ACTIVE CONTRACTION OF THE THORACOLUMBAR FASCIA - INDICATIONS OF A NEW FACTOR IN LOW BACK PAIN RESEARCH WITH IMPLICATIONS FOR MANUAL THERAPY

Robert Schleip, Werner Klingler MD, Frank Lehmann-Horn PhD
Applied Physiology, Ulm University, Germany
Email: robert.schleip@medizin.uni-ulm.de

INTRODUCTION
The fascia and ligaments are usually considered to be a passive element in the neuro-myofascial dynamics of joint stability. The presence of contractile connective tissue cells and the ability of fascia to contract on its own suggests that fascia may play a more active role in joint dynamics and regulation.

BACKGROUND AND PURPOSE
Tensile transmission across the thoracolumbar fascia (TLF) serves as an important element for back stability(1). Previous viscoelastic examinations of the TLF exposed to in vitro isometric stretch demonstrated an unexpected ability for autonomous fascial contraction within several minutes (2). Since the musculature of visceral organs exhibits a similar capacity, a histological examination of the TLF for intrafascial cells with smooth muscle like contractility had been suggested (2,3). Contractile cells containing smooth muscle actin have been found not only in wound healing and pathologically contracted fascia (Morbus Dupuytren, club foot, frozen shoulder) but also in normal ligaments (4,5), tendons (6) and fascia (7,3). The current study therefore examines the existence of cells containing contractile smooth muscle actin in the TLF. Additionally some of the possible variables influencing TLF active contraction are investigated.

Figure 1. Left side: tissue section of lumbar fascia from a man of 19 yrs with dense population of cells staining positively for alpha smooth muscle actin (here in black) and with high degree of collagen crimp. Right side: section from man of 76 yrs. with hardly any collagen crimping and no positively stained cells in this area.
MATERIAL AND METHODS
Immunohistochemistry: 39 tissue samples of the superficial lamina of the posterior layer of the TLF at the level of L2 and L4 from 11 human cadavers of 3 different age groups were taken (Group A: 19-26 yrs, 4 males. Group B: 54-56 yrs, 1 female, 2 male. Group C: 71-76 yrs, 1 female, 3 male). Samples were analyzed by immunohistochemistry for cells containing alpha smooth muscle actin using a monoclonal antibody.

In vitro contraction tests: Test pieces of fresh porcine lumbar fascia (size 30 mm x 2 mm x 2 mm) were dissected along the main longitudinal fiber direction. The samples were fixed at either end and oriented vertically in an organ bath containing Krebs-Ringer solution (pH buffered, supplied with 95% O2 and 5% CO2) at 35°C. Samples were prestretched with a constant 5% isometric strain. Force registrations were performed after an adaptation period of at least 30 minutes to ensure steady state conditions. The tension force of the tissue was recorded by a computer over a testing period of 15 min., during which the tissue was either challenged with electrical stimulation or with chemical stressors, thereby testing for a possible response to acetylcholine, caffeine, the NO-donator glyceroltrinitrate, and a potassium enriched solution.

RESULTS
Immunohistochemistry: Cells containing alpha smooth muscle actin were found in all tissue samples. Mean density of these cells in longitudinal sections was 128/mm² (± 51/mm²) in group A (<32 yrs), 13/mm² (±8/mm²) in group B (54-56 yrs) and 12/mm² (± 3/mm²) in group C (<70 yrs). Mean average density of contractile cells was 79/mm², with an average cellular diameter of 4.2 µm and a length of 18.1 µm. Density seemed to be related to the degree of collagen crimping, which had a mean amplitude of 8 µm (± 1.5 µm) in the youngest group, 4.4 µm (± 0.7 µm) in the middle group, and 3.9 µm (± 0.6 µm) in the eldest group. The youngest group showed significantly more contractile cells (p<0.002) and higher crimp amplitude (p>0.0005) compared with any of the two other groups.

In vitro contraction tests: Caffeine in concentrations up to 50 mM did not result in reproducible significant tension increase or decrease, neither did acetylcholine (10⁻⁸ to 10⁻⁶ M) or a potassium enriched solution of up to 80 mM. Yet electrical stimulation of 5 Hz (7 V, 2 ms) tends to increase the force (mean increase 2.1%, ± 1.7%, n=12), a 20 Hz stimulation (7V, 2 ms) tends to decrease it (by 1.6%, ±1.4%, n=10), and the NO-donator glyceroltrinitrate tends to decrease the force as well (by 1.2%, ± 1.5%, with 2 mg/ml, n=9). Because of the small number of repetitions for each tested variable, these in vitro results still require further verification.

CONCLUSION
Contractile cells containing smooth muscle actin are commonly present in the posterior layer of the thoracolumbar fascia. The density of these cells is higher in younger people and correlates positively with the amplitude of crimp in collagen fiber arrangement. Assuming the known potential force of smooth muscle cells or contractile myofibroblasts, the amount of cells could be sufficient to result in significant fascial contractions such as in compartment syndrome.

Using in vitro testing of fresh porcine lumbar fascia, a transient temporary contraction or release can be triggered by electrical as well as chemical stimulation. We suggest that these preliminary results support the possibility that active contraction of intrafascial smooth muscle like cells could play a contributing role in temporary or chronic changes in force transmission of the thoracolumbar fascia.
fascia. Further studies including antibodies specific for smooth muscle cells and in vitro tests with human samples are needed to get a more specific understanding of fascial contraction. If verified by future research, active fascial contractility could offer new insights for the understanding of low back stability, compartment syndromes, and myofascial release therapies.

![Graph showing comparison of density of intrafascial contractile cells and amount of collagen crimp between 3 age groups](image)

**Figure 2.** Comparison of density of intrafascial contractile cells and amount of collagen crimp between 3 age groups.

**REFERENCES**


Citation: