Journal of Bodywork & Movement Therapies (2015) 19, 310–326

Available online at www.sciencedirect.com

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journal homepage: www.elsevier.com/jbmt

FASCIA SCIENCE AND CLINICAL APPLICATIONS: EXTENSIVE REVIEW

A unifying neuro-fasciagenic model of somatic dysfunction — Underlying mechanisms and treatment — Part I

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Received 3 October 2014; received in revised form 6 January 2015; accepted 8 January 2015

KEYWORDS
Fascia;
Somatic dysfunction;
Fascial dysfunction;
Fascial mechanisms;
Osteopathic manipulative treatment;
Osteopathic models;
Fasciagenic model;
Fascial treatment;
Fascial release;
Manual therapy

Summary This paper offers an extensive review of the main fascia-mediated mechanisms underlying various dysfunctional and pathophysiological processes of clinical relevance for manual therapy. The concept of somatic dysfunction is revisited in light of the diverse fascial influences that may come into play in its genesis and maintenance. 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 fascial model that integrates neural influences into a multifactorial and multidimensional interpretation of dysfunctional process as being partially, if not entirely, mediated by the fascia.

The fascia

Fascia is an ubiquitous tissue permeating the entire body. It surrounds, supports, suspends, protects, connects and divides muscular, skeletal and visceral components of the organism (Kumka and Bonar, 2012). The fascia plays different physiological and functional roles related to joint stability, general movement coordination, proprioception, nociception (Tozzi, 2012), transmission of mechanical forces (Huijing, 2009); and is associated with wound healing, tissue repair and many connective tissue pathologies such as Dupuytren contracture and the effects of post-operative adhesions (Gabbiani, 2003). By investing each tissue at multiple hierarchical levels, the fascia embodies the element of structural interconnectedness around, within and between body constituents, whilst allowing simultaneous sliding and gliding motions. Since it appears to shape every body constituent, it has been referred to as both an 'organ of form' (Varela and Frenk, 1987) and as an 'organ of innerness' due to its phenomenological dimension of 'in between' the 'outer' (skin) and the 'inner' (visceral

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http://dx.doi.org/10.1016/j.jbmt.2015.01.001
1360-8592/

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endothelium) boundaries of the body (Van der Wal, in preparation). Instead of consisting of different superimposed layers, gliding on each other, it has been proposed as a single architecture with various levels of form and complexity (Guimbertau, 2012). It has been defined as an 'ectoskeleton' (Wood Jones, 1944), in relation to its continuity and function of muscle attachment, enveloping, force transmission and body-wide proprioception. Even at a cellular level, fascia displays an interconnected arrangement with soft tissue fibroblasts within the ECM forming an extensive reticular network, via their cytoplasmic expansions, that permeates the whole body (Langevin et al., 2004). Fibroblasts may actually form adherens and gap junctions at the intercellular levels of contact, allowing also for a more concerted response to mechanical loading (Ko et al., 2000). Furthermore, each fibroblast's cytoskeleton is structurally connected to the external environment, either directly with contiguous cells or through the extracellular matrix (ECM) constituents (Hinz and Gabbiani, 2003a; Fletcher and Mullins, 2010). The entirety of this system may indeed represent a body-wide signaling network (Langevin, 2006) that depends on the relationship between cells and surrounding matrix. Mechanical tension signals from the ECM are transferred through transmembrane mechanoreceptors to the cytoskeleton and cell nuclei, while being transduced into chemical information — via mechanotransduction - so impacting on various aspects of cell behavior and metabolism via the modulation of gene expression (Wang et al., 2009). On the one hand, cells synthetize, secrete, modify and degrade ECM constituents, and on the other, mechanical properties of the ECM affect cytoskeletal organization and cell behavior (Chiquet et al., 2009; Guilak et al., 2006). Cell growth, differentiation, metabolism, contractility, proliferation and apoptosis are under the influence of forces transmitted from the extracellular matrix to the cell via 'focal adhesion' proteins (Goldmann, 2014; Chicurel et al., 1998), and from the cytoskeleton to the nucleus via linker complexes (Isermann and Lammerding, 2013). Focal adhesions are highly flexible and dynamic complexes that are constantly changing and reassembling in response to tensional forces on both sides of the cell membrane. Under increases in tension, these adhesion contacts can become stronger (Parsons et al., 2010) or be released (Liu et al., 2011), and can initiate biochemical signaling cascades for cytoskeletal remodeling and actin polymerization (Machesky and Hall, 1997). Thus they represent a clear pathway for active tensional regulation, whilst mediating vital cell functions, as well as playing a role in disease development (Hoffman, 2014). Indeed, defects in cellular and extracellular mechanics, caused by protein misregulation (Jaalouk and Lammerding, 2009), mutations and/or changes in the expression of proteins linking the cell nucleus with the cytoskeleton (Isermann and Lammerding, 2013), may be implicated in the etiology and development of multiple diseases such as cardiomyopathies and cancer.

