21 Fascial Syndromes Emerging, Treatable Contributors to Musculoskeletal Pain

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INTRODUCTION

Fascia is essential to the overall health and function of the human body and is a key component to understanding the human structure. Fascia is both a tissue and a system. The collagen content of fascia is capable of slowly remodeling itself over time, based on the mechanical forces to which it is subjected. This has both positive and negative ramifications. Understanding how the complexities of repetitive force transmission through the body's fascial web can affect posture and function will guide the physician or clinician to a new understanding of the complexities of chronic pain and physical dysfunction where traditional musculoskeletal models prove insufficient.

This chapter details the emerging integral role of fascia in chronic musculoskeletal pain, as well as improving fluid flow to enhance metabolic function.

DEFINING OUR TERMS

For most of medical history, fascia was thought to be an inert, biologic packing material and, as such, it held very little medical interest; however, that view is rapidly changing. In the year 2000, PubMed showed 364 new papers under the topic. By contrast, in 2017, 475 articles were listed for the first 6 months alone. This growing trend can be traced back to the First International Fascia Research Congress, which took place at the Harvard School of Medicine in 2007 and drew over 500 researchers and clinicians. The fifth such conference is to be held in Berlin, Germany, in 2018. Significant was their definition of fascia not just as a tissue, but also as a system: *The fascial system consists of the three-dimensional continuum of soft, collagen containing, loose and dense fibrous connective tissues that permeate the body. It incorporates elements such as adipose tissue, adventitae and neurovascular sheaths, aponeuroses, deep and superficial fasciae, epineurium, joint capsules, ligaments, membranes, meninges, myofascial expansions, periostea, retinacula, septa, tendons, visceral fasciae, and all the intramuscular and intermuscular connective tissues including endo-/peri-/mysium.*

The fascial system interpenetrates and surrounds all organs, muscles, bones and nerve fibers, endowing the body with a functional structure, and providing an environment that enables all body systems to operate in an integrated manner [1].

COMPONENTS AND RECENT RESEARCH

Fascia is composed of fiber and fluid, collagen and ground substance, also collectively referred to as the extracellular matrix (ECM). The fiber is predominantly collagen, of which there are at least 15 distinctive types [2]. The most abundant in connective tissue is collagen Type I, which accounts for 90% [3]. These fibers wind together in a triple helix, giving the fascia tremendous tensile strength that, gram for gram, is stronger than steel [4]. Like steel, collagen is highly

ductile, meaning that when subjected to extreme forces it will slowly deform, rather than break like porcelain or glass.

Ground substance is host to a variety of proteoglycans, which are composed of smaller molecules called glycosaminoglycans (GAGs). One specific GAG worth clinical consideration is hyaluronan. Hyaluronan is the hydraulic fluid that keeps the muscles and joints gliding properly and is produced in the sliding layers between the epimysium of the muscles by a class of cells called fasciacytes. Changes in the viscosity of hyaluronan could compromise the sliding behaviors of the underlying muscles and fascia, creating symptomatic pain [5].

It is further theorized that the ECM, with its abundance of hydrophilic GAGs, acts very much like a sponge. While drinking water is necessary for proper metabolic function, it is not enough. It is the mechanical stimulation of the fascial tissues via compression stretching, and upping the core body temperature, serving to wring out the sponge, allowing old waste water to be carried away and new fresh water to be taken up by the tissue, helping to clean up any metabolic traffic jams. Whether that happens via movement or manual therapy is considered moot at this point.

The fascia also contains an abundance of cell types including mast cells, T cells, macrophages, lymphocytes, adipocytes and the recently discovered telocytes [6]. Telocytes are mechanosensitive cells that are vital to many physiological processes like stem cell upkeep, tissue repair and immune function. Telocytes share genetic material with other cells via extracellular vesicles. This discovery lends additional credence to the theory that the fascial system is a body-wide, cellular signaling network [7,8].

