Fascia and Pain.
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The human body must be considered as a functional unit, where every area is in communication with another through the fascial continuum, consequently originating perfect tensegritive equilibrium.1 Medical literature does not suggest a sole definition of fascia, because it varies in terms of thickness, function, composition, and direction depending on its location. The fascial tissue is equally distributed throughout the entire body, enveloping, interacting with, and permeating blood vessels, nerves, viscera, meninges, bones, and muscles, creating various layers at different depths, and forming a tridimensional metabolic and mechanical matrix. The fascia becomes an organ that can affect an individual’s health.1

The fascial continuum is essential for transmitting the muscle force, for correct motor coordination, and for preserving the organs in their sites: the fascia is a vital instrument that enables the individual to communicate and live independently.1,2

The connective tissue can directly convey pain signals; in fact, it contains nociceptors that can translate mechanical stimuli into pain information.1 Furthermore, if there are nonphysiological mechanical stimuli, the proprioceptors can turn into nociceptors.1 There are some researchers who suggest strongly that any change in the viscoelasticity of the fascial system activates the nociceptors.1 Reduced sliding of the various layers limits the functionality of the endocannabinoid system. There is a close relationship between the endocannabinoid or endorphin system and the fibroblasts. The cannabinoid receptor, or CB1 (is the most common neuroreceptor), is mainly housed in the nervous system, but it can be found in the fascial system and in the fibroblasts as well, particularly near the neuromuscular junction.1,2 This relationship is believed to better manage any inflammation and pain information originating in the fascial tissue, as the fascia undergoes continuous remodeling during the day.2 It is hypothesized that the axoplasmic flow originating in the dorsal ganglionic roots carries some molecules to the distal nerve endings, in an attempt to reduce pain information deriving from the nociceptors in the fascial continuum, such as CB1.1,2,3 Normally, CB1 closes Na+ and opens K+ channels, hyperpolarizing the nociceptor, preventing peripheral sensitization and pain.3 If there is a mechanical barrier owing to a reduction of the fascial sliding, the axoplasmic flow will be hindered, with consequent onset of hyperalgia, because CB1 can not be transported in the distal nerve.3

2. Bordoni B, Zanier E. Understanding fibroblasts in order to comprehend the osteopathic treatment of the fascia. Evidence-Based Complementary and Alternative Medicine, 2015, article ID 860934, 7 pages, in press.