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Chronic low back pain influences trunk neuromuscular control during unstable sitting among persons with lower-limb loss

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ABSTRACT

Background: Persons with unilateral lower-limb loss are at increased risk for developing chronic low back pain. Aberrant trunk and pelvis motor behavior secondary to lower-limb loss potentially alters trunk postural control and increases demands on the trunk musculature for stability. However, it is unclear whether trunk postural control is associated with the presence or chronicity of low back pain within this population.
Research question: Is there a potential role of impaired trunk postural control among persons with lower limb loss and chronic low back pain?
Methods: Two groups of males with unilateral lower-limb loss (n = 18 with chronic low back pain; n = 13 without pain) performed an unstable sitting task. Trunk postural control was characterized using traditional and non-linear measures derived from center-of-pressure time series, as well as trunk kinematics and the ratio of lumbar to thoracic erector spinae muscle activations.
Results: Traditional and non-linear center-of-pressure measures and trunk muscle activation ratios were similar between groups, while participants with chronic low back pain demonstrated greater trunk motion and reduced local dynamic stability.
Significance: Our results suggest that persons with both lower-limb loss and chronic low back pain exhibit impaired trunk postural control compared to those with limb loss but without pain. Aberrant trunk motor behavior may be a response to altered functional requirements of walking with a prosthesis. An inability to adequately control the trunk could lead to spinal instability and pain in the presence of repetitive exposure to aberrant motor behavior of these proximal structures during everyday activities.

1. Introduction

Low back pain (LBP) is an exceptionally common secondary condition associated with lower-limb amputation (LLA), with point prevalence rates nearly twice that in the general population (52–89% vs. 6–33%, respectively) [1,2]. Though LBP is often considered multifactorial [3], there is a growing body of evidence supporting biomechanical origins, specifically for altered motor behaviors serving in a causal role for the onset and/or persistence of LBP within this population. As examples, characteristic gait findings among persons with LLA include larger trunk motions [4], rigid trunk-pelvis coordination patterns [5], and increased trunk muscle activation [6]. Altered movements and trunk muscle recruitment are, in turn, posited to influence spinal loading, thereby increasing the risk of LBP [7]. Further, trunk mechanics during gait were found to be moderately correlated with trunk postural control in unstable sitting [8]; while observed in a cohort of persons without LLA, this correlation does suggest an important role of trunk postural control in gait. Notwithstanding the characteristic adaptations in trunk and pelvis motor behavior among persons with LLA, it is currently unknown whether alterations in trunk postural control contribute to the high prevalence of LBP.

Trunk postural control can be assessed using an unstable sitting paradigm, in which the need to balance on an unstable chair (with the lower limbs supported) isolates trunk-pelvic control by minimizing the

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influence of the lower extremities [9,10]. Deficits in trunk postural control with LBP have been identified in non-LLA cohorts of persons with vs. without LBP [10,11] as well as individuals with vs. without LLA [12]. Specifically, decreased performance, indicated as increased center-of-pressure (COP) displacements, is observed among individuals with LBP [10,11,13]. van Dieën [13] noted that individuals with a current episode of LBP exhibit lower COP frequencies, consistent with previous work that suggested individuals with LBP adopt a rigid, or "guarding", postural control strategy [14]. Individuals with LBP also demonstrate preferential recruitment of global movers (i.e., longissimus) vs. spinal stabilizers (i.e., iliocostalis) and larger trunk movements during an unstable sitting task. Preferential recruitment of global trunk muscles suggests individuals with LBP adopt a motor behavior/control strategy that may increase lumbar instability in the presence of large thoracolumbar movement [15]. Similar motor behaviors are observed among persons with LLA, among whom increased erector spinae activation with increased COP displacement is indicative of poor trunk postural control and may increase the intervertebral neutral zone [12]. Increases in this zone may lead to decreased spinal stability and ultimately pain [16].

Existing evidence suggests that persons with LLA and LBP have altered trunk and pelvic motor behaviors during activities such as gait [17–19]. These alterations are often suggested to be the result of impaired trunk postural control, although this has yet to be comprehensively investigated. Further, it is unclear if trunk postural control is associated with the presence or persistence of LBP within the LLA population. The purpose of this study was therefore to investigate the potential role of impaired trunk postural control among persons with LLA and LBP using an unstable sitting paradigm. We hypothesized that, among persons with LLA, those with LBP would demonstrate impaired trunk postural control, evidenced by larger COP displacements, smaller ratios of lumbar/thoracic muscle activation, and smaller relative motion between the trunk and pelvis.

2. Methods

2.1. Participants

Thirty-two persons with traumatic, unilateral LLA participated in this study – 19 with LBP and 13 without LBP (Table 1). LBP status was determined via the NIH recommended minimal data set, where chronicity is defined as pain that persisted for at least three months and with pain on at least half the days in the past six months [20]. All participants were at least one year removed from initial injury, reported no musculoskeletal injures to the contralateral (i.e., unaffected) limb, and were ambulatory without the use of an assistive device (e.g., cane, walker, etc.). Individuals with phantom limb pain, and/or discomfort, regardless of cause (> 4/10 on a visual analog scale for pain) with 100% weight bearing in socket or pain that interfered with performance of functional activities were also excluded. Prior to data collection, participants gave informed consent to protocols approved by the local Institutional Review Board.

Table 1
Mean (SD) participant characteristics in the lower limb amputation groups with and without low back pain (LBP). Reported p-values represent group comparisons (pooled transtibial and transfemoral with and without LBP) from unpaired t tests. TT: transtibial, TF: transfemoral, VAS: visual analog scale (0–10). Note, VAS Pain values represent intensity at time of testing.

	LBP	No Pain	*p*-value
Age (years)	35.8 (7.3)	33.1 (7.5)	0.32
Height (cm)	179.6 (7.2)	178.6 (4.8)	0.64
Mass (kg)	91.9 (15.8)	89.5 (14.8)	0.66
Time Since Injury (years)	8.7 (4.1)	6.1 (4.2)	0.11
Level of Amputation (TT/TF)	11/7	11/2	NA
Oswestry Disability Index (% disability)	29.9 (24.5)	7.4 (9.1)	0.002
Pain Intensity (VAS)	1.8 (1.5)	0.2 (0.6)	0.001

2.2. Experimental procedures

Participants sat on an unstable chair [12] with their eyes open, and were instructed to keep the chair level and their arms crossed. The distance between each spring and the central ball-pivot was standardized across all participants and balance trials. Three 60-second practice trials were completed initially to attenuate learning effects [21]. A fourth trial was then completed and used for data analyses. Three-dimensional trunk and pelvis kinematics were tracked (120 Hz) with 12 retro-reflective markers using an 18-camera motion capture system (Qualisys, Göteborg, Sweