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The Fascial System in Musculoskeletal Function and Myofascial Pain

Kimia Grace Ganjaei[1] · Jeremiah W. Ray[2,3] · Brandee Waite[2] · Kevin J. Burnham[2,4]

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Abstract

Purpose of Review This article aims to discuss the structure and function of fascial systems and the potential role in myofascial pain syndromes.

Recent Findings New terminology differentiates anatomic structure (fascia) and function (fascial system), improving the conceptual framework and communication. Fascia has been shown to be innervated and biologically active and may have roles in nociception, proprioception, and myofascial force transmission. A number of factors may modify the function of fascial tissues through altering stiffness. A new cell type, "fasciacytes," produces hyaluronic acid, a molecule critical to fascial lubrication. Fascial contribution to myofascial pain syndromes remains unclear, though plausible mechanisms connect them, and direct evidence of fascia-mediated pain exists. Current evidence is limited to support fascia-directed therapies for myofascial pain syndromes.

Summary Developing evidence implicates fascial tissue in musculoskeletal function and myofascial pain syndromes. Further investigation into fascial physiology and pathophysiology is needed to translate this knowledge into clinical care.

Keywords Fascia · Myofascial · Pain · Proprioception · Function · Mechanotransduction

Introduction

The term *fascia* is commonly used in anatomical descriptions; however, the specific definition has been elusive [1•]. Similarly, fascia is inherent in the concept of myofascial pain, though this nomenclature is often inconsistent, and the associated pathophysiologic understanding remains incomplete [2]. Recent work has provided more concrete terminology and insights into the role(s) that fascia may play in musculoskeletal function and myofascial pain. This review aims to provide an overview of the terminology, physiology, and pathophysiology of the fascial system and its potential roles in the musculoskeletal function and myofascial pain.

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✉ Kevin J. Burnham
kjburnham@ucdavis.edu

[1] Internal Medicine Residency, Department of Internal Medicine, UC Davis Health, Sacramento, CA, USA

[2] Sports Medicine Section, Department of Physical Medicine and Rehabilitation, UC Davis Health, Sacramento, CA, USA

[3] Department of Emergency Medicine, UC Davis Health, Sacramento, CA, USA

[4] Division of General Internal Medicine, Geriatrics & Bioethics, Department of Internal Medicine, UC Davis Health, 4860 Y Street, Suite 0400, Sacramento, CA 95817, USA

Definition and Terminology

Some of the difficulty surrounding fascial terminology may arise from the coexistence of incompletely overlapping meanings for the term fascia. More precise and useful definitions, differentiating between the anatomical term *a fascia* and a broader, functional term, *fascial system*, have been proposed (Table 1). For the purposes of this article, we will generally focus on the concept of the *fascial system*. The fascial system is a "three-dimensional continuum of soft, collagen-containing, loose and dense fibrous connective tissues that permeate the body," which allows integrated body system operation [1•].

Fascia can be broadly separated into superficial, parietal, visceral, and deep fascia. Deep fascia surrounds bones, muscles, nerves, and vasculature. For the purposes of this article, we will focus on deep muscular fascia, of which there are two subtypes: the epimysial and aponeurotic fascia. Epimysial fascia consists of multiple thin layers which cover and are strictly connected with muscle. The epimysium surrounds multiple

Table 1 Definitions and categorization of fascial terminology. Reproduced with permission from Adstrum et al. [1]

Term	Category	Definition
Fascia	Anatomic	A fascia is a sheath, a sheet, or any other dissectible aggregations of connective tissue that forms beneath the skin to attach, enclose, and separate muscles and other internal organs.
Fascial system	Functional	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, adventitiae 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-/epimysium.
		The fascial system surrounds, interweaves between, and interpenetrates 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.

fascicles while the perimysium surrounds one fascicle. The epimysium and perimysium help transmit lateral forces between adjacent muscle fibers within a single muscle [2]. Aponeurotic fascia consists of parallel layers of collagen fibers. The aponeurosis can become a tendon, which can consolidate groups of muscle fascicles into myofascial insertions or tendons. Myofascial insertions are fascial expansions that wrap across tendons and beyond a specific tendon insertion site. These insertions can connect with fascial layers of other muscle groups [3].

