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ORIGINAL ARTICLE

Central Sensitization and its Association with Pain, Psychosocial Factors, Disability, Functional Muscle Performance, and Depression in Gujarati-Speaking Adults with Chronic Low Back Pain: A Cross-Sectional Study

Dhameliya Pooja Ashokbhai[1], Bid Dibyendunarayan Dhrubaprasad[2]

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ABSTRACT

Background: Chronic low back pain (CLBP) is the leading cause of disability worldwide and is increasingly recognized as a condition characterized by central sensitization (CS). Despite the growing evidence of CS as a multidimensional driver of pain, psychosocial dysfunction, and physical impairment, research on the Indian population, particularly Gujarati-speaking adults, is scarce.

Objective: To investigate the association between CS and pain intensity, fear–avoidance beliefs (FABs), functional disability, back extensor muscle endurance, and depression in Gujarati-speaking adults with CLBP.

Methods: This cross-sectional study included 150 adults (102 females, 48 males; mean age, 42.30 ± 10.24 years) with CLBP from multiple physiotherapy and orthopedic clinics in Surat, Gujarat, India. CS was measured using the Central Sensitization Inventory Gujarati version (CSI-G). Additional instruments included the Numerical Pain Rating Scale (NPRS), Fear-Avoidance Beliefs Questionnaire–Gujarati (FABQ-G), Oswestry Disability Index–Gujarati (ODI-G), Extensor Endurance Test (EET), and Patient Health Questionnaire-9–Gujarati (PHQ-9-G). Pearson's correlation and multiple linear regression analyses were performed using IBM SPSS v20.0.

Results: CS was positively correlated with pain intensity ($r = 0.869$, $p < 0.001$), FABs ($r = 0.840$, $p < 0.001$), disability ($r = 0.825$, $p < 0.001$), and depression ($r = 0.730$, $p < 0.001$) and negatively correlated with back extensor endurance ($r = -0.390$,

AUTHOR'S AFFILIATION:

[1] Private Physiotherapy Practitioner, Surat, Gujarat, India.

[2] Head of the Department of Musculoskeletal Sciences, The Sarvajanik College of Physiotherapy, Rampura, Surat, Gujarat, India.

CORRESPONDING AUTHOR:

Bid Dibyendunarayan Dhrubaprasad, Head of the Department of Musculoskeletal Sciences, The Sarvajanik College of Physiotherapy, Rampura, Surat, Gujarat, India.

E-mail: dnbid71@gmail.com

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$p < 0.001$). Regression analysis identified pain intensity ($\beta = 0.384$), disability ($\beta = 0.248$), depression ($\beta = 0.183$), and fear-avoidance beliefs (FABs) ($\beta = 0.174$) as significant predictors of CS. Extensor endurance was not a significant independent predictor ($p = 0.229$).

Conclusion: CS is very strongly associated with pain, psychosocial distress, and disability in Gujarati-speaking patients with CLBP. CS screening and biopsychosocial assessment should be routine in CLBP management, and interventional research is needed to test targeted treatments.

KEYWORDS

• Central sensitization • Chronic low back pain • Fear-avoidance beliefs • Oswestry Disability Index • Depression • Biopsychosocial model • PHQ-9

INTRODUCTION

Chronic low back pain (CLBP), defined as pain in the lumbar region persisting beyond 12 weeks, is one of the most prevalent musculoskeletal conditions.[1] CLBP is the leading cause of years lived with disability worldwide, affecting all age groups.[2] The lifetime prevalence of low back pain is 84%, with 20% of working adults experiencing chronic forms.[3] In India, a meta-analysis reported a point prevalence of 48% and a lifetime prevalence of 66%, particularly affecting women and rural communities.[4] Mechanistic research on CLBP in Gujarati-speaking adults remains limited, representing a gap in the regional pain science.

The pathophysiology of CLBP has evolved beyond the biomechanical explanations. CS, defined as the increased responsiveness of nociceptive neurons in the CNS to normal or subthreshold inputs, is now considered key to sustaining pain in many patients with CLBP.[5,6] CS results from neuroplastic changes in the CNS, such as enhanced synaptic efficacy in dorsal horn pathways, upregulation of NMDA receptor activity, microglial activation with pro-inflammatory cytokines, and failure of inhibitory control systems.[7] These changes lower pain thresholds, expand receptive fields, and sustain pain without peripheral tissue damage, resulting in hyperalgesia and allodynia.[5]

The clinical consequences of CS extend beyond the amplification of pain across multiple domains.[8] Pain is disproportionate to structural pathology.[9] Psychosocial factors interact with CS mechanisms, and central excitability amplifies threat perception.[10] In patients with CLBP, functional disability reflects pain severity and fear-driven activity restriction.[11] Muscle performance decreases due to deconditioning.[11] Depression shares neurobiological pathways with CS, creating a cycle of pain and distress.[12]

Most research has examined CS's association with individual outcomes in isolation, mainly in Western populations. Studies investigating the relationships between CS, pain, psychosocial factors, disability, muscle performance, and depression in Indian cohorts are lacking. Recently available Gujarati-validated assessment instruments now enable culturally appropriate research on this population.

This study was designed to address these concerns. Using a cross-sectional design and a battery of validated Gujarati-language instruments, this study aimed to quantify CS and characterize its simultaneous association with pain intensity, FABs, functional disability, back extensor muscle endurance, and depression among Gujarati-speaking adults with CLBP.

We hypothesized that CS would be positively associated with pain intensity, fear-avoidance beliefs, functional disability, and depression, and negatively associated with back extensor muscle endurance in Gujarati-speaking adults with CLBP. Furthermore, higher levels of pain, disability, fear-avoidance beliefs, and depression were hypothesized to significantly predict increased central sensitization.

METHODS

Study Design and Setting

This cross-sectional study was conducted over one year at multiple physiotherapy and orthopedic clinics in Surat, Gujarat,

India. The multicenter approach enhanced sample representativeness and minimized selection bias. Ethical approval was granted by the Institutional Ethics Committee of The Sarvajanik College of Physiotherapy (reference: SMT/SCOP/IEC/24-25/683, dated October 16, 2024). Participants provided written informed consent in Gujarati prior to their enrolment.

