Fascia can be divided into two general categories: subcutaneous fascia and deep fascia, also known as muscular fascia. Subcutaneous fascia, as the name implies, is located immediately deep to the skin. It is largely composed of adipose (fat) cells, giving it its gel-like consistency. Collagen fibers and fibroblast cells are embedded within this mix of adipose cells. Deep fascia is located deeper in the body and is primarily comprised of densely packed collagen fibers, accounting for its description as fibrous fascia. Fibroblasts are located between these collagen fibers (Figure 1).

Endomysium, perimysium and epimysium, which create the tendons and aponeuroses of muscles, are examples of deep fibrous fascia, as are ligaments and intermuscular septa. The loosely packed structure of subcutaneous fascia allows for a healthy blood supply, giving it strong reparative properties when injured. Alternatively, the dense arrangement of fibrous fascia does not allow adequate room for blood vessels; therefore, fibrous fascia has a poor blood supply and does not heal well when injured. In addition to collagen fibers and fibroblasts, both...
Recent research suggests that fascia does not function only in a passive tensile manner, but instead, like muscle tissue, creates contractile active tension forces.

types of fascia contain elastic fibers that give them the ability to shorten back to their original length after being stretched.

The traditional understanding of the role of fascia within the musculoskeletal system is that it is an inert noncontractile tissue. Because of its high concentration of collagen, it has been understood to be extremely effective at withstanding tensile (pulling) forces without being torn. However, any pulling force it possesses has been considered to be passive. That is, when the fascial tissue is pulled upon, its collagen fibers passively resist being lengthened and stretched, having the ability to transfer these pulling forces to other tissues. For example, when a muscle belly contracts and pulls upon its tendons, these fibrous tissue tendons transfer that pulling force to the bony attachments of the muscle. Indeed, collagen fibers have long been heralded for their passive tensile strength.

Until recently, muscle tissue has been considered unique in its ability to contract. In other words, only muscle tissue can create active tension forces. However, recent research has provided a more sophisticated understanding of the contractile role of fascia within the musculoskeletal system. What is now becoming clearly understood is that fascia does not function only in a passive tensile manner, resisting and transmitting tensile forces. Instead, like muscle tissue, it can also create contractile active tension forces.

We now know that fascia also contains another type of cell called myofibroblasts (Figure 2). The root “myo” comes from the Latin word for muscle, indicating that myofibroblasts have contractile activity. The distinction between fibroblasts and myofibroblasts seems to be the presence of a protein called alpha smooth muscle actin (α-SMA) in myofibroblasts. α-SMA seems to be the principle molecule that confers upon fascia its contractile ability.

The degree of myofibroblasts present within a fascial tissue varies, but seems to be related to the degree of body mechanics
Although the new evidence of the active contractile strength of fascia is significant, the importance of fascia’s passive strength should not be dismissed. After all, the total tension force of muscle/myofascial tissue is the combination of its active and passive forces. Fascia is extremely resistant to pulling forces, therefore it does not lengthen out. This is important to the functioning of a muscle in two ways. First, when a muscle contracts, it exerts its pulling force upon its fascial tendon or aponeurosis which then transfers that pulling force to the muscle’s bony attachment. Therefore the muscle’s active contraction pull is efficiently and strongly transferred to its attachments to create movement. Second, fascia’s resistance to being lengthened also adds a passive tension force to the muscle. If an external force stretches a muscle to the limit of its fascia’s ability to lengthen, the resistance to stretch by the fascia creates a passive tension force that opposes the force of stretch upon the muscle. This passive resistance force adds to the active contractile tension force of the muscular and fascial tissues. Thus, fascia contributes to a muscle’s total tension force both actively and passively. (It should be added that fascia’s resistance to lengthening creates a stable skeletal framework for the muscle that prevents the muscle from being overstretched and torn, therefore fascia also serves a protective function for muscle.)

