FASCIAL DYSFUNCTION

Manual Therapy Approaches

Edited by

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PREFACE

For generations anatomists have carefully been trimming away and discarding connective tissues in order to reveal attractive images of muscles, joints and organs that appear in textbooks – images that are often unrecognizable to anyone who has observed the same structures during dissection.

Quite literally, fascia ended up on the cutting-room floor in the interests of presenting a coffee-table artwork, unrelated to physical reality.

Noted Dutch anatomist Jaap van der Wal has even suggested (2009a) that major anatomy texts should be located on the fiction shelves of book stores! He reports: *'I was trained to consider fasciae as connective layers that had to be removed, because they 'covered' something… one had to separate, to dis-sect and the revealed structures ('organs') had to be 'cleaned,' 'cleared' of connective tissue. Connective tissue was something like a covering or sleeve over and in between the dissected structures, often it had to be removed during the dissection procedure.'*

Fascia/connective tissue was seemingly a nuisance to the anatomist, with very little effort by scientists to study or understand its multiple functions.

Research into fascia was therefore largely neglected for decades, with some notable exceptions – including Grinnell (2007): fibroblast mechanics; Hinz & Gabbiani (2010): fibrosis and wound healing; Huijing (1999): force transmission; Ingber (2010): mechanotransduction and tensegrity; Langevin (2006): signaling mechanisms; Purslow (2002): connective tissue structure; Reed & Rubin (2010): fluid dynamics; Solomonow (2009): ligaments; Stecco et al. (2009): continuity of fascial anatomy; Tesarz et al. (2011): neurology of fascia; van der Wal (2009a, 2009b): architecture of fascia; Willard (2007): fascial continuity.

While these examples may seem to indicate a rich degree of research activity, the reality was that for many years, in the main body of science, fascia had been the forgotten tissue – an apparently unimportant, unexciting and superfluous structure that needed to be removed (during dissection) in order for the more glamorous organs, muscles, nerves etc. to be observed and examined.

And then – in 2007 – the first multidisciplinary international congress on Fascia Research (FRC1) was organized and held at Harvard Medical School Conference Centre, Boston.

The event was conceived by clinicians, therapists, practitioners – mainly but not exclusively from the Rolfing/Structural Integration, osteopathic and massage professions. The concept was simple: to invite the best research scientists in the world to come to an event where they could present their findings to an audience of mainly, but not entirely, practitioners who were anxious to understand what mechanisms were producing the clinical results they were seeing daily with their patients – and that remained largely unexplained.

To the genuine surprise of the organizers, most scientists agreed to present – and the event was a phenomenal success.

Scientists were surprised to find an enthusiastic audience of non-scientists and clinicians who were thrilled to be able to pose questions to scientists, many of whom had little idea of the relevance of their studies to manual therapists.

After Boston came Amsterdam (at the Free University, 2009) and then Vancouver (2012). A 4th FRC will take place in 2015 in Washington DC.

The effects of these conferences on worldwide fascia study has been astonishing.

For example, in 2012 the scientist/clinician (and one of the driving forces in the initiation of the Fascia Research Conferences), Tom Findley MD PhD, noted that *'the number of peer-reviewed scientific papers on fascia indexed in Ovid Medline or Scopus has grown from 200 per year in the 1970s and 1980s to almost 1000 in 2010'* – and this trend has continued.

Each of the fascia research events has built on previous ones, with an increasing dialogue emerging between practitioners and scientists, as they inform and question and learn, from each other.

However, a negative effect has also emerged – the misinterpretation of evidence, a sort of pop-version of fascia research, in which complex processes and mechanisms have been over-simplified to the point of the absurd, frequently by under-informed therapists and practitioners, and this is the main reason for compiling this book.

The book aims to explain the clinical relevance of the avalanche of complex scientific information that has emerged from the research conferences in particular, and recent fascia research (which has exploded into action) in general.

The multiple roles of fascia in the body, and what can go wrong, are outlined in the first section of this book, as are chapters describing assessment and palpation methods, and a summary of mechanisms that might explain the effects of various forms of manual treatment.

Section II contains a series of chapters that individually detail a number of the major fascia-related methods of treatment, with evidence for their usefulness, and proposed mechanisms of action.

This book should be seen as work in progress – a translation of current research-based knowledge, designed to counterbalance the plethora of misinformation related to fascial function, dysfunction and treatment.

As new evidence emerges, a currently constant process, so will there be a need for ongoing translation – so that science continues to inform practice.

Leon Chaitow
Corfu, Greece 2014

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Chapter 1

THE CLINICAL RELEVANCE OF THE FUNCTIONS OF FASCIA: TRANSLATING THE SCIENCE

Leon Chaitow

This chapter explores fascia's remarkable functions from the perspective of the manual therapist, highlighting the clinically relevant connections between fascial function, dysfunction, and fascia's anatomical and physiological features.

As outlined in this chapter, fascia has multiple functions, and maintaining and restoring these when they are disturbed – for a variety of reasons ranging from aging to trauma – should be a primary focus of practitioners/therapists.

Definitions - what fascia is and what it does

At present there is no generally accepted way of categorizing fascia. Schleip (2012a) has noted there are currently at least three common ways of codifying fascia:

- The Federative International Committee on Anatomical Terminology (1998) describes fascia as *'sheaths, sheets or other dissectible connective tissue aggregations'* including *'investments of viscera and dissectible structures related to them'* (Terminologia Anatomica 1998)
- *Gray's Anatomy for Students* (Standring et al. 2008) describes fascia as *'masses of connective tissue large enough to be visible to the unaided eye'* noting that *'fibres in fascia tend to be interwoven'* and that it includes *'loose areolar connective tissue'* such as the subcutaneous *'superficial fascia'*
- The most recent international Fascia Research Congress (Schleip et al. 2012b) characterizes fascia as: *'fibrous collagenous tissues which are part of a body wide tensional force transmission system.'*

In order to enhance fascial function when it has been lost or is under strain, we need to:

- Understand the roles of fascia – what it is and what it does (Ch. 1)
- Be aware of how fascia can become dysfunctional – and what symptoms are then likely to result (see Ch. 2)
- Have the ability to evaluate, observe, palpate and assess fascial function and dysfunction, which is the theme of Chapters 3 and 4 (by Tom Myers and this author)
- Be aware of methods that can prevent dysfunction, as well as being able to effectively restore and/or enhance its functionality. Detailed evaluations of 15 separate models of fascial care and treatment are offered in Chapter 5, and in Section 2 (comprising Chs 6-20). These chapters examine what is known about the most widely used fascia-focused therapeutic methods – their methodologies, mechanisms (as far as these are currently understood) as well as the evidence of therapeutic effects (as far as this is available).

An evidence-informed picture should emerge that can be used in clinical reasoning when deciding

on therapeutic choices, as well as providing the basis for explaining possible fascial-involvement to patients/clients. Effective clinical choices in management of existing fascia-related problems should therefore result.

This book's terminology

Taking account of the various definitions listed above, and where appropriate, this book describes individual fascial tissues and structures by considering:

- The functional role of particular tissues, for example *separating* fascia
- The anatomical structures related to the tissues under discussion, for example *cervical* fascia
- Additional descriptors may be given, for example *loose* or *dense* connective tissue
- The relative hierarchical position may be described, for example *superficial* or *deep* fascia.

Note: In this book, due to the current lack of universal agreement regarding terminology, the following descriptors may be found in different chapters or quotes, all referring to the same connective tissue layer: superficial, subcutaneous, loose, non-dense, areolar, pannicular.

The importance of clinically relevant (and accurate) translation of research

The increased interest in fascia, resulting from recent research congresses and symposia and the explosion of research-based publications on the subject, has led to the development and promotion of a variety of 'new' methods of treatment. Many of these attempt to validate themselves via reference to research studies, with a significant number being trademarked (™), or in attempting to protect their uniqueness, by adding a registration symbol (®).

Some of these copyrighted, registered modalities are included as individual chapters in Section 2. The authors of those chapters have explained the methods and the foundations on which the modality has been constructed – that is, the way in which scientific research has been translated into a clinical approach.

This trend towards copyrighting methods emphasizes the need for practitioners, clinicians, and therapists to have the ability to exercise critical evaluation of the evidence presented to them, and be able to then make informed decisions. One of the main aims of this book is to provide the tools that will lead to sound judgments being exercised.

Clinical practice informed by research evidence

Apart from a summary of fascia's anatomical and physiological features, this chapter outlines key aspects of recent fascial research, while also offering translation of new information where this is potentially clinically relevant.

In order to successfully achieve prevention, assessment and successful treatment of fascial dysfunction, we depend on accurate interpretation of basic science findings. The more clearly that we understand fascial anatomy and physiology, and the more we are aware of the implications of research findings, the better able we will be to recognize the roles fascia may play in a variety of painful and dysfunctional conditions.

