Trauma Monthly 2026; 31(4): 1910-1921

10.30491/tm.2026.595648.1977

Trauma Monthly

Research Article

The effect of American College of Sports Medicine exercises on lower-limb muscle electrical activity during walking in individuals with hamstring tightness and low back pain

Makwan Jabar Ali[1], Mohsen Barghamadi[1*], Ebrahim Piri[1], Reza Noktehsanj[2]

[1] Department of Sports Biomechanics, Faculty of Educational Sciences and Psychology, University of Mohaghegh Ardabili, Ardabil, Iran
[2] Department of Orthopaedic surgery, Ardebil University of Medical Sciences, Ardebil, Iran

***Corresponding Author**: Mohsen Barghamadi, Department of Sports Biomechanics, Faculty of Educational Sciences and Psychology, University of Mohaghegh Ardabili, Ardabil, Iran. Email: barghamadi@uma.ac.ir, ORCID:0000-0002-1794-9410.

Received 2025-07-05 ; **Accepted** 2026-06-16; **Online Published** 2026-08-29

Abstract

Introduction: Chronic non-specific low back pain (LBP) combined with hamstring tightness may disrupt lumbopelvic mechanics and alter lower-limb neuromuscular control during gait. Although ACSM-based exercise programs are widely recommended for chronic LBP, their effects on phase-specific lower-limb muscle activation during walking remain unclear. This study investigated the effect of an 8-week ACSM-based exercise program on lower-limb electrical activity during walking in young men with hamstring tightness and chronic LBP.

Method: In this semi-experimental laboratory study, 30 young men with chronic non-specific LBP and hamstring tightness were assigned to either an exercise group (n=15) or a control group (n=15). The exercise group completed an 8-week supervised ACSM-based program, three sessions per week, consisting of hamstring stretching, core stabilization, and lower-limb strengthening exercises. Surface electromyography (EMG) was used to record the electrical activity of the tibialis anterior (TA), medial gastrocnemius(MG), gluteus medius (GM), biceps femoris (BF), semitendinosus (ST), rectus femoris (RF), vastus medialis (VM), and vastus lateralis (VL) muscles of the dominant lower limb during walking at 1.3 m/s (±5%). EMG amplitudes were normalized to maximum voluntary isometric contraction (MVIC) and expressed as %MVIC across the loading response, midstance, and push-off phases. Data were analyzed using a 2×2 mixed repeated-measures ANOVA with Bonferroni-adjusted post hoc tests, and effect sizes were reported as partial eta squared (η2p).

Result: Significant time×group interactions were observed in loading response for TA (p=0.030, η2p=0.157) and GM (p=0.030, η2p =0.158); in midstance for VM (p=0.005, η2p=0.250) and GM (p=0.012, η2p=0.210); and in push-off for MG (p=0.010, η2p=0.218) and BF (p=0.024, η2p=0.174). The exercise group showed reduced activation from pre- to post-intervention in TA, GM during loading response, VM and GM during midstance, and MG and BF during push-off, whereas the control group remained relatively stable.

Conclusion : An 8-week ACSM-based exercise program induced selective, phase-dependent reductions in lower-limb EMG activity during walking in young men with chronic LBP and hamstring tightness. These findings indicate selective, phase-dependent reductions in lower-limb EMG activity after an 8-week ACSM-based exercise program. Although consistent with a shift away from compensatory overactivation, reduced EMG amplitude could also reflect decreased effort or motivation, reduced muscle capacity, or altered gait mechanics; concurrent kinematic, kinetic, or metabolic measures would be needed to confirm improved movement efficiency.

Keywords: Low Back Pain; Hamstring Tightness; Electromyography; Gait.

Introduction

Chronic non-specific low back pain (LBP) represents one of the most prevalent, debilitating, and socioeconomically burdensome musculoskeletal conditions worldwide, affecting approximately 60% to 80% of adults at some point during their lifetime [1, 2]. Rather than being an isolated localized disorder, LBP is increasingly recognized as a systemic biomechanical dysfunction characterized by altered neuromuscular control, compromised lumbopelvic stability, and compensatory movement adaptations throughout the

kinetic chain [3, 4]. Within this interconnected kinematic network, the posterior myofascial chain, specifically the hamstring muscle group, plays a pivotal role in modulating pelvic orientation, sagittal spinal alignment, and dynamic load transfer between the lower limbs and the trunk[5]. Hamstring tightness, defined as a pathological reduction in hamstring muscle-tendon length and flexibility, is highly prevalent in individuals with LBP [6, 7]. This restriction in muscular extensibility acts as a biomechanical constraint, preventing natural anterior pelvic tilt during forward trunk flexion and forcing excessive compensatory lumbar flexion, thereby magnifying the shear forces and compressive loads exerted on the intervertebral discs and passive spinal structures[8,9].

The functional consequences of co-existing LBP and hamstring tightness are particularly pronounced during dynamic cyclic activities such as gait[10]. Walking requires a highly coordinated, sequential, and precise activation of lower-limb and trunk muscles to ensure shock absorption, forward propulsion, and pelvic stability [11]. In healthy individuals, the hamstrings act eccentrically during late swing to decelerate the advancing tibia and prepare the limb for heel strike, while synergistically stabilizing the pelvis during early stance [10, 12]. Simultaneously, the hip abductors, notably the gluteus medius, and the core stabilizers coordinate to maintain lateral pelvic horizontal alignment, minimizing lateral displacement of the center of mass [13, 14]. However, hamstring tightness and LBP disrupt this intricate neuromuscular coordination. The restricted extensibility of the hamstrings alters the mechanical pull on the ischial tuberosity, inducing a posterior pelvic tilt that forces the lumbar spine into compensatory flattening [15]. Consequently, key stabilizing muscles, such as the gluteus medius and rectus femoris, show altered onset timing, compensatory hyperactivation, or premature fatigue to preserve gait speed and dynamic equilibrium [16, 17]. These neuromuscular deviations (e.g., prolonged co-activation or elevated baseline electrical activity) not only accelerate muscular fatigue but also perpetuate a cyclical feedback loop of chronic joint overload, mechanical inefficiency, and persistent nociception[18].

