The emerging strategy of *fascia unloading*, defined as reducing tension in the interconnected fascia layers in response to the mechanical load applied to the tissue during movement, has recently gained popularity as a potential method for enhancing injury rehabilitation and promoting muscular performance. Kinesio Taping (KT) is a therapeutic procedure that is believed to facilitate fascial unloading. The practice gained international exposure at the 2008 Beijing Olympic Games, where athletes from various sports (i.e., track and field, cycling, tennis, and badminton) wore kinesiotape (Figure 1). More recently, sports commentators have noted the use of KT by cyclists competing in the 2010 Tour de France and the 2011 Asian Football Confederation Cup in Qatar. Although KT is commonly used by elite athletes, the mechanism by which this technique alters fascial structures remains speculative. The purpose of this report is to describe fascia physiology and the theory of fascia unloading, with a focus on the use of the KT method during rehabilitation and its influence on exercise performance.

**Fascia Physiology**

Early reports referred to fascia, a connective tissue that surrounds and separates muscle tissue, as a covering for muscles that is a continuation of tendon with limited...
function. It was later described as an exoskeleton, suggesting a greater physiological role. Some now suggest that fascia has contractile components, which can integrate proprioceptive signals and assist in load bearing (e.g., the lumbar fasciae). Fascia has been described as having plastic properties because it deforms when a load is imposed and complete recovery of its normal state may take several hours. The tissues that have primarily been studied include the thoracolumbar fascia, the iliotibial tract, and the plantar aponeurosis. Fascia myofibroblasts are cells that are capable of exerting continuous force over long periods of time, which may influence the structural stability of the tissue. Myofibroblasts may represent an intermediate cell type between a smooth muscle cell and a fibroblast. The sustained contractile ability of myofibroblasts may play a role in chronic contractures, such as Dupuytren’s contracture of the palmar fascia or adhesive capsulitis in the shoulder. These cells are not stimulated to contract by a neural impulse, which suggests that they are not subject to conscious control. There appear to be two factors that induce long-duration, low-energy contraction of myofibroblasts: (a) mechanical tension within the tissue and (b) binding of specific cytokines and other agents (i.e., nitric oxide, histamine, mepyramine, and oxytocins) to cell membrane receptors. Angiotensin and caffeine, which are calcium channel blockers, and norepinephrine and acetylcholine, which are neurotransmitters, have no effect on these cells. Direct neural stimulation of skeletal muscles by the somatic nervous system involves acetylcholine, whereas smooth muscles are activated by the parasympathetic nervous system through release of norepinephrine. The fact that myofibroblasts do not respond to neural stimulation may have implications for therapeutic fascia loading and unloading techniques that may be used for pain management.

**Fascia Loading**

Long-duration contraction of the connective tissue may play a role in acute or chronic musculoskeletal pain. Fascia contraction occurs very slowly over a period of 20-30 minutes and may be sustained for more than an hour before slowly subsiding. The contraction develops in response to a sustained load. The lower the pH (i.e., an acidic environment) causes myofibroblasts to contract. Therefore, conditions such as a breathing pattern disorder, emotional stress, or consumption of acid-producing foods could induce a general stiffening of the fascia.

Athletes often complain of muscular pain that is not caused by a specific traumatic incident. They often describe muscles as being locked, inferring that there is tightness within the affected limb. Muscle locking has been described in the literature as an eccentrically loaded muscle (locked long) or a concentrically loaded muscle (locked short). Connective tissue can be remodeled by the positioning and movements of the body segments. Repetitive movement of a specific muscle group can produce a thickening or shortening of the superficial and/or deep fascia surrounding the activated muscle, which may provide more stability and allow the muscle to generate more power. During the process of fascia remodeling, inadequate lengthening (regular stretching) may produce a dysfunctional state that could increase risk for fascia tearing. The primary goal of the athletic trainer or therapist (AT) will be restoration of optimal functional status through (a) restoration of normal range of motion, (b) development of neuromuscular control, and (c) remediation of strength deficits. Fascial manipulation and KT are two therapeutic procedures that are increasingly implemented by ATs to assist in this process.

**Fascia Unloading for Rehabilitation**

Fascia damage (i.e., microtearing and/or inflammation) is believed to be common among athletes and is believed to be under-diagnosed. Only pathology in thick fascia bands, such as the plantar fascia, is easily identified through diagnostic imaging. Unlike muscle fibers, which signal a need for development of sarcomeres when heavily loaded, fascia is extremely susceptible to microtears when stretched quickly (e.g., high-intensity eccentric loading). If the fascia stretch is applied slowly over a long period of time, however, it may undergo plastic deformation. Skeletal muscle fibers can be described as relatively elastic, whereas fascia behaves more like a plastic material. Although fascia microtears may cause discomfort, they may not be detectable through diagnostic imaging. The fascia is innervated by free nerve endings that convey nociceptive neural signals. In fact, nociceptors are most abundant in the skin and the outer layers of connective tissue. A pain signal is transmitted from the fascia to the spinal cord, and ultimately to the brain, but the exact pathway for transmission of the pain impulse can vary. When healing is complete, nociceptive input should