RESEARCH ARTICLE

A Kinematic Analysis of Automatic Postural Responses during Predicted and Unpredicted Postural Perturbations in People with Low Back Pain

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Abstract

Objectives: Limited evidence exists on alterations in postural response kinematics following external perturbations in individuals with chronic low back pain (LBP). Therefore, this study aimed to investigate differences in automatic postural responses between individuals with chronic LBP and asymptomatic controls during forward translation of the support surface.

Methods: A total of 21 participants with chronic non-specific low back pain (LBP) and 21 age- and sex-matched healthy adults participated in this study. Participants were exposed to both predicted and unpredicted perturbations through forward translation of the support surface, which were analyzed using a motion analysis system. Angular displacements of the trunk and lower limbs were measured across four predefined time intervals corresponding to anticipatory postural adjustments (APA) and compensatory postural adjustments (CPA).

Results: In the unpredicted condition, trunk angular displacement during the APA1 phase was significantly lower in the LBP group compared with the control group ($P = 0.04$). A significant main effect of group was observed for hip ($P = 0.009$, $\eta p^2 = 0.17$), knee ($P = 0.01$, $\eta p^2 = 0.16$), and ankle ($P = 0.01$, $\eta p^2 = 0.14$) displacements during the CPA1 phase. Moreover, a significant group effect was found for knee ($P = 0.01$, $\eta p^2 = 0.20$) and ankle ($P = 0.04$, $\eta p^2 = 0.09$) displacements during the CPA2 phase. Participants with LBP exhibited greater lower-limb joint displacements than controls under predicted and unpredicted conditions.

Conclusion: Individuals with chronic low back pain (LBP) demonstrated altered kinematic strategies of the trunk and lower-limb joints in response to forward translation of the support surface. These findings suggest clinicians should consider evaluating and addressing automatic postural responses in this population.

Level of evidence: III

Keywords: Case control studies, Kinematics, Low back pain, Lower extremity, Postural balance

Introduction

Low back pain (LBP) is the most prevalent musculoskeletal disorder and the leading cause of years lived with disability worldwide.[1] The reported prevalence of LBP ranges from 30% to 80%, depending on the population studied.[2] Moreover, more than two-thirds of individuals with LBP experience a recurrence within 12 months after recovery.[3] Therefore, understanding the impairments associated with LBP is essential to inform intervention strategies and improve overall health outcomes.

A significant issue for individuals with low back pain (LBP) is impaired postural control during quiet standing,[4,5] self-initiated movements,[6] and particularly in response to external perturbations,[7] which is associated with an increased risk of falls. Researchers have employed various external stimuli, such as platform translations and virtual reality environments, to investigate automatic postural responses.[8] When the body is perturbed, the central

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nervous system engages two primary postural mechanisms—anticipatory and compensatory postural adjustments (APAs and CPAs, respectively)—to maintain stability against the perturbation.[9] APAs facilitate postural readiness before an expected perturbation, whereas CPAs restore the center of mass position following an expected or unexpected perturbation.[10] In predictable conditions, participants are informed about the direction and timing of each perturbation; in unpredictable conditions, no cues are provided regarding either.[11] These mechanisms induce alterations in electromyographic activity, center of pressure, and kinematics depending on the direction and magnitude of the applied forces.[12–14] The two forms of postural adjustments operate in coordination to preserve stability in response to external perturbations,[15] and the presence of APAs modulates the extent of compensatory muscle activity required.[16]

A systematic review with meta-analysis reported that individuals with chronic low back pain (LBP) exhibit delayed activation of trunk muscles—including the transverse abdominis, internal oblique, external oblique, and rectus abdominis—in response to both predicted and unpredicted external perturbations compared with healthy individuals. Furthermore, those with chronic LBP demonstrate delayed initiation of lumbar spine flexion during sudden weight release,[17] and show both a lag and a reduction in center of pressure displacement in response to unpredicted perturbations.[18,19] However, there is limited evidence regarding the kinematic responses of the lower limbs following external perturbations in individuals with chronic LBP. The lumbar spine plays a crucial role in transmitting forces and coordinating lower body movements.[20] Analyzing trunk and lower-limb kinematic changes in response to postural perturbations is essential for understanding the compensatory strategies employed by individuals with LBP and, potentially, for identifying therapeutic implications, particularly in rehabilitation medicine. Therefore, this study aimed to compare trunk and lower-limb kinematics between individuals with chronic non-specific LBP and healthy controls during the anticipatory (APA) and compensatory (CPA) phases of predicted and unpredicted external perturbations.

