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European Fascia Research Project Report

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What happens in fascia when we touch it? According to Ida Rolf, fascia is a plastic medium capable of responding to manual pressure. Yet, we still don't know where this adaptability comes from. When we feel a fascial release under our hands, which of the elements in fascia is responding? Is it the collagen or elastin fibers, the fibroblasts or perhaps the groundsubstance? Can fascia really change its tonus independent of the involved musculature? And finally, how does fascia respond to different types of pressure over varying amounts of time?

If these questions haven't stirred up your interest or curiosity, take a look at Figure 1. What would you expect to feel when `rolfing` this piece of alive fascia?

Fig. 1: 'Is it possible to 'rolf' a piece of fresh fascia? This piece of lumbar fascia from a pig has been preserved in a special organ bath. Most of the tissue features (like cellular responsiveness, water content, piezoelectric conductivity, temperature, etc.) are still the same as in the living animal.

Fascinating questions such as these motivated our group to conduct a basic research project, starting in August 2003. Our goal was to understand more about fascial plasticity and about fascial responsiveness to manipulation in general. The experiment just described, was a small playful excursion during our otherwise more serious scientific research work[1]. While we plan to publish our research in scientific journals in 2006, an interim report will be delivered at the 5th World Congress on Low Back and Pelvic Pain in November 2004. This article is our way of sharing our experiences and insights with our bodywork colleagues.

Abbreviations:

FB:	fibroblast (the main cell type in fibrous connective tissue)
IH:	immunohistology (tissue analysis which uses antibodies for marking specific particles)
IVCT:	in vitro contraction test
MFB:	myofibroblast (a contractile type of fibroblast)
NO:	nitrous oxide
SM:	smooth muscle
SMC:	smooth musce cells
TGF:	transforming growth factor, (a multifunctional protein that plays a central role in the regulation of cell growth and differentiation)

Driven by curiosity

One of the scientific starting points of our project was a classic paper by Yahia et al. on the biomechanical behavior of fascia[2]. Using the human lumbodorsal fascia, this extensive in vitro study explored the viscoelastic properties of dense connective tissues, such as stress relaxation and creep. In other words, they measured the degree of elastic and plastic changes of fascia in response to mechanical loading. During these tests the researchers also discovered a new and unexpected behavior: When stretched at a constant length, the tensional resistance of the tissue slowly relaxed (as was expected); yet when the same tissue was stretched again after an appropriate time of rest, the tissue had not only regained its original strength, but it proved to be even stronger than before. Termed '*ligament contraction*', this remarkable tissue behavior reminded the researchers of a similar stretch response in visceral organ tissue, and they recommended a histological study of the lumbodorsal fascia for contractile cells with smooth muscle properties.

While this particular histological study of fascia has not yet been done, smooth muscle cells (SMC) were discovered in the fascia of the lower leg a few years later; and it was suggested by Staubesand[3] and others[4] 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 questions have been raised as to whether the number of such contractile cells in fascia is sufficient to have any significant effect[5].

The Research Teams

The original group began with two enthusiastic Rolfers fascinated by the diverse properties of fascia and inspired by the possibilities hinted at in the research of Yahia and Staubesand. The project was framed as a PhD dissertation project in human biology. After several months of literature review and personal counselling by Rainer Breul PhD (a Munich university anatomy professor witha focus on fascia), Jochen Staubesand PhD (now emeritus professor of anatomy and the first person to discover intrafascial SMCs) and others, we decided to explore these questions following two main methodological approaches. The first approach consists of in vitro contraction tests of fresh fascia strips in response to chemical, electrical and mechanical stimulation, and the second approach is a histological study of contractile cells in human lumbar fascia.

Since those early days, the scope of the project has gained momentum and increased substantially and now includes several research teams at different universities. Although more than a dozen people are involved, their activities are primarily coordinated by the three authors of this report.

Fig. 2: Part of the histological team in Munich. Left to right: Julia Vogts, biologist, Joerg Massmann PhD, chair of the department, Christiane Barth, technical assistant, Ursula Meister, technical assistant.

The in vitro contraction test (IVCT) team now proudly runs a special '*Fascia Research Lab'* at the Department of Applied Physiology, University of Ulm, Germany (Fig. 3). Under the guidance of the head of department, Frank Lehmann-Horn PhD, we conduct in vitro tests with fresh strips of animal and human fascia. The histological team is led by Joerg Massmann PhD, a former work colleague of Dr. Staubesand and presently a leading researcher on SMC anomalies. Since October 2003, his Munich team of histologists has been working regularly on our fascia research project (Fig. 2). Using monoclonal antibodies, more than 90 immunohistochemical analyses of fascia have been carried out so far, most of them with tissue sections from human lumbar fascia.

Starting our own Fascia Research Lab

After having conducted the initial test runs in a commercial lab over several months, Dr. Lehmann-Horn's offer to use his university lab for our research, provided a giant step forward for our project. With technical assistance we successfully modified the unique muscle-IVCT equipment which the department is well known for, to suit our specific needs. With this state-of-the-art equipment we can perform computer assisted tensional force measurements of fascia slips, which are suspended in equilibrated physiological solution, with controlled temperatures and a constant supply of 95% oxygen and 5% carbon gas. These ideal physiological test conditions allow us to keep cellular responsiveness in fascia alive for up to 6 hours during our in vitro tests (Fig. 8).

Fig. 3: Members of the Fascia Research Lab, University of Ulm. Left to right: Ursula Mohr, technical assistant, Frank Lehmann-Horn MD PhD, senior scientist & research director, Sunisa Chaiklieng BSc, Robert Schleip MA, Werner Klingler MD.

About half of the fascia we use is pig tissue, which we collect early in the morning from the local slaughter house. Our other samples are from lab mice, and since September 2004 we have had the privilege of acquiring fresh samples of human fascia lata once a week from the surgery.

International networking contacts

We are working in conjunction with Myron Spector PhD and his group at the Harvard-M.I.T. Division of Health Sciences in Boston. Their research is focused on the ability of connective tissue cells to become contractile[6]. It was their suggestion to use the cytokine TGF-beta as a contraction stimulant in our current in vitro studies. We have also received the support of Jochen Staubesand PhD from Freiburg, Germany and Giulio Gabbiani PhD from Geneva, an international authority on myofibroblasts.

Our collaboration with Priscilla Barker's team at Melbourne university, which is studying the innervation of the human lumbar fascia has been another source of inspiration. Currently they are repeating and expanding on an earlier histological study by Bednar[7], who found no evidence of sensory nerve endings in the lumbar fascia in low back pain patients as compared with healthy people. While this finding may concur with the observed lack of proprioceptive accuracy in low back pain patients' experience of their pelvis and lower back position[8], it is also reminiscent of a similar correlation between diminished peripheral sensation and a distorted cortical perception in phantom pain and tinnitus[9,10]. If verified, these insights could open exciting avenues and further research into proprioceptive training as well as a specific manual stimulation of fascial mechanoreceptors to facilitate an increased development and function in these receptors. Barker's research group will also present their results at the upcoming world congress in Melbourne and both teams are eagerly anticipating exchanging their experiences.

Learning from others

An important part of our research project has been and continues to be a thorough review of all the current literature in our field. This is how we learned that connective tissue cells are now considered to be quite similar to foetal stem cells, i.e. they are cells which are still fairly undifferentiated and therefore maintain the ability to adapt their nature to different functional needs. All fibroblasts (FB), for example, maintain the ability to become contractile by expressing smooth muscle (SM) actin stress fibers as well as special focal adhesions on their membrane. Not only FBs, but also chondroblasts and osteoblasts can change their morphology to become "*connective tissue cells with muscle*" [6]. For FBs this happens regularly in wound healing as well as in pathological tissue contractures like palmar fibromatosis (Dupuytren disease), frozen shoulder[11], Peyronie's disease, plantar fibromatosis, or club foot[12].

Yet the same kind of contractile cells have also been found to make up a significant portion of the FBs in healthy people; e.g. in the cruciate ligament of the knee[13], the Achilles tendon, the periodontal ligament, or in digital flexor tendons. When taking the maximum contraction force of such cells as determined in a cell culture and multiplying that with their reported density, it seems clear to us that the outcome could be sufficient to allow a significant and palpable effect on local tissue tension.

