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# An Integrated Approach to Treatment of Chronic Low Back Pain: Based on Neuroscience of Pain



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## Abstract

Prevalence and recurrent rate of chronic low back pain is high and causes work incapacity, increasing the cost of both medical resources and the economic burden on society. The neuroscience of pain and chronic pain indicated by the ascending and descending pathways and the pain matrix in the brain is altered. Mesolimbic and prefrontal areas altered neuronal responses to pain. Furthermore, attention and emotional modulatory pathways have mechanisms on pain modulation related to different areas of the pain matrix. Neurophysiological changes in somatosensory and motor cortex changes lead to changes in the motor control of the impaired laterality recognition and body images. Additionally, physical pain and virtual pain share the same areas of activation in the pain matrix, but activate differently. Changes in ascending and descending pain modulatory systems, the PAG-RVM pathway, the dorsal horns of the spinal cord and peripheral receptors, influence central sensitization through different mechanisms.

Current peripheral mechanisms of pain describe low back pain as changes in muscle, joint, and movement patterns, related to a deficit in proprioception and motor control. Traditional approaches, including modality and bio-mechanical theory with treatment result in room for improvement. Fascia and its attached core muscle is the target of new treatment strategies for improving trunk stability for patients with chronic low back pain. Fascia expansion of various muscles from proximal to distal parts forms a myofascial chain that resembles the Chinese Meridian, serving functions in force transmission, protection, circulation and homeostasis. Fascia lesion stimulating nociceptors is considered the source of low back pain. Top-down mechanism approaches for pain management, including motor control, motor imagery, cognitive behavior therapy, and pain education demonstrate promising results. The need of an integrating model for dealing with chronic pain is urgent.

**Keywords:** Low back pain; Fascia; Pain management; Chinese meridian

**Abbreviations:** ACC: Anterior Cingulate Cortex; PFC: Prefrontal Cortex; RVM: Rostral Ventromedial Medulla; S2: Secondary Somatosensory Cortex; AMY: Amygdala; S1: Primary Sensory Cortex

## Introduction

### Prevalence and recurrent rate of chronic low back pain

The high recurrence rate and prevalence rate of chronic low back pain causes work incapacity and increases the cost of both medical resources and the economic burden on society. The incidence of low back pain is about 84%, with a high recurrence rate and prevalence rate [1]. Low back pain has been considered as a kind of disability [2]. Treatments are mainly focused on decreasing pain through traction and exercise, but with only short-term effects. Chronic fascial low back pain is a common musculoskeletal disorder, and is categorized according to the source of pain: muscle, ligament, and soft tissue-related, neurological, joint-related, and other [3]-[4]. There are 65% of patients with muscle-related low back pain. Muscle-related low back pain is considered an early stage of the disorder, and has potential of reversal. Pain recurrence would lead to dysfunction,

decrease in quality of life, increased consumption of social resources, and loss of productivity may be inevitable.

### Neuroscience of pain and chronic pain: the ascending and descending pathways and the pain matrix in the brain

A recent review examined the area of neuroplasticity changes of the pain matrix associated with chronic musculoskeletal disorders [5]. The pain matrix includes the associated sensory cortex, the anterior cingulate cortex (ACC), and the insula cortex. Changes in certain areas of the brain are associated with specific signs/symptoms of patients with chronic pain.

### Changes in mesolimbic and prefrontal areas altered neuronal responses to pain

Changes in mesolimbic, prefrontal and pain matrix, including the insula, cingulate cortex, and nucleus accumbens lead to the altered response to pain, regard to the "unpleasantness"

and spontaneous pain, change ineffective, cognitive, and/or motivational aspects of pain, which may be presented as signs and symptoms. Besides, changes in psychological aspects of pain, such as fear avoidance and catastrophization, are also affected [6].

### **Attention and emotional modulatory pathways have mechanisms on pain modulation related to different area of pain matrix**

Attentional pathways include the ACC, prefrontal cortex (PFC), periaqueductal gray [7], and rostral ventromedial medulla (RVM) while emotional pathways include the nucleus spiriformis lateralis (SPL), secondary somatosensory cortex (S2), insula, and amygdala (AMY). When focusing on pain, the activity in primary sensory cortex (S1), insula, and ACC, is stronger than when distracting from the pain. As for the emotional pathway, negative emotional states produced by looking at emotional faces, listening to unpleasant music, or smelling unpleasant odors [8], altered pain-evoked cortical activation in ACC consistently. These researches suggest the existence of different mechanisms and modulations of the pain matrix behind attentional and emotional modulatory pathways [9].