Participants

Eligible participants were adults aged 20–60 years with a physician-confirmed diagnosis of CLBP for over three months and a pain intensity of 4–10 on the NPRS scale. Participants had to be independently ambulatory and able to perform daily activities without assistance. Consecutive sampling identified eligible patients during routine consultations by the treating clinicians. The exclusion criteria included specific spinal pathologies (neoplasm, fracture, spondylolisthesis, spondylolysis, spinal stenosis, and ankylosing spondylitis), history of spinal surgery, pregnancy, psychiatric medication use, permanent disability, and red flag signs.

Sample Size

The sample size was calculated using G*Power 3.1.9.4 software with a two-tailed correlational model (power = 0.80, α = 0.05, effect size = 0.25), yielding a minimum requirement of 120 participants. The target was increased to 150 to account for potential data loss and support multiple regression analyses, consistent with established practices in chronic pain research.

Outcome Measures

CS was assessed using the CSI-G[13], a 25-item self-report questionnaire scored 0–100 on a five-point Likert scale; scores ≥ 40 indicate clinically significant CS.[14] Pain intensity was measured using the Numerical Pain Rating Scale (NPRS), an 11-point scale ranging from 0 (no pain) to 10 (worst pain imaginable).[15] FABs were evaluated using the FABQ-G,[16] a 15-item instrument scored 0–90, with higher scores indicating stronger avoidance beliefs.[10] FABQ scores were categorized as low (0–48), moderate (49–64), or high (65–90) scores. The FABQ-G comprises 15 items compared to 16 in the original English version of the FABQ[17], as item 8 was removed due to its lack of contextual relevance in Gujarat. Functional disability was quantified using the ODI-G[18] expressed as a percentage (0–100%); scores of 21–40% represented moderate disability.[19] Back extensor muscle endurance was assessed using the EET, in which participants maintained a prone trunk extension position, and the time held (in seconds, maximum 300 s) was recorded.[20] Depression was measured using the PHQ-9-G, a nine-item instrument scored 0–27, with a score of 15–19 indicating moderately severe depression.[21]

Procedure

Data were collected in one session for each participant. After consent and demographic recording, the participants completed self-report questionnaires (CSI-G, NPRS, FABQ-G, ODI-G, and PHQ-9-G) in a quiet environment with standardized instructions. The EET was followed, and the mean of the two trials was noted.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, version 20.0. Normality was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Pearson's correlation coefficient (r) was used to examine bivariate relationships between the CSI-G and all outcome variables, which were interpreted as weak ($r < 0.3$), moderate (0.3–0.5), strong (0.6–0.8), and very strong (> 0.8). Multiple linear regression analysis was used to identify independent predictors of the CSI-G score, with age, sex, and body mass index (BMI) included as covariates. Statistical significance was set at $p < 0.05$.

RESULTS

Participant Characteristics

The participant characteristics are presented in Table 1.

Table 1: Demographic and anthropometric characteristics of the study sample

Characteristic	Value	Notes
Participants, n	150	Clinical sample
Female, n (%)	102 (68.0)	Sex distribution
Male, n (%)	48 (32.0)	Sex distribution
Age, years	42.30 ± 10.24	Mean ± SD
Height, cm	161.83 ± 8.72	Mean ± SD
Weight, kg	70.68 ± 13.47	Mean ± SD
Body mass index, kg/m²	27.07 ± 5.07	Overweight on average

Characteristic	Value	Notes
Age group 20–30 years, n	22	Young adults
Age group 31–45 years, n	70	Largest subgroup
Age group 46–60 years, n	58	Older adults

A total of 150 participants were included in the study, comprising 102 women (68%) and 48 men (32%). The mean age was 42.30 ± 10.24 years, mean height was 161.83 ± 8.72 cm, mean weight was 70.68 ± 13.47 kg, and mean body mass index was 27.07 ± 5.07 kg/m², placing the sample in the overweight category. The age distribution showed the largest subgroup in the middle-aged adult category (31–45 years; n = 70), followed by older adults (46–60 years; n = 58) and young adults (20–30 years; n = 22).

Descriptive Statistics of Clinical Outcome Measures

The mean CSI-G score was 45.98 ± 13.94, indicating moderate CS severity. The mean pain intensity was 6.55 ± 1.02, indicating moderate pain. The mean FABQ-G total score was 57.63 ± 6.51, indicating elevated fear-avoidance beliefs, with the majority of participants classified[17] in the high FABQ category (n = 95), followed by moderate (n = 51), and low (n = 4) scores. The mean ODI-G score was 38.42 ± 7.82%, corresponding to moderate functional disability. Back extensor endurance was markedly reduced (mean EET: 42.52 ± 30.75 s) with substantial heterogeneity across all participants. The depression scores on the PHQ-9-G averaged 16.19 ± 3.40, corresponding to moderately severe depression symptoms.

Prevalence and Distribution of CS

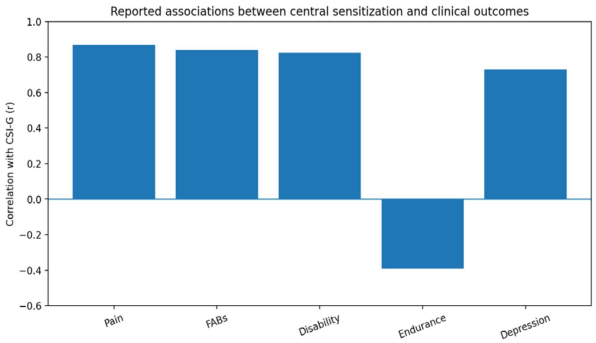
Of the 150 participants, 95 (63.3%) met the CSI-G threshold for clinically significant CS (score of ≥ 40). CS positivity was substantially more prevalent among women (76 of 102, 74.5%) than among men (19 of 48, 39.6%), indicating a notable sex difference in CS distribution within this sample.

