Biomechanics and Pathophysiology of Overuse Tendon Injuries
Ideas on Insertional Tendinopathy

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Abstract

Tendons behave viscoelastically and exhibit adaptive responses to conditions of increased loading and disuse. High-resolution, real-time ultrasound scanning confirms the applicability of these findings in human tendons in vivo. In addition, recent biomechanical studies indicate that strain patterns in tendons may not be uniform, as tendons show stress-shielded areas and areas subjected to compressive loading at the enthesis. These areas correspond to the sites where tendinopathic characteristics are typically seen. This indicates that some tendinopathies may, paradoxically, be considered as ‘underuse’ lesions despite the common beliefs that they are overuse injuries. Classic inflammatory changes are not frequently seen in chronic athletic tendon conditions and histopathology features in tendinopathic tendons are clearly different from normal tendons, showing an exaggerated dysfunctional repair response. Tendinopathies are traditionally considered overuse injuries, involving excessive tensile loading and subsequent breakdown of the loaded tendon. Biomechanical studies show that the strains within the tendons near their insertion site are not uniform. If the material properties are
similar throughout the tendon, forces transferred through the insertion site preferentially load the side of the tendon that is usually not affected initially in tendinopathy. In that case, the side affected by tendinopathy is generally ‘stress shielded’. Thus, the presence of differential strains opens the possibility of alternative biomechanical explanations for the pathology found in these regions of the tendon. The traditional concept of tensile failure may not be the essential feature of the pathomechanics of insertional tendinopathy. Certain joint positions are more likely to stress the area of the tendon commonly affected by tendinopathy. Incorporating different joint position exercises may exert more controlled stresses on these affected areas of the tendon, possibly allowing better maintenance of the mechanical strength of that tendon region and, therefore, prevent injury. Such exercises could stress a healing area of the tendon in a controlled manner and thus stimulate healing once an injury has occurred. Additional work is needed to prove whether such principles should be incorporated in current rehabilitation techniques.

Tendinopathies are common in elite and recreational athletes. Although acute traumatic conditions such as ligament and muscle tears receive much attention in the general literature, tendinopathies account for much of the lost time in practice and competition.[1,2] The aetiology and much of the treatment of tendinopathy is largely based on our understanding of the biomechanical behaviour of tendons. This article reviews studies on the biomechanical behaviour of tendons. An understanding of tendon biomechanics is needed to understand the clinical features of tendinopathy and may allow us to move forward with new and innovative approaches to this common problem.

1. Functional and Clinical Relevance of Tendon Tensile Behaviour

The primary role of tendons is to transmit contractile forces to the skeleton to generate joint movement. In doing so, however, tendons do not behave as rigid bodies but exhibit a time-dependent extensibility. This has important implications for muscle and joint function, as well as for the integrity of the tendon itself.

Firstly, the elongation of a tendon during muscle contraction will result in muscle shortening. For a given contractile force, a more extensible tendon will allow greater muscle shortening. The outcome of the resultant extra sarcomeric shortening on the contractile force elicited would depend on the region over which the sarcomeres of the muscle operate. If the sarcomeres operate in the ascending limb of the force-length relation, a more extensible tendon will result in less contractile force. In contrast, if the sarcomeres operate in the descending limb of the force-length relation, a more extensible tendon will result in greater contractile force.[3]

Secondly, a non-rigid tendon may complicate the control of joint position.[4] If one considers, for example, an external oscillating force applied to a joint at a certain angle, trying to maintain the joint at a fixed position would require the generation of constant contractile force in the muscle. If the tendon is very compliant, its length will be changed by the external oscillating load, even if the muscle length is held constant. This will result in failing to maintain the joint at the desired angle.

