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Research Paper

Prediction of the elastic strain limit of tendons

A.M. Reyes[a], H. Jahr[b], H.T.M. van Schie[c,d], H. Weinans[a,e], A.A. Zadpoor[a,*]

[a]Department of Biomechanical Engineering, Faculty of Mechanical, Maritime, and Materials Engineering, Delft University of Technology (TU Delft), Mekelweg 2, Delft 2628 CD, The Netherlands
[b]Department of Orthopedic Surgery, University Hospital, RWTH Aachen University, Aachen, Germany
[c]UTC Imaging, Stein, The Netherlands
[d]Department of Physiotherapy, Faculty of Medicine, Nursing and Health Sciences, Monash University, Frankston, Australia
[e]Department of Orthopedics and Department of Rheumatology, Utrecht University Medical Center, Utrecht, The Netherlands

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ABSTRACT

The elastic strain limit (ESL) of tendons is the point where maximum elastic modulus is reached, after which micro-damage starts. Study of damage progression in tendons under repetitive (fatigue) loading requires a priori knowledge about ESL. In this study, we propose three different approaches for predicting ESL. First, one single value is assumed to represent the ESL of all tendon specimens. Second, different extrapolation curves are used for extrapolating the initial part of the stress–strain curve. Third, a method based on comparing the shape of the initial part of the stress–strain curve of specimens with a database of stress–strain curves is used. A large number of porcine tendon explants (97) were tested to examine the above-mentioned approaches. The variants of the third approach yielded significantly ($p<0.05$) smaller error values as compared to the other approaches. The mean absolute percentage error of the best-performing variant of the shape-based comparison was between $8.14\pm6.44\%$ and $9.96\pm9.99\%$ depending on the size of the initial part of the stress–strain curves. Interspecies generalizability of the best performing method was also studied by applying it for prediction of the ESL of horse tendons. The ESL of horse tendons was predicted with mean absolute percentage errors ranging between $10.53\pm7.6\%$ and $19.16\pm14.31\%$ depending on the size of the initial part of the stress–strain curves and the type of normalization. The results of this study suggest that both ESL and the shape of stress–strain curves may be highly different between different individuals and different anatomical locations.

1. Introduction

Achilles tendon injuries are very common among athletes (Schepsis et al., 2002; Shannon, 2011; Werd, 2007) with an incidence of 11 in 100,000 per year (Clayton, 2008), most commonly affecting middle-aged athletic men (Dudhia et al., 2007; Werd, 2007). One of the main risk factors leading to these injuries is overuse (Flick et al., 2006; Jung et al., 2009;

*Corresponding author. Tel.: +31 15 2781021; fax: +31 15 2784717.
E-mail address: a.a.zadpoor@tudelft.nl (A.A. Zadpoor).

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Thornton and Hart, 2011). It has been observed that repeated loading of tendon without allowing for enough recovery time in between could subject the tendon to a degenerative condition that will leave the tendon more susceptible to failure even when submaximal loads are applied (Kannus and Józsa, 1991; Thornton and Hart, 2011). This repetitive loading may include abnormal mechanical loading such as changes in magnitude, frequency, duration or direction (Thornton and Hart, 2011). Moreover, other risk factors such as age, vascular supply, training errors, misalignment, flexibility, and strength deficits may also contribute to initiation of chronic tendon injuries (Almekinders and Temple, 1998). Nonetheless, there is a lack of understanding of the etiology of overuse injuries and that may result in unreliable and inconsistent diagnosis (Almekinders and Temple, 1998; Riley, 2004).

The degenerative condition preceding the injury is known as tendinosis. It is manifested as a group of varied morphological, structural, and phenotype changes such as cell rounding (Riley, 2008), collagen disorientation and fiber separation (Khan et al., 2000), focal variations in cellularity and vascularization, and an increased amount of non-collagenous matrix such as a higher volume density of GAG-rich areas (Movin et al., 1997). Furthermore, mechanical changes such as a decrease in Young's modulus (Thornton and Hart, 2011) are also characteristics of tendinosis.

