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 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
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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 tensitional 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 musculo-skeletal 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 myo-fascial 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-
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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 behaviour, 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

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- **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).
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).
Chapter 1

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.

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.

- 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 (interceptors). Schleip (2011) suggests that these interceptors 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.

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 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 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 remodelling (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
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.
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Myofibroblasts (Hoffman et al. 2011)

- These contractile cells derive from fibroblasts that have been stimulated to change their form and function, as a result of mechanical load and consequent deformation. Myofibroblasts contain actin and myosin, giving them the ability to contract. They help to repair, reconstruct and remodel injured tissue by helping create new ECM (see ECM description under Fibroblasts, above) – and by exercising high contractile force. If these processes become unregulated, tissue contracture (as in Dupuytren’s disease) and development of fibrosis may result. Fibrosis is discussed further in Chapter 2.

- The two key features that are involved in transformation of fibroblasts to myofibroblasts are mechanical load, and the chemical TGF-β1. Particular levels of mechanical stress are required to induce myofibroblast development, such as occur in trauma.

- Myofibroblasts are embedded in the ECM, sensing changes in tension, using specialized matrix adhesions – the structures that allow them to stick to surfaces in their microenvironment, and with which they interact to help orchestrate physiological tissue repair (see Fig. 1.5).
Collagen

- The word ‘collagen’ derives from the classical Greek word for glue, ‘kola’, and is made up of different combinations and concentrations of proteins, bundled together in a variety of fibers. Collagen molecules bind together to form microfibrils.

- Several microfibrils wrap around each other in a spiral formation, to create collagen fibrils, and when fibrils are spiralled together a collagen fiber is the end-result.

- When collagen fibers twist around each other, fibrillar bundles are formed – to create tendons and ligaments – geared to cope with mechanical stress.

- When under a traction force the spirals wind together for even greater stability, enormously high tensile strength is achieved – greater than steel.

- The mechanical strength of what are essentially otherwise relatively unstable triple helices, is largely achieved thanks to the multiple links between the triple helix of micro-fibers, fibrils and fibers (Fullerton 2006) as well as to internal osmotic pressure (Masic et al. 2015) (Fig. 1.3).

- The cross-links are biochemical bridges found in all the triple helices – for the formation of which vitamin C is an important ingredient (Van den Berg 2010).

- ‘Changes in tissue shape lead to electrical voltage changes, as molecules use piezoelectrical activity to organize the architecture of the tissue’ (van den Berg 2010). (Piezoelectric: the ability of some materials (including fascia) to generate an electric potential in response to applied mechanical stress.)

- Purslow and Delage (2012) report that cross-linkages stabilize collagen molecules in muscular fascia, can become excessive due to ageing – 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.

- Collagen provides support, shape, and stability, while elastin – an associated protein in connective tissue (described later in this chapter) – determines fascia’s degree of flexibility (Langevin & Huijing 2009).

- Collagen has, as a primary role, the creation of mechanical support in many tissues, such as those of fascia, bone, tendon, skin and blood vessels.

- Water is also an integral part of collagen structure, and its mechanical properties depend on hydration, with dehydration leading to a powerful shrinkage-contraction of the collagen molecule (Masic et al. 2015).

- Tissue features, such as fiber directions, are largely dependent on the tensional and compressive (tensegrity) forces to which they are obliged to adapt. ‘Contractile forces of the cell stresses, exerted through changes during development, interstitial fluid pressure, and physical activity, alter the strain on fibrils and reinforce collagen in the direction of loading’ (Bhole et al. 2009).

- Most collagen (around 90%) in the body is Type 1 – for example, found in the skin; however, there are many other collagen types (Ross...
FIGURE 1.3
(a) The triple collagen bundle depends for stability on internal stress features including hydraulics. If stresses are not balanced, the triple helix frays like rope and eventually unravels and collapses at the cleavage point in the collagen. The analogy of a rugby scrum suggests that they are held in place by balanced forces, but they buckle and collapse in a similar manner. After collapse, the scrum is reformed. Similarly, in collagen, after metalloproteinases clean up the cleavage site, new collagen helix fiber is created. (b) Osmotic pressure/hydration of fascial fibers. (b) Redrawn from Masic et al. (2015).
Buckling exposes collagen to specific enzymes (matrix metalloproteinases or MMPs) at cleavage sites, initiating degradation, and subsequent repair and regeneration.

‘The underlying dynamic molecular changes in collagen structure, which may involve single atoms, were readily observed through tracking of enzyme binding’ (Dittmore et al. 2016).

**Collagen’s self-repair process?**

- Flynn et al. (2010) observed that: ‘applied mechanical strain preferentially preserves collagen fibrils in the presence of enzyme-related degradation.’ Therefore, when there is optimal tensional support, collagen degrades more slowly.

- Humphrey et al. (2014) explain that 'Mechanical loads on trans-membrane complexes and cytoskeletal structures are fundamental to the cell–ECM interactions that govern mechanical homeostasis in health – [requiring] – that cells first sense the mechanics of the ECM and then regulate it to maintain the desired properties; loss of these complementary homeostatic processes leads to fibrosis, mechanical failure or other pathologies.'

- Dittmore et al. (2016) reported that unbalanced tensional support affecting collagen fibers periodically results in spontaneous deformation and collapse (‘buckling’) of individual fibers, at what they term ‘cleavage sites’. This process triggers self-generated repair and remodeling: ‘Collagen fibrils resemble nanoscale cables that self-assemble and our experiments reveal unanticipated defects that form along the collagen fibrils .... the initiation sites of collagenase activity, representing a strain-sensitive [thermally-labile] mechanism for regulating tissue remodelling.’

- Triple-helix collagen fibrils contain billions of sites, at 1 micro-meter intervals, vulnerable to spontaneous buckling – if the tensional forces that support them are not optimal.

- When under appropriate tension, the number of buckling (cleavage) sites decrease.

**Key Point**

Balanced tension – externally derived, as well as from internal osmotic pressure (i.e. tensegrity) – allows collagen to maintain structural integrity and optimal function, for longer than if mechanical tensions are unbalanced or inadequate.

The homeostatic processes associated with collagen degradation, self-repair and remodeling, are seen to relate directly to features such as balanced mechanical tension and adequate hydrostatic pressure.

**Smooth muscle cells**

- Unsurprisingly, smooth muscle cells (SMCs) are located in smooth muscle tissue such as are found in the walls of viscera and blood vessels.

- Perhaps more surprisingly, SMCs are also embedded in connective tissues. These non-striated, spindle-shaped cells are capable of slow rhythmic involuntary contractions. Schleip et al. (2005) suggest that their presence in fascia may relate to fascial tone, influencing musculoskeletal dynamics.

SMCs are sensitive to changes in the pH of the tissues, constricting when alkalinity increases, as occurs with a chronic upper chest breathing pattern or hyperventilation (Chaitow et al. 2013, Krapf et al. 1991)
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Key Point

In Chapter 2 there is a short discussion of the effects of altered pH on fascia – sometimes resulting from patterns of overbreathing (such as hyperventilation). This causes smooth muscles to constrict, with potential relevance for fascial tone.

Telocytes

Telocytes are specialized mechanosensitive connective-tissue cells that have roles in tissue homeostasis and that are capable of organizing and accomplishing – alone or together with fibroblasts, macrophages and others – multiple tasks associated with facilitating cellular repair, regeneration and remodeling, throughout the body (Cretoiu et al. 2015, Edelstein 2016).

