Chapter 1
The functions of fascia: translating research into clinical relevance

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This chapter explores fascia's remarkable functions from the perspective of the manual therapy practitioner, highlighting the practical and clinically relevant connections between fascial function, dysfunction, and fascia's anatomical and physiological features, as informed by recent research.

Fascia, as explained in this chapter, has multiple functions, and maintaining and restoring these when they are disturbed or dysfunctional – for any of a variety of reasons – should be a primary focus of practitioners/therapists.

In order to enhance fascial function, 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 mainly Ch. 2)
- Have the ability to evaluate, observe, palpate and assess fascial function and dysfunction, which is the theme of Chapters 3 (by Tom Myers) and 4 (by this author)
- Be aware of methods that can prevent dysfunction, as well as being able to effectively restore and/or enhance its functionality (see mainly Ch. 5)
- Cautiously interpret important basic science research that has helped to explain many of the underlying mechanisms operating in response to manual and movement therapies (see mainly Ch. 5)
- Understand different models of fascial care, treatment and management, which are also offered in Chapter 5, and in Section II (comprising Chs 6–21). Those chapters examine what is known about the most widely used fascia-focused therapeutic methods – their methodologies, mechanisms, as well as the evidence of therapeutic effects (as far as this is available).

An evidence-informed picture emerges, that can be used as a guide in clinical reasoning when deciding on therapeutic choices, as well as providing the basis for explaining possible fascial involvement relating to their symptoms to patients/clients.

A range of effective clinical choices, for the management of fascia-related problems, emerges from this information-rich background.

Definitions – what fascia is and what it does

At present, there are a variety of definitions, some based on fascia's morphology – its form, structure and architecture – as well as definitions deriving from fascia's multiple functions.

As can be seen in Box 1.1, there is no generally accepted way of categorizing or defining fascia. This unsatisfactory situation resulted in the formation – by the Fascia Research Society (https://fasciaresearchsociety.org/) – in 2015, of the Fascia Nomenclature Committee (FNC). Since then the FNC has worked on improving the language describing fascia's multiple aspects and functions.

Various definitions are listed in Box 1.1.

Box 1.1 Defining fascia

Morphological definitions of fascia include:

- Terminologia Anatomica (FIPAT 2011): '*Fascia consists of sheaths, sheets or other dissectible connective tissue aggregations…* [This term] *includes not only the sheaths*

of muscles but also the investments of viscera and dissectible structures related to them.'

- Gray's Anatomy (Standring 2016): '*Fascia is a term applied to masses of connective tissue, large enough to be visible to the unaided eye. Its structure is highly variable but, in general, collagen fibres in fascia tend to be interwoven and seldom show the compact, parallel orientation seen in tendons and aponeuroses.*'

Functional definitions include:

- Fascia Research Congress (Findley & Schleip 2007): '*Fascia is the soft tissue component of the connective tissue system that permeates the human body forming a whole-body continuous three-dimensional matrix of structural support. It interpenetrates and surrounds all organs, muscles, bones and nerve fibres, creating a unique environment for body systems functioning.* [It includes] *all fibrous connective tissues, including aponeuroses, ligaments, tendons, retinacula, joint capsules, organ and vessel tunics, the epineurium, the meninges, the periostea, and all the endomysial and intermuscular fibres of the myofasciae.*'
- Schleip et al. (2012a): *'One could describe Fascia 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.'*
- Kumka and Bonar (2012): *'Fascia is an uninterrupted viscoelastic tissue which forms a functional 3-dimensional collagen matrix. It surrounds and penetrates all structures of the body extending from head to toe, thus making it difficult to isolate and develop its nomenclature...* [it] *is virtually inseparable from all structures in the body and acts to create continuity amongst tissues to enhance function and support.*'

Towards a more comprehensive FNC definition

The Fascia Nomenclature Committee (FNC) has summarized some of fascia's agreed functions: '*including (but not limited to) architectural/structural, neurological functions, biomechanical force transmission, morphogenesis, and cellular signal transmission*' – while recognizing that it also '*interpenetrates and surrounds all organs, muscles, bones and nerve fibres*'.

The current suggested definition of fascia by the FNC (still being actively debated and refined) is:

- Fascia Nomenclature Committee (FNC) (Adstrum 2017): '*The fascial system consists of the three-dimensional continuum of soft, collagen-containing, loose and dense fibrous connective tissues that permeate the body. It incorporates elements such as adipose tissue, adventitia and neurovascular sheaths, aponeuroses, deep and superficial fasciae, epineurium, joint capsules, ligaments, membranes, meninges, myofascial expansions, periostea, retinacula, septa, tendons, visceral fasciae, and all the intramuscular and intermuscular connective tissues including endo-/peri-/epimysium. The fascial system surrounds, interweaves between, and*

interpenetrates all organs, muscles, bones and nerve fibres, endowing the body with a functional structure, and providing an environment that enables all body systems to operate in an integrated manner.'

