Full Title: Individuals with Chronic Low Back Pain Show Impaired Adaptations of Lumbar Extensor Muscle Reflex Amplitude During Unexpected Perturbations

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Disclosure

Competing interest statement

The authors have no competing interest to declare.

Conflict of interest

The authors have no actual or potential conflicts of interest, including any financial, personal, or other relationships with other people or organisations within 3 years of beginning the submitted work that could inappropriately influence, or be perceived to influence, their work.

Abbreviations:

Body mass index (BMI)

High-density surface electromyography (HDsEMG)

Low back pain (LBP)

Maximal voluntary isometric contractions (MVIC)

Nervous system (NS)

Pain Catastrophizing Scale (PCS)

Root mean square (RMS)

Standard deviation (SD)

Tampa Scale for Kinesiophobia (TSK)

Visual analog scales (VAS)

ABSTRACT

Purpose: This study investigated the adaptability of trunk muscle responses to a series of unexpected external perturbations in patients with chronic low back pain (LBP). **Methods**: Thirty-seven adult participants, including 19 without LBP (control group) and 18 with chronic LBP, were submitted to 15 repetitions of trunk perturbations applied in a posterior-to-anterior direction, inducing trunk flexion. High-density surface electromyography (HDsEMG) was used to analyze lumbar muscle reflex amplitude. A two-way repeated measures ANOVA (2x2) was conducted to compare group differences and the effect of trial repetition over time (first five trials vs last five trials of perturbations). **Results:** Significant interaction effects were found on both sides (Left: $p = 0.038$; Right: $p = 0.007$). Post hoc comparisons revealed a decrease in response amplitude only in the control group between the first and last five perturbations, with reductions of 5.0% on the left side ($p=0.026$, Bonferroni-corrected) and 5.7% on the right side ($p=0.030$, Bonferroni-corrected). In contrast, individuals with chronic LBP showed no significant adaptation through repetition in the reflex response amplitude of the lumbar extensor muscles (post-hoc both sides: $p > 0.05$). **Conclusion:** Individuals with chronic LBP fail to adapt reflex amplitudes to repeated perturbations, possibly due to impaired proprioception, reduced motor variability and neuroplastic changes observed in individuals with chronic LBP. These changes might limit their ability to optimize responses repeated perturbations, potentially compromising spinal stability and increasing functional cost.

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Keywords:

Lumbar muscles, trunk, perturbations, chronic low back pain, adaptations, high-density surface electromyography.

INTRODUCTION

Low back pain (LBP) is one of the most prevalent musculoskeletal conditions and the leading cause of years lived with disability (Cieza et al., 2020, Wu et al., 2020). Neuromuscular adaptations in individuals with chronic primary LBP, defined as persistent low back pain without a clear underlying pathology (Nicholas et al., 2019), are important to understand, as they can predict functional disability (Dubois et al., 2014) and possibly influence pain persistence. It is hypothesized that these adaptations influence muscle function and, given the role of back muscles in motor control, may lead to structural changes and alterations in pain perception or injury risk (Hodges and Danneels, 2019). These adaptations may be the result of proprioceptive impairments, possibly due to central alterations in sensorimotor processing (Meier et al., 2019, Tong et al., 2017, Kantak et al., 2022), which manifest as changes in force accuracy, steadiness, movement variability and muscle activity (Meier et al., 2019, Tong et al., 2017). Studies assessing neuromuscular adaptations in LBP individuals during voluntary tasks have shown alterations in lumbar muscle activation patterns, such as redistribution of muscle activity (Sanderson et al., 2019b, Sanderson et al., 2024, Sanderson et al., 2019a, Arvanitidis et al., 2021), increased trunk muscle coactivation (van Dieën et al., 2003b, van Dieën et al., 2003a), and changes in muscle activation levels, with some studies reporting decreased (Ahern et al., 1988, Cassisi et al., 1993) or increased (Lima et al., 2018) lumbar muscle activity.

In addition to voluntary tasks, individuals with chronic LBP often show abnormal reflexive responses in trunk muscles. Meta-analyses have shown delays in lumbar muscle activation during unexpected postural perturbations in individuals with chronic LBP (Abboud et al., 2017). Moreover, studies have reported increased reflex amplitudes of lumbar muscles in response to sudden perturbations in both chronic and acute LBP (Larivière et al., 2010, Jones et al., 2012a, Gao et al., 2014, Jones et al., 2012b). Trunk flexors reflex amplitude, however, have shown mixed results (Abboud et al., 2017). While these findings improve our understanding of pain neuromuscular alterations in LBP individuals, the majority of studies investigating chronic LBP often rely on single trial or averaged responses across multiple trials, which fail to assess the nervous system's (NS) ability to adapt motor responses overtime. A solution to this limitation is the assessment of trial-to-trial adaptation. In healthy individuals, the NS has been shown to adapt neuromuscular responses to perturbations based on prior exposure (Skotte et al., 2004), potentially reducing functional cost through repetitions. However, under acute experimental pain induced by delayed-onset lumbar muscle

soreness, which share common features with clinical LBP, such as inflammation and muscle damage, the NS was unable to adapt reflex muscle activity amplitude to repeated unexpected trunk perturbations (Abboud et al., 2021). Yet, how individuals with chronic LBP adapt to repeated perturbations remains unknown, as no studies have directly assessed trial-to-trial adaptations in LBP populations.

Therefore, this study aims to investigate the adaptability of trunk muscle responses to a series of unexpected external perturbations in patients with chronic LBP. It was hypothesized that individuals with LBP are not able to adapt their neuromuscular responses across trunk perturbation trials.

METHODS

Participants

Thirty-seven adult participants, including 19 individuals without LBP (control group) and 18 individuals with chronic LBP were recruited. Participants were recruited through advertisements in kinesiology and chiropractic clinics, as well as from the university community and via Facebook ads. Based on a power analysis conducted with G*Power 3.1 (Faul et al., 2007), 34 participants were required to achieve a power of 0.8 for a repeated-measure ANOVA with a within-between interaction (2x2) and a moderate effect size (f = 0.25). To account for a potential 10% attrition rate, the sample size was increased to 37 participants. Participants in the control group were included if they had not experienced any episodes of LBP in the last six months and were aged between 18 and 60 years. For participants with chronic LBP, the inclusion criteria required the presence of low back pain located between the lower ribs and the gluteal folds for at least three months, with or without radiating pain to the lower limbs and aged between 18 and 60 years. Exclusion criteria for both groups included a history of spinal surgery, cancer, spinal fractures, spinal metastases, or current pregnancy. All participants' medical histories were verified by an experienced chiropractor (A.H.). The project received approval from the Research Ethics Board for human research of the "Université du Québec à Trois-Rivières" (CER-22-294-07.06). All participants gave written informed consent. The study was conducted following the principles of the Declaration of Helsinki (2013).

Study Design

This experimental study was conducted during a single session in which lumbar extensor muscles were assessed through a series of 15 repeated unexpected trunk perturbations in the anteroposterior direction, inducing trunk flexion. At the beginning of the session, LBP participants completed a series of French validated questionnaires to assess various psychological and disability-related factors. These included:

1. Tampa Scale for Kinesiophobia (TSK) (Woby et al., 2005, French et al., 2002) : This scale assesses the fear of movement or re-injury due to pain, often referred to as kinesiophobia. It contains 17 items scored on a 4-point Likert scale (from 1 = Strongly Disagree to 4 = Strongly Agree). The total score ranges from 17 to 68, with higher scores indicating greater fear of movement, and a score above 37 typically suggests high kinesiophobia.

2. Beck Depression Inventory (Bourque and Beaudette, 1982): The Beck Depression Inventory is used to assess the severity of depressive symptoms. It consists of 21 multiple-choice questions, each rated from 0 to 3, leading to a total score range from 0 to 63. Scores are categorized as follows: 0-13 (minimal depression), 14-19 (mild depression), 20-28 (moderate depression), and 29-63 (severe depression).

3. Pain Catastrophizing Scale (PCS) (French et al., 2005): The PCS measures the degree to which a person catastrophizes their pain, which includes rumination, magnification, and feelings of helplessness. It consists of 13 items, each scored from 0 to 4, leading to a total score range from 0 to 52. Higher scores indicate greater pain catastrophizing, with scores above 30 generally indicating a high level of catastrophizing.

