

EDITORIAL

Chronification of low back pain: getting to the spine of the problem



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‘Houston, we have a problem.’ This is how I would best describe the state of affairs in low back pain management. Economically, the cost of low back pain management in U.S. has been on a rising trend. Martin et al. reported medical costs for spine problems from a Medical Expenditure Panel Survey from 1997 to 2005.¹ They found that the annual per capita age- and sex-adjusted medical costs for respondents with spine problems to be \$4695 (95% CI, \$4181–\$5209) in 1997, compared with \$6096 (95% CI, \$5670–\$6522) in 2005. Epidemiologically, the prevalence of chronic low back pain specifically, has also been on the rise. Freburger et al. found the prevalence of chronic low back pain to be on the rise over a 14-year interval, from 3.9% (95% confidence interval [CI], 3.4–4.4%) in 1992 to 10.2% (95% CI, 9.3–11%) in 2006.² How did we get to this point and where did we go wrong along the way? I would like to approach these two questions by exploring two root mechanisms that may yield tangible solutions within the realm of rehabilitation science and clinical practice: (1) peripheral biomechanical nociceptive mechanisms and (2) centrally mediated pain processing mechanisms within the brain. To simplify the complexity of low back pain and its heterogeneous nature, I propose to parse this patient population into three preliminary groups based on these mechanisms: (1) those with biomechanical mechanisms to their pain, (2) those with cognitive-affective mechanisms to their pain, and (3)

those with a combination of both, which I presume to be the majority of this patient population. I will introduce a cognitive-biomechanical model as a conceptual guide to help manage low back pain that potentially stems from these two root mechanisms.

As I recall my first year as a new graduate from physical therapy school, I cringe thinking about all of my failures (defined as less than favorable outcomes) on these patients that naively allowed me to ‘practice’ as a licensed professional. Can you imagine being seen by a physician freshly graduated from medical school without any form of residency, fellowship, or specialization training? On a number of low back pain patients, I remember providing extension exercises, flexion exercises, physical modalities, “core”/trunk strengthening, and mobilization/manipulation. I did it all. Sometimes it worked, and sometimes it didn’t. It was as if outcomes were based on a roll of the dice. We have all encountered these unfortunate patients, even the ones who failed medical management such as opiates, neuropathic medications, injection therapies, radiofrequency nerve ablation, and even spinal surgery. I challenge us to think of the patients who did respond favorably – what would prevent the recurrence of their problem? How is “core”/trunk strengthening going to help someone who does not modify the way they lift heavy boxes at work? How are extension or flexion exercises

going to help someone who continues to sit in a slouched position in front of a computer? How is any form of pharmacologic or injection therapy going to help the recurrence of low back pain that is associated with faulty walking mechanics? How sustainable is pain relief from a lumbar fusion if the pain is associated with a lifting task that results in a lumbar paraspinal muscle strain? Part of the solution to these thought-provoking questions is to turn our attention to the biomechanical mechanisms associated with low back pain.

The medical management of low back pain using the biomedical model is handicapped without consideration of the biomechanical factors that contribute to low back pain and its associated common complaints, such as lifting, bending, sitting, standing, or walking. Treatment without a diagnosis is like treating blindfolded. More specifically, treatment of low back pain without a biomechanically based cluster of a diagnosis (hypothesized tissue source [pain generator], tissue load [tensile, compressive, or shearing], and identified pathomechanics [movement fault]) is likely one of the fundamentally neglected factors associated with low back pain chronification in the rehabilitation arena. As a physical therapy profession that specializes in the 'movement system,' understanding the tissue source of pain, the adverse loads on this tissue source, and pathomechanics driving the loads on this identified source is crucial because our intervention is dependent on these findings. For example, trunk stabilization has been a treatment for low back pain for years, but what mechanism are we actually addressing? Would this intervention be a logical solution for someone suffering from a muscle strain as a result of poor lifting mechanics? Trunk strengthening may theoretically make it worse if the muscles continue to be strained during such exercise program. Obviously, one size does not fit all when it comes to low back pain and we are aware that there is a need for a logical diagnostic framework. In a systematic review with meta-analysis, Smith et al. concludes that "There is strong evidence stabilization exercises are not more effective than any other form of active exercise in the long term [for patients with low back pain]." ³ This conclusion is not surprising considering the patients used in these studies were not homogeneously grouped, nor were they biomechanically classified.