The most abundant cell in the fascia is the fibroblast, the custodian of the ECM. Along with producing cytokines, interleukins and other immune function cells, the fibroblast also synthesizes and remodels collagen based on the tension between the cell and the ECM. So, when the tension is low, there is little collagen production. When under high tension, the fibroblast will increase collagen production and cell proliferation [9]. Direction sensitive, the fibroblast will organize itself based on the pull of the underlying matrix [10]. Subjects with low back pain (LBP) showed a 25% greater thickness in their lumbar fascia when compared to pain-free controls [11]. The lack of regular movement or total immobility will give the fibroblasts little to no stimulation, which has a negative impact on the formation of healthy collagen and also causes a loss of crimp (Figure 21.1).

Crimp is the two-directional, lattice-like collagen weave that accounts for the springy, elastic movements that are a property of the fascia [12]. Animals studies show that immobilization promotes the development of crosslinks among the fibers, inhibiting natural movement [13].



FIGURE 21.1 (a) Electron microscope image showing collagen fibers in a normal soleus perimysium running in three different distinct orientations. (b) How immobilization destroys the collagen network and renders the orientations indistinguishable. (Reproduced from Jarvinen et al. with permission.)

Appropriate fibroblast stimulation through movement and manual therapy can restore healthy crimp formation [14].

An in vitro study subjected an active culture of fibroblasts to sufficient repetitive strain to incur hardening of the collagenous elements of the cytoskeleton. These stressed cells had a 30% higher rate of apoptosis non-stressed controls. Moreover, stressed cells treated to compression with stretch, the core components of myofascial release, reduced apoptosis to slightly below the level of the control group and restored other negative factors to near their pre-stressed levels [15].

ANATOMY AND THE FASCIAL SYSTEM

While anatomy books like to divide fascia into discrete units and aggregates, it is vital to remember that all these parts, pieces and layers are part of one system. Starting just under the skin with the more superficial or areolar layer to the deep fascia or fascia profunda. The deep fascia comprises all the layers that interact with the musculoskeletal body (Figure 21.2). The deep fascia is highly organized and very much like an elastic, full-length body stocking – the innermost layer peeling away to form an epimysium, a pocket around each muscle. These epimysial pockets are free to glide due to hyaluronan [5]. This layer continues to the bundled perimysium, down to each individual muscle fiber wrapped in its own endomysium. This honeycomb arrangement allows for load sharing among the individual myofibers. Electron microscope studies have also revealed collagen fibers running in a more perpendicular fashion, creating a longitudinal network through the epimysium to the adjacent antagonistic muscle [16]. Other imaging studies clearly show the collagen fibers getting smaller and smaller, going all the way down to and through individual cell walls [17].

The deep fascia also includes the dynaments, which are highly specialized connective tissue structures organized in series with muscle fascicles, rather than in parallel, as has traditionally been thought. This calls into question the idea that ligaments are only active during the end ranges of joint movement [18].

Another significant development was a series of body-wide maps organized around the mostly vertical lines of fascial force transmission [19]. These maps, known as the anatomy trains [20,21], adhere to specific rules regarding direction, depth and direct and mechanical connections in certain planes of movement (via shared skeletal connections) and the fact that muscles also "attach" to other muscles via the fascia, and not just bone (Figure 21.3). The anatomy trains have survived the scrutiny of the cadaver laboratory [22]. A recent systematic review of anatomical dissection studies [23] looked for independent evidence for 6 of the 12 maps, finding sufficient corroboration for 3. The most verified map is the superficial back line (SBL) (Figure 21.4).

Covering the dorsal aspect of the body, the SBL comprises the plantar fascia to the periostea of the calcaneus, which has a fascial connection to the gastrocnemius via the Achilles tendon. From the gastrocnemius, we have a functional coupling with the hamstring, making an important distinction. Functionally, when the knee is flexed, the fascia of the upper and lower leg functions separately. When extended, they link, rather like two pairs of hands linked at the wrist (think of trapeze artists) to form one functional unit. Fascial crosslinks have also been observed at this junction [23]. The hamstrings continue to the ischial tuberosity, which continues across to the lateral border of the sacrum via the sacrotuberous ligament. Numerous dissections have revealed that the superficial aspects of the ligament are continuous with the tissue on either side of the ischial tuberosity; and have shown the ability to lift the superficial fibers of the sacrotuberous ligament away from the body while still maintaining a strong continuity with the hamstrings and the fascia of the erector spinae [21,22]. From the erector spinae, we travel up to the galea apoeneurotica to the epicranial fascia. Though we have started from the ground and worked our way up, this is arbitrary. Fascial force transmission works in both directions.