As Bei et al. (2015) explain: ‘Telocytes [are found in] skeletal muscle interstitium [close to] capillaries, nerve fibres, satellite cells and myocytes.’ – and – ‘Functionally telocytes form a 3D interstitial network by homocellular and heterocellular communication… through close associations with fibroblasts, smooth muscle cells, endothelial cells, immunoreactive cells and nerve endings, which suggests conventional roles in mechanical support, immune surveillance and intercellular communication and signaling… involved in the maintenance of tissue homeostasis.’ Such diverse functions necessarily involve a range of forms of information transfer – featuring diverse communication mechanisms.

Telocytes are distributed in the interstitial extracellular matrix of all body tissues. They have telopodes (Fig. 1.4) involved in intercellular communication with other telocytes, as well as surrounding structures, such as blood vessels, nerve endings, smooth muscles, glandular elements, and those covering epithelia.

Edelstein & Smythies (2014) have discussed the possible role of telocytes in pathology: ‘[They trans- mit] integrated signals to neighbouring cells... their interrelationship with neural stem cells and neurogenesis in the context of neurodegenerative disease is just beginning to be explored.’
‘The consensus is that telocytes [probably] form an extensive inter-cellular information transmission and executive system... utilizing electric currents, small molecules, exosomes and possibly electrical events within the cytoskeleton to modulate homeostasis stem cell activity, tissue repair, peristalsis, anti-cancer activity and other complex functions in many organs.’

Can telocytes be influenced biomechanically?

Humphrey et al. (2014) have observed – in relation to fascial homeostasis – that: ‘Soft connective tissues resident cells continually read and respond to environmental cues to promote homeostasis, including maintenance of mechanical properties of the extracellular matrix (ECM) – fundamental to cellular and tissue health.’

Since telocytes are – like fibroblasts and myofibroblasts – mechanosensitive, what practitioners using manual and exercise-based therapies need to know is whether – and to what degree – telocytes can be therapeutically influenced by movement and/or externally applied mechanical loading (or unloading) procedures. At this time, such influences remain uncertain.

For other breaking news please see the foreword by Tom Findley on the interstitium.

The extracellular matrix (ECM)

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 comprise the ECM.

Fascia’s key cells, the fibroblasts, synthesize the ECM and collagen, in response to mechanical load, that provides structural and biochemical support to the surrounding cells. The surface of the cells that produce many of the ECM’s constituent materials – the fibroblasts – are directly connected to it by GAGs and collagen fibers (using adhesion complexes – described below).

This matrix provides the route of transport for nutrients and waste materials between the outer aspect of the endothelial barrier to the parenchymal (functional) cells of any tissue.

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).

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 the 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).
- Using various communication strategies, cells inform adjacent cells of their physical and chemical responses to changes in mechanical load, perceived by their CMACs, that are anchored to the ECM.
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- This is particularly relevant during wound healing. When myofibroblasts are activated (from fibroblasts) 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, strong stress-fiber bundles – but when placed on a soft surface their focal adhesions do not develop, remaining relatively small (Fig. 1.5).

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 mechanical load modify its behaviour, with profound effects on structure and function. Manual therapy’s influence on such processes is discussed in Chapter 5, while chapters in Section II outline individual therapy models.

**Elastin**

- This key ECM constituent allows tissues in the body to resume their shape after stretching or contracting; for example, where superficial fascia needs to allow significant sliding motion, as on the dorsum of the hand, levels of elastin are increased to allow for restoration of position and shape, following movement.

**Fibrillin**

- This constituent of the ECM is essential for the formation of elastic fibers that provide strength and flexibility to connective tissue. When it is genetically mutated, Marfan syndrome results.

**Fibronectin**

- This is a glue-like substance that, for example, binds to integrins, the cell-spanning receptor proteins.

**Integrins**

- Aerial-like protein projections, vital for cell-to-cell and cell-to-ECM communication. Myers (2012) has explained this superbly: ‘The ECM is connected to cell membranes and through them to the cytoskeleton via hundreds or thousands of binding integrins on the cell surface. Forces from outside the cell are transmitted via these adhesive connections to the inner workings of
the cell. Thus, we can now understand that each cell, as well as ‘tasting’ its chemical milieu, is ‘feeling’ and responding to its mechanical environment ... forces also move in the other direction – from the cell to the ECM – in the case of muscular or (myo)fibroblast contraction that gets conveyed through the membrane to the surrounding ECM.’

**Transforming growth factor beta-1 (TGF-β1):**

- This is a secreted protein that performs many cellular functions, including the control of cell growth, cell proliferation and cell differentiation. New collagen is formed in response to mechanical loading (as in exercise or manual therapy) that stimulates, among other substances, TGF-β1. According to Langevin (2006), brief stretching decreases the effects of TGF-β1 in the production of additional collagen – which may be relevant to manual therapy techniques aimed at reducing the risk of scarring/fibrosis. Inactivity has been shown to dramatically reduce collagen proliferation function in muscle tissues – but not in tendons (Kjaer et al. 2009).

**Fascial lubricants**

**Proteoglycans (PGs), glycosaminoglycans (GAGs)**

- These water-loving, mucus-like substances, largely made up of protein and sugar molecules in varying combinations, form the ground substance – a loosely packed feature of the ECM. These substances have important roles in assisting diffusion of nutrients and waste products, as well as offering a home for chondroitin and other sulfates, and various collagen fibers with stabilizing, compressive or tensile functions.

**Hyaluronic acid (HA)**

- This component of PG and GAG has lubricating functions and assists in maintaining the viscosity of the ECM. A layer of lubricating hyaluronic acid lies between the deep fascia and muscles (Langevin et al. 2009).

- Load effects on HA: Using sophisticated mathematical modeling methods, Roman et al. (2013) compared the lubrication effects of HA when three different forms of manual load were applied: constant sliding, perpendicular vibration, tangential oscillation. The degree of effectiveness was judged by increased fluid-pressure of HA, since ‘the pressure generated in the fluid between the muscle and the fascia ... [during treatment] ... causes the fluid gap to increase’. The findings were that ‘perpendicular vibration and tangential oscillation may increase the effects of the treatment in the extracellular matrix, providing additional benefits in manual therapies that currently use only constant sliding motions.’

- Langevin et al. (2011b) have shown that reduction of fascia’s gliding potential in the thoracolumbar area (described technically as ‘reduced thoracolumbar shear strain’) correlates with increased thickness of the TLF and, in males, seems to predispose to low back pain. This gender-specific link between a free sliding motion of fascia in the TLF, the thickness of the connective tissue layers, and low back pain, remains unexplained.

**Fluid dynamics and fascia**

- When Klingler and colleagues (2004) examined the effects of stretching on the water-binding capabilities of the ground substance of pig [and human] connective tissue, they found that the water content reduced – as though squeezing a sponge. This effectively reduced the stiffness of the tissues. After around 30 minutes the water content increased again, so that several hours after the stretch there was an increase in hydration. In these conditions fibroblasts increase their produc-
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- Meert (2012) notes that the fluid in the ECM ‘creates a transport space for nutrients, waste materials and messenger substances and actually facilitates homeostasis between the extracellular and the intracellular region. In addition, the lymphatic system filters this supply out of the ocean of interstitial fluids and drains it into the venous system.’

- Water molecules build dipoles and therefore water flow also means the flow of energy and information. It is therefore not a total surprise that some investigators suggest that chains of water molecules along collagen fibrils are acupuncture meridians (Ho 2008).

- ‘Fibroblasts respond to connective tissue tension by homeostatic adjustment of interstitial fluid pressure and transcapillary fluid flow. Transmission of forces from fibroblasts to the extracellular matrix ... causes changes in interstitial hydrostatic pressure ... influencing the response to injury and inflammation’ (Langevin et al. 2005).