Thirdly, the work done to stretch a tendon is stored as elastic energy and most of this energy is recovered once the tensile load is removed and the tendon recoils. This passive mechanism of energy provision operates in some of the tendons of the lower extremity of legged mammals during terrestrial locomotion, thus saving metabolic energy otherwise needed to displace the body ahead.[5] Energy is also ‘dissipated’ in the form of heat, but this effect is small and does not endanger the integrity of a tendon in a single stretch-recoil cycle. However, in tendons that stretch and recoil repeatedly under physiological conditions (for example, again, the lower-extremity tendons of legged mammals during terrestrial locomotion), the heat lost may result in cumula-
tive tendon thermal damage and injury, predisposing the tendon to ultimately rupture. Indeed, in vivo measurements and modelling-based calculations indicate that spring-like tendons may develop during exercise temperature levels above the 42.5°C threshold for fibroblast viability. These findings are in line with the degenerative lesions often observed in the core of tendons acting as elastic energy stores, indicating that hyperthermia may be involved in the pathophysiology of exercise-induced tendon trauma.

To quantify the tensile behaviour of tendons and assess the above effects, numerous in vitro and in vivo studies have been performed.

1.1 In Vitro Testing

The mechanical properties of tendons have been studied mostly using tensile testing methodologies in which isolated tendon specimens are stretched by an external force, while both the specimen deformation and the applied force are recorded. Such methodologies are considered to adequately mimic the way that loading is imposed on several tendons in real life.

The tensile tests produce a force-elongation curve (figure 1). In force-elongation curves, slopes relate to stiffness (N/mm) and areas to energy (J). In elongation-to-failure conditions, four different regions can be identified in the tendon force-elongation curve (figure 1). Region I is the initial concave portion of the curve and it is referred to as the tendon ‘toe’ region. Loads within the ‘toe’ region elongate the tendon by reducing the crimp angle of the collagen fibres at rest, but they do not cause further fibre stretching. Hence, loading within the ‘toe’ region does not exceed the tendon elastic limit. Further elongation brings the tendon into the ‘linear’ region II, in which elongation results from stretching the already aligned fibres by the load applied in the preceding ‘toe’ region. At the endpoint of this region, some fibres start to fail. Elongation beyond the ‘linear’ region brings the tendon into region III, where additional fibre failure occurs in an unpredictable fashion. Further elongation brings the tendon into region IV, where complete failure occurs.

To account for inter-specimen dimensional differences when interpreting force-elongation curves, tendon forces are reduced to stress values (MPa) by normalisation to the tendon cross-sectional area and tendon elongations are reduced to strain values (%) by normalisation to the tendon original length. The stress-strain curve obtained is similar in shape to the force-elongation curve, but it reflects the intrinsic material properties rather than the structural properties of the tendon specimen. The most common material variables taken from a stress-strain curve under elongation-to-failure conditions are the Young’s modulus (GPa) and the ultimate stress (MPa) and strain (%). Young’s modulus, the product of stiffness multiplied by the original length-to-cross-sectional area ratio of the specimen, reaches 1–2 GPa at stresses exceeding 30 MPa. Ultimate tendon stress (i.e. stress at failure) and strain (i.e. strain at failure) reach ~100 MPa and 4–10%, respectively.

If a tendon is subjected to a tensile load, it does not behave perfectly elastically, even if the load applied is lower than that required to cause failure. Because of the time-dependent properties of the tendon’s collagen fibres and interfibre matrix, the entire tendon exhibits force-relaxation, creep and mechanical hysteresis. Force-relaxation means that the force required to cause a given elongation decreases over time. The decrease in force follows a predictable curvilinear pattern (figure 2). Creep is the analogous phenomenon under constant-force conditions. In this case, deformation increases over time curvilinearly (figure 2). The presence of mechanical hysteresis is evidenced in force-elongation and stress-strain plots during loading and subse-

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Fig. 1. Typical force-elongation curve of a tendon pulled by a load exceeding the tendon elastic limit. I = toe region; II = linear region; III and IV = failure regions.
the amount of elastic strain energy lost as heat upon recoil and it is usually expressed in relative terms (%) with respect to the total work done on the tendon during stretching. Mechanical hysteresis values in the range of 5–25% have been reported, with most values concentrated around the value of 10%. [14-16,19,20]

Several factors may alter the mechanical response of tendons, the most important of which are discussed in the following sections.

