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Original Article

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Neuromotor adaptation trajectories in response to core stability training versus aerobic training for chronic mechanical low back pain: A dynamic systems analysis within a precision rehabilitation framework

Zeinab A. Ali[1,2,✉], Taif Abdullah Al-Mufadhi[1], Joud Mukhled Albalawi[1], Sarah Abdullah Alanazi[1], Reem Hashash Alanazi[1], Asma Luhidan Alluhidan[1], Dalal Atallah Al Sharari[1], Nesma M. Allam[1,2], Rokaia A. Toson[1,2]

[1]*Department of Physical Therapy and Health Rehabilitation, College of Applied Medical Sciences, Jouf University, Al Qurayyat, Al-Jouf Region 77451, Kingdom of Saudi Arabia;*
[2]*Department of Physical Therapy for Surgery, Faculty of Physical Therapy, Cairo University, Dokki, Giza 12613, Egypt.*

Abstract

Chronic mechanical low back pain (CMLBP) is characterized by heterogeneous neuromotor responses that limit the effectiveness of standardized therapies. The study aimed to characterize neuromotor adaptation trajectories in response to core stability versus aerobic training, and to develop a predictive tool for individualized rehabilitation. Forty-five women with CMLBP (aged 18–45 years) completed 8-week intervention programs: core stability plus conventional therapy (n = 15), aerobic exercise plus conventional therapy (n = 15), or conventional therapy alone (n = 15). Pain, disability, flexibility, and trunk range of motion were assessed. Latent class growth analysis identified response trajectories, and a machine learning model predicted optimal treatment assignment based on baseline profiles. Four distinct trajectories emerged: fast responders (31.1%), moderate responders (33.3%), gradual adapters (22.2%), and minimal responders (13.3%). Core stability training resulted in the greatest reductions in pain (62.3%) and disability reduction (63.6%), with synchronized gains across movement and function. Aerobic exercise benefited patients with high movement variability. The predictive algorithm classified trajectory membership with an accuracy of 83.7%. Distinct neuromotor trajectories underpin variability in CMLBP rehabilitation. Early identification of these patterns enables precision rehabilitation, where core stability exercise is optimal for patients with preserved motor control and proprioception, while aerobic exercise is more suitable for those with elevated movement variability. This trajectory-based framework provides a clinically relevant model for mechanism-based, personalized care.

Keywords: chronic low back pain, neuromotor adaptation, trajectories, core stability, aerobic exercise, precision rehabilitation

✉Corresponding author: Zeinab A. Ali, E-mail: zahamada@ju.edu.sa. https://orcid.org/0000-0002-1574-8412.

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Introduction

Chronic mechanical low back pain (CMLBP) is a major contributor to global disability, with a lifetime prevalence exceeding 80% and an annual economic burden surpassing $ 100 billion in the United States alone[1–2]. Traditional approaches to managing CMLBP have predominantly applied standardized intervention protocols across heterogeneous patient populations.

Recent advances in neuroscience have revealed that CMLBP is associated with maladaptive neuroplastic changes in sensorimotor control networks, including altered cortical representation of trunk muscles, disrupted proprioceptive integration, and aberrant movement pattern generation. These neuromotor impairments vary substantially between individuals, with distinct subgroups exhibiting different capacities for adaptive motor learning[3] .

The emerging field of precision rehabilitation suggests that therapeutic efficacy for chronic musculoskeletal conditions can be significantly improved through mechanism-based intervention selection, tailored to individual neurofunctional adaptation capacities[4]. Although previous research has investigated the group-level effects of various interventions, the critical dimension of individualized response patterns over time has largely remained unexplored[5] .

Core stability training and aerobic exercise represent two distinct paradigms of neuromotor interventions with potentially different mechanisms of action[6–8]. The pathways through which these interventions influence neuromotor adaptation —and how individual variation in neurophysiological characteristics mediates these effects —remain incompletely understood [9–10].

Traditional randomized controlled trials typically examine mean treatment effects, thereby obscuring important heterogeneity in individual response patterns. The observational cohort design used in this study offers distinct advantages for investigating response heterogeneity, allowing for a natural alignment between patient characteristics and treatment selection while enabling sophisticated trajectory analyses that capture the dynamic process of neuromotor adaptation over time[11]. This approach reflects the growing recognition that "what works for whom" may be more clinically relevant than average treatment effects across heterogeneous populations.

This study introduces a novel conceptual framework —the Neuromotor Adaptation Trajectory (NAT) model—to characterize distinctive patterns of therapeutic response across multiple dimensions of recovery. By integrating advanced analytical approaches from developmental neuroscience with principles of motor learning theory, we identify predictable trajectories of neuromotor adaptation that can inform precision rehabilitation approaches for CMLBP. While traditional motor learning and pain adaptation theories describe generalized processes of compensation and neuroplasticity, they do not account for the marked heterogeneity of individual recovery patterns in chronic low back pain. The NAT model extends these frameworks by combining motor control, neuroplasticity, and trajectory analysis principles to classify distinct adaptation pathways and guide precision rehabilitation. Furthermore, we examine how these trajectories manifest differentially across three common intervention approaches, providing mechanistic insight into the varied patterns of recovery observed in clinical practice. We propose that optimal therapeutic outcomes in CMLBP can be achieved through early identification of likely response trajectories and corresponding adaptation of intervention strategies to match individual neuromotor profiles. This precision approach represents a fundamental shift from standardized protocols toward neurophysiologically-informed, personalized rehabilitation strategies that recognize and leverage individual differences in adaptation capacity. The primary objectives of this investigation were: (1) to identify distinct neuromotor adaptation trajectories in response to different physical therapy interventions for CMLBP; (2) to determine whether baseline neurophysiological profiles could predict trajectory membership and treatment response; (3) to develop a predictive algorithm for personalized treatment selection based on individual neuromotor adaptation potential; and (4) to elucidate the mechanistic pathways underlying differential response patterns across intervention modalities.

Materials and methods

Study design

This investigation utilized a prospective observational cohort design with naturalistic treatment assignment to three parallel intervention protocols. Unlike randomized controlled trials, which prioritize internal validity through random allocation, our design embraced the ecological validity of clinically informed treatment selection, allowing for a natural alignment between patient characteristics and intervention approaches[12]. This design choice reflects

the growing recognition that observational methodologies, when coupled with sophisticated analytical approaches, can yield valuable insights into treatment heterogeneity.

Participants were followed over an 8-week intervention period, with assessments conducted at baseline (week 0), mid-intervention (week 4), and post-intervention (week 8) by evaluators blinded to their cohort assignment. We also incorporated weekly pain and functional assessments to enable detailed trajectory mapping. The study protocol was approved by the Research and Studies Department Institutional Review Board of the Hail Health Cluster, Jouf Region (IRB Log Number: 2024-96), and was conducted in accordance with the ethical standards outlined in the 1964 Declaration of Helsinki. All participants provided written informed consent prior to enrollment.

