

Review

Do people with low back pain walk differently? A systematic review and meta-analysis

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Abstract

Background: The biomechanics of the trunk and lower limbs during walking and running gait are frequently assessed in individuals with low back pain (LBP). Despite substantial research, it is still unclear whether consistent and generalizable changes in walking or running gait occur in association with LBP. The purpose of this systematic review was to identify whether there are differences in biomechanics during walking and running gait in individuals with acute and persistent LBP compared with back-healthy controls.

Methods: A search was conducted in PubMed, CINAHL, SPORTDiscus, and PsycINFO in June 2019 and was repeated in December 2020. Studies were included if they reported biomechanical characteristics of individuals with and without LBP during steady-state or perturbed walking and running. Biomechanical data included spatiotemporal, kinematic, kinetic, and electromyography variables. The reporting quality and potential for bias of each study was assessed. Data were pooled where possible to compare the standardized mean differences (SMD) between back pain and back-healthy control groups.

Results: Ninety-seven studies were included and reviewed. Two studies investigated acute pain and the rest investigated persistent pain. Nine studies investigated running gait. Of the studies, 20% had high reporting quality/low risk of bias. In comparison with back-healthy controls, individuals with persistent LBP walked slower (SMD = -0.59 , 95% confidence interval (95%CI): -0.77 to -0.42) and with shorter stride length (SMD = -0.38 , 95%CI: -0.60 to -0.16). There were no differences in the amplitude of motion in the thoracic or lumbar spine, pelvis, or hips in individuals with LBP. During walking, coordination of motion between the thorax and the lumbar spine/pelvis was significantly more in-phase in the persistent LBP groups (SMD = -0.60 , 95%CI: -0.90 to -0.30), and individuals with persistent LBP exhibited greater amplitude of activation in the paraspinal muscles (SMD = 0.52 , 95%CI: 0.23 – 0.80). There were no consistent differences in running biomechanics between groups.

Conclusion: There is moderate-to-strong evidence that individuals with persistent LBP demonstrate differences in walking gait compared to back-healthy controls.

Keywords: Biomechanics; Low back pain; Running; Walking

1. Introduction

The experience of acute low back pain (LBP) is almost universal, with up to 80% of people experiencing an acute episode of LBP in their lifetimes.¹ However, the greatest burden to individuals and society comes from the pain and disability associated with persistent LBP.^{2,3} Persistent LBP is characterized by symptoms lasting or recurring over months and years.⁴

Recently, researchers have differentiated between persistent LBP that is experienced almost every day (chronic LBP) and persistent LBP that follows a more episodic pattern (recurrent LBP).^{5,6} Although there are attempts to standardize definitions for recurrent and chronic patterns of persistent LBP,^{5,7,8} these definitions have not yet been widely adopted.

Walking and running gaits are frequently assessed in individuals with acute and persistent LBP during clinical evaluations and as part of observational and interventional research. Adaptations in gait biomechanics in individuals with LBP may include changes in spatiotemporal characteristics like speed or

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step length, kinematic characteristics like joint/segmental motion or coordination between joints/segments, kinetic characteristics like joint forces and torques, and electromyography (EMG) characteristics like amplitude and timing of muscle activation. The magnitude of trunk motion and joint loading during gait is relatively low.^{9–11} Despite this, due to the repetitive, cyclical nature of walking and running, adverse loading over time in response to changes in gait mechanics in the trunk or lower limbs may contribute to the onset, recurrence, or persistence of LBP symptoms.¹² Conversely, changes in gait mechanics in individuals with LBP may represent adaptive strategies used to mitigate the loading associated with locomotion and to protect pain-producing tissues. Recent work has highlighted the inconsistent evidence for biomechanical differences during tasks such as gait in individuals with persistent LBP compared to pain-free controls.¹³ In part, this is due to small sample sizes in individual studies. It is also potentially a result of heterogeneity in clinical back pain presentations.¹³ There is currently little consensus on how to effectively subgroup individuals with back pain based on their clinical presentation or movement characteristics. Therefore, it is critical to determine whether there are biomechanical traits that generalize across individuals with LBP during important functional activities such as walking and running. This will facilitate development of appropriate rehabilitation strategies for back pain management. Of the 2 recent reviews investigating gait in individuals with LBP,^{14,15} neither performed a quantitative synthesis of the results, and only one included EMG data.¹⁵

The aims of this systematic review, therefore, were to review and quantitatively synthesize evidence for differences in walking and running gait biomechanics in individuals with acute and persistent LBP compared to back-healthy controls.

2. Methods

This review was conducted based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (PRISMA). The protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO: CRD42018078746).

2.1. Search strategy

The search was conducted in the PubMed, CINAHL, SPORTDiscus, and PsycINFO databases without date restriction. The search terms combined keywords or MeSH terms for gait AND low back pain and were tailored for specific databases. The search strategy is shown in full in the supplementary materials. After removal of duplicates, 2 authors (JAS and VW) double-screened titles and abstracts based on the inclusion criteria (described in detail below). Full text manuscripts of remaining articles were then retrieved and additionally screened. Reference lists from retrieved articles, previous systematic reviews, and NCBI citation alerts were also checked. The search was initially conducted in June 2019. In September 2020, the search was repeated using identical search terms in the same databases to identify studies published since the original search.

2.2. Inclusion criteria

Included studies were peer reviewed, original research works that were available in English. Eligible studies compared gait variables between a group of individuals with acute or persistent LBP and a group of back-healthy controls. Study types included case-control, cross-sectional, and prospective cohort studies. Studies had to include objectively quantified gait data in 1 or more of the following categories: (a) spatio-temporal data (speed, distance, step and stride characteristics); (b) kinematic data (peak excursion or total range of motion in the thoracic or lumbar spine, pelvis, or lower extremities or coordination in kinematics between 2 or more joints/segments); (c) kinetic data (net joint moments, joint impulse, work, power); (d) ground reaction force data (vertical or horizontal ground reaction forces); (e) EMG (amplitude or timing of activation in the trunk or lower extremity musculature). Gait paradigms included overground and treadmill steady-state walking and running as well as walking under dual-task conditions involving additional mechanical or cognitive tasks. In studies that included pre- and post-intervention data, only the pre-intervention outcomes were included in this review. Studies were excluded if they were conference abstracts, case reports, dissertations, or review articles, if they did not report comparisons between individuals with and without LBP, or if LBP was experimentally induced in previously asymptomatic participants.

2.3. Study quality assessment

The quality of the reporting of the included studies was assessed along with risk of bias using a 16-criteria checklist^{16–19} (Table 1). A positive score was given for each criterion met by the study. A total quality score was calculated as the sum of all positive scores from Criteria 3 through 16 relevant to the study type (8, 12, and 9 for cross-sectional, case-control, and prospective cohort studies, respectively), and a percentage of the possible maximum score was calculated. Each study was independently scored by 2 authors (JAS scored all studies, and SPL, JB, and HLT each scored one third of the studies). Where there was a difference in scores, the 2 scoring authors discussed the criteria for which the scoring discrepancy occurred and reached a consensus on a final score. Studies were designated as having high reporting quality if they scored 50% or more.¹⁸

2.4. Data extraction

The following data were extracted from all eligible studies: study design, sample size, study inclusion/exclusion criteria, study population demographic characteristics, any additional metrics characterizing the LBP cohort, and the biomechanical outcomes. Data were extracted and double-checked by 3 authors (HS, VW, JAS).

Data were synthesized qualitatively if there were at least 2 articles with different populations that reported equivalent outcomes. Data were pooled for meta-analysis for outcomes in which there were equivalent data available from more than 3

Table 1
Checklist for assessment methodological quality for cross sectional, case-control, and prospective cohort study designs.