Our original hypothesis, which is shared by Professor Staubesand, was that the contractile cells in fascia would probably have the same physiology and innervation as visceral or vascular SMCs. This hypothesis had a certain appeal to the manual therapists in our team, since its verification would nicely support Staubesand's credo of an intimate two-way connection between fascia and the autonomic nervous system. Nevertheless, this hypothesis now appears unlikely to be substantiated in the light of recent advances in FB research. Here´s why: FBs exist in a certain heterogeneity. Those phenotypes which contain SM-actin stress fibers are often called myofibroblasts (MFB), and many of those have several features in common with SMCs. Yet MFBs have been clearly shown to express different proteins and to use different messenger substances and energy processes for contraction than SMCs. While most SMCs can be easily influenced by input from either sympathetic or parasympathetic nerves, contraction of MFBs is regulated by specific cytokines (like TGF, or fibronectin) and by mechanical tension[14].

In vitro fascial contraction

Now we'd like to share some of our discoveries with you, starting with our first line of approach, the in vitro tests at our *Fascia Research Lab*. Similar to the classical experiments of Yahia, we suspend strips of lumbar fascia in a physiological organ bath (Fig. 5). Both ends of the strip are attached to a computerized testing apparatus which can both elongate the strip in a controlled fashion and also measure the resistance force with a precision of 100 nano-Newtons. When working with pig fascia, we use strips of 40 mm x 5 mm x 1 mm; when testing the lumbar fascia in humans or mice, our strips are about a half that size (Fig. 4).

Fig.4 (left): **a piece of fresh porcine lumbar fascia**, ready to be cut into strips for testing

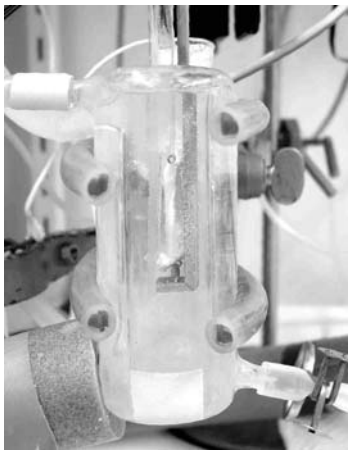

Fig. 5 (right): **Test strip suspended in a temperature controlled double-walled glass container** with constant supply of pH-balanced Krebs-Ringer solution, bubbled with 95% O^2 and 5% CO^2.

We were able to show that Yahia's observed '*ligament contraction'* not only happens in human fascia, but also in mice and pigs; i.e. when we stretch a fascial strip for 15 minutes, allow it to rest for 30 minutes, and then stretch it again, the tissue resistance tends to be stronger the second time. In May this year, we began to repeat the same tests with fascia in which the cells had been destroyed by deep freezing in liquid nitrogen and subsequent rapid thawing. We wondered if we still could get a similar '*Yahia effect'* of a relative contraction at the repeated stretch with these tissue samples? And yes we clearly did, although to a slightly lesser degree. This now leads us to believe that the observed contraction does involve, at least partially, some non-cellular factors.

Water, the surprising element

Based on the contributions of Alfred Pischinger[15], James Oschman[16] and Mae-Wan Ho[17], we began to look at the water content in the ground substance. By carefully measuring the wet weight of our fascial strips at different experimental stages plus the final dry weight (after later drying the strips in an oven), we found the following pattern: During the isometric stretch period, water is extruded, which is then refilled in the following rest period. Interestingly if the stretch is strong enough and the following rest period long enough, more water soaks into the ground substance than before. The water content then even increases to a higher level than before the stretch[18].

Could this be the explanation for the observed '*ligament contraction'* in both Yahia's and our experiments? In the literature we found mixed information on the effect of increased hydration on connective tissues:

some studies (e.g. with cartilage) show that increased hydration leads to an increase in stiffness, others show an opposite tendency. To clarify the effect of hydration on our tissues, we performed a series of tests in which we replaced our usual physiological solution with distilled water (which tends to increase tissue hydration) or with 25% sucrose (which dehydrates the tissue). The results were quite clear: Increased hydration increases the elastic modulus i.e. stiffness. The picture starts to look like this to us. When fascia is being stretched, water is being extruded from the ground substance and simultaneously there are some temporary relaxation changes in the longitudinal arrangement of the collagen fibers. When the stretch is finished, the longitudinal relaxation of the fibers takes a few minutes to revert (provided the strain has not been too strong and there have been no micro-injuries); yet the water continues to be soaked up into the tissue, to the degree that the tissue even swells and becomes stiffer than before.