Normality Testing

Kolmogorov–Smirnov and Shapiro–Wilk tests revealed non-normal distributions for most study variables ($p < 0.05$). Given the large sample size (n = 150), parametric statistical tests were considered appropriate and applied throughout the study. Pearson's correlation was used for bivariate analyses, and multiple linear regression was used for multivariate analyses.

Correlation Between CS and Outcome Variables

Pearson's correlation analysis revealed significant associations between CS and all outcome variables (Table 2). A very strong positive correlation was observed between the CSI-G and NPRS ($r = 0.869$, $p < 0.001$), indicating that higher CS scores were associated with greater pain intensity. CS was also very strongly correlated with the total FABQ-G ($r = 0.840$, $p < 0.001$) and ODI-G ($r = 0.825$, $p < 0.001$) scores, reflecting strong associations with FABs and functional disability, respectively. A strong positive correlation was found between the CSI-G and PHQ-9-G scores ($r = 0.730$, $p < 0.001$), confirming a substantial association between CS and depression symptoms. A moderate negative correlation was observed between the CSI-G and EET scores ($r = -0.390$, $p < 0.001$), indicating that participants with higher CS scores demonstrated lower back extensor endurance (Table 2) (Graph 1).



Graph 1: Summary of the correlations between the CSI-G scores and clinical outcome measures

Table 2: Pearson's Correlation Between CS (CSI-G) and Clinical Outcome Measures (N = 150)

Variable	r	Interpretation	p-value
Pain Intensity (NPRS)	0.869	Very Strong positive	< 0.001
Fear-Avoidance Beliefs (FABQ-G)	0.840	Very Strong positive	< 0.001
Functional Disability (ODI-G)	0.825	Very Strong positive	< 0.001
Back Extensor Endurance (EET)	−0.390	Moderate negative	< 0.001
Depression (PHQ-9-G)	0.730	Strong positive	< 0.001

Multiple Linear Regression

Multiple linear regression identified four significant independent predictors of the CSI-G score. Pain intensity was the strongest predictor ($B = 5.255$, $\beta = 0.384$, $p < 0.001$),

followed by functional disability (B = 0.442, β = 0.248, p < 0.001), depression (B = 0.749, β = 0.183, p < 0.001), and FABs (B = 0.374, β = 0.174, p = 0.024). Back extensor endurance did not independently predict CS in the multivariate model (B = −0.020, β = −0.044, p = 0.229) (Table 3).

Table 3: Multiple Linear Regression Analysis — Predictors of CS (CSI-G Score) (N = 150)

Predictor	B	SE	β	t	p-value	95% CI
(Constant)	−38.230	5.014	—	−7.625	< 0.001	−48.141 to −28.320
Pain Intensity (NPRS)	5.255	1.100	0.384	4.776	< 0.001	3.080 to 7.429
Fear-Avoidance Beliefs (FABQ-G)	0.374	0.164	0.174	2.276	0.024	0.049 to 0.698
Functional Disability (ODI-G)	0.442	0.108	0.248	4.100	< 0.001	0.229 to 0.655
Back Extensor Endurance (EET)	−0.020	0.017	−0.044	−1.209	0.229	−0.053 to 0.013
Depression (PHQ-9-G)	0.749	0.201	0.183	3.727	< 0.001	0.352 to 1.145
Model Fit	$R^2 = 0.835$		Adjusted $R^2 = 0.826$		F(8, 141) = 89.33, p < 0.001	

Dependent variable: *CSI-G Total. Covariates included age, sex, and BMI. B = unstandardized coefficient, SE = standard error, and β = standardized coefficient. R^2 and Adjusted R^2 were calculated using $\Sigma(\beta_i \times r_i)$; k = 8 (5 predictors + 3 covariates).*

The overall regression model was statistically significant [F(8, 141) = 89.33, p < 0.001], explaining **83.5% of the variance** in the CSI-G scores (R^2 = 0.835). After adjusting for the number of predictors and covariates (age, sex, BMI), the model retained strong explanatory power (Adjusted R^2 = 0.826), indicating minimal overfitting and confirming that pain intensity, functional disability, depression, and fear-avoidance beliefs collectively accounted for a large proportion of the variance in central sensitization severity in this sample.

DISCUSSION

This study is the first large-scale examination of CS associations across five domains: pain intensity, psychosocial factors, functional disability, back extensor muscle endurance, and depression in Gujarati-speaking adults with CLBP. The findings affirm CS as a significant, multidimensional phenomenon in this population and support the biopsychosocial model of chronic pain as an appropriate framework for its assessment and management (Figure 1).