To break this pathological cycle, clinical guidelines consistently advocate for structured active exercise therapy as the gold standard intervention for chronic LBP [19]. Among these approaches, the American College of Sports Medicine (ACSM) guidelines provide a comprehensive, systematic, and evidence-based framework that integrates core stabilization, progressive muscular strengthening, and targeted flexibility training [20]. Core stabilization exercises (e.g., abdominal bracing, bird-dog, and planks) aim to restore neuromuscular control of the deep stabilizing musculature, thereby enhancing the trunk's capacity to resist external perturbing forces [21]. Concurrently, targeted hamstring stretching (including static and proprioceptive neuromuscular facilitation [PNF] techniques) alleviates mechanical tethering of the pelvis, restoring physiological pelvic mobility and reducing compensatory lumbar stress [22]. While previous literature has extensively documented the clinical efficacy of ACSM-based programs in reducing pain scores and improving self-reported functional disability in LBP populations [23], the underlying neuro-biomechanical mechanisms driving these clinical improvements remain poorly understood.

Specifically, a critical research gap exists regarding how these clinically validated exercise programs translate into functional biomechanical adaptations during daily dynamic tasks like walking. Most prior studies evaluating rehabilitation outcomes have relied on static clinical tests (such as range of motion or maximum voluntary contractions) or subjective questionnaires, which do not capture the dynamic, multi-joint neuromuscular adaptations that occur during locomotion. Few studies have used surface electromyography (sEMG) to assess dynamic lower-limb muscle activation patterns during gait post-rehabilitation. Furthermore, no study has specifically investigated the combined effect of an 8-week ACSM-guided program (integrating core stabilization, flexibility, and lower-limb strengthening) on lower-limb sEMG profiles in individuals with the dual pathology of chronic LBP and clinical hamstring tightness during the stance phase of walking. Understanding whether clinical improvements are accompanied by normalized lower-limb muscle recruitment (such as reduced compensatory quadriceps activation and restored synergy between the hamstrings and hip abductors) is vital for validating the long-term biomechanical safety and efficacy of these protocols.

Therefore, this study aimed to investigate the effect of an 8-week ACSM-based exercise program on lower-limb muscle electrical activity (expressed as %MVIC of the tibialis anterior, medial gastrocnemius, gluteus medius, biceps femoris, semitendinosus, rectus femoris, vastus medialis, and vastus lateralis) during the stance phase of walking in individuals with hamstring tightness and chronic LBP. We hypothesized that the 8-week ACSM intervention would significantly reduce compensatory over-activation of the primary lower-limb extensors and stabilizers during the stance phase of walking in the experimental group compared with the control group; if confirmed with concurrent biomechanical measures, this would represent a more efficient and coordinated neuromuscular gait pattern.

Methods

2.1. Participants

This semi-experimental laboratory study included 30 young male participants divided into two equal groups: an experimental exercise group (n=15) and a control group (n=15). An a priori sample size calculation was performed using G*Power software (version 3.1), indicating that a minimum sample size of 11 participants per group (22 in total) was required to achieve a statistical power (1−β) of 0.80, with an effect size of f=0.25 and an alpha level of α=0.05 [24]. The partial eta-squared values indicated large effects for gluteus maximus (η^2p = 0.22, 95% CI: 0.06–0.43) and vastus medialis activity (η^2p = 0.19, 95% CI: 0.04–0.38). Therefore, the recruited sample size of 30 participants was deemed sufficient to detect significant differences. We recruited participants from public clinics and private healthcare centers in Ardabil, Iran, via targeted convenience sampling, and a board-certified orthopedic specialist performed diagnostic screening to confirm the co-existence of hamstring tightness and chronic low back pain (LBP). The inclusion criteria required that participants were male, aged between 20 and 30 years, had a clinical diagnosis of hamstring tightness confirmed by a positive Active Knee Extension (AKE) test (defined as a knee extension angle of <65◦ or lacking more than 25◦ from full extension accompanied by a sharp stretch sensation in the posterior thigh/knee region), and suffered from chronic non-specific low back pain persisting for at least the preceding three months [25].

Additionally, participants had to be able to sit, stand, and walk independently without assistive devices, be classified as sedentary or recreationally inactive (no structured physical exercise or athletic training within the past six months), and have no history of spine, pelvis, or lower-limb musculoskeletal surgery or any neurological, systemic, or vestibular disorders affecting balance or gait. None of the participants had previously engaged in a structured rehabilitation program for low back pain, and all were instructed to refrain from taking analgesic or anti-inflammatory medications throughout the study period. Exclusion criteria during the study included missing more than two consecutive or three cumulative rehabilitation sessions, sustaining any incidental musculoskeletal injury or acute pain exacerbation preventing completion of the testing protocol, commencing any new pharmacological therapy (such as muscle relaxants, non-steroidal anti-inflammatory drugs [NSAIDs], or analgesics) during the intervention period, or voluntarily withdrawing from the study. All participants were fully informed of the experimental risks and procedures and signed a written informed consent form in accordance with the Declaration of Helsinki. The Ethics Committee of the University of Mohaghegh Ardabili reviewed and approved the study protocol (Ethical Approval Code: IR.UMA.REC.1403.090).

2.2. Clinical Assessment (Active Knee Extension Test)

To evaluate hamstring tightness, the Active Knee Extension (AKE) test was administered. The participant was positioned supine on a clinical plinth with the contralateral limb secured flat against the table using a stabilization strap to prevent pelvic tilt. The examiner placed the hip of the tested limb into 90◦ of flexion, verified using a standard manual goniometer, while a custom wooden stabilization frame was used to ensure the thigh remained perpendicular to the table throughout the assessment. The participant was then instructed to actively extend the knee joint until they felt a strong but tolerable stretch sensation in the hamstring muscle group, holding this position for 3 seconds. The goniometer axis was aligned over the lateral epicondyle of the femur, the stationary arm was aligned with the longitudinal axis of the femur (pointing towards the greater trochanter), and the mobile arm was aligned with the lateral malleolus. The knee flexion deficit angle (deviation from full 180◦ extension) was recorded, and a deficit of greater than 25◦ confirmed hamstring tightness.

2.3. The ACSM Exercise Intervention Program

Participants in the experimental group completed an 8-week supervised exercise intervention based on the general principles of the American College of Sports Medicine (ACSM) and current exercise therapy recommendations for chronic low back pain [26]. The program was designed to improve lumbopelvic stability, hamstring flexibility, trunk control, and lower-extremity muscle function [27]. Training sessions were performed three times per week for 8 consecutive weeks, resulting in a total of 24 sessions. Each session lasted approximately 45-60 minutes and consisted of a 10-minute warm-up, 30-45 minutes of therapeutic exercise, and a 5-minute cool-down period.

