changes increase from middle age (Tempelhof *et al.* 1999).

A high prevalence rate of rotator cuff tears and tendinopathy has been observed amongst siblings which could indicate that there is a genetic component to the disease process (Tashjian *et al.* 2014). Results showed that 32.3% of patients in the rotator cuff tear group studied reported that family members had a history of rotator cuff problems or surgery compared to only 18.3% of the controls.

Links between waist circumference, metabolic syndrome and smoking and the prevalence of rotator tendinopathy have been observed (Rechardt *et al.* 2010). In a large study conducted in the Finnish population, the authors suggest that altered glucose metabolism and vascular pathology caused by increased adiposity and smoking may contribute to tendon damage.

Consistent loading beyond the physiological capacity of a tendon is considered to be an important factor in the aetiology of tendinopathy (Scott *et al.* 2013). In a study of Elite swimmers 73/80 (91%) swimmers reported shoulder pain. Most (84%) had a positive

impingement sign, and 69% of those examined with MRI had supraspinatus tendinopathy (Sein *et al.* 2010).

KEY REFERENCE
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Clinical Presentation: Rotator Cuff Tendinopathy

The most common symptom of rotator cuff tendinopathy is shoulder and arm pain, especially during overhead activities (Hermans *et al.* 2013). The sensation is described as dull pain that becomes sharp and stabbing during overhead motion. The athlete or sportsperson with rotator cuff tendinopathy complains of pain with overhead activity such as throwing, swimming and overhead shots in racquet sports. Activities undertaken at less than 90^0 of abduction are usually pain free (Brukner and Khan 2007).

Pain is often located in the region of the deltoid muscle, ranging from its origin at the clavicle, acromion process, and scapular spine to its insertion at the middle part of the humerus (Hermans *et al.* 2013).

Other symptoms include night pain particularly when sleeping on the effected shoulder, weakness, stiffness, or crepitus that may be heard during shoulder movement. Inspection of the supraspinatous and infraspinatus fossae above and below the scapular spine can show atrophy. Swelling seldom occurs but can be a sign of inflammatory or traumatic changes (Hermans *et al.* 2013).

Key References
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Assessment and Diagnosis of Rotator Cuff Tendinopathy

Rotator cuff tendinopathy diagnosis is typically made by a combination of history and physical examination. Traditionally the examination of the shoulder has been based on the assertion that it possible to isolate individual structures and mechanically stretching or compressing the tissue of interest will help with diagnosis. However muscle contraction does not occur in isolation with EMG results showing multiple muscle activations during shoulder special tests (Brookham et al. 2010).

Shoulder Assessment: Physical Assessment

KEY RESOURCE: BJSM SHOUDLER EXAM
https://www.youtube.com/playlist?list=PLB2E91E9C20E30B25

The assessment of the shoulder region often involves taking extensive history combined with physical assessment in an attempt to diagnose shoulder pathology such as rotator cuff tendinopathy. Historical musculoskeletal assessment owes its foundations to the concept that it is possible to isolate individual structures and apply mechanical procedures that either compresses of stretches the tissue of interest (Lewis 2009). Lewis (2009) argues that the commonly used orthopaedic special tests should be thought of as pain or symptom provocation tests, without the ability to contribute to a structural diagnosis.

The clinical tests used to identify structural pathology in current use include the O'Brien active compression test for superior labral pathology, the posterior capsule length test to assess the extensibility of the posterior glenohumeral capsule and Jobe's ''supraspinatus test'' to assess the strength and pain response from the supraspinatus musculotendinous unit. Other tests include the Neer sign, which has been embraced with other tests, such as the Hawkins' test and the Internal Rotation Resistance Stress Test, as clinical methods to implicate the acromion as the cause of the presenting shoulder symptoms (Lewis 2009).

A high quality systematic review and meta-analysis of various shoulder physical tests failed to recommend that any one test or combination of tests was appropriate in the diagnosing of shoulder conditions (Hegedus *et al.* 2012).

Is there a solution?

The diagnosis of Rotator tendinopathy is challenging, with both imaging-based and clinical diagnostic approaches having numerous flaws (Lewis 2009). As noted above, Hegedus et al (2012) reported that none of the shoulder clinical diagnostic tests provide an accurate diagnosis of pathology. However, the evidence does demonstrate that there was marginal better accuracy with a combination of tests. Michener et al (2009) found that, a cut-off of three out of five positive tests (painful arc, empty can, external rotation resistance, Neer, and Hawkins and Kennedy) best predicted rotator cuff tendinopathy, while less than three positive tests ruled it out (Michener *et al.* 2009).

A review by Hermans et al (2013) concluded that the painful arc sign and pain or weakness on resisted external rotation were the best predictors of any degree of Rotator Cuff disease (Hermans *et al.* 2013). The best indicators for rotator cuff tear were found to be a positive internal or external rotation lag sign. Figure 3.2 outlines the recommended tests for the clinical diagnosis and evaluation of rotator cuff disease. The review also produced an excellent summary of its findings which can be found here:

KEY RESOURCE
http://jama.jamanetwork.com/article.aspx?articleid=1733724#SummaryVideo

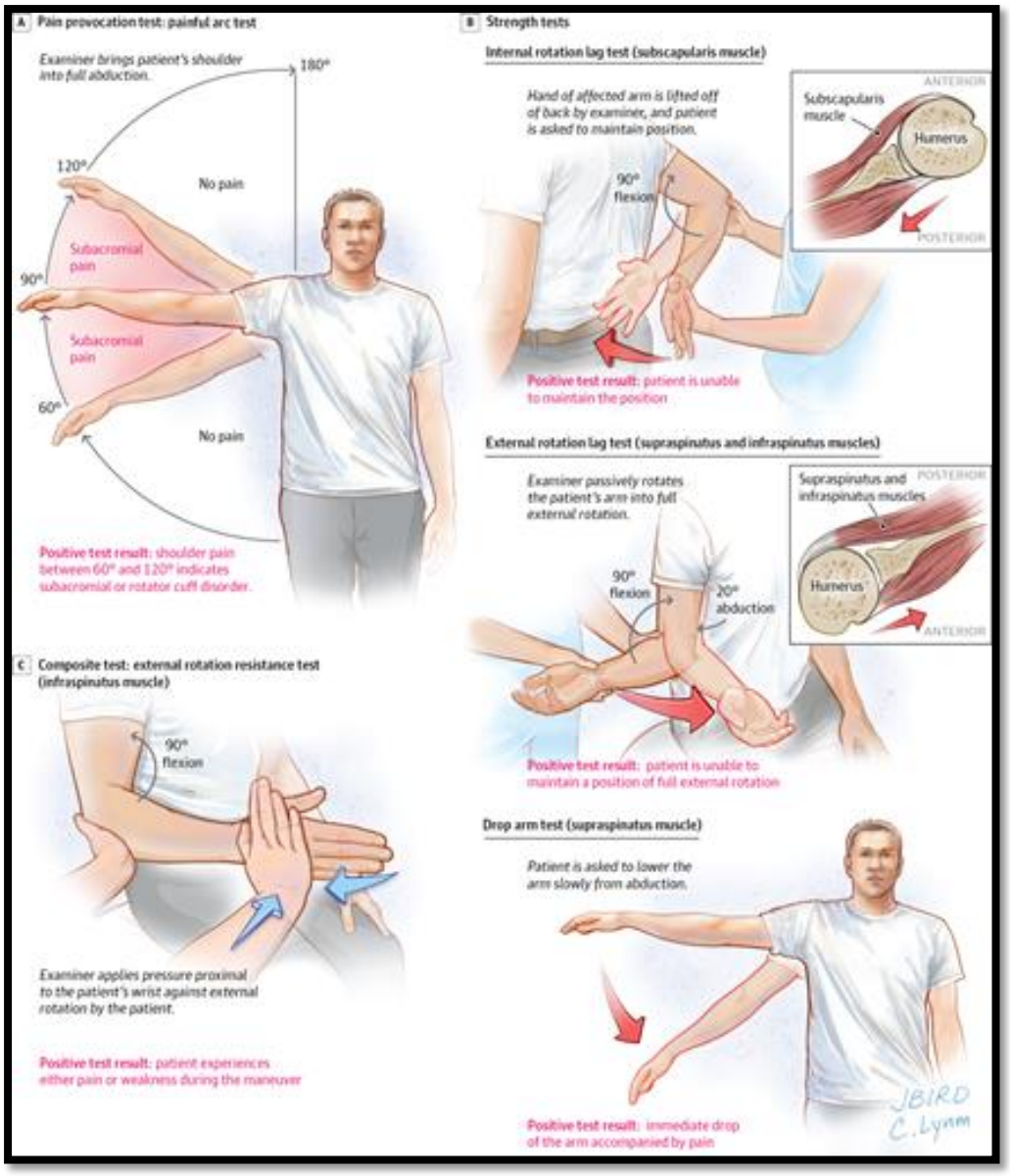


Fig 3.2: A, The positive result of the painful arc test, a pain provocation test, is characteristic of shoulder pain during abduction of the arm between 60° and 120°, suggesting a subacromial impingement syndrome or rotator cuff disorder due to compression of the rotator cuff muscles and subacromial bursa between the humeral head, acromion, or coracoid process. B, Strength tests assess muscle function of a specific rotator cuff muscle, producing weakness, pain, or both, especially when the patient has a partial rotator cuff tear. During such tests, the patient either moves the arm toward a certain position or maintains a certain position of the arm or shoulder against gravity. The internal rotation lag test evaluates the subscapularis muscle. The external rotation lag test assesses both the supraspinatus and infraspinatus muscles. The drop arm test assesses the integrity of the supraspinatus muscle. A positive test result is an immediate drop of the arm accompanied by pain. C, The external rotation resistance test is a composite test of the infraspinatus muscle. The test is positive when the patient experiences either pain or weakness during the maneuveur (Hermans et al. 2013).

Imaging

Ultrasonography

Many clinicians recognise ultrasonography as the gold standard for the initial evaluation of rotator cuff tendinopathy (Broadhurst and Simmons 2007). It allows clinicians to examine tendons whilst they are in motion and comparison with the contralateral shoulder is beneficial. It is also advantageous because of its ease of use, relative low cost and lack of exposure to radiation (Broadhurst and Simmons 2007). Changes observed on diagnostic ultrasound for rotator cuff pathology include: hypoechoic areas within the tendon and thickening of the tendon with or without internal hypo- or hyperechoic foci (Naredo et al. 2002).

The findings of ultrasonography for diagnosis of rotator cuff tendinopathy must be taken with caution though. Ultrasonography is found to correlate poorly with both surgery and arthrography for diagnosing rotator cuff tears (Martin-Hervas et al. 2001; Burk et al. 1989). Also, a study carried out in Japan on 664 individuals found that asymptomatic tears were actually twice as common in their subjects as symptomatic tears with asymptomatic rotator cuff tears accounting for 65.3% of all tears and asymptomatic tears for 34.7% (Minagawa et al. 2013). A similar finding was seen by Templehof et al. (1999) where similarly asymptomatic rotator cuff tears were found in a large proportion of their subjects. Furthermore, they found that the older the subjects were the higher likelihood they had of demonstrated tears on ultrasonography. From this, it remains unclear to what extent are rotator cuff tears deemed a normal degenerative change with age, and what potentially triggers an asymptomatic tear into a symptomatic one causing pain and functional impairment.

Magnetic Resonance Imaging (MRI)

Undoubtedly MRI use for diagnosing full-thickness rotator cuff tears is evident with a large meta-analysis finding a sensitivity of 92.1% and a specificity of 92.9% when surgery was used as the reference standard (de Jesus et al. 2009). However, the use of MRI in diagnosis of rotator cuff tendinopathy is challenged. With respect to partial thickness tears, sensitivity and specificity are reported as significantly lower values (de Jesus et al. 2009). While good reliability (ICC=0.85) was found for the use of MRI in rotator cuff tendinopathy, this was only achieved for highly experienced radiologists. Inter-rater reliability between highly

experienced and less experienced radiologists was found only to be fair at ICC=0.55 (Sein et al. 2007). Similar to ultrasonography, the ability to achieve an accurate structural diagnosis is lessened due to a poor correlation between radiological imaging findings and symptoms.

It remains clear that the additional value of these diagnostic tests has been seriously challenged due to the presence of abnormal rotator cuff morphology in subjects absent of pain and/or functional deficit (Littlewood 2012).

Differential Diagnosis of Shoulder Pain

Adhesive Capsulitis

Adhesive Capsulitis or Frozen Shoulder is a self-limiting painful shoulder condition where both active and passive movement is limited (Burnabk et al. 2008). It is broken into 3 phases. The first stage is called the *painful stage* where the gradual onset of pain worsens and typically lasts 3-9 months. Movement becomes slightly restricted but this issue is more apparent in the second stage – the *freezing stage*. While pain does not necessarily worsen, movement becomes increasingly more restricted, especially in shoulder external rotation plus flexion and internal rotation. This typically lasts for 4-12 months. The final stage – the *thawing stage* – results in pain and movement gradually improving but can be lengthy and lasts from 12-42 months (Manske and Prohaska 2008).

Adhesive Capsulitis is divided into 2 categories:

- Primary: which is idiopathic and insidious
- Secondary: due to trauma and subsequent immobilization. (Manske and Prohaska 2008)

Adhesive Capsulitis has an incidence of 3-5% in a general population however people with diabetes mellitus have a much higher incidence of 20% (Manske and Prohaska 2008). Adhesive Capsulitis is also associated with other co-morbidities, with thyroid problems consistent with diagnosis (Burbank et al. 2008). Patients that may present with Adhesive Capsulitis are usually aged 40 years or greater, with incidence peaking between 40-70 years. Also women are more commonly affected than men (Ewald 2011).

Subjective assessment: Patient may report anterior shoulder pain, pain at night (especially lying on affected shoulder), stiffness and difficulty dressing.