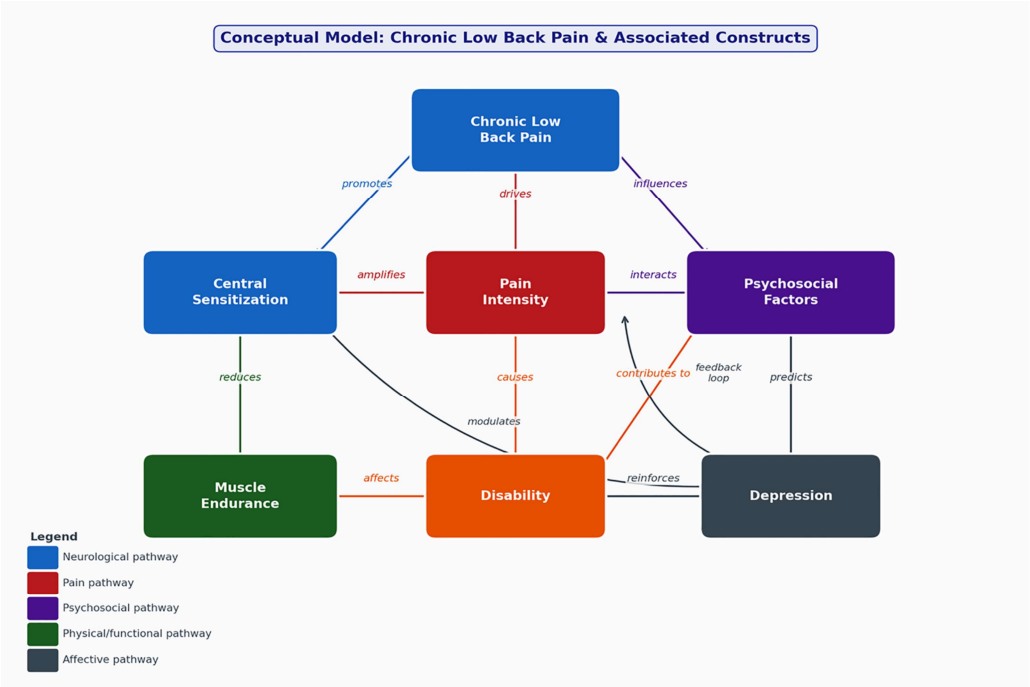
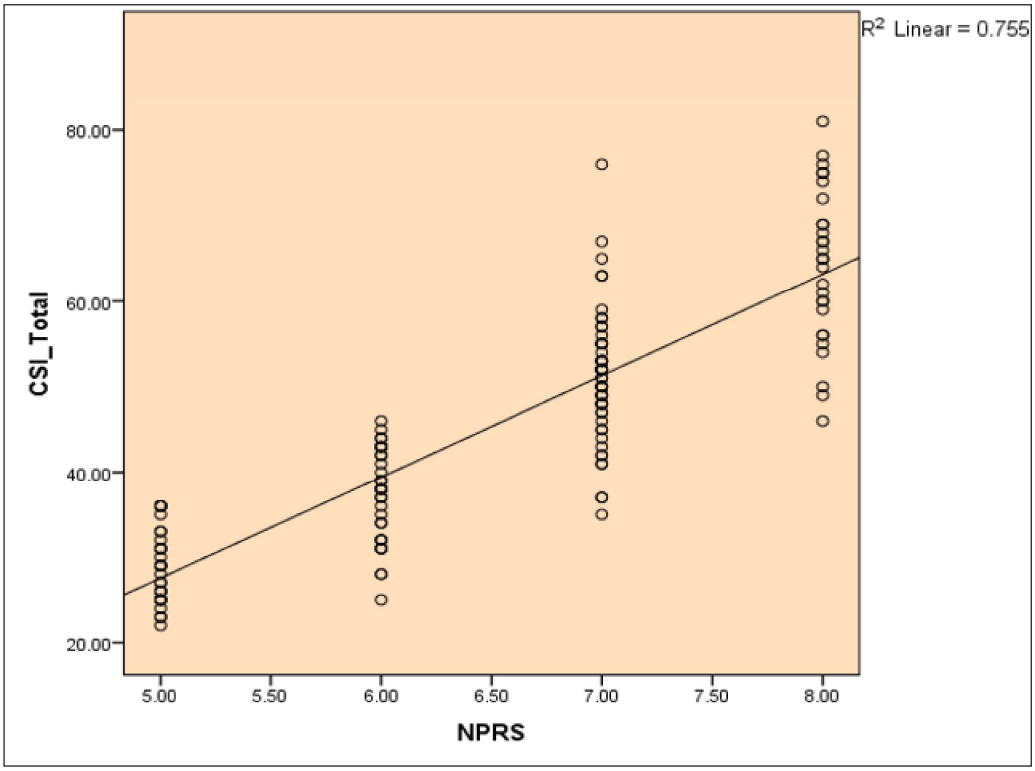


Figure 1: Conceptual model illustrating the observed associations between chronic low back pain, central sensitization, pain intensity, psychosocial factors, disability, muscle endurance, and depression.

CS and Pain Intensity

The strong correlation between CS and pain intensity ($r = 0.869$, $p < 0.001$) (Graph 2) is a key finding that aligns with CS as a primary pain amplifier in CLBP.[5] CS-mediated pain originates from CNS hyperexcitability rather than from peripheral tissue injury. At the neurochemical level, sustained nociceptive input drives long-term potentiation in the spinal cord dorsal horn through NMDA receptor activation, increased glutamate and substance P release, and reduced gamma aminobutyric acid (GABA)-ergic inhibition.[6,7] Microglial activation amplifies these changes via the release of proinflammatory cytokines IL-1β and TNF-α, which lower the nociceptive thresholds and maintain central hyperexcitability.[7]