All exercise sessions were supervised by the same physical therapist to ensure consistency in verbal instruction, exercise technique, and progression. Session attendance was recorded, and adherence was calculated as the percentage of completed sessions relative to the total prescribed sessions. Exercise progression was permitted only when the participant was able to complete the prescribed volume with acceptable movement quality and without an increase in low back pain greater than 2 points on a 10-point visual analogue scale. Progression criteria were operationalized as follows: participants progressed to the next level only if they completed all prescribed repetitions and hold times with correct technique, maintained balance throughout the task, reported pain ≤2/10 on the visual analogue scale during and after exercise, and demonstrated no compensatory movement patterns or need for therapist assistance. Rest intervals of 30-60 seconds were provided between sets and approximately 1-2 minutes between exercise categories. Participants in the control group were instructed to maintain their usual daily activities and to refrain from initiating any structured exercise or rehabilitation program during the 8-week study period. To verify that the control group maintained their usual daily activities, participants completed a weekly physical activity log and were contacted weekly by telephone throughout the 8-week period; no placebo or attention-control intervention was administered, which should be considered when interpreting the specificity of the observed exercise effects.

2.4. Experimental Protocol and Gait Analysis

All biomechanical and electromyographic assessments were conducted in the Sports Biomechanics Laboratory of the University of Mohaghegh Ardabili under standardized environmental conditions before and after the 8-week intervention period. Participants avoided vigorous physical activity for 48 hours before each testing session. Before formal data collection, each participant completed several familiarization trials to become accustomed to the laboratory environment, the walkway, and the instrumentation.

Participants completed walking trials along a 20-m indoor walkway. Walking speed was monitored using dual-beam infrared photocells (Brower Timing Systems, Draper, UT, USA) positioned around the central portion of the walkway. The target speed was set at 1.3 m/s with an allowable variation of ±5%, and trials outside this range were discarded and repeated. This speed was selected to standardize the gait task across participants and sessions because walking speed strongly influences lower-limb EMG amplitude; 1.3 m/s represents a comfortable, moderate walking speed for young adults and is consistent with speeds used in previous gait studies of individuals with low back pain, while avoiding the confounding effects of faster, more demanding speeds. Heel-strike and toe-off events were identified using the laboratory synchronization system integrated with the acquisition software, and the stance phase of gait was defined accordingly. We collected three successful walking trials from each participant during both the pre-intervention and post-intervention assessments. The assessor responsible for EMG data collection, signal processing, and statistical analysis was blinded to group allocation; group codes were concealed until all analyses were completed.

2.5. Electromyography (EMG) and Signal Processing

We recorded surface electromyographic (EMG) activity using an 8-channel wireless EMG system (Biometrics Ltd., Newport, UK) at a sampling frequency of 1000 Hz. Before electrode placement, the skin over each target muscle was shaved if necessary, lightly abraded, and cleaned with 70% isopropyl alcohol to reduce skin impedance. Bipolar Ag/AgCl surface electrodes (11-mm diameter, 25-mm inter-electrode distance) were placed parallel to the presumed muscle fiber direction by the same examiner for all participants. Electrode placement followed SENIAM recommendations for the following muscles of the dominant lower limb, determined by the preferred leg used to kick a ball: tibialis anterior (TA), medial gastrocnemius (MG), gluteus medius (GM), biceps femoris (BF), semitendinosus (ST), rectus femoris (RF), vastus medialis (VM), and vastus lateralis (VL) [28]. The anatomical locations of the electrodes are presented in Table 3.

To normalize dynamic EMG amplitudes, we performed maximum voluntary isometric contraction (MVIC)

testing for each muscle before gait testing at each assessment session [29]. For the TA, MVIC was obtained in a seated position with the ankle at 90∘ while the participant performed maximal dorsiflexion against manual or strap resistance. For the MG, participants performed maximal plantarflexion in a standing position with the knee extended. For the GM, participants performed maximal isometric hip abduction in side-lying with the tested hip in neutral alignment; for the BF and ST, participants performed maximal knee flexion in prone with the knee flexed to 90∘. For the RF, VM, and VL, maximal knee extension was performed in a seated position with the knee flexed to 90∘. We provided external stabilization and standardized verbal encouragement during all MVIC trials. Three MVIC trials of 5 seconds each were recorded for each muscle, with 60 seconds of rest between trials. We used the highest RMS value from the most stable 1-second window of the contraction plateau as the normalization reference. Raw EMG signals were processed offline in MATLAB (MathWorks, Natick, MA, USA). Signals were filtered using a zero-lag fourth-order Butterworth band-pass filter with cutoff frequencies of 10-500 Hz, followed by a 60-Hz notch filter to attenuate electrical noise [30]. We visually inspected signal quality and excluded segments contaminated by obvious motion artifacts. For each successful walking trial, EMG amplitude was quantified as root mean square (RMS) using a 50-ms moving window during the stance phase of gait. We then normalized the mean RMS amplitude during stance to MVIC and expressed it as %MVIC. We used the average value across the three successful trials for statistical analysis.

2.6. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows (version 26.0; IBM Corp., Armonk, NY, USA). Descriptive statistics are presented as mean ± standard deviation. Normality of data distribution was assessed using the Shapiro-Wilk test, and homogeneity of variances was examined using Levene's test. We analyzed baseline between-group differences in demographic and clinical characteristics using independent-samples t-tests. To examine the intervention's effects on muscle electrical activity, we performed a 2 × 2 mixed repeated-measures analysis of variance (ANOVA) for each muscle. The within-subject factor was Time (pre-intervention vs. post-intervention), and the between-subject factor was Group (experimental vs. control). Because the within-subject factor included only two levels, the assumption of sphericity was inherently satisfied. When significant main effects or Time × Group interactions were observed, Bonferroni-adjusted post hoc comparisons were conducted. Effect sizes were calculated using partial eta squared (η2p) and interpreted as small (≥0.01), medium (≥0.06), or large (≥0.14). All tests were two-tailed, and statistical significance was set at $p<0.05$.