While not meant to supplant the traditional origin/insertion model, the anatomy trains present a model for holistic anatomy, the understanding of which can lead the clinician or physician to different insights, and a model for understanding biotensegrity.



FIGURE 21.2 The layers of the deep fascia from the epimysium of the muscle to the endomysium; the individual wrapping for each muscle fiber. (Reproduced with permission from Handspring Publishing Ltd, taken from *Fascia: What It Is and Why It Matters* by David Lesondak, 2017.)

BIOTENSEGRITY

Biotensegrity is a biomechanical first proposed by orthopedic surgeon Stephen Levin [24,25]; it leverages the truss or the three-dimensional triangle model. Trusses can withstand tensile forces much better than a square frame design. The Saint Louis Arch is constructed on the truss model. So is the arch of the foot.



FIGURE 21.3 Does muscle really "attach" to bone? In this fresh tissue dissection, viewed from the profound side, there is no clear distinction or border between the splenius, rhomboid and serratus muscles. Screen capture from video by author. (Photo courtesy of Thomas Myers and the Laboratories for Anatomical Enlightenment.)

Arising from the worlds of art and architecture, tensegrity is defined as any structure that employs contiguous tensional members and discontinuous compression members in such a way that each member operates with maximum efficiency and economy. In the biotensegrity model, the bones are the discontinuous compression members and the surrounding connective tissue, the fascial system, provides the tensional framework. Clinically, subjects who have undergone massage for shoulder pain utilizing tensegrity principles showed a statistically significant increase in both passive and active range of motion (ROM) during flexion and abduction [26].

Tensegrity is also the basic property of the cellular structure. Experiments have shown that mechanical restructuring of the cell via the cytoskeleton tells the cell what to do [27–29]. In short, when cells were stretched, they thrived. And, similar to Meltzer's study [15], cells prevented from stretching went into apoptosis. This process happens via integrins, a cell receptor that binds the cell to the ECM with collagen fibers. When stimulated by pressure and vibration, the integrin transmits this tension directly to the nucleus, altering gene expression and activating mechanotransduction.

Clinically, a massage after strenuous exercise was shown to both increase the production of antiinflammatory agents and create new mitochondria by inducing mechanotransduction [30]. There is now little doubt that manual therapies can alter genetic expression [31]. While joint degeneration can still occur even in the absence of inflammatory agents [32], it is worth noting that such studies do not take into account strain hardening and repetitive motion syndromes.

RELEVANT METABOLIC PATHWAYS

While not much is known about the fascia and nutrition, adequate amounts of Vitamin C are vital for collagen Type I synthesis [33]. Otherwise, the basics of an anti-inflammatory diet are recommended, including essential omega-3 fatty acids with an overall low ratio of omega-6 to omega (3:1 or 5:1), though the typical intake ratio in the diet of most Americans is much higher [34]. Omega-3 supplements offer a safe alternative in helping balance this ratio [35], but certain doses can lower platelet aggregation [36].



FIGURE 21.4 The superficial back line (SBL) in both unembalmed (a) and embalmed (b) dissections. (b) Overlaid on the skeleton model for clarity. Photos by author. (Photo courtesy of Thomas Myers and the Laboratories for Anatomical Enlightenment.)

Certain spices and herbs also contain bioactive substances shown to reduce inflammation. They have the added benefit of regulating inflammation through multiple pathways, as opposed to modifying just a single enzyme of an inflammatory cascade. These spices and herbs include capsaicin, curcumin, ginger, licorice, saffron and turmeric [37,38], although high doses of these can create toxicity and also interfere with certain medications.

Olive oil is a good source of oleocanthol, which inhibits the production of pro-inflammatory COX-1 and COX-2 enzymes, with 3.5 tbsp the equivalent of 200 mg of ibuprofen [39]. Individuals whose diets are rich in olive oil generally exhibit reduced inflammatory markers [40]. The catechins found in green tea and red wine also protect against free radicals and oxidative cell damage [41], further inhibiting inflammatory markers and responses.