One possible and profound conclusions is that: Fascia seems to adapt in 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.

Cellular contraction

Nevertheless, at this time we believe that there is probably also a cellular effect, which may allow a slow fascial contraction. Our reasoning behind this is based on the literature study of such fairly common fascial contractures as in frozen shoulder, palmar fibromatosis, or club foot. Recent studies have shown, that these aberrations are due to cellular contraction within fascia; and that the amount of contraction responds to mechanical stress as well as specific chemical messengers. If such adaptive processes show up pathologically in different parts of the body, it seems also probable – although not certain – that there also exists a span of different degrees of fascial contracture among normal healthy people. Interestingly, palmar fibromatosis rarely disappears without any intervention, whereas such spontaneous improvements are quite common in frozen shoulder. This makes us wonder whether similar fascial contractures exist to a minor degree in normal people and that some of these may have the same temporary nature and spontaneous responsiveness as was observed in the examples of frozen shoulder.

Fig. 6: Rainer Breul PhD, anatomy professor at the Ludwing Maximilian University, Munich. Regular advisor and enthusiastic supporter of our project.

It was our advisor Professor Breul, who made the suggestion to build a testing apparatus in which we could perform IVCTs with fresh pig fascia to test the response to several chemical stimulatory transmitters (Fig. 6). Based on Staubesand´s suggestion, that the contractile cells within fascia would most likely be sympathically innervated and may work like SMCs, we conducted our first test rows with the addition of the sympathetic transmitter adrenaline. Although three of our first tests did appear to elecit a contractile response (and we consequently toasted one evening with a small bottle of champagne), more than 30 tests later we now believe these results were mere statistical deviations and that adrenaline does not have any significant effect on fascial contraction, at least not within a time period of up to 45 minutes.

After gathering suggestions from several different experts, we then tested other substances and solutions, such as acetylcholine, caffeine, and a depolarizing potassium solution; all without any significant statistical

effect. Since each substance needed to be tested on different samples and protocols, the testing with just these three solutions kept us busy for several weeks.

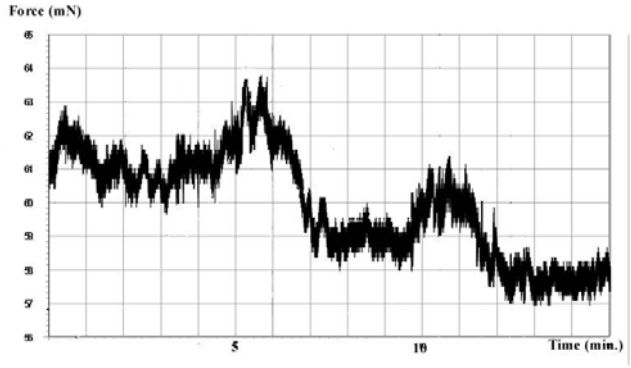

Fig. 7: Example of an IVCT experiment. The addition of a NO-donator substance at minute 5 and minute 10 is followed each time by a tension decrease.

We also got some positive responses. When we tested two SM relaxants which are known as vasodilators (nifedipin and glyceroltrinitrate), we got a significant relaxation effect with the second one (Fig. 7). This second substance works as a NO-donator, i.e. it produces nitrous oxide, which acts as a gaseous transmitter and can pass through most of the body´s membranes. Additionally we got a significant effect in response to electrical stimulation: a frequency of 5 Hz showed a clear force increase, and a stimulation with 20 Hz a decrease. Since by then we had become obsessed with constantly questioning our results and checking for possible experimental artifacts, we observed one day, how in most of the 20 Hz-applications there was a slightly stronger turbulence in the solution than without electric stimulation. While such processes known as electrophoresis are hard to prevent, we finally came up with an idea to check their effect upon our force measurements with a piece of thin elastic rubber replacing our fascial strips. Would we still get a similar response pattern from the different electrical stimulations? Yes we did. We therefore concluded that our previously observed electrical effect on fascia was a mere experimental artifact of the electric stimulation, and not due to a change in cellular contraction.