- What do studies on cells and tissues in a lab actually mean, when it comes to management of fascia-related pain and dysfunction?
- What can we learn from mathematical modeling evaluations of fascial function (see Ch. 5)?
- How do such studies inform treatment methods (see Chs 2 and 4)?
- How does anatomical research, for example emerging from dissection findings, translate into clinical reasoning (see Chs 3 and 4)?
- How might information deriving from imaging studies offer the manual clinician information that is clinically useful (see Chs 3 and 4)?

(Bio)Tensegrity defined

- 'Tensegrity' is an invented word that combines elements of 'tensional integrity.' It describes a structural shape that is determined by the closed, continuous, tensional behaviors of the components of the system – rigid struts and flexible connecting elements, which respond compliantly to tension and compression (Fig. 1.1).
- Levin and Martin (2012) observe that biotensegrity: *'reverses the centuries-old concept*

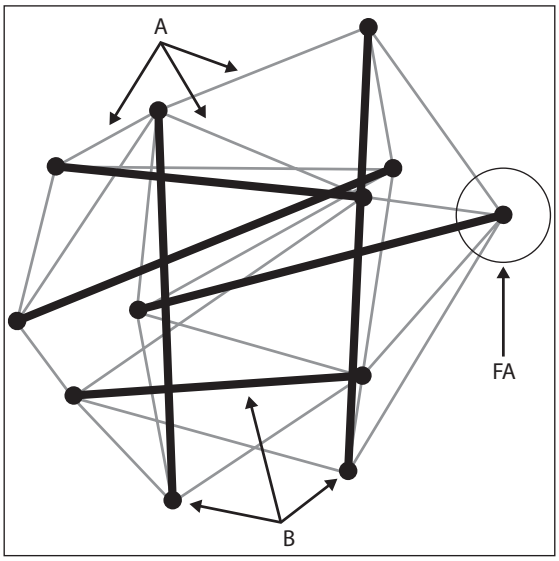

FIGURE 1.1 Biotensegrity model. A pre-stressed tensegrity model representing biotensegrity architecture at all size scales throughout the body – at molecular, tissue, organ and organ system levels – all with compression and tension elements. A = tension features: microfilaments cells, muscle, tendon, ligament, fascia. B = compression: DNA helix, microtubules, extracellular matrix, ribs, bones, fascia. FA = focal adhesion: points of integration between tensional and compressive elements at a cellular level. *Adapted from Swanson 2013*.

> *that the skeleton is the frame upon which the soft tissue is draped, and replaces it with an integrated fascial fabric with "floating" compression elements (bones in vertebrates), enmeshed within the interstices of the tensioned elements.'*

- Ingber (1993) has demonstrated that cells function as independent pre-stressed tensegrity structures, and that molecules, tissues and organs can all be viewed as tensegrity complexes.
- Within these hierarchical biological tensegrity systems (biotensegrity), individual pre-stressed cells are poised and ready to receive mechanical signals and to convert them into biochemical changes, termed mechanotransduction (see below).

The concept of fascially-linked, continuous chains, slings, trains and loops of myofascial structures is discussed later in this chapter. See Box 1.1 for a summary of some of the main functional features of fascia.

Key Point

The (Bio)tensegrity model should remind us that compressive or tensional load has mechanical (and chemical) *mechanotransduction* effects – and that architectural shape matters – and that as it changes so do its functions (see Fig. 1.1). (Mechanotransduction is described later in this chapter. It refers to the ways cells convert mechanical stimuli into chemical activity.)

Box 1.1

Examples of functional characterizations of fascia (Kumka & Bonar 2012)

- **Linking fascia:** This comprises dense connective tissue which can be classified as active or passive, and which 'includes fasciae of muscles, fasciae of regions (head and neck, trunk, limbs), aponeuroses, tendinous arches and neurovascular sheaths.' (Terminologia Anatomica 1998).
  - **Active linking fascia:** contains numerous pain and mechanoreceptors; is active during movement and in stabilizing of joints, and critical for force-transmission (see later in the chapter). It may have the ability to contract to offer pretension to muscles. Example: thoracolumbar fascia; IT tract.
  - **Passive linking fascia:** maintains continuity between structures; has proprioceptive functions; it is passively involved in force transmission via loading from muscles. Examples: ligamentum nuchae, plantar aponeurosis.
- **Fascicular fascia :** This comprises a mixture of both loose and dense connective tissues that provide the architectural shape of muscles:

  - It surrounds muscles (epimysium), as well as separating muscle fibers (perimysium), while covering each muscle fiber (endomysium).
  - Fascicular fascia merges to form dense myotendinous structures. This intramuscular fascicular fascial network acts to both spread and focus forces inside muscles, as well as between synergistic muscles – and via linking fascia – to antagonist muscles. In addition, it provides a range of protective tunnels and pathways for nerves, blood vessels and lymphatic structures.
- **Compression fascia :** This dense connective tissue structure envelops and compartmentalizes the limbs involving sheet-like layers.
  - For example, the crural fascia of the lower limb exists as stocking-like coverings that variously offer compression and tension, while strongly affecting muscular efficiency and venous return. The dense layers are separated by loose connective tissue that facilitates sliding, gliding motions between them, allowing differential actions of individual strata.
- **Separating fascia :** Largely comprising loose connective tissue, this sometimes gossamer-thin material creates envelopes, bags, compartments, tunnels, sheaths and linings that separate organs and body regions, reducing friction while offering shock-absorbing and sliding potentials, in response to movement, tension and distension.
  - Examples include pericardium, peritoneum and synovial sheaths.

Kumka and Bonar (2012) emphasize the ubiquitous nature of fascia when they offer an example of all four of these suggested categories – in the thigh:

- *'Illiotibialband (Linking)*
- *Perimysium of the quadriceps femoris muscle (Fascicular)*
- *Fascia lata (Compression)*
- *Subcutaneous tissue (Separating)'.*

Key Point

The clinical relevance of the notes in Box 1.1 relate to concepts of continuity – of chains, strings and slings, involving fascial connections. Specific clinical implications for manual therapies are discussed in this chapter under subheadings such as *Force transmission* and *Mechanotransduction*.

Fascia: *resilience* as a descriptor

Schleip et al. (2012a) describe fasciae as: ...*'The soft-tissue component of the connective tissue system that permeates the human body. One could also describe them as fibrous collagenous tissues that are part of a body wide tensional force transmission system. The complete fascial net then includes not only dense planar tissue sheets (like septa, muscle envelopes, joint capsules, organ capsules and retinacula), which might also be called 'proper fascia,' but it also encompasses local densifications of this network in the form of ligaments and tendons. Additionally it includes softer collagenous connective tissues like the superficial fascia or the innermost intramuscular layer of the endomysium...the term fascia now includes the dura mater, the periosteum, perineurium, the fibrous capsular layer of vertebral discs, organ capsules as well as bronchial connective tissue and the mesentery of the abdomen.'*

Fascia is part of all the soft tissues of the body:

- Fascia binds, packs, protects, envelopes and separates tissues.
- Fascia invests and connects structures, providing the scaffolding that permits and enhances transmission of forces.
- Fascia has sensory functions, from the microscopic level (for example, individual cell-to-cell communication) to the involvement of large fascial sheets, such as the thoracolumbar fascia (TLF).
- Fascia provides the facility for tissues to slide and glide on each other.

- Fascia also offers a means of energy storage – acting in a spring-like manner via pre-stressed fascial structures, such as the large tendons and aponeuroses of the leg, during the gait-cycle, for example. Think of kangaroos or cats!
- The multiple functions of the connective tissue matrix, with its combined qualities of strength and elasticity – of biotensegrity – can be described by the single word *resilience*. This can be defined as having the ability to adapt to distorting forces and, where appropriate, the ability to return to the original form and position, which is very much the quality of the fascial web. *Resilience* also describes the ability to rapidly recover from illness or injury (see Box 1.2).

Fascia's functional characteristics

The definitions and concepts relative to fascia (above) offer useful ideas as to how we might make clinical sense of the fascial components of the body (Langevin et al. 2011a, Swanson 2013). What emerges is that:

- Fascia is connected to all other tissues of the body, microscopically and macroscopically – so that its three-dimensional collagen matrices are architecturally continuous – from head to toe, from individual cells to major organs.
- Fascia has important colloidal viscoelastic, elastic and plastic properties (see Box 1.2).
- Fascia is richly innervated – participating in proprioception and sensing of pain (see Box 1.3).
- Fascia is functional, not passive. It is dynamic and active – participating in movement and stability.