4. Oswestry Disability Index (ODI) (Vogler et al., 2008): The Oswestry Disability Index is used to measure the degree of disability related to low back pain. It contains 10 sections, each scored from 0 to 5, with a total score range from 0 to 50. The score is then multiplied by 2 to give a percentage score (0-100%), with higher scores indicating greater disability. A score of 0-20% suggests minimal disability, 21-40% suggests moderate disability, 41-60% suggests severe disability, and 61-100% indicates very severe disability.

These scales were used to better understand the participant's condition. Additionally, participants with LBP were asked to rate their pain using visual analog scales (VAS), where the left end represented no pain, and the right end, represented extreme pain. Participants were asked to rate their pain at two specific time points: the beginning of the protocol and at the end of the protocol, after the 15 perturbations.

Trunk Perturbation Protocol

Three maximal voluntary isometric contractions (MVICs) were performed in the posteroanterior direction (trunk flexion) in a semi-seated position, with their legs secured to minimize lower limb contributions (Figure 1). Using their trunk, participants were instructed to pull against the resistance of a cable installed over their shoulders as hard as possible for 5 seconds. This same position was used for the subsequent perturbation protocol. A one-minute rest was provided between each MVIC attempt to minimize muscle fatigue. After determining the highest MVIC value, participants were required to perform an isometric contraction representing 20% of their maximum trunk flexion MVIC consistent with previous study (Abboud et al., 2023). Perturbations were delivered via a cable attached to a custom-made harness positioned on the participant's torso. This cable was connected to a force gauge (Model LSB350; Futek Advanced Sensor Technology Inc., Irvine, CA, USA), which was in turn connected to a small motor. The perturbations were triggered by a custom-made device attached to the motor. A similar set-up has been previously described and used in other studies (Larivière et al., 2010, Radebold et al., 2000). Participants used visual feedback on a computer screen to maintain the target force of 20% MVIC. Once this target was reached, a perturbation was triggered after a randomized delay of 1, 3, or 5 seconds to prevent anticipation. The perturbation applied a force in the posterior-to-anterior direction, inducing trunk flexion. Participants wore noise-canceling earmuff (Stanley, RTS-63011, Honeywell Safety Products Australia, Pty Ltd) to eliminate any auditory cues from the trigger device. Between perturbations, participants were instructed to return to the neutral trunk position, perpendicular to the ground, in preparation for the next perturbation. The cable length remained consistent across all perturbation trials, ensuring that participants maintained the same neutral position for each perturbation.

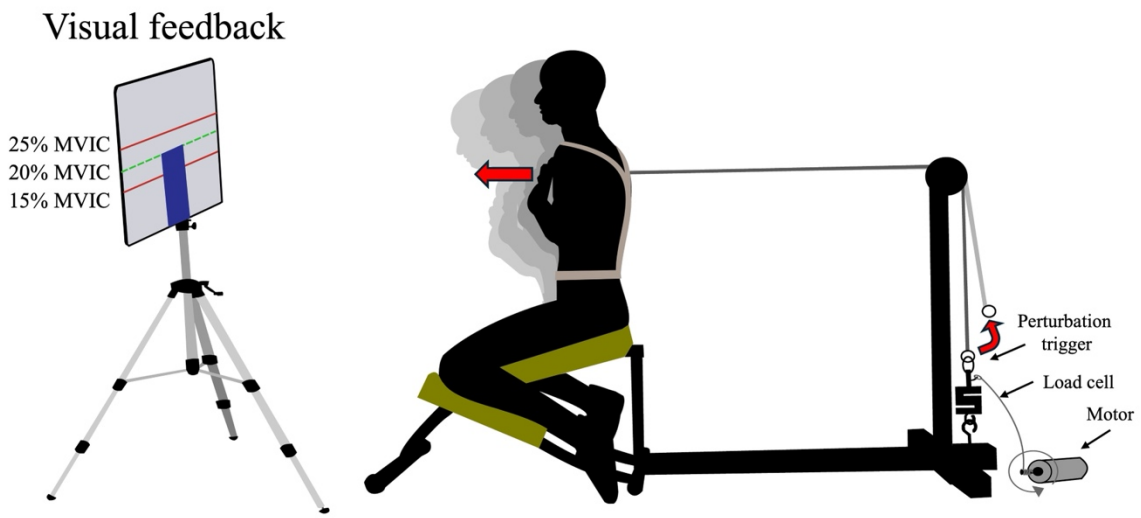

Fig. 1 Illustration of the trunk perturbation protocol. The red arrows indicate events occurring during a trunk perturbation.

Data collection

HDsEMG

The muscle activity of the right and left lumbar extensor muscle was measured using high-density surface electromyography (HDsEMG), two grids of 64 electrodes each (semi-disposable adhesive matrix; model GR10MM0808, 3mm electrode diameter, 10 mm inter-electrode spacing, 8X8, OTBioelettronica, Torino, Italy). HDsEMG was used to record muscle activity across the entire lumbar region, providing a more comprehensive understanding of reflex adaptations in the lumbar extensor muscles. This is particularly relevant as patients with LBP are known to show altered lumbar muscle activity, with changes in muscle redistribution (Sanderson et al., 2019b, Sanderson et al., 2024, Sanderson et al., 2019a, Arvanitidis et al., 2021). The grids were placed over the lumbar extensor muscles, with the center of the grid positioned at the L3 level, approximately 1 cm lateral to each side of the lumbar spinous processes (Criswell, 2010). The ground electrode was placed on the right posterior superior iliac spine. Prior to grid placement, the skin over the lumbar region was shaved, cleaned using fine-grade sandpaper (Red DotTrace Prep; 3M, St. Paul, MN) and rubbing alcohol (ethyl alcohol 70%). Signals from the HDsEMG were amplified (256-channel EMG-USB2; OTBioelettronica, Torino, Italy) by a factor of 2000X. Signals from the HDsEMG were sampled at

2048 Hz and digitized using a 12-bit A/D converter. To ensure consistency, the same investigator placed the HDsEMG for all participants. HDsEMG signals were normalized to the maximal root mean square (RMS) value recorded over a 1-second epoch during trunk extension MVIC task. During these tasks, participants had to pull posteriorly on a cable as hard as possible in trunk extension for 5 seconds. Three trunk extension MVICs were performed. The highest MVIC value was considered for EMG normalization, and the normalized signal was used to assess muscle activity amplitude variables (baseline muscle activity and reflexes amplitude).

Bipolar EMG

The muscle activity of the left and right rectus abdominis and obliquus externus was recorded using single differential Delsys Surface EMG sensor with a common mode rejection ratio of 92 dB at 60 Hz, a noise level of 1.2 µV, a gain of 10 V/V ± 1%, an input impedance of 10^{15} Ω, a bandwidth of 20–450 ± 10% (Model DE2.1, Delsys Inc., Boston, MA, USA). Electrodes on the rectus abdominis were placed parallel to the muscle fibers and located approximately 2 cm lateral from the umbilicus over the muscle belly. Electrodes for the obliquus externus were placed lateral to the rectus abdominis and directly above the anterior superior iliac spine, halfway between the crest and the ribs parallel to the muscle fibers. Both these locations are in accordance with CRAM recommendations for electrode placement (Criswell, 2010). Prior to electrodes placement, the skin of the region was shaved and clean using the same procedure as previously described. Signals from the bipolar EMG were amplified by a factor of 1000X. Signals were sampled at 2048 Hz and digitized using a 12-bit A/D converter. The same investigator placed the bipolar EMG for all participants. Trunk flexor EMG signals were normalized using the maximum RMS value obtained from a 1-second epoch during the trunk flexion MVIC.

Data Analyses

The signals were processed and analyzed using MATLAB (v.2024a; The MathWorks, Natick, MA). Both the HDsEMG signals and bipolar EMG signals were digitally band-pass filtered using a 4th-order

Butterworth filter with a frequency range of 20–400 Hz. To mitigate interference from the 60 Hz power line and its harmonics, second-order Butterworth notch filters were used.

HDsEMG

HDsEMG data were analyzed independently for the left and right sides. Differential HDsEMG signals were calculated by subtracting consecutive monopolar signals along the craniocaudal direction (fiber orientation). The final grid configuration consisted of 7×8 channels. In addition to Butterworth filtering, a visual inspection of the Fast Fourier Transform and raw signals was performed to identify electrodes affected by contact issues, noise, or artifacts. Electrodes were classified as problematic when they differed from neighboring signals. Specifically, signals were considered problematic if their frequency spectrum fell outside the EMG range or if the amplitude was excessively high compared to neighboring electrodes. This visual inspection method serves as an additional quality control step, complementing the objective filtering method to ensure signal accuracy. Problematic electrodes were replaced using an interpolation method that used data from adjacent electrodes along the craniocaudal axis (fiber orientation). The interpolation involved averaging the signals from the two adjacent electrodes. For electrodes located in the first or last row, where adjacent electrodes were unavailable, the problematic electrodes were excluded from the analysis to maintain data quality. Recordings were excluded if more than 10% of electrodes from the same grid exhibited unstable or poor signal quality (Gallina et al., 2022). HDsEMG data from three participants (two in the control group and one with LBP) were excluded on the left side, while data from one participant in the control group were excluded on the right side.