Materials and Methods

Study design

The present research was a case–control study approved by the Ethics Committee of Ahvaz Jundishapur University of Medical Sciences (approval number: IR.AJUMS.REC.1401.546). The study was conducted between October 2023 and April 2024. Written informed consent was obtained from all participants before their enrollment in the study.

Participants

Twenty-one participants with chronic low back pain (LBP; 16 males and five females) and 21 healthy individuals (16 males and five females) participated in this study. The two groups were matched for sex, age, weight, and height [Table 1]. Participants with LBP were included if they had been diagnosed with non-specific low back pain (i.e., no identifiable pathoanatomical cause),[21] were between 18 and 50 years of age, and had a history of LBP lasting 12 weeks or longer. Control participants were included if they had no history of back injury during the previous year. Exclusion criteria for all participants included any history of trauma, spinal or lower-extremity surgery, radicular symptoms, rheumatoid arthritis, neurological disorders, cardiovascular diseases, or diabetes. Furthermore, all participants were required to have no uncorrected visual or hearing impairments, no cognitive deficits, and to be free from medications that could affect balance.

Table 1. Demographics and clinical characteristics of participants

Demographic data	LBP group (n=21) Mean (SD)	Control group (n=21) Mean (SD)	P- value
Age (years)	33.95 (7.21)	29.23 (3.83)	.34
Height(cm)	176.85(9.78)	176.67(8.04)	.47
Weight(kg)	83.23(16.08)	77.52(14.74)	.11
VAS (1-10)	4.52(1.59)	-	NA
ODI (0-100)	19.19(6.44)	-	NA
Duration of LBP (month)	9.90(8.16)	-	NA

LBP: Low back pain, SD: Standard deviation; VAS: Visual analogue scale; ODI: Oswestry disability index; NA: not applicable.

Experimental procedures

Participants were exposed to forward slip perturbations in the sagittal plane using a movable platform to simulate external disturbances (100 mm displacement within 1 s; average acceleration = 0.1 m/s^2). Initially, participants were familiarized with the perturbation protocol. They were then asked to stand barefoot at the center of the platform with their feet shoulder-width apart and their hands placed on their chest. For safety reasons, participants wore a harness. They were instructed to gaze at a target 1.5 meters above them on the wall. Two perturbation conditions were tested: predictable, in which the timing of the disturbance was communicated to the participant beforehand, and unpredictable, in which the timing remained unknown. Perturbations were administered in a randomized order, and each condition was repeated three times. To minimize fatigue, a one-minute rest interval was provided between each trial.

Kinematic analysis

A three-dimensional motion analysis system (Qualisys Medical AB, Gothenburg, Sweden) was used to assess the lumbar region's and lower limbs' kinematics. Red reflective markers were placed at specific anatomical landmarks, including the spinous processes of the first, sixth, and twelfth thoracic vertebrae (T1, T6, and T12) and the fifth lumbar vertebra (L5); the anterior superior iliac spine (ASIS) and posterior superior iliac spine (PSIS) on both sides; the greater trochanter and lateral femoral condyle; the lateral malleolus of the ankle; the calcaneus; and the heads of the first and fifth metatarsals. Additionally, three triad marker clusters were attached to T6, T12, and L5, with one marker positioned on the spinous process and two others placed 2.5 cm laterally on each side. Kinematic data were collected at a sampling frequency of 200 Hz.

