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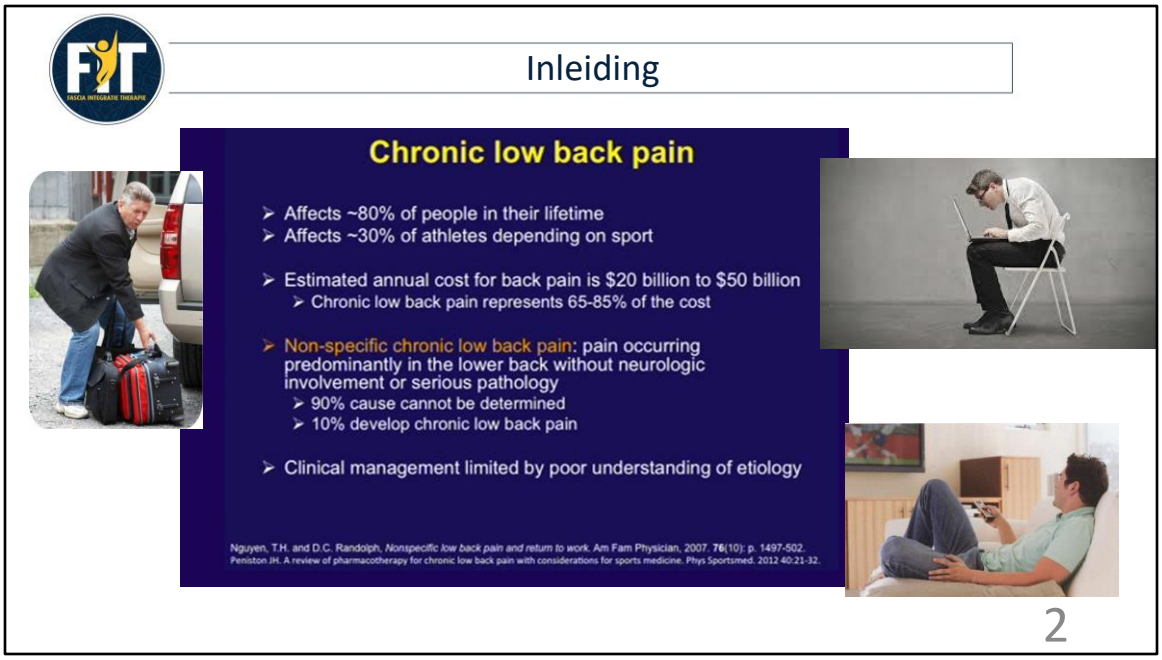
L.W.K. – anatomie en pathologie

Opleiding fasciatherapie F.I.T.
Module onderste extremiteit

Short description of content: low back pain is often described as non-specific. Recent research points the way to more specific knowledge about the pathology of low back pain. Knowledge of these new insights points the way to a more specific approach to low back pain.
Learning objectives: The student is aware of these new insights and can use this in the clinical reasoning process for low back pain (after performing the myofascial examination)



The lifetime prevalence of non-specific (common) low back pain is estimated at 60–70% in industrialized countries (one-year prevalence 15–45%, adult incidence 5% per year).

- WHO 2013 Béatrice Duthey

<< As a clinician, it is easy to gain the impression that structural changes in the LPH complex are always accompanied by symptoms; however, it is known that the incidence of spondylosis and osteoarthrosis is just as great in patients with symptoms as in patients without symptoms (Bogduk 1997, Lawrence et al 1966, Magora & Schwartz 1976, Torgerson & Dotter 1976). In addition, it is common for the joints or regions adjacent to the painless one with the structural change to become symptomatic. Regardless of which joint or region is producing pain, it is known that pain can have an impact on the function of the LPH complex. Lee, 2011>>

Nonspecific diagnosis often results in non-specific treatment

<<Why is it that myofascial pain is frequently not recognized?
One reason is certainly the fact that pain very often radiates from the muscle as referred pain. The site at which pain is felt and the site of origin of the pain are not

identical, and this fact makes it difficult to determine the connection between the problem in the muscle (mTrP) and the clinical presentation of the pain.
On the other hand, myofascial pain is a clinical diagnosis — the active mTrPs can be reliably identified only by means of palpation using clearly defined diagnostic criteria. There are no abnormal findings on radiologic or chemical lab tests. Unfortunately, myofascial pain is still misdiagnosed far too often, with the cause of pain being wrongly attributed to an abnormal finding on an imaging study. The result is that patients do not receive proper treatment and may even receive unnecessary surgery, since what is being treated is not the cause of the pain. Gautschi 2019>>

Red flags
Red flags are signs or signals that are alone or in combination, indicate a possible (serious), specific cause of the low back pain, which requires additional diagnostics is needed.
There is consensus about the following red flags:
• Onset of low back pain after the age of 50, continuous pain regardless of posture or movement, nocturnal pain, general malaise, malignancy in the history, unexplained weight loss, increased blood sedimentation rate (BSE) → malignancy?
• Recent fracture (<2 years ago), previous vertebral fracture, age over 60 years, low body weight (<60 kg / BMI <20 kg / m2), older with hip fracture, long-lasting corticosteroid use, local knock, pressure and shaft pressure pain of the spine, noticeable reduction in height, enhanced thoracic kyphosis → osteoporotic vertebral fracture?
• Onset of low back pain before age 20, male, iridocyclitis, unexplained peripheral arthritis, or inflammatoire history of bowel disease, especially nocturnal pain, morning stiffness> 1 hour, less pain when lying down / movement / exercise, good response to NSAIDs, increased BSE Ankylosing spondylitis?
• Severe low back pain following trauma → vertebral fracture?
• Onset of low back pain before age 20, palpable steps in the course of the spinous processes at the height of L4-L5 → severe spondylolisthesis? KNGF guideline for the low back >>

Algemeen beleid (KNGF richtlijn)

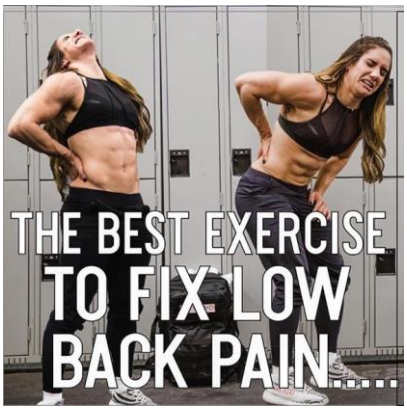
Beleid bij aspecifieke lage rugpijn met een afwijkend beloop zonder dominante aanwezigheid van psychosociale herstelbelemmerende factoren

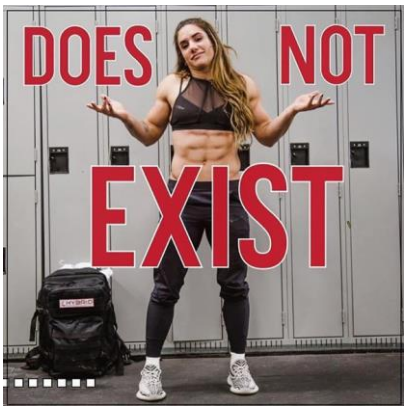
- Vermijd passiviteit, stimuleer activiteit (beweging, activiteiten, werken)
- Stel gerust (toename pijn ⇏ beschadiging van structuren in de rug)
- Oefenprogramma (op dat aansluit bij de behoefte van de patiënt en de eigen expertise en ervaring als therapeut).
- Stoornissen in gewrichtsfuncties: artrogene mobilisatie of manipulatie* en/of - kortdurende massage of warmtetherapie ter vermindering van de pijn.
- Check aanpak werkverzuim

3

<< Policy for non-specific low back pain with abnormal course without dominant presence of psychosocial factors that hinder recovery (profile 2)
• Avoid advice that leads the patient to passivity and promote a physically active lifestyle.
• Indicate that increase in pain is not associated with damage to structures in the back.
• Stimulate (dosed) movement, build up activities and continuing to work or resuming work (possibly with temporary adjustment of that activities).
• Draw up an exercise program that meets your needs of the patient and their own expertise and experience as therapist.
• For joint function disorders, consider:
- arthrogenic mobilization or manipulation * and / or - short-term massage or heat therapy reduction of pain.
• In case of absence from work for longer than 4 weeks, ask for appointments that have been made with the company doctor and discuss if necessary, the policy with this doctor and / or the occupational physiotherapist.
KNGF guideline for the low back >>

Oefentherapie bij lage rugpijn





Kunnen we meer specifiek zijn?

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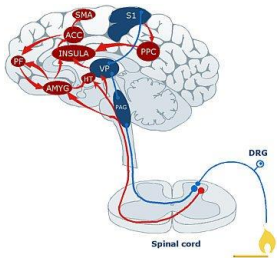
Nociceptieve pijn

Pijn = Nociceptie ✖ Gevoeligheid zenuwstelsel

(issues in tissues)



CSI: central sensitivity inventory

- Angst
- Alertheid
- Stress
- Vermoeidheid
- Cognities

etc.

Rx

5

All nociceptive pain is constructed of two elements: nociception and the sensitivity of the nervous system
It is paramount that we assess both in <u>all</u> our patients

<< Acute pain can be largely driven by central mechanisms. Persistent pain can also be largely driven by peripheral mechanisms. That is, persistent or chronic pain states may have central components, but these are not necessarily the dominant mechanism for every patient simply because the pain experience has persisted for a long period of time. While evidence supports that 'the relationship between pain and the state of the tissues becomes less predictable as pain persists (Moseley 2007), we need to remember that the pain experience is uniquely individual. Regardless of whether the pain is a newly occurring event or a persistent experience, it is a multidimensional experience, and thus any person presenting with pain should be evaluated with a framework in mind that allows for the consideration If all these factors. As Butler (2000, p. 53) notes: Overlap of mechanisms is the key feature because the boundaries are often fuzzy. There will be differing contributions of mechanisms to the injury state over time, person and injury.
It is reasonable to suggest that even if peripheral mechanisms only contribute 20% to the complete picture, addressing that 20% in addition to the other approaches will

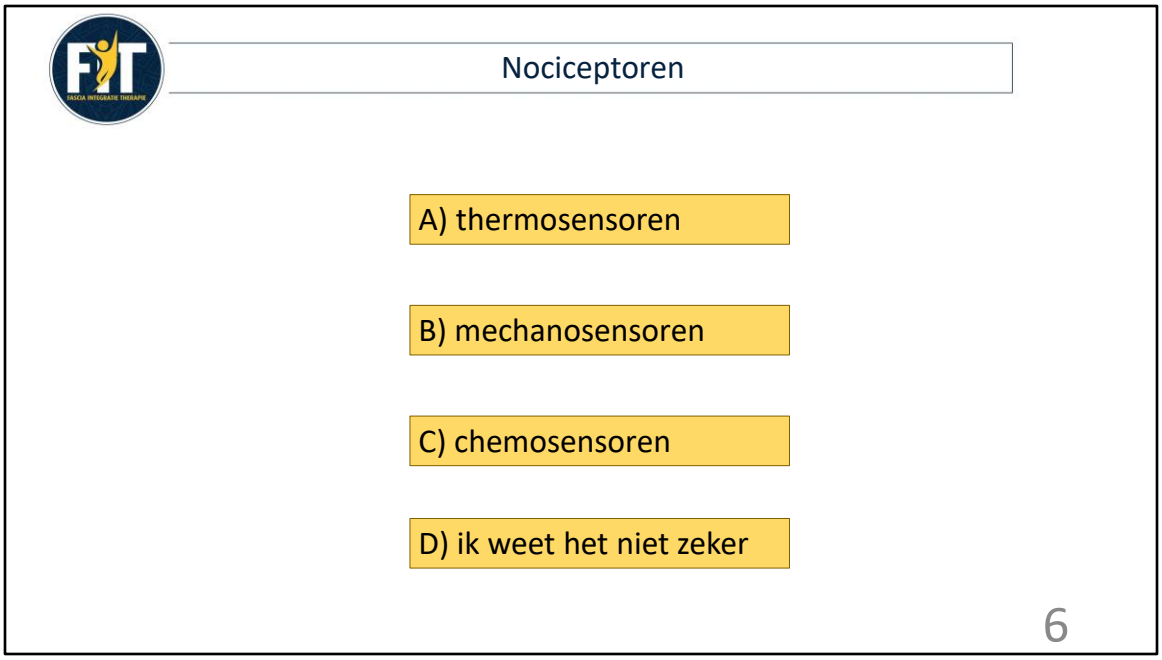
provide the greatest chance for the best outcome. Furthermore, it is likely that by addressing the physical impairments, psychosocial variables will also be impacted, further advancing the goals of treating drivers of central sensitization. It is also crucial to recognize that our patients change as a result of their changing life circumstances and our interactions with them (both physical and personal). Thus, during the course of treatment continual re-evaluation is necessary to adapt the treatment programme accordingly. Sticking to a rigid plan based on an initial placement into a subgroup may result in the provision of sub-optimal care.
In the brain, an individual's thoughts and feelings (cognitions + emotions = perception) are integrated and can influence the output mechanisms, which include:

- Somatic or motor (altered posture, altered motor control);
- Autonomia (increased sympathetic response for 'fight or flight');
- Neuroendocrine (increased stress, heightened emotions, hormonal changes);
- Neuroimmune.

Lee 2012>>

<< George's Gem Number 1 'To be able to think of disease as an entity, separate from man and caused by an identifiable substance, apparently has great appeal to the human mind. Perhaps the persistence of such views in medicine reflects the operation of psychological processes to protect the physician from the emotional implications of the material with which he deals.' [12]
We share this view but we have a very important caveat: that disturbed state or function of particular tissues, for example because of injury, tissue overload, inflammation, aberrant activation of primary nociceptors, injury to nerves, altered response profiles of immunocompetent tissues all constitute valid and potentially major contributors to a pain state. Moseley 2017>>

<<The most unfavorable prognosis occurs in patients who have a firmly fixed idea (*idée fixe*) that a peripheral tissue injury is the cause of their widespread pain symptoms. This misleading idea can arise from inappropriate medical information (in the context of chronification or in line with a nocebo effect) or as the result of membership in self-help groups, but can also be a symptom of a hypochondriacal disorder. Gautschi 2019>>



<<Nociception

Nociceptors are sensory receptors that innervate peripheral tissues, and they consist of free nerve endings. A nociceptor responds to noxious stimuli, defined as damaging or potentially damaging stimuli. Once activated, they convey information to the central nervous system through thinly myelinated A6 and unmyelinated C-fibres. Watson 2020>>

Nociceptors are neural sensors with the task to protect the cells. They signals possible dangers in the form of heat, mechanical stress and changes in the chemical balance of the extracellular fluid. So there are thermosensors, mechanosensors and chemosensors, and many of them are in the form of polymodal sensors.

<<Nociceptors
A nociceptor is a sensory receptor that is capable of transducing and encoding actually or potentially tissue-damaging stimuli (noxious stimuli). Nociceptors convert mechanical, thermal, and chemical energy into electrical signals and carry this information to the CNS. nociceptors are unencapsulated receptors, termed free nerve

endings, that respond to noxious stimuli; they include Aδ (Group III; thinly myelinated axons) and C fibers (Group IV; unmyelinated axons). Sluka 2016>>

<<Most or all afferent nociceptive neurons innervating fascial tissues are hypothesized to be polymodal. responding to mechanical stimuli, heat stimuli, and chemical stimuli (e,g., bradykinin, capsaicin). This hypothesis is based on several groups of experimental studies:
The afferent innervation of the knee joint capsule (cat, rat: Schaible, 2013: Schaible & Grubb, 1993) and of skeletal muscle (mostly gastrocnemius-soleus muscle in cat and rat: Mense, 199 3. 2013). In fact, many small-diameter muscle afferents probably innervate fascial tissues of the skeletal muscle. Unmyelinated afferent fibers innervating the paraspinal tissues in rats (Bove & Light, 1995). Afferent C- and Aδ-fibers innervating the retroperitoneal space including blood vessels, nerves, lymph nodes, vertebral column (periosteum, ligaments, intervertebral discs) and fat tissue in cats (Bahns et al, 1986). Jänig 2017>>

<<Nociceptors can be defined as sensory receptors that are activated by noxious stimuli that damage or threaten the body's integrity. Nociceptors belong to the slowly conducting afferent A delta and C fibres. They are classified according to their responses to mechanical, thermal, and chemical stimuli. SKIN NOCICEPTORS: In the skin, high-threshold mechano-nociceptors (HTMs) and mechano-heat nociceptors of A and C fibres (AMHs and CMHs) are frequently found. CMHs are usually called polymodal C fibres (CPMs) if they also show chemosensitive properties. Sensations of sharp pain are evoked by intraneural micro stimulation of nociceptive A delta fibres, whereas stimulation of C fibres causes dull pain sensations. NOCICEPTORS OF DEEP TISSUES AND VISCERA: Slowly conducting afferents of deep tissues (muscles, joints) are primarily classified according to their mechanosensitivity. High-threshold afferents in somatic and visceral tissues are specifically activated by noxious mechanical stimuli. Many visceral afferents, however, are already activated by peristaltic contractions encoding the stimulus intensity over a wide range. High proportions of somatic and visceral nociceptors can be excited or sensitised by various irritants and inflammatory mediators such as capsaicin, bradykinin, prostaglandins, leukotrienes, serotonin, histamine, and free radicals. As a special class of nociceptors, mechano-insensitive or "silent" afferents have been found in nearly all tissues. Silent afferents become mechanosensitive only after long noxious stimulation, e.g., during an inflammation. FREE NERVE ENDINGS: "Free nerve endings", which are regarded as the morphological correlatives of nociceptors, usually consist of bundles of unmyelinated fibres. With electron microscopy varicose segments of the sensory axon are visible that are characterised by free areas of axolemma, accumulations of mitochondria and vesicles, and a modified axoplasm. These presumptive receptive sites are periodically arranged along the whole course of the sensory endings at a length of up to several hundred microns. Additionally, the

fine sensory endings are branched, forming tree-like structures, and frequently innervate different types of tissues. Studies correlating structure and function of articular afferents provide evidence for a close relationship between topographical and functional properties of sensory endings. High-threshold afferents (nociceptors) seem to terminate in structures of dense connective tissue. Proportions of nociceptors contain neuropeptides such as substance P and calcitonin gene-related peptide, which are released from the activated nociceptive terminals and cause neurogenic inflammation, including precapillary vasodilatation and postcapillary plasma extravasation.
Messlinger K [What is a nociceptor?]. [Article in German]. Anaesthesist 1997 Feb;46(2):142-53.>>

<< Free Nerve Endings. The afferent fibers of the free nerve endings are, for the most part, not myelinated (type IV fibers, diameter 0.5–1.0 µm). Beside these, thin, myelinated fibers also occur as afferents (type III fibers, diameter 1–4 µm). Both types, myelinated and non-myelinated, end in free nerve endings, which are myelinated. Free nerve endings are by far the most numerous receptor found in humans (Mense 2007). They form a giant, hidden network. Because they end in the interstitial space, they can also be called interstitial receptors (Schleip 2003). Using the electron microscope, different forms of free nerve endings can be differentiated. Some endings show a close spacial relationship to collagen fiber bundles, which could indicate a mechanosensitivity function (Mense 2007).

In terms of function, differentiation can be made between mechanoreceptors, nociceptors, thermoreceptors, and chemoreceptors, and many are considered to be multimodal. The majority of free nerve endings have a mechanosensitive function, of which approximately 50% have a high threshold and only react to strong mechanical impact. The other half with a lower threshold respond even to minimal pressure effects (Schleip 2003) and weak movement stimuli (Mense 2007). A study shows that the interstitial receptors in the vicinity of the jaw muscles react to slight change in mandibular position and to very minimal fascial shift, so that the free nerve endings are assigned, not only a nociceptive function, but a proprioceptive function as well (Sakada 1974). Gautschi 2019>>

<< This general description of the biology of nociceptive primary afferent neurons also applies to those innervating fascial tissues.

Neurophysiology of afferent neurons innervating fascial tissues

Most or all afferent nociceptive neurons innervating fascial tissues are hypothesized to be polymodal. responding to mechanical stimuli, heat stimuli, and chemical stimuli (e,g., bradykinin, capsaicin). This hypothesis is based on several groups of experimental studies:

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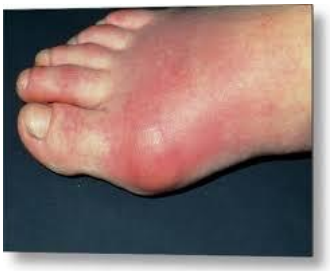
Afferent C-fibers innervating the pia mater of sacral ventral roots in cats (jiinig & Koltzenburg, 1991). Figure 28.3 demonstrates the discharge of an afferent C-fiber innervating the perineurium of the ventral root to light stretch of the ventral root S2.
Afferent fibers innervating fascial tissues are predominantly excited by mechanical stimuli. Furthermore, many of them are probably also excited by heat stimuli. Finally, all of them are probably excited by chemical stimuli as they occur during inflammation. Whether fascial tissues are innervated by normally mechanoinsensitive (mechanically extremely high-threshold) afferent fibers, we do not know (see Michaelis et al.. 1996: Schaible & Grubb. 1993: Schmidt et al., 1995).

Jänig in Liem 2017>>

Thermosensoren?



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Thermosensors are mainly located in the skin. Their threshold is high, around 42,5°C. So they will be activated when someone is (nearly) burning himself. It is very unlikely that they play an important role in chronic pain where people don't experience these high temperatures.

Threshold for thermosensors is too high. Only cause pain with (nearly) burning.
Thermoreceptors – Respond to temperature change for duration of stimulus.
So it is not likely they play an important role in (chronic) pain conditions.

Mechanosensoren?

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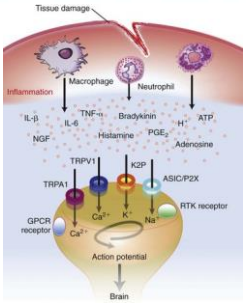
Mechansensors are mailly located in the fascia, they have a connection with collagen structures to be able to measure the mechanical stresses in the tissues. Many patients feel more pain with meachanical loading so it is tempting to think that mechanosensors are the primary source of nociception in pain. But when you look closer it is clear that this cannot be the case. When a person lifts a heavy weight, say 100 kg, all the mechanosensors in the whole body are activated, still the person will feel no pain. Under normal circumstances loading of mechanosensors won't cause pain. Only when the tissues tear, or are about to tear, pain will result. This amount of loading does not happen during activities of daily living. So also mechanosensors are not the primary nociceptors in chronic pain.

Chemosensoren?



Ontsteking

Ischaemie

pH ↓

Sensitisatie nocisensoren!

"Practically all lesions of the fascia are accompanied by a sterile inflammation."
Mense 2019

9

This leaves us with the chemosensors. Chemosensors don't have a high threshold, they constantly monitor the chemical environment of the cells, the interstitial fluid or 'groundsubstance'. The groundsubstance is for the cells what the sea is for fish, it is vital for function and survival. Especially changes in pH are critical for cell function. It is only logical that chemosensors are the primary nociceptors of the body as survival of the cells is more important than mechanical damage. When chemosensors are activated this will result in sensitisation of the other nociceptors. So now mechanical loading can result in pain. So mechanical nociception is secondary to chemical nociception. If mechanical loading causes pain there must be a change of the chemical environment of the cells

<< It is well established that the immune system, and factors released from immune cells (e.g. cytokines and chemokines), play a critical role in the generation of both acute and chronic pain (Fig. 3.5). Watson 2020

But how can we explain that mechanical loading does cause pain in pain patients? Mechanosensors can also be sensitised by central sensitisation, then you will expect

widespread pain. It is interesting to see that even in patients with fibromyalgia, generally seen as the most clear nociplastic condition, the pain is nog equally spread over the whole body, but certain areas or movements are more painful then others. In chronic pain there will often be a combination of more localised chemical sensitisation and central sensitisation.

The immune system is not only activated during overt injury but also during normal loading of tissues and especially during (ongoing) overload.

Substances released by immune cells include serotonin, bradykinin, prostaglandins, cytokines and chemokines. During inflammation, cytokines are released by infiltrating macrophages at the site of injury, and these pro-inflammatory cytokines (e.g. IL-l|3,IL-6, TNFa) sensitize nociceptors and produce mechanical and thermal hyperalgesia in animals and humans (Cunha et al 1992; Feldmann et al 1995 Ferreira et al 1988; Kaneko et al 2000; Kaneyama et al 2002 McNearney et al 2004; Ozaktay et al 2002; Watkins et al 1994 Watkins et al 1995). As stated above, these inflammatory mediators can sensitize the ASIC and TRPVl channels to make them more responsive to acidic, mechanical and thermal stimuli. Together these data can increase input to the central nervous system to result ultimately in the perception of pain.

> Nerve growth factor. NGF is a neurotrophic factor that is produced by muscle and during tissue injury (Amano et al 1991; Hayashi et al 2011; Wu et al 2009) and activates and sensitizes nociceptors through its TrkA (high affinity) receptors (Hoheisel et al 2007; Murase et al 2010; Snider & McMahon 1998). Injection of NGF in humans and animals produces long-lasting hyperalgesia and results in an upregulation of proteins involved in pain transmission, including substance P, TRPVl and Navl.8, and alters N-methyl-D-aspartate (NMDA) glutamate receptor sensitivity (Basbaum et al 2009; Ji et al 2002; Svensson et al 2003a; Wong et al 2014) which enhances nociceptor excitability.

Watson 2020>>

<< nonneural activators and inflammatory mediators
Many substances are released from inflammatory cells that can directly activate

and/or sensitize primary afferent fibers. It is now well established that the immune system, and factors released from immune cells (e.g., cytokines, chemokines), plays a critical role in the generation of both acute and chronic pain.

Evidence for mast cells, neutrophils, macrophages, dendritic cells and T cells shows their involvement in a variety of pain conditions (Dawes 2013), and for macrophages in the analgesia by nonpharmacological treatments like acupuncture and exercise (da Silva 2015, Leung 2015). Substances released by immune cells include serotonin, bradykinin, prostaglandins, cytokines, and chemokines. Serotonin, released from platelets, activates muscle nociceptors and causes pain in humans (Fock 1976, Richardson 1986). Bradykinin, which is released from plasma after tissue injury and is present in inflammatory exudates, sensitizes nociceptors and produces pain and heat hyperalgesia in humans (Cesare1996, Dirosa 1971, Kirchhoff1990, Koltzenburg 1992, Manning 1991, Neugebauer 1989, Petho 2001). Prostaglandins are metabolites of the arachidonic acid cascade and are produced in response to tissue injury.
Prostaglandins directly excite and sensitize nociceptors through receptors located on primary afferent fibers (Chen 1999, Schaible 1988). The nonsteroidal anti-inflammatories (NSAIDs) produce their effects by reducing prostaglandin production through inhibition of the enzyme cyclooxygenase, which is involved in the breakdown of arachidonic acid.
During inflammation, cytokines are released by infiltrating macrophages at the site of injury and synoviocytes in joints. The proinflammatory cytokines, including interleukins (IL-ip, IL-6) and tumor necrosis factor (TNFa), are increased in the synovial fluid from patients with arthritis; they sensitize primary afferent nociceptors and produce mechanical and thermal hyperalgesia (Cunha 1992, Feldman 1995, Ferreira 1988, Kaneko 2000, Kaneyama 2002, McNearney 2004, Ozaktay 2002, Watkins 1995, 1994). Although the actions of each of these inflammatory mediators are described individually, many mediators act together to enhance the inflammation and/or hyperalgesia, producing a potentiated response.

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Phase III trials is being carefully monitored because there are reports of enhanced joint destruction in the active arm of the study compared with the placebo arm. Adenosine triphosphate (ATP) is found and released from muscle fibers during exercise, keratinocytes, and synoviocytes (Bangsbo 2001, Jankowski 2013, Loredo1998, Stucky 2004). When injected into human subjects, it causes pain, and when injected into animals, it causes hyperalgesia. ATP binds purinergic receptors (P2X), particularly P2X2 and P2X3, which are found on nociceptors, resulting in activation and sensitization (Burnstock 2013, Dessem 2010, Shinoda 2008). Combining ATP with lactate and decreased pH can produce a potentiated effect enhancing nociceptor activity, hyperalgesia, and pain in animals and humans (Birdsong 2010, Light 2008, Pollak 2014, Seo 2011). Sluka 2016>>

<<The peripheral nervous system and the immune system perform a series of similar functionalities such as recognizing, responding, and adapting to external or internal stimuli despite significant morphological differences. The peripheral nervous system actively communicates and coordinates with the immune system to function as a unified defense system. The peripheral nervous system is highly regulated by the immune system, especially under inflammatory conditions. On the other hand, the nervous system can modulate the immune system via neurotransmitters and chemokines released by the peripheral nerve endings, particularly from nociceptors. In both physiological and pathological conditions, peripheral nociceptive (including pruriceptive) neurons may express a variety of immune-related receptors, such as chemokine receptors and immunoglobulin (Fc) receptors that are usually found on immune cells. Certain ligands such as chemokines and immune complexes may induce abnormal neuronal hyperexcitability and even ectopic action potential discharges, therefore producing the sensation of pain and/or itch in immune-related diseases. The immune-sensing mechanisms of peripheral nociceptors may play an important role in the development of chronic pain and pruritus and may indicate novel therapeutic strategies for these pathological conditions.

Peripheral nociceptors, or primary nociceptive (and pruriceptive) neurons, are the first layer of nocisensitive sensors innervating almost all the tissues and organs, especially the epithelial of body surface (Dubin and Patapoutian 2010).

Peripheral nociceptors expressing a variety of immune-related receptors may directly and actively interact with the immune system in both physiological and pathological conditions and may play a critical role in pain and itch related to immune diseases.

The primary site of coupling between neurons and immune cells is the peripheral tissue where “naked” free terminals of peripheral nociceptive neurons innervate the epidermis of skin, cornea, or mucosa. These nerve endings express various membrane receptors that are accessible to chemical factors in the peripheral tissue.