Bipolar EMG

A visual inspection of the Fast Fourier Transform and raw signals was also performed on bipolar EMG to identify problematic electrodes. Any problematic electrode was removed from analyses. Data from five participants (three LBP and two in the control group) were removed for the bipolar EMG on both the left and right sides, for both the obliquus externus and rectus abdominis muscles, due to a thick layer of abdominal adiposity that prevented signal recording. To mitigate data loss, enhance robustness and given

that no significant differences were observed between the left and right sides ($p > 0.05$), the data from both sides were averaged for analysis.

Variables

Dependent variables were calculated for all trials. To evaluate adaptation, the effect of trial repetition was assessed by comparing the mean values of the first five perturbation trials to those of the last five perturbation trials for all dependent variables, following previous methodology (Abboud et al., 2016).

Baseline

Baseline muscle activation was assessed by calculating the RMS of the signals across all channels within a 500 ms window preceding the perturbation onset.

Response latency

Response latency was defined as the time interval (in milliseconds) between the onset of the perturbation and the onset EMG reflex response. HDsEMG signals were processed using a dual-pass, zero-lag, sixth-order Butterworth low-pass filter with a 50 Hz cut-off frequency and response onset was determined when the RMS EMG signals exceeded three standard deviations (SDs) above baseline muscle activity (Hodges and Bui, 1996). The onset was then assessed using a 25 ms sliding window, following previous methodology (Larivière et al., 2010, Abboud et al., 2023). Reflex latency was confirmed through visual inspection of all electrodes for the first perturbation by the same investigator. This step ensured that the automated onset detection method worked correctly for each participant. Responses were classified as voluntary and excluded from analysis if the response latency exceeded 200 ms. Moreover, responses faster than 15 ms were removed from the analyses as they were classified as aberrant responses (Zedka et al., 1999, Cholewicki et al., 2005).

Response Amplitude

The EMG peak response was initially identified, after which the response amplitude was calculated as the mean RMS value over a 100-ms window centered on the peak. The EMG peak had to occur in a 300-

ms window following the perturbation onset to be considered in the analysis. To evaluate the contribution of the trunk flexor muscles, response amplitude was also calculated for the abdominal muscles using the same method.

Centroid Coordinates of Response Amplitude

To determine the spatial distribution of lumbar extensor muscle activity in response to perturbations, the mediolateral and craniocaudal coordinates of the centroid were calculated. The centroid was defined as the location corresponding to the mean of the channels with RMS response values that exceeded 70% of the maximum RMS across all channels (Gallina et al., 2022). The centroid was computed on the EMG response amplitude. The right-side array was flipped along the x-axis, to ensure that higher x-coordinates consistently represented a more medial position of the centroid on both sides to simplify the interpretation of spatial coordinates.

Statistical Analyses

Statistical analyses were conducted using SPSS Statistics for Mac, version 28 (SPSS Inc., IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test and visual inspection assessed data distribution, guiding the choice of parametric or non-parametric tests. Participants' characteristics (i.e., age, gender, weight, height, body mass index (BMI)) as well as the perturbation force (defined as 20% of the participant's MVIC exerted forward prior to the perturbation), were summarized using frequency distributions for categorical variables and means and SD for continuous variables. For group comparisons, independent samples t-test was used for continuous variables, and the chi-square test was used for categorical variables. Repeated measures ANOVAs evaluated the effect of trial repetition (first vs. last 5 perturbations), group differences and interactions on dependent variables. When necessary, Bonferroni post hoc tests were performed. A significance level of $p < 0.05$ was used for all analyses, with effect sizes reported as partial eta-squared (η^2) for the ANOVAs. Results are presented as means and SD.

RESULTS

Demographic

Demographic characteristics are presented in Table 1. The two groups did not differ significantly in any demographic variable or in perturbation force.

Table 1 Participants' characteristics

Characteristics	Control (N=19) (n (%) or mean ± SD)	LBP (N=18) (n (%) or mean ± SD)	Statistical test (t or X^2)	P-value	Effect size
Age (years)	32.79 ± 11.70	34.22 ± 10.40	T = 0.39	p = 0.697	d=0.129
Gender	M : 10 (52.6) F : 9 (47.4)	M : 9 (50.0) F : 9 (50.0)	X^2 = 0.026,	p = 0.873	V = 0.026
Weight (kg)	75.17 ± 10.63	80.91 ± 20.68	t = 1.07	p = 0.292	d=0.352
Height (m)	1.73 ± 0.09	1.71 ± 0.10	t = -0.93	p = 0.361	d=0.305
BMI (Kg/m^2)	25.04 ± 3.83	27.62 ± 6.04	t = 1.56	p = 0.128	d=0.512
Perturbation force (N)	130.30 ± 52.47	130.06 ± 40.39	t = -0.16,	p = 0.988	d=0.005

Questionnaire

The results and interpretation of the questionnaires for LBP participants are presented in Table 2, highlighting generally low psychometric scores and moderate pain intensity.

Table 2 Questionnaire scores for individuals with LBP (n=18)

Questionnaire	Score (mean ± SD)	Interpretation
Oswestry Disability Index (%)	24.12±9.14%	Moderate disability
Beck Depression Inventory (/63)	5.67±4.39	Minimal depression
TSK (/68)	31.72±6.45	Low kinesiophobia
PSC (/52)	16.00±8.21	Low pain catastophising
VAS Baseline (/10)	2.94±1.89	Mild to moderate pain intensity
VAS post perturbations (/10)	4.23±2.43	Moderate pain

Baseline

Baseline activity was not altered across trials on either side (Left: F (1,32) = 1.519, p = 0.227, partial η^2 = 0.045; Right: F (1, 34) = 2.846, p = 0.101, partial η^2 = 0.077). Similarly, no significant group differences were observed on baseline muscle activity on either side (Left: F (1, 32) = 0.010, p = 0.921, partial η^2 = 0.000; Right: F (1, 34) = 0.147, p = 0.704, partial η^2 = 0.004). Additionally, no significant interaction effects were found on either side (Left: F (1, 32) = 0.376, p = 0.544, partial η^2 = 0.012; Right: F (1, 34) = 0.151, p = 0.700, partial η^2 = 0.004). Mean and SD are presented in table 3.

Response Latency

Latency did not change across trials on either side (Left: F (1,32) = 1.174, p = 0.287, partial η^2 = 0.035; Right: F (1, 34) = 0.107, p = 0.746, partial η^2 = 0.003). No significant group differences were observed on latency on either side (Left: F (1, 32) = 0.370, p = 0.548, partial η^2 = 0.011; Right: F (1, 34) = 0.031, p = 0.861, partial η^2 = 0.001). Additionally, no significant interaction effects were found on either side (Left: F (1, 32) = 3.721, p = 0.065, partial η^2 = 0.104; Right: F (1, 34) = 1.935, p = 0.173, partial η^2 = 0.054). Mean and SD are presented in table 3.

Response amplitude

Lumbar extensor muscle

The lumbar extensor response amplitude did not show any changes across trials on either side (Left: F (1,32) = 1.298, p = 0.263, partial η^2 = 0.039; Right: F (1, 34) = 0.101, p = 0.753, partial η^2 = 0.003). Similarly, no significant group differences were observed on response amplitude on either side (Left: F (1, 32) = 0.316, p = 0.578, partial η^2 = 0.010; Right: F (1, 34) = 0.523, p = 0.474, partial η^2 = 0.015). Significant interaction effects were found on both sides (Left: F (1, 32) = 4.659, p = 0.038, partial η^2 = 0.127; Right: F (1, 34) = 8.301, p = 0.007, partial η^2 = 0.196). Post hoc comparisons revealed a decrease in response amplitude only in the control group between the first and last five perturbations, with reductions of 5.0% on the left side (p=0.026, Bonferroni-corrected) and 5.7% on the right side (p=0.030, Bonferroni-corrected). No other post hoc comparisons were significant. Mean and SD are presented in table 3 and in figure 2 and 3.