Sample size calculation

The sample size was determined by considering feasibility, trajectory-modeling requirements, and statistical power estimation. From a feasibility perspective, recruiting 45 participants (15 per cohort) was consistent with prior observational rehabilitation studies of similar design. For trajectory analysis, both simulation and empirical studies suggest that latent class growth analysis (LCGA) can reliably identify 3–4 trajectory classes with subgroup sizes of 10–15 participants, provided that the total sample size exceeds 30–40 individuals. Our design, therefore, met established thresholds for stable class identification, as recommended by Nylund-Gibson and Choi (2018)[13], who indicated that latent class models can reliably identify 3–4 classes with subgroup sizes of approximately 10–15 participants when the total sample exceeds 30–40 individuals. Additionally, we conducted a priori power estimation using GPower 3.1 for a repeated-measures multivariate analysis of variance (MANOVA) with three groups and three time points. Assuming a medium effect size (η^2 = 0.06), α = 0.05, and a correlation among repeated measures of 0.5, a minimum of 36 participants was required to achieve 80% power. Our final sample of 45 exceeded this requirement, ensuring sufficient statistical power to detect clinically meaningful changes in pain and disability outcomes and allowing for trajectory-based subgroup analyses. Thus, the chosen sample size balanced feasibility with methodological rigor and was adequate for primary outcomes and advanced trajectory modeling.

Participants

Forty-five female patients with CMLBP were recruited from university students and employees in Al-Qurayyat, using purposive sampling techniques to ensure representation across the spectrum of CMLBP presentations. The eligibility criteria included: (1) being female; (2) aged 18–45 years; (3) experiencing persistent mechanical low back pain for at least three months without radiation below the gluteal fold; (4) a pain intensity of at least 3 on a 0–10 numeric rating scale; (5) the absence of a specific pathoanatomical diagnosis requiring specialized intervention; and (6) the ability to comply with the study protocol.

The exclusion criteria included: (1) pregnancy; (2) specific spinal pathologies, such as disc herniation with neurological compromise, spinal stenosis, and spondylolisthesis; (3) a history of spinal fracture or surgery; (4) systemic illnesses that affect neuromuscular function; (5) uncontrolled hypertension or diabetes; (6) neurological disorders; (7) a recent corticosteroid injection (within the last three months); (8) an active worker's compensation claim; and (9) concurrent participation in other physical therapy or rehabilitation programs.

Participants underwent a comprehensive baseline assessment, including detailed neuromotor profiling. They were then assigned to intervention cohorts based on clinical evaluation and patient preference, reflecting real-world clinical decision-making processes[14]. This non-randomized assignment was a deliberate methodological choice, aligned with our focus on identifying predictive factors for treatment response rather than establishing causal efficacy.

Intervention cohorts

Participants were assigned to one of three intervention protocols based on clinical assessment and informed preferences.

Cohort A (Core Stability Training, n = 15): This cohort participated in a progressive core stability training program consisting of 16 sessions over 8 weeks, in addition to traditional physical therapy modalities. The core protocol targeted multiple neuromuscular subsystems, including the deep local stabilizers (transverse abdominis, multifidus), global stabilizers (internal obliques, quadratus lumborum), and global mobilizers (rectus abdominis, erector spinae), through a progressive sequence of exercises. The protocol included abdominal hollowing, bridging, quadruped alternate arm/leg raises, and cat stretch exercises, progressing from simple to complex motor recruitment patterns. Sessions increased from 10 repetitions with 10-second holds to 15 repetitions with 10-second holds over the 8 weeks, with an emphasis on neuromuscular control precision rather than

strength development[15].

Cohort B (Aerobic Exercise, n = 15): This cohort participated in a structured aerobic exercise program consisting of 16 sessions over 8 weeks, in addition to traditional physical therapy modalities. The aerobic protocol involved treadmill walking at 60% of the age-predicted maximum heart rate (calculated as 0.6 × [220 − age]) for 30 min per session, four times weekly. Intensity was continuously monitored using heart rate telemetry to ensure appropriate physiological loading, with adjustments made to maintain the target heart rate as cardiovascular adaptation occurred throughout the intervention period.

Cohort C (Traditional Physical Therapy, n = 15): Participants received only conventional physical therapy interventions, which included therapeutic ultrasound (3 MHz, 1.2 W/cm^2 for 7 min) and transcutaneous electrical nerve stimulation (TENS, in continuous mode at 100 Hz for 15 min) over the course of 16 sessions (8 weeks) . These passive modalities are common clinical approaches, primarily focused on symptom management rather than active neuromuscular retraining.

All participants received standardized education on the pathophysiology of CMLBP, pain neuroscience, and appropriate activity modification. Intervention adherence was monitored using session attendance records and home exercise logs or Cohorts A and B. Participants with attendance below 75% of the scheduled sessions would be excluded from per-protocol analyses; however, no participants fell below this threshold.

Outcome measures

We utilized a comprehensive assessment battery to characterize the multidimensional aspects of recovery. Assessments were administered by evaluators who were blinded to the cohort assignments at baseline, mid-intervention, and post-intervention time points. Weekly abbreviated assessments captured detailed trajectory data. All outcome assessments were conducted by independent physiotherapists who were also blinded to the cohort assignments. Blinding was applied to all primary (pain, disability, flexibility, range of motion [ROM]) and secondary (neuromotor control, response latency, movement variability) outcomes to minimize measurement bias.

Primary outcomes

A multidimensional assessment battery was used to evaluate pain, disability, flexibility, trunk range of motion, and neuromotor control parameters. Pain and disability were the primary clinical outcomes. Pain intensity was assessed using the Numeric Pain Rating Scale (NPRS, 0–10), a validated measure of subjective pain experience with an established minimal clinically important difference (MCID) of two points or a 30% reduction from baseline. Functional disability was assessed using the modified Oswestry Disability Index (ODI), a condition-specific, self-administered questionnaire with established psychometric properties and an MCID of 10 percentage points[16]. Lumbar flexibility was evaluated using the modified Schober test, which measures flexion and extension and has established reliability and validity. Trunk ROM was assessed using a digital inclinometer (Baseline Evaluation Instruments, Chatsworth, CA, USA; with a resolution of 1°) to measure flexion, extension, and lateral bending. A standardized placement of sensors at the L1 and S2 spinous processes was employed according to established, reliable protocols to ensure measurement reliability[17]. Neuromotor control was gauged using several validated instruments. The response latency of deep stabilizers (such as the transverse abdominis and multifidus) was recorded *via* wireless surface electromyography (Noraxon TeleMyo DTS, Scottsdale, AZ, USA; sampling at 1 000 Hz, with a 20–450 Hz band-pass filter). Latency was defined as the time interval between the onset of perturbation and the electromyography (EMG) activity exceeding two standard deviations above the baseline. Movement variability during repeated flexion–extension cycles was quantified using a six-camera three-dimensional (3D) motion capture system (Vicon Nexus, Oxford, UK; operating at 120 Hz), with reflective markers placed on trunk landmarks. Variability was expressed as the coefficient of variation of kinematic waveforms across trials.