Domain and item number	Description	CS	CC	PC
Study objective				
1	Positive, if the study had a clearly defined objective	+	+	+
Study population				
2	Positive, if the main features of the study population are described (sampling frame and distribution of the population according to age and sex)	+	+	+
3	Positive, if cases and controls are drawn from the same population and a clear definition of cases and controls is given and if subjects with the disease/symptom in the past 3 months are excluded from the control group		+	
4	Positive, if the participation rate is at least 80% or if the participation rate is 60%–80% and the non-response is not selective (data shown)	+	+	+
5	Positive, if the participation rate at main moment of follow-up is at least 80% or if the non-response is not selective (data shown)			+
Measurements				
6	Positive, if data on history of the disease/symptom is collected and included in the statistical analysis	+	+	+
7	Positive, if the outcome is measured in an identical manner among cases and controls		+	
8	Positive, if the outcome assessment is blinded with respect to disease status	+	+	
9	Positive, if the outcome is assessed at a time before the occurrence of the disease/symptom		+	
Assessment of the outcome				
10	Positive, if the time-period on which the assessment of disease/symptom was based was at least 1 year			+
11	Method for assessing injury status: physical examination blinded to exposure status (+); self-reported: specific questions relating to symptoms/disease/use of manikin (+), single question (–)	+	+	+
12	Positive, if incident cases were included (prospective enrollment)		+	
Analysis and data presentation				
13	Positive, if the measures of association or group comparisons estimated were presented including confidence intervals	+	+	+
14	Positive, if the analysis is controlled for confounding or effect modification: individual factors	+	+	+
15	Positive, if the analysis is controlled for confounding or effect modification: other factors	+	+	+
16	Positive, if the number of cases in the final multivariate model was at least 10 times the number of independent variables in the analysis	+	+	+
Total possible score (sum of Items 3–16)		8	12	9

Abbreviations: CC = case-control; CS = cross sectional; PC = prospective cohort.

studies with different cohorts. Where there were multiple articles from the same author groups, the demographic characteristics of the study populations were checked to ensure that outcomes from the same population were not double-counted in the meta-analysis. Where necessary, authors of studies that did not report group means/standard deviations (SDs) were contacted to provide these data. Group averages/SDs were calculated from confidence intervals, standard errors, effect sizes, and median and interquartile ranges as needed using standard methods. For the pooled analyses, group averages/SDs from LBP or male/female sub-groups reported in some studies were combined.²⁰ A random effects model was used to calculate standardized mean differences (SMD) and 95% confidence intervals (95% CIs) for the SMD between the LBP and back-healthy groups (Review Manager Version 5.4.1; Cochrane Training, London, UK).²¹ Effect sizes of ≥ 0.8 were considered large, and those from 0.5 to < 0.80 were considered moderate. Group differences were significant if the *p* value for the test of overall effect was < 0.05 . The mean difference between groups in the original measurement units was calculated for those significant group comparisons where all studies used the same units of measurement, or it was calculated as a percentage of the control group value for outcomes with varying units of measurement. The heterogeneity in the results within the pooled analyses was evaluated for each outcome using the χ^2 test to detect significant heterogeneity and the I^2 statistic to quantify the heterogeneity, with I^2 greater than 0.75 indicative of substantial heterogeneity.²² Studies were excluded from pooled data analyses if the 95% CI for the group effect in that study did not overlap with the confidence interval for the SMD effect and if the removal of the outlier study did not affect the direction or significance of the pooled effect.²²

The level of evidence for the pooled analyses was defined using the following criteria:^{23,24}

- 1) Strong evidence—homogenous data ($\chi^2 p \geq 0.05$) pooled from studies of which at least 2 were high quality;
- 2) Moderate evidence—either heterogeneous data ($\chi^2 p < 0.05$) pooled from studies of which one was high quality, or homogenous data ($\chi^2 p \geq 0.05$) from lower quality studies;
- 3) Limited evidence—heterogeneous data ($\chi^2 p < 0.05$) from lower quality studies.^{23,24}

3. Results

The initial search identified 3272 articles (Fig. 1). Following the removal of duplicates, 2202 articles were available for further evaluation. An additional 7 articles were identified manually and during the repeat search in 2020. The abstracts and titles of 2209 articles were screened. Lastly, 124 full-text articles were retrieved and assessed for inclusion. A total of 97 articles were included. Attempts were made to contact authors of 22 studies that did not present group data for one or more variables of interest, and responses were received for 7 studies. Median score for reporting quality/risk of bias was 33% (range

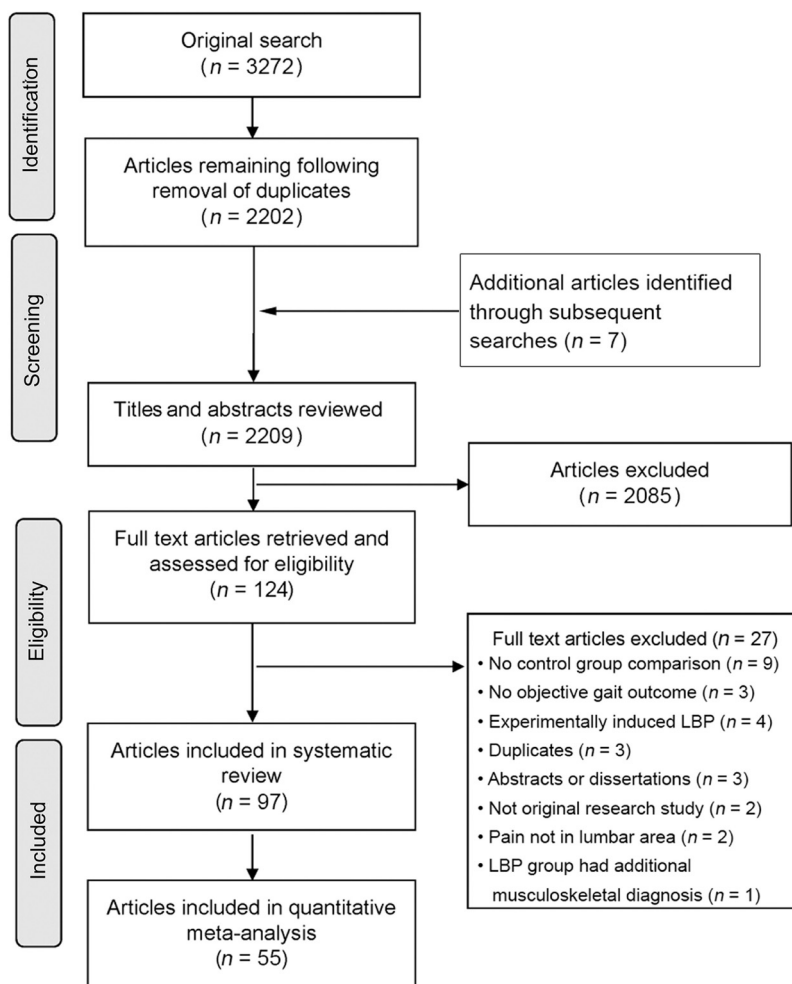


Fig. 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (PRISMA) flow diagram summarizing study selection processes.

13%–89%, Supplementary materials). Only 19 out of 97 studies received high scores for quality (scoring >50%, Table 2). The range of scores is similar to those reported in other systematic reviews investigating factors associated with musculoskeletal disorders that used the same quality assessment tool.^{18,19} Most studies did not report the participation rate; therefore, the potential influence of non-response was unclear. Very few studies reported blinding of researchers or presented confidence intervals in their analyses. Fifty-five articles were included in the quantitative meta-analysis.

