Achilles tendinopathy in the dysrepair stage rehabilitation

Environment, Air

<u>A critical review of the key considerations of rehabilitation for an Achilles</u>
<u>Tendinopathy in the dysrepair stage</u>

The Achilles tendon is the insertion of the distal gastrocnemius and soleus, transmitting load to the calcaneus. It's the strongest and thickest tendon in the body, but has a high injury rate in runners. (Wezenbeek et al 2017; Paavola et al 2002). It consists of elastin and type 1 collagen embedded in the extracellular matrix of water and proteoglycans; and of the dry mass of the tendon 70% is collagen and 1 – 2% is elastin. The collagen fibres are packed together in parallel bundles and the tenocytes and tenoblasts, which produce the protein and carbohydrates, are arranged in between them. It doesn't have a true synovial sheath, instead it is surrounded by a paratenon which reduces friction during movement against other tissues. (Longo, Ronga, & Maffulli 2018; Van Sterkenburg, & Van Dijk 2011; Maffulli, Sharma, & Luscombe 2004; Paavola et al 2002).

There isn't an exact aetiology for Achilles tendinopathy (AT) (Beyer et al 2015, Paavola et al 2002) but clinical experience has linked it to extrinsic factors of previous injury and training errors, and intrinsic factors of tendon vascularity, muscle weakness and lower limb misalignment (Azevedo, Lambert, Vaughan, O'Connor & Schwellnus 2009). AT is linked with rapid increases in physical activity placing a significant biomechanical demand on the tendon, followed by an inadequate resting period not allowing for a complete healing response. (Papa 2012). This repetitive strain on the tendon which cannot tolerate any more tensile loading results in tendon fibres tearing microscopically producing inflammation and pain. (Cook & Purdam

2009; Paavola et al 2002). Cook and Purdam (2009) categorised tendinopathies as reactive, dysrepair and degenerative, the dysrepair stage is more advanced than reactive where the extracellular matrix has significantly broken down, there is an increase in protein production and proteoglycans which results in the disorganisation in the matrix. Tenocytes proliferate haphazardly, the parallel arrangement of the collagen fibres is lost becoming irregular and there is an influx of type 3 collagen (Longo et al 2018).

For the AT to heal there needs to be management of loading and exercise which can stimulate the matrix structure (Cook & Purdam 2009). Conservative treatment should be targeted at relieving symptoms, including limb misalignment and muscle weakness, but needs to stimulate cell activities, restructure the matrix and encourage protein production. Key considerations for AT are to complete eccentric exercises to increase collagen production in abnormal tendons and improve the matrix structure, restore the full ankle joint dorsiflexion which decreases the strain placed on the Achilles Tendon, and ensuring muscle firing in gluteal medius to dissolve forces during the early stance phase to reduce load on the Achilles tendon (Munteanu & Barton 2011; Cook & Purdam 2009; Paavola et al 2002).

The first key consideration is eccentric exercise and this is considered the gold standard for rehabilitating AT (Wetke, Johannsen & Langberg 2015). Eccentric exercise for AT was developed in 1984 by Curwin and Stanish (cited in Cook, Khan & Purdam 2002) and progressed by Alfredson et al (1998) by introducing heavy load eccentric exercise. In this study the

findings were that all 15 participants in the study were back to their pre-injury running after 12 weeks with no requirements for surgery, and further research has recommended to treat AT with eccentric exercise for 3 months before trying other forms of treatment options. (Grävare, Silbernagel & Crossley 2015). Follow up reviews have been published between 1 – 5 years after trials and have found participants have fully recovered using eccentric exercise and suffered no symptoms or reduction in function (Silbernagel, Brorsson, & Lundberg 2011; Gärdin, Movin, Svensson & Shalabi 2010).