On the sensory side, injury can affect the mechano-receptors in muscle, ligament, joint capsule and skin that provide input to the nervous system about the position of a body segment or its movement. This can either be due to direct injury of the mechanoreceptor or trauma or inflammation of the structure that contains the mechanoreceptor. Such changes may be the result of repeated loading of the structure!"] as opposed to frank trauma. Alternatively, pain is associated with changed processing or interpretation of sensory information. For instance, despite availability of information from the back muscles in people with back pain, this information is often ignored.t'^' In chronic pain there is reorganisation of the sensorimotor cortex and the extent of reorganisation is related to the duration of pain. Any of these changes in sensory function would mean that the nervous system has corrupted input or interpretation of sensory information. This has two major impacts on the control of movement. (Vincenzino, 2011).
Wang 2016>>

<< The host evolves redundant mechanisms to preserve physiological processing and homeostasis. These functions range from sensing internal and external threats, creating a memory of the insult and generating reflexes which aim to resolve inflammation. Impairment in such functioning leads to chronic inflammatory diseases. By interacting through a common language of ligands and receptors, the immune and sensory nervous systems work in concert to accomplish such protective functions. While this bidirectional communication helps to protect from danger, it can contribute to disease pathophysiology. Thus, the somatosensory nervous system is anatomically positioned within primary and secondary lymphoid tissues and mucosa to modulate immunity directly. Upstream of this interplay, neurons detect danger which prompts the release of neuropeptides initiating i) defensive reflexes (ranging from withdrawal response to coughing); and ii) chemotaxis, adhesion and local infiltration of immune cells. The resulting outcome of such neuro-immune interplay is still ill-defined, but consensual findings start to emerge and support neuropeptides as blockers of TH1-mediated immunity but also as drivers of TH2 immune responses. However, the modalities detected by nociceptors revealed broader than mechanical pressure and temperature sensing and include signals as various as cytokines and pathogens to immunoglobulins and even microRNAs. Along these lines, we aggregated various dorsal root ganglion sensory neurons expression profiling datasets supporting such wide-ranging sensing capabilities and to help identify novel danger detection modalities of these cells. Thus, revealing unexpected aspects of nociceptor neurons biology might prompt the identification of novel drivers of immunity, means to resolve inflammation and strategies to safeguard homeostasis. ?? >>

<<The nervous system as well as the immune system use common signaling molecules for intra- and inter-system communications. Specifically, both entities produce a similar array of peptide and non-peptide transmitters that act on a

common set of receptors present in the two systems. One important set of such signaling molecules are cytokines. The wide distribution of cytokine receptors throughout the body, including the immune and the nervous system allows direct communication between these two entities. In addition to cytokines the nervous system and immune system also communicate with each other using shared ligands such as neurotransmitters and neuroendocrine hormones, and their respective receptors. Some of the most important clinical interactions between these two systems are associated with the "sickness response" as well as pain and analgesia. This "sickness response" which has been frequently attributed to inflammatory cytokines, strongly resembles the core symptoms of fibromyalgia and other Central Sensitivity Syndromes (CSS). Therefore a large number of research studies have focused on the relationship between peripheral cytokines and CSS. However, a lack of consistent associations was observed between CSS symptoms and peripheral cytokines which seem to suggest that maybe cytokines abnormalities of the central nervous system contribute to the pathogenesis of these illnesses. Better knowledge of cytokine -nervous system interactions may ultimately benefit the development of interventions that improve CSS manifestations including the "sickness response" and chronic pain. Staud 2015>>

<<In animal experiments, muscle inflammation of 12-days duration leads to a marked increase in the innervation density of nerve endings containing substance P (Mense et al. 2001). Because many of the substance P–containing nerve endings are nociceptors, this means that there was an increase in the innervation density of nociception-sensitive structures. A noxious stimulus in the muscle activates in such a situation more nociceptive endings and thereby triggers increased pain. Gautschi 2019>>

Sleeping nociceptors (silent afferents) may compromise up to 25% of deep somatic tissues. These afferents aggressively respond to inflammatory chemicals, remaining active for prolonged periods of time after exposure and causing adverse plastic changes in the CNS.

<< He found that the proportion of second-order neurons with fascial input from the TLF significantly increased in animals with chronic myositis in the low back. These recent articles suggest not only that the muscle fascia could be a source of nociception in a physiological condition, but also that it could play crucial roles in hyperalgesic conditions (e.g., myofascial pain syndrome). Taguchi 2008>>

<< Sensitization of a neuron is characterized by increased spontaneous activity, a decrease in threshold of response to noxious stimuli, an increase in responsiveness to the same noxious stimuli, and/or an increase in receptive field size. Recording the activity of peripheral nerves before and after induction of acute inflammation,

Schaible and Schmidt (1985, 1988) show increased spontaneous activity and responsiveness to noxious and innocuous joint movement in primary afferent fibers of Groups II, III, and IV. Similar changes occur following inflammation of the muscle with carrageenan (Berberich 1988, Diehl 1988) or following ischemia of the muscle (Mense 1983). Following peripheral inflammation, silent nociceptors begin to respond to both innocuous and noxious stimuli, such as pressure and joint movement.

Ion Channels

Several ion channels, found on peripheral terminals of primary afferent fibers, may also be important in the response to noxious stimuli. Low pH is found in inflamed tissues, is released in response to fatiguing exercise, and produces pain in humans and animals (Frey- Law 2008, Hamamoto 1998, Reeh 1996, Sluka 2001). Acid-sensing ion channels (ASICs) are located in DRG neurons, activated by low pH, and are importantly involved in pain from muscle and joint (Gregory 2015, Ikeuchi 2008, Molliver 2005, Price 2001, Sluka 2003, 2007, 2013, 2009). ASICs in DRG increase after inflammation of muscle or joint, and blockade of ASICs reduces the hyperalgesia associated with inflammatory and noninflammatory pain (Sluka 2009), ASIC3 has been extensively studied and plays a significant role in musculoskeletal pain (for review, see (Sluka 2015)).

The vanilloid receptor-1, TrpVl, is activated by the exogenous ligand capsaicin, located in DRG, responds to low pH, and mediates hyperalgesia to heat stimuli (Caterina 2000, 1999). In humans, intradermal or intramuscular injection of capsaicin produces pain and unpleasantness (Witting 2000). Further, inflammatory mediators can activate and sensitize TRPVl through multiple second messengers, making it more responsive to peripheral stimuli such as heat or acid (Ringkamp 2013). Clinically, low-dose capsaicin creams (<1%) are effective for treatment of neuropathic and musculoskeletal pain conditions such as postherpatic neuralgia and osteoarthritis (Anand 2011). Higher concentration patches are also used, 8% capsaicin, for treatment of neuropathic pain (Anand 2011) .These treatments are thought to desensitize the nociceptors and produce a localized loss of nerve fiber terminals in the skin (Anand 2011).

Sluka 2016>>

<<A drop in pH is probably one of the main activators of peripheral nociceptors, as many painful disturbances of muscle are associated with low pH in muscle tissue. Nerve growth factor (NGF) also has a connection to muscle pain: NGF is synthesized in muscle and activates muscle nociceptors (e2). NGF synthesis is increased when a muscle is inflamed (e3). >>

<< Since most mechanoreceptive free nerve endings in normal joints are only stimulated by extreme joint movements, they are probably not normally significant

sources of position and movement sense. However, as with muscular free nerve endings, when there is inflammation, a large proportion of the free nerve endings are sensitised by the milieu of chemical substances produced during the inflammatory process (Grigg et al., 1986; He et al., 1988). In turn, this may result in abnormal joint position sense. Strimpakos 2011a>>

<< He found that the proportion of second-order neurons with fascial input from the TLF significantly increased in animals with chronic myositis in the low back. These recent articles suggest not only that the muscle fascia could be a source of nociception in a physiological condition, but also that it could play crucial roles in hyperalgesic conditions (e.g., myofascial pain syndrome). Taguchi 2008>>

<< Silent Nociceptors
Some nociceptors are normally silent, but after tissue injury, they become activated and respond to noxious stimuh. For example, Schaible and Schmidt [1988] showed that before experimental knee joint inflammation, some Group III and IV nociceptors do not spontaneously fire or respond to noxious knee joint movement. After the inflammation, however, these nociceptors fire spontaneously, and now respond to noxious joint movement Substances released as a result of the injury may sensitize the nociceptors, allowing them to fire to lower-intensity stimuli. Silent nociceptors were initially located in joint tissue, but have since been located in skin and viscera as well (Gebbart 1996, Schaible 1988, Schmidt 1995). Approximately one-third of nociceptors innervating the joint, skin, or viscera are silent and become activated after tissue damage.
Sluka 2016>>

<< Silent or “sleeping” nociceptors represent a particular group of nociceptors which become sensitized under inflammatory conditions; they only respond to mechanical stimulation after first going through such a sensitization phase (Mense and Gerwin 2010a).
Gautschi 2019>>

<< Concerning nociception, this analysis confirms the previous study by Schubert et al. (2006), which showed there were more receptors in fascia affected by Dupuytren’s disease, considered an inflammatory disorder (Baird et al., 1993; Meek et al., 1999; Qureshi et al., 2001). In some similar connective tissue diseases such as Achilles tendinosis (Schubert et al., 2005), patellar tendinopathy (Schwartz et al., 2015; Fu et al., 2002) and thoracolumbar inflammation (Bednar et al., 1995; Hoheisel et al., 2015; Mense and Hoheisel, 2016), substance P-positive nerve fibers have been seen to sprout: substance P is a neurotransmitter involved in both fibroblast stimulation (Lai et al., 2003) and nociception (Mashaghi et al., 2016). The effect could be attributable to increased secretion of nerve growth factor (Lubahn et al., 2007), which acts on

both nerve fibers and fibroblasts, causing differentiation into myofibroblasts. These results implicate pathological innervation in amplifying fibrosis and this could be the main cause of pain onset, indicating a connection between the fibrotic and painful forms of the disorder. Stecco C 2018>>

<<However, under abnormal mechanical stimulation, a pathological change in fascial innervation may occur, resulting in dysfunctional ingrowth of nociceptive fibers (Sanchis-Alfonso and Rosello-Sastre, 2000) that generates or maintains inflammation (Herbert and Holzer, 2002). Even the epineurium and perineurium are innervated by nervi nervorum, which can in turn cause neurogenic inflammation and consequently evoke dysesthetic (distal) and nerve trunk (local) pain when a mechanical stimulus is applied along the nerve path (Bove, 2008).
In addition, evidence suggests that nociceptive input rising from the thoracolumbar fascia may contribute to pain in non-specific low back pain (although not to localized pressure hyperalgesia) (Schilder et al., 2014). This may also produce changes in the corresponding spinal area. Hoheisel and Mense (2014) noted that following induced inflammation in the thoracolumbar fascia, an expansion of the spinal target region of fascia afferents has been demonstrated, together with the appearance of new receptive fields that could explain the spread of pain in patients with non-specific low back pain. Therefore, irritation of primary afferent fibers in the fascia is capable of initiating the release of neuropeptides, eventually setting up a neurogenic inflammation, with peripheral (Deising et al., 2012) and central sensitization, and altering the texture of surrounding connective tissue via the interaction of fibroblast and immune cells (Mense, 2001). This process may trigger a cascade of either local or global responses: chronic pain and connective tissue remodeling (Langevin and Sherman, 2007), altered mechanoreceptor feedback and muscle control (Panjabi, 2006), followed by further connective tissue alterations, neural adaptation, and eventually cortical reorganization (Flor, 2003). This process may expand to include influences on endocrine and autonomic pathways (Benarroch, 2006), as well as on sensory, cognitive and affective areas of the brain, that may in turn respond to control pain (Peyron et al., 2000). Recent studies have also shown that myofascial pain may alter the activity of related higher centres accounting for a reduction of sensory processing, followed by an altered motor output (Schabrun et al., 2013), together with a reorganization of the motor cortex associated with deficits in postural control (Tsao et al., 2008). Such evidence may explain several local, segmental and global effects of a given fascial dysfunction, proposing fascia as a possible nociceptive source for the establishment of somatic dysfunction and of its features, such as tenderness and tissue texture changes. Tozzi 2015>>

<<The effects of a fasciitis on the sensory innervation
For quantitative evaluation of the fasciitis effects, only the units stained for PGP 9.5, CGRP, and SP were selected. An experimental fasciitis was induced by injection of

Freund complete adjuvant into the fascia. Twelve days later the density of innervation for each fibre type was determined. The fasciitis was supposed to not only mimic the symptoms of a clinical fasciitis but also of other long-lasting painful alterations of the fascia. The histological sections showed a localized swelling at the injection site and massive infiltrations of leukocytes in all layers of the fascia. At high magnifications eosinophilic granulocytes and lymphoid cells were visible. The histological picture resembled an eosinophilic inflammation. (Lebeaux 2012) There was no difference in the morphology between free nerve endings in the inflamed and intact fascia. The inflamed fascia exhibited a higher density of CGRP-ir and SP-ir units (Figure 7B,C). However, the density of PGP 9.5-containing units – which represented all fibres and endings in the fascia – was not higher (Figure 7A). This apparent discrepancy is due to the fact that the TH-ir sympathetic units had a much lower density in the inflamed fascia (not shown). The reason for this decrease in TH-ir fiber density is not known. Schaible and Straub have likewise described a changed sympathetic supply to joints when they were experimentally inflamed. (Schaible2013) The inflammation-induced changes in innervation density occurred in the subcutaneous tissue and the inner layer; they were almost absent in the middle layer. Moreover, we never found a SP-ir fibre in this layer, and this was valid for the intact and inflamed fascia. The mechanism through which the fibre length increases in inflamed tissue is obscure. The two main possibilities are sprouting or increased branching of the fibers. In a study on afferents from the intact and inflamed gastrocnemius-soleus muscle, also NGF-ir and growth associated protein 43- [(GAP 43-)] ir fibers were increased. (Reinert 1998) This finding points to sprouting as the most important mechanism. So, an increased density of nociceptive fibers in inflamed tissue appears to be a general phenomenon. Mense 2019>>

<< Changes in innervation can occur pathologically in fascia. Sanchis-Alfonso and Rosello-Sastre (2000) report the ingrowth of nociceptive fibres and immuno-reaction to substance P in the lateral knee retinaculum of patients with patellofemoral malalignment problems.
Bednar et al (1995) found an alteration in both the histological structure (inflammation and microcalcifications) and the degree of innervation of the TLF in patients with chronic lumbalgia, indicating a possible role of the fascia in lumbar pain. In particular, these authors found a loss of nerve fibres in the TLF of back pain patients. Stecco C 2015>>

<<Fascial pH and factors influencing its levels. Several free nerve endings in the fascia inform the insular cortex of the forebrain about physiological tissue conditions, such as pH changes and warmth (Craig, 2002). Tozzi 2015 >>

<< There is a family of pain receptors called acid sensing ion channels (asic Ia, Ib, 2a, 2b, 3 and 4). 'Acid sensing' here means: 'acid sensitive', and these receptors send out

a signal that the brain will experience as pain. In the event of a small change in acidity, pH, this signal is enhanced. (Wemmie 2013) Many processes affect those small variations in this acidity, including inflammations. These ASIC proteins are found on many types of nerve cells, but as a single molecule they are inactive. At first, three of these asic molecules have to form a complex together, before they give a functional signal resulting in pain. Water has a neutral pH, and when the pH decreases, it is called acidic. If it increases we call it alkaline. At a normal pH in the blood and tissues, the majority of these ASIC receptors are in a neutral state and therefore inactive. Suppose you tap your hand, then you will feel that tapping like something mechanical, but it doesn't hurt at all. But if you have an injury, for example a wound or you have had a minor burn, and then you tap your hand in the same way, it may hurt a lot. This is due to the irritation, damage or inflammation, in which three ASIC receptors have formed a trimeric complex, in order to become active, and then they will give a pain signal in this configuration. Once all the ASIC receptors have been placed in the trimeric form, the pain becomes very intense. There is a kind of volume control in the pain system which stays, under normal circumstances, at a low level. In addition to this strengthening of the pain stimulus, the number of receptors can also become higher, due to the increased degree of inflammation. Capel 2019>>

Wat test je?

Distraction

Thigh Thrust

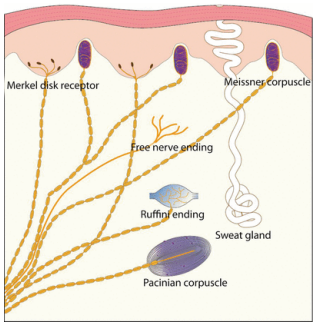
Compression

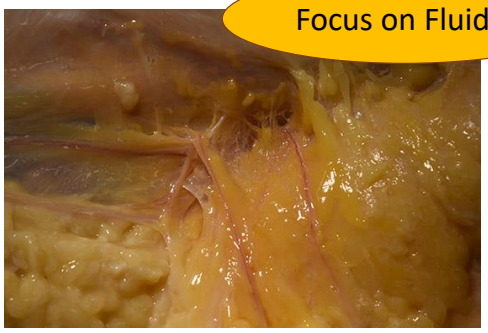
FABER

Gaenslen

10

Chemosensoren in ‘wet’ tissues





Focus on Fluids!

It is all about the environment!

11

<<The primary purpose of connective tissue is protection of the cells.
Single-celled microorganisms essentially possess all the major functional abilities that our multicellular bodies possess. They can absorb and remove chemical substances. They have organs, or organelles, that can absorb, convert, reduce, and utilize nutrients as energy. They have sensory and controlling systems - the integrated membrane proteins (channel proteins) that transport substances into and out of the cell. But there are also receptor proteins that transfer information from the outside to the inside of the cell, controlling the activity of the cell nucleus and. thus, the genetic material.' The single cell possesses contractility and, in this way, it can change its form, and it is mobile.
The only major multicellular-type functional characteristic that the single cell does not possess is protection against mechanical loading. The thin cell membrane, with its phospholipid components, does not lend the cell a significant amount of mechanical protection. This was not a problem when cells first evolved billions of years ago, because the first microbes were presumably filled with water and were living in water, thus the cell membrane was not exposed to higher mechanical loadings (vdBerg 2011).

Due to these developments, there was a necessity for the cells of the multicellular

organism to be protected against mechanical loading. To achieve this protection, the cells produce an extracellular matrix. This extracellular matrix is connected to the cell membrane by integrins.' - If the cell is mainly loaded with tensile loading, then it primarily produces collagen libers - mostly collagen type I. Collagen type I is the main collagen in the human body (making up 95% of the body's collagen). It is also the thickest and the strongest of all collagens. Vd Berg 2017 Ch 5>>

Primary function of fascia

- Providing a container for the liquid environment of the cell
- Protection against mechanical loading
- Providing a system of fluid management (blood/lymph)

To properly understand the anatomy, physiology and pathology of fascia, it is useful to look at the three basic ingredients of fascia: Cells, fibers, groundsubstance
E.C.M = extra cellular matrix = fibers+ groundsubstance

<< The term extracellular matrix (ECM) is applied to the sum total of extracellular substance within the connective tissue. Essentially it consists of a system of insoluble protein fibrils and soluble complexes composed of carbohydrate polymers linked to protein molecules (i.e. they are proteoglycans) which bind water. Mechanically, the ECM has evolved to distribute the stresses of movement and gravity while at the same time maintaining the shape of the different components of the body. It also provides the physico-chemical environment of the cells imbedded in it, forming a framework to which they adhere and on which they can move, maintaining an appropriate porous, hydrated, ionic milieu, through which metabolites and nutrients can diffuse freely.'" Myers 2017 ??>>

<<It is the task of the fascia and all of the connective tissue to protect the cells, the tissues, and the entire body against mechanical loading - including tensile loading, compression, and friction - as well as to enable the repair process after injury. vd Berg in Tozzi 2017 Ch 6>>

Cells in the matrix: you can compare this to people in a house. The house protects the people against rain and storm, but most important for survival of the people is the air in the house.

<<It is the task of the fascia and all of the connective tissue to protect the cells, the tissues, and the entire body against mechanical loading - including tensile loading, compression, and friction - as well as to enable the

repair process after injury. vd Berg in Tozzi 2017 Ch 6>>

<<Despite the high degree of differentiation and specialization of organelles and organelle systems, the cells of higher organisms retained their original characteristics. The cell, as a component of a macroorganism, also swims in an 'ocean' - the interstitial fluid, which is also described as an 'internal environment'. Meert 2012 Ch 1>>

<<This hydrophilic, viscous lubricant is found throughout fascia, skin and neural tissue. Hyaluronan (HA) is involved in a variety of physiological and mechanical functions such as wound healing, lubrication, cellular signalling, maintaining osmotic balance and tissue hydration (Liao et al. 2005, Matteini et al. 2009). Collectively the ECM defines the shape and form of our cells. It provides a framework to which the cells can adhere, move about in and communicate through. The ECM creates a medium by which appropriate balance can be maintained between porosity, hydration and ionic environment, thus allowing nutrients and metabolites to diffuse freely into and out of our cells. The ECM acts as an immune barrier. It is also a repository for metabolites and toxins, and for storing fat.

HA is present between the sublayers of aponeurotic fascia and between the deep fascia and the underlying muscles. In skeletal muscles HA is present in the epimysium, perimysium and endomysium (Piehl-Aulin et al 1991; Laurent et al 1991; Mc Combe et al 2001). Perivascular and perineural fascia also contain high levels of HA. Smith 2016>>

<<A further important role played by the fascial system is the management of liquids, such as lymph and blood. In fact, the fascia activates the flow of lymph and blood toward the different structures meant for containing the liquids; this activity is attributable to the innate contractile property of the fascia but also to different pressure gradients generated by the various fascial layers, which compel the liquids to flow (Findley 2013, Lee 2013, Li H-Y 2008, Park 2013, Soh 2009). The flowing of liquids is important for cellular health. Bordoni 2015>>

<<. Meert (2012) notes that the fluid in the ECM: 'creates a transport space for nutrients, waste materials and messenger substances and actually facilitates homeostasis between the extracellular and the intracellular region. In addition, the lymphatic system filters this supply out of the ocean of interstitial fluids and drains it into the venous system.'

The clinical relevance of fluid dynamics and fascia points to fluid/water having a major influence on flexibility and stiffness, as well as on the distribution of substances, such as nutrients, pro- and anti-inflammatory products, and the drainage of debris during processes such as inflammation and tissue repair - with influences on homeostatic function.

Fascia also plays an important role in fluid balance and physiology. Water content is dependent on changes in interstitial fluid pressure resulting from a dynamic interaction between the osmotic pull exerted by negatively charged, normally under-hydrated glycosaminoglycans that are abundant in fascia, and the mechanical stiffness and tension of collagen fibers that resist water extrusion and thus tissue swelling (Mow and Ratcliffe, 1997). It appears that any decrease in collagen tension leads to a reduction in interstitial hydrostatic pressure and causes fluids to be taken up by the constituents of the ECM. The role of fibroblasts is also crucial in determining collagen tension through cell-matrix contacts, acting as modulators of fluid dynamics by adjusting their size and matrix tension in response to changes in osmotic pressure (Langevin et al., 2013). In turn, hydrostatic pressure has been shown to act as a mechanical stimulus inducing cell-matrix processes of mechanotranduction that directs cell behavior in a variety of tissues, including cell movement within the

ECM (Polacheck et al., 2014), cytoskeletal polymerization and cellular tuning of sensitivity to fluid pressure, and to accommodate variable levels of stress (Myers et al., 2007). In particular, intermittent cyclic hydrostatic pressure applied to bone-derived cells induces alterations in mRNA levels for a specific subset of genes involved in connective tissue remodelling and differentiation (Tasevski et al., 2005). However, during inflammation, changes in physical properties of the connective tissue, involving hyaluronic complexes, may influence transcapillary exchange resulting in as much as a hundred-fold increase in fluid flow (Reed et al., 2010). This is caused by an integrin-mediated lowering in interstitial fluid pressure following a release of cellular tension exerted on the collagen network that allows glycosaminoglycans to expand and take up fluids. Therefore, a reciprocal influence exists between mechanical force, cell response and interstitial fluid dynamics. A sustained static stretch applied to fascia may produce an extrusion of water in the tissue followed by a compensatory increase in matrix hydration (Schleip et al., 2012a), resembling a sponge-like effect that may be significant for fascial function. Chaitow 2014>>

<< The entire system is also serving as a pump, distributing the fluids which bath individual cells, and intimately involved in water metabolism. To organize the fascial system in thereby to alter the flow of fluids and

therefore the delivery of nutritients to cells throughout the body.
The clinical relevance of fluid dynamics and fascia points to fluid/water having a major influence on flexibility and stiffness, as well as on the distribution of substances, such as nutrients, pro- and anti-inflammatory products, and the drainage of debris during processes such as inflammation and tissue repair - with influences on homeostatic function. Meert 2012 Ch 1>>

<<Only a small proportion of the body fluids (approximately 4-6 l) circulates as blood in the average adult human weighing 70 kg. Of this circulating blood, only around one-quarter is in the arterial system (under greater pressure), while around three-quarters is in the venous system (at lower pressure). A large proportion of the body fluids (approximately 10-12 1) is found outside the vascular system: around 7-9 I in the interstitium, the tissue between the cells, with around 3 1 of lymph flowing in the lymphatic vessels. In order to enable the exchange of substances between these systems, the flow of body fluids needs to be quite slow and the blood pressure low. In certain organs and tissue regions, such as cartilage, the cornea and the valves of the heart, vessels are completely absent and the exchange of substances takes place by diffusion. The greatest proportion of body fluids is located in the intracellular space (around 28 1).

It is therefore to the intracellular region that cell metabolism must look, since its field of operations is dependent on the supply of raw materials (nutrients) and the removal of waste substances (products of metabolism) via the interstitial space to serve the actual field of operations within the cell. Meert 2012 Ch 1>>

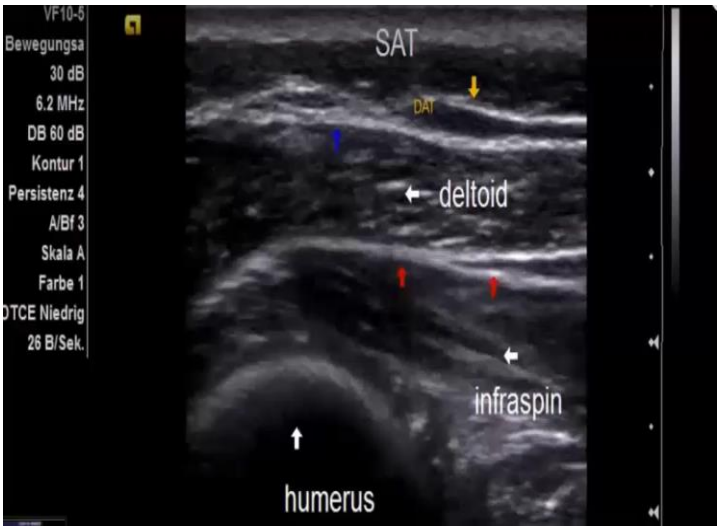
<<On average, people ingest 2300 ml of water per day in food and drink. Metabolic conversion of nutrients accounts for a further 200 ml, resulting in an estimated total intake of 2500 ml per day for human beings (McBride, 1998).
This water is then excreted in a variety of ways. The kidneys excrete about 1500 ml, the skin approximately 500 ml, the lungs approximately 300 ml and the gastrointestinal tract roughly 200 ml per day. Consequently, up to 2500 ml of water is excreted each day, in order to maintain an overall balance between intake and elimination of water (McBride, 1998).

This means that extracellular fluid can be found in three groups of regions in the body:
vessels: blood plasma and lymph
free-flowing in connective tissue: interstitial fluid or lymph (including CSF)
distinctly separate areas: synovial fluids, serous fluids (pleural, pericardial and peritoneal) and excretory fluids (urine and sweat).

Meert 2012 Ch 2>>

<<Pressure changes and unhindered mobility are very important for fluid exchange (and best to be influenced). In addition, osmolar gradients and the electrical potentials of particles are also included.
Marcer, 2005 >>

Groundsubstance: Friction free movement



12

<< For the collagen fibers to move friction-free against each other during loading, the cell produces a small amount of ground substance that is able to bind water, resulting in a lubricating film. The ground substance and the bound water are vital for the cells, because all of our cells must 'feed' themselves through the process of diffusion - just like the original single-cell microbes did on Earth. Diffusion requires water. Berg in Lima 2017 Ch2>>

<<The HA (hyaluronan) appears to provide a lubricating surface as fascia glides smoothly over muscles and tendons. Because of its considerable charge at neutral pH, HA is surrounded by an enormous volume of solvent water that can exert pressures on various adjacent structures. Stecco 2013>>

<<Lubrication and protection against friction
Due to the ground substance and the water bound to it within the extracellular matrix, tissue movement can take place without friction. During movement the deformation of the matrix forces the ground substance to release water, which will be absorbed again when the tissue is relaxed again. This water moves through the tissue but also leaks into neighbouring tissues. An example is the intervertebral disc, where leaking water will pass through the endplate into the vertebral body. The same phenomenon occurs in the articular cartilage, the bursae, and tendon sheets. In the paratenon, the outer sheet of the tendon constantly produces a fluid similar to synovial fluid. In fact, all tissues permanently form a fluid film to ensure lubrication during movement.
This friction-free movement is extremely important, because permanent friction would adversely affect health. Unrelieved friction would mean that the body would never be able to stabilize its core temperature at a constant level of approximately 37° C.
vd Berg in Tozzi 2017 Ch 5>>

Fascia as a tissue of separation: creates space which serves as an interface for ease of sliding and gliding of structures and tissues in relation to one another. Spatial separation supports unimpeded motion and motion ensures the healthy functioning of tissues and structures and the sliding-gliding interface.

HA helps reduce the impact of compressive forces in synovial-joint related tissues and improves slide/glide between various structural proteins and layers of adjacent tissue (e.g. between various layers of fascia and between muscle fibres) (McCombre et al. 2001, Stecco et al. 2011).

