

The European Fascia Research Project

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Introduction

A.T.Still, founder of osteopathy, as well as I.P.Rolf, founder of Structural Integration, suggested that skilled practitioners can feel a fascial release in response to their manual touch. Standard biomechanical teaching however assumes that fascia serves a passive mechanical role only, transmitting tension which is generated by muscle activity or external forces. Recently, several indications have been published which suggest that fascia may be able to actively contract in a smooth muscle-like manner and consequently influence musculoskeletal dynamics. If verified, the existence of such a smooth muscle-like fascial contractility would have important implications for the understanding and further development of fascial manipulation. Our basic research project specifically examines this exciting possibility. While preliminary results of our project have already been presented elsewhere^{[i][ii]}, the following report includes some of our more recent findings.

Background

In what appears to be the most thorough examination of the viscoelastic behavior of a normal (non pathological) fascial sheet so far, Yahia et al. 1993^[iii] reported an unexpected discovery of fascial behavior, which they termed '*ligament contraction*'. In this in vitro study, pieces of human lumbar fascia were isometrically stretched for 15 minutes, then allowed to rest for 30 or 60 minutes, and then stretched again. Contrary to the authors' expectation, the resistance force of the tissues proved to be stronger at the repeated stretch compared with the previous time, i.e. they had become stiffer. After carefully ruling out other possible explanations for this response, the authors discussed the congruence of this behavior with similar in vitro stretch responses of visceral musculature, and they concluded that the most likely explanation would be the presence of smooth-muscle like cells in this fascia. They therefore suggested a histological study for these cells.

Three years later the German anatomist Staubesand reported his discovery of smooth muscle-like cells in the fascia of the lower leg. He documented this with electron microscopy^[iv]. It was suggested by Staubesand^[v] and others^[vi] that these intrafascial cells might enable the fascia to contract and relax via the control of the autonomic nervous system independent of the muscular tonus. While this explanation opens some exciting perspectives for myofascial bodyworkers, it has never been proven, and reasonable questions have been raised as to whether the number of such contractile cells in fascia is sufficient to have any significant effect^[vii].

These two studies motivated our group to start a special research project, to examine whether the lumbar fascia can actively contract. We followed three major approaches: First - a literature review on what is already known in this field. Secondly - a histological search for contractile cells in human lumbar fascia. And finally, in vitro contraction tests with fascia.

Fascial contractures

In the literature review we found many examples of tissue contractions caused by connective tissue cells called myofibroblasts. This happens naturally in wound healing, but also in several chronic fascial contractures. In the hand, it presents as palmar fibromatosis, a.k.a. Dupuytren disease, or as a pad like thickening of the knuckles. In the foot the same process as Dupuytren disease is called plantar fibromatosis. And in club foot, the myofibroblasts contraction is focused on the medial side. In frozen shoulder, the contraction happens in the shoulder capsule. Interestingly, the capsular contractures in frozen shoulder often heal spontaneously, without any therapeutic intervention; whereas this is almost never the case with the palmar fascia in Dupuytren contracture. Considering the wide spread existence of pathological fascial contractures, it seems likely to us that there may be lesser degrees of fascial contractions in normal people which may influence biomechanical behavior.



Fig. 1: Contracture of palmar fascia in Dupuytren disease. The increased activity of a special contractile type of fibroblast causes a permanent thickening and shortening of the palmar fascia, which then prevents full extension of the hand and often includes visible nodules around flexor tendons. Similar fascial contractures, which are due to the same cellular activity, are reported to occur on other frequently loaded tissues, e.g. at the foot or shoulder joint.