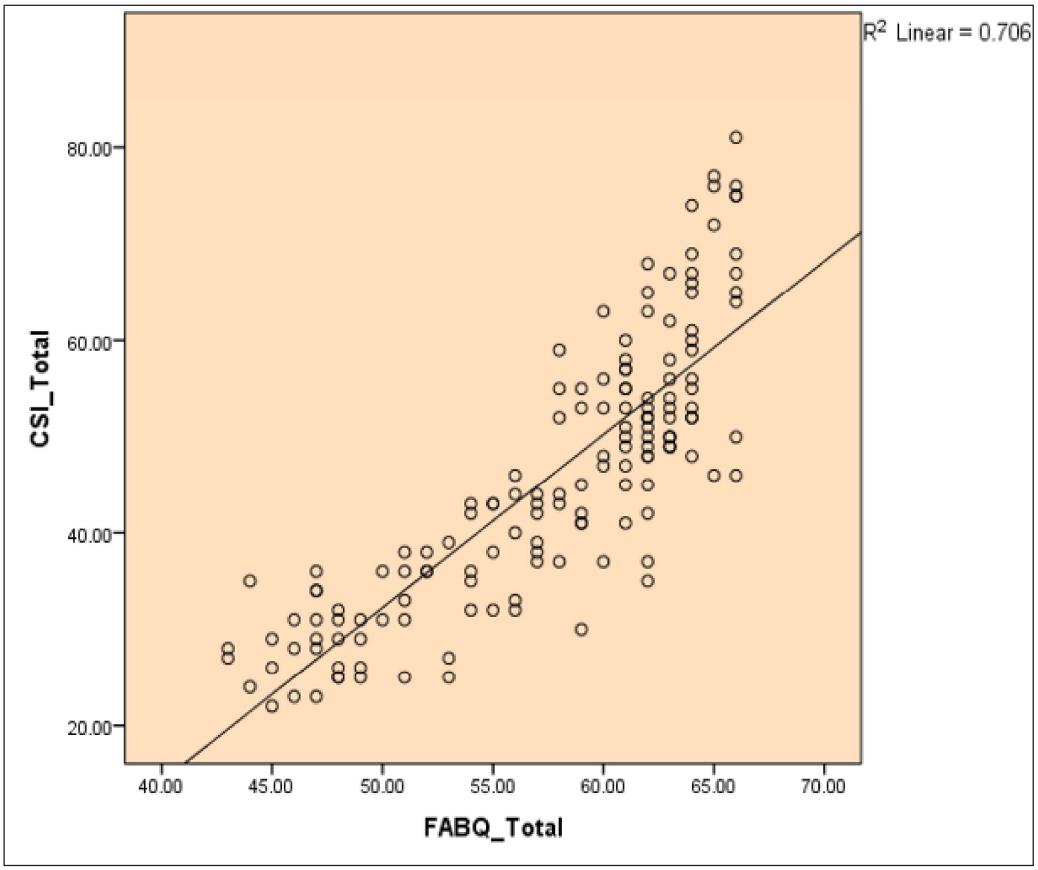
Graph 2: Scatter plot CSI-G vs. NPRS

Once established, CS can maintain pain independently of peripheral input, explaining why many patients with CLBP report severe pain disproportionate to imaging findings.[8] Woolf emphasized that increased excitability and reduced inhibitory control can intensify pain even with modest peripheral input,[5] while Latremoliere and Woolf described central neuroplasticity as generating pain hypersensitivity.[6] Individuals with higher CSI-G scores may experience more intense and persistent pain that is less proportional to tissue findings. Den Bandt *et al.*[22] showed CS-positive patients had higher pain ratings during mechanical stimulation than CS-negative patients. Aoyagi *et al.*[9] identified a CS-positive subgroup of patients with CLBP with elevated pain scores and reduced muscle performance. These findings confirm CS as an active driver of pain in CLBP, accounting for substantial between-patient variance in pain severity.

The pain–CS correlation requires careful interpretation. Strong correlations may reflect overlap between self-reported symptom measures, as the CSI includes symptoms common to chronic pain. This association does not imply that pain intensity and CS are identical; rather, they are related manifestations of shared syndromes. This aligns with Arendt-Nielsen's work showing CS as a multidimensional phenomenon in chronic pain conditions.[23] Regression analysis confirmed pain intensity as the strongest CS score predictor ($\beta = 0.384$, $p < 0.001$), indicating a bidirectional relationship where CS drives pain and vice versa.[22] Clinically, high CLBP pain scores may reflect central mechanisms, necessitating different therapies.

CS and Psychosocial Factors

The strong correlation between CS and FABs ($r = 0.840$, $p < 0.001$) (Graph 3) shows that psychosocial factors are integral to CS pathophysiology and not merely comorbidities. The fear-avoidance model (FAM)[10] described by Vlaeyen and Linton explains that when pain signals threaten, catastrophizing leads to kinesiophobia and activity-avoidance. This avoidance reduces mechanoreceptive and proprioceptive input, which normally aids in descending pain inhibition and maintains central excitability.[11] Reduced physical activity leads to musculoskeletal deconditioning, generating greater nociceptive input during movement and reinforcing fear and catastrophizing.[24]



Graph 3: Scatter plot CSI-G vs. FABQ-Total

The mean FABQ-G total score of 57.63 ± 6.51 suggests a generally high level of fear-avoidance beliefs among participants, with most individuals classified in the high FABQ category (n = 95), followed by moderate (n = 51), and a small proportion in the low category (n = 4), indicating elevated fear-avoidance tendencies within the study population.

Roussel *et al.*[25] found that patients with CLBP and CS showed higher catastrophizing and kinesiophobia than those without CS, indicating that central hyperexcitability amplifies the pain response. The regression model showed that FABs independently predicted CS severity ($\beta = 0.174$, $p = 0.024$), highlighting that interventions targeting maladaptive pain beliefs may provide both neurophysiological and psychological benefits.

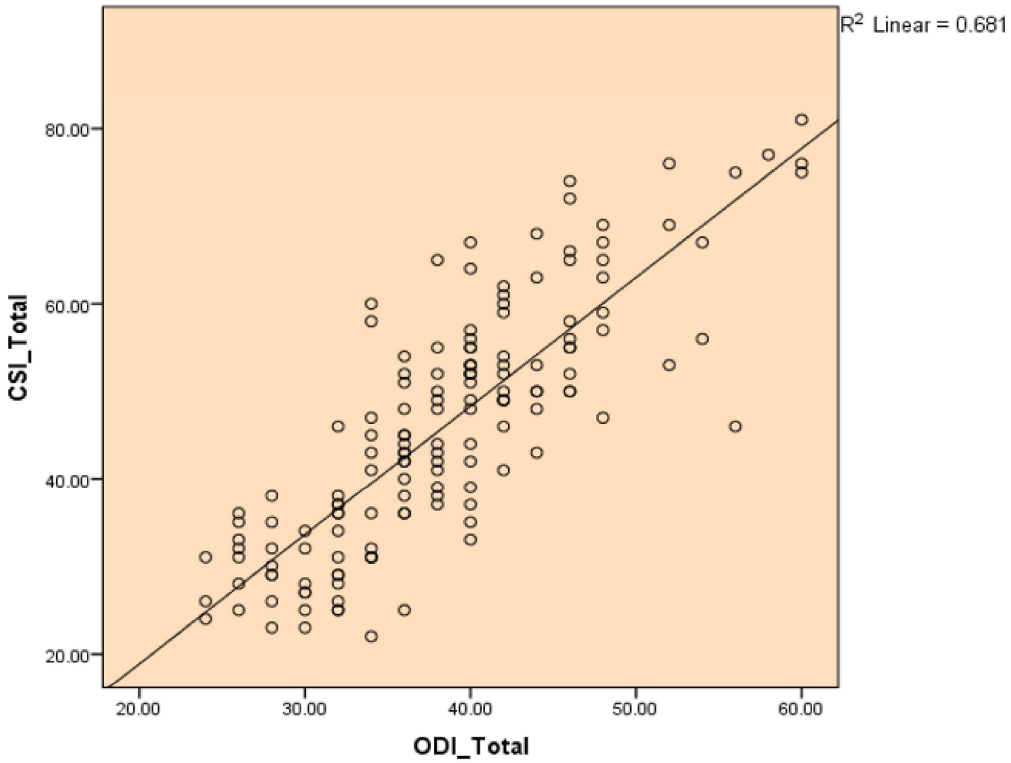
Psychological stress from fear and anxiety dysregulates the HPA axis, causing abnormal cortisol secretion that promotes neuroinflammation and glial activation, maintaining CS.[7] Fear activates brain regions like the amygdala and anterior cingulate cortex, modulating pain control pathways and amplifying nociceptive processing.[8] These interactions explain why psychosocial interventions, particularly cognitive behavioral therapy and pain neuroscience education, reduce FABs and CS-related pain and should be part of standard physiotherapy for CS-positive CLBP.[26]