Data processing

The onset of postural perturbation was determined using a reflective marker positioned directly on the force plate. Baseline joint displacement was calculated within a −500 to −450 ms window preceding the perturbation onset (0 ms). This baseline value was subtracted from all subsequent outputs to ensure that the observed changes reflected actual differences rather than baseline variability. Angular displacements of the lumbar spine, hip, knee, and ankle were computed by comparing the positions of the body segments at the initial and final frames of each trial during the defined APA and CPA phases. Considering the approximate 50-ms electromechanical delay between skeletal muscle activation and muscle tension development, all phase windows were shifted forward by 50 ms. The analyzed phases were defined as follows: APA1 (−250 to −100 ms), APA2 (−100 to +50 ms), CPA1 (+50 to +200 ms), and CPA2 (+200 to +350 ms). Kinematic analysis was performed to quantify lower-limb and trunk segment positions. The measured variables included: ankle angle (the angle between the horizontal line and the lateral line through the fibular head), knee angle (the angle between the lateral fibular head and the line connecting the greater trochanter to the femoral head), and hip angle (the angle between the horizontal line and the greater trochanter). Trunk displacement for each phase was calculated as the linear distance between the C7 and PSIS markers in the sagittal plane, representing horizontal trunk motion.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 29.0; IBM Corp., Armonk, NY, USA). Demographic variables were compared between groups using independent-samples t-tests after confirming the normality of data distribution. A two-way mixed repeated-measures analysis of variance (ANOVA; 2 × 2 design) was conducted to examine the main effects and interaction effects of prediction condition (predictable vs. unpredictable) and group (LBP vs. control) on the dependent kinematic variables. Post hoc comparisons were performed using the Bonferroni correction. Effect sizes for all parameters were reported as partial eta squared (ηp^2), with values interpreted as small ($0.01 \leq \eta p^2 < 0.06$), medium ($0.06 \leq \eta p^2 < 0.14$), and large ($\eta p^2 \geq 0.14$) effects.[22] The level of statistical significance was set at $p < 0.05$.

Results

The characteristics of the participants are presented in Table 1. There were no statistically significant differences between the LBP and control groups in age, height, weight, or sex distribution. On average, participants with LBP reported minimal levels of pain and disability, and none experienced an exacerbation of pain during or after the experimental procedures. Table 2 presents the mean and standard deviation (SD) of angular displacements of the trunk and lower-limb joints across the four phases under both predicted and unpredicted conditions [Table 2]. The ANOVA revealed a significant group-by-prediction interaction for trunk displacement during the APA1 phase, with a large effect size ($P = 0.01$, $\eta p^2 = 0.16$) [Table 3]. Post hoc analysis indicated that, in the unpredicted condition, trunk displacement was significantly lower in the LBP group compared with the control group ($P = 0.04$). No significant group differences were observed in trunk displacement during the APA2 or CPA phases [Table 3]. In addition, no significant group-by-prediction interactions were found for lower-limb joints across the four phases ($P > 0.05$). However, a significant main effect of group was identified for hip ($P = 0.009$, $\eta p^2 = 0.17$), knee ($P = 0.01$, $\eta p^2 = 0.16$), and ankle ($P = 0.01$, $\eta p^2 = 0.14$) displacements during the CPA1 phase, all with large effect sizes. Similarly, during the CPA2 phase, the main effect of group was significant for knee ($P = 0.01$, $\eta p^2 = 0.20$) and ankle ($P = 0.04$, $\eta p^2 = 0.09$) displacements, representing large and medium effect sizes, respectively. Participants with LBP exhibited greater lower-limb joint displacements than controls during predicted and unpredicted CPA phases [Tables 2 and 3]. Furthermore, a significant main effect of prediction was found for the trunk segment during the APA2 phase, and for both the trunk and ankle during the CPA2 phase [Table 3]. All participants demonstrated greater trunk and ankle displacements under the unpredicted condition compared with the predicted condition [Table 2].

Discussion

This study compared postural responses to support-surface perturbations between individuals with chronic low back pain (LBP) and age- and sex-matched healthy controls. The results indicated that, under the unpredictable condition, participants with LBP exhibited reduced trunk displacement compared with controls during the APA1 phase. Furthermore, in predicted and unpredicted conditions, individuals with chronic LBP demonstrated greater lower-limb joint displacements (hip, knee, and ankle) during the CPA phases following forward translation of the support surface compared with the control group. While previous studies have examined kinematic characteristics in individuals with LBP during tasks such as the stand-to-sit transition[23] and in those with degenerative joint disease,[24] the present study is the first to investigate lower-limb kinematics associated with trunk motion in response to external perturbations among individuals with non-specific