Objective assessment: Restricted movement in all directions, especially external rotation; pain with movement and eased immediately with rest and no crepitus (Leeds Community Healthcare NHS 2012).

Glenohumeral Instability

Glenohumeral instability refers to disorders affecting the capsular-labral complex of the shoulder, which mainly refers to dislocation and subluxation (Burbank et al. 2008). Patients typically present as younger than 40 years and tend to report a specific event or trauma prior to the dislocation or subluxation often associated with sports or falls. Subjectively, the patient may report a feeling of a 'dead arm' and numbness around the lateral aspect of the shoulder (Burbank et al. 2008). Also the patient may describe the position of injury in an abducted/extended/externally rotated position. Objective assessment testing consistent with diagnosis are positive laxity texts – apprehension & relocation tests and sulcus sign (Leeds Community Healthcare NHS 2012).

Acromioclavicular (AC) Joint Osteoarthritis

AC joint pathology is usually well localised. As a result, tenderness on palpation of the AC joint is highly consistent with diagnosis (Burbank et al. 2008). The patient may report a history of a previous injury to the joint and report of a history of heavy weight-lifting. Similar to osteoarthritis at any other joints in the body, it is characterized by pain and swelling, especially with activity. This activity typically consists of movements involving cross-body adduction, extreme internal rotation and forward flexion (Burbank et al. 2008). While radiographical imaging may be helpful, they may be difficult to interpret and support diagnosis as most people will show signs of AC osteoarthritis by the age of 40-50 years (Burbank et al. 2008).

Glenohumeral Osteoarthritis

Glenohumeral osteoarthritis can present as gradual shoulder pain and loss of ROM. Patients are typically over 50 years, have a history of arthritis, previous injury to the shoulder, and findings of pain, ROM loss and crepitus on objective assessment are consistent with diagnosis (Burbank et al. 2008).

Referred Pain

Shoulder pain may arise from another structure. These include:

- Neural impingement in the cervical spine due to disc herniation or spinal stenosis.
- Visceral: Pain due to gall bladder can result in right shoulder pain, angina can result in left shoulder pain, and diaphragmatic irritation can reproduce ipsilateral shoulder pain (Leeds Community Healthcare NHS 2012).

Red flags

Indications for immediate referral:

- Unexplained deformity or swelling.
- Pulmonary or vascular compromise.
- Suspected malignancy or cancer.
- Significant or unexplained sensory/motor deficits.
- Significant weakness not due to pain.

Indications for urgent referral

- Displaced or unstable fracture.
- Failed attempted reduction of a dislocated shoulder.
- Massive tear of the rotator cuff (greater than 5 cm tear)

(Mitchell et al. 2005)

Conservative Interventions for Rotator Cuff Tendinopathy

Evidence for Eccentric Exercise in Rotator Cuff Tendinopathy

The use of eccentric exercise in rehabilitation has increasingly gained attention in the literature recently as a specific training modality for rotator cuff tendinopathy. Histological changes observed in the rotator cuff (specifically the supraspinatus) have been found to have similarities with those of the Achilles and patellar tendinosis (Kahn *et al* 1999). Furthermore, eccentric exercise has been proposed as an effective conservative treatment in the management of tendinopathy of the Achilles and patellar tendons (Alfredson *et al* 1998; Murtaugh and Ihm 2013). Therefore, it stands to reason that rotator cuff tendinopathies may also benefit from the advocacy of eccentric exercises.

However, very few studies have examined specifically the effect of eccentric exercise alone in rotator cuff tendinopathy and the quality of evidence to support its use is minimal. Those studies which have examined eccentric exercises in rotator cuff tendinopathy are outlined below.

Jonsson *et al* (2006) conducted a pilot study and showed good clinical results of eccentric training for the supraspinatus and deltoid muscles in chronic painful participants. The subjects had pain for a mean of 41 months and were recruited from a waiting list for surgical treatment. Each participant performed eccentric ‘empty can’ exercises that were allowed to be painful as long as the pain had ceased by the following session. This was completed for the whole 12 week period, and based on Alfredson’s protocol with 3 sets of 15 repetitions being completed twice daily. Once the 3 sets could be completed pain-free, progression of the exercise was achieved through adding weights to reach a new painful training level. After the commencement of the training period, a significant reduction in pain intensity was observed through the visual analogue scale (VAS), and a significant improvement in function was noted as measured by the Constant-Murley score. A 1-year follow-up showed that 5/9 patients were extremely satisfied with their status and withdrew from the waiting list for surgical treatment.

Bernhardsson *et al* (2011) carried out a similar study examining the effects of a treatment programme focusing on specific eccentric training with progressive loading of the supraspinatus and infraspinatus rotator cuff tendons on pain intensity and function in subjects with shoulder impingement. It differed from Jonsson *et al* (2006) in that the programme consisted of a greater number of exercises. Overall, there was 5 exercises in the training programme – 2 for warm-up and scapular control (shoulder shrug and scapular retraction), 1 stretching exercise (upper trapezius), while the 2 main exercises were eccentric exercise of the supraspinatus and infraspinatus in side-lying. Interestingly, the frequency parameters used above by Jonsson *et al* (2006) were also used in this study. This training programme was found to be effective in reducing pain and increasing function. 8/10 subjects had significantly reduced pain intensity levels as measured by the VAS in relation to their baseline score. Intriguingly, the mean VAS change of this study was 30 mm, while Jonsson *et al* (2006) noted a similar change with a 31 mm difference pre and post the eccentric training. These values are translated into a more clinical context in that previous research has established 14 mm as a minimal clinically important difference (MCID) in patients with rotator cuff disease (Tashjian *et al* 2009). Function as measured by both the Patient-Specific Functional Scale and the Constant-Murley score was found to have increased significantly in all patients.

Camargo *et al* (2012) used an isokinetic dynamometer to evaluate and provide an eccentric training programme to the shoulder abductors in patients with shoulder impingement. This was performed at 60°/s and the range of motion (ROM) trained was 60° → 80° to 20°. The training was completed twice per week for 6 weeks with 3 sets of 10 repetitions performed on each training day with a 3 minute rest interval between each set. The Disabilities of the Arm, Shoulder and Hand (DASH) Questionnaire evaluated functional status and symptoms and was found to have significantly lower values immediately after commencement of the 6 week programme and this was further maintained at follow up 6 weeks later. Practicality issues around this type of exercise arise however as the researchers used an isokinetic device to both evaluate baseline but to also deliver the actual intervention. Problems could potentially arise in incorporating this into a clinical setting.

One obvious limitation from the 3 studies outlined above is that no control group was used in any of the studies. Bernhardsson *et al* 2011 made an attempt to introduce a control. A single-subject experimental design was used with a baseline period of 3 weeks followed by the 12

week treatment period. As such each subject's baseline period acted as a control. This is someway improved in that the replication of the design across all the individuals of the study (n=10) enhanced the generalizability of the results. The lack of a clearly identified control group does however prevent the researchers from ruling out the influence of natural recovery on the results, however unlikely it may be due to the chronicity of the subjects included.

However, more recent studies have utilised a randomised control trial (RCT) methodology to examine the effect of eccentric exercises in rotator cuff tendinopathy.

Maenhout *et al* (2013) examined if there was a superior value to treatment of rotator cuff tendinopathy if heavy load eccentric training was added to conservative treatment. The control group completed the traditional rotator cuff training which consisted of 2 exercises - both internal and external rotation resisted with a theraband. Each exercise was performed only once a day for 3 sets of 10 repetitions. These exercises were performed for a count of 6 seconds, 2 seconds each for the concentric, isometric and eccentric phases of the exercise. Load was increased by changing colour of the band as pain permitted. The exercise group completed the same exercises while also completing the heavy load eccentric exercise which was the eccentric phase of 'full can' abduction in the scapular plane with a weight. This was performed at a speed of 5 seconds for 3 sets of 15 repetitions. This was repeated twice daily. Again this was the same frequency parameters used by both Jonsson *et al* 2006 and Bernhardsson *et al* 2011. On top of these home exercises, both groups received 9 physiotherapy sessions over the course of the 12 week intervention. The results showed that isometric strength at 90° abduction was the only significant difference between both groups upon completion of the intervention, with the heavy load eccentric group scoring significantly higher over the control group. However, both groups did have equally significant changes for decreased pain and improved function as measured by the Shoulder Pain and Disability Index (SPADI) but the eccentric group was not superior to the control for these outcomes. Subsequently, it can be concluded from this study that there was no significant benefit in adding heavy eccentric loading to a standard theraband -resisted internal and external rotation training programme for subacromial impingement syndrome (Maenhout *et al* 2013).

This study by Maenhout *et al* (2013) scored 6/10 on the PEDro scale and failed to score on concealed allocation and failed to blind subjects, therapists and assessors. As a result, performance bias may be relevant as the therapist and investigators expectations may have influenced results

Another RCT was carried out by Holmgren *et al* (2012) evaluating the effect of 12 weeks of strengthening eccentric exercises of the rotator cuff and concentric or eccentric exercise of the scapula stabilisers on the need for surgery in patients with SAIS. The programme consisted of six different exercises – 2 eccentric exercises for the rotator cuff, 3 concentric/eccentric for the scapular stabilisers and a posterior capsule stretch. See Table 3.1 for details on the frequency parameters. The control group carried out 6 unspecific exercises without an external load and these exercises weren't progressed or modified throughout the whole rehabilitation period. In addition, both groups received a subacromial corticosteroid injection two weeks before starting the exercise programme and 7 physiotherapy sessions over the 12 weeks.

The exercise group that included the use of eccentric exercises had significantly greater improvement in pain and function, as evaluated by both the Constant-Murley score and DASH, than the control. A significantly lower proportion of patients in the exercise group chose to ultimately undergo surgery – 20% vs 63%.

The results of this RCT are much more favourable than that of Maenhout *et al* 2012. However, it must be noted that all subjects in both studies did receive physiotherapy treatment concurrently over the 12 weeks making it difficult to fully attribute all the changes in the measures used to that of the exercises. However, the authors make the point that offering a variety of treatments best represent current practice in physiotherapists (Littlewood *et al* 2012) making their intervention more transferrable and applicable to clinical practice.

Table 3.1: Summary of eccentric training exercises for rotator cuff tendinopathy.

Study	Inclusion Criteria	Sample Size	Intervention	Length of Intevention	Frequency parameters
Bernhardsson *et al* (2011)	Shoulder pain > 30mm on VAS for 3+ months; Age:18-65; 3+/5 +ve: Neer, Jobe, Hawkins, Painful arc, TOP of supraspinatus	n = 10	5 exercises - 2 warm-up (shoulder shrug and scapular retraction), upper trapezius stretch, eccentric strengthening of supraspinatus and infraspinatus side-lying	12 weeks	3 sets of 15 repetitions; Twice daily; 7 days per week
Camargo *et al* (2012)	Pain on 3 of Neer, Hawkins, Jobe, Speed; Painful ROM during shoulder elevation;	n = 20	Isokinetic dynamometer at 60°/s with ROM trained from 80° to 20°	6 weeks	3 sets of 10 repetitions; Once daily 2 days per week.
Holmgren *et al* (2012)	Age: 30-65; Pain on proximal lateral aspect of the upper arm > 3months; 3 or more +ve: Neer, Hawkins, Jobes, Patte's manoeuvre	n = 97 (control = 46; eccentric exercise = 51)	Control: 6 unspecific neck and shoulder exercises with no external load Eccentric: 2 eccentric exercises for rotator cuff, 3 concentric/eccentric exercises for scapula stabilisers, and posterior shoulder stretch	12 weeks	Control: each exercises repeated 10 times and each stretching exercise repeated 3 times twice daily Eccentric: Weeks 1-8 – twice daily strengthening – 3 sets of 15 repetitions stretch - 30-60s 3 times Week 9-12 – as above but once daily
Jonsson *et al* (2006)	+ve Hawkins; +ve Neer; On waiting list for surgery (conservative Rx failed)	n = 9	'Empty Can' using sling + progression using weight.	12 weeks	3 sets of 15 repetitions; Twice daily; 7 days per week
Maenhout *et al* (2013)	18 years +; Unilateral shoulder pain > 3 months; Painful arc; 2/3 +ve tests: Hawkins, Jobe, Neer; 2/4 painful resisted tests – 'full can', resisted abduction @ 90°, resisted internal and external rotation	n = 61 (control = 30; heavy load eccentric = 31)	Control: Internal and External rotation with theraband Eccentric: as above plus 'Full can' + weight. repetitions	12 weeks	Control exercises: 3 sets of 10 repetitions; Once daily – each repetition lasts 6 seconds. Eccentric exercise: Each repetition lasts 5 seconds 3 sets of 15 repetitions; Twice daily; 7 days per week

Key References
• Littlewood, C., May, S. and Walters, S. (2013) 'A review of systematic reviews of the effectiveness of conservative interventions for rotator cuff tendinopathy', *Shoulder & Elbow*, 5(3), 151-167.

Littlewood et al (2013) carried out a review of the systematic reviews which examined the effectiveness of conservative interventions for rotator cuff tendinopathy.

Below are the main findings from each of the conservative treatment which were examined.

Exercise combined with Manual Therapy for Rotator Cuff Tendinopathy

Littlewood *et al* (2013) found 11 systematic reviews relating to the effectiveness of exercise combined with manual therapy for rotator cuff tendinopathy. The results of these 11 systematic reviews were based on 5 primary studies (Bang and Deyle 2000, Citaker *et al* 2005, Cloke *et al* 2008, Conroy and Hayes 1998, Senbursa *et al* 2007). These 5 studies were of poor to moderate quality however so the conclusions drawn from the systematic reviews may need to be taken with caution. The methodological quality was scored on PEDro as – 3/10 (Citaker *et al* 2005), 4/10 (Senbursa *et al* 2007), 5/10 (Cloke *et al* 2008), 6/10 (Bang and Deyle 2000; Conroy and Hayes 1998).

