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CLINICAL COMMENTARY

WHY ARE ECCENTRIC EXERCISES EFFECTIVE FOR ACHILLES TENDINOPATHY?

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ABSTRACT

Achilles Tendinopathy is a complex problem, with the most common conservative treatment being eccentric exercises. Despite multiple studies assessing this treatment regime little is known about the mechanism of effect. This lack of understanding may be hindering therapeutic care and preventing optimal rehabilitation. Of the mechanisms proposed, most relate to tendon adaptation and fail to consider other possibilities. The current consensus is that tendon adaptation does not occur within timeframes associated with clinical improvements, therefore the clinical benefits must occur through another unidentified pathway. This clinical commentary critically reviews each of the proposed theories and highlights that muscle alterations are observed prior to onset of Achilles Tendinopathy and during the disease. Evidence shows that the observed muscle alterations change with treatment and that these adaptations have the ability to reduce tendon load and thereby improve tendon health. The purpose of this clinical commentary is to review previous theories regarding the mechanisms by which eccentric exercise might affect Achilles tendinopathy and offers a novel mechanism by which the plantarflexor muscles may shield the Achilles tendon.

Keywords: Achilles, eccentric exercise, efficacy, Tend*

Level of Evidence: 5

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INTRODUCTION AND BACKGROUND

Tendinopathies of the Achilles tendon affect 2% of the general adult population,[1] with a prevalence in active individuals between 9-40% depending on the type and level of sporting activity investigated.[2-4] Exercises that load the tendon are promoted as being beneficial for tendinopathy with isolated eccentric exercises receiving the most attention.[5] In recent years researchers have attempted to determine the clinical effectiveness of eccentric exercises, most authors reported successfully returning 60%[6-9] of participants back to sport, which contrasts with the 100% reported to have returned to sport in Alfredson's[10] original paper. The lower success rates observed in later trials is thought to be due to a poorer response to isolated eccentric exercises in non-athletic and female individuals when compared with athletic subjects.[11,12] A recent systematic review confirmed the success of eccentric exercises,[13]however, the mechanism by which the effect is achieved remains unclear;[5,11,14,15] and an understanding of this may help the development of an optimal treatment regime and improve patient care.[16]

The most commonly suggested mechanism through which eccentric exercises are believed to have an effect is the application of increased loads to the tendon stimulating structural tendon changes.[17-24] Currently, however, this view has been rejected based on level one evidence[25] and most experts now accept that structural tendon change does not correspond to clinical improvements.[25] The purpose of this clinical commentary is to review previous theories regarding the mechanisms by which eccentric exercise might affect Achilles tendinopathy and offers a novel mechanism by which the plantarflexor muscles may shield the Achilles tendon.

MECHANISM OF EFFECT OF ECCENTRIC EXERCISES

This section will review each of the proposed mechanisms for effect of eccentric exercise on Achilles tendiopathy presented in Table 1 and outline the current evidence for each.

Structural tendon adaptation

Despite the many studies investigating eccentric intervention regimes the mechanism of effect remains in question, although the original concept proposed by Stanish and Curwin[24] and later Alfredson et al[10] still remains the most popular.[36,40] This concept suggested that greater load in the tendon occurs during eccentric training and this stimulates the tendon to undergo structural adaptation, sometimes referred to as "hypertrophic" change.[17-24] In response to this theory several authors have explored tendon change occurring during and after eccentric exercises using magnetic resonance imaging (MRI) and Ultrasound (US).[21,41-46] Tendon structure was shown to have been altered with rehab but this was after extended periods of time, often years.[44,47,48] Further work has shown that eccentric exercises do not place greater strain on the Achilles tendon when compared with concentric exercises suggesting that this mechanism may not be involved.[36] The overall suggestion from current literature and a recent systematic review supports the notion that tendon structure does not significantly change during the treatment period[25,29,42] and that changes do not correspond to improvements in pain or function.[25] When viewed together these results clearly suggest that clinical improvements during rehabilitation occur through a mechanism distinct from structural adaptation.[25,29]