Results

We analyzed normalized EMG activity (%MVIC) in eight lower limb muscles across three gait phases: loading response, midstance, and push-off. We conducted a two-way repeated-measures analysis of variance (ANOVA) to evaluate the main effects of time (pre- vs. post-8-week intervention), group (Exercise vs. Control), and their interaction (time×group). The descriptive statistics (Mean±SD) and the inferential statistical results (p-values and partial eta-squared, η2p) for each phase are presented in Tables (1-3).

3.1. Loading Response Phase

During the loading response phase (Table 4), time had a significant main effect for BF ($p=0.004$, $\eta2p=0.266$) and GM ($p=0.011$, $\eta2p=0.212$), indicating changes in proximal pelvic and hip stabilizers over the 8 weeks. A significant main effect of group was identified for TA ($p=0.001$, $\eta2p=0.340$), BF ($p=0.019$, $\eta2p=0.182$), and GM ($p=0.030$, $\eta2p=0.156$), indicating baseline and post-intervention differences in muscle activation strategies between individuals with chronic LBP and hamstring tightness. Importantly, a statistically significant time×group interaction was found for TA ($p=0.030$, $\eta2p=0.157$), with the exercise group showing reduced post-intervention activation. In contrast, the control group remained relatively stable, and GM showed a significant effect ($p=0.030$, $\eta2p=0.158$), where the exercise group demonstrated a substantial reduction in the abnormally high activation levels observed before the intervention. No significant main or interaction effects were observed for the medial MG, VM, VL, RF, and ST during this phase ($p>0.05$).

3.2. Midstance Phase

During the midstance phase (Table 5), which represents the single-limb support period, the ANOVA revealed a significant main effect of time for the VM ($p=0.028$, $\eta2p=0.161$) and GM ($p=0.009$, $\eta2p=0.220$). A significant main effect of group was identified for the MG ($p=0.015$, $\eta2p=0.198$) and GM ($p=0.003$, $\eta2p=0.295$). The exercise group showed overall higher activation patterns than the control group. A significant time×group interaction was observed for VM ($p=0.005$, $\eta2p=0.250$), showing that the exercise group decreased activation post-intervention, whereas the control group

remained stable, and for GM (p=0.012, η2p=0.210). The exercise group showed a significant reduction in GM activation post-intervention, which may suggest reduced compensatory muscular demands, although this interpretation is tentative without kinematic and kinetic data.

3.3. Push-Off Phase

During the push-off phase (Table 6), the propulsion generation was characterized by significant main effects of time for the MG (p=0.003, η2p=0.285) and BF (p=0.018, η2p=0.190). A significant main effect of group was found for the MG (p=0.005, η2p=0.255), VL (p=0.022, η2p=0.178), and BF (p=0.012, η2p=0.212). A significant time×group interaction effect was obtained for MG (p=0.010, η2p=0.218). The exercise group showed reduced peak push-off activation post-intervention, indicating optimized calf muscle motor drive during the propulsive phase, and BF (p=0.024, η2p=0.174). The exercise group showed decreased hamstring co-contraction post-intervention, which could indicate reduced joint stiffness; whether this reflects improved movement economy requires confirmation with kinetic and metabolic measures.

Table 1. Demographic and Clinical Characteristics of the Participants.

Characteristics	Experimental Group (n=15)	Control Group (n=15)	*p*-value
Age (years)	24.13±2.82	23.87±2.64	0.798
Height (cm)	176.47±5.12	175.93±4.87	0.771
Body Mass (kg)	74.20±6.81	73.80±7.15	0.876
BMI (kg/m2)	23.78±1.84	23.81±2.03	0.966
AKE Angle (°)	148.60±4.12	149.13±3.89	0.722
LBP Duration (months)	4.87±1.46	4.67±1.29	0.697
Note: Group comparisons performed using independent samples t-tests (α=0.05). BMI = Body Mass Index; AKE = Active Knee Extension; LBP = Low Back Pain.			

Table 2. The 8-Week ACSM Rehabilitation and Exercise Protocol.

Exercise Category	Specific Exercises	Prescription (Sets × Reps / Duration)	Progression Criteria (Weeks 1–4 vs. Weeks 5–8)
Warm-Up	Light walking, pelvic tilts, and active cat-camel stretches.	10 minutes	Gradual increase in active lumbar range of motion.
Stretching & Flexibility	Active-assisted hamstring stretches, hip flexor stretches, and piriformis stretches.	3 sets × 30-second hold (bilateral)	Weeks 1–4: Static stretching. Weeks 5–8: Proprioceptive neuromuscular facilitation (PNF) contract-relax techniques.
Core & Lumbar Stabilization	Quadruped bird-dog, abdominal bracing (draw-in maneuver), side planks, and standard bridges.	3 sets × 10–12 repetitions (or 15–30 s isometric hold)	Weeks 1–2: Stable surface, 15-second holds, bilateral support. Weeks 3–4: Stable surface, 20-second holds, increased repetitions. Weeks 5–6: Unstable surface (foam pad / Swiss ball), 20-second holds. Weeks 7–8: Unstable surface (foam pad / Swiss ball), 30-second holds or single-limb variation if tolerated.
Lower-Limb Strengthening	Wall squats, lunges, and calf raises.	3 sets × 10–15 repetitions	Weeks 1–4: Body weight only, 3 × 10–12 repetitions. Weeks 5–8: External resistance added gradually (resistance band or light dumbbells), 3 × 12–15 repetitions.
Cool-Down	Diaphragmatic breathing and gentle child's pose stretches.	5 minutes	Focus on relaxation and heart rate normalization.
Note. Progression was individualized according to pain tolerance, movement quality, and the ability to complete the prescribed exercise volume safely.			

Table 3. Electrode Placement Sites According to the SENIAM Protocol.

Muscle	Anatomical Electrode Placement Site
Tibialis Anterior (TA)	Upper 30% of the line connecting the tip of the fibula to the medial malleolus of the ankle.
Medial Gastrocnemius (MG)	Most prominent region of the medial muscle belly.
Gluteus Medius (GM)	50% of the distance along the line connecting the anterior superior iliac spine to the greater trochanter.
Biceps Femoris (BF)	50% of the line connecting the lateral condyle of the tibia to the ischial tuberosity.
Semitendinosus (ST)	50% of the line connecting the medial condyle of the tibia to the ischial tuberosity.
Rectus Femoris (RF)	50% of the distance along the line connecting the anterior superior iliac spine to the superior border of the patella.
Vastus Medialis (VM)	Distal 80% of the line connecting the anterior superior iliac spine to the patella, at approximately 55° relative to the longitudinal axis of the thigh.
Vastus Lateralis (VL)	Upper 35% of the distance along the line connecting the anterior superior iliac spine to the lateral border of the patella.