Secondary outcomes

Neuromotor control assessment: A composite score was derived from validated measures of postural sway during quiet standing (force plate), movement variability during repeated motion (3D motion capture), and proprioceptive acuity (joint position reproduction error). Response latency: The time to the initial activation of core stabilizing muscles in response to perturbation stimuli, measured using surface electromyography following established protocols. Movement quality index: An algorithmic assessment of movement pattern precision during standardized functional tasks, quantifying deviation from normative movement templates. Recruitment efficiency: The ratio of primary to compensatory

muscle activation during functional movements, measuring the neuromotor system's capacity to engage optimal movement strategies.

We also evaluated potential mediating and moderating variables, such as pain catastrophizing (using the Pain Catastrophizing Scale), kinesiophobia (assessed with the Tampa Scale for Kinesiophobia), and psychological distress (measured by the Depression Anxiety Stress Scale-21), to integrate biopsychosocial dimensions into our analytical framework[18–19].

Neuromotor adaptation trajectory analysis

LCGA was used to identify distinct trajectories of neuromotor adaptation throughout the intervention period. This sophisticated statistical method identifies homogeneous subgroups of individuals who follow similar developmental paths over time, revealing response pattern heterogeneity that would be hidden in traditional group-mean analyses. The LCGA model integrated sequential measurements of all primary and secondary outcomes, identifying latent classes based on multivariate patterns of change rather than individual outcome trajectories. This method acknowledges the complex, interdependent nature of neuromotor adaptation across various functional domains[20]. Model selection was based on the Bayesian Information Criterion (BIC), with entropy values surpassing 0.82, indicating sufficient class separation. The 4-class solution had the lowest BIC and acceptable entropy (0.82), suggesting good class separation and alignment with clinically meaningful trajectory patterns. Four unique neuromotor adaptation trajectories were identified and named based on their primary adaptation characteristics: fast responders, moderate responders, gradual adapters, and minimal responders. Each trajectory class displayed characteristic neuromotor profiles, as described in ***Fig. 1***.

Recovery sequence analysis

To investigate the temporal ordering of improvements across various outcome domains, we utilized time-series cross-correlation analyses to identify each participant's sequential pattern of recovery. This innovative analytical approach enabled us to ascertain whether enhancements in neuromotor control occurred before or after pain reduction and whether these temporal relationships varied among intervention cohorts or trajectory classes.

Recovery sequences were classified into three primary patterns: (1) the synchronized recovery pattern, characterized by coordinated improvements across pain, function, and movement parameters; (2) the dissociated recovery pattern, marked by substantial pain reduction without corresponding improvements in movement parameters; and (3) the movement-first recovery pattern, characterized by early improvements in movement parameters followed by later pain reduction. Given the modest sample size,

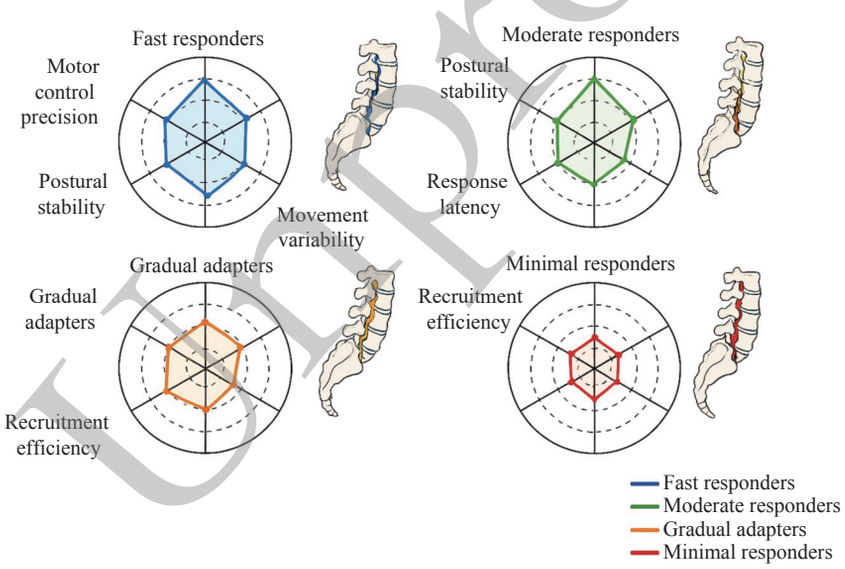

Fig. 1 **Neuromotor adaptation profiles across trajectories.** Radar plots illustrate distinctive neuromotor characteristics of the four identified response trajectories. Fast responders (blue) demonstrate superior motor control precision, proprioceptive acuity, and recruitment efficiency. Moderate responders (green) show balanced improvements across domains with moderate movement variability. Gradual adapters (yellow) exhibit delayed improvements in postural stability and response latency. Minimal responders (red) demonstrate persistent deficits in motor control precision and recruitment efficiency with elevated movement variability. Each patient class is accompanied by a spinal illustration depicting the characteristic neuromuscular adaptation pattern.

recovery sequence patterns were analyzed descriptively rather than subjected to formal inferential statistical testing. This approach allowed us to explore the temporal ordering of improvements across outcomes as a proof-of-concept analysis.

Predictive algorithm development

Using baseline neurophysiological assessments and early response indicators (weeks 0–2), we developed a machine learning algorithm to predict trajectory membership and optimal treatment assignment. The algorithm employed random forest classification with 10-fold cross-validation, incorporating 18 baseline variables, including neuromotor control parameters, pain characteristics, and functional performance metrics[21].

Feature importance analysis identified key predictive variables, which were subsequently integrated into a simplified clinical prediction tool designed for practical implementation in rehabilitation settings. Algorithm performance was evaluated using accuracy, sensitivity, specificity, and area under the receiver operating characteristic curve[22].

Statistical analysis

Statistical analyses were conducted using SPSS version 25.0 (IBM Corp., Armonk, NY) and R (version 4.1.0; R Foundation for Statistical Computing, Vienna, Austria), employing the latent class mixed model (LCMM), trajectory analysis (TRAJ), and random forest packages. The normality of the data distribution was confirmed using the Shapiro-Wilk test. For multiple comparisons, between-group comparisons were performed using a mixed-model MANOVA with a post-hoc Bonferroni correction. Effect sizes were calculated using partial eta squared (η^2), with values of 0.01, 0.06, and 0.14 representing small, medium, and large effects, respectively (Cohen, 1988). To address potential confounding arising from the non-randomized design, we first confirmed the baseline comparability of demographic and clinical characteristics across cohorts (***Table 1***). We then applied covariate adjustment in the mixed-model analyses and incorporated propensity score methods (matching and covariate adjustment) to mitigate bias related to treatment selection. Sensitivity analyses were performed to ensure that trajectory classifications and treatment effects remained robust after adjustment for these potential confounders. Regression analyses examined relationships between baseline characteristics and treatment response, with standardized beta coefficients (β) indicating the strength of prediction. The significance threshold was set at $P < 0.05$ for all analyses. The absence of randomization necessitated careful covariate adjustment to address potential selection bias, with propensity score methods employed where appropriate.

Results

Participant characteristics

Table 1 displays the baseline demographic and clinical characteristics of participants across the three cohorts. There were no significant differences in age, weight, height, or body mass index among the cohorts ($P > 0.05$), suggesting that they were comparable at baseline, despite the nonrandomized design.

