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Abstract

Fascia represents an intricate system of connective tissue that permeates throughout the human body. Its matrix of continuous fibers support, protect, divide and suspend both superficial and deep anatomical structures. While once thought to be a passive mesh network, new evidence suggests fascia is much more complicated. Now recognized as an active physiological component of the human body, myofascial health and function has been given much attention clinically. Of the techniques aimed to treat and restore fascial structure and function, myofascial release has been found to promote stability, increase range of motion and most importantly alleviate musculoskeletal pain. This form of soft tissue therapy deserves more academic and clinical attention for its positive effects on the fascial health.

Keywords

myofascial release, myofascia, fascia, soft-tissue therapy, connective tissue

Disciplines

Medicine and Health Sciences | Musculoskeletal System | Tissues

Comments

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Myofascial Release

Introduction:

In the modern era of clinical medicine, musculoskeletal dysfunction is almost exclusively a diagnosis of isolated muscles, bones, ligaments and tendons. Having reduced much of our anatomy into parts, we have simultaneously lost consideration of the whole system collectively (Carey, 2010; Schleip, Jager, & Klingler, 2012). By analyzing the body components in isolation, the underlying organic structure which stabilizes and immobilizes these component parts is often overlooked. A form of connective tissue, fascia has likely suffered reduced scientific attention given its uninteresting categorization and suspected function. Fortunately, recent evidence suggests that fascia is not inert like once believed, and that its function extends well beyond serving as a mechanical connector. In fact, its role is so heavily implicated in musculoskeletal dynamics that studies suggest that 85% of people will experience myofascial pain/dysfunction at one point in their lives (Cathcart, McSweeney, Johnston, Young, & Edwards, 2019; Jafri, 2014; Kalichman & Ben David, 2017; Shah et al., 2015). With such significant health implications, fascia has been an active and ongoing topic of research, with many mechanisms and pathways still yet to be understood.

Of the several techniques associated with treating fascial impairment, myofascial release is the most popular and well-studied. Myofascial release is a form of soft-tissue therapy which targets fascial restrictions and is used to relieve pain, restore function and normalize fascial tissue health and length (Jafri, 2014; Kim, Park, Goo, & Choi, 2014; Laffaye, Da Silva, & Delafontaine, 2019; Simmonds, Miller, & Gemmell, 2012; Tozzi, 2015a; Tozzi, 2015b). While various techniques of myofascial release exist, the most prevalent forms involve manual manipulation and self-myofascial release (i.e. foam rolling). It is the purpose of this paper to articulate the newly understood function and physiology of fascia and to analyze the effectiveness and suspected mechanisms behind myofascial release.

What is fascia?:

In its simplest and often limited understanding, fascia refers to the passive, three-dimensional network of connective tissue that permeates throughout the human body. Providing structural support, cushioning and attachments to other tissues, fascia is often neglected when compared to the "active" components it envelops (i.e. muscles, tendons and ligaments) (Higgins, 2011; Schleip et al., 2012; Tozzi, 2015a; Tozzi, 2015b). While true, this explanation improperly communicates how truly pervasive and valuable this tissue really is. Unable to be segmented into parts like bones and muscles, the continuity of this ubiquitous structure must be recognized from a broader perspective. Only then is it apparent that this interconnected matrix surrounds, suspends, supports, protects, connects and divides all skeletal, muscular, and visceral components of the body (Barnes, 1997; Chaitow, 2012; Grieve et al., 2015; Tozzi & Ost, 2012; Tozzi, 2015a; Tozzi, 2015b). From regions of thick fascial density in the lumbar region of the spine, to the miniscule coverings of myofibrils, fascia can be responsible for up to 60% of one's muscle mass (Carey, 2010).

Like most connective tissues, fascia is largely composed of collagen fibers, elastin and ground substance wound into bundles that run parallel to the natural lines of tension in the body (Tozzi & Ost, 2012; Tozzi, 2015a; Tozzi, 2015b). Fibroblasts and fibrocytes are also necessary components of the tissue as they are responsible for maintaining the cellular environment and producing collagen (Simmonds et al., 2012). Together with other connective structures, fascia is derived from mesenchyme; the embryonic foundation for bone, cartilage, blood and lymph. While all fascia is fundamentally similar, fascial components slightly vary based upon the layers in which they permeate within the body; superficial, deep or serous connective layers. For instance, superficial fascia consists of increased amounts of adipose tissue, where as deep fascia contains more densely packed collagen fibers (Beardsley & Skarabot, 2015; Higgins, 2011; Price, 2008).