One other factor needs to be considered when looking at the total contractile tension force of muscle/myofascial tissue, that is its elasticity. Elasticity is a passive pulling force that causes a structure to bounce back toward its center after being stretched (think of an elastic band), therefore this pulling force can be added to the total pulling force of a muscle. Fascia is inelastic, so the elasticity of a muscle is provided by the muscle tissue itself. Specifically, within muscle fibers, there is a large protein called titin that connects the myosin filament to the actin filaments and Z lines of the sarcomere. Titin has a region that is coiled and springlike, which provides the elasticity to muscle/myofascial tissue. Elasticity is an passive force, however, unlike the passive force of fascia that simply resists being lengthening out, titin’s elasticity adds a pulling force that continues to act until the muscle is returned to its resting length.

Elasticity is extremely efficient. A muscle that is stretched can rely upon its passive elasticity to create movement without any active contractile force created by the sliding filament mechanism. For example, if I stretch my upper back into extension by concentrically contracting my extensor muscles of the upper spine, when I want to return to the initial position, I do not need to actively contract my spinal flexors, rather I need only relax. Upon relaxation of the spinal extensor musculature, my stretched muscular tissues in the front of the trunk will naturally pull me back into flexion. In this manner, the motion of our body can be very efficient. A muscle on one side of a joint expends energy to contract and move us in one direction, and we can then passively return to our starting position merely by relaxing and letting the stretched tissues on the other side of the joint elastically recoil back, without the expenditure of energy and effort.
Fascial tissues with high concentrations of myofibroblasts have been found to have sufficiently strong force to impact musculoskeletal mechanics—meaning their active pulling forces are strong enough to contribute to body movement.

Physical stress that the fascial tissue is experiencing. As greater and greater tensile forces are placed upon the fascia, more and more myofibroblasts develop from the normal fibroblasts of the fascial tissue. For this reason, myofibroblasts are also known as “stress fibers.” These myofibroblasts can then create an active pulling force that counters and opposes the pulling force that the fascial tissue is experiencing. For this reason, myofibroblasts are found in the greatest concentration in fascial tissues that have been injured and are undergoing wound healing. Fascial tissues with high concentrations of myofibroblasts have been found to have sufficiently strong force to impact musculoskeletal mechanics—that is, their active pulling forces are strong enough to contribute to movement of the body.

Even noninjured fascial tissues have been found to contain myofibroblasts, albeit to a lesser degree. In cases where an overt physical trauma is absent, these myofibroblasts have developed in response to the accumulation of pulling tensile forces placed upon these tissues. Lower myofibroblast concentrations, while not sufficiently strong to impact musculoskeletal dynamics, have been found to exert sufficient isometric contraction pulling force to maintain their tissue integrity in the face of the tensile forces they are experiencing. In particular, the thoracolumbar fascia of the back, plantar fascia of the foot, and the fascia lata of the thigh have been found to be particularly well populated with myofibroblasts.

The importance and application of this knowledge are multifold. First, the field of kinesiology needs to consider fascial contractile forces when evaluating the contractile forces of the musculoskeletal system. Albeit far weaker in degree than muscular contraction forces, these fascial contraction forces are significant enough that they should be factored in. In the field of pathology, this knowledge points to the ability of fascial scar tissue to exert contractile forces that are helpful in closing and binding together wounded tissue. However, it also points to the possibility that if myofibroblastic contraction is excessive compared to the demands placed upon the fascial tissue within which it is located, it may create an unhealthy—perhaps even destructive—effect on fascial and other nearby tissues. This would most likely occur in noninjured tissues that are experiencing chronic tensile stresses, such as chronically tight and taut tissues (e.g., tight muscles and taut ligaments). In these pathologically contracted fascial tissues, bodywork and movement therapies may be particularly valuable and effective by helping to change the stress loads that are placed upon the fascia and minimizing myofibroblastic development.

The research on fascial contraction is still emerging. As our knowledge base increases, however, the curricula of massage therapy and other bodywork and movement professions may need to pay close attention and allow for this more sophisticated understanding of the structure and function of fascia to be incorporated and taught in both core curriculum and continuing education classes.*