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Research Paper

Brain Wave Patterns in Patients With Chronic Low Back Pain: A Case-control Study

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ABSTRACT

Introduction: Research evidence indicates that maladaptive reorganization of the brain plays a critical role in amplifying pain experiences and pain chronification; however, no clear evidence of change exists in brain wave activity among patients with chronic low back pain (CLBP). The objective of this study was to assess brain wave activity in patients with CLBP, compared to healthy controls.

Methods: Twenty-five patients with CLBP and twenty-four healthy controls participated in the study. A quantitative electroencephalography device was used to assess brain wave activity in eyes-open and eyes-closed (EO and EC) conditions. The regional absolute and relative power of brain waves were compared between the groups.

Results: Our results showed a significant increase in the absolute power of theta (F=5.905, P=0.019), alpha (F=5.404, P=0.024) waves in patients with CLBP compared to healthy subjects in both EC and EO conditions. Patients with CLBP showed a reduced delta absolute power in the frontal region (F=5.852, P=0.019) and augmented delta absolute power in the central region (F=5.597, P=0.022) in the EO condition. An increased delta absolute power was observed in the frontal (F=7.563 P=0.008), central (F=10.430, P=0.002), and parietal (F=4.596, P=0.037) regions in patients with CLBP compared to the healthy subjects in the EC condition. In the EC condition, significant increases in theta relative power (F=4.680, P=0.036) in the parietal region were also found in patients with CLBP.

Conclusion: The increased absolute power of brain waves in people with CLBP may indicate cortical overactivity and changes in the pain processing mechanisms in these patients.

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Highlights

- Chronic low back pain (CLBP) increases the alpha, theta, and delta power in the brain.
- CLBP is associated with increased brain wave activity in the frontal, central, and parietal regions.
- Our findings suggest altered central pain processing in CLBP.

Plain Language Summary

Traditional diagnosis and treatment of CLBP are mainly focused on peripheral pathology. But, the modern neuroscience approach to pain highlights the role of cortical plasticity in chronic musculoskeletal pain. In this regard, several studies found structural and functional changes in the brain in patients with chronic pain. Detailed knowledge about cortical changes in CLBP can improve our understanding of mechanisms involved in CLBP, opening a new window to better treatment of LBP (Low back pain). This study investigated brain wave activity in patients with CLBP compared to healthy individuals. Our findings suggest increased brain activity in various parts of the brain in patients with chronic LBP. This finding indicates that CLBP treatment should focus on both peripheral and cortical factors rather than local tissue damage.

1. Introduction

Low back pain (LBP) is the most common chronic pain, affecting 70%–85% of the adult population at some point in life (Becker et al., 2010). About 10%-30% of people with LBP develop chronic pain, leading to early retirement and high healthcare costs (Becker et al., 2010; Johannes et al., 2010). Treatment of CLBP comprises rest, pharmacological treatment, and physiotherapy, which are usually effective only in the short-term (Qaseem et al., 2017; Saragiotto et al., 2016). The increasing worldwide prevalence of CLBP and inadequate long-term outcomes of current treatment urged a reassessment of the traditional approaches for CLBP treatment.

Traditional diagnosis and treatment of CLBP mainly focus on peripheral pathology and mechanical abnormalities (Ferreira et al., 2006); however, the modern neuroscience approach to pain highlights the role of cortical plasticity in pain chronification and amplification (Fregni et al., 2007). From a neuroscience perspective, maladaptive reorganization of the brain neural networks plays a critical role in chronic pain conditions (Apkarian, 2011; De Vries et al., 2013). In this regard, neuroimaging studies revealed structural and functional changes in the brain in chronic musculoskeletal pain conditions, supporting the neuroscience approach to chronic pain (Kregel et al., 2015). For instance, changes in the brain's EEG activity were also found in individuals with neuropathic pain and fibromyalgia (González Roldán et al., 2013; Sarnthein et al., 2006). Similarly, researchers reported changes in the brain morphology and function in patients with CLBP as compared to pain-free controls (Apkarian et al., 2004; Giesecke et al., 2004; Schouppe et al., 2019). Employing magnetic resonance imaging (MRI), magnetoencephalography (MEG), and transcranial magnetic stimulation (TMS), previous studies on patients with CLBP have evidenced a significant reduction in gray matter density in prefrontal and thalamic regions (Apkarian et al., 2004) as well as alterations in primary somatosensory (Kregel et al., 2015) and motor cortex representation of the back (Tsao et al., 2008).

Previous studies investigated cortical changes in people with chronic pain, using TMS, magnetoencephalography (MEG), functional MRI, positron emission tomography (PET), and electroencephalography (EEG) (Apkarian et al., 2005; Schabrun et al., 2014). Recently, quantitative electroencephalography (qEEG) has also been considered by researchers, as it provides reliable information about brain function during rest, sensory stimulation, and cognitive tasks. In addition, this method is safe, inexpensive, and non-invasive, making it a valuable apparatus for clinical practice and research applications (De Vries et al., 2013; Spronk et al., 2011). Nevertheless, despite the wide applicability of qEEG to evaluate cortical changes in chronic musculoskeletal conditions, relatively little knowledge exists on brain wave activity changes in CLBP patients.