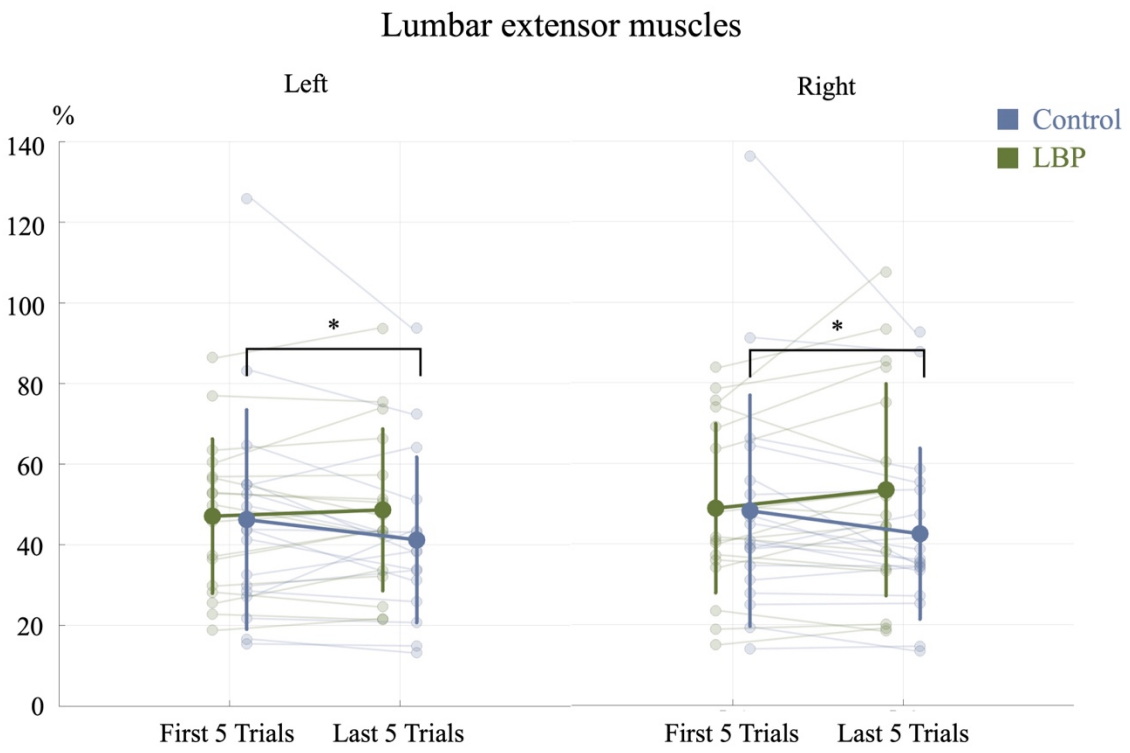

Fig. 2 Comparisons of lumbar extensor muscle reflex amplitude between the first 5 and last 5 trials for both groups. The control group is shown in blue, and the LBP group in green. Individual data points are connected by lines. Statistically significant differences ($p < 0.05$) are indicated by '*'. Data are presented as mean ± standard deviation. The y-axis represents muscle activation as a percentage of the maximal voluntary contraction.

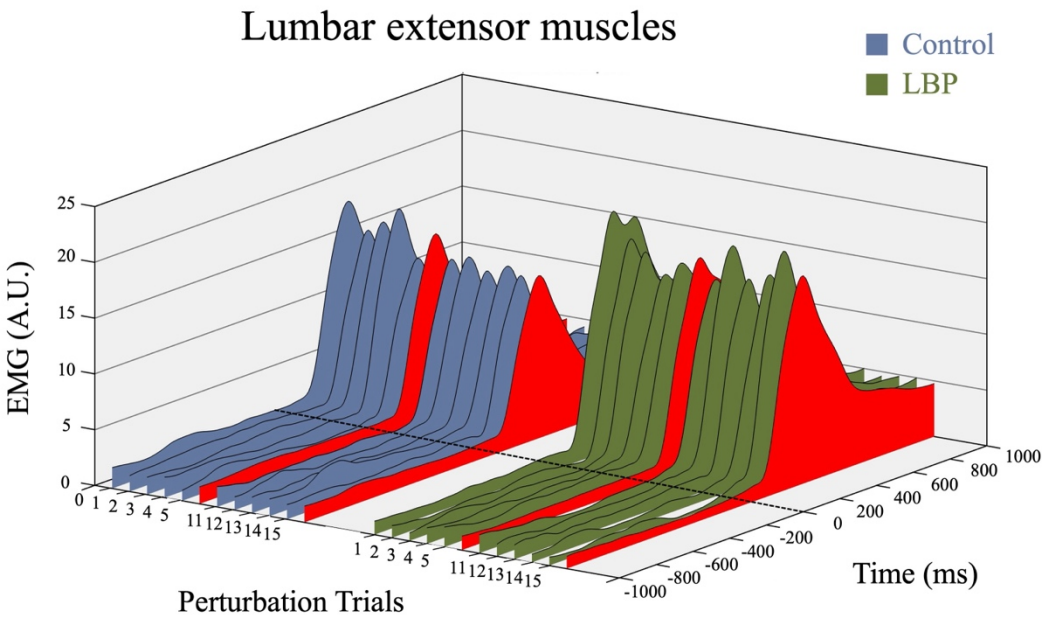

Fig. 3 Mean EMG activity across HDsEMG channels on both sides. The control group is shown in blue, and the LBP group in green. The first (1-5) and last (11-15) 5 trials are displayed for each group, with the red trial indicating the mean of the preceding five trials. The black dotted line marks the perturbation onset. (A.U.: Arbitrary Units).

Trunk Flexor

The reflex amplitude of both abdominal muscles varied across trials (obliquus externus: $F(1,30) = 9.382$, $p = 0.005$, partial $\eta^2 = 0.238$; rectus abdominis: $F(1,30) = 6.372$, $p = 0.017$, partial $\eta^2 = 0.175$). The mean reductions were 25.83% for the obliquus externus and 26.97% for the rectus abdominis. No significant group differences were observed for either muscle (obliquus externus: $F(1,30) = 0.373$, $p = 0.546$, partial $\eta^2 = 0.012$; rectus abdominis: $F(1,30) = 0.309$, $p = 0.583$, partial $\eta^2 = 0.010$). Additionally, no significant interaction effects were found (obliquus externus: $F(1,30) = 0.000$, $p = 1.000$, partial $\eta^2 = 0.000$; rectus abdominis: $F(1,30) = 0.002$, $p = 0.964$, partial $\eta^2 = 0.000$). The means and standard deviations are presented in Table 4 and in figures 4 and 5.