Fascia itself is an umbrella term that refers to the biologic fabric that envelopes muscles to joint capsules and organ capsules, but this broad categorization should serve as an indication as to how all-encompassing this component is to the musculoskeletal system (Carey, 2010; Langevin & Huijing, 2009; Schleip et al., 2012). The extent of these connective tissues can be more fully understood when one considers that cadaver dissections can isolate fascial bundles that originate in the feet and span the whole length of the body up to the skull (Carey, 2010; Grieve et al., 2015; McKenney, Elder, Elder, & Hutchins, 2013). Acting as one interconnected entity overlapping in various orientations, fascia can be considered the body's cohesive matrix (Chaitow, 2012; Schleip, 2003a).

Of the structural functions associated with connective tissue, fascia most importantly serves to transmit mechanical forces between surrounding muscles and body systems. Acting as a tension distributor, fascia and the rest of the connective network are pulled from varying angles and expected to dynamically distribute tension to allow for unrestricted, locomotive and coordinated movement. (Beardsley & Skarabot, 2015; Carey, 2010; Chaitow, 2012; Laffaye et al., 2019; Schleip et al., 2019). According to a lecture from Anthony Carey, up to half of all force generated in a single contraction is transmitted to nearby connective tissues and muscles (2010). Lumbar fascia, for example, has been shown to transfer force to the hips and lower limbs in order to provide stability to one's lumbar spine and pelvic girdle. Considering the integrated network of this scaffolding, tension created in one region of the body will ultimately correlate to tension in another. Thus, fascia must manage these dynamic forces to allow for coordinated mobility (Tozzi & Ost, 2012).

Utility of Fascia:

Until recently, fascia was thought to be largely an inert structure that alone served mechanical purposes. However, established and ongoing research suggests a deep complexity within the function and involvement of fascia. It is now believed that fascia functions as a very active, dynamic and plastic tissue that is fundamental to the economy of the body and its health (Laimi et al., 2018; Tozzi, 2015a; Tozzi, 2015b). No longer disregarded, myofascia is now studied for its involvement in cell signaling, excitation-contraction coupling, neuromuscular inputs, local circulation and energy metabolism. Serving roles well beyond mechanical purposes, fascia's utility has become an active and ongoing topic of study, with much of its complexity yet to be understood (Jafri, 2014). From dense collections of nerves to contractile muscle cells, newly found fascial components suggest a much more impactful purpose (Simmonds et al., 2012).

One important finding within fascia has been the discovery of its contractile elements and properties. First identified as containing actin and myosin filaments, fascia is now known to contain smooth muscle cells within its collagen matrix. While unexpected when first discovered, these contractile cells are better understood when considering their fibroblast origins. As fibroblasts differentiate into myofibroblasts, they too gain muscle cell building properties (Carey, 2010; Schleip, 2003a; Schleip, 2003b; Schleip et al., 2019). Presumably, it is the expression of myofibroblasts within fascia which ultimately leads to contractility and the creation of tension (Tozzi & Ost, 2012). Combined with evidence of changes in fascial tonus, the presence of these muscular cells suggests that the facia has a degree of active contractility and regulation of our musculoskeletal dynamics. In a recent study, Schleip and colleagues found evidence that fascial shortening and stiffening occurs often and that myofibroblast density seems to regulate such contractile properties (2019). Uniquely, this same study found that fascial contractility occurs at a much slower rate than their muscular counterparts; at least two times slower (Schleip et al., 2019). The time frame of such contractions precludes fascia from actively providing short-term, direct effects on mechanical motion. Considering these calculations, the current assumption is that fascia develops pre-tension on muscular structures and ultimately plays an important role in neuromuscular coordination. Recent evidence also has concluded that long-term fascial contractility leads to collagen matrix remodeling. In other words, fascia, like muscle, changes its structure in response to the local forces/demands its subjected to (Ajimsha, Daniel, & Chithra, 2014; Carey, 2010; Chaitow, 2012; Schleip et al., 2019). Such findings conclude that while fascia may not actively regulate stability on a moment to moment basis, the prolonged altering of tension and mechanosensation likely contributes to necessary coordination (Schleip et al., 2019).