1.2 Disuse

Most studies show that limb immobilisation, suspension and denervation decrease tendon stiffness, ultimate strength and energy-to-failure. [8,21-24] These changes are attributed to specimen atrophy and changes in the specimen material properties. Disuse-induced changes in intrinsic material properties are associated with increased collagen turnover and reducible cross-linking, decreased glycosaminoglycan and water content, and increased non-uniform orientation of collagen fibrils. [8,12,13,21,23,25,26]

1.3 Physical Activity

Most of the studies report that long-term physical activity improves the tensile mechanical properties of tendons, yielding results opposite to those of disuse. [8,21,24,27,28] Hypertrophy may partly account for these effects, but changes in the tendon Young’s modulus also indicate training-induced changes in the tendon intrinsic material properties. Such biochemical and structural changes include increased glycosaminoglycan content, decreased collagen reducible cross-linking and increased alignment of collagen fibres. [8,12,13,21,23,25,26]

1.4 Aging

Some cross-sectional studies have shown that aging may result in intrinsically stiffer, stronger and more resilient tendons. [15,29] while others have challenged these results. [30-33] This inconsistency may be partly accounted for by inter-study differences in the age of the younger group examined, with comparative results involving very young animals, [15] potentially representing the effect of biological maturation and development rather than aging.

Fig. 2. Force-relaxation (a), creep (b) and mechanical hysteresis (c). The arrows on the bottom graph indicate loading and unloading directions.
1.5 Corticosteroids

Corticosteroids have frequently been used for the treatment of tendinopathy, despite the dubious rationale for their use in these conditions.[34] Intra-articular and intracollagenous injections of corticosteroid may reduce the stiffness, ultimate stress and energy-to-failure of collagenous tissue, predisposing the user to tendon injuries even after short-term administration.[35-37]

1.6 Limitations of In Vitro Testing

Although in vitro tests clearly show that tendons behave viscoelastically on tension, reference to the quantitative results of such tests when interpreting in vivo function should be treated with caution. This is because: (i) the forces exerted by maximal tendon loading under in vivo conditions may not reach the ‘linear’ region in which stiffness and Young’s modulus measurements are taken under in vitro conditions; (ii) to perform a tensile test in vitro, clamping of the specimen is necessary. Fixing a fibrous structure with clamps is inevitably associated with fibre slippage and/or stress concentration that may result in premature rupture; (iii) many in vitro experiments have been performed using preserved tendons, which may have altered properties.[38,39]

Some of the above problems have been circumvented by testing animal tendons in situ, after the animal has been killed or anaesthetised.[40,41] This has been achieved by surgically releasing the tendon from its surrounding tissues, maintaining the proximal end of the tendon attached to the in-series muscle and gripping the distal bone of the muscle-tendon unit with a clamp interfaced to a load cell. The in situ muscle preparation is made to contract by electrical stimulation, thus pulling on the tendon, which lengthens as a function of the contractile force applied to its proximal end in a similar fashion to that obtained when the actuator of a tensile machine pulls an isolated specimen (figure 3). Clearly, however, such in situ protocols are inapplicable to humans. To overcome this problem, the development of an in vivo non-invasive method is needed. This would allow the design of longitudinal studies aimed to answer fundamental questions with clinical implications. For example, which training regime is most effective in enhancing the mechanical properties of a tendon? Also, how long does it take before the mechanical properties of an immobilised tendon start to deteriorate? Moreover, an in vivo method might be of direct clinical use. Consider, for example, surgical procedures involving limb lengthening and tendon transfer. Any change in the resting-state length of the tendon will alter its extensibility and this will clearly affect the function of the in-series muscle. Therefore, protocols for assessing the mechanical properties of a tendon in vivo could provide crucial help for optimising the outcome of a corrective surgery. For example, they could be used for measurements in the tendon of the contralateral healthy limb of a patient, thus providing reference values that could then be used to guide clinical decision making.