Table 4. Mean ± SD and ANOVA results of normalized EMG activity (%MVIC) during the Loading Response phase.

EMG	Control group		Exercise group		Main effect of time	Main effect of group	Main effect of time *group
	Before	After	Before	After	P (η2p)	P (η2p)	P (η2p)
TA	22.4 ± 3.1	22.1 ± 2.9	29.8 ± 4.2	23.5 ± 3.3	0.082 (0.105)	0.001* (0.340)	0.030* (0.157)
MG	12.3 ± 2.1	12.0 ± 1.9	13.1 ± 2.4	12.8 ± 2.0	0.450 (0.021)	0.380 (0.028)	0.720 (0.005)
VM	18.5 ± 2.8	18.2 ± 2.6	19.4 ± 3.1	18.9 ± 2.7	0.320 (0.035)	0.410 (0.025)	0.810 (0.002)
VL	19.1 ± 3.0	18.8 ± 2.7	20.2 ± 3.3	19.7 ± 2.9	0.290 (0.040)	0.350 (0.031)	0.880 (0.001)
RF	15.4 ± 2.2	15.1 ± 2.0	16.2 ± 2.5	15.8 ± 2.1	0.390 (0.027)	0.440 (0.022)	0.790 (0.003)
ST	14.2 ± 2.1	13.9 ± 1.8	15.0 ± 2.3	14.6 ± 1.9	0.350 (0.032)	0.390 (0.027)	0.850 (0.002)
BF	16.5 ± 2.4	14.1 ± 2.0	20.8 ± 3.1	16.9 ± 2.5	0.004* (0.266)	0.019* (0.182)	0.150 (0.075)
GM	18.2 ± 2.5	17.9 ± 2.3	26.4 ± 3.8	19.5 ± 2.7	0.011* (0.212)	0.030* (0.156)	0.030* (0.158)

Table Note: All participants (both exercise and control groups) had chronic non-specific low back pain and hamstring tightness. Tibialis Anterior (TA), Medial Gastrocnemius (MG), Gluteus Medius (GM), Biceps Femoris (BF), Semitendinosus (ST), Rectus Femoris (RF), Vastus Medialis (VM), And Vastus Lateralis (VL). *: $p<0.05$.

Table 5. Mean ± SD and ANOVA results of normalized EMG activity (%MVIC) during the Midstance phase.

EMG	Control group		Exercise group		Main effect of time	Main effect of group	Main effect of time *group
	Before	After	Before	After	P (η2p)	P (η2p)	P (η2p)
TA	11.2 ± 1.8	11.0 ± 1.6	12.1 ± 2.0	11.8 ± 1.7	0.510 (0.016)	0.280 (0.043)	0.790 (0.003)
MG	22.4 ± 3.2	22.0 ± 2.9	29.5 ± 4.1	28.7 ± 3.8	0.380 (0.028)	0.015* (0.198)	0.650 (0.008)
VM	20.2 ± 2.9	19.9 ± 2.7	28.4 ± 3.9	21.5 ± 3.1	0.028* (0.161)	0.085 (0.103)	0.005* (0.250)
VL	21.5 ± 3.1	21.1 ± 2.8	23.0 ± 3.4	22.4 ± 3.0	0.320 (0.035)	0.310 (0.037)	0.810 (0.002)
RF	12.8 ± 1.9	12.5 ± 1.7	13.9 ± 2.2	13.4 ± 1.8	0.410 (0.025)	0.290 (0.041)	0.750 (0.004)
ST	15.3 ± 2.3	15.0 ± 2.1	16.7 ± 2.5	16.2 ± 2.2	0.350 (0.032)	0.220 (0.055)	0.890 (0.001)
BF	14.8 ± 2.2	14.5 ± 1.9	16.1 ± 2.4	15.6 ± 2.1	0.420 (0.024)	0.250 (0.049)	0.820 (0.002)
GM	19.5 ± 2.7	19.1 ± 2.5	32.4 ± 4.6	23.2 ± 3.2	0.009* (0.220)	0.003* (0.295)	0.012* (0.210)

Table Note: All participants (both exercise and control groups) had chronic non-specific low back pain and hamstring tightness. Tibialis Anterior (TA), Medial Gastrocnemius (MG), Gluteus Medius (GM), Biceps Femoris (BF), Semitendinosus (ST), Rectus Femoris (RF), Vastus Medialis (VM), And Vastus Lateralis (VL). *: $p<0.05$.

Table 6. Mean ± SD and ANOVA results of normalized EMG activity (%MVIC) during the Push-off phase.

EMG	Control group		Exercise group		Main effect of time	Main effect of group	Main effect of time *group
	Before	After	Before	After	P (η2p)	P (η2p)	P (η2p)
TA	14.5 ± 2.1	14.2 ± 1.9	15.8 ± 2.4	15.3 ± 2.1	0.380 (0.028)	0.210 (0.058)	0.780 (0.003)
MG	35.6 ± 4.8	35.1 ± 4.5	49.8 ± 6.2	38.4 ± 5.1	0.003* (0.285)	0.005* (0.255)	0.010* (0.218)
VM	16.4 ± 2.3	16.1 ± 2.0	17.8 ± 2.6	17.3 ± 2.3	0.390 (0.027)	0.250 (0.049)	0.850 (0.002)
VL	17.2 ± 2.5	16.9 ± 2.2	22.4 ± 3.2	21.6 ± 2.9	0.280 (0.041)	0.022* (0.178)	0.720 (0.005)
RF	13.1 ± 1.9	12.8 ± 1.7	14.3 ± 2.1	13.9 ± 1.8	0.440 (0.022)	0.270 (0.045)	0.890 (0.001)
ST	18.5 ± 2.6	18.2 ± 2.4	20.1 ± 2.9	19.5 ± 2.7	0.350 (0.032)	0.240 (0.051)	0.810 (0.002)
BF	20.4 ± 2.9	20.0 ± 2.7	30.2 ± 4.1	22.8 ± 3.1	0.018* (0.190)	0.012* (0.212)	0.024* (0.174)
GM	15.8 ± 2.2	15.5 ± 2.0	17.4 ± 2.5	16.9 ± 2.2	0.410 (0.025)	0.190 (0.063)	0.790 (0.003)

Table Note: All participants (both exercise and control groups) had chronic non-specific low back pain and hamstring tightness. Tibialis Anterior (TA), Medial Gastrocnemius (MG), Gluteus Medius (GM), Biceps Femoris (BF), Semitendinosus (ST), Rectus Femoris (RF), Vastus Medialis (VM), And Vastus Lateralis (VL). *: $p<0.05$.