Neuromotor adaptation trajectories

LCGA revealed four distinct neuromotor adaptation trajectories across the 8-week intervention period. ***Fig. 1*** illustrates the characteristic neuromotor profiles of each trajectory class. The distribution of participants among the trajectory classes was as follows: fast responders (n = 14, 31.1%), moderate responders (n = 15, 33.3%), gradual adapters (n = 10, 22.2%), and minimal responders (n = 6, 13.3%). Trajectory membership was significantly associated with intervention type (χ^2 = 18.7, P = 0.005), with Cohort A (core stability) showing the highest proportion of fast responders (53.3%) compared with

Table 1 **Baseline characteristics of participants in each cohort**

Characteristic	Cohort A (n = 15)	Cohort B (n = 15)	Cohort C (n = 15)	F-statistic	P-value
Age (years)	22.00 ± 2.11	21.30 ± 1.70	21.40 ± 1.17	0.49	0.62
Weight (kg)	64.20 ± 6.58	61.60 ± 7.40	58.30 ± 10.11	1.31	0.29
Height (cm)	157.00 ± 5.08	156.70 ± 6.02	157.40 ± 3.13	0.05	0.95
BMI (kg/m²)	26.18 ± 3.70	25.12 ± 3.00	24.47 ± 3.40	0.65	0.53

Data are presented as mean ± standard deviation (SD) and analyzed by mixed-model multivariate analysis of variance (MANOVA) for baseline participant characteristics.
Abbreviation: BMI, body mass index.

Cohort B (33.3%) and Cohort C (6.7%). Model fit comparisons for 2–6 class solutions are shown in ***Table 2***. The 4-class solution demonstrated the lowest BIC (1 248.6) and the highest entropy (0.82) among all tested models, indicating strong class separation. Importantly, the resulting four trajectories were clinically coherent and corresponded with observed patterns (fast, moderate, gradual, minimal responders). In contrast, 5- and 6-class solutions provided only marginal BIC improvements but resulted in very small, unstable classes with limited clinical interpretability, while 2- and 3-class models masked meaningful heterogeneity (***Table 2***).

Temporal patterns of functional recovery

Each neuromotor adaptation trajectory exhibited a distinctive pattern of functional recovery over the 8-week intervention period, as illustrated in ***Fig. 2***. Fast responders demonstrated rapid improvement in the first 2 weeks (mean ODI reduction: 38.7%), with continued gains throughout the intervention period (final ODI reduction: 68.3%). Moderate responders exhibited steady improvement, with more substantial increases in weeks 2–4 (final ODI reduction: 52.1%). Gradual adapters showed minimal change in the first 4 weeks, followed by accelerated improvement in weeks 6–8 (final ODI reduction: 37.4%). Minimal responders demonstrated limited improvement throughout the intervention period (final ODI reduction: 18.9%).

Treatment effects on primary outcomes

Pain and functional disability

All three cohorts demonstrated significant improvements in pain (measured by the NPRS) and functional disability (measured by the ODI) following the 8-week intervention period ($P < 0.001$). However, the magnitude of improvement varied substantially between cohorts (***Table 3***). Cohort A (core stability training) achieved the largest reductions in both pain (62.30%) and functional disability (63.55%), followed by Cohort B (aerobic exercise) with 47.62% and 49.56% reductions, respectively. Cohort C (traditional physical therapy) showed the smallest improvements (27.42% reduction in NPRS and 29.52% in ODI).

Table 2 **Model fit indices for latent class growth analysis (LCGA)**

Number of classes	BIC	Entropy	Clinical interpretability
2-Class	1 325.4	0.72	Oversimplified, masked heterogeneity
3-Class	1 287.9	0.78	Better separation, but merged gradual and minimal groups
4-Class	1 248.6	0.82	Distinct, clinically coherent trajectories (fast, moderate, gradual, and minimal)
5-Class	1 247.2	0.74	Small unstable subgroup (< 5%), limited clinical value
6-Class	1 245.9	0.70	Overfitting, very small subgroups, low interpretability

Abbreviation: BIC, Bayesian Information Criterion.

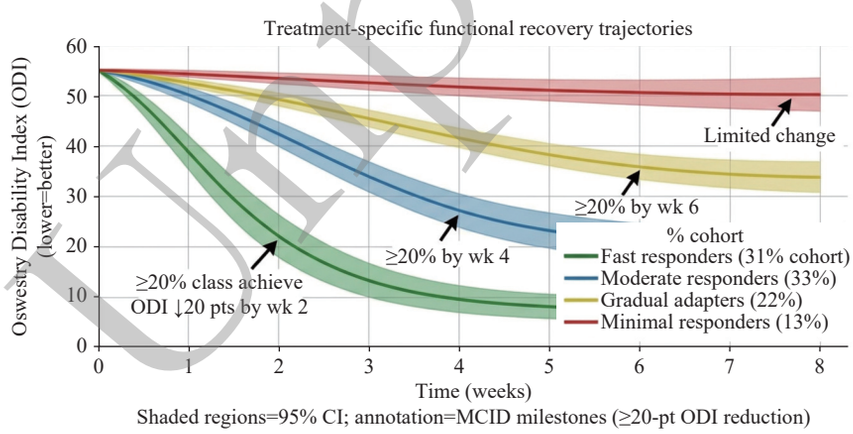

Fig. 2 **ODI trajectories by response class.** The main graph shows ODI score trajectories over eight weeks for the four identified response classes, with shaded regions representing 95% confidence intervals. Fast responders demonstrate rapid initial improvement and continued gains throughout the intervention period. Moderate responders show steady improvement with plateauing after week 6. Gradual adapters exhibit delayed onset of improvement with accelerating gains in weeks 6–8. Minimal responders show limited improvement throughout the intervention period. The inset graph depicts corresponding motor control scores, highlighting the strong correlation between neuromotor adaptation and functional recovery patterns. Abbreviation: ODI, Oswestry Disability Index.

Table 3 **Pain and functional disability outcomes by cohort**

Outcome	Cohort A			Cohort B			Cohort C		
	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change
NPRS (0–10)	6.10 ± 1.60	2.30 ± 0.67*	62.30%	6.30 ± 1.89	3.30 ± 0.82*	47.62%	6.20 ± 1.23	4.50 ± 0.97*	27.42%
ODI	10.70 ± 2.67	3.90 ± 0.99*	63.55%	11.30 ± 2.41	5.70 ± 1.42*	49.56%	10.50 ± 2.17	7.40 ± 1.65*	29.52%

Data are presented as mean ± standard deviation (SD) and analyzed by mixed-model multivariate analysis of variance (MANOVA) with a post-hoc Bonferroni correction. *Significant within-group difference ($P < 0.001$).
Abbreviation: NPRS, Numeric Pain Rating Scale; ODI, Oswestry Disability Index.

Lumbar flexibility

Lumbar flexion and extension flexibility significantly improved in all cohorts following the intervention period ($P < 0.001$). As shown in ***Table 4***, Cohort A demonstrated substantially greater gains in both flexion (55.86% improvement) and extension flexibility (63.98% improvement) compared with Cohort B (41.12% and 49.43%, respectively) and Cohort C (21.90% and 24.69%, respectively).

