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Active fascial contractility: Fascia may be able to contract in a smooth muscle-like manner and thereby influence musculoskeletal dynamics

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Summary Dense connective tissue sheets, commonly known as fascia, play an important role as force transmitters in human posture and movement regulation. Fascia is usually seen as having a passive role, transmitting mechanical tension which is generated by muscle activity or external forces. However, there is some evidence to suggest that fascia may be able to actively contract in a smooth muscle-like manner and consequently influence musculoskeletal dynamics. General support for this hypothesis came with the discovery of contractile cells in fascia, from theoretical reflections on the biological advantages of such a capacity, and from the existence of pathological fascial contractures. Further evidence to support this hypothesis is offered by in vitro studies with fascia which have been reported in the literature: the biomechanical demonstration of an autonomous contraction of the human lumbar fascia, and the pharmacological induction of temporary contractions in normal fascia from rats. If verified by future research, the existence of an active fascial contractility could have interesting implications for the understanding of musculoskeletal pathologies with an increased or decreased myofascial tonus. It may also offer new insights and a deeper understanding of treatments directed at fascia, such as manual myofascial release therapies or acupuncture. Further research to test this hypothesis is suggested.

Introduction

Dense irregular connective tissue sheets in the human body — such as aponeuroses, joint capsules, or muscular envelopes like the endo-, peri- and epimysium — are usually referred to as fascia. Ligaments and tendons may be regarded anatomically as local thickenings of fascial sheets, which are adapting to increased local tension with a denser and more parallel fiber arrangement. Aside from ligaments and tendons, several other examples demonstrate that fascia plays an important role in musculoskeletal dynamics: stiffness of the plantar fascia contributes to stability of the foot [1]; the lumbar fascia limits spinal mobility [2]; and tension transmission across the epimysium contributes to muscle force [3,4].

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While this is currently accepted medical knowledge, it is also assumed that fascia is solely a passive contributor to biomechanical behavior. Contrary to this common conception, the authors propose the hypothesis, that human fascia may be able to spontaneously adjust its stiffness in a time period ranging from minutes to hours and thereby contribute more actively to musculoskeletal dynamics. If verified by future research, the existence of active fascial contractility could have implications for the understanding and treatment of musculoskeletal disorders which are associated with increased or decreased myofascial tension or with diminished neuromuscular coordination. The authors will review here four general indications as well as two experimental in vitro reports as evidence for the hypothesis. Finally, the implications of this new perspective will be discussed and suggestions will be offered for testing the hypothesis.

Evidence

The presence of contractile cells in fascia

Recent findings by Spector and others have shown that fibroblasts, as well as chondro- and osteoblasts, are ''connective tissue cells with muscle'', i.e., that they have an innate capacity to express the gene for α-smooth muscle actin (ASMA) and to display contractile behavior [5]. Expression can be triggered by environmental factors, such as increased mechanical stimulation as well as specific cytokines. With fascia this expression happens naturally in wound healing and in several pathological situations. Additionally, fibroblasts which contain ASMA stress fibers have been found in normal tendons [6] and ligaments [7,8]. Cells containing ASMA stress fibers are known to be either contractile smooth muscle cells or to be a contractile phenotype of fibroblasts with smooth muscle-like features, now known as myofibroblasts [9]. Furthermore, the potential contraction force of myofibroblasts has been shown to be correlated to the degree of ASMA expression [10].

While no quantitative immunohistochemical examination has yet been published for cells containing ASMA in normal fascial sheets, the existence of cells resembling smooth muscle cells was accidentally discovered by Staubesand in normal crural fascia and has been documented with electron microscopy [11,12]. Since the crural fascia has a similar morphology to the lumbar fascia or to the muscular epimysial envelopes, it seems reasonable to extrapolate that the crural fascia is not the only fascial sheet with this property. It therefore can be cautiously assumed, that contractile cells are probably also present in other dense human fascial sheets, as have already been found in tendons, ligaments and in the crural fascia. Given the presence of contractile fibroblasts in normal fascia, it is postulated that the regular expression of this cellular phenotype would most likely serve a functional purpose; i.e., that these cells are at times used for smooth muscle like contractions.