<<Movement in the body does not only depend on the mobility of the joints but also on the movement possibilities between different collagen layers and between, for example, nerves and their environment. This gel is also found between the different sheets of the aponeurotic fascia and in the endo, peri -and epimysium (McCombe et al. 2001).>>

<< Cold temperature makes the groundsubstance more viscous, so that there is more stability, while the mobility increases with heat. A warm-up is not just about warming up the muscles, but maybe even more about warming up the groundsubstance.>>

<<This friction-free movement is extremely important, because permanent friction would adversely affect health. Unrelieved friction would mean that the body would never be able to stabilize its core temperature at a constant level of approximately 37° C. Vd Berg 2017 Ch 5>>

<<The ultrasound study highlighted a mean thickness of 1.88 mm of the fascia lata, 1.68 mm of the rectus sheath, and 1.73 mm of the sternocleidomastoid fascia. The HA within the deep fascia facilitates the free sliding of two adjacent fibrous fascial layers, thus promoting the normal function associated with the deep fascia. If the HA assumes a more packed conformation, or more generally, if the loose connective tissue inside the fascia alters its density, the behavior of the entire deep fascia and the underlying muscle would be compromised. This, we predict, may be the basis of the common phenomenon known as ‘‘myofascial pain.’’
The retention of HA after exercise, combined with an endomysial location supports the concept that HA not only lubricates but also facilitates movements between muscle fibers Stecco C 2011>>

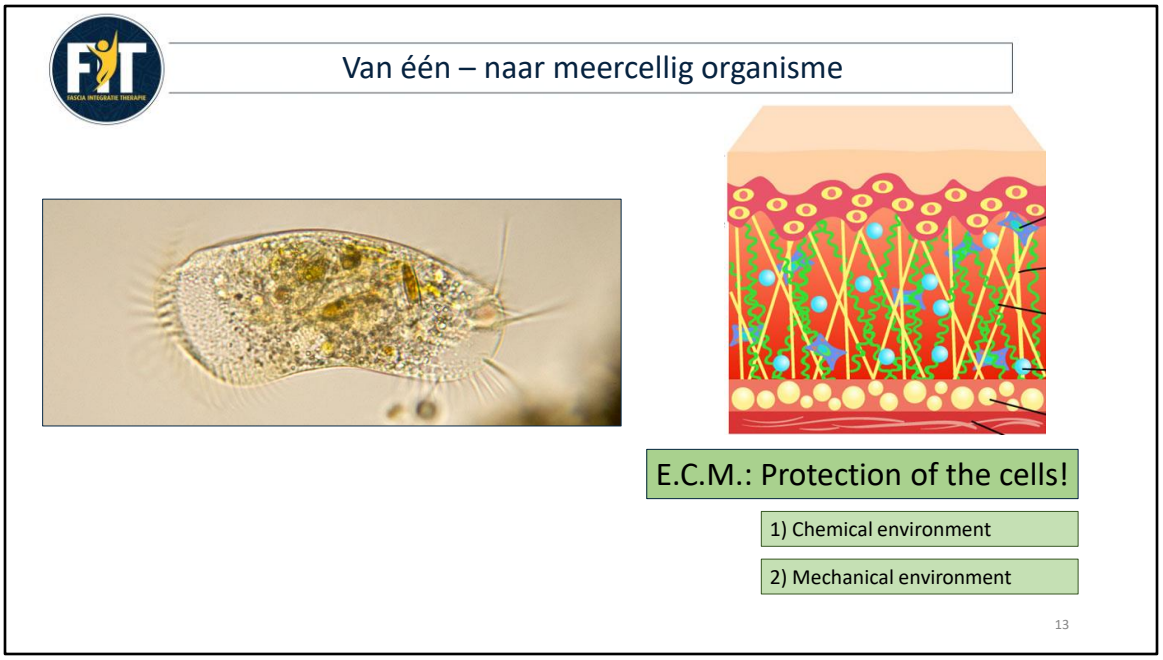
<<However, until now, the precise content of hyaluronan within fasciae has been unknown. This study quantifies for the first time the hyaluronan content of human fascial samples obtained from a variety of anatomic sites. Here, we demonstrate that the average amount varies according to anatomic site, and according to the different kinds of sliding properties of the particular fascia. For example, the fascia lata has 35 lg of hyaluronan per gram of tissue, similar to that of the rectus sheath (29 lg g_1). However, the types of fascia adherent to muscle contain far less hyaluronan: 6 lg g_1 in the fascia overlying the trapezius and deltoid muscles. In the fascia that surrounds joints, the hyaluronan increases to 90 lg g_1, such as in the retinacula of the ankle, where greater degrees of movement occur. Surprisingly, no significant differences were detected at any site as a function of age or sex (P-value > 0.05, t-test) with the sole exception of the plantar fascia.

As has been demonstrated recently (Klein et al. 1999; Stecco et al. 2011; Cowman et al. 2015a,b), fascia presents a lubricating layer of hyaluronan (HA), allowing sliding between fascial sublayers and between fasciae and adjacent muscles, bones, and joints. HA is the major glycosaminoglycan (GAG) of the extracellular matrix (ECM). It is composed of D-glucuronic acid and N-acetyl-D-glucosamine, linked via alternating b-(1?4) and b- (1?3) glycosidic bonds. Polymers of HA can range in size from a few kDa up to 8 MDa. The molecular size strongly influences the biological, physiological, and pathological functions of the molecule (Cowman et al. 2015a,b). The HA content in in vivo tissues is usually constant and is the result of the simultaneous action of the synthetases, HAS1, HAS2, HAS3 (Itano & Kimata, 1996; DeAngelis, 1999), the hyaluronidases (HYAL) in its various isoforms (Stern & Jedrzejas, 2006; Csoka & Stern, 2013), and other degrading molecules, such as reactive oxygen species (Triggs-Raine & Natowicz, 2015).
Fede 2018>>

<< The layers of loose connective tissue within deep fasciae were studied with particular emphasis on the histochemical distribution of hyaluronan (HA). Samples of deep fascia together with the underlying muscles were taken from neck, abdomen and thigh from three fresh nonembalmed
cadavers. Samples were stained with hematoxylin–eosin, Azan-Mallory, Alcian blue and a biotinylated HA-binding protein specific for HA. An ultrasound study was also performed on 22 voluntary subjects to analyze the thickness of these deep fasciae and their sublayers. The deep fascia presented a layer of HA between fascia and the muscle and within the loose connective tissue that divided different fibrous sublayers of the deep fascia. Stecco C 2011>>

<< For the collagen fibers to move friction-free against each other during loading, the cell produces a small amount of ground substance that is able to bind water, resulting in a lubricating film. The ground substance and the bound water are vital for the cells, because all of our cells must 'feed' themselves through the process of diffusion - just like the original single-cell microbes did on Earth. Diffusion requires water. Berg in Lima 2017 Ch2>>

<< Fascia also has this lubrication effect. Fascia incorporates a water-dense vascular system, which enables different fasciae to slide friction-free against each other. Guimbertuea J 2010, 2005, Langevin 2009). The fluid production in the fascia can be disturbed under pathologic circumstances, such as decreased circulation caused by increased sympathetic reflex activity. An example would be the development of subcutaneous connective tissue zones, in which we find reduced mobility as well as a decrease in the possibility of lifting the skin from the bottom layer. Vd Berg 2017 Ch 5>>



<<The primary purpose of connective tissue is protection of the cells.
Single-celled microorganisms essentially possess all the major functional abilities that our multicellular bodies possess. They can absorb and remove chemical substances. They have organs, or organelles, that can absorb, convert, reduce, and utilize nutrients as energy. They have sensory and controlling systems - the integrated membrane proteins (channel proteins) that transport substances into and out of the cell. But there are also receptor proteins that transfer information from the outside to the inside of the cell, controlling the activity of the cell nucleus and. thus, the genetic material.' The single cell possesses contractility and, in this way, it can change its form, and it is mobile.
The only major multicellular-type functional characteristic that the single cell does not possess is protection against mechanical loading. The thin cell membrane, with its phospholipid components, does not lend the cell a significant amount of mechanical protection. This was not a problem when cells first evolved billions of years ago, because the first microbes were presumably filled with water and were living in water, thus the cell membrane was not exposed to higher mechanical loadings (vdBerg 2011).

Due to these developments, there was a necessity for the cells of the multicellular

organism to be protected against mechanical loading. To achieve this protection, the cells produce an extracellular matrix. This extracellular matrix is connected to the cell membrane by integrins.' - If the cell is mainly loaded with tensile loading, then it primarily produces collagen libers - mostly collagen type I. Collagen type I is the main collagen in the human body (making up 95% of the body's collagen). It is also the thickest and the strongest of all collagens. Vd Berg 2017 Ch 5>>

Primary function of fascia

- Providing a container for the liquid environment of the cell
- Protection against mechanical loading
- Providing a system of fluid management (blood/lymph)

To properly understand the anatomy, physiology and pathology of fascia, it is useful to look at the three basic ingredients of fascia: Cells, fibers, groundsubstance
E.C.M = extra cellular matrix = fibers+ groundsubstance

<< The term extracellular matrix (ECM) is applied to the sum total of extracellular substance within the connective tissue. Essentially it consists of a system of insoluble protein fibrils and soluble complexes composed of carbohydrate polymers linked to protein molecules (i.e. they are proteoglycans) which bind water. Mechanically, the ECM has evolved to distribute the stresses of movement and gravity while at the same time maintaining the shape of the different components of the body. It also provides the physico-chemical environment of the cells imbedded in it, forming a framework to which they adhere and on which they can move, maintaining an appropriate porous, hydrated, ionic milieu, through which metabolites and nutrients can diffuse freely.'" Myers 2017 ??>>

<<It is the task of the fascia and all of the connective tissue to protect the cells, the tissues, and the entire body against mechanical loading - including tensile loading, compression, and friction - as well as to enable the repair process after injury. vd Berg in Tozzi 2017 Ch 6>>

Cells in the matrix: you can compare this to people in a house. The house protects the people against rain and storm, but most important for survival of the people is the air in the house.

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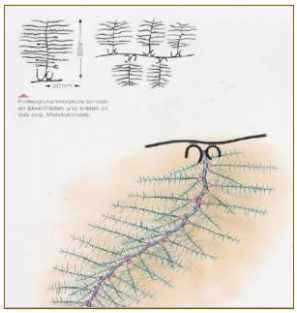
<<Despite the high degree of differentiation and specialization of organelles and organelle systems, the cells of higher organisms retained their original characteristics. The cell, as a component of a macroorganism, also swims in an 'ocean' - the interstitial fluid, which is also described as an 'internal environment'. Meert 2012 Ch 1>>

<<This hydrophilic, viscous lubricant is found throughout fascia, skin and neural tissue. Hyaluronan (HA) is involved in a variety of physiological and mechanical functions such as wound healing, lubrication, cellular signalling, maintaining osmotic balance and tissue hydration (Liao et al. 2005, Matteini et al. 2009). Collectively the ECM defines the shape and form of our cells. It provides a framework to which the cells can adhere, move about in and communicate through. The ECM creates a medium by which appropriate balance

can be maintained between porosity, hydration and ionic environment, thus allowing nutrients and metabolites to diffuse freely into and out of our cells. The ECM acts as an immune barrier. It is also a repository for metabolites and toxins, and for storing fat.

HA is present between the sublayers of aponeurotic fascia and between the deep fascia and the underlying muscles. In skeletal muscles HA is present in the epimysium, perimysium and endomysium (Piehl-Aulin et al 1991; Laurent et al 1991; Mc Combe et al 2001). Perivascular and perineural fascia also contain high levels of HA. Smith 2016>>

Hyaluron: Matrix in een matrix



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<<GAGs are not flexible enough to assume a globular form and stay extended occupying an ample surface in relationship to their volume. The high density of negative charges attracts water forming a hydrated gel; this gel is responsible for the turgidity and viscoelasticity of the connective tissue (CT) and controls the diffusion of various metabolites. In particular, it permits the rapid diffusion of water-soluble molecules but inhibits the movement of large molecules and bacteria. Its viscoelasticity allows the tissue to return to its original form after stress, and enables the collagen fibers to move without friction against each other, to absorb forces that affect the tissue and to protect the collagen network from excessive stress.

This HA-rich layer also protects muscles and supports recovery from injury, and stimulates satellite cell proliferation following loss of muscle fibres. Changes in this HA-rich matrix contribute to pain, inflammation and loss of function. HA is abundant during the earliest stages of wound healing and functions to open up tissue spaces through which cells can travel. By binding to cell receptors and interacting with the cytoskeleton it confers motility to the cells.

HA is particularly plentiful during embryogenesis and in tissues undergoing rapid growth, and is present wherever repair and regeneration occur. Depending on its chain length, especially when it becomes fragmented, HA has recently been shown to have a wide range of opposing biological functions: such as becoming angiogenic, inflammatory and immunostimulatory.

The turnover of HA is about 2-4 days compared to 7-10 days for the other sulfated GAGs. This means that the HA cells must remain active, otherwise there is the risk of reduction in the quantity of ground substance. The residual products of the GAGs have a feedback effect on the cells and this controls

synthesis. It has been established that mechanical distortion of CT cells represents a stimulus for ECM synthesis (Adhikari et al 2011). Stecco C, 2015>>

<< In an active area of the body, the ground substance changes its state constantly to meet local needs; in a 'held' or 'still' area of the body, it tends to dehydrate to become more viscous, more gel-like, and to become a repository for metabolites and toxins. Myers 2014>>

<<The ECM plays an important role in wound healing and repair. It serves as a repository for signaling molecules and mediates signals from other cells to promote cell proliferation and differentiation. The ECM is responsive to mechanical strain and tensional loading (tissue deformation). Mechanical forces exert influence on the tissue structural elements (microfilaments) and the molecular composition of the ECM (Benjamin & Ralphs 1998, Milz et al. 2005). Strain type, degree, direction and duration can influence ECM composition and impact fibroblast functions that guide healing and adaptation responses (Purslow2002, Ingber 2003, Standley & Meltzer 2008, Stecco et al. 2009, Blechschmidt & Gasser 2012).

Hyaluronic Acid (HA) is present between the sublayers of aponeurotic fascia and between the deep fascia and the underlying muscles. In skeletal muscles HA is present in the epimysium, perimysium and endomysium (Piehl-Aulin et al 1991; Laurent et al 1991; Mc Combe et al 2001). Perivascular and perineural fascia also contain high levels of HA. HA occurs both as individual molecules, and as macromolecular complexes that contribute to the structural and mechanical properties of fascia. HA is a lubricant that allows normal gliding of joint and connective tissues. It is likely that these gliding interactions are influenced by the composition and efficacy of the HA-rich instructional matrix. Changes in this HA-rich matrix contribute to pain, inflammation and loss of function, and may in fact be critical to these changes in pathology (Lee & Spicer 2000). The HA-rich layer between aponeurotic fascia and muscle seems to function to protect the muscle, to support recovery from injury and to stimulate satellite cell proliferation following loss of muscle fibres. This layer also plays a role in the inflammatory process and HA is prevalent

Depending on its chain length, HA has a wide range of biological functions and sometimes opposing ones. Its high molecular weight form is found in normal, quiescent tissues, while fragmented HA indicates tissues under stress exhibiting highly angiogenic, inflammatory and immunostimulatory influences.

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PCs and GAGs have many different functions. First, they stabilize the connective tissue by binding to collagen and elastic fibers, cells and water. They predominantly absorb forces that affect the unformed tissue and protect the collagen network from excessive stress. The other important task of the ground substance is the absorption of compression force on cartilage, intervertebral discs in the nucleus area, etc. (Buckwalter et al. 1988; Eyre et al. 1989).

<<M. Foldi et al. state that a third of the hyaluronic acid content of the body is broken down daily and

replaced. This occurs principally in the regional lymph nodes and to some extent in the liver.

Hyaluronidase is sometimes also called 'spreading factor'. It loosens the structure of the connective tissue, thus facilitating fluid exchange between the connective tissue and the vascular system. Various tissues produce and secrete hyaluronidase - the skin, the spleen and the testicle - and » certain bacteria can release this enzyme too. Granulocytes, by modifications of the pH or the electrolyte concentration or by the presence of inhibitor substances (antibodies, anti-enzymes), can also form lysosomes filled with hyaluronidase and release them.
The connective tissue matrix is, so to speak, in a 'labile equilibrium'. To a certain extent, it is fluctuating, under the influence of numerous factors, between two extremes:
A very permeable phase (sol state): rich in 'free' water and poor in 'colloids' (macromolecules or GAG). Hyaluronic acid, in particular, is depolymerized by the effect of hyaluronidase in short chains. The matrix becomes more fluid and thus more permeable to hormones, neurotransmitters, inorganic ions (Na+, Ca2+, K+), electrical and energetic signals, viruses, opsonins, interferon, erythropoietin, antibodies and also antigens. The negative electrical field of the surface declines, and this, in its turn, attracts anions (for example, CI", HC03~, P043~, S04~2, proteins).
A less permeable phase (gel state): poor in 'free' water and rich in 'colloids' or macromolecules. Many water molecules are bound. The matrix becomes more viscous, less permeable and more saturated. The electromagnetic fields lose their coherence. The negative electrical field of the surface becomes more powerful, and this, in turn, attracts cations (for example, Na^+, K^+, Ca^{2+}, Mg^{2+}). Meert 2012>>

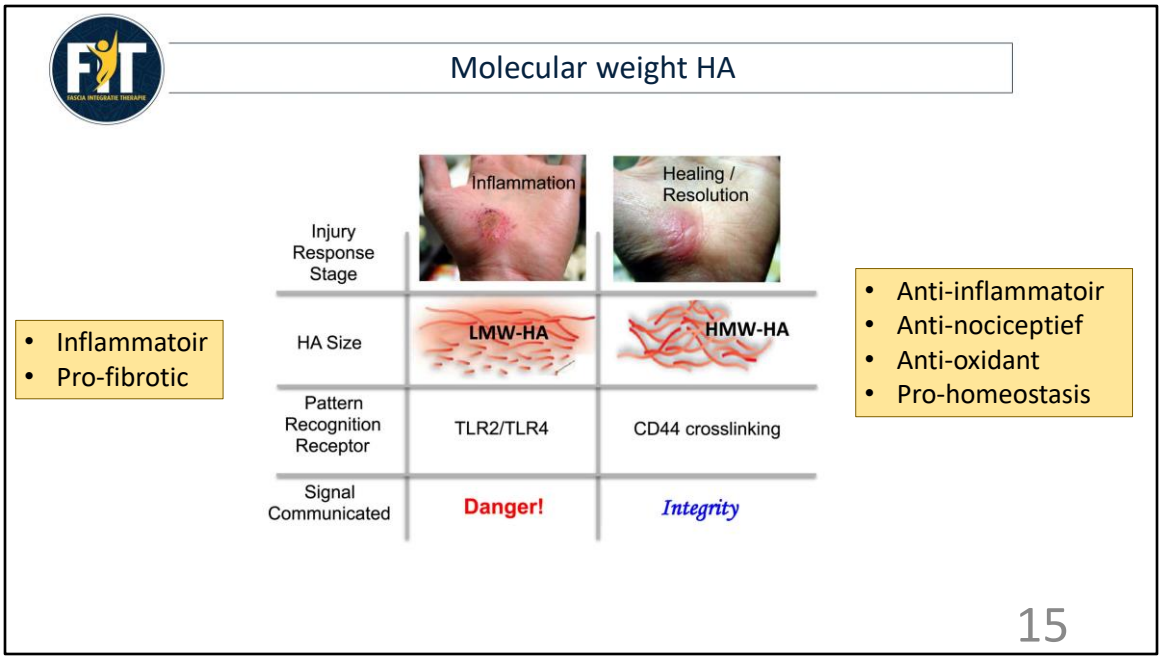
<<The heavy negative loading of the GAGs and PGs causes the capacity to bind to water and because of its viscoelasticity allows the tissue to return to its original form after stress. The binding of water also enables the collagen fibers to move without friction against each other. The stored water simultaneously serves as a transport route for nutrients and waste products as well (Grodzinsky 1983; Fleischmajer et al. 1990; Currier & Nelson 1992; Aaron & Bolander 2005; Van den Berg 2010).>>

<<Hyaluronan (HA) is an anionic linear polymer that is ubiquitously expressed in the extracellular matrix (ECM) of mammalian tissues, where it forms loose and elastic matrices (Fraser JR 1997, Dicker KT 2014). HA is a space filling molecule that makes the ECM an appropriate environment for cell movement and proliferation and confers elastoviscous biomechanical properties to the tissues (Balazs EA 1998, Evanko SP 2007). Caires 2014>>

<< Physical properties, size, and molecular weight distribution

High molecular weight (HMW) HA is a hydroviscous substance, a characteristic that dictates many of its macromolecular effects. Given its extremely hydrophilic nature, HA can bind up to 1000× its weight in water, thus forming a very voluminous, expanded random-coil

structure in aqueous physiological solutions. Interestingly, however, the structure of HMW HA varies with the context in which it is found. Thus, the glycocalyx of vascular endothelial cells consists of a brush-like border (Zhang X 2018), the HA surrounding the cumulus cells around the oocyte is tightly bound and circular (Nagyova E2018), and when surrounding chondrocytes, HA gives rise to a biomechanical structure that protects cartilage (Huang Y 2016). These variations of HMW HA likely arise from the macromolecular structure imparted by interacting HA-binding proteins and proteoglycans. Thus, HA most likely acts as an insulating coat or spatial buffer, permitting lowmolecular-weight molecules such as electrolytes and nutrients to diffuse, but blocking high-molecularweight proteins or even cells from reaching the cell surface, as elegantly demonstrated in the red blood cell exclusion assay (Lee GM 1993). Further, the endothelial HA glycocalyx prevents inflammatory cells from directly interacting with the endothelium by maintaining a nonadhesive surface and, in platelets, HA prevents adherence and degranulation (Petrey AC 2016). Degradation of HA, however, reduces its barrier function thus making the HA matrix much more permeable and accessible to cell interactions. Garantziotis S 2019>>



Overuse/trauma (?) – inflammation - Degradation HA - Low molecular weight (reduced polymerization) – more water – low viscocity
Inflammation: reduced concentration HA and reduced average molecular weight

<<HA can be cleaved by reactive oxygen species generated during inflammation (Henderson 1991, Halliwel 1995) It is also consistent with data reporting that high MW HA down-regulates inflammatory cell activity (Darzynkiewicz Z 1971, Forrester JV 1980), and that HA fragments stimulate innate immune system activity (Jiang D 2007, Pure E 2009). Band PA 2015>>

<<Hyaluronan (HA) is present in the extracellular matrix of all body tissues, including synovial fluid in joints, in which it behaves as a filter that buffers transmission of mechanical forces to nociceptor nerve endings thereby reducing pain. Using recombinant systems, mouse-cultured dorsal root ganglia (DRG) neurons and in vivo experiments, we found that HA also modulates polymodal transient receptor potential vanilloid subtype 1 (TRPV1) channels. HA diminishes heat, pH and capsaicin (CAP) responses, thus reducing the opening probability of the channel by stabilizing its closed state. Accordingly, in DRG neurons, HA

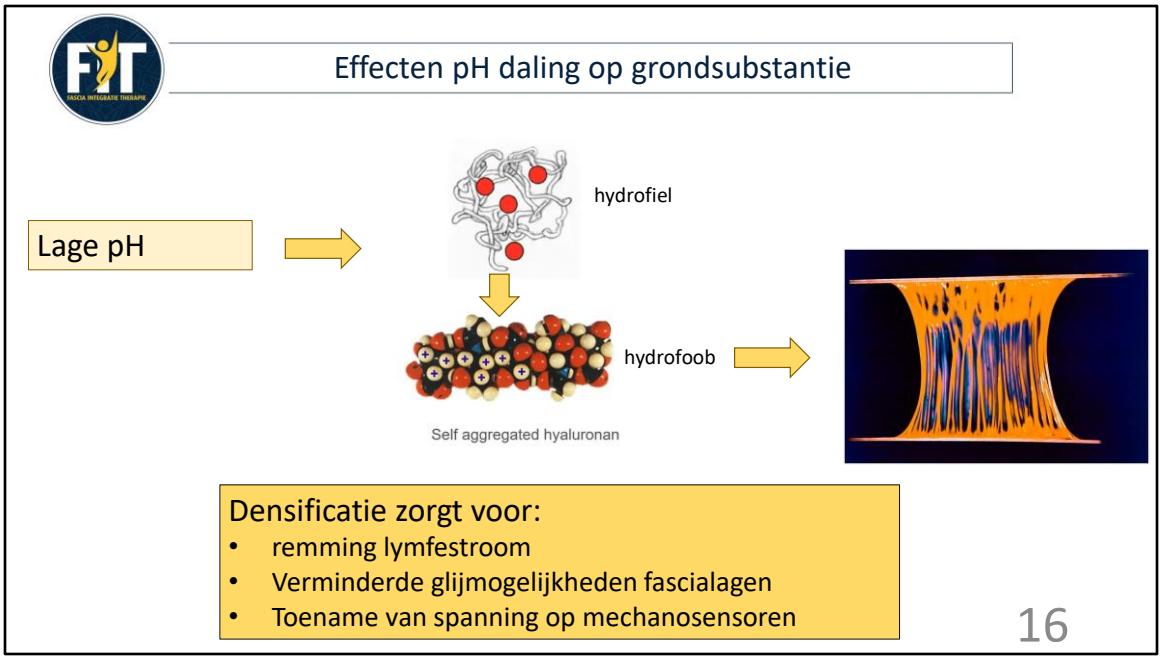
decreases TRPV1-mediated impulse firing and channel sensitization by bradykinin. Moreover, subcutaneous HA injection in mice reduces heat and capsaicin nocifensive responses, whereas the intra-articular injection of HA in rats decreases capsaicin joint nociceptor fibres discharge. Collectively, these results indicate that extracellular HA reduces the excitability of the ubiquitous TRPV1 channel, thereby lowering impulse activity in the peripheral nociceptor endings underlying pain. Caires 2015>>

<<HA size is one of the major determinants of its activity. Several studies have demonstrated that HA >1 MDa is anti-inflammatory and promotes epithelial cell homeostasis and survival. Thus, HA, at doses of 1 mg/ml or greater in its HMW form, inhibits inflammatory cell chemotaxis, phagocytosis, elastase release, and respiratory burst activity [16–19]. HMW HA

Removal of HA by early treatment of myocardial infarction with hyaluronidase results in reduced myocardial fibrosis and infarct size []. HA fragments consist of LMW HA (<~500–700 kDa), and much smaller HA oligosaccharides (8–30-dimer lengths). Collectively, these fragments increase the expression of proinflammatory chemokines and iNOS in macrophage cell lines as well as in alveolar macrophages from injured lungs.
Additionally, HMW HA accelerates cutaneous wound healing (King SR 1991) and reduces adhesion formation after intra-abdominal surgery (Urman B).
The reasons for this pronounced sizedependency are not

entirely clear. Several factors, including receptor clustering and engagement, cellular uptake, intra- vs extracellular signaling, and interactions with HA ligands such as heavy chains of inter-α-inhibitor (IαI), potentially related to linear or globular structures of different-sized HA chains, likely play a role.

The decoration of HA with IαI heavy chains alters the adhesive properties of HA to invading cells, as do aggrecan and versican, which confers pro-and antiinflammatory activity. Importantly, the association of HA with these proteins changes dynamically during tissue injury, inflammation, and organ development, thus lending great plasticity and versatility to the biological properties of HA matrices.
Garantziotis S 2019>>



Densification = problem of the groundsubstance: thickened, hardened, increased viscocity

<<The loose connective tissue of the fasciae is an important reservoir for water, salts and other elements. It is also a potential reservoir for the accumulation of degradation products, such as lactate. An accumulation of these products over time could alter the biomechanical properties of the loose connective tissue. Alteration of the loose connective tissue, as we have seen, can adversely affect the sliding motion of fascial layers and can cause myofascial dysfunction. Interactions among HA, lactate and alterations of pH in fascial tissues are of particular interest. Juel et al (2004) documented that when the pH inside muscles reaches 6.6, the athlete is at the stage of exhaustion. At pH 6.6, the viscosity of HA present in the endomysium and perimysium of muscles significantly increases. Gatej et al (2005) documents that at pH 6.6 (normal value is pH 7.4) the complex viscosity of HA approaches 5 Pa's instead of the typical 3.8 Pa's. This increase in viscosity can explain the typical stiffness experienced by athletes after prolonged intense activity (marathons, endurance games, etc.). This type of stiffness usually disappears with rest and a full restoration of painless range of motion occurs. The acidification of the fascial loose connective tissue is not the only component that creates stiffness. Overuse syndromes or trauma

could alter the viscosity of the fascia, promoting stiffness throughout the surrounding areas. One reason for postexercise pain and loss of motion remaining in a percentage of people may be that they have a high-viscosity profile. Overused or post-traumatic areas with increased viscosity may already exist and a high-viscosity profile may be maintained. From a clinical point of view, such areas can be defined as undergoing densification. They constitute potential trigger point type areas and may be responsible for the myofascial pain syndrome. Stecco C 2015>>

<<Hyaluronan (HA) is often increased under inflammation and is partly responsible for holding the water in edematous tissue. Hyaluronan and proteoglycans in high concentrations can make for a very viscous and sticky pro-inflammatory matrix. (de la Motte 1999, 2003) Hyaluronan is also increased in Achilles tendons following immobilization. (Okita 2004) Evanko, 2009>>

<<A densification ' in the loose connective tissue, rather than a fibrosis, occurs due to the increased viscosity of tissue. This increased viscosity is caused by the larger HA fragments and HA molecule entanglement. Normalizing of the HA molecule by deep compression, friction, heat and increased alkalinity are factors found to change the 'gel' into a more fluid medium, thus allowing restoration of the normal sliding function of the fascia. Stecco and colleagues (2013) hypothesize that the myofascial pain syndrome with its stiffness and pain is caused primarily by a I densification of the loose connective tissue. Chaitow 2014 H12>>

<<If the Hyaluronan (HA) assumes a more packed conformation, or more generally, if the loose connective tissue inside the fascia alters its density, the behavior of the entire deep fascia and the underlying muscle would be compromised. This, we predict, may be the basis of the common phenomenon known as myofascial pain. Besides, there is much evidence (Swerup 1996, Wilkinson 1983) that the activation of the receptors is strongly dependent on the viscoelasticity of the surrounding tissue. So, it is known that the HA is one of the most important elements that determines the viscoelasticity of a tissue. Its important presence inside the fascia could permit us to suppose that its alteration could modify the activation of the receptors inside the fascia. This could be the origin of the common phenomenon of myofascial pain. Stecco C 2011 >>