Eccentric exercise, when the muscle contracts whilst being lengthened by forces applied to it, is proposed to cause structural adaptations in the tendon which increases length and decreases stiffness (Verrall, Dolman & Best 2018). It has been discussed that the progressive loading mechanism of eccentric exercise stimulates the synthesis of type 1 collagen which aids in tendon repair, affects tendon compliance and reduces tenocyte activity and increases tendon strength (Mascaró et al 2018; Verrall, Schofield, Brustad & Physio 2011; Sayana & Maffulli 2007). It is commented that most of these findings are clinical options and it is still largely unknown why eccentric exercises produce excellent clinical results, and as the Randomised Control Trial's (RCT) conducted haven't measured the outcomes on tissue remodelling through loading, the outcomes discussed may not be empirically reliable (Mascaró et al 2018; Murphy et al 2018B; Verrall et al 2018).

A successful rehabilitation programme for AT needs to be based on the individual's functional capacity, clinical reasoning and the stage of tendon healing (Grävare et al 2015). Achilles tendinopathy at the dysrepair stage

needs to focus on progressive loading and exercise to improve the mechanical or structural properties of the tendon (Mascaró et al 2018; Cook & Purdam 2009). The rehabilitation programme needs to ensure the muscle complex is rehabilitated, not just the tendon in isolation and to include speed and endurance, as if there is inadequate loading, speed or endurance this can negatively affect rehabilitation and result in a lack of musculotendinous strength to return to activity. (Cook et al 2002).

Studies on eccentric exercises compared to other modalities or immobilisation has produced results for effective and timely rehabilitation for AT, with Alfredson et al's (1998) RCT showing that the intervention group were back to pre-injury exercise after 12 weeks with no pain or loss of functionality whilst the control group who rested before surgery still experienced pain and after surgery they returned to their pre-injury running but it took 24 weeks rather than 12. Further studies supported the findings that eccentric exercise should be the first approach to treat AT (Rowe et al 2012; Verrall et al 2011; Magnussen, Dunn & Thomson 2009;) and from Rowe et al's (2012) study they determined that eccentric exercises have the strongest supporting evidence for being the first treatment option for AT.

There have been studies that have not supported Alfredson et al's findings or challenged the protocols set down in their study. Wetke et al's (2015) study found only 26% of their participants improved with eccentric training alone and challenged studies conducted in controlled situations with researchers seeking high compliance from their participants cannot be replicated in a clinic setting, however their training regime was cautious and progressed

slower compared to Alfredson et al's protocol. Sayana and Maffulli's (2007) study found that under 60% of non-athletic participants recovered using eccentric training alone but they didn't have a control group to compare results, and Chester, Costa, Shepstone, Cooper & Donell (2008) discussed studies have been on recreational athletes and may not be comparable for sedentary individuals Their RCT found there wasn't statistically significant differences between using eccentric exercises compared to other conservative modalities, but was limited by the participants demographics and sample size. Rompe, Furia & Maffulli (2009) found they couldn't reproduce the results of previous studies despite following the same procedure, with only 60% successful outcomes. Murphy et al (2018) reviewed studies on eccentric exercise and determined the quality has been low. From 46 studies only 4 have a sample size larger than 50 participants and 18 of these studies did not use a valid or reliable measure for self-reported pain. These findings show there is conflicting evidence for the effectiveness of eccentric exercise rehabilitating AT in the dysrepair stage.

The second key consideration for AT is the kinetic chain and this focuses on lack of dorsiflexion (DF) in the ankle and Gluteus Medius (GM) weakness in the hip which have been identified as intrinsic factors for AT (Franettovich, Honeywill, Wyndow, Crossley & Creaby 2014). AT is commonly linked to runners as when the foot makes contact with the ground, the forces are absorbed by soft tissues and joints as they travel up the kinetic chain, with the Achilles Tendon essential for this process as it is elongated when the foot connects with the ground, storing energy which is used during the second

part of the stance contributing to the driving force production (Lorimer & Hume 2014). Lack of DF is the loss of the posterior glide of the talus in the ankle mortise and this restricts the forward progress of the tibia over the talus, placing abnormal stresses on the foot and ankle (Amis 2016; Denegar, Hertel & Fonseca 2002) This will mean the Achilles tendon is not at maximal stretch so therefore it must absorb the same load in a shorter range of movement and with less time (Donoghue, Harrison, Laxton & Jones 2008; Cook et al 2002).