### **Neurophysiological changes in somatosensory and motor cortex changes lead to changes in the motor control of the impaired laterality recognition and body images**

Changes in the somatosensory cortex include expansion, restriction, or shifting of the representation of the somatosensory map, therefore, increase in 2-point discrimination, underperformed lateral recognition and changes in body image perception may be presented [10]-[12]. When the primary motor cortex is influenced, altered brain mapping of motor areas is identified [11]; [13]. Changes in corticospinal excitability were also noted. Therefore, impaired ability to selectively recruit individual muscles, and the ability to perform co-contraction were presented. Brain functional connectivity of the sensorimotor network in patients with low back pain using fMRI has revealed significant decrease in the left supplementary motor area, left precentral gyrus, and lobules IV and V of the cerebellum. In contrast, an increase in the right middle frontal gyrus and superior frontal gyrus has been discovered [12]. Therefore, it is likely that both the peripheral and the central neural systems should be considered as important factors when discussing low back pain.

### **Physical pain and virtual pain shared the same areas of activation in the pain matrix, but activate differently**

“Shared representation theory of social pain” suggests that rejection and related experiences piggyback on brain systems evolve to represent physical pain [14]-[16], and the overlapping activity in previous studies have strengthened this theory. However, a recent study that examined the “core pain processing

region”, dorsal anterior cingulate cortex with fMRI revealed different patterns in activation evoked by physical pain and rejection, suggesting a different pathway of signal transmission and processing for physical pain and rejection [9]. A possible explanation may be that while physical pain is a bottom-up stimulus, rejection is a top-down signal received by the brain. It is possible that chronic low back pain could be induced physically, psychologically, and socially.

### **Changes in ascending and descending pain modulatory systems, PAG-RVM pathway, dorsal horn of the spinal cords and peripheral receptors, influence central sensitization through different mechanisms**

Not only changes in central but also in peripheral receptors contribute to the neuronal changes. When changes occur at peripheral receptors, there is an increased release of neurotransmitters from the peripheral through the projection of the dorsal horn to the upper neural system [17] and changes about the input and output characteristics are presented, contributing to central sensitization [7]. Changes in descending pain modulatory systems, PAG-RVM pathway and descending inhibition of pain (disturbed conditioned pain modulation), therefore, contribute to central sensitization [18]-[19]. Changes in the dorsal horn of spinal cord lead to increased transmission of nociceptive and neuropathic stimuli [5]; [20]-[21], as well as changes in membrane permeability. Through the changes of descending pain modulatory systems and the dorsal horn of spinal cord, pain threshold decreased, and central sensitization may be presented.

### **Bottom-up Approaches to Treating Chronic LBP**

#### **Current peripheral mechanism of pain describes low back pain in the changes of muscle, joint, movement pattern, related to deficit in proprioception and motor control**

To explore the possible cause of low back pain, one possible theory is the bottom up theory. When there is injury to the muscle, the body must compensate to maintain its functional ability, which leads to altered joint position and movement pattern. As a result, under-stretched or tension-concentrated muscles or joints will stimulate the pain receptors within, and the signals will be transmitted to the higher neural system. A recent review about lumbo-pelvic kinematics in people with and without back pain suggested that though there are no differences between lordosis curves, reduced lumbar ROM, speed, and proprioception has been discovered. Changes in proprioception and motor control have also been thought as a possible reason behind low back pain [22]. As a previous review suggested, patients with low back pain performed more error during a reposition test, and when asked to reposition with a slower speed, patients with low back pain required more practice to achieve the target, indicating altered motor control and compromised proprioception. Besides, a recent study has shown that when performing a sit-to-stand-to

sit task, patients with low back pain needed more time to finish the task compared to those without [12].

**Traditional approaches including modality and bio-mechanical theory with treatment result in room for improvement**

Traditional therapy is mainly focused on symptoms relief. Both heat and cryotherapy are typically applied for short-term, immediate effects on patients. Using electrical stimulation alone cannot increase the chances of recovery for function. Traction applied to patients with low back pain only helped to decrease the pain, but with mild effect. A 2013 Cochrane Database systemic review regarding the effect of mechanical traction on low back pain patients without sciatica pain revealed that there was moderate-quality evidence suggesting that in the aspect of pain intensity, there was mild or no decrease in pain when comparing traction to non-treatment [23]. Furthermore, manual therapy and exercise are recommended for treatment of low back pain in the 2016 treatment guideline [24]-[27].