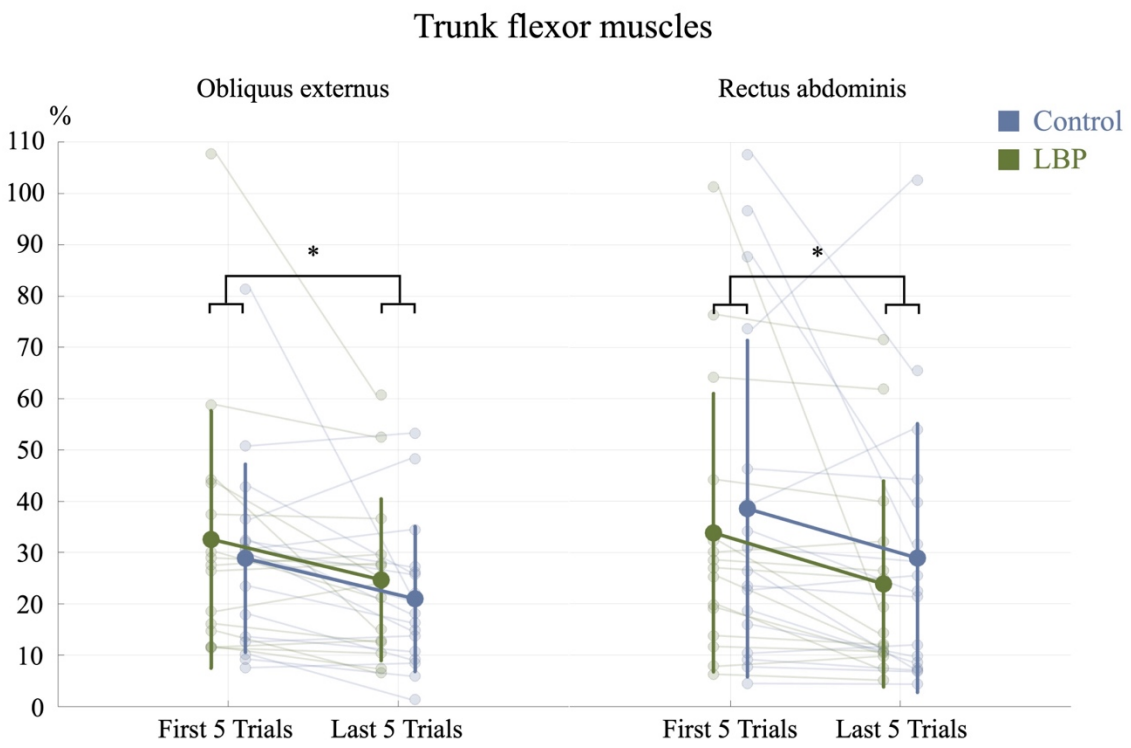

Fig. 4 Comparisons of trunk flexor muscle reflex amplitude between the first 5 and last 5 trials for both groups. The control group is shown in blue, and the LBP group in green. Individual data points are connected by lines. Statistically significant differences ($p < 0.05$) are indicated by '*'. Data are presented as mean ± standard deviation. The y-axis represents muscle activation as a percentage of the maximal voluntary contraction.

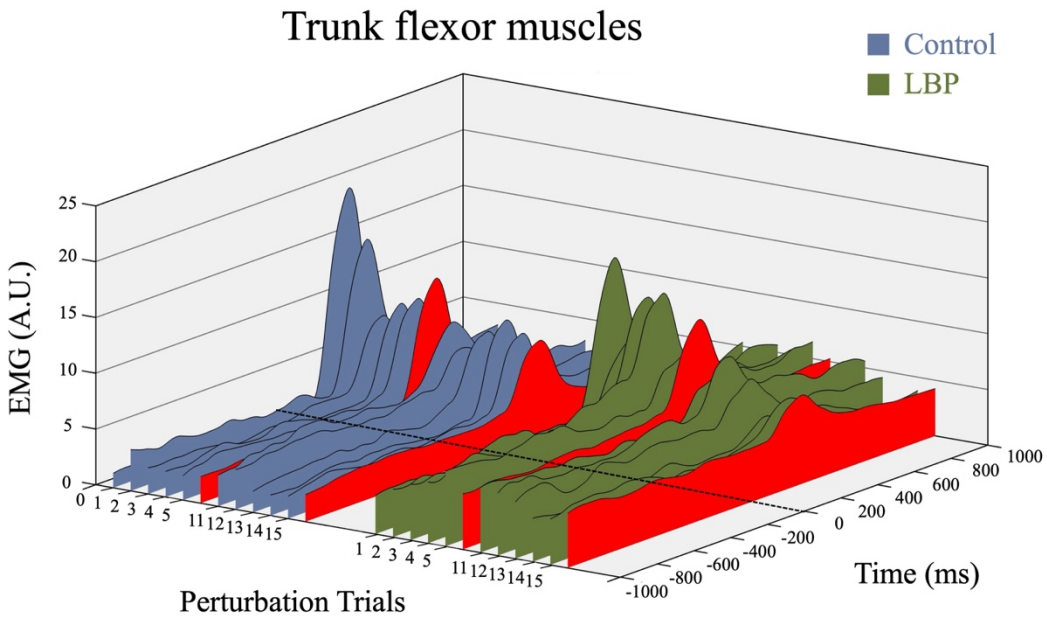

Fig. 5 Mean EMG activity across both trunk flexor muscle (obliquus externus and rectus abdominis). The control group is shown in blue, and the LBP group in green. The first (1-5) and last (11-15) 5 trials are presented for each group, with the red trial indicating the mean of the preceding five trials. The black dotted line marks the perturbation onset. (A.U.: Arbitrary Units).

Centroid of Response Amplitude

Mediolateral

The mediolateral centroid coordinates remained unchanged across trials on either side (Left: $F(1,32) = 0.808$, $p = 0.376$, partial $\eta^2 = 0.025$; Right: $F(1, 34) = 3.209$, $p = 0.082$, partial $\eta^2 = 0.086$). Similarly, no significant group differences were observed on mediolateral centroid coordinates on either side (Left: $F(1, 32) = 2.467$, $p = 0.126$, partial $\eta^2 = 0.072$; Right: $F(1, 34) = 0.632$, $p = 0.432$, partial $\eta^2 = 0.018$). Additionally, no significant interaction effects were found on either side (Left: $F(1, 32) = 1.641$, $p = 0.209$, partial $\eta^2 = 0.049$; Right: $F(1, 34) = 0.892$, $p = 0.353$, partial $\eta^2 = 0.026$). Mean and SD are presented in table 3.

Craniocaudal

The craniocaudal centroid coordinates remained unchanged across trials on either side (Left: $F(1,32) = 0.696$, $p = 0.410$, partial $\eta^2 = 0.021$; Right: $F(1, 34) = 0.377$, $p = 0.543$, partial $\eta^2 = 0.011$). No

significant group differences were observed on craniocaudal centroid coordinates the left side (Left: F (1, 32) = 2.844, p = 0.101, partial η^2 = 0.082. However, significant group differences were observed on the right side (Right: F (1, 34) = 5.341, p = 0.027, partial η^2 = 0.136). Post hoc analysis indicated a more cranial centroid location in the LBP group compared to the control group (p = 0.027, Bonferroni-corrected). No significant interaction effects were found on either side (Left: F (1, 32) = 0.025, p = 0.876, partial η^2 = 0.001; Right: F (1, 34) = 0.470, p = 0.498, partial η^2 = 0.014). Mean and SD are presented in table 3 and figure 6.

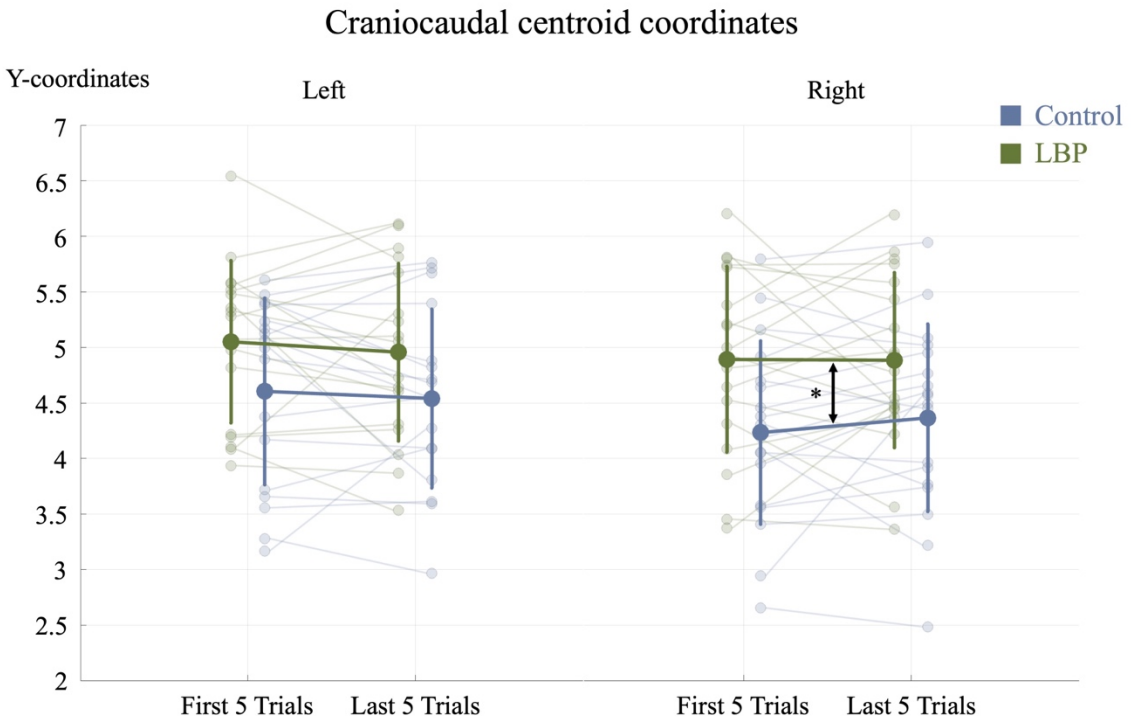

Fig. 6 Comparisons of craniocaudal centroid coordinates between the first 5 and last 5 trials for both groups. The control group is shown in blue, and the LBP group in green. Individual data points are connected by lines. Statistically significant differences ($p < 0.05$) are indicated by '*'. The difference between groups on the right side is 0.589 cm. Data are presented as mean ± standard deviation. The y-axis represents the craniocaudal (y) centroid coordinates.

