Fascia research: What do we know and implications for the practicing therapist

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Fluid Flow - Fascia regulates fluid flow in the extracellular matrix, and fluid flow causes fascial remodeling. The ground substance in the extracellular matrix is prevented from absorbing fluid by tension that fibroblast cells put on extracellular matrix fibers. When this tension is relaxed, the extracellular matrix can absorb fluid rapidly so that transfer of fluid from the capillaries can increase 20-100 fold in a very short time.

Clinical relevance: Presumably the increase in tissue fibrosis will stabilize the extracellular matrix, independent of the connective tissue generated tension, which will at least partially control the edema but will decrease the tissue's ability to increase and decrease tension to regulate further variations in fluid flow. This fibrosis may be clinically useful for tissues which are too lax and may be the underlying mechanism of prolotherapy and other tissue tightening techniques. The massive stiffening of tissues seen within the first few hours after a motor vehicle accident may also be due to this mechanism. Since anti inflammatory compounds reduce the drop in interstitial pressure and edema formation, very large doses of oral non steroidal anti inflammatory medications within the first hour after trauma may prevent this stiffening. Similarly, immediate application of pressure from manual therapies may also prevent the edema from forming in the first place.

Therapies designed to locally increase edema such as Chinese cupping may increase the adaptability of the fluid flow adjustment systems by temporarily increasing fluid flow. Therapies designed to reduce lymphedema must take into account the tissue changes which take place with prolonged decrease in interstitial flow, including the increased tissue compliance or "overstretching" of the interstitial matrix. Even if lymphatic channels are re-connected, the removal of lymph nodes decreases lymph resorption and puts a greater load on the fluid removal through the lymphatics.

Finally, organs must be viewed in the context of the surrounding connective tissues and distant blood and lymphatic fluid flow, and specific organ pathology cannot be fully understood or treated without taking those tissues into account.

Mechanotransduction The living cell is a mechanical structure with a force balance between compression bearing microtubules and tension bearing bundles of actomyosin filaments. The cells are anchored to the extracellular matrix by clusters of integrin receptors which connect extracellular proteins to intracellular actin associated molecules. These receptors also serve to sense physical forces outside the cell and transmit that information through mechanical connections throughout the cell to the nucleus as well as multiple locations in the cell. This cytoskeleton provides both mechanical structure and direction to biochemical reactions within the cell. The cell can thus convert external mechanical signals into internal biochemical reactions.

Accomplishments in cell mechanics of particular interest to fascial research include the effect of fluid shear stress forces on the endothelium and on the osteocyte, the control of cell shape and

function by extracellular matrix stiffness, and impact of mechanical forces on remodelling of bone, muscle and cartilage. Two mechanisms have been identified which cells use to protect themselves from excessive mechanical stretch: first, cytoskeletal stiffening of the microfilament and microtubule lattice, and second, fluidization which allows a marked increase in mobility of macromolecules which ordinarily are restricted in motion by molecular crowding.

Clinical relevance - A better understanding of mechanochemical control mechanisms may let us correct mechanical loading or mechanochemical signalling in adult conditions. Use of the experimental methods developed may allow us to explore the effects of externally applied forces such as repetitive stress disorders or manually applied therapies.

The major fascial cell, the fibroblast Fibroblasts synthesize organize and remodel collagen, depending on the tension between the cell and the extracellular matrix. The myofibroblast produces a copious amount of collagenous extracellular matrix and generates tensile forces which play a large role in wound healing and excessive fibrosis in many tissues. This cell develops primarily from local fibroblasts. By changing shape, the fibroblast can affect stiffness and viscosity of connective tissue within minutes, consistent with the mechanotransduction models of microtubule network expansion and actomyosin generated tension proposed by Ingber.

Clinical relevance - Connective tissue actively regulates matrix tension in response to stretch as a normal, dynamic physiological process. Understanding how cells respond to forces can lead to potential treatments to decrease fibrosis in cases where the forces remain high. Yet to be explored are techniques to increase fibrosis in tissues which are too lax.

Microscopic anatomy of the fascia Deep fascia has parallel longitudinal collagen bundles and rudimentary elastic laminae, giving it both high tensile strength and elasticity. The deep fascia is a highly vascular structure with a superficial and a deep layer, each with an independent rich vascular network of capillaries, venules, arterioles, and lymphatic channels. At the junction between the deep fascia and the muscle, without any special secretory cells, the fascia is able to maintain a lubricating layer of hyaluronic acid. The deep fascia is not just a tough barrier structure of collagen and elastin, but is a metabolically active vascular layer which provides gliding and protective functions. There is a multifibrillar network of microvacuoles ranging in size from a few to a few hundred microns. The smaller vacuoles are found in tissues which have greater excursion. These fibers provide a supporting framework for vascular, lymphatic and nervous connections between tissues which routinely move, such as tendon and tendon sheath.

Clinical relevance - fascial layers are able to produce a lubricant, hyaluronic acid, which allows sliding between the fascia and neighboring muscle. The architecture of the fascia allows continuity of nerves, blood and lymph vessels between the sliding tissues. With trauma to the muscle, the overlying fascia no longer produces the sliding layer of hyaluronan. Anatomic and clinical studies will be necessary to identify and improve methods to maintain sliding after tissue trauma.

Fascial anatomy There is widespread presence of myofascial extension from the muscle at its distal end into fascial layers of the more distal extremity. Ligaments crossing a joint are not independent of the neighboring muscle, but rather are an intimate part of the fascial portion of the end of the muscle and can thus be tensed or relaxed by the muscle. There also are direct myofascial connections between neighboring muscles, so that up to 50% of the muscle force may be transmitted to neighboring tissue rather than to the tendon of insertion. There also are preserved fascial sheaths to muscles which are present in primates but no longer in humans – the muscle has disappeared in evolution but the enveloping fascia is still present.

Clinical relevance – traditional muscular anatomy as presented in the anatomy textbooks does not take into account the multiple fascial connections both laterally along the muscle and longitudinally at the ends of the muscles. This anatomy is quite evident in all extremities where muscle crossing joints is quite clear, but has not been studied in as much depth in trunk muscles.

Pain can have muscle, fascial, and nerve components. Myofascial trigger points have local thickening of muscle fibers, while muscle tender points to palpation have no identifiable specific pathology. Nerves are surrounded by three layers of fascia and can become sensitized to normal motion by more proximal nerve irritation. Delayed onset muscle soreness is seen 1-2 days after eccentric muscle contraction, and increased fascial sensitivity can be seen in this condition.

Clinical Relevance:

Understanding the physiology of pain and pain perception can help the clinician initiate and evaluate treatment although there are also strong psychological components.

Fascia references. BOLD ones are available for free at <u>http://www.ncbi.nlm.nih.gov/pubmed/</u>

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