Discussion

This study showed that an 8-week ACSM-based exercise program produced phase-specific neuromuscular adaptations during walking in men with chronic non-specific LBP and hamstring tightness. The principal changes were observed in the TA and GM during loading response, VM and GM during midstance, and MG and BF during push-off, indicating that the intervention did not reduce muscle activity globally, but rather modified activation in muscles most directly involved in impact attenuation, pelvic control, single-limb stability, and propulsion. This selective pattern matters because it aligns with a transition from a compensatory motor strategy to a more efficient gait

pattern. However, this interpretation requires confirmation with concurrent kinematic and kinetic data. A clinically meaningful interpretation is that, before the intervention, individuals with LBP and hamstring tightness likely relied on a protective stabilization strategy during walking. Contemporary pain-adaptation models propose that in the presence of pain or movement-related threat, motor output is redistributed to increase trunk or joint stiffness and preserve function, often at the cost of movement efficiency [31, 32]. In gait, this may manifest as excessive activation of muscles responsible for distal control, frontal-plane pelvic stabilization, or knee stiffening. The elevated pre-intervention activation in key stance-phase muscles in the exercise group supports this interpretation. It suggests that chronic LBP is associated not only with pain but also with altered neuromuscular organization during locomotion [33,31].

The changes in TA and GM during loading response are particularly relevant. Loading response requires rapid weight acceptance, shock absorption, and control of trunk and pelvic motion. Increased TA activity may represent a distal stiffening strategy to secure foot placement and regulate tibial advancement more cautiously at initial contact, especially in individuals with impaired proximal control [34]. In parallel, increased GM activation may reflect a compensatory demand for frontal-plane pelvic stabilization during the transition to single-limb support [35, 36]. The reduction in both muscles after the intervention is consistent with participants requiring less neuromuscular guarding to accomplish early stance control. However, reduced effort or altered gait mechanics remain alternative explanations. This is biomechanically plausible because exercise programs combining flexibility and stabilization can improve intersegmental coordination and reduce the need for excessive co-contraction during functional tasks[19-20].

The findings for GM and VM during midstance further strengthen this interpretation. Midstance is a mechanically demanding phase in which the body progresses over a single support limb, requiring coordinated control of the pelvis, hip, and knee. The persistence of elevated GM activity before intervention suggests that frontal-plane control remained a major challenge in the exercise group, in agreement with evidence showing altered hip abductor function in people with chronic LBP [35, 37]. The concomitant reduction in VM activity after training may indicate less reliance on a knee-stiffening strategy once proximal control improved. This is an important point, because individuals with LBP may compensate for reduced lumbopelvic stability by shifting stabilizing demands distally, thereby increasing quadriceps recruitment during stance [32, 38]. The post-intervention decline in both GM and VM is therefore consistent with improved force-sharing across the kinetic chain and a reduced need for compensatory stabilization at the hip and knee. However, direct confirmation would require concurrent joint kinetic measures. The changes in MG and BF during push-off likely reflect improved propulsion mechanics and reduced posterior-chain over-recruitment. Push-off depends on coordinated plantarflexor output, hip extension, and efficient energy transfer through the lower extremity. In individuals with chronic LBP, increased MG and BF activation may indicate a strategy to preserve forward progression when proximal control or lower-limb coordination is compromised [33, 39]. Hamstring tightness may further accentuate this pattern by increasing passive resistance around the hip and pelvis, thereby disrupting normal segmental sequencing during terminal stance. The reduction in MG and BF activity after the intervention suggests that the participants achieved propulsion with less compensatory effort. This interpretation is consistent with the notion that improved hamstring extensibility and trunk–hip control can reduce unnecessary muscular demand and enhance locomotor economy [19, 40]. An important strength of the present findings is that not all recorded muscles changed significantly. This selective response argues against the possibility that the intervention lowered overall EMG amplitude because of reduced effort or measurement variability. Instead, the changes were concentrated in muscles that are biomechanically well positioned to compensate for deficits in lumbopelvic control and hamstring extensibility. Such selectivity increases the biological plausibility of the results and supports the interpretation that the intervention influenced task-relevant motor control rather than producing a generalized suppression of activation .

From a mechanistic perspective, the results fit within a kinetic-chain framework. Hamstring tightness may restrict normal pelvic motion and alter sagittal-plane mechanics, while chronic LBP may impair feedforward and reactive trunk control. Together, these impairments can shift stabilization demands toward the hip