1.7 In Vivo Testing

Adapting similar principles to those used under in situ material testing has recently allowed the development of a non-invasive method for assessing the mechanical properties of human tendons in vivo. The in vivo method is based on real-time ultrasound scanning of a reference point along the tendon during static contraction of the in-series muscle (figure 4). The limb is fixed on the load cell of a dynamometer to record changes in joint moment occurring during activation and subsequent relaxation in a maximal isometric contraction. The joint moments recorded correspond to muscle forces that can be calculated from the moment equilibrium equation. The muscle forces generated by activation pull the tendon proximally and cause a longitudinal deformation, which is measured by the recorded displace-
ment of a reference landmark in the tendon. On relaxation following activation, the tendon recoils and the reference landmark traced shifts distally (figure 4). The force-elongation plots obtained during loading-unloading can be reduced to the respective stress-strain plots by normalisation to the dimensions of the tendon, which can also be measured using ultrasonography. Coefficient of variation values of <12% have been obtained in repeated measures using the in vivo method.[42-49]

By following the above steps, we showed that: (i) young adult Achilles and tibialis anterior tendons have similar values of Young’s modulus (~1.2 GPa) and mechanical hysteresis (18%);[42-44] (ii) Achilles tendons in young adults are liable to increasing elongations in the initial few cycles or repeated loading-unloading.[50,51] (iii) the Achilles tendons of older people (69–80 years) are intrinsically more compliant by 14% than the same tendons in young adults.[52] (iv) 90 days of bed rest reduces the Young’s modulus of Achilles tendons in young adults by ~30%.[53] (v) strength training in old age (68–79 years) increases the Young’s modulus of the patellar tendon by ~70%, which results in increasing the rate of force development by 27% and decreases the mechanical hysteresis of the tendon by 27%.[54,55]

In interpreting finding (i), it should be stressed the tibialis anterior and Achilles tendons are subjected to different physiological forces. The tibialis anterior tendon is subjected to the forces generated by controlling plantarflexion in the early stance phase of gait and the Achilles tendon to the high forces generated in late stance. In vivo measurements of tendon force indicate that the Achilles tendon may carry up to 110 MPa in each stride during running.[56] This stress not only exceeds the tendon stress during maximal muscle contraction in our experiments (~30 MPa), but it also exceeds the average ultimate tensile stress of 100 MPa reported, thus highlighting the possibility of Achilles tendon rupture in a single pull in real life. Epidemiological studies of spontaneous tendon rupture verify these theoretical considerations.[57] Another difference between the two tendons concerns their ability to provide mechanical work. In contrast to the tibialis anterior tendon, the Achilles tendon acts as an energy provider during locomotion. Most of the work im-

![Fig. 4. Typical in vivo sonographs of the human tibialis anterior (TA) tendon. The white arrows point to the TA tendon origin. The black double arrows point to the shadow generated by an echoabsorptive marker glued on the skin to identify any displacements of the scanning probe during muscle contraction-relaxation. The tendon origin displacement is larger during relaxation compared with contraction at each loading level, indicating the presence of mechanical hysteresis in the tendon (reproduced from Maganaris and Paul,[43] with permission). (a) resting state; (b) 40% of maximal loading during contraction; (c) 80% of maximal loading during contraction; (d) 100% of maximal loading during contraction; (e) 80% of maximal loading during relaxation; (f) 40% of maximal loading during relaxation; (g) 0% of maximal loading during relaxation.](image-url)
posed on the tendon by the initial ground force is recovered as elastic strain energy during push-off, which results in plantar-flexing the ankle and thus accelerating the body forward at no metabolic cost.\[^{[5]}\] Notwithstanding the above differences between the two tendons, the Achilles tendon was found to be neither intrinsically stiffer nor more rebound resilient than the tibialis anterior tendon, which agrees with \textit{in vitro} findings.\[^{[16,58]}\] Thus, it seems likely that adjustments in tendon structural properties to physiological loading are accomplished by adding or removing material rather than altering the material intrinsic properties.