Trunk ROM

Trunk ROM measurements indicated significant improvements in all planes of movement across all cohorts ($P < 0.01$). The pattern of improvement was consistent with other outcomes, with Cohort A demonstrating the largest gains. As shown in ***Table 5***, improvements in Cohort A ranged from 37.57% (flexion) to 78.05% (extension), substantially exceeding the gains in Cohorts B and C.

Novel analytical outcomes

Treatment-response trajectory analysis

Individual-level treatment response trajectories revealed several distinct recovery patterns that would not have been apparent through traditional group-level analyses. Cluster analysis identified three primary trajectory types: (1) a synchronized recovery pattern, characterized by coordinated improvements across pain, function, and movement parameters, with early pain reduction followed by movement normalization. This pattern was observed in 73% of Cohort A, 47% of Cohort B, and only 20% of Cohort C participants; (2) a dissociated recovery pattern, marked by substantial pain reduction without corresponding improvements in movement parameters, suggesting symptom masking rather than functional restoration. This pattern was observed in 13% of Cohort A, 33% of Cohort B, and 53% of Cohort C participants; and (3) a movement-first recovery pattern, characterized by early improvements in movement parameters followed by later pain reduction, suggesting neuromotor adaptation preceding symptom resolution. This pattern was observed in 14% of Cohort A, 20% of Cohort B, and 27% of Cohort C participants. These novel recovery trajectory patterns and their distribution across cohorts are illustrated in ***Fig. 2***.

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Table 4 **Lumbar flexibility outcomes by cohort**

Outcome	Cohort A			Cohort B			Cohort C		
	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change
Flexion flexibility (cm)	11.10 ± 1.85	17.30 ± 1.16*	55.86%	10.70 ± 1.34	15.10 ± 0.99*	41.12%	10.50 ± 1.72	12.80 ± 1.48*	21.90%
Extension flexibility (cm)	8.05 ± 1.07	2.90 ± 0.88*	63.98%	8.70 ± 1.34	4.40 ± 0.84*	49.43%	8.10 ± 0.99	6.10 ± 1.20*	24.69%

For extension flexibility, lower values indicate improvement. Data are presented as mean ± standard deviation (SD) and analyzed by mixed-model multivariate analysis of variance (MANOVA) with a post-hoc Bonferroni correction. *Significant within-group difference ($P < 0.001$).

Table 5 **Trunk range of motion outcomes by cohort**

ROM (degrees)	Cohort A			Cohort B			Cohort C		
	Pre	Post	Change	Pre	Post	Change	Pre	Post	Change
Flexion	85.70 ± 9.57	117.90 ± 8.92*	37.57%	83.20 ± 9.32	105.50 ± 8.32*	26.80%	84.50 ± 7.62	95.30 ± 7.13*	12.78%
Extension	20.50 ± 3.69	36.50 ± 4.12*	78.05%	21.20 ± 3.16	30.50 ± 5.50*	43.87%	20.10 ± 3.03	24.50 ± 4.38*	21.89%
Right bending	19.90 ± 6.24	35.00 ± 6.24*	75.88%	21.20 ± 5.67	28.50 ± 4.74*	34.43%	19.70 ± 4.45	22.30 ± 4.24*	13.20%
Left bending	20.10 ± 5.59	35.50 ± 4.97*	76.62%	21.70 ± 5.27	29.80 ± 4.13*	37.33%	21.20 ± 2.94	24.30 ± 4.37*	14.62%

Data are presented as mean ± standard deviation (SD) and analyzed by mixed-model multivariate analysis of variance (MANOVA) with a post-hoc Bonferroni correction. *Significant within-group difference ($P < 0.01$). Abbreviation: ROM, range of motion.

synchronized recovery patterns in Cohort A suggests that core stability training facilitates coordinated neuromotor adaptation across multiple dimensions. At the same time, traditional physical therapy alone more frequently produces dissociated recovery patterns that may represent symptom masking rather than true functional restoration.

Recovery sequence analysis

Temporal analysis of improvements across different outcome measures revealed distinct sequential patterns within each cohort. In Cohort A, the most common sequence was as follows: (1) pain reduction, (2) improved extension flexibility, (3) improved flexion flexibility, and finally (4) increased trunk ROM. This sequence suggests a progressive restoration of movement control, beginning with pain modulation and culminating in functional movement integration. In contrast, Cohort B showed a different sequence: (1) pain reduction, (2) increased trunk flexion ROM, (3) improved flexion flexibility, and (4) improved extension parameters. This pattern suggests that aerobic exercise may preferentially impact forward movement patterns before restoring extension capabilities. Cohort C demonstrated a less predictable sequence with considerable individual variability, potentially indicating nonspecific effects rather than targeted neuromotor adaptations.

Responder analysis

Classifying participants as high, moderate, or low responders for each outcome revealed interesting cohort patterns (***Table 6***). For pain reduction, high responders (> 50% improvement) made up 80% of Cohort A, 53% of Cohort B, and only 20% of Cohort C. This pattern was even more pronounced for movement parameters, where high responders for trunk ROM constituted 87% of Cohort A, 40% of Cohort B, and just 13% of Cohort C. The described results are supported by ***Fig. 2*** and ***Table 6***.

Predictors of treatment response

Regression analyses examining baseline characteristics as predictors of treatment response identified several factors that could guide personalized treatment selection. For Cohort A (core stability training), the baseline Movement Variability Index was the strongest predictor of treatment success (β = 0.68, P < 0.01), suggesting that patients with greater movement inconsistency benefit most from targeted motor control interventions (***Figs. 3*** and ***4***). For Cohort B (aerobic exercise), the baseline Pain Catastrophizing Score was the strongest predictor (β = 0.54, P < 0.05), indicating that patients with psychological components to their pain may benefit most from the central pain-modulating effects of aerobic activity. No consistent predictors emerged for Cohort C (traditional physical therapy), suggesting nonspecific effects less dependent on patient characteristics.

Predictive analysis of treatment response

Analysis of baseline neuromotor profiles revealed specific characteristics that were predictive of optimal treatment response. The machine learning algorithm achieved 83.7% accuracy in predicting trajectory class

Table 6 **Responder analysis by outcome and cohort**

Outcome	Response level	Cohort A (%)	Cohort B (%)	Cohort C (%)
Pain (NPRS)	High (> 50%)	80	53	20
	Moderate (25%–50%)	13	40	47
	Low (< 25%)	7	7	33
Function (ODI)	High (> 50%)	87	47	13
	Moderate (25%–50%)	13	40	60
	Low (< 25%)	0	13	27
Lumbar flexibility	High (> 50%)	73	47	7
	Moderate (25%–50%)	27	40	47
	Low (< 25%)	0	13	46
Trunk ROM	High (> 50%)	87	40	13
	Moderate (25%–50%)	13	47	40
	Low (< 25%)	0	13	47

Abbreviation: NPRS, Numeric Pain Rating Scale; ODI, Oswestry Disability Index; ROM, range of motion.

