

Is compressive load a factor in the development of tendinopathy?

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ABSTRACT

Tendons are designed to take tensile load, but excessive load can cause overuse tendinopathy. Overuse tendinopathy results in extensive changes to the cells and extracellular matrix, resulting in activated cells, increase in large proteoglycans and a breakdown of the collagen structure. Within these pathological changes, there are areas of fibrocartilaginous metaplasia, and mechanotransduction models suggest that this response could be due to compressive load. As load management is a cornerstone of treating overuse tendinopathy, defining the effect of tensile and compressive loads is important in optimising the clinical management of tendinopathy.

This paper examines the potential role of compressive loads in the onset and perpetuation of tendinopathy, and reviews the anatomical, epidemiological and clinical evidence that supports consideration of compressive loads in overuse tendinopathy.

Excess training volume or too much training that uses the elastic function of tendons are the key elements that induce tendon overload and are important factors in the onset of athletic tendinopathy, the clinical syndrome of pain and dysfunction in a tendon. Tendons have evolved primarily to transmit tensile load and have a fibrous tissue structure that accommodates this, hence it is traditionally thought that the nature of the overload is purely tensile. However, some recent papers have provided evidence for compressive load at or near regions where tendinopathy occurs. This concept requires further examination as better insight into the pathoetiology of tendinopathy and the loads that initiate pathology may improve treatment in this common, recurrent and difficult to manage condition.

Almekinders *et al*¹ were the first to consider compression, or a differential in tensile loads, as a concept for overload of tendons. They suggested that strains at the Achilles tendon insertion were not uniform and proposed that the joint side of the tendon was exposed to less tensile load (stress shielded) and may be subjected to compressive loads.² This somewhat complex model of differential strains in a tendon has withstood some scrutiny, but the clinical applications have been limited. Further consideration of aspects of compression in common presentations of tendinopathy and their relation to a clinical paradigm is required.

WHAT IS THE EVIDENCE THAT COMPRESSION AFFECTS TENDON?

Collagen-based connective tissue (fibrous, fibrocartilage, cartilage and bone) can respond to different

types of loads by altering their tissue structure to be suitable for applied loads.³ Gillard *et al*⁴ demonstrated this in uninjured rabbit tendons when they first removed and then reinstated compressive loads, resulting in tissue change from fibrous tissue towards fibrocartilage at the point of compression, with a return of fibrous tissue on removal of compression.

Milz *et al*⁵ also showed that compression can drive adaptive changes within the tendon matrix. Using 3-D reconstruction of the Achilles insertion, areas of fibrocartilage were seen at, and proximal to, the insertion, adjacent to the superior calcaneal tuberosity. The presence of fibrocartilage, proximal to the attachment, supports the concept of compressive forces affecting tenocyte phenotype and therefore tendon structure.⁶

These adaptive responses are driven by tendon cells, which respond to cyclic compression by becoming more rounded (chondrocytic) and by expressing large proteoglycans such as aggrecan.^{7,8} Many authors have reported an increase in the deposition of aggrecan and type II collagen in the areas of compression within tendons,⁶ notably on the deep surface of tendons adjacent to a bony prominence. Biomechanical modelling⁹ and *in vitro* cell cultures subjected to mechanical stimuli¹⁰ strongly implicate hydrostatic pressure as the stimulus for this response. These changes are also described at the deep surfaces of most entheses where compression is a feature.

WHAT IS THE RELATIONSHIP BETWEEN TISSUE ADAPTATION TO COMPRESSION AND TENDINOPATHY?

The cell and matrix changes in tendinopathy, seen when tendons are examined after surgery for recalcitrant symptoms, are extensive. Normal tendon is fibrous tissue with a highly structured type I collagen-based extracellular matrix with minimal cells and neurovascular structures. Tendon pathology is a more cellular tissue with substantial matrix changes including increased amount of large aggregating proteoglycans⁸ with a very high turnover,¹¹ a change in collagen type (type III) with increased collagen turnover and disorganisation, as well as neurovascular ingrowth.¹²

These changes with pathology are similar to those in the fibrocartilage (fibrocartilaginous metaplasia),¹³⁻¹⁴ and this is a universal response regardless of the site.¹⁵ Mechanotransduction models¹⁶ would suggest that this is a result of some form of compressive load being placed on the tendon, as compressive loading in soft

connective tissues tends to favour fibrocartilage formation.¹⁷ There are, however, some differences between tendon pathology and fibrocartilage (table 1).

