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Clinical Studies

The association of hip range of movement, and its side-to-side asymmetries, and non-specific lower back pain in adults aged 40 years and older

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ABSTRACT

Background: The hip joint's close association and coupling with the lumbar spine may influence its axes of rotation during closed-chain movement. Consequently, altered hip range of movement (ROM) may potentially foment the symptoms of non-specific lower back pain (NSLBP), warranting its investigation. A quantitative, cross-sectional, analytical design was employed to determine whether NSLBP has an association with altered hip ROM and dominance-aligned hip side-to-side asymmetries.

Methods: Ninety-three convenience sampled participants with and without NSLBP were enrolled. These were assigned to the Lumbar Pain Group (LPG), comprising 61 individuals, 32 males and 29 females or the Control Group (CG), consisting of 32 pain-free volunteers, 18 males and 14 females. Individuals with hip conditions were excluded from the study. Participants completed a Baecke questionnaire and their BMI, Beighton score and leg dominance was established. The following asymptomatic, dominance aligned hip ROM was measured with an Inertial Measurement Unit (IMU): hip flexion ROM in supine; hip extension ROM in prone; Modified Thomas Test (MTT) with knee extension; straight leg raise (SLR); abduction in supine; adduction in supine and hip internal (IR) and external rotation (ER) in prone, sitting and supine in 30° flexion using a framework.

Results: The LPG and CG showed similar hip ROM. However, significant hip side-to-side asymmetries presented in the LPG's sagittal, frontal and transverse planes but only presented in the CG's transverse plane hip ranges. The dominant side usually displayed the smaller range.

Conclusions: Without further evidence, it is unlikely that symmetrically atypical coupled hip ranges should be ignored in the treatment of NSLBP, but a stronger case exists for attention to the hip ROM side-to-side asymmetries. Compounded, multi-planar hip ROM side asymmetries may be one of the causes of NSLBP.

Introduction

In spite of lower back pain's high lifetime prevalence of 84%, of which 90% is attributed to NSLBP, NSLBP remains a challenging condition to treat [1,2]. This is because it cannot be attributed to a specific source [3]. Composite movement is determined by the sum of individual ranges from different joints along the kinetic chain. Adjacent to one another, the hip and lumbar spine couple to achieve these ends [4,5]. Closed-chain movement sees the pelvis moving atop the hips and, in a similar fashion, the lumbar spine moving over the pelvis. Thus, limited or excessive hip ROM potentially changes the lumbar spine's axes of rotation, placing undue strain and stress on the soft tissues and bone of the lumbar spine [6,7]. Consequently, hip ROM has an important role in lumbar mechanisms of injury and symptom generation [8,9].

To our knowledge, the effects of leg dominance on hip ROM and its association with lower back pain has not been studied in the general population. Earlier research explored dominance's effects in sporting cohorts [10–14] but omitted the characteristic in other contexts [9,15–21]. As dominance has been suggested as a potential cause of NSLBP [22], this too warranted investigation.

Exploring whether hip treatment has a place in NSLBP management, this research set out to determine whether hip hypomobility or hypermobility is associated with NSLBP. Secondly, to establish whether hip ranges' side-to-side differences (asymmetry) are associated with NSLBP. Sorted by leg dominance, these asymptomatic

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hip ranges were compared in adults with and without this lumbar condition.

Methods

Study design

This was a quantitative, cross-sectional, analytical study. This study received ethics clearance from the Human Research Ethics Committee (Medical) of the University of the Witwatersrand.

Setting

The pilot, reliability and main study testing took place at the University of the Witwatersrand's Physiotherapy Department and the researcher's private practice.

Participants

A systematic review reported that low back pain is most prevalent among people from 40 to 80 years of age [23], peaking between the ages of 41 and 50 [19]. Consequently, individuals over the age of 40 were recruited for the study.

Qualifying participants were grouped based on their history of NSLBP: those with and those without. The LPG consisted of individuals with recurrent (defined as multiple episodes of pain occurring on less than half the number of days in a 12-month period) or chronic localized non-specific lumbar pain (experiencing symptoms on more than half the number of days in a 12-month period, occurring in a single or multiple episodes) [14]. The CG comprised volunteers who had had lower back pain for less than two weeks in the past year, in order to exclude exercise induced muscle soreness [13]; had not received musculoskeletal treatment within the previous six months [24] and/or regularly taken anti-inflammatories or analgesics, ≥2 times per week [25] for a lumbar condition. Individuals with hip conditions were excluded from the study - defined by current pain in the groin, anterior thigh, lateral hip or buttock; those receiving or had received musculoskeletal treatment, within the previous six months [24] or were regularly making use of anti-inflammatories or analgesics, ≥2 times per week [25], for a hip condition. Furthermore, participants were excluded if they had congenital hip problems; lumbar, knee or hip surgery; histories of hip and knee trauma; body mass index (BMI) ≥29.9 kg/m^2; neurological conditions; significant leg length discrepancy ≥20 mm; systemic inflammatory conditions; spinal pathologies as per Van Dillen et al. [14]; chronic symptomatic knee problems; prolonged steroid use; were pregnant or osteoporotic [13,14,26,27,28]. Participants were rescheduled if they had exercised on the assessment day or had taken anti-inflammatories or analgesics 24 hours prior to testing.