Results from Conroy and Doyle (1998) and Bang and Deyle (2000) led researchers to conclude that exercise combined with manual therapy improved significantly in terms of pain, function and ROM in the short term compared to exercise alone (Green *et al* 2003, Desmules *et al* 2003, Michener *et al* 2004, Faber *et al* 2006).

However, more recent evidence has begun to question the advocacy or additional benefit of manual therapy with exercise. The evidence shows that active interventions like manual therapy, as well as multimodal physiotherapy and surgery confer no additional benefit than exercise alone (Littlewood *et al* 2013).

Table 3.2: Exercise combined with Manual Therapy for rotator cuff tendinopathy – Adapted from Littlewood et al. (2013)

Study	Population	Main findings	AMSTAR score
Green et al (2003)	Rotator cuff tendinitis	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	9/11
Desmeules et al (2003)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	5/11
Michener et al (2004)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	7/11
Trampas and Kitsios (2006)	Subacromial impingement syndrome	Evidence shows no significant difference in terms of pain or function compared to PNF with exercise	6/11
Faber et al (2006)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	5/11
Ho et al (2009)	Subacromial impingement syndrome	Conflicting evidence regarding short-term effectiveness compared to exercise alone	3/11
Kuhn et al (2009)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	4/11
Kromer et al (2009)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	5/11
Nyberg et al (2010)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	4/11
Braun and Hanchard (2010)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on improving pain, function and ROM compared to exercise alone	7/11
Kelly et al (2010)	Subacromial impingement syndrome	Conflicting evidence regarding effectiveness compared to exercise alone	6/11

Multimodal Physiotherapy for Rotator Cuff Tendinopathy

Due to the lack of high quality evidence on which to base practice, there is huge variability in treatments offered by physiotherapists for rotator cuff tendinopathy (Littlewood *et al* 2012). A survey of UK physiotherapists showed that treatment ranged from advice/education, stretching, strengthening, manual therapy, electrotherapy and corticosteroid injections and various combinations of these. While this multi-modal approach clearly reflects clinical practice, is this approach effective?

Littlewood *et al* (2013) found 7 systematic reviews which related to the effectiveness of multimodal physiotherapy for rotator cuff tendinopathy. The quality of these systematic reviews ranged from poor to moderate quality scoring 4-7/11 on the AMSTAR.
Results found that compared to no treatment or placebo, evidence supports the effectiveness of multimodal physiotherapy in the medium-long term. However, the short term effectiveness is not statistically significant.

Table 3.3: Effectiveness of Multimodal Physiotherapy for rotator cuff tendinopathy – Adapted from Littlewood et al. (2013)

Study	Population	Main findings	AMSTAR score
Kuhn et al (2009)	Rotator cuff impingement	Evidence finds no significant difference compared to surgery	4/11
Dorrestijin et al (2009)	Subacromial impingement syndrome	Evidence finds no significant difference compared to surgery	5/11
Kromer et al (2009)	Subacromial impingement syndrome	Evidence supports effectiveness in the medium-term on pain and function compared to no intervention	5/11
Nyberg et al (2010)	Subacromial impingement syndrome	Evidence supports effectiveness in the medium-term on pain and function compared to no intervention	4/11
Braun and Hanchard (2010)	Subacromial impingement syndrome	Evidence supports effectiveness in the medium-term on pain and function compared to no intervention	7/11
Brantingham et al (2011)	Rotator cuff disorders	Evidence supports effectiveness	4/11
Hanratty et al (2012)	Subacromial impingement syndrome	Evidence supports short-term effectiveness on pain and function and long-term on function.	6/11

Corticosteroid Injection for Rotator Cuff Tendinopathy

Corticosteroids are a class of medication that are related to cortisone and is used reduce inflammation. While corticosteroids can be taken either orally, inhaled or intravenously, corticosteroid injections are commonly used around the shoulder joint in conjunction with an anaesthetic (Garnham 2003). Littlewood *et al* (2013) found 6 systematic reviews that related to the effectiveness of corticosteroid injections for rotator cuff tendinopathy. The systematic reviews were of variable quality with scores on the AMSTAR ranging from 5-9/11.

Evidence from high quality studies which scored 8 or 9/11 (Arroll and Goodyear-Smith (2005), Buchbinder *et al* (2003), Green *et al* (1998), Green *et al* (2003)) are supportive of the short-term effectiveness of corticosteroid injections in reducing pain, increasing function and ROM compared to placebo. Furthermore, a lower quality systematic review by Johansson *et al* (2002) had similar findings. However, although of lower quality than the other systematic review, the most recent systematic review by Kroester *et al* (2007) had conflicting results on effectiveness of corticosteroid injections on pain, function or ROM. A conclusion was drawn that absence of a clear consensus and more evidence is needed before a definitive judgement on corticosteroid injections for rotator cuff tendinopathy could be made.

Table 3.4: Effectiveness of Corticosteroid Injections for rotator cuff tendinopathy – Adapted from Littlewood et al. (2013)

Study	Population	Main findings	AMSTAR
Green et al (1998)	Rotator cuff tendinitis	Evidence is supportive of short-term effectiveness for improving ROM compared to placebo Evidence does not support short-term effectiveness for reducing pain compared to placebo	9/11
Johansson et al (2002)	Subacromial impingement syndrome	Evidence supports short and long-term effectiveness for improving pain and ROM compared to injection of local anaesthetic	5/11
Buchbinder et al (2003)	Rotator cuff disease	Evidence is supportive of short-term effectiveness for improving pain, function and ROM compared to placebo	9/11
Green et al (2003)	Rotator cuff tendinitis	Evidence supports its effectiveness compared to physiotherapy interventions	9/11
Arroll and Goodyear-Smith 2005)	Rotator cuff tendinitis	Evidence supports short and long-term effectiveness for improving pain and ROM compared to placebo	8/11
Koester et al (2007)	Rotator cuff disease	Conflicting evidence on effectiveness for pain, function and ROM compared to placebo	5/11

Laser Therapy for Rotator Cuff Tendinopathy

Laser therapy is a relatively new therapeutic modality and is not commonly used for treatment of rotator cuff tendinopathy.

LASER: Laser therapy is the Amplification of light by Stimulated Emission of Radiation. Theoretically, laser energy is transmitted to induce cell proliferation and stimulate cellular function (Watson 2013). In studies that have examined laser therapy, the average intensity and duration of treatment programs appears to be 20–30 minutes of low intensity laser therapy five times a week for two to three weeks.

Littlewood *et al* (2013) found 6 systematic reviews which related to the effectiveness of laser therapy for rotator cuff tendinopathy. The quality ranged from 4-9/11 on the AMSTAR. There was agreement among 5 of the reviews that the evidence didn't support the effectiveness of laser therapy (Green *et al* 2003, Grant *et al* 2004, Faber *et al* 2006, Kromer *et al* 2009, Tumilty *et al* 2010). Nyberg *et al* (2010) found support for the effectiveness of high-intensity laser from one RCT. However an overall conclusion that laser therapy has a lack of evidence to be used clinically was made due to the fact that the predominant lack of positive findings clearly outweighed the results of one RCT.

Table 3.5: Effectiveness of Laser therapy for rotator cuff tendinopathy – Adapted from Littelwood et al. (2013)

Study	Population	Main findings	AMSTAR score
Green et al (2003)	Rotator cuff tendinitis	Evidence does not support effectiveness compared to placebo	9/11
Grant et al (2004)	Rotator cuff pathology	Evidence does not support effectiveness compared to placebo	5/11
Faber et al (2006)	Subacromial impingement syndrome	Evidence does not support short-term effectiveness for function compared to placebo	5/11
Kromer et al (2009)	Subacromial impingement syndrome	Conflicting evidence regarding short-term effectiveness compared to placebo	5/11
Nyberg et al (2010)	Subacromial impingement syndrome	Evidence supports the effectiveness of high-intensity laser for pain compared to ultrasound	4/11
Tumilty et al (2010)	Rotator cuff tendinopathy	Conflicting evidence regarding effectiveness	5/11

Therapeutic Ultrasound for Rotator Cuff Tendinopathy

Therapeutic ultrasound (US) is a form of mechanical (vibration) energy which is used as a method of stimulating the tissue beneath the skin's surface via very high frequency sound waves, typically between 800,000 Hz and 2,000,000 Hz (Watson 2013). The normal human sound range is from 16 Hz to around 15000-20000 Hz with anything upwards of this is referred to as ultrasound (Watson 2013). Ultrasound is used as a physiotherapy treatment for its physiological effects which include argumentation of blood flow, increased capillary permeability and tissue metabolism, enhancement of tissue extensibility, elevation of pain threshold and alteration of neuromuscular activity leading to muscle relaxation (Downing and Weinstein 1986).

Littlewood *et al* (2013) found 5 systematic reviews (Johansson *et al* 2002, Green *et al* 2003, Michener *et al* 2004, Faber *et al* 2006, Kromer *et al* 2009) relating to the effectiveness of ultrasound for Rotator Cuff Tendinopathy. Based on the AMSTAR, the quality of the systematic reviews varied from 5-9/11. Nevertheless, all 5 systematic reviews consistently concluded that the evidence does not support the use or effectiveness of therapeutic ultrasound.

Table 3.6: Effectiveness of Ultrasound for rotator cuff tendinopathy – Adapted from Littlewood et al. (2013)

Study	Population	Main findings	AMSTAR score
Johansson et al (2002)	Subacromial impingement syndrome	Evidence does not support effectiveness for pain or ROM compared to placebo	5/11
Green et al (2003)	Rotator cuff tendinitis	Evidence does not support effectiveness for pain or ROM compared to placebo	9/11
Michener et al (2004)	Subacromial impingement syndrome	Evidence does not support effectiveness for pain or ROM compared to placebo	7/11
Faber et al (2006)	Subacromial impingement syndrome	Evidence does not support short-term effectiveness for functional limitations compared to placebo	5/11
Kromer et al (2009)	Subacromial impingement syndrome	Evidence does not support effectiveness for pain or ROM compared to placebo or acupuncture	5/11

Pulsed Electromagnetic Energy for Rotator Cuff Tendinopathy

Pulsed Electromagnetic Energy (PEME)/Pulsed Electromagnetic Field (PEMF) therapy uses electrical energy to direct a series of magnetic pulses through injured tissues. It is believed that these pulses can help to improve vascularisation and stimulate cellular repair. Littlewood *et al* (2013) found 4 systematic reviews (3 of which only looked at the same study from 1984) relating to the effectiveness of pulsed electromagnetic energy for rotator cuff tendinopathy. These systematic reviews (Green *et al* 2003, Grant *et al* 2004, Kromer *et al* 2009, Nyberg *et al* 2010) ranged from 4-9/11 on the AMSTAR. Evidence supporting its use is conflicting with some early reviews supporting short-term effectiveness compared to placebo. However, later reviews showed conflicting evidence in that it was no more effective than placebo for improving pain and function. Subsequently, it was concluded that the evidence of Pulsed Electromagnetic Energy for Rotator Cuff Tendinopathy is non-supportive of effectiveness.

Table 3.7: Effectiveness of Pulsed Electromagnetic Energy for rotator cuff tendinopathy – Adapted from Littlewood et al. (2013)

Study	Population	Main findings	AMSTAR score
Green et al (2003)	Rotator cuff tendinitis	Evidence is supportive of short-term effectiveness compared to placebo	9/11
Grant et al (2004)	Rotator cuff pathology	Evidence is supportive of short-term effectiveness compared to placebo	5/11
Kromer et al (2009)	Subacromial impingement syndrome	Conflicting evidence regarding short-term effectiveness compared to placebo	5/11
Nyberg et al (2010)	Subacromial impingement syndrome	Evidence not supportive of effectiveness	4/11

Extracorporeal Shock Wave Therapy for Rotator Cuff Tendinopathy

KEY RESOURCE

Santry Sports Surgery Clinic Information Video - Shockwave Therapy or ESWT. Explained: https://www.youtube.com/watch?v=Fq5yqiWByX4

Extracorporeal Shock Wave Therapy (ESWT) is a method of treating tendinopathies in which a shockwave is delivered to the tissue via a compressed air impulse exerted by the hand piece (Watson 2013). Originally pioneered as a non-invasive treatment for the physical destruction of kidney stones, ESWT has been used for many tendinopathies including the rotator cuff.

Many variables are associated with this therapy, such as the type of shock wave generator (electrohydraulic, electromagnetic or piezoelectric), type of wave (radial or focal), intensity (total energy per shock wave/per session), frequency of the shock waves and the protocol and application and repetitions (no. of shocks). All these variables makes comparisons between studies quite difficult and contributes to a lack of agreement on this type of therapy (Kaux *et al* 2011).

The shockwave enters the tissue and radiates out and dissipates the energy of the shockwave which is thought to be responsible for the generation of physiological effects and stimulation of cellular activity which is thought to cause inhibition of nocioceptors and thus provide an analgesic effect (Kaux *et al* 2011).

Littlewood *et al* (2013) found 4 systematic reviews which related to the effectiveness of ESWT for rotator cuff tendinopathy. According to the AMSTAR, all 4 systematic reviews scored 5/11. As well as scoring similar for quality, all 4 reviews consistently agreed that the evidence didn't support the effectiveness of ESWT compared to placebo for rotator cuff tendinopathy.

Table 3.8: Effectiveness of Extracorporeal Shockwave Therapy (ESWT) for rotator cuff tendinopathy - Adapted from Littlewood et al. (2013).