Compressive loads have been shown to induce tendon pathology. Soslowky *et al*¹⁸ investigated the effect of different loads on rat supraspinatus tendon and examined the effect of compressive load, tensile load and the combination of compressive and tensile load.¹⁹ They showed that compressive load (by interposing tissue between the tendon and acromion) in itself had minimal effect in the tendon, tensile load (running downhill) was clearly detrimental, but the combination of loads was especially damaging to the tendon. Increased cross-sectional area and decreased mechanical properties were maximal in tendons exposed to both compressive and tensile loads.

ARE THERE COMPRESSIVE LOADS WHERE TENDON PATHOLOGY OCCURS?

Although tendinopathy is the most common diagnosis, nearly all clinical tendinopathy occurs at, or adjacent to, the bone–tendon junction, hence this should be more accurately called an enthesopathy. The normal tendon attachment will transition from tendon through fibrocartilage to mineralised fibrocartilage to bone over a relatively short distance (<2 mm).²⁰ However, additional structures proximal to the insertion have an active role in the transmission of force from the tendon to the bone. Benjamin *et al*²¹ have provided key insights into the structures and the loads around the fibrocartilaginous tendon attachments, and called the summation of the insertion and these additional structures, ‘the enthesis organ’. The key characteristics of the organ are as follows: the tendon will insert into the bone at an angle after a bony prominence, that there is often a bursa between the tendon and the bone, and fibrocartilage is expressed on the opposing bone and tendon surfaces to absorb the compression of the tendon against the bone (figure 1). The enthesis organ has two functions: compression of the tendon against the bone reduces tensile load on the insertion and it confers a mechanical advantage to the muscle–tendon unit.²¹

In examining the loads on the enthesis organ, especially proximal to the insertion, these areas of the tendon are nearly always exposed to both tensile and compressive loads, especially in some joint positions, suggesting that this may predispose to pathology at the bony prominence before insertion. Although this paper discusses compression, it is important to acknowledge that the load in the tendon near an enthesis will vary through its thickness and length and essentially be a combination of tensile and compressive loads rather than solely compressive load. For simplicity, we will continue to discuss it as ‘compression’.

Importantly, many clinical presentations of tendinopathy at the bone–tendon junction occur at, or adjacent to, the area of compression of the tendon against the bone proximal to the

insertion, not at the actual insertion of the tendon to the bone. Abnormal clinical and imaging findings are seen at the site of compression proximal to the tendon insertion,^{22,23} and strongly suggest that compression where bone and tendon approximate are important considerations in the onset of tendinopathy. Pathology prior to the insertion fits a number of problematic tendons, and the compression may be immediately proximal to, or more removed from, the insertion (table 2). Some tendons have a fixed bony prominence proximal to the insertion, others have a prominence that is modified by movement and effective in some joint positions, while others still have a movement modified prominence that is external to the bone–tendon muscle continuum and is prominently dependent on movement of other joints.

Despite the concept offering insight into many clinical presentations, there are several tendons that lack a bony prominence and compression is unlikely to have an important role in the onset of tendinopathy. These include the flexor tendons of the forearm that insert into the medial epicondyle of the humerus and the insertion of the patellar tendon into the patella. The lateral elbow enthesitis is discussed below as it has a bony prominence only in some forearm positions.

HOW DOES THIS RELATE TO TENDINOPATHY?

Regions of tendons that anatomically abut a bony prominence such as the tibialis posterior at the medial malleolus have been examined to better understand the fibrocartilaginous metaplasia within these regions that occurs adjacent to the bone.^{9,24} Wren *et al*²⁵ used a poroelastic model to propose that zones of compression have low-fluid permeability because of the strong water binding properties of aggrecan, which in effect restricts fluid flow and serves to protect the solid components within the matrix, presumably the cells and collagen. Conversely, they propose the tensile regions have a feature of higher fluid permeability, and are less well suited to high cyclic compression loads.

Between the compressive (fibrocartilage adjacent to the bone) and tensile (fibrous tissue in the region removed from the bone) zones of the tendon that abut a bone is a transition zone with graduated features of both. This gradation from compressive to tensile morphology occurs from the deep to the superficial aspects of the tendon. Along the deep aspect of the tendon, this transition occurs proximally, and in some cases distally.