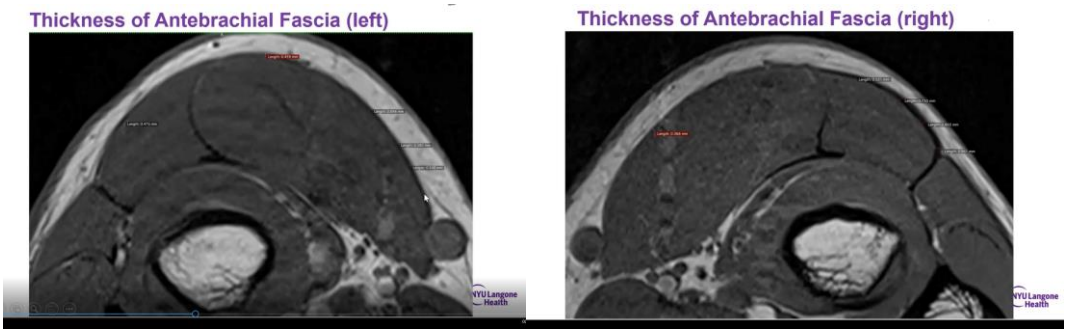
<<Changes in pH, ionic content and temperature may represent key environmental and metabolic factors influencing fascial viscosity (Thomas 2012). For instance, an increase in body temperature, as occurs during physical exercise, may reduce fascial stiffness by reducing tissue viscosity, while breathing exerts a significant influence on pH. In most breathing pattern disorders, a state of hypocapnia can occur, resulting in elevated pH levels due to respiratory alkalosis (Chaitow et al., 2014). In turn, this may lead to smooth muscle cell contraction and even spasm, with profound implications

on fascial, visceral and vasal tone (Foster et al., 2001). Such events may be commonly found in a variety of patients, since breathing pattern disorders have traditionally been associated with anxiety, stress (Magarian, 1982) and some chronic connective tissue disorders (Garcia-Campayo et al., 2011). Conversely, a more acidic ECM environment seems to exert a modulating action on the metabolism and protein synthesis of connective tissue cells, such as fibroblasts and chondrocytes (Ohshima and Urban, 1992), ranging from a predisposition to inflammatory reactions and tissue damage during high levels of acidosis (Levick, 1990); impaired remodelling and tissue repair with pH lower than 6.5; and to beneficial effects on the composition of both gelatinous and fibrous matrix (Mwale et al., 2011). Finally, myofibroblast contractility in vitro has been shown to increase with a lowering of pH (Pipelzadeh and Naylor, 1998), suggesting a potential influence on fascial tone. In conclusion, changes in breathing patterns and temperature, and presumably of physical activities (Shen et al., 2012) and nutrition (Arent et al., 2010) are able to modulate tissue pH levels through environmental and metabolic changes, and oscillations of these may strongly influence fascial function and dysfunction Tozzi 2015>>

<<Changes in this HA-rich matrix contribute to pain, inflammation and loss of function, and may in fact be critical to these changes in pathology (Lee & Spicer 2000). The HA-rich layer between aponeurotic fascia and muscle seems to function to protect the muscle, to support recovery from injury and to stimulate satellite cell proliferation following loss of muscle fibres. This layer also plays a role in the inflammatory process and HA is prevalent Smith 2016>>

<< The thickening of the skin causes ischaemia and therefore nociceptive pain via the peripheral nervous system (Holey 1995). In addition to local pain, the presence of increased subcutaneous fluid will cause an alteration in the osmotic pressure in cells. There is retention of sodium and associated excretion of potassium, resulting in water retention that interferes with the neuromuscular conducting mechanism (Ebner 1975). The physiological consequence of this is underlying muscle atrophy, which may then lead to the development of myofascial trigger points, a further source of pain and dysfunction. Multiple literature sources describe the mechanisms of which subcutaneous panniculosis can perpetuate visceral disturbance and that the association between cutaneous dysfunction and visceral disturbance is so high that examination of the skin can be used as a predictor of potentially undiagnosed visceral disease (Korr 1949, Wilson 1956, Grainger 1958, Beal 1985, Tillman & Cummings 1992). (Chaitow 2012 H 12) >>

Tenniselleboog

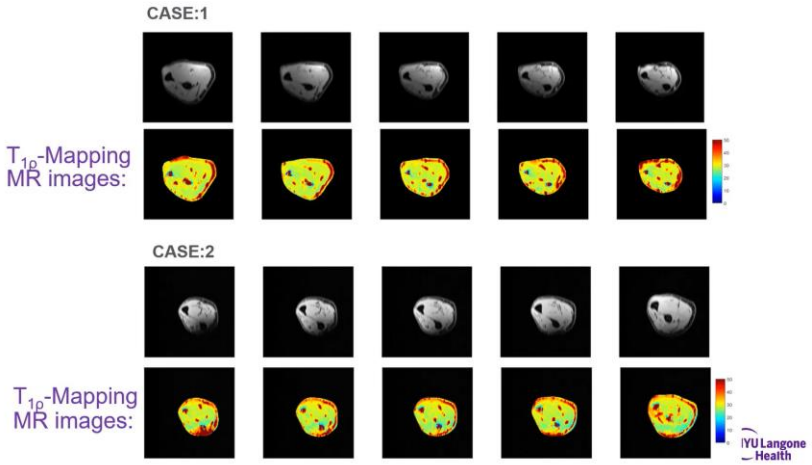


Goede zijde

Aangedane zijde

17

Nieuwe MRI techniek



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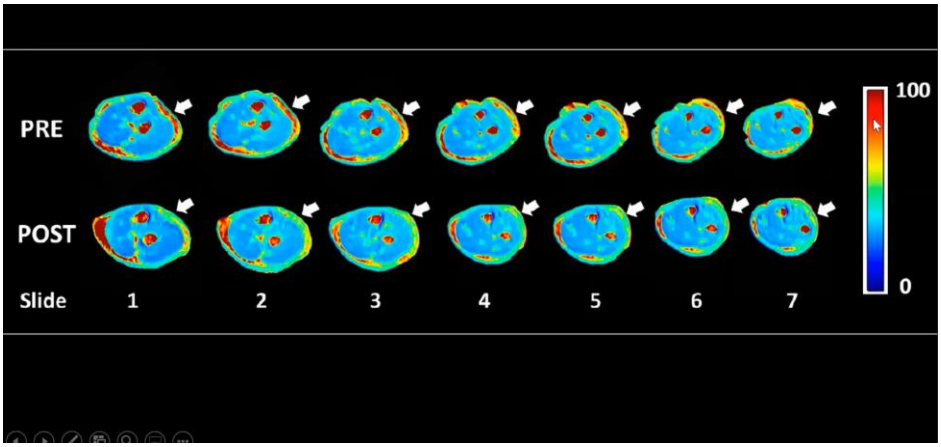
Liquify the fascia



19

Densificaties

TRIGGER POINTS AND FASCIAL DENSIFICATION

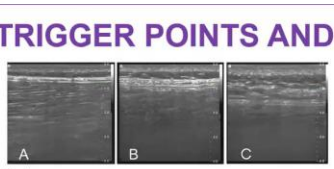
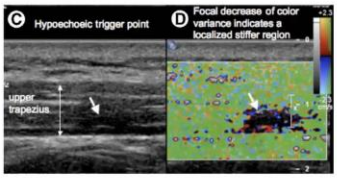


Figure 4
Examples of ultrasound images illustrating thin (A), thick (B) and multilayered (C) perimuscular connective tissue morphology

The LBP group had approximately 25% greater fascia thickness

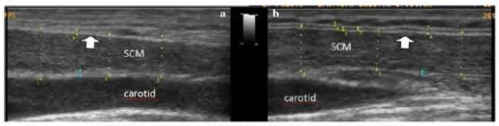
Langevin HM et at 2009-2016

Reducing fascia mobility with a 52% reduction in shear



The hypoechoic and stiffer nodules

Sikdar S et al 2009

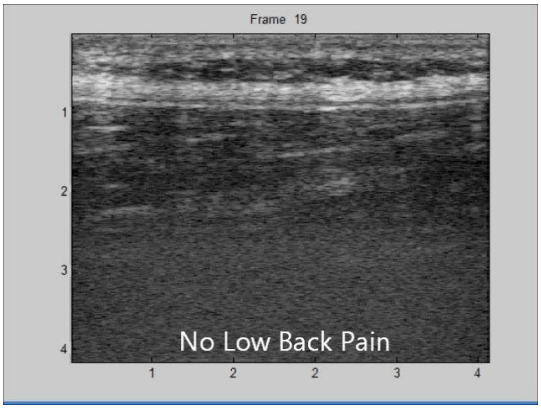


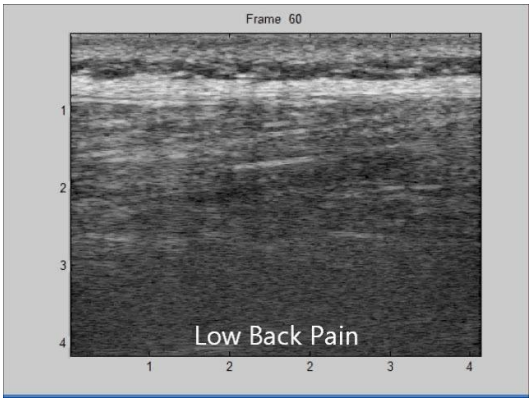
a SCM in control subject (*arrow* fascia). b SCM in a patient (*arrow* fascia)

Chronic neck pain group had a hypoechoic and thicker fascia

Stecco A et al 2013

Research Langevin (2011): Verminderde verschuifbaarheid TLF





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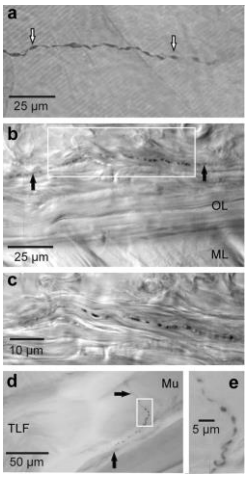
Research Mense, Tesarz, Taguchi

Fascia has 10x more sensory fibers than muscle!

Free nerve endings especially in superficial fascia and superficial layer of deep fascia



(Tesarz 2011)

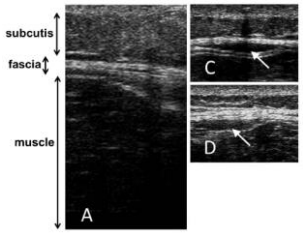
(Mense 2010, Tesarz 2011, Taguchi 2007, Schleip 2012, 2013, Schilder,)

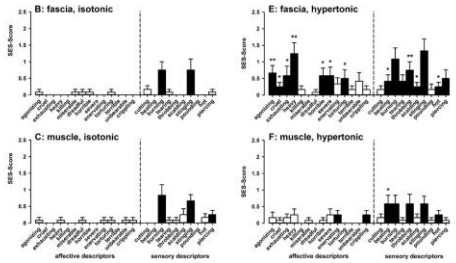
22

<< We found that the proportion of second-order neurons with fascial input from the TLF significantly increased in animals with chronic myositis in the low back. These recent articles suggest not only that the muscle fascia could be a source of nociception in a physiological condition, but also that it could play crucial roles in hyperalgesic conditions (e.g. myofascial pain syndrome). Taguchi in Schleip 2012>>

<<Somatic neuro-fascial interaction A review by Benjamin (2009) describes several studies that showed the presence of primary afferents and nerve fibers within fascia. This innervation is denser in the most superficial layers between the dermis and the deep fascia, and it follows a segmental pattern that resembles the myotome and dermatome distribution (Tesarz et al., 2011), supporting the concept of fascia as a 'sensory organ'. In particular, the presence of mechanoreceptors in fascia suggests a role in dynamic proprioception, force transmission and motor control (Stecco et al., 2007). Research also shows that fascia, rather than muscle tissue, is involved in the delayed onset of muscle soreness following physical exercise, suggesting a role in pain generation in normal physiological conditions (Gibson et al., 2009). Tozzi 2015>>

TLF als bron van lage rugpijn?





(Schilder 2013, 2016)

"These findings show that the thoracolumbar fascia is the deep tissue of the back that is most sensitive to chemical stimulation, making it a prime candidate to contribute to nonspecific LBP."

(Langevin 2007, 2011, Tesarz 2011, Taguchi 2008, Schilder 2013, 2016)

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Schilder A, Hoheisel U, Magerl W, Benrath J, Klein T, Treede RD. Sensory findings after stimulation of the thoracolumbar fascia with hypertonic saline suggest its contribution to low back pain. Pain. 2013 Sep 26. doi:pii: S0304-3959(13)00522-8. 10.1016/j.pain.2013.09.025.

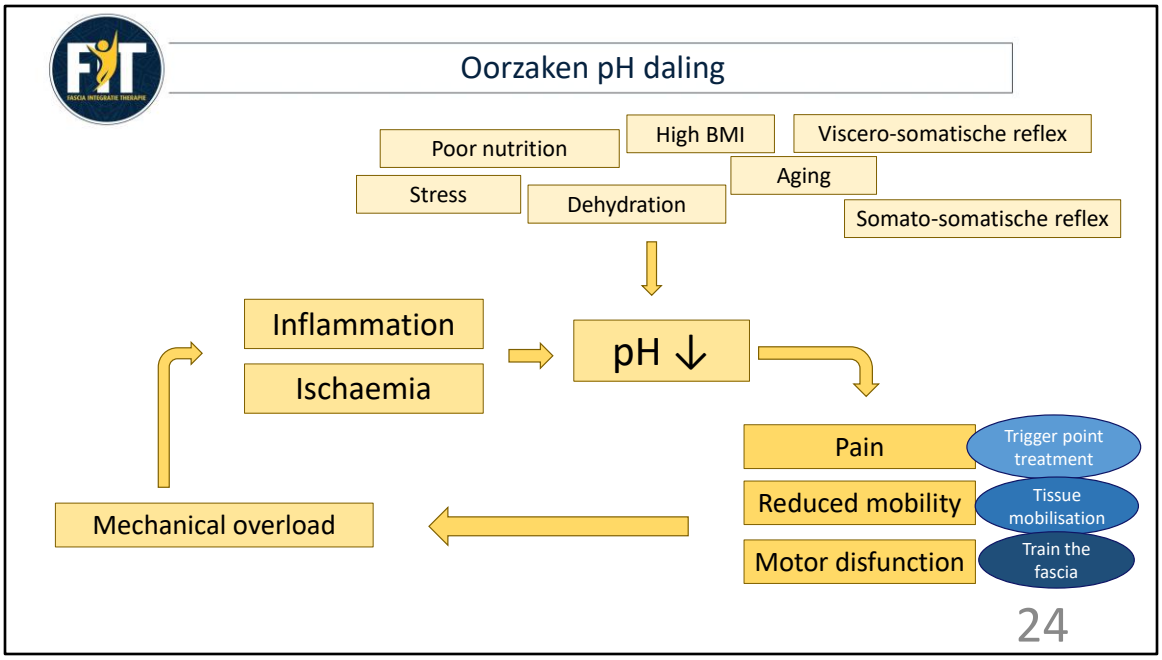
Abstract: Injection of hypertonic saline into deep tissues of the back (subcutis, muscle, or the surrounding fascia) can induce acute low back pain (LBP). So far, no study has analyzed differences in temporal, qualitative, and spatial pain characteristics originating from these tissues. The current study aimed to investigate the role of the thoracolumbar fascia as a potential source of LBP. 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 ($P < 0.001$), primarily based on longer pain durations and, to a lesser extent, on higher peak pain ratings.

Pressure hyperalgesia was only induced by injection of hypertonic saline into muscle, but not fascia or subcutis. Pain radiation and pain affect evoked by fascia injection exceeded those of the muscle ($P < 0.01$) and the subcutis significantly ($P < 0.05$). Pain descriptors after fascia injection (burning, throbbing, and stinging) suggested innervation by both A- and C-fiber nociceptors. These findings show that the thoracolumbar fascia is the deep tissue of the back that is most sensitive to chemical stimulation, making it a prime candidate to contribute to nonspecific LBP but not to localized pressure hyperalgesia.

Most of the neurons with receptive fields in the TLF had convergent input from the skin and other deep tissues or regions in the low back, abdominal wall, hip, and proximal/distal leg. This finding may explain the diffuse nature of nonspecific low back pain in patients.

This research reveales that the TLF is an important source of nociception Bij myositis was er meer nociceptie vanuit de fascia dan uit de spieren. (Hoheisel, in Schleip 2012)>

<< During flexion of the trunk, for example, activation of the abdominal muscles stretch the TLF along a particular direction, while activation of the gluteus maximus and latissimus dorsi stretch the TLF in another direction. It is the presence of various autonomous fibrous planes within the TLF that permit particular muscles to contract without opposition from other muscles that are inserted into the same fascia. If trauma, surgery or an overuse syndrome alters the sliding system within an aponeurotic fascial plane, new lines of force inside this fascia may form. This could adversely affect other muscles, not directly related to the injured plane. This altered activity of muscles could be considered a compensation, and could be present also far from the original problem. The anatomical element that joins the old and new pain is the fascia. Stecco C 2015>>



<<Many muscle disturbances are connected to a reduction in the pH value in the tissue, and a lower pH value (acid environment) is one of the main causes for the chronification of muscle pain. Mense and Gerwin 2010a>>

<<When connective tissue becomes dysfunctional the problems that arise are in proportion to the support the tissues provide when they are healthy. Several mechanisms have been identified to explain how dysfunction develops in the subcutaneous tissue. These are as the result of visceral referred pain, in tissue superficial to myofascial trigger points, in the cutaneous distribution of inflamed peripheral nerves, and superficial to or referred from areas of joint dysfunction (Ebner 1975, Travell & Simons 1993, Beco 2004, Maigne 1996).

Viscerosomatic reflex
One potential mechanism is described because dichotomizing sensory neurons have a branch to both the uterus and to the skin. Uterine inflammation could cause excitation of the visceral branch of the afferent neuron, leading to antidromic activation of the somatic branch causing neurogenic plasma extravasation. Wesselmann also hypothesized that visceral afferent neurons may excite cutaneous afferent neurons. The result of this spinal mechanism is antidromic activation of

cutaneous afferent fibres, again resulting in plasma extravasation. Finally, the paper discussed the possibility that sympathetic post-ganglionic nerve terminals must be intact. This was demonstrated by a decrease in plasma extravasation when the anterior spinal root was not intact (Wesslemann & Lai 1993).

Superficial to muscles with myofascial trigger points
Travell & Simons (1993) have reported a strong association between active myofascial trigger points and subcutaneous connective tissue restrictions.
Dermographia/fibrositis commonly occurs most often over muscles of the back of the neck, shoulders and torso, and less frequently over limb muscles. In panniculosis, the subcutaneous tissue exhibits increased viscosity suggestive of thixotropy. It is proposed by Travell and Simons that the connective tissue restrictions may be related to sympathetic nervous system activity involving mechanisms operating in the underlying myofascial trigger points. Treating the panniculosis can relieve myofascial trigger point activity and/or make the underlying myofascial trigger point more responsive to treatment.

Dermatomes of inflamed neural structures
The 'pinch-roll' test (of the subcutaneous tissue in the territory of a peripheral nerve) is commonly accepted as a clinical indicator of inflamed neural tissue. Referred pain is accompanied by hyperalgesia of the skin and subcutaneous tissues in the involved dermatomes. This hyperalgesia or hypersensitivity can be revealed by gently grasping a fold of skin between the thumbs and forefingers, lifting it away from the trunk and rolling the subcutaneous surfaces against one another in a pinch and roll fashion. The entire dermatome may be affected or only partial tissue changes may be seen (Maigne 1996, Beco 2004). Connective tissue restrictions and altered neural dynamics Ischaemia or thickness associated with subcutaneous panniculosis can compromise neural gliding mechanisms, particularly when the peripheral nerves innervate or transect the dysfunctional tissue region (Butler 2004).

The thickening of the skin causes ischaemia and therefore nociceptive pain via the peripheral nervous system (Holey 1995). In addition to local pain, the presence of increased subcutaneous fluid will cause an alteration in the osmotic pressure in cells. There is retention of sodium and associated excretion of potassium, resulting in water retention that interferes with the neuromuscular conducting mechanism (Ebner 1975). The physiological consequence of this is underlying muscle atrophy, which may then lead to the development of myofascial trigger points, a further source of pain and dysfunction. Multiple literature sources describe the mechanisms of which subcutaneous panniculosis can perpetuate visceral disturbance and that the association between cutaneous dysfunction and visceral disturbance is so high that examination oft he skin can be used as apredictor of potentially undiagnosed visceral disease (Korr 1949, Wilson 1956, Grainger 1958, Beal 1985, Tillman & Cummings

1992). Chaitow L 2012 H 12>>

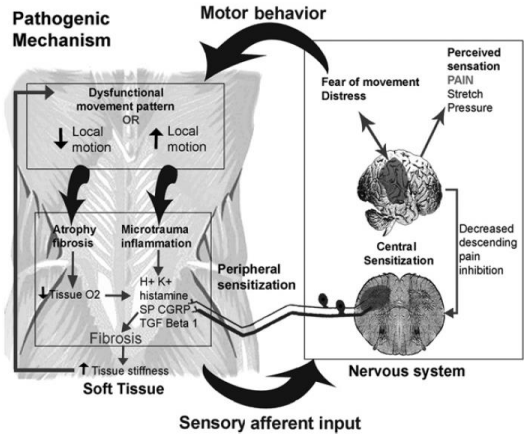
<<Body fat, or adipose tissue, is inflammatory. About 60% of the cells in adipose tissue are macrophages, the robocops of the immune system, and one of the principal sources of inflammatory cytokines. Overweight or obese people, with a higher body mass index, will generally have higher blood levels of cytokines and CRP than slimmer people. (Das UN 2001)
Or obesity could cause depression immunologically by increasing the total number of macrophages in the body and increasing the levels of cytokines in the blood. However, it is at least clear that obesity both causes inflammation and increases the risk of depression.
Bullmore 2019>>

<<When we examine the conditions that can cause the pH level to drop below 6.5. we can see that this is often related to the nutritional habits of people in the industrialized world. Acid-producing compounds are especially common in animal proteins, sugar (including sugar-containing fluids and canned foods), alcohol, coffee, and black tea. Stress and smoking may also have an acidic influence on the pH level. By contrast, nutrients with an alkaline pH can be found in fruits and vegetables. When one examines the average nutritional habits of patients in the industrialized world, it is evident that acidic nutrients are consumed in far greater quantities then alkaline nutrients (van den Berg. 2011; Vormann et al., 2001; Birklein et al„ 2000; Woo et al., 2004: Finlayson et al.. 1964; Bibby et al, 2005; Bibby & Urban. 2004; Murphy & McPhee, 1965; Kellum et al.. 2004). Vd Berg 2017 ch 9>>

<< pH can be influenced by, among other things, a

dysfunctional breathing pattern (Chaitow et al., 2014) and nutrition (Arent et al., 2010)>>

Voorbeeld Thoraco Lumbar Fascia



(Langevin 2007)

Figure 1 Pathophysiological model for chronic low back pain linking somatic and behavioral components.

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<<However, under abnormal mechanical stimulation, a pathological change in fascial innervation may occur, resulting in dysfunctional ingrowth of nociceptive fibers (Sanchis-Alfonso and Rosello-Sastre, 2000) that generates or maintains inflammation (Herbert and Holzer, 2002). Even the epineurium and perineurium are innervated by nervi nervorum, which can in turn cause neurogenic inflammation and consequently evoke dysesthetic (distal) and nerve trunk (local) pain when a mechanical stimulus is applied along the nerve path (Bove, 2008). In addition, evidence suggests that nociceptive input rising from the thoracolumbar fascia may contribute to pain in non-specific low back pain (although not to localized pressure hyperalgesia) (Schilder et al., 2014). This may also produce changes in the corresponding spinal area. Hoheisel and Mense (2014) noted that following induced inflammation in the thoracolumbar fascia, an expansion of the spinal target region of fascia afferents has been demonstrated, together with the appearance of new receptive fields that could explain the spread of pain in patients with non-specific low back pain. Therefore, irritation of primary afferent fibers in the fascia is capable of initiating the release of neuropeptides, eventually setting up a neurogenic inflammation, with peripheral (Deising et al., 2012) and central sensitization, and altering the texture of surrounding connective tissue via the interaction of fibroblast and immune cells (Mense, 2001). This process may trigger a cascade of either local or global responses: chronic pain

and connective tissue remodeling (Langevin and Sherman, 2007), altered mechanoreceptor feedback and muscle control (Panjabi, 2006), followed by further connective tissue alterations, neural adaptation, and eventually cortical reorganization (Flor, 2003). This process may expand to include influences on endocrine and autonomic pathways (Benarroch, 2006), as well as on sensory, cognitive and affective areas of the brain, that may in turn respond to control pain (Peyron et al., 2000). Recent studies have also shown that myofascial pain may alter the activity of related higher centres accounting for a reduction of sensory processing, followed by an altered motor output (Schabrun et al., 2013), together with a reorganization of the motor cortex associated with deficits in postural control (Tsao et al., 2008). Such evidence may explain several local, segmental and global effects of a given fascial dysfunction, proposing fascia as a possible nociceptive source for the establishment of somatic dysfunction and of its features, such as tenderness and tissue texture changes. Tozzi 2015>>

<< Do emotional states affect overall strategy for function and task performance? Studies suggest that emotional states do play a significant role in human function, and are often reflected in the musculoskeletal system (Hodges & Moseley 2003, Moseley & Hodges 2005). In addition to their functional complaints, many patients with pain present with symptoms similar to those seen in individuals who have experienced traumatic events. Negative emotional states such as fear, anxiety, and insecurity can express themselves in maladaptive defensive, or aggressive, postures that correlate with altered muscle activity and further strain on the musculoskeletal system.
Clinically, it appears that if an individual does not have the coping mechanisms necessary to confront their symptoms, they learn to avoid activities that result in pain (Vlaeyen & Linton 2000). This avoidance can persist due to their fear of re-injury or an underlying belief that they are unable to perform because of their condition (fear-avoidance). The muscles of the region can reflect this fear and can become hypertonic, thereby increasing force closure that subsequently results in sustained excessive compression of the LPH complex. This can perpetuate pain. Furthermore, emotional states can contribute to peripheral and/or central sensitization of the nervous system (Butler 2000, Butler & Moseley 2003, Moseley & Hodges 2005), which in turn can create substantial barriers to rehabilitation.

Langevin HM, Sherman KJ. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. Med Hypotheses.2007 ;68(1):74-80. Epub 2006 Aug 21.
Summary Although chronic low back pain (cLBP) is increasingly recognized as a complex syndrome with multifactorial etiology, the pathogenic mechanisms leading to the development of chronic pain in this condition remain poorly understood. This article presents a new, testable pathophysiological model integrating connective

tissue plasticity mechanisms with several well-developed areas of research on cLBP (pain psychology, postural control, neuroplasticity). We hypothesize that pain-related fear leads to a cycle of decreased movement, connective tissue remodeling, inflammation, nervous system sensitization and further decreased mobility. In addition to providing a new, testable framework for future mechanistic studies of cLBP, the integration of connective tissue and nervous system plasticity into the model will potentially illuminate the mechanisms of a variety of treatments that may reverse these abnormalities by applying mechanical forces to soft tissues (e.g. physical therapy, massage, chiropractic manipulation, acupuncture), by changing specific movement patterns (e.g. movement therapies, yoga) or more generally by increasing activity levels (e.g. recreational exercise). Non-invasive measures of connective tissue remodeling may eventually become important tools to evaluate and follow patients with cLBP in research and clinical practice. An integrative mechanistic model incorporating behavioral and structural aspects of cLBP will strengthen the rationale for a multidisciplinary treatment approach including direct mechanical tissue stimulation, movement reeducation, psychosocial intervention and pharmacological treatment to address this common and debilitating condition.

<<The human lumbar fascia as potential generator of low back pain Panjabi's new explanatory model of low back pain injuries suggests that a single trauma or cumulative microtrauma causes subfailure injuries of paraspinal connective tissues and their embedded mechanoreceptors, thereby leading to corrupted mechanoreceptor feedback and resulting in further connective tissue alterations and neural adaptations (Panjabi 2006). Our group subsequently proposed an extension of that model which includes the posterior layer of the lumbar fascia as a potential focus of such microtrauma and resulting muscle control dysfunction. Factors raised in support of that explanation include the long distance of this layer from the axis of spinal flexion (Fig. 1) as well as its lesser stiffness compared with spinal ligaments (Schleip et al. 2007).

Langevin reports that the posterior layer of the lumbar fascia tends to be thicker in chronic low back pain patients. In addition it expresses less shear motion during passive trunk flexion (Langevin et al. 2009). Surgical examinations of the posterior layer of the lumbar fascia revealed frequent signs of injury (Bednar et al. 1995) and of inflammation (Dittrich 1963, Dittrich 1964) in low back pain patients. In addition new histological examinations at our laboratory have shown a high density of myofibroblasts, the existence of which is usually associated with excessive loading or injury repair – in the same fascial layer (Schleip et al. 2007). And finally, injection of the inflammatory agent Freund Adjuvans solution into the rat lumbar muscles resulted in a dramatic increase in the proportion of dorsal horn neurons with input from the posterior lumbar fascia (Taguchi et al. 2009).

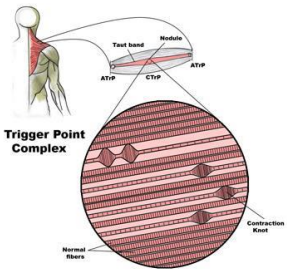
Conclusions
Fascial tissues serve important load-bearing functions. Severe tensional loading can induce temporary viscoelastic deformation and even microtearing. The innervation of fascia indicates a potential nociceptive function. Microtearing and/or inflammation of fascia can be a direct source of musculoskeletal pain. In addition, fascia may be an indirect source of physical problems such as back pain due to a sensitization of fascial nerve endings associated with inflammatory processes in other tissues within the same segment.
Stecco 2013>>

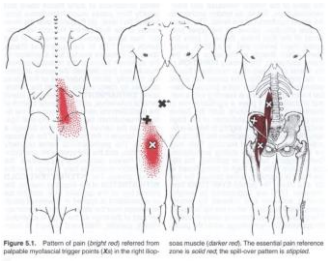
Perhaps a clinical realisation arising from this is that not only are ergonomics key, but also paying attention to other causes of creep on the ligamentous system, such as the hypnotic effect of computer and TV screens, the sedative effects of alcohol consumption or of chronic sleep deprivation; potentially switching the body off from its own mechanoreceptive feedback, may offer greater understanding in preventing low back injury. (Matt Wallden 2010)
, 2011>>

Wat is een myofasciaal trigger point?

Een sterk drukpijnlijke plaats in een harde streng in een skeletspier.

Met uitstraling naar een specifieke ‘referral zone’





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<< The most commonly accepted definition describes a TrP as a hyperirritable spot in a taut band of a skeletal muscle, which is painful on compression, stretch, overload, or contraction of the muscle and usually has a distinct referred pain pattern (Simons and Travell, 1999).

Referred pain, the most characteristic sign of a trigger point (TrP), is a central phenomenon initiated and activated by peripheral sensitization, whereby the peripheral nociceptive input from the muscle can sensitize dorsal horn neurons that were previously silent. TrP are a peripheral
source of nociception, and act as ongoing nociceptive stimuli contributing to pain propagation and widespread pain. Several studies support the hypothesis that TrP can induce central sensitization, and appropriate TrP treatment reduces central sensitization. In contrast, preliminary evidence suggests that central sensitization can also promote TrP activity, although further studies are needed. Proper TrP management may prevent and reverse the development of pain propagation in chronic pain conditions, because inactivation of TrP attenuates central sensitization.

