



Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.elsevier.com/jbmt



EDITORIAL

Understanding mechanotransduction and biotensegrity from an adaptation perspective

The concepts represented by *mechanotransduction* and *biotensegrity*, need to be appreciated if we are going to make sense of an important evolution in our understanding of how the body works. These have profound implications for manual therapy.

Mechanotransduction relates to the way mechanical load is translated by cells into chemical responses – instantly – switching off, or switching on, specific processes, such as inflammation, in response to forces such as those that occur during manually applied compression and/or stretching (as examples), as well as during movement (including internal motions, for example associated with respiration and/or circulation).

The word *tensegrity*, as used by the architect Buckminster Fuller in the 1960s, was a shorthand way of describing “tensional integrity” – in which compressive and tensional forces operate in relation to each other, providing the basis of form to structures – such as a geodesic dome – or in the human body, in cells and larger structures.

Biotensegrity describes tensegrity in living structures, for example the architecture of cells – together with the way this structure relates to its immediate environment – the extracellular matrix, and how cells attach/adhere to surrounding structures (Swanson, 2013; Ingber, 2003; Levin, 2006).

Selye (1950) described general and local adaptation syndromes (GAS) and (LAS) - that provide useful frameworks within which to appreciate of the interactions between our individual biomechanical/biochemical and psychosocial attributes, and multiple environmental influences. Selye observed that the body responds to external sources of stress (biochemical, biomechanical, environmental, psychosocial etc) in predictable biological ways as it attempt to restore internal homeostasis. An initial hormonal stress response involves sympathetic arousal (‘fight or flight’) – with subsequent adaptation phases if stress is prolonged – leading ultimately to adaptation exhaustion, ill-health and collapse.

One way of envisaging the symptoms on display by patients is to conceive them as representing failed (or failing) adaptation – and to determine therapeutic interventions

that have as objectives reduction of adaptive load and/or enhancement of functionality (greater ability to manage ‘load’) – without burdening the system with excessive adaptive *demands*.

The Biotensegrity model can be seen as a natural extension of Selye’s original observations. As Randal Swanson (2013) explains: “*Biotensegrity provides a conceptual understanding of the hierarchical organization of the human body and explains the body’s ability to adapt to change. Further, biotensegrity explains how mechanical forces applied during osteopathic manipulative treatment could lead to effects at the cellular level, providing a platform for future research on the mechanisms of action of osteopathic manipulative treatment.*”

It is suggested that the words ‘osteopathic manipulative treatment’ in the quotation from Swanson, may for the purposes of this editorial, usefully be replaced by – for example – ‘manual or movement-related treatment.’

Swanson continues to detail research over the past quarter century that clearly demonstrates that cells function as tensegrity structures, and that molecules as well as tissues, such as those involving bone and body organs all involve this architectural design.

Standley and Meltzer (2008) et al. as well as Meltzer et al. (2010) have offered experimental evidence that demonstrate that manual methods such as myofascial release and strain/counterstrain, are capable of reversing inflammatory cellular behaviour within minutes. Standley et al. demonstrated an increase in the expression of numerous inflammatory genes and an increase in apoptotic rate when fibroblasts were subjected to 8 h of repetitive motion strain. When these distressed cells received just 60 s of the equivalent of either myofascial release, or strain/counterstrain, their behaviour changed dramatically – returning to normal.

These experimental examples offer a glimpse of what may be happening during application of these and other manual methods, to the human body, as they ‘adapt’ to therapeutic interventions – in line with Selye’s stress-hypothesis.

For a full appreciation of what may lie beyond this 'glimpse' Swanson's (2013) paper is highly recommended.

References

- Ingber, D.E., 2003. Mechanobiology and diseases of mechano-transduction. *Annu. Mediaev.* 35, 564–577.
- Levin, Stephen, 2006. Tensegrity, the new biomechanics. In: Hutson, M., Ellis, R. (Eds.), *Textbook of Musculoskeletal Medicine*. Oxford University Press, Oxford.
- Meltzer, K.R., Cao, T.V., Schad, J.F., et al., 2010. In vitro modeling of repetitive motion injury and myofascial release. *J. Bodywork Move. Therapies* 14 (2), 162–171.
- Selye, H., 1950. *Stress*. Acta Inc Medical Publishers, Montreal.
- Standley, P.R., Meltzer, K., 2008. In vitro modeling of repetitive motion strain and manual medicine treatments: potential roles for pro- and anti-inflammatory cytokines. *J. Bodywork Move. Therapies* 12 (3), 201–203.
- Swanson, R.L., 2013. Biotensegrity: a unifying theory of biological architecture with applications to osteopathic practice, education, and research. *J. Am. Osteopath. Assoc.* 113 (1), 34–52.

Leon Chaitow, ND DO, Editor-in-Chief,
School of Integrated Health, University of Westminster,
21, Siddons Lane, 144 Harley Street, London NW15NF, UK

*Tel.: +44 20 7224 4220; fax: +44 20 7486 1241.
E-mail addresses: leonchaitow1@gmail.com,
leonchaitow1@mac.com

27 February 2013