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- Reed et al. (2010) studied the ways in which fluid moves between peripheral lymph and blood vessels and the interstitial tissues. During inflammation, for example, they found that the physical properties of the loose connective tissues (involving GAGs and hyaluronic acid, as described above) can change within minutes, resulting in as much as a hundred-fold increase in fluid flow. They propose that: ‘connective tissue cells apply tensile forces on ECM fibres that in turn restrain the under-hydrated ground substance from taking up fluid and swelling.’ Connective tissue is seen to be an active feature of fluid balance and physiology.

- Pollack (2013) has reported on the types and quality of water in the living body:
  - ‘Experimental evidence confirms the existence of an ordered, liquid crystalline, phase of water ... [this] envelops every macromolecule in the body, including those of the fascia ... most water [in the body] is liquid crystalline water.’
  - ‘Mechanical pressure and radiant energy convert bulk water into liquid crystalline water that is characteristic of healthy tissue and necessary for normal physiological function.’

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**Key Point**

The clinical relevance of fluid dynamics and fascia points to fluid/water having a major influence on flexibility and stiffness, as well as on the distribution of substances, such as nutrients, pro- and anti-inflammatory products, and the drainage of debris during processes such as inflammation and tissue repair – with influences on homeostatic function.
The bigger picture: structural characteristics of fascia

Describing the different fascial structures independently, as below, may detract from the reality, that the fascial network is continuous; every part of it is joined to every other part, structurally and functionally.

This has been succinctly expressed as follows by Schleip et al. (2012b, Introduction): ‘The fascial body is one large networking organ, with many bags and hundreds of rope-like local densifications, and thousands of pockets within pockets, all interconnected by sturdy septa as well as by looser connective tissue layers.’

The text below describes some of the different forms, types and locations of fascia and its geography (see also Box 1.6) followed by a summary of how this translates into one of the most clinically useful aspects of current research evidence – load transfer.

Fascial layers and bags (Willard 2012a)

Tensional forces, resulting from muscular contractions and load-demands, are spread to adjacent – and distant – tissues via fascial sheets, as well as by means of densified threads, strings, straps, wrappings, and rope-like connections (tendons, ligaments, retinacula, etc.).

Fascia also comprises a complex variety of bags, septa, pockets and envelopes that contain, separate and divide tissues and structures – while in many instances allowing a sliding, gliding facility that provides the basis for frictionless movement between soft-tissue layers. This can be lost or reduced by adhesions and increased density.

The geography of fascia can be broken into broad functional categories:

1. Superficial (or adipose, areolar, loose, panniculair) connective tissue/fascia – holds organs in place and attaches epithelial tissue to other underlying tissues
2. Deep (or dense) – also known as axial (or investing) fascia
3. Meningeal fascia – surrounds the nervous system
4. Visceral fascia – surrounds and supports organs.

1 Superficial connective tissue (also known as adipose, loose, areolar or panniculair) fascia

- Superficial fascia (see below for more on this topic) in different areas of the body contains distinctive layers of fat that provide insulation and cushioning. In the heel, this cushioning is in the form of fibro-adipose tissue.
- A superficial layer of loose connective tissue and fat surrounds the torso and extremities, but not the external orifices. This allows sliding between itself and denser, deeper fascia that wraps around and invests muscle.
- Blood vessels and nerves pass to and from deeper structures, through the superficial fatty layer.
- This loosely packed subcutaneous – superficial – fascia is the connective tissue that is most accessible (and amenable) to manual therapy interventions (Box 1.5).

Box 1.5 Loose connective tissue and stretching

- Langevin et al. (2005) reported that sustained, light (under 20% of available elasticity) stretch produced a significant time-dependent increase in fibroblast cell body perimeter and cross-sectional area: ‘this study [has] important implications for our understanding of normal movement and posture, as well as therapies using mechani-
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Major features of superficial fascia

- Contains lymphatic channels
- Has a shock-absorbing function – for example, in the heel
- Acts as a heat insulator and thermal regulator
- Stores energy in the form of triglycerides
- Provides channels for veins and, in some areas, well-protected, large nerve fibers, as well as containing numerous mechanoreceptors (Schleip et al. 2012b)
- Sometimes houses vestigial muscle structures – for example, the platysma in the neck
- Contains elastic fibers that allow skin (dermis, epidermis) stretching, and which also create tensile and elastic properties that facilitate a degree of lengthening followed by a return to the original state
- Connects deeper fascia with the body surface, housing fatty lobules (small subdivisions)
- Contains ground substance – the extracellular matrix (ECM) – that fills the spaces between cells, and which has multiple properties that determine the orientation of collagen fibrils, as well as various fluids that allow movement such as the sliding, gliding functions of these tissues

Fourth (2009) notes that animal and human studies indicate that the ideal degree of stretch, required to lengthen loose connective tissue, should not exceed 20% of the available elasticity, with 5% to 6% usually being adequate.

‘When connective tissue stretches (e.g. via physical extension or mechanical stimulation with an acupuncture needle), fibroblast cells that help produce and maintain the connective-tissue matrix become enlarged and flattened. We suggest that focal adhesion complexes on the surface of the fibroblasts detect stretching, and initiate signalling mediated by the protein Rho. The cell then releases ATP into the extracellular space encouraging a change in cell shape, possibly involving breakdown products with analgesic effects. In addition, the Rho pathway instigates remodelling of the cell’s focal adhesions, which mediate where and how the cell attaches to the extracellular matrix, leading to relaxation of connective tissue’ (Langevin 2013).

Langevin has also observed that when an acupuncture needle is rotated – as is normal practice during treatment – collagen fibers wrap around the needle-shaft, leading to traction of the tissues to a distance of several centimetres. This stimulates fibroblasts to increase levels of hyaluronic acid as well as ATP, improving sliding potential and modifying pain (Burnstock 2010).

See subheading Cell-matrix adhesion complexes (CMACs) for an explanation of ‘focal adhesions’.

When subjected to mechanical strain (such as light sustained stretch), fibroblasts within muscle fascia secrete interleukin-6, which has been shown to significantly influence differentiation that is essential for muscle repair, and to powerfully influence inflammatory processes (i.e. it has the potential to trigger either pro- or anti-inflammatory effects depending on other factors) (Hicks et al. 2012).

Fibroblasts synthesize the ECM and collagen in response to mechanical stimuli – such as stretching.
Chapter 1

• Reed et al. (2010) have summarized major features of the ground substance of superficial fascia, as follows: ‘The ECM of the loose connective tissue, constituting the interstitial matrix, has three principal components:
  o collagens constituting the stiff scaffolding for organs and organisms
  o elastic fibres and microfibrils
  o the ground substance composed from proteoglycans and hyaluronan, as well as glycoproteins.’

• Superficial fascia also contains various important cell types including:
  o adipocytes for fat storage
  o fibroblasts (see notes above)
  o various protective blood cells, such as neutrophils and macrophages
  o mast and plasma cells
  o sweat glands.

Key Point
Clinically significant aspects of loose, superficial, connective tissue lie in its relative accessibility to compression, stretching and/or needling – as examples. Load aiming to enhance length has been suggested to require light elongation pressure (well under 20%) – sustained for minutes not seconds.

2 Deep (axial or investing) fascia
The deep connective tissue is subdivided into ‘regular’ and ‘irregular’ forms:

• Regular: in which collagen fibers lie in a parallel fashion – as in tendons (see notes below on Tendons and tenocytes)

• Irregular: in which collagen fibers are not parallel with each other, found for example below the skin.

In both forms, collagen fibers are richly interspersed with fiber-forming fibroblast cells.

As the name implies, deep (axial) fascia extends deep into the body, surrounding and merging the major muscles, tendons, ligaments, and aponeuroses (flat, broad tendon-like sheets that join muscles and the body parts that muscles act on) of the trunk, extending into the limbs, providing protection and lubrication.

Force (load) transfer during muscle contraction is an important feature of deep fascia – discussed below.