Three main theories are proposed to explain the rupture of tendons: a mechanical, a vascular, and a neural theory (Riley, 2004). In this study, we will only consider the first theory according to which repetitive micro-trauma is the cause of the lesion and is analogous to fatigue failure in engineering materials (Dudhia et al., 2007; Riley, 2004). It is assumed that micro-trauma, represented by matrix damage, overwhelms the capacity of cells to repair structural defects (Riley, 2004). Furthermore, micro-damages such as collagen fiber damage and cross-link rupture can make fibers slide over each other, denature, become inflamed, and cause pain (Devkota and Tsuzaki, 2007). This theory is consistent with the increased incidence in relation to age and the presence of degeneration predominantly in the active population, but does not explain by itself the fact that pain is not always present (Rees et al., 2006).

It is hypothesized that certain loading patterns should be responsible for the above-mentioned morphological, structural, and phenotype changes. Nonetheless, in order to elucidate the complex relationship that exists between them, the concept of micro-damage has to be further studied (Devkota and Tsuzaki, 2007) with a focus on the early stages of the problem (Almekinders and Temple, 1998; Fung and Wang, 2010).

Some experiments have studied the fatigue behavior of tendon with different loading settings and with different specific goals (Devkota and Tsuzaki, 2007; Fung and Wang, 2010; Ker, 2007a; Pike et al., 2000). Only a few of them have focused on the early stages of loading (Fung and Wang, 2010; Nakama and King, 2005; Sun et al., 2010). However, the applied load is always in terms of the ultimate tensile load and applied equally to all the tendons in the experiment. This loading protocol does not take into consideration the variability that may exist between individuals: a variability that can also be found even within the same tendon (Sharma and Maffulli, 2005a; Thorpe and Stark, 2010). As a result, the applied load may lie at different distances from the elastic limit (inflection point in the stress–strain curve) for each of the tested tendon specimens resulting in inconsistent mechanical and biological readings. It is therefore important to be able to predict the elastic limit of tendons in order to apply the load at the same distance from this critical point.

The aim of this study is to propose and evaluate three different approaches for prediction of the elastic strain limit of tendons. Stress–strain curves of tendon explants show several distinct regions. Within the initial region (toe region), the displacement of the tendon explant is mainly derived by the "straightening of the crimp wave" (Bosch, 2009). Beyond this point, the fibers start to stretch in a region that is called the linear region. The linear region is comprised of two sub-regions. In the first sub-region, straining occurs due to molecular elongation (Amar, 2009; Frontera, 2003) followed by fibril straining and fibril recruitment, meaning that gradually more fibrils start to be strained (Natali et al., 2005; Rigozzi et al., 2011). This training process reaches a point (start of the second sub-region) where partial rupture of the cross-links between collagen molecules occurs (Frontera, 2003; Sharma and Maffulli, 2005b). The strain point just before micro-ruptures occur corresponds with the maximum value of Young's modulus (Crevier-Denoix et al., 2005) and marks the end of reversible and damage-free elongation (Denoix, 2002; Haraldsson, 2008). The strain associated with the maximum modulus is therefore considered the strain value beyond which accumulation of micro-damage starts. Further straining of the tendon will result in more fiber failures and macroscopic damage.

In order to evaluate the proposed methods, *in vitro* experiments were carried out with porcine superficial and deep digital flexor tendons. The interspecies generalizability of the best-performing approach is evaluated by applying it for predicting the elastic strain limit of horse tendons.

2. Materials and methods

2.1. *Materials*

Superficial (SDFT) and deep (DDFT) digital flexor tendons of the hindlimbs of Yorkshire pigs were used for the experiments. These are fast growing pigs (Ezekwe and Martin, 1975) and, based on their weights (35–54.9 kg), their estimated age was between 4 and 6 months. The pigs were therefore not completely skeletally mature. According to Thornton, Achilles tendinosis may incur between 18 and 55 years old while it is only after the age of 30 that clinical problems appear (Thornton, 2011). In fact, in basketball players, some patellar tendinosis may commence even before 18 years old (Cook et al., 2000). It may therefore be important to study the behavior of tendons in earlier ages (before skeletal maturity) to fully understand the etiology of tendon diseases. Because of that reason and easier access to 4–6 months pigs, they were used in the current study. A total of 97 explants were obtained of which 40 were isolated from SDFTs and 57 from DDFTs. The tendons tested in this study are uniaxially loaded elastic energy-storing tendons that are biomechanically