Myofascial trigger points (TrP) are one of the most overlooked and ignored causes of musculoskeletal pain. There is evidence suggesting that TrP are a common primary

dysfunction and not necessarily secondary to other diagnoses (Mense, 2010). In other words, TrP may occur in the absence of any underlying medical condition and can constitute an independent cause of pain. TrP can, however, also be co-morbid with a variety of medical musculoskeletal conditions, including osteoarthritis of the hip or knee (Bajaj, 2001), or with visceral conditions, for example endometriosis (Jarell, 2011), interstitial cystitis (Weis, 2001), irritable bowel syndrome, dysmenorrhea, or prostatitis (Doggweiler-Wigul, 2004).

Although latent TrP do not induce spontaneous pain, they can provoke motor dysfunction, e.g. muscle weakness, inhibition, increased motor irritability (Ibarra et al, 2011), muscle cramps (Ge et al, 2008), and altered motor recruitment (Lucas et al, 2010). During the past decade, an increasing number of researchers have shown an interest in the etiology and clinical relevance of latent TrP (Ge et al, 2011).

TrP are located within discrete bands of contractured muscle fibers called taut bands. A taut band signifies a contracture arising endogenously within a limited number of muscle fibers, but not involving the entire muscle (Simons and Travell, 1999). Studies have observed that taut bands have higher stiffness (Chen et al, 2007), reduced vibration amplitude (Sikdar et al, 2009), higher peak systolic velocities, and negative diastolic velocities (Sikdar et al, 2010)] compared with normal muscle sites.

Although different theories have been proposed, the integrated hypothesis is the most accepted model for explaining the pathophysiology of muscle TrP (Bron et al, 2012). In summary, the integrated hypothesis proposes that abnormal depolarization of the post-junctional membrane of motor endplates causes a localized hypoxic energy crisis associated with sensory and autonomic reflex arcs that are sustained by complex sensitization mechanisms. The presence of spontaneous electrical activity or endplate noise, and the clinical evidence that treating TrP eliminates or significantly reduces this endplate noise, support the notion that TrP are located in close proximity to dysfunctional motor endplates (Ge, 2011). A recent study reported that TrP in the upper trapezius muscle are located in well-defined areas proximal to innervation muscle zones (Barbero, 2013). Although current evidence supports the hypothesis that TrP are associated with dysfunctional motor endplates, the function of muscle spindles in the etiology of TrP has been also investigated (Ge et al, 2009, Partanen, 1999, Partanen, 2010) .

Because the intensity of referred pain and the size of the referred pain area are positively correlated with central nervous system excitability, it is now generally assumed that muscle referred pain is a central sensitization process mediated by a peripheral sensitization phenomenon, with additional sympathetic activity facilitation and dysfunctional descending pain inhibition (Arendt-Nielsen, 2001). Hoheisel et al. reported that new receptive fields emerged within minutes after experience of

noxious stimuli (Hoheisel, 1993), explaining the delay between the noxious input and development of referred pain (Graven-Nielsen, 1997). In clinical practice referred pain appears within seconds of stimulation of a TrP, suggesting that induction of these axonal changes is a rapid process. TrP are more effective than non-TrP regions at inducing neuroplastic changes in the dorsal horn neurons (Kuan, 2007).

Maintenance of referred pain is dependent on ongoing nociceptive input from the site of primary muscle pain (Arendt-Nielsen, 2001). There is currently insufficient data to determine which sensitization mechanism, peripheral or central, is more relevant to the development of referred pain.

Studies conducted by Shah et al. revealed that the concentrations of bradykinin (BK), calcitonin gene-related peptide (CGRP), substance P, tumor necrosis factor-α (TNF-α), interleukins 1β, IL-6, and IL-8, serotonin (5-HT), and norepinephrine were significantly higher at active TrP than at latent TrP or non-TrP points (Shah, 2005). Interestingly, concentrations of the same biochemical and algogenic substances in a pain-free area of the gastrocnemius muscle were also higher for subjects with active TrP in the upper trapezius muscle compared with subjects with latent TrP or non-TrP points (Shah, 2008). More recently, Hsieh et al. confirmed the presence of multiple biochemical substances in the immediate proximity of TrP (Hsieh, 2012). Both the studies by Shah et al. (Shah, 2005, 2008) and Hsieh et al. (Hsieh, 2012) provided evidence that therapeutic intervention with dry needling could modulate and normalize the chemical environment of TrP, supporting the theory that TrP may be a source of peripheral nociceptive input.

Li et al. observed nociceptive (hyperalgesia) and nonnociceptive (allodynia) hypersensitivity at TrP, suggesting that TrP sensitize nociceptive and non-nociceptive nerve endings (Li, 2009). Wang et al. reported that blocking large-diameter myelinated muscle afferents increased pressure pain and referred pain thresholds at TrP, but not at non-TrP regions, suggesting that non-nociceptive large-diameter myelinated muscle afferents are also involved in TrP pain (Wang, 2010). These studies establish the presence of nociceptive pain hypersensitivity at TrP and confirm that TrP are a focus of peripheral sensitization.

Mense suggested that, because TrP constitute a continued peripheral nociceptive afferent barrage into the central nervous system, the presence of multiple TrP in the same or different muscles or the presence of TrP for prolonged periods of time can sensitize spinal cord neurons and supra-spinal structures (Mense, 1994).

Xu et al. reported that painful stimulation of latent TrP induced central sensitization in healthy subjects, because stimulation of TrP increased pressure hypersensitivity of extra-segmental tissues (Xu, 2010). A few studies observed that stimulation of TrP

induced enhanced activity of brain areas including the primary and secondary somatosensory cortex, the inferior parietal cortex, and the mid and anterior insula (Niddam 2008, 2009), supporting the hypothesis that TrP can induce central sensitization.

Anesthetic injections into active TrP significantly reduced mechanical hyperalgesia, allodynia, and referred pain for individuals with migraine (Giamberardino, 2007), fibromyalgia (Affaitati, 2011), and whiplash (Freeman, 2009).

There are, however, several studies revealing that asymptomatic healthy subjects also have TrP—because the subjects are pain free, these would be classified as latent TrP, (Ibarra 2011, Lucas 2010, Ge 2008 2009 2011, Wang 2010 2012, Jarell 2011, Bajaj 2001, Fernandez-de-las-Penas 2007 2009 2010, Fernandez-Carnero 2007, Bron 2011, Barbero 2013). Latent TrP are not spontaneously painful, but they do provide nociceptive barrage into the dorsal horn.(Ibarra 2011, Lucas 2010, Ge 2008 2009 2011, Wang 2010 2012). Although there is no evidence supporting the hypothesis that TrP are a result of central sensitization, in clinical practice individuals with higher levels of central sensitization present with multiple TrP.

Depending on the chronicity of the disorder and the associated disability, patients should receive pain neuroscience education addressing the neurobiology of pain and pain mechanisms, fear, anxiety, and other psychosocial variables (Puentedura 2012). Patients need to develop different strategies for optimizing normal functional movement and to undertake active and specific or more global exercises, including aerobic exercise.

Inactivation of TrP is associated with attenuation of central sensitization (Giamberardino 2007, Affaitati 2011, Freeman 2009) and induction of spinal inhibition (Srbely 2008 2010) Fernandez-de-las-Penas 2014>>

<< Taut band 50-100% stiffer than surrounding muscle tissue. Chen 2008>>

Both latent and active TrP's lead to documented referred pain:
Travell and Simons 1999, Gautschi 2019, Donnelly 2019, Ballantyre 2010, Mense 1994, Fernandez-Carnero 2007, Fernandez-de-las-Penas 2014, Hong 1997, Farasyn 2007, Rubin 2010, Gibson 2006, Vecchiet 1993, 1997, 1999, Rubin 2012, Hooshmand 1993, Hwang 2005, Jaeger 1985, Kleier 1985, Koelbaek 1999, Fernandez-de-las-Penas 2007, 2010, 2012, Giamberardino 1999, Graven-Nielsen 1997, Hong 1996, Alonso-Blaco 2012, Choi 2015, Xu 2010, Zhang 2009, Ge 2011, 2009, Li 2009

"Myofascial trigger points are the most commonly overlooked cause of chronic pain." (Prof. David G. Simons)

Myofascial pain has been described as the most common diagnosis responsible for chronic pain and disability, but it is also considered the most commonly missed diagnosis. (Hendler 1993, Fricton 1990) This may be due in part to the fact that physical therapy literature rarely includes reference to trigger points.

Because some patients perceive that clinicians do not take pain complaints seriously, they may be surprised when a clinician elicits a familiar pain complaint by compressing myofascial trigger points. Validating a pain complaint in this manner can be an important step in establishing a therapeutic relationship.

Estimations of the prevalence of trigger points with chronic pain patients tranges between 55% and 95%.

<<Myofascial pain is the most common component of musculoskeletal pain conditions. As a form of muscle pain, myofascial pain can often be described as aching, cramping, deep, and difficult to localize. Muscle pain is distinguished from cutaneous pain in that muscle pain involves nociceptive-specific neurons in the brainstem and spinal cord (Sessle 2000, Arendt-Nielsen 2002). In addition, muscle pain activates unique cortical areas that are associated with affective or emotional components of pain (Svensson 1997). Although muscle nociception is inhibited more intensely by descending pain-modulating pathways (XianMin 1990, Fields 1999), persistent muscle nociception is more effective than cutaneous nociception at inducing maladaptive neuroplastic changes within the dorsal horn (Wall 1984). Such neuroplastic changes underlie the clinical observation that muscle pain is often persistent and difficult to resolve.

Myofascial pain syndrome (MPS) is a term used to describe a pain condition that can be acute (less than three months in duration) or, more commonly, chronic and that stems from the muscle and its surrounding connective tissue (e.g., fascia). Although the specific pathophysiological basis of myofascial trigger point (MTrP) development and symptomatology is unknown, several promising lines of scientific study (e.g.. biochemical, tissue imaging, and somatosensory testing) as well as a recent systematic and comprehensive evaluative approach (including measures of range of motion, strength, and self-reports of pain, fatigue, mood, and health status) have revealed objective abnormalities. For many clinicians and investigators, the finding of one or more MTrPs is required to assure the diagnosis of MPS.
The contemporary use of the term 'MPS' implies a specific condition that is distinguished from other soft tissue pain disorders such as fibromyalgia, tendonitis, or bursitis (Robert 2007). It presents as regional pain, sometimes with referred pain, often accompanied by increased tension and decreased flexibility. It has been reported to coincide with other diseases and syndromes associated with pain, for

example, rheumatic diseases and fibromyalgia (Borg-Stein 2002). MPS has also been associated with other pain conditions including radiculopathies, joint dysfunction, disk pathology, tendonitis, cranio-mandibular dysfunction, migraines, tension-type headaches, carpal tunnel syndrome, computer-related disorders, whiplash-associated disorders, spinal dysfunction, pelvic pain and other urologic syndromes, post-herpetic neuralgia, and complex regional pain syndrome (Borg-Stein 2002). In addition, MTrPs have been associated clinically with a variety of medical conditions including those of metabolic, visceral, endocrine, infectious, and psychological origin (Hong 1996). Shah in Liem 2017>>

<<A medical approach to treat chronic pain of the neuromusculoskeletal system that does not appropriately incorporate the widespread phenomenon of referred pain into its diagnostic and therapeutic strategy is often doomed to failure and serves just to foster chronification of the pain.

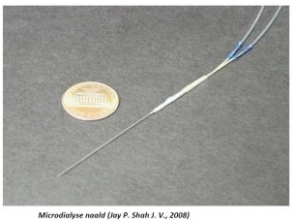
"The trigger point, as we currently define it, is certainly the most common manifestation of pain in the locomotor system, if not in the entire organism." (Prof. Karl Lewit in the Introduction to Dejung 2009)

In one study of patients examined in specialized pain centers, active mTrPs were found in 85% of 283 patients (Fishbain 1986), and in another, 93% of 96 patients (Gerwin 1995). In a study of 296 patients in a dental clinic specializing in head and neck pain, Friction (1990) determined that the pain had a primary muscular cause in 55% of the patients.
On the basis of this data, Travell and Simons concluded that: "Active myofascial trigger points are clearly very common and are a major source of musculoskeletal pain and dysfunction" (Travell and Simons 1999). This assessment has been corroborated by multiple studies showing that mTrPs very commonly play a role in patients with many types of problems, including: tension headaches (Alonso-Blanco et al. 2012a, Bendtsen et al. 2011, Buchmann et al. 2007, Couppé et al. 2007, Fernandez-de-las-Penas et al. 2006b, 2006c, 2009a, and 2010, von Stülpnagel et al. 2009); migraine (Buchmann et al. 2008, Calandre et al. 2006, Fernandez-de-las-Penas 2006d, Giamberardino et al. 2007, Tali et al. 2014); neck pain (Fernandez-de-las-Penas et al. 2007, Gerber et al. 2014, Munoz-Munoz et al. 2012, Vazquez-Delgado et al. 2009); problems after whiplash injury (Castaldo et al. 2014, Dommerholt et al. 2015, Ettlin et al. 2008, Freeman et al. 2009); nonspecific back pain (Borg-Stein et al. 2006, Chen and Nizar 2011, Iglesias-Gonzales et al. 2013, Nice et al. 1992, Nioo and van der Does 1994, Simons et al. 1983); nonspecific neck, shoulder, and arm pain (Fernandez-de-las-Penas et al. 2012), shoulder pain (Buchmann et al. 2009, Bron 2011, Bron et al. 2011, Ge et al. 2007, Hains et al. 2010a, Hidalgo-Lozano et al. 2013, Paz et al. 2014, Sergienko and Kalichman 2015, Sola et al. 1955) and shoulder pain associated with subacromial impingement (Alburquerque-Sendín 2013, Hidalgo-

Lozano et al. 2010); lateral elbow pain (Fernandez-Carnero et al. 2007, 2008, Fernandez-de-las-Penas 2012, Gonzalez-Iglesias et al. 2011, Shmushkevich and Kalichman 2013); forearm and hand pain (Hwang et al. 2005); posture and stress-related pains associated with computer use (Treaster et al. 2006); knee pain (Henry et al. 2012, Mayoral et al. 2013); temporomandibular joint (Ardic et al. 2006, Fernández-Carnero et al. 2010, Itoh et al. 2012, Vazquez et al. 2010); tinnitus (Rocha and Sanchez 2007); and pains in the bladder and urogenital region (Anderson et al. 2006, 2009, 2011, Doggweiler-Wiygul 2004, FitzGerald et al. 2009, Jarrell 2004, Weiss 2001). Gautschi 2019>>

Biochemisch milieu TrP

Microdialyse:
(dr. Jay Shah, 2005, 2008)



Verstoord biochemisch milieu

hoge concentraties: protonen (lage pH), bradykinine, kaliumionen, substance P, etc.

Ontsteking

- Sensitisatie (perifeer – centraal)
- Fascia veranderingen: verklevingen

Chronisch probleem!

27

<<The concentration of the hydrogen ion (H^+) has a pronounced influence on the sensitization of muscle nociceptors. The higher the H^+ion concentration (i.e., the lower the pH value), the stronger the reaction (Mense 2013). Many muscle disturbances are connected to a reduction in the pH value in the tissue, and a lower pH value (acid environment) is one of the main causes for the chronification of muscle pain (Mense and Gerwin 2010a). A low pH value is detected in the vicinity of mTrPs (Shah et al. 2005, 2008b).

Nociceptor Density
In animal experiments, muscle inflammation of 12-days duration leads to a marked increase in the innervation density of nerve endings containing substance P (Mense et al. 2001). Because many of the substance P–containing nerve endings are nociceptors, this means that there was an increase in the innervation density of nociception-sensitive structures. A noxious stimulus in the muscle activates in such a situation more nociceptive endings and thereby triggers increased pain. Gautschi 2019>>

<<The active trigger point has identifiable pathophysiologic changes. Levels of substance P, calcitonin gene related peptide, bradykinin, and assorted cytokines, are

elevated, indicating a chemical inflammation. Trigger point milieu pH is low, about pH 5, consistent with hypoxia and ischemia. Persistent, low-amplitude, high frequency electrical discharges that look like endplate potentials are characteristic. The taut band can be visualized using high definition ultrasonography and magnetic resonance sonography. Central sensitization in MPS has been documented in humans by functional magnetic resonance image scanning.
Gerwin R 2005>>

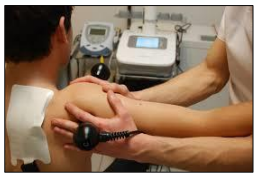
<<One mechanism of central sensitization is the release of substance P together with glutamate from presynaptic terminals of nociceptive fibers from muscle. One important characteristic of a latent TrP is that it does not elicit spontaneous pain but evokes referred pain. Myofascial TrP's are not merely a peripheral phenomenon, the input from TrP's leads to hyperexcitability of central neurons that manifests itself in allodynia, hyperalgesia, and pain referral. These central changes are mainly based on an increase in the synaptic efficacy of central connections induced by nociceptive input. (How do muscle lesions such as active and active trigger points influence central nociceptive neurons? Mense S 2010>>

<<Interestingly, concentrations of the same biochemical and algogenic substances in a pain-free area of the gastrocnemius muscle were also higher for subjects with active TrP in the upper trapezius muscle compared with subjects with latent TrP or non-TrP points (Shah, 2008). More recently, Hsieh et al. confirmed the presence of multiple biochemical substances in the immediate proximity of TrP (Hsieh, 2012). Both the studies by Shah et al. (Shah, 2005, 2008) and Hsieh et al. (Hsieh, 2012) provided evidence that therapeutic intervention with dry needling could modulate and normalize the chemical environment of TrP, supporting the theory that TrP may be a source of peripheral nociceptive input.
Fernandez-de-las-Penas 2014>>

‘liquify the fascia’

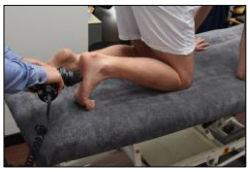
Roman 2013:

Warmte



Verschuivingen

Vibratie



Melt and Move!

Increase tissue flow by
movement, stretching, breathing

28

Roman et al. (2013), in their detailed investigation and mathematical modeling of fascia's gliding processes, note that: The fluid pressure of hyaluronic acid (HA) increases substantially as fascia is deformed during manual therapies.

Therapy - ‘Liquify the fascia’:

- Vibration
- Warmth
- Shearing forces

Hands off if possible, hands-on if necessary. Thickened paint can be liquefied again by stirring, but if there are lumps in the paint you must use your hands to dissolve the lumps. In many cases, movement is sufficient to get the ground substance fluid again, but with local adhaesions you sometimes have to use your hands.

<<It has been found that the lack of sliding between layers of fascia might be due to molecular entanglement of HA molecules resulting in increased viscosity and that increased temperature is necessary to restore normal HA fluidity Piehl-Aulin 1991>>

<< An increase of several degrees Celsius, e.g., in limbs from roughly 33°C to 39°C, leads to significantly higher viscoelasticity of fascia. In this case, muscle is less limited by fascial resistance and range of motion is increased. Hence, in most situations, this resembles a gain of function during exercise. At lower temperatures, i.e., at rest, the viscoelastic properties are adapted to serve stabilization and load-bearing function. Klingler in Schleip 2012 H7.18>>

<<If the resulting colloid is slowly stirred with a stick or spoon, movement will be smooth, but any attempt to move it rapidly will be met with a semi-rigid resistance (known as 'drag'). This quality of colloids is known as thixotropy - most evident in the extracellular matrix. The thixotropic property of colloids means that the more rapidly force is applied (load), the more rigidly will the tissue respond - hence the likelihood of fracture when rapid force meets the resistance of bone. If force is gradually applied, 'energy' is absorbed by, and stored in, the tissues, with potential therapeutic implications (Binkley & Peat 1986). Chaitow 2014>>

<<The viscoelastic properties of fascia have been observed in numerous studies which have analysed

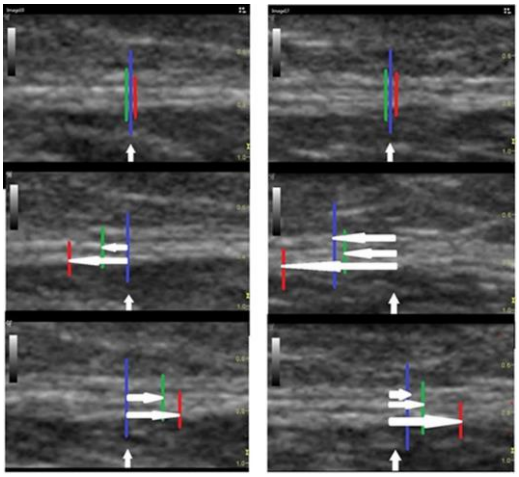
different structures: thoracolumbar fascia (Yahia et al. 1993), fascia lata (Wright & Rennels 1964), subcutaneous fascia of rats (Iatridies et al. 2003), plantar and nasal fascia (Chaudhry 2007).
The clinical utilization of the viscoelastic properties of fascia has been described by various authors: Rolf (1977), Barnes (1990), Threlkeld (1992), Cantu & Grodin (2001), Barnes (1997), Pilat (2003), Schleip et al. (2005), Pilat (2009). Recent theories hypothesized that different chemical mediators may be involved in this process (Vaticón 2009), although further research is clearly needed. Chaitow 2012 H11>>

<<A (therapeutic) stretch lasting for a few minutes could then potentially un-restrain the matrix and promote transcapillary fluid flow and temporary matrix swelling. Fibroblasts, in turn, can either release their matrix contacts resulting in a further drop of interstitial fluid pressure or remodel the contractile cytoskeleton and adhesive matrix contacts, so as to develop a counter-tension sufficient to restore tension equilibrium (Langevin et al., 2013). This model would also fit with the fascial hydrodynamic response reported by Schleip et al. (2012). In response to mechanical stimuli, such as compression and stretch, fascia may exhibit a sponge-like behaviour, showing a squeezing and refilling response under the opposing forces of the restraint of collagen network and the osmotic pull of proteoglycans complex. Interestingly, the fluid pressure might increase more during tangential oscillation (2-4 Hz) and perpendicular vibration (15-60 Hz) with respect to the fascial layer than during constant sliding or back-and-forth motion, as predicted by 3D mathematical modelling methods (Roman et al., 2013; Chaudhry et al., 2013). This would cause the flow to occur more around the edges of the area under manipulation - due to an increased pressure gradient - producing an enhanced lubrication and an improved sliding potential between fascial layers and muscle tissue. Thus, the use of vibratory and oscillatory techniques and not just constant sliding motions should be considered, especially when interstitial fluid dynamics need to be improved such as in

the case of fibrotic tissue.>>

<<In tissue conditions associated with pathological water content, such as inflammatory processes, edema, or the increased accumulation of free radicals, squeezing the tissue can result in adequate rehydration. Klingler 2017>>

Toename mobiliteit



(Luomala 2013)

<<

- Importantly, Chaudhry and colleagues (2007) note that in order to achieve a viscoelastic deformation (such as stretch) without causing tissue damage, there should be no slow increase in the applied force. Instead, a fairly constant force should be maintained, for up to 60 seconds, in order to allow for a plastic stress-relaxation response of the tissues. (Chaiot 2014)
- Palpated change may relate to enhanced sliding facility of associated superficial fascial layers, which had been reduced or lost following inflammation or trauma (Chaitow 2014)
- The ECM is responsive to mechanical strain and tensional loading (tissue deformation). Mechanical forces exert influence on the tissue structural elements (microfilaments) and the molecular composition of the ECM (Benjamin & Ralphs 1998, Milz et al. 2005). Strain type, degree, direction and duration can influence ECM composition and impact fibroblast functions that guide healing and adaptation responses (Purslow2002, Ingber 2003, Standley & Meltzer 2008, Stecco et al. 2009, Blechschmidt & Gasser)
- It is suggested that alterations in HA amount and organization may play a role in tissue changes (e.g. tissue softening and improved slide/glide) following fascial manipulations (Evanko & Wight 1999). ??>>

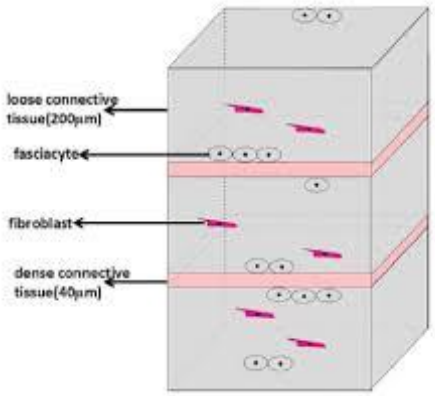
<<Sustained combinations of compressive force involving different vectors - for example, pressure, torsion, shear - that engage tension against restriction barriers, deep in soft tissues (adhesions, fibrosis, scarring) have been shown in elastography images to result in relative structural and functional normalization (Martinez Rodriguez et al. 2013, Borgini et al. 2010). Chaitow in Chaitow 2014 H5>>

<<Whenever a cc (centrum of coordination) present muscle contracture and fascial alterations, the aim of treatment is always to liquefy the fascia, rather than to release the contracture. Once the fascial afferents are normal, that is, no longer nociceptive, then the muscle tone normalises itself. Only manipulation of a limited area will transform friction into heat, modifying the consistency of the fascia's extracellular matrix, which is heat sensitive. Fascial manipulation acts on different tissues:

- It mobilises the hypodermis or the subcutaneous loose connective tissue
- It modifies the consistency of the deep fascia's extracellular matrix
- It restores gliding between the endofascial collagen fibres
- It ruptures adhesions between the layers of deep fascia in the trunk
- It recreates elasticity of the connective tissue skeleton (epimysium, endomysium)

Stecco, 2009>>

Fasciacyten



- Grondsubstantie wordt gevormd door fasciacyten
- Turn-over Hyaluron is 2-4 dagen
- Beweging is een stimulans voor productie
- Bij ontsteking wordt er meer gevormd
- Afgevoerd als lymfe

30

<<The synovial fluid is probably produced by the fasciacytes, which are commonly present inside fasciae and produce hyaluronan (HA). In areas subjected to friction, fasciacytes probably become more organized and produce more HA to facilitate gliding between the various fascial layers. Stecco C, 2015>>

<<This HA-rich layer also protects muscles and supports recovery from injury, and stimulates satellite cell proliferation following loss of muscle fibres. Changes in this HA-rich matrix contribute to pain, inflammation and loss of function. HA is abundant during the earliest stages of wound healing and functions to open up tissue spaces through which cells can travel. By binding to cell receptors and interacting with the cytoskeleton it confers motility to the cells.

HA is particularly plentiful during embryogenesis and in tissues undergoing rapid growth, and is present wherever repair and regeneration occur. Depending on its chain length, especially when it becomes fragmented, HA has recently been shown to have a wide range of opposing biological functions: such as becoming angiogenic, inflammatory and immunostimulatory.

The turnover of HA is about 2-4 days compared to 7-10 days for the other sulfated

GAGs. This means that the HA cells must remain active, otherwise there is the risk of reduction in the quantity of ground substance. The residual products of the GAGs have a feedback effect on the cells and this controls synthesis. It has been established that mechanical distortion of CT (connective tissue) cells represents a stimulus for ECM synthesis (Adhikari et al 2011). Stecco C, 2015>>

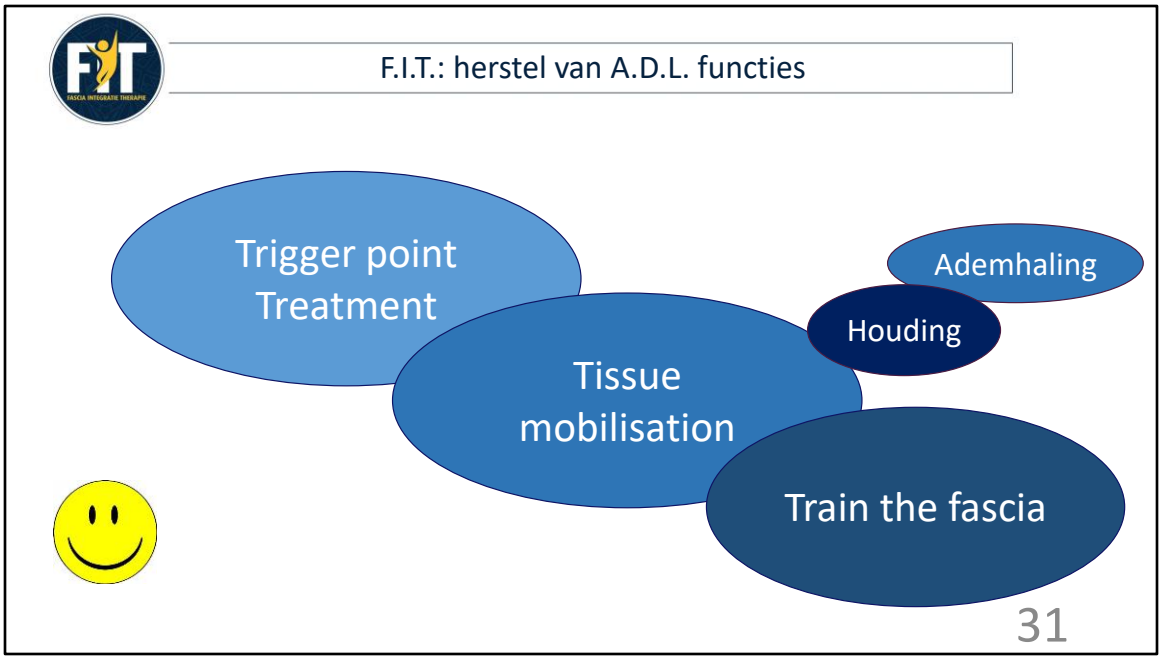
<<HA metabolism (synthesis and degradation) and microenvironmental regulation

HA metabolism is intriguing in that one third of the body's HA undergoes turnover daily (Triggs-Raine B 2015). The balance of HA synthesis and breakdown of HA either by hyaluronidases or oxidative and nitrative stresses define HA content and form. HA production is regulated at multiple levels including enzyme expression, post-transcriptional control by micro-RNAs and antisense HAS expression, and/or posttranslational modifications. HA is synthesized by at least three distinct, apparently functionally redundant synthases. However, the HASs appear to have different biological roles depending on specific contexts.

In addition to enzyme-mediated degradation, HA can be fragmented by reactive oxygen and nitrogen

species. Oxygen radicals degrade HA in vitro (Kennet EC 2007, 2009, Li M 1997), and activation of neutrophil myeloperoxidase can do the same in vivo (Moseley R 1997). Thus, HA fragments become part of the front-line defense to injury by activating innate and adaptive immunity.