To put things in a more biomechanical perspective, let's look at one example of a low back pain case (Fig. 1A and B). In this case illustration, there is left sided low back pain presumed to be coming from excessive facet loading on the left lumbar region with specific complaints of standing. This case demonstrates an asymmetrical and excessive right pelvic drop/obliquity that can result in symptomatic left sided facet compression (Fig. 1A and B). ⁴ Fundamentally, no amount of trunk strengthening alone, would be able to normalize these pathomechanics or facet loads. However, it has been shown that hip abductor weakness is associated with greater frontal and transverse plane trunk excursions, which may result in excessive facet loading and pain, as illustrated in this case. ⁵ This provides a plausible conceptual framework in which hip strength and pelvic obliquity may play a relevant role in patients with mechanical facet pain as a result of appropriately identified pathomechanics. There is growing evidence showing an association between low back pain and gluteal weakness. ⁶⁻¹⁶ For example, Cooper et al. have shown that there is a higher prevalence of glu-

teus medius weakness in people with chronic low back pain compared to healthy controls. ¹⁷ Being able to identify these key physical impairments may lead to targeting the biomechanical underpinnings that result in low back pain. In this illustrated case example, it would be to address the hip weakness and normalize the functional standing pathomechanics, perhaps in addition to addressing relevant trunk weakness. This would therefore reduce the pathomechanical loads on the facets presumed to be the prospective pain-generating tissue source.

Not all patients with low back pain fit into any one particular box, such as the biomechanical model presented here. We have all had our fair share of "crazy patient" experiences; or to be more politically correct, "complex cases" that make it a long and labored day. Aside from the comorbidities that make these cases complex, I'm referring to those patients who have unconventional pain behavior presentations; those that seem to have their pain all over, stating, "it just hurts;" and when you go on to perform the physical examination, everything is painful, there is a constant level of pain, or the pain presentation does not fit a particular mechanical pattern. Somehow, these patients with chronic low back pain have commonly sought out and received practically every intervention option known to mankind, ranging from physical therapy modalities, manual therapy and exercise, to pharmacological and injection therapy management, to ultimately some sort of surgical intervention that puts them at risk for a failed outcome. Providers have typically and euphemistically documented these patients to be "poor historians" or "complicated" and cram them into some sort of diagnostic "box," but the fact of the matter remains – there are other underlying mechanisms other than the peripheral nociceptive one (biomedical model) that warrants further exploration. I now turn to the other end of the nervous system – the brain, a pain manufacturing plant.

At the most rudimentary level, I believe it is the interaction between psychosocial (cognitive-affective) and biomechanical variables that complicates our low back pain patients. If I could simply treat every musculoskeletal pain condition like a mechanic (a.k.a. biomechanist), life would be quite simple – normalize their motion, strength, and movement patterns, and call it a day. But when you throw pain into the clinical picture with cognitive-affective variables, things get potentially tortuous – and this, I presume results in what we call the "difficult or complex patient." Patient management now becomes more abstract and qualitative (biopsychosocial) vs. objective and quantitative (biomedical). Fortunately, the advent of advanced brain neuroimaging such as functional magnetic resonance imaging (fMRI), has made it more feasible to explore, discover, and understand how pain is influenced (and vice versa) by its interdependent psychosocial and biomechanical counterparts.