Study	Population	Main findings	AMSTAR score
Grant et al (2004)	Rotator cuff pathology	Evidence is not supportive of effectiveness against placebo	5/11
Harniman et al (2004)	Rotator cuff tendinitis	Evidence is not supportive of effectiveness in terms of pain, function or ROM compared to placebo	5/11
Faber et al (2006)	Subacromial impingement syndrome	Evidence is not supportive of effectiveness at any time point (short-term, medium-term or long-term) on functional limitations compared to placebo	5/11
Huisstede et al (2011)	Rotator cuff tendinosis	Evidence is not supportive of effectiveness against placebo or other interventions	5/11

Table 3.9: Effectiveness of Acupuncture for rotator cuff tendinopathy – Adapted from Littlewood et al. (2013)

Study	Population	Main findings	AMSTAR score
Johansson et al (2005)	Subacromial pain	Evidence supportive of short-term effectiveness in terms of reducing pain and improving function compared to placebo	5/11
Grant et al (2004)	Rotator cuff pathology	Evidence supportive of short-term effectiveness in terms of reducing pain and improving function compared to placebo	5/11
Michener et al (2004)	Subacromial impingment syndrome	Evidence is conflicting in the short-term and not supportive of effectiveness in the mid-term for improving pain, function and ROM compared to placebo	7/11
Green et al (2003)	Rotator cuff disease	Evidence is conflicting in the short-term and not supportive of effectiveness in the mid-term for improving pain, function and ROM compared to placebo No significant difference compared to corticosteroid injection for pain or ROM No significant difference compared to ultrasound for pain or ROM	9/11
Trampas and Kitsios (2006)	Subacromial impingement syndrome	Evidence is supportive of effectiveness for improving pain compared to ultrasound	6/11
Nyberg et al (2010)	Subacromial impingement syndrome	Conflicting evidence compared to placebo for improving pain and function	4/11

Acupuncture for Rotator Cuff Tendinopathy

Acupuncture is an intervention involving the insertion and manipulation of fine needles into the body to stimulate specific neurochemical and both connective and contractile tissue responses to achieve a therapeutic effect (Bannan 2013).

Littlewood *et al* (2013) found 6 systematic reviews related to the effectiveness of acupuncture for rotator cuff tendinopathy. The quality of the systematic reviews varied from 4-9/11 on the AMSTAR. Initial reviews by Johansson *et al* (2002) and Grant *et al* (2004) both supported the short-term effectiveness of acupuncture compared to placebo only. This conclusion was based on 1 RCT by Kleinhenz *et al* (1999) in which patients either received 4 weeks of acupuncture or a sham treatment. However, the ability of the researchers to be able to successfully deliver a sham acupuncture treatment may be dubious as all the participants of the control arm of the study may not have been fully blinded to treatment.

Medium term effectiveness of acupuncture is not supported by the later reviews (Michener *et al* 2004, Green *et al* 2005, Trampas and Kitsios 2006, Nyberg *et al* 2010) and neither is effectiveness compared to active interventions including ultrasound or corticosteroid injections. However, these have already been shown to be poorly effective treatments themselves as discussed previously. One study found that acupuncture combined with exercise is more beneficial compared to ultrasound (Johansson *et al* 2005). However, considering the positive findings shown by exercise as seen above, it is difficult to ascertain and ascribe any of this treatment effect to acupuncture.

Platelet Rich Plasma for Rotator Cuff Tendinopathy

Another conservative intervention which is gaining increasing popularity recently is Platelet-Rich Plasma (PRP) injections. PRP is what the name suggests: blood plasma that has been enriched with platelets. Platelets participate in the body's natural response to injury and once activated by mediators at the site of injury, the platelets undergo degranulation and release bioactive proteins or growth factors that aid in wound-healing. These PRP injections involve injecting someone's own concentrated platelets, protein-releasing bodies and other healing growth factors into a damaged tendon. The intention behind PRP is to provide the tendon with these healing growth factors to stimulate healing of bone and soft tissue. With respect to tendons, some evidence has shown that PRP treatment may in fact enhance tendon healing due to increasing the number of tenocytes and increased production of Type I and Type III collagen (Zhang 2010).

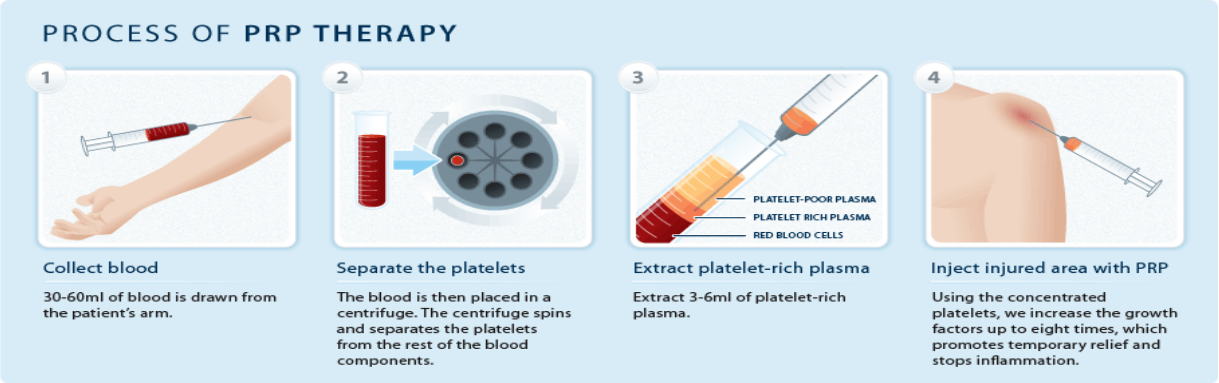


Fig 3.3: Process of Platelet-Rich Plasma Therapy/Injections

KEY RESOURCE

Santry Sports Surgery Clinic Information Video – All you need to know about Platelet Rich Plasma (PRP) Muscle Injection: https://www.youtube.com/watch?v=CasixaimHug

Evidence?

A prospective study by Scarpone *et al* (2013) evaluated pain and function in 18 subjects who received 1 ultrasound-guided PRP injection. The mean VAS at baseline was 7.5±0.3 and this dropped to 0.5±0.3 after 12 weeks and 0.4±0.2 after 52 weeks. Furthermore, 17/18 participants were "completely satisfied" or "satisfied" with their functional status after 12 weeks. However, long-term follow up score for 'satisfaction' after 52 weeks is not recorded.

While, these results are promising due to significant improvement in pain levels and functional status, the lack of a control group severely impact the credibility of these results.

However, Kesikburun *et al* (2013) did use a control group to compare the effect of PRP injections vs placebo on pain and function in rotator cuff tendinopathy. 20 subjects received the PRP injection while 20 control subjects received a saline solution injection using real-time ultrasound for guidance. All patients then completed a 6 week rehabilitation exercise program.

The Western Ontario Rotator Cuff Index (WORC) was used as the primary outcome measure The SPADI, pain on Neer's Impingment sign (VAS) and passive shoulder ROM were secondary outcome measures used.

In contrast to the previous study, the authors here found that comparisons between groups revealed no significant difference between the groups in WORC, SPADI and VAS at 1 year follow up. Within group analysis showed significant improvements compared to baseline in all outcome measures but the authors ultimately concluded that PRP was no more effective than placebo.

KEY RESOURCE

UW Health Sports Rehabilitation - Platelet-Rich Plasma Rehabilitation Guidelines: http://www.uwhealth.org/files/uwhealth/docs/sportsmed/sports_med_PRP.pdf

Efficacy of Surgery for Rotator Cuff Tendinopathy

The decision to undergo surgery is indicated in patients where conservative treatments have failed to improve on clinical outcomes (Boykin *et al* 2010). The financial impact surgery has is huge with approximately 40,000 surgeries performed in the US for rotator cuff problems at a cost of approximately $14,000 per surgery in 2002 (Oh *et al* 2007).

Many surgical techniques exist.

- **Acromioplasty**: which is also referred to as "subacromial decompression" is one such surgical technique whereby the anterior edge and the under surface of the anterior part of the acromion are removed.
- **Bursectomy**: involves removal of the subacromial bursa and can be performed with or without acromioplasty. This is some surgeons preferred technique as the coracoacromial arch is not altered as a result (Donigan and Wolf 2011).
- **Radiofrequency (RF) based microtenotomy**: a relatively new surgical procedure whereby a radiofrequency based probe is used to perform microdebridement of the supraspinatus tendon (Toliopoulos *et al* 2014).

These techniques have evolved over the years from open procedures to arthroscopic. This helps to avoid wide dissection and subsequent large scarring as a result of open procedures and has been attributed to quicker operating times, improved post-operative mobility and less complications, such as infection (Toliopoulos *et al* 2014).

Efficacy of surgery vs exercise in the management of rotator cuff tendinopathy

Toliopoulos *et al* (2014) carried out a systematic review which examined efficacy of surgery in rotator cuff tendinopathy and found moderate evidence from 4 RCTs that acromioplasty is not superior to exercise in the treatment of rotator cuff tendinopathy.

Haahr *et al* (2005) found that the arthroscopic subacromial decompression group had an increase of 15.5% and 19.9% at 6 and 12 months respectively on the Constant-Murley score, while the exercise group had increases of 20.1% and 21.3% at 6 and 12 months respectively. These differences between the 2 treatment groups were not statistically significant.

Ketola *et al* (2009) also concluded that acromioplaty combined with exercise was no more effective than exercise alone in the long-term. Their 2-year follow-up showed that changes in

VAS pain scale were not significantly different with a mean change of -3.7/10 for the exercise group and a mean change of -3.9/10 for the surgery group.

Brox *et al* (1999) compared a supervised exercise programme to arthroscopic subacromial decompression and to a placebo also which involved detuned laser treatment. It was found that both the supervised exercise and surgery demonstrated superior outcomes to the placebo, while there was no significant difference between these 2 treatment on the Neer score at follow-ups of 6 months and 2 ½ years.

While all 3 of the above studies used different outcome measures making it difficult to compare studies, it is evident that all 3 studies conclude that surgery holds no additional benefit than exercise alone.

One study by Rahme *et al* (1998) did conclude in favour of surgery due to a higher proportion of subjects in the surgery group showing >50% reduction in pain scores at both 6 months and 1 year. However, this is a non-validated outcome measure and limits the conclusions that can be drawn from these results.

Outcome Measures

Constant-Murley Score (CMS)

The CMS is a 100-point scale consisting of 4 subscales which help to determine the level of pain and functionality of a patient due to a shoulder injury (Roy et al. 2010). The higher the score, the higher function of the individual (Constant et al. 2008)

The CMS is broken down as such:

Subscale	Score
Pain	15
Activities of Daily Living	20
Range of Motion	40
Strength	25
Total	*100*

Table 3.10: Constant Murley Score (Constant et al 2008)

Roy et al. (2012) conducted a systematic review to review the psychometric evidence of the CMS. They found that the overall inter-rater reliability of the CMS was excellent (ICC: 0.89; 95% CI: 0.79-0.94). Construct validity was established with strong correlations associated with other shoulder outcome measures (Roy et al.2012). Responsiveness was confirmed also (Roy et al. 2012).

Kukkonen et al. (2013) reported a minimal clinically important difference (MCID) of 10 points on the CMS. The MCID is the smallest change in status on the outcome measure which is considered to be clinically relevant (Littlewood et al. 2012).

Please see Appendix (Online) for a link to the CMS.

Western Ontario Rotator Cuff Index

The Western Ontario Rotator Cuff (WORC) Index is a condition-specific, self-report questionnaire developed by Kirkley et al. (2003) used to assess Quality of Life (QoL) in patients with shoulder complaints as a result of Rotator Cuff tendinopathy.

It consists of 21 questions which relate to five different domains of shoulder dysfunction: Physical symptoms, Sports/Recreation, Work, Lifestyle and Emotional well-being (de Witte et al. 2012). Each question is scored from 0-100 on 100mm visual analogue scale, with each question scored as a percentage.

De Witte et al. (2012) examined the psychometric properties of the WORC and found high reliability and good validity and responsiveness when tested on people with Rotator Cuff tendinopathy. The WORC is shown to have a minimal detectable change (MDC) of 7.1 (Lopes et al. 2008). An MCID of 12 points has been reported (Kirkley et al. 2003).

One limitation observed by the authors was that the WORC failed to discriminate as well as the DASH score between individuals with severe and less severe shoulder symptoms (de Witte et al. 2012). However, a major strength of the WORC is it takes account of ADLs, mental health and QOL dimensions and not just physical findings (de Witte et al. 2012).
Please see Appendix (Online) for a link to the WORC Index with instructions on its completion and scoring.