Volunteers were recruited through signage in surrounding medical practices and sports clubs; posts on Facebook community groups; leaflets at sporting events and via electronic pamphlet dissemination to sporting association's members. Interested parties were emailed an information sheet, a screening questionnaire and an informed consent form beforehand.

Instrumentation, outcomes measures and variables

Data on the participant's age, gender, height (stadiometer, Wenzhou Kindcare Import and Export Company, Wenzhou, China; Model KT-GF06A), weight (body scale, Tanita Corporation, Tokyo, Japan; Model UM-075), mobility (Beighton score, [29]) and participation in work, sport and leisure (Baecke habitual activity questionnaire, [30]) were collected.

Leg dominance was determined by posing the question to the participants: "If you would kick a ball on a target, which leg would you use to kick the ball?". This phrase has been reported to have a 100% agreement between self-reported and observed leg dominance (for bilateral mobilizing activities) in healthy men and women [31]. To help facilitate communication, the word "kick" was substituted for their word "shoot."

The following non-dominant and dominant side's passive hip ROM were measured and discerned by an IMU and its application (Shemesh Health Solutions Pty Ltd, Johannesburg, South Africa): hip flexion in supine; hip extension in prone; MTT with knee extension; SLR; abduction in supine; adduction in supine and hip internal and ER in prone, sitting and supine on a 30° framework. The MTT, with knee extension, was employed to measure iliopsoas length.

Procedures

The pilot study consisted of 11 university students and age-appropriate adults sourced from the public. Ten physiotherapy students and volunteers under the age of 40 were enlisted for the reliability study. The test-retest reliability study analysis was performed between one and two days apart. The main study's convenience sample consisted of 93 participants.

On arrival, a volunteer completed a Baecke habitual activity questionnaire and their BMI and Beighton Scale score were established. Dominance was determined post hoc. Passive joint range testing was used to negate inaccuracies caused by muscle weakness. The findings of multiple validation studies, once conducted to appreciate the IMU's functionality in differing test positions, and skeletal landmarks identified and marked on the day, governed the IMU's placement and orientation. The participant's non-restricting clothing was kept free of the sensor, which was fixed in position with elastic strapping. Drawn in lots, the following hip ranges were block randomized and measured: hip flexion in supine, SLR and MTT positions; extension in prone; abduction and adduction in supine; and rotation in prone, sitting and supine (with the hip angled over a 30° wedge; see Fig. 1A, B and C). The side and the order of hip rotation position testing were also randomized. This reduced the increasing joint flexibility's effects on the final results. Details of the participant positioning, skeletal landmarks, IMU's placement, the application's settings and the testing procedure appears in Online Supplementary Table 1.

The author alone performed all the passive hip ROM assessments to reduce inter-tester inconsistencies [13]. Passive end range was noted once the examiner identified the joint range limit, palpated a pelvic compensation [7,14] or if the participant communicated the onset of lumbar pain or incidentally reported a strong, tolerable, pain-free stretch [32]. Each joint range was measured three times and an average calculated using an Excel 2013 macro (Microsoft, Redmond, Washington) [14,33].

The researcher was blinded to each hip range's measurement's results [7,33]. The only exception was the MTT outcome, where because gravity and muscle flexibility determined the end point, the researcher had no effect on the result. The participant, with sight of the IMU's measurements on a linked Ipad (Apple Incorporated, Cupertino, California), memorized and disclosed the results for capture at the end of each specific range's testing. Participants were encouraged to communicate any memory lapses. In the event this occurred, another trial was performed. On the odd occasion, when a fault was suspected, a fourth trial was performed. This was carried out to ensure accurate performance of the passive movement and to eliminate any error. The researcher was once again blinded to the fourth trial's result. The three most aligned trial's measurements were then recorded [34]. The testing took place on wooden plinths and stools to negate metal furnishing's effects on the IMU's magnetometer.

Study size

An a priori sample size was calculated for the comparison of dominant and non-dominant hip ROM (matched pairs) with an expected

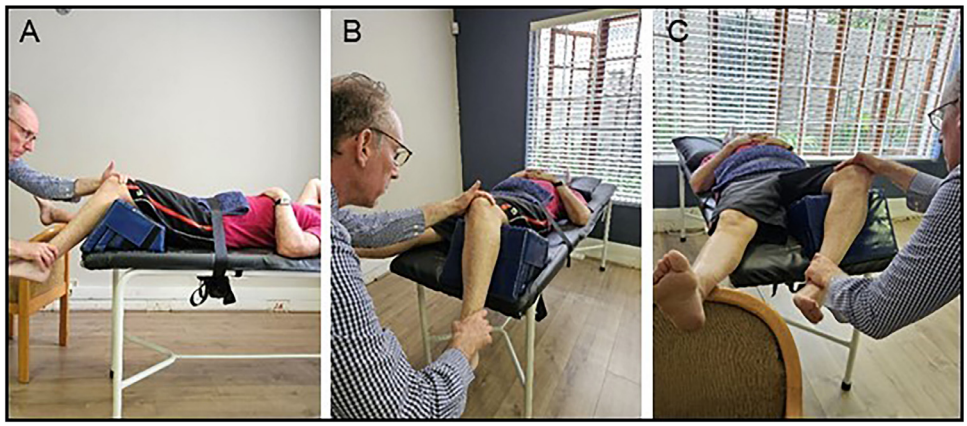


Fig. 1. (A) Participant positioned supine with hip in 30° flexion. (B) IR testing. (C) ER testing over the 30° wedge with contralateral leg in abduction and supported over the back of a wooden chair. The testing conditions above were replicated for publishing purposes, some of the original equipment was not available at the time.

effect size (ES) of 0.3, and alpha error probability of 0.05 and a power of 0.80. A sample of 90 participants was required.