The medial frontal cortex (MFC) for example, is purported to play a key role in motor function, cognitive control, emotion, and pain. ¹⁸ This area of the brain is of particular interest to me because it is a large piece of the "pain puzzle" whose functional organization formulates a clear clinical framework. In a large-scale meta-analysis of almost 10,000 fMRI studies, de la Vega et al. identified 3 broad functional zones of the MFC: (1) the posterior zone

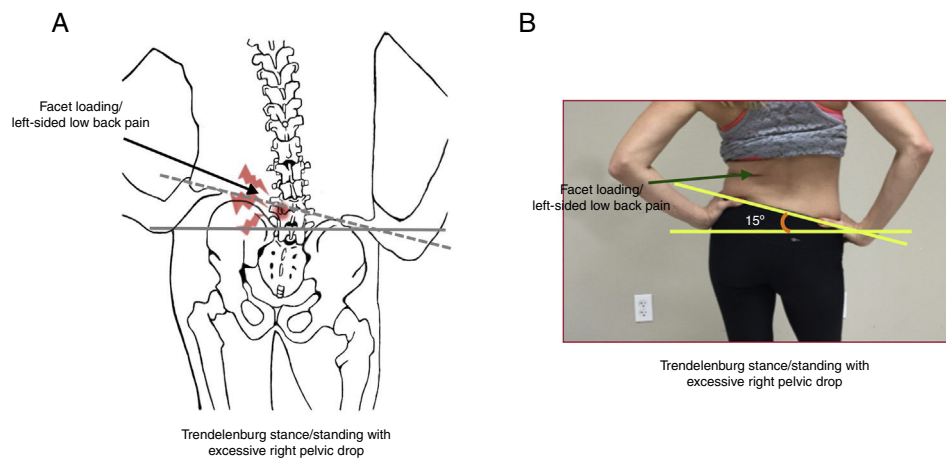


Figure 1 (A) Illustration of left sided-facet loading with an associated excessive right pelvic drop. (B) Photo case example of a patient with left sided low back pain during prolonged static standing. Note the potential for left sided-facet loading with an associated excessive right pelvic drop during this static standing position.

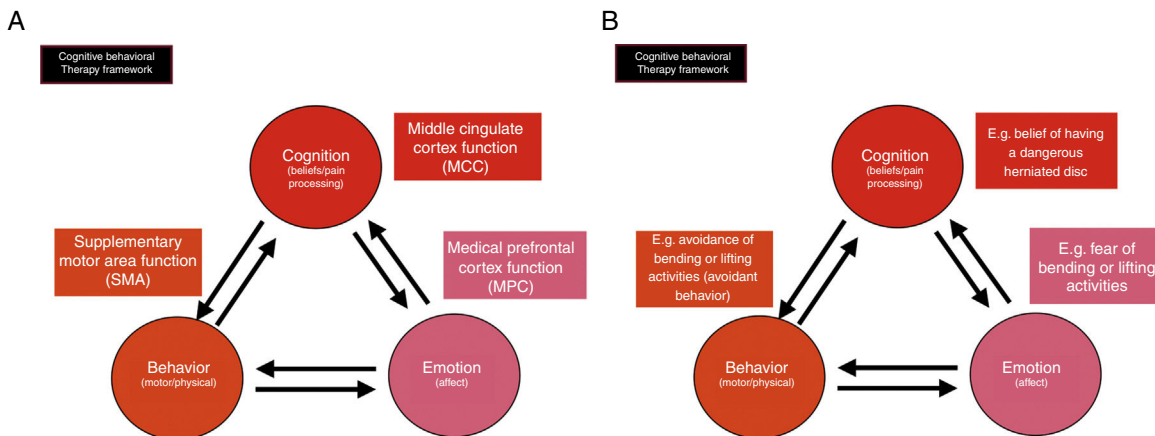


Figure 2 (A) Cognitive-Behavioral Therapy (CBT) framework showing the interdependency between cognition, affect, and behavioral variables noted in the colored circles. Also shown in colored rectangles are the brain regions known to be associated with these functions. (B) Clinical low back pain case example applied within the CBT framework.

