The functional morphology of 'the tennis wrist'

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Abstract

At present there is no consensus in the literature concerning the aetiology of lateral epicondylitis and a causal treatment. Microscopic lesions in the common origin of wrist and finger extensors, which are caused by excessive strain, are thought to be the underlying cause of lateral epicondylitis. At least 40 different courses of treatment have been described. However, it is still unknown which treatment is considered the best. In 1988, Riezebos and Lagerberg published about the tennis wrist from a functional morphology perspective (Riezebos & Lagerberg 1988).
Several findings, which cannot be explained by excessive strain, are understandable within the concept of lateral epicondylitis caused by an internal derangement of the wrist. The aim of this review is to falsify the model of lateral epicondylitis, referred to as 'the tennis wrist', which has prevailed for more than 20 years.

(key-words: tennis elbow, tendinopathy, etiology, musculoskeletal manipulations, wrist)

Introduction

Lateral epicondylitis (LE) has been clinically diagnosed (Goguin & Rush 2003; Boisaubert et al 2004) on the basis of pain around the epicondyl. The pain either occurs or increases during resistance to the extension of the wrist or 3rd metacarpal (Struijs et al 2003; Hong et al 2004) and the palpation of the lateral epicondyl (Radpasand 2009; Assendelft et al 2009). Gripping is painful, certainly when force is required. Mundane activities, such as shaking hands and holding a cup, become difficult (Trudel et al 2004; Vicenzino et al 2007).
LE has been diagnosed as the most common complaint in elbow problems (Hong et al 2004). In 80% of the cases, the symptoms are chronic (Radpasand 2009). Statistical data reveal that LE can be associated with long-term absenteeism from work (Hong et al 2004; Bisset et al 2005). A recovery period of 6 months to 2 years has been reported (Smidt et al 2002; Coombes et al 2009).

The aetiology of LE is unknown (Slater et al 2003; Manchanda & Grover 2007; Alizadehkhaiyat et al 2009). The underlying cause of lateral epicondylitis is thought to be attributed to microscopic lesions in the common origin of the wrist and finger extensors caused by excessive strain (Benjamin et al 2006; Manchanda & Grover 2007; Cutlip et al 2009; Magnusson et al 2010), especially in the extensor carpi radialis brevis muscle (ECRB) (Alfredson et al 2000; Boisaubert et al 2004; Alizadehkhaiyat et al 2009). At least 40 different types of treatment have been described (Bisset et al 2005; Stasinopoulos & Johnson 2007; Radpasand 2009). However, it remains unclear which treatment is the best.
The first article about LE was published in 1873 (Goguin & Rush 2003; Hong et al 2004). Since then various pathophysiological hypotheses have been described (Snijders et al 1987; Stoeckart et al 1991; Hägg & Milerad 1997).

In 1988, Riezebos and Lagerberg published about the tennis wrist from a functional morphological perspective (Riezebos & Lagerberg 1988). LE could be regarded as the functional morphological changes that occur in the ECRB resulting from a scapholunate internal derangement (Frankel et al 1971; Tasker & Waugh 1982; Dimitroulis 2005) during wrist extension.
The starting point for this article is the 'expiry date' of the controversial principle of 'the tennis wrist', which has prevailed for the past 20 years. For manual therapy, investigating the possibilities of a functional morphological approach is important, as the *functional* morphology enables intervention in the causal function problems underlying the symptoms. The manual therapist then treats the primary disrupted function and not the secondary 'abnormal' form or 'diseased' structure.

Sub-questions
1. Is an inflammatory infiltrate found in tendon tissue? Does the tendon tissue undergo morphological changes in LE?
2. Do these changes occur on the basis of an altered mechanical strain in the tendon?
3. Can an altered ratio rolling/gliding in the joint lead to an altered mechanical strain in the tendon?
4. Can an altered positioning of the movement axes in joints lead to an altered ratio rolling/gliding?

Method
The database search was carried out in August, 2010. Using specific search strings, PubMed searched for articles related to the functional morphological changes that occur in tendon tissue as a result of an internal derangement in the spanned joint.
The MeSH search terms used are: 'tendons', 'tendon injuries', 'tennis elbow', 'adaptation, physiological' and the free search term, 'wrist'.