DIAGNOSING FASCIAL CONDITIONS

The question remains as to how to diagnose fascial conditions. Ultrasound has proven a reliable diagnostic tool. Studies have correlated areas of pain with differences in fascial thickness when

compared to control groups [11,42]. But not all of us have access to ultrasound equipment, or the appropriate time to use it on every patient if we do.

The most common symptoms of fascial dysfunction include:

- Decrease in local and/or general ROM
- Soft tissue pain when performing simple movements
- Compromised motor control, coordination in activities of daily living (ADLs)
- Reduced flexibility, lack of resilience
- Bad posture/body-wide patterns of compensation
- Dull aches or pains that never truly go away and/or do not respond to conventional orthopedic treatments
- Diminished proprioception and/or interoception that often manifests as a perceived clumsiness/bumping into things and uncertainty about physiological feelings

While each of these conditions could indicate a non-fascial condition, one of the simplest ways to assess if it is a fascial problem is patho-anatomical assessment. Bony landmarks related to the area of pain are palpated to look for asymmetries that would correlate with the symptoms. For example, in the case of LBP, is one iliac crest higher than the other? Does the anterior superior iliac spine (ASIS) deviate more than 7° with the posterior superior iliac spine (PSIS) in the sagittal plane? And so on.

Upon discovering such asymmetries and pointing them out to the patient, are they capable of easily correcting them? Or does the correction create further distortion and/or pain? A good rule of thumb is as follows: If asymmetries are present, manual therapy is indicated. If asymmetries are not present or are easily corrected when pointed out, a more movement-based solution is indicated.

These distinctions are important to accurately refer the patient to the proper fascia-based therapist.

FASCIAL THERAPIES

Fascia-related therapies are multiplying rapidly. They can be most easily categorized as manual or movement therapy. Many therapists combine elements of both, but that does not necessarily indicate a more efficacious treatment. The following is a list of some of the therapies with a reputation for achieving results.

- *Manual therapy*: Structural integration including Rolfing, anatomy trains structural integration drop (kinesis myofascial integration) and Hellerwork; Myofascial Release, Trigger Point Therapy, Fascial Manipulation; and Visceral Manipulation.
- *Movement therapy*: Fascial movement; MELT method; fascial fitness; and yoga. Recommended yoga modalities include therapeutic, yin, Iyengar and restorative.
- *Other*: Fascial stretch therapy, which combines assisted stretching with traction and proprioceptive neuromuscular facilitation (PNF); and acupuncture. While not generally recognized as a fascial modality, acupuncture has been shown to interact with the fascia in ways that induce mechanotransduction [43] and influence the behavior of fibroblasts [44]. A preliminary investigation has also revealed the likelihood that the meridian system of traditional Chinese medicine organizes itself along fascial planes [45]. Regional treatment for musculoskeletal disorders are also referred to as dry needling.

While certain aspects of treatment differ, they all share the quality of increasing proprioception and tactile discrimination, the diminishment of which has been shown to be involved with many pain syndromes [46,47]. To minimize adhesions, treatment is recommended by 3 weeks after surgery, sooner if superficial soreness has diminished.

The following table serves only as a general guideline. On an individual basis, therapists may be more highly skilled with treating certain conditions than others, and as such may have higher individual effectiveness ratings. It is recommended that you seek out and assess qualified practitioners in your area.

Therapies/ Conditions	Manual Therapies							
	Structural Integration	Myofascial Release	Trigger Point Therapy	Fascial Manipulation	Visceral Manipulation			
Low back pain	•	•	۲	•				
Fibromyalgia	a	\checkmark^{a}	b a					
Thoracic outlet/carpal tunnel	•	✓	۳	۲				
"Smartphone neck"	۲	۲	۲	•				
Concussion	a		~					
Scoliosis	a			~	✓ ^a			
GERD, IBS, reflux, etc.		•			•			
Running/sports injuries	•	•	•	•				
Post organ replacement	₩a		~	~	a			
Post joint replacement	•	✓	~					
Stroke contractures	~	~	۲	۳				

Note: \blacksquare , very effective; \checkmark , effective.