3.1. Participant numbers and demographics

Of the included articles, 93 were case-control, 3 were cross-sectional,^{25–27} and 1 was a prospective cohort study.²⁸ Nine studies investigated running gait.^{27,29–36} Of the 97 included articles, there were 83 different study cohorts resulting in a total of 3364 individuals with ongoing LBP and 2315 back-healthy controls. Of the studies that reported participants' sex, the LBP groups included 767 males and 2075 females, and the control groups included 653 males and 961 females. The range of mean age was 21.4–73.6 years for the LBP groups and 18.7–73.5 years for the control groups. Participants with LBP

were described as having back pain, nonspecific or idiopathic LBP, chronic LBP, or recurrent LBP. However, as the criteria for these categories varied between studies, we did not subgroup participants based on these descriptors. Persistent LBP was commonly defined based on duration of symptoms, with minimum duration varying from 6 weeks to 1 year and the most frequent criterion being 3 months (Table 2). Two studies included participants described as having acute LBP, defined as symptom duration of less than seven days, who were re-tested once symptoms had resolved.^{37,38} Four studies sharing the same full or partial cohort included a separate group with a history of resolved LBP.^{29,30,33,34} Nineteen studies^{25,39–41,46,48,49,51,68,78,81,92,95,97,100,121,123,124,126} quantified minimum or maximum pain severity as part of their inclusion criteria (Table 2). Seven studies required LBP to be severe enough to impact function.^{32,36,39–43} Fourteen studies used pain frequency as a measure to define chronicity or recurrence of episodes.^{32,36,39–41,44–52} Location of LBP was defined in 16 studies, with location predominantly described as occurring below the costal margin and above the gluteal folds (Table 2).

Fourteen of the studies^{48,49,53–64} sub-grouped individuals with LBP based on pathoanatomical diagnoses. Among these

Table 2
Summary of included studies.

Author (year)	Sample size	LBP symptom inclusion criteria
Al-Obaidi et al. (2003) ⁶⁷	31 LBP; 24 Control	Duration > 7 weeks
Amir Rashedi Bonab et al. (2020) ⁵³	50 LBP; 20 Control	Physician diagnosis of lumbar disc herniation or chronic LBP; Symptoms within past 3 months
Ansari et al. (2018) ¹⁰⁰	21 LBP; 21 Control	Duration > 3 months; Intensity < 6/10
Anukoolkarn et al. (2015) ¹¹⁹	40 LBP; 40 Control	Duration > 3 months
Arendt-Nielsen et al. (1996) ⁹⁵	10 LBP; 10 Control	Diagnosis of idiopathic LBP; Intensity > 3/10
Bagheri et al. (2019) ⁷⁹	15 LBP; 15 Control	Duration > 3 months
Bagheri et al. (2020) ⁸⁰	15 LBP; 15 Control	Duration > 3 months
Becker et al. (2018) ⁶⁴	30 LBP; 20 Control	Diagnosis of stenosis, degenerative instability, or disc herniation with leg pain/neurogenic claudication
Carvalho et al. (2016) ¹²⁰	9 LBP; 9 Control	Duration > 3 months
Christe et al. (2017) ⁷⁵	11 LBP; 11 Control	Duration > 3 months
Cimolin et al. (2011) ⁸⁴	8 LBP; 20 Control (10 obese)	Duration > 3 months
Coyle et al. (2018) ⁴⁹	19 LBP; 19 Control	Duration ≥ 3 months; Frequency ≥ 4 days/week; Intensity ≥ 3/10; Pain radiation to/below knee when walking
Coyle et al. (2019) ⁴⁸	20 LBP; 20 Control	Duration ≥ 3 months; Frequency ≥ 4 days/week; Intensity ≥ 3/10; Pain radiation to/below knee when walking
Crosbie et al. (2013) ⁵⁰	19 LBP; 19 Control	Frequency ≥ 2 recurrences after initial episode; Episode duration > 24 h
da Fonseca et al. (2009) ⁶⁵	17 LBP; 11 Control	Duration > 6 months
Demirel et al. (2020) ¹²¹	66 LBP; 21 Control	Duration > 3 months; Intensity > 3/10
Ebrahimi et al. (2017) ⁹²	10 LBP; 10 Control	Duration > 3 months; Intensity 4–6/10; Disability 21%–60%
Farahpour et al. (2016) ⁶⁸	15 LBP; 30 Control (15 with pronation)	Intensity > 30/100; Disability index > 10
Farahpour et al. (2018) ⁸²	15 LBP; 30 Control (15 with pronation)	Intensity > 30/100; Disability index > 10
Gombatto et al. (2015) ⁷⁶	18 LBP; 18 Control	Diagnosis of LBP
Gutke et al. (2008) ²⁸	Prospective cohort of 308	Diagnosis of LBP or pelvic pain based on clinical assessment
Hamacher et al. (2014) ¹²²	12 LBP; 12 Control	Duration > 3 months
Hamacher et al. (2016) ¹²³	12 LBP; 12 Control	Duration > 3 months; Intensity ≥ 4/10
Hamacher et al. (2016a) ¹²⁴	14 LBP; 14 Control	Duration > 3 months; Intensity ≥ 4/10
Hamill et al. (2009) ²⁹	11 LBP; 22 Control (11 with history of resolved LBP)	Duration ≥ 4 months
Hanada et al. (2011) ⁹⁸	9 LBP; 9 Control	Duration > 8 months
Hart et al. (2009) ³²	25 LBP; 25 Control	Frequency ≥ 3 episodes over 3 years, or 5 episodes in total; Limitation to daily activities during episodes
Hart et al. (2009a) ³⁶	25 LBP; 25 Control	Frequency ≥ 3 episodes over 3 years, or 5 episodes in total; Limitation to daily activities during episodes
Healey et al. (2005) ¹²⁵	11 LBP; 11 Control	Duration > 6 months
Hemmati et al. (2017) ¹²⁶	40 LBP; 40 Control	Duration > 3 months; Intensity 3–5/10; Intensity < 3/10 at time of testing
Henchoz et al. (2015) ¹²⁷	13 LBP; 13 Control	Duration > 3 months
Hicks et al. (2017) ⁵¹	54 LBP; 54 Control	Duration > 3 months; Frequency ≥ 4 days/week; Intensity ≥ 3/10
Hines et al. (2018) ⁹³	25 LBP; 27 Control	Duration ≥ 6 weeks; ≥ 1 episode in week of testing
Huang et al. (2011) ⁵⁴	12 LBP; 12 Control	Lumbar disc herniation on CT scan
Jimenez-del-Barrio et al. (2020) ⁸⁶	20 LBP; 20 Control	Duration ≥ 3 months; Pain between costal margin and gluteal folds
Keefe et al. (1985) ⁷¹	18 LBP; 18 Control	Duration ≥ 6 months
Kendall et al. (2010) ⁸¹	10 LBP; 10 Control	Duration ≥ 6 weeks; Intensity ≥ 3/10; Pain between costal margin and gluteal folds
Kim et al. (2015) ¹²⁸	10 LBP; 10 Control	Duration > 2 months; Pain between inferior scapulae and cleft of buttocks
Kim et al. (2017) ⁹⁹	30 LBP; 15 Control	Duration > 7 weeks; Pain between costal margin and gluteal folds
Kuai et al. (2017) ⁵⁶	7 LBP; 26 Control	Diagnosis of lumbar disc herniation based on imaging and confirmed by 2 orthopedic physicians
Kuai et al. (2017a) ⁵⁵	7 LBP; 26 Control	Diagnosis of low lumbar disc herniation based on imaging and confirmed by 2 orthopedic physicians
Kuai et al. (2018) ⁵⁷	7 LBP; 26 Control	Diagnosis of low lumbar disc herniation awaiting surgery
Lamoth et al. (2002) ¹²⁹	39 LBP; 19 Control	Duration > 3 months; History of seeking medical treatment and physician diagnosis of nonspecific chronic LBP
Lamoth et al. (2006) ⁸⁹	12 LBP; 12 Control	Duration > 3 months; History of seeking medical treatment and physician diagnosis of nonspecific LBP
Lamoth et al. (2006a) ⁶⁶	22 LBP; 17 Control	Duration > 3 months; History of seeking medical treatment and physician diagnosis of nonspecific LBP
Lamoth et al. (2008) ⁹¹	12 LBP; 14 Control	Duration > 3 months; History of seeking medical treatment and physician diagnosis of nonspecific LBP

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Table 2 (Continued)

Author (year)	Sample size	LBP symptom inclusion criteria
Lee et al. (2002) ¹³⁰	40 LBP; 48 Control	Not specified
Lee et al. (2007) ⁶²	40 LBP; 20 Control	Current episode of LBP with or without unilateral referred leg pain; Currently receiving medical treatment
Lee et al. (2011) ¹³¹	30 LBP; 30 Control	Duration ≥ 3 months
MacRae et al. (2018) ⁸⁵	16 LBP; 16 Control	Duration ≥ 3 months
Manciopi et al. (2017) ⁴²	15 LBP; 15 Control	Duration > 12 months; ≥ 1 episode in previous 6 months; Pain between T12 and gluteal folds; Limitation to work or necessity for treatment
Müller et al. (2015) ³¹	11 LBP; 11 Control	Physician diagnosis of chronic, nonspecific LBP
Nadler et al. (2002) ²⁷	Cross-sectional cohort of 211	LBP in the previous year requiring treatment
Naliboff et al. (1985) ¹³²	68 LBP; 35 Control	Duration ≥ 6 months
Newell et al. (2010) ¹³³	12 LBP; 12 Control	Duration > 6 weeks; Physician diagnosis of nonspecific LBP
Novy et al. (1999) ⁷⁴	79 LBP; 46 Control	Not specified
Pakzad et al. (2016) ⁹⁷	30 LBP; 15 Control	Duration > 3 months; Intensity > 2/10; Disability > 12%; Pain between lower ribs and gluteal folds
Papadakis et al. (2009) ⁵⁸	35 LBP; 35 Control	Diagnosis of stenosis based on imaging
Papi et al. (2019) ¹³⁴	20 LBP; 20 Control	Nonspecific pain in the lower back
Poosapati Arjunan et al. (2010) ³⁵	4 LBP; 9 Control	Duration between 6 weeks and 4 months; Mild to moderate intensity
Prins et al. (2016) ⁷⁸	15 LBP; 15 Control	History of chronic LBP with symptoms in the previous 3 months; Intensity $\geq 2/10$
Queiroz et al. (2015) ¹³⁵	71 LBP; 142 Control; (71 with other musculoskeletal pain)	Duration ≥ 6 weeks
Rahimi et al. (2020) ⁸³	20 LBP; 20 Control	Duration > 3 months; Physician diagnosis of nonspecific LBP
Rodrigues et al. (2017) ¹³⁶	41 LBP; 42 Control	Duration > 3 months
Ryan et al. (2009) ¹³⁷	15 LBP; 15 Control	Duration > 3 months
Seay et al. (2011) ³⁴	14 LBP; 28 Control (14 with history of resolved LBP)	Duration ≥ 4 months
Seay et al. (2011a) ³³	14 LBP; 28 Control (14 with history of resolved LBP)	Duration ≥ 4 months
Seay et al. (2014) ³⁰	14 LBP; 28 Control (14 with history of resolved LBP)	Duration ≥ 4 months; Intensity mild to moderate
Selles et al. (2001) ⁹⁰	6 LBP; 6 Control	Duration ≥ 1 year
Simmonds et al. (1997) ¹³⁸	23 LBP; 23 Control	Not specified
Simmonds et al. (1998) ¹³⁹	44 LBP; 48 Control	Not specified
Simmonds et al. (2012) ⁶³	40 LBP; 20 Control	Current episode of LBP with or without unilateral referred leg pain; Currently receiving medical treatment
Smith et al. (2016) ⁴⁰	14 LBP; 14 Control	Duration > 1 year; Frequency ≥ 2 episodes in the previous year; Intensity < 0.5/10 at time of testing; Limitation to function during episodes; Unilateral pain between the 12th rib and gluteal fold
Smith et al. (2016a) ³⁹	14 LBP; 14 Control	Duration > 1 year; Frequency ≥ 2 episodes in the previous year; Intensity < 0.5/10 at time of testing; Limitation to function during episodes; Unilateral pain between the 12th rib and gluteal fold
Smith et al. (2017) ⁴¹	14 LBP; 14 Control	Duration > 1 year; Frequency ≥ 2 episodes in the previous year > 24 hours; Intensity < 0.5/10 at time of testing; Limitation to function during episodes; Unilateral pain between the 12th rib and gluteal fold
Spengelink et al. (2002) ¹⁴⁰	47 LBP; 10 Control	Duration ≥ 6 months
Sung et al. (2017) ⁷²	37 LBP; 45 Control	Recently recovered from episode of LBP
Sung et al. (2017a) ⁴⁷	51 LBP; 59 Control	Frequency ≥ 1 year incidence of recurrence; Recently recovered from episode of LBP
Swain et al. (2019) ⁴³	26 LBP; 21 Control	≥ 1 episode within the last 2 months; Limitation to dance practice during episode(s); Pain between the 12th rib and gluteal folds
Tagliaferri et al. (2019) ²⁵	Cross-sectional cohort of 1182	Intensity $\geq 1/10$ at time of testing
Tanigawa et al. (2018) ²⁶	Cross-sectional cohort of 52	Presence of pain in low back, pubic symphysis, or sacroiliac joints
Taylor et al. (2003) ³⁷	8 LBP; 8 Control	Duration < 7 days; Pain in lumbar region with or without radiation to upper leg
Taylor et al. (2004) ³⁸	12 LBP; 11 Control	Duration < 7 days; Pain in lumbar region with or without radiation to upper leg
Teixeira da Cunha-Filho et al. (2010) ⁷³	30 LBP; 30 Control	Duration > 3 months; Patient seeking medical care
Tomkins-Lane et al. (2012) ⁶⁰	94 LBP; 32 Control	Diagnosis of spinal stenosis or non-stenotic LBP based on clinical assessment with or without imaging
Tong et al. (2007) ⁶¹	36 LBP; 12 Control	Diagnosis of spinal stenosis or non-stenotic LBP based on clinical assessment with or without imaging
van den Hoorn et al. (2012) ⁸⁷	13 LBP; 12 Control	Not specified

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Table 2 (Continued)

Author (year)	Sample size	LBP symptom inclusion criteria
van der Hulst et al. (2010) ¹²	63 LBP; 33 Control	Duration >3 months; Pain between scapulae and buttock clefts with or without radiation to legs
van der Hulst et al. (2010a) ⁹⁶	63 LBP; 33 Control	Duration >3 months; Pain between scapulae and buttock clefts with or without radiation to legs
Vickers et al. (2017) ¹⁴¹	25 LBP; 30 Control	Duration ≥6 months
Vogt et al. (2001) ⁴⁴	34 LBP; 22 Control	Frequency ≥ 1/2 day in past 12 months (single or multiple episodes); Clinician diagnosis of idiopathic chronic LBP; Pain between T12 and gluteal folds
Vogt et al. (2003) ⁵²	17 LBP; 16 Control	Frequency ≥ 1/2 day in past 12 months (single or multiple episodes); Physician diagnosis of idiopathic chronic LBP; Pain between T12 and gluteal folds
Voloshin et al. (1982) ¹⁴²	24 LBP; 39 Control	Not specified
Weiner et al. (2006) ⁴⁵	163 LBP; 160 Control	Duration ≥3 months; Frequency daily or almost every day; Intensity moderate or greater
Yamakawa et al. (2004) ⁵⁹	60 LBP; 22 Control	Diagnosis of spinal stenosis based on imaging; Criteria for non-stenotic LBP not specified
Yazdani et al. (2018) ⁹⁴	11 LBP; 13 Control	Duration ≥6 months; Physician diagnosis of idiopathic chronic LBP
Zahraee et al. (2014) ⁴⁶	20 LBP; 20 Control	Duration ≥6 months; Frequency on most days per week; Intensity >2/10; Pain between T12 and gluteal folds; Mechanically induced symptoms

Note: Studies in bold received a high-quality/bias score (>50%).