Table 3 Mean and SD for each variable of the lumbar extensor muscles on both the left and right sides of the trunk.

Variables	Group	Trial repetition	Left	Right

Baseline (% RMS)	Control	First 5 trials	14.93±12.97	14.49±13.27
		Last 5 trials	14.75±13.76	14.13±12.88
	LBP	First 5 trials	14.72±8.10	16.17±11.67
		Last 5 trials	14.20±8.17	15.59±11.28
Response latency (ms)	Control	First 5 trials	59.96±18.80	57.65±18.58
		Last 5 trials	62.12±24.15	60.72±21.28
	LBP	First 5 trials	60.72±19.16	60.51±21.99
		Last 5 trials	53.04±22.56	55.56±23.45
Lumbar extensor muscle response amplitude (% RMS)	Control	First 5 trials	46.23±27.20	48.22 ± 28.69
		Last 5 trials	41.22±20.49	42.52±21.19
	LBP	First 5 trials	47.10±19.14	48.84±21.00
		Last 5 trials	48.65±20.02	53.42±26.26
Mediolateral centroid coordinates	Control	First 5 trials	6.08±0.70	5.79±0.86
		Last 5 trials	6.29±0.79	5.86±1.05
	LBP	First 5 trials	5.78±0.99	5.48±0.84
		Last 5 trials	5.74±0.83	5.72±0.76
Craniocaudal centroid coordinates	Control	First 5 trials	4.60±0.84	4.23±0.82
		Last 5 trials	4.54±0.81	4.37±0.84
	LBP	First 5 trials	5.05±0.73	4.89±0.84
		Last 5 trials	4.96±0.80	4.88±0.79

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Table 4 Mean and SD of the response amplitude for the obliquus externus and rectus abdominis muscles.

Variables	Group	Trial repetition	Obliquus externus	Rectus abdominus
Abdominal muscle response amplitude (% RMS)	Control	First 5 trials	28.87±18.31	38.55±32.83
		Last 5 trials	20.96±14.13	28.92±26.18
	LBP	First 5 trials	32.56±25.10	33.85±27.12
		Last 5 trials	24.68±15.78.	23.86±20.05

DISCUSSION

The main aim of this study was to investigate the adaptability of trunk muscle responses to a series of unexpected external perturbations in patients with chronic LBP. We hypothesized that individuals with LBP are not able to adapt their neuromuscular responses across trunk perturbation trials. Our findings partially supported this hypothesis, showing that while individuals with chronic LBP were not able to adapt the reflex response amplitude of lumbar extensor muscles, they were able to reduce the reflex co-contraction amplitude of trunk flexor muscles across trunk perturbations.

Impact of Chronic Low Back Pain on Reflex Response Amplitude

The lack of adaptation through repetition in reflex response amplitude of lumbar extensor muscles observed in individuals with chronic LBP may be explained through motor control variability models. According to recent theories, motor strategies in chronic LBP initially involve an increase in motor variability due to the perturbation caused by the nociceptive input resulting in a redistribution of muscle activity hypothesized to serves as a protective mechanism to minimize further pain or tissue damage (Madeleine, 2010, Meier et al., 2019). However, the persistence of these protective strategies often leads to a reduction in motor variability over time (Madeleine, 2010). This reduction can result in excessive loading on spinal tissues, which can contribute to reduced proprioceptive input (Meier et al., 2019). Additionally, neuroplastic changes, including alterations in the primary motor cortex (M1) and sensorimotor regions, may disrupt the organizational structure of these areas such as overlapping regions which may impair the NS ability for

flexible motor responses (Tsao et al., 2011). The combination of impaired proprioception, reduced motor variability, and neuroplastic changes may alter the ability to explore and adopt optimal motor strategies, thereby limiting the ability to dynamically adjust motor output in response to repeated unexpected perturbations.

Despite the observed deficits in lumbar extensor muscles, individuals with LBP showed the ability to adapt the co-contraction reflex amplitude of trunk flexor muscles, comparable to the control group. Such adaptation in trunk flexor muscles suggests that LBP alterations of adaptability may primarily affect muscles located in the painful region, while muscles involved in co-contraction, away from the painful region, are still able to decrease muscle activity during repeated trunk perturbations. This could be explained by the impact of pain on muscle function alterations in the lumbar region (Matheve et al., 2023), which can alter proprioception and the processing of proprioceptive information (Meier et al., 2019). These local proprioceptive changes and muscle function alterations may impact the ability to modulate reflex amplitude in the lumbar extensor muscles, as previously discussed, without a significant impact in the trunk flexor muscle allowing for adaptation. Although these findings are interesting, the underlying explanations remain speculative. Future research should examine the mechanisms involved.

Impact of Chronic Low Back Pain on Centroid Coordinates

A more cranial location in lumbar muscle activation was observed in patients with chronic LBP compared to controls. This cranial activation pattern in chronic LBP has been observed in several other studies during voluntary movements (Sanderson et al., 2019b, Sanderson et al., 2024, Sanderson et al., 2019a, Arvanitidis et al., 2021). This suggests that individuals with chronic LBP may recruit the upper motor unit territories of the erector spinae muscle (Gallina et al., 2024, Abboud et al., 2020). The cranial recruitment may be explained by increased intramuscular fatty infiltration and muscle atrophy in the lower lumbar muscles of those with LBP, compared to healthy controls (Matheve et al., 2023, Mardulyn et al., 2025, Gildea et al., 2013). These degenerative changes likely reduce the functional capacity of the lower muscles, leading to a compensatory shift in activation toward more cranial regions (Matheve et al., 2023). Moreover, these cranial regions likely innervate fibers attached to the thoracic vertebrae, helping to reduce tension in the lumbar region where the pain is localized, thereby protecting the area from further injury (Bogduk, 2005,

Hodges and Tucker, 2011). However, this cranial recruitment may be less efficient, as a more caudal activation could be biomechanically advantageous by engaging a larger muscle volume, distributing the load more effectively, and using a longer lever arm (Bogduk, 2005, Sanderson et al., 2019b).

Impact of Chronic Low Back Pain on Response Latency and Baseline

Baseline muscle activity was similar between individuals with chronic LBP and controls, consistent with findings from other perturbation studies (Miller et al., 2013, Liebetrau et al., 2013, Jones et al., 2012b). Regarding response latency, no significant differences were observed in this study. Findings on response latency in LBP patients are heterogeneous, with some studies reported longer latency (Radebold et al., 2001, Shenoy et al., 2013, Radebold et al., 2000), while others showed shorter latency (Freddolini et al., 2014), and some found no difference between groups (Larivière et al., 2010, Leinonen et al., 2001). This heterogeneity may be attributed to differences in assessment methods, as most studies rely on bipolar EMG. Bipolar EMG may not capture the behavior of the entire muscle since the lumbar extensor muscles, such as the erector spinae, can be activated at different vertebral levels (Abboud et al., 2020). The heterogeneity in electrode placement could influence the results, leading to inconsistent findings across studies.

In the present study, no differences in response latency were observed when assessing the entire lumbar region using HDsEMG. While HDsEMG offers greater precision in capturing muscle activity across the lumbar region, further studies are needed to confirm and generalize these findings.

Limitations

One limitation of this study is the lack of assessment of deep trunk muscles, such as the transversus abdominis and multifidus. These muscles could help explain the findings of this study, as their reflex responses are known to be altered in individuals with chronic low back pain (Hodges et al., 2001). Future research should include these muscles to have a comprehensive understanding of the impact of chronic LBP on the adaptability of the NS.

Another limitation is the normalization of RMS signals based on an MVC, which can be challenging in individuals with LBP. Pain may impair their ability to perform an MVC accurately, leading to potential inaccuracies in muscle activity normalization. However, the level of EMG activity during the first five trials was similar between groups, which suggests that this limitation may have had a minimal impact on our results.

Additionally, the participants were younger than the age range with the highest prevalence of LBP (around 50 to 55 years) (Ferreira et al., 2023). The age range of 18 to 60 years was selected to minimize the effects of aging on neuromuscular control in those over 60 (Crawford et al., 2016, Charles and Bates, 2023). However, this choice may limit the generalizability of our findings to the broader population.

