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A predictive model for classifying low back pain status based on lumbopelvic kinematics measured using inertial measurement units: a cross-sectional study

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Abstract

Background Low back pain (LBP) is a leading cause of disability worldwide. Impaired lumbopelvic control contributes to chronic and recurrent LBP, often presenting as aberrant movement patterns. This study aimed to investigate whether inertial measurement units (IMUs) can classify individuals with no LBP (NoLBP), chronic LBP (CLBP), and a history of LBP (HxLBP) based on lumbopelvic kinematics.

Methods A total of 141 participants (47 per group) performed ten standardized lumbopelvic movement control tests while wearing IMU sensors. Kinematic parameters, including mean velocity (MV), peak-to-peak amplitude (P2P), and area under the curve (AUC) of acceleration, were extracted. One-way ANOVA was used to compare kinematic differences across groups, and binary logistic regression models were developed to identify predictors for classification. Robustness analyses using 10-fold cross-validation with the least absolute shrinkage and selection operator (LASSO) were also performed.

Results Significant group differences were found in MV, P2P, and AUC across multiple movement tests ($P < 0.05$). The most pronounced differences were observed between NoLBP and CLBP: individuals with CLBP were characterized by slower trunk flexion (odds ratio [OR] = 0.94, 95% CI: 0.90–0.98), greater AUC during prone hip rotation (OR = 2.78, 95% CI: 1.45–5.34), and greater P2P during trunk rotation (OR = 1.32, 95% CI: 1.12–1.55). Robustness analyses confirmed the robustness and stability of the classification models.

Conclusion IMU-derived kinematic parameters provide objective measures of impaired movement control and may support clinical identification of individuals at risk for chronic or recurrent LBP.

Keywords History of low back pain, Kinematics, Movement control, Inertial measurement units, Classification model

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Introduction

Low back pain (LBP) is a highly prevalent health condition and the leading cause of years lived with disability worldwide [1]. Spinal dysfunction, specifically impaired lumbopelvic control, is considered a major contributor to chronic and recurrent LBP [2–4]. This impairment is often characterized by clinically observed aberrant movement patterns during functional movements, such as slow motion, high movement variability, and delayed activation of stabilizer muscles [2, 4, 5]. Greater lumbar excursion during prone hip extension and prolonged standing has been noted in LBP patients [6, 7].

Clinical assessment of lumbopelvic movement control relies heavily on visual observation, which is subjective and prone to inter-rater variability. While these methods are valuable, they may lack the sensitivity to detect subtle movement impairment, particularly in individuals with a history of LBP (HxLBP) who may be currently asymptomatic. This presents a significant clinical gap, as HxLBP is a strong predictor of future LBP recurrence [8].

Subtle alterations in lumbopelvic control in individuals with LBP and HxLBP may not be readily detectable through clinical observation. To address this limitation, objective kinematic assessments using inertial measurement units (IMUs) offer a promising solution. IMUs are portable, cost-effective tools capable of quantifying three-dimensional motion through acceleration and angular velocity measurements [9–11]. Kinematic parameters, such as mean velocity (MV), peak-to-peak amplitude (P2P), and area under the curve (AUC) of acceleration, have been shown to be sensitive to detect movement control impairments, including instability catch (sudden deceleration and acceleration during movement) and out-of-plane deviations (movement away from the primary plane of movement) [9, 10].

Despite HxLBP being a known risk factor for recurrent LBP, limited studies have examined kinematic profiles across the continuum of LBP, including asymptomatic individuals with prior episodes. This study aimed to determine whether IMU-derived kinematic parameters can distinguish between individuals with no LBP (NoLBP), chronic LBP (CLBP), and HxLBP. Furthermore, we aimed to develop a classification model using these parameters to aid in early detection and inform targeted rehabilitation strategies for preventing chronic or recurrent LBP. We hypothesized that (1) individuals with CLBP would exhibit significantly reduced movement velocity and greater kinematic irregularity (i.e., higher P2P and AUC values) compared to NoLBP, (2) individuals with HxLBP would demonstrate intermediate kinematic profiles, slower than NoLBP but more stable than those with CLBP, and (3) specific IMU-derived parameters could classify participants into the three groups using logistic regression models.

Methods

Study design

This study employed a cross-sectional observational design to evaluate lumbopelvic kinematics across three groups: NoLBP, CLBP, and HxLBP. This study was approved by the University Institutional Review Board (COA No. MU-CIRB 2020/084.1806) and adhered to the principles outlined in the Declaration of Helsinki. Written informed consent for publication of identifying information/images in an online open-access publication was also obtained.

Participants

Participants aged between 20 and 40 years were recruited using a convenience sample. We have purposely selected this age because related studies have demonstrated that individuals with LBP aged below 40 years were more likely to have impaired lumbopelvic control and would benefit from motor control exercise. In addition, individuals older than 40 years were more likely to have a specific low back condition, such as degenerative spine, spondylosis or spinal stenosis. Recruitment was conducted via advertisements and word of mouth within the university and surrounding communities. The inclusion criteria for the NoLBP group included no LBP episodes requiring treatment or affecting daily activities within the past 12 months. For the HxLBP group, participants were included if they had experienced at least two LBP episodes in the past six months that interfered with function or required treatment [3, 8, 12]. Participants could have intermittent ('on-and-off') symptoms during this period but were asymptomatic on the day of testing. The CLBP group included individuals with active LBP persisting for more than three months and a pain intensity of 3–6 out of 10 on the numerical pain rating scale (NPRS) within the last 24 h [1, 13]. Exclusion criteria for all participants included systemic diseases, neurological signs, prior spinal surgery, inflammatory joint disease, osteoporosis, pregnancy, musculoskeletal problems outside the lumbar region (e.g., hip, knee, or shoulder pathology) that could affect trunk or lumbopelvic movement, vestibular dysfunction, severe psychosocial issues, or concurrent medical treatment that would prevent participation. Based on the sample size guideline for discriminant analysis [14], the formula $n = i \times 20$, where i is the number of predictive variables. A previous study identified seven relevant kinematic parameters differentiating movement patterns [9], resulting in a target of at least 140 participants. Thus, 141 participants (NoLBP = 47, CLBP = 47, and HxLBP = 47) were enrolled, ensuring balanced representation across the three groups.