Terminology used in this book

Taking account of the various definitions listed above, 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
- Fascia's relative hierarchical position may be described, for example, 'superficial or deep' fascia.
- Due to the current lack of universal agreement regarding terminology, the following descriptors (and others) may also be found in different chapters or quotes, all referring to the same connective tissue layers: superficial, subcutaneous, loose, non-dense, areolar, pannicular.

(Bio)Tensegrity

- 'Tensegrity' 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).
- Tensegrity is an invented word that combines elements of 'tensional integrity'.
- Levin and Martin (2012) observe that biotensegrity: '*reverses the centuries-old concept 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*

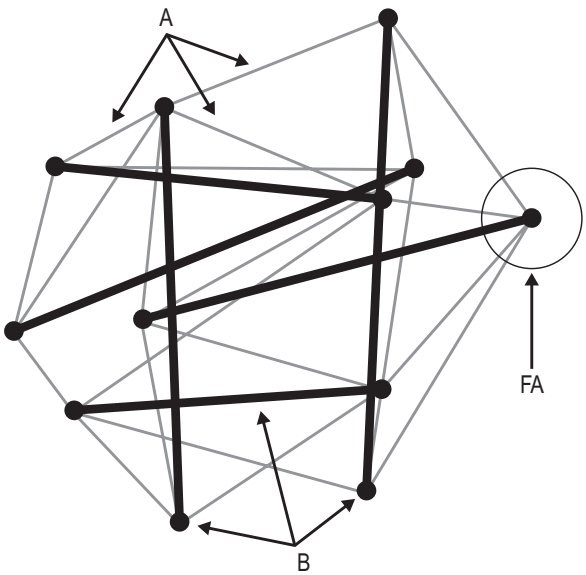

Figure 1.1
Biotensegrity model. A pre-stressed tensegrity model representing biotensegrity architecture at all size scales throughout the body – at molecular, tissue, organ and organ system levels – all with compression and tension elements. A = tension features: microfilaments cells, muscle, tendon, 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.*

(bones in vertebrates), enmeshed within the interstices of the tensioned elements.'

- As Scarr (2014) has observed: '*The musculoskeletal system is not about 'muscles moving bones', but a dynamic interplay between tension and compression*'. The concept of continuously linked chains, trains, slings and loops of myofascial tissues, transmitting and absorbing load throughout the body, is discussed later in this chapter, in particular under the subheading *Force transmission, load transfer and fascia*.
- 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. This extremely important cellular function, termed *mechanotransduction*, is discussed in more detail below.
- Kumka (personal communication, 2013) offers a clinician's perspective: '*the morphological characteristics of fascia – its location, relationships, innervations etc. – are the 'highways' through which fascia should be approached by clinicians*'.

Some of the main functional features of fascia are listed below.

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 – because as shape changes so do functions (see Fig. 1.1). (Mechanotransduction is described later in this chapter. It refers to the ways cells convert mechanical stimuli into chemical activity.)

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 (Box 1.2).
- Fascia is richly innervated – participating in proprioception, interoception and sensing of pain (Box 1.3).
- Fascia is functional, not passive. It is dynamic and active – participating in movement and stability.
- Fascia is part of all the soft tissues of the body, where it binds, packs, permeates, protects, envelopes and separates tissues.
- Fascia invests and connects structures, providing the scaffolding that permits and enhances transmission and absorption 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 vast 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 tensegrity structures, such as the large tendons and aponeuroses of the leg, during the gait cycle, for example. Think of kangaroos or cats!
- Leaving aside the processes of mechanotransduction (described more fully below), how the body regulates itself and adapts to its envi-

ronment 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 the 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 the 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 fibres 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 as applied to low back pain*.

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.

Wolff'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 that colloids offer to applied load increases proportionally to the velocity of force application. For a simple example of colloidal behavior, consider a thick mixture of flour and water. If the resulting colloid is slowly stirred with a stick or spoon, the 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. 13) methodology, as well as aspects of neuromuscular therapy (see Ch. 14). 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 become 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.'*
- See also notes under subheading later in the chapter, *Fascia – resilience as a descriptor... and the seeds of dysfunction.*

- 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.
- The stiffness of collagen/fascia relates to the thixotropic colloidal nature of its viscoelastic properties, as well as to osmotic pressure – the fluid content of collagen. *'...water plays a crucial role in stabilizing the structure of the collagen molecule and is an essential and active part of the protein unit.'* (Masic et al. 2015).
- In a manual medicine context, hysteresis is the rate at which connective tissue responds to the loading and unloading of a compressive (deforming) force. More specifically it is defined as the difference in viscoelastic behavior (energy loss) (Chila 2003).
- For example: *'Altered hysteresis characteristics in tissues that were previously 'boggy' or edematous, might be recognized by a specific lag time in tissue recoil, following diagnostic palpation, compared to 'normal' or to fibrotic tissues'* (Barnes et al. 2013)
- Cantu and Grodin (2001) used 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

Fascia's multiple functions and characteristics, as well as its global presence throughout the body, suggest that it is likely to be involved in almost all aspects of dysfunction and disease – either as an effect or as part of the etiological sequence leading to dysfunction and disease. This would be more readily understood if fascia were seen to be a veritable system – such as the circulatory, or nervous systems.