‘Dynaments’ instead of ligaments?
Rather than the traditionally held view of muscles acting on joints through ligaments, a different view is expressed by anatomist Jaap van der Wal (2009, 2012): ‘Nearly all the deep and superficial regular dense connective tissue (RDCT) layers are organized in series with muscle fascicles (presented as muscle compartment walls). Collagenous fibres that run from bone to bone – thought to be stressed passively by displacement of the articulating bones – hardly occur. Instead, there occur broad aponeurotic layers of RDCT to which relatively short muscle fascicles insert, which, on the opposite side, are directly attached to skeletal elements. Such configurations of muscle fascicles attached to the periosteum of one articulating bone, and via a layer of RDCT indirectly attached to another articulating bone, could be considered ‘dynamic ligaments’ (van der Wal 2009). Van der Wal has named these muscle-ligament units ‘dynaments’.

Myers (2011) succinctly summarizes this: ‘We simply cannot divorce the muscles and ligaments. They are linked in series and part of one joint stabilizing and moving system. The relevant architecture of the fascia-muscle arrangement is the dynament, not the
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muscle.’ He points out that: ‘The pure (and rare) ligament, like the cruciate, is [indeed] bone–fascia–bone, but most ligaments are in series: bone–fascia–muscle–fascia–bone, as in the hamstrings or rotator cuff.’

Tendons and tenocytes

- Tenocytes are fibroblast-like cells from which the mature tendon is formed. In response to mechanical forces (mechanotransduction) leading to ECM deformations, they synthesize the extracellular matrix and assist in collagen fiber formation, the basic units of tendons (Zhang & Wang 2013).

- Within tendons, tenocytes are arranged in parallel lines. The local extracellular matrix deforms as compressive, tensile, shear and strain forces combine to act on the cells, causing them to change their composition and structure, in response.

- Tenocytes in healthy tendons respond differently to mechanotransduction (for example, regarding gene expression) compared with tenocytes located in degenerative tendon tissue, which do not respond (Buschmann & Bürgisser 2017).

- Zhang & Wang (2013) report that: ‘Tendons can be roughly divided into positional tendons and energy storing tendons. Positional tendons are loaded along their long axis and enable muscles to move bones, while energy storing tendons (in addition to their positional role) store energy when they are stretched. The energy storing tendons can experience strains of up to 10%, as opposed to the positional tendons where maximum strains are between 2% and 3% in living tissues.’

- Buschmann & Bürgisser (2017) reported on the analysis of tendon stem cells (TSCs) extracted from tendon tissues that were either unstretched, 4% stretched (regarded as low-level load) or 8% stretched (regarded as high-level load). NOTE: the 4% and 8% describe the degree of stretch applied to the whole tendon – however, the degree of load reaching the cells would be far smaller.

- The findings showed that moderate mechanical loading (4%) was equivalent to appropriate exercise and that this enhanced tendon anabolism. In contrast, although high mechanical loading (8%) still enhanced gene expression, it resulted in ‘aberrant differentiation of TSCs into non-tenocytes (e.g. adipocytes, chondrocytes, and osteocytes’, that could lead to degenerative tendinopathy (Choi et al. 2014). See also notes towards the end of this chapter, under the heading Basic science tendon studies, and Fig. 1.11).

3 Meningeal fascia (surrounds nervous system)

This is encased within the axial fascia, surrounding and protecting the structures of the nervous system.

4 Visceral and mediastinal fascia

Willard (2012b) has summarized the visceral fascia, thus: ‘Visceral fascia can be traced from the cranial base into the pelvic cavity. It forms the packing surrounding the body cavities where it is compressed against the somatic body wall. It also forms the packing around visceral organs, many of which it reaches by passing along the suspensory ligaments such as the mesenteries. This fascia also functions as a conduit for the neurovascular and lymphatic bundles as they radiate outward from the thoracic, abdominal, and pelvic mediastinum to reach the specific organs.’

Visceral fascia surrounds and supports organs and provides the ‘packing tissue’ for the midline structures of the body (Drake et al. 2010) – it is continuous from the nasopharyngeal and cervical region, all the way through the thorax (mediastinum), passing via the diaphragm, through the abdomen, to the pelvic...
floor. In the midline, in addition to the abdominal plexus and the autonomic nerves, it houses major vessels, such as the aorta, and the caval venous anastomosis, as well as the thoracic duct. The visceral fascia effectively envelopes all the major organs, invests the pleural and peritoneal linings, and forms the neurovascular sheaths. The mediastinal fascia, largely comprising loose connective tissue, therefore forms the central compartmental cavity of the thorax, housing major organs as well as neural and vascular structures.

Manual therapy methods exist that have potential therapeutic influences for the mediastinum (Barral & Mercier 2004).

**Key Point**

The forms and locations of the various fascial features are clinically relevant because of the potentials offered to the manual therapist by the continuity that exists between most structures lying between the base of the skull and the pelvic floor.

**Extremity and trunk differences in deep fascia**

The deeper fasciae of the extremities differ significantly from that of the trunk. Deep fascia associated with muscles in the limbs slides freely, whereas, in the trunk, muscles are more adherent to the deeper fascia.

The deep fasciae of the limbs not only envelop muscles. Evidence shows they comprise two or three dense layers or sheets with undulating arrangements of parallel collagen bundles that may include some elastic fibers. Deep, dense, fascial layers are usually separated by thin layers of loose connective tissue that cushion and allow the deeper layers to slide on each other, so providing frictionless mechanical adaptability.

Where hard and soft tissues meet, where fascia connects to bone, there are local areas of concentrated tensional stress-fibers, in which collagen links to, and anchors, layers of deep connective tissue to each other, or consolidates these into a retinaculum (stabilizing bands around tendons) or fibrocartilage (particularly string-cartilage, such as the meniscus of the knee). The thin, sheet-like, layers of deep, dense fascia are oriented, in relation to each other, at approximately 78°. This orientation apparently allows reduced friction as fascial sheets slide over underlying layers, improving fascia’s ability to take up the strain.

If a myofascial load is exerted onto muscle, force is automatically transmitted onto the intramuscular connective tissue via the layer of fascia that wraps the muscle (epimysium). For this force to be transmitted adequately, the connections need to be firm, not loose. Huijing and Langevin (2009) argue that since some fascial structures that are not dense are capable of transmitting some muscular force, ‘the term ‘loose connective tissue’ for such structures is inadequate, and the term ‘areolar’ is preferred’ (Fig. 1.6).

![Figure 1.6](image-url)

*Schematic image of the deep fasciae of the limbs, showing the composition and fiber direction in three different layers. Adapted from Figure 1.5.2 from Stecco C, Stecco A 2012 Deep fascia of the lower limbs. In: Schleip R et al. (eds): Fascia: the tensional network of the human body. Churchill Livingstone Elsevier, Edinburgh, Ch. 1, p. 34.*
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**Key Point**
The clinical significance of the different orientations of fascial planes should be taken into account – for example – to achieve optimal applications of directions of the load during manual treatment methods that target perceived restriction barriers.

**Muscle fascia** (Fig. 1.7)

- Every muscle is wrapped in a layer of connective tissue – the epimysium – that links it to bone via the tendons.
- The muscle itself is separated into smaller units of muscular bundles, or fascicles, by a fascial network – the perimysium.
- Fascicles are further separated into muscle fibers by the endomysium that forms a continuous connecting matrix that shear-links adjacent fibers, to coordinate force transmission, while keeping fibers in a stable relationship (Purslow 2008).
- Intramuscular septa are tough fascial sheets that separate extremity compartments and muscles – for example, anterior and posterior crural septa; lateral and medial femoral septa; and the lateral and medial humeral septa.