It is possible that the transition zone is affected in the development of tendinopathy in the following manner. Excessive loading has the propensity to partially deplete the bound water normally present in the tensile and transitional zones. This movement of water has been postulated by Grigg *et al*²⁶ who reported a loss in tendon diameter in the tensile zone of the Achilles mid-tendon when the tendon was subjected to repeated eccentric load.

Table 1 Similarities and differences between normal tendon, fibrocartilage and tendon pathology⁵⁸

	Normal tendon	Fibrocartilage	Pathological tendon
Cells	Few spindle-shaped cells	No cell proliferation Cells rounder	Cell proliferation Cells rounder, more endoplasmic reticulum
Proteoglycans	Minimal mostly decorin and biglycan	5–10 times more than in tensile tissue, mostly aggrecan	3 times more than tensile tissue, 25x metabolic rate of normal tendon ¹¹ Biglycan and aggrecan increase, decorin maintained ⁸
Collagen	Predominately type I	Type I and II	Type I collagen, some type II, substantial increase in type III collagen
Collagen structure	Ordered collagen network	Ordered collagen network	Disorganised collagen network
Vascularity	Minimal	None to minimal	Variable but can be abundant

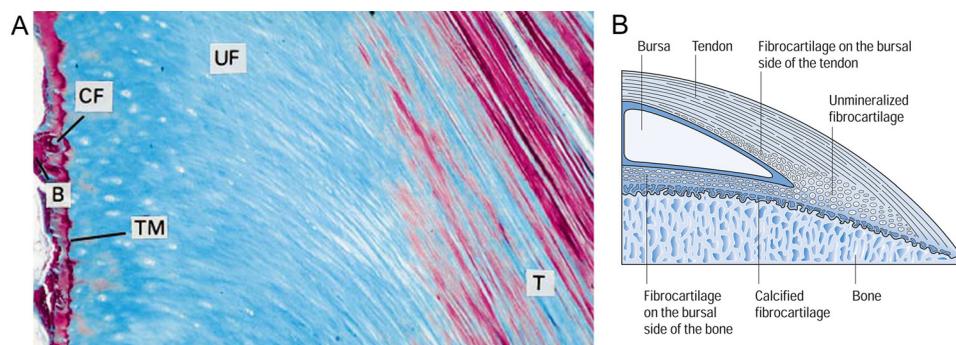


Figure 1 (A) Tendon attachment to bone. The collagen fibres in the tendon (T) are angled on approach, transitioning through fibrocartilage (UF) before attaching to the bone (B) through calcified fibrocartilage (CF). Masson's trichrome, x60. Reproduced with permission from Benjamin and Ralphs, *Journal of Anatomy* 1998 (193): 484. (B) Tendon attachment to bone and the enthesis organ. Schematic demonstrating the complex nature of the tendon attachment to the bone and the fibrocartilage where the tendon and bone are adjacent. This figure was published in *Physical Therapies in Sport and Exercise* Kolt and Snyder-Mackler (Eds) Tendon: 30, Copyright Elsevier (2007).

Table 2 The compressive anatomy of tendons susceptible to enthesopathy

Tendon	Anatomical site of compression	Position of compression
Achilles insertion	Superior calcaneus	Ankle dorsiflexion
Tibialis posterior	Medial malleolus	Anatomically permanent pivot
Biceps long head	Bicipital groove	Shoulder extension
Supraspinatus	Greater tuberosity	Shoulder adduction
Hamstring (upper)	Ischial tuberosity	Hip flexion
Gluteus medius and minimus	Greater trochanter	Hip adduction
Adductor longus/rectus abdominus	Pubic ramus	Hip abduction/extension
Peroneal tendons	Lateral malleolus	Anatomically permanent pivot
Quadriceps	Femoral condyle	Deep knee flexion
Pectorals	Humeral tuberosity	External rotation

The loss of water may expose the tenocytes in these regions to greater intrinsic cyclic compressive load. This may provoke the tenocytes to respond through the synthesis of large water binding proteoglycans, a process well described in tendinopathy and known to occur within days.¹¹ As described by Wren, this would reduce the permeability of this region of the tendon tissue, thereby protecting against further insult. Further loading of this swollen region however, may perpetuate the response and result in a failure to achieve equilibrium.²⁷

Some validation for the transition zone as being the area affected is provided by Scott *et al*²⁸ who induced supraspinatus tendinopathy in the rat. The authors described the typical features of tendinopathy (cell rounding, aggrecan deposition) extending beyond the normal fibrocartilaginous zone of insertion, both proximally and superficially into regions normally occupied by normal spindle shaped tenocytes.