Shoulder Pain and Disability Index (SPADI)

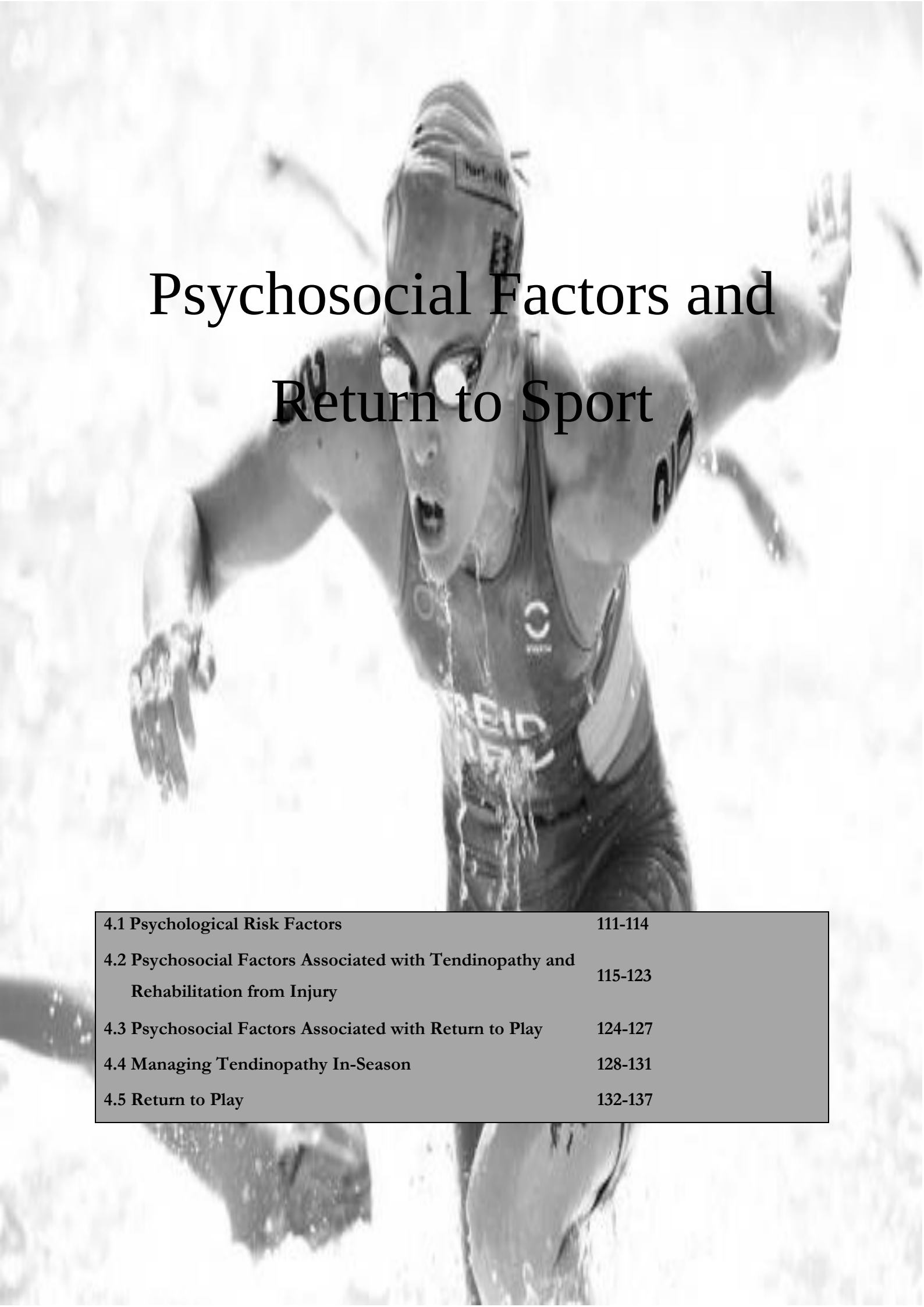
The SPADI is used to measure shoulder pain and disability. It consists of 13 questions separated into 2 domains: Pain (5 items) and Disability (8 items). Each item is scored from 0-10 on a numerical rating scale (NRS), and has been found easier to administer and score than the earlier VAS version (Williams et al. 1995). It takes <5 minutes to complete the SPADI (Williams et al. 1995).
One item omitted is allowed, however anymore and the no score is calculated (Roach et al. 1991).

Roy et al. (2009) found that the SPADI has reliability coefficients of ICC ≥ 0.89. Internal consistency has shown a similarly high value with Cronbach α > 0.90 (Roy et al. 2009, Hill et al. 2011). Paul et al. (2004) and Roy et al. (2009) confirmed good construct validity, correlating with other shoulder outcome measures such as the DASH. The SPADI has been shown good responsiveness and can adequately discriminate between improving patients and those who are deteriorating (Williams et al. 1995, Roy et al. 2009). Finally, a MCID of 8 points has been reported (Paul et al. 2004).

Please see Appendix (Online) for a link to the SPADI and its instructions and scoring.

Disabilities of the Arm, Shoulder and Hand (DASH)

The DASH questionnaire has been demonstrated to perform consistently well in relation to reliability, validity, responsiveness to change (Haldorsen *et al.* 2014). It is a 30-item, self-report questionnaire designed to measure physical function and symptoms in patients with disorders of the shoulder, elbow, wrist and hand and takes approximately 3 – 4 minutes to complete. The DASH Outcome Measure was jointly developed by the Institute for Work & Health in Ontario and the American Academy of Orthopaedic Surgeons (AAOS). It is suitable for a broad range of shoulder pain presentations, including rotator cuff disorders, shoulder osteoarthritis, shoulder fractures, and frozen shoulder. The DASH outcome measure has been specifically validated for use with rotator cuff disorders (Huang *et al.* 2015).



Psychosocial Factors and Return to Sport

Psychological Factors as Risk Factors for Sports Injury

While the mechanical and physiological factors are more commonly identified as risk factors for sports injury, there is significant evidence to demonstrate that psychological factors are also implicated in their etiology (Wiese-Bjornstal 2010). Knowledge of psychological predictors of injury in elite sport is far from comprehensive, and findings to date have been somewhat contradictory (Galambos *et al.* 2005). A number of models have been created which emphasize a relationship between psychological risk factors and injury occurrence (Ivarsson and Johnson 2010). Among the most influential is the stress-injury model outlined by Williams and Andersen (1998).

This model proposes that certain psychological factors can predispose an individual to become more stressed when exposed to pressure. Increased stress can in turn lead to physiological and psychological changes that can increase the athlete's risk of injury. The model proposes to divide psychological risk factors into three main categories: personality factors; history of stressors; and coping resources. Figure 4.1 demonstrates the interplay between these factors and stress responses which can lead to injury.

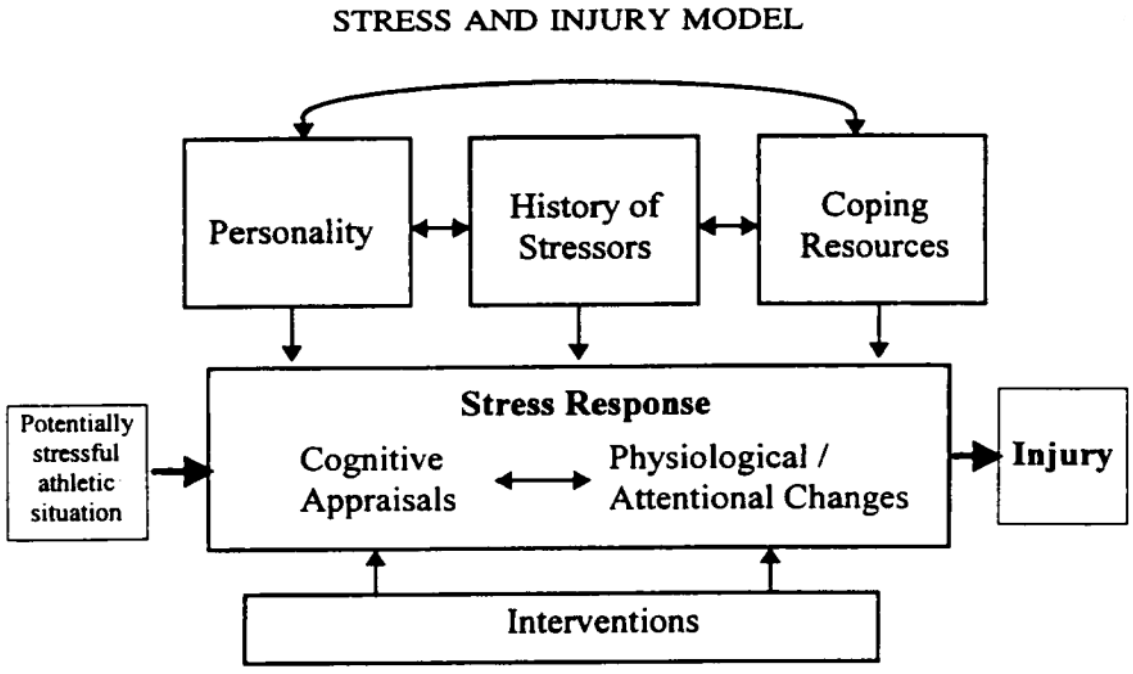


Fig 4.1: Stress and injury model as outlined by Williams and Andersen (1998) demonstrating the relationship between psychological risk factors and injury occurrence.

Personality Factors

A study by (Johnson 2007) provided an overview of the stress and injury model with regard to psychological antecedents to injury and discussed the variables involved for each category of factors. For personality, the author identified the following characteristics that have all demonstrated relationships with increased stress response and subsequent injury risk:

- ***Locus of Control:*** Injuries positively correlated with external locus of control
- ***Competitive Trait Anxiety (CTA):*** i.e. perceiving competitive situation as threatening. Positive relationship between high CTA and high injury rate and severity of injuries
- ***Perfectionism:*** Related to increased injury risk
- ***Mood Status*****:** Athletes with a positive state of mind early in season incurred few injuries compared to athletes with a less positive mind
- ***Self-Confidence/Esteem:*** Low self-esteem positively correlated with injury

History of Stressors Factors

The history of stressors category is composed of daily hassles and life event stress. Whilst it is unclear whether daily hassles such as being late or unexpected arguments have an effect in this model, life event stress does appear to be influential. Suggested examples of potentially stressful life events are moving city, divorce, illness of family member etc. The memory of a previous injury and the stress this may cause for the athlete would also fall into this category (Johnson 2007)

Coping Factors

A study by Ivarsson and Johnson (2010) set out to examine the relationship between these psychological factors and injury risk in forty-eight male soccer players with a mean age of twenty-two. They found that a number of psychological factors, such as high stress levels and ineffective coping could increase the injury risk among athletes. Regarding coping factors, the final category of factors outlined by Williams and Andersen (1998), they found that the two coping factors, self-blame and acceptance could together explain 14.6 % of injury occurrence.

Interventions to Target Psychological Risk Factors

A study by Maddison and Prapavessis (2005) involved 470 rugby players who completed measures corresponding to variables in the Williams and Andersen (1998) stress and injury model. They found that social support, the type of coping, previous injury, and competitive anxiety interacted together to maximize life stress and increase risk of injury. In addition, it was investigated if those identified as at risk of injury would benefit from a cognitive behavioural stress management (CBSM) programme targeting the modifiable psychological risk factors through education. The results supported a reduction in injury vulnerability for those who completed a six session CBSM intervention. Specifically, the amount of time missed due to injury was reduced in the intervention group compared to the control group. A similar pattern of results was shown for occurrence of injury, although this effect was not statistically significant. However, overall these results advocate the use of CBSM programmes to target these factors, a finding which is in keeping with previous research (Johnson *et al.* 2005; Perna *et al.* 1998).

Key Points

There is significant evidence to demonstrate that certain psychological factors can predict injury (Wiese-Bjornstal 2010).

The stress-injury model outlined by Williams and Andersen (1998) proposes that certain psychological factors can predispose an individual to become more stressed when exposed to pressure and Johnson (2007) discussed the variables involved in each of the categories of factors outlined:

- Personality Factors: locus of control, competitive trait anxiety, mood status, perfectionism, self-confidence/esteem
- History of Stressors Factors: stressful life events, memory of previous injury etc.
- Coping Factors: self-blame and acceptance coping

Maddison and Prapavessis (2005) found that a CBSM intervention resulted in a reduction in injury and reduced amount of time missed due to injury in the intervention group compared to the control group.

Key References
• Johnson, U. (2007) 'Psychosocial antecedents of sport injury, prevention, and intervention: an overview of theoretical approaches and empirical findings', International journal of sport and exercise psychology, 5(4), 352-369.
• Ivarsson, A. and Johnson, U. (2010) 'Psychological factors as predictors of injuries among senior soccer players. A prospective study', Journal of Sports Science and Medicine, 9(2), 347-352.
• Williams, J. M. and Andersen, M. B. (1998) 'Psychosocial antecedents of sport injury: Review and critique of the stress and injury model", Journal of applied sport psychology, 10(1), 5-25.

Psychosocial Factors Associated with Tendinopathy and Rehabilitation from Injury

Introduction

Aside from the obvious physical health consequences, sports injuries also have the potential to cause a great deal of psychological disturbance for sports participants. Common psychological consequences of sports injuries include increased anger, depression, anxiety, tension, fear, and decreased self-esteem. There can often be an immediate imbalance and disruption to the lives of the injured athletes due to the loss of health as well as the lack of ability to achieve their full athletic potential (Reese *et al.* 2012). With this in mind, and given the high prevalence of tendinopathy in athletes (Lian et al 2005; Ostor et al 2005; Paavola et al 2002), the importance of acknowledging psychosocial factors in dealing with this type of injury is evident. Competence in recognising the psychological impact involved and confidence in incorporating strategies to target these issues in management plans are essential skills to utilise in practice (ACOS *et al.* 2006).

Despite the gradual growth of literature available regarding the psychology of injury, research has shown that chartered physiotherapists report a lack of formal training in sport psychology and rely heavily on previous experience when dealing with this aspect of injury rehabilitation (Arvinen-Barrow *et al.* 2008). Taking this into account, a recent systematic review of the literature by Heaney *et al.* (2015) aimed to identify the most appropriate content to include in formal education programmes that aimed to educate sports injury rehabilitation professionals on the psychology of injury and how it is relevant in practice.

The scope of this short course is not sufficiently broad to cover the psychosocial aspects of dealing with injury in such detail as a specific psychology education programme. However, for the purpose of this short course, the following three broad topics which were recommended in this review by Heaney *et al.* (2015) to be included in such programmes will be discussed with regard to tendinopathy:

1. ***Understanding of the psychological impact of injury***
2. ***Interventions and psychological skills/techniques***
3. ***Referral and Professional Boundaries***

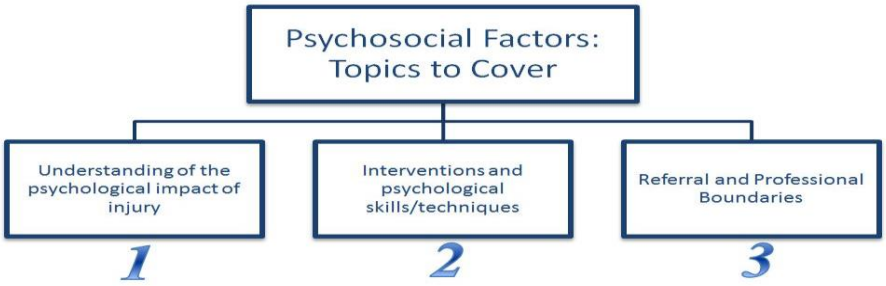


Figure 4.2: Areas that should be covered in sport psychology education for sport injury rehabilitation professionals as outlined by Heaney *et al.* (2015).