Somatic dysfunction

Nowadays, the aim of manual therapy is to promote health and to support the inherent self-regulatory capacities within a dynamic interaction of body, mind and spirit (Rogers et al., 2002) and this is achieved in the osteopathic field by focusing on the musculoskeletal system as the interface of the body's homeostatic potential (Hruby, 1992). Therefore, it is paramount to identify and resolve any dysfunction that may compromise health. As such, the term 'somatic dysfunction' has been defined as any "impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial and myofascial structures" (E.C.O.P., 2011), related to neural and/or vascular elements, that might underlie pathophysiologic conditions. The observational and palpatory features include objective findings such as local changes in tissue texture and temperature, structural and/or functional asymmetries and restriction of motion (DeStefano, 2011b), and subjective elements such as tenderness on palpation and/or altered sensitivity to touch (DiGiovanna, 2005).

Since its origins, osteopathic research has focussed on the mechanisms underlying somatic dysfunction and on its features by exploring related neurological interactions. Louisa Burns (1907) was the first to conduct proper scientific studies to investigate the dysfunctional visceral and somatic reflexes associated with 'osteopathic lesions' (the old term for 'somatic dysfunction'). Through animal autopsy, she found vascular changes in the fascial tissues — 'hemorrhage per diapedesin' — associated with vertebral dysfunctions that showed signs of inflammation, exudation and altered tissue texture (Burns, 1925). These findings confirmed the idea that tissue motion restrictions or alterations associated with 'lesions' were a result of connective tissue inflammation as previously suggested by Marion Clark (1906). Experiments by Cole (1951), Denslow et al. (1947) and Korr (1979) then demonstrated the presence of aberrant somato-visceral reflexes as the basis for the existence of facilitated areas in the spine that corresponded with the palpable features of the 'osteopathic lesion'. Hix (1976), Beal (1985), Kelso et al. (1982), and more recently Fryer et al. (2010, 2006, 2004) continued the research in a similar direction; and most of these findings became organized into the nociceptive model as formulated by Van Buskirk (1990) (Box 1.1).

This model primarily interprets somatic dysfunction from a neurological perspective and as the result of a neurogenic inflammation caused by the release of proinflammatory neuropeptides (such as substance-P and somatostatin) from primary afferent nociceptors following mechanical, chemical or thermal noxious stimulus. If persistent, as in the case of tissue damage, the activation of these nerve fibers alters their own thresholds to produce an area of primary hyperalgesia (peripheral sensitization). The latter, and the associated edematous response, are proposed to underlie respectively the increased sensitivity to touch and the tissue texture changes found in somatic dysfunction. Excessively active primary afferent fibers also relay action potentials into the dorsal horn of the spinal cord and release excitatory amino acids and substance-P. This, in turn, may induce phosphorylation events, altered membrane properties, subsequent gene inductions and the release of facilitatory compounds such as dynorphin — that produces lowered thresholds of activation — up to pathologic changes in the