Properties and Physiology of the Fascial System

The properties of the fascial system can be conceptually split into microlevel (molecular and cellular responses) and macrolevel (mechanical) properties. The extracellular matrix (ECM) contains both molecular and cellular components. The cellular cytoskeleton is connected to the ECM through a molecular chain; this chain allows molecular interactions to occur in a dynamic, bidirectional fashion. Thus, functional and structural changes in the ECM can result in cellular adaptations and vice versa. Additionally, the ECM can serve as molecular storage system, housing, and releasing biologically active molecules involved in organ or tissue regulation. The emanation or activation of these agents can be triggered by mechanical stress, suggesting a mechanism by which exercise can induce regional or systemic effects [4••].

At the macrolevel, in situ animal and human imaging studies have shown intermuscular and extramuscular fascial tissues provide pathways for force transmission. The magnitude of this force transmission in vivo remains uncertain and may be affected by the stiffness of the myofascial tissue; in turn, the fascial tissue stiffness may be modified by a number of factors or the consequence of injury, disease, surgery, or aging. The myofascial network derived from multidirectional connections between fascial tissues of skeletal muscles suggests local forces (i.e., muscular contraction) may alter adjacent tissue mechanics [4••].

Signaling Within the Extracellular Matrix

The ECM plays a central role in promoting and regulating inflammatory responses to mechanical stress on the fascial system, controlling leukocyte extravasation, vascular permeability, collagen remodeling, and systemic signaling via exercise-induced myokines, which alter metabolism [4••]. Myofibroblasts are also within the connective tissue with the contractile ability [5•]. Myofibroblasts within the ECM require regulation as abnormal myofibroblast growth and excess type III collagen has been associated with fascial disorders such as Dupuytren's contracture [6].

Additional receptors discovered within fascia have diagnostic and therapeutic implications. For example, greater fascial tissue concentrations of estrogen receptors were found in pre-menopausal compared to post-menopausal women. The clinical significance of this finding requires additional research but suggests there may be sex-specific modulation of collagen synthesis [4, 7]. Endocannabinoid receptors were also found in human fascia and in myofibroblasts. While the role of endocannabinoids in pain modulation is still being fully elucidated, CB1 and CB2 receptors have been found to decrease pro-inflammatory cytokines and fibrosis. In a recent study, Fede et al.

demonstrated cannabinoid receptor agonism led to the production and release of vesicles containing hyaluronic acid (HA), suggesting the endocannabinoid system may play a role in remodeling fascial tissues [8, 9].

Viscoelasticity and Hyaluronic Acid

Cells of the deep fascia secrete HA, the predominant space-filling polymer within the loose connective tissue of the ECM. HA acts as a lubricant, facilitating connective tissue gliding [10]. Recent research suggests fibroblast-like cells, termed "fasciacytes," may play a key role in HA production [11••] (Fig. 1). Aponeurotic fascia appears to have higher average HA concentrations than epimysial fascia; however, retinacular tissue appeared to have the highest concentration [12•].

The polymer structure of HA varies with pH and temperature. Short HA chains assemble in lower and disassemble in higher environmental pH; these polymer changes alter the viscoelastic properties. Exercise, through the production of lactic acid, reduces pH, and leads to the recombination of short HA chains. This increases fascial tissue viscosity and may contribute to a feeling of stiffness after exercise. This process should reverse within approximately 30 min following normalization of pH through lactic acid clearance [3]. In contrast, HA depolymerizes as the temperature increases to 40 °C, decreasing viscosity [10].

Similarly, HA viscoelasticity is affected by pressure. Hyaluronic acid is a non-Newtonian fluid (thixotropic), reducing viscosity with increased pressure. Manual therapies increase fascial pressure, causing reduced HA viscosity and augment flow within the fascial tissues. This may suggest a mechanism through which manual therapies affect fascial tissue sliding [13].