These structures facilitate load sharing and load transfer so that a continuous functional mechanical, three-dimensional network can operate.

**Box 1.6 The main 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’ (FIPAT 2011).

  - **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; iliotibial (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, planter aponeurosis.

- **Fascicular fascia**: This comprises a mixture of both loose and dense connective tissues that provide the architectural shape of muscles:

  - It surrounds whole muscles (epimysium), as well as separating muscle fascicles (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.

  - An unexplained feature of the layers of perimysial collagen fibers – for example, those surrounding muscle fascicles – is that their
long axis always lies at 55° to the longitudinal axis of the fascicle, when the muscle is at its relaxed (resting) length – in humans and animals (Lewis & Purslow 1989).

- **Compression fascia**: This dense connective tissue structure envelopes and compartmentalizes the limbs involving sheet-like layers.
  - For example, the crural fascia of the lower limb exists as stocking-like coverings that variously offers compression and tension, while strongly affecting muscular efficiency and venous return. These 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:

- Illiotibial-band (linking)
- Perimysium of the quadriceps femoris muscle (fascicular)
- Fascia lata (compression)
- Subcutaneous tissue (separating).

**NOTE**: See text below, under the heading Guimberteau’s alternative sliding model, that challenges the concept of separate ‘layers’ of fascia, as described above, and elsewhere in this chapter (and book).

**Key Point**

The clinical relevance of the notes in Box 1.6 relates to concepts of continuity – of chains, strings and slings, involving fascial connections. Some of the specific clinical implications of fascial continuity, in manual therapies, are discussed in this chapter under subheadings such as Force transmission, load transfer and fascia and in Box 1.4: Mechanotransduction.

**Guimberteau’s alternative sliding model – ‘there are no layers’**

A different model is suggested (Guimberteau & Bakhach 2006, Guimberteau 2012) in which it is observed that: ‘The traditional notion of different fascias or the sliding, gliding, collagenous system, historically referred to as paratenon, connective or areolar tissues, focuses on the separateness of these structures. Electron scanning microscopy suggests that this system does not consist of different, superimposed layers. In reality, there is a single, tissular architecture with different specializations. To emphasize its functional implications, we call this tissue the multimicrovacuolar collagenous (dynamic) absorbing system (MVCAS).’

Guimberteau and colleagues’ observations suggest that:

- This intercellular environment contains a highly hydrated proteoglycan gel, with high lipid content. Its sides comprise intertwined vacuoles composed of collagen and elastins.
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- The MVCAS is seen as the building block of: ‘an inter-organic network, functioning at different levels and performing three major mechanical roles: (i) responding to any kind of mechanical stimulus in a highly adaptable and energy-saving manner; (ii) preserving the structures, providing information during action and springing back to its original shape; (iii) ensuring the interdependence and autonomy of the various functional units.’

They also note that:

- ‘…the microvacuolar system’s function is to maintain the peripheral structures close to, but not mechanically affected by the body action in progress. Conversely, it also offers resistance, first minimally then increasing as the load increases.’

- The ability of the MVCAS to respond resiliently changes when negative influences such as edema, trauma, inflammation, obesity and/or ageing occur, creating changes in microvacuolar shape.

**Key Point**

The concept of ‘layers’, as compared to a ‘continuum of fascial microvacuoles’, in which some areas of this single continuous tissue
(fascia) are seen to represent areas of specialization, with greater density when compared with other areas, can easily be reconciled by accepting that both may be seen as accurate descriptors.

Whether a ‘specialized region’ is called a ‘layer’, or something else, should not matter. For practical purposes, ‘layers’ will be the descriptor used in this book.

**Force transmission, load transfer and fascia**

Schleip (2003a,b) has described fascia as: ‘...the dense irregular connective tissue that surrounds and connects every muscle, even the tiniest myofibril, and every single organ of the body forming continuity throughout the body’.

How might this summary of fascia’s ubiquitous presence change the way we understand movement and locomotion?

One key element involves relearning the way that force is transmitted. We have been taught to think of specific muscles contracting with force then being transferred in a linear manner by means of, for example, aponeuroses and tendons, thereby producing joint movement.

Illustrations of muscular activity in standard anatomical atlases usually involve removal of fascial elements to reveal what is commonly and inaccurately presented as the primary mechanical feature of movement – specific muscles – so ignoring vital fascial connective continuities in which force is transmitted in multiple directions simultaneously: sometimes laterally, sometimes obliquely, and sometimes linearly.

For example, structures normally described as the muscles of the hip, pelvis, and leg interact with arm and spinal muscles via the thoracolumbar fascia, which allows effective load transfer between the spine, pelvis, legs, and arms in an integrated system (Fig. 1.8).

**The lumbar interfascial triangle (LIFT)**

(Willard et al. 2012)

As noted above, the TLF integrates forces deriving from passive connective tissues as well as numerous active muscular structures, including aponeurotic and fascial layers that separate paraspinal muscles from the muscles of the posterior abdominal wall.

- The superficial posterior layer of the TLF is mainly an aponeurosis of latissimus dorsi and serratus posterior inferior, while deep to this is the retinacular sheath that encapsulates the paraspinal muscles that support the lumbosacral spine.

- Where this sheath meets the aponeurosis of transversus abdominis it forms a raphe (a seam-like ridge) – a dense septum. This is the junction of the hypaxial (anterior to the spine) and epiaxial (posterior to the spine) fascial compartments – where it forms the lumbar interfascial triangle (LIFT).

- This remarkable structure (a ‘roundhouse’ in Myers terminology – see Ch. 3) helps distribute the load from abdominal and extremity muscles into, and from, the TLF.

- All layers of TLF fuse to merge with the posterior superior iliac spine and the sacrotuberous ligament, assisting to support the lower lumbar spine and sacroiliac joint.

- Load reaching the LIFT from the abdominal muscles, latissimus dorsi, the lower extremity, and pelvic muscles are therefore appropriately distributed, in order to assist in stabilizing the spine, trunk and pelvis.
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Biomechanical and functional connections

Scalenes and the thorax

Because of the fascial sheets and membranes that envelop and connect them, the scalene muscles are continuous with thoracic structures – ranging from the pectoral muscles to the pericardium. It is therefore unwise to conceive of scalene dysfunction without taking account of what they are connected to – via fascia – anatomically and functionally.

Key Point

The clinical relevance of knowledge of the ECM and of GAGs (such as hyaluronic acid) highlights the importance of sliding/gliding functions – whether between fascial layers or involving a different mechanism. In essence, the ability for frictionless muscular activity is the ideal. There is some evidence that different forms of the therapeutic load may help to create enhanced sliding potential. See Chapter 5 for more on the specific forms of load.

Figure 1.8

Deep layer of the thoracolumbar fascia and attachments to gluteus medius and attachments between the deep layer and the erector spinae muscle.
Stecco and Stecco (2012) note that: ‘intermuscular and epimysial fasciae serve as areas of insertion for muscle fibres that [...] can mechanically reach a skeletal element without necessarily being attached directly to the bone’. This is apparently also true for connections to the superficial fasciae (such as fascia cruris and fascia antebrachii), which provide broad insertion areas for muscle fibers (Fig. 1.9).

Shoulder, trunk, cervical fascial continuity

Stecco and Stecco (2012) have reported on the results of their numerous dissections. For example: ‘The deep fasciae of the shoulder present characteristics that are similar to both the fasciae of the trunk and of the extremities. In particular, the fasciae of the pectoralis major, deltoid, trapezius, and latissimus dorsi muscles form a unique layer, enveloping all of these muscles and passing over the serratus anterior, where it forms a strong fascial lamina.’