Contractile cells in fascia

In our second approach, the histological studies, we collected pieces of the deep fascia from human cadavers and treated them with an antibody for smooth muscle actin stress fibers. Cells containing these fibers are assumed to be either smooth muscle cells or contractile myofibroblasts. Using monoclonal antibodies, more than 100 immunohistochemical analyses of fascia have been carried out so far. While most of these were done with tissue sections from human lumbar fascia, we also included dozens of samples from plantar fascia and the Fascia lata. Additionally we included lumbar fascia samples from quadruped mammals (pigs, rats and mice). We found these contractile cells in all our samples. But there was a significantly higher density in the younger human age group than in the two older groups (Fig.2).

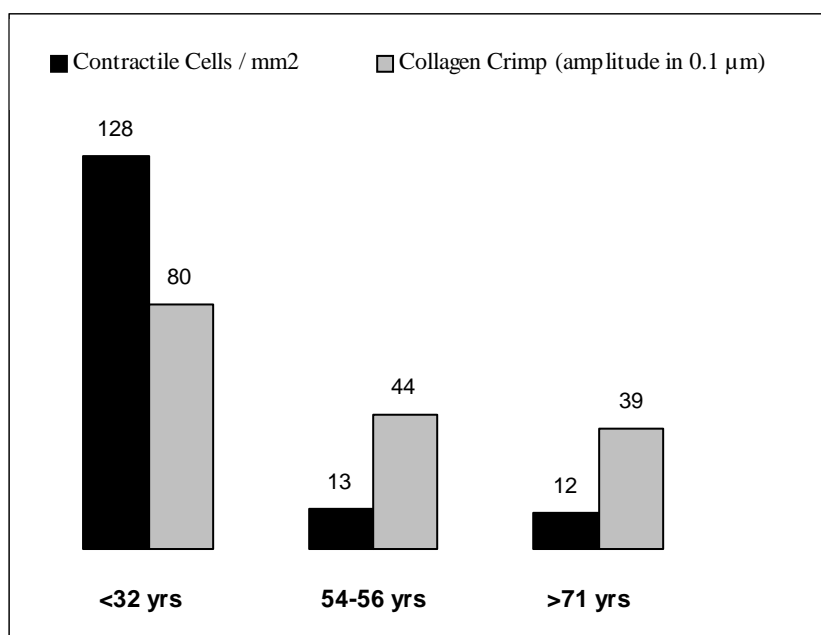


Fig. 2 . Comparison of density of intrafascial contractile cells and amount of collagen crimp between 3 age groups. The youngest age group had a significantly higher density of contractile cells than the two other age groups; and the density generally correlated positively with the amplitude of collagen crimp

We also discovered that there is a positive correlation between the density of contractile cells and the amount of crimp formation (waves) in

collagen fibers. I.e. in areas with a more straight fiber arrangement, hardly any contractile cells are found; whereas their density is much higher in areas with more wave-like collagen fibers (Fig. 3). At this stage we do not know the causal relationship behind this correlation. It could be that the cellular contraction creates the waves (which is what some authors

suggest for the contractile fibroblast in tendons^[viii]); and it could be also that a fibroblast suspension between waves increases tensional input to the fibroblasts in everyday usage, so that these cells are stimulated to become more contractile. Another interesting observation is that density of contractile cells tends to be higher areas around blood vessels and/or fat cells.

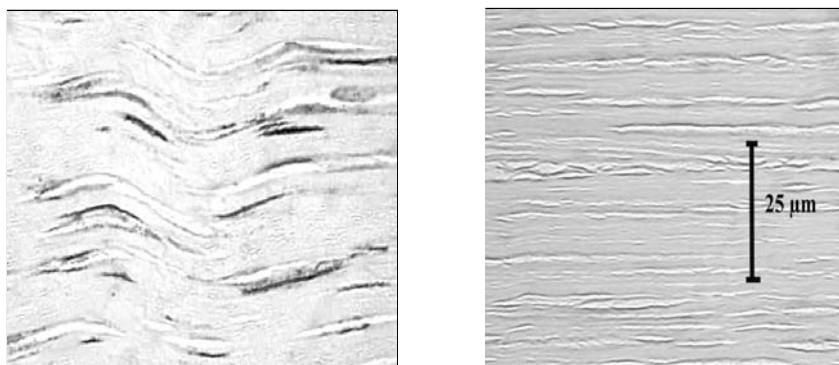


Fig. 3: Left side: **Typical tissue section of lumbar fascia** from a 19 year old man with dense population of cells staining positively for alpha smooth muscle actin (here in black) and a high degree of collagen crimp. Contrasting that on the right side is a section from a 76 year old man with hardly any collagen crimping and no positively stained cells in this area.