The regression model showed that FABs independently predicted CS severity (β = 0.174, p = 0.024), suggesting that targeting maladaptive pain beliefs could offer neurophysiological and psychological benefits. Psychological stress from fear and anxiety dysregulates the HPA axis, causing abnormal cortisol secretion, promoting neuroinflammation and glial activation, maintaining CS.[7] Fear activates brain regions like the amygdala and anterior cingulate cortex, modulating descending pain control pathways and amplifying nociceptive processing.[23] These interactions support including psychosocial interventions, particularly CBT and pain neuroscience education (PNE), as standard physiotherapy components for CS-positive CLBP.[27]

CS and Functional Disability

The very strong positive correlation between CS and functional disability ($r = 0.825$, $p < 0.001$) (Graph 4) confirms that CS contributes substantially to activity limitations in patients with CLBP, independent of pain severity alone.[28] Functional disability in this sample was moderate (mean ODI-G: 38.42 ± 7.82%), encompassing limitations in personal care, lifting, walking, sitting, standing, sleeping, and occupational participation. The International Classification of Functioning, Disability, and Health (ICF) framework recognizes disability as a product of physiological impairments, psychological responses, and social contextual factors, all of which are engaged by CS.[28]

CS impairs functional capacity through various mechanisms. Central hyperexcitability lowers the pain threshold during physical tasks like sustained postures and lifting.[8] CS causes motor cortex reorganization, showing altered postural control and muscle activation in patients with CLBPs.[11] These changes lead to inefficient movement and increased musculoskeletal strain. Initially adaptive protective strategies become maladaptive when chronic, restricting function and causing deconditioning.



Graph 4: Scatter plot CSI-G vs. ODI-G

CS impairs functional capacity through multiple mechanisms. Central hyperexcitability lowers the pain threshold during tasks such as sustained postures and lifting.[23] CS-related motor cortex reorganization alters postural control and muscle activation patterns[11], leading to inefficient movements and increased strain. Initially, adaptive protective strategies, such as muscle guarding, become maladaptive when chronic, restricting function and causing

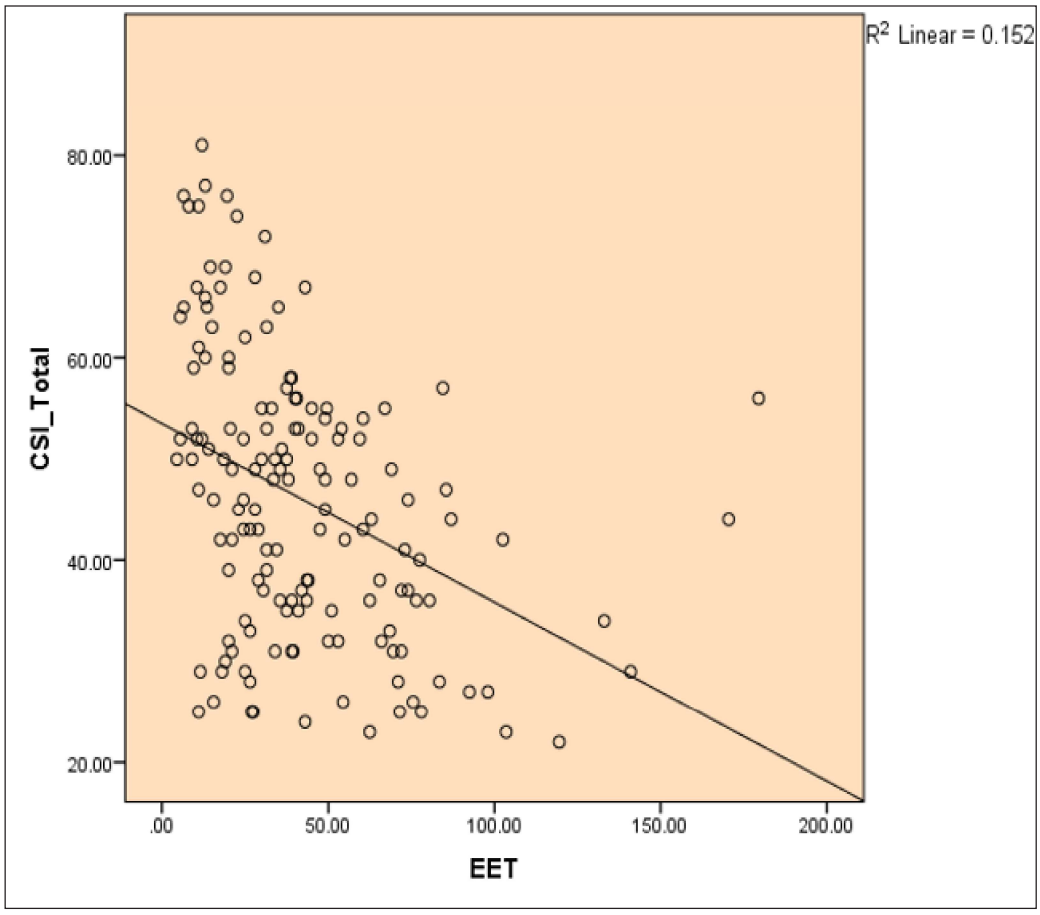
deconditioning. Tagliaferri *et al.* emphasized evaluating CLBP across multiple domains, including function, emotional well-being, and treatment response, highlighting the need to assess beyond pain scores alone.[29]

Arendt-Nielsen *et al.* found that CS affects functional impairment beyond pain intensity in chronic pain conditions.[23] The regression model showed that disability was the second strongest predictor of CS ($\beta = 0.248$, $p < 0.001$), and the link was not solely explained by pain intensity. This bidirectional relationship, in which fear, inactivity, altered motor patterns, and reduced movement confidence perpetuate CS and disability, suggests that rehabilitation must target central pain processing and its physical effects. Graded motor imagery, activity pacing, and motor control retraining address these needs.[26,30,31]

CS and Back Extensor Muscle Endurance

Back extensor endurance in this sample was markedly reduced (mean EET: 42.52 ± 30.75 s) relative to the normative values of 60–120 s reported in pain-free adults, confirming significant muscle performance deficits in this CLBP population. The moderate negative correlation between CS and EET ($r = -0.390$, $p < 0.001$) (Graph 5) indicates that higher CS levels are associated with poorer back extensor endurance, which is consistent with the concept that CS disrupts normal neuromuscular function through altered motor cortical representation and pain-related inhibition of muscle activation.

Hodges and Moseley[11] demonstrated that LBP delays transversus abdominis and multifidus activation during limb movements, indicating a shift toward superficial muscle strategies. This compromises spinal stability, increases tissue loading, and reduces EET performance. Research has shown that changes in cortical representation correlate with pain chronicity in patients with back pain, impairing the stabilization of muscle activation. CS-mediated pain amplification during trunk extension may cause early EET termination, leading to low mean values and wide standard deviations among participants.



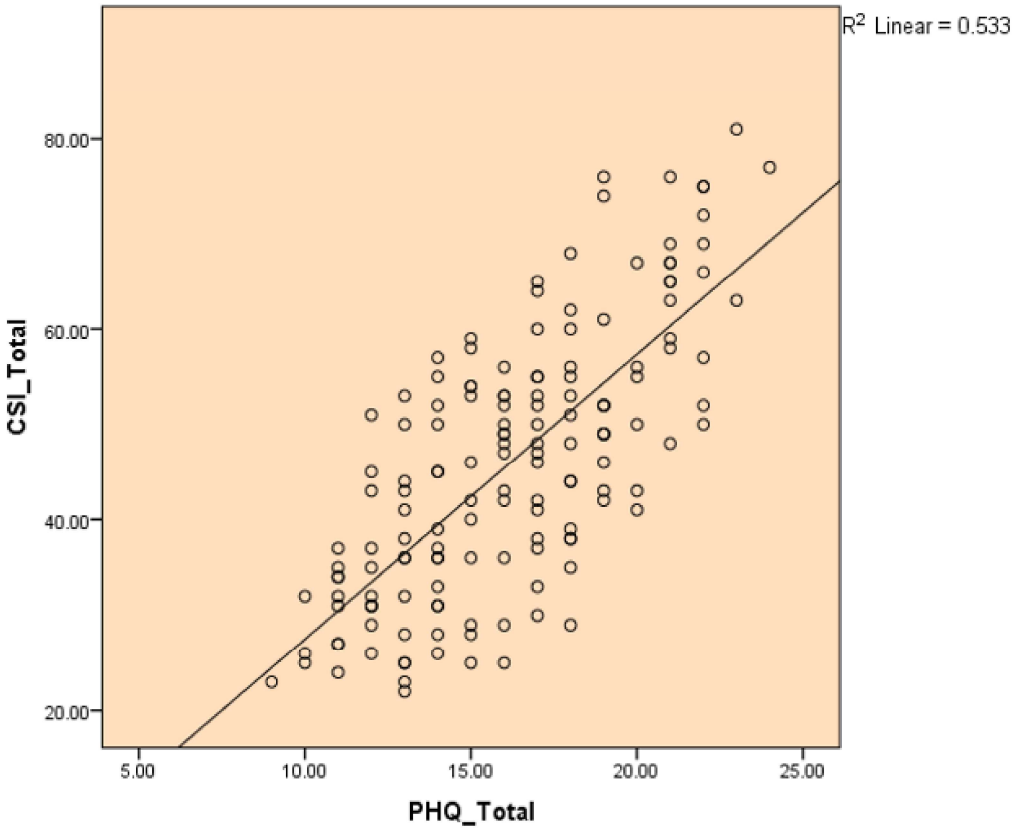
Graph 5: Scatter plot CSI-G vs. EET

Hodges and Moseley showed LBP delays transversus abdominis and multifidus activation during limb movements, shifting from deep-to-superficial muscle strategies, compromising spinal stability, and increasing tissue loading.[11] Cortical representation changes with pain chronicity impair stabilizing muscle activation, and CS-mediated pain amplification during trunk extension may cause early EET termination in patients with CLBPs. The wide standard deviation for endurance values (±30.75 s) suggests participant heterogeneity, likely due to occupation, activity level, symptom duration, coping style, and prior rehabilitation, highlighting the need for individualized functional assessments.