Statistical methods

Statistical analyses were conducted using IBM SPSS Statistics Version 28 (IBM, Armonk, New York). No data were missing. Significance was set at p <.05 for all comparisons. Analysis was performed on all the hip ranges cited above and the MTT adjusted, SLR measurements [35]. The captured left and right side hip variable data was later organized in accordance with each participant's side dominance. As per the American Statistical Association's guidelines [36], both p-values and ES are noted for statistically important results. In this vein, terminology conveying degrees of evidence has replaced binary "significant" and "insignificant" wording as per Muff et al.'s [37] recommendations. Consequently, p-values above 0.1 display no evidence; 0.05 to 0.1 are weakly evident; 0.01 to 0.05 show moderate evidence; values of 0.001 to 0.01 present strong evidence and 0.0001 to 0.001 depict very strong evidence.

A Chi-squared test of independence was performed to determine whether there was an association between gender and lower back pain. The group's age, BMI, Beighton scores, Baecke activity indices and hip range variables were compared with the two-tailed independent t-test or the Mann Whitney U test depending on normality. ES were calculated using Cohen's d (parametric) or the formula: $r = Z/\sqrt{N}$ [38] (non-parametric) depending on normality.

For the comparison of the LPG and CG's side-to-side hip range differences, a paired samples t-test or Wilcoxon Signed Rank test was utilized to analyses the variables. Again, ES were calculated using Cohen's d (parametric) or the formula: $r = Z/\sqrt{N}$ [39] (non-parametric) depending on normality. ES were classified as small (d = 0.2), medium (d = 0.5) or large (d = 0.8).

Results

Participants

Three hundred and twelve people applied for the study. The study's total convenience sample consisted of 93 participants, 50 men and 43 women, ranging from 41 to 81 years of age (mean age 54.84 ± 10.45 years) with a mean BMI of 25.0 ± 2.9 kg/m^2. The LPG consisted of 61 individuals, 32 males and 29 females, and the CG comprised 32 volunteers, 18 males and 14 females. Six of the LPG participants were left-footed and two presented in the Control. The overall average Beighton Scale score was 2.31 ± 1.87 and the mean Baecke questionnaire results for work, sport, leisure and total indices were 2.39 (± 0.58), 3.07 (± 0.95), 2.79 (± 0.57) and 8.25 (± 1.53), respectively.

Main results

A Chi-squared test of independence showed no association between gender and lumbar pain status (p=.73). Reliability testing of the 30 variables' left and right sides found an average Cronbach's Alpha score of .900; with range of .032 to .982. The Cronbach's alpha and 95% confidence intervals are quoted in Table 1. The group's age, BMI and Beighton scores were similar (Table 2). Although their work activity was the same, a difference was found between their Baecke sport, leisure and total indices. A small effect was evident for the Baecke sport (ES = 0.230) and total indices (ES = 0.282), while there was a medium effect between groups in the Baecke leisure index (ES = 0.567); all of which indicated that the CG was more active than the LPG.

No difference was found between the LPG and the CG's hip ROM variables (Table 3). The quoted SLR results are MTT adjusted [35]. In spite

Table 1
Test-retest reliability study results

Variable	Cronbach's alpha	95% Confidence interval
MTT (Knee Extended) Left Hip	0.954	0.830–0.990
MTT (Knee Extended) Right Hip	0.922	0.711–0.982
SLR Left Hip	0.969	0.880–0.992
SLR Right Hip	0.958	0.846–0.990
Hip Flexion Left	0.840	0.394–0.962
Hip Flexion Right	0.908	0.544–0.972
Hip Extension Left	0.911	0.242–0.968
Hip Extension Right	0.935	0.723–0.982
Hip Abduction Left	0.929	0.736–0.984
Hip Abduction Right	0.032	−4.656 −0.778
Hip Adduction Left	0.927	0.732–0.983
Hip Adduction Right	0.932	0.751–0.984
Hip IR Supine 30° Left	0.860	0.442–0.962
Hip ER Supine 30° Left	0.950	0.814–0.989
Hip Total Rotation Supine 30° Left	0.973	0.860–0.992
Hip IR Supine 30° Right	0.942	0.786–0.987
Hip ER Supine 30° Right	0.853	0.436–0.967
Hip Total Rotation Supine 30° Right	0.934	0.756–0.985
Hip IR Prone Left	0.804	0.234–0.955
Hip ER Prone Left	0.957	0.844–0.990
Hip Total Rotation Prone Left	0.887	0.575–0.974
Hip IR Prone Right	0.977	0.913–0.994
Hip ER Prone Right	0.778	0.119–0.937
Hip Total Rotation Prone Right	0.937	0.766–0.985
Hip IR Sitting Left	0.968	0.814–0.990
Hip ER Sitting Left	0.954	0.829–0.989
Hip Total Rotation Sitting Left	0.982	0.932–0.996
Hip IR Sitting Right	0.972	0.841–0.991
Hip ER Sitting Right	0.945	0.789–0.986
Hip Total Rotation Sitting Right	0.979	0.923–0.995