Abbreviations: CT = computer tomography; LBP = low back pain.

articles, 32 participants were diagnosed with lumbar disc herniation.^{53–57} One hundred and eight participants were described as having spinal stenosis.^{58–61} Forty participants had LBP with pain referred to the lower limbs.^{62,63} Thirty-nine participants were described as having radiculopathy.^{48,49} Lastly, 1 article recruited a pain group with LBP diagnosed as stenosis, degenerative instability, or disc herniation.⁶⁴ For the control groups, most studies required controls to be healthy, pain-free individuals, and they were frequently matched by sex and age to the experimental group. Exclusion criteria varied widely across studies. Eighty studies explicitly excluded participants whose LBP was associated with known pathoanatomical diagnoses such as radiculopathy and participants who had a history of spinal surgery.

3.2. Walking gait

Due to limited available evidence, data were not synthesized or pooled from the 2 articles reporting walking gait in individuals with acute LBP. All of the following analyses are for study cohorts with persistent LBP. Findings from the meta-analysis for persistent LBP, including metrics of strength of evidence, are summarized in Table 3.

3.2.1. Spatiotemporal characteristics

Thirty studies and a total of 1570 participants were included in the pooled analysis of preferred walking speed in individuals with persistent LBP. Individuals with persistent LBP walked more slowly than back-healthy individuals (SMD = -0.59, 95%CI: -0.77 to -0.42), test for overall effect $p < 0.001$; heterogeneity $I^2 = 58%$, $\chi^2 p < 0.001$) (Fig. 2A). The mean difference in speed between groups was 0.12 m/s. Two studies that quantified gait biomechanics across a range of controlled treadmill speeds noted that individuals with LBP were not able to maintain gait at controlled speeds of greater than approximately 1.4 m/s.^{65,66} Pooled data with 687 participants indicated that individuals with LBP had shorter stride

length when walking at preferred speed (SMD = -0.38, 95%CI: -0.60 to -0.16, effect $p < 0.001$; $I^2 = 45%$, $\chi^2 p = 0.05$) (Fig. 2B). The mean difference in stride length between groups was 0.05 m. Pooled evidence from 510 participants demonstrated no significant difference between groups in cadence (SMD = -0.19, 95%CI: -0.46 to 0.09, effect $p = 0.18$; $I^2 = 53%$, $\chi^2 p = 0.03$) (Fig. 2C). Duration of single limb support did not differ between groups (SMD = -0.17, 95%CI: -0.56 to 0.23, effect $p = 0.41$; $I^2 = 72%$, $\chi^2 p = 0.001$).^{47,67–71} Five studies with a total of 385 participants reported step width, and pooled analysis also showed no difference between groups (SMD = 0.34, 95%CI: -0.06 to 0.74, effect $p = 0.10$; $I^2 = 72%$, $\chi^2 p = 0.006$).^{42,47,51,70,72} In studies investigating distance walked in 5 min, individuals with LBP walked significantly shorter distances than did healthy controls.^{73,74}

3.2.2. Kinematic characteristics—single segment/joint

The peak-to-peak amplitude of lumbar motion was modeled with markers fixated around the spinal levels of T12,⁴⁴ L1,^{43,50} L2,⁶⁶ and L3,^{43,75,76} referenced to the global coordinate system, to L5, or to the pelvis. Pooled analyses of 5 studies with 193 participants examining the magnitude of lumbar motion during walking gait demonstrated that there was no significant difference in amplitude of motion in the axial plane in individuals with persistent LBP (SMD = 0.07, 95%CI: -0.26 to 0.39, effect $p = 0.69$; $I^2 = 21%$, $\chi^2 p = 0.28$).^{43,44,50,66,75} The peak-to-peak frontal plane lumbar motion was pooled from 6 studies and also demonstrated that there was no difference between LBP and control groups (SMD = -0.13, 95%CI: -0.39 to 0.13, effect $p = 0.32$; $I^2 = 0%$, $\chi^2 p = 0.95$).^{43,44,50,66,75,76} As few studies investigated sagittal plane lumbar motion, these data were not pooled, but no studies reported a significant difference between individuals with and without LBP.^{44,50,76}

Upper trunk motion was modeled with markers fixated on the sternum,^{42,77} acromioclavicular joints,³¹ and/or the spinal levels of C7,^{31,42} T1,^{43,75} T3,⁶⁶ and T6.^{50,78} Motion was referenced to the global coordinate system, or to the lower thoracic,

Table 3

Summary of meta-analysis results for walking gait. The direction of the difference between groups is indicated for comparisons with a significant standardized mean difference. Effect sizes and the strength of the evidence are shown for all comparisons.

Outcome	Group difference ^a	Effect size ^b	Strength of evidence ^c
Preferred walking speed	LBP < control	Moderate	Moderate
Stride length	LBP < control	Small	Strong
Duration of single limb support	n/s	Small	Moderate
Cadence	n/s	Small	Moderate
Step width	n/s	Small	Moderate
Upper lumbar axial motion	n/s	Small	Strong
Upper lumbar frontal motion	n/s	Small	Strong
Upper trunk axial motion	n/s	Small	Strong
Upper trunk frontal motion	n/s	Small	Strong
Upper trunk sagittal motion	n/s	Small	Moderate
Pelvis axial motion	n/s	Small	Moderate
Pelvis frontal motion	n/s	Small	Strong
Hip sagittal motion	n/s	Small	Limited
Axial in-phase thorax/pelvis coordination	LBP > control	Moderate	Strong
Peak vertical ground reaction force first peak	n/s	Small	Limited
Peak vertical ground reaction force second peak	n/s	Small	Limited
Amplitude of lumbar paraspinal activation	LBP > control	Moderate	Moderate

^a Standardized mean difference between groups.

^b Effect sizes of <0.5 are small; 0.5–0.79 are moderate; >0.79 are large.

^c Strong evidence = homogenous data pooled from studies of which at least 2 were high quality; moderate evidence = either heterogeneous data pooled from studies of which one was high quality, or homogenous data from lower quality studies; limited evidence = heterogeneous data from lower quality studies.

Abbreviations: LBP = low back pain; n/s = no significant.

lumbar, or pelvic segments. Nine studies with 307 participants reported data for peak-to-peak axial plane motion in the thorax during walking that could be pooled.^{31,42,43,50,66,75,77–79} There was no difference between groups (SMD = –0.10, 95%CI: –0.33 to 0.13, effect $p = 0.40$; $I^2 = 0\%$, $\chi^2 p = 0.56$) (Fig. 3A). Evidence pooled from 6 studies demonstrated that the amount of frontal plane motion also did not differ between groups (SMD = –0.16, 95%CI: –0.45 to 0.12, effect $p = 0.26$; $I^2 = 13\%$, $\chi^2 p = 0.33$).^{42,43,50,66,75,79} Of the studies investigating sagittal plane motion that could be pooled, there was no significant difference between individuals with and without LBP (SMD = –0.54, 95%CI: –1.30 to 0.22, effect $p = 0.17$; $I^2 = 0\%$, $\chi^2 p = 0.40$).^{31,42,50,79} Intra-subject stride-to-stride variability of lumbar or thoracic kinematic motion was reported in several studies, but without consistent methodological approach or findings.^{44,66,80}

Data from 5 studies with 179 participants investigating pelvis kinematics in the axial plane during walking were pooled.^{31,44,50,66,78} Amplitude of peak-to-peak pelvis motion

was modeled with markers on the sacrum^{44,50,66,78} and greater trochanters³¹ and was usually referenced to a global coordinate system. There was no significant difference between groups (SMD = –0.12, 95%CI: –0.42 to 0.18, effect $p = 0.43$; $I^2 = 50\%$, $\chi^2 p = 0.09$). Similarly, in the frontal plane, the amplitude of pelvic motion did not differ between groups (SMD = –0.09, 95%CI: –0.75 to 0.58, effect $p = 0.80$; $I^2 = 43\%$, $\chi^2 p = 0.15$).^{44,50,66,81} Sagittal plane pelvis kinematics were only available in 2 studies, and neither reported a significant group difference.^{44,50}

Eight studies reported hip kinematics during steady-state gait.^{50,52,56,82–86} Pooled data available from 4 of these studies with 128 participants indicated no difference in total sagittal plane hip motion in individuals with LBP (SMD = –0.08, 95%CI: –0.43 to 0.27, effect $p = 0.65$; $I^2 = 0\%$, $\chi^2 p = 0.94$). In the frontal plane, two studies reported reduced motion,^{83,84} with a large effect size occurring in a study of obese adults,⁸⁴ and 2 reported no difference.^{56,82} In the axial plane, 2 studies reported no difference,^{56,82} and one reported decreased motion in individuals with LBP.⁸³ Knee flexion during late stance or swing phase was reduced in 3 out of 5 studies that reported knee kinematics,^{31,82–84,86} but there were no consistent trends evident for frontal or axial plane knee motion or for ankle motion.

3.2.3. Kinematic characteristics—inter-segmental coordination

Multiple studies investigated coordination of kinematic motion between spinal segments during steady-state gait.^{33,34,50,66,77,78,87–91} Most examined coordination in motion between the thorax and pelvis across a variety of controlled walking speeds. Time- and frequency-domain techniques were used to quantify phase relations between segments. In the axial plane, pooled analyses of 185 participants demonstrated that motion between the thoracic spine and the lumbar spine/pelvis was significantly more in-phase in individuals with LBP than in controls (SMD = –0.60, 95%CI: –0.90 to –0.30, effect $p < 0.001$; $I^2 = 0\%$, $\chi^2 p = 0.96$) (Fig. 3B). The mean difference in relative phase between groups was 25%. This finding was supported by three out of four additional studies from which data could not be pooled.^{33,87,88,90} Multiple studies also investigated the stride-to-stride variability of inter-segmental coordination in the axial plane during steady-state gait,^{33,66,87,89–91} with 3 reporting less variability in individuals with LBP.^{87,89,90}

Fewer studies reported frontal or sagittal plane intersegmental coordination, and there was insufficient data available to pool the findings. In the frontal plane, coordination between the thorax and lumbar spine/pelvis was reported as being more in-phase in individuals with LBP^{33,34,50} or the same.⁶⁶ In the sagittal plane, one study reported more in-phase coordination in individuals with LBP,⁹² and 2 others sharing the same cohort reported no difference.^{33,34} Stride-to-stride variability in sagittal thorax–pelvis coordination was reported as being less in individuals with LBP⁹² or the same.³³

3.2.4. Kinetic characteristics

Five studies reported kinetic measures in the lower extremities during walking.^{82,84,85,93,94} In 3 studies examining sagittal

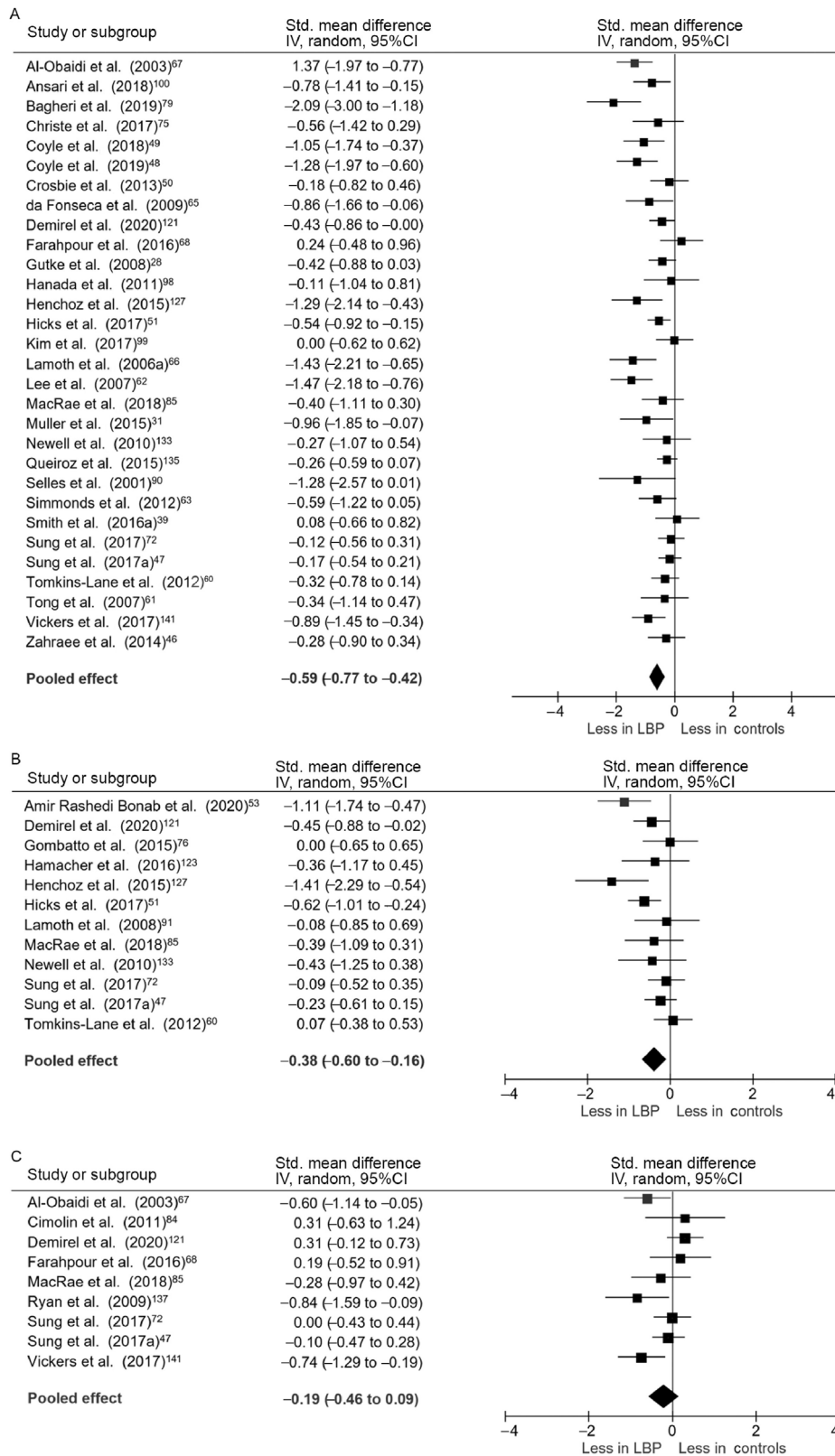


Fig. 2. Meta-analysis of spatiotemporal gait variables. (A) Preferred walking speed, (B) Stride length, and (C) Cadence. 95%CI = 95% confidence interval; IV = inverse variance; LBP = low back pain; Std. = standardized.

plane total net joint moments at the hip, there were no differences between individuals with and without LBP.^{82,85,93}

3.2.5. Ground reaction forces

Five studies investigated ground reaction forces during walking gait,^{46,62,65,68,94} of which 4 studies with 138 participants had data that could be pooled. There was no difference between groups when walking at preferred speed in peak vertical ground reaction forces during either the first (SMD = 0.29, 95%CI: -0.54 to 1.11, effect $p = 0.49$; $I^2 = 82\%$, $\chi^2 p < 0.001$) or second vertical force peaks (SMD = -0.21, 95%CI: -0.86 to 0.46, effect $p = 0.56$; $I^2 = 72\%$, $\chi^2 p = 0.01$).