A neural component of fascia was first speculated in the early 2000's when both myelinated and unmyelinated nerve fibers were found to exist within the thick collagenous structures of fascia. Along with finding rich vascular structures in deep layers of fascia, several studies have identified neural innervation of the fascia. More specifically, fascial tissues are densely innervated by mechanoreceptors including Pacinian (which reduce mechanical sensitivity), Ruffini corpuscles (mechanoreceptors) and interstitial Type III/IV mechanoreceptors (Arguisuelas et al., 2019; Beardsley & Skarabot, 2015; Simmonds et al., 2012). Proprioceptors, including Golgi receptors, and nociceptors have also been isolated in connective tissues (Carey, 2010; Schleip, 2003a). With such densely varied neural components and a known contractile property, fascia has been appropriately linked to the autonomic nervous system. With direct communication to the regulatory nervous system, fascia can be characterized as a responsive, adaptable and plastic tissue (Bertolucci, 2008). Given this deeply supported neural involvement, fascia is now suspected to play an important role in dynamic proprioception and structural adaptability to autonomic signaling (Bertolucci, 2008; Tozzi & Ost, 2012).

The newly identified matrix of vascular components also implicates fascia in cellular signaling and the regulation of homeostatic agents. From hormones like norepinephrine and calcitonin to various proteins, enzymes and even oxygen, the blood dynamics in the connective tissue likely play a contributing role in the distribution of necessary biochemicals (Barnes, 1997; Higgins, 2011; Jafri, 2014). Associated with such functions, a fluid flow model of myofascia assumes that the fascial ground substance is the conduit of necessary water that rehydrates the surrounding tissues (Carey, 2010; Laffaye et al., 2019; Schleip, 2003a; Tozzi & Ost, 2012; Tozzi, 2015b). The attention given to fascia within the past twenty years highlights the newfound importance the tissue plays in maintaining and regulating our musculoskeletal structure.

Fascia Pathophysiology:

As with all other anatomical structures, fascia is not immune to damage or dysfunction. In healthy circumstances, fascia is as a pliable, resilient and dynamic structure that functions in one interconnected matrix. Unfortunately, these conditions are prone to disruption from several causes. Acute injury of connective tissues is often the result of trauma, immobilization or repetitive movement injuries which reduce the sliding ability and extensibility of fascia, ultimately contributing to tension, pain, neuromuscular dysfunction and impaired functional capacity (Barnes, 1997; Higgins, 2011; Kim et al., 2014; Meltzer et al., 2010).

The presentation of such connective injuries is referred to as myofascial trigger points. Commonly arising in the perimysium (the connective covering of muscle fibers), myofascial trigger points are highly localized areas of hypercontracted and stiffened tissue that are often palpable, sensitive and painful (Jafri, 2014; Kalichman & Ben David, 2017; Schleip et al., 2019).

The development of such trigger points arises originally from an injury that initiates an inflammatory response within a focal region of one's musculoskeletal system. Such inflammation can be triggered directly from chemoattractants at the injured site or following an exacerbated sympathetic neural response (Jafri, 2014; Kim et al., 2014). Inflammatory responses, while important, form cellular adhesions to restrict and reduce subsequent injuries. Unfortunately, such adhesions also introduce barriers and limitations within the often-pliable fascial network (i.e. collagen formation, scar tissue) (Carey, 2010). Studies show that inflammatory responses also result in increased fascial apoptotic rates leading to significant losses in myofibroblast density (Meltzer et al., 2010). Considering the homeostatic and regulatory functions of myofibroblasts in maintaining the connective tissue matrix, it is conceivable that such an inflammatory response would lead to biomechanical and architectural dysfunction within the fascial system. When combined with the adhesive barriers formed as a result of inflammation, the fascial network becomes stiff, brittle, and incapable of adequately distributing tension.

The stiffening of the fascial network understandably places both stress and limitations on the musculoskeletal system at large. The continuity of the fascia only serves to exacerbate dysfunction, as tension in one part of the body will overflow and create undue tension at other neighboring sites (Ajimsha, 2011; Ajimsha et al., 2014; Grieve et al., 2015). A slippery slope, this excessive and increased pressure can only help to potentiate further sympathetic stimuli as the body tries to overcome these musculoskeletal restrictions (Jafri, 2014; Kim et al., 2014). Like a game of tug of war, these sympathetic stimuli lead to increased muscle contractility, creating ever more tension upon the system (Higgins, 2011) Similarly, the pressure generated within this network is believed to result in restricted vascularization. Like a domino effect, this issue may lead to ischemic, acidic (pH) and viscous tissue which only destabilizes the impaired fascial network more (Barnes, 1997; Higgins, 2011; Price, 2010; Tozzi & Ost, 2012). As alluded to, the mechanisms by which fascial trigger points are initiated, amplified and perpetuated are variable and are likely due to a range of contributing factors (Shah et al., 2015)

Given this irregular and unregulated tension within the musculoskeletal system, it is not difficult to ascertain the immense clinical implications of such injuries. Due to the way fascia permeates through muscles and the inability of the connective tissue to distribute force properly, this network creates major muscular imbalances in length and in recruitment. Ultimately these dysfunctions can lead to functional impairments such as decreased neuromuscular coordination, decreased strength, decreased range of motion, decreased muscular endurance and altered structural alignment (Higgins, 2011; Ozsoy et al., 2019). Aside from functional losses, damage to one’s fascia in the form of trigger points oftentimes causes significant pain and discomfort. Again, considering fascia’s rich innervation of neural components, it is fair to reason nociceptors are activated under such consistent and strong pressures. Unfortunately, given the ranging factors that aggravate and perpetuate these fascial injuries, clinical presentations often are chronic and quite debilitating; this disease-like presentation is referred to as myofascial pain syndrome (Higgins, 2011; Jafri, 2014; Shah et al., 2015)

In helping to understand the population of those that are most affected by myofascial pain, it is necessary to specify that both age and psychological stress can serve as predisposing factors. The aging process has been found to dramatically change ones fascial structure in terms of decreased pliability, resiliency, altered alignment and increased thickness (Carey, 2010; Ozsoy et al., 2019; Schleip et al., 2019). Similarly, psychological stress has been tied to an increase in sympathetic stimulation and hypercontractility due to certain levels of hormones associated with stress (Jafri, 2014).

Fascial Therapy:

While several techniques exist to relieve myofascial restrictions and to restore proper neuromuscular function, the most widely practiced model is myofascial release. While myofascial release is a broad term that represents a handful of massage-like strategies, all of them aim to normalize the extensibility and sliding properties of fascia while reducing the pressure on affected pain receptors (Ajimsha, 2011; Ajimsha et al., 2014; Cathcart et al., 2019; Laimi et al., 2018). The popularity and legitimacy of this technique has been on the rise as scientists gain a fuller understanding of the full extent of fascia's biomechanical, signaling and homeostatic functions. Although a handful of systematic reviews have cast doubt on the effectiveness of myofascial release, many more research studies have recently demonstrated the technique's array of benefits (Ajimsha et al., 2014; Laimi et al., 2018; McKenney et al., 2013)

As aforementioned, myofascial release represents a handful of techniques that aim to alleviate fascial damage and restore mobility. All forms of myofascial release involve manipulation of the myofascia through low-load, long duration stretches (Tozzi & Ost, 2012). The two subsets of myofascial release include both direct and indirect pressures. Direct myofascial techniques exert sustained pressure whereas indirect techniques use gentler pressure for longer durations. In the clinical treatment of myofascial injury, the most common method for applying these pressures is through manual massage. Offered by skilled practitioners, these myofascial release therapies involve the stretching of fascia both to and beyond one's collagenous barrier (Laimi et al., 2018; Tozzi & Ost, 2012). Separately, self-myofascial release is a technique often used for more acute fascial injuries and for general injury prevention. Self-myofascial release is most commonly performed using a foam roller. This technique works under the same principles of manual release, however in this case the individual uses their own body weight to produce the pressure on the damaged soft tissue (Beardsley & Skarabot, 2015; Grieve et al., 2015; Kalichman & Ben David, 2017; Laffaye et al., 2019; Laimi et al., 2018). Just like how myofascial dysfunctions are affected by a wide range of factors, the mechanisms by which myofascial release works are broad and not fully understood. Nonetheless, myofascial release presumably works via structural, homeostatic, and neural mechanisms.

One proven effect of myofascial is its modification of tissue architecture. The pressure stimulation exerted on the soft tissues of the body has been proven, through ultrasound imaging, to reduce thickness, collagen density and adhesion of the fascial network (Chaitow, 2012; Tozzi & Ost, 2012; Yoshimura et al., 2019). The stretching of soft tissues in myofascial release is also expected to lengthen elastin within the fascia; making it more pliable. Separately, manipulation of these tissues has been shown to influence the synthesis of proteoglycans and collagen, resulting in rehabilitation of the fascia (Chaitow, 2012).

Another mechanism through which this therapy is said to work is by increasing bloodflow to the restricted tissues. As with all massages, manipulation helps evacuate and circulate blood faster in tissue; especially restricted and ischemic tissues. Even increased microcirculation at the level of the fascia is expected to increase with myofascial release techniques (Jafri, 2014). Aside from providing more oxygen and nutrients to weakened tissue, the increased fluid flow has also been found to stimulate fibroblast proliferation and the differentiation between fibroblasts and myofibroblasts. Hence, myofascial therapy is assumed to play a role in fibrogenesis and fascial repair (Tozzi & Ost, 2012).

Another suspected pathway for how myofascial release restores health and function of the fascia is through changes in viscosity of the connective tissue’s ground substance. Theorized by the fluid flow model, studies have shown that fluid dynamics within the fascia have an active role in establishing the viscosity of the extracellular matrix and therefore the pliability of the soft tissues. Like squeezing water out of a sponge, massaging musculoskeletal tissues can help extravasate fluid and change fascia viscosity from a gel-like state to something more fluid (Carey, 2010; Chaitow, 2012; Schleip, 2003b). The compression provided by manipulation is expected to help dispel inflammatory mediators, metabolic wastes and excess fluid (Laffaye et al., 2019; Tozzi & Ost, 2012). In this mechanism, the technique of myofascial release helps to restore pliability of the fascia and ultimately mobility and range of motion of the injured site.

The performance of myofascial release is also proven to promote and improve healing of the injured site. This technique has a demonstrated affect on an individual's inflammatory response. Acting through mechanically stimulated fibroblasts, myofascial release helps to reduce edema and the inflammatory response in order for the body to start healing sooner (Cao, Hicks, Zein-Hammoud, & Standley, 2016; Kim et al., 2014; Meltzer et al., 2010). Evidence of this has effect on inflammation has been shown by normalized apoptotic rates and decreased delayed onset muscle soreness following myofascial release (Jafri, 2014; Meltzer et al., 2010).

The last, and most important mechanism by which myofascial release attains favorable outcomes is through its affect on neuromuscular physiology and the autonomic system. Because fascia is densely innervated by mechanoreceptors that are sensitive to manipulation, an increase in the afferent connection central nervous system will result in a decrease of sympathetic activation, and therefore a reduction of myofascial tonus (Arguisuelas et al., 2019; Bertolucci, 2008; Schleip, 2003a; Tozzi & Ost, 2012).

Regardless of the mechanisms by which the therapy is believed to work, myofascial release studies have yielded several favorable outcomes. Several studies have found the treatment to be effective at alleviating chronic-lower back pain (Ajimsha et al., 2014; Arguisuelas et al., 2019; Kalichman & Ben David, 2017; Simmonds et al., 2012). Other research has demonstrated myofascial release is successful at increasing range of motion in major joints (i.e. hip, knee & ankle) (Beardsley & Skarabot, 2015; Cathcart et al., 2019; Grieve et al., 2015; Laffaye et al., 2019; Yoshimura et al., 2019). Other scientific papers have demonstrated benefits of myofascial release on stability and function (Ozsoy et al., 2019; Simmonds et al., 2012).

The Future of Fascia:

With a newfound interest from the scientific community, fascia is likely to serve as a major focus in future musculoskeletal research. Assuming advances in tissue imaging I imagine the components of fascia will become better understood and more regionally studied in the body (ex. How does myofascia in the lumbar region vary from myofascia in the plantar?) (Schleip et al., 2019). Given the successes of myofascial release in alleviating injured tissue and normalizing function, its expected that the technique continues. With the current techniques of myofascial therapy so variable, much of the systematic reviews of the treatment appear diluted with inconsistent methods. For this reason, I suspect myofascial techniques will become more and more regulated in order to optimize the treatment’s effects. It is also likely that further research will be performed to better understand the complex networks that are involved in myofascial release (ex. How is pain reduced through myofascial therapy?) (Laffaye et al., 2019).

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