Brain waves are patterns of rhythmic or repetitive neural activity produced by synchronized oscillations of a group of neurons communicating with each other (Kropotov, 2010). According to frequency, brain waves are classified into delta, theta, alpha, beta, and gamma waves (Jatoi & Kamel, 2018). Delta wave (1–4 Hz) is associated with deep sleep and a very relaxed state of mind (Guyton, 2011). A study showed delta activity is related to sustained pain and homeostatic processes (Knyazev, 2012). Theta wave (4-8 Hz) is involved in emotions, focused concentration, and mental tasks (Tatum et al., 2008). A review showed an increased theta activity in chronic pain patients (Pinheiro et al., 2016). Alpha wave (8–12 Hz) represents a state of wakeful rest (Jatoi & Kamel, 2018). Research evidence shows that alpha wave is involved in basic cognitive processes and momentary memory storage, and inhibitory processes (Kropotov, 2010; Klimesch et al., 2007). Moreover, a review study showed increased alpha power in chronic pain patients (Pinheiro et al., 2016). Beta wave (12-30 Hz) is related to active thinking, concentration, and computation (Jatoi & Kamel, 2018). Previous studies reported altered beta power in patients with chronic pain (Ploner et al., 2017).

To our knowledge, quantitative brain activity in chronic back pain has been investigated so far by Schmidt et al., who found no significant difference in brain activity between people with chronic back pain and their healthy counterparts. However, the investigation of brain activity in Schmidt et al's study was limited to eyes-closed condition and parieto-occipital region (Schmidt et al., 2012). Therefore, we aimed to compare brain waves in people with CLBP and healthy subjects across three cortical regions in both eyes-open and eyes-closed (EO and EC) conditions. Prior studies suggest that chronic pain conditions are associated with increased brain wave activity (Pinheiro et al., 2016; Olesen et al., 2011). Thus, we hypothesized that CLBP is associated with increased brain wave activity compared to healthy individuals.

2. Materials and Methods

This case-control study involved 25 participants (37.12±5.86 years, 27.15±2.7 kg/m^2), diagnosed with CLBP, and 24 healthy participants (34.45±4.01 years, 28.64±3.69 kg/m^2), with no history of chronic pain. The number of participants was calculated using our preliminary data on 10 subjects in each group (β=0.20 and α=0.05) for between-group differences in the regional theta power as the most-noticed brain wave abnormality in patients with chronic pain (Ploner et al., 2017). Based on this analysis, a minimum of nineteen participants per group was required.

For CLBP patients, the inclusion criteria were a history of persistent LBP for more than six months or recurring LBP for at least three incidents in the last year requiring activity limitation or medical intervention (Gombatto et al., 2013); the age of 30-50 years; and average pain severity of greater than 30 on a 0-100 mm visual analogue scale (VAS) in the last week. The healthy subjects were included if they had no history of chronic pain and were aged 30-50 years.

The exclusion criteria included the use of medications affecting brain wave activity, alcoholism and addiction, a history of inflammatory joint disease, systemic metabolic disorder, and/or neurological disorders, such as headache, seizure, polyneuropathy and psychological disorders, spondylolysis and spondylolisthesis, spinal surgery, trauma to low back, disc herniation based on an MRI report, pregnancy, and physiotherapy in the last six months.

Data collection

In this study, the regional brain wave activity in both EO and EC (EO, EC) conditions, pain intensity, and functional ability were assessed.

Brain wave activity

The absolute and relative power of brain wave activity was recorded by quantitative Electroencephalograph (qEEG: Mitsar-EEG-201, Mitsar Co. Ltd, Saint-Petersburg, Russia) in both EO, and EC conditions. QEEG is a method based on the quantification of brain wave amplitudes and is designed to process EEG signals (Kropotov, 2010). The absolute power index measures the energy of the electrodes in a certain region of the brain in different frequency bands in microvolt sq (Cunha et al., 2004). Relative power is power in each band, relative to the total frequency bands (Kropotov, 2010).

For the qEEG recording, the participants sat on a reclining chair in a semi-dark room, with 19 electrodes placed on their scalps, according to the international 10-20 system (Jasper, 1958). The reference electrodes were placed on the earlobes. The maximum electrode impedance was 5 K ohms. The low and high cut filters were set at 0.53 and 30 Hz, respectively, and the notch filter was set at 45-55 Hz. The sampling rate was 500 per second. Participants were asked to remain still during the recordings.

All EEG recordings were obtained between 8 AM to 12 PM to avoid sleepiness. To prevent caffeine-induced theta decrease, the participants refrained from caffeinated drinks on the day of the qEEG recording (Landolt

et al., 2004). In the EO condition, the subjects were requested to keep EO and maintain their gaze on a fixation mark on the wall. In the EC condition, participants were instructed to close their eyes but not to sleep.

QEEG frequency bands include delta (1.0-4.0 Hz), theta (4.0-8.0 Hz), alpha (8.0-12.0 Hz), and beta (12.0-25.0 Hz). The EEG data collection was done for five minutes per EO and EC conditions. The raw file of the EEG data was then sent to the Neuroguide software system for analysis and removal of artifacts (Kropotov, 2010). The EEG recordings were also visually inspected for artifact deletion. Artifact deletion was done based on eye movements, muscle movements, and drowsiness using the artifact rejection toolbox of the neuroguide system. Next, the artifact-free epochs were chosen for spectral analysis by the same software system, which uses spectral analysis to decompose the EEG pattern into simple components (sinusoidal waves) using fast fourier transformation (FFT), a method for converting brain wave data from the time domain to the frequency domain so that absolute and relative power indices are created (Kropotov, 2010). The values obtained from FFT absolute and relative power were entered into the SPSS statistical software for analysis. The average absolute and relative power of each frequency band was calculated for brain regions relevant to pain processing, i.e. the frontal (Fp1, Fp2, F3, F4, F7, F8, Fz), central (C3, C 4, C z), and parietal (P3, P4, Pz) regions.