Improved sturdiness — an evolutionary benefit

Our biological make-up has been shaped through a Darwinian process of selective survival, including countless fight and flight reactions. Life-threatening situations often involve rapid and strenuous activities. Survival in these situations not only depends on luck, wit, speed or muscular strength, but also on the mechanical sturdiness of one's limbs; i.e., not breaking a leg or dislocating an ankle while jumping or running for one's life can be a useful advantage. It would make biological sense that animals equipped with an additional mechanism to muscular coordination for a temporary increase in tissue stiffness would have a distinct advantage.

When exposed to several hours or even days of high stress situations, an innate capacity to increase fascial stiffness may be invaluable. The ability of fascia to actively contract, mediated by mechanical strain, plus specific stress related cytokines, would consequently provide us with a useful secondary myofascial tonus regulation system. Given the genetic capacity of fibroblasts to become contractile, it seems feasible that our bodies now may contain the ability to activate this advantage when challenged by periods of high mechanical and/or emotional stress.

Effect on neuromuscular coordination

In addition to this mechanical advantage, increased fascial stiffness offers a further benefit. Ligaments contain mechanoreceptors which provide sensory feedback for muscular coordination [13,14]. Without their feedback, motor coordination is significantly impaired. The same kind of mechanoreceptors are also found in broad fascial sheets, and it is assumed that they serve a similar proprioceptive function [15—17]. This is congruent with the recent finding, that patients with chronic low back pain demonstrate fewer mechanoreceptors in their lumbar fascia as well as impaired

lumbopelvic proprioception and motor coordination [18,19]. Interestingly, low threshold mechanoreceptors apparently influence muscle activity via the γ-muscle spindle system, while high threshold mechanoreceptors exert effects directly onto the α-motorneurons [20]. An increased fascial stiffness would therefore be expected to result in many muscular responses elicited by fascial mechanoreceptors in a shift from a low threshold activated γ-system response towards a much quicker high-threshold activated α-motorneuronal reaction.

A temporary decrease of ligament stiffness in cats has been shown to result in the stimulation of fewer ligamentous mechanoreceptors and in decreased periarticular muscle activation [21]. It seems likely that this response would be similar in the fascial tissues found in humans. A temporary increase in fascial stiffness would consequently improve fascial proprioception and increase muscular activation. An animal or person with an enhanced fascial stiffness would therefore have the advantage of a generally more precise and more rapid muscular reflex coordination in response to fascial proprioception, as well as the increased sturdiness. While a chronically increased fascial tonus may over time have metabolic and physiological drawbacks, the ability to temporarily increase fascial stiffness may have helped our ancestors to cope in situations demanding an increased motor performance.

Existence of chronic fascial contractures

The ability of fascia to contract is further demonstrated by the widespread existence of pathological fascial contractures. Probably, the most well known example is Dupuytren disease (palmar fibromatosis), which is known to be mediated by the proliferation and contractile activity of myofibroblasts. Lesser known is the existence of similar contractures in other fascial tissues which are also driven by contractile myofibroblasts, e.g., plantar fibromatosis, Peyronie disease (induratio penis plastica), club foot, or — much more commonly — in the frozen shoulder [22] with its documented connective tissue contractures [23]. Given the widespread existence of such strong pathological chronic contractures, it seems likely that minor degrees of fascial contractures might exist among normal, healthy people and have some influence on biomechanical behavior.

One could argue, that there may be general differences between long term chronic contractures and the proposed ability of fascia to temporarily contract in a smooth muscle like manner. Interestingly, in the condition ''frozen shoulder'' the fascial contracture sometimes improves spontaneously within a few days [24,25]. This seems to indicate a fairly rapid release of cellular contractions, rather than long term morphological changes in the collagen architecture. Another supporting indicator for a similar physiological basis are the experiments with granulation tissue, in which myofibroblast driven tissue contractions were significantly increased by the addition of pharmacological smooth muscle agonists, with clearly significant effects during as little as half an hour [26].

While none of these indications are conclusive on their own, collectively they add considerable support to the hypothesis, that fascia may be able to influence biomechanical behavior by an active temporary contraction of intrafascial myofibroblasts. As well as these general theoretical indications, there are two experimental studies, which offer more concrete evidence for our hypothesis.