Inclusion criteria
Articles meeting the following criteria have been included:
1. Publications from the past 15 years (arbitrary).
2. Publications preferably about humans.
3. Consistent with the aforementioned sub-questions.
4. Publications in English, French, German and Dutch.

Studies at all levels of evidence are eligible for this literature study. The publications included have been rated according to a citation analysis, the journal impact factor and the H index.

Figure 1.

Evaluation 1
The first evaluation takes place on the basis of the research objective.

Evaluation 2
After having studied the full-text version, those articles that appeared to be inconsistent with the sub-questions, were excluded.

Results

The search resulted in 157 articles, which have been subjected to further assessment. Furthermore, references found in the literature have been screened and evaluated for their relevance to the research objective. Of the 180 original articles, 63 were excluded after the first evaluation, which means that 117 articles were included for the second evaluation. After the second evaluation, 102 articles remained.

The morphology of epicondylitis
Histopathological research has demonstrated that few macrophages, lymphocytes or neutrophils are found in chronically-overstrained tendon tissue (Benjamin et al 2006; Magnusson et al 2010).
The histopathological findings for tendinopathy are characterised by the rupturing and thinning of collagen fibres, increased vascularisation, granulation tissue and an increase in proteoglycans (PG) in the extracellular matrix (Benjamin et al 2006; Riley 2008).
The pathological changes are described as 'a-functional'. It would therefore seem better to talk about 'tendinosis' (Goguin & Rush 2003; Slater et al 2003; Doschak & Zernicke 2005).
Angiofibroblastic tendinosis refers to degenerative changes that occur when a tendon is repeatedly subjected to microtrauma (Kjaer 2004; Boisaubert et al 2004; Coombes et al 2009).
In tendinopathy, molecular changes exhibit an increased expression of type III collagen, decorin, aggrecan and biglycan. These changes indicate recovery processes and adaptive responses to changes in mechanical strain (Hoffmann & Gross 2007; Riley 2008; Lui et al 2010). Decorin and biglycan are 'small leucine-rich repeated proteoglycans' (SLRP) that contribute to collagen fibril structure, whereas aggrecan is a large highly hydrophilic PG that plays a role in adaptation to compressive forces (Riley 2008; Lui et al 2010).

The mechanics of the tendon and the enthesis
The tendon consists of hierarchical layers in which Type I crimped collagen fibres are accurately arranged in the pulling orientation (Yoon & Halper 2005; Riley 2008).
The tendon responds to mechanical strain by altering its structure, composition and mechanical properties. This process is termed 'mechanotransduction'. Intermolecular, covalent cross-linking ensures mechanical stabilization of collagen fibrils and the ultimate tension-absorbing properties of the tendon (Chiquet et al 2003; Martin et al 2003; Screen et al 2006).

Figure 2.

A strong correlation exists between fibre orientation of collagen connective tissue and the functional requirements imposed upon the tissue. If force transfer of tissue is anisotropic, then the functional properties of the resulting structure are as well (Ottani et al 2001; Kjaer et al 2006). In an aponeurotic tendon the force transfer occurs in a non-uniform manner (Maganaris 2002; Thomopoulos et al 2006), which can lead to intratendinous shear strain (Magnusson et al 2008; Benjamin et al 2008; Burkholder 2008).
However, the enthesis is adapted to the effects of non-uniform force transfer as a consequence of an insertional angle change. A correlation exists between the quantity of fibrous cartilage in entheses and the extent to which an insertional angle change occurs during movements (Benjamin et al 2006). In

'fibrocartilaginous entheses' (FCE), chondrogenesis has taken place (Milz et al 2005; Riley 2008; Thomopoulos et al 2010).

Figure 3.