Pain intensity

Pain intensity was assessed with a 100 mm (visual analog scale) VAS, with 0 for a painless state and 100 for the worst imaginable pain. The participants were requested to rate their mean pain intensity in the previous week (Carlsson, 1983).

Oswestry disability index

The Oswestry disability index questionnaire was used to rate participants' functional ability (Mousavi et al., 2006). This questionnaire consists of 10 six-item sections with a range score of 0%-50%. The disability score of 0%-20% indicates minimal disability, 21%-40% moderate disability, 41%-60% severe disability, 61%-80% crippled, and 81%-100% bed-ridden, with worsening symptoms (Fairbank & Pynsent, 2000).

Statistical analysis

First, the normality of data was assessed by the K-S test. Mixed ANOVA tests (3 regions×2 conditions×groups) were used to compare differences in the regional absolute and relative power between the groups in both conditions. A univariate test was used to contrast differences in the regional absolute and relative power of the brain waves between the groups when the main effect of the group was significant. The level of significance was set at $P<0.05$ and the confidence interval was considered as 95%CI for all analyses. The data were analyzed in SPSS software, version 22 (IBM, Chicago, IL).

3. Results

No significant difference was observed between the groups for demographic characteristics of age and BMI ($P>0.05$). Participants with CLBP had a mean pain intensity of 48±11.45 and a disability score of 27.52±9.4/100 based on VAS and the Oswestry disability index, respectively. The participants in the control group had an Oswestry score of zero.

Table 1 and 2 present the Mean±SE of the absolute and relative power of brain waves for healthy controls and CLBP patients in both conditions. Tables 3 and 4 present the absolute and relative power of brain waves in the frontal, central, and parietal regions in both EO and EC conditions. Three-way interaction was not significant for the absolute power of theta, alpha, and beta waves and the relative power of the delta and alpha waves. In other cases, the three-way interaction was significant.

A significant increase was observed in the regional absolute power of theta ($F=5.905$, $P=0.019$), (mean difference=6.86, 95% CI (1.18, 12.55) and alpha ($F=5.404$, $P=0.024$), (mean difference=13.97, 95% CI (1.82, 26.07) waves in patients with CLBP in both condition (Table 3). In the EO condition, patients with CLBP showed a reduced delta absolute power in the frontal region ($F=5.852$, $P=0.019$) and augmented delta absolute power in the central region ($F=5.597$, $P=0.022$) (Table 5). In the EC condition, significant increases were found in the regional absolute power of delta in the frontal ($F=7.563$, $P=0.008$), central ($F=10.430$, $P=0.002$), and parietal ($F=4.596$, $P=0.037$) regions in patients with CLBP compared to the healthy subjects (Table 5).

Significant increases in the relative power of theta wave ($F=4.680$, $P=0.036$) in the parietal region were also found in patients with CLBP in the EC condition (Table 6). We detected no significant differences between the groups in the regional absolute, and relative power of the beta wave ($P>0.05$) (Tables 3 and 6).

Table 1. Mean±SE of the absolute power of brain waves for CLBP patients and healthy controls in both conditions

Healthy Controls in Both Conditions							
Eye Condition	Brain Waves	Mean±SE					
		CLBP Group			Healthy Control		
		Frontal	Central	Parietal	Frontal	Central	Parietal
Eyes open	Delta	16.95±1.22	17.91±1.15	17.3±1.29	21.18±1.24	14.02±1.17	13.63±1.32
	Theta	10.98±1.76	14.13±2.48	13.34±2.68	7.29±0.49	7.96±0.59	7.03±0.60
	Alpha	10.08±1.91	20.03±4.46	34.17±8.26	5.57±0.54	8.69±0.96	12.88±2.34
	Beta	7.65±0.84	9.84±1.68	11.10±2.01	5.94±0.39	6.70±0.49	7.16±0.55
Eyes closed	Delta	17.28±0.81	16.49±0.86	16.09±1.08	14.06±0.83	12.5±0.88	12.77±1.10
	Theta	12.89±1.79	18.98±3.77	21.06±5.31	8.41±0.54	9.61±0.73	9.87±1.05
	Alpha	22.92±3.80	36.45±6.45	66.88±14.52	15.04±1.67	21.23±2.45	43.26±7.01
	Beta	7.54±0.83	12.25±2.07	15.54±2.78	6.45±0.43	9.14±0.75	12.35±1.10

CLBP: Chronic low back pain; SE: Standard error.

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4. Discussion

In this study, we compared regional brain wave activity in CLBP patients with healthy subjects. Our findings showed significant increases in the regional absolute power of brain waves in patients with CLBP compared to their healthy counterparts. Cortical overactivity in patients with CLBP may indicate altered pain processing mechanisms in CLBP when compared to healthy individuals.