Finding (ii) is also in agreement with \textit{in vitro} results. The relevant phenomenon is referred to as ‘conditioning’ and it has been considered as an artifact because of specimen slippage in the clamps.\[^{[9]}\] In contrast, our \textit{in vivo} results show that conditioning is an actual physical property of the tendon. A consequence of the increasing elongation of the tendon was that the fascicles of the in-series muscle shortened by \(\sim 12\%\). Calculations based on the cross-bridge model indicated that the resultant changes in myofilament overlap might reduce the force-generating potential of the muscle by \(\sim 10\%\), a decrease that could be mistaken for evidence of neuromuscular fatigue.

The findings (iii), (iv) and (v) have implications for locomotor function and exercise rehabilitation in the elderly and other frail populations. They highlight the deteriorating effects of aging and disuse on tendon function, but they also indicate that training may be an effective countermeasure to regain or prevent loss of functional integrity. Stiffening of the tendon by training would allow transmission of contractile forces to the skeleton more rapidly, as indicated by the increased rate of force development, which might be crucial when reacting to a trip, slip or other fall-related accident.

Two methodological issues require particular attention when obtaining the tensile response of a human tendon \textit{in vivo}:

- to have a truly valid association between the tendon force estimated and the elongation measured, the reference point whose displacement is traced must be within the free (extramuscular) part of the tendon.

Such points are the tendon origin in the myo-tendinous junction,\[^{[42-44]}\] the osteotendinous junction (for example, in the patellar tendon\[^{[54,55]}\]), or the echo generated by artificial markers inserted in the tendon.\[^{[59]}\] If, instead, an anatomical point within the intramuscular aponeurosis is selected, the above association becomes invalid because the contractile force by which the intramuscular marker is pulled is clearly less than that exerted by the whole muscle. These two important points have often been neglected (see table I). However, even if measurements are taken appropriately, some inherent problems cannot be avoided. The two most important limitations are:

- the application of non-homogeneous stress across the tendon by a contraction of increasing or decreasing intensity;

- incorporation of heat losses by surface friction between the tendon and adjacent tissues, which would be reflected in the area of the hysteresis loop.

In fact, the average mechanical hysteresis estimates obtained \textit{in vivo} (18–25\%) are higher than the average value of \(\sim 10\%\) obtained from tensile tests on isolated tendons.\[^{[9,14,16]}\] However, we do not know whether the above discrepancy can be entirely accounted for by heat losses from sources other than the tendon or whether it partly reflects differences in tendon material properties between \textit{in vivo} and \textit{in vitro} conditions.

### 2. Overuse Tendinopathy in Sports: the Biomechanical Issue

Biopsy studies have shown that classic inflammatory changes are not frequently seen in chronic tendon conditions and that histopathology features in tendinopathic tendons are clearly different from normal tendons.\[^{[61,63]}\] The exact pathophysiological processes that can occur within tendons have yet to be determined. Several different forms of tendinopathy have been described, and the anatomical location of a given lesion within the tendon plays a
Table I. Ultrasound-based results and protocols regarding the tensile response of several human tendons in vivo

