



RESEARCH UPDATE

Pathophysiological Model for Chronic Low Back Pain — Integrating Connective Tissue and Nervous System Mechanisms

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From abstract

Although chronic low back pain (cLBP) is increasingly recognized as a complex syndrome with multifactorial etiology, the pathogenic mechanisms leading to the development of chronic pain in this condition remain poorly understood.

This article presents a new, testable pathophysiological model integrating connective tissue plasticity mechanisms

with several well-developed areas of research on cLBP (pain psychology, postural control, neuroplasticity).

We hypothesize that pain-related fear leads to a cycle of decreased movement, connective tissue remodeling, inflammation, nervous system sensitization and further decreased mobility.

The integration of connective tissue and nervous system plasticity into the model of cLBP will potentially illuminate the mechanisms of a variety of treatments that may reverse these abnormalities by applying mechanical forces to soft tissues (e.g. physical therapy, massage, chiropractic manipulation, acupuncture), by changing specific movement patterns (e.g. movement therapies, yoga) or more generally by increasing activity levels (e.g. recreational exercise).

Non-invasive measures of connective tissue remodeling may eventually

become important tools to evaluate and follow patients with cLBP in research and clinical practice.

These authors also note:

“Historically, mechanistic models for cLBP have tended to focus on musculoskeletal tissues, on the nervous system, or on behavior. In this paper, we propose a new, dynamic and integrative pathophysiological model for cLBP bringing together recent research on movement and neuroplasticity along with well-established connective tissue remodeling mechanisms.”

The authors propose that “plasticity in both connective tissue and nervous systems, linked to each other via changes in motor behavior, play a key role in the natural history of cLBP, as well as the response of cLBP to treatments and placebos.”

The “association between symptoms and imaging results (X-ray, CT, MRI) has been consistently weak, and up to 85% of patients with low back pain cannot be given a precise pathoanatomical diagnosis using these methods.”

“The generally poor predictive value of diagnostic imaging in cLBP, and the often disappointing effects of many ‘lesion-specific’ treatments such as intra-articular corticosteroid injections, has spurred research efforts toward ‘non-structural’ psychological and behavioral aspects of cLBP, and away from tissue pathology.”

“Rest may be initially important in the face of acute low back injury (e.g. disc

herniation, muscle sprain), it is increasingly recognized that timely resumption of physical activity is critical to successful rehabilitation.”

“In addition to abnormal movement patterns, patients with cLBP have been shown to have generalized augmented pain sensitivity and cortical activation patterns suggesting abnormal central pain processing.”

“Ongoing pain is associated with widespread neuroplastic changes at multiple levels within the nervous system and including primary afferent neurons, spinal cord, brainstem, thalamus, limbic system and cortex.”

Neuroimaging has shown that there are distinct “brain networks” involved in acute vs. chronic pain. Chronic pain is specifically related to regions for cognition and emotions.

Chronic back pain results in neuronal or glial loss in the pre-frontal and thalamic gray matter.

Current models view chronic pain as a multisystem output, the “pain neuro-matrix” includes both sensory and motor components.

“We hypothesize that connective tissue remodeling occurs in cLBP as a result of emotional, behavioral and motor dysfunction.” **[Very Important]**

“We further hypothesize that increased connective tissue stiffness due to fibrosis is an important link in the pathogenic mechanism leading to chronicity of pain, fear-avoidance and

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