

# Association Between Symptoms of Central Sensitization and Cognitive Behavioral Factors in People With Chronic Nonspecific Low Back Pain: A Cross-sectional Study

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## ABSTRACT

**Objective:** The objective of this cross-sectional study was to analyze the relationship between symptoms of central sensitization (CS) and important cognitive behavioral and psychosocial factors in a sample of patients with chronic nonspecific low back pain.

**Methods:** Participants with chronic nonspecific low back pain for at least 3 months were included in the study. They completed several questionnaires and a functional test. Pearson's correlation was used to analyze associations between symptoms of CS and pain behavior, functioning, pain, pain catastrophizing, kinesiophobia, and illness perceptions. Additionally, a between-group analysis was performed to compare patients with and without clinically relevant symptoms of CS.

**Results:** Data from 38 participants were analyzed. Significant associations were found between symptoms of CS and all other outcomes, especially current pain ( $r = 0.510$ ,  $P = .001$ ), mean pain during the past 7 days ( $r = 0.505$ ,  $P = .001$ ), and pain catastrophizing ( $r = 0.518$ ,  $P = .001$ ). Patients with clinically relevant symptoms of CS scored significantly worse on all outcomes compared with persons without relevant symptoms of CS, except on functioning ( $P = .128$ ).

**Conclusions:** Symptoms of CS were significantly associated with psychosocial and cognitive behavioral factors. Patients exhibiting a clinically relevant degree of symptoms of CS scored significantly worse on most outcomes, compared with the subgroup of the sample with fewer symptoms of CS. (*J Manipulative Physiol Ther* 2017;xx:1-10)

**Key Indexing Terms:** *Low Back Pain; Central Nervous System Sensitization; Catastrophizing; Illness Behavior*

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## INTRODUCTION

Chronic nonspecific low back pain (CNLBP) is a major problem today. Up to 84% of the general population experiences an episode of LBP during their life, and for about 90% of them, no specific medical cause can be found.<sup>1-3</sup> Instead, modern pain neuroscience has confirmed that the involvement of impaired central pain modulation or central sensitization (CS) might explain the patients' pain and symptoms.<sup>4,5</sup> Central sensitization can be defined as a process of abnormal and intense enhancement of pain caused by increased neuronal responses to stimuli in the central nervous system.<sup>5-10</sup> This central hyperexcitability is associated with altered sensory processing in the brain,<sup>5,9,11</sup> malfunctioning of endogenous pain inhibitory systems,<sup>5,9,12</sup> increased activity of pain facilitatory pathways,<sup>5,9,11,13</sup> and temporal summation of second pain and/or wind-up,<sup>9,11,13</sup> which leads to dysfunctional endogenous analgesic control.<sup>12</sup> Recent studies have also highlighted the role of an overactive network of brain areas in the CS process.<sup>5,9,14</sup> Increased activity is found to be present in brain areas involved in acute pain sensations (eg, insula, anterior cingulate cortex, and prefrontal cortex), as well as in other brain regions, such as different brain stem nuclei, the dorsolateral frontal cortex, and the parietal associated cortex.<sup>14,15</sup> Furthermore, decreased  $\gamma$ -aminobutyric acid neurotransmission<sup>16</sup> and long-term potentiation of neuronal synapses in the anterior cingulate cortex<sup>17</sup> are contributing to an overactive brain neuromatrix.<sup>9</sup> Besides these top-down mechanisms, different bottom-up mechanisms are also involved in CS, such as peripheral injury and other stressors (eg, infections), triggering the release of proinflammatory cytokines and the activation of spinal cord glia with cyclooxygenase-2 and prostaglandin E2 expression in the central nervous system.<sup>9,18-21</sup> Together, these mechanisms lead to hyperalgesia and allodynia in regions close to and outside the primary pain region.<sup>5,9</sup>

Symptoms of CS are present in a wide range of disorders, for example, fibromyalgia,<sup>22</sup> chronic whiplash-associated disorders,<sup>23</sup> irritable bowel syndrome,<sup>24,25</sup> and chronic fatigue syndrome.<sup>26</sup> However, only a subgroup of CNLBP patients appear to manifest features of CS.<sup>6,8,27-29</sup> Evidence exists that subgroups of CNLBP patients exhibit altered temporal summation, hyperalgesia, and allodynia for electrical, pressure, and heat stimuli in regions outside the primary painful region. Concerning the malfunctioning of descending anti-nociceptive mechanisms in CNLBP patients, conflicting evidence was found.<sup>30,31</sup>

It remains to be established why CS is present only in some patients with CNLBP. Genetic predisposition<sup>4</sup> and the influence of psychosocial and cognitive behavioral factors, such as depressive feelings,<sup>32</sup> pain catastrophizing,<sup>33</sup> and fear avoidance behavior, and more specifically kinesiophobia,<sup>33-35</sup> have been reported in patients with CNLBP. These factors may enhance central hyperexcitability

through the activation of limbic brain regions, which, in turn, contributes to and sustains CS.<sup>15,28,30,36,37</sup>

The relationship between CS and pain catastrophizing or fear avoidance beliefs in patients with CNLBP has not yet been studied. By unraveling which psychosocial and cognitive behavioral factors underlie, cause, or maintain the process of CS in CNLBP patients, more targeted interventions could be integrated into their treatment, which might lead to better outcomes for this population.

The first objective of the present cross-sectional study was to analyze associations between symptoms of CS and pain behavior, functioning, pain intensity, illness perceptions, kinesiophobia, and pain catastrophizing in people with CNLBP. Second, this study aimed to compare the aforementioned outcome measures between patients manifesting and those not manifesting symptoms of CS to a clinically relevant degree.

It was hypothesized that symptoms of CS would be positively correlated with pain, pain behavior, catastrophizing, kinesiophobia, and negative illness perceptions and inversely correlated with functioning. The second hypothesis stated that persons with clinically relevant symptoms of CS have worse psychosocial outcomes related to CNLBP compared with people with fewer of these symptoms.

## METHODS

**Study Design and Setting**

This cross-sectional study was reported in accordance with the Strengthening The Reporting of OBServational studies in Epidemiology statement.<sup>38</sup>

Participants were initially recruited to participate in a randomized, controlled, triple-blind trial in which an experimental treatment was compared with a placebo treatment to examine the effectiveness of a new electrotherapy device for the treatment of patients with CNLBP.<sup>39</sup> The protocol was approved by the University Hospital Brussels (Universitair Ziekenhuis Brussel)/Vrije Universiteit Brussel ethics committee and was registered with [clinicaltrials.gov](http://clinicaltrials.gov) (No. NCT02256410).

The study was conducted at the University Hospital Brussels, Belgium, from November 2013 to February 2015. All participants provided their informed consent prior to inclusion in the study.

The present cross-sectional study comprises a subanalysis of the baseline values of the outcome measures of the initial trial.

**Participants**

Participants, both male and female, were recruited in the pain clinic of the University Hospital Brussels, through an online questionnaire and through flyers and posters in health care centers and pharmacies in Brussels, Belgium. Potential participants were screened during a telephone

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