Fibrous cartilage facilitates the dissemination of tension at the area of transition between hard and soft tissue so that the transition is resilient to compression and shear forces. This characteristic is reflected in the molecular composition, namely the presence of specific hyaline cartilage molecules, in particular sulphated GAGs, aggrecan and Type II collagen (Milz et al 2005; Thomopoulos et al 2010). Tendons, which have been subjected to compression forces, develop cartilaginous characteristics (Kraushaar & Nirschl 1999; Ottani et al 2001).
Pathological changes are found at the very locations where 'strain', i.e. elongation, occurs the least. A clinically recognisable enthesopathy mainly occurs in the deeply situated parts of an enthesis where such regions are compressed by the superficial parts, and where the presence of fibrous cartilage is usually more apparent (Benjamin et al 2006; Coombes et al 2009).

The mechanics of epicondylitis

The origin of the ECRB is located on the movement axis of the elbow (London 1981; Lieber et al 1997b). With its relatively short fibres and large moment arm, the ECRB acts as a mechanical torque for wrist extension. This design results in an ECRB that is isometrically strong (Lieber et al 1997a; Lieber & Fridén 1998).
Electromyographic investigations reveal that the ECRB exhibits more electrical activity than the extensor carpi radialis longus muscle (ECRL) (Tournay & Paillard 1953; Riek et al 2000) during extension and gripping. In patients with LE, the ECRB exhibits an increased electrical activity compared to a healthy control group (Bauer & Murray 1999; Shetty & Randolph 2000).
An increase or decrease in the activity of the wrist extensors and flexors following electrical stimulation of the scapholunate interosseous ligament (SLIL) is observed, dependent on the wrist position. Increased activity is encountered in the antagonists of wrist positions and may possibly have a protective function for the joint (Hagert 2010).

The arthrokinematics of epicondylitis

In vivo it has been found that the scaphoid rotates more upon flexion and extension than the lunate, which implies intercarpal rotations (Kobayashi et al 1997; Sun et al 2000; Wolfe et al 2000; Moojen 2003). Intra-articular kinematics are described using arthrokinematic terminology such as rolling, gliding and spinning (Biscević et al 2005; Baeyens et al 2006). Rolling and gliding take place simultaneously (Schomacher 2009).
Joints have incongruent profiles and subsequently no fixed rotational axis (Lagerberg 2007). The instant centre of rotation (ICR) therefore shifts in the joint during movement (Gertzbein et al 1986; Sears et al 2006; Schomacher 2009). For the joint to move, the ICR should always be normal to the contact point. With this, each tendency of the rotational axis to shift is accompanied by a shift of the contact point. Any displacement of the ICR away from the perpendicular of the tangent in the **point of joint contact** leads to compression or separation of the profiles (Montgomery et al 1998).
The direction in which the moment arms of ligaments exert force on the profiles accurately determines the position of the centrode (Herzog & Read 1993; Leardini 2001; Biscević et al 2005). Abnormal positions of a centrode lead to changes in the rolling/gliding ratio (Frankel et al 1971) and vice-versa. Movement-impaired joints exhibit a reduced glide and an increased roll between the surfaces (Schomacher 2009). A joint limitation develops when the direction of the speed of movement on the surfaces has the tendency to compress both profiles. The movement blocks and further flexion or extension is no longer possible (Frankel et al 1971; Montgomery et al 1998).
The SLIL is the principle stabilizing structure between the scaphoid and the lunate (Moojen et al 2002; Kai et al 2006; Milz et al 2006; Short et al 2007). Ligament damage of the SLIL is the most common cause of carpal instability and is a condition that eventually results in severe impairment of radiocarpal function (Blevens et al 1989; Milz et al 2006). After section of the SLIL, normal articular contacts are altered, the contribution of the scaphoid increases and that of the lunate decreases during flexion and extension

(Blevens et al 1989; Tang et al 2002; Kai et al 2006; Henry 2008) and the scapholunate movement becomes variable (Werner et al 2005).
A kinematic change of the scaphoid after rupture of the SLIL alters the position of the surrounding tendons and their centre of rotation, which leads to a change in tendon excursion (Tang et al 1997). Immobilisation also results in instability caused by the ligaments being more compliant, resulting in ligamentous laxity (Wren et al 2000; Doschak & Zernicke 2005; Woo et al 2006; Magnusson et al 2008).