1. Understanding of the psychological impact of injury

Qualitative research by Clement *et al.* (2015) documented injured athletes' psychosocial responses during the different phases of injury rehabilitation. These four phases were: reaction immediately post injury; reaction post diagnosis; reaction to rehabilitation; and finally reaction to return to sport. Within each of these phases the authors examined the athletes' cognitive appraisal (i.e. how they view the situation) as well as the athletes' emotional response and behavioural response. The relationships between these factors play a large role in the athlete's rehabilitation and psychological health post injury. Figure 4.3 below outlines how these factors interact as per Walker *et al.* (2007).

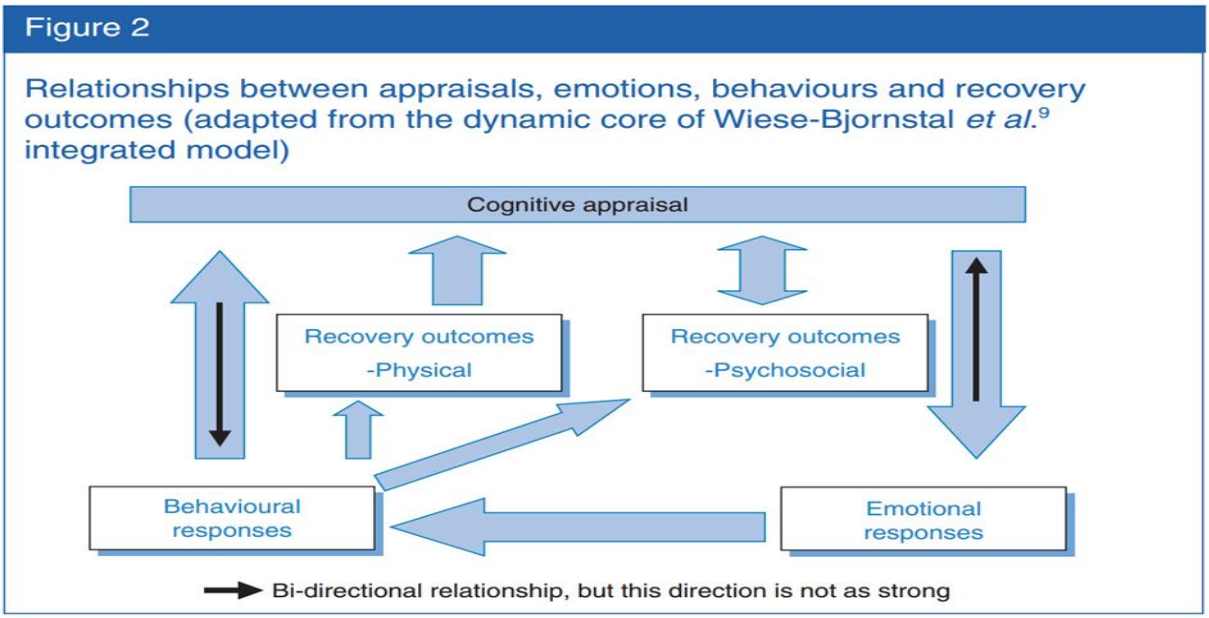


Figure 4.3 – Overview of relationships between cognitive appraisals, emotional responses and behavioural responses including how these relationships affect recovery outcomes, both physical and psychological (Walker *et al.* 2007)

Phase 1 – Reaction Immediately Post Injury

Overall, athletes' initial cognitive appraisals of the injury were predominately negative. The more athletes perceived their injury to be severe or expected to be away from their sport for a significant period of time the more negative their cognitive appraisals were and vice versa. The athletes' emotional response was affected significantly by these initial cognitive appraisals in that if they perceived the injury to be significantly serious and devastating, his or her emotional response was also highly negative.

Focusing specifically on tendinopathy, it is evident that differentiating between acute tendon injuries and chronic overuse tendinopathies would be an important consideration. The various types of tendinopathy could vary significantly with regard to the athletes' perception of severity and subsequent emotional response. The theme that emerged as the most common behavioural response during this phase was seeking social support, particularly from significant others, with some emphasising the importance of the support received from family members.

Phase	Cognitive Appraisal	Emotional Response	Behavioural Response
Reaction Immediately Post Injury	Perceived injury severity influencing appraisal	Negative thoughts leading to negative emotions	Seeking social support

Table 4.1: Overview of injured athlete's psychosocial responses during reaction immediately post injury phase of injury rehabilitation as outlined by Clement *et al.* (2015).

Phase 2 – Reaction Post Diagnosis

The first major point of change in the initial appraisals was after diagnosis. Once athletes were fully aware of the injury severity their initial response to injury changed depending on the diagnosis. In the majority of cases, the news of the diagnosis fostered a positive outlook on the situation. This may be due to initially fearing the worst after the injury has occurred and subsequently learning that the injury is not as serious or devastating as they initially feared. However, it should not all the subjects experienced a more positive outlook post diagnosis and this depends largely on the initial cognitive appraisal of severity.

Relating back to tendinopathy specifically, informing the athlete regarding the treatment protocols and prognosis involved in such an injury, it seems that this information can in many cases help the patient to realise that the severity is not so high that recovery is possible. The emotional responses on this phase simply involved changed emotions due to the new knowledge of the actual severity, and similarly to the previous phase seeking social support was the most common behavioural response.

Phase	Cognitive Appraisal	Emotional Response	Behavioural Response
Reaction Post Diagnosis	Cognitive reappraisal after diagnosis	Knowing severity changed emotions	Seeking social support

Table 4.2: Overview of injured athlete's psychosocial responses during reaction to diagnosis phase of injury rehabilitation as outlined by Clement *et al.* (2015).

Phase 3 – Reaction to Rehabilitation

Athletes described varying cognitive appraisals during the reaction to rehabilitation phase that were mainly concerned with thoughts questioning the rehabilitation process. At the start of rehabilitation in particular, it appeared that athletes' cognitive appraisals of their rehabilitation were mixed and underpinned with thoughts about the perceived value of their rehabilitation programmes, their willingness to get on with the process, and the perceived difficulty of these programmes. The most common emotional response to rehabilitation was frustration. Regarding behavioural responses, once more seeking social support was the predominant response with family again the most common source.

However, it appeared that during the reaction to rehabilitation phase, the role of sports medicine professionals as a source of social support became amplified. These professionals were able to foster positivity in the athlete and encourage targeting the end goals of rehabilitation. For tendinopathy, many treatment protocols involve completing exercises despite a certain level of pain being present (Kongsgaard et al 2005). This can be difficult for the rehabilitating athlete given that occasional flare-ups in pain are to be expected with this type of protocol and reassurance from the physiotherapist that emphasises the bigger picture would be a very valuable source of social support during this phase.

Phase	Cognitive Appraisal	Emotional Response	Behavioural Response
Reaction to Rehabilitation	Thoughts that question rehabilitation process	Feeling frustrated	Cautious and seeking social support

Table 4.3 – Overview of injured athlete's psychosocial responses during reaction to rehabilitation phase of injury rehabilitation as outlined by Clement *et al.* (2015).

Phase 4 – Reaction to Return to Sport

Regarding cognitive appraisal during the return to sport phase, it appeared that all of the athletes in this study had gained perspective as a result of the injury and rehabilitation process. The athletes gained an appreciation of being able to play sport once more and having regained their full physical capacity to do so. However, negative appraisals were also evident as some of the athletes felt anxious regarding how well their injury would "hold up" to the demands of returning to sport. The emotional response during this phase were related to these differing cognitive appraisals, with positive excitement at returning to sport and fear of re-injury the dominant responses encountered.

As for the behavioural responses during the return to sport phase, caution was the most common as the athletes consciously refrained from returning to their full pre-injury activity levels so as to reduce the risk of re-injury. This is an interesting point to note with regard to tendinopathy given that return to sport post successful rehabilitation from this type of injury needs to be gradual and progressive (Podlog and Eklund 2006) and fortunately this is appears to be the natural tendency of the athlete.

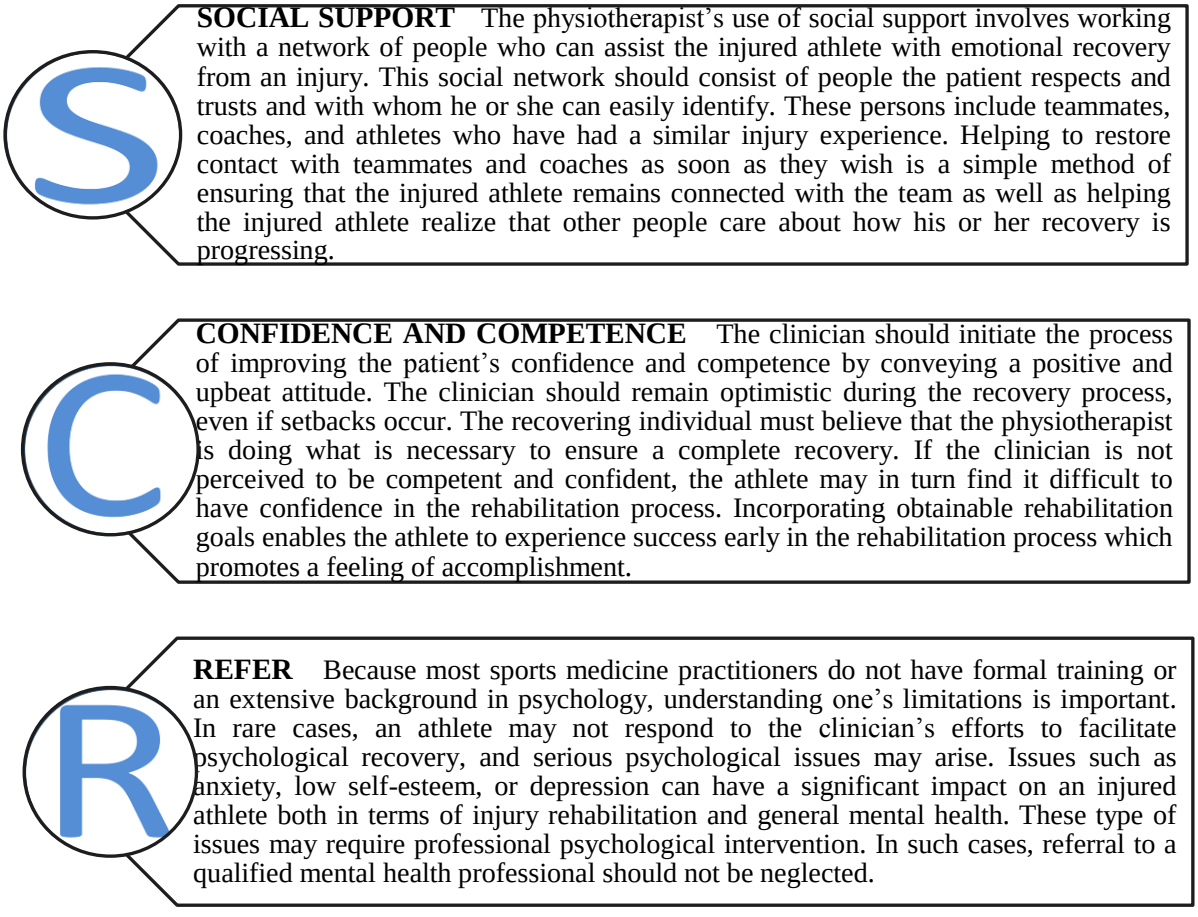
Phase	Cognitive Appraisal	Emotional Response	Behavioural Response
Reaction to Return to Sport	Lessons learned	Feelings of excitement and re-injury anxiety	Cautious in return to play

Table 4.4: Overview of injured athlete's psychosocial responses at reaction to return to sport phase of injury rehabilitation as outlined by Clement *et al.* (2015).

2. Interventions and psychological skills/techniques

Hinderliter and Cardinal (2007) acknowledge that most sports injury rehabilitation professionals only have a limited amount of training in psychology and may not feel comfortable addressing the psychological needs of the athlete. With this in mind, they developed an acronym that represents six concepts that may be used to address the psychological aspects of recovery from a physical injury, intended as simple outline to guide a psychological approach to patient care. The acronym **SCRAPE** stands for **S**ocial Support, **C**onfidence and Competence, **R**efer, **A**ccommodate, **P**sychological skills, and **E**ducate. While the wide variance in patient responses and subjective nature of addressing psychosocial issues hinders the development of a comprehensive guide, this provides a general framework to guide approaching psychological aspects of recovery from physical injury.

Figure 4.4 – Diagram displaying The SCRAPE Approach to "Psychological Rehabilitation for Recovery from Injury" as outlined by Hinderliter and Cardinal (2007)



ACCOMODATE The physiotherapist should adjust the athletes care in a way that satisfies their unique needs but not necessarily what the patient may want. The practitioner must remember that different communication methods or modes of treatment may be required in each case. After identifying the athlete's needs, the treatment protocol should be individualised. This individualisation will facilitate patient engagement in the recovery process and will assist in the development of a personal relationship between the patient and the clinician. Flexibility in scheduling can also help to allow the patient more input into the design of some aspects of the rehabilitation plan.

PSYCHOLOGICAL SKILLS Techniques such as imagery, relaxation, and goal setting can have a positive influence on the recovery of the injured athlete. Teaching and employing these psychological skills may improve the athlete's confidence, motivation, effort, and self-image. One technique which can be especially useful is the use of a rehabilitation journal. The injured athlete is asked to keep track of the various aspects of his or her recovery process in a confidential diary. Looking back at previous accomplishments, the athlete may gain confidence in the care that has been delivered and may become more committed to the recovery process.