Key Point

Kumka (personal communication, 2013) offers a clinician's perspective: *'morphological characteristics of fascia – its location, relationships, innervations etc. – are the 'highways' through which fascia should be approached by clinicians.'*

Box 1.2

Fascial properties – thixotropy, plasticity, elasticity, viscoelasticity and the processes of drag, hysteresis and creep

Fascia has a remarkably diverse set of properties – and these have implications for manual therapists. Two key principles should be kept in mind when considering fascial characteristics:

Hooke's Law: Stress imposed on tissues (that is, the degree of force being applied) is directly proportional to the strain produced (e.g. change in length) within the elastic limits of the tissues. See elasticity and plasticity discussion below.

Wolffe's Law: Tissues (e.g. bone, fascia) remodel in response to forces or demands placed upon them. Chen and Ingber (2007) describe how mechanical forces are transmitted into the cytoskeleton and the nuclear matrix of cells, where biochemical and transcriptional changes occur through the process of mechanotransduction.

- Fascia is a **colloid**, defined as comprising particles of solid material, suspended in fluid. The amount of resistance colloids offer to applied load increases proportionally to the velocity of force application. For a simple example of colloidal behaviour, consider a thick mixture of flour and water. If the resulting colloid is slowly stirred with a stick or spoon, movement will be smooth, but any attempt to move it rapidly will be met with a semi-rigid resistance (known as '**drag**'). This quality of colloids is known as **thixotropy** – most evident in the extracellular matrix (described later in this chapter).
- **Collagen** is the most widely distributed protein in the body and this is responsible for the colloidal properties of fascia.
- The thixotropic property of colloids means that the more rapidly force is applied (load), the more rigidly will the tissue respond – hence the likelihood of fracture when rapid force meets the resistance of bone. If force is gradually applied, 'energy' is absorbed by, and stored in, the tissues, with

potential therapeutic implications (Binkley & Peat 1986).
- **Energy-storage** is also a feature of preparation for movement – as explained below (Schleip et al. 2012a).

Gentle, sustained, manual load is a requirement if drag and resistance are to be reduced when attempting to induce changes in those fascial soft-tissue structures most amenable to change i.e. the more superficial, loose fascial layers, rather than the dense, deeper, fasciae.

- Soft tissues display variable degrees of **elasticity** (springiness, resilience or 'give') in order to withstand deformation when load is applied. The elastic property of fascia is possible because these tissues have the ability to store some of the mechanical energy that is applied to them. They are then able to utilize this when returning to their original shape and size when load is removed.
- This process of energy storage, and energy loss, is known as **hysteresis** (Comeaux 2002). The properties of hysteresis (and creep, described below) offer possible explanations for myofascial release (or induction, see Ch. 14) methodology, as well as aspects of neuromuscular therapy (Ch. 15). These qualities should be taken into account during technique application.
- If load is excessive or frequently repeated, it may overcome the elastic potential of tissues, leading to plastic deformation. Permanent change, or a semi-permanent **plastic** distortion, of the connective tissue matrix may result, with a return to normal only achievable with the introduction of sufficient energy to allow a reversal of the deformation process, ideally by means of slowly applied manual therapies (Doubal & Klemera 2002).
- Olson and Solomonow (2009) offer a potent example of the effects of exhausted elasticity resulting from repetitive load: *'viscoelastic tissue properties becomes compromised by prolonged repetitive cyclic trunk flexion-extension which in turn influences muscular activation. Reduction of tension in the lumbar viscoelastic tissues of humans occurs during cyclic flexion-extension and is compensated by increased activity of the musculature in order to maintain stability. The ligamento-muscular reflex is inhibited during passive activities but becomes hyperactive following active cyclic flexion, indicating that moment requirements are the controlling variable. It is conceived that prolonged routine exposure to cyclic flexion minimizes the function of the viscoelastic tissues and places increasing demands on the neuromuscular system which over time may lead to a disorder and possible exposure to injury.'*
- Greenman (1996) has described how fascia manages loads and stresses, in both plastic and elastic ways, with its responses depending – variously – on the type, speed, duration and amount of the load. When load is gradually applied to fascia, elastic reactions follow in which slack is reduced as tissues respond. Persistent load leads to what is colloquially referred to as '**creep**', in which the shape of tissue slowly lengthens or distorts, due to the viscoelastic property of connective tissue. An example of creep is the process of gradual compression affecting intervertebral discs when standing upright.
- Stiffness of any tissue relates to its viscoelastic properties and, therefore, to the thixotropic colloidal nature of collagen/fascia.
- Cantu and Grodin (2001) use the term '**deformation characteristics**' to describe what they see as the 'unique' feature of connective tissue. This term incorporates the combined **viscous (permanent, plastic)** deformation characteristic, as well as the spring-like **(temporary, elastic)** deformation potentials, as summarized above.