Biomechanical in vitro evidence

In what appears to be the most thorough examination of the viscoleastic behavior of a normal (non pathological) fascial sheet so far, Yahia et al. [27] reported an unexpected discovery of fascial behavior, which they termed 'ligament contraction'. In this in vitro study pieces of human lumbar fascia were isometrically stretched for 15 min, then allowed to rest for 30 or 60 min, and then stretched again. Contrary to the authors' expectation, the resistance force of the tissues proved to be stronger at the repeated stretch compared with the previous time, i.e., they had become stiffer. After carefully ruling out other possible explanations for this response, the authors discussed the congruence of this behavior with similar in vitro stretch responses of visceral musculature, and they concluded that the most likely explanation would be the presence of smooth-muscle like cells in this fascia.

Pharmacological in vitro evidence

The second line of experimental evidence comes from recent research into the pharmacological control of wound contraction. In order to understand more about the contractile behavior of myofibroblasts in wound healing, several authors conducted in vitro contraction tests with fascia in response to pharmacological substances. While most authors performed their studies with injured or pathological fascia only, Pipelzadeh and Naylor

[28–30] included tissue from normal superficial fascia of rats. Suspending thin strips of this fascia in a superfusion system, they were able to induce clear and reversible tissue contractions in response to mepyramine, calcium chloride, as well as adenosine. The contractile behavior of this fascia was found to be similar to that of injured fascia from rats, which again was fairly congruent with the contractility of human myofibroblasts reported elsewhere [31,32]. The rapid onset, the reversibility, the repeatability and the dose dependency of the contractile responses in all these tissues suggest that cellular receptors are responsible for the observed effects. Given the usual caveats of extrapolating from in vitro animal data to living humans, these results appear as congruent with the hypothesis of a cellular driven active contractility in normal fascia.

Implications

Assuming that human fascia does contract in vivo as proposed in our hypothesis, how strong would the resulting force be? For an estimation of this we chose the data from the in vitro experiments with human lumbar fascia by Yahia et al., reported earlier. With a tissue strip of 1.5 mm × 1.0 mm × 30 mm the maximal measured force increase during an isometric stretch was 1.5 N. If we hypothetically apply the same force ratio to whole fascial sheets in the human body, it seems clear that such fascial contractions could have substantial biomechanical influences. As an example, the superficial lamina of the lumbar fascia, with a reported horizontal cross sectional area of 71 mm × 0.53 mm at the level of the third lumbar vertebra (plus adjusting for the 45° oblique fiber angulation in this fascial layer) would have a theoretical bilateral contraction force of 38 N.

This would put the force of active fascial contractions within a biomechanically significant range, at which it could cause a lumbar paraspinal compartment syndrome [33]. It is also in a range where a decreased fascial tonus can contribute to spinal segmental instability, which is frequently associated with the onset of low back pain [34,35]. Similarly a loss of fascial tone could also be responsible for sacroiliac pain, which is often caused by a lack of force closure of the sacroiliac joint [36] and resulting hypermobility (an example of this is the high incidence of pelvic pain during pregnancy due to hormonal changes [37]). Manual deep tissue therapies, such as Rolfing or myofascial release, which claim to influence fascial tone [38], may be able to benefit from more specific understanding (and new questions) from this new perspective. It is also possible, that acupuncture, which has been recently shown to be intimately linked with fascial anatomy [39,40], may be better understood and its effectiveness improved.

The authors therefore suggest that this hypothesis be tested with further research. A first step could be a quantitative immunohistochemical examination of human fasciae for cells containing ASMA stress fibers. Additionally a replication of the pharmacological in vitro contraction experiments of Pipelzadeh and Naylor could be done with human surgical fascia. If verified, this might not only have interesting therapeutic implications, but the new appreciation of fascia would also pose new questions: How is fascial contractility related to microinjuries, to hypoxia, to stress or infection related cytokines? How does it respond to different hormonal or pharmacological agents? Why does the fascial contraction in frozen shoulder often heal spontaneously, while this is rarely the case with the palmar fascia in Dupuytren contracture? How do different types of static and cyclic mechanical stimulation influence fascial contractility? As intriguing as these questions may be, before attempting any clinical research, first an exploration of the hypothesis through basic research is suggested.

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