3.2.6. Muscle activation characteristics

Fifteen studies included EMG measures of the paraspinal and abdominal musculature during walking, using preferred and controlled walking speeds.^{12,35,39,42,52,64,66,82,89,95-100} Of these, 14 used surface EMG electrodes, and one used intramuscular EMG. Most studies investigated amplitude of muscle activation, with two investigating timing of activation.^{39,52} During steady-state gait, pooled analyses of data from 210 participants for surface EMG of the low lumbar paraspinal musculature across the entire stride cycle or within the stance phase indicated that individuals with LBP had greater amplitude of activation (SMD = 0.52, 95%CI: 0.23-0.80, effect $p < 0.001$; $I^2 = 1\%$, $\chi^2 p = 0.40$) (Fig. 3C). On average, individuals with LBP exhibited 31% greater activation than back-healthy controls.

For the abdominal musculature, 2 high-quality studies reported increased rectus abdominis activity during some or all phases of gait,^{12,97} 1 reported decreased activity,⁹⁸ and 2 studies, including 1 high-quality study, reported no difference in activity between groups.^{82,99} Three studies reported no difference in amplitude of external oblique activation,^{12,82,99} and 1 high-quality study reported increased activation during some subphases of gait.⁹⁷ One study reported increased peak activation in internal oblique compared to controls,⁸² 1 reported variable results depending on subphases of gait,⁹⁸ and another reported decreased activity during several gait subphases.⁹⁹

3.3. Running gait

Although there were nine articles that reported the biomechanics of running gait,^{27,29-36} 6 of these studies appeared to report data from the same 2 populations.^{29,30,32-34,36} As a result, running data were only available for 5 different population cohorts, and there were insufficient comparable data for meta-analyses. One study investigated Division 1 collegiate athletes.²⁷ Four articles shared the same cohort of recreational runners that were required to run at least 20 km per week in order to participate in the study.^{29,30,33,34} Two articles shared the same cohort of participants, who were described as being recreationally active.^{32,36} Two articles did not report any requirement for running distance/frequency or activity level in its participants.^{31,35} Most of the articles reported the preferred running speed of the participants, but running biomechanics were quantified during running at a controlled speed in 5 of the articles.^{29,30,33-35}

3.3.1. Spatiotemporal characteristics

Preferred running speed did not differ between groups.^{31,33,36}

3.3.2. Kinematic characteristics—single segment/joint

During running, the extent of upper trunk motion in the axial plane was reported as being less in individuals with LBP³¹ or the same,^{32,34} and there was no difference in sagittal^{31,32,34} or frontal plane motion.^{32,34} One study utilizing a controlled running speed found that axial plane pelvic motion was reduced in individuals with persistent LBP,³¹ and another using participant preferred running speed reported that it was greater in individuals with LBP.³⁴ Two studies reported sagittal plane hip kinematics during running and found no difference in motion between individuals with and without LBP.^{29,36} Similarly for running, sagittal plane knee motion either did not differ^{31,32} or was reduced²⁹ in individuals with LBP, and sagittal plane ankle motion was not significantly different.^{29,31}

3.3.3. Kinetic characteristics

During running, sagittal plane hip moments did not differ between groups.^{29,36} One running study reported increased external flexion moment at the knee in individuals with LBP,³⁶ but there was no difference in another study.²⁹

4. Discussion

To our knowledge, this is the first comprehensive systematic review and meta-analysis of walking and running gait in individuals with LBP. Individuals with persistent LBP walk differently than back-healthy controls. These differences are most evident in spatiotemporal characteristics, in patterns of inter-segmental coordination, and in paraspinal muscle activation. Current evidence does not indicate that LBP is associated with a difference in the amplitude of motion in the trunk or lower extremities during walking. There is insufficient evidence to determine whether running biomechanics are altered in individuals with LBP.

Pooled data demonstrated that individuals with persistent LBP choose to walk more slowly than individuals without back pain. The mean difference between groups exceeded previously reported values for the minimal clinically important difference in preferred walking speed across a range of studies in adults.¹⁰¹ In 2 studies that reported that LBP patients were unable to walk at controlled fast speeds,^{65,66} it was unclear whether this inability was due to pain, fear of pain, or deconditioning. Al Obaidi et al.⁶⁷ examined influences on preferred walking speed and found that fear avoidance and pain anticipation significantly predicted reduced walking speed in individuals with persistent LBP. Stride length was also reduced in individuals with LBP, but the mean difference for this comparison did not exceed reported values for minimal detectable change.^{102,103} It is possible that individuals with LBP use a strategy of slower walking velocity and slightly reduced stride length to minimize the kinematic and kinetic demands of walking.^{37,62}

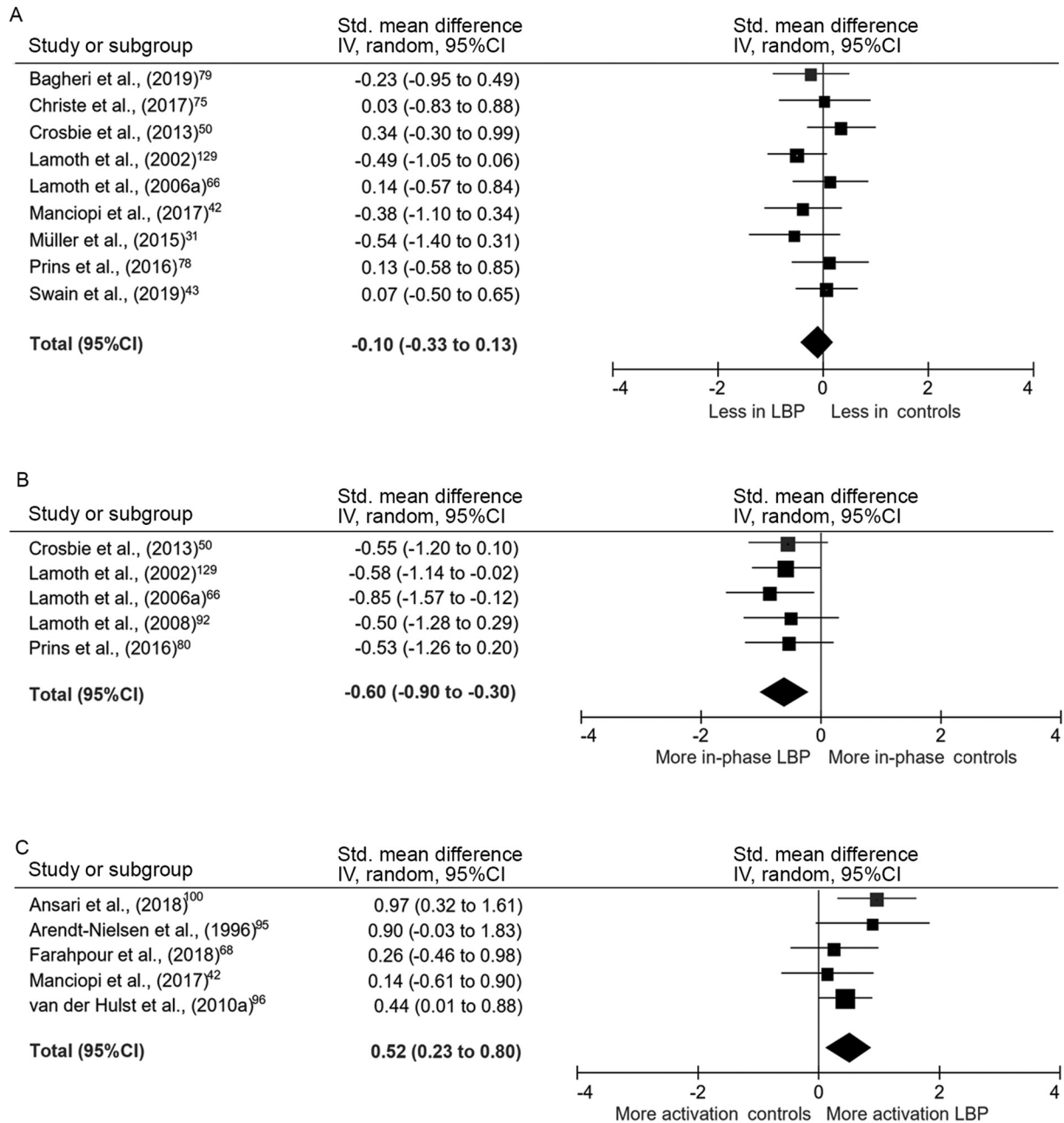


Fig. 3. Meta-analysis of kinematic and EMG gait variables. (A) Axial plane thoracic motion, (B) Axial plane inter-segmental coordination, and (C) Amplitude of paraspinal activation. 95%CI = 95% confidence interval; EMG = electromyography; IV = inverse variance; LBP = low back pain; Std. = standardized.