This study detected increased regional brain activity of the delta and theta frequency bands in patients with CLBP. These findings suggest increased brain oscillations of the regions tested, indicating cortical neuroplastic changes in patients with chronic LBP. Consistent

with our findings, an extensive shift in brain organization and morphology was observed in patients with CLBP in neuroimaging studies using TMS, MRI, and

Table 2. Mean±SE of the relative power of brain waves for CLBP patients and healthy controls in both conditions

Eye Condition	Brain Waves	Mean±SE					
		CLBP Group			Healthy Control		
		Frontal	Central	Parietal	Frontal	Central	Parietal
Eyes open	Delta	41.81±2.22	34.22±2.75	30.35±2.95	44.60±1.65	36.34±1.44	34.06±1.66
	Theta	19.75±1.04	21.18±1.30	17.09±1.52	17.91±0.70	19.43±0.67	16.61±0.41
	Alpha	17.06±1.63	25.19±2.50	34.30±3.46	13.99±0.98	20.86±1.42	26.72±2.31
	Beta	15.14±0.85	15.77±1.18	15.40±1.15	15.12±0.75	17.03±0.94	17.93±0.93
Eyes Closed	Delta	32.17±2.39	25.32±2.28	21.08±2.38	32.31±1.67	24.68±1.50	19.10±1.60
	Theta	19.62±1.31	21.32±2.07	18.52±2.52	17.75±0.72	17.80±0.92	12.77±0.68
	Alpha	32.67±2.64	40.30±4.01	53.28±8.54	30.40±2.23	35.87±2.32	48.35±2.79
	Beta	12.84±0.87	15.51±1.45	15.98±1.72	13.93±0.65	16.96±0.76	16.12±1.01

CLBP: Chronic low back pain; SE: Standard error.

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Table 3. Analysis of variance for absolute power of brain waves at brain regions of frontal, central and parietal in eye-closed and eye-open conditions for healthy controls and patients with CLBP

Main Effect			Theta (uV2)	Alpha (uV2)	Beta (uV2)
Main effect	Group	F	5.905	5.404	2.336
		P	0.019*	0.024*	0.133
	Eye condition	F	7.549	38.132	40.162
		P	0.008*	0.0001*>	0.0001*>
	Region	F	5.035	22.050	22.624
		P	0.008*	0.0001*>	0.0001*>
Interaction	Eye condition group**	P	0.231	0.267	0.554
	Region group***	P	0.066	0.129	0.260

**Shows the interaction of absolute power for group and eye condition,

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***Shows the interaction of absolute power for group and brain region.

voxel-based morphometry (VBM) (Apkarian et al., 2004; Tsao et al., 2008). For instance, previous studies found changes in trunk muscle representation at the motor cortex (Tsao et al., 2008), and reduced neocortical gray matter volume (Apkarian et al., 2004) in patients with CLBP. Extensive brain activity changes in people with CLBP can be explained based on the neuromatrix theory (Melzack, 1999). According to the neuromatrix theory, pain is produced by specific "neurosignature" patterns of nerve impulses, generated by a complex neuronal network, distributed in different regions of the brain (SAAB, 2014). Contrary to our finding, Schmidt et al. found no significant difference between people with CLBP and their healthy counterparts in the brain activity, averaged across the parieto- occipital sites by investigating brain activity in the parieto-occipital region. However, limitations in the study by Schmidt et al, such as the use of medications by about 50% of participating subjects, investigation of brain activity only in the parieto-occipital region and eye-closed condition make it hard to compare our findings to theirs (Schmidt et al., 2012).

Table 4. Analysis of variance for relative power of brain waves at brain regions of frontal, central, and parietal in eye-closed and eye-open conditions for healthy controls and patients with CLBP

Main Effect	F and P	Delta (%)	Alpha (%)
Group	F	0.144	1.332
	P	0.706	0.254
Eye condition	F	47.000	38.778
	P	<0.0001*	<0.0001*
Region	F	129.597	52.380
	P	<0.0001*	<0.0001*
Eye condition group**	P	0.043*	0.782
Region group***	P	0.870	0.569

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CLBP: Chronic low back pain; *Significant at P<0.05. Relative power is power in each band, relative to the total frequency bands.

**Shows the interaction of relative power for group and eye condition,

*** Shows the interaction of relative power for group and region.

Table 5. Univariate analysis of delta absolute power between patients with CLBP and healthy individuals in both conditions

Eye Condition	Absolute Power (uV2)	F	P	Mean Difference (95 % CI)
Eyes open	Frontal	5.852	0.019*	-4.23 (-7.74, -0.71)
	entralC	5.597	0.022*	3.88 (0.58, 7.19)
	Parietal	3.940	0.053	3.67 (-0.04, 7.39)
Eyes closed	Frontal	7.563	0.008*	3.21 (8.64, 5.57)
	entralC	10.430	0.002*	3.99 (1.50, 6.48)
	Parietal	4.596	0.037*	3.31 (0.20, 6.43)

CLBP: Chronic low back pain; CI: Confidence interval.

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*Significant at P<0.05.

Our findings showed a significant overactivity of the delta wave in people with CLBP compared to healthy controls in the EC condition. However, we found reduced delta wave activity in the frontal region in the EO condition. An overactivity of delta wave was also observed in patients with chronic pancreatitis and migraine (Olesen et al., 2011; Bjørk et al., 2009). Similar to our findings, Hargrove et al. found lower delta absolute power in the frontal region in patients with fibromyalgia (Hargrove et al., 2010).

We also found significant increases in the regional absolute power of theta in patients with CLBP in both conditions. In the EC condition, a significant increase was observed in the relative power of theta wave in patients with CLBP. Increased theta activity is a common finding in patients with chronic pain (Pinheiro et al., 2016). For instance, theta wave overactivity was found in patients with fibromyalgia (González Roldán et al., 2013; Fallon et al., 2018) and neurogenic pain (Sarnthein et al., 2006; Stern et al., 2006). Animal studies suggest that theta waves in the parietal region can be predictive of pain

Table 6. Univariate analysis of theta and beta relative power between patients with CLBP and healthy individuals in both conditions

Brain Wave	Relative Power (%)		F	P	Mean Difference (95% CI)
Theta	Eyes open	Frontal	2.078	0.156	1.83 (-0.72,4.40)
		entralC	1.369	0.248	1.74 (-1.25, 4.74)
		Parietal	0.088	0.768	0.47 (-2.75, 3.71)
	Eyes closed	Frontal	1.515	0.225	1.87 (-1.18, 4.93)
		entralC	2.322	0.134	3.51 (-1.12, 8.15)
		Parietal	4.680	0.036*	5.75 (-0.40, 11.10)
Beta	Eyes open	Frontal	0.000	0.987	0.02 (-2.29, 2.32)
		entralC	0.684	0.412	-1.25 (-4.31, 1.80)
		Parietal	2.887	0.096	-2.53 (-5.54, 0.46)
	Eyes closed	Frontal	0.972	0.329	-1.08 (-3.30, 1.13)
		entralC	0.753	0.390	-1.44 (-4.80,1.90)
		Parietal	0.005	0.941	-0.14 (-4.21, 3.91)

CLBP: Chronic low back pain; CI: Confidence interval.

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*Significant at P<0.05.

(LeBlanc et al., 2014). Increased theta activity in chronic pain was explained by TCD theory (Llinás et al., 2005).

Thalamocortical dysrhythmia refers to a state in which thalamic neurons change from high-threshold tonic firing to low-threshold calcium spike bursts in the theta–delta range caused by the suppression of excitatory inputs onto thalamic cells (Llinas & Jahnsen, 1982; Jahnsen & Llinas 1984 a, b; Steriade, Datta & Pare, 1990; Jeanmonod, Magnin & Morel, 1996). In this regard, studies showed the behavior of the thalamus neurons alters in chronic pain disorders, which in turn disrupts the normal thalamocortical rhythm (De Vries et al., 2013; Pascoal-Faria, et al., 2015). This disruption results in alterations in cortical processing in the frequency domain, including excess theta power (De Vries et al., 2013).

Our results showed an increased alpha power in the regional absolute power in patients with CLBP compared to the healthy controls in both conditions. Consistent with our findings, several studies reported increased alpha activity in patients with chronic pain conditions, such as chronic jaw pain (Wang et al., 2019), trigeminal neuralgia (Wang et al., 2014), and chronic pancreatitis (Drewes et al., 2008). Increased alpha activity indicates lower cortical excitability and a higher perceptual threshold (Foxe & Synder, 2011). Increased alpha activity in patients with chronic pain may correspond with functional suppression of attention (Wang et al., 2019). Research findings also showed that higher alpha power is associated with a lower subjective perception of pain (Babiloni et al., 2016).

Brain waves, including delta, theta, and alpha are related to cognitive performance (Klimesch, 1999; Prichep et al., 1994). Cognitive dysfunction is commonly observed in chronic pain patients (Berryman et al., 2013). For instance, a review study showed that increased theta and alpha power are associated with a reduction in the amplitude of evoked potentials in cognitive tasks in patients with chronic pain (Pinheiro et al., 2016). Also, another study reported that increased theta power is related to reduced cognitive efficiency in patients with fibromyalgia (Donaldson et al., 2003). Therefore, it is suggested that future studies assess the correlation between brain wave patterns and cognitive function in patients with chronic low back pain.

5. Conclusion

Our findings indicate a widespread overactivity of brain waves in patients with CLBP compared to their healthy counterparts. These alterations in brain wave activity may play a principal role in chronification and recurrence of pain in people with LBP. Cortical changes associated with CLBP present a new target for treating CLBP. Future studies aiming at the effects of cortical stimulation on central neuroplastic changes in CLBP will further our understanding of the cortical changes associated with LBP and possible treatment strategies to tackle this clinical dilemma.

Limitations

The present study had several limitations. In this study, we focused on the absolute and relative power indices between the groups. We did not investigate other indices of qEEG, such as coherence, power ratio, peak frequency, phase lag, and amplitude asymmetry. We did not use source localization techniques, such as LORETA to detect the exact region of the brain involved. Thus, future studies are required to examine brain source localization along with qEEG analysis.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article. This study was approved by the Ethics Committee of Ahvaz Jundishapur University of Medical Sciences (Code: IR.AJUMS.REC.1396.713) and was registered in Iranian Registry for Clinical Trial (Code: IRCT20140705018362N3).

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Authors' contributions

Data curation and writing–original draft: Maryam Sadat Larie; Project administration and formal analysis: Maryam Sadat Larie, Fateme Esfandiarpour; Supervision and funding acquisition: Fateme Esfandiarpour; Investigation: Maryam Sadat Larie, Fateme Esfandiarpour and Forough Riahi'; Methodology: All authors. Writing–review, and editing: Fateme Esfandiarpour, Forough Riahi and Mohamad Parnianpour.

Conflict of interest

The authors declared no conflict of interest.