Instruments and measures

Inertial Measurement Units (IMUs) (Delsys Trigno, Delsys Inc., Boston, MA, USA) were used to measure lumbopelvic motion. Five IMU sensors were placed at the T3, L1, and S1 spinous processes, as well as bilaterally on the femur (5 cm. superior to the lateral epicondyle) or ankle (5 cm. superior to the lateral malleolus), depending on the specific movement test (Fig. 1). Data were acquired using EMGworks Acquisition software (version 4.7.8) at 370 Hz, which is the manufacturer-specified sampling rate of the IMU system. This system has been validated with an optical motion capture system and used in several studies [9, 15, 16]. Our previous study demonstrated excellent test-retest reliability, using movement pattern consistency (coefficient of multiple determination = 0.85) [16].

Procedure

Demographic data (age, sex, BMI) were collected. Clinical data for the HxLBP and CLBP groups, including pain scale scores, disability levels, onset characteristics, duration, and frequency of episodes, were collected using the NIH Minimal Data Set [13]. Participants also completed the Modified Oswestry Disability Questionnaire (MODQ) [17] and the Tampa scale of kinesiophobia (TSK) [18]. These clinical measures were collected to describe symptom severity and functional impact (and to confirm group classification), thereby enabling interpretation of the clinical relevance and generalizability of the kinematic findings, even though these measures were not entered as predictors in the primary analyses. After demographic and clinical data collection, participants were asked to expose their lumbopelvic area, and the researcher attached IMU sensors on body landmarks.

Ten movement control tests were used to assess lumbopelvic movement control (Fig. 2). These tests were selected based on four key criteria: (1) clinical feasibility—simple and time-efficient to perform in both research and clinical settings; (2) ease of administration—requiring minimal equipment and space; (3) established inter-rater agreement in identifying aberrant movement patterns; and (4) compatibility with IMU sensor placement, minimizing signal interference or sensor dislocation during dynamic tasks.

The selected tasks represent a broad spectrum of functional movements commonly affected in patients with low back pain, encompassing sagittal, frontal, and transverse plane control of the trunk and pelvis. This multidimensional approach aligns with previous literature emphasizing the need for plane-specific evaluation of motor control impairments in LBP populations [4, 5].

Moreover, the test battery includes positions that vary in weight-bearing demand (e.g., standing, quadruped, sitting, and prone), which is crucial for detecting task-specific deficits that may not appear in static or single-plane assessments. Similar multi-positional movement control tests have demonstrated clinical utility and reliability in identifying movement impairments in both symptomatic and asymptomatic individuals [19–21]. Importantly, the selected movements have minimal overlap in movement strategy, reducing redundancy and allowing for comprehensive analysis of neuromuscular control across different contexts while maintaining validity in IMU-based kinematic capture.

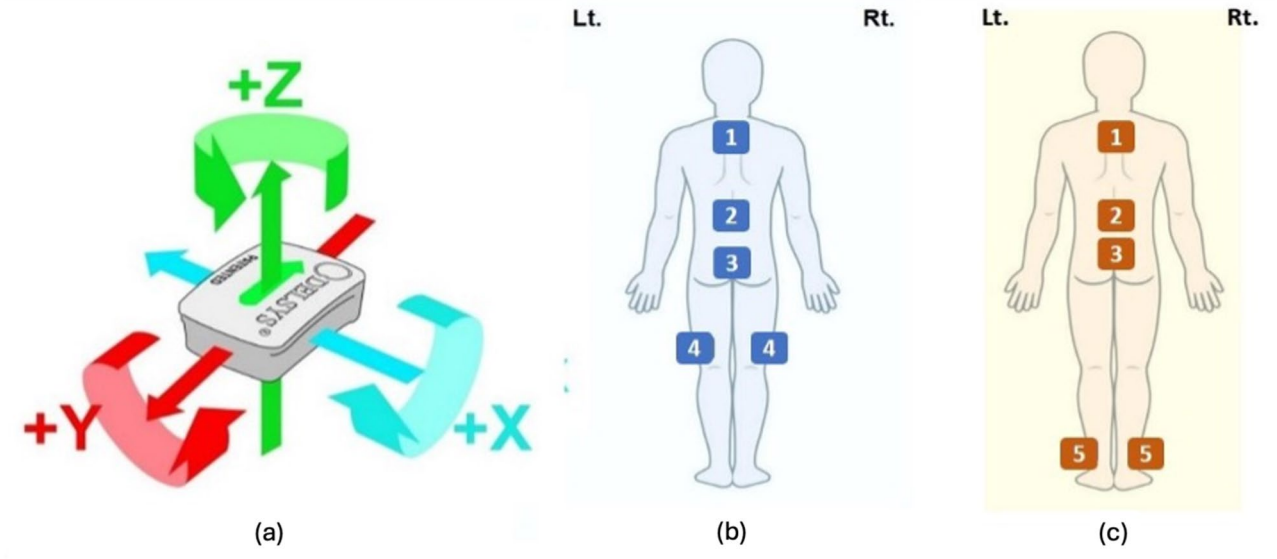


Fig. 1 Delsys sensors sensing axis (**a**), body landmark and sensor locations (**b**) and (**c**). Sensor 1 is a thoracic sensor over the spinous process T3. Sensor 2 is a lumbar sensor over the spinous process L1. Sensor 3 is the pelvis sensor over the process of S1. Sensors 4,5 are the femoral and ankle sensors (depending on the tests) over 5 cm. superior to the right and left femoral lateral epicondyle and lateral malleolus, respectively

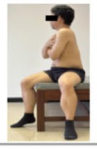
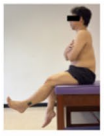
Lumbopelvic movement tests	Start and ending position	Picture
1. Trunk flexion	**Starting position**: Standing with feet shoulder-width apart **Ending position**: Trunk flexion with lumbar flexion and knee extension	
2. Trunk extension	**Starting position**: Standing with feet shoulder width apart and hands on the pelvis **Ending position**: Trunk extension with lumbar and knee extension	
3. Trunk lateral bending	**Starting position**: Standing with feet shoulder-width apart **Ending position**: Trunk lateral bend to right or left side	
4. Trunk rotation in sitting	**Starting position**: Sitting straight with hands on the shoulders **Ending position**: Trunk rotation to the right or left side	
5. Quadruped backward	**Starting position**: 4-point-kneeling, hips in 90° flexion, with a slightly curved lower back **Ending position**: Pelvis moving backward to 120° hip flexion	
6. Quadruped forward	**Starting position**: 4-point-kneeling, hips in 90° flexion, with a slightly curved lower back **Ending position**: Pelvis moving forwards to 60° hip flexion	
7. Sit with knee extension	**Starting position**: Sitting straight **Ending position**: Sitting with knee extension	
8. Prone with knee flexion	**Starting position**: Prone with hip in the neutral position **Ending position**: Knee flexion with 90°	
9. Prone with hip rotation	**Starting position**: Prone with hip in the neutral position and knee flexion at 90° **Ending position**: Prone with hip in the neutral position and knee flexion at 90°	
10. Prone with hip extension	**Starting position**: Prone with hip in the neutral position **Ending position**: Prone with hip in extension	

Fig. 2 Lumbopelvic movement tests

The researcher provided standardized verbal instructions and demonstrations for the participants. Practice trials were provided to ensure that the participants understood the test. While the participants were performing the test, there was no corrective feedback or command from the raters or researcher. All participants were asked to perform these tests in a random sequence (3 consecutive repetitions for each test), while kinematic data were concurrently recorded.

Data analysis

Kinematic data were processed using LabVIEW software version 2012 (National Instruments, USA). Raw IMU data were filtered using a second-order zero-phase low-pass Butterworth filter with a 10 Hz cut-off frequency [9]. Start and stop events were identified with a cut-off threshold of 5% of maximum velocity. Data were time-normalized to 101 data points (100% of the movement cycle) [9]. Each task was performed three times, and time-normalized data from the three repetitions were averaged point-by-point to generate a single composite waveform for parameter calculation, thereby reducing trial-to-trial variability while preserving the representative movement pattern. Lumbar angular velocity during trunk flexion, extension, lateral bending, and sitting with trunk rotation was used to calculate mean velocity (MV) and angular acceleration. Pelvic MV and angular acceleration were measured during sitting with knee extension, quadruped forward and backward movement, prone hip extension, prone knee flexion, and prone hip rotation, and peak-to-peak amplitude (P2P) and area under the curve (AUC) were also calculated [9, 22]. Figure 3 illustrates a kinematic analysis workflow for deriving kinematic parameters from IMU data. Test-retest reliability of these kinematic parameters was assessed in our previous study, demonstrating moderate to excellent reliability ($ICC_{2,k}$ = 0.95, 0.72, and 0.91, respectively) [9]. The 95% confidence minimal detectable change (MDC) values were 1.9 occurrences, 0.98 deg/sec (P2P), and 16.71 units (Area), respectively. P2P and deviation (DEV) of the secondary plane of movement were identified, indicating the out-of-plane deviations.

Although NoLBP, HxLBP, and CLBP may represent points on a continuum, we analyzed them as discrete categories to facilitate pairwise classification and enhance clinical interpretability. This categorical approach has been used in prior LBP research to reveal distinct motor control strategies and kinematic differences among patient subgroups [3, 22, 23]. Direct binary comparisons allowed us to evaluate which kinematic parameters best differentiate each clinically relevant grouping.

Statistical analysis

Statistical analyses were performed using SPSS Software, Version 22.0 (IBM Corp., Armonk, NY, USA). Descriptive statistical analysis was performed on the demographic