^a Indicates modalities work well in conjunction for this condition.

Therapies/ Conditions	Movement Therapies				Other	
	Fascial Movement	MELT Method	Fascial Fitness	Yoga	Fascial Stretch Therapy	Acupuncture
Low back pain	✓	•		•	•	~
Fibromyalgia	a a	a		\checkmark^a	e a	a
Thoracic outlet/carpal tunnel		~			۳	~
"Smartphone neck"	۲	•		•	۲	✓
Concussion	✓	✓		~		W ^a
Scoliosis	₩a	a		•		
GERD, IBS, reflux, etc.						•
Running/sports injuries	•	•	•		•	
Post organ replacement	¥			~		~
Post joint replacement	۲	۲	۲	•	۲	
Stroke contractures					~	~

Note: $\mathbf{\Psi}$, very effective; \checkmark , effective.

 $^{\rm a}$ $\,$ Indicates modalities work well in conjunction for this condition.

CASE STUDY

PATIENT PROFILE

This is the case of a 46-year-old businesswoman who likes to practice yoga, initially complaining of numbness in her left lateral forefoot in March 2016. The symptoms were mild and intermittent until 6 months later when she felt a "pull" in her low back while performing a backbend in class. After several weeks of constant LBP, she began treatment with a chiropractor who diagnosed her with a "low left pelvis". Her relief was "occasional", but after 6 weeks of care her foot numbness spread to the whole forefoot. She also reported an apparent redness and mild swelling of the left foot. There was a recurring feeling like her legs and the soles of both feet were "on fire".

She began to receive acupuncture treatments that helped initially, but overall the symptoms began to worsen. Chiropractic treatment was placed on hold, an anti-inflammatory diet was recommended and x-rays of the lumbar spine ordered.

The x-rays revealed narrowing of the disc space from L3 to S1 and a sacral anteversion. A magnetic resonance image (MRI) was ordered, but was not done due to insurance issues. A week later, tingling and numbness radiculopathies in the lower extremities worsened and the patient now had irradiation to her arms as well. A neurologist was consulted, who ordered an MRI that was again denied. After three physical therapy treatments, her left leg radiculopathy persisted. Finally, a year after her symptoms started and on the advice of her physician, she was referred to our office for evaluation and further treatment.

CLINICAL OBSERVATIONS

Overall, the patient is a healthy individual of great physicality, given her longevity in the fitness industry. Patho-anatomical assessment revealed relevant postural anomalies: the left hemipelvis had a pronounced anterior tilt in the sagittal plane. Likewise, the left leg showed a hyperextended knee. The right hip, by comparison, was within 7° of neutral. There was a lateral rotation in the left hip in the transverse plane and the left hip had a left tilt (meaning the right iliac crest appeared higher) in the frontal plane. A very slight right bend of the lumbar spine could also be seen and palpated. All of these anomalies fit with her presenting symptoms. Pain was self-rated as 6–7 on the pain scale. Neuropathy/radiculopathy 7–8 on the left leg and 4–5 on the right leg.

TREATMENT PLAN

The basic plan was for a series of weekly visits with the objective of horizontalizing the pelvis to reduce uneven tension and wear in the lower back. This would occur in a logical progression from superficial to deep. Each session would be designed to deal with the most presenting symptoms while at the same time preparing her for subsequent treatments. The duration of treatment in specific areas does not have a specific dose response, but rather is dictated by "endfeel" – the pliability of the tissue and the degree of change in the patient's comfort and mobility.

First treatment: The SBL (see section titled 'Anatomy and the Fascial System') was chosen to provide immediate relief and gauge the patient's response. As the right side of the lower SBL was locked long and the left side was locked short, lengthening and differential techniques to promote glide between muscles were used on the left. Sacral multifidi treated with appropriate compression and slow nutation and counter-nutation. Given the probability of herniation, the erector spinae group was treated by putting the patient in supported spinal flexion with deep breathing providing the requisite stretch. Sub-occipitals release.

Results: While no visible changes were present, the patient reported an ease in tension.