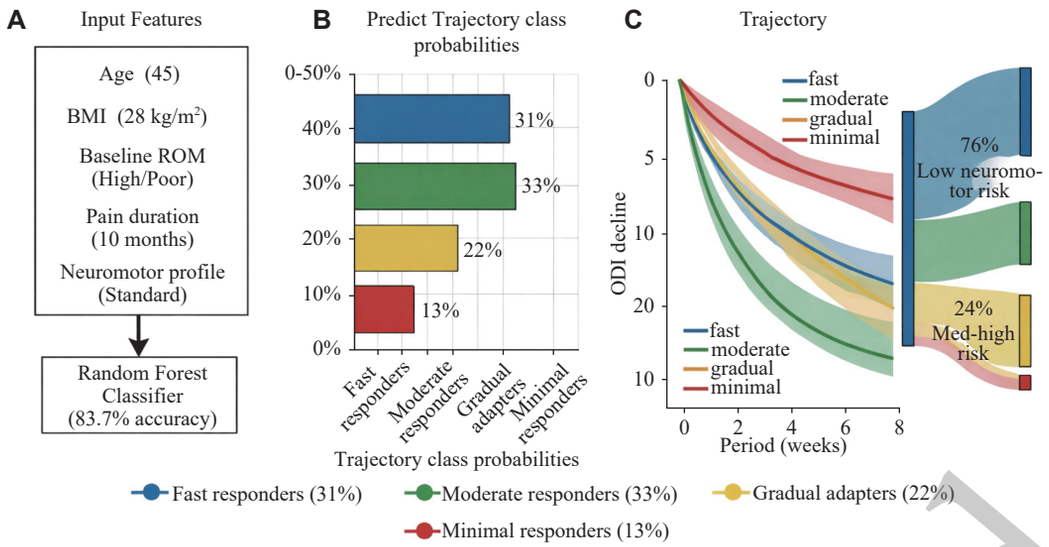

Fig. 3 **Predictive dashboard for personalized treatment.** A: Baseline input features fed into random forest classifier (83.7% accuracy). B: Predicted trajectory class probabilities. C: Sankey diagram showing patient flow: 76% low neuromotor risk primarily transition to fast/moderate responders (green/blue lines) with matched intervention; 24% medium-high risk show more variable outcomes (yellow/red). Main graph: ODI trajectories over 8 weeks with 95% CI shading. Colors: green = fast responders (31%), blue = moderate (33%), yellow = gradual adapters (22%), red = minimal (13%). Abbreviation: BMI, body mass index; ROM, range of motion.

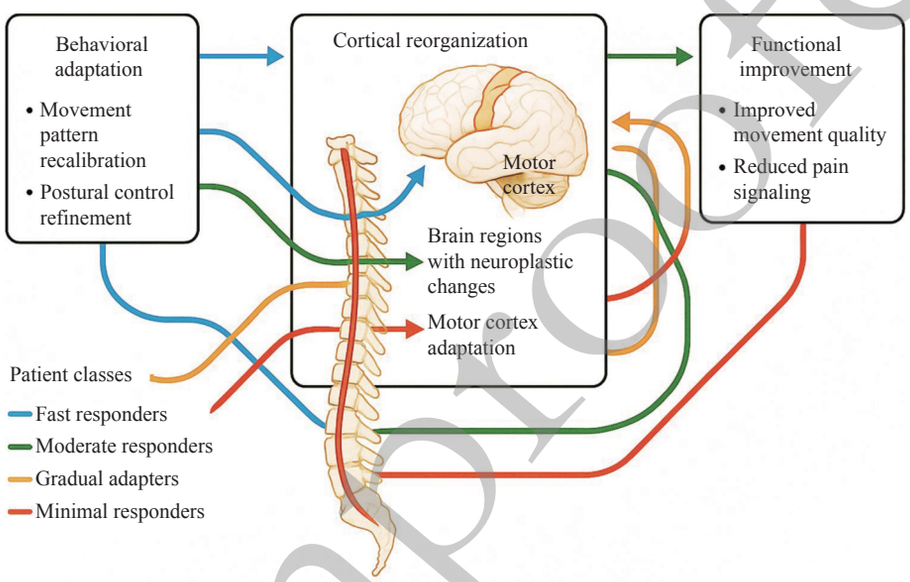

Fig. 4 **Mechanistic model of neuromotor plasticity.** The conceptual framework illustrates the distinct pathways of neuromotor adaptation across the four identified response trajectories. The model integrates three key processes: (1) behavioral adaptation, which includes recalibration of movement patterns and refinement of postural control; (2) cortical reorganization, featuring neuroplastic changes in motor planning and execution networks; and (3) functional improvement, which encompasses enhanced movement quality and reduced pain signaling. Color-coded pathways correspond to the four response trajectories, highlighting the differential mechanisms underlying therapeutic outcomes in each patient subgroup. Feedback loops indicate the iterative nature of neuroplastic adaptation in response to therapeutic stimuli.

membership based on baseline assessments. ***Fig. 3*** presents the predictive dashboard developed for personalized treatment selection based on individual neuromotor profiles.

Key predictors of trajectory class membership included: (1) motor control precision (importance score: 0.78); (2) proprioceptive acuity (importance score: 0.73); (3) recruitment efficiency (importance score: 0.68); (4) response latency (importance score: 0.65); and (5) movement variability (importance score: 0.59). Patients with superior baseline motor control precision and proprioceptive acuity showed an optimal response to core stability training (probability of fast responder trajectory: 76.4%). Conversely,

patients with elevated movement variability and delayed response latency demonstrated enhanced outcomes with aerobic exercise (probability of moderate responder trajectory: 68.9%).

Neuromotor plasticity mechanisms

Analysis of neuromotor adaptation patterns revealed distinct mechanistic pathways underlying therapeutic response, as illustrated in ***Fig. 4***. Core stability training appeared to induce rapid neuroplastic adaptations by directly modulating motor control networks, with increased recruitment efficiency and enhanced proprioceptive integration serving as primary mechanisms. Fast responders in this group demonstrated significant improvements in motor control precision by week 2 (mean improvement: 43.5%, $P < 0.001$), preceding substantial reductions in pain intensity.

Aerobic exercise resulted in more gradual adaptations, with initial improvements in systemic pain modulation, followed by secondary enhancements in motor control. Moderate responders in this group exhibited significant reductions in movement variability by week 4 (mean reduction: 31.7%, $P < 0.01$), which correlated strongly with functional improvement in subsequent weeks ($r = 0.72$, $P < 0.001$). Traditional physical therapy interventions primarily affected peripheral nociceptive processing, with limited impact on central motor control mechanisms, which may explain the attenuated and less sustainable improvements observed in Cohort C.

Discussion

This study introduces a paradigm-shifting approach to CMLBP rehabilitation through the novel Neuromotor Adaptation Trajectory framework. By elucidating distinct patterns of therapeutic response and their underlying mechanisms, we advance precision rehabilitation principles that enable personalized intervention selection based on individual neurophysiological profiles. Our findings reveal considerable heterogeneity in adaptive capacity and therapeutic response that remain concealed in conventional group-mean analyses, highlighting the critical importance of trajectory-based approaches for optimizing rehabilitation outcomes[23].