(supplementary motor area and pre-supplementary motor area) was associated preferentially with motor function, (2) the middle zone (middle cingulate cortex) with pain and cognitive control, and (3) the anterior zone (medial prefrontal cortex) with affective processing such as fear or emotion.¹⁸ I've come to realize that these 3 broad functional zones of the MFC fit nicely within a cognitive behavioral therapy framework that illustrates how cognition (beliefs), emotions (affect), and behavior (motor control) all interdependently influence each other within the context of pain (see Fig. 2A). For example, take a patient with low back pain who *avoids* any type of bending (avoidant behavior) because he *believes* that he has a herniated disc (cognition/belief of having a dangerous herniated disc), and is *afraid* to engage in any type of bending or lifting activities (fear affect) (see Fig. 2B). Take note of the next patient you encounter with low back pain and see if you can identify the following components: (1) a behavioral component (e.g. *non compliance* with exercise program – “I haven’t had time to do my exercises”), (2) a belief/cognitive component (e.g. “I *believe* these exercises will make my pain worse”), and (3) an emo-

tion/affective component (e.g. “I’m *frustrated* that I was given these exercises to do at home, especially since they won’t help”). Now, see if you can change any one of these components and see how it might influence the other two components. This should give you some insight into the world of cognitive behavioral therapy and increase your awareness of the cognitive-affective dimensions of pain.

It has been a fascinating revelation to notice that the majority if not all patients who present with a complaint of pain also happen to have associated motor control impairments. Misera et al. have shown a neural mechanism that may help explain why patients who experience pain commonly have accompanied motor impairments.¹⁹ In their novel fMRI study paradigm, they found an overlapping functional activity of the MCC (pain and cognitive processing) and SMA (motor control) during independent trials of motor control and pain processing tasks, as well as when the tasks were done simultaneously.¹⁹ This may explain why some patients with motor control impairments (SMA) also have pain that is complicated by cognitive/belief processing issues, since the MCC is not only associated with pain processing, but cog-

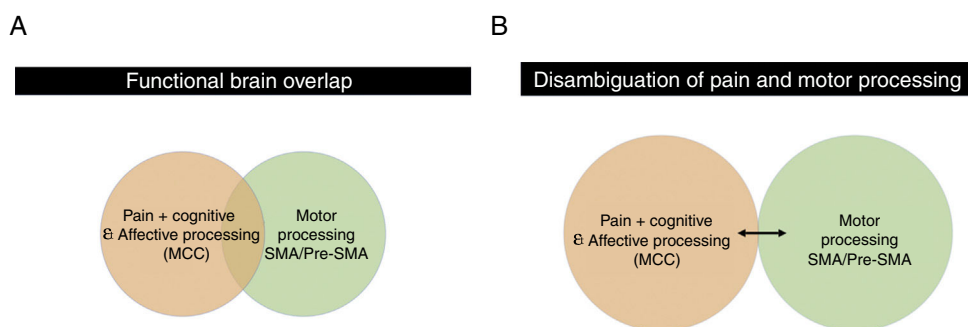


Figure 3 (A) Functional brain overlap of the SMA/Pre-SMA and MCC. This simplistic figure potentially explains why a large percentage of patients who present with pain in the physical therapy clinic also have underlying motor control impairments. The larger the overlap, the potentially more complicated a patient may be. (B) Disambiguating pain and motor processing. This figure illustrates less overlap between motor and pain/cognitive and affective processing, which may theoretically result in a less complicated patient presentation, or interaction between the two variables.

nition/belief processing as well. With the presumption that pain and cognitive-affective variables are what complicates the clinical picture, the strategy should be to disambiguate the overlapping functional activity of the SMA and MCC, or interdependency of motor control (biomechanics), pain, and cognitive-affective variables (see Fig. 3A and B). To clinically do so, we need to have a better clinical understanding of the cognitive-affective variables, aside from having a better grasp of the biomechanical concepts.

The attempt to understand and manage psychosocial variables predictive of chronic low back pain disability is not novel. For example, we know that higher pain catastrophization, decreased self-efficacy, increased fear of movement and avoidance, poor expectations of recovery, depression, pain-related anxiety in patients with low back pain is associated with chronic low back pain-related disability, as shown in multiple studies.^{20–28} What might be novel, is a strategic and organizational scheme that would help address the cognitive-affective variables associated with low back pain chronification. I believe it is crucial to find the key factor that mediates these modifiable variables. One such factor that can result in a cascading effect of reactions is the threat value of pain. The interpretation of an event as threatening has been shown to increase stimulus salience and to amplify the perception of pain and non-painful stimuli.^{28–34} I would like to introduce the Cognitive Threat Perception Model of Pain (Fig. 4). Starting with a cognitive high threat value, a patient may likely interpret an amplified pain response/interpretation that would result in an increased fear avoidance or fear of movement emotion and behavior, that would then result in an increase in catastrophizing thoughts, that would result in a rumination or attention perseverating effect on the pain, which would ultimately end up with a feeling of helplessness (decreased self-efficacy) and depression. In other words, threat may essentially be the catalyst that sparks this “domino effect” that has been shown to result in low back pain-related disability.