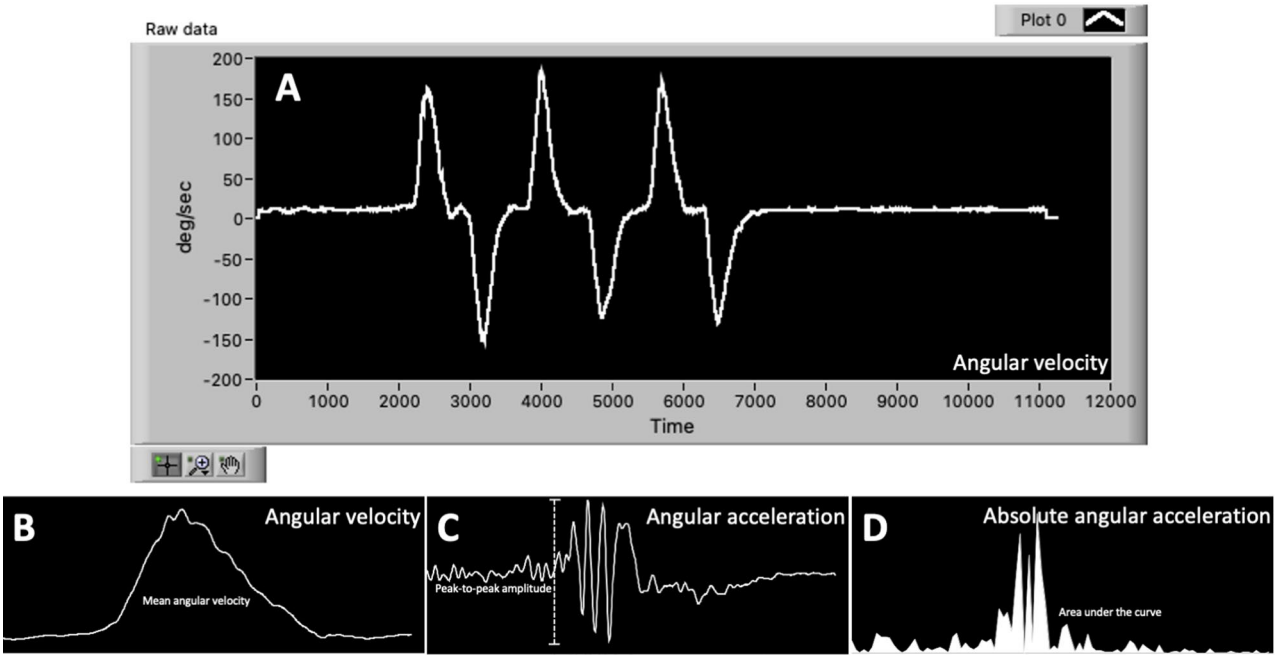


Fig. 3 An example of a kinematic analysis workflow for deriving kinematic parameters from IMU data: **A** Representative raw trunk flexion angular velocity trace (deg/s) over time for one trial (3 repetitions), **B** Mean angular velocity (MV) calculated over one repetition (forward bend phase), **C** Angular acceleration (first derivative of angular velocity during forward bend phase) used to compute peak-to-peak amplitude (P2P; difference between maximum and minimum values) to capture sudden deceleration–acceleration events, and **D** Absolute angular acceleration used to compute the area under the curve (AUC), reflecting the overall magnitude of acceleration–deceleration over time

data. Normality was assessed using the Shapiro-Wilk test, and homogeneity of variances was evaluated using Levene's test. We found that our data were normally distributed, and the homogeneity of variances was assumed. Between-group comparisons of demographic variables (e.g., age, BMI) were conducted using one-way ANOVA, and chi-square tests were used for categorical variables. Clinical data between the CLBP and HxLBP groups (e.g., onset, episode frequency, time since last episode, pain intensity, MODQ, and TSK) were compared using independent t-tests. The significance level was set at $P < 0.05$. Group comparisons of kinematic parameters among NoLBP, CLBP, and HxLBP were performed using one-way ANOVA. Post-hoc analyses were conducted using the least significant difference (LSD) test due to its sensitivity in detecting group-level differences.

Kinematic parameters with P-values < 0.05 from the post-hoc LSD tests were entered into binary logistic regression models to classify group membership between each pair: (1) NoLBP vs. CLBP, (2) NoLBP vs. HxLBP, and (3) CLBP vs. HxLBP. Binary outcomes were coded as 0 or 1. Prior to modeling, multicollinearity was assessed using the variance inflation factor (VIF < 10) and tolerance (> 0.1), and linearity of continuous predictors with the logit was tested using the Box-Tidwell transformation.

A stepwise model selection approach was applied ($P < 0.05$ for entry, $P > 0.10$ for removal). Model fit was evaluated using the Hosmer-Lemeshow goodness-of-fit test ($P > 0.05$), Cox & Snell and Nagelkerke pseudo-R^2 values, and classification accuracy. Regression results were reported as unstandardized coefficients (B), standard errors (SE), odds ratios (OR), and 95% confidence intervals (CI).

To assess the robustness of the classification models, additional analyses were performed using 10-fold stratified cross-validation with shuffling and logistic regression with least absolute shrinkage and selection operator (LASSO, L1) regularization. Ten-fold cross-validation provides reliable estimates of out-of-sample performance by reducing variance compared with single-sample splits. LASSO regularization constrains model complexity, mitigates multicollinearity, and improves generalizability by penalizing less informative predictors [24, 25]. Robustness analyses were performed using Python (version 3.11, scikit-learn library). Together, these methods provide stronger evidence for the stability and reproducibility of our findings.

Results

Demographic and clinical characteristics of the NoLBP, CLBP, and HxLBP groups

A total of 141 participants completed the study, with 47 participants in each of the three groups (NoLBP, CLBP, and HxLBP). No significant differences were observed in baseline demographic characteristics (age, sex, BMI) among the three groups. However, there was a significant difference in the time since the last episode between the CLBP and HxLBP groups ($P < 0.05$), as shown in Table 1.

Comparison of the different kinematic parameters across ten lumbopelvic movement tests among NoLBP, CLBP, and HxLBP

One-way ANOVA revealed significant differences ($P < 0.05$) in kinematic parameters across the three groups in four of the ten lumbopelvic movement tests (Table 2). Post-hoc analysis using LSD indicated that the mean velocity of trunk flexion (TF_MV) was significantly higher in the NoLBP group compared to both the CLBP and HxLBP groups, suggesting faster trunk flexion among asymptomatic individuals. Conversely, the mean

Table 1 Demographic and clinical characteristics of the NoLBP, CLBP, and HxLBP participants

Variables	Participants ($n = 141$)			*P*-value
	NoLBP ($n = 47$) Mean ± SD	CLBP ($n = 47$) Mean ± SD	HxLBP ($n = 47$) Mean ± SD	
Age (years)	30.2 ± 5.4	30.1 ± 5.9	29.9 ± 6.0	0.96
Sex (%female)	72.34%	78.72%	59.57%	0.12
Height (m)	1.64 ± 0.07	1.62 ± 0.08	1.65 ± 0.09	0.174
Weight (kg)	61.6 ± 11.7	63.5 ± 15.6	63.8 ± 15.6	0.690
BMI (kg/m^2)	22.9 ± 4.0	24 ± 4.9	23.1 ± 4.4	0.45
Onset (months)	N/A	14.7 ± 11.0	12.2 ± 11.8	0.84
Frequency of episodes (per year)	N/A	28.6 ± 33.0	14.77 ± 28.7	0.12
Time since the last episode (days)	N/A	6.09 ± 8.1	40.7 ± 41.4	< 0.001*
Duration of an episode (days)	N/A	2.4 ± 2.0	4.7 ± 13	0.06
Pain intensity during the episode (0 = no pain, 10 = worst pain that can be imagined)	N/A	4.6 ± 1.5	4.1 ± 1.4	0.60
MODQ (0–100%)	N/A	16.26 ± 12.21	14.4 ± 12.68	0.40
TSK (17–68)	N/A	37.74 ± 6.6	38.16 ± 5.6	0.75

NoLBP no low back pain, *CLBP* chronic low back pain, *HxLBP* history of low back pain, *BMI* body mass index, *MODQ* Modified Oswestry Disability Questionnaire, *TSK* Tampa Scale of Kinesiophobia, *N/A* not applicable; * $P < 0.05$

Table 2 Kinematic parameters and group comparisons among NoLBP, CLBP, and HxLBP

Variable	NoLBP Mean ± SD	CLBP Mean ± SD	HxLBP Mean ± SD	ANOVA		Post-hoc Comparisons	
				P-value	Effect size (Eta squared)	Mean Difference (*P*-value)	Effect size (Cohen's d)
TF_MV (deg/sec)	56.53 ± 15.10	50.07 ± 14.45	49.40 ± 13.51	0.032	0.049	NoLBP > CLBP: 6.46 (0.031)	0.437
						NoLBP > HxLBP: 7.13 (0.017)	0.498
LB_R_MV (deg/sec)	8.23 ± 3.20	8.18 ± 3.22	6.49 ± 4.74	0.045	0.044	HxLBP < NoLBP: -1.73 (0.028)	0.428
						HxLBP < CLBP: -1.68 (0.033)	0.416
LB_R_P2PF (deg/sec)	0.09 ± 0.05	0.13 ± 0.08	0.08 ± 0.05	0.004	0.077	CLBP > NoLBP: 0.034 (0.012)	0.478
						CLBP > HxLBP: 0.043 (0.002)	0.629
LB_R_AUCF (units)	0.84 ± 0.80	1.18 ± 1.00	0.66 ± 0.48	0.006	0.072	CLBP > NoLBP: 0.34 (0.039)	0.376
						CLBP > HxLBP: 0.52 (0.002)	0.669
PHR_R_P2PT (deg/sec)	0.06 ± 0.03	0.09 ± 0.05	0.07 ± 0.05	0.020	0.055	CLBP > NoLBP: 0.029 (0.005)	0.601
PHR_R_AUCT (units)	1.00 ± 0.74	1.47 ± 0.97	1.10 ± 0.73	0.016	0.058	CLBP > NoLBP: 0.475 (0.006)	0.547
						CLBP > HxLBP: 0.368 (0.032)	0.427
Rot_L_P2PF (deg/sec)	7.79 ± 3.12	10.60 ± 5.72	8.20 ± 4.17	0.005	0.073	CLBP > NoLBP: 2.81 (0.003)	0.610
						CLBP > HxLBP: 2.4 (0.01)	0.479
Rot_L_DEVF (units)	231.75 ± 96.02	294.78 ± 158.66	239.92 ± 118.62	0.036	0.047	CLBP > NoLBP: 63.03 (0.018)	0.481
						CLBP > HxLBP: 54.86 (0.038)	0.392

NoLBP No low back pain, *CLBP* Chronic low back pain, *HxLBP* History of low back pain, *TF_MV* mean velocity of trunk flexion, *LB_R_MV* mean velocity of lateral bend to right, *LB_R_P2PF* peak-to-peak of sudden deceleration and acceleration in the frontal plane of lateral bend to right, *LB_R_AUCF* area under the curve of sudden deceleration and acceleration in the frontal plane of lateral bend to right, *PHR_R_P2PT* peak-to-peak of sudden deceleration and acceleration in the transverse plane of prone right hip rotation, *PHR_R_AUCT* area under the curve of sudden deceleration and acceleration in the transverse plane of prone right hip rotation, *Rot_L_P2PF* peak-to-peak of sudden deceleration and acceleration in the frontal plane of sitting with left rotation, *Rot_L_DEVF* deviation of sudden deceleration and acceleration in the frontal plane of sitting with left rotation

