The first Fascia Research Congress, held in Boston in 2007, brought together top scientists and alternative medical practitioners with one common goal: to explore the latest and best scientific research findings on the human fasciae in all its forms and functions.

Co-initiator and organiser, Robert Schleip’s interest in fascia began with his work as a Rolfer. He later travelled to the ‘scientific world’, completing a PhD in fascia biology. This article is based on Robert Schleip’s presentation - ‘Alice in Wonderland: Getting Curiouser and Curiouser’ – given at the third World Fascia Congress in Vancouver, 2012 …

Organisers of the Fascia Research Congress, Thomas Findley and Robert Schleip came from opposite ends of the spectrum. While Schleip traversed the ‘fascial matrix’ from clinician to scientist, Findley did the reverse. Initially, Findley worked as a medical scientist, studying the science behind rehabilitation medicine, before subsequently studying Rolfing and becoming the director of research at the Rolf Institute of Structural Integration in Boulder, Colorado. Their goals for the conference were twofold: to inform practitioners of the science behind what they were doing in terms of fascia, while demonstrating to scientists the clinical applications of their work.

While combining the two groups may seem natural, bridging the gap wasn’t so easy, with some scientists hesitant to attend, fearing their reputations would be damaged. Despite this initial reluctance on the part of scientists, the first Congress was a resounding success and, more than five years later, the Congress is still providing for the exchange of ideas and current research on the human fasciae.

Incorporating science into practice
Schleip has compared his journey into the world of science to a ‘trip to wonderland’; and has even been quoted as calling himself a ‘born-again scientist’. In his talk at the third World Fascia Congress, Schleip discussed areas where his visits to ‘science wonderland’ bore fruit:

Fascia and fluid dynamics
It is recognised that water constitutes around 68 per cent of the volume of fascial tissues, and fascia is responsible for regulating fluid flow in the extracellular matrix. This fluid flow, in turn, can cause fascial remodelling. In an in vitro study (using animal fascia), Schleip and co-workers at the University of Ulm (Figure 1) measured the water content in facial tissues before and immediately after the tissue was stretched.1

The study found that during tissue loading (fascia stretch), water was significantly extruded from the tissue and then, after the manipulation, the tissue gained hydration. The study also found that these water content changes were associated with significant changes in tissue stiffness. This phenomenon is due to the behaviour of the ground substance in the extracellular matrix, which is prevented from absorbing fluid by tension exerted by fibroblast cells on the extracellular matrix fibres. When this tension is relaxed, the extracellular matrix can absorb fluid rapidly.

Another study, conducted by Rutkowski and Swartz, described how subtle change in fluid shear on cell culture profoundly changes the fibroblasts2. Fibroblasts are most responsive to fluid shear - ie to the slow motions of the water around them - as sensed through their antenna-like cilia (soft tentacles).

Figure 1. Changes in the mean water content of a porcine lumbar fascia before and following a 15 minutes stretch with a 4% strain (After Schleip et al., 2012)
The study found that a large portion of the impact of collagen stretch is less due to the direct effect of transmitting that stretch to the cell membrane than to the sensation of the fluid shear induced by collagen fibre reorientation, which, in turn, is sensed by the hair-like cilia.

This can be illustrated as follows: imagine how much the hairy tip of a painter’s brush would bend if you move it at a steady speed through a fluid medium. Or imagine moving a finger through yoghurt. Both the speed as well as the viscosity of the fluid medium will influence the amount of shear. The clinical implication is that if you move very slowly at a constant speed through a dense tissue area, then the tiny cilia of the fibroblasts will be bent only very gently by the resulting fluid shear. This seems to stimulate the cilia to produce an enzyme (MMP-1), which starts to break down excessive collagen over a matter of hours. These findings have implications when considering the speed and the amount of pressure to use in clinical practice. For example, Schleip found that when he paid more attention to the fluid dynamics (rather than, for example, solely focusing on breaking up the fibrous tissue) and worked slowly and gently, he gained better results. According to Schleip, practitioners should now not only be thinking about stimulating the mechanoreceptors or golgi tendon organ, but also be aware of how the fluid moves.

**Science gives clinicians an objective tool for evaluating the stiffness of tissue**

Most palpation is subjective, and therapists often cannot remember how ‘stiff’ the tissue was before and after treatment. Schleip suggested that a more effective way of measuring tissue stiffness is to use a myometer (the MyotonPRO). He argued that the quantitative digital measurement provided by the myometer is reliable and useful for assessing the biomechanical properties of myofascial tissues. These tools create a constant pre-load of the soft tissue via a movable indentation probe, which is then rapidly released. The tissue response (damping oscillation) is subsequently measured. This kind of tool can provide a more objective way to measure the effectiveness of a treatment.

**Innervations of the lumbar fascia provide clinicians with a clue to the treatment of lower back pain**

New research into the innervation of fascia has challenged the assumption that to profoundly alter the fascia and combat pain, you must work deeply. A recent study by Tesarz et al. (2011), published in Neuroscience quantitatively evaluated the density of sensory nerves in the different layers of the fascia.