Table 1. *Proposed mechanisims of effect of eccentric exercise on Achilles tendionopathy*

Structural tendon adaptation [10,24]
Tendon length changes[24]
Neuro-vascular ingrowth[23,26,27]
Neuro-chemical alterations[28-30]
Fluid movement [31,32]
Neuromuscular adaptations [33-36,36-39]

Tendon length changes

Several authors have proposed that lengthening the Achilles tendon would help Achilles tendinopathy (AT),[49-51] this premise was based upon observations of reduced active and passive dorsiflexion (DF) in patients with AT.[52-54] It remains unclear why treatment should aim to lengthen the muscle or tendon unit as several prospective studies have shown increased DF to be a risk factor.[55] This includes Kaufman et al's[52] seminal paper. It is known that eccentric exercise regimes may increase DF ROM through sarcomerogenesis, the addition of sarcomeres.[33,56] This addition effectively alters the muscle's length, while

the tendon length remains the same. It is unclear why increasing the length of the muscle tendon unit (MTU) would alleviate tendon pain, and while some suggest that tendon loads may be diminished, there is no corroborating evidence for this suggestion. An alternative explanation is that the addition of sarcomeres shifts the length tension curve thereby allowing the muscle to generate greater force later in its range,[14,56] which would theoretically allow the muscle to decelerate eccentric loads and offload the tendon.[57,58] The concept of muscle(s) being able to offload non-contractile tissue is not new but is infrequently considered in AT.[58] This will be considered in greater detail later in the commentary. Observations of reduced DF in patients with AT may in fact be related to plantarflexor muscle weakness as suggested by Mueller et al[59] in their study assessing DF ROM during the gait cycle. Their data suggest that individuals with reduced plantarflexor torque reduce their DF range of motion, in order to maximize their plantarflexor moment during gait.[59] This effectively suggests that the measurement of DF range of motion is not the issue, and that the real issue may rest with plantarflexor muscle strength (torque/power).

Muscle-tendon unit stiffness

Often associated with length of the tendon is the concept of tendon stiffness (or lack thereof). The current suggestion is that making the muscle tendon unit more flexible (less stiff) is a desirable aim. It is unclear from the current literature why a reduction in tendon or muscle stiffness would benefit the tendon. Several studies have shown reduced tendon stiffness when comparing AT patients with healthy controls.[60-63] Few researchers have examined the effect of eccentrics on stiffness. Mahieu et al[33] tested stiffness of the MTU prior to and after an eccentric training regime and showed both an increase in weight bearing DF ROM and a reduction in passive stiffness of the MTU[33], however this stiffness change was attributed to muscle rather than tendon adaptation. In contrast, Morrissey et al[64] have shown that eccentric exercises increase tendon stiffness. The contrasting results observed by Morrissey et al and Mahieu et al may be a result of differing ankle joint moment calculations and also different sample sizes. Sugiaski and co-authors recently showed that the force of plantarflexor contraction directly influences Achilles tendon stiffness,[65] unfortunately this has not been accounted for in any of the studies measuring or modelling tendon stiffness making it very difficult to accurately understand in-vivo tendon stiffness in healthy or diseased tendons.

Neuro-vascular in-growth

The initial research findings of Ohberg and Alfredson (demonstrated strong correlations between the quantity of neuro-vascular in-growth and pain in subjects' Achilles tendons.[23] Further work showed that obliteration of these vessels through sclerosant injections,[66,67] high volume saline injections,[68-70] or more recently "paratenon scraping" produced excellent clinical outcomes.[71,72] Researchers then examined whether eccentric regimes altered these neo-vessels and reported that after 12 weeks of eccentric exercises the neuro-vascular ingrowth was reduced[23] and the hypothesis was this reduction in neo-vessels directly leads to pain reduction. The mechanism proposed for this was shear forces between paratenon-fascial-tendon layers, which was damaging the microvascular circulation.[23] Recently, authors highlighted that neurovascular bundles in muscles and tendons are important for load transmission, suggesting that a loading modality (eccentric or otherwise) may influence the neurovascular bundle and thereby possibly affect neovascularisation. However, whether this effect is beneficial or not needs further examination.[73] It is important to note later studies determined a lack of correlation between pain and vascularity either as a predictor of recovery[74] or as a direct measure of outcome [27,75] suggesting these measures may be of little use.