Table 2
Comparison between the lumbar pain group and the control group's characteristics

Variable	Lumbar Pain Group (n = 61)	Control Group (n = 32)	Cohen's d Effect Size	Levene's Equality of Variances test (p-value)	Two-tailed p-value. Asymp. Sig. (2-tailed)	95% CI
Age(y)** Median(IQR)	51(46.00–60.5)**	57.5(46.25–67.75)**	0.084		0.416	
BMI(kg/m^2)** Median(IQR)	25.4(23.1–27.3)**	25(23.48–26.8)*	0.032		0.759	
Beighton Scale** Median(IQR)	2(0–4)**	2(1–3)**	0.018		0.865	
Baecke Work Index** Median(IQR)	2.13(2–2.63)**	2.38(2.13–2.85)*	0.134		0.197	
Baecke Sport Index** Median(IQR)	2.75(2–3.75)**	3.5(2.81–4)*	0.230		0.027	
Baecke Leisure Index* Mean ± SD; SEM	2.68 ± 0.56; 0.07*	2.99 ± 0.53; 0.09*	0.567	0.523	0.011	−0.55 - −0.07
Baecke Total Index** Median(IQR)	8(6.75–9.13)*	9(7.78–10.22)**	0.282		0.007	

*denotes a variable with a normal distribution.
**denotes a variable with a skewed distribution.
CI, Confidence Interval; IQR, Interquartile Range; SD, Standard Deviation; SEM, Standard Error of the Mean.

of the similarity seen between the group's hip ranges, a number of the group's hip ROM variables showed side-to-side differences. Although evident in both groups, a greater number occurred in the LPG. Namely, of the 15 tested hip range variables, 11 showed non-dominant to dominant side asymmetry ES in the pain group above 0.2, ranging from 0.226 to 0.509 (Table 4). Five of the 15 variables presented with non-dominant to dominant side differences in the CG – here the ES ranged from 0.249 to 0.392 (Table 5). The LPG's asymmetrical ranges spanned all three planes of movement, while the CG's presented in the transverse plane only.

Discussion

These results indicate that hip ROM per se has no association nor bearing on lumbar pain. This is in line with previously published studies where similarity was noted between the LPG and CG's hip flexion [11,15]; extension [11,15,16]; MTT [10,17]; SLR [9,17,18]; abduction [11,15,16,19,20]; adduction [11,16]; and rotation [11,12,19]. In contrast to our study, Tsai et al. [11] tested active hip ROM and Tanaka et al. [15] compared the ROM of osteoarthritic and non-pathological hips in group's with and without lower back pain.

Adegoke and Fapojuwo [16] attributed their group's similar hip ranges to hip treatment that the LPG received prior to testing, however individuals receiving this treatment within the six months prior to data collection were excluded from our study. Nevertheless, lower back pain has been ascribed to other factors. Namely, erector spinae weakness [11,20]; altered movement patterns [18]; lumbar flexor and extensor strength imbalances [40] and eccentric lumbar extensor weakness [41]. These characteristics may play a greater role in lumbar symptom generation and provocation.

Remarkable in light of the similarities between our group's hip ROM, a greater number of hip ROM non-dominant to dominant side asymmetries presented in the LPG. Namely, of the 15 hip side variables tested, 11 were different in the LPG, with five in the CG. Notably, when compared to the the non-dominant side, the dominant side's range was more often smaller. This occurred in nine of the LPG's hip ROM variables which showed an effect above 0.2. Two other LPG's variables showed this characteristic, but they had an ES of 0.101 and 0.099. Twelve of the variables in the CG had a relatively smaller range on the dominant side, but five's ES were greater than 0.2. The statistically evident side-to-side asymmetries spanned all three planes of movement in the LPG but presented only in the transverse plane in the CG. This multi-planar presentation may be paramount, explaining why in spite of transverse plane side asymmetries, the CG's participants were asymptomatic. Prevalent primarily on the dominant side, these seemly small asymmetries may compound with combined, closed-chain movements, limiting pelvic excursion over this hip, resulting in shearing and torsioning across the lumbar spine. Presumably, the combination of multiple axial asymmetrical hip range restrictions may cause or be the result of a chronic lumbar condition.

Historically, hip range asymmetries were considered to be the consequence of repertition and dominance (resulting in adaptive changes) [42], early-onset arthritic change or femoroacetabular impingement [43]. In this study, repetitive activity's effects were an unlikely explanation for the LPG's wider ranging side asymmetry because of the group's diversity. It is possible that osteopathic change could explain these side asymmetries, but without radiological investigations this is difficult to confirm. Although the authors could not verify whether these degenerative disorders were more prevalent in the LPG, the similarity of the group's age, BMI and Baecke Work Index might mitigate these concerns to some degree. Furthermore, although participants with a history of hip injury were included in both groups, the exclusion of congenital hip disorders, leg length discrepancies, chronic symptomatic knee problems and hip and knee fractures and surgeries, would have also potentially limited the prevalence of osteopathy, weakening this explanation for the side discrepancies seen. Moreover, one might expect the CG's significantly higher sport and leisure activity to effect greater hip joint wear and tear, resulting in wider ranging side differences in this group, but this was not the case.