Key Point

Awareness of these multiple fascial qualities offers clinicians insights into the multiple ways in which mechanical load can influence what they are touching. Another aspect of that contact is of course how the nervous system is influenced, and also by fluid dynamics – both of which are discussed later in this chapter.

Box 1.3

Major fascial reporting stations

- **Golgi receptors:** These are plentiful in dense connective tissue. In myotendinous junctions and ligaments of peripheral joints they are known as **Golgi tendon organs**, where they respond to muscular contraction. Other Golgi receptors respond to active (but probably not passive) stretching movements – with immediate tonus decrease in related motor fibers. The extent to which manually applied load can elicit Golgi responses remains unclear (Schleip 2003).
- **Pacini and Paciniform mechanoreceptors:** These intrafascial receptors are found in dense connective tissue. Pacini bodies in muscle fascia, myotendinous junctions, deep capsular layers and spinal ligaments are reported to respond to changes in pressure and vibration – but not sustained compression – with effects leading to enhanced proprioceptive feedback and motor control.
- **Ruffini mechanoreceptors:** These are located in dense connective tissue, ligaments of the peripheral joints, dura mater, and outer capsular layers. Some respond to rapid pressure changes, but the majority are affected by sustained pressure, or slow rhythmic – deep – strokes, as well as to lateral (tangential) stretch forces. The effects include reduced sympathetic activity.
- **Interstitial (e.g. Types 3 and 4) mechanoreceptors:** These offer sensory information, and are far more plentiful in – for example – muscle spindles and fascia – than are Pacini and Ruffini reporting stations. The highest density is located in the periosteum. Ten percent are myelinated (Type 3), the remaining being unmyelinated (Type 4). Some are responsive to rapid pressure, others to fascial (and skin) stretching. Others are low threshold – responding to touch that is 'as light as a painter's brush' (Mitchell & Schmidt 1977). They are also known as *interstitial myofascial* tissue receptors (interoceptors). Schleip (2011) suggests that these interoceptors have autonomic influences – on blood pressure, for example.
- The clinical employment of suitable manual strategies in order to influence different neural receptors is explored further in Chapter 5.

Key Point

Awareness of the ways in which different degrees, durations and directions of load may influence the neural structures within fascia offers clinically relevant therapeutic options; for example, light, brief, tangential load (affecting Pacini mechanoreceptors), as compared with moderate, sustained stretch (affecting Golgi tendon organs). A sharp 'cutting/pricking' sensation is a commonly reported sensation when dysfunctional fascia is being stretched or compressed.

Innervation of fascia

- Leaving aside the processes of mechanotransduction (as mentioned above and described more fully below), how the body regulates itself and adapts to its environment depends, to a large extent, on neural reporting that offers the brain information regarding internal and external requirements. Interpretation of such information, received from pain receptors and mechanoreceptors of varying types, determines the way the body responds to the demands of life.
- Proprioceptors are mechanoreceptors that constantly monitor joint position, tendon load, ligament tension, and the status of muscle-tone and contraction. Golgi tendon organs (see Box 1.3) are examples of specialized proprioceptors that are involved in preservation of joint integrity. Proprioception from fascia is largely provided by the mechanoreceptors located within fascial structures, as well as from what has been termed the 'ectoskeleton' (Benjamin 2009). This describes a virtual 'soft tissue skeleton' in which mechanoreceptors in muscles connect

to the fascial layers to which muscle fascicles insert, as part of the process of force transmission (discussed later in this chapter).

- Stecco et al. (2007) have demonstrated the presence of a variety of neural structures in deep fascia – including Ruffini and Pacini corpuscles. This strongly suggests that fascia participates in perception of posture, as well as motion, tension and position (see Box 1.3).

- Additionally, the TLF is densely innervated with marked differences in the distribution of the nerve endings, over various fascial layers: the subcutaneous tissue (superficial fascia) contains a dense presence of sensory mechanoreceptors, such as Pacini receptors and Ruffini endings (see Box 1.3). Substance P-positive free nerve endings – assumed to be nociceptive – are exclusively found in these layers: *'The finding that most sensory fibers are located in the outer layer of the fascia, and the subcutaneous tissue, may explain why some manual therapies that are directed at the fascia and the subcutaneous tissue (e.g. fascial release) are often painful'* (Tesarz et al. 2011).