EDUCATE Educating the athlete on injury pathology, typical recovery time, necessary restrictions, and the rationale for therapeutic interventions may help the athlete become a more active participant in the recovery process. The athlete's active involvement in his or her care can help to reinforce other aspects of the SCRAPE approach. When educating the patient, the physiotherapist should clearly articulate and reinforce important points and should avoid excessive use of medical terminology. The clinician may also provide written materials or illustrations designed to reinforce specific concepts. The athlete should be encouraged to keep such materials with his or her journal.

The psychological strategies discussed in the SCRAPE approach such as goal setting, positive self-statements, cognitive restructuring, and imagery may facilitate recovery from injury (Hinderliter and Cardinal 2007). The mechanism by which these strategies are effective may be through helping to reduce stress and increasing coping mechanisms and social support. However, problematic emotional reactions can lead to increased stress at various stages of the rehabilitation cycle and it is essential that the physiotherapist is alert to signs of increased stress so that they can attempt to intervene accordingly. The following are a list of behaviours to watch out for which may indicate increased stress as outlined by (ACOS *et al.* 2006):

- Unreasonable fear of re-injury
- Continued denial of injury severity and response to recovery
- General impatience and irritability
- Rapid mood swings
- Withdrawal from significant others

- Extreme guilt about letting the team down
- Dwelling on minor physical complaints
- Obsession with the question of return-to-play

In order to attempt to prevent these possible negative emotional and behavioural responses from occurring, as well as dealing with them when they do occur, the physiotherapist will need to use their skills in this area to steer the athlete back onto the right track with regard to their psychological recovery from injury. Podlog *et al.* (2014) provide an overview of the necessary skills that sports injury rehabilitation professionals can utilise as part of the fundamentals of a psychological injury intervention plan to address these possible negative responses.

Table 4.5: Table displaying an overview of the necessary skills that sports medicine professionals can utilise as part of the fundamentals of a psychological injury intervention plan as outlined by Podlog et al. (2014)

Skill 1	**Injury Education** - Providing proactive practical information about injury, healing, and rehabilitation empowers the athlete encouraging personal investment in the recovery process and facilitating compliance with treatment tasks.
Skill 2	**Rehabilitation and Return to Sport Goals** - Understanding of rehabilitation goals helps athletes create a sense of personal responsibility and increases their expectations of return to sport by creating a clear path toward recovery
Skill 3	**Building the Rehabilitation Team** - Building a team that helps meet the challenges and demands of the new rehabilitation environment helps the athlete overcome feelings of isolation related to separation from the sport and teammates
Skill 4	**Managing Emotions** - Understanding how to identify and cope with the distress inherent in injury helps improve mood and controls the ups and downs of rehabilitation.
Skill 5	**Visualizing The Stages Of Recovery** - Fostering confidence in the athletes' ability to cope with injury adversity and endure rehabilitation is facilitated by depicting a positive future perspective on the rehabilitation process.
Skill 6	**Focus And Distraction Control** - Facilitating a task focus and providing guidance in maintaining focus can enable athletes to deal with the uncertainty of rehabilitation and remain appropriately focused on physical, technical, and psychological skills.

Skill 7	**Working Through Pain** - Managing pain effectively enables the athlete to trust the rehabilitation process, maintain a stable emotional state, and make effective decisions regarding activity and limits.
Skill 8	**Building Confidence In Return To Play** - Accepting fear and treating it as a tool to guide decision making enables the athlete to transition effectively to sport.
Skill 9	**Mental Toughness And The Survival Mind Set** - Focusing on controlling the controllable aspects such as personal thoughts and actions enable the athlete to gain skills in coping with adversity and can facilitate recovery.
Skill 10	**Becoming A Renewed Athlete** - Assimilating lessons learned from injury and re-embracing the aspirations that led to initial participation enable the athlete to return to sport renewed and reinvigorated.

3. Referral and Professional Boundaries

It is important to note that the skills discussed in the previous section which physiotherapists can use to target psychosocial issues associated with injury may not be sufficient in dealing with some cases. As outlined as part of the SCRAPE approach (Hinderliter and Cardinal 2007), an athlete may not respond to the clinician's efforts to facilitate psychological recovery, and more serious psychological issues may arise. These types of issues may require professional psychological intervention and referral to a qualified mental health professional should not be neglected.

Unfortunately, specific criteria outlining the indications for onward referral in this manner are not currently available. It has been recognised in the literature that there is a need for tools to facilitate assessment of serious psychological issues as well as greater communication between the mental health community and sports injury rehabilitation professionals (Mann *et al.* 2007). With this in mind, it is evident that the physiotherapist when dealing with sports injuries should use their own good judgement in identifying when referral to a qualified mental health professional is warranted.

Psychosocial Factors Associated with Return to Play

It has increasingly been recognised that physical and psychological readiness to return to play after injury do not always coincide (Ford and Gordon 1998). Podlog and Eklund (2007) used the self-determination theory to examine the psychosocial literature on the return to sport following injury. This theory focuses on the effects of varying degrees of self-determination on human behaviour, health and well-being (Ryan and Deci 2007). In simple terms, it focuses on how different individuals will vary in their tendency to behave in effective and healthy ways. By doing this, they were able to identify three main areas of concern that athletes can often face on return to sport following injury:

- **Competence Based Concerns** – concerns over one's own ability to remain uninjured; concerns over a lack of physical fitness; doubts over an inability to reach future performance goals.

- **Autonomy Based Concerns** – being pressured into returning to sport before they are physically and/or psychologically ready to do so, be it by coaches, teammates etc.

- **Relatedness Based Concerns** - feelings of estrangement and separation from one's teammates as well as a loss of social identity

It is proposed that by addressing these three areas of concern during the return to sport period, the athlete will be facilitated to make a full return psychologically as well as physically.

Strategies aimed at addressing Competence Based Concerns

Returning athletes may have fears and concerns about how their body will withstand the demands of sport (Bianco *et al.* 1999) and may also be concerned regarding future performances and the ability to fulfil personal or external expectations (Bianco 2001). The following strategies that can be used to address these concerns were described by Podlog and Dionigi (2010), with some being similar to those used during the rehabilitation phase.

1. By adopting a team approach to rehabilitation that incorporates the knowledge and expertise of various rehabilitation experts.

2. By implementing an individualized goal-setting programme that focuses on skill development, task-specific processes, and flexible deadlines.

3. By using role models if possible who can help alleviate returning athlete concerns through informational and emotional support, as well as through inspiration and motivation (i.e. having previously returned from injury)

Strategies aimed at addressing Autonomy Based Concerns

Research examining the consequences of varying degrees of autonomy has found that the type of motivation to return to sport may have a significant impact upon athletes' psychological return outcomes (Podlog and Eklund 2005). The following strategies that can be used to address autonomy concerns were described by Podlog and Dionigi (2010):

1. Autonomy can be encouraged through instilling positive thinking via cue words and self-affirming statements that focus athletes' attention on matters under their own control.

2. By maintaining direct lines of communication with other treatment professionals the possibility of restriction of the athlete's decisional autonomy regarding the timing of a return to sport following injury can be minimised.

Strategies aimed at addressing Relatedness Based Concerns

Maintaining a sense of connectedness to coaches, teammates and training partners can be difficult when an athlete is injured and this can lead to feelings of alienation and isolation when returning to sport. The following strategies that can be used to address relatedness concerns were described by Podlog and Dionigi (2010):

1. By providing athletes with meaningful tasks and activities such as return-to-sport exercises which can be carried out in the same vicinity as teammates in an effort to maintain their social involvement and social identity as an athlete.

2. However, it is important that the athlete has progressed sufficiently before they return to training with teammates. Otherwise they may have to be excluded from the majority of the training procedures and this would only heighten the sense of isolation experienced by the athlete.

KEY POINTS

Sports injuries have the potential to cause a great deal of psychological disturbance for sports participants with common consequences including increased anger, depression, anxiety, fear and decreased self-esteem (Reese et al 2012)

Heaney et al (2015) outlined the core content that sport injury rehabilitation professionals should be educated on regarding the psychology of injury and how it is relevant in practice:

1. Understanding of the psychological impact of injury at various stages:
    - Reaction immediately post injury
    - Reaction post diagnosis
    - Reaction to rehabilitation
    - Reaction to return to sport
2. Interventions and psychological skills/techniques – the following are examples of methods that can be used to aid this process:
    - The SCRAPE approach to psychological rehabilitation for recovery from injury as outlined by Hinderliter and Cardinal (2007)
    - Skills that can be used to aid psychological intervention in sports injury recovery as outlined by Podlog *et al.* (2014)
3. Referral and professional boundaries

It is increasingly recognised that physical and psychological readiness to return to sport after injury do not always coincide (Ford and Gordon 1998).

Three main areas of concern that athletes can encounter at this point were identified by Podlog and Eklund (2007):

1. Competence Based Concerns
2. Autonomy Based Concerns
3. Relatedness Concerns

Strategies aimed at addressing these concerns in order to facilitate a full psychological as well as physical return to sport following injury as per Podlog and Dionigi (2010) have been discussed.

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Management Tendinopathy In-Season

While abstaining from playing sport during rehabilitation prior to return to play has been recommended in the literature (Paavola *et al.* 2000), this may not be the most practical or even most beneficial approach to dealing with tendinopathy. Unfortunately, there is a scarcity of evidence recommending a system of modified rest in which the pain-provoking activity should be limited or avoided (Paavola *et al.* 2002) with the current literature in this area focusing mainly on AT.

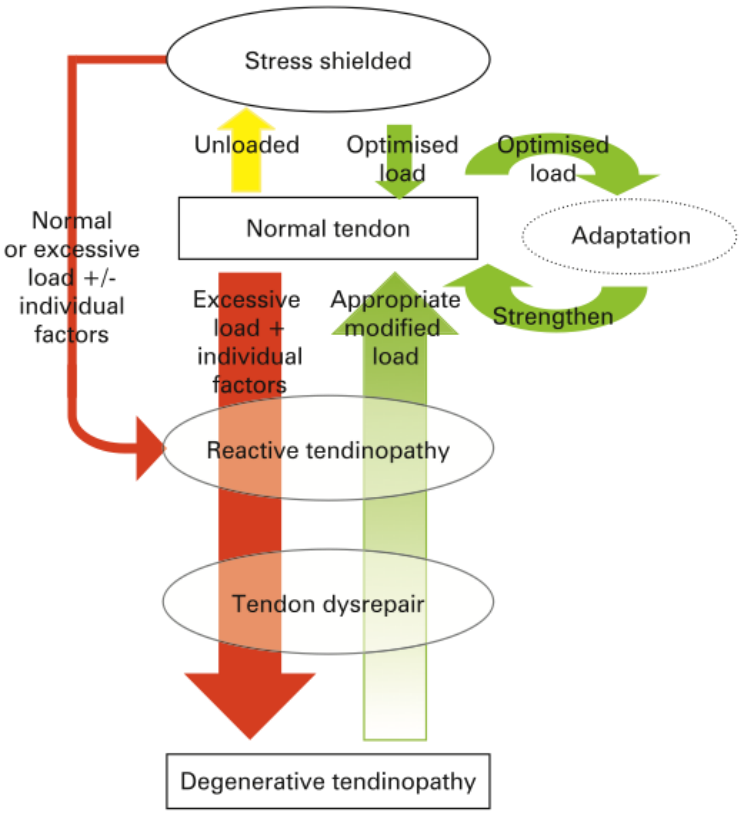
Cook and Purdam (2013) discussed the challenge of managing tendinopathy in competing athletes. Tendinopathy begins with a mismatch between the tendon's load capacity and load placed on the tendon. This occurs most commonly through a sudden or substantial change in the load it is subjected to. This can include a return to sport from an injury or after the off season, where the load capacity of the tendon is reduced due to a loss of a regular loading stimulus. As tendons respond very slowly to load, a tendinopathic response is triggered if the magnitude or temporal distribution exceeds the tendon's threshold. Examples of overloads that can cause tendinopathic responses in the TA are demonstrated in Table 4.6.

Type of overload	Example
Single high-intensity session	Repeated uphill running,
Increased frequency of training	High-load training more than five times a week
Different drills	Rapid introduction of plyometric training
High loads when fatigued	Sprints at the end of training
Change in footwear	Shoes that provide less support, or stiff soles, shoes that mandate a forefoot strike or have a lower heel wedge
Change in surface	Running in soft-sand, running on uneven surfaces
Training with muscle stiffness	Training sessions following heavy-weight session

Table 4.6: Table demonstrating overloads that can cause tendinopathic responses in the TA as outlined by Cook and Purdam (2013).

The continuum model of tendinopathy outlined by Cook and Purdam (2009) is demonstrated in Figure 4.5. This model provides an overview of the background for the mechanisms by which load management can prevent worsening of symptoms of tendinopathy while continuing with sports participation.

Fig 4.5: Contimuum model of tendinopathy as outlined by Cook and Purdam (2009)



If a tendon is overloaded in an athlete with underlying degenerative tendinopathy participating in sports, they will present with a reactive on degenerative tendinopathy. In addition to the high loads, changes in mechanical properties such as stiffness and elasticity at the interface between areas of differing tendon pathology may predispose degenerative tendons to become reactive in previously unaffected portions of the tendon. By managing the load that the tendon is exposed to the reactive tendinopathy should return the degenerative tendon to its functional low pain status (Cook and Purdam 2013).

Load Management In-Season (Cook and Purdam 2013)

- Consideration of total load on the tendon is an important concept, as a combination of small overloads may induce a reactive response. Care with rapid increase or an excessive tensile load is a major consideration.
- Reduction of compressive loads and particularly the combination of compression and tensile loads is especially important while the athlete continues with sport participation (Cook and Purdam 2012).
- Loads that reduce pain should be introduced reasonably early. Loading to decrease pain will maintain tendon stimulus. There is literature to support the use of isometric exercise in pain conditions for this purpose (Kosek and Ekholm 1995). In reactive tendons, isometric contraction with some load appears to be beneficial in decreasing pain for

several hours. These exercises should be completed in the mid to inner range of the muscle-tendon unit to reduce compression. In highly irritable tendons, a bilateral exercise, shorter holding time and fewer repetitions per day may be indicated.