DF can cause subtalar pronation as when the talus doesn't move back fully into the ankle mortise it causes the subtalar joint to excessively pronate producing torsional action on the Achilles Tendon which places disproportionate tensile stress on the medial fibres of the Achilles causing microtears (Wezenbeek et al 2017; Hein, Janssen, Wagner-Fritz, Haupt & Grau 2014; Ryan et al 2009). Munteanu & Barton's (2011) study didn't support the theory that the torsional action on the Achilles tendon was linked to AT but they did find participants suffered reduced DF and the authors suggested this is a compensatory strategy to limit the loading through the tendon. Kaufman, Brodine, Shaffer, Johnson & Cullison's (1999) study found participants with rear foot inversion and decreased DF with knee extension is associated with AT whilst Rabin, Kozol & Finestone's (2014) study measured DF with the knee bent to imitate the position of the limb when running and found restricted DF was foretelling of developing AT in their participants. The authors concluded their study was the first of its kind to find a positive association with DF and AT but limitations of their study were a small sample

size, and the participants were army recruits who underwent rigorous physical training so this isn't a representative sample.

GM is an essential muscle in the running gait cycle as its role is to stabilise the hip during the early stance phase and if there is muscular weakness in GM it can cause internal rotation of the femur, impacting the kinetic chain as it causes medial deviation at the knee, tibial abduction and subtalar pronation at the ankle, which as previously discussed is seen as an intrinsic factor for AT (Bell-Jenje, et al 2016; Azevedo et al 2009). When there are abnormal biomechanics in the kinetic chain it can lead to increased loading and stress on multiple anatomic structures and GM weakness reduces the hips impact absorbing capacity increasing the load on the Achilles tendon (Bramah, Preece, Gill & Herrington 2018; Doherty et al 2016; Munteanu & Barton 2011). Kulig, Loudon, Popovich Jr, Pollard & Winder's (2011) study found altered kinematics when jumping for dancers with AT, including hip adduction and internal knee rotation compared to the control group. Franettovich et al (2014) found there was delayed neuromotor control in GM with runners with AT and suggested there is a link in the mechanics from the hip to the ankle which needs to be considered for rehabilitation whilst Bramah, Preece, Gill & Herrington's (2018) study showed that runners with injuries have a greater peak contralateral pelvis drop and lean their trunk forward more, due to reduced strength or neuromuscular function at the hip. These studies all considered GM weakness in runners who already have AT so it is difficult to conclude if GM weakness is a risk factor for AT or a result of AT.

Whilst risk factors for AT have been discussed include subtalar pronation, dorsiflexion range of movement and muscle strength, these have largely been developed from clinical perspective rather than very limited primary epidemiological data (O'Neill, Watson & Barry 2016), so an evidence-based conclusion on whether addressing DF and GM weakness will improve AT cannot be drawn but based on clinical evidence any muscle weakness or abnormal joint mobility in the kinetic chain needs to be addressed as part of the rehabilitation program (Grävare et al 2015).

To conclude despite decades of research AT continues to be challenging for researchers and clinicians with no conclusive aetiology (Murphy et al 2018B; Beyer 2015). Eccentric exercise has been termed the gold standard for rehabilitating AT and follow up studies have found that participants' AT has resolved and they continue to be symptom free up to five years after the study (Van der Plas et al 2012; Silbernagel et al 2011; Gärdin et al 2010). It can be concluded, therefore, that eccentric exercises is effective to rehabilitate AT but there are gaps in the literature to be able to produce evidence-based best practice for practitioners including a standardised exercise prescription protocol and effective healing stage specific interventions (Rowe et al 2012). There also needs to be a conclusive scientific explanation for eccentric exercise as there is no understanding for the mechanism underpinning this therapeutic approach (Murphy et al 2018B). Literature does view DF and GM weakness as intrinsic factors for AT but there is limited research available so further empirical research needs to be conducted to confirm this link. Based on clinical opinion, however, it is

important they are considered when rehabilitating the AT (Franettovich et al 2014).

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