Garantziotis S 2019>>



<<The presence of active TrPs in neck pain has previously been documented in the literature (Fernandez-de-Las-Penas C 2007, Dommerholt J. 2005, Freeman MD,2009, Castaldo M, 2014).Active TrPs exhibit greater concentrations of inflammatory and nociceptive substances (substance P, cytokines, etc.) compared with latent TrPs (Shah JP,, 2008). These substances sensitize local nociceptors suggesting an explanation for higher neck pain and lower PPTs in active TrP areas. These results would be further supported by the fact that the injection of algogenic substances has been used to mimic muscle pain and to induce pressure hypersensitivity in healthy subjects (Svensson P,, 2003). Further, if the nociceptive input from the periphery is long-lasting, this may lead to an increased barrage to the central nervous system that can finally increase excitability and synaptic efficacy of neurons in central nociceptive pathways developing central sensitization and therefore lowering PPTs in distant pain-free areas (Shah JP 2008, Imamura M 2016). The fact that nociceptive stimulation of latent TrPs can induce central sensitization in healthy subjects would support this hypothesis (Xu YM 2010). The results suggest that active TrPs can contribute to the development of pain, disability and local as well as widespread pressure pain hypersensitivity in patients with neck disorders. This would support the importance of a treatment directed towards active TrPs deactivation as this may reduce pain and increase pressure pain sensitivity both locally and widespread, as previously found in

patients with shoulder pain (Hidalgo-Lozano A 2011).
Castaldo 2017>>

<<Mobilise
I'm sure I've said it before and I'll say it again. Mobility is the one thing that the body must make use of if movement is to be possible. Mobility in all joints creates an awareness of what is possible in each and every joint, thus creating a safe awareness of movement potential about a mechanically advantageous point for the joint. We call it 'centre'. To know your centre is to be efficient, have flow, conserve energy and to be liberated from the shackles of limitation and stabilisation.
The tighter a muscle is, the less distance it will allow a joint to travel and the more another joint will have to travel to accomplish the overall goals of the body and brain. Now we have entered the realm of compensatory movement. This can only occur when the body is performing suboptimally.
The body is a closed system that has the capacity to redistribute its global tension and compression relationships around the body to facilitate all movement. Ward 2013>>

TrP treatment

Treatment approach based on the knowledge of fascial component of the TrP

Ischaemic compression versus F.I.T. technique

1. Liquify Hyaluronan (Stecco A 2013)
2. Excite Ruffini's → Sympaticus ↓ (Schleip 2003, Staubesand and Li 1996)

From distal to proximal: protect valves in veins

32

<<Technique II produces the same therapeutic effects described under technique I above — it removes the "inflammatory soup," stimulates metabolic processes, and reflexively relaxes the taut bands associated with the mTrP. In addition, connective tissue adhesions and shortened areas of the intramuscular collagen tissue (pathological crosslinks) are released. At the same time, it is possible that the myosin and actin filaments, which are bound together in the form of a stable rigor complex, can be mechanically torn apart. Under the now favorable conditions (hyperemia rather than hypoxia), the muscle tissue can regenerate.

The gliding movement of the therapist's hand is carried out extremely slowly, so that the collagen structures can undergo non-reversible lengthening. A very slow, powerful force applied for a long time is required to stretch collagenous structures (Bogduk 2000)

Deep connective tissue techniques performed powerfully and in "slow motion" can stretch collagen fibers by about 5% (van Wingerden 1995). In addition to the muscle's superficial fascia, the deep intermuscular fascia and connective tissue structures, which had been contracted within the muscle as the result of pathophysiological modification processes, are also stretched.

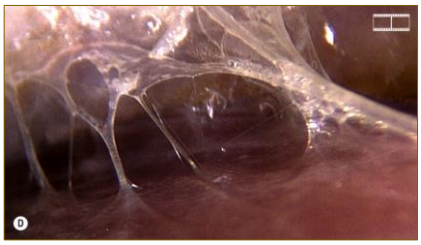
At the same time, the powerful pressure and stretch stimuli excite the fascial mechanoreceptors, a great number of which are located in the superficial and intermuscular fascia (Schleip 2003, Staubesand and Li 1996). This action reflexively decreases the tone of the motor unit involved (the taut band), the global resting muscle tone, and the sympathetic nerve activity (Schleip 2003).
The overall effect is that a “muscle release” takes place. The muscle becomes more supple and distensible. This improves not only the functional capability of the muscle, but the perfusion and metabolic activity as well, allowing the muscle to regain its ability to regenerate.

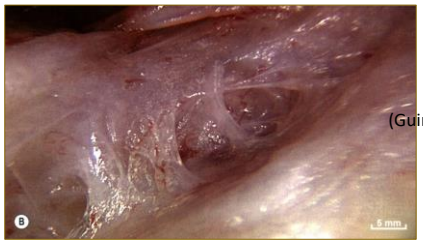
If this technique is performed as a more summary variation, with minimal to medium pressure intensity, then the reflexive effects of technique III predominate: The muscle tone is perceptibly reduced, both locally and globally. Patients perceive this as pleasant and beneficial, so it is good to use this technique when concluding a trigger point treatment session. For patients who are very sensitive to pain, this gentle manual technique is often a good way to begin a therapy session.
In contrast, however, this fascia-stretching technique can also be very specific and concise. In this variation, the taut band involved is selectively stretched out slowly, using the thumb or a wooden trigger point tool over the entire course of the fiber (▶ Fig. 7.17 **a**). This version accentuates the structure-specific effect of technique III on the connective tissue.

When used in the extremities, the fascial stretching technique (technique III) should always be performed from distal to proximal to protect the valves in the veins. Gautschi 2019

Trigger point therapy as a treatment concept. Sustainable therapy of myofascial pain often requires the use of trigger point treatment techniques; used alone, however, they are often not sufficient. Experience has shown that, especially when symptoms are chronic, it may be necessary to bring other factors into the TrP treatment program to achieve sustained resolution of myofascial problems. The following are questions that must be posed, systematically evaluated, and resolved: What factors lead to the development of TrPs? What perpetuates the trigger point activity? What prevents the active mTrPs from spontaneously reverting to the latent phase. Often multiple strategic strands must be woven together to prevent mTrPs from reactivating. Gautschi 2019>>

Tissue mobilisation





(Guimberteau 2015)

Verstoring van fluid flow

33

<< MVCAS responsiveness and resiliency vary depending on the level of pathology present (such as edema, trauma, inflammation, obesity and aging), all of which create identifiable and unique changes in microvacuolar shape.
Edema is accommodated by increased intravacuolar pressure and collagenic distension, without any organic tissue destruction, but with fibrillar distraction limiting further distention and movement. Upon reduction of edema, restitutio in integrum will be the rule.
Open trauma destroys the precise interactions of the MVCAS. Hemorrhage, liquid extravasations, edema and hyperemia will disturb the mechanical balance, and the sliding system will require more force against resistance. Movement will be difficult. Tissues will become adherent from direct trauma and from lack of motion, which will further perturb mobility.
Inflammation induces intravacuolar hyperpressure with fibrillar dilacerations, creating small megavacuola and completely perturbing movement. Therefore, tissue is destroyed, similar to trauma reaction, and the restitutio in integrum will never be obtained. Permanent functional sequelae will be the result. Guimberteau in Schleip 2012 H3.6>>

<< Fascia also has this lubrication effect. Fascia incorporates a water-dense vascular

system, which enables different fasciae to slide friction-free against each other (Guimberteau 2010, 2005, Langevin 2009). The fluid production in the fascia can be disturbed under pathologic circumstances, such as decreased circulation caused by increased sympathetic reflex activity. An example would be the development of subcutaneous connective tissue zones, in which we find reduced mobility as well as a decrease in the possibility of lifting the skin from the bottom layer. Vd Berg 2017 Ch 5>>

<< MVCAS responsiveness and resiliency vary depending on the level of pathology present (such as edema, trauma, inflammation, obesity and aging), all of which create identifiable and unique changes in microvacuolar shape (Plate 3.6.4).
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<<The fluid production in the fascia can be disturbed under pathologic circumstances, such as decreased circulation caused by increased sympathetic reflex activity. An example would be the development of subcutaneous connective tissue zones, in which we find reduced mobility as well as a decrease in the possibility of lifting the skin from the bottom layer. The tension and mobility changes found in osteopathic listening tests are additional examples of these changes. Vd Berg 2017 Ch 5>>

<<The interstitial environment and matrix (also called the ground substance; Pischinger and Heine, 2004; Heine, 1997) essentially alternate between a gel state and a sol state, reducing permeability and making the transport of nutrients and waste substances more difficult, rendering congestion more likely and increasing the risk of an inflammatory reaction.
Mechanical tensions in the connective tissue surrounding the anastomosis, congestion around the capillary system and imbalances of the autonomic system can lead to functional errors and the unphysiological activation or closing of arteriovenular anastomoses. Meert 2012 Ch 1>>

<< 'Permeability of the connective tissue': changes in the sol-gel state of the connective tissue influence the transport of fluids. For example, prolonged compression of connective tissue will press fluids out of the tissue, and fibrocytes can cause increased fibrosis in the long term as a reaction. Meert 2012 Ch 2>>

<< Although this has been known for quite some time, the mechanism responsible for this matrix ‘‘under-hydration’’ has only recently been elucidated. In a series of elegant experiments, Reed et al. showed that tension exerted by fibroblasts onto the collagen network actively restrains the loose matrix and prevents water from entering the tissue [Wiig et al., 2003; Reed et al., 2010].>>

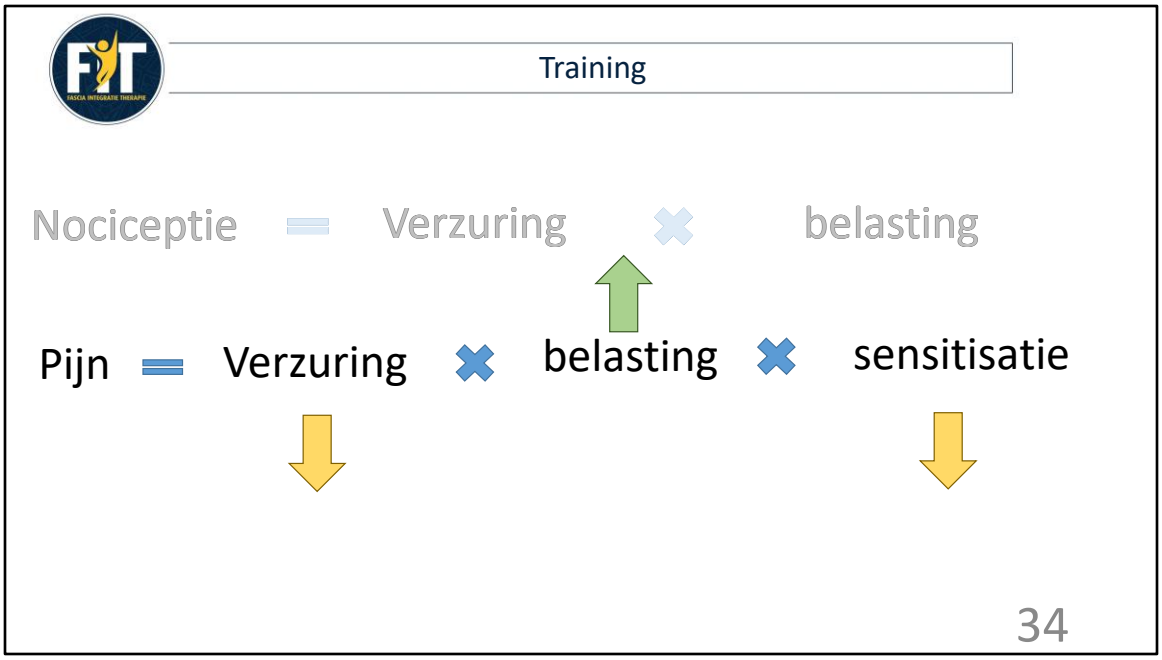
<< When the circulation within tissue is reduced - as the result of increased sympathetic reflex activity, atherosclerosis, or smoking - fascia cannot produce enough fluid and friction-free movement becomes impossible. One of the effects of manual therapy is that we decrease sympathetic reflex activity, which then leads again to increased mobility (van den Berg, 2011).
Vd Berg 2017 Ch 9>>

<<Hauck (1992) demonstrated the existence of a 'low-resistance pathway' along the connective tissue fibres for the transinterstitial fluid movement from the capillaries to the lymphatic vessels. We suggest that the disposition of the collagen and elastic fibres inside the superficial fascia could guide the lymphatic flux in the correct direction. If superficial fascia is altered, then lymphatic drainage will be compromised. Stecco C 2015>>

<<Pressure changes and unhindered mobility are very important for fluid exchange (and best to be influenced). In addition, osmolar gradients and the electrical potentials of particles are also included. Marcer, 2005 >>

<<One highly relevant consideration in terms of practice is the need to establish sufficient mobility in the myofascial area around the blood vessels to avoid any unnecessary reactions (vasoconstriction) of the vessels. Meert 2012 Ch 1>>

<< Obesity begins both with adipocytes replacing glycolycans in the vacuola and with distension of vacuola and fibers. At this stage slow, progressive weight loss will still result in a return to the original morphology. Movement within the tissue is reduced and gravitation becomes more important in determining tissue morphology. In the second stage, vacuola are in extreme dilatation and the distension of fibers changes to dilaceration causing transformation to megavacuola that will in turn be filled by further adipocytes, changing body form. Only surgery will recreate tissue tension at this stage, by resecting excess skin and fat. Guimberteau in Schleip 2012 Ch 3.6>>



To reduce pain for many patients (and therapists) reduction of load is the first thing they will try. Right after an injury that is a good choice but after the recovery phase it is not! Loading is crucial for optimal recovery so reduction of inflammation and reduction of sensitisation should be the first treatment targets.

IASP: (new) definition of pain

"An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage."

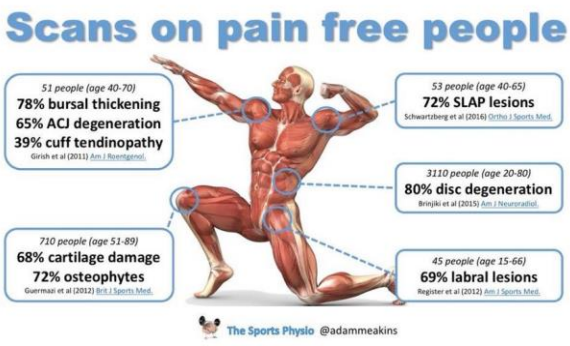
Pain persists in response to being overly sensitive to a perceived threat of harm to your body, despite the absence of tissue damage

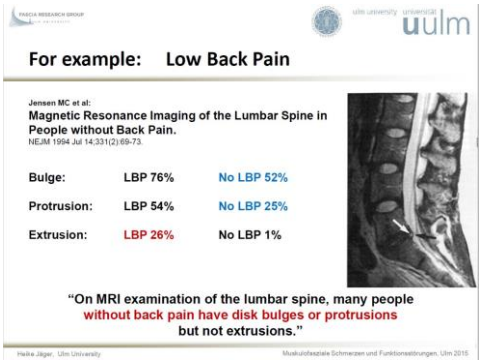
World Physiotherapy Day

Het gaat niet primair om weefselschade maar om homeostase!

35

Relatie objectieve afwijkingen en pijn



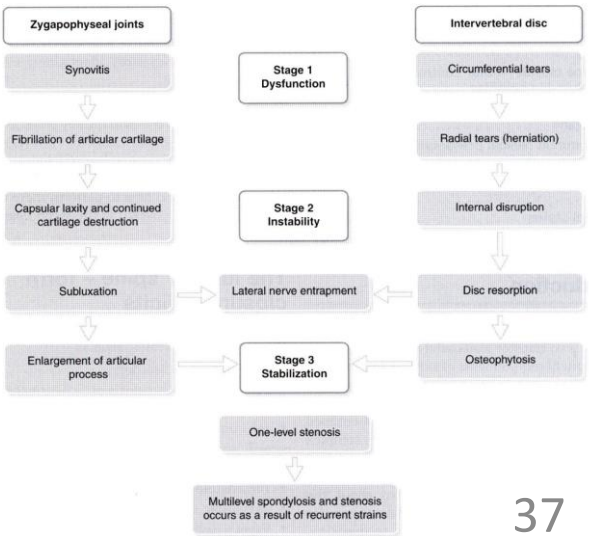


Maybe we have been focussing on the wrong tissues?

36

Structurele veranderingen lage rug

- Macro trauma
- Minor repetitive trauma



(Kirkaldy-Wilhs, 1983)

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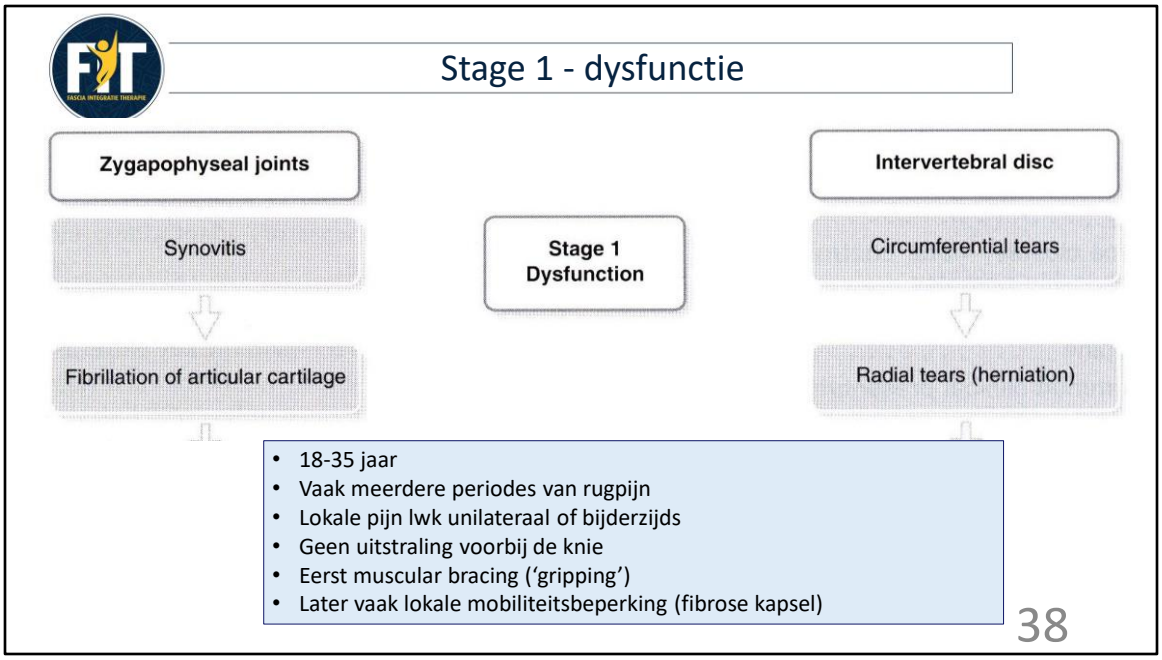
<< the most common cause of structural changes in the passive system of the lumbar spine occurs secondary to sudden macrotrauma (Taylor et al 1990, Twomey et al 1989) or minor repetitive trauma over time (Farfan 1973, 1978, Kirkaldy-Willis et al 1978, Kirkaldy-Willis & Hill 1979, Kirkaldy-Willis 1983, Taylor & Twomey 1986) (Fig. 5.1). The repetitive use of non-optimal strategies for transferring loads through the LPH complex often creates uneven load sharing and eventually tissue breakdown and pain.

Non-optimal strategies for transferring load often create excessive stress on the joints of the LPH complex and, over time, can lead to structural changes, particularly in the lumbar spine. These changes are often attributed to age (Kirkaldy-WiUis & Hill 1979, Kirkaldy-Willis 1983, Taylor & Twomey 1986, 1992) and Bogduk (1997) suggests they are 'natural consequences of the stresses applied to the spine throughout life. Lee, 2011>>



<<These changes are consistent with stage 1 dysfunction (see Fig. 5.1) according to Kirkaldy-Wilhs (1983). Continued use of non-optimal strategies, combined with intermittent acute (recurrent) flare-ups of back pain, can result in progressive pathoanatomical changes ultimately leading to significant form closure deficits and potentially segmental instability (Fig. 5.6) (Bogduk 1997, Kirkaldy-Willis etal 1978, Kirkaldy-Wilhs & Hill 1979, Kirkaldy-Willis 1983, Panjabi 1992a,b, Taylor & Twomey 1986, Taylor et al 1990, Twomey et al 1989). Note that in the Kirkaldy-Wilhs model, 'instability' is defined as the loss of passive system integrity with an increased neutral zone;

The history
This individual is usually young (18-35 years of age] and the mode of onset of pain is commonly insidious (postural) or sudden (trauma). They may report that this is an initial episode of low back pain or it may be a recurrent episode. After an initial acute episode of low back pain, there is a suggestion that subsequent acute episodes of low back pain be termed recurrent episodes of a chronic problem, as the underlying mechanisms contributing to recurrent low back pain are likely to be different from those of a first-time traumatic episode, and recurrence of pain after an acute episode is a common problem (Carey et al 1999, Pengel et al 2003).

The pain may be unilateral or bilateral and is usually localized to the low back (region between T12/ 12th rib and the iliac crest) and can radiate distally as far as the foot. Dysesthesia is not often reported unless there is an injury to the intervertebral disc that has prolapsed or herniated into the spinal canal/ lateral recess. The aggravating activities include the extremes of range of motion (forward/backward bending, rotation), prolonged standing or sitting, and lifting. Rest in the supine lying position (knees over a bolster) usually affords rehef.
In the first few days after a traumatic injury (acute inflammatory phase, see Table 7.2), the patient often has marked difficulty walking and getting out of a chair as both tasks require motion of the lumbar spine and are often provocative.

Often: muscular defense (butt, trunk, back gripping)

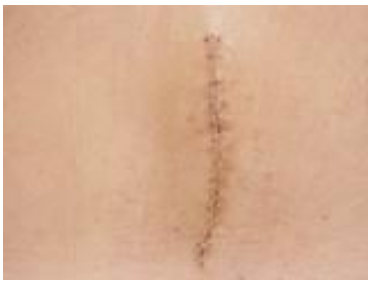
Form closure, force closure, and motor control
Initially, severe pain restricts a detailed examination of segmental mobility, integrity of the passive system (form closure mechanism), and the active and motor control systems (force closure mechanism). As the pain subsides and the overactive superficial muscle system is released (Chapter 10), the tests for mobility and motion control can be done. A variable response will be found and depends on the extent of the structural changes.

If in the later phases of this event (remodeling/ maturation phase from 4 weeks to 1 2 months; see Table 7.2) restrictive capsular adhesions have created a fibrotic stiff joint, the neutral zone of motion will be reduced and the elastic zone will reveal a very firm end feel (Fig. 5.16C). The tests for the integrity of the passive restraints (Chapter 8) will be normal. If, however, there has been an attenuation of the ligaments/capsule or loss of tensile strength or height of the intervertebral disc, the tests for the integrity of the passive restraints may be positive and this finding puts the impairment into Kirkaldy-Willis' stage 2 -instability (see Fig. 5.1).

The results of the tests for the active and control systems are variable and almost always positive. In other words, there are deficits in the neuromuscular behavior of the deep and superficial muscle systems, and possible structural changes in the active systems, the patterns of which are indeterminate and require specific assessment. Lee, 2011>>

Discopathie – wel of geen operatie?

Clinical outcome of instrumented fusion for the treatment of failed back surgery syndrome: a case series of 100 patients. Arts MP1, Kols NI, Onderwater SM, Peul WC. Acta Neurochir (Wien). 2012



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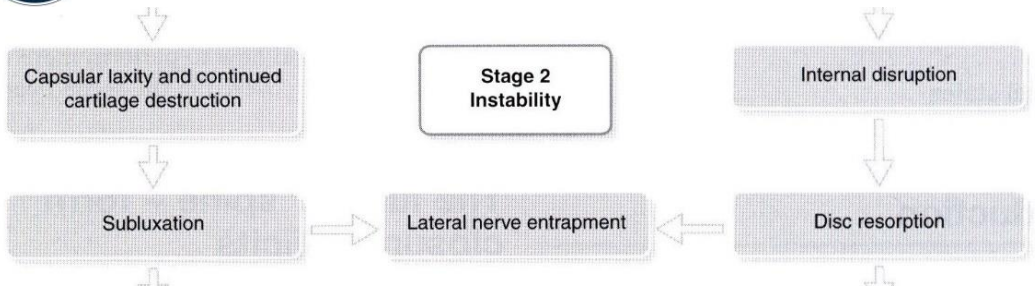
<<After a bladder rectum disorder and a high degree of paresis (emergency indication) are excluded, conservative treatment is the treatment of choice with very good chances of success (90%).
In the medium and long term, the prognosis of conservative (i.e. non-surgical) and surgically treated disc hernia patients is equally good. A large-scale randomized clinical study with over 500 disc hernia patients showed that there was no statistically significant difference between operated and non-operated (i.e. conservatively treated) patients over the 2-year period (Weinstein et al. 2006). This is confirmed by studies carried out as early as 1982, which found that the conservative and operative therapy of the herniated disc after a period of 4-10 years is almost equivalent in terms of clinical results (Weber 1982). In many cases, the surgical procedure for a disc hernia is an impatience intervention.
Nerve root block: It could be shown that the lumbar nerve root block (fluoroscopic application of a steroid to the nerve root) in 60% of the patients with an indication for surgery led to a rapid relief from symptoms, so that surgery was not necessary (Narozny et al. 2001). The therapeutic efficiency of nerve root blockade has been demonstrated in a prospective, randomized, double-blind study (Riew et al. 2000), and the lumbar nerve root block is therefore an efficient measure to support conservative treatment in mild radiculopathy.

In the sacral block, the local anesthetic is injected into the sacral epidural space together with steroids via the sacral canal hiatus, and the solution can rise to the level of the thoraco-lumbar transition; As a result, irritation and inflammation situations, such as occur in connection with acute lumboradicular syndromes in disc herniation or with lumbar spinal stenosis or foraminal stenosis, subside (Grob and Dvorak 1998). Complications are extremely rare if a lumbar or sacral nerve root block is carried out properly. Cave: The risk of serious side effects (tetraplegia) in cervical nerve root block is relatively high (Scanlon et al. 2007). Gautschi 2019>>

Stage 2 - instability



1. Acute blokkade
2. Flexion control impairement
3. Flexion control impairement
4. Rotational control impairement
5. Multidirectional control impairement

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Spit

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<<Lumbar dysfunction with acute pain - stage 2, acute locked back
The intra-articular meniscoid (see Fig. 10.48 A-C) of a moderately degenerated zygapophyseal joint can become trapped during a flexion/rotation load (lift and twist) when the movement and load are poorly controlled. The resulting response of the neuromuscular system (increased activation of the superficial muscles of the trunk) stabilizes/fixates the lumbar segment in what appears to be a 'locked' posture and the findings are as follows.

The history
The mode of onset is usually sudden and the patient vividly recalls the precipitating event. The pain is often unilateral and segmentally specific and intensely aggravated by any motion that inferiorly glides the impaired zygapophyseal joint. The joint most commonly affected is L4-5. The pain may radiate distally down the buttock and posterolateral thigh. Resting in a flexed, laterally translated posture (away from the side of pain) often affords some relief. This is a key finding and differentiates this condition from a prolapsed intervertebral disc, which is usually intolerant to flexion postures.

Standing posture

The patient presents with a classical flexed and laterally translated posture and cannot achieve a neutral lumbar spine posture in any position. Segmentally, the impaired joint is flexed and rotated away from the impaired side. The pelvis is posteriorly tilted and the hip joints are often flexed.

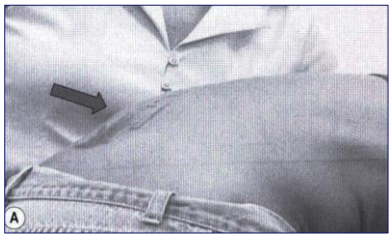
Load transfer tests
Any attempt to correct the postural deformity meets with marked increase in pain. All movements are painful and limited. The protective neuromuscular response restricts all motion at L4-5 and L5-S1. If the pain has significantly altered motor control, unlocking of the ipsilateral hemipelvis and/or buckling/giving way of the impaired lumbar segment may occur.

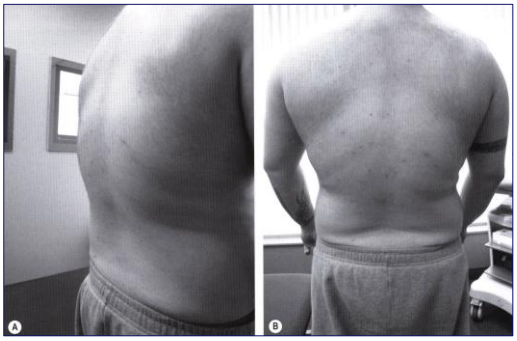
Form closure, force closure, and motor control
Positionally, the impaired joint is held in flexion and contralateral rotation (away from the side of pain). The neutral zone of motion is completely blocked and the end feel is springy (Fig. 5.16F). Marked inhibition of the deep muscle system is usually present.

Treatment
This condition requires a specific high acceleration, low amplitude thrust technique that is described in Chapter 10. Subsequently, the patient will require instruction on ideal strategies (for posture and movement) and may need specific training to restore function of the deep muscles in order to protect this segment from future episodes of 'locking,' since the underlying form closure deficit remains (Chapters 11, 12).

The history
This individual is usually middle aged (35-50 years of age) and has a long history of intermittent low back pain with repeated episodes of exacerbation and resolution. Alternately, this may be the first episode that is not resolving in expected timeframes. The low back pain may be unilateral or bilateral and can refer as far as the distal extent of the segmental dermatome. Dysesthesia is common, though not universal, due to the potential for neurovascular impedance at the intervertebral foramen and/or presence of sensitization of the peripheral or central nervous systems and altered CNS processing (Chapter 7). The aggravating activities frequently include sustained end-range postures (flexion and/or extension of the lumbar spine with or without rotation) and those activities that induce them (prolonged standing or sitting out of neutral spine, prolonged forward or backward bending of the trunk). Rest in the supine lying position with the knees supported over a bolster usually affords relief. The findings of four different segmental impairments will be described. Lee 2011>>

LWK – segmentale instabiliteit





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<< Fig. 4.13 • (A) Note the excessive segmental flexion at L4-5 (arrow). This segment has 'hinged' and translated posteriorly during flexion of the lumbar spine in forward bending. This is non-physiological coupling of motion. (B) Note the horizontal skin crease at L4-5. This segment has 'hinged' and translated anteriorly during extension of the lumbar spine in backward bending. This is also non-physiological coupling of motion.

