In separate sessions, 12 healthy subjects received ultrasound-guided bolus injections of isotonic saline (0.9%) or hypertonic saline (5.8%) into the erector spinae muscle, the thoracolumbar fascia (posterior layer), and the overlying subcutis. Subjects were asked to rate pain intensity, duration, quality, and spatial extent. Pressure pain thresholds were determined pre and post injection.

Injections of hypertonic saline into the fascia resulted in:
- significantly larger area under the curve of pain intensity over time than injections into subcutis (P<0.01) or muscle (P<0.001),
- pain radiation evoked by fascia injection exceeded those of the muscle (P<0.01) and the subcutis significantly (P<0.05),
- Pressure hyperalgesia was only induced by injection of hypertonic saline into muscle, but not fascia or subcutis.


Sensory findings after stimulation of the thoracolumbar fascia with hypertonic saline suggest its contribution to low back pain.
Patel and Lieber (1997) and Huijing (1999) have shown that:

- 70% of the transmission of muscle tension is directed (in series) through tendons
- 30% of muscle force is transmitted through the connective structures in parallel

Origin of muscular fibers from the deep fascia that presents a thickening in correspondence with these insertions.

Many muscles have myofascial expansions. When these muscles contract, they also stretch the deep fascia connected with the expansion.

Lacertus fibrosus (aponeurosis) continues from the biceps tendon and merges with the antebrachial fascia.

The relationships between the expansions of the pectoral girdle muscles (i.e. pectoralis major, latissimus dorsi and deltoid) and brachial fascia were analyzed.

Specific spatial organization (Stecco et al, CTO, 2008)
In the last years several researches have demonstrated the presence of many free and encapsulated nerve terminations, particularly Ruffini and Pacini corpuscles, inside the fasciae.

**WHAT IS FASCIA**

In reality, in the majority of cases, it is not clear whether it is fascial densification or fascial fibrosis that is involved. This lack of certainty causes not only confusion in terminology, but also implies that very different treatment modalities can be applied to fascia in an attempt to relieve pain.
The deep fasciae are a complex structure formed by at least two components:
- two or three layers of parallel collagen fibre bundles
- Loose connective tissue interposed

An alteration of the collagen tissue could give a fascial fibrosis,
An alteration of the loose connective tissue a fascial densification

Tissue viscoelasticity shapes the dynamic response of mechanoreceptors

FIBROUS COMPONENT OF THE DEEP FASCIA

The deep fasciae are formed by collagen fibres type I and type III, disposed in many directions.

APONEUROTIC FASCIAE:
from an irregular fibrous tissue to a multilayer organization

Layer I
Layer II
Lateral region of the tight
The fascia is not an elastic tissue, the elastic fibres are less than 1%. As a fishing-net, the fascia could adapt to muscular volume variations and to stretches, but over a cut-off the fascia becomes tensioned and consequently is able to transmit the forces at a distance.
The multilayer structure of the deep fasciae of the limbs

The presence of loose connective tissue interposed between adjacent layers permits local sliding, and so from a mechanical point of view the single layers could be considered independently.

The loose connective tissue is composed by:
- Water
- Ions
- Glycosaminoglycans (with a prevalence of hyaluronan)

Hyaluronan is secreted by specific cells inside the fascia, which are called fasciacytes.

Hyaluronan is a lubricant that allows normal gliding of joint and connective tissue.

Hyaluronan occurs both as individual molecules, and as macromolecular complexes that contribute to the structural and mechanical properties of fascia.

Fascial damage (i.e. surgery or trauma) always causes an inflammatory reaction that promotes the healing process. Three sequential, yet overlapping, phases of the reparative healing process occur:
- Inflammation
- Proliferation (fibroblasts grow and form a new, provisional ECM by excreting collagen type III and then type I collagen and fibronectin. In this phase, the collagen forms an irregular connective tissue that has the main function of closing the wound gap)
- Remodeling for the correct healing of the deep fascia it is fundamental that collagen fibres remodel and realign according the local tensile stress. Only now the connective tissue can transmit forces at a distance.)
Remodeling can last for years, depending on the size and nature of the wound. In actuality, this process is fragile and susceptible to interruption or failure. In particular, it seems that a fundamental role is played by the mechanical stress acting on the injury site, that guides the neuroinflammatory response. If the tissue in which tensile state can be observed was previously in an unbalanced condition or is immobilized, the remodeling process does not lead to physiological spatial reconstitution, but instead causes random deposition of collagen fibers.

Male, 65 ys, diabetic, amputation after 10 months of immobility following trauma.

For example, in the leg, a horizontal scar causes a tensile state three times greater than a vertical scar.

CAUSES OF ALTERATIONS IN THE FIBROUS COMPONENT: IMMOBILIZATION

Slimani et al (2012) demonstrated that immobilization causes pronounced muscle connective tissue thickening. During early recovery there are sustained increased expression of markers of CT remodeling and increased nuclear apoptosis.

CAUSES OF ALTERATIONS IN THE FIBROUS COMPONENT: DIABETES

• Duffin et al (2002) demonstrated that patients with type I diabetes have a plantar fascia significantly thicker compared to normal controls.
• Li et al (2013) demonstrated that diabetes alter the physical properties of collagen structures and tissue behavior:
  • reduce tissue stress relaxation (p<0.01)
  • Reduce drastically fiber-sliding with a compensatory increase in fiber-stretch.