As Kumka expresses it: '*Fascia is an innervated, continuous, functional organ of stability and motion.*' (See also Box 1.6.)

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 2003a,b).

- **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 a 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 affects Pacini mechanoreceptors
- moderate, sustained stretch affects Golgi tendon organs

A sharp 'cutting/pricking' sensation is a commonly reported sensation when dysfunctional fascia is being stretched or compressed.

Clinically relevant fascial features

As noted, fascia provides structural and functional continuity between the body's hard and soft tissues, as a 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 II.

All of these functions and attributes of fascia are interesting; however, some have greater relevance than others.

An important clinically relevant fascial feature that deserves attention is the way in which fascial cells respond to different forms and degrees of mechanical load – i.e. mechanotransduction.

A great deal of emphasis is to be found in these opening chapters, and in the discussions of the different therapeutic models in Section II, relating to physical, and mechanical, influences on fascia's behavior. In a way, this emphasis is deliberate, in order to counterbalance neurophysiological interpretations as to the effects of therapeutic interventions.

That neurophysiology is a major feature of almost all dysfunction is not in question (see Box 1.3) – however, many clinically relevant effects are unrelated to neurophysiology, and result directly from mechanically induced changes in cellular shape – hence the emphasis given to mechanotransduction (Box 1.4) (Coppieters & Butler 2008, Hakim & Grahame 2003).

Box 1.4 Mechanotransduction

Mechanotransduction describes the multiple ways in which cells respond to different degrees of mechanical load: torsion, tension, shear, compression, stretch, bend and friction – resulting in rapid modification of cellular behavior 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 fibroblasts and telocytes (described below), and their immediate environment, including the soup-like extracellular matrix (ECM) network (described below), in which they function.

1. Mechano-coupling occurs when – for example – compression or shear force transduces into chemical signals, within or between cells, altering metabolism, internal biochemistry and gene expression (Wipff & Hinz 2008).
2. Effector cells in connective tissues respond to mechanical loading by synthesising protein, promoting tissue repair and remodeling (Khan & Scott 2009, Kjaer et al. 2009).
3. Fibroblasts (described in detail below) respond to the *degree, direction, frequency and duration* of mechanically imposed strain, triggering both pro- and anti-inflammatory responses (as appropriate at the time), as well as range of motion (Standley & Meltzer 2008).
4. In a 2012 study, Hicks et al. observed that cyclic short-duration stretches (CSDS) – such as occur during repetitive motion strain – lead to musculoskeletal injury and an inflammatory response (involving the affected fibroblasts). Myofascial release (see Ch. 13) uses an acyclic, long-duration stretch. When this was applied to traumatized myofascial cells, in a laboratory setting, they started to secrete interleukin 6 – which is vital to modulation of the inflammatory processes associated with injury healing and repair.
5. Huang et al. (2013) have summarized the possible ways in which cellular behavior may be influenced biomechanically through the effects of mechanotransduction: '*Mechanotherapies that target mechanotransduction signalling pathways can mainly aim to modulate one of their four phases:*
   i. *the mechanocoupling phase, where the external mechanical signal is converted into a mechanical signal in the vicinity of the cell*
   ii. *biochemical coupling, where the local mechanical signal is transduced into a biochemical signal, resulting ultimately in genetic or protein changes*

iii. *signal transmission, where the biochemical signal is then passed from the sensor cells to the effector cells*

iv. *the effector cell response.'*

It is reasonable to question to what degree the forms of externally applied load – as used in manual therapies and exercise – are transmitted to mechanosensitive cells in the tissues (see Box 1.7, later in this chapter).

The examples given here, of mechanotransduction processes, offer a glimpse of the potentials for mechanical influences on cell behavior, for example as a result of exercise, manual therapy and/or acupuncture.

Key Point

The extent to which mechanotransduction effects can be influenced by manual or movement therapies (due to different degrees of imposed mechanical load) remains underresearched. However, there is evidence that alteration of local tissue tension influences post-traumatic healing, via mechanotransduction affecting the biotensegrity architecture of cells. These fascial features are discussed more fully in Chapter 5.

Fascia's specialized cells, structures and functions (Benjamin 2009, Schleip et al. 2012b)

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 of fascia-related cells is not comprehensive, but highlights the major elements involved in fascial structure and function.

Fibroblasts

- Fibroblasts are the commonest cell type in connective tissues. They secrete collagen proteins that help to maintain the structural framework of the extracellular matrix (ECM) – that remarkably diverse mesh that surrounds cells, and 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, above) (Fig. 1.2).
- 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 pro-collagen 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.*'
- 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.