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Table 2. Mean (SD) of kinematic measures of postural performance in response to forward perturbations

			LBP group Mean (SD)	Control group Mean (SD)
Trunk displacement	**APA1**	*predicted*	1.22(1.86)	2.05(3.26)
		Unpredicted	.75(.67)	1.09(1.23)
	APA2	*predicted*	3.55(1.56)	3.23(2.36)
		Unpredicted	4.39(2.37)	6.39(6.77)
	CPA1	*predicted*	25.09(33.48)	33.80 (35.84)
		Unpredicted	26.61(24.68)	34.74(32.79)
	CPA2	*predicted*	29.18(39.15)	31.18(35.16)
		Unpredicted	46.09(35.52)	45.03(40.70)
Hip displacement	**APA1**	*predicted*	.008(.26)	.19(.33)
		Unpredicted	.06(.12)	.06(.22)
	APA2	*predicted*	3.00(1.92)	2.58(.69)
		Unpredicted	2.56(1.72)	2.42(2.18)
	CPA1	*predicted*	5.91(6.03)	2.83(5.96)
		Unpredicted	4.27(6.25)	2.23(4.49)
	CPA2	*predicted*	4.11(3.49)	4.02(4.10)
		Unpredicted	4.76(3.74)	1.59(5.19)
Knee displacement	**APA1**	*predicted*	.21(.23)	.22(.49)
		Unpredicted	.05(.16)	.15(.29)
	APA2	*predicted*	1.19(.96)	1.14(.72)
		Unpredicted	1.62(.68)	.84(.92)
	CPA1	*predicted*	20.49(8.47)	15.77(7.43)
		Unpredicted	18.57(7.69)	13.78(6.53)
	CPA2	*predicted*	6.71(3.27)	3.54(3.28)
		Unpredicted	5.97(3.62)	3.54(2.50)
Ankle displacement	**APA1**	*predicted*	.13(.15)	.17(.42)
		Unpredicted	.04(.13)	.10(.07)
	APA2	*predicted*	3.09(1.16)	2.93(1.15)
		Unpredicted	3.40(1.54)	2.97(1.39)
	CPA1	*predicted*	10.05(3.28)	8.40(3.13)
		Unpredicted	10.15(5.58)	7.23(3.31)
	CPA2	*predicted*	2.66(3.97)	.59(3.22)
		Unpredicted	3.04(4.20)	2.60(3.70)

LBP; low back pain, SD; standard deviation, APA: anticipatory postural adjustments phase; CPA; compensatory postural adjustments phase

Table 3. Summary of analysis of variance for kinematic measures of postural performance: F ratios (P values; partial eta squared)

		$APA_{1\ phase}$	**$APA_{2\ phase}$**	**$CPA_{1\ phase}$**	**$CPA_{2\ phase}$**
Interaction effect					
Group × Prediction	*Trunk*	7.31(.01;.16)	2.20 (.14;.05)	.008 (.93;.001)	.07 (.78;.002)
	Hip	.58 (.44;.01)	.14 (.70;.004)	.55 (.46;.01)	2.90 (.09;.06)
	Knee	.28 (.59;.007)	.41 (.52;.01)	.01 (.98;.001)	.47 (.49;.01)
	Ankle	.03 (.85;.001)	.24 (.62;.006)	1.05 (.31;.02)	3.43 (.07;.09)

Table 3. Continued

Main effect					
Group	*Trunk*	1.08 (.30;.02)	.67 (.41;.01)	.67 (.93;.01)	.01(.92;.001)
	Hip	.58 (.44;.01)	.52 (.47;.01)	7.65 (.009;.17)	3.13 (.08;.07)
	Knee	.38 (.53;.01)	.74 (.39;.01)	6.54 (.01;.16)	7.65 (.01;.20)
	Ankle	.81 (.37;.02)	1.05(.31;.02)	6.55 (.01;.14)	4.33(.04;.09)
Prediction	*Trunk*	1.96 (.16;.01)	6.16 (.01;.14)	.16 (.69;.004)	7.22(.01;.16)
	Hip	.58 (.44;.01)	.67 (.41;.01)	1.04(.31;.02)	.96 (.33;.02)
	Knee	1.70 (.19;.04)	.01 (.90;.002)	1.39 (.24;.03)	.47 (.49;.01)
	Ankle	1.89 (.17;.04)	.35 (.55;.009)	.03 (.85;.001)	5.48 (.02;.12)