HOW DOES THIS CONCEPT HELP CLINICALLY?

Common aggravating activities for insertional tendinopathy can be somewhat explained by this concept. For example, load in dorsiflexion such as walking bare foot or on sand is aggravating for an Achilles tendon insertion problem. Patients with an Achilles insertional tendinopathy may have no pain when hopping on their toes, but complain of pain with Achilles tendon load in ankle dorsiflexion (eg, push-off). Similarly, wearing shoes with a substantial heel raise will help decrease pain in the Achilles insertion.²⁹

Table 3 Clinical options to reduce compression

Tendinopathy	Modification	Effectiveness
Achilles insertion	Heel raise	Effective
Tibialis posterior	Orthotics and heel raise	Limited
Hamstring (upper)	Limit sitting/ lunging	Moderate
Gluteus medius and minimus	Lumbopelvic control, sleep supine	Effective
Adductor longus	Limit loads in abduction/extension	Moderate
Peroneal tendons	Heel raise	Limited
Quadriceps	Limit loads in deep knee flexion	Moderate

Load on the gluteal tendon in hip adduction such as walking with poor lumbopelvic stability, induces a similar compressive load and often provokes a painful response. Although Soslowsky reported minimal impact from solely compressive loads,¹⁹ clinically there appears to be pain associated with sustained, compressed positions. For example, sleep disruption from lying on the affected side is a hallmark sign for those with gluteal tendinopathy³⁰ as is sitting for those with hamstring tendinopathy.³¹

Stretching might be provocative in these enthesal tendinopathies where there is some tension in the muscle as well, such as stretching the Achilles insertion over the edge of a step. Overall, it is probably better to manage muscle compliance and length with massage techniques rather than stretching in tendinopathy that has a compressive element.

Consideration of compression at the bony prominence proximal to the insertion can help explain the sexual dimorphism of pelvic tendinopathies. Clinically, it is well known that older women are five to six times more likely than men to get gluteal tendinopathy³² and men are similarly over-represented in adductor tendinopathy.³³ Applying the enthesis organ and compression model to these tendinopathies, it is possible that increased neck of femur angles will increase the resultant compression of the tendon against the greater tuberosity in women more than in men. Fearon³⁴ demonstrated the resilience of this concept in the study which reported that women with gluteal tendon tears had greater coxa vara than women with severe osteoarthritis of the hip. Conversely men have to be in considerably less abduction/extension before there is compression of the adductor longus/rectus abdominus tendon complex against the pubic ramus; this may in part explain why men are more likely to get adductor tendinopathy than women.³⁵

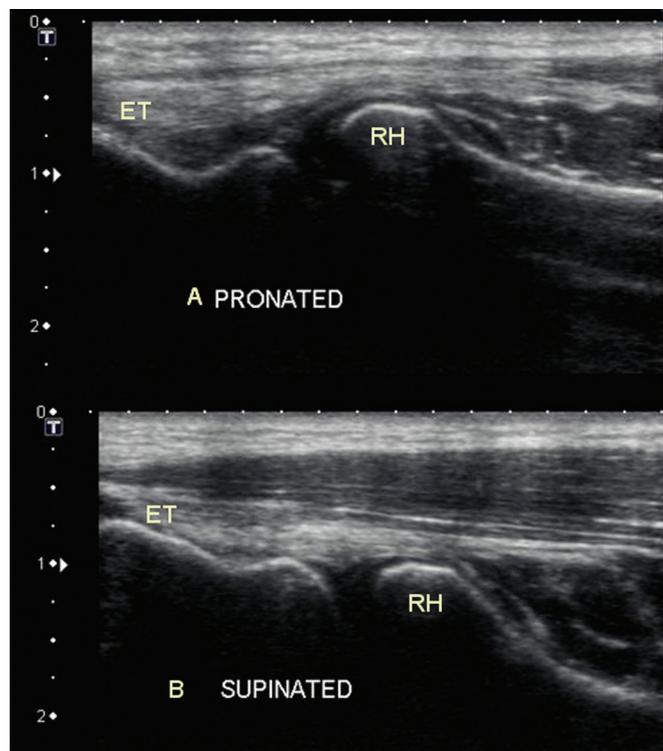


Figure 2 The radial head in pronation (A) and supination (B) providing some protection of the insertion in pronation but not supination. ET, extensor tendon, RH, radial head.