Furthermore, the participants primarily had moderate levels of disability, low levels of kinesiophobia, low pain catastrophizing, and mild to moderate pain intensity. Consequently, the findings of this study may not be generalizable to individuals with higher levels of psychological impairments or more severe pain conditions. Future research should aim to include individuals with greater psychological impairments and higher pain intensities to provide a broader understanding of these results.

Finally, the current study did not differentiate between types of pain, such as nociceptive, nociplastic, or neuropathic, nor their relative contributions. Recent advances suggest that different pain types may have distinct impacts on motor control, highlighting the importance of differentiating pain types in future studies (Nijs et al., 2024, Shraim et al., 2022, Shraim et al., 2023). Furthermore, we did not include details on the duration of pain or whether the pain was radiating, which could limit the interpretation of the findings.

Conclusion

In conclusion, this study investigated the adaptability of trunk muscle responses to a series of unexpected external perturbations in patients with chronic LBP. The results showed that while individuals with chronic LBP failed to adapt the reflex response amplitude of lumbar extensor muscles, they were able to adapt the reflex contraction amplitude of trunk flexor muscles. These results may be attributed to impaired proprioception, reduced motor variability and neuroplastic changes commonly observed in individuals with

LBP. These changes might limit their ability to optimize responses, potentially compromising spinal stability and increasing functional cost.

REFERENCES

ABBOUD, J., DUCAS, J., MARINEAU-BéLANGER, É. & GALLINA, A. 2023. Lumbar muscle adaptations to external perturbations are modulated by trunk posture. *European Journal of Applied Physiology*, 1-12.

ABBOUD, J., KUO, C., DESCARREAUX, M. & BLOUIN, J. S. 2020. Regional activation in the human longissimus thoracis pars lumborum muscle. *The Journal of Physiology,* 598, 347-359.

ABBOUD, J., LARDON, A., BOIVIN, F., DUGAS, C. & DESCARREAUX, M. 2017. Effects of muscle fatigue, creep, and musculoskeletal pain on neuromuscular responses to unexpected perturbation of the trunk: a systematic review. *Frontiers in Human Neuroscience,* 10, 667.

ABBOUD, J., LESSARD, A. & DESCARREAUX, M. 2021. Superficial lumbar muscle recruitment strategies to control the trunk with delayed-onset muscle soreness. *European Journal of Applied Physiology,* 121, 2573-2583.

ABBOUD, J., NOUGAROU, F. & DESCARREAUX, M. 2016. Muscle activity adaptations to spinal tissue creep in the presence of muscle fatigue. *PloS one,* 11, e0149076.

AHERN, D. K., FOLLICK, M. J., COUNCIL, J. R., LASER-WOLSTON, N. & LITCHMAN, H. 1988. Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and non-patient controls. *Pain,* 34, 153-160.

ARVANITIDIS, M., BIKINIS, N., PETRAKIS, S., GKIOKA, A., TSIMPOLIS, D., FALLA, D. & MARTINEZ-VALDES, E. 2021. Spatial distribution of lumbar erector spinae muscle activity in individuals with and without chronic low back pain during a dynamic isokinetic fatiguing task. *Clinical Biomechanics,* 81, 105214.

BOGDUK, N. 2005. *Clinical anatomy of the lumbar spine and sacrum*, Elsevier Health Sciences.

BOURQUE, P. & BEAUDETTE, D. 1982. Étude psychometrique du questionnaire de dépression de Beck auprès d'un échantillon d'étudiants universitaires francophones. *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement,* 14, 211.

CASSISI, J. E., ROBINSON, M. E., O'CONNER, P. & MACMILLAN, M. 1993. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low-back pain patients and controls. *Spine,* 18, 245-251.

CHARLES, J. P. & BATES, K. T. 2023. The functional and anatomical impacts of healthy muscle ageing. *Biology,* 12, 1357.

CHOLEWICKI, J., SILFIES, S. P., SHAH, R. A., GREENE, H. S., REEVES, N. P., ALVI, K. & GOLDBERG, B. 2005. Delayed trunk muscle reflex responses increase the risk of low back injuries. *Spine,* 30, 2614-2620.

CIEZA, A., CAUSEY, K., KAMENOV, K., HANSON, S. W., CHATTERJI, S. & VOS, T. 2020. Global estimates of the need for rehabilitation based on the Global Burden of Disease study 2019: a systematic analysis for the Global Burden of Disease Study 2019. *The Lancet,* 396**,** 2006-2017.

CRAWFORD, R. J., FILLI, L., ELLIOTT, J., NANZ, D., FISCHER, M., MARCON, M. & ULBRICH, E. 2016. Age-and level-dependence of fatty infiltration in lumbar paravertebral muscles of healthy volunteers. *American Journal of Neuroradiology,* 37**,** 742-748.

CRISWELL, E. 2010. Cram's introduction to surface electromyography. *Jones &Bartlett Learning*.

DUBOIS, J.-D., ABBOUD, J., ST-PIERRE, C., PICHé, M. & DESCARREAUX, M. 2014. Neuromuscular adaptations predict functional disability independently of clinical pain and psychological factors in patients with chronic non-specific low back pain. *Journal of Electromyography and Kinesiology,* 24**,** 550-557.

FAUL, F., ERDFELDER, E., LANG, A.-G. & BUCHNER, A. 2007. G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior research methods,* 39**,** 175-191.

FERREIRA, M. L., DE LUCA, K., HAILE, L. M., STEINMETZ, J. D., CULBRETH, G. T., CROSS, M., KOPEC, J. A., FERREIRA, P. H., BLYTH, F. M. & BUCHBINDER, R. 2023. Global, regional, and national burden of low back pain, 1990–2020, its attributable risk factors, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. *The Lancet Rheumatology,* 5**,** e316-e329.

FREDDOLINI, M., STRIKE, S. & LEE, R. Y. 2014. The role of trunk muscles in sitting balance control in people with low back pain. *Journal of Electromyography and Kinesiology,* 24**,** 947-953.

FRENCH, D. J., NOëL, M., VIGNEAU, F., FRENCH, J. A., CYR, C. P. & EVANS, R. T. 2005. L'Échelle de dramatisation face à la douleur PCS-CF: Adaptation canadienne en langue française de l'échelle «Pain Catastrophizing Scale». *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement,* 37**,** 181.

FRENCH, D. J., ROACH, P. J. & MAYES, S. 2002. Peur du mouvement chez des accidentés du travail: L'Échelle de Kinésiophobie de Tampa (EKT). *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement,* 34**,** 28.

GALLINA, A., ABBOUD, J. & BLOUIN, J.-S. 2024. Vestibular control of deep and superficial lumbar muscles. *Journal of Neurophysiology,* 131**,** 516-528.

GALLINA, A., DISSELHORST-KLUG, C., FARINA, D., MERLETTI, R., BESOMI, M., HOLOBAR, A., ENOKA, R. M., HUG, F., FALLA, D. & SøGAARD, K. 2022. Consensus for experimental design in electromyography (CEDE) project: High-density surface electromyography matrix. *Journal of Electromyography and Kinesiology,* 64**,** 102656.

GAO, Y., SHI, J.-G., YE, H., LIU, Z.-R., ZHENG, L.-B., NI, Z.-M., FAN, L.-Q., WANG, J. & HOU, Z.-H. 2014. Adaptation of muscles of the lumbar spine to sudden imbalance in patients with lower back pain caused by military training. *The journal of spinal cord medicine,* 37**,** 774-781.

GILDEA, J. E., HIDES, J. A. & HODGES, P. W. 2013. Size and symmetry of trunk muscles in ballet dancers with and without low back pain. *Journal of orthopaedic & sports physical therapy,* 43**,** 525-533.

HODGES, P., CRESSWELL, A. & THORSTENSSON, A. 2001. Perturbed upper limb movements cause short-latency postural responses in trunk muscles. *Experimental Brain Research,* 138**,** 243-250.

HODGES, P. W. & BUI, B. H. 1996. A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography. *Electroencephalography and Clinical Neurophysiology/Electromyography and Motor Control,* 101**,** 511-519.

HODGES, P. W. & DANNEELS, L. 2019. Changes in structure and function of the back muscles in low back pain: different time points, observations, and mechanisms. *Journal of orthopaedic & sports physical therapy,* 49**,** 464-476.

HODGES, P. W. & TUCKER, K. 2011. Moving differently in pain: a new theory to explain the adaptation to pain. *Pain,* 152**,** S90-S98.

