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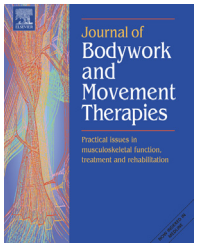

Can fascia's characteristics be influenced by manual therapy?

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Summary This essay looks into some fundamental properties of collagen and attempts to relate what the manual therapist does to the necessity of maintaining the collagen's strength under loads. In so doing we point out some difficulties in gathering relevant data applicable to the clinic and propose direction for further research.

Back in the 70's life was much simpler. Biomechanics was based on mathematical models derived from the 1940's ideas of Bartelink (1957) whereby muscles and bones were the dominant structures manipulating the animal in space under the control of the central nervous system. This idea was received with unabridged enthusiasm, and even to this day, many believe that this is the revealed biomechanics' gospel. That this concept survived for so long against the contradictions it generated is a testimony to the unique ability of the art of medicine to keep an attractive idea alive in spite of a ruthless experimental annihilation.

The contribution of collagen was largely ignored in spite of an extensive description of its architecture in the first edition of Gray's Anatomy back in the late 19th century, and more recently described by Willard and Vleeming (2012). Since the forces transmitted by collagen are difficult to assess in vivo (as opposed to muscles whose function can be monitored using electromyography) it was easier to consider that the primary function of collagenous structures was to prevent excessive motion of bones and joint articulations.

That simplistic view was quickly shown to be untenable in the case of spinal mechanics. For example, the hip extensor's power cannot be transmitted to the upper extremities via the erectores because these muscles are not large enough to do the job (Farfan, 1975, 1978; Gracovetsky, 1981, 1986). In addition, even if they were, their anatomical positioning would result in a compressive load that would crush the intervertebral joints. And instead of trying to explain this paradox, the expedient thinking of the time was to arbitrarily decide that our anatomy was deficient and responsible for the functional limitations leading to the problem of dreadful low back pain.

In the late 70's and early 80's, Harry Farfan (1975) introduced the heretical concept that the thoracolumbar fascia played a central role in the force transmission system from the foot to the arms. The magnitude of the fascial contribution to heavy lifting is impressive: it is on the order

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of 3—4 times what the erector spinae muscles can produce. In other words, the collagen's fascia was no longer something that could be marginalized. It was a central component of our architecture (Gracovetsky, 1977, 2008).

In truth, the ferocious unsolicited criticism that followed did dampen somehow over the following 20 years, but even then the question of the precise function of the fascias remained open? A consensus emerged to the effect that fascias were made of collagen fibers, not unlike steel cables, but with temperature dependent nonlinear characteristics. Hence, as the story went, the only unknown was the precise attachment of these fibers to the bone and/or muscles, and it was believed that once that geometrical problem was resolved, the function of the musculo-skeletal system would be understood.

In parallel with these events, a different type of representation evolved under the label of "Tensegrity". Tensegrity is a model that neatly constructs complicated shapes (such as the anatomy of the animal) using essentially two components, that is a compressive one (call it bone) and tensile one (call it ligament or collagen). A good description of the approach can be found in Scarr (2014). An excellent illustration was proposed by Guimberteau (2005) in a series of videos showing the incredible adjustment and coordination under stress of the various components under the surface of the skin.

But the need to actually represent realistic situations, such as the hip joint, implied complex tensegrity representations of very high orders that implicates so many nonlinear elements that the benefit of its' simplicity is somewhat lost in the description of the physiological anatomy. Perhaps more damning is the fact that although simple structures like the icosahedron guarantees that the bones will remain under compressive load, this feature may not be possible with complex tensegrity components of very high order. In other words, there may exist loading configurations under which the virtual tensegrity bone may end up being subjected to tension. That would be regrettable since Wolff's law (1892) tells us that bone reacts to stress, and hence bone that is subjected to tension may begin to degenerate and weaken, thereby reducing the ability of the animal to survive external loading.

Which raises the interesting question: What type of external loads will guarantee that the tensegrity model will always work as it proponents say it would? That may very well define the optimum environment of the living organisms. Or to put it in another way, can the environment define the type of tensegrity models that are appropriate for the animal?