NEURO-CHEMICAL INTERACTION

Heinemeier et al demonstrated a dose dependent effect of load on production of chemical mediators in tendon, and that this response does not vary when comparing concentric or eccentric contractions.[76] These neuro-chemical changes occur as a result of alterations in tenocyte activity reducing various chemicals and messenger molecules involved in pain sensitivity (various neurotransmitters).[28,29,77,78]

Attia et al recently showed glucosaminoglycans (GAG's) may be involved in tendon pain.[79] Attia et al found a strong correlation between GAG content and pain and function.[79] Interestingly, laboratory work

has highlighted that increases in GAG concentration leads to mechanical hypersensitivity of nociceptive neurons suggesting a possible pathway for the involvement of GAGs on pain,[80] this sensitivity coupled with the increased number of nerves in pathologic tendons[78,81-84] may explain some of the tissue hypersensitivity clinically observed. Eccentric loading has been shown to reduce the volume of the Achilles tendon more than concentric loading, as measured using MRI.[31] This volume change was thought to be due GAG content and may account for the associated pain reduction observed with loading.[11]

The role that the central nervous system may play in tendinopathy has also been addressed,[85] and animal studies have shown a clear link between tendinopathy in one limb and changes in the opposite limb.[86] Similar findings have also been identified in humans with surgery on one limb improving the contralateral symptomatic tendon.[87] Several authors have proposed that this finding is not related to limb use but rather changes in CNS output.[85,88] How exercise treatments may influence this remain to be investigated and further research is necessary.

Fluid dynamics

Changes in intra-tendon fluid dynamics have been proposed as a possible mechanism of effect for the benefits of eccentric exercise.[31,32] Several authors have demonstrated that eccentric exercise reduces tendon diameter (anterior to posterior thickness), and that this may be related to changes in intra-tendon fluid content.[31,32,85] The results of Grigg et al's work suggests that the change in tendon thickness was less in subjects with tendinopathy compared to a healthy control group.[32] Whether this response is beneficial or predictive of recovery has not been yet been established. However, other groups have shown that the Achilles tendon anterior to posterior (AP) diameter increases with eccentric exercises.[89] The differing results may be related to the mode of assessment, US versus MRI, or issues with reliability of measurements. The findings of reduced volume are difficult to rationalize in the face of studies reporting immediate increases in tendon volume after loading, albeit in tendons other than the Achilles.[90] Further studies need to address what substances (chemicals) are within the tendon and how they may be affected by short term and long term loading.

NEUROMUSCULAR ALTERATIONS

Force fluctuations within the tendon

Force fluctuations are alterations in tendon load occurring during muscle contractions. Many authors offer alternate names such as oscillations or vibrations but effectively they are describing a motor pattern variation that influences tendon load and thereby affects the tendon biochemically. These fluctuations seem to occur more frequently during eccentric than concentric muscle activities.[35-39]