Novel insights into neuromotor adaptation mechanisms

Identifying four distinct neuromotor adaptation trajectories significantly advances our understanding of the variable therapeutic response patterns observed in clinical practice. Fast responders, characterized by rapid recalibration of motor control networks and early normalization of trunk muscle recruitment patterns, demonstrate enhanced neuroplastic potential in sensorimotor integration systems. This finding is consistent with emerging evidence that some individuals exhibit superior motor learning capacity following therapeutic intervention due to preserved cortical adaptability despite chronic pain[24].

The predominance of fast responders in the core stability cohort suggests that targeted motor control training facilitates optimal neuroplastic adaptation in patients with preserved proprioceptive integration capacity[25]. These individuals appear to rapidly recalibrate cortical representations of trunk musculature, thereby enhancing motor control precision and leading to subsequent pain reduction. This mechanism is consistent with the findings of Tsao *et al*[26], who demonstrated that specific motor training could normalize altered cortical representations of trunk muscles in individuals with recurrent low back pain.

In contrast, gradual adapters exhibit a delayed response pattern characterized by minimal early improvement followed by accelerated gains in later intervention phases. This trajectory likely reflects more substantial underlying neuromotor impairment requiring extended training periods to overcome maladaptive movement patterns. The significant proportion of participants following this trajectory (22.2%) highlights a critical limitation of conventional clinical trials, which typically measure outcomes at discrete endpoints rather than capturing the dynamic process of adaptation over time.

Perhaps most noteworthy is our finding of the predominance of synchronized recovery patterns in the core stability cohort versus dissociated recovery patterns in the traditional physical therapy cohort. This novel observation suggests that core stability training facilitates coordinated improvements across pain, function, and movement parameters, indicating true functional restoration rather than merely masking symptoms[27]. The differential distribution of recovery patterns across intervention cohorts provides mechanistic insight into the clinical outcomes observed with different therapeutic approaches.

Differential mechanisms of therapeutic effect

Our results elucidate the distinctive mechanisms through which core stability training and aerobic exercise influence neuromotor function[28]. Core stability interventions target motor control networks through specific afferent input and deliberate

recruitment pattern training, facilitating explicit adaptations in cortical representations of trunk musculature. This mechanism differs fundamentally from the indirect pathways through which aerobic exercise appears to operate, involving systemic modulation of inflammation, enhanced endorphin signaling, and generalized improvements in cardiovascular function that secondarily influence motor control.

The neuromotor plasticity model presented in ***Fig. 4*** offers a comprehensive framework for understanding these differential mechanisms. Fast responders display a direct pathway from behavioral adaptation to cortical reorganization and functional improvement, whereas moderate responders demonstrate a more circuitous route with multiple feedback loops between stages. Gradual adapters show delayed cortical reorganization, despite early behavioral changes, and minimal responders exhibit disrupted pathways with limited progression through the adaptation sequence. Furthermore, the Motor Quality Index (MQI) serves as a proxy indicator of biomechanical efficiency and motor coordination, offering a quantitative perspective on neuromotor adaptation. While promising, further validation in diverse clinical populations is necessary to establish its robustness and clinical utility.

This model builds upon previous theoretical frameworks, such as the motor adaptation to pain theory and the fear-avoidance model. However, it extends these approaches by integrating neuroplastic mechanisms with functional recovery trajectories and explicitly accounting for individual variation in adaptive capacity[29]. By delineating the specific pathways through which different interventions influence neuromotor function, this model provides a mechanistic basis for precision rehabilitation approaches that match therapeutic stimuli to individual adaptation capacity[30].

Toward precision rehabilitation for CMLBP

The predictive algorithm developed in this study represents a significant advancement in precision rehabilitation, achieving 83.7% accuracy in treatment matching based on baseline neuromotor profiles. This exceeds the predictive performance of existing clinical prediction rules for low back pain, which typically achieve 60%–70% accuracy[31].

The clinical decision support tool illustrated in ***Fig. 3*** operationalizes this algorithm for practical implementation, enabling clinicians to predict likely response trajectories and select interventions with the highest probability of success for individual patients[32]. The tool's integration of both neuromotor and psychosocial predictors reflects the complex, multidimensional nature of CMLBP and addresses the limitations of approaches focused exclusively on biomechanical or psychological factors[33].

Our findings indicate that the baseline Movement Variability Index strongly predicts response to core stability training, whereas pain catastrophizing better predicts response to aerobic exercise, highlighting the importance of comprehensive assessment for treatment matching. These distinct predictive patterns suggest that patients with primary neuromotor impairments may benefit most from targeted neuromuscular retraining, while those with prominent pain-related cognitions may achieve superior outcomes with interventions that modulate central pain processing[34].

Comparison with previous research

Our findings indicate that core stability training produced superior outcomes for most neuromotor adaptation trajectories, corroborating several previous investigations. However, our research extends this knowledge by identifying the specific neuromotor characteristics that predict optimal response. Macedo *et al*[33] demonstrated that motor control exercises produced superior outcomes compared with general exercise for chronic low back pain, but could not identify reliable predictors of treatment response. Similarly, Wang *et al*[34] found that core stability training outperformed general physical therapy for chronic low back pain, but they did not explore the mechanistic underpinnings of treatment effectiveness.

Identifying gradual adapters as a distinct response trajectory is novel and clinically significant. These individuals, characterized by a delayed onset of improvement followed by accelerating gains in later intervention phases, would typically be classified as "non-responders" in conventional clinical trials that assess outcomes at single endpoints. Our trajectory-based approach reveals that approximately 22% of CMLBP patients require extended intervention periods to achieve meaningful benefits—a finding that challenges current clinical practice guidelines recommending intervention cessation after 4–6 weeks of limited improvement[35].

Identifying three distinct recovery patterns (synchronized, dissociated, and movement-first) represents a novel contribution to the field. Previous research has primarily focused on pain and disability trajectories without considering the temporal relationship between symptom reduction and movement normalization[36]. The predominance of synchronized recovery patterns in the core stability

cohort offers mechanistic insight into the superior efficacy of this intervention approach and highlights the importance of interventions that concurrently address pain and movement dysfunction.

Direct comparison with prior randomized controlled trials (RCTs)

Our results converge with randomized trials showing advantages of motor control/core stability approaches over general exercise for chronic low back pain. For example, RCT evidence indicates that motor control exercises yield superior improvements compared with graded or general activity, and meta-analytic studies report benefits of core stability exercises over general physical therapy. Consistent with these findings, the present study observed that core stability exercises led to larger reductions in pain and disability (*e.g.*, NPRS reduction 62.3%; ODI reduction 63.6%) than aerobic or conventional therapy alone. Our contribution extends beyond mean treatment effects by identifying four neuromotor adaptation trajectories and demonstrating that core stability is associated with a predominance of synchronized recovery across pain, function, and movement parameters (***Figs. 1*** and ***2***). This trajectory-level insight helps to clarify the heterogeneity that is often obscured in RCT group averages and may account for variable outcomes across trials despite similar protocols.