Earlier studies considered dominance's effects when sport was the focus [10–14] but omitted it when sport was not [9,15–21]. Dominance, however, presents a potential cause of hip side asymmetries in the general population. This may be due to the sequential effects of leg dominance on habitual posturing, hip side asymmetries and lower back pain [22]. Peterson Kendall et al. [35] also noted the influence of handedness on posture, muscle length and strength. Their observations mirror Hruska's [22], who described a similar dominance related pattern, but rather noted its effects on hip joint ROM and spinal curvatures and its consequential role in lower back pain provocation. This study's findings may support this conviction. Namely, compounded, multi-planar hip ROM restrictions on the dominant side could alter hip movement in single stance and weight shifted movements, placing uniquely greater stresses on the lumbar spine in those presenting with these features.

The differing side asymmetries in the groups' MTT and hip extension results may also be of interest. The CG's hip extension and MTT measures show no side difference but this is not the case in the LPG, where the MTT dominant and non-dominant sides are similar but the hip extension results show an asymmetry. As both ranges test hip extension, parity would be expected. Compensatory lumbar rotation effected by the psoas majors' anterior insertion onto the transverse processes, may provide the additional hip extension range in the prone position, which is controlled

Table 3
Comparison between the lumbar pain group and the control group passive hip ROM

Variable	Lumbar Pain Group (n = 61)	Control Group (n = 32)	Cohen's d Effect Size	Levene's Equality of Variances test (p-value)	Two-tailed p-value. Asymp. Sig. (2-tailed)	95% CI
MTT (Knee Extended) Non-Dominant Hip(°)* Mean ± SD; SEM	−9.12 ± 6.83; 0.87	−8.43 ± 4.81; 0.85	0.111	0.028	0.613	−3.38 – 2.01
MTT (Knee Extended) Dominant Hip(°)* Mean ± SD; SEM	−9.87 ± 6.14; 0.79	−7.67 ± 5.61; 0.99	0.370	0.604	0.094	−4.79 – 0.38
SLR Non-Dominant Hip(°)** Median(IQR)	73.00(60 – 90.67)**	73.5(60.83 – 81.67)*	0.058		0.574	
SLR Dominant Hip(°)* Mean ± SD; SEM	72.98 ± 17.17; 2.20	70.75 ± 14.83; 2.62	0.136	0.136	0.536	−4.89 – 9.34
Hip Flexion Non-Dominant(°)* Mean ± SD; SEM	130.37 ± 8.72; 1.12	130.06 ± 10.74; 1.90	0.032	0.200	0.883	−3.80 – 4.40
Hip Flexion Dominant(°)* Mean ± SD; SEM	129.08 ± 9.93; 1.27	129.16 ± 8.60; 1.52	0.008	0.575	0.972	−4.19 – 4.04
Hip Extension Non-Dominant(°)* Mean ± SD; SEM	32.47 ± 7.87; 1.01	33.27 ± 4.73; 0.84	0.115	0.003	0.599	−3.82 – 2.22