All of these changes were demonstrated for tendons, but it is probable that this also applies to fasciae, causing loss of fascial viscoelasticity. This has potentially important implications for tissue remodeling and mechanically regulated cell signaling.
CAUSES OF ALTERATIONS IN THE FIBROUS COMPONENT: AGING

• Trindade et al (2012) demonstrate that the human deep temporal fascia is stiffer in older than in younger persons. Thus, increasing age creates stiffer, stronger and more stable connective tissues, although much less flexible.

• Wojtysiak (2013) demonstrated that in newborn pigs the perimuscular collagen fibrils of the longissimus lumborum muscle have a wavy disposition and form a loose network. Only with increasing age do the arrangement of collagen fibrils becomes denser and more regular. These factors can influence the shear force value of connective tissue and the underlying muscles.

CAUSES OF ALTERATIONS IN THE FIBROUS COMPONENT: OVERUSE

- The strain patterns in fasciae may not be uniform, so it is probably that overuse and underuse sites inside fasciae exist.
- Connective tissues exhibit adaptive responses to conditions of increased loading and disuse.
- If the adaptive response is inadequate, the fasciae hoard local alterations that change the distribution of the lines of force inside fasciae.

IN THE FIBROUS COMPONENT: OVERUSE

CAUSES OF ALTERATIONS

IN THE LOOSE CONNECTIVE TISSUE: DENSIFICATION

The loose connective tissue assures the autonomy of adjacent fibrous layers. Only if the loose connective tissue has low viscosity the fibrous layers can:
- transmit forces along different directions without interfering with each other
- adapt to volume variations of the underlying muscles during contraction.

The densification of the loose connective tissue, represented with a red flash, alters the gliding between the two fibrous layers. The transmission of the forces can be altered in a way that is not easily defined. The tissue around the densification point can be subjected to intense mechanical stress.

Stecco A et al; RMI study and clinical correlations of ankle retinacula damage and outcomes of ankle sprain. Surg Radiol Anat. 2011 Dec
CAUSES OF DENSIFICATION:
STRENUOUS EXERCISES

Piehl-Aulin et al (1985) demonstrate a transient accumulation of hyaluronan following exercise. Similarly to a synovial joint, increased production of HA is the initial attempt to increase the gliding efficiency between two surfaces.

Tadmor et al (2002) show that when hyaluronan is organized into layers, viscosity increases considerably with increasing distance between the two surfaces.

The increased viscosity of the loose connective tissue inside the fascia may cause decreased gliding between the layers of collagen fibers of the deep fasciae. This may be perceived by patients as an increase in fascial stiffness.
The three-dimensional superstructure of HA chains breaks down progressively when temperature is increased, with a consequent decrease in viscosity (Gabor et al, 2003). This may explain the effects of many physical therapies that increase temperature (laser, etc.) and with warming up in general.

CAUSES OF DENSIFICATION:
LOW TEMPERATURE

CAUSES OF DENSIFICATION:
LOW pH

The hyaluronan shows stable condition in alkaline solution, but in acid solution its viscosity increases dramatically (Gatei 2005).

After strenuous exercises the muscles pH can reach a value of 6.60 with an increase of approximately 20% in HA viscosity.

This may be perceived by patients as an increase in fascial stiffness.
Pajetadze (1998) demonstrated that, when superfused with lactic acid, the contraction of the myofibroblasts of the superficial fascia of rats was significantly increased. Trabold et al. (2003) demonstrated that lactic acid stimulates collagen synthesis.

The pH induces also an increase of the fibrosis and of the fascial tension. Trabold et al. (2003) demonstrated that lactate stimulates collagen synthesis.

Hyaluronan is a thixotropic substance. Dintenfass (1966) demonstrates that synovial fluid has thixotropic and elastic (instantaneous dilating) properties. He finds that its viscosity decreases with an increase in shear rate, but it is pressure-resistant under sudden impacts.

This property can be assumed also for the key element of the fascial loose connective tissue and explains why immobility reduces fascial gliding and consequently, range of motion. Besides, the movements and massages can reduce its viscosity.

The loose connective tissue is an important reservoir of water and salts for surrounding tissues. But it also has the capacity to accumulate various of waste products. The biomechanical properties of loose connective tissue may be altered depending upon water content and waste products accumulation.
FIBROSIS OR DENSIFICATION?

Densification
- Alteration of loose connective tissue
- Alter the gliding

Fibrosis
- Damage of the fibrous component
- Affects the capacity of loading transmission

The two alterations cause different problems for the fascial function and need different treatments.

DENSIFICATION

This water-mediated supramolecular assembly was shown to break down progressively when the temperature was increased to over \( \sim 40 \) °C.

Matteini P. 2009

Luomala T et al. 2014

Stecco A et al. 2013
It is more difficult to modify because: only a local inflammatory process can destroy the pathologic collagen fibers and permit deposition of new collagen fibers.

FIBROSIS

Inf lammat ion

Noble P.W, 2002

"Under conditions of stress hyaluronan becomes depolymerized and lower molecular mass polymers are generated"  Stern R. 2006

It is only with a clear understanding of fascial anatomy and structure that it will be possible to make accurate differential diagnoses;

Only then it will be possible to prescribe correct treatments.

Thanks