Recommendations: The patient revealed that she still teaches hot yoga, which seems to exacerbate her symptoms. I suggested reducing the heat by 5° in the classes.

Second treatment – Subjective: The patient reported "feeling great" for 1 hour past treatment, followed by a gradual onset of symptoms. Pain remained at 6–7. She reduced the heat in the hot yoga classes by 5° and reported a 50% reduction in burning sensation post class. The patient also shared information regarding a "difficult childbirth" in 2012 when a tailgut cyst was removed post-partum. *Treatment:* Left quadriceps in a cephalad direction. Lower deep front line. Deep posterior compartment released with slow active flexion/extension of foot bilaterally. Left thigh anterior intermuscular septum treated in cephalad direction, posterior septum caudally. Right thigh septa treated cephalad and differentially to free adhesions caused by compression due to favoring. Left quadratus. Supported erector spinae. Quadratus lumborum. *Results*: While no visible changes were present, the patient reported feeling "different" with ease in overall tension.

Third treatment – Subjective report: The patient reported feeling the "best I've felt in a long time" on the first day following the previous treatment. The second day, she still felt "good" with a gradual return of symptoms, but not as intense, by Day 3. The pain was self-rated as 5–6 on the pain scale. Neuropathy/radiculopathy 5–6 on the left leg and 2–3 on the right leg. *Treatment*: Left side: pectineus, adductor longus, gluteus medius, quadratus femoris. Bi-lateral psoas with the patient going into bridge. Erector spinae and sub-occipitals.

Fourth treatment – Subjective: Overall, the patient felt "better" for most of the week. The perception of "nerve stuff" was more prevalent for the first 2 days, but had now diminished to neuropathy/ radiculopathy 4–5 on the left leg and 1–2 on the right leg. *Objective*: The left pelvis was visibly less rotated and closer to horizontal. The overall pain level, especially LBP was reported at 4–5, with brief spikes to 6–7. *Treatment*: The focus was on refining left-side restrictions. Pectineus psoas tendon, obturator externus. Multifidi on both sides. AMPs were based on the pelvic clock. Erector spinae.

Fifth treatment – Subjective: Neuropathy/radiculopathy 3–4 on the left leg and 0–1 on the right leg. The left pelvis was within 7° of horizontal. LBP was reported at 2–3. *Treatment*: The focus was on the back functional line, which links the latissimus dorsi on the right with the gluteus maximus on the left via the lumbodorsal fascia. Bi-lateral psoas releases coupled with slow spinal extension. Coccygeal ligaments. Erector spinae were treated with the patient seated with AMP of slow extension while treatment proceeded caudally.

Sixth treatment – Subjective report: The patient reported a good week. Her check up with the neurologist was positive. An MRI was now considered unnecessary. *Treatment*: Peroneals, edges of the illiotibial band as necessary where adhesions to the vastus lateralis and biceps femoris were palpated. Iliac crest and quadratus lumborum, lattissimus. Bi-lateral with left side focus.

At this point, the next three treatments were scheduled 2–3 weeks apart. These last few treatments involved some fine-tuning of the previous sessions. By the end of the ninth and last treatment, the patient reported an 85%–90% improvement. She returned to the gym and resumed some light running. She is scheduled for a routine 6-month check-up.

CONCLUSION

Healthy fascia is vital to the regulation, maintenance and overall function of the human body. Improving tissue quality, hydration and function improves musculoskeletal performance, enhances metabolic uptake and restores resiliency and intracellular communication. Fascia-oriented interventions are suggested in cases of chronic pain as an alternative to surgery and/or medication. Even as this chapter goes to press a new space has just been discovered [48]. Called the interstitium, it's a contiguous, fluid-filled area of the body between the skin and muscles and organs. Lined with collagen bundles, it is analogous to the superficial fascia. There is also a continuity between the interstitium and the lymphatic system. The interstitium's primary function seems to be hydration and cellular transport, potentially opening up whole new frontiers of understanding and treating disease, as well as promoting health and wellness.

