BACKGROUND: Deep fascia has been reported as a potential source of pain due to the abnormal stimulation of embedded nerve receptors that can become enmeshed within pathological changes in this tissue. Fibrosis (accumulation of excessive amounts of fibrous connective tissue) and densification (an increase in the density of fascia) have been proposed as typical pathological or dysfunctional changes that can occur to deep fascia. These changes can modify the mechanical features of deep fascia, thereby causing alteration or dysfunction in related organs or muscles. The mechanical imbalance and connective tissue matrix disorders found in fibrosis and densification differ. Understanding these differences is important when selecting specific treatment modalities to relieve chronic pain syndromes.

APPROACH: Review of available literature

RESULTS: In order to identify the different alterations that can lead to pain, this review presents an overall description of deep fasciae, focusing on its multi-layered structure. It includes an analysis of the mechanical properties of deep fascia, such as its viscoelastic properties and non-linear stress-strain behaviour. The properties of loose connective tissue are discussed, as well as the characteristics of hyaluronic acid and its role within these layers.

CONCLUSIONS: Fibrosis consists of an alteration of the fibrous component of fasciae that involves a random accumulation of collagen fibers. This is difficult to modify manually. Densification is an alteration of the viscosity of the loose connective tissue within fascia caused by a concentration of hyaluronic acid [1]. Densification does not alter collagen fiber bundles but, nevertheless, changes the tensile status of the fascia and affects the sliding component. This type of alteration appears to be reversible through appropriate manual therapy [2]. Surgery, trauma, diabetes or aging are among such changes that
anatomically alter the fibrous layers of fasciae, causing fascial fibrosis. Overuse syndromes, exercise or diet may affect the loose connective tissue component of the deep fascia by causing densification, which then alters its sliding component.