They continue: ‘All of these fasciae adhere firmly to their respective muscles due to a series of intramuscular septa that extend from the internal surface of these fasciae, dividing the muscle itself into many bundles.’ It is inconceivable that load on any of these muscles would not directly affect all the others mentioned (see Fig. 1.8). This pattern of ‘separate’ muscles being bound together by fascial structures is repeated in the limbs, on the back, in the cervical region, etc.

How load from the hamstrings is distributed

Franklyn-Miller et al. (2009) have demonstrated – using micro-strain gauges – that the degree of force used in a hamstring stretch results in a variety of unexpected load transfers:

- 240% of the imposed strain is transferred to the iliotibial (IT) tract
- 145% of the hamstring load is transferred to the ipsilateral lumbar fascia, via the sacrotubercous ligament

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- 103% to the lateral crural compartment
- 45% to the contralateral lumbar fascia
- 26% to the plantar fascia.

Strain transmission, via fascial continuities during stretching, can therefore be seen to affect many tissues, other than the muscle to which load is being applied.

The researchers report that ‘strain is distributed through a consistent pattern… [that] correlates closely with collagen fibre orientation.’ This suggests the possibility that apparent muscular restrictions might, in fact, be fascial in origin, and that the source of such dysfunction may lie at a distance from where it is perceived.

The latissimus dorsi–hip–gluteus maximus connection

Using volunteers, Carvalhais et al. (2013) were able to show that active latissimus dorsi tensioning – for example, when the shoulder was actively adducted and the scapular depressed, using force equivalent to 25% of a maximum voluntary contraction – produced lateral rotation of the contralateral hip, together with increased passive (also contralateral) gluteus maximus (GM) stiffness.

Gluteus maximus and knee pain

Stecco et al. (2013) have also identified a force transmission link from the thoracolumbar fascia, via GM, to the IT tract, and onwards to the knee, suggesting that ‘hypertonicity of gluteus maximus could explain increased tension in the lumbar region, causing low back pain and the lateral region of the knee.

In all (12) subjects gluteus maximus presented a major insertion into the fascia lata, so large that the iliotibial tract could be considered a tendon of insertion of the gluteus maximus … [explaining] … transmission of the forces from the thoracolumbar fascia to the knee [and] possibly explaining why hypertonicity of the GM could cause an IT band friction syndrome (ITBFS) or, more generally, knee pain.

Resolution of ITBFS can only be achieved when the biomechanics of the hip are properly addressed.

Fascial connections validated by electromyography

The muscular associations connected to a myofascial kinematic chain (described as the superficial back line by Myers 2009 – see details later in this chapter under subheading Muscle chains, tracks and trains and more fully in Ch. 3, particularly Fig. 3.8b) have been tested using electromyography (Weisman et al. 2014).

The findings of this study, which aimed to ‘map the association of muscle activations along the superficial back line (SBL) using separate conditions of an active range of motion with and without resistance and passive range of motion’, were of clinical relevance:

- During maximum isometric contraction of the right gastrocnemius, there were strong activation signals recorded at the electrodes placed on: right hamstrings, posterior superior iliac spine (PSIS), left and right 12th thoracic segment (T12), and right upper trapezius.

- During maximal isometric cervical extension (prone) there were strong activation signals recorded at the electrodes placed on the upper trapezius, T6, T12, PSIS, with moderate but significant activation in the hamstrings.
Clinical implications and considerations

We can see, for example, that knee pain might emerge from GM dysfunction and that GM (and the hip) may be strongly influenced by contralateral latissimus dorsi activity, via the thoracolumbar fascia.

Data from Stecco studies (described above) have shown the direct connection between latissimus dorsi, upper trapezius, the scalenes and the pectoral muscles. Could knee pain reflect fascial, or other, restrictions involving any of these muscles, or could dysfunction in any of them relate to influences from the knee?

We can also see links between gastrocnemius function and, for example, the ipsilateral upper trapezius muscle; and the cervical muscles and the hamstrings.

These examples highlight the need to revise previously held ideas as to how the body works biomechanically. This revision of previously held concepts is a necessary process in order to appreciate advances in understanding fascial function – as is the need to learn the topography, the geography, the connections and the architecture of the soft tissues, including fascia – not excluding it, as happens in many anatomy texts. We need to know where, as well as how, fascia influences function.

Stated simply, fascia integrates and organizes both posture and movement.

Key point

The clinical relevance that is becoming apparent from these studies relates to the need for therapists to become aware – as evidence emerges – of the pathways of force transmission in different areas of the body.

The example described above of the knee–hamstring–GM–hip–TLF–latissimus, etc. connections suggests that influences at a distance need to be considered when seeking both the etiological and the maintaining features of pain and restriction.

Muscle chains, tracks and trains

This section does not try to provide a comprehensive list of the many attempts that have been made to catalogue the connections that make up myofascial load sharing.

Richter (2012) described a number of different models in which myofascial links, slings, etc. form the basis for understanding biomechanical function. He explains: ‘Even if these models are sometimes very different, they all have one thing in common: they show the locomotor system and the myofascial tissues as being one unit that always functions as a whole.’

Probably the most widely employed model is known as ‘Anatomy Trains’ (Myers 2009), that describes theoretical lines, tracks and junctions (meridians), etc. – involving the multiple connections that make up the ‘global, geodesic tension complexes’ of the locomotor system ‘that simultaneously stabilize and allow adjustments within the skeletal frame’. These are summarized below, and more fully in Chapter 3.

A functional separation of structures within this network involves the 600+ muscles of the body, as well as the multiple, more deeply situated force-transmission systems, including fascial sheets, ligaments, tendons, capsules, etc.

Myers acknowledges that the Anatomy Trains model is one that will continue to develop, but that it provides a ‘design argument’ that matches tensile theory and load-transfer research, and therefore has clinical relevance – as he describes in the context of assessment in Chapter 3.

When reflecting on the connections, as catalogued by Myers, bear in mind van der Wal’s
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observation (2009) that no muscle attaches to bone anywhere in the body, and that osseous connections are always made via the intervening connective tissue structures (se notes on ‘dynaments’ earlier in this chapter).

Myers’ meridians/lines

1. Superficial Front Line: toe extensors, anterior crural compartment, quadriceps, rectus abdominis and abdominal fasciae, sternalis and sternal fascia, sternocleidomastoid.


3. Lateral Line: fibularis muscles, lateral crural compartment, IT tract, hip abductors, lateral abdominal obliques, internal and external intercostals, sternocleidomastoid, and splenii.


5. Superficial Back Arm Line: trapezius, deltoid, lateral intermuscular septum, extensor group.


7. Superficial Front Arm Line: pectoralis major, latissimus dorsi, medial intermuscular septum, flexor group, carpal tunnel.


10. Back Functional Line: latissimus, lumbosacral fascia, GM, vastus lateralis (see Fig. 1.8).


12. Deep Front Line: tibialis posterior, long toe flexors, deep posterior compartment, popliteus, posterior knee capsule, adductor group, pelvic floor, anterior longitudinal ligament, psoas, iliacus, quadratus lumborum, diaphragm, mediastinum, longus muscles, hyoid complex, the floor of mouth, jaw muscles.

Other models

As mentioned, many different models exist that interpret possible fascial chains and connections.

• As examples, see the discussions in Chapter 9 on Fascial Manipulation®, in which local areas of coordination – and perception – are identified in relation to myofascial sequences.

• Also: see the similar but different model – described fully in Chapter 17 – Global Postural Re-education, from which this quote is taken: ‘Biomechanical synergies, hegemonic and defence mechanisms and chain-organized neuro-myofascial structures, are some of the reasons for body compensations that justify the need for a global approach’ (Souchard 2015).