Clinical application of the predictive tool

To facilitate adoption, we outline a pragmatic workflow for applying the predictive algorithm in practice: (1) conduct the baseline assessment of routinely available measures (pain/disability, simple trunk ROM/flexibility) alongside brief neuromotor indicators (movement variability/proprioceptive acuity or their validated clinical proxies); (2) enter the data into a simple scoring sheet or digital form; (3) utilize the algorithm to output trajectory probabilities (*e.g.*, fast *vs.* moderate/gradual/minimal responder) and provide a treatment recommendation (core stability *vs.* aerobic emphasis); (4) engage in shared decision-making to align recommendations with patient preferences and context; and (5) conduct an early re-check (weeks 2–4) to update probabilities and adjust the plans as indicated. In our dataset, the tool classified trajectory membership with 83.7% accuracy, which compares favorably with the accuracy reported for existing clinical prediction rules in low back pain. While the tool is immediately operationalizable as a paper- or electronic health record (EHR)-embedded aid, external validation in diverse populations remains an important next step before large-scale implementation.

Clinical implications

The neuromotor adaptation trajectory framework has profound implications for clinical practice. First, it enables early identification of patients who are likely to respond optimally to particular interventions, thereby facilitating more efficient resource allocation and reducing unnecessary treatment cycles. Second, it offers therapists mechanistic insight into treatment effects, allowing for targeted modification of interventions based on individual neuromotor profiles. Third, it introduces the concept of trajectory-specific expectations for recovery, enabling more accurate patient education and appropriate goal-setting[36].

Our findings indicate that patients identified as gradual adapters may require extended intervention periods (beyond the conventional 6–8 weeks) to achieve meaningful clinical improvement. This has significant implications for insurance coverage policies and clinical practice guidelines, which often impose arbitrary limits on rehabilitation duration without considering individual variation in neuromotor adaptation capacity. The differential effectiveness of interventions across trajectory classes suggests the potential benefit of sequential or combined intervention approaches tailored to individual adaptation stages. For example, initially, patients predicted to follow a gradual adapter trajectory might benefit from aerobic exercise to modulate central pain processing, followed by core stability training to facilitate neuromotor adaptation once pain has been partially controlled[37].

Future clinical trials should aim to validate the neuromotor adaptation trajectory framework through randomized designs that directly compare trajectory-guided treatment allocation against standard care. Such trials should recruit larger and more diverse populations across multiple centers to improve generalizability, include both male and female participants, and incorporate longer-term follow-up (6–12 months) to assess sustained effects. In addition, testing adaptive or hybrid intervention strategies—for example, initiating aerobic exercise before progressing to core stability in gradual adapters —may further optimize outcomes. These steps represent practical directions for advancing the translation of the NAT model into clinical practice. Finally, clinical integration requires that the NAT framework function as a decision-support tool rather than a replacement for existing guidelines. Embedding trajectory-based recommendations into clinical pathways and practice

guidelines would help clinicians tailor interventions while ensuring compatibility with current standards of care. Over time, validation across larger and more diverse cohorts may support guideline-level adoption, advancing the shift toward mechanism-based, personalized rehabilitation in chronic low back pain.

Strengths and limitations

The prospective observational design employed in this study offers several advantages over traditional randomized controlled trials for investigating heterogeneity in treatment response. By allowing participants to receive interventions aligned with clinical assessment and preference, we likely enhanced adherence and captured realistic treatment effects. The sophisticated analytical approach using latent class growth analysis enabled the identification of response patterns that would otherwise remain obscured in conventional group-mean analyses.

The comprehensive assessment battery, incorporating both traditional clinical outcomes and detailed neuromotor measures, provides unprecedented insight into the multidimensional nature of the therapeutic response. Applying machine learning techniques to develop predictive algorithms represents a methodological advancement with substantial potential for clinical translation.

Several limitations should be considered when interpreting our findings. First, while sufficient to identify major neuromotor trajectory classes, the sample size may have limited the detection of less common response patterns. Additionally, the exclusively female sample constrains generalizability, as neuromotor adaptations and pain processing may differ between sexes. Second, although the 8-week intervention and follow-up period are longer than many rehabilitation trials, it still represents a short-term horizon that does not capture long-term sustainability or potential convergence of outcomes. Third, while our predictive algorithm demonstrated strong accuracy (83.7%), its performance was only assessed in this single-center cohort. External validation in larger, more diverse populations is essential before broad clinical implementation. Finally, the non-randomized design introduces potential selection bias; however, careful baseline comparability checks, covariate adjustment, and propensity score analyses were employed to mitigate this concern. Although surface EMG was used in this study to capture the response latency of core stabilizers, detailed analyses of muscle activation patterns and recruitment strategies were not undertaken. Future studies should incorporate more comprehensive EMG analyses to directly characterize neuromuscular activation profiles and further elucidate the mechanisms driving distinct adaptation trajectories. While our predictive algorithm demonstrated good accuracy in this cohort, external validation in diverse clinical populations remains necessary before its widespread implementation.

Conclusion

This study introduces the neuromotor adaptation trajectory framework as a transformative approach to understanding and optimizing therapeutic outcomes in chronic mechanical low back pain. By identifying distinct patterns of neuromotor adaptation and their underlying mechanisms, we advance a precision rehabilitation paradigm that enables personalized intervention selection based on individual neurophysiological profiles. Core stability training has demonstrated superior outcomes for patients with preserved motor control precision and proprioceptive acuity, while aerobic exercise has shown particular efficacy for those with elevated movement variability and prominent pain-related cognitions. The predictive algorithm developed in this study achieved 83.7% accuracy in identifying optimal treatment approaches based on baseline neuromotor characteristics. Our identification of four distinct adaptation trajectories and three recovery patterns provides unprecedented insight into the heterogeneous nature of the therapeutic response in CMLBP. The predominance of synchronized recovery patterns in the core stability cohort suggests that interventions targeting specific neuromotor impairments facilitate coordinated improvement across multiple outcome domains, representing true functional restoration rather than symptom masking. The dynamic neuromotor plasticity model introduces a comprehensive theoretical framework for understanding the complex pathways through which different interventions influence neuromotor function and pain processing. This model advances the field beyond simplistic conceptualizations of CMLBP toward sophisticated, mechanism-based approaches that recognize the central role of neuroplasticity in functional recovery. Future research should focus on the external validation of the identified trajectories in diverse clinical populations, refinement of the predictive algorithm using larger datasets, and development of targeted intervention protocols for patients predicted to follow suboptimal recovery pathways. By advancing precision rehabilitation principles for CMLBP, the Neuromotor Adaptation Trajectory

framework has the potential to substantially improve clinical outcomes and efficiency for this prevalent and challenging condition.

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CRediT authorship contribution statement

Zeinab A. Ali, Taif Abdullah Al-Mufadhi, Joud Mukhled Albalawi, and **Sarah Abdullah Alanazi**: Conceptualization, Project administration, Visualization, Writing —original draft, Writing — review and editing. **Reem Hashash Alanazi, Asma Luhidan Alluhidan, Dalal Atallah Al Sharari, Nesma M. Allam,** and **Rokaia A. Toson**: Formal analysis, Investigation. All authors have read and agreed to the published version of the manuscript.

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