**Fascia and its attached core muscle is the target of new treatment strategy for improving trunk stability for patients with chronic low back pain**

New treatment strategy focusing on training and motor learning of core muscle, and clinical guideline has suggested that it can effectively decrease pain and increase function. In a study, including 414 participants in 5 trials revealed that core muscle exercise was superior to other types of exercise, but the effects only last for 6 months [28]. Locating in the deepest layer of muscle around the spine, and connects to the fascia, core muscle contains high amount muscle spindle to be responsible for proprioception of spine. Most of core muscle contains muscle, tendons, such as multifidus, that has more than 50% of

tendinous ingredients. Transverse abdominis connects to fascia to form a natural brace providing lumbar spine stability [29].

Thoracolumbar fascia consists of anterior, middle, and posterior parts to separate erector spinae, abdominal muscle, Quadratus lumborum, and Psoas major; and then connects back muscles to form a supportive belt, and expands upward to thoracic, neck and finally occipital area, downward to sacrum. The myofascial system with the vertebral spines forms a tension-compression balance system, tensegrity. The change of the tension of the muscle fascia system results in morphological changes and movement changes [30].

**Fascia expansion of various muscles from proximal to distal parts form myofascial chain resembles Chinese Meridian, serving functions in force transmission, protection, circulation and homeostasis**

Recent research explores the connection between deep fascia and muscle. The anatomical structure of fascia [31], mechanical interaction with muscles, proprioceptive role in protection and pain processing, and their role in circulation in maintaining homeostasis may provide us new ideas about how does low back pain occur. Deep fascia are well-organized dense fibrous layers that interact with muscles, and provide sliding and gliding mechanism [32]. Myofascial expansion is the connection from a skeletal muscle or tendon and inserts into the aponeurotic fascia, functioning in stabilizing tendons, and reducing the stress of bony attachment [32]. Fascia retinacula or reinforcement areas are richly innervated, and are considered to be a dynamic pulley system, which mechanically change the line of pull of muscles during motion, and proprioception providing sensory input for coordination. Fascia is well vascularized and with lymphatic channels to serve functions in circulation and fluid hemostasis

**Fascia lesions stimulating nociceptor is considered the source of low back pain**

Table 1: Bottom-up Approaches to Treating Chronic LBP.

| Bottom up approach     | Area related        | Changes/method  | Sign and symptoms  | Author  |
|------------------------|---------------------|---|--|---|
| Manual therapy         | Dorsal horn         | Increased membrane excitability of neuron/neuro-physiological recording<br>Decreased descending modulation/fMRI | Hyperalgesia and allodynia<br>Pain thresholds decreased          | Boadas-Vaello et al. [17]<br>Snodgrass SJ et al. [43]                                       |
| Peripheral stimulation | Peripheral receptor | Change of muscle receptor excitability<br>Increased stimulation of nociceptive stimuli/nerve recording          | Peripheral sensitization<br>Contributes to central sensitization | Snodgrass SJ et al. [43]  |
| Therapeutic exercise   | Muscle Fascia       | Increased nociceptive input from thoraco-lumbar fascia to dorsal horn   | Non-specific low back pain                                       | Saragiotto et al. [25]<br>Mense & Hoheisel [33]<br>Wang et al. [28]<br>Stecco & Hammer [32] |

It is believed that within muscles are many muscle spindles, so when contracting muscles, signals will be detected and received. As a result, injury to muscle may stimulate nociception and cause pain. But recent studies suggest that muscle spindles lay not within the muscle, but in the fascia particularly at the junction of the perimyosium and epimyosim [32]. Therefore, it is possible that the pull of the fascia stimulates certain receptors causing proprioceptive response of muscles. Abundant free and encapsulated nerve endings have been found in the thoracolumbar fascia. Nerves are more numerous in the superficial and intermediate sublayers, but not in the deep

layers. Thus, when muscle contracts fascia is being stretched. When the force is prolonged or excessive, it stimulates the nociceptors at fascia and lead to pain. A recent study inducing experimental inflammation at the thoracolumbar fascia of rats showed increased density of nociceptive receptors on fascia, suggesting a possible connection to low back pain [33]. Injecting inflammatory substances into thoracolumbar fascia tissue in *in-vivo* condition resulted in strong pain, in comparison to injecting into muscle tissue, which supports the concept that the nociception from fascia is the source of low back pain (Table 1), [34].