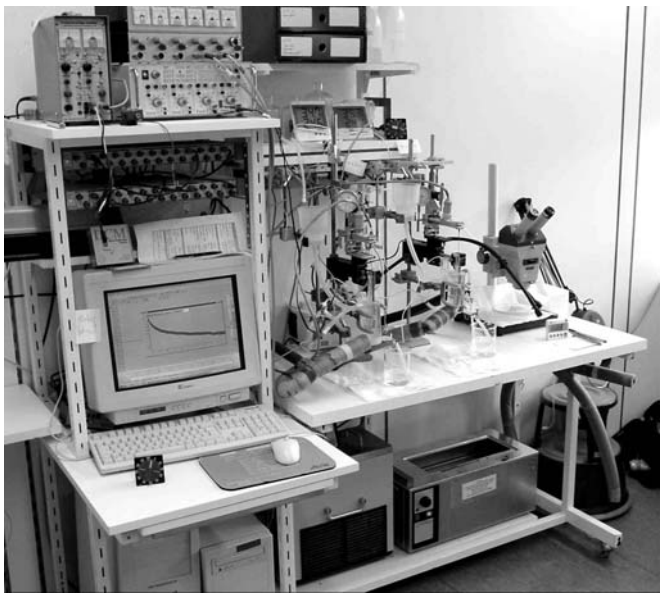

Fig. 8 : A portion of our Fascia Research Lab at the University of Ulm, Germany. The test vessel with the organ bath is hooked up to the computerized force displacement transducer which records any changes in tissue resistance force. Solutions can be exchanged and titrated in a controlled fashion. Tissues can be stimulated electrically with different voltages and frequencies.

Currently we are following Gabbiani's suggestions by checking our fascial strips with 1-hour long protocols for a response to the agents endothelin-1, histamine and angiotensine, as well as with a 4-hour long protocol with the cytokine TGF-beta. While we have not completed these time consuming tests, our preliminary results already indicate a contractile effect of histamine.

Our histological discoveries

What did we find out with our second line of approach, the tissue analysis via immunohistology (IH)? With good luck, enthusiasm, private money, patience and a lot of paperwork we managed to establish a clinical support network in Munich and to obtain fresh tissue sections from human autopsies. Within 1 day after death small samples of fascia are taken out, put in formaldehyde and brought by courier to our histology lab. Here they are first embedded in paraffin and then stored until we start our IH procedure. Consisting of 18 steps, the IH treatment includes the application of a special antibody which marks only those cells which contain alpha-SM-actin stress fibers and are considered to be contractile. The really interesting part for us is in the following days, as we watch those cells in special microscopes. Our sense of excitement in this particular part of our work comes from the fact, that this is to our knowledge the first time that contractile cells in fascia have been systematically observed in normal people, and that patterns in the arrangement and density of these cells can be recognized.

Fig. 9 shows the results of IH analysis of the lumbar fascia from 11 human cadavers. We found FBs containing alpha-SM-actin in all specimens. Mean density of these cells in longitudinal sections was 79 per mm^2. Assuming the known potential force of MFBs, the amount of cells could be sufficient to result in significant fascial contractions such as in compartment syndrome. Interestingly, the density of contractile cells was statistically higher in our younger age group (<32 yrs) than in the two older groups. However, one cadaver of an elderly exhibited a high number of stained FBs. Unfortunately there is no information on the physical activity of this individual.