Hip Extension Dominant(°)* Mean ± SD; SEM	31.11 ± 6.19; 0.79	32.07 ± 4.88; 0.86	0.166	0.241	0.449	−3.46 – 1.55
Hip Abduction Non-Dominant(°)** Median(IQR)	44.33(39.33 – 47.5)**	43.5(38.67 – 51.75)*	0.008		0.936	
Hip Abduction Dominant(°)* Mean ± SD; SEM	44.97 ± 6.08; 0.78	45.49 ± 5.89; 1.04	0.086	0.853	0.695	−3.13 – 2.09
Hip Adduction Non-Dominant(°)* Mean ± SD; SEM	29.05 ± 5.42; 0.69	30.29 ± 5.17; 0.91	0.233	0.927	0.289	−3.56 – 1.07
Hip Adduction Dominant(°)* Mean ± SD; SEM	27.44 ± 5.01; 0.64	28.65 ± 4.12; 0.73	0.255	0.140	0.247	−3.25 – 0.85
Hip IR Supine 30° Non-Dominant(°)* Mean ± SD; SEM	40.99 ± 10.38; 1.33	40.18 ± 11.08; 1.96	0.077	0.627	0.725	−3.79 – 5.42
Hip ER Supine 30° Non-Dominant(°)* Mean ± SD; SEM	61.74 ± 7.09; 0.91	61.97 ± 9.12; 1.61	0.029	0.304	0.895	−3.62 – 3.17
Hip Total Rotation Supine 30° Non-Dominant(°)* Mean ± SD; SEM	102.74 ± 12.25; 1.57	102.15 ± 16.10; 2.85	0.043	0.050	0.844	−5.34 – 6.52
Hip IR Supine 30° Dominant(°)* Mean ± SD; SEM	44.01 ± 9.60; 1.23	41.01 ± 9.49; 1.68	0.314	0.781	0.154	−1.14 – 7.15
Hip ER Supine 30° Dominant(°)* Mean ± SD; SEM	57.82 ± 9.53; 1.22	58.91 ± 9.29; 1.64	0.115	0.551	0.600	−5.18 – 3.01
Hip Total Rotation Supine 30° Dominant(°)* Mean ± SD; SEM	101.83 ± 13.44; 1.72	99.92 ± 16.17; 2.86	0.133	0.422	0.545	−4.34 – 8.17
Hip IR Prone Non-Dominant(°)* Mean ± SD; SEM	42.71 ± 11.23; 1.44	40.74 ± 9.48; 1.68	0.185	0.391	0.400	−2.65 – 6.60
Hip ER Prone Non-Dominant(°)* Mean ± SD; SEM	56.89 ± 9.74; 1.25	57.19 ± 8.41; 1.49	0.032	0.753	0.884	−4.33 – 3.74
Hip Total Rotation Prone Non-Dominant(°)* Mean ± SD; SEM	99.60 ± 12.39; 1.59	97.93 ± 11.96; 2.12	0.137	0.725	0.532	−3.63 – 6.98
Hip IR Prone Dominant(°)* Mean ± SD; SEM	41.94 ± 10.29; 1.32	40.54 ± 9.27; 1.64	0.140	0.429	0.522	−2.92 – 5.72
Hip ER Prone Dominant(°)* Mean ± SD; SEM	55.08 ± 10.02; 1.28	54.00 ± 7.81; 1.38	0.116	0.144	0.597	−2.96 – 5.12
Hip Total Rotation Prone Dominant(°)* Mean ± SD; SEM	97.02 ± 12.64; 1.62	94.54 ± 10.67; 1.89	0.207	0.307	0.347	−2.73 – 7.69
Hip IR Sitting Non-Dominant(°)* Mean ± SD; SEM	42.98 ± 9.58; 1.23	41.89 ± 9.71; 1.72	0.114	0.743	0.602	−3.07 – 5.27
Hip ER Sitting Non-Dominant(°)* Mean ± SD; SEM	51.44 ± 6.87; 0.88	50.66 ± 6.04; 1.07	0.119	0.502	0.586	−2.07 – 3.65
Hip Total Rotation Sitting Non-Dominant(°)* Mean ± SD; SEM	94.43 ± 11.57; 1.48	92.54 ± 11.61; 2.05	0.163	0.921	0.458	−3.14 – 6.91
Hip IR Sitting Dominant(°)* Mean ± SD; SEM	39.43 ± 7.23; 0.93	40.09 ± 7.67; 1.36	0.090	0.447	0.680	−3.87 – 2.53
Hip ER Sitting Dominant(°)* Mean ± SD; SEM	52.12 ± 7.62; 0.98	51.95 ± 7.31; 1.29	0.023	0.968	0.916	−3.09 – 3.43
Hip Total Rotation Sitting Dominant(°)* Mean ± SD; SEM	91.55 ± 11.35; 1.45	92.04 ± 12.31; 2.18	0.042	0.550	0.847	−5.56 – 4.57