REFERENCES

- 1. Adstrum S. et al. (2017) Defining the fascial system. J Bodyw Mov Ther. 21(1):173–177.
- 2. Lindsay, M. (2008) *Fascia: Clinical Applications for Health and Human Performance*. Clifton Park, NY: Delmar.
- 3. Vuokko, K. (2002) Intramuscular extracellular matrix: Complex environments of muscle cells. *Exercise Sports Sci Rev.* 30(1):20–25.
- 4. Lodish, H. et al. (2000) Molecular Cell Biology, 4th edn. W. H. Freeman. New York
- 5. Stecco, C. et al. (2011) Hyaluronan within fascia in the etiology of myofascial pain. *Surg Radiol Anat.* 33(10):891–896.
- 6. Cretoiu, D. et al. (2016) Telocytes and their extracellular vesicles: Evidence and hypotheses. *Int J Mol Sci.* 17:1322.
- 7. Langevin, H. (2006) Connective tissue: A body-wide signaling network? Med Hypotheses. 66:1074–1077.
- 8. Oschman, J. (2003) Connective tissue as an energetic and informational continuum. *Struct Integration*. 31(3):5–15.
- 9. Grinnell, F. (2007) Fibroblast mechanics in three-dimensional collagen matrices (DVD recording). First International Fascia Research Congress, Boston, MA (online). Available: http://www.fasciacongress. org.
- 10. Kirkwood, J. and Fuller, G. (2009) Liquid crystal collagen: A self-assembled morphology for the orientation of mammalian cells. *ACS Biomatter Sci Eng.* 25:3200–3206.
- 11. Langevin, H., Stevens-Tuttle, D., Fox, J.R., and Badger, J.G. (2009) Ultrasound evidence of altered lumbar connective tissue structure in human subjects with chronic low back pain. *BMC Musculoskelet Disord*. 10:151.
- 12. Staubesand, J., Baumbach, K.U.K., and Li, Y. (1997) La structure fine de l'aponévrose jambière. *Phlèbologie*. 50:105–113.
- 13. Järvinen, T.A. et al. (2002) Organization and distribution of intramuscular connective tissue in normal and immobilized skeletal muscles. An immunohistochemical polarization and scanning electron microscopic study. *J Muscle Res Cell Motil.* 23(3):245–254.
- 14. Schleip, R. and Müller, D.G. (2013) Training principles for fascial connective tissues: Scientific foundation and suggested practical application. *J Bodyw Mov Ther.* 17(1):103–111.
- 15. Meltzer, K. et al. (2010) In vitro modeling of repetitive motion injury and myofascial release. *J Bodyw Mov Ther.* 14(2):162–171.
- 16. Guimberteau, J.-C. (2012) Muscle attitudes (DVD). Endovivo Productions, Pessac, France. Available www.endovivo.com.
- 17. Passerieux, E. et al. (2006) Structural organization of the perimysium in bovine skeletal muscle: Junctional plates and associated intracellular subdomains. *J Struct Biol.* 154:206–216.
- van der Wal, J. (2009) The architecture of the connective tissue in the musculoskeletal system: An often overlooked functional parameter as to proprioception in the locomotor apparatus. *Int J Ther Massage Bodywork*. 2(4):9–23.
- 19. Huijing, P.A. (2009) Epimuscular myofascial force transmission: A historical review and implications for new research. International Society of Biomechanics Muybridge Award Lecture, Taipei, 2007. J Biomech. 42(1):9–21.
- 20. Myers, T. (1997) The "anatomy trains". J Bodyw Mov Ther. 1(2):91-101.
- 21. Myers, T.W. (2013) Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists, 3rd edn. Edinburgh: Elsevier.
- 22. Myers, T. and Lesondak, D. (2010) Anatomy trains revealed; dissecting the myofascial meridians (DVD release). Singing Cowboy Productions, Pittsburgh, PA.
- 23. Wilke, J., Krause, F., Vogt, L. and Banzer, W. (2016) What is evidence-based about myofascial chains: A systematic review. *Arch Phys Med Rehabil*. 97(3):454–461.
- 24. Levin, S.M. (1981) The icosahedron as a biologic support system. *Proceedings of the 34th Annual Conference on Engineering in Medicine and Biology*, Houston, Texas, Volume 23, p. 404.
- 25. Levin, S.M. (2010) Biotensegrity and dynamic anatomy. Lecture (DVD). S.M. Levin, McLean, VA.
- 26. Kassolik, K. et al. (2013) Comparison of massage based on the tensegrity principle and classic massage in treating chronic shoulder pain. *J Manipulative Physiol Ther.* 36(7):418–427.
- 27. Ingber, D.E. (1998) The architecture of life. Sci Am. 278(1):48-57.
- 28. Ingber, D.E. (2003) Tensegrity I. Cell structure and hierarchical systems biology. J Cell Sci. 116(Pt 7):1157–1173.