APA: anticipatory postural adjustments; CPA; compensatory postural adjustments phase

Several possible explanations may account for the altered postural responses observed in the LBP group compared with asymptomatic controls. First, proprioceptive impairments in the lumbopelvic region, combined with delayed activation of trunk muscles,[25–27] may influence the initial trunk positioning and the automatic postural reactions in individuals with LBP during perturbations. The central nervous system relies on accurate sensory information about lumbar spine position and motion to determine the location of the center of mass, particularly when rapid postural adjustments are required. Inaccuracy in lumbopelvic proprioceptive input among individuals with chronic LBP may lead to sensory reweighting, increasing dependence on lower-limb joints for maintaining balance. Second, the reduced lumbar range of motion in all planes commonly associated with LBP[20] may be compensated for by increased displacements of the lower-limb joints (hip, knee, and ankle), as observed in the present study. Increased stiffness in the LBP group before perturbation onset—likely due to changes in the passive properties of muscles and connective tissues, and to higher electromyographic activity of the back muscles (multifidus and erector spinae),[28]—may also explain the lower trunk displacement observed. This maladaptive postural response was most evident during unpredicted forward perturbations in individuals with chronic LBP. Such a response may be attributed to delayed spinal reflexes,[29] alterations in corticospinal control loops,[30] and increased muscle stiffness acting as a protective mechanism in unpredictable situations. This adaptive strategy may reduce the reliance on the trunk while promoting greater dependency on the lower-limb joints for maintaining postural stability.

The results of this study are consistent with previous research demonstrating impaired balance responses in individuals with chronic LBP following unexpected perturbations. A meta-analysis conducted in 2018 reported delayed activation of trunk muscles and delayed center of pressure responses to unexpected perturbations in individuals with chronic LBP compared with healthy controls.[7] However, few studies have examined kinematic strategies and alterations in the lower limbs in response to perturbations among individuals with LBP.[28,29] Our findings align with those of Jacobs et al.,[31] who demonstrated that individuals with LBP adopt modified kinematic strategies to minimize trunk extension by employing greater knee flexion in response to support-surface rotations. Similarly, Mok et al.[17] reported prolonged anterior translation of the spine in the LBP group compared with controls in response to sudden loading. In contrast, Dean et al.[24] found no significant differences in spine displacement following unpredicted forward perturbations between patients with symptomatic lumbar disc degeneration (LDD) and asymptomatic controls. This discrepancy may be explained by spinal pathology in participants with disc degeneration (Pfirrmann grade ≥6 at one or more lumbar levels) and differences in the magnitude of the applied perturbations. Although considerable variability exists among previous studies—particularly in terms of the type, magnitude, and predictability of perturbations, as well as participant characteristics and outcome measures—most of them support the current findings, indicating that both anticipatory (APA) and compensatory (CPA) postural adjustments are altered in individuals with chronic LBP. Therefore, effective rehabilitation strategies should be considered to specifically target these automatic postural responses.

Limitation

This study considered only forward translation of the support surface. Varying the direction of perturbations—such as lateral, backward, or vertical (upward/downward) translations—may elicit different postural responses in individuals with non-specific chronic LBP. Future research should therefore explore these additional perturbation directions to provide a more comprehensive understanding of postural control mechanisms in this population. Second, electromyographic activity of the trunk and lower-limb muscles and kinetic measurements such as changes in the center of pressure during perturbations were not evaluated in the present study. Additionally, cortical evoked potentials associated with postural responses were not recorded through electroencephalography (EEG). Finally, the participants in this study represented individuals with non-specific LBP and minimal disability; therefore, the findings cannot be generalized to populations with different levels of impairment or specific pathoanatomical causes of LBP.