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The surprising matrix

In our in vitro tests, we take pieces of porcine or rat lumbar fascia, and suspend thin strips in an organ bath. This allows us to add specific drugs to the bath and measure the tensional response with a force transducer (Fig. 4). Recently we were also able to repeat these test with fresh human fascia from surgeries. Based on Staubesand's suggestion, that the contractile cells in fascia might behave similar to smooth muscle cells, we started with adrenaline and acetylcholine, at different dosages. There was no response. Then we used the vasodilator substance nifedipine, again without any clear response. That was when we began to question whether there may be other factors aside from cellular contraction, which may explain the reported tissue hardening in repeated stretches.

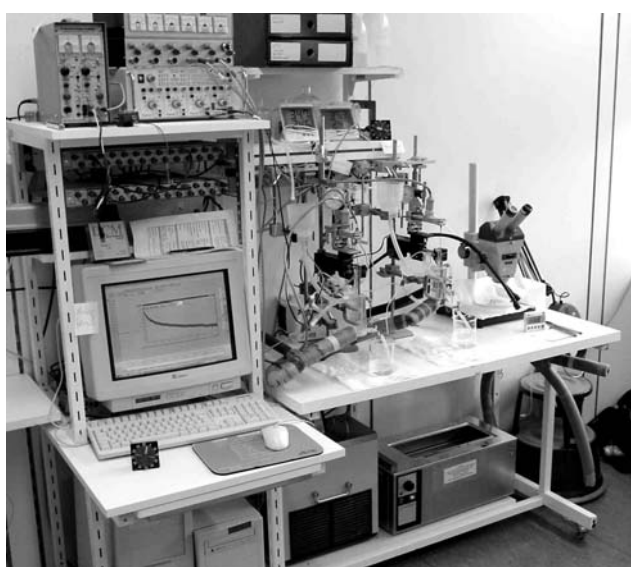


Fig.4 : A portion of our Fascia Research Lab at the University of Ulm, Germany. The test vessel with the organ bath is hooked up to the computerized force displacement transducer which records any changes in tissue resistance force. Solutions can be exchanged and titrated in a controlled fashion. Tissues can be stimulated electrically as well as pharmaceutically while tensional changes are registered.

Based on the work of Alfred Pischinger^[ix], James Oschman^[x] and Mae-Wan Ho^[xi]

we looked at the water binding qualities of the matrix, specifically the ground substance. We took strips of porcine lumbar fascia and measured the water content at various stages. Our results are shown in Fig. 5. Before the stretch, the average water content was 68%.

Immediately after a 15-minute stretch, the water content was significantly lower. Within approximately 30 minutes, the water content had returned to the original level. Then we had a real surprise. We discovered that if the strain was strong enough and the rest period long enough, the water content would continue to rise to an even higher value than before the stretch.