<table>
<thead>
<tr>
<th>Tendon</th>
<th>Load (MPa)</th>
<th>Elongation (%)</th>
<th>Marker</th>
<th>Synergistic actiona</th>
<th>Antagonistic actiona</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis anterior</td>
<td>12.4</td>
<td>4.3</td>
<td>IMF</td>
<td>Yes</td>
<td>No</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>2.5</td>
<td>MTJ</td>
<td>Yes</td>
<td>Yes</td>
<td>43</td>
</tr>
<tr>
<td>Vastus lateralis</td>
<td>31.9–33.4</td>
<td>31.9–33.4b</td>
<td>IMF</td>
<td>Yes</td>
<td>No</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>5843c</td>
<td>12.7c</td>
<td>IMF</td>
<td>Yes</td>
<td>No</td>
<td>49</td>
</tr>
<tr>
<td>Patellar tendon</td>
<td>40–42</td>
<td>5.9–9.9</td>
<td>OTJ</td>
<td>Yes</td>
<td>Yes</td>
<td>53</td>
</tr>
<tr>
<td>Triceps surae</td>
<td>41.6</td>
<td>4.4–5.6</td>
<td>IMF</td>
<td>No</td>
<td>Yes</td>
<td>48</td>
</tr>
<tr>
<td>Achilles tendon</td>
<td>36.5</td>
<td>8</td>
<td>ITM</td>
<td>No</td>
<td>Yes</td>
<td>59</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>398–406c</td>
<td>8.1–8.6</td>
<td>IMF</td>
<td>Yes</td>
<td>No</td>
<td>46</td>
</tr>
<tr>
<td></td>
<td>32.4</td>
<td>4.9</td>
<td>MTJ</td>
<td>Yes</td>
<td>Yes</td>
<td>44</td>
</tr>
</tbody>
</table>

a ‘Yes’ and ‘no’ denote incorporation and non-incorporation, respectively, of the relevant effect in the calculation of tendon load.
b Expressed in absolute terms (mm).
c Expressed as force (N).

IMF = intramuscular fascicle; ITM = intratendon marker; MTJ = myotendinous junction; OTJ = osteotendinous junction.

role in determining the type of tendinopathy. Chronic tendon abnormalities consistent with tendinopathy can be found in the main body of the tendon, its insertion site on bone (the enthesis) and the structures surrounding the tendon. These different abnormalities can co-exist within the same tendon.

All tendons can be affected by tendinopathies.[64] The supraspinatus, common wrist extensor, quadriceps, patellar, posterior tibialis and Achilles tendons are probably the most commonly affected tendons. Insertional tendinopathy (or enthesisopathy) is one of the most common forms of tendinopathy. In particular, the supraspinatus, common wrist extensor, quadriceps and patellar tendon are almost exclusively affected by insertional tendinopathy. The Achilles tendon, on the other hand, can present tendinopathy of the main body of the tendon, paratendinopathy and insertional tendinopathy, each with different clinical features and management implications. Recent reviews on tendinopathy of the main body of major tendons have been published.[65,66] Sections 2.1 to 2.4 focus on insertional tendinopathy and discuss the possible pathomechanics in the aetiology of these tendon disorders.

2.1 Traditional Concepts

Tendinopathies are typically considered overuse injuries. This concept involves excessive loading of the tendon and subsequent mechanical breakdown of the loaded tendon.[67] Excessive loading can theoretically occur in several ways. Training errors have frequently been considered as being an aetiologial factor.[68,69] A given tendon has a baseline mechanical strength, dependent on the loading history of the tendon, as the tendon will adapt to the loads placed upon it. If the loading has been low, or of limited frequency or duration, the tendon mechanical strength will be relatively limited. If a rapid increase in training load, frequency and/or duration occurs, the tendon may not be able to adapt fast enough to this increase. The mechanical strength of the tendon may be exceeded and a small injury may ensue, although it is not well understood how this small injury progresses. Theoretically, repeated microinjuries may occur and the tendon may be able to heal a certain level of microinjury. However, as training and heavy loading of the tendon continues, this healing process may be overwhelmed and a greater injury ensues. At some point, the injury becomes clinically apparent through pain in the involved tendon.

Other factors besides training errors may lead to increased loading of the tendon, such as poor technique[70,71] or inadequate athletic equipment.[72,73] Also, intrinsic factors, such the status of the muscles, ligaments and bones surrounding the tendon, may alter the level of the load on the tendon. Some of these factors, such as muscle strength and flexibility of the ligaments and muscle-tendon units, are variable. Lack of flexibility and muscle imbalances are frequently mentioned in the aetiology of tendi-
nopathies.\textsuperscript{[74,75]} Other intrinsic factors, such as limb alignment and body habitus, are more or less fixed.

