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Fascia strain hardening: A new mesoscopic approach

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1. Introduction

The mechanical characterization of fasciae tissues is crucial in medical research. However, the mechanical behavior of the thoracolumbar fascia (TLF), which is linked to conditions like low back pain (Casato 2019), remains underexplored. A better understanding of its properties could improve prevention and management of such pain.

Recent studies (Schleip 2012) have noted changes in the mechanical properties of the TLF during *ex vivo* tensile tests, but the underlying mechanisms remain unclear. This study aims to address this gap with a mesostructural explanation and a supporting mathematical model.

2. Methods

2.1 Thoracolumbar fascia mesostructure

The TLF is a multi-layered structure composed of collagen fiber bundles embedded within a soft proteoglycan matrix (Benetazzo, 2011). From a mechanical perspective, it can be modelled as a network of parallel, undulated collagen fibers.

2.2 Strain hardening observation

The strain hardening phenomenon, first observed during *ex vivo* tensile tests on mouse TLF, describes a stiffness increase after conditioning (Schleip 2012). Under physiological conditions, researchers noted that a second loading, using the same loaded conditions,

resulted in greater tissue stiffness than the first. Our team observed the same phenomenon in human TLF tensile tests (Creze 2025). Samples were first loaded to 10N along the superficial fiber direction, held at constant strain for 5 minutes, unloaded to 0N, and then loaded up to 50N. In all cases, the tissue exhibited increased stiffness and faster stiffening during the second loading compared to the first.

2.3 Strain hardening hypothesis

This study explored a hypothesis based on the tissue's mesostructure. In its resting state, the superficial collagen fiber bundles were crimped. During the initial loading, many fibers unfolded, increasing tissue stiffness. During relaxation, while strain remained constant, the applied force decreased, suggesting fiber reorganization. Stretched fibers began to fold again. The greater stretch led to the more pronounced folding. This process resulted in a modified crimp pattern, ultimately altering the tissue's mechanical behavior in subsequent loading phases.

2.3 Semi-analytical loading model

A semi-analytical model of 88 parallel nonlinear springs, representing collagen fibers, was developed to test the hypothesis by simulating the tissue's force-strain relationship. The total applied force was the sum of forces on each spring (*Equation 1*), considering that mechanical behavior was mainly driven by superficial fibers aligned

with the test direction. To represent the crimped collagen fibers, each spring had bi-linear behavior with a slack threshold. Below this threshold, stiffness was zero; above it, stiffness was set at 37.64 N/%. The slack (s_i) thresholds were randomly distributed across the springs following a normal distribution as many biological parameters. Differences in mechanical behavior resulted from changes in slack between the two loads.

During the relaxation phase, the slack of each unfolded fiber increased regarding its initial slack. The increase was driven by Equation 2 where ds is the slack increase, dF is the force decrease, K is the collagen stiffness and s and s_i represents the slack of the fiber and the slack of every unfolded fiber respectively.

$$F = \sum_i K(\varepsilon - s_i(F)) \quad \text{Equation 1}$$

$$s(F - dF) = s(F) + \frac{dF}{K} \frac{1}{\sum_i (\frac{1}{s_i})} \frac{1}{s} \quad \text{Equation 2}$$

2.3 Experimental comparison

The model was evaluated against experimental data (Fig. 1). The initial slack distribution was adjusted to accurately replicate the 10N tensile test results (blue curve). The tissue was loaded to the strain observed in the 10N test, then relaxed until the force decreased by 7N. Applying the model, new slack values were computed, and a second tensile test was conducted, from the lowest updated slack strain to the maximum strain reached in the 50N test.

3. Results and discussion

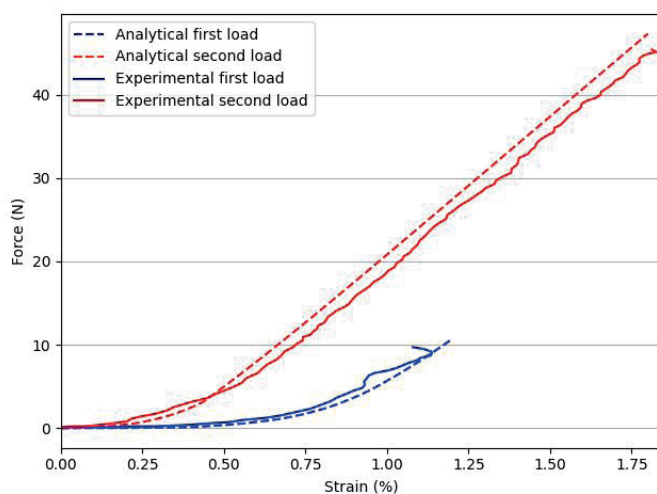


Figure 1. Experimental force/strain behaviour and semi-analytical force/strain behaviour.

The 10N tensile test showed strong agreement between experimental data and the model ($r^2=0.99$), as expected from the distribution parameters calibration. While the model accurately replicated experimental stiffness ($r^2=0.98$), it exhibited a faster stiffening response, leading to an overestimation of force ($1.98N \pm 0.35N$).

The semi-analytical model captured strain hardening, mirroring the experimental observations of the phenomenon. Discrepancies stem from model simplifications, as many tissue structures were omitted to maintain a computationally efficient approach. Including the matrix would have slightly increased the tissue's initial stiffness, improving the fit at the start of the 50N test and enhancing overall accuracy.

Despite some differences, the model supports our hypothesis that crimped collagen fibers drive strain hardening. This approach is opposed to the Schleich *et al* (Schleich 2012) hypothesis which focuses on the matrix stiffness.

4. Conclusions

This study investigated the origin of strain hardening in the TLF using a simple semi-analytical model to test a mesostructure-based hypothesis. Strain hardening likely originates from collagen fiber folding during tissue relaxation. While this hypothesis replicated the phenomenon, no microscopic evidence confirms it. Furthermore, it remains uncertain whether this phenomenon occurs under physiological in vivo loading of the TLF. Mesostructure-based models, like discrete element models, can aid in understanding and replicating biological tissue mechanics.

Conflict of Interest Statement

None.

Contributor Roles

AL: Methodology, Writing original draft, investigation; CV: Writing review and editing; SL: Writing review and editing; JG: Writing review and editing.

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