Conclusion

Individuals with non-specific chronic low back pain (LBP)

demonstrated distinct kinematic strategies compared with asymptomatic controls in response to forward translation of the support surface. Participants with chronic LBP exhibited reduced trunk displacement during the APA1 phase under the unpredicted condition, while displaying greater lower-limb joint displacements (hip, knee, and ankle) during the CPA phases under both predicted and unpredicted conditions compared with the control group. These findings suggest that individuals with chronic LBP adopt compensatory movement strategies that shift postural control from the trunk to the lower limbs when exposed to destabilizing perturbations. Therefore, assessing spine and lower-limb kinematics under dynamic, destabilizing conditions may serve as a more sensitive indicator of altered postural control in chronic LBP populations.

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References

1. GBD 2021 Low Back Pain Collaborators. 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. Lancet Rheumatol. 2023;5(6):e316-e329. doi:10.1016/S2665-9913(23)00098-X.
2. Shokri P, Zahmatyar M, Falah Tafti M, et al. Non-spinal low back pain: Global epidemiology, trends, and risk factors. Heal Sci reports. 2023;6(9):e1533. doi:10.1002/hsr2.1533.
3. da Silva T, Mills K, Brown BT, et al. Recurrence of low back pain is common: a prospective inception cohort study. J Physiother. 2019;65(3):159-165. doi:10.1016/j.jphys.2019.04.010.
4. Berenshteyn Y, Gibson K, Hackett GC, Trem AB, Wilhelm M. Is standing balance altered in individuals with chronic low back pain? A systematic review. Disabil Rehabil. 2019;41(13):1514-1523. doi: 10.1080/09638288.2018.1433240.
5. Park J, Nguyen VQ, Ho RLM, Coombes SA. The effect of chronic low back pain on postural control during quiet standing: A meta-analysis. Sci Rep. 2023;13(1):7928. doi:10.1038/s41598-023-34692-w.
6. Garcez DR, da Silva Almeida GC, Silva CFO, et al. Postural adjustments and impairments in older adults with chronic low back pain. Sci Rep. 2021;11(1):4783. doi: 10.1038/s41598-021-83837-2.
7. Knox MF, Chipchase LS, Schabrun SM, Romero RJ, Marshall PWM. Anticipatory and compensatory postural adjustments in people with low back pain: a systematic review and meta-analysis. Spine J. 2018;18(10):1934-1949. doi: 10.1016/j.spinee.2018.06.008.
8. Kędziorek J, Błażkiewicz Mi. The impact of external perturbations on postural control. Acta Bioeng Biomech. 2024;26(2). doi: 10.37190/abb-02422-2024-02.
9. Santos MJ, Kanekar N, Aruin AS. The role of anticipatory postural adjustments in compensatory posture control: 2. Biomechanical analysis. J Electromyogr Kinesiol. 2010;20(3):398-405. doi:10.1016/j.jelekin.2010.01.002.
10. Santos MJ, Kanekar N, Aruin AS. The role of anticipatory postural adjustments in compensatory posture control: 1. Electromyographic analysis. J Electromyogr Kinesiol. 2010;20(3):388-397. doi: 10.1016/j.jelekin.2009.06.006.
11. Adkin AL, Campbell AD, Chua R, Carpenter MG. The influence of postural threat on the cortical response to unpredictable and predictable postural perturbations. Neurosci Lett.