WHAT ABOUT TRUE ENTHESOPATHIES?

There are several tendons that have no bony prominence, and the tendon pathology occurs solely at the bone–tendon junction, these include the medial elbow and the patellar tendon (although some may argue that the patellar tip acts as a prominence).³⁶ These tendons and their entheses are primarily subject to tensile load, so it is interesting that the primary changes in pathology are exactly those seen at the other insertional tendinopathies and are histologically closer to the fibrocartilage than the fibrous tissue.¹⁵

The lateral elbow also suffers mostly from a true enthesopathy, however the enthesis organ is more complex. The enthesis has a variable bony prominence in the radial head, which appears to act as a bony prominence in forearm pronation and mid-prone, but not in supination (figure 2). This may be an anatomical adaptation, as maximal extensor muscle loads generally occur in the pronated position. In this position, the tendon will be compressed against the radial head to reduce load on the insertion. However, in supination there is no protection for the enthesis of the extensor muscles from such a prominence. This is functional, as the main muscle loads in supination are usually placed on the flexors (as in picking up objects with palm up). However, large extensor muscles load in supination, such as gripping with repeated pronation and supination, places a direct load on the enthesis. This is seen clinically when lateral elbow tendinopathy presents after repeated pronation and supination such as heavy use of a screwdriver or wringing out clothes.

WHAT ABOUT THE ACHILLES MID-TENDON?

A tendon where the tensile load predominates is the mid-substance of the Achilles tendon, which is exposed to very high tensile and elastic loads. The loading explains many

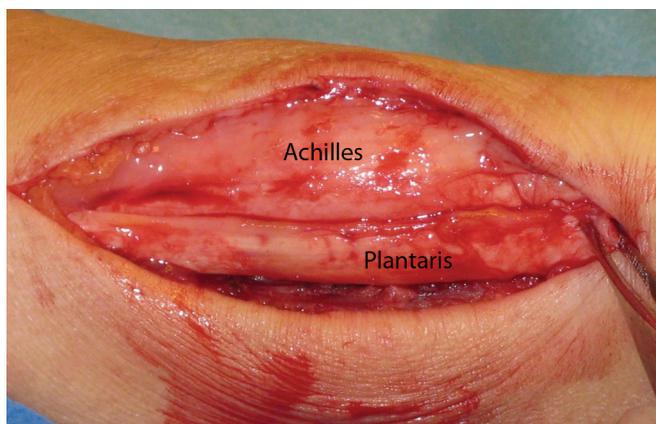


Figure 3 The relationship between the Achilles and plantaris tendon.

populations who succumb to Achilles tendinopathy; the badminton player,³⁷ the sprinter and the distance runner.³⁸ However, there are many people who succumb to tendinopathy in the mid-Achilles that do not have substantial tensile tendon overload, in fact it is not uncommon in sedentary people.³⁹

There is potential for some compressive loads even in the mid-tendon, Almekinders *et al*¹ discuss internal shear due to differential forces on the posterior and anterior aspects of the tendon. An additional internal tendon shear force may come from the differential contribution of the gastrocnemius and soleus fibres to the Achilles tendon.^{40–41} Recent publications suggest that the plantaris tendon may also apply a compressive force in some athletes, where the plantaris tendon is invaginated into the Achilles (figure 3).⁴² As the plantaris tendon is stiffer,⁴³ it is suggested that it places a shearing or compressive load on the Achilles especially, in dorsiflexion/eversion, creating tendinopathy in either or both the Achilles and the plantaris. This subset of Achilles tendinopathy may explain why raising the height of the heel in the shoe can have such a variable effect on mid-Achilles pain, perhaps being very helpful in those with an invaginated plantaris tendon and less so (or not at all) for those who solely have a mid-Achilles tendinopathy.