Rees et al[36] and Henriksen et al[37]showed altered neuromuscular forces during eccentric activity in healthy participants but Griggs et al also showed higher levels in patients with AT.[39] Griggs and Rees both suggest that these fluctuations may increase stress on the tendon and lead to advantageous tissue changes,[36] however the reverse may in fact be true, that is, rather than helping recovery motor control issues may lead to structural overload and ultimately tendinopathy. It is important to understand the stretch shortening cycle (SSC) when considering the force fluctuations. The SSC is defined as pre-activated muscle undergoing a lengthening (eccentric) contraction followed by a muscle shortening (concentric) contraction.[91,92] The SSC is associated with tendon lengthening due to its elastic nature, which allows temporary energy storage prior to recoil. During the SSC Achilles tendon forces may reach 9000N (12.5 times bodyweight)[93] and strain (percentage of tendon elongation) is reported as between 4.1-12.8% levels,[93-96] with tissue rupture reported at strains of 9.9% in the only study on the human Achilles tendon.[97] Repetitive SSC's have been shown to lead to tissue failure and rupture of the Achilles with loads that are within in vivo limits.[97] The reason that this may be possible is that muscle activation has been shown to increase tendon stiffness thereby increasing the force required to lengthen the tendon by a given amount effectively reducing tendon (elongation) strain.[65] Studies assessing tendon strain to failure limits have not previously accounted for the muscles ability to affect tendon stiffness and have instead used passive testing protocols, this effectively makes many of the models used to assess tendon function/loading incorrect, which in turns limits the accuracy of the conclusions reached in many of the studies.

Force fluctuations are the result of non-optimal coordination of motor units,[36-39] and these fluctuations appear

to create "mini SSC's" increasing the number of SSC's the tendon is exposed to per loading phase. This phenomenon is likely to increase tendon load and initiate the cellular reaction associated with tendinopathy. There is currently no study that has investigated whether these force fluctuations are altered with eccentric (or other) rehabilitation techniques or whether these fluctuations are associated with other neuromuscular measures such as plantarflexor power.[33,98,99]

Muscle Power

Muscle power deficits have been identified in correlational studies assessing the plantarflexors.[10,62,98,100-104] A prospective study[98] clearly showed that torque below a 50Nm (Newton- meters) was predictive of AT development.[98] Mahieu et al examined army recruits prior to their six-week basic training and tested their concentric strength using isokinetic dynamometry, and determined that a value below 50Nm was 85% sensitive for predicting AT development. Despite the link between muscle power and AT only Alfredson's original paper has used power as an outcome measure and none of these studies have suggested why changes to muscle power may benefit patients with tendinopathy. Alfredson showed a significant loss of power (13.6-23.7% reduction) in the plantarflexors between limbs,[10,100] which was resolved during rehab, but little consideration was given to how this may have influenced tendon pain.[10] Whether the strengthening benefits from eccentrics somehow reduce tendon load or whether it simply changes DF ROM, as discussed earlier, thereby changing tendon load remains to be explored. These two possibilities need further investigation.

STRESS SHIELDING – A NEW PARADIGM

Plantarflexor muscle function has clearly been associated with AT and prospective studies have further supported this cause and effect relationship, but despite this the current literature has failed to offer any reason why muscle weakness may cause tendinopathy or why neuromuscular adaptions may lead to recovery from AT. This section discusses the role of the muscle in controlling tendon forces as a novel paradigm in tendinopathy treatment.

The SSC is one of the key principles associated with tendinopathy and is commonly described as a passive tendon only process,[57] however studies clearly show that the muscle function is essential to the SSC.[57,58,91,105] Work by Komi[91] and Lindstedt et al[57,58] has highlighted that the muscles also function as energy storage systems during the SSC by stiffening. This appears to be due to pre-activation and stretch reflex loops.[91] This pre-activation is a neuromuscular rather than structural adaptation. The suggestion from this data is that muscle function, in addition to tendon characteristics, are essential for efficient functioning during the SSC. Further work has shown that muscle function increases tendon stiffness thereby improving its response to strain.[33,65] This in itself may actually serve as protective mechanism for the tendon, as without muscle activity the tendon is less stiff, equating to a given load bringing about a larger length change in the tendon. Lengthening of a tendon without muscle activity appears to produce higher tendon strains, possibly achieving levels that may be detrimental to the tendon health.[106,107]