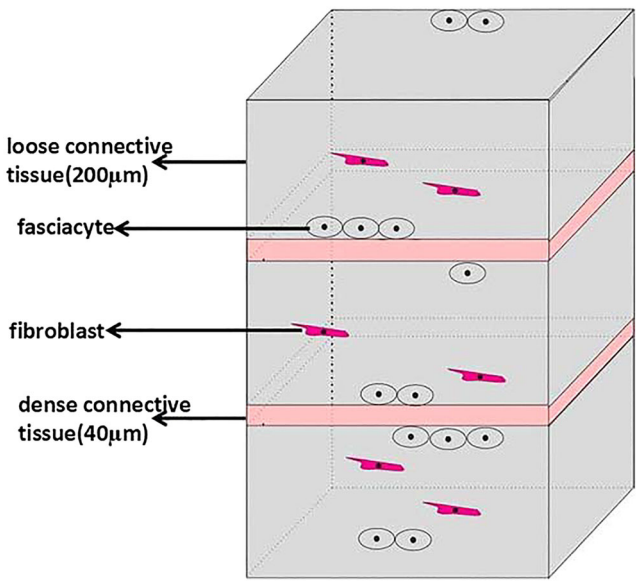

Fig. 1 Fasciacyte diagram

Fascial Tissue Stiffness

A variety of factors are thought to affect fascial stiffness (Table 2). An inflammatory reaction to fascial damage from trauma or surgery progress to healing through fibroblast proliferation and collagen deposition. However, proper healing requires remodeling of the collagen network to align with local tensile stresses. Corruption of this process may yield randomly oriented collagen fibers. Connective tissues increase in strength but become stiffer with age. Diabetes causes glycation of many peptides, including collagen, as well as alterations in the types of collagen produced. Collagen glycation and abnormal crosslinking may lead to stiffer and thicker fascia [10]. Likewise, the fibrosis in Dupuytren's contracture in patients with diabetes is thought to be due to collagen crosslinking by glycation end products [2].

Innervation

The fascial system is involved in proprioception, nociception, and active contraction. Several studies have demonstrated extensive innervation of the deep fascia, with free nerve endings of both Aδ, C, and postganglionic sympathetic fibers. Aδ

Table 2 Factors impacting mechanical stiffness of fascial tissue and their hypothesized impact. Up arrows indicate a positive effect (increased stiffness), down arrows indicate a negative effect (decreased stiffness), and double arrows an ambiguous association. *ECM*, extracellular matrix. Adapted from Zügel et al. [4]

Impact on fascial stiffness	Factor
Ambiguous (↔)	Estrogen
	Genetic predisposition
	Hyaluronan
	Tissue hydration
Decreased (↓)	Corticosteroids
	Stretch-induced tissue elongation
Increased (↑)	Aging
	Cellular contractility
	Crosslinking
	ECM matrix deposition
	Neuro-muscular diseases
Uncertain	Trauma-induced scars

fibers are predominantly sensitive to mechanical stimuli, whereas C fibers (nociceptors) respond to both mechanical and chemical stimulation; however, the mechanical stimulation threshold of the C fibers in deep fascia appears to be twice that of skin or muscle [14••].

Force Transmission and Myofascial Chains

The effect of skeletal muscles is traditionally defined as a muscle's actions over two osseous structures. However, some estimates suggest 70% of the muscular contractile force is conveyed to bone, while 30% is transmitted to perimuscular fascial elements [14••]. Subsequently, local and distant fascial connections between various muscles may also affect function. These connections are defined as myofascial chains; the existence of which could help explain referred pain and dysfunction in distant anatomic structures. A systematic review in 2016 sought evidence for these proposed chains. The authors concluded "most skeletal muscles are directly linked by connective tissue," with strong evidence for three myofascial chains (superficial back line, back functional line, front functional line) [15]. Homogeneity in this review was limited, as included studies varied in quantity and measurement of force application and transfer. In addition, most studies were performed through cadaveric dissection. Formalin fixation increases collagenous tissue crosslinking and alters hyaluronic acid, in addition to the temperature changes required to preserve specimens. Cadaveric dissection may be useful, in that it eliminates contraction of the underlying muscle, but in vivo applicability may be limited [15]. A subsequent systematic review in 2019 of the upper limb suggests connective tissue continuity between the neck and shoulder region with the forearm [16].