Table 3 Logistic regression models for group classification

Comparison	Parameter	B	S.E.	OR (95% CI)	*P*-value
NoLBP vs. CLBP[a]	TF_MV	-0.06	0.02	0.937 (0.89–0.97)	< 0.001
	PHR_R_AUCT	1.02	0.33	2.78 (1.44–5.33)	0.002
	Rot_L_P2PF	0.27	0.08	1.31 (1.12–1.55)	< 0.001
NoLBP vs. HxLBP[a]	TF_MV	-0.03	0.01	0.95 (0.92–0.98)	0.006
CLBP vs. HxLBP[b]	LB_R_AUCF	-0.96	0.36	0.38 (0.18–0.77)	0.008
	PHR_R_AUCT	-0.81	0.34	0.44 (0.22–0.86)	0.017

NoLBP No low back pain, *CLBP* Chronic low back pain, *HxLBP* History of low back pain, *B* Unstandardized coefficient, *S.E.* Standard error, *OR* Odds ratio, *CI* Confidence interval, *TF_MV* mean velocity in trunk flexion, *PHR_R_AUCT* area under the curve of sudden deceleration and acceleration in transverse plane of prone right hip rotation, *Rot_L_P2PF* peak-to-peak of sudden deceleration and acceleration in the frontal plane of sitting with left rotation, *LB_R_AUCF* Area under the curve in the frontal plane of lateral bend to right

[a] Reference category "No low back pain"

[b] Reference category "Chronic low back pain"

velocity of lateral bending to the right (LB_R_MV) was significantly lower in the HxLBP group than in both the NoLBP and CLBP groups, indicating reduced lateral bending velocity. Furthermore, the CLBP group exhibited significantly higher values for (1) peak-to-peak of sudden deceleration and acceleration in the frontal plane during the lateral bend to the right (LB_R_P2PF), (2) area under the curve of sudden deceleration and acceleration during frontal plane lateral bending to the right (LB_R_AUCF), (3) area under the curve of sudden deceleration and acceleration in the transverse plane during prone right hip rotation (PHR_R_AUCT), (4) peak-to-peak of sudden deceleration and acceleration during frontal plane sitting with left rotation (Rot_L_P2PF), and (5) deviation of sudden deceleration and acceleration during frontal plane sitting with left rotation (Rot_L_DEVF) compared to both NoLBP and HxLBP, reflecting increased instability and movement variability in multiple planes. Additionally, peak-to-peak of sudden deceleration and acceleration in the transverse plane during prone right hip rotation (PHR_R_P2PT) was significantly greater in the CLBP group relative to the NoLBP group, further supporting the presence of movement control impairments in individuals with chronic symptoms.

Kinematic model for classifying NoLBP, CLBP, and HxLBP

Binary logistic regression models identified distinct kinematic predictors for differentiating between groups (Table 3). For NoLBP vs. CLBP, three parameters were retained: TF_MV (OR = 0.94, 95% CI 0.90–0.98, $P = 0.002$), PHR_R_AUCT (OR = 2.78, 95% CI 1.45–5.34, $P = 0.002$), and Rot_L_P2PF (OR = 1.32, 95% CI 1.12–1.55, $P < 0.001$). The model showed acceptable fit (Hosmer–Lemeshow $P = 0.129$), Nagelkerke $R^2 = 0.38$, and an

overall classification accuracy of 74.5% (78.7% for NoLBP and 70.2% for CLBP). Robustness analysis using LASSO with 10-fold cross-validation yielded a similar mean accuracy of 76.1%.

For NoLBP vs. HxLBP, TF_MV was the only significant predictor (OR = 0.97, 95% CI 0.94–1.00, $P = 0.022$). The model showed Hosmer–Lemeshow $P = 0.306$, Nagelkerke $R^2 = 0.08$, and a classification accuracy of 61.7% for both groups. Cross-validation produced a comparable mean accuracy of 61.2%.

For CLBP vs. HxLBP, LB_R_AUCF was a significant predictor (OR = 0.37, 95% CI 0.18–0.75, $P = 0.006$), while PHR_R_AUCT showed a trend toward significance (OR = 0.61, 95% CI 0.36–1.04, $P = 0.070$). The model fit was adequate (Hosmer–Lemeshow $P = 0.487$), Nagelkerke $R^2 = 0.20$, with overall classification accuracy of 62.8% (53.2% for CLBP and 72.3% for HxLBP). Cross-validation yielded a mean accuracy of 64.0%.

Discussion

This study investigated lumbopelvic movement control using IMU-based kinematic measurements in individuals with NoLBP, CLBP, and HxLBP during ten movement control tests. We found significant differences across the three groups in four tests. Furthermore, logistic regression identified parameters that provided moderate classification ability. These findings suggest that clinically feasible IMU-derived kinematic measures, obtained with a standardized placement and processing pipeline, can capture relevant aspects of movement control.

Demographic and clinical characteristics

The HxLBP group reported a significantly longer time since their last LBP episode (approximately 41 days) compared to the CLBP group (approximately 6 days), indicating more frequent recurrent LBP episodes in those with CLBP. Fluctuating pain severity in CLBP can activate lumbopelvic movement control dysfunction, leading to compensatory movement patterns that exacerbate recurrent pain and disability.