Caution: some ‘lines’ remain unvalidated

A systematic review of evidence derived from cadaveric dissection studies, concluded that: ‘Although there is strong empirical support for the existence of the superficial back line (all 3 transitions verified, based on 14 studies), back functional line (all 3 transitions verified, based on 8 studies) and front functional line (based on 6 studies), evidence is ambivalent with regard to the spiral line and lateral line, and poor for the superficial front line’ (Wilke et al. 2016).
The review concludes that: ‘The system of myo-fascial meridians represents a promising approach to transfer tensegrity principles into practice. Therapists may use the myofascial chains as a conclusive orientation, but they should be aware that the functional implications remain to be studied.’

The lines described in Anatomy Trains are discussed more thoroughly in Chapter 3.

**Key Point**

The clinical relevance of an awareness of muscular chains and links (whether the Anatomy Trains model or one of the many others that have been described) cannot be overstated. Understanding load sharing and the reciprocal function of myofascial continuities widens clinical choices in relation to manual and exercise, treatment and rehabilitation approaches.

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**Translation of basic science into clinical relevance**

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

The increasing interest in fascia by practitioners and therapists, resulting from recent research congresses, symposia, and the sheer volume of research-based publications on the subject, has led to the development and promotion of a variety of ‘new’ methods of treatment.

Many treatment approaches 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 II of this book. The authors of those chapters have explained the methods as well as the foundations on which the modality has been constructed – that is, the ways in which scientific research has been interpreted into a clinical approach, and also, where possible, the results of clinical studies.

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 to be able to then make informed decisions.

Therefore, one of the main aims of this book is to provide the tools that will lead to informed judgments being exercised, particularly where evidence from basic science research is extrapolated to support clinical methods.

**Can clinical practice be reliably informed by basic science research evidence?**

In some examples, translational evidence may be used to suggest – not prove – the potential value of a particular treatment method.

An example of this is the research evidence that when cells, such as fibroblasts, are treated in a certain way in controlled laboratory settings, their behavior changes.

For example, as described below, modeled myofascial release (see Ch. 13) – or counterstrain (see Ch. 15) – have been shown in laboratory studies to modify inflammation (Standley & Meltzer 2008).

Research results from such studies may be translated to suggest – not prove – that these methods might have similar effects when used in treatment settings.

In order to successfully achieve prevention, assessment and successful treatment of fascial dysfunction, accurate interpretation of basic science findings
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is necessary. The more clearly we understand fascial anatomy and physiology – from the cell to the larger tissue features – and the more we are aware of the implications of research findings, the better able we should be to recognize the roles fascia may play in treatment and management of a variety of painful and dysfunctional conditions.

However – caution is necessary for interpretation of basic science research information into therapeutic action.

**Modeled myofascial release (and counterstrain), applied to fibroblasts**

Studies of osteopathic methods, in which myofascial release (Meltzer et al. 2010), see Chapter 13, have been modeled and applied to fascial cells (fibroblasts), in laboratory settings. The results appear to offer possible explanations for known clinical outcomes when these treatment methods are used in practice.

There is obvious potential relevance of information regarding the dosage elements of treatment, such as the optimal degrees, duration and directions of applied load (stretch, torsion, compression, etc.).

**Example: Modeled repetitive strain and myofascial release**

In a 2010 study, Meltzer et al. harvested human fibroblast cells that were spread onto pre-stressed (tension had been increased by 10%), flexible collagen-coated membranes, which were a part of an instrument that could apply precise degrees of load (magnitude, duration, direction and frequency) to the membranes to which the cells were adhered, via a vacuum mechanism.

Different protocols were followed in which:

1. Cells were not subjected to any strain for the duration of the experiments.
2. Cells were subjected to repetitive motion strain (RMS) for 8 hours continually (10% added load/stretch every 1.6 seconds), and then tested immediately upon cessation of RMS.
3. Myofascial release (MFR): Cells were subjected to modeled MFR for 60 seconds, involving the addition of a sustained 10% loading of the flexible membrane) and then tested immediately upon cessation of MFR.
4. Cells were subjected to the 8 hours of RMS – followed three hours later by 60 seconds of MFR, and then tested immediately upon cessation of MFR.

Findings:

- Repetitive strain altered the shape (morphology) of the fibroblasts; increased levels of apoptosis (programmed cell death/suicide'), and increased levels of inflammatory cytokines.
- However, when the induced damage of 8 hours of repetitive strain was followed (3 hours later) by 60 seconds of modeled MFR, these negative effects reversed significantly (some completely), including reduced apoptosis and inflammation.
- In 2007, Meltzer & Standley et al. had performed a very similar study, and achieved almost identical results, when they modeled counterstrain (by unloading the flexible membrane by 6%) for 60 seconds, following repetitive strain of fibroblasts (see Ch. 15).
- Duration, direction and degree of load are all features that need to be identified if manual and movement interventions are to be optimally applied to assist the self-regulating functions, described by these and other studies.

**Basic science tendon studies** *(Fig. 1.10)*

In their review of the effects of different forces (degree, direction, etc.) required for optimal healing of damaged tendons, Wang et al. (2012) note that: ‘Tendons are mechanoresponsive… they have the capacity to...
 adapts themselves to altered mechanical loading' and that: 'Repetitive mechanical stretching has two opposite effects – anti-inflammatory at small magnitudes and pro-inflammatory at large magnitudes.'

In regard to the degree of load, this translates in clinical settings as follows:

- **Low levels** (2%) of mechanical load to damaged tendons were found to reduce tensile strength as well as collagen production (among other factors).

- **High levels** of mechanical load (8%), applied to damaged tendons interfere with collagen organization, and increase levels of inflammatory mediators, while also increasing tendon stem cell (TSC) differentiation into non-tenocytes.

- In contrast, **moderate** degrees of mechanical load (4%) to damaged tendons increased tensile strength and collagen synthesis, while increasing TSC differentiation into tenocytes and decreasing inflammatory mediators.

- In a different study, involving fibroblast behavior during the repair of a damaged bioengineered tendon, Zein-Hamoud and Standley (2015) identified that the degree (~6%), direction (heterobaxial), and duration (4–5 min) of load had beneficial effects on tendon healing.

### Caution required

But – the question arises as to what studies of cells and tissues in a laboratory may actually mean, when it comes to management of fascia-related pain and dysfunction.

Although the clinical translation of such basic science research into cell behavior is potentially helpful in identifying possible underlying mechanisms involved in manual treatments – it is important that we translate such research evidence cautiously.

- For example, studies of the effects of different dosages of load, applied (for example) to fibroblasts, commonly ignores other cells, such as stem cells, that are likely to be mechanically influenced and affected during manual treatment of the human body (Cao et al. 2015).

- Inflammation changes following trauma may not become evident for days following – for example – repetitive strain (Maclntyre et al. 2001), whereas anti-inflammatory effects in some of the studies only reflect immediate influences (Meltzer et al. 2010).

- In addition, we cannot know how applied load/force is absorbed or transmitted to deeper
tissues – in real life, compared with modeled methods, where highly controlled laboratory conditions apply, and where precise measurable forces are applied to cells (Zein Hamoud & Standley, 2015).

- Another question arises: How is a practitioner to know the difference between loading tissues with the beneficial approximately 6% as compared with 3% or 12% in order to match these findings?
- Box 1.7 (below) considers aspects of this vital issue – how load may be transferred into the tissues beneficially.

**Box 1.7 Living tissues are more like a fiber-reinforced composite than spaghetti**

- Burkholder (2008) has offered descriptions of some of the intricacies of load transfer into tissues and has discussed the mind-numbing complexity of ways in which mechanical load to living muscle is transferred and transmitted.

- Burkholder suggests that our task is to: ‘consider the shape changes and forces associated with mechanical signals and juxtapose them with observed changes in biochemical signalling, to determine what might contribute to the cellular perception of mechanical signals.’