At the end of the day, tensegrity or not, the 17th century classical modelling of Borelli remained not far away, albeit sometimes hidden by some modern fancy names such as "finite elements analysis".

But the considerable investment into mathematics, following Borelli's proposals could not overcome the need to explore different aspects of biomechanics. Other factors had to be considered.

The root of the problem is that the constitutive elements of the model (be it compressive or tensile) are nonlinear structural components that are assigned fixed properties, that depend upon the mechanical stress applied to them, their temperature, and/or the action of various enzymes that may independently degrade the tissues and compromise the integrity of their structure. The combination of these elements creates a very messy model of the role of the fascia in life.

And so, given the sudden importance of collagen fascia, many began to look closely into its atomic structure and in particular the parameters that may influence the response of the triple helix of tropocollagen (Dittmore, 2016; Ruberti, 2005; Susilo, 2016). It was found that collagen was remarkably resistant to proteolysis (a breakdown of proteins into smaller polypeptides or amino acids), unless specialized matrix metalloproteinases (MMPs) could bind along the collagen fibril at specific places called 'cleavage sites'. Once bound to a cleavage site along the molecular structure of the individual collagen helix, MMP would start degrading the structure, a process central to remodelling and repairing the collagen, in order to ensure survival of the animal following injury.

The first snag in understanding what exactly happens, is the near impossibility of studying the response of collagen fibrils in vivo. Besides the nonexistence of appropriate instruments to track the actual behavior of an individual collagen fibril, the experiments would pose difficult ethical issues, since human subjects would be negatively impacted by the destructive nature of these experiments. For that reason, most of the available data was collected *in vitro*, under conditions sometimes so different from the in vivo situation, that extrapolating the data into the clinical setting would, at the very least, demand extensive validation.

But there is another layer of difficulty, in that the conclusions of the *in vitro* experiments were often reached via mathematical representations of what the experimenters thought would be the issues at hand. That further raises the issue of validation of the mathematical models used in data acquisition, processing and procedures, before any conclusions could be applied to the clinic. We are therefore left with the double jeopardy of appreciating the application of the conclusions derived from these *in vitro* data, into any clinical setting.

Within these words of caution, considerable attention turned to the creation and destruction of these cleavage sites. First it was found that the triple helix arrangement was in fact unstable at all temperatures, meaning that the nice longitudinal fiber arrangement would collapse like a coil, because the energy of the coiled collagen structure is lower than the energy of the original elongated fibrillar structure. Hence the molecules of collagen will always try to rearrange themselves to stay in the minimum energy configuration.

That would not be so bad if by so doing the coiled collagen fibrils opened up sites at which the MMP could begin cleaving the collagen into pieces, by a process not yet fully understood. But that desirable feature for remodeling and repair is not helping collagen to support loads. And so the question is now to understand how the degradation process could be stopped and reversed to reinforce the collagen and makes it capable of sustaining the very large forces that are needed to support the spinal structures and many others.

It therefore appears reasonable to consider that one possible implicit objective of appropriate manual therapy

(or appropriate exercise) might be to stop and reverse the destructive action of MMP with the attendant strengthening of the collagen fibrils.

Since the natural tendency of tropocollagen to coil, creates cleavage sites by releasing energy, reversal of the process demands restoring the energy that was released during the coiling process. This is pretty much the way in which the combination of oxygen and hydrogen releases considerable energy to create a very stable water molecule. Conversely, to break up the bonds and restore the free hydrogen and oxygen atoms demands putting back the energy released by the creation of the water molecule, as in the addition of heat. Heating the collagen is therefore one possible solution to the problem, because heat is nothing more than mechanical energy whereby the oscillating atoms of two structures are put in contact. Note that the amplitude of oscillation of an atom is also what is termed its temperature. What happens next is similar to a boxing match. Whoever swings the most (a higher temperature) delivers the best punches to the (lower temperature) receiver. Mechanical energy is then transferred from one boxer towards his somewhat distressed opponent. That is sometimes defined as heat transfer.