Comparing between NoLBP and CLBP

Individuals with CLBP showed slower trunk flexion velocity and greater kinematic irregularity during lateral bending, prone hip rotation, and trunk rotation. This finding is consistent with prior reports that individuals with CLBP may adopt slower movement speeds during functional tasks; however, movement speed alone does not identify the underlying mechanism. Slower movements may reflect a range of factors, including pain-related protective strategies, perceived instability or stiffness, reduced physical capacity/conditioning, cautious task execution, or altered sensorimotor control. Accordingly, previously proposed mechanisms such as fear-avoidance behaviors [26] and sensorimotor dysfunction [27] should be interpreted as possible explanations rather than direct inferences from the present data. Christe et al. found that individuals with CLBP had reduced angular amplitude and velocity during functional movements, leading to decreased lumbar spine motion and greater reliance on hip movements to minimize pain [28]. The CLBP group also demonstrated higher P2PF, AUCF, and AUCT values during lateral bending, prone hip rotation, and sitting with trunk rotation, indicating an inability to smoothly control trunk movement in these planes. This manifests as instability catches and out-of-plane movements due to muscle guarding or co-contraction, reflecting compensatory strategies to avoid pain [29]. The NoLBP group exhibited smoother and more controlled movements.

The kinematic classification model demonstrated moderate ability to distinguish individuals with CLBP from NoLBP using MV during trunk flexion, AUCT during prone hip rotation, and P2PF during sitting with rotation. MV during trunk flexion highlighted movement speed as an indicator of chronic pain, with slower movements in those with CLBP likely stemming from physical limitations and/or behavioral adaptations, which is consistent with studies linking increased superficial muscle activity and impaired sensorimotor control to slow movement in individuals with LBP [29]. Higher AUCT during the prone hip rotation predicted CLBP, suggesting greater variability in transverse plane rotation may increase CLBP risk, which aligns with previously reported compensatory over-motion in the lumbar region [30]. P2PF during sitting with trunk rotation indicated instability during trunk rotation in the secondary plane, which may be associated with uncoordinated muscle activation and impaired control, and is consistent with previously reported hyper-rotation and maladaptive movement strategies in individuals with CLBP [31].

Comparing between NoLBP and HxLBP

Clinical observations suggest HxLBP may predispose individuals to CLBP [12]. Our study showed higher MV during trunk flexion and lateral bending in the NoLBP group compared to HxLBP, aligning with research indicating reduced lumbar velocity in LBP [32]. Despite no current pain in HxLBP, slower movements may persist asymptomatically, indicating long-term adaptations, possibly from fear-avoidance behaviors [33]. Slower lateral bending in HxLBP may also result from quadratus lumborum tightness, restricting motion and affecting lumbar posture [34], and highlights the importance of clinicians being aware of potential movement control adaptations early in HxLBP.

Regression analysis indicated that MV during trunk flexion significantly differentiates individuals with HxLBP

from those with NoLBP. An increase in angular velocity reduce the likelihood of being classified as HxLBP, indicating slower trunk flexion is associated with HxLBP characteristics. While few studies directly address HxLBP kinematics, Hidalgo et al. [35] demonstrated a reliable model based on range of motion (ROM) and trunk movement speed to identify individuals with non-specific LBP. Slower trunk flexion speeds in HxLBP, similar to CLBP, support that longer movement duration are typical in LBP [36]. These alterations in HxLBP, even without current pain, suggest subtle, persistent movement control impairments after acute LBP episodes. These impairments could increase the future LBP recurrence risk, emphasizing the need to identify and address these deficits in rehabilitation programs which may be able to prevent the transition from HxLBP to CLBP.

Comparing between CLBP and HxLBP

Our study revealed that individuals with HxLBP had reduced MV during lateral bending, which may be associated with asymmetrical lateral bending, a previously reported risk factor for developing CLBP [37]. Notably, individuals in the HxLBP group moved slower than those with CLBP; however, movement velocity alone does not identify the underlying mechanism, and our data cannot determine whether slower movement represents a protective strategy, a persistent alteration in motor control, or another factor. One possibility is that people with HxLBP adopt a more cautious movement strategy during trunk motion, potentially reflecting perceived threat or fear of reinjury, altered sensorimotor control, reduced physical conditioning, or task-specific confidence. Prior prospective work has also identified physical factors (e.g., flexibility/ROM characteristics) that may be associated with LBP risk [32], but we did not directly measure quadratus lumborum tightness or other muscle-specific properties. Lower P2P and AUC values in HxLBP compared to CLBP during lateral bending, prone hip rotation, and sitting with trunk rotation indicate better movement control. In contrast, individuals with CLBP demonstrated poorer movement control, particularly in the primary plane of movement during lateral bending and prone hip rotation, and a higher number of instability catches in the secondary plane during trunk rotation. These findings suggest that CLBP is associated with movement control impairments and aberrant movement patterns, while HxLBP is characterized by slower velocities and fewer abnormal patterns.

AUCF and AUCT during lateral bending and prone hip rotation distinguished individuals with HxLBP from those with CLBP, suggesting that an increase in AUC significantly reduces the likelihood of HxLBP, reflecting smoother movement control in HxLBP individuals and better spinal control. While motor control changes are linked to a higher risk of recurrent pain in individuals with HxLBP [38], our results imply that those with HxLBP have less compensatory control than CLBP, potentially due to their pain-free status and fewer recurrence episodes, which have less impact on disrupting spinal control balance.

Lateral bending to the right was impaired in individuals with CLBP, reflecting asymmetrical movement and differences in kinematic characteristics, and supports previous studies linking lateral bending imbalance to spinal dysfunction [39]. Although our study did not assess participants' dominant sides, the findings suggest movement imbalances can impact spinal control. Additionally, reduced right hip rotation was less likely in HxLBP compared to CLBP, potentially due to limited hip rotation.

Robustness analyses

To evaluate the stability of our logistic regression models, we conducted robustness analyses using 10-fold cross-validation and LASSO regularization. The results confirmed that the same predictors identified in the logistic regression models remained consistent across folds, supporting the reliability of the classification models.