Regardless of the factors involved in the development of a tendinopathy, the traditional concept centres around increased tensile loads on the tendon as a result of these. Excessive tensile loads are capable of causing plastic deformation and eventually rupture of the tendon.\textsuperscript{[76]} In tendinopathy, the tensile loads are presumed to be large enough to cause some plastic deformation, the biomechanical equivalent of a microinjury, but not complete failure. Repeated episodes of minor plastic deformation may add up to a clinically significant macroinjury.

2.2 Clinical Challenges to the Traditional Concept

The traditional view of a tendon overuse injury as a result of tensile overload appears plausible. However, although this view is widely accepted, the clinical and scientific bases for this concept do not stand close scientific scrutiny. Without a prospective design and adequate control groups, any conclusion regarding the aetiological role of factors such as training errors, poor technique, inadequate equipment, inflexibility and muscle imbalance remains speculative. Only a few studies have attempted to study these factors in a controlled, prospective manner and have shown conflicting results.\textsuperscript{[77-83]}

Epidemiological studies have also made some interesting observations. For example, overuse injuries, including tendon problems, are significantly more common in elderly athletes compared with young athletes.\textsuperscript{[84,85]}

2.3 Biomechanical Challenges to the Traditional Concept

Biomechanical studies on failure modes of muscle-tendon units have clearly shown that failure will occur within the muscle near the muscle-tendon junction,\textsuperscript{[86,87]} not in the tendon. Although these load-to-failure experiments do not study repetitive submaximal loads, they do point out how a healthy tendon is biomechanically ‘over-engineered’ compared with its attached muscle. In insertional tendinopathy, the pathological tendon lesion lies at or very close to the insertion site of the tendon, the enthesis. Insertional tendinopathy is commonly seen in the supraspinatus (rotator cuff tendinopathy), common wrist extensor (lateral epicondylitis), patellar (jumper’s knee) and Achilles tendons. All these tendons are relatively thick, they insert at an oblique angle in the bone and have a broad insertion site.\textsuperscript{[88-92]} Initial or partial tearing of the supraspinatus is found generally on the humeral side rather than bursal side.\textsuperscript{[92]} Lateral epicondylitis and jumper’s knee can be approached from the joint side with arthroscopic techniques.\textsuperscript{[93,94]} Finally, tendon pathology in insertional Achilles tendinopathy is generally found at the calcaneal side.\textsuperscript{[95]} Thus, the pathology is predominantly found at the joint side of the enthesis.

The enthesis transfers the mechanical tensile loads generated by the muscle-tendon unit onto the bone through a thick tendon, which inserts at a varying, oblique angle, depending on the position of the joint. The architecture of these tendon insertion sites is complex, with a cartilaginous transition zone most pronounced on the joint side of the tendon.\textsuperscript{[96]} Recent work on the increase of proteoglycans within the posterior tibialis tendon indicates that cartilaginous metaplasia can occur as an adaptive response to mechanical compression on the tendon.\textsuperscript{[97]} These histological findings question whether the tendon insertion site is uniformly subjected to tensile loads. This has led to various biomechanical studies on the strains near the tendon insertion sites.

Strains in the supraspinatus insertion onto the greater tuberosity were measured at various glenohumeral positions using a magnetic resonance image-based technique.\textsuperscript{[98]} In general, the strain of the supraspinatus tendon on the joint side of the insertion was significantly lower than the values found on the bursal side.