An alternative explanation that invokes a purely compressive load is the role of the posterior retinaculum.²⁰ Precedence for this may be found in the retinacular metaplasia and consequent compressive load on the tendon as the causal factor in de Quervain's tendinopathy⁴⁴ and trigger finger.⁴⁵ The Achilles tendon bowstrings over the retinaculum when the ankle is plantarflexed and this may explain how those who are not active at all or use minimal elastic and tensile loads can succumb to Achilles tendinopathy.^{46–47}

HOW DOES THIS CONCEPT HELP CLINICAL UNDERSTANDING?

The tendon insertion and the enthesis organ are well engineered to absorb most functional loads, and will adapt over time to higher levels of load. However, these tissues are slow to adapt to high loads and slow to resolve after insult. Overload may trigger a tendinopathic response and mere abolition of the overload in the short term is usually insufficient to resolve the tendinopathy and return the athlete to full function.

Load management in tendinopathy has primarily relied on reducing the volume and/or intensity of the training load, as well as amendment of any aberrant biomechanical issues.

Reducing compressive loads in insertional tendinopathies provides an important further unloading strategy for the sensitised tendon (table 3). Reduction of compressive loads is often simple, such as changing training strategies, reducing stretching or adding a heel raise, and an athlete with minimal pain may continue some training loads with these interventions.

Complete rest from tensile loads for a tendinopathy is contraindicated as it can decrease mechanical strength of the tendon,⁴⁸ and total removal of load can induce tendinopathic changes due to the lack of a mechanical stimulus.⁴⁹ Subjecting tendons to tensile, moderate isometric loads while protecting against compression may improve recovery. This has been demonstrated by Jonsson *et al*⁵⁰ who applied tensile load through limited joint range that reduced the compression of the calcaneus on the tendon in dorsiflexion. Using full range of ankle movement in eccentric exercise was successful in only 30% of those with insertional Achilles tendinopathy,⁵¹ whereas by limiting dorsiflexion to plantargrade only in the exercise regimen, 70% reported improvement.⁵⁰

DISCUSSION

It is well documented that the tendinopathic area is located on the deep surface of most tendons at, or close to, the bone-tendon junction. This has appeared incongruous with the tensile overload theory because there is less elongation in this region than in the superficial portion of the tendon.^{2 52} The potential role of compression of the deeper transitional layers (and deep proximal/distal fibres of the tendon against the bone) that results in pathology may provide an explanation for this finding. However, very little work to date has focused on the tendon enthesis or the transitional zones within the enthesis. Further understanding of the load parameters required to provoke or inhibit tendinopathy changes in this zone is required. Further more, the complementary effects of a range of cytokines, in particular transforming growth factor β and insulin-like growth factor 1, in this process, and perhaps in time the inhibition of these at appropriate phases of the condition, should also be considered.^{28 53}

The pathoetiology of tendinopathy is still not well understood, and many models have been proposed. Some of these models propose that tensile overload tears collagen that results in tendon pathology.^{54 55} If compressive loads have an important role in the development of tendinopathy then these models are left lacking, and models that propose a cell-led response to load appear more robust.⁵⁶ This may be especially true of the insertional tendinopathies, where combinations of tensile and compressive loads exist.

Compressive overload may also explain the resistant nature of these tendinopathies. As the matrix changes and the tendons swell, the compression is higher and likely to occur earlier in joint range, making the condition progressive. The deposition of aggrecan (and its breakdown products)¹¹ and this association with a decreased permeability of the matrix may also explain the slow resolution. In essence, the perpetuation of swelling in tendinopathy may in itself create a space-occupying lesion, further increasing the internal compression stimulus on the matrix.

The changes in the tendon collagen should also be considered, as tendinopathy is widely reported to increase the amount of type III collagen deposited in the matrix. An interesting corollary to this hypothesis of compression is that although type II collagen has been identified in the normally

appearing fibrocartilage subjected to compressive loads in the enthesis it has been reported only occasionally in tendinopathy.⁵⁷ Further investigation of this collagen type is required as it relates to cell lineage, mechanotransduction elements, load parameters and attendant cytokines.

CONCLUSION

Although the science is incomplete in substantiating a role for compression in the typical tendinopathies encountered in clinical practice, we have endeavoured to provide a cellular, biomechanical and clinical level for such a hypothesis to improve understanding and management of tendinopathy. Further research of the effects of provocative loading on the enthesis would be valuable. This needs to be complemented with clinical trials that examine the effect of reducing compression in the management of tendinopathy. Consideration of enthesal or external compression within the pathoetiological and management paradigm may improve understanding and management of tendinopathy.

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