NoLBP vs. CLBP: The model consistently selected (1) the mean velocity during trunk flexion, (2) the area under the curve (AUC) of sudden deceleration and acceleration in the transverse plane during prone right hip rotation, and (3) the peak-to-peak of sudden deceleration and acceleration in the frontal plane of sitting with left rotation.

NoLBP vs. HxLBP: The mean velocity of trunk flexion remained the sole predictor.

CLBP vs. HxLBP: The model consistently retained the AUC of sudden deceleration and acceleration in the frontal plane of lateral bend to right, together with the AUC of sudden deceleration and acceleration in the transverse plane of prone right hip rotation.

Cross-validated accuracies were comparable to, or slightly higher than, those of the original models, indicating that the observed predictors were not sample-specific and were internally generalizable within the study population.

Study limitations

The findings of this study should be considered alongside several limitations. First, the convenience sampling method used in this study may limit the generalizability of the findings to other populations with LBP. Future studies should include more diverse samples, including individuals of different ages, occupations, and pain characteristics.

Second, the study focused on a limited set of unidimensional kinematic parameters. This was a deliberate choice to enhance clinical feasibility, as these measures can be

easily derived from portable IMUs and were selected for their potential interpretability in relation to clinically observed movement patterns. However, our study did not directly test the correspondence between these kinematic features and clinician-observed signs. Future studies should explicitly evaluate this link by comparing IMU-derived metrics with standardized clinical observation ratings and examining their agreement and validity.

Third, while our classification models achieved moderate accuracy (61.7–74.5%), particularly in distinguishing NoLBP from CLBP, the performance was lower for differentiating CLBP from HxLBP. This reflects the clinical overlap between these groups and indicates that the current models should be considered proof-of-concept. Future studies should integrate additional kinematic features and advanced machine learning approaches to enhance classification accuracy and clinical applicability.

Fourth, the same kinematic variables were used for both group comparisons and classification, which may bias performance estimates despite cross-validation. In addition, no external validation dataset was available, further limiting the ability to confirm the generalizability of our models. Future work should validate models on independent datasets and explore additional metrics to improve robustness and clinical applicability.

Finally, practical considerations may constrain clinical implementation. Although wearable sensors can be relatively portable, access to IMU systems, software, and expertise for data collection and processing varies across clinics, and associated costs and workflow demands may limit uptake. These feasibility constraints should be considered when interpreting the implications for early detection and targeted rehabilitation. Future studies should therefore report implementation factors (e.g., equipment costs, setup time, training requirements, and data-processing burden) and evaluate streamlined protocols to support real-world clinical adoption.

Clinical implications and future directions

Our kinematic analysis identified distinct movement patterns for each group, leading us to develop a specific kinematic classification model using statistical regression to differentiate between the three groups. An IMU-based assessment technology combined with our classification model objectively quantified movement-control–related kinematic features, including out-of-plane deviations across three planes of motion. These findings are hypothesis-generating and may inform future research on clinically feasible assessment and whether these kinematic features relate to rehabilitation outcomes. These findings enhance our understanding of natural spinal movement control and offers potentially useful clinical assessment methods through the interpretation of clinically relevant kinematic parameters, which may offer insights into the future design of personalized preventive and rehabilitative programs to reduce recurrent LBP.

Conclusion

This study demonstrated that IMU-based kinematic analysis has moderate ability to differentiate movement patterns among individuals with NoLBP, CLBP, and HxLBP, providing a valuable tool for assessing lumbopelvic movement control strategies. Our findings suggest that IMUs offer a promising approach to enhance clinical decision-making in LBP management by providing objective and quantitative measures of movement impairments and potentially identifying individuals at risk for chronic or recurrent LBP. In addition, robustness analyses using 10-fold cross-validation and LASSO regularization confirmed the stability and reliability of the classification models, supporting their potential applicability in clinical and research settings. Future research should focus on confirming these results in larger, more diverse populations, investigating the longitudinal relationship between movement patterns and LBP progression, and evaluating the efficacy of IMU-guided personalized rehabilitation programs.

Abbreviations

AUC	Area under the curve
ANOVA	Analysis of variance
BMI	Body mass index
CI 95%	Confidence intervals
CLBP	Chronic low back pain
DEV	Deviation
EMG	Electromyography
HxLBP	History of low back pain
ICC	Intraclass correlation coefficient
IMUs	Inertial measurement units
LASSO	Least absolute shrinkage and selection operator
LB	Lateral bend
LBP	Low back pain
LSD	Least significant difference
MDC	Minimal detectable change
MODQ	Modified Oswestry Disability Questionnaire
MV	Mean velocity
NoLBP	No low back pain
NPRS	Numerical pain rating scale
OR	Odd ratio
P2P	Peak-to-peak
TSK	Tampa scale of kinesiophobia
PHR	Prone hip rotation
ROM	Range of motion
Rot	Rotation
SE	Standard errors
TF	Trunk flexion
VIF	Variance inflation factor

Supplementary Information

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Supplementary Material 1.

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Authors' contributions
SK has significantly contributed to conceptualization, data curation, formal analysis, methodology, and writing-original draft. KK and NR have substantially contributed to data curation and formal analysis. RV has substantially contributed to writing-review & editing. JR has significantly contributed to formal analysis and writing-review & editing. PW has significantly contributed to conceptualization, data curation, formal analysis, funding acquisition, and writing-original draft.

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Data availability
The datasets used and/or analyzed during this study would be available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This study was approved by Mahidol University Institutional Review Board (COA: MU-CIRB 2020/084.1806). All participants provided written informed consent before participation.

Consent for publication
Written informed consent for publication was also obtained.

Competing interests
The authors declare no competing interests.

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