Fascia also possesses contractile ability independent of muscle. Myofibroblasts, sharing characteristics of both fibroblasts and smooth muscle cells, have been demonstrated in human fascial specimens from several sites, including plantar fascia, fascia lata, lumbar fascia; the highest concentration has been observed in lumbar fascia [17, 18]. The epimysium connects to muscle spindles and this allows for transmission of muscle stretch information. Aponeurotic fascia is also innervated with Ruffini and Pacinian corpuscles, and as the aponeurotic fascia forms myofascial insertions, they may coordinate balanced muscle action with the input from these mechanoreceptors [2].

Imaging of Fascial Tissues

While fascia can be imaged via computed tomography (CT) and magnetic resonance imaging (MRI), ultrasound (US) has become advantageous for both static and dynamic assessment of fascia (Fig. 2) [19]. Applying US fascial imaging research to the clinical setting remains challenging. There are no normative US data on expected fascial thickness for any fascial compartment, as there are only a few studies of healthy fascia, and there is significant heterogeneity in fascial measurement protocols—from the chosen axis, probe location, number of measurements taken, and even, occasionally, terminology defining the fascia between different studies. Additionally, patient positioning may affect fascial thickness and stiffness, further limiting inter-study comparisons [19]. Nonetheless, limited studies suggest that US measurements of fascial thickness may have acceptable reliability [20–23].

Axial strain elastography (ASE) has been used to qualitatively evaluate fascial elasticity. Due to operator variability with ASE, shear wave elastography (SWE) has emerged as a promising objective adjunct to US and clinical assessment in measuring of fascial elasticity in the diagnosis of plantar fasciitis [24].

Myofascial Pain Syndrome

Myofascial pain syndrome (MPS) is an amalgamation of sensorimotor, autonomic, and neuropathic symptoms that arise from muscle and soft tissue and have often been associated with the stimulation of myofascial trigger points (TP). TP are based on the observation by Travell and Simmons—who originally defined MPS—that palpable knots were present which referred pain to local or distal fascia through a referred pain pattern [25]. There is a lack of consensus on what constitutes a TP. A recent study of experts from 12 countries suggested that the diagnostic criteria for a trigger point include a taut band, hypersensitive spot, and referred pain anywhere from that spot. The quality of the pain from a TP could be aching, tingling, burning, or other sensation [26]. It sometimes can cause a decreased range of motion in adjacent structures [26, 27]. An active trigger point is distinguished from a latent one in that an active trigger point reproduces the patient's symptoms at a distant site [26]. However, some authors question a causal association between TP and MPS, and argue TPs are neither diagnostic nor particularly relevant in treatment of MPS [28].

MPS can manifest in acute and chronic forms; risk factors include previous trauma, poor ergonomics during repetitive activity, structural changes such as spondylosis, and systemic conditions such as hypothyroidism or certain vitamin deficiencies. MPS is common in clinical practice and typically diagnosed through a combination of history, physical exam, and exclusion of alternative etiologies [25]. There is no consensus on the exact symptomatology of myofascial pain [25, 29]. A survey among physicians noted most agreement in the following symptoms after palpation of a specific muscle area: "point tenderness" (particularly "within a tight band of muscle"), "local twitch response," "referred pain," "repeated

