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New evidence of a dynamic fascial maintenance and self-repair process

Leon Chaitow

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Leon Chaitow

New evidence of a dynamic fascial maintenance and self-repair process

Dittmore et al (2016) have described experimental evidence that collagen operates a self-healing process involving what they term “*cleavage-vulnerable binding regions*”.

These sites are arrayed periodically at $\sim 1\mu\text{m}$ (one millionth of a meter) intervals, along collagen fibrils.

They note that the triple-helix of fibrillary collagen, in its most common form, assembles into highly organized networks that provide the scaffolding for the extracellular matrix, tendons, bones, and other load-bearing structures.

The essence of the model that emerges from the Dittmore et al experiments is that, when collagen fibrils are in what are termed an ‘*intact*’ phase, molecules are in a straight conformation. This is a ‘*high-energy*’ state, that periodically results in the accumulation of internal strain, relieved by the collagen uncoiling to form ‘*buckled*’ molecular configurations (termed ‘*cleavage sites*’). This buckling process exposes the unloaded collagen, allowing enzymes, such as specialized matrix metalloproteinases (MMPs), to bind to it, before initiating proteolysis – followed by subsequent repair and remodeling.

The data provided by Dittmore et al suggests that fibrillar collagen self-regulates its own maintenance in this way - by a constant process of repairing collagen fibrils, on a cellular level.

The importance of tissue tension

Importantly, Dittmore et al also note that tension-dependent stabilization against degradation by MMPs, has been demonstrated in normal tissue.

Others, such as Susilo et al (2016) have also reported that: “*mechanical loading induces stabilizing changes internal to the fibrils themselves, or in the fibril-fibril interactions.*”

This suggests that the self-repair, remodeling sequence may be delayed (i.e. made less necessary) by the presence of appropriate levels of tension, since, as has been explained

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