Discussion

The concept of a scapholunate internal derangement being a cause of LE makes several findings comprehensible, which cannot be explained by excessive strain (Boisaubert et al 2004; Magnusson et al 2008; Cutlip et al 2009).

The selective involvement of the ECRB in LE (Slater et al 2003; Manchanda & Grover 2007; Puchner 2008) and the increased electrical activity this muscle exhibits (Bauer & Murray 1999; Shetty & Randolph 2000) are mentioned frequently. In 'the tennis wrist' model, a scapholunate internal derangement is assumed to be responsible for a meticulously described sequence of events.
In this scapholunate internal derangement, the rolling component of the scaphoid becomes too large during extension (Crisco JJ et al 1999; Wolfe et al 2000; Kai et al 2006; Henry 2008), which could lead to a subluxation with increased capsular tension in the complex. As compensation, the ECRB is activated to tilt the 3rd radius (3rd metacarpal, capitate, lunate) as a 'block' with the scaphoid to reduce the associated ligamentous tension (Riezebos & Lagerberg 1988).

In arthrokinematics, it is assumed that the convexity has a 'natural' tendency to roll in the absence of ligamentous guidance (Schomacher 2009). 'The tennis wrist' model also assumes that an internal derangement results in an increased arthrokinematic roll as the glide appears later in the movement phase.
However, in view of the low friction coefficient of joint cartilage, it is questionable whether the rolling component will occur at an unguided moment. When ligamentous guidance is lacking, the position of the ICR is undefined. The ICR defies all arthrokinematic laws, albeit it briefly. It no longer needs to be on the perpendicular of the tangent in the contact point. In short, the ICR is subject to the capriciousness of the resultant outcome of all moments acting on the joint.

In the model, the increased capsular tension in the scapholunate complex results in pain and a response of the ECRB. When electrically stimulating the SLIL, Hagert found either an increase or a decrease in the activity of the wrist extensors and flexors, depending on the wrist position (Hagert 2010). In this study, no distinction was made between the ECRL and the ECRB. However, it does illustrate the tendency of the body to defend itself against the potential threat of end positions.

London positions the rotational axis of the elbow through the centre of the arch formed by the trochlear sulcus and the humeral capitellum (London 1981). Thus the origin of the ECRB falls on the movement axis of the elbow. The mechanical effect of a muscle is determined by the distance from the line of action of the muscle to the ICR. The moment arm that the ECRB has over the elbow is negligible in that sense. This implies that the ECRB fulfills its function during gripping irrespective of the elbow position.
With its relatively short fibres and large moment arm, the ECRB provides a large mechanical torque for wrist extension, resulting in an ECRB that is isometrically strong (Lieber et al 1997a; Lieber & Fridén 1998). From these publications, it can be deduced that the ECRB is suitable for isometric loads, as is generally the case during gripping.

If excessive strain can be held responsible for LE, the questions still remain as to why the symptoms do not quickly disappear during rest (Hong et al 2004; Manchanda & Grover 2007; Alizadehkhaiyat et al 2009; Assendelft et al 2009), and why symptoms are found in patients who seldom perform activities requiring considerable exertion (Goguin & Rush 2003; Milz et al 2004).
According to the 'tennis wrist' model, immobility causes internal derangement. This explains why patients, who do not perform physically-demanding activities, can be affected (Riezebos & Lagerberg

1988; Goguin & Rush 2003; Milz et al 2004). Immobility or a 'lack of use' is a very dangerous stimulus to the musculoskeletal system. Immobility results in a 'weaker' connective tissue (Woo et al 1987; Akeson et al 1987; Yasuda & Hayashi 1999; Woo et al 2006; Magnusson et al 2008; Puchner 2008) and subsequently to less ligamentous guidance of the joint. The tendency of a joint with an internal derangement to subluxate results in excessive capsular tension and movement impairment.