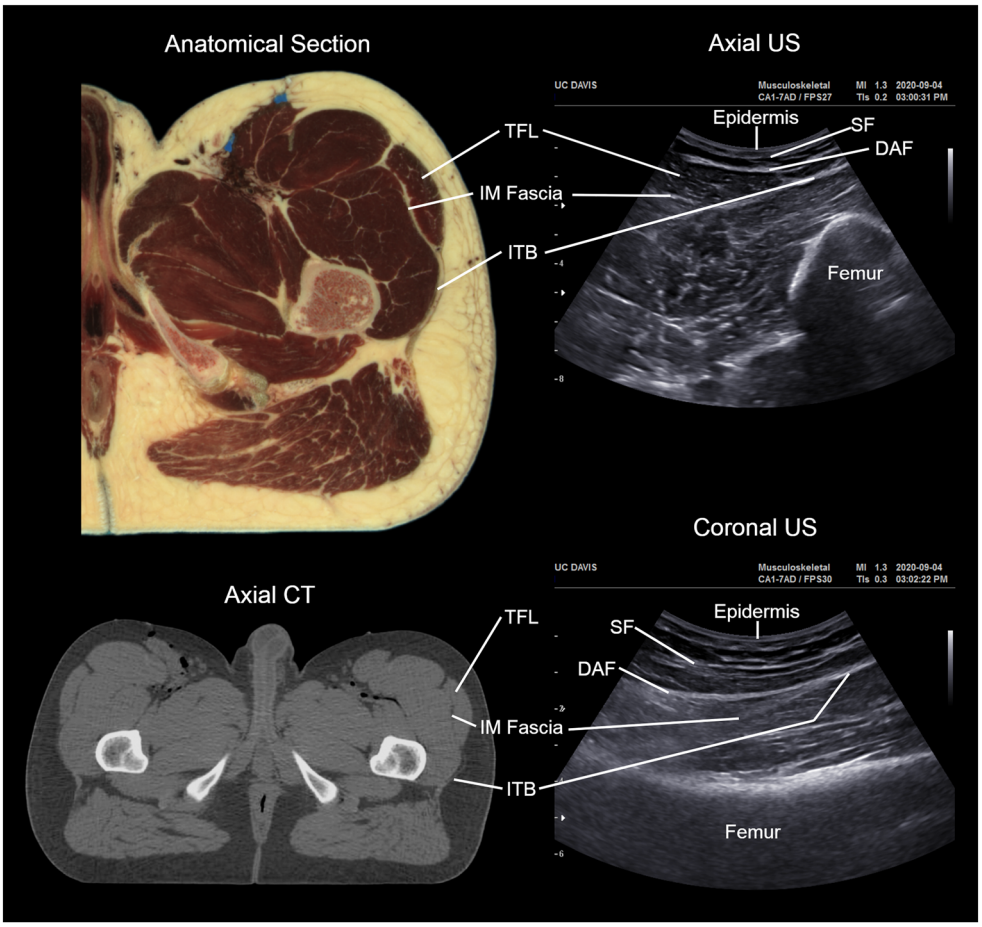

Fig. 2 Examples of deep fascia of the anterolateral thigh on anatomical cross-section, axial CT, and axial and coronal ultrasound. CT, computed tomography; DAF, deep aponeurotic fascia; IM, intermuscular; ITB, iliotibial band; SF, superficial fascia; US, ultrasound. Anatomic section and axial CT image from the Visible Human Project®, courtesy of the U.S. National Library of Medicine. Ultrasound images are from the authors

palpation reproduces usual pain" [29]. A local twitch response to an active TP on physical examination has been anecdotally reported to be helpful, but this is not universally sought for in diagnosis [26, 29].

The Role of Fascia in Myofascial Pain

MPS has historically been characterized as a muscular syndrome and the contribution of fascia to MPS is nebulous to most physicians. Recent research has identified various pathologies in the deep fascia. Many theories propose to explain the connection between these identified pathologies and symptomatology; however, the precise mechanisms driving MPS remain poorly understood. A major etiology for MPS is prolonged muscle contraction through repetitive activity, often in the presence of poor ergonomics. Rather, the pathophysiology of MPS could be due to a mismatch in muscle energy requirement and expenditure and subsequent neuromuscular dysfunction, which manifests as an alteration in myofascial physiology [30].

During normal muscle contraction, blood flow through the low-pressure capillary bed is temporarily blocked. Once muscles relax, normal blood flow resumes. When muscles are persistently in a state of low-level contraction, the intramuscular pressure is high enough to impair oxygen diffusion to muscle and fascia and impede oxidative metabolism. Certain muscles may be at higher risk of generating pressures which impede capillary flow; for example, even 10% of the maximum voluntary contraction of the supraspinatus creates pressures greater than the associated capillary bed [31]. Chronic hypoxia from capillary occlusion decreases local pH through the production of lactic acid. As discussed previously, the macrostructure of HA is inversely related to pH, which has been theorized to contribute to "densification" of the fascia. Stecco et al. have suggested that muscle overuse increases large molecular weight HA and hence viscosity. This may encumber the normal "gliding" of muscle within the loose connective tissue and alter the local biomechanics [2, 3].