Although it is not known how much energy can be transferred from the skin surface to the deeper layers, it can be speculated that at least some of the therapist's energy, applied to the skin, will end up being transferred. It follows that controlling the delivery of mechanical energy (heat) to the coiled collagen should (presumably) uncoil it, close the cleavage sites, and prevent MMP from binding and degrading the collagen. The amount of energy needed to reverse the creation of cleavage sites is not really known, but is estimated to be small, and within the range of what might be provided by external action. This should not be confused with the considerable amount of energy needed to deform the human fascia, as calculated by a model describing the fascia as a stable material with elastic nonlinear properties (Chaudhry et al., 2008).

Therapists will recognize here the benefit of heating pads or any other heat delivery techniques such as massage etc. In that regard the video illustrations of Jean Claude Guimberteau also demonstrate how a force applied to the surface of the skin ends up being dissipated deep into the tissues via a dense interconnected network of collagenous tissues (Guimberteau, 2012).

Another interesting property of the uncoiled collagen is that mechanical stress, aligned with the fibrils, inhibits the creation of cleavage sites and hence prevents the subsequent degradation. Yoga teachers know that stretching tissues in various positions has beneficial effects. Aligning the forces with the collagen fibril orientation may be made easier by attenuating the impact of gravity, and that may explain the benefits of hydrotherapy. But perhaps more importantly, this may highlight why the beginning of stressful exercises must start with a warm up period. Indeed, the athlete needs to neutralize the creation of cleavage sites before MMP begins the process of degradation, to protect the collagenous fascias from damage by excessive stresses to the structure.

This raises two additional questions. Who is directing the use of the available energy for opening or closing the sites on which MMP attach? Is it the central nervous system (via production of enzymes or something else)? Or is it an automated system strictly based upon the molecular physical differences with local feedback? And if an injury occurs, does the affected collagen release a chemical signal asking for help? It would be advantageous to have the collagen repair and regeneration functions as a local event, pretty much like the growth of nails continue for $^{1}/_{2}$ hour after death. The concept of having a non-neural mechanism directing the allocation of energy resources has been suggested by the experiments of Grinnell (2008).

However this is still not good enough. It may be true for specific fascias such as the lumbodorsal fascia and other tendons that are clearly positioned for transferring forces. But what about the other collagenous fascias that are deep inside the body, in a stable temperature and humidity environment while subjected to minimal stresses, especially when sleeping. If nothing stops MMP, then we would wake up as a ball of gelatin. Since people do not routinely liquefy themselves during a good night's sleep, the hunt for the responsible element(s) for stopping the degradation due to MMP must continue.

We might have to consider the role of respiration and any other automatic mechanical motion such as the cardiac pulse, intestinal contractions, etc. These heat generators of might encourage the closing of the cleavage sites. In addition, the role of the gravitational field cannot be ignored since it is always stressing some collagen with a corresponding need for the muscles to redistribute and relieve the stress. For instance, as we sleep, the impact of gravity on our body mass stresses the collagen holding the body parts. Since collagen is a viscoelastic material, it elongates to a point where local pain forces a change in the sleeping position. In other words, there is a continuous cyclic loading/unloading of collagen in all parts of the body. This is particularly true for the omnipresent cardiac pulse which cyclically stresses and relaxes the vascular network of arteries with its attendant collagenous support. Hence, besides providing blood to the system, the heart insures its share of maintenance of the collagenous cardio-vascular network.

Conversely, a too aggressive elimination of the cleavage sites would break the equilibrium between the creation/removal of collagen. Perhaps too much collagen production might be responsible for the so called "adhesions" which in turn might explain the need and benefits of myofascial release-type techniques.

In that regards, it appears that long stays in zero gravity would not be a good idea since it would directly affect negatively the status of collagen everywhere in the body.

Note that the tensegrity representation of biomechanical tissues linking collagen to the surrounding bone network implies that the weakening of collagen results in a corresponding weakening of the bone via what is known as Wolff's Law (1892), since the stress through the structures is modified and possibly lowered. Although the relationship has not been explored, it is theoretically possible to encourage bone repair by collagen (fascial) manipulation. It may not be coincidental that bone is also an assembly of a different type of collagen fibers.