In this functionally disrupted situation, the ECRB is required to function in changed positions (Lieber et al 1997a). Only in specific positions can the operating force be evenly distributed over the collagen fibres. In positions other than 'known ones', 'shearing moments' occur in the flat origin (Riezebos & Lagerberg 1988). There is external evidence for the existence of tension concentrations referred to in the 'tennis wrist' model.
The direction (Wang 2006; Magnusson et al 2008) of the forces in the aponeurotic origin of the ECRB no longer concur with the direction in which collagen fibres are oriented (Ottani et al 2002). The force transfer takes place out-of-plane (Thomopoulos et al 2006), resulting in changes in intratendinous shear strain (Benjamin et al 2006; Magnusson et al 2008; Burkholder 2008).
There are indications in the literature for the remodelling proposed in the model.
The histopathological findings for tendinopathy are characterised by 'angiofibroblastic hyperplasia' (Boisaubert et al 2004; Coombes et al 2009). There is evidence for the presence of fibrous cartilage as a normal characteristic of entheses (Benjamin & Ralphs 1998; Milz et al 2004).
Aggrecan plays a role in the adaptation to compressive load (Doschak & Zernicke 2005; Riley 2008; Lui et al 2010). The molecular composition of various fibrous connective tissues is directly related to the respective mechanical tissue functions (Milz et al 2005).
It would seem that tissue in the tendon, ideally suited to resisting tension from one single direction, gradually transforms into tissue that needs to be resilient against tensional forces from various different directions. Consequently, it must also be resilient to compressive forces that arise from deformation of the tissue. Type I collagen connective tissue, with fibres accurately arranged at micrometer level, is replaced by Type III collagen and cartilage.
It is unlikely that 'the body' replaces mechanically-specific tissue without there being a clear mechanical need for this, since remodelling (Ottani et al 2001; Wang 2006), requires a high metabolic activity.
The formation of cartilage in the tendon should therefore be seen as a normal, non-diseased development in response to compressive load.

Other than Riezebos and Lagerberg's publication (Riezebos & Lagerberg 1988), nothing else has ever been written about 'the tennis wrist'. Therefore, the main focus has been on factors relevant to this topic.
As the model assumes a functional adaptation of the ECRB, the MeSH term, 'adaptation', was used. This might have resulted in selection bias.
As there was no scoring method for descriptive studies, the publications included were evaluated by means of a citation analysis, the journal impact factor and the H index.
The higher the score for one or more ratings, the more authoritative the reference.

Interpretation and proposal

If ligamentous guidance is reduced, an internal derangement occurs as a result of which altered (scapholunate) kinematics develop (Montgomery et al 1998; Brownhill et al 2006).

The movement in the joint becomes erratic and unpredictable (Weiler & Bogoch 1995; Scarvell et al 2005; Werner et al 2005; Foumani et al 2009).
A condition for an equilibrium in the joint is that all moments cancel each other out and yield a reaction force without a resulting joint moment. This 'zero joint reaction moment' can only proceed through the ICR (Pierce & Li 2005; Li et al 2006; Lagerberg 2007).
The friction coefficient in a synovial joint is about 0.01 (Carter et al 2007; Ateshian 2009). In comparison, the friction coefficient of ice on ice is 0.1. Force transfer between profiles can therefore only occur perpendicularly. If the reaction force deviates by a single degree, the profiles would immediately slip (Lagerberg 2007). Ligaments guarantee the alignment of the reaction force without exerting a moment on the joint (Lagerberg 2007; Kleipool & Blankevoort 2010). In cases of ligament injury or immobility, the

contact points proceed on the basis of the prevailing load conditions, resulting in the joint becoming a highly unpredictable mechanical linkage (Lagerberg 2007).

The tendon is confronted with tension concentrations that in terms of direction and intensity are variable and unpredictable (Pierce & Li 2005; Thomopoulos et al 2010).

The fibre orientation in the tendon no longer satisfies the requirements imposed on it. It therefore undergoes a remodelling (Ottani et al 2001; Riley 2008).

Cartilage is resilient against variable tension concentrations due to variable shear and compression forces (Benjamin et al 2006; Lui et al 2010).