Muscle microdilution of TP in patients with myofascial pain demonstrated elevated levels of bradykinin, substance P, serotonin, inflammatory cytokines, and calcitonin gene-related peptide (CGRP), which affect nociception. The release

of these mediators is thought to relate to local tissue hypoxia [32]. As described previously, fascial tissues contain nociceptors; inflamed thoracolumbar fascia has been observed to have an increase in substance P fibers [33]. In addition to mediating nociception, substance P increases local microcirculation and vessel permeability, leading to edema, which activates fascial mechanoreceptors through pressure [34]. In a clinical correlate, patients with the patellofemoral syndrome were found to have elevated substance P within the lateral retinaculum on immunohistochemical analysis [35].

Direct evidence of fascia-mediated pain also exists. High-frequency stimulation of lumbar fascia was shown to increase pain sensitivity long after stimulus removal, suggesting the importance of hyperalgesia from long-term potentiation in central processing of the initial fascial injury [36]. Interestingly, hypertonic saline injected into thoracolumbar fascia elicited more pain than injection in muscle, though only muscle injection caused pressure hyperalgesia [37].

An alternate pathway influencing myofascial pain may be impaired proprioception. Direct fascial tissue injury, such as a ligamentous sprain, may alter its proprioceptive ability. Stecco et al. showed damage to the ankle retinacula altered proprioception and functional ankle stability [38]. A defect or scar may corrupt the proprioceptive map of the fascial system and contribute to persistent instability of the previously injured joint. Inadequate biomechanical compensation may lead to repeated injury, instability, inflammation, and pain [2]. A potential clinical connection can be hypothesized in hypermobile Ehlers-Danlos syndrome (hEDS). hEDS patients have abnormally lax connective tissue, though the genetic and pathologic bases remain unclear. These patients also demonstrate impaired proprioception, associated joint pain, and myofascial pain [39–42].

Myofascial Pain Therapeutics and Fascia

Injections

There are vast treatment options for myofascial pain, but this article will focus on therapies potentially targeting fascia. Despite common use in practice, dry needling and pharmacologic injections of muscles and TPs using lidocaine and botulinum toxin against placebo have yielded mixed results with significant heterogeneity between studies; these options at most provide short-term pain relief [43]. Injections targeted to the fascia may provide improved results. Hydrodissection is defined as using injected fluid to dissect fascial places under US guidance. This has been used to free entrapped nerves and to treat tendinopathy [44–46]. In an intriguing study, Domingo et al. reported a case series of 25 patients treated with an interfascial block for myofascial pain of the upper muscles of the back. 5 cadaver injections were also performed to demonstrate the injectate within the interfascial space. In 25 participants, local anesthetic interfascial injection improved mean visual analog scale pain scores immediately post-injection (6.4 to 1.0 at rest; 7.6 to 1.6 in motion). The efficacy of this treatment is suggested to derive from the presence of nerves within the fascia [47].

In the authors' experience, interfascial injections for upper back myofascial pain have yielded more durable results than standard trigger point injections. To contrast, TP injections are typically performed by injecting a small amount of fluid into the identified muscular area of dysfunction, followed by passing the needle through the area and seeking a muscle twitch response. In an interfascial injection (or hydrodissection), US is used to guide deposition of a larger volume of injectate between the fascial layers. Similarly, in patients with chronic myofascial hamstring pain, we have observed improvement with large volume hydrodissection of areas of impaired fascial movement on US. The mechanism of this improvement remains unknown; however, alterations in ECM viscosity or nociceptor stimulation within the fascia seem plausible.

While not specifically focused on myofascial pain, Raghavan et al. injected human recombinant hyaluronidase into upper limb muscles and found significantly improved muscle stiffness from spasticity after cerebral injury [48]. Patients undergoing this therapy improved both average passive and active range of motion with a statistically significant improvement in many upper limb movements ($p < 0.05$). Immobilization is also associated with increased viscosity of HA [5], which suggests that hyaluronidase has therapeutic potential in myofascial symptoms.