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References

Apkarian A. V. (2011). The brain in chronic pain: Clinical implications. *Pain Management, 1*(6), 577–586. [DOI:10.2217/pmt.11.53] [PMID] [PMCID]

Apkarian, A. V., Bushnell, M. C., Treede, R. D., & Zubieta, J. K. (2005). Human brain mechanisms of pain perception and regulation in health and disease. *European Journal of Pain, 9*(4), 463–484. [DOI:10.1016/j.ejpain.2004.11.001] [PMID]

Apkarian, A. V., Sosa, Y., Sonty, S., Levy, R. M., Harden, R. N., & Parrish, T. B., et al. (2004). Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *The Journal of Neuroscience, 24*(46), 10410–10415. [DOI:10.1523/JNEUROSCI.2541-04.2004] [PMID] [PMCID]

Babiloni, C., Brancucci, A., Del Percio, C., Capotosto, P., Arendt-Nielsen, L., & Chen, A. C., et al. (2006). Anticipatory electroencephalography Alpha rhythm predicts subjective perception of pain intensity. *The Journal of Pain, 7*(10), 709–717. [DOI:10.1016/j.jpain.2006.03.005] [PMID]

Becker, A., Held, H., Redaelli, M., Strauch, K., Chenot, J. F., & Leonhardt, C., et al. (2010). Low back pain in primary care: Costs of care and prediction of future health care utilization. *Spine, 35*(18), 1714–1720. [DOI:10.1097/BRS.0b013e3181cd656f] [PMID]

Berryman, C., Stanton, T. R., Jane Bowering, K., Tabor, A., McFarlane, A., & Lorimer Moseley, G. (2013). Evidence for working memory deficits in chronic pain: A systematic review and meta-analysis. *Pain, 154*(8), 1181–1196. [DOI:10.1016/j.pain.2013.03.002] [PMID]

Bjørk, M. H., Stovner, L. J., Engstrøm, M., Stjern, M., Hagen, K., & Sand, T. (2009). Interictal quantitative EEG in migraine: A blinded controlled study. *The Journal of Headache and Pain, 10*(5), 331–339. [DOI:10.1007/s10194-009-0140-4] [PMID] [PMCID]

Carlsson, A. M. (1983). Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. *Pain, 16*(1), 87–101. [DOI:10.1016/0304-3959(83)90088-X] [PMID]

Cunha, M., Bastos, V. H., Veiga, H., Cagy, M., McDowell, K., & Furtado, V., et al. (2004). [Changes in cortical power distribution produced by memory consolidation as a function of a typewriting skill (Portuguese)]. *Arquivos De Neuro-Psiquiatria, 62*(3A), 662–668. [DOI:10.1590/S0004-282X2004000400018] [PMID]

de Vries, M., Wilder-Smith, O. H., Jongsma, M. L., van den Broeke, E. N., Arns, M., & van Goor, H., et al. (2013). Altered resting state EEG in chronic pancreatitis patients: Toward a marker for chronic pain. *Journal of Pain Research, 6,* 815–824. [DOI:10.2147/JPR.S50919] [PMID] [PMCID]

Donaldson, M., Mueller, H., Donaldson, S., & Sella, G. E. (2003). QEEG patterns, psychological status and pain reports of fibromyalgia sufferers. *American Journal of Pain Management, 13*(2), 60-73. [Link]

Pinheiro, E. S., de Queirós, F. C., Montoya, P., Santos, C. L., do Nascimento, M. A., & Ito, C. H., et al. (2016). Electroencephalographic patterns in chronic pain: A systematic review of the literature. *Plos One, 11*(2), e0149085. [DOI:10.1371/journal.pone.0149085] [PMID] [PMCID]

Drewes, A. M., Gratkowski, M., Sami, S. A., Dimcevski, G., Funch-Jensen, P., & Arendt-Nielsen, L. (2008). Is the pain in chronic pancreatitis of neuropathic origin? Support from EEG studies during experimental pain. *World Journal of Gastroenterology, 14*(25), 4020–4027. [DOI:10.3748/wjg.14.4020] [PMID] [PMCID]

Fairbank, J. C., & Pynsent, P. B. (2000). The oswestry disability index. *Spine, 25*(22), 2940–2952. [DOI:10.1097/00007632-200011150-00017] [PMID]

Fallon, N., Chiu, Y., Nurmikko, T., & Stancak, A. (2018). Altered theta oscillations in resting EEG of fibromyalgia syndrome patients. *European Journal of Pain, 22*(1), 49–57. [DOI:10.1002/ejp.1076] [PMID] [PMCID]

Ferreira, P. H., Ferreira, M. L., Maher, C. G., Herbert, R. D., & Refshauge, K. (2006). Specific stabilisation exercise for spinal and pelvic pain: A systematic review. *The Australian Journal of Physiotherapy, 52*(2), 79–88. [DOI:10.1016/S0004-9514(06)70043-5] [PMID]

Foxe, J. J., & Snyder, A. C. (2011). The role of alpha-band brain oscillations as a sensory suppression mechanism during selective attention. *Frontiers in Psychology, 2*, 154. [DOI:10.3389/fpsyg.2011.00154] [PMID] [PMCID]

Fregni, F., Freedman, S., & Pascual-Leone, A. (2007). Recent advances in the treatment of chronic pain with non-invasive brain stimulation techniques. *The Lancet. Neurology, 6*(2), 188–191. [DOI:10.1016/S1474-4422(07)70032-7] [PMID]

Giesecke, T., Gracely, R. H., Grant, M. A., Nachemson, A., Petzke, F., & Williams, D. A., et al. (2004). Evidence of augmented central pain processing in idiopathic chronic low back pain. *Arthritis and Rheumatism, 50*(2), 613–623. [DOI:10.1002/art.20063] [PMID]

Gombatto, S. P., Norton, B. J., Sahrmann, S. A., Strube, M. J., & Van Dillen, L. R. (2013). Factors contributing to lumbar region passive tissue characteristics in people with and people without low back pain. *Clinical Biomechanics, 28*(3), 255–261. [DOI:10.1016/j.clinbiomech.2013.01.005] [PMID] [PMCID]

González-Roldán, A. M., Muñoz, M. A., Cifre, I., Sitges, C., & Montoya, P. (2013). Altered psychophysiological responses to the view of others' pain and anger faces in fibromyalgia patients. *The Journal of Pain, 14*(7), 709–719.[DOI:10.1016/j.jpain.2013.01.775] [PMID]

Hall, J. E., & Hall, M. E. (2020). *Guyton and Hall Textbook of Medical Physiology*. Amsterdam: Elsevier. [Link]

Hargrove, J. B., Bennett, R. M., Simons, D. G., Smith, S. J., Nagpal, S., & Deering, D. E. (2010). Quantitative electroencephalographic abnormalities in fibromyalgia patients. *Clinical EEG and Neuroscience, 41*(3), 132–139. [DOI:10.1177/155005941004100305] [PMID]

Llinás, R., & Jahnsen, H. (1982). Electrophysiology of mammalian thalamic neurones in vitro. *Nature, 297*(5865), 406–408. [DOI:10.1038/297406a0] [PMID]

Jahnsen, H., & Llinás, R. (1984). Electrophysiological properties of guinea-pig thalamic neurones: An in vitro study. *The Journal of Physiology, 349*, 205–226. [DOI:10.1113/jphysiol.1984.sp015153] [PMID] [PMCID]

Jahnsen, H., & Llinás, R. (1984). Ionic basis for the electro-responsiveness and oscillatory properties of guinea-pig thalamic neurones in vitro. *The Journal of Physiology, 349*, 227–247. [DOI:10.1113/jphysiol.1984.sp015154] [PMID] [PMCID]

Jasper, H. H. (1958). The ten-twenty electrode system of the international federation. *Electroencephalography and Clinical Neurophysiology, 10*, 371-375. [Link]

Jeanmonod, D., Magnin, M., & Morel, A. (1996). Low-threshold calcium spike bursts in the human thalamus. Common physiopathology for sensory, motor and limbic positive symptoms. *Brain, 119* (Pt 2), 363–375. [DOI:10.1093/brain/119.2.363] [PMID]

Jatoi, M. A., & Kamel, N. (2018). *Brain source localization using EEG signal analysis*. London: Taylor & Francis. [DOI:10.1201/9781315156415]

Johannes, C. B., Le, T. K., Zhou, X., Johnston, J. A., & Dworkin, R. H. (2010). The prevalence of chronic pain in United States adults: Results of an Internet-based survey. *The Journal of Pain, 11*(11), 1230–1239. [DOI:10.1016/j.jpain.2010.07.002] [PMID]

Klimesch, W. (1999). EEG Alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews, 29*(2-3), 169-195. [DOI:10.1016/S0165-0173(98)00056-3]

Klimesch, W., Sauseng, P., & Hanslmayr, S. (2007). EEG Alpha oscillations: The inhibition-timing hypothesis. *Brain Research Reviews, 53*(1), 63–88. [DOI:10.1016/j.brainresrev.2006.06.003] [PMID]

Knyazev. G.G. (2012). EEG delta oscillations as a correlate of basic homeostatic and motivational processes. Neuroscience and Biobehavioral Reviews, 36(1),677-95. [DOI:10.1016]/j.neubiorev.2011.10.002] [PMID]

Kregel, J., Meeus, M., Malfliet, A., Dolphens, M., Danneels, L., & Nijs, J., et al. (2015). Structural and functional brain abnormalities in chronic low back pain: A systematic review. *Seminars in Arthritis and Rheumatism, 45*(2), 229–237.[DOI:10.1016/j.semarthrit.2015.05.002] [PMID]

Kropotov, J. (2010). Introduction. In J. D, Kropotov(Ed.), *Quantitative EEG, event-related potentials and neurotherapy* (pp. 1-9). Cambridge: Academic Press. [DOI:10.1016/B978-0-12-374512-5.50037-1]

Landolt, H. P., Rétey, J. V., Tönz, K., Gottselig, J. M., Khatami, R., & Buckelmüller, I., et al. (2004). Caffeine attenuates waking and sleep electroencephalographic markers of sleep homeostasis in humans. *Neuropsychopharmacology, 29*(10), 1933–1939. [DOI:10.1038/sj.npp.1300526] [PMID]

LeBlanc, B. W., Lii, T. R., Silverman, A. E., Alleyne, R. T., & Saab, C. Y. (2014). Cortical theta is increased while thalamocortical coherence is decreased in rat models of acute and chronic pain. *Pain, 155*(4), 773–782. [DOI:10.1016/j.pain.2014.01.013] [PMID]

Llinás, R., Urbano, F. J., Leznik, E., Ramírez, R. R., & van Marle, H. J. (2005). Rhythmic and dysrhythmic thalamocortical dynamics: GABA systems and the edge effect. *Trends in Neurosciences, 28*(6), 325–333. [DOI:10.1016/j.tins.2005.04.006] [PMID]

Melzack R. (1999). From the gate to the neuromatrix. *Pain, 82*(Suppl 6), S121–S126. [DOI:10.1016/S0304-3959(99)00145-1] [PMID]

Mousavi, S. J., Parnianpour, M., Mehdian, H., Montazeri, A., & Mobini, B. (2006). The oswestry disability index, the roland-morris disability questionnaire, and the quebec back pain disability scale: Translation and validation studies of the Iranian versions. *Spine, 31*(14), E454–E459. [DOI:10.1097/01.brs.0000222141.61424.f7] [PMID]

Olesen, S. S., Hansen, T. M., Graversen, C., Steimle, K., Wilder-Smith, O. H., & Drewes, A. M. (2011). Slowed EEG rhythmicity in patients with chronic pancreatitis: Evidence of abnormal cerebral pain processing? *European Journal of Gastroenterology & Hepatology, 23*(5), 418–424. [DOI:10.1097/MEG.0b013e3283457b09] [PMID]

Pascoal-Faria, P., Yalcin, N., & Fregni, F. (2015). Neural markers of neuropathic pain associated with maladaptive plasticity in spinal cord injury. *Pain Practice, 15*(4), 371–377. [DOI:10.1111/papr.12237] [PMID]

Ploner, M., Sorg, C., & Gross, J. (2017). Brain rhythms of pain. *Trends in Cognitive Sciences, 21*(2), 100–110. [DOI:10.1016/j.tics.2016.12.001] [PMID] [PMCID]

Prichep, L. S., John, E. R., Ferris, S. H., Reisberg, B., Almas, M., & Alper, K., et al. (1994). Quantitative EEG correlates of cognitive deterioration in the elderly. *Neurobiology of Aging, 15*(1), 85-90. [DOI:10.1016/0197-4580(94)90147-3]

Qaseem, A., Wilt, T. J., McLean, R. M., Forciea, M. A., Clinical Guidelines Committee of the American College of Physicians., & Denberg, T. D., et al. (2017). Noninvasive treatments for acute, subacute, and chronic low back pain: A clinical practice guideline from the American college of physicians. *Annals of Internal Medicine, 166*(7), 514–530. [DOI:10.7326/M16-2367] [PMID]

Saab, C. Y. (2013). *Chronic pain and brain abnormalities*. Cambridge: Academic Press. [Link]

Saragiotto, B. T., Maher, C. G., Yamato, T. P., Costa, L. O. P., Costa, L. C. M., & Ostelo, R. W. J. G., et al. (2016). Motor control exercise for nonspecific low back pain: A cochrane review. *Spine, 41*(16), 1284–1295. [DOI:10.1097/BRS.0000000000001645] [PMID]

Sarnthein, J., Stern, J., Aufenberg, C., Rousson, V., & Jeanmonod, D. (2006). Increased EEG power and slowed dominant frequency in patients with neurogenic pain. *Brain, 129*(Pt 1), 55–64. [DOI:10.1093/brain/awh631] [PMID]

Schabrun, S. M., Jones, E., Elgueta Cancino, E. L., & Hodges, P. W. (2014). Targeting chronic recurrent low back pain from the top-down and the bottom-up: A combined transcranial direct current stimulation and peripheral electrical stimulation intervention. *Brain Stimulation, 7*(3), 451–459. [DOI:10.1016/j.brs.2014.01.058] [PMID]

Schmidt, S., Naranjo, J. R., Brenneisen, C., Gundlach, J., Schultz, C., & Kaube, H., et al. (2012). Pain ratings, psychological functioning and quantitative EEG in a controlled study of chronic back pain patients. *Plos One, 7*(3), e31138. [DOI:10.1371/journal.pone.0031138] [PMID] [PMCID]

Schouppe, S., Van Oosterwijck, S., Danneels, L., Van Damme, S., & Van Oosterwijck, J. (2020). Are functional brain alterations present in low back pain? A systematic review of EEG studies. *The Journal of Pain, 21*(1-2), 25–43. [DOI:10.1016/j.jpain.2019.06.010] [PMID]

Spronk, D., Arns, M., Barnett, K. J., Cooper, N. J., & Gordon, E. (2011). An investigation of EEG, genetic and cognitive markers of treatment response to antidepressant medication in patients with major depressive disorder: A pilot study. *Journal of Affective Disorders, 128*(1-2), 41–48. [DOI:10.1016/j.jad.2010.06.021] [PMID]

Steriade, M., Datta, S., Pare, D., Oakson, G., Dossi, R.C. (1990) Neuronal activities in brain-stem cholinergic nuclei related to tonic activation processes in thalamocortical systems. *Journal of Neuroscience, 10*(8), 2541-59. [DOI:10.1523/JNEUROSCI.10-08-02541.1990] [PMID] [PMCID]

Stern, J., Jeanmonod, D., & Sarnthein, J. (2006). Persistent EEG overactivation in the cortical pain matrix of neurogenic pain patients. *NeuroImage, 31*(2), 721–731. [DOI:10.1016/j.neuroimage.2005.12.042] [PMID]

Tatum, W. O., Husain, A. M., Benbadis, S. R., & Kaplan, P. W.(2008). *Handbook of EEG interpretation*. New York: Demos Medical Publishing. [Link]

Tsao, H., Galea, M., & Hodges, P. W. (2008). Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. *Brain, 131*(8), 2161-71. [DOI:10.1093/brain/awn154] [PMID]

Wang, Y., Li, D., Bao, F., Ma, S., Guo, C., & Jin, C., et al. (2014). Thalamic metabolic alterations with cognitive dysfunction in idiopathic trigeminal neuralgia: A multivoxel spectroscopy study. *Neuroradiology, 56*(8), 685–693. [DOI:10.1007/s00234-014-1376-5] [PMID]

Wang, W. E., Roy, A., Misra, G., Ho, R. L. M., Ribeiro-Dasilva, M. C., & Fillingim, R. B., et al. (2019). Altered neural oscillations within and between sensorimotor cortex and parietal cortex in chronic jaw pain. NeuroImage. *Clinical, 24*, 101964. [DOI:10.1016/j.nicl.2019.101964] [PMID] [PMCID]

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