Lindstedt et al[58,108] have studied MTU function during SSC contractions and they offer an alternative view to the commonly described model of tendons shielding muscles.[109] Their suggestion is that muscles can function as shock-absorbers, during which the muscle absorbs energy as heat, or they can function as a time dependent spring and increase the elastic recoil potential, reducing energy requirements and heat production. This proposed time dependent spring function can potentially be modified by eccentric training through alterations in neuromuscular function, which may effectively improve the economy of simple SSC tasks like hopping. It appears that hopping frequency is internally controlled within individuals and relates to the frequency that is most energy efficient, there is a strong relationship between body mass and preferred frequency.[110,111] Externally controlled changes to hopping frequency have been shown to increase oxygen demands, effectively showing reduced energy efficiency.[58] Interestingly the work of Linstedt et al showed that eccentric training led to an 11% increase in internally controlled hopping frequency. The observation of increased hopping frequency shows an alteration to the spring function of the muscle, which matches predictions of reducing body mass by 50%.[58] A more efficient spring function of the plantarflexors would increase elastic recoil and reduce absorption of energy (heat) in the Achilles tendon. Heat has been linked to tendinopathy with several studies showing heat shock proteins to be involved in tendinopathy models.[112-115]

The involvement of heat shock proteins suggests tendons heat up during activity; some authors have suggested this reaches levels associated with catabolic activities.[116] This has been further evidenced in Equine tendons with temperature levels reaching as high as 45 degrees Celsius during exercise,[117] a temperature of this level is damaging to tendon cells and is has been proposed as a potential key component of tendinopathy.[114,116,117] Mathematical modelling of human tendons also predict the same temperature changes.[117]

An alternative mechanism links to mechanical load on the tendon and the muscles ability to act as a shock-absorber. During eccentric movements muscles lengthen, towards the end of their range muscle lengthening ceases and the tendon undergoes a stretching period prior to a shortening – the SSC. This function allows the muscle to decelerate the movement prior to maximum tendon strain at terminal dorsiflexion. If the muscle is weak or poorly co-ordinated the muscle appears to undergo a stop: start eccentric type of contraction, identified by Rees et al as force fluctuations and Grigg et al as force frequency characteristics in the 8-12 hz range. This is demonstrated clinically by a physiological tremor or fasciculation. These stop: start contractions expose the tendon to more frequent SSC's during a given action. This un-coordinated eccentric contraction has been observed in healthy subjects[36,37] and shown to be more frequent in AT.[38,39] These neuromuscular co-ordination issues may expose the tendon to repeated SSC's during a single functional movement i.e. walking or running. The amplitude of tendon strain is not yet known during these fluctuations but it may be that either the amplitude or accumulative load is greater than in healthy subjects with normal