abductors, ankle dorsiflexors, quadriceps, and plantarflexor–hamstring complex during walking [13, 36, 38]. The ACSM-based intervention likely addressed these constraints through multiple pathways: stretching may have reduced passive posterior-chain stiffness, strengthening may have improved load tolerance, and stabilization exercises may have enhanced proximal control. The net result appears consistent with a more physiologic distribution of muscular effort across stance, with less dependence on compensatory overactivation. Future studies should verify this interpretation using kinetic and metabolic measures. These findings also carry clinical significance. Exercise therapy for chronic LBP is often justified based on pain relief and functional improvement, yet the movement-based mechanisms underlying these benefits are less frequently demonstrated. This study suggests that one plausible mechanism is normalization of gait-related neuromuscular control. Excessive activation of stabilizing and propulsive muscles may be initially protective, but persistent overactivity can increase energetic cost, promote fatigue, and reinforce maladaptive motor behavior [31, 32, 41]. By reducing this excess demand in a phase-specific manner, multimodal exercise may improve not only symptoms, but also the efficiency and sustainability of walking in individuals with LBP and hamstring tightness. Overall, the present data support a focused clinical message: multimodal exercise appears more effective than a single-component approach for restoring gait-related neuromuscular behavior in this population. The combination of flexibility, core stabilization, and lower-limb strengthening seems particularly relevant because it targets both the mechanical and motor-control contributors to compensatory gait strategies. Therefore, the observed reductions in TA, GM, VM, MG, and BF activity are consistent with a more economical, better-coordinated locomotor pattern after intervention. However, reduced EMG amplitude could also reflect decreased effort or muscle capacity and should be interpreted cautiously without concurrent kinematic, kinetic, or metabolic data. Although the reductions in lower-limb EMG activity (e.g., GM from 26.4% to 19.5% MVIC during loading response) were statistically significant, their clinical meaningfulness warrants careful interpretation. Because validated clinical outcome measures of pain and disability (e.g., VAS, Oswestry Disability Index, Roland–Morris Questionnaire) were not collected, and because no minimal clinically important difference (MCID) thresholds currently exist for surface EMG-derived parameters, we cannot directly quantify the functional significance of these changes for pain, disability, or quality of life. To facilitate clinical interpretation, we additionally report standardized response means for the primary outcomes: gluteus maximus during loading response (SRM=0.86), vastus medialis during midstance (SRM=0.72), and medial gastrocnemius during push-off (SRM=0.69). These SRM magnitudes, in the large range according to Cohen's benchmarks, indicate that the observed reductions were robust at the individual level, suggesting the intervention produced clinically perceptible changes in neuromuscular activity rather than trivial effects significant only at the group level. Nevertheless, because EMG amplitude does not directly reflect patient-perceived function, these results should be regarded as evidence of improved neuromuscular output that may lay the groundwork for functional improvement, and future randomized controlled trials incorporating validated clinical outcomes are needed to establish the clinical significance of these changes.

Several limitations should be considered when interpreting the present findings. First, the sample was limited to young men, which restricts the generalizability of the results to women, older adults, and other clinical populations with chronic LBP. Second, although surface EMG provides useful information about muscle activation patterns, it does not directly quantify muscle force, deep muscle function, or the mechanical contribution of individual segments during gait. Third, kinematic and kinetic variables were not assessed concurrently; therefore, the interpretation that the intervention improved movement efficiency and lumbopelvic control remains inferential rather than directly confirmed. In addition, the authors did not collect validated clinical outcome measures of pain and disability (e.g., visual analog scale, Oswestry Disability Index, or Roland–Morris Questionnaire); therefore, they could not examine the relationship between the

observed neuromuscular changes and clinical improvements in pain or functional disability. Future studies should include such measures to determine whether clinically meaningful improvements accompany the observed EMG changes. Finally, the study did not include long-term follow-up, so the durability of the observed neuromuscular adaptations remains unclear.

Conclusion

The present study indicates that an 8-week ACSM-based exercise program can induce selective, phase-dependent changes in lower-limb muscle activity during walking in men with chronic non-specific LBP and hamstring tightness. The reductions observed in TA and GM during loading response, GM and VM during midstance, and MG and BF during push-off are consistent with a shift away from compensatory overactivation. However, reduced EMG amplitude could also reflect decreased effort, reduced muscle capacity, or altered gait mechanics. Future studies should incorporate kinematic, kinetic, and metabolic measures to confirm improved movement efficiency. Although the a priori sample-size calculation indicated adequate power for the prespecified medium effect size, the modest sample size (n=15 per group) may have limited the ability to detect smaller, potentially clinically meaningful effects; therefore, interpret the findings cautiously and confirm them in larger studies. Furthermore, the authors did not collect kinematic data (e.g., pelvic tilt, hip and knee joint angles, and trunk motion); therefore, interpretations of improved lumbopelvic control, pelvic stability, and movement efficiency remain inferential and should be confirmed in future studies incorporating concurrent kinematic and kinetic measurements.

Acknowledgment

The authors would like to express their sincere appreciation to all participants who voluntarily took part in this study.

Conflict of Interest Disclosures

The authors declare that they have no competing interests.

Funding Sources

None.

Authors' Contributions

M.B.: Conceptualization, Methodology, Supervision, Writing, Review & Editing. M.J.A., E.P. and R.N.: Data Curation, Formal Analysis, Investigation, Writing, Original Draft. All authors reviewed and approved the final version of the manuscript.

Ethical Statement

All procedures performed in this study involving human participants were conducted in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its subsequent amendments. Ethical approval was obtained from the Ethics Committee of the University of Mohaghegh Ardabili (Ethics Code: IR.UMA.REC.1403.090).

Declaration of Generative AI and AI-assisted technologies

The authors declare that no generative artificial intelligence (AI) and no AI-assisted technologies were used in the writing of this manuscript, data analysis, figure preparation, or in the generation of any text, images, or tables.

References

1. Maher C, Underwood M, Buchbinder R. Non-specific low back pain. The Lancet. 2017;389(10070):736-47.
2. Hartvigsen J, Hancock MJ, Kongsted A, Louw Q, Ferreira ML, Genevay S, et al. What low back pain is and why we need to pay attention. The Lancet. 2018;391(10137):2356-67.
3. Wu A, March L, Zheng X, Huang J, Wang X, Zhao J, et al. Global low back pain prevalence and years lived with disability from 1990 to 2017: estimates from the Global Burden of Disease Study 2017. Annals of translational medicine. 2020;8(6):299.
4. Ferreira ML, De Luca K, Haile LM, Steinmetz JD, Culbreth GT, Cross M, et al. Global, regional, and national burden of low back pain, 1990–2020, its attributable risk factors, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. The Lancet Rheumatology. 2023;5(6):e316-e29.
5. O'sullivan P. It's time for change with the management of non-specific chronic low back pain. BMJ Publishing Group Ltd and British Association of Sport and Exercise Medicine; 2012. p. 224-7.
6. Xu H-R, Zhang Y-H, Zheng Y-L. The effect and mechanism of motor control exercise on low back pain: a narrative review. EFORT open reviews. 2023;8(7):581-91.
7. van Dieʌn JH, Selen LP, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. Journal of electromyography and kinesiology. 2003;13(4):333-51.
8. Panjabi MM. The stabilizing system of the spine. Part I.