This study found strong evidence for altered phase relations between motion in the thorax and the pelvis during walking in individuals with persistent LBP. In back-healthy controls, the pattern of coordination, or relative motion, between the upper trunk and pelvis in the axial plane is speed dependent, becoming more anti-phase as speed increases.⁶⁶ Even when walking at controlled speeds, individuals with LBP exhibited greater in-phase movement patterns. This may be due to a reduced ability to dissociate movement between the trunk and pelvis in these individuals. Anti-phase coordination during fast walking helps to generate elastic recoil between the thorax and the pelvis and may also contribute to minimizing total body angular

momentum in the axial plane.¹⁰⁴ Therefore, the reduction in anti-phase coordination in individuals with LBP may help to explain their decreased gait speed and reduced stride length.

Our meta-analysis demonstrates that individuals with LBP have greater lumbar paraspinal activation during walking. Phasic muscle activity in the paraspinals occurs bilaterally at initial contact and during the double support phases of the gait cycle.^{105,106} This activation controls sagittal and frontal plane motion between the trunk and the pelvis.¹⁰⁷ The amplitude of this activity is low, typically less than 20% of maximum voluntary activation for walking,^{39,108} although this increases to up to 100% of maximum for fast running.¹⁰⁸ Acutely,

increased activation during gait may be adaptive if it serves to reduce motion and protect pain-sensitive tissues. After the acute phase, it may also be a compensation for the muscle weakness related to atrophy and fatty infiltration that occurs in multifidus in response to back pain¹⁰⁹ or for proprioceptive dysfunction.^{110,111} However, over time this increased activation in individuals with LBP may contribute to recurrence due to increased compressive spinal loading.¹¹² Increased paraspinal activation may also be the cause of the reduced anti-phase coordination described above as a result of increased axial stiffness limiting dissociation of motion between the upper trunk and the pelvis.^{87,104}

In comparison with the paraspinals, abdominal muscle activity during locomotion is much more variable between individuals and more dependent upon locomotor speed.^{105,107,108} This variability within healthy individuals is perhaps due to the redundancy of the abdominal muscle system and likely accounts for the lack of consistent differences in abdominal activation in individuals with LBP in the present review. It should be noted that all but one of the studies in this review used surface EMG. Surface EMG cannot selectively quantify activation in the transversus abdominis and multifidus muscles.^{113,114} Isolated postural impairment of these deep muscles has been a focus of LBP research and treatment for some years. However, our findings are consistent with a recent systematic review of anticipatory postural adjustments indicating that the postural function of the superficial muscles in the abdominal and paraspinal systems are also affected by LBP.¹¹⁵

Current evidence does not consistently demonstrate a significant difference in joint or segmental excursion in the trunk or lower extremities during walking or running in individuals with LBP. This may be because the joint range of motion utilized in the thoracic and lumbar spines during walking and slow running is a small proportion of the available range.¹⁰⁸ This is in contrast with other activities, such as standing forward flexion, where significant reductions in lumbar range of motion in individuals with LBP have been observed.¹¹⁶ The amplitude of hip and knee motion during walking and running is a greater proportion of available range, but the current evidence does not consistently support interdependency between back pain and lower limb gait kinematics.

This review identified methodological challenges that limit the current understanding of associations between gait biomechanics and LBP. As noted earlier, many studies quantified gait biomechanics at participants' preferred walking speeds. This makes it difficult to determine whether the observed differences in characteristics like stride length in individuals with LBP are due to these individuals walking more slowly, or whether they are still evident when walking speed is controlled. Unfortunately, as very few studies quantified gait characteristics at prescribed gait velocities for all participants or adjusted for gait velocity in their analyses,^{51,62,66,76} there is insufficient evidence to separate the influence of slower gait velocity from the independent effects of LBP on these characteristics. Future research should focus on assessment of walking and running gait at a range of speeds in individuals with LBP. This will demonstrate how these individuals modulate

gait biomechanics in response to increasing mechanical demands as speed increases. It will also allow researchers to clearly differentiate between an inability to maintain faster walking speed and a preference to walk more slowly in LBP populations. Importantly, all but one of the studies in this review used a case-control or cross-sectional design. As there are so few prospective studies tracking changes in gait biomechanics and LBP status over time, it is still not known whether walking or running biomechanics affect the development of or persistence of symptoms. It is also not clear how observed biomechanical differences in individuals with LBP influence joint or tissue loading. As a result of these limitations in existing research, it cannot currently be determined whether the biomechanics of gait associated with LBP are adaptive or maladaptive. To answer this important question, future studies should probe longitudinal relationships between gait biomechanics and symptoms.

The limited number of available studies that investigate running precluded meta-analyses of the running biomechanics in individuals with LBP. Additionally, in this review we were unable to probe differences in gait between sub-groups of individuals with LBP. The inconsistent sub-grouping or classification of individuals with persistent LBP remains problematic. Multiple classification systems based on biomechanical or kinesiopathological factors have been proposed, but none are fully supported by available evidence.¹¹⁷ Some studies investigated patient sub-groupings based on age, sex, weight, pain severity, or psychosocial factors. Other studies in this review recruited participants based on pathoanatomical diagnoses, such as herniated lumbar discs, degenerative instability, or spinal stenosis. Studies varied in how these pathoanatomical diagnoses were made, and the inconsistent relationship between pathoanatomical findings and clinical presentation or outcome is now well known.¹¹⁸ However, the heterogeneity of the participants included in this review results in greater generalizability of the findings to the broader clinical population. Finally, the quality of the reporting in the included studies was low overall, with only 19 studies receiving a high-quality assessment score.

5. Conclusion

We found that individuals with a history of persistent LBP exhibit different biomechanical characteristics during walking gait than back-healthy controls. Differences are most evident in spatiotemporal characteristics, coordination between the thorax and pelvis coordination, and paraspinal muscle activation. However, it is not known whether the strategies evident in individuals with LBP during gait are adaptive or maladaptive. Prospective research following the transition from acute to persistent pain or symptom resolution will provide insight into the effect of these altered gait mechanics on the trajectory of back pain symptoms over time.

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Authors' contributions

JAS designed the study, supervised the literature search, conducted quality scoring, extracted the data, performed some of the analyses, and drafted the manuscript; HS extracted the data, performed some of the analyses, and drafted the manuscript; JJB assisted with study design, conducted quality scoring, and edited the manuscript; HLT assisted with study design, conducted the quality scoring, and edited the manuscript; VW conducted the literature search, extracted the data, and edited the manuscript; SPL assisted with study design, conducted the quality scoring, and edited the manuscript. All authors have read and approved the manuscript, and agree with the order of presentation of the authors.

Competing interests

The authors declare that they have no competing interests.

Supplementary materials

Supplementary materials associated with this article can be found in the online version at doi:10.1016/j.jshs.2022.02.001.

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