![Figure 2. The distribution of CGRP and Substance P (SP)-immunoreactive nerve fibers in the thoracolumbar fascia (redrawn from Tesarz et al., 2011).](image)

(a) Mean nerve fibre length of CGRP and SP. Almost all fibres were found in the outer layer of the fascia and the subcutaneous tissue. The middle layer was free of SP-positive fibres.

(b) Distribution of CGRP and SP-containing receptive free nerve endings expressed as a percent of the total number of CGRP-containing or SP-containing fibers in each fascia layer. SP-containing free nerve endings were restricted to the outer layer of the thoracolumbar fascia and the subcutaneous connective tissue while CGRP-containing free nerve endings were also found in the inner layer of the thoracolumbar fascia.
Both the subcutaneous tissue and the outer layer showed particularly dense innervations featuring sensory fibres - SP-positive free nerve endings - which are assumed to be nociceptive.

In contrast, the dense layer of the lumbar fascia has no nociceptive nerve endings. Because of its dense sensory innervation, including nociceptive fibres, the TLF may play an important role in lower back pain. Because of its dense sensory innervation, including nociceptive fibres, the TLF may play an important role in lower back pain.

The findings of this study suggest that most myofascial pain may arise in the superficial layer, indicating that it may be more effective to work superficially, to stimulate proprioceptive and nociceptive nerve endings.

**The influence of sympathetic activation on fascial tonicity**

Vladimir Janda, a key figure in the 20th Century rehabilitation movement, was one of the first physicians to combine therapy and medicine in a ‘hands-on’ approach. Janda observed a close relationship between the autonomic nervous system (ANS) and fascial tonicity, implying that sympathetic activation may lead to an increased cellular contraction within fascial tissues. This observation was confirmed by recent findings suggesting that sympathetic activation induces an increased TGF-β1 expression - a cytokine that is known as the most potent stimulator of myofibroblast contraction.

Figure 3 illustrates a possible two-way interaction between ANS activation and fascial tonicity. In addition to indicating the influence of the ANS on cellular contractility in fascia, this diagram also emphasises the potential influence of therapeutic fascial stimulation on ANS tuning.

Stimulation of non-nociceptive mechanosensory free nerve endings can influence ANS tuning. In addition, stimulation of Ruffini corpuscles - which are reportedly particularly sensitive to slow shear application - tends to inhibit sympathetic activation.

**The rhythmic oscillations of fascial tissues**

A study by Follonier et al. (2010) demonstrated that myofibroblasts tended to oscillate in synchronicity when they were placed in a close physical contact with each other (Fig. 4). When connective tissue cells were put together in a cell culture medium with a collagen grid, they showed periodic oscillations: in particular, they expressed rhythmic calcium oscillations, which were accompanied by contractions of the cells. The observed oscillations had a mean period length of 100 seconds.

Schleip posed an intriguing question: can the very slow rhythm observed in these cell cultures - with one cycle taking more than one-and-a-half minutes - be related to the so-called ‘long tide’ oscillations in biodynamic craniosacral therapy? (The so-called ‘breath of life’ has a reported period length of 100 seconds.) Schleip invited interested therapists and scientists to subject this supposition to rigorous testing.

**Clinicians leading the way**

In addition, Schleip also suggested an area of consideration where scientists could learn from therapists:

**Scientists need to embrace collaboration**

Schleip maintains: “If you want to understand connective tissue, it works well to adapt some of the properties of that connecting tissue within your own social structure.”

Competition in the scientific world is strong: researchers compete to publish first and, therefore, their findings or data are usually not shared and are kept secret. This attitude is exacerbated by competition for funding.
Schleip urged scientists to be more open about their work and to collaborate more broadly, without fearing that their ideas might be stolen. He invited scientists to “…incorporate some of the networking strengths (that are very familiar to the complementary therapist) in their collaboration efforts in order to understand the organ of networking.”

REFERENCES


This article is based on and adapted from an original contribution published in Terra Rosa E-magazine No. 12, June 2013 (http://www.scribd.com/doc/146701416/Terra-Rosa-E-magazine-No-12#fullscreen)

You can watch Robert Schleip’s presentation on YouTube: http://www.youtube.com/watch?v=millGLLmXlc

Robert Schleip (PhD MSc) is director of the Fascia Research Group at Ulm University in Germany. He has been a Rolfing and Feldenkrais teacher for more than 20 years. Frustrated with his own explanations for the supposed tissue changes in manual therapy, he entered the field of connective tissue research in 2004 and has been fascinated with this new field of exploration ever since. His laboratory research finding on active fascial contractility was honored with the Vladimir Janda Award for Musculoskeletal Medicine. He was also one of the driving forces behind the first Fascia Research Congress (Harvard Medical School, Boston 2007) and the subsequent international fascia congresses. He is author of numerous books and other publications and still maintains a part-time private practice as a Rolfing and Feldenkrais practitioner.

Figure 4. Myofibroblasts expressed rhythmic calcium oscillations. The graph on the left showed recording of fluorescence activity of five individual cells, which were previously stained with Flura-2. The analysis revealed a common peak around 99 ± 32 seconds of the cells, as well as a second maximum of 221 ± 21 s. Based on Follonier Castella et al. (2010)