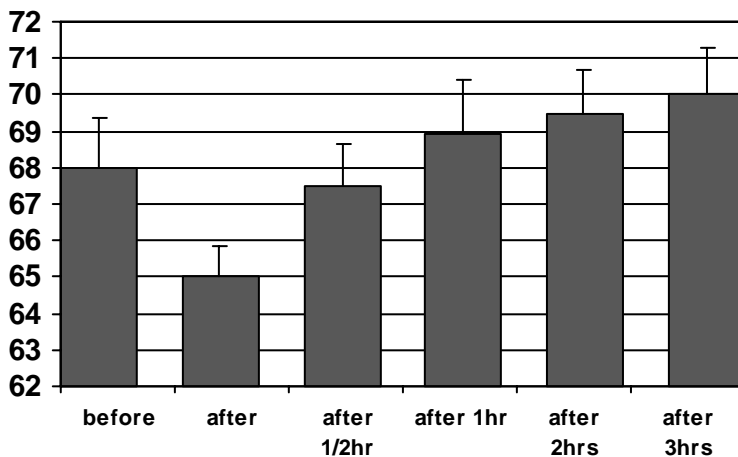


Fig. 5: Water content (in % of total tissue weight) of lumbar fascia before and after a 15 minute stretch.

In order to understand the viscoelastic effect of this, we increased the tissue hydration by putting distilled water into the bath. Here we measured the elastic stiffness in Mpa (mega-Pascale), compared with the effects of a sucrose solution, which dehydrates the tissue. The results were quite clear: an increase in water content increased the elastic stiffness of the tissues (see Fig. 6).

	Water content	Stiffness (Mpa)
Hypotone solution n=6	↑	+42%
Hypertone solution n=6	↓	-27%

Fig. 6: Water content effects stiffness. An increase in water content, induced by immersion of the fascial tissue in a hypotone solution, resulted in an increased tensional stiffness. Conversely, a decrease in water content, induced by a hypertone solution, resulted in a decreased tensional stiffness

This led us to the following conclusion. When the fascia is stretched, there are longitudinal relaxation changes in the collagen fibers and the water is squeezed out, much like what happens when you squeeze water out of a sponge. Within a few minutes the collagen fibers recover their original state. Meanwhile, water continues flooding into the tissue to an even higher percentage than before, substantially increasing the elastic stiffness.

One possible and profound conclusions is that: Fascia seems to adapt with very complex and dynamic water changes to mechanical stimuli, to the degree that the matrix reacts in smooth-muscle-like contraction and relaxation responses of the whole tissue. It seems likely that much of what we do with our hands in Structural Integration and the tissue response we experience may not be related to cellular or collagen arrangement changes,

but to sponge like squeezing and refilling effects in the semi-liquid ground substance with its intricate scrub-like arrangement of water binding glycosaminoglycans and proteoglycans. Since age related tissue changes are associated with a decreased water content, this brings up the question: Could slow but strong tissue draining moves that are a part of our work prove to increase hydration? Future studies with in-vivo measurements of the tissue water content taken hours and days after such treatments might offer interesting 'anti aging' perspectives for our field.

Test tube fascial contractions

While this may not be regarded as an active contraction process, we finally had some success with our in vitro contraction experiments (Fig.7). When we applied glyceroltrinitrate – a powerful nitrous oxide donator, which works a little like viagra - and applied that to the lumbar fascia of mice, we got a clear and significant relaxation response.



Fig. 7: In vitro contraction test: a piece of fresh porcine lumbar fascia is vertically suspended inside an organ bath. The tissue is isometrically stretched for 1 hour; then a pharmaceutical agonist is added into the bath and any tension changes in the tissue are registered.

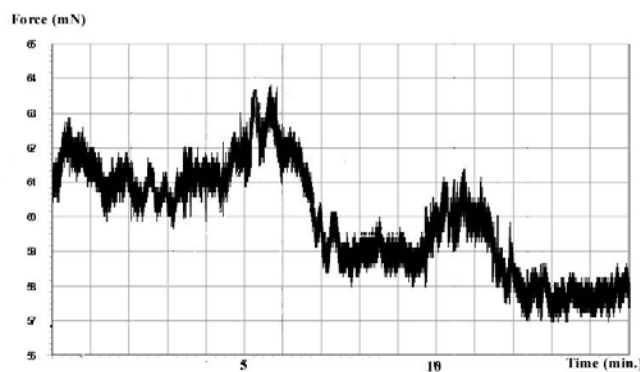


Fig. 8: Example of an IVCT experiment. The addition of a NO-donator substance at minute 5 and minute 10 is followed each time by a tension decrease.