Chondrogenesis and osteogenesis are the result of functional morphological adaptations and are not pathologic (Benjamin & Ralphs 1998; Milz et al 2005; Li et al 2010).
In the preliminary stages of calcific tendinitis, the tendon transforms into fibrocartilagenous tissue due to the metaplasia of tenocytes into chondrocytes (Hughes & Bolton-Maggs 2002; Lam et al 2006). The tendon becomes infiltrated with an extracellular matrix of cartilage when it is subjected to compressive loading (Ottani et al 2001).
Calcified tissue contributes to the functional recovery of the enthesis (Newsham-West et al 2007). Intratendinous calcifications and enthesophytes are found in LE (Levin et al 2005). Therefore, the changes that take place in LE cannot be termed 'degenerative' or 'a-functional'. The tendon is actively developing under the prevailing variable mechanical conditions to create tissue that is resilient to the 'freaky' tension in the tendon. Even when forces in tendon tissue are opposing and, as such, extremely conflicting demands are made on it, the tendon transforms into a structure where the 'average' function is integrated.

The changes that occur in the tendon can be described as 'tendopoiesis'. The suffix poiesis means 'construction' and 'development'.

Conclusions

1. No inflammatory infiltrate is found in tendon tissue in LE.
   In tendinopathy, tendon tissue undergoes morphological changes to complete the remodelling.
2. These changes can take place in response to changes in the mechanical load.
3. These changes in the mechanical strain of the tendon can result from an altered ratio rolling/gliding in the spanned joint.
4. An altered position of the ICR results in an altered rolling/gliding ratio.

The origin of the ECRB undergoes functional morphological changes in LE in order to complete a remodelling. The remodelling can possibly be attributed to a scapholunate internal derangement, resulting in highly variable and unpredictable tensions in the origin of the ECRB.

The study has not been able to falsify the model. So therefore, even though it was first proposed more than 20 years ago, the model has not lost its relevance.

A retrospective cohort study into epicondylitis symptoms in patients with scapholunate instability or in patients, who were injured after a fall, would provide more clarity on the aetiology of LE.

According to the model, scapholunate mobility needs to be restored. Arthrokinematic mobilisation techniques required for this do justice to the results of scientific research (Coyle & Robertson 1998), which has revealed that the Type I collagen connective tissue of impaired joints exhibits an increased compliance. We therefore recommend that a randomised controlled trial be performed with a study population of sufficient size (Struijs et al 2003; Boisaubert et al 2004) with a long-term follow-up (Bisset et al 2005; Puchner 2008).

Tendopoeisis is the term that covers the process described in this article.

Figure 1.

Flow chart of the literature search.