When flexion couples with posterior translation [Fig. 4.13A), or extension couples with anterior translation [Fig. 4.13B), a hinge, or kink, in the curve is palpable and often visible; these kinematics are non-physiological in that the direction of translation is the reverse of what should be occurring during the osteokinematic motion.

A patient witin a segmental flexion/posterior translation instability at L5-S1. Note tine segmental kyphosis at L5-S1 in standing (arrow) (A) and its accentuation in sitting (B). In addition, note the posterior pelvic tilt (arrow) and loss of the lumbar lordosis in sitting.

Lumbar dysfunction - stage 2 flexion control impairment

An individual with a flexion control impairment often presents with a segmental kyphosis (Fig. 5.17A), which is exaggerated in flexion (forward bending) - The segmental kyphosis is also evident in sitting (Fig. 5.17B). The individual tends to stand and sit with the pelvis posteriorly rotated, the impaired segment flexed and posteriorly translated and the upper lumbar and lower thoracic spine extended. The mode of onset is often flexion/rotation activities, sudden or repetitive. The back pain is usually aggravated by sustained or repetitive flexion tasks (shoveling, gardening, rowing, etc.). The tests for the integrity of the passive system (passive restraints) (Chapter 8) are positive; there is increased anteroposterior translation in the neutral zone and a softer end feel in the elastic zone. The tests for the active and control systems for the impaired segment are positive in flexion loading (i.e. loads requiring control of flexion such as resisted arm extension at 90° shoulder flexion) and the specific motor control and active system deficits are variable (response is indeterminate). When there is loss of segmental control, the deep fibers of multifidus at that segment are usually impaired in function bilaterally and, if the condition is persistent, there are also structural changes in the muscle (atrophy and fatty infiltration) bilaterally (Fig. 5.18). The responses of TrA and the pelvic floor are variable (absent, delayed, asymmetrical) and often not coactivated with the impaired dMF. The superficial muscle system response is variable with at least one muscle (EO, lO, RA, sMF, ES, etc.) being hyperactive.

A patient with a segmental extension/anterior translation instability at L5-S1. (A) In standing, the segmental hinge is not apparent; however, in backward bending (B) the classical 'skin crease' and hinge at L5-S1 is easily seen. Note also the lack of segmental extension in the upper lumbar spine. This is another example of a motion control and a movement impairment occurring at different levels in the same lumbar spine

Lumbar dysfunction - stage 2 extension control impairment
An individual with an extension control impairment presents with an excessive segmental lordosis that is exaggerated in extension (backward bending) (Fig. 5.19A,B) (Video 5.2 {g). The pelvis can be either anteriorly tilted (see Fig. 10.22A,B) or posteriorly tilted (Fig. 5.19B) and can remain there during backward bending. The upper lumbar spine and lower thoracic spine often remain flexed during backward bending such that the motion hinges around the 'unstable' [defined a la Kirkaldy-Willis) segment (Video 5.3 •^)- The mode of onset is often extension and/or rotation activities, sudden or repetitive, and the back pain is usually aggravated by sustained or repetitive extension or rotation tasks (prolonged standing, running, swimming, etc.).
The tests for the integrity of the passive system (passive restraints) (Chapter 8) are positive; there is increased posteroanterior translation in the neutral zone and a softer end feel in the elastic zone. The tests for the active and control systems for the

impaired segment are positive in extension loading (i.e. loads that require control of extension such as resisted arm elevation) and the specific motor control and active system deficits are variable (response is indeterminate). When there is loss of segmental control, the deep fibers of multifidus at that segment are usually impaired in function bilaterally and, if the condition is persistent, there are also structural changes in the muscle (atrophy and fatty infiltration) bilaterally (see Fig. 5.18). The responses of TrA and the pelvic floor are variable (absent, delayed, asymmetrical) and often not coactivated with the impaired dMF. The superficial muscle system response is variable with at least one muscle (EO, lO, RA, sMF, ES) being hyperactive.

Lumbar dysfunction - stage 2 rotation control impairment

An individual with a rotation control impairment may present in acute pain with a segmental lateral shift. If the condition is persistent, the pain is less intense and the shift less obvious. In this case, the segment is often flexed and rotated. This loss of segmental lordosis is exaggerated in sitting, the pelvis is often posteriorly tilted, and an intrapelvic torsion (IFT) is common. The mode of onset is often a rotation injury, usually in flexion (lift and twist), and recurrences of pain and impairment are common. The back pain is aggravated by all tasks that require rotation of the lumbar spine (walking, running, twisting, etc.). The tests for the integrity of the passive restraints (Chapter 8) are positive unilaterally; there is increased unilateral posteroanterior or anteroposterior translation in the neutral zone and a softer end feel in the elastic zone. The tests for the active and control systems for the impaired segment are positive in tests requiring rotational control (bilateral or unilateral) and the specific motor control and active system deficits are variable (response is indeterminate). When there is loss of segmental rotation control, the deep fibers of multifidus at that segment are usually impaired in function unilaterally and, if the condition is persistent, there are also structural changes in the muscle (atrophy and fatty infiltration) unilaterally. The responses of TrA and the pelvic floor are variable although most often asymmetrical in response to a verbal cue. In addition, there is loss of coactivation of TrA with the dMF often in a contralateral pattern (left dMF and right TrA). The superficial muscle system response is variable with at least one muscle (EO, lO, RA, sMF, ES) being hyperactive.

Treatment

In treatment, the goal is to restore segmental mobility to the lumbar spine such that the load is equally distributed. This will require that the patient be taught ideal strategies for postures and movements necessary for their activities of daily living (Chapters II, 12). The impaired joint is commonly compressed (see Fig. 5.16D) by overactivation of the superficial muscle system, which will require release (Chapter 10) prior to retraining segmental control via retraining coordination of the deep and superficial muscle systems (Chapter II) followed by integration into functional tasks (Chapter 12). Note the difference in the extension hinge at L5-S1 during backward bending before and after treatment.

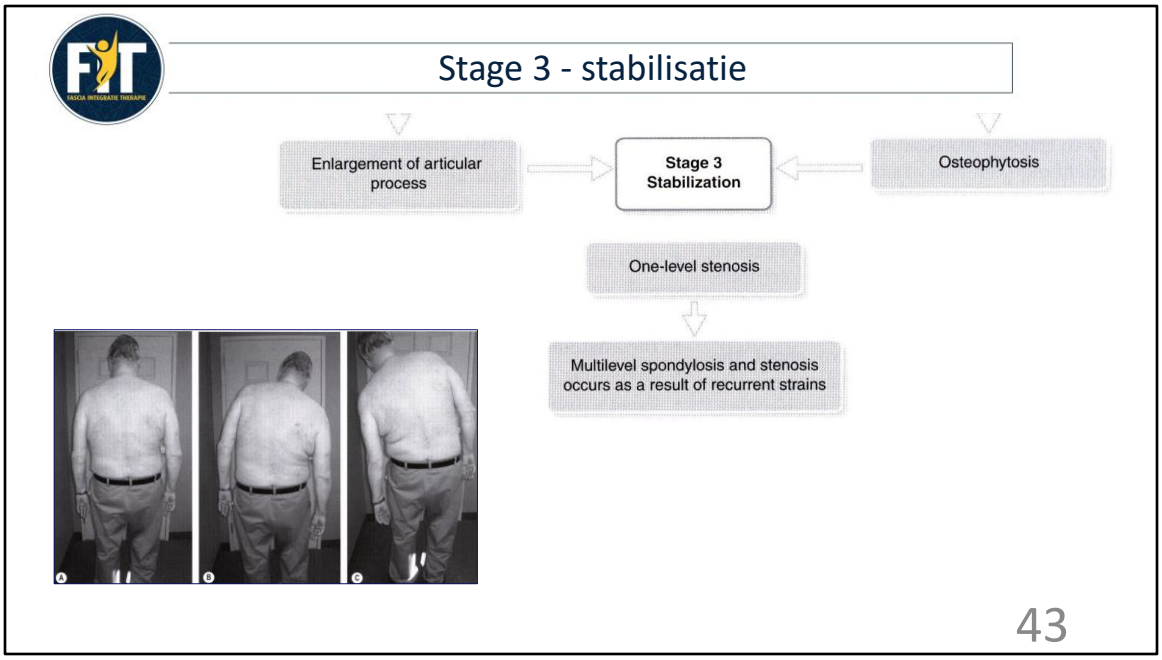
Lumbar dysfunction - stage 2 multidirectional control impairment

The segment that lacks control in multiple directions is severely impaired. This individual has often had multiple episodes of acute back pain with increasing levels of disability after each event. The back pain is aggravated by all loading tasks in every direction and there is minimal range of functional (controlled) motion. It is difficult to assess the integrity of the passive system (passive restraints) (Chapter 8), as there is usually marked co-contraction bracing of the superficial muscle system preventing accurate motion analysis of the deeper structures. The tests for the active and control systems for the impaired segment are positive and the specific motor control and active system deficits are variable (response is indeterminate) and often involve multiple muscle groups. The deep fibers of multifidus are usually impaired in function bilaterally at the impaired segment and there are consistent structural changes in the muscle (atrophy and fatty infiltration) bilaterally (see Fig. 5.18). The response of TrA is often absent and coactivation of the superficial muscle system common (EO, lO, RA, sMF, ES).

Treatment

The individual with a segmental multidirectional control impairment is very difficult to treat conservatively. Although the approach is the same as for the unidirectional control impairment (Chapters 10-12), the response to treatment is often not ideal. These individuals may require a consultation for surgical stabilization; however, prolotherapy should be tried first.

Lee, 2011>>



<< Collectively, these anatomical changes allow the superior articular process of the inferior vertebra to sublux upwards and forwards during axial rotation of the trunk (Fig. 5.8). This motion consequently narrows the lateral recess, potentially affecting the function of the structures within the intervertebral foramen (Butler 2000, Shacklock 2005, Sunderland 1978).
The posterior zygapophyseal joints can enlarge to develop osteophytes, the intervertebral disc can become fibrotic, and traction spurs may develop on the anterior and/or posterior aspect of the vertebral body, occasionally leading to spontaneous fusion (Fig. 5.9A,B). According to Kirkaldy-WilUs (1983), these changes occur during the third stage of the degenerative process (stabilization) (see Fig. 5.1),

and the patient is now often painfree, hypomobile, and no longer unstable. The risk at this stage is the development of fixed central and/or lateral recess stenosis due to osseous trespass on the spinal canal and/or lateral recess with attendant peripheral symptoms of neurogenic vascular claudication. Note that, although the affected segments may no longer be a source of peripheral nociception, the biomechanical effects of these pathoanatomical changes usually create altered stresses on other regions in the kinetic chain and therefore can be a cause of new sources of pain.

Lumbar dysfunction with chronic or persistent pain - stage 3

Continued use of non-optimal strategies combined with intermittent acute flare-ups of back pain can result in progressive pathoanatomical changes, ultimately leading to significant form closure deficits. In stage 3 [stabilization] the osteophytes on the posterior zygapophyseal joints and intevertebral disc can create spontaneous fusion (see Fig. 5.9A]. According to Kirkaldy-Willis [1983), when these changes occur the patient is painfree. The risk at this stage is the development of fixed central and/or lateral stenosis due to osseous trespass on the spinal canal and/or lateral recess with attendant peripheral symptoms of neurogenic vascular claudication. The primary complaint is dysesthesia and pain in the lower extremities often induced by tasks that require extension and rotation of the lumbar spine such as walking. Forward bending and sitting in flexion affords temporary relief, as does intermittent lumbar traction. The lumbar lordosis is often reduced and mobility extremely restricted in this stage [Fig. 5.21 A-C). Motor control training to balance activation of the superficial and deep muscle systems [release any hypertonicity in the superficial muscle system and train better strategies for load transfer using more activation of the deep muscle system) are sometimes beneficial; however, if the structural changes are excessive, surgical intervention may be required. Optimizing mobility and control in adjacent joints in the kinetic chain [e.g. thorax, hips) as well as changing strategies for function can help unload the affected segments by creating better load sharing during functional tasks, and can improve functional status as well as prevent further deterioration of the pathoanatomical changes.

Standing posture

The fibrotic, stiff SIJ is not evident in postural analysis; however, the SIJ that is myofascially compressed secondary to overactivation of the external rotators of the hip has a classical appearance [Fig. 5.22B). Normally, the lumbopelvic region should resemble the shape of a pyramid [Fig. 5.22A) with a wide pelvic base narrowing superiorly at the waist. When an individual develops a strategy for transferring load that uses predominantly the deep external rotators of the hip joint as well as the ischiococcygeus, the constant activation of these muscles compresses the inferior aspect of the SIJ. This is called a butt-gripping strategy [Fig. 5.22B). The bilateral butt-gripper has a buttock that resembles an inverted pyramid and, in standing, a large divot posterior to the greater trochanter can be seen and palpated. The bilateral butt-gripper tends to stand with the pelvic girdle posteriorly tilted, the L4-5 and L5-S1 joints flexed, and the lower extremities externally rotated. If the leg does not appear to be externally rotated at the foot, then a compensatory rotation occurs at the knee and through the foot. The unilateral butt-gripper often stands with the pelvis rotated in the transverse plane and an IPT.

Load transfer tests

In both forward and backward bending, the fibrotic, stiff SIJ will create an IPT and the

asymmetry will be consistent each time the individual moves in the sagittal plane. Axial rotation will be hmited towards the side of the restriction and lateral bending will be limited away from the side of the restriction. A SIJ that is myofascially compressed unilaterally by the external rotators of the hip joint will create similar findings, although the direction of the lateral bending restriction can be variable. In addition, an IPT will be produced and the direction depends on the specific muscles involved; some may create an altered axis of motion for the hip. The apex of the forward bending curve is often in the thorax (Fig. 5.23, Video 5.4 fj]).
During the one leg standing test, the individual with the fibrotic, stiff SIJ will have reduced motion during flexion of the ipsilateral hip (compared to the opposite SIJ] and will have no difficulty transferring load (no unlocking during single leg loading). The myofascially compressed joint may move symmetrically during flexion of the ipsilateral hip or may not, depending on the strategy used for the task, and it frequently 'unlocks' during single leg loading. The ASLR test is often negative when the SIJ is fibrotic or stiff. There is a notable difference in the effort required to lift one leg off the table, and adding more compression to the pelvic girdle does not reduce the effort required to perform this task; in fact, compression may make it more difficult to lift the leg. This finding suggests that the pelvis is already excessively compressed (Video 5-5 -fi]]- The myofascially compressed SIJ will have variable responses to compression during the ASLR, depending on which muscles are overactive (creating different vectors], and to what degree they are overactive. Generally, it is more difficult to lift one leg off the table, but only certain patterns of compression across the pelvis (Chapter 8) will reduce the effort to perform the task; other patterns of compression will make the task harder. In the extremely compressed SIJ (where all parts of the joint are held by hypertonic muscles], any compression pattern makes the leg harder to lift.

Form closure, force closure, and motor control

The fibrotic, stiff SIJ has a reduced neutral zone of motion at all three parts of the SIJ (superior, middle, and inferior], whereas the myofascially compressed SIJ commonly has one part of the joint restricted and the location depends on which muscle is hypertonic. The elastic zone of the fibrotic, stiff joint has a consistently hard end feel, whereas the myofascially compressed joint has a muscular resistance quality. In both cases, the form closure mechanism is intact (normal passive restraints]. The results of the tests for the active and control systems (force closure and motor control] are variable and almost always positive. In other words, there are deficits in the neuromuscular behavior and/or integrity of the deep and superficial muscle systems, the pattern of which is indeterminate and requires specific assessment (Chapter 8].

Treatment

This section describes the specific therapy indicated for restoring mobility of the SIJ following a traumatic sprain of the joint as it is this injury that often leads to a stiff,

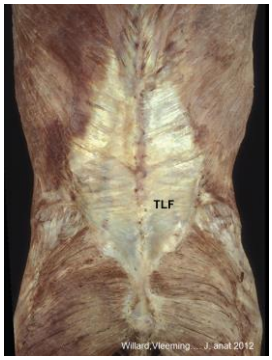
fibrotic SIJ if not properly managed. If the injury results in an intra-articular synovitis, several pain provocation tests will be positive (Chapter 8] and the goal of treatment at this time is to reduce the load through the joint such that healing can occur. The SIJ is a difficult joint to rest as most postures/ positions compress the joint. Clinically, it appears that the best resting position for the painful SIJ is sidelying with the painful side uppermost and the hip and knee supported on a pillow between the legs. Weight bearing activities such as walking, standing, and sitting should be minimized during the first few days. A cane can help to reduce the loading through the pelvis when vertical. Sacroiliac belts increase compression of the joint and often increase pain during this stage of healing.

As the pain and inflammation settles, passive and active range of motion of the joint should be encouraged (Chapter 10]. If the patient presents several weeks or months after the initial injury, it is possible that the SIJ has become stiff and fibrotic. A specific mobihzation technique is the treatment of choice (Chapter 10]. In treatment, the goal is to restore symmetrical mobility between the left and right SIJ such that loads are equally shared and rotation forces are not distributed to the low back or hip. This will require that the patient be taught ideal strategies for postures and movements necessary for their activities of daily living (Chapters II, 12] after the stiff SIJ is mobilized.

The myofascially compressed SIJ is treated with specific release techniques directed towards the specific muscles responsible for the excessive compression (Chapter 10]. Subsequently, retraining of intrapelvic control via activation and coordination. Lee, 2011

A patient with marked central canal stenosis from LI to L5. (A) Note the loss of lumbar lordosis in his standing posture and the marked limitation of (B) left and (C) right sideflexion. This is a very stable, non-painful lumbar spine. His primary complaint was bilateral numbness in his legs and feet that occurred while walking. Lee, 2011>>

E01 - TLF



(Willard 2012)

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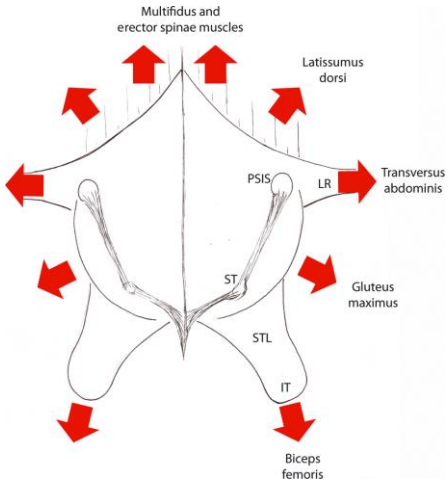
<<Tesarz J, Hoheisel U, Wiedenhöfer B, Mense S. Sensory innervation of the thoracolumbar fascia in rats and humans. Neuroscience. 2011 Oct 27;194:302-8 Abstract—The available data on the innervation of the thoracolumbar fascia (TLF) are inconsistent and partly contradictory. Therefore, the role of the fascia as a potential source of pain in the low back is difficult to assess. In the present study, a quantitative evaluation of calcitonin gene-related peptide (CGRP) and substance P (SP)-containing free nerve endings was performed in the rat TLF. A preliminary nonquantitative evaluation was also performed in specimens of the human TLF. The data show that the TLF is a densely innervated tissue with marked differences in the distribution of the nerve endings over the fascial layers. In the rat, we distinguished three layers: (1) Outer layer (transversely oriented collagen fibers adjacent to the subcutaneous tissue), (2) middle layer (massive collagen fiber bundles oriented obliquely to the animal's long axis), and (3) inner layer (loose connective tissue covering the paraspinal muscles). The subcutaneous tissue and the outer layer showed a particularly dense innervation with sensory fibers. SP-positive free nerve endings—which are assumed to be nociceptive—were exclusively found in these layers. Because of its dense sensory innervation, including presumably nociceptive fibers, the TLF may play an important role in low back pain. >

<<Changes/dysfunction of the thoracolumbar fascia can be triggering and/or perpetuating factors for chronic back pain (Langevin et al. 2011, Schleip et al. 2004, Tesarz et al. 2008).
o Many of the muscles responsible for low back pain (erector spinae muscles, quadratus lumborum, internal oblique and transversus abdominis, latissimus dorsi, serratus posterior inferior muscle) come in direct contact with the thoracolumbar fascia, Because mTrPs very often cause connective tissue changes, mTrPs in the muscles mentioned above can result in restructuring and dysfunction of the thoracolumbar fascia.
o Conversely, a dysfunctional thoracolumbar fascia can induce irritation and faulty loading of the muscles and the formation of mTrPs.
° Changes in the thoracolumbar fascia change the sensory impulse stream of the fascial mechanoreceptors (and possibly the nociceptors as well) -> irritation of the proprioceptors and the motor and sensorimotor function.
o In patients with chronic low back pain, the sheer stress in the thoracolumbar fascia is reduced by about 20% (Langevin et al. 2011). Gautschi 2019>>

TLF



(Willard – 2012)

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<< The material of a good support system will often reflect a combination of resilience, a multidirectional weave, and reinforced strength through multiple layers. The lumbar fascia does fit this design profile. The fascial part of the corset consists of three layers: the posterior lumbar fascia (PLF), the medial lumbar fascia (MLF), and the anterior lumbar fascia (ALF) (Vleeming 2007). The PLF extends from the midline to the thoracic and lumbar spinous processes and their ligaments, and attaches to the posterior ilium, crossing the midline to the opposite ilium. The MLF firmly invests the lumbar transverse processes, its inter-transverse ligaments, as well as the inferior iliac crest and the ilio-lumbar ligament. The transversus abdominis (TA) is continuous with this fascia, and with the abdominal fascia in the front. There, the PLF and MLF form an envelope that houses the multifidus. The ALF arises from the anterior transverse processes and forms a small envelope with the MLF; this envelope houses the quadratus lumborum (QL) before merging with the TA. The QL is concentrically active with the diaphragm upon inhalation and thereby contributes to stability through tensing its own fascial envelope. Blom M-J in Schleip 2012>>

<<In increasing compression the biceps femoris and gluteus maximus muscles play a role (Vleeming et al. 1989a,b, 1992b 1996; DonTigny 1990; Vleeming 1990; Van Wingerden et al. 1993). Both muscles are attached to the sacro-tuberous (and

partially the sacrospinous) ligament which functionally bridges the SI joint. Obviously, pain in the area of the SI joints is not necessarily a local problem; it can be symptomatic of a failed load transfer system (Snijders et al. 1993a, b).
The strong thoracolumbar fascia (TLF) (Tesh et al. 1987) can be used for load transfer from the trunk to the legs (Mooney et al. 2001). The posterior (PLF) and middle layer (MLF) of this fascia are of special interest because of the multiple connections with muscles. The main interest is whether muscle-induced tension of this fascia can assist in effectively transferring load between spine, pelvis, legs, and arms.
From an anatomical point of view the following was noticed:
In all preparations, the posterior layer of the thoracolumbar fascia covers the back muscles from the sacral region through the thoracic region as far as the fascia nuchae. At the level of L4-L5 and sacrum strong connections exist between the superficial and deep lamina. The transverse abdominal and internal obhque muscles are indirectly attached to the thoracolumbar fascia through a dense raphe formed by fusion of the middle layer (Adams & Dolan 2007) of the thoracolumbar fascia and both laminas of the posterior layer. Vleeming A in Schleip 2012 H 1.6>>

Superficiale fascia TLF

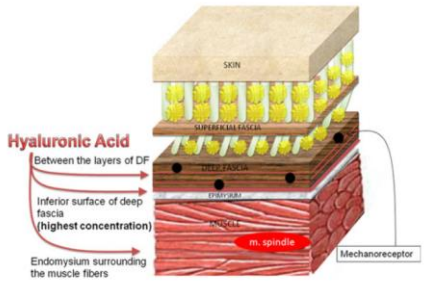
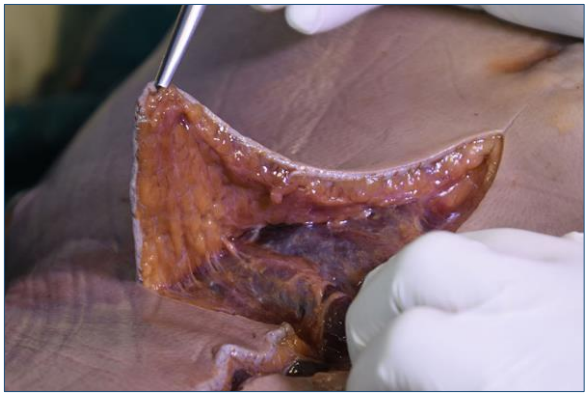


Fig. 5 Idealized image of the deposition profile of hyaluronan



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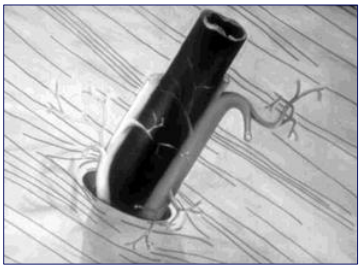
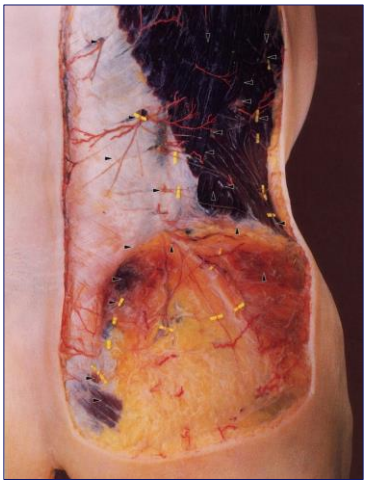
<< Dissection of the back exposes the superficial fascia as a fibrous, adipose layer in the middle of the subcutaneous adipose tissue (Fig. 6.1). It is very thick, especially in the proximal portion of the back, while the deep fascia over the trapezius muscle is thin and adheres to the muscle.

Abu-Hijleh et al (2006) showed that the thickness of the superficial fascia of the back varies significantly among humans, but generally is thicker in females. The superficial fascia of the back is rich in adipose tissue in most areas. The fat cells are usually distributed within the fibrous tissue of the superficial fascia, which gives the appearance that the superficial fascia consists of multiple sublayers. Abu-Hijleh et al (2006) demonstrated that the collagen fibres in two adjacent sublayers run at right angles to one another. Stecco C 2015>>

Chronic Construction Injury (CSI)





(Walter Thiel)

A.C.N.E.S. Anterior
La.C.N.E.S. Lateral Cutaneous Nerve Entrapment Syndrome
Po.C.N.E.S. Posterior

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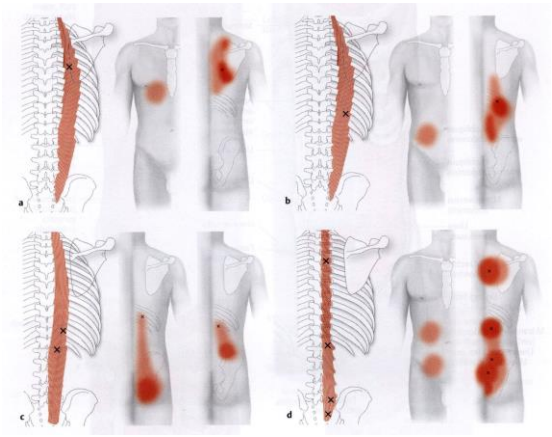
Skin nerves can get trapped in the fascia layer that they pierce. This is called a chronic constriction injury and can cause TrP-like pain

E02 - erector



(Gautschi 2012)

Check TrP's buikspieren!

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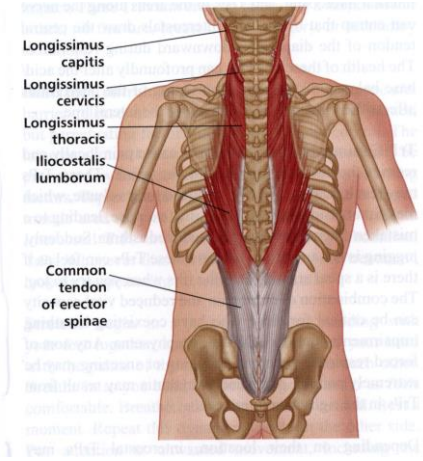
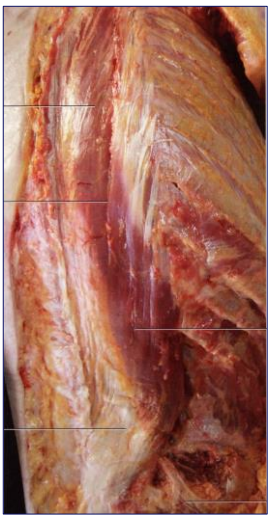
Mm. rotatores / multifidi can be accessed via proc. spinosi. (to lateral)
Lumbar multifidi present as more vertical, consistent with movements in a sagittal plane, and is larger and longer. Additionally, multifidus fibers have significant vertical orientation at L5-S1 and T11-T12, where lumbar and thoracic spines (respectively) have the greatest degree of flexion and extension. Consistent with this concept, the / most oblique fibers of multifidus are located at T1-T2, the joint segment with the greatest degree of axial rotation

E02 Erectors - iliocostalis





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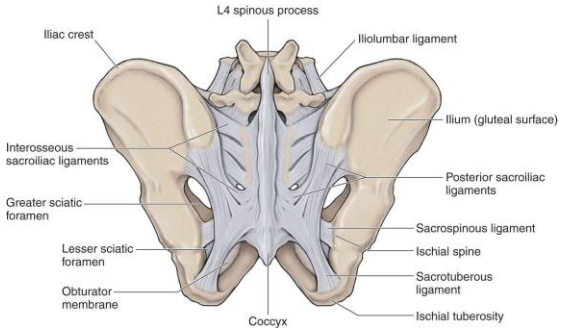
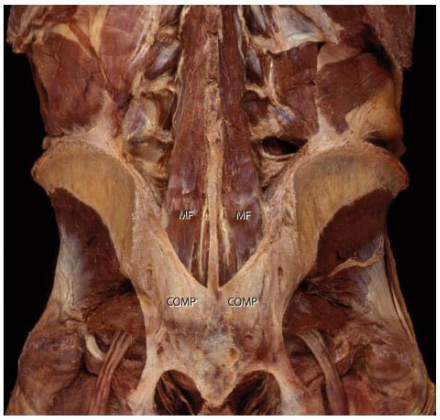
In the fascia around the sips you often find a lot of fibrosis.