*denotes normal distribution, presented as mean, SD, SEM and analyzed with the two-tailed independent t-test.
**denotes skewed distribution, presented as median, IQR and analyzed with the Mann-Whitney U Test.
CI, Confidence Interval; ER, External Rotation; IQR, Interquartile Range; IR, Internal Rotation; MTT, Modified Thomas Test; SD, Standard Deviation; SEM, Standard Error of the Mean; SLR, Straight Leg Raise. The MTT is reported with negative mean and median scores, indicating the limb's end position below the horizontal. Each side's internal and ER measures are combined to report that side's total rotation - this is repeated for each sagittal plane position, namely, supine 30°, prone and sitting.

Table 4
Comparison of the lumbar pain group's non-dominant and dominant side's passive hip ROM

Variables	Lumbar Pain Group Non-Dominant Hip ROM	Lumbar Pain Group Dominant Hip ROM	Cohen's d Effect Size	Two-tailed p-value. Asymp. Sig. (2-tailed)	95% CI
MTT (Knee Extended)(°)* Mean ± SD; SEM	−9.12 ± 6.83; 0.87	−9.87 ± 6.14; 0.79	0.226	0.082	−0.10 – 1.62
SLR(°)** Median(IQR)	73.00(60 – 90.67)**	69.67(58.50 – 84.00)	0.454	<0.001	
Hip Flexion(°)* Mean ± SD; SEM	130.37 ± 8.72; 1.12	129.08 ± 9.93; 1.27	0.252	0.054	−0.02 – 2.59
Hip Extension(°)* Mean ± SD; SEM	32.47 ± 7.87; 1.01	31.11 ± 6.19; 0.79	0.277	0.034	0.10 – 2.61
Hip Abduction(°)** Median(IQR)	44.33(39.33 – 47.5)**	45.33(41.00 – 48.50)	0.090	0.484	
Hip Adduction(°)* Mean ± SD; SEM	29.05 ± 5.42; 0.69	27.44 ± 5.01; 0.64	0.325	0.014	0.34 – 2.87
Hip IR Supine 30˚(°)* Mean ± SD; SEM	40.99 ± 10.38; 1.33	44.01 ± 9.60; 1.23	0.396	0.003	−4.97 - −1.06
Hip ER Supine 30˚ (°)* Mean ± SD; SEM	61.74 ± 7.09; 0.91	57.82 ± 9.53; 1.22	0.488	<0.001	1.86 – 5.98
Hip Total Rotation Supine 30˚(°)* Mean ± SD; SEM	102.74 ± 12.25; 1.57	101.83 ± 13.44; 1.72	0.101	0.435	−1.40 – 3.21
Hip IR Prone(°)* Mean ± SD; SEM	42.71 ± 11.23; 1.44	41.94 ± 10.29; 1.32	0.099	0.443	−1.23 – 2.77
Hip ER Prone(°)* Mean ± SD; SEM	56.89 ± 9.74; 1.25	55.08 ± 10.02; 1.28	0.227	0.081	−0.23 – 3.85
Hip Total Rotation Prone(°)* Mean ± SD; SEM	99.60 ± 12.39; 1.59	97.02 ± 12.64; 1.62	0.334	0.011	0.60 – 4.55
Hip IR Sitting(°)* Mean ± SD; SEM	42.98 ± 9.58; 1.23	39.43 ± 7.23; 0.93	0.509	<0.001	1.77 – 5.35
Hip ER Sitting(°)* Mean ± SD; SEM	51.44 ± 6.87; 0.88	52.12 ± 7.62; 0.98	0.112	0.383	−2.22 – 0.86
Hip Total Rotation Sitting(°)* Mean ± SD; SEM	94.43 ± 11.57; 1.48	91.55 ± 11.35; 1.45	0.458	<0.001	1.27 – 4.49

*denotes normal distribution, presented as mean, SD and analyzed with the Paired samples t-test;
**denotes skewed distribution, presented as median, IQR and analyzed with the Wilcoxon Signed Rank Test.
CI, Confidence Interval; ER, External Rotation; IR, Internal Rotation; MTT, Modified Thomas Test; SD, Standard Deviation; SLR, Straight Leg Raise. The MTT is reported with negative mean and median scores, indicating the limb's end position below the horizontal. Each side's internal and ER measures are combined to report that side's total rotation - this is repeated for each sagittal plane position, namely, supine 30˚, prone and sitting.

by the MTT's flexed spinal position. This potential lumbar hypermobility may offer one explanation for the LPG's symptoms.

In double stance, these observed hip range side asymmetries would only be pertinent in certain lumbar ranges of movement - when both sides are acting. This is possible at end range lumbar flexion and extension. The sides' differing end position could cause a torsion of the pelvis and spine. This mechanism may also play a role during bilateral hip flexion and its functional counterparts, squatting and initiation of sit-to-stand [44]. This effect is dependent on the magnitude of the asymmetry and the degree of deviation from the sagittal plane, as it is countered by hip abduction and/or external rotation which releases stretch on the posterior capsule. This was demonstrated clinically by Sahrmann [8], who noted a change in pelvic angulation in persons with unilaterally decreased hip flexion on rocking backward from a four-point kneeling starting position. The pelvis levelled when the restricted hip was placed in greater external rotation.

In two-legged stance, hip rotation side asymmetries would have no collective bearing on lumbar rotation. As 27% of a non-symptomatic cohort displayed equal bilateral internal and external rotation range [7], most people's lumbar rotation is checked by either the left or right hip, depending on its version. This was true of this sample, where both groups exhibited similar version, displaying no difference between their dominant and non-dominant IR and ER ROM in the prone position.

Lumbar lateral flexion range could however be affected by asymmetrically tight hip abductors in bilateral stance, but seemly presents in unusual positions. Sahrmann [8] reported another clinical example that demonstrated asymmetrically tight hip abductors producing a lateral pelvic tilt and lumbar lateral flexion when standing with feet together. The LPG's different adduction side asymmetry could potentially have this effect, but people seldom stand and move in this position. This study's group's symmetrical side-to-side abduction range may not have influenced the LPG's symptoms but in the rare circumstance when certain individuals are in the side splits position, hip abduction asymmetry could theoretically have a bearing on the spine's frontal plane alignment.

In summation, it is conceivable that in spite of NSLBP sufferers having the same hip ranges as their healthy counterparts, their higher incidence of asymmetrical hip ROM restrictions, compounded through combined movements, could lead to lumbar injury and pain.

Limitations

The conclusions reached are limited to persons with NSLBP. Cohorts with for example, spinal stenosis, might present with different hip range abnormalities.

Excluding pain participants with a higher psychosocial risk [45], would have enhanced the composition of the LPG. A more distinctly biomechanically fomented group may have revealed a different result.

Participants who qualified for the LPG were not questioned on whether any lumbar treatment or exercises they had received, could have potentially affected their hip ROM.