- Provocative tests and objective scoring methods should be used to monitor tendon pain. As the VISA scale scores substantially higher on pain during higher level activity, therefore it is only responsive on a month-to-month basis. The athlete can monitor tendon response to training loads by completing a simple loading test daily at a similar time. Table 4.7 demonstrates some suggested provocative tests that can be used to score tendon pain.

Provocative clinical tests useful to monitor tendon pain

Tendon	Low-load clinical test	High-load clinical test
Achilles	Single leg heel raise	Hop
Patellar tendon	Decline squat	High single leg jump, landing from a height
Hamstring tendon	Single leg bent knee bridge	Single leg dead lift
Gluteal tendon	Single leg stance	Hop

Table 4.7: Some suggested provocative tests that can be used to score tendon pain

Silbernagel *et al.* (2007) evaluated the effect of continued sport and the associated loading on the TA while completing an eccentric exercise regime. The participants were followed over 1 year, with one group continuing to load their tendons and participate in sport while the other group limited this type of activity. There were no significant differences between groups at 6, 12 and 26 weeks or 1 year.

Continued sport, as long as symptoms allow, appears to have a specific positive effect on calf-power and jump performance which is not gained with the Silbernagel loading programme alone even though it includes faster calf loading and stretch-shorten cycle rehabilitation (Malliaras *et al.* 2013).

Key Points

Tendinopathy is a prevalent injury in athletes and is very common in the competition season when loads are high (Cook and Purdam 2013).

(Silbernagel *et al.* 2007) found that no negative effects could be demonstrated from continuing TA-loading activity, such as running and jumping, with the use of a pain-monitoring model, during treatment.

By managing the load that the tendon is exposed the reactive tendinopathy should return the degenerative tendon to its functional low pain status (Cook and Purdam 2013). This involves:

- Considering total load on tendon
- Reduction of compressive loads
- Isometric exercises - beneficial in decreasing pain
- Using provocative tests and objective scoring methods to monitor tendon pain

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Return to Play in Sport

Return-to-Play (RTP) is the decision-making process of returning an injured or ill athlete to practice or competition (Herring *et al.* 2012). RTP can be important to an athlete for a number of reasons with common motives including achieving personal goals, a love of playing sport, socialising with teammates and preserving athletic identity (Podlog and Eklund 2006). Returning an athlete to sport participation is a complex and often difficult decision. Within competitive sport this process often involves aggressive rehabilitation while avoiding increased risk to the athlete. RTP has not been well defined in the literature and as a result there has been little progress toward identifying systematic approaches to clinical decision making in sport (Shultz *et al.* 2013). A consensus statement by Herring *et al.* (2012) states that with regard to RTP it is essential the team physician confirm:

- Restoration of sport-specific function to the injured part
- Restoration of musculoskeletal, cardiopulmonary and psychological function, as well as overall health of the injured or ill athlete
- Restoration of sport-specific skills
- Ability to perform safely with equipment modification, bracing, and orthoses
- The status of recovery from acute or chronic injury
- Psychosocial readiness
- The athlete poses no undue risk to themselves or the safety of other participants
- Compliance with federal, state, local and governing body regulations and legislation

Unfortunately, well established RTP guidelines do not exist for the vast majority of conditions (this is the case for all three of Achilles, rotator cuff and patellar tendinopathies) and patients are greatly dependent on their clinician's ability to take a broad spectrum of factors into account in order to reach the optimal decision. Of course, individual decisions regarding the return of an injured athlete to sport will depend on the specific facts and circumstances relating to each individual patient (Herring *et al.* 2012) and this perhaps has prevented the establishment of comprehensive protocols for various conditions.

With this in mind, Creighton *et al.* (2010) developed a 3-step decision-based RTP model for an injury or illness that is specific to the individual practitioner making the decision. They aimed to synthesize the available literature and propose a model for RTP which would help clarify the processes that clinicians use consciously and subconsciously when making these decisions. A recent study by Shrier *et al.* (2014) set out to validate this proposed model and concluded that by grouping the large number of factors affecting RTP decision making into specific domains the process of RTP decisions was simplified. The authors also highlighted the value of utilising this model in educating relatively inexperienced sports injury rehabilitation professionals on the RTP process.

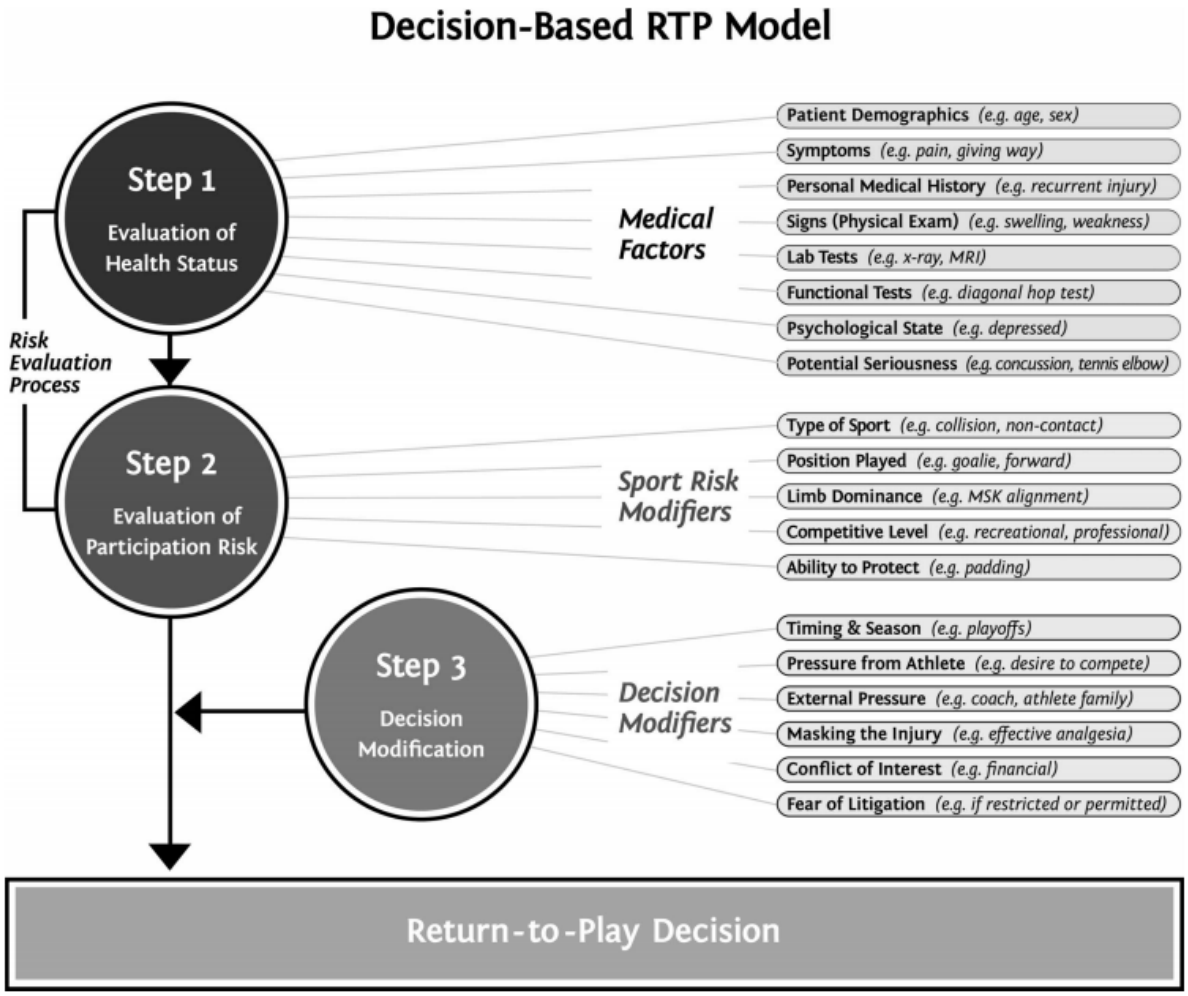

Figure 4.6: Decision-Based RTP Model as outlined by Creighton *et al.* (2010) which outlines a 3 step process and illustrates the interplay between these steps.

The model incorporates three general steps in the process of RTP and the interplay of these steps is demonstrated in Figure 4.6. The 3 steps outlined include:

- ***Step 1:*** Evaluation of Health Status
- ***Step 2:*** Evaluation of Participation Risk
- ***Step 3:*** Decision Modification

Step 1: Evaluation of Health Risk (Creighton *et al.* 2010)

This involves a complete evaluation of the health status for the injury in question based on a subjective examination together with functional and laboratory testing when indicated. While some research uses only this health status evaluation in the RTP decision making process, this model identifies this evaluation as simply the first step stress and the findings should not be considered in isolation. Table 4.8 outlines the rationale behind inclusion of the various medical factors involved in step 1.

Table 4.8: Table detailing rationale for relevance of factors to RTP decision making process

Factor	Rationale for Relevance to RTP Decision Making Process
Patient Demographics	Variances in sex and age influence the health status of an individual secondary to hormonal/age-related factors affecting factors such as tissue regeneration abilities
Symptoms	Subjective reporting of the present history of the injury provides essential information in establishing the health status of the individual with pain in particular recognised as an important factor (Park *et al.* 2004) given its association with incomplete healing.
Personal Medical History	The personal medical history can help to provide the physiotherapist with a context with which to evaluate the health status. In particular, determining if it is the first instance of injury or a recurrent problem can influence RTP decisions from both a psychological (Bauman 2005) and physical recovery perspective
Signs	General recommendations for physical signs used by clinicians: Strength – at or near pre-injury levels ; Range of Motion - at or near pre-injury levels ; Joint Stability – no instability ; Tenderness – injury site should not be tender ; Inflammation or Swelling – none present ; Effusion – none present
Laboratory Tests	Imaging techniques can indicate some of the physiological abnormalities that suggest incomplete healing.

Functional Tests	Even when tissue may be fully healed biologically, deficits can remain that are secondary to the injury and functional testing which simulate the sport specific actions the individual needs to RTP. Regarding tendinopathy specifically, Silbernagel *et al.* (2006) developed a test battery to evaluate if AT caused functional deficits on the injured side compared with the non-injured side in patients. In patellar tendinopathy given that it is more prevalent in sports involving repetitive jumping, simulation of this movement would be a rational functional test to use.
Psychological State	See psychosocial factors involved in return to sport section
Potential Seriousness	The health status is also affected by the specific tissue injured, its extent, and the subsequent potential for healing. With this in mind it is clear that the RTP decision making process would vary significantly between chronic and acute tendinopathies.

Step 2: Evaluation of Participation Risk (Creighton *et al.* 2010)

The main disadvantage of allowing RTP is a high risk of re-injury. A study by Hägglund *et al.* (2006) investigated 263 elite footballers and found that 87% of those injured in a given season would also suffer from an injury the following season compared with 48% of those who had no injury in the first season. There are a number of factors that need to be taken into consideration in evaluating the sports participation risk for each individual athlete.

Generally, the higher the degree of contact allowed in a given sport the higher the risk of injury that the participants are exposed to (Kovacic and Bergfeld 2005) and this is an important factor to consider. In addition the competitive level at which the athlete will be competing at will affect RTP as at the same health status higher levels of competition are associated with higher health risk (Orchard *et al.* 2005).

The specific risk factors for achilles, rotator cuff and patellar tendinopathies have been outlined in this booklet. If the athlete is returning to a sport or sporting position where the specific risk factors for their injury are present (as will often be the case as may have been cause of original injury), this will affect the RTP decision making process. For example, the RTP decision would differ between an outfield soccer player with a rotator cuff injury and a goalkeeper with the same injury.

Step 3: Decision Modification (Creighton *et al.* 2010)

The authors acknowledge that there are additional factors aside from health status and participation risk that may influence the RTP decision making process. These factors are termed "Decision Modifiers". These factors are not restricted to the athlete and can involve family, teammates and coaches. As can be seen in Figure 4.6, the decision modification step is set aside from the other steps. This is because while decision modification factors cannot be used to determine RTP in isolation without the context of the previous steps, no information on decision modification is provided by these previous steps. Examples of such factors which can modify the RTP decision making process include:

- ***Timing:*** If the injury occurs during the off-season for an athlete then it may be more beneficial for them to delay return to sport until it is necessary in order to maximise recovery. Conversely, in-season injuries may be affected by desire to partake in certain events due to prestige, financial gain, etc.
- ***Pressure from Athlete or Others:*** Although the sports injury rehabilitation professional may be inclined to discount pressure from these types of sources, in certain instances those other than the clinician may be in a better position to evaluate other factors such as job security, family and personal situations etc.

KEY POINTS

Return-to-Play (RTP) is the decision-making process of returning an injured or ill athlete to practice or competition (Herring *et al*. 2012)

Unfortunately, well established RTP guidelines do not exist for the vast majority of conditions and patients are greatly dependent on their clinician's ability to take a broad spectrum of factors into account in order to reach the optimal decision (Herring *et al.* 2012)

Creighton *et al.* (2010) developed a 3-step decision based RTP model for an injury or illness that is specific to the individual practitioner making the decision which incorporates three steps:

1. ***Evaluation of Health Status***
    - Factors involved: patient demographics, symptoms, personal medical history, signs, laboratory tests, functional tests, psychological tests, potential seriousness.
2. ***Evaluation of Participation Risk***
    - This involves considering the specific risks that each individual athlete will be exposed to on return to pay and how this affects the RTP decsion making process
3. ***Decsion Modification***
    - There are additional factors that can occasionally influence the process such as timing of injury be it in-season or during the off-season or or pressure from others such as teammates, family etc.

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