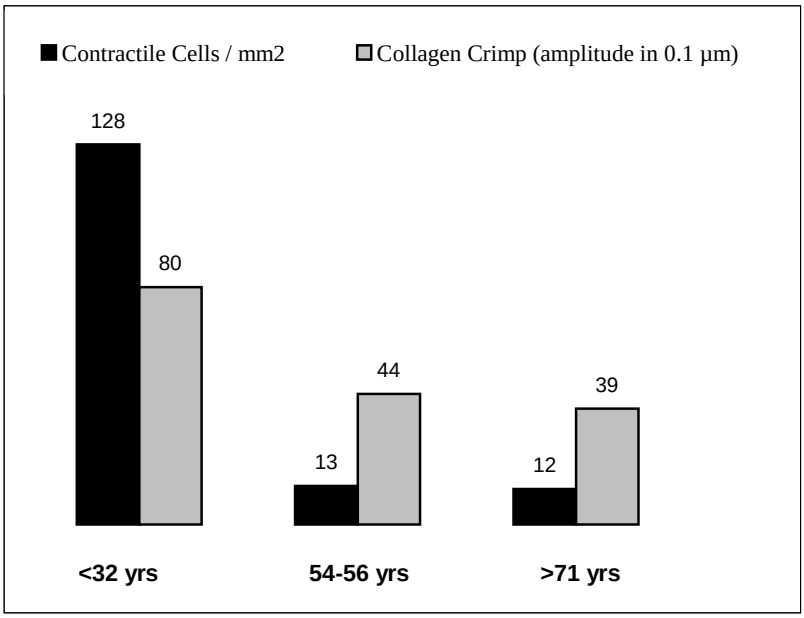

Fig. 9 . Comparison of density of intrafascial contractile cells and amount of collagen crimp between 3 age groups. The youngest age group had a significantly higher density of contractile cells than the two other age groups; and the density generally correlated positively with the amplitude of collagen 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. 10). 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 FBs in tendons[19]); and it could be also that a FB suspension between waves increases tensional input to the FBs in everyday usage, so that these cells are stimulated to become more contractile.

Interestingly when doing the same IH analysis with the lumbar fascia of two of our young lab mice, we found an amazing density of FBs; yet none of them had any SM-actin stress fibers. Before jumping to any premature conclusions, we decided to wait for the IH results from the following tissues which we had already collected: lumbar fascia & fascia lata of 5 more mice, lumbar fascia & fascia lata of 6 pigs, fascia

lata & plantar fascia of 5 humans. We expect that the results will help us to understand more about the general function of contractile cells in fascia. It is possible, that we may discover that the existence of such cells is primarily related to micro-injuries and resultant repair processes. On the other hand, it is also possible that they will suggest, that density of such cells is mainly driven by everyday tensional stimulation (as our observed correlation with the amount of collagen crimp seems to indicate). This would support the notion that fascial contractility serves indeed as a secondary and adaptable tension control system in the body.

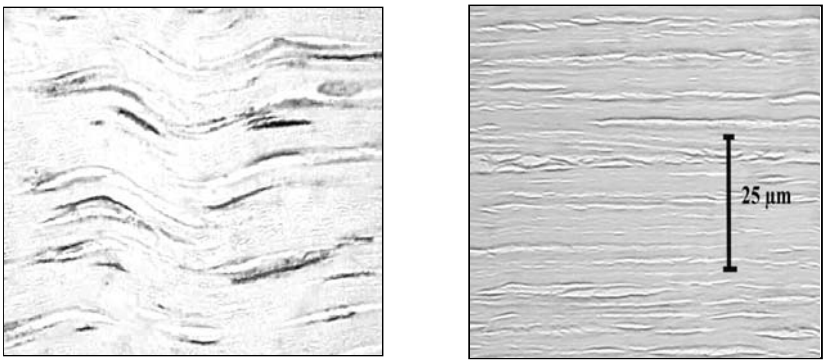

Fig. 10: Left side: **Typical tissue section of lumbar fascia** from a 19 year old man with dense population of cells staining positively for alpha smooth muscle actin (here in black) and a high degree of collagen crimp. Contrasting that on the right side is a section from a 76 year old man . with hardly any collagen crimping and no positively stained cells in this area.

Treasures ahead

This research project continues to feel like a real '*treasure hunt*' to all of us involved. Already at this stage we are certain that our results will contribute towards shifting the traditional concept of fascia as a passive tension transmitter to a new picture of fascia as a dynamically adaptable organ. Together with the work of Barker et al. on the sensory innervation of fascia, our findings support the notion of Andrew Taylor Still and Ida Rolf, that fascia is much more alive and responsive than commonly assumed and that working through this adaptable medium can have profound effects on the whole organism.