Note: The TLF is described further and is given particular attention in Chapter 9, The Fascial Manipulation® method applied to low back pain.

Key clinically relevant fascial features

As noted, fascia provides structural and functional continuity between the body's hard and soft tissues, as an ubiquitous elastic–plastic, sensory component that invests, supports, separates, connects, divides, wraps and gives cohesion to the rest of the body – while sometimes allowing gliding, sliding motions – as well as playing an important role in transmitting mechanical forces between structures (Huijing 2007).

The individual elements contained in that summary ('elastic', 'plastic', 'sensory', 'separating', 'gliding' etc.) need to be unravelled and individually discussed – as they are in the opening chapters of the book and in many of the discussions of clinical methods in Section 2.

All of these functions and attributes of fascia are interesting; however, some have greater clinical relevance than others. Potentially clinically relevant fascial features that deserve attention include the ways in which fascial cells respond to different forms and degrees of mechanical load (mechanotransduction), as well as the multiple connecting, wrapping and linking aspect of fascia and how these impact therapeutic assessment and treatment.

Mechanotransduction

Mechanotransduction describes the multiple ways in which cells respond to different degrees of load: torsion, tension, shear, ease, compression, stretch, bending and friction – resulting in rapid modification of cellular behaviour and physiological adaptations – including gene expression and inflammatory responses. Mechanotransduction in connective tissues involves both physical and chemical communication processes that take place between specialized cells, such as myofibroblasts, and their immediate environment, including the soup-like extracellular matrix (ECM) network in which they function. Mechanotransduction processes that involve collagenase and TGF-β1 (transforming growth factor beta-1) are of particular importance and are explained below.

> **Key Point**
>
> The extent to which mechanotransduction effects (due to different forms and degrees of load on cells) can be influenced by manual therapy remains speculative. However, there is evidence that alteration of local tissue tension can influence post-traumatic healing, via mechanotransduction, by means, for example, of changes in collagenase and/or TGF-β1 production. These fascial features are discussed below and more fully in Chapter 5.

Extracellular matrix (ECM)

The ECM is the physical microenvironment in which cells operate. The ECM also provides the opportunity for cells to anchor themselves (using *adhesion complexes* – described below).

The space around and between cells comprises an intricately organized elastic mesh of locally

secreted protein, collagen fibers and polysaccharide molecules, as well as ion-rich water and glycosaminoglycans (GAGs) – such as hyaluronic acid – that make up the ECM. Fascia's key cells, the fibroblasts, synthesize the ECM and collagen in response to load.

- The surface of the cells that produce the ECM's constituent materials – fibroblasts – are directly connected to it by GAGs and collagen fibers
- Extracellular collagen fibers in the matrix turn over rapidly, up to 50% in just 24 hours, demonstrating an active ever-changing nature (Hocking et al. 2009)
- Two principle factors drive the development of myofibroblasts: mechanical stress and transforming growth factor beta-1 (TGF-β1):
  - Myofibroblasts feel stress using specialized matrix adhesions (see below)

Cell matrix adhesion complexes (CMACs)

Cells anchor themselves to the scaffolding of the ECM using soluble adhesive substances. These tie proteoglycans and collagen fibers to receptors on the cell surface. Using this structural architectural framework (see notes on tensegrity, earlier in the chapter), cells sense and convert mechanical signals into chemical responses allowing them to instantly react to external load. Therefore – in addition to their adhesive functions – cell adhesion molecules help to modulate signal transduction:

- *'CMACs are exceptionally flexible and dynamic complexes, and their components undergo rapid and regulated turn-over to maintain delicately balanced streams of mechanical and chemical information. Besides the critical role of CMACs in cell migration, signalling through these complexes provides influence over virtually every major cellular function, including for example cell survival, cell differentiation and cell proliferation.'* (Lock et al. 2008)
- Quite literally cells inform adjacent cells of their physical and chemical responses to altered load. In this process physical load is also transferred to adhesion complexes – the virtual 'limbs' of cells that 'anchor' to the ECM.
- This is particularly relevant during wound healing. When myofibroblasts are activated to perform as structural/architectural stabilizers of the repairing wound, it has been found that they perform these roles most efficiently when the tissues they are operating in are firm/tense, rather than being flaccid/relaxed – with these features (firm/soft) being recognized by their surface receptors, the adhesion features.
- Wipff and Hinz (2009) note that when placed on rigid plastic, myofibroblasts respond by enlarging and developing thick stress-fiber bundles – but when placed on a soft surface their focal adhesions do not develop, remaining relatively small (see Fig. 1.2 and Plate 1).