- ‘The mechanical properties of intramuscular connective tissue are particularly difficult to determine, making the distribution of passive tension between passive myofibers and matrix nearly impossible to estimate.’

- ‘The principal feature that distinguishes whole muscle from cultured myotubes is the integration with a three-dimensional (3-D) extracellular matrix and extensive connections among adjacent fibers. This mechanical integration has the effect of homogenizing deformations, meaning that gradients in shape changes will be smaller than they might be in an isolated fiber. A whole muscle is much more like a fiber-reinforced composite than it is like a bundle of spaghetti.’

- Gravovetsky (2016) has considered the issues relating to the transfer of forces, from the skin surface to deeper tissues and suggests that: ‘[While] it is not known how much energy can be transferred from the skin surface to the deeper layers, it can be speculated that at least some of the therapist’s energy, applied to the skin, will end up being transferred. It follows that controlling the delivery of mechanical energy (heat) to coiled collagen should (presumably) uncoil it, close the cleavage sites, and prevent matrix metalloproteinases [enzymes] from binding and degrading the collagen. The amount of energy needed to reverse the creation of cleavage sites is not really known but is estimated to be small, and within the range of what might be provided by external action.’

- ‘The video illustrations of Jean Claude Guimberteau […] demonstrate how a force applied to the surface of the skin ends up being dissipated deep into the tissues via a densely interconnected network of collagenous tissues’ (Guimberteau 2012).

- As to the evidence from the studies by Flynn et al. (2010), Humphrey et al. (2014) and Dittmore et al. (2016) regarding collagen homeostasis – as summarized earlier in this chapter – the clinical relevance would seem to be that balanced tone/tensions are conducive to collagen well-being, while unbalanced patterns are not.
In support of those observations, Gracovetsky offers the following thoughts: ‘Franchi (2010) [used] electron microscopy to study changes in collagen fiber organization when the tissues are put under stress, [and] demonstrated that the well-ordered fibrils become disorganized under stress, thereby interfering with an orderly sliding motion... [The resulting] ‘hardening’ may explain why [manual therapies such as myofascial release techniques] can reduce the amount of disorganization within the collagen fibrils and permit a freer movement.’

Unsurprisingly, therefore, the solution to the maintenance of collagen and therefore fascial health would seem to align with the maintenance of an optimally balanced musculoskeletal status, potentially assisted by manual and movement therapeutic methods.

Fascia: resilience as a descriptor... and the seeds of dysfunction

- The multiple functions of the connective tissue matrix, with its combined qualities of strength and elasticity (i.e. of biotensegrity), can be described by the single word resilience.

- The quality of resilience refers to the ability (for example) of cells or tissues to adapt to distorting forces, and, where appropriate, to be able to return to their original form and position – something that is true of the fascial web.

- Resilience also describes the ability of an individual to rapidly recover from illness or injury.

- When resilience fails – or to put it differently, when adaptation demands overwhelm resilience, as a result of any of a variety of factors, ranging from age to pathology, overuse or trauma – dysfunction is the result. Failed, or failing, adaptation might involve alterations in the optimal transfer of information (for example, force transmission), reduced coordination, and/or loss of gliding functions – leading to movement disorders, pain and unbalanced degrees of tension in the soft tissues of the body.

How fascial problems start

The multiple ways in which fascial dysfunction can manifest are explored in Chapter 2. As with many musculoskeletal symptoms, the causes of fascial dysfunction include overuse (e.g. repetitive strain), misuse (poor functional use patterns), disuse, and/or abuse (trauma).

Fascial dysfunction may result from slowly evolving trauma (disuse, overuse and misuse) or sudden injury (abuse) leading to inflammation and inadequate remodeling (such as excessive scarring or development of fibrosis):

- ‘Densification’ may occur involving distortion of myofascial relationships, reducing sliding facilities and altering muscle balance and proprioception (Stecco & Stecco 2009). As a result of such changes, chronic tissue loading forms ‘global soft-tissue holding patterns’ (Myers 2009).

- ‘When fascia is excessively mechanically stressed, inflamed or immobile, collagen and matrix deposition becomes disorganized, resulting in fibrosis and adhesions’ (Langevin 2008).

- Densification and loss of fascial sliding function have been clearly demonstrated by Langevin et al. (2011b). Individuals with chronic low back pain showed a 25% greater thickness of the TLF than individuals without low back pain. The gliding potential between fascial layers was also shown to be significantly reduced in these individuals.
Stecco et al. (2013) suggest that a combination of irritation, inflammation, acidification and densification of loose connective tissue may lead to myofascial pain as a result of ‘free nerve endings becoming hyperactivated’ resulting in local inflammation, pain and sensitization. These changes can be reversed by manual therapy interventions that reduce stiffness, density and viscosity – and improve pH; all potentially possible via manual therapies.

Fascia is also greatly affected by the aging process (and inactivity that is possibly related to illness or concurrent pain):

- As we age skin changes, characterized by the evolution of wrinkles, which reflects the reduction in superficial fibroblast and collagen cells.
- Collagen fibers gradually become less organized, more tangled, and tissues lose their defined shape (i.e. they sag) and elastic recoil potentials.
- A part of that inevitable – but variable – process involves loss of elastin, so that from around the third decade of life this process is measurable (Kirk & Chieffi 1962).
- At the same time, there is an atrophy of fat cells, so that ‘quantitative and qualitative characteristics of the fibro-adipose connective system are changed and its viscoelastic properties become reduced’. Additionally, the skin and underlying superficial fascia stretch and relax, leading to ptosis of the soft tissues, as well as altered shape involving fat deposition and cellulite (Macchi et al. 2010).

Therapeutic options relative to these and other fascial changes are explored in Chapter 5 and throughout Section II, where there are separate chapters on 16 different modalities that all target fascial dysfunction.

**Next:** In the next chapter, the features and factors relating to the evolution of fascial dysfunction are explored.

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**References**


Benjamin M 2009 The fascia of the limbs and back – a review. J Anat 214:1–18


Bouffard N et al 2009 Tissue stretch decreases procollagen-1 and TGF-β1 in mouse subcutaneous fascia. Abstract. 2nd Fascia Research Congress. Free University of Amsterdam, Amsterdam


Chaitow L, Bradley D, Gilbert C 2013 Recognising and treating breathing disorders. Churchill Livingstone, Edinburgh


The functions of fascia: translating research into clinical relevance


Klingler W, Schleip R, Zorn A 2004 European Fascia Research Project Report. 5th World Congress Low Back and Pelvic Pain, Melbourne


Langevin HM 2010 Tissue stretch induces nuclear remodeling in connective tissue fibroblasts. Histochem Cell Biol 133:405–415


Langevin HM et al 2011b. Reduced thoracolumbar fascia shear strain in human chronic low back pain. BMC Musculoskelet Disord 12:203


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Reed RK, Lidén A, Rubin K 2010 Edema and fluid dynamics in connective tissue remodelling. J Mol Cell Cardiol 48(3):518–523


Ross M, Pawlina W 2011 Histology, 6th edn. Lippincott Williams & Wilkins, Baltimore, p 218


Souchard P 2015 Déformations Morphologiques de la Colonne Vertébrale. Elsevier Masson, France


Stecco L, Stecco C 2009 Fascial manipulation: Practical Part. Piccin, Padova


Tortora G et al 2007 Microbiology: an introduction. Pearson Benjamin Cummings, San Francisco


van der Wal JC 2009 The architecture of connective tissue as parameter for proprioception – an often overlooked functional parameter as to proprioception in the locomotor apparatus. Int J Ther Massage Bodywork 2(4):9–23


Author Queries

AU1: Pls add wording to direct reader to figure in Meltzer article.