- Ingber, D.E. (2003) Tensegrity II. How structural networks influence cellular information processing networks. J Cell Sci. 116(pt 8):1397–1408.
- 30. Crane, J.D. et al. (2012) Massage therapy attenuates inflammatory signaling after exercise-induced muscle damage. *Sci Transl Med.* 4:119ra13.
- 31. Banes, A.J. (2012) Mechanical loading & fascial changes: Tendon focus. Plenary lecture, Third International Fascia Research Congress (Conference Proceedings DVD), Vancouver, BC.
- van den Berg, W.B. (1998) Joint inflammation and cartilage destruction may occur uncoupled. Springer Semin Immunopathol. 20:149–164.
- Boyear, N., Galey, I., and Bernard, B.A. (1998) Effect of vitamin C and its derivatives on collagen synthesis and cross-linking by normal human fibroblasts. *Int J Cosmet Sci.* 20(3):151–158.
- 34. Kris-Etherton, P.M. et al. (2000) Polyunsaturated fatty acids in the food chain in the United States. *Am J Clin Nutr.* 71:179S–188S.
- 35. Melanson, S. F. et al. (2005) Measurement of organochlorines in commercial over-the-counter fish oil preparations: Implications for dietary and therapeutic recommendations for omega-3 fatty acids and a review of the literature. *Arch Pathol Lab Med.* 129(1):74–77.
- 36. Sander, K. and Sanders-Gendreau, K. (2007) The college student and the anti-inflammatory diet. *Explore* 3:410–412.
- 37. Craig, W. (1999) Health-promoting properties of common herbs. Am J Clin Nutr. 70 (Suppl):491S-499S.
- Aggarwal, B. et al. (2009) Molecular targets of nutraceuticals derived from dietary spices: Potential role in suppression of inflammation and tumorigenesis. *Exp Biol Med.* 234:825–849.
- 39. Beauchamp, G.K. et al. (2005) Ibuprofen-like activity in extra-virgin olive oil. Nature. 437:45-46.
- 40. Chrysohoou, C. et al. (2004) Adherence to the Mediterranean diet attenuates inflammation and coagulation process in healthy adults: The ATTICA study. *J Am Coll Cardiol*. 44:152–158.
- 41. Adcocks, C., Colin, P., and Buttle, D. (2002) Catechins from green tea (*Camellia sinesis*) inhibit bovine and human cartilage proteoglycan and Type II collagen degradation. *Vitro J Nutr.* 132:341–346.
- 42. Stecco, A. et al. (2014) Ultrasonography in myofascial neck pain: Randomized clinical trial for diagnosis and follow-up. *Surg Radiol Anat*. 36:243–253.
- 43. Langevin, H. et al. (2001) Mechanical signaling through connective tissue: A mechanism for the therapeutic effect of acupuncture. *FASEB*. J15:2275–2282.
- Langevin, H. et al. (2011) Fibroblast cytoskeletal remodeling contributes to connective tissue tension. J Cell Physiol. 226:1166–1175.
- 45. Langevin, H. and Yandow, J.A. (2002) Relationship of acupuncture points and meridians to connective tissue planes. *Anat Rec.* 269:257–265.
- 46. Lee, A.S. et al. (2010) Comparison of trunk proprioception between patients with low back pain and healthy controls. *Arch Phys Med Rehabil*. 91(9):1327–1331.
- Moseley, L. (2008) Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain. *Pain.* 137:600–608.
- 48. Benias, P.C. et al. (2018) Structure and distribution of an unrecognized interstitium in human tissues. *Sci Rep.* 8(1):4947.