Function, dysfunction, adaptation, and enhancement. Journal of spinal disorders. 1992;5(4):383-9.
9. Kendall F, McCreary E, Provance P. Muscles, testing and function. Medicine and Science in Sports and Exercise. 1994;26(8):1070.
10. Gou Y, Lei H, Chen X, Wang X. The effects of hamstring stretching exercises on pain intensity and function in low back pain patients: a systematic review with meta-analysis of randomized controlled trials. SAGE Open Medicine. 2024;12:20503121241252251.
11. Gajdosik R, Lusin G. Hamstring muscle tightness: reliability of an active-knee-extension test. Physical therapy. 1983;63(7):1085-8.
12. Norkin CC, White DJ. Measurement of joint motion: a guide to goniometry: Fa Davis; 2016.
13. Esola MA, McClure PW, Fitzgerald GK, Siegler S. Analysis of lumbar spine and hip motion during forward bending in subjects with and without a history of low back pain. Spine. 1996;21(1):71-8.
14. McGill S. Low back disorders: evidence-based prevention and rehabilitation: Human Kinetics; 2025.
15. Da W. Biomechanics and motor control of human movement. XIKUA Boletнn Cientнfico de la Escuela Superior de Tlahuelilpan. 2013;1(1):1-21.
16. Burnfield M. Gait analysis: normal and pathological function. Journal of Sports Science and Medicine. 2010;9(2):353.
17. Sadler S, Cassidy S, Peterson B, Spink M, Chuter V. Gluteus medius muscle function in people with and without low back pain: a systematic review. BMC musculoskeletal disorders. 2019;20(1):463.
18. DA N. Kinesiology of the musculoskeletal system. Foundations for Physical Rehabilitation. 2002:251-310.
19. Hayden JA, Ellis J, Ogilvie R, Malmivaara A, van Tulder MW. Exercise therapy for chronic low back pain. Cochrane Database of Systematic Reviews. 2021.(9)
20. Searle A, Spink M, Ho A, Chuter V. Exercise interventions for the treatment of chronic low back pain: a systematic review and meta-analysis of randomised controlled trials. Clinical rehabilitation. 2015;29(12):1155-67.
21. Wang X-Q, Zheng J-J, Yu Z-W, Bi X, Lou S-J, Liu J, et al. A meta-analysis of core stability exercise versus general exercise for chronic low back pain. PloS one. 2012;7(12):e52082.
22. Smrcina Z, Woelfel S, Burcal C. A systematic review of the effectiveness of core stability exercises in patients with non-specific low back pain. International journal of sports physical therapy. 2022;17(5):766.
23. De Luca CJ. The use of surface electromyography in biomechanics. Journal of applied biomechanics. 1997;13(2):135-63.
24. Faul F, Erdfelder E, Lang A-G, Buchner A. G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behavior research methods. 2007;39(2):175-91.
25. Pirdaus D, Shofi G. EFEKTIVITAS MICRO-DOSE ECCENTRIC TRAINING TERHADAP FLEKSIBILITAS HAMSTRING PADA ATLET SEKOLAH BERDASARKAN ACTIVE KNEE EXTENSION TEST. Nusantara Hasana Journal. 2026;6(1):836-44.
26. Tan T, Tan X, Xie J, Zeng B, Xie J, Wang H, et al. Effects of exercise dose based on the ACSM recommendations on pain and disability in non-specific low back pain patients: a systematic review and meta-analysis of randomized controlled trials. Frontiers in Physiology. 2026;17:1725132.
27. Crookham J. A guide to exercise prescription. Primary Care: Clinics in Office Practice. 2013;40(4):801-20.
28. Piri E, Sobhani V, Jafarnezhadgero A, Arabzadeh E, Shamsoddini A, Zago M, et al. Effect of double-density foot orthoses on ground reaction forces and lower limb muscle activities during running in adults with and without pronated feet. BMC Sports Science, Medicine and Rehabilitation. 2025;17(1):54.
29. Chuang TD, Acker SM. Comparing functional dynamic normalization methods to maximal voluntary isometric contractions for lower limb EMG from walking, cycling and running. Journal of electromyography and kinesiology. 2019;44:86-93.
30. Potvin JR, Brown SH. Less is more: high pass filtering, to remove up to 99% of the surface EMG signal power, improves EMG-based biceps brachii muscle force estimates. Journal of Electromyography and Kinesiology. 2004;14(3):389-99.
31. Hodges PW, Tucker K. Moving differently in pain: a new theory to explain the adaptation to pain. Pain. 2011;152(3):S90-S8.
32. Van Dieʌn JH, Flor H, Hodges PW. Low-back pain patients learn to adapt motor behavior with adverse secondary consequences. Exercise and sport sciences reviews. 2017;45(4):223-9.
33. Lamoth CJ, Meijer OG, Daffertshofer A, Wuisman PI, Beek PJ. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. European Spine Journal. 2006;15(1):23-40.
34. Winter DA. Biomechanics and motor control of human gait: normal, elderly and pathological1991.
35. Nelson-Wong E, Gregory DE, Winter DA, Callaghan JP. Gluteus medius muscle activation patterns as a predictor of low back pain during standing. Clinical biomechanics. 2008;23(5):545-53.
36. Cooper NA, Scavo KM, Strickland KJ, Tipayamongkol N, Nicholson JD, Bewyer DC, et al. Prevalence of gluteus medius weakness in people with chronic low back pain compared to healthy controls. European Spine Journal. 2016;25(4):1258-65.
37. Kendall KD, Schmidt C, Ferber R. The relationship between hip-abductor strength and the magnitude of pelvic drop in patients with low back pain. Journal of Sport Rehabilitation. 2010;19(4):422-35.
38. Arendt-Nielsen L, Graven-Nielsen T. Muscle pain: sensory implications and interaction with motor control. The Clinical journal of pain. 2008;24(4):291-8.
39. Crossley KM, Bennell KL, Cowan SM, Green S. Analysis of outcome measures for persons with patellofemoral pain: which are reliable and valid? Archives of physical medicine and rehabilitation. 2004;85(5):815-22.
40. Page P. Current concepts in muscle stretching for exercise and rehabilitation. International journal of sports physical therapy. 2012;7(1):109.
41. Steiger F, Wirth B, De Bruin E, Mannion A. Is a positive clinical outcome after exercise therapy for chronic non-specific low back pain contingent upon a corresponding improvement in the targeted aspect (s) of performance? A systematic review. European Spine Journal. 2012;21(4):575-98.