The therapeutic relevance of fluid dynamics and the ECM are described below.

> **Key Point**
>
> The clinical relevance of an understanding of the nature and functions of the ECM includes awareness that, for example, various forms of load modify its behavior with profound effects on structure and function. Manual therapy's influence on such processes is discussed in Chapter 5, while chapters in Section 2 outline individual therapy models.

Specialized cells, structures and functions of fascia (Benjamin 2009)

Fascia holds the body together, involving a body-wide tensional network of sometimes dense and fibrous, and sometimes elastic and flimsy (gossamer thin), collagenous, soft tissues.

Note: This list is not comprehensive, but highlights the major elements involved in fascial structure and function:

- **Collagen:** Derived from the classical Greek word for glue, kola, collagen is made up of different combinations and concentrations of proteins, bundled together in a variety of fibers. The architecture of collagen is sometimes described in terms of the directions of these fibers, as well as the thickness and density of the resulting structure. Collagen provides support, shape, and stability, while the ratio with

which it is merged with elastin (see below) determines its degree of flexibility (Langevin & Huijing 2009). Tissue features, such as fiber directions, are largely dependent on the tensional and compressive demands to which they are being adapted. Most collagen (around 90%) in the body is Type 1 – for example, found in skin; however, there are many other collagen types (Ross & Pawlina 2011). Purslow and Delage (2012) report that cross-linkages stabilize collagen molecules in muscular fascia, but that these cross-links can become excessive due to aging – as well as being influenced by diet, and the toxic effects of, for example, tobacco smoke. Nutritional and lifestyle influences on fascial function – and the emergence of dysfunction through aging or trauma – are discussed in Chapter 2. Major influences on collagen production involve substances discussed later in this section – see information under the subheadings *Collagenase* and *Transforming growth factor beta-1 (TGF-β1).*

- **Fibroblasts:** These are the commonest cell-type in connective tissue. They secrete collagen proteins that maintain the structural framework of the extracellular matrix – that remarkably diverse mesh that surround cells, which provides scaffolding as well as being a communication network. Fibroblasts alter their function in response to activity and load that modifies their shape (see discussion on mechanotransduction). Kumka and Bonar (2012) have noted that:
  '*Fibroblasts are highly adaptable to their environment, and show a capacity to remodel in response to the direction of various mechanical stimuli, producing biochemical responses. If function changes, as with increased mechanical stress, or prolonged immobilization, deoxyribonucleic acid (DNA) transcription of procollagen in the fibroblasts will change types (e.g., collagen type I into collagen type III), or undifferentiated cell types may adapt towards a more functionally appropriate lineage.*'
- **Collagenase:** When fibroblasts are subjected to either continuous or cyclical load (stretch, shear forces or compression – mechanical – or, for example, involving edema) they secrete collagenases, enzymes that break the peptide bonds in collagen, preventing excessive connective tissue formation, for example during wound healing (Tortora et al. 2007).
- Cyclical stretching (or compression) of fibroblasts – involving approximately 10% of available elasticity – doubles collagenase production.
- In contrast, continuous stretching is only 50% as effective (Langevin 2010, Carano & Siciliani 1996). Additionally, Bouffard et al. (2009) report that brief, light, stretching of tissues that house fibroblasts promotes collagenase production, decreasing the formation of new collagen structures, therefore reducing the likelihood of fibrosis. There are numerous other mechanotransduction processes; however, the example given here offers a sense of the potentials for mechanical (via exercise and/or manual therapy and/or acupuncture) influences on cell behavior.

Key Point

Of potential clinical importance is the observation that lightly loaded cells lose their sensitivity to mechanical deformation after 10–15 minutes, requiring a rest period or a different stimulus to recommence collagenase secretion.

The observation that intermittent load has a greater influence on collagenase production than sustained load, is also clinically relevant.

In general, varying degrees and forms of load – including exercise, light, heavy, sustained, cyclical mechanical stimulus – modifies cellular behaviour and gene expression, influencing tissue remodelling – involving enzymes and various growth factors such as TGF-β1.

Unsurprisingly, exercise enhances collagen formation, while inactivity dramatically diminishes this, in muscles but not in tendons.

- **Myofibroblast:** These derive from fibroblasts that have been stimulated to change their form and function, as a result of mechanical load and consequent deformation. Myofibroblasts have some of the characteristics of smooth-muscle cells, containing actin