Sacrale multifidi





(Willard 2012)

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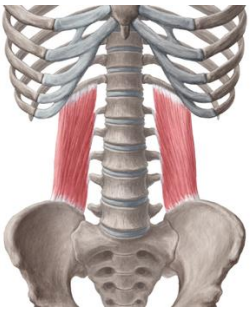
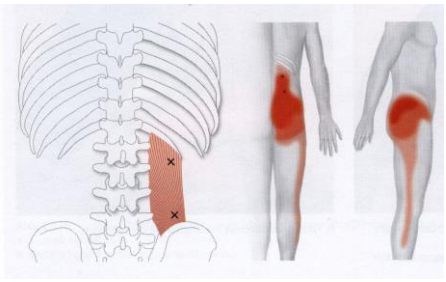
E03 – Quadratus lumborum





(Gautschi 2012)

51

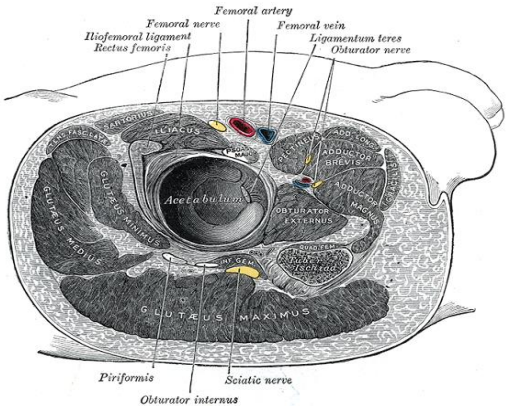
Lumbar multifidi present as more vertical, consistent with movements in a sagittal plane, and is larger and longer. Additionally, multifidus fibers have significant vertical orientation at L5-S1 and T11-T12, where lumbar and thoracic spines (respectively) have the greatest degree of flexion and extension. Consistent with this concept, the / most oblique fibers of multifidus are located at T1-T2, the / joint segment with the greatest degree of axial rotation

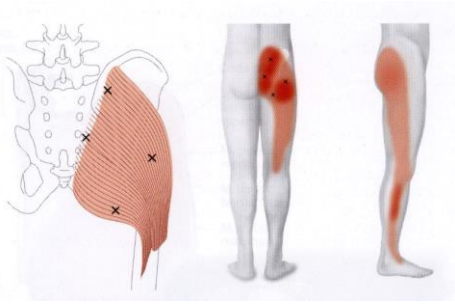
<<The m. Quadratus lumborum is a muscle group in which many proprioceptors occur. These sensors provide important information that is very important for the total coordination and control of the position of the spine. You could say that the m. quadratus lumborum are the eyes and ears of the m. erector spinae. Systems in which certain muscles provide important sensory information for controlling larger movement systems occur in more places in the body. The abdominal muscles are the eyes and ears of the m. iliopsoas, and so on. Bosch 2012>>

<<QL can also refer ain to oOuter aspect of the groin (can refer to labia in women, testicle or lateral penis in men) Wise

2018>>

E04 – Gluteus maximus





Pijn bij lang zitten

(Gautschi 2012)

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The treatment of choice is PIR of the glueus maximus, during which the levator ani also contracts and relaxes at the same time.
Based on clinivally experience and on the therapeutic results it can be assumed that tension in the gluteus maximus and the levator ani is the main cause of a tender coccyx, that is it represents a tendomyopathy of these muscles.

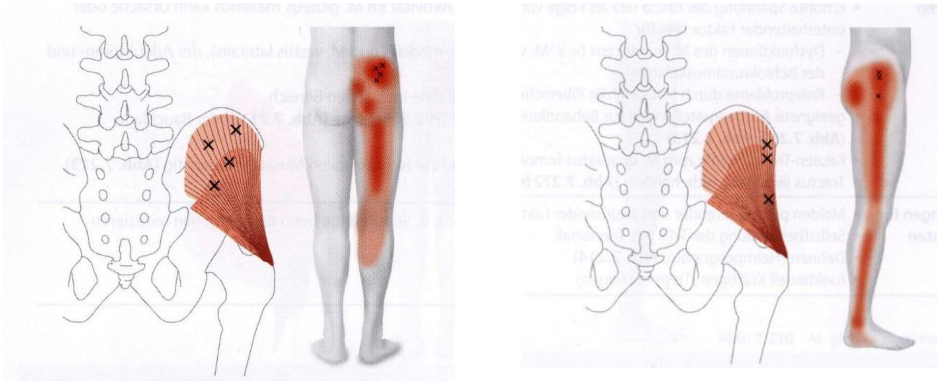
Gmax attaches 85% of the fibers to the tractus iliotibialis, Gmax connects contralateral latissimus with the tractus iliotibialis

TrP in m. Gluteus max is related to increased stress in tensor fascia latae and in combination with fascia thoracolumbalis.
But don't forget the relationship with vastus medialis, lateralis, adductors, hamstrings and gastrocnemius. Also check the tension in the iliotibialis tract.
But primary TrP is usually the QL.

<<• Active mTrPs frequently present
• Increased tension in the fascia lata as the result of TrP activity in the gluteus maximus can be the cause of or a perpetuating factor for:
o dysfunction of the quadriceps (especially the vastus medialis and vastus lateralis),

the adductors and the hamstring muscles;
o knee problems due to irritation of the distal thigh fascia.
The fascial separation technique is important in separating the gluteus maximus from the quadratus femoris and the hamstring muscles
Treat the iliotibial tract as well Gautschi 2019>>

E05 – Gluteus medius / E06 minimus



(Gautschi 2019)

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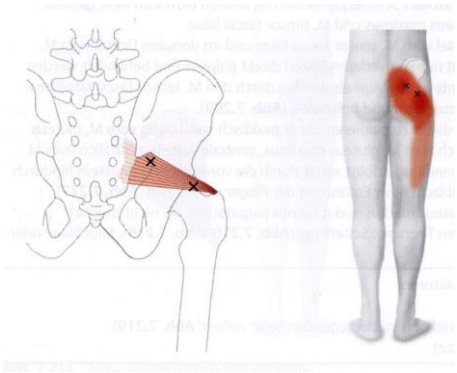
TrP appearance m. Gluteus minimus, is often confused with hernia-like symptoms

<<They are among the muscles that very frequently exhibit TrPs. So-called "pseudoradicular" symptoms and therapist pseudo-sciatica are. in most cases, myofascial pain syndromes with active mTrPs in the gluteus medius and minimus, but also in the gluteus maximus and the tensor fasciae latae.
The tensor fasciae latae overlies the anterior third of the gluteus medius, and the gluteus maximus its posterior third. It can, therefore, only be directly palpated and treated in its middle third, the anterior and posterior parts of the muscle require examination and treatment through the tensor fasciae latae and gluteus maximus, respectively
There is no access to direct palpation of the gluteus minimus — it is virtually completely covered by the gluteus medius. The posterior parts are covered additionally by the gluteus maximus muscle, and the anterior parts by the tensor fasciae latae Its treatment must be performed through the muscles mentioned above and is quite demanding. Therefore, wooden trigger point tool should be used to ease the burden on the therapist's fingers.
It is frequently impossible to make an unequivocal distinction between the gluteus medius and minimus by means of palpation. Gautschi 2019>>

<<Trigger points in the gluteus medius (and sometimes minimus) can refer pain and sensation around the buttocks, hip girdle, and down the leg as well as into the testicles. Wise 2018>>

E07 - piriformis



(Gautschi 2012)

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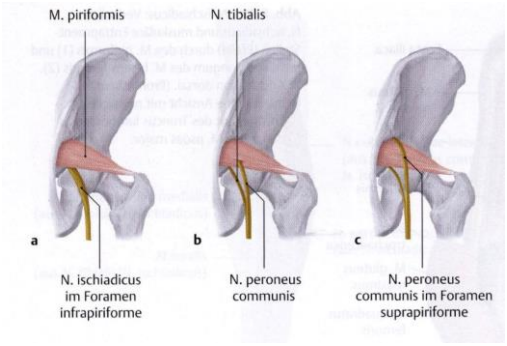
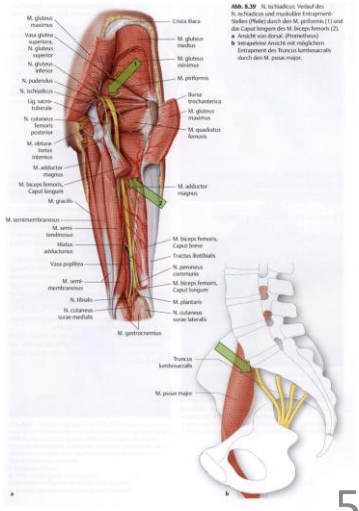
Referred pain from m. Piriformis. This can also be caused by ischemic pressure, when sitting with something in the wallet for a long time.

<<Participates in SI joint dysfunction Piriformis syndrome: combination of myofascial pain (referred pain) and nerve compression syndrome (sciatic nerve)

The incidence of the piriformis syndrome/of TrPs in the piriformis is overestimated. TrPs in the gluteus maximus, medius, and minimus are more commonly the cause of the symptoms. The piriformis may be partially or completely absent.
Palpation: Divide the line between the PSIS and the greater trochanter into thirds; going from proximal to distal, the point marking the first third lies over the place where the piriformis emerges from the greater sciatic foramen -> deep palpation required.
Palpation and therapy must be conducted through the relaxed gluteus maximus muscle

Piriformissyndroom





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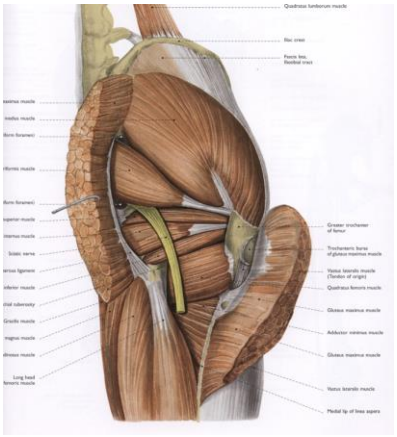
<<This syndrome is still considered a controversial diagnosis and an uncommon cause of sciatic pain (Halpin 2009, Miller 2012). According to Miller, the proposed criteria for the classification of piriformis syndrome are:
Buttock and leg pain made worse with sitting, stair climbing and/or leg crossing.
Pain and tenderness to palpation of the sciatic notch area (piriformis muscle) and pain with increased piriformis muscle tension.
No evidence of axonal loss to the sciatic nerve on electrophysiological testing.
No evidence of abnormal imaging or other entity that could explain the presenting features of sciatica (e.g. radiculopathy, tumor, etc.).
• Reduction of >60% of buttock and leg pain with diagnostic injection into the piriformis muscle under radiographic imaging (fluoroscopic or ultrasound) and/ or EMG guidance.

The sciatic nerve usually passes underneath the piriformis muscle, but many anatomical variations exist and the sciatic nerve could also travel through the piriformis muscle or pass over the piriformis muscle or split in two and pass directly around the piriformis muscle. Nobody has demonstrated an association of a topographic relationship between the sciatic nerve and piriformis muscle and the incidence of piriformis syndrome. Since the sciatic sheath is a continuation of the

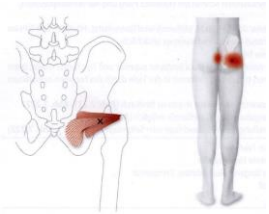
piriformis fascia, an increased tension in this fascia may alter the normal function of the sciatic sheath, causing symptoms similar to nerve compression. Stecco C 2015>>

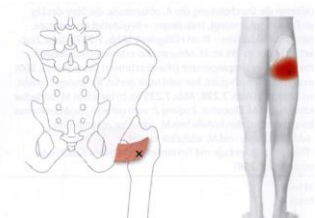
Legs on top of each other: piriformis 21% stretched

E08 – kleine exorotatoren



Heup exorotatoren:
m. quadratus femoris
m. obturatorius externus
m. obturatorius internus
mm. gemelli





(Gautschi 2012)

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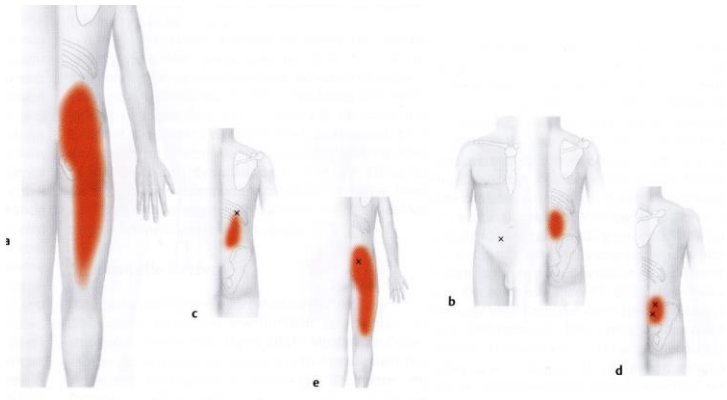
Test: endo hip in the prone position

Exorotators lie deep below the gluteus max. And are usually related to coxarthrosis.
You cannot differentiate the muscles from each other

Apart from the obturatorius externus, all muscles cause pain in the buttock.
The obt. externus gives pain in the groin.

Obt. ext and gemelli are not only exorotators but also extensors and therefore tilt the pelvis backwards
Obt. Int is a flexor and therefore tilts the pelvis forwards

Gecombineerde TrP's



(Gautschi – 2013)

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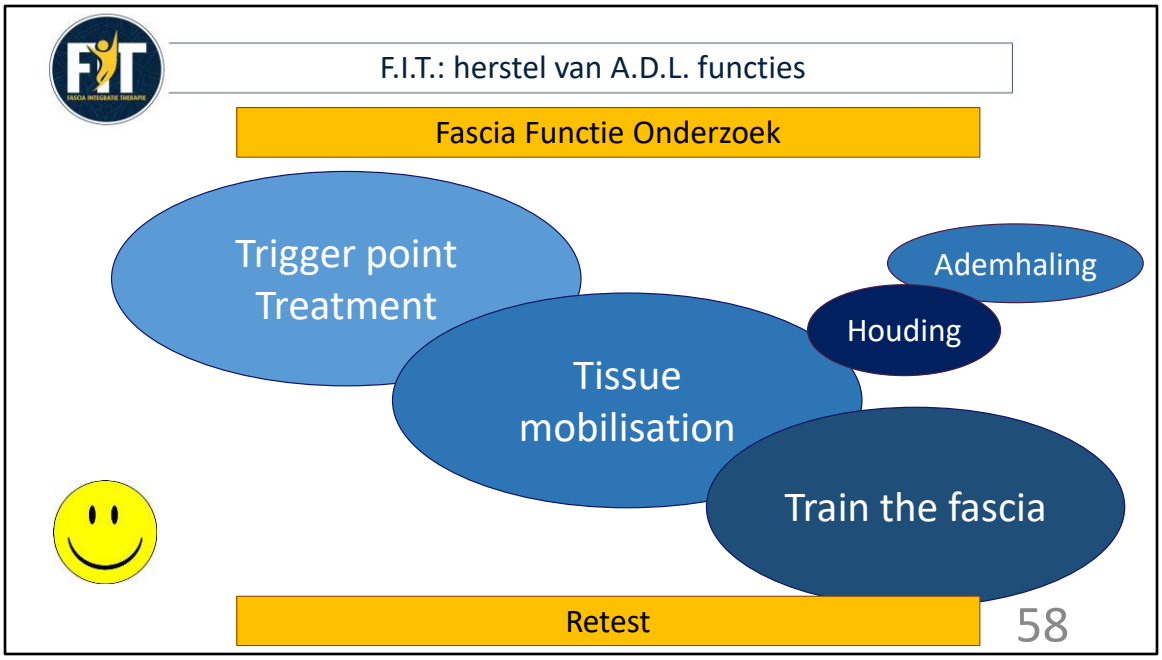
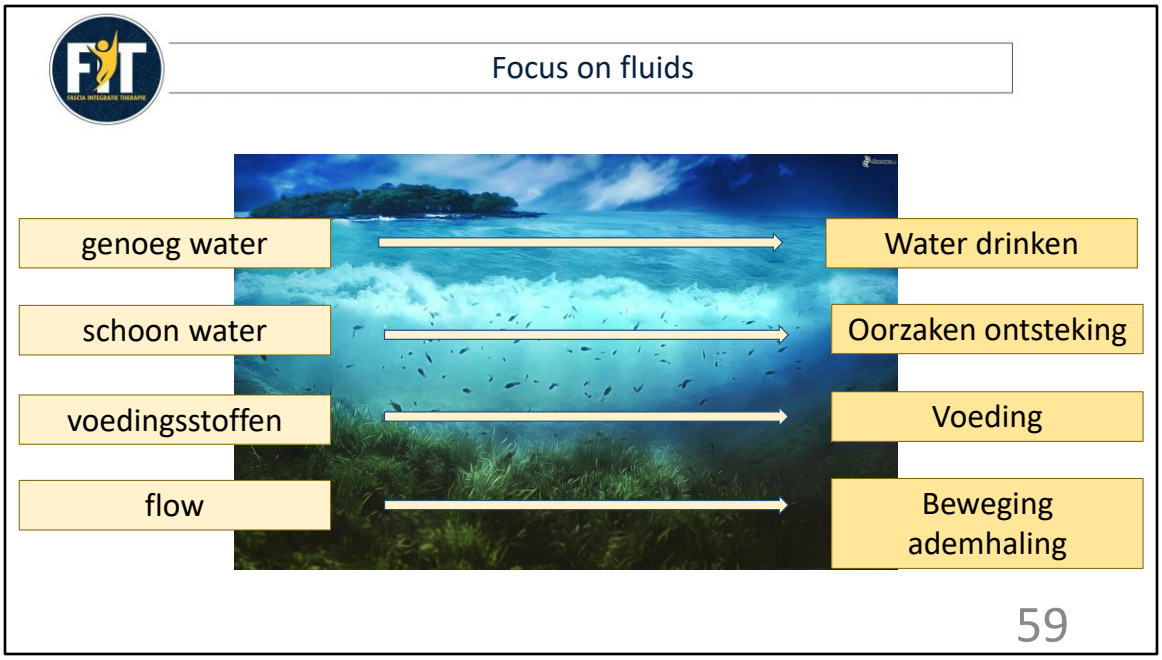
<<Several muscles are often involved in the pain process; The clinical pain pattern often arises from the superimposition of the Referred Pain areas of several mTrPs, which are located in different muscles (-> composite pain patterns
b. psoas
c. iliocostalis thoracis
d. multifidus
e. Gmax
Gautschi 2019>>



<<The presence of active TrPs in neck pain has previously been documented in the literature (Fernandez-de-Las-Penas C 2007, Dommerholt J. 2005, Freeman MD,2009, Castaldo M, 2014).Active TrPs exhibit greater concentrations of inflammatory and nociceptive substances (substance P, cytokines, etc.) compared with latent TrPs (Shah JP,, 2008). These substances sensitize local nociceptors suggesting an explanation for higher neck pain and lower PPTs in active TrP areas. These results would be further supported by the fact that the injection of algogenic substances has been used to mimic muscle pain and to induce pressure hypersensitivity in healthy subjects (Svensson P,, 2003). Further, if the nociceptive input from the periphery is long-lasting, this may lead to an increased barrage to the central nervous system that can finally increase excitability and synaptic efficacy of neurons in central nociceptive pathways developing central sensitization and therefore lowering PPTs in distant pain-free areas (Shah JP 2008, Imamura M 2016). The fact that nociceptive stimulation of latent TrPs can induce central sensitization in healthy subjects would support this hypothesis (Xu YM 2010). The results suggest that active TrPs can contribute to the development of pain, disability and local as well as widespread pressure pain hypersensitivity in patients with neck disorders. This would support the importance of a treatment directed towards active TrPs deactivation as this may reduce pain and increase pressure pain sensitivity both locally and widespread, as previously found in

patients with shoulder pain (Hidalgo-Lozano A 2011).
Castaldo 2017>>

<<Mobilise
I'm sure I've said it before and I'll say it again. Mobility is the one thing that the body must make use of if movement is to be possible. Mobility in all joints creates an awareness of what is possible in each and every joint, thus creating a safe awareness of movement potential about a mechanically advantageous point for the joint. We call it 'centre'. To know your centre is to be efficient, have flow, conserve energy and to be liberated from the shackles of limitation and stabilisation.
The tighter a muscle is, the less distance it will allow a joint to travel and the more another joint will have to travel to accomplish the overall goals of the body and brain. Now we have entered the realm of compensatory movement. This can only occur when the body is performing suboptimally.
The body is a closed system that has the capacity to redistribute its global tension and compression relationships around the body to facilitate all movement. Ward 2013>>



Groundsubstance is for the cells what the sea is for fish

<<Adaptation of body structure is essential to maintaining life. The body constantly reshapes itself through a diversity of adaptive mechanisms that in concert act to maintain structural homeostasis. Some of these adaptive mechanisms operate nearly instantaneously (e.g. viscoelastic response), whereas others take effect over months or years.
Structural homeostasis can alternatively be termed structural homeokinetics, to emphasize that the body is balancing processes in time, rather than maintaining a fixed steady state.
Bordoni 2015>>

<< We can regard these ground substances as both facilitators and as barriers between the blood and all the cellular surfaces, chemical filters which regulate many interactions. Damage to their elements through malnutrition, trauma, fatigue, stress and the like results in the impairment of these supportive functions by the depletion of fluid volume, by the build-up of foreign particles and toxins, or by altering the chemical properties of the mucopolysaccharides. Such disruption strikes at the very basis of healthy metabolic activity. Healthy ground substance works constantly to

help maintain a supportive chemical and physical equilibrium between all the body tissues. Juhan 2003>>

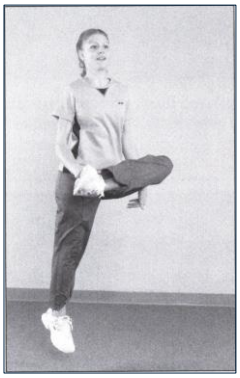
<< Thixotropy, from the Greek words thixis (touch) and tropos (turning or changeable), is a term that is new to many people across the spectrum of clinicians who use manual therapeutics. It is not, however, new to physiologists involved in the study of muscle and tissue mechanics. Thixotropy describes a state of stiffness of a fluid that is dependent on the history of movement. A number of common substances exhibit thixotropy. Tomato catsup is probably the most common. After sitting in the bottle, catsup becomes very stiff and difficult to get out of the bottle. With just a little stirring, however, the stiffness decreases substantially.""

Thixotropy is a physical property of muscle and other tissues and not a response to some neurophysiological event. The mere act of moving a substance with thixotropic properties will result in a reduction of stiffness. The reverse is also true: if a thixotropic substance remains still for a given period of time (which varies depending on the substance), the substance will become stiffen
Cantu 2012>>

Therapie

60

Free hips = happy back



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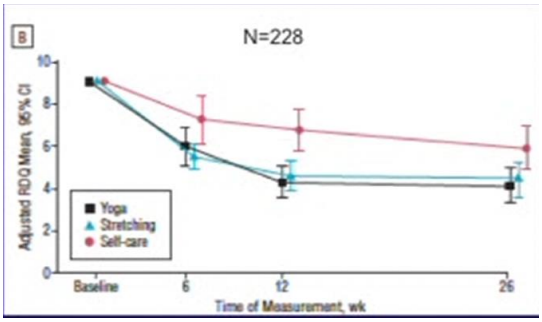
<<Conventional wisdom maintains that freer hips mean happier backs, and research both in Japan (1) and in the USA (2) generally supports this.

Simply being put into this "baby crawling" or "bullfrog" position often gives a therapeutic stretch to the hip joints; however, while we are here, we can increase hip mobility by releasing the gluteals. While stabilizing your client's leg with your own, use the flat of your forearm to gently lean into the medial attachments of the gluteus maximus just below the iliac crest (Figures 10.5 and 10.6). Tendinous attachments have concentrations of Golgi tendon organs. These respond to sustained pressure, so you will get the best results by waiting with slow, nearly static pressure here, rather than sliding or moving your touch. Use moderate pressure, with a slight vector of pressure towards yourself, in order to ease or nudge the gluteus away from its bony attachments on the ilium

Specific kinds of hip mobility have been correlated with low back health. Internal hip rotation, hip flexion, and hip extension in both sexes, and hamstring flexibility in men, all have a negative correlation with back pain (that is, people with those types of mobility generally have less back pain) (3). Luchau 2015>>

Stretching - yoga



(Sherman, K.J. et al, Arch Intern Med, 2011, 171 (22): p. 2019-2026)

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Sherman: large scale randomized controlled trial of yoga or stretch for chronic low back pain

Corey (2012)

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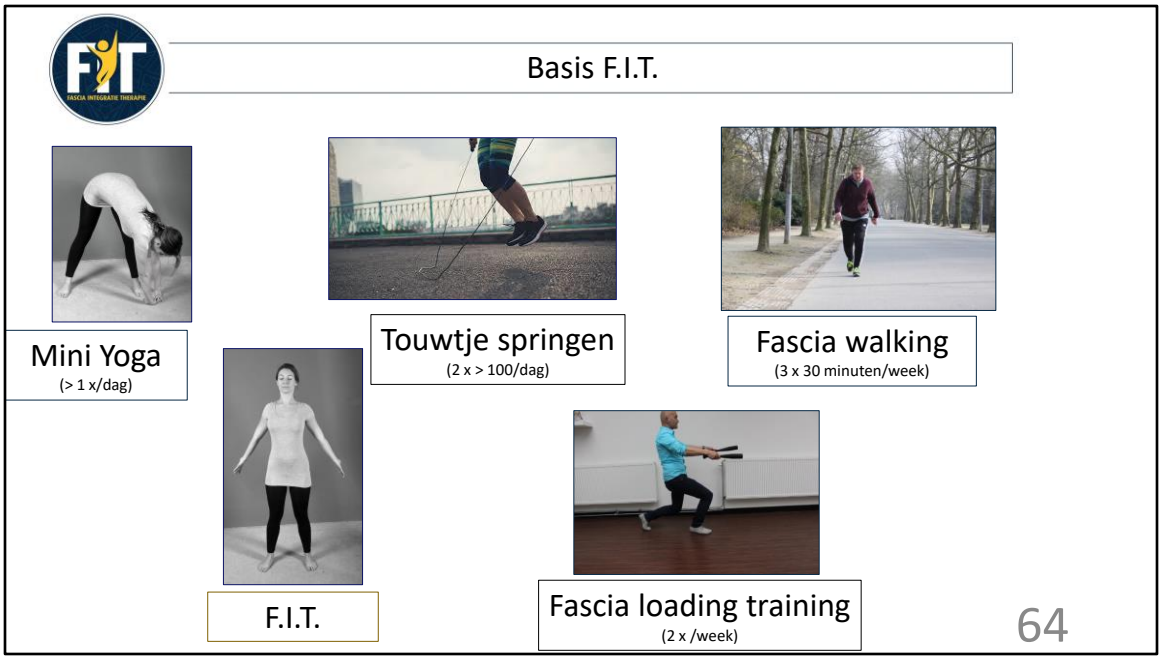
Korte stretch: anti-inflammatory, anti-fibrotic, verminderde mechansosensitiviteit, beter looppatroon

Corey SM, Vizzard MA, Bouffard NA, Badger GJ, Langevin HM. Stretching of the back improves gait, mechanical sensitivity and connective tissue inflammation in a rodent model. PLoS One. 2012; ;7(1):e29831. doi: 10.1371/journal.pone.0029831. Epub 2012 Jan 6

Abstract

The role played by nonspecialized connective tissues in chronic non-specific low back pain is not well understood. In a recent ultrasound study, human subjects with chronic low back pain had altered connective tissue structure compared to human subjects without low back pain, suggesting the presence of inflammation and/or fibrosis in the low back pain subjects. Mechanical input in the form of static tissue stretch has been shown in vitro and in vivo to have anti-inflammatory and anti-fibrotic effects. To better understand the pathophysiology of lumbar nonspecialized connective tissue as well as potential mechanisms underlying therapeutic effects of tissue stretch, we developed a carrageenan-induced inflammation model in the low back of a rodent. Induction of inflammation in the lumbar connective tissues resulted in altered gait, increased mechanical sensitivity of the tissues of the low back, and local macrophage infiltration. Mechanical input was then applied to this model as in

vivo tissue stretch for 10 minutes twice a day for 12 days. In vivo tissue stretch mitigated the inflammation-induced changes leading to restored stride length and intrastep distance, decreased mechanical sensitivity of the back and reduced macrophage expression in the nonspecialized connective tissues of the low back. This study highlights the need for further investigation into the contribution of connective tissue to low back pain and the need for a better understanding of how interventions involving mechanical stretch could provide maximal therapeutic benefit. This tissue stretch research is relevant to body-based treatments such as yoga or massage, and to some stretch techniques used with physical therapy.



Stretch-Shortening-Cycle (SSC). Sports science categorises reactive strength or the SSC as a separate motor quality. However, it relies strongly on maximum strength, innervation patterns of the muscles and the elastic recoil properties of fascial connective tissues (Schmidtbleicher & Gollhofer, 1985)
Compared to the pure concentric rate of force development, without a preceding eccentric movement phase, in the SSC a higher force is achieved (Komi, 2000). Currently there are two explanation models for this phenomenon. A neu-rophysiological model and a mechanical model. In the neurophysiological model it will be suggested, that the rapid pre-stretch of the muscle spindles during the eccentric phase, leads to a stretch reflex. As a result result, it develops a more powerful muscle contraction, due to the activation of more motor units (Komi, 1992). The faster the eccentric loading phase, the stronger the concentric muscle contraction (Bohm et al., 2006). A common example for a stretch reflex is the knee jerk reflex.

In the SSC, fatigue is absolutely counterproductive. It causes a stretch reflex decrease and a loss of elastic energy potential (Komi, 2000). This leads to a reduced muscle and joint stiffness, which can cause injury. Thus, turning plyometrics into a conditioning programme, which is usually designed to develop energy systems, is not

advisable. One key to avoid fatigue is to keep a specific work-rest-ratio between sets. For rehabilitation, Chu & Cordier (2000) suggest a ratio of 1:5-1:10. When the exercise takes 10 seconds, 50-100 seconds rest would be appropriate. True Shock Training, for example, depth jumps, demands considerably longer rest periods. Depending on the intensity 3-10 minutes rest is advised (Bubeck, 2002) (Sialis, 2004).

Dynamic alignment

When the spine is in line…..

……you feel fine!

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Lage rugpijn - kernpunten

- **FFO: focus op functie!**
- TrP's: m.n. quadratus lumborum, psoas, glutei
- Verschuifbaarheid thoraco lumbale fascia
- Mobiliteit heupen
- Kracht Gmax – heup scharnier
- Dynamic alignment / lengte
- Ademhaling

- Voeten / fascia plantaris

Be specific!

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Uiteindelijke doel

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