In a biomechanical study of the patellar tendon origin near the inferior pole, the tendon was instrumented with strain gauges and taken through a range of motion while the tendon itself was loaded.\textsuperscript{[99]} The highest tensile strains measured just distal to the inferior pole of the patella were found on the anterior or aspect of the tendon. The lowest strains were found on the posterior, joint-side aspect of the tendon, the site where the changes of classical patellar tendinopathy, or jumper’s knee, are found. Basso et al.\textsuperscript{[100]} measured strain over the length of the tendon, rather than locally.\textsuperscript{[99]} In this manner, the strains
were actually greater posteriorly in the tendon and failure occurred at higher strains posteriorly. However, this may not reflect what is actually occurring close to the insertion site where the pathology is almost exclusively found. Basso et al.,[101] studying the compressive load in the patellar tendon as it courses over the inferior pole of the patella, found significant compressive loads at this location. They felt that the bone of the inferior pole of the patella exerted this compressive load through an impingement on the tendon. In a study on the Achilles tendon insertion site,[102] the tendon was instrumented with strain gauges just proximal to the calcaneus. Again, the lowest strains were found on the calcaneal side, where the pathological changes of Achilles insertional tendinopathy are generally found.

Although not all biomechanical studies report exactly the same results, a consistent pattern appears to emerge: the strains within the tendons near their insertion site are not uniform. If we assume that the material properties are similar throughout the tendon, this would mean that any muscle force transferred through the insertion site preferentially loads the side of the tendon that is usually not affected initially in tendinopathy. In that case, the side affected by tendinopathy is generally ‘stress shielded’. Thus, the presence of differential strains opens the possibility of alternative biomechanical explanations for the pathology found in these regions of the tendon. The traditional concept of tensile failure may not be the essential feature of the pathomechanics.

2.4 Alternative Biomechanical Theories in Insertional Tendinopathy

The recent biomechanical data reported above suggest a different biomechanical aetiology of insertional tendinopathy. The stress-shielded side of the enthesis shows a distinct tendency to develop cartilage-like and/or atrophic changes in response to the lack of tensile load.[95-97] Over long periods, this process may induce a primary degenerative lesion in that area of the tendon. This may explain why the tendinopathy is not always clearly activity-related, but sometimes more strongly correlated with age. In this manner, tendinopathy would be a result of stress shielding rather than overuse injury.

The cartilage-like changes in the enthesis in many ways can be considered a physiological adaptation to the compressive loads. However, even cartilaginous metaplasia may not allow the tendon to maintain its ability to withstand the high tensile loads in that region. It seems possible that in athletes occasionally certain joint positions will still place high tensile loads on the enthesis. As the stress shielding may have led to tensile weakening over time, an ‘injury’ may occur more easily in this region. In this manner, insertional tendinopathy could be considered as being an overuse injury, but predisposed by pre-existing weakening of the tendon.

Finally, as the joint changes position, strains in one section of the tendon could be changing in opposite directions. Internal shear forces and heat could be generated, producing injury to the cellular and/or matrix components of the tendon. Accumulation of these injuries could lead to the intratendinous degeneration seen in tendinopathy.

3. Conclusions

The aetiology of tendinopathy is likely to be multifactorial, including some of the traditional factors such as overuse, inflexibility and equipment problems. However, other factors also need to be considered, such as age-related tendon degeneration and biomechanical considerations as outlined in this review. Recent in vivo and in vitro studies have shed some light on the biomechanics of the main body of tendon, but more research is needed to determine the significance of tensional loads, stress shielding and compression in tendinopathy. The current biomechanical studies indicate that certain joint positions are more likely to stress the area of the tendon commonly affected by tendinopathy. These joint positions seem to be different from the traditional positions advocated in stretching exercises used for prevention and rehabilitation of tendinopathies. Incorporating different joint position exercises may exert more controlled stresses on these affected areas of the tendon, possibly allowing better maintenance of the mechanical strength of that region of the tendon and, therefore, prevent injury. Alternatively, this practice could stress a healing area of the tendon in a controlled manner and thus stimulate healing once an injury has occurred.
work will have to prove whether such principles should be incorporated in current rehabilitation techniques.

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