Myofascial Release

A proprietary treatment protocol, "Fascial Manipulation®," based on fascial system theory, has been shown in small randomized trials to improve symptoms in nonspecific low back pain and chronic ankle instability [49–51]. However, a systematic review of myofascial release treatment effects on fibromyalgia and low back pain did not have effect sizes to reach the minimally clinically important difference for pain and disability for most of the included studies [52•]. Another systematic review of instrument-assisted soft tissue mobilization (IASTM) found moderate to large effect for improved immediate range of motion in uninjured patients (effect size: 1.11–2.48) with mostly moderate effect 2–4 weeks post treatment (effect sizes: 0.89–1.62). This study also found significant immediate improvement in pain with carpal tunnel syndrome immediately post treatment (effect size > 1.50) with small to moderate improvement in grip and pinch (effect sizes 0.54–0.82) and small to moderate improvement in thoracic back pain and functioning at 1 week (effect sizes < 0.79) [53•]. Foam rolling pre-exercise was found to increase

flexibility with an effect size of 0.34 (CI 0.12, 0.55, $p < 0.01$) but had a questionable improvement in sprint performance (effect size 0.28, CI − 0.01, 0.57, $p = 0.06$) in another systematic review [54•]. Most of these systematic reviews have been limited by heterogeneous studies of variable quality.

Extracorporeal Shock Wave Therapy

Two recent systematic reviews of MPS of the trapezius concluded extracorporeal shock wave therapy (ESWT) may alleviate pain; they differed in whether ESWT was superior to other conventional therapies (e.g., dry needling, trigger point injection) [55, 56]. Another systematic review found ESWT improved pain and the pain pressure threshold with large effect sizes in MPS of the neck and shoulder regions, but did not significantly reduce disability [57•]. In plantar fasciitis, ESWT appears to be effective for reducing pain and was found to be superior compared with other treatments at 1–6 months post treatment and is likely safe at 1 year of follow-up [58–60]. The mechanisms by which ESWT may impact MPS remain unclear. Hypotheses include increased muscle perfusion or angiogenesis or altering pain signaling pathways [57•]. ESWT has been shown to cause selective degeneration of unmyelinated nerve fibers in a rat model [61]. Further research is needed to ascertain if ESWT alters fascial tissues.

Additional Therapeutic Interventions

Limited additional evidence may link other therapeutic modalities to fascia, including heat, cannabidiol, and acupuncture:

- Superficial heat alleviates pain and improves pain tolerability with applied pressure in patients with neck pain ($p < 0.01$) and plantar heel pain ($p < 0.01$) as compared with sham heat [62]. A potential mechanism for this observation is that heat may lower fascial viscosity through depolymerization of HA [3].
- Cannabidiol (CBD) is being sold commercially for pain, but there is still limited research on its effects. The presence of endocannabinoid receptors in fascia suggests a potential therapeutic target. A study of myofascial pain from temporomandibular disorder found that after 2 weeks, transdermal CBD reduced pain by 70%, while the placebo did not show any statistically significant difference. In addition, CBD reduced EMG activity of the masseter muscle compared to placebo (left: 11% versus 3.3% reduction, right 12.6% versus 0.23% reduction) [63•].
- A key reaction elicited during acupuncture is "de qi," termed "needle grasp." The needle is inserted into the patient and then rotated, after which resistance to needle removal develops. This phenomenon appears to relate to the formation of a connective tissue "whorl" around the needle, rather than muscle-needle interactions. Additionally, Langevin et al. demonstrated correlations between intermuscular or intramuscular connective tissue planes with more than 80% of acupuncture points and 50% of meridian intersections [64]. Based on these results and other findings, Finando et al. argue that the fascia may be the mechanism of action in acupuncture [65].

Conclusions

Evolving information suggests fascia plays a role in the musculoskeletal function and myofascial pain. Further research into the physiology and pathophysiology of the fascial system, delineation of myofascial chains, optimal techniques for fascial imaging, and clinical trials targeting the fascial system in pain syndromes will be crucial to translate these findings into clinical care.

Compliance with Ethical Standards

Conflict of Interest KB—None
JWR—None
KGG—None
BW—non-conflict disclosure—Scientific Advisory Board, Level 41 AI

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