Journal of Bodywork & Movement Therapies 22 (2018) 235–236

Contents lists available at ScienceDirect

Journal of Bodywork & Movement Therapies

journal homepage: www.elsevier.com/jbmt

Editorial

Fascial well-being: Mechanotransduction in manual and movement therapies

Mechanotransduction refers to the multiple ways in which mechanosensitive cells respond to different degrees, directions, frequency and duration of mechanical load, such as torsion, tension, shear, compression, stretch, bend and friction. Mechanotransduction in connective tissues, involves both physical and chemical communication processes that occur between specialized cells, such as fibroblasts and telocytes, and their immediate environment, - the soupy, mesh-like extracellular matrix (ECM) network, in which they operate.

- In mechanosensitive cells, mechanical stimuli result in architectural shape modification, altering cellular behavior and physiological adaptation – potentially resulting in modified gene expression, enhanced protein synthesis, modulated inflammatory responses, more efficient repair and remodeling, and more. (Kahn and Scott 2009, Kjaer, 2009, Kumka and Bonar, 2012; Standley and Meltzer, 2008; Cao et al., 2015; Wipff and Hinz, 2008).
- Depending on many variables, mechanical signaling may have quite contrasting effects. For example, in a 2012 basic science study investigating the effects of different forms of mechanical load applied to fibroblasts, Hicks et al. (2012) observed that 8 hours of cyclic short-duration stretches - such as occur during repetitive motion strain – result in an inflammatory response from the affected fibroblasts. In contrast, modelled Myofascial Release – involving acyclic, long-duration (60 seconds), light (approximately 10%) stretch – applied to the already distressed fibroblasts - under controlled laboratory conditions - significantly reduced that inflammatory process.

The question is whether or not such changes - that can be predictably achieved in laboratory settings – can be replicated using clinically applied manual or movement methods?

1. Mechanotransduction and collagen's self-repair processes

Studies of the homeostatic functions of collagen, have offered insights into underlying processes and mechanisms that might, potentially, be influenced by manual and movement therapies.

- Flynn et al (2010) observed that: "*applied mechanical strain preferentially preserves collagen fibrils in the presence of enzyme related degradation.*" Suggesting that when there is optimal (external and internal) tensional support, collagen degrades more slowly
- Humphrey et al. (2014) explain that "*Mechanical loads on transmembrane complexes and cytoskeletal structures are fundamental to the cell–ECM interactions that govern mechanical homeostasis in health* –[requiring] - *that cells first sense the mechanics of the ECM and then regulate it to maintain the desired properties; loss of these complementary homeostatic processes leads to fibrosis, mechanical failure or other pathologies.*"
- Dittmore et al. (2016) have reported that unbalanced tensional support, affecting collagen fibrils, results in periodic spontaneous deformation and collapse ('*buckling*') - at what are termed 'cleavage sites'. This process triggers self-generated repair and remodelling: "*Collagen fibrils resemble nanoscale cables that self-assemble and our experiments reveal unanticipated defects that form along the fibrils … the initiation sites of collagenase activity, representing a strain-sensitive [thermally-labile] mechanism for regulating tissue remodeling.*" They also report that "*The underlying dynamic molecular changes in collagen structure, which may involve single atoms, were readily observed through tracking of enzyme binding.*"

It appears that triple-helix collagen fibrils contain billions of sites, at 1 μm intervals, that are vulnerable to spontaneous buckling – when the tensional forces that support them are not optimal. Buckling then exposes collagen to specific enzymes (Matrix Metalloproteinases or MMPs) at the cleavage sites, initiating degradation, and subsequent repair and regeneration. However, when under appropriate tension, the number of buckling (cleavage) sites decrease.

The question is whether or not manual or movement methods can be employed to enhance collagen stability?

- Also, is it possible to reliably translate into clinical settings, basic science evidence from studies in which cell behavior has been shown to be altered under minutely controlled conditions?
- In other words, what do studies of cells and tissues in a laboratory, actually mean clinically, when it comes to management of fascia-related pain and dysfunction? Although clinical translation of basic science research into cell behaviour is potentially helpful in identifying underlying mechanisms involved in manual and movement methods – translation of such research evidence needs to be cautious.
- For example, laboratory based studies of the effects of different dosages of load (degree, direction, duration etc) of mechanical load, applied (for example) to fibroblasts (Cao et al., 2015), cannot evaluate the effects on those, and other, cells, in the context of the environment in which they would be found in the living human body.
- In addition, we do not know how, and to what degree, applied load/force is absorbed or transmitted from the skin surface, to

https://doi.org/10.1016/j.jbmt.2017.11.011
1360-8592/© 2017 Published by Elsevier Ltd.

deeper tissues - in real life - compared with modelled methods, where highly controlled laboratory conditions apply, and where precise measurements of all parameters of forces affecting specific cells, is possible.

2. Insights

Burkholder (2008) has offered descriptions of some of the intricacies of load transfer into tissues, and has discussed the mind-numbing complexity of ways in which mechanical load to living muscle is transferred and transmitted.