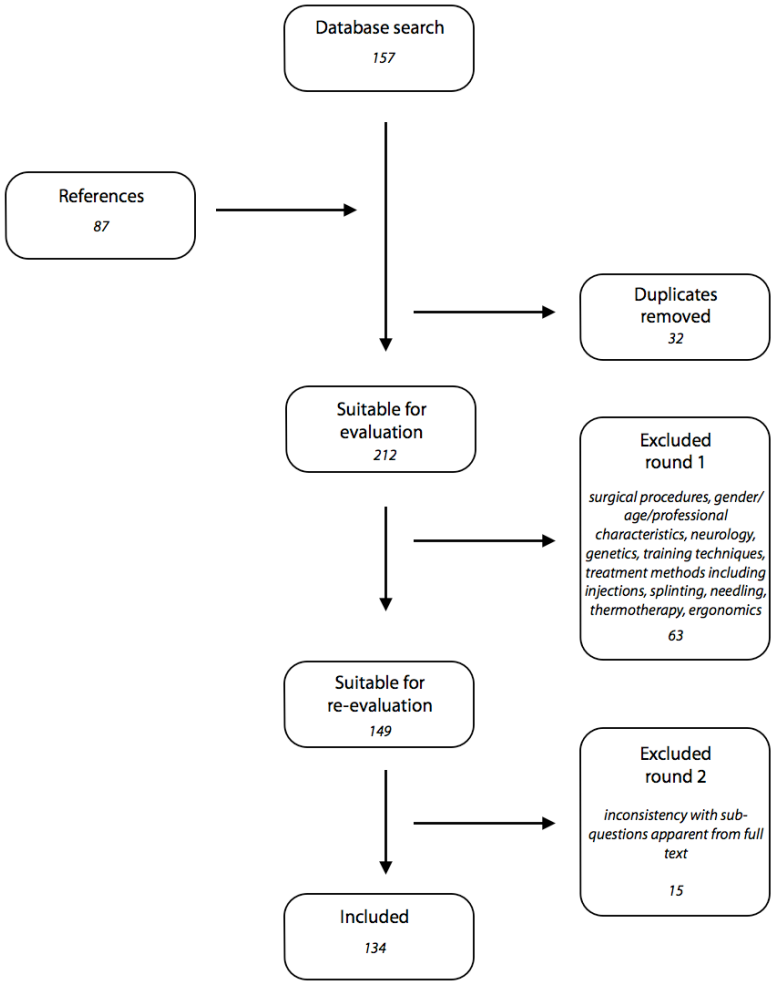

Figure 2.

The various hierarchical levels of tendon tissue.
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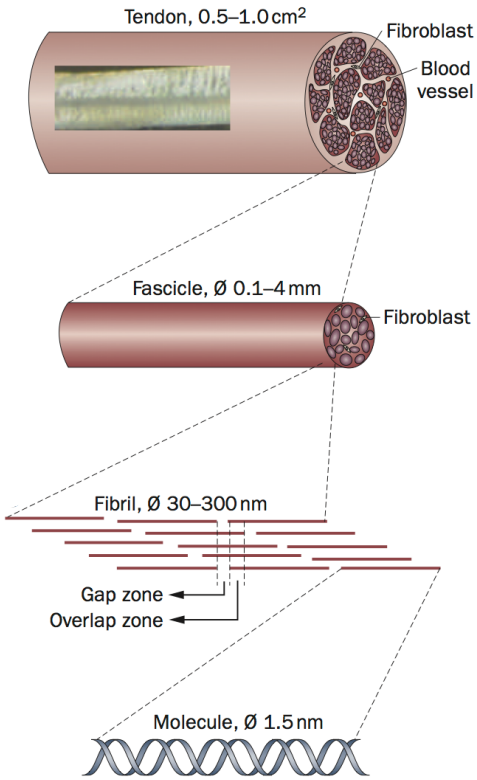


The tendon is built up from collagen fascicles. The inset shows two bordering collagen fascicles. The crimp pattern of the fascicle is visible.

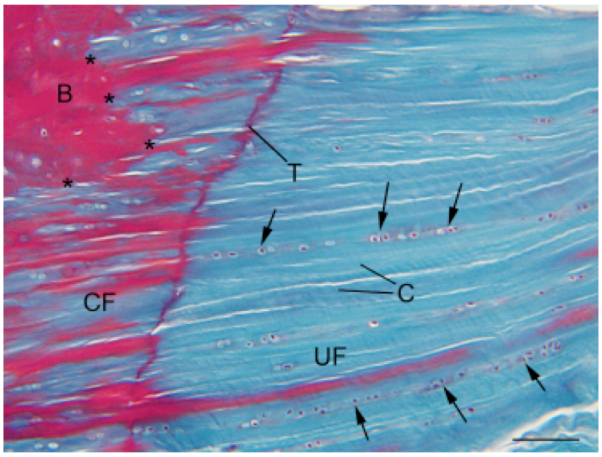
The fascicle is made up of collagen fibrils, fibroblasts, proteoglycans, glycoproteins and glycosaminoglycans.

The collagen fibril has a 'quarter-stagger-arrangement' of collagen molecules. Crosslinking of the collagen fibrils safeguards the integrity of the fibril.

The collagen molecule is built up from three protein chains (α-chains) that form a triple helix.

Figure 3.

Upon approaching the bone (B) the tendon becomes fibrocartilaginous. A section of non-calcified fibrous cartilage (NF) is separated from an area of calcified fibrous cartilage (CF) by a boundary (T). T is the 'hard-soft' boundary of the enthesis. The cells (➔) in the fibrous cartilage are arranged longitudinally to the collagen fibres (C).
scale line=100 µm.
Reproduced by kind permission of John Wiley and Sons from (Benjamin & McGonagle 2009).



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