Although the participant's knee was passively extended, for greater accuracy, the MTT should have also been performed in hip abduction [8,21]. This would have negated the effects of tensor fascia lata tightness on the final iliopsoas and SLR length measurements. In spite of this omission, only five members of the LPG and three of the CG's non-dominant,

Table 5
Comparison of the control group's non-dominant and dominant side's passive hip ROM

Variables	Control Group Non-Dominant Hip ROM	Control Group Dominant Hip ROM	Cohen's d Effect Size	Two-tailed p-value Asymp. Sig. (2-tailed)	95% CI
MTT (Knee Extended)(°)* Mean ± SD; SEM	−8.43 ± 4.81; 0.85	−7.67 ± 5.61; 0.99	0.143	0.278	−2.16 – 0.64
SLR(°)* Mean ± SD; SEM	72.99 ± 16.08; 2.84	70.75 ± 14.83; 2.62	0.143	0.072	−0.22 – 4.70
Hip Flexion(°)* Mean ± SD; SEM	130.06 ± 10.74; 1.90	129.16 ± 8.61; 1.52	0.086	0.349	−1.04 – 2.85
Hip Extension(°)* Mean ± SD; SEM	33.27 ± 4.73; 0.84	32.07 ± 4.88; 0.86	0.249	0.062	−0.06 – 2.46
Hip Abduction(°)* Mean ± SD; SEM	45.04 ± 7.25; 1.28	45.49 ± 5.89; 1.04	0.066	0.588	−2.12 – 1.22
Hip Adduction(°)* Mean ± SD; SEM	30.29 ± 5.17; 0.91	28.65 ± 4.12; 0.73	0.348	0.063	−0.10 – 3.39
Hip IR Supine 30° (°)* Mean ± SD; SEM	40.18 ± 11.08; 1.96	41.01 ± 9.49; 1.68	0.080	0.556	−3.69 – 2.02
Hip ER Supine 30° (°)* Mean ± SD; SEM	61.97 ± 9.12; 1.61	58.91 ± 9.29; 1.64	0.333	0.025	0.41 – 5.72
Hip Total Rotation Supine 30° (°)* Mean ± SD; SEM	102.15 ± 16.10; 2.85	99.92 ± 16.17; 2.86	0.138	0.104	−0.49 – 4.95
Hip IR Prone(°)* Mean ± SD; SEM	40.74 ± 9.48; 1.68	40.54 ± 9.27; 1.64	0.021	0.869	−2.23 – 2.63
Hip ER Prone(°)* Mean ± SD; SEM	57.19 ± 8.41; 1.49	54.00 ± 7,81; 1.38	0.392	0.030	0.32 – 6.05
Hip Total Rotation Prone(°)* Mean ± SD; SEM	97.93 ± 11.96; 2.12	94.54 ± 10.67; 1.89	0.295	0.011	0.82 – 5.95
Hip IR Sitting(°)* Mean ± SD; SEM	41.89 ± 9.71; 1.72	40.09 ± 7.67; 1.36	0.199	0.168	−0.80 – 4.38
Hip ER Sitting(°)* Mean ± SD; SEM	50.66 ± 6.04; 1.07	51.95 ± 7.31; 1.29	0.190	0.239	−3.49 – 0.90
Hip Total Rotation Sitting(°)* Mean ± SD; SEM	92.54 ± 11.61; 2.05	92.04 ± 12.31; 2.18	0.042	0.720	−2.32 – 3.33

*denotes normal distribution, presented as mean, SD and analyzed with the Paired samples t-test.
**denotes skewed distribution, presented as median, IQR and analyzed with the Wilcoxon Signed Rank Test. CI, Confidence Interval; ER, External Rotation; IR, Internal Rotation; MTT, Modified Thomas Test; SD, Standard Deviation; SLR, Straight Leg Raise. The MTT is reported with negative mean and median scores, indicating the limb's end position below the horizontal. Each side's internal and ER measures are combined to report that side's total rotation - this is repeated for each sagittal plane position, namely, supine 30°, prone and sitting.

dominant or both MTT results exceeded or was equal to 1°. The LPG's and CG's MTT outcomes that exceeded this 1° threshold, ranged from 1° to 7.33°, and 1.33° to 4° respectively. These MTT ROM would have had a bearing on their complementary SLR outcomes [35].

Lastly, it should be highlighted that although statistically relevent, group differences may be clinicaly indistinuishable. Further research is necessary to validate these findings.

Recommendations

In the effort to establish end range, applying a substantial force during passive hip range assessment, may inadvertently correct a restriction in the ROM. Although practically challenging and vulnerable to biases, future research may consider measuring the onset of hip joint resistance (what Maitland [46] referred to as R1) rather than end range, in symptomatic and asymptomatic individuals. Comparison of these ranges, may shed light on differences between experimental and CGs which have previously been missed.

Conclusions

This study found that the LPG and CG's dominant and non-dominant hip ranges of movement were similar. In spite of these findings, a greater number of non-dominant to dominant hip range side asymmetries presented in the LPG. Consequently, without further evidence, it is unlikely that symmetrically atypical coupled hip ranges should be ignored, but rather a stronger case exists for the identification and treatment of hip range side asymmetries in NSLBP patients. This treatment should focus on the dominant side. Combined and compounding hip ROM side asymmetries in closed-chain movement may alter the lumbar spine's axes of rotation, leading to altered torsional forces and lower back injury. Linking hip ROM faults to aggravating activities would facilitate an appreciation of mechanisms of injury, enhance technique selection and negate or curb future episodes.

Declarations of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary materials

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