We also found, that Dr. Ian Naylor and his research group at the University of Bradford (U.K.) had recently reported positive results with a number of additional substances. This group, which we are now collaborating with, is specialized in research on the pharmaceutical regulation of wound healing and Dupuytren disease. For this purpose they have also started to conduct tests with normal uninjured fascia. Using the lumbar fascia from rats, they reported a clear contraction response with the substance mepyramine which is generally considered to be the most reliable agonist in myofibroblast research ^[xii].

On our invitation Dr. Naylor visited our lab, and in return, we worked with Dr. Naylor in his laboratory at Bradford university during two one-week visits so far. We were able to repeat his positive contraction results with rat lumbar fascia in response to mepyramine. Additionally with rat testicular capsule (a fascial organ capsule with a high density of

myofibroblasts) we could demonstrate contractile responses to the smooth muscle agonist adenosine as well as to the hormone oxytocine. Repeating the in vitro test with mepyramine on fresh human surgical fascia at our own laboratory at Ulm university, we were also able to confirm the existence of an active fascial contractility for human fascia.

What does fascia have in common with sea shells?

Based on a very early suggestion by Prof. Gabbiani, original co-discoverer of myofibroblast cells, we recently explored whether the slow and sustained contraction of fascial myofibroblasts may have some similarity with the catch musculature of ocean mussels which are known for their ability to sustain contractions for very long times. When members of our team together with Dr. Naylor applied this ocean muscle tissue to our in vitro contraction tests with mepyramine, we got an almost identical response curve as we usually got with our fascia tissues. This suggests that the contractility of fascial myofibroblasts may not only include some smooth muscle features, yet it may also include strong similarities with the kinetics of molluscan catch muscles. This would explain, how fascial myofibroblasts can hold a particular tension for very long times, while using only a fraction of the energy for that compared with skeletal or smooth muscle cells.

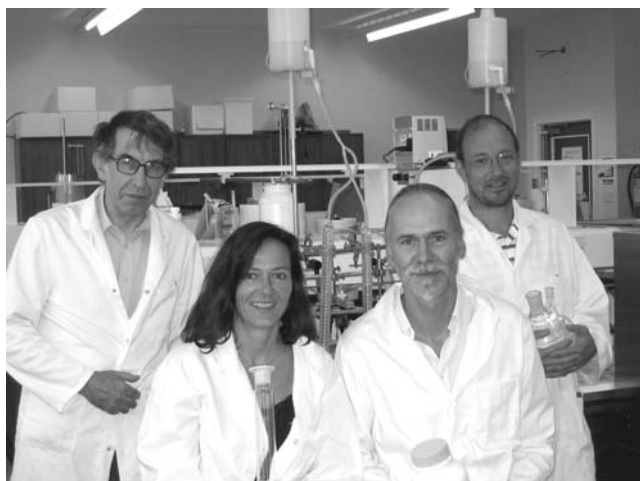


Fig. 9: Members of the European Fascia Research Group working together with Dr. Ian Naylor.
(Left to right: Dr. Ian Naylor, Birgit Frank, Robert Schleip, Adjo Zorn Ph.D.)

Implications for passive muscle tone

Some of our histological tissue sections did not only include the proper fascia but also small pieces of related skeletal muscle tissue. When we started to look at these portions more carefully, we discovered much to our surprise, that the intramuscular connective tissue often had a higher relative density of contractile cells than the proper extramuscular fascia. Quite often the intramuscular fascial layer of the perimysium, which envelopes bundles of muscle fibers, was particularly packed with myofibroblasts. We speculate that this may be related to the spatial proximity of intramuscular blood vessels (and probably also nerves) which tend to travel along this layer. It is known that tonic muscles such as the soleus or the upper trapezius generally include a thicker perimysium; which also makes them the tougher meat nutritionally. Interestingly these are the same muscles which are more prone for chronic shortening or stiffening as compared with phasic muscles. E.g. persons with spastic myopathies often show increased problems in motor performance, particularly in locomotion, due to chronic shortening of the soleus or other tonic muscles.