Now that I have dichotomized the problem of low back pain management into two basic mechanisms (peripheral/biomechanical and central/cognitive-affective), the question is what do we do about it? I would like to introduce a cognitive-biomechanical framework within the domain of physical therapy as a tangible solution that

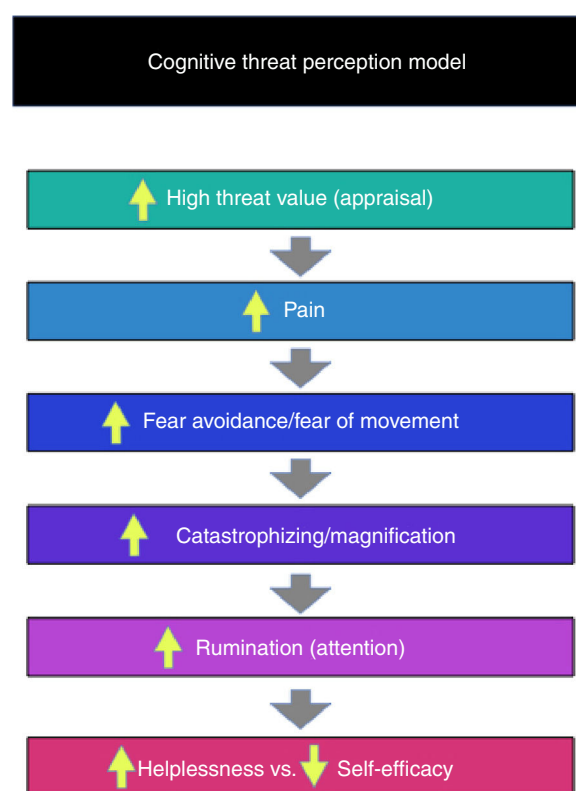


Figure 4 Cognitive Threat Perception Model. High threat value (appraisal) may trigger a cascading “domino effect” of increased pain and negative psychosocial responses that have been shown to be predictive of low back pain and disability.

would address the biomechanical as well as psychosocial (cognitive-affective) factors that comprise the underpinnings related to the chronification of low back pain. I believe a combined cognitive-biomechanical approach to low back pain management is warranted; this would include addressing biomechanical factors, as well as the cognitive-affective factors contributing to the evolution of low back pain.

What is interesting about a cognitive-biomechanical approach to low back pain, is that I believe many physical therapists unknowingly do it, albeit, inadvertently. The

cognitive-affective part of this approach involves a lot of operant conditioning and graded activity/exposure underpinnings that physical therapists commonly do as per their routine practice. Graded activity for example, involves positive reinforcement, education, exercise pacing, and being goal-oriented. The physical therapy analog to this also includes the same elements where the patients also have goals that are set (goal oriented), and the therapists oftentimes naturally provide coaxing and praise when their patients do well (positive reinforcement). The therapists will also educate them (reconceptualizing or framing/reframing their cognition/beliefs, or education) and pace the patient through their exercise program so as not to cause harm. Although I do admit to bastardizing the operant conditioning process here, the point is to illustrate that there may be other underlying mechanisms we are treating when we simply prescribe therapeutic exercises for our patients. From a cognitive behavioral framework, I would like to emphasize that the patients are getting a “story” to their problem – more specifically, a biomechanical story that connects all the dots to help explain and rationalize their pain. Admittedly, there is still much research that is needed in this area, but there is also much needed evidence for a lot of what is done in physical therapy. Moreover, there is a large need to study the underlying mechanisms behind what we do – thus my interest in peripheral versus central mechanisms of pain. I am hopeful that future research, both of my own and others in this field, will uncover more of these simple truths to help solve this complex problem.

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