2008;435(2):120-125. doi:10.1016/j.neulet.2008.02.018.
12. Mok NW, Brauer SG, Hodges PW. Postural recovery following voluntary arm movement is impaired in people with chronic low back pain. Gait Posture. 2011;34(1):97-102. doi: 10.1016/j.gaitpost.2011.03.021.
13. Radebold A, Cholewicki J, Panjabi MM, Patel TC. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine (Phila Pa 1976). 2000;25(8):947-954. doi: 10.1097/00007632-200004150-00009.
14. Mok NW, Brauer SG, Hodges PW. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine (Phila Pa 1976). 2007;32(19):E537-E543. doi: 10.1097/BRS.0b013e31814541a2.
15. Cesari P, Piscitelli F, Pascucci F, Bertucco M. Postural Threat Influences the Coupling Between Anticipatory and Compensatory Postural Adjustments in Response to an External Perturbation. Neuroscience. 2022;490:25-35. doi: 10.1016/j.neuroscience.2022.03.005.
16. Mehravar M, Yadollah-Pour N, Tajali S, Shaterzadeh-Yazdi MJ, Majdinasab N. The role of anticipatory postural adjustments and compensatory control of posture in balance control of patients with multiple sclerosis. Journal of Mechanics in Medicine and Biology. 2015;15(05):1550087.
17. Mok NW, Brauer SG, Hodges PW. Changes in lumbar movement in people with low back pain are related to compromised balance. Spine (Phila Pa 1976). 2011;36(1):E45-E52. doi: 10.1097/BRS.0b013e3181dfce83.
18. Etemadi Y, Salavati M, Arab AM, Ghanavati T. Balance recovery reactions in individuals with recurrent nonspecific low back pain: effect of attention. Gait Posture. 2016;44:123-127. doi: 10.1016/j.gaitpost.2015.11.017.
19. Henry SM, Hitt JR, Jones SL, Bunn JY. Decreased limits of stability in response to postural perturbations in subjects with low back pain. Clin Biomech. 2006;21(9):881-892. doi: 10.1016/j.clinbiomech.2006.04.016.
20. Errabity A, Calmels P, Han WS, et al. The effect of low back pain on spine kinematics: A systematic review and meta-analysis. Clin Biomech. 2023;108:106070. doi: 10.1016/j.clinbiomech.2023.106070.
21. Balagué F, Mannion AF, Pellisé F, Cedraschi C. Non-specific low back pain. Lancet. 2012;379(9814):482-491. doi: 10.1016/S0140-6736(11)60610-7.
22. Cohen J, eds. SPA for the BS. In: Statistical Power Analysis for the Behavioral Sciences. 2nd ed. Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers;1988.
23. Dehcheshmeh FG, Nourbakhsh MR, Farsani ZA, Bazrgari B, Arab AM. Kinematic Analysis of Pelvic and Lower Limb Joints during Stand-to-sit Movement in Individuals with Chronic Low Back Pain: A cross-sectional study. Arch Bone Jt Surg. 2024;12(8):587. doi: 10.22038/ABJS.2024.76840.3551.
24. Deane JA, Lim AKP, Phillips ATM, McGregor AH. Symptomatic individuals with Lumbar Disc Degeneration use different anticipatory and compensatory kinematic strategies to asymptomatic controls in response to postural perturbation. Gait Posture. 2022;94:222-229. doi: 10.1016/j.gaitpost.2021.03.037.
25. Lee AS, Cholewicki J, Reeves NP, Zazulak BT, Mysliwiec LW. Comparison of Trunk Proprioception Between Patients With Low Back Pain and Healthy Controls. Arch Phys Med Rehabil. 2010;91(9):1327-1331. doi:10.1016/j.apmr.2010.06.004.
26. Brumagne S, Cordo P, Lysens R, Verschueren S, Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine (Phila Pa 1976). 2000;25(8):989-994. doi:10.1097/00007632-200004150-00015.
27. Pinto SM, Cheung JPY, Samartzis D, et al. Differences in Proprioception Between Young and Middle-Aged Adults With and Without Chronic Low Back Pain. Front Neurol. 2020;11. doi:10.3389/fneur.2020.605787.
28. Vatovec R, Voglar M. Changes of trunk muscle stiffness in individuals with low back pain: a systematic review with meta-analysis. BMC Musculoskelet Disord. 2024;25(1):155. doi: 10.1186/s12891-024-07241-3.
29. Cholewicki J, Silfies SP, Shah RA, et al. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine (Phila Pa 1976). 2005;30(23):2614-2620. doi: 10.1097/01.brs.0000188273.27463.bc.
30. Taube W, Schubert M, Gruber M, Beck S, Faist M, Gollhofer A. Direct corticospinal pathways contribute to neuromuscular control of perturbed stance. J Appl Physiol. 2006;101(2):420-429. doi: 10.1152/japplphysiol.01447.2005.
31. Jacobs J V, Roy CL, Hitt JR, Popov RE, Henry SM. Neural mechanisms and functional correlates of altered postural responses to perturbed standing balance with chronic low back pain. Neuroscience. 2016;339:511-524. doi: 10.1016/j.neuroscience.2016.10.032.