Burkholder suggests that our task is to:

"consider the shape changes and forces associated with mechanical signals and juxtapose them with observed changes in biochemical signalling, to determine what might contribute to the cellular perception of mechanical signals."

"The mechanical properties of intramuscular connective tissue are particularly difficult to determine, making the distribution of passive tension between passive myofibers and the matrix, nearly impossible to estimate. The structure of that matrix is a woven network of stiff collagen fibrils, and the mechanical behavior of relaxed muscle, is consistent with reorientation of that matrix around isovolumic myofibers) [suggesting that] fibers may undergo lengthening deformation resulting from transverse compressive forces, rather than tension or shear on molecular adhesions. It is also worth noting that this is an illustration of "tensegrity," the idea that local changes in the shape of a structure depend on the global integration of that structure."

"The principal feature that distinguishes whole muscle from cultured myotubes is the integration with a three dimensional (3-D) extracellular matrix and extensive connections among adjacent fibers. This mechanical integration has the effect of homogenizing deformations, meaning that gradients in shape changes will be smaller than they might be in an isolated fiber. A whole muscle is much more like a fiber-reinforced composite than it is like a bundle of spaghetti."

'Gracovetsky (2016) has considered the issues relating to the transfer of forces, from the skin surface to deeper tissues, and has suggested that:

"[While] 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 matrix metalloproteinases [enzymes] 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"

"The video illustrations of Jean Claude Guimberteau [http://www.endovivo.com/en/strolling,under,skin,dvd.php] 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).

As to the evidence from the studies by Flynn et al (2010), Humphrey et al (2014), and Dittmore et al. (2016) – regarding collagen homeostasis – as summarised above - the clinical relevance would seem to be that balanced tone/tensions are conducive to collagen well-being, while unbalanced patterns are not.

In support of those observations, Gracovetsky offers the following thoughts:

"Franchi et al. (2010) *[used] electron microscopy to study the changes in the collagen fibers organization when the tissues are put under stress [and] demonstrated that the well-ordered fibrils become disorganized under stress, thereby interfering with an orderly sliding motion …. [The resulting] "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."*

Unsurprisingly therefore, the solution to maintenance of collagen and therefore fascial) health, would seem to align with maintenance of an optimally balanced musculoskeletal status, potentially assisted by manual and movement therapeutic methods.

References

Burkholder, T., 2008. Mechanotransduction in skeletal muscle. Front. Biosci. 12, 174–191.
Cao, T., et al., 2015. Duration and magnitude of myofascial release in 3Dimensional bioengineered tendons: effects on wound healing. JNL Am. Osteopath. Assoc. 115 (2), 72–82.
Dittmore, A., et al., 2016. Internal strain drives spontaneous periodic buckling in collagen and regulates remodeling. Proc. Natl. Acad. Sci., 201523228
Flynn, B.P., et al., 2010. Mechanical Strain Stabilizes Reconstituted Collagen Fibrils against Enzymatic Degradation by Mammalian Collagenase Matrix Metalloproteinase 8 (MMP8). PLoS ONE 5 (8), e12337. https://doi.org/10.1371/journal.pone.0012337.
Franchi, M., et al., 2010 Mar. Tendon and ligament fibrillar crimps give rise to left-handed helices of collagen fibrils in both planar and helical crimp. J. Anat. 216 (3), 301–309.
Gracovetsky, S., 2016. Can fascia's characteristics be influenced by manual therapy? J. Bodyw. Mov. Ther. 20, 893–897.
Guimberteau, J.C., 2012. Inside Architectures. Film Video 28' DVD - Distribution. Endovivo.
Hicks, M., et al., 2012. Mechanical strain applied to human fibroblasts differentially regulates skeletal myoblast differentiation. J. Appl. Physiol. 113 (3), 465–472.
Humphrey, J., et al., 2014. Mechanotransduction and extracellular matrix homeostasis. Nat. Rev. Mol. Cell Biol. 215 (12), 802–812.
Khan, K., Scott, A., 2009. Mechanotherapy. Br. J Sports Med. 43, 247–251.
Kjaer, M., 2009. From mechanical loading to collagen synthesis. Scand. J. Med. Sci. Sports 19 (4), 500–510.
Kumka, M., Bonar, B., 2012. Fascia: a morphological description and classification system based on a literature review. Can. Chiropr. Assoc. 56 (3), 1–13.
Standley, P., Meltzer, K., 2008. Effects of Repetitive Motion Strain (RMS) & Counter-Strain (CS), on fibroblast morphology and actin stress fiber architecture. J. Bodyw. Mov. Ther. 12 (3), 201–203.
Wipff, P., Hinz, B., 2008. Integrins and the activation of latent transforming growth factor beta1-an intimate relationship. Eur. J. Cell Biol. Sep. 87 (8–9), 601–615.

Leon Chaitow, Editor-in-Chief
Corfu, Greece
E-mail address: chaitow1@gmail.com.