Based on these considerations, we treated rat soleus muscle tissue in our organ bath with a 'knock out protocol' of high caffeine additions into the bath until it was clear that the myocells in the tissue were depleted and no more able to contract. At that point we added our usual myofibrblast contractile agent mepyramine, which triggered nevertheless a clear and long lasting contractile tissue response. Since the tissue had previously shown to be

unable to contract to electrical stimulation as well as to additional dosages of caffeine, we tend to assume that this subsequent response is due to an active intramuscular connective tissue contraction.

Since recent research by a group around Prof. Huijng's in Amsterdam^[xiii], ^[xiv] indicates that intramuscular connective tissue significantly influences passive muscle elasticity, we now speculate that an active shortening of the intramuscular perimysium may contribute to chronic stiffness of tonic muscles which is so often found in pathological conditions as well as in many of us 'normal people'^[xv].

Potential strength of fascial contractility

To calculate the potential in vivo contraction force of the lumbar fascia, we chose the data from the experiments with human lumbar fascia by Yahia et al., reported earlier. With a tissue strip of 1.5 mm x 1.0 mm x 30 mm the maximal measured force increase was 4.2 N. If we hypothetically apply the same force ratio to whole fascial sheets in the human body, it seems clear that such fascial contractions could have substantial biomechanical influences. As an example, the superficial lamina of the lumbar fascia, with a reported horizontal cross sectional area of 71 mm x 0.53 mm at the level of the third lumbar vertebra (plus adjusting for the 45 degrees oblique fiber angulation in this fascial layer) would have a theoretical contraction force of 51 N (equal to 5.1 kg).

This would put the force of active fascial contractions within a biomechanically significant range, at which it could cause a lumbar paraspinal compartment syndrome^([xvi]). It is also in a range where a decreased fascial tonus can contribute to spinal segmental instability, which is frequently associated with the onset of low back pain^([xvii], [xviii]). Similarly a loss of fascial tone could also be responsible for sacroiliac pain, which is often caused by a lack of force closure of the sacroiliac joint^([xix]) and resulting hypermobility (an example of this is the high incidence of pelvic pain during pregnancy due to hormonal changes).

Interim conclusions

To summarize our conclusions to date:

- Chronic contractures of frequently loaded tissues are common adaptations, driven by cellular contraction within fascia.
- Fascia is capable of performing smooth muscle-like, slow contractions, through a surprising regulation of its water content. Since dehydration is an intrinsic aspect of aging, specific stretching routines or manual therapies may be worthwhile study projects in anti-aging.
- Our in vitro contraction results as well as those of Dr. Naylor give evidence that a short term fascial contractility, happening over minutes, does exist. This may have important implications for the understanding of back stability as well as deep tissue therapies such as Structural Integration.
- Certainly, fascia proves to be a truly fascinating tissue, and warrants further investigation

Practical applications

Already at this point, this research has changed our Rolwing work. Our insights about the scrub-like water binding nature of the semi-fluid matrix, now bring up images and a more detailed caring for the sponge-squeezing and refilling effects of our work. It suddenly makes new sense, why a repeated slow-draining stroke followed by appropriate rest sometimes

makes all the difference. And why such treatment often works wonders in rejuvenating dried-out tissues. On the other hand, having observed thousands of spindle shaped cells floating in the collagenous matrix in our microscopes, our working fingers now frequently feel like they are contacting similar fish-like creatures under our hands. Future results of this project may help us to understand more specifically, which of our effects are due to dynamic changes in the water content, and which may be due to cellular contractile changes. These insights may also help us to find out how different durations and amounts of manual pressure may result in different short term or long term fascial effects.

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