EET was not a significant predictor of CSI-G in the multivariate regression model (β = −0.044, p = 0.229) due to shared variance with pain intensity and disability. This indicates that reduced endurance stems from CS as a chronic pain outcome. Muscle performance is vital for CLBP rehabilitation and recovery. Exercise should retrain deep trunk stabilizers, progress to functional integration with biofeedback, and include pain education and activity pacing to restore movement confidence and reduce pain-related threat perception.[26,27]

CS and Depression

The strong positive correlation between CS and depression (r = 0.730, p < 0.001) (Graph 6) is consistent with the substantial literature documenting the co-occurrence and neurobiological interdependence of chronic pain and mood disorders.[12] The prevalence of depression in CLBP populations is estimated to be 30–50%, far exceeding general population rates.[12] In this sample, the mean PHQ-9-G score of 16.19 ± 3.40 indicated moderately severe depressive symptoms, confirming the clinical significance of this comorbidity.



Graph 6: Scatter plot CSI-G vs. PHQ-9-G

The CS–depression relationship is bidirectional and involves shared neurochemical mechanisms. Both conditions involve the dysregulation of serotonergic and noradrenergic pathways, impairing nociceptive inhibition. Reduced serotonin and norepinephrine levels in depression impair inhibitory control, facilitating CS.[12] HPA axis dysregulation promotes neuroinflammation and central hyperexcitability in the brain. Changes in the prefrontal cortex,

anterior cingulate cortex, insula, and amygdala provide neural substrates for pain amplification and emotional distress.

Bair *et al.* found that pain and depression are often comorBid amplifying each other through shared pathways.[12] Kroenke *et al.* showed a reciprocal relationship in which baseline pain predicted later depression and vice versa.[21] Depression is an independent CS predictor ($\beta = 0.183$, $p < 0.001$), indicating that untreated depression may perpetuate central pain amplification. These findings support the integration of PHQ-9-G screening into routine CLBP assessment and highlight the need for multidisciplinary management combining physiotherapy, psychology, and medical care for CS–depression comorbidity.

Population-Specific Considerations

This study presents the first multi-domain examination of CS in Gujarati-speaking patients with CLBP, filling a gap in Indian pain research. The mean CSI-G score of 45.98 matches international CS levels, as Scerbo *et al.*[32] reported CSI scores of 40–55 in chronic pain populations, indicating similar CS mechanisms across cultures. Neblett *et al.*[33] established CS severity thresholds across populations, supporting their cross-cultural applicability. The use of Gujarati-validated instruments (CSI-G, FABQ-G, ODI-G, and PHQ-9-G) ensured cultural and linguistic accuracy, minimized misunderstandings, and improved response reliability, especially for sensitive psychological and functional disclosures. These findings are directly applicable to clinical practice in Western India.

The predominance of females (68%) aligns with the literature showing that women report CLBP and seek healthcare more than men. While sex-specific differences were not tested and should not be overinterpreted, this pattern requires further study. The mean BMI of 27.07 kg/m² (overweight) indicates obesity's role as a CLBP risk factor, with adipose tissue producing proinflammatory adipokines that may facilitate CS.[7] The predominance of middle-aged adults (31–45 years) highlights the socioeconomic impact of CS-related disability and the importance of early intervention.

The prevalence of clinically significant CS was 63.3%. This estimate requires caution, as the sample was from treatment-seeking settings and the CSI is a screening tool, not a definitive tool. The CSI-G indicates a CS-related symptom burden, not a direct physiological test. Nonetheless, many patients with chronic low back pain may exhibit symptom profiles consistent with central sensitization, supporting the need for clinicians to consider CS when pain is disproportionate, persistent, or accompanied by widespread symptoms and emotional distress.[17,33]

Clinical Implications

These findings have direct implications for physiotherapy practice. CS screening using the CSI-G (cut-off ≥40) should be incorporated into CLBP assessment protocols.[33] Identifying CS-positive patients enables mechanism-informed care. Pain neuroscience education, which views pain as CNS processing rather than tissue damage, reduces FABs, catastrophizing, and pain intensity in patients with CLBP.[34,31] Graded exposure therapy reintroduces feared activities and disrupts fear avoidance.[10] Motor control retraining with cardiovascular exercise addresses deconditioning while stimulating endorphin release.[26] CBT modifies pain beliefs and improves patient function.[24] For depression-CS overlap, multidisciplinary management combining physiotherapy, psychology, and medical care offers superior outcomes.

Patients with high pain, fear-avoidance, disability, and depression are likely to have CS profiles, where tissue-based explanations and passive modalities are insufficient to explain symptoms. Exercise should enhance movement confidence, endurance, and function, and reduce threat perception. A mechanism-informed, patient-centered approach aligns with contemporary pain rehabilitation principles and addresses this cohort's multidimensional burden.[35,36]

STRENGTHS AND LIMITATIONS

The assessment of five outcome domains using validated Gujarati instruments provided comprehensive CS associations that were previously unavailable for this population. Recruitment across Surat clinics enhanced sample diversity. This study addresses the gap in pain research in the Gujarati-speaking Indian population.

This study has some limitations. Sampling from clinical settings in one city may limit generalizability. A high FABs may indicate a sampling bias. Self-reported outcomes may have been biased. CS was assessed behaviorally without QST. The female-dominant sample (68%) limited gender-specific findings. Key confounders, such as occupational demand, physical activity, sleep, and medication use, were not controlled.

CONCLUSION

CS is clinically significant in Gujarati-speaking adults with CLBP, demonstrating strong associations with pain intensity, fear-avoidance beliefs, functional disability, and depression, and a moderate inverse relationship with back extensor muscle endurance. Pain intensity was the strongest independent predictor of CS severity, followed by disability, depression, and fear avoidance beliefs. Back extensor endurance did not independently predict CS when other variables were controlled.

These findings reinforce the biopsychosocial model of CLBP, in which CS is strongly associated with cognitive-behavioral responses, physical dysfunction, and emotional distress in a self-reinforcing cycle of pain.

Future research should prioritize longitudinal and interventional designs, incorporate QST, and expand across diverse Indian subpopulations to build an evidence base that is long overdue in the world's most populous nation.

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