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FASCIA SCIENCE AND CLINICAL APPLICATIONS: EXTENSIVE REVIEW

A unifying neuro-fasciagenic model of somatic dysfunction – Underlying mechanisms and treatment – Part II

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Summary This paper offers an extensive review of the main fascia-mediated mechanisms underlying various therapeutic processes of clinical relevance for manual therapy. The concept of somatic dysfunction is revisited in light of the several fascial influences that may come into play during and after manual treatment. A change in perspective is thus proposed: from a nociceptive model that for decades has viewed somatic dysfunction as a neurologically-mediated phenomenon, to a unifying neuro-fascial model that integrates neural influences into a multifactorial and multidimensional interpretation of manual therapeutic effects as being partially, if not entirely, mediated by the fascia. By taking into consideration a wide spectrum of fascia-related factors – from cell-based mechanisms to cognitive and behavioural influences – a model emerges suggesting, amongst other results, a multidisciplinary-approach to the intervention of somatic dysfunction. Finally, it is proposed that a sixth osteopathic 'meta-model' – the connective tissue-fascial model – be added to the existing five models in osteopathic philosophy as the main interface between all body systems, thus providing a structural and functional framework for the body's homoeostatic potential and its inherent abilities to heal.

Introduction

In osteopathic practice there are three main manual approaches that are directed towards the fascia: 1) *direct approach* – the affected tissue is brought against the restrictive barrier, described as a "functional limit that abnormally diminishes the normal physiologic range" (E.C.O.P., 2011a). This is maintained until tensions modify; 2) *indirect approach* – tissues are brought away from the restrictive barrier while a position of ease (a balanced

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tension in all planes and directions) is found and maintained up to a release; 3) *combined approach* — both the point of ease and the restrictive barrier are consecutively engaged in an interactive fashion (Ward, 2003). Although myofascial and fascial-ligamentous release techniques are the most commonly applied fascial approaches amongst American osteopathic physicians (Johnson and Kurtz, 2003), there are a multitude of fascia related techniques that utilize various levels of aggressiveness (Sergueef and Nelson, 2014), from *balanced ligamentous tension technique* to *counterstrain*, from articulatory to cranial and visceral techniques, including soft tissue work from inhibitory pressure to effleurage manoeuvres.

Osteopathic treatment of fascia has shown to be effective for a wide variety of conditions, from local musculoskeletal causes, such as acute joint injury (Eisenhart et al., 2003) to general mood disorders such as depression (Plotkin et al., 2001). Other non-osteopathic manual modalities have shown similar results, possibly because of the common therapeutic influence and stimulation of the myofascial complex (Simmonds et al., 2012).

Several mechanisms may underlie therapeutic changes in the fascia.

Fascia-related mechanisms involved in the treatment of somatic dysfunction

Structural changes

Structural modifications in the connective tissue may occur immediately or just after treatment and may account for the palpable changes following manipulation. Myofascial release of the thoracolumbar fascia in patients with chronic low back pain has shown an increase in thickness of fascial layers that remained for at least 24 h (Blanquet et al., 2010). This suggests a sustained change in the architecture and/or hydration of the fascia being worked on. In addition, US measurements applied immediately before and after manual intervention, showed highly significant differences in collagen fibre density and orientation in the structure of the matrix in the dermis, reflecting palpable differences in tension and regularity (Pohl, 2010). These findings are consistent with the re-organization and remodelling of collagen fibres, which have been suggested to result from myofascial work (Martin, 2009) through a breakdown of abnormal collagen cross-links and an increased matrix hydration.

Since abnormal palpable findings (such as altered texture) in connective tissue might be related to abnormal cross-links between collagen fibres, it has been shown that human fibroblasts respond better to cyclical (3 min stress-3 minutes relaxation, of about 7% of their length) rather than static stretch by increasing the production of collagenase by 200% (Carano and Siciliani, 1996).

This enzyme has a potential role in collagen remodelling in dysfunctional tissue by breaking cross-linking peptide bonds, thus preventing excessive connective tissue formation, as occurs during wound healing. However, the repetitive mechanical stretch-induced collagenase activity can also be suppressed by hormonal (oestradiol and progesterone) influences (Zong et al., 2010), as might occur during the menstrual cycle or in hormonal therapy.

A static load may also break abnormal tissue collagen crosslinking and stimulate fibroblast differentiation under the influence of IL-6, with a potential role in tissue repair and remodelling (Hicks et al., 2012; Khan and Scott, 2009). In addition, the duration of the load appears to be a significant factor. It seems that brief periods of stretching may decrease the effects of TGF-β1 production of additional collagen, thus reducing the risk of fibrosis or scarring (Langevin et al., 2006). Scars may generate pain syndromes that can be relieved by a direct manual approach to the involved connective tissue (Kobesova and Lewit, 2000), and this could be applied in the first 12 h following surgery to reduce inflammatory reactions and the risk of adhesion formation (Chapelle and Bove, 2013).

Cell-based mechanisms

As will be described in this section, various forms of manual loading, whether sustained or cyclical, that differ in direction, speed, magnitude and frequency, appear to exert a strong impact on cell behaviour, gene expression and tissue remodelling through growth factors and enzyme activation.

Several cell-based mechanisms may potentially represent crucial factors in the achievement of a palpable release during manual fascial work. Some of these are described in Table 1.

Fibroblasts in vitro and in vivo have shown an almost immediate response to traction, pressure and shear forces, followed by a series of changes in chemical signalling pathways and gene activation, ATP release, actin polymerization, and also differential stretch-activated calcium channel signalling (Wall and Banes, 2005; Stoltz et al., 2000). Although most of the proposed mechanisms may require hours or even days before producing desirable effects on tissue texture and function, some of them may take place within minutes from the starting point of a therapeutic manouver. Langevin et al. (2013) note that in response to sustained changes in tissue length, fibroblasts may rapidly modulate such tension by remodelling their cytoskeleton and changing their contractile apparatus. Within minutes they could remodel their cell-matrix contacts (focal adhesions) along the direction of tissue stretch (Ciobanasu et al., 2013; Geiger et al., 2009), or expand microtubule network and actomyosin activation so as to maintain tensional homoeostasis through an equal counter-tension (Eastwood et al., 1998). This may produce a counterforce in the matrix tension that might be palpable. Tensional load appears to be perceived by the cell at a nuclear level too. Ex vivo and in vivo studies demonstrate that fibroblasts respond within minutes to mechanical stretching by dynamically remodelling their cytoskeleton with perinuclear redistribution of alpha-actin (Langevin et al., 2005, 2006; 2010). Although this property of rapidly responding to mechanical stress appears to be specific to areolar connective tissues only, it remains significant for fascial work because loose connective tissues form the interface between subcutaneous and perimuscular layers, and are potentially engaged in manual interventions. However, cytoskeletal remodelling failed to occur when distinct matrix properties were produced in gel, as for denser and stiffer connective tissue with increased cross-linked collagen (Abbott et al., 2013). This shows the