**Top-down Mechanism Approaches for Pain Management Including Motor Control, Motor Imagery, Cognitive Behavior Therapy and Pain Education Demonstrate Promising Results**

**Table 2:** Top-down mechanism approaches for pain management.

| Top Down Approach   | Area Related  | Changes/Method  | Sign and Symptoms  | Author   |
|---|---|---|--|--|
| Cognitive-behavioral<br>Neurophysiology education of pain<br>Placebo effect   | Pain matrix<br>Limbic system<br>Thalamus<br>Cerebellum<br>Prefrontal cortex | Decreased grey matter at insula, ACC and PFC/MRI<br>Decreased white matter integrity/DTI<br>Learning with movement and negative outcomes/fMRI<br>Attentional modulation/fMRI<br>Emotional modulation/fMRI | Unpleasantness<br>Spontaneous pain<br>Maladaptive behavior patterns<br>Fear avoidance, anxiety, depression<br>Catastrophization, somatization, worry and increased vigilance | Bushnell et al. [20]; Heinricher et al. [10]; Ehde et al. [36]; Engers et al. [37]; Moseley [38]; Wetherell et al. [39]; Veehof et al. [41]; Snodgrass et al. [43]; Pelletier et al. [5]; Seminowicz & Davis, 2006; Ochsner et al. [2] |
| Sensory discrimination training and implicit imagery<br>External focus of attention with motor learning<br>Transcranial magnetic stimulation (TMS)<br>Peripheral electrical stimulation | Sensory motor cortex<br>Associative area                                    | Altered somatosensory maps/ MRI cortical re-organization/rest fMRI<br>Changes in muscle and movement representations and cortico spinal excitability/ TM  | Disturbances in body image<br>Co-contraction<br>Difficulty in individual muscles activation  | Heinricher et al. [10]; Tsao et al. [13]; Moseley et al. 2008;   |
| Cognitive based general exercise  | Descending modulatory systems, PAG-RVM                                      | Decreased descending inhibition of pain/ functional neuro-imaging   | Hyperalgesia and allodynia<br>Pain threshold decreased   | Bushnell et al. [20]; Lee et al. [7]; Heinricher et al. [10]; Ossipov et al. [18]; Porreca et al. [19]   |

Motor control training for 2 weeks induced a shift in the motor area controlling trunk muscles in patients with chronic low back pain, indicating the post-training neuroplasticity of the motor cortex in patients with chronic pain [13]; [35]. To address the behavioral change in fear avoidance as well as other symptoms associated with chronic low back pain, intervention targeting cognitive-behavioral approach has been developed and applied, with promising results [20]. To promote neuroplasticity, repetition and intensity are required for adaptive change. Re-conceptualizing pain, neurophysiology of pain focusing on anatomical and physiological information, education as well as

increasing the understanding of noxious stimuli processing of patients, which has been demonstrated with promising effects in decreasing pain, are associated with changes in brain activation [36]-[38].

Addressing maladaptive thoughts has been demonstrated with the effects of improving mood, decreasing pain intensity for up to 6 months in patients with chronic pain [36]; [39], while acceptance-based interventions such as acceptance commitment therapy has been applied to healthy subject and with the effects of neuroplasticity in the insula and S1 [40] as well as the

functional connectivity between the medial PFC and the the insula [41]; [39]. Cognitive-based intervention such as motor imagery can influence brain function and cortical processes [42], including sensorimotor areas. Though with positive results, it can only optimize its effects if subjects are able to sustain their attention (Table 2), [43].

## Conclusion

### The need of an integrating model for dealing with chronic pain is urgent

Chronic low back pain may have multiple causes. Therefore, single model (e. g. Bottom-up) can only partially explain the mechanism behind LBP. The need of integration includes the structural and physiological basis of myofascial trigger points and myofascial expansion, force transmission, neuroplasticity and neuroscience in pain management and pain prevention. Chronic low back pain is a bio-psychosocial issue further complicated by different cultural factors; requiring multilevel classification and identification of the biomarker and risk factors for optimal management and prevention. Therefore, when dealing with chronic low back pain, various soft tissues could be considered as targets, while the education of pain as well as other approaches dealing with plastic changes in the central neural system should be addressed to maximize the effect of the treatment.

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