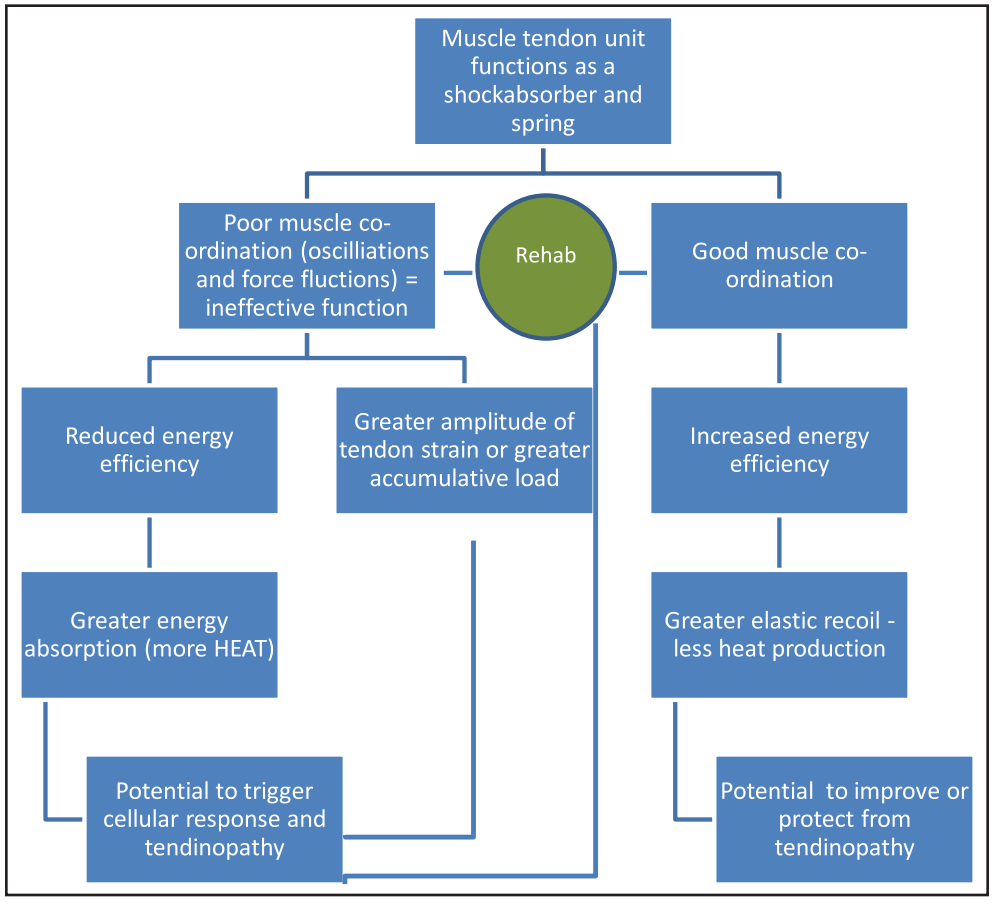

Figure 1. *Flow chart depicting the potential role muscle co-ordination may have in tendinopathy. The MTU as a whole can function as a shock-absorber and absorb energy in the form of heat. Changes in muscle stiffness associated with eccentric training or good pre-existing function leads to less energy absorption and more recovery of energy through elastic recoil, this may protect the tendon by preventing excessive heat absorption/production.*

co-ordination. The greater frequency of these SSC exposures is likely to influence the tendons rate of wear and ultimately its ability to repair. This potentially links to the development and perpetuation of tendinopathy, but may also be a key consideration during rehabilitation. Further work is needed to determine if changes in neuromuscular function are responsible for rehabilitation outcomes and if these oscillations are matched to greater tendon strain or absorption of energy in the form of heat production.

CONCLUSION

Current literature regarding AT suggests that eccentric exercises are effective in the treatment of pain and the restoration of function. There are numerous potential explanations supporting the rationale for effectiveness of eccentric regimes, however many of the suggested explanations have not been fully investigated.

Of the potential mechanisms, changes in neuromuscular output appear to be the most promising but are currently poorly understood and under researched. The changes necessary for benefit may include increased MTU stiffness, increased strength, and shifts in the length tension curve. It is possible that these neuromuscular changes reduce the load on the tendon by "smoothing muscle contractions" (force fluctuations) and thereby reduce maximal or accumulative tendon strain. This may affect tendon homeostasis.

Future research should focus on neuromuscular alterations associated with AT and determine how treatment influences these parameters. Additionally, combining studies of neuromuscular parameters and alterations with investigations of intra-tendon fluid dynamics and bio-chemical interactions would provide a more in-depth understanding of the mechanisms involved. The proposed studies could focus on plantarflexor power (concentric and eccentric), force fluctuations during eccentric movements, shift(s) in the angle of peak torque, and any changes that may occur in the SSC. Further studies would need to determine how neuromuscular changes influence ground reaction force during functional tasks and relate these forces to tendon strain. This data may be important in prospective risk factor studies.

Table 2. *Key points*

Key points:

- Structural tendon adaptation does not correspond to clinical improvements in those with Achilles tendinopathy
- Other possible mechanisms are more likely to explain clinically observable changes than structural adaptation
- Various neuromuscular changes may be responsible for the clinical benefit associated with eccentric exercise

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