There are other possible mechanisms that may play a significant role in the hardening of the collagen structure. Many have argued that the water concentration in the

tropocollagen would stiffen the molecular chains by establishing links between the each of the individual strand of the triple helix collagen molecule and even between the triple helix fibrils themselves (Leikin, 1997). However the data is not always congruent with other investigations such as (Rana, 2012) who describe the links as either aldol or aldol histidine at the N terminus or C terminus of the tropocollagen assembly.

It may be that Leikin's preparation of his collagen's specimens before subjecting them to the Raman Spectroscopy procedure significantly changed the water concentration of the tissues after removal from the animal. Leikin work is also supported to some extend by Masic (2014) who was interested in the characteristics of a drier collagen that correlated with an increase in stiffness of the collagenous structures. The unresolved clinical question is to prove that the in vitro experimental removal of water is something that happens in vivo.

Similarly, the data from the mechanical experiments of Schleip (2012) are problematic in that they demonstrate a very rapid drop in water content following the application of tensile force on the collagen fibril which is then followed by a very slow (i.e. 2—3 hours) rehydration process. If we accept the premise that water content would significantly affect the stability of the collagen molecular structure, it becomes difficult to explain how weight lifters could repeat lifts within seconds (hence presumably with a drier and weaker collagen) without any structural damage. This is not to say that water has no role; Evidence is emerging highlighting the importance of hydration in the mechanical properties of collagen (Bella, 2016).

Franchi (2010) took a different approach by using scanning electron microscopy to study the changes in the collagen fibers organization when the tissues are put under stress. His remarkable images demonstrate that the well-ordered fibrils became indeed much disorganized under stress, thereby interfering with an orderly sliding motion. This feature is similar to what can be observed during the hardening process of many linear and nonlinear materials subjected to tension beyond their elastic range.

When that occurs, the atomic planes can no longer slide nicely over each other since the rough gliding surfaces would induce a significant friction and hence resistance to further deformation (Fratzl et al., 1998). This "hardening" may explain why repeated massage and/or the application of myofascial release techniques can reduce the amount of disorganization within the collagen fibrils and permit a freer movement.

And so the centuries-old manual therapy techniques appear to be based upon an independent emerging body of evidence in physics and chemistry. For example, it is very humbling to realize that hundreds if not thousands of years ago, by sheer observation, trial and error, many smart people were able to identify a set of beneficial movements and exercises that defines, for example, what is termed "Yoga". And all this, of course, without the means of appreciating in what way a specific action would impact the basic molecular components of the musculo-skeletal system.

What appears to be missing, besides the validation of the proposed manual or movement approaches, is the specific way in which collagen is regenerated after being degraded by MMP. Understanding this process by direct experiments rather than mathematical models will go a long way towards clarifying the healing of the tissues injured or damaged. But the $64,000 question for the therapist is to understand the relation between a treatment modality and the associated response of the collagenous fascias. To answer that requires a collective investigation that transcends the plethora of treatments derived from several schools of thoughts. Why do we need to have Osteopathy, Chiropractic, Physical therapy, Rolfing, Yoga (and many more similar philosophical approaches) if the active parameters are the same? Admittedly, this concept will be met with some resistance, but at the end of the day the question remains: Is it possible that the claimed differences are really a simple matter of semantics?

In the search for the relation between modality and collagen fascia, one can expect to find a considerable amount of overlapping techniques. Logic would then dictate that only the common elements producing demonstrable good clinical results ought to be retained to avoid redundancy. At the end of the day, these common elements would represent the foundation of a unified method for treatment which by definition would be independent of the clinician's philosophical perspective and only dependent upon the subject's presentation and the physics of the phenomenon's involved.

If history is any guide, these considerations of a yet to be fully understood mechanism of action for manual therapy will end up being dissected, vigorously challenged, and eventually replaced. In the meantime, it is hoped that this short essay may help many to walk over these treacherous waters and encourage students to undertake the research needed to secure better answers.

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