Additionally our research may shed some light upon - and open new therapeutic avenues for - pathological conditions which are associated with an increased or decreased fascial tone. Examples include plantar fascitis, fibromyalgia, compartment syndromes (including those in lower arms and erector spinae), carpal tunnel syndrome, flat foot, fascial contractures (as in frozen shoulder, Dupuytren disease, plantar fibromatosis or club foot), or several of the common muscular contractures as in tensional headache, low back pain, tennis or golfer's elbow, etc.

As far as we know, we are currently the only experimental research group on fascial contractility in normal (healthy) people. Unless we stumble unintentionally over some pharmaceutically marketable insights, it is unlikely that we will receive any government or industry funding. And although it may be partially disappointing, it is indeed understandable and reasonable, that the Rolf Institute´s research committee feels obliged to the membership to invest their funds in clinical research which proves the efficiency of Structural Integration, rather than into basic research. By definition, the outcome of basic research is unpredictable. Yet we feel, that finding out more about what is still unknown about our work, is most needed in our field, and that is how we can best foster the development or our work. Currently our research is driven entirely by our personal passion. It is financed out of our own private practices, and supported by a lot of volunteer work and free service from other people and institutions who we have managed to infect with our enthusiasm. Our sincere appreciation also goes to the European Rolfing Association, which continues to support us in many helpful ways.

Fig.11: Happy brainstormers at the 'Berlin Think Tank': Divo Gitta Müller, Robert Schleip, Adjo Zorn PhD

We feel quite honored and privileged to be invited to present our interim results at the upcoming 5th Interdisciplinary World Congress on Low Back and Pelvic Pain[1], and we expect this to attract some attention and networking contacts for our project. We feel this remarkable invitation is also reflective of a general increase in interest in fascia among low back pain researchers. We plan to publish our results in scientific journals in 2006, and will of course also continue to share our discoveries and questions with you, our fascial bodywork colleagues. If you, dear reader, know of any private person or a foundation with the means and willingness to support such a unique research project, please do connect them with us[20]. And yes, we love to have more people join in our efforts and network with us in what feels like an important and exciting journey of discovery (Fig. 11).

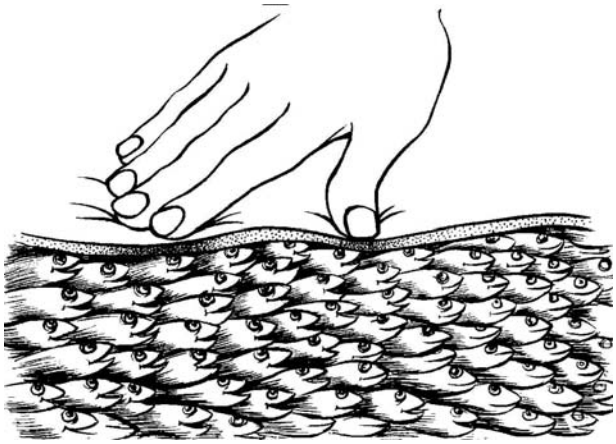

Fig 12: Knowing about the existence of contractile cells in fascia can influence your touch perception. These spindle-shaped (fish-like) cells can cause chronic tissue contractures, yet they have also been shown to be responsive to mechano-stimulation. (Illustration reprinted with permission of Elsevier Science from: Schleip R 2003 Fascial plasticity – a new neurobiological explanation. Journal of Bodywork and Movement Therapies 7(2):104-16)

Last but not least: our research has changed our Rolfing 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 (Fig. 12). Such beautiful and enriching touch perceptions already make the whole journey worthwhile for us. Yet they also foster our growing curiosity. Wouldn't it be nice to understand even more about these subtle tissue responses under our hands?

NOTES:

[1] Our subjective impression in this experiment was, that we felt a similar responsiveness to our experience of working with fascia in the living human body. Of course, aside from the obvious possibility of human interaction, we were also missing the response transmission from many external factors, such as connected musculature, breathing, etc..

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