JONES, S. L., HENRY, S. M., RAASCH, C. C., HITT, J. R. & BUNN, J. Y. 2012a. Individuals with non-specific low back pain use a trunk stiffening strategy to maintain upright posture. *Journal of Electromyography and Kinesiology,* 22**,** 13-20.

JONES, S. L., HITT, J. R., DESARNO, M. J. & HENRY, S. M. 2012b. Individuals with non-specific low back pain in an active episode demonstrate temporally altered torque responses and direction-specific enhanced muscle activity following unexpected balance perturbations. *Experimental brain research,* 221**,** 413-426.

KANTAK, S. S., JOHNSON, T. & ZARZYCKI, R. 2022. Linking pain and motor control: conceptualization of movement deficits in patients with painful conditions. *Physical Therapy,* 102**,** pzab289.

LARIVIèRE, C., FORGET, R., VADEBONCOEUR, R., BILODEAU, M. & MECHERI, H. 2010. The effect of sex and chronic low back pain on back muscle reflex responses. *European journal of applied physiology,* 109**,** 577-590.

LEINONEN, V., KANKAANPää, M., LUUKKONEN, M., HäNNINEN, O., AIRAKSINEN, O. & TAIMELA, S. 2001. Disc herniation-related back pain impairs feed-forward control of paraspinal muscles. *Spine,* 26**,** E367-E372.

LIEBETRAU, A., PUTA, C., ANDERS, C., DE LUSSANET, M. H. & WAGNER, H. 2013. Influence of delayed muscle reflexes on spinal stability: model-based predictions allow alternative interpretations of experimental data. *Human movement science,* 32**,** 954-970.

LIMA, M., FERREIRA, A. S., REIS, F. J. J., PAES, V. & MEZIAT-FILHO, N. 2018. Chronic low back pain and back muscle activity during functional tasks. *Gait & posture,* 61**,** 250-256.

MADELEINE, P. 2010. On functional motor adaptations: from the quantification of motor strategies to the prevention of musculoskeletal disorders in the neck–shoulder region. *Acta Physiologica,* 199**,** 1-46.

MARDULYN, T., DELAFONTAINE, A., JISSENDI, P. & FABECK, L. 2025. Lumbar Muscle Fatty Infiltration and Atrophy in Patients with Low Back Pain and Degenerative Spinal Pathologies: A CT Imaging Study. *Journal of Clinical Medicine,* 14**,** 2125.

MATHEVE, T., HODGES, P. & DANNEELS, L. 2023. The Role of Back Muscle Dysfunctions in Chronic Low Back Pain: State-of-the-Art and Clinical Implications. *Journal of Clinical Medicine,* 12**,** 5510.

MEIER, M. L., VRANA, A. & SCHWEINHARDT, P. 2019. Low back pain: the potential contribution of supraspinal motor control and proprioception. *The Neuroscientist,* 25**,** 583-596.

MILLER, E. M., BAZRGARI, B., NUSSBAUM, M. A. & MADIGAN, M. L. 2013. Effects of exercise-induced low back pain on intrinsic trunk stiffness and paraspinal muscle reflexes. *Journal of biomechanics,* 46**,** 801-805.

NICHOLAS, M., VLAEYEN, J. W., RIEF, W., BARKE, A., AZIZ, Q., BENOLIEL, R., COHEN, M., EVERS, S., GIAMBERARDINO, M. A. & GOEBEL, A. 2019. The IASP classification of chronic pain for ICD-11: chronic primary pain. *Pain,* 160**,** 28-37.

NIJS, J., KOSEK, E., CHIAROTTO, A., COOK, C., DANNEELS, L. A., FERNáNDEZ-DE-LAS-PEñAS, C., HODGES, P. W., KOES, B., LOUW, A. & OSTELO, R. 2024. Nociceptive, neuropathic, or nociplastic low back pain? The low back pain phenotyping (BACPAP) consortium's international and multidisciplinary consensus recommendations. *The Lancet Rheumatology*.

RADEBOLD, A., CHOLEWICKI, J., PANJABI, M. M. & PATEL, T. C. 2000. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine,* 25**,** 947-954.

RADEBOLD, A., CHOLEWICKI, J., POLZHOFER, G. K. & GREENE, H. S. 2001. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine,* 26**,** 724-730.

SANDERSON, A., CESCON, C., HENEGHAN, N. R., KUITHAN, P., MARTINEZ-VALDES, E., RUSHTON, A., BARBERO, M. & FALLA, D. 2019a. People with low back pain display a different distribution of erector spinae activity during a singular mono-planar lifting task. *Frontiers in Sports and Active Living,* 1**,** 65.

SANDERSON, A., CESCON, C., MARTINEZ-VALDES, E., RUSHTON, A., HENEGHAN, N., KUITHAN, P., BARBERO, M. & FALLA, D. 2024. Reduced variability of erector spinae activity in people with chronic low back pain when performing a functional 3D lifting task. *Journal of Electromyography and Kinesiology,* 78**,** 102917.

SANDERSON, A., MARTINEZ-VALDES, E., HENEGHAN, N. R., MURILLO, C., RUSHTON, A. & FALLA, D. 2019b. Variation in the spatial distribution of erector spinae activity during a lumbar endurance task in people with low back pain. *Journal of anatomy,* 234**,** 532-542.

SHENOY, S., BALACHANDER, H. & SANDHU, J. 2013. Long latency reflex response of superficial trunk musculature in athletes with chronic low back pain. *Journal of Back and Musculoskeletal Rehabilitation,* 26**,** 445-450.

SHRAIM, M. A., MASSé-ALARIE, H., SALOMONI, S. E. & HODGES, P. W. 2023. The effect of skilled motor training on corticomotor control of back muscles in different presentations of low back pain. *Journal of Electromyography and Kinesiology,* 71**,** 102782.

SHRAIM, M. A., SLUKA, K. A., STERLING, M., ARENDT-NIELSEN, L., ARGOFF, C., BAGRAITH, K. S., BARON, R., BRISBY, H., CARR, D. B. & CHIMENTI, R. L. 2022. Features and

methods to discriminate between mechanism-based categories of pain experienced in the musculoskeletal system: a Delphi expert consensus study. *Pain,* 163**,** 1812-1828.

SKOTTE, J. H., FALLENTIN, N., PEDERSEN, M., ESSENDROP, M., STRøYER, J. & SCHIBYE, B. 2004. Adaptation to sudden unexpected loading of the low back—the effects of repeated trials. *Journal of Biomechanics,* 37**,** 1483-1489.

TONG, M. H., MOUSAVI, S. J., KIERS, H., FERREIRA, P., REFSHAUGE, K. & VAN DIEëN, J. 2017. Is there a relationship between lumbar proprioception and low back pain? A systematic review with meta-analysis. *Archives of physical medicine and rehabilitation,* 98**,** 120-136. e2.

TSAO, H., DANNEELS, L. A. & HODGES, P. W. 2011. ISSLS prize winner: smudging the motor brain in young adults with recurrent low back pain. *Spine,* 36**,** 1721-1727.

VAN DIEëN, J. H., CHOLEWICKI, J. & RADEBOLD, A. 2003a. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine,* 28**,** 834-841.

VAN DIEëN, J. H., SELEN, L. P. & CHOLEWICKI, J. 2003b. Trunk muscle activation in low-back pain patients, an analysis of the literature. *Journal of electromyography and kinesiology,* 13**,** 333-351.

VOGLER, D., PAILLEX, R., NORBERG, M., DE GOUMOENS, P. & CABRI, J. Cross-cultural validation of the Oswestry disability index in French. Annales de readaptation et de medecine physique: revue scientifique de la Societe francaise de reeducation fonctionnelle de readaptation et de medecine physique, 2008. 379-385.

WOBY, S. R., ROACH, N. K., URMSTON, M. & WATSON, P. J. 2005. Psychometric properties of the TSK-11: a shortened version of the Tampa Scale for Kinesiophobia. *Pain,* 117**,** 137-144.

WU, A., MARCH, L., ZHENG, X., HUANG, J., WANG, X., ZHAO, J., BLYTH, F. M., SMITH, E., BUCHBINDER, R. & HOY, D. 2020. Global low back pain prevalence and years lived with disability from 1990 to 2017: estimates from the Global Burden of Disease Study 2017. *Annals of translational medicine,* 8.

ZEDKA, M., PROCHAZKA, A., KNIGHT, B., GILLARD, D. & GAUTHIER, M. 1999. Voluntary and reflex control of human back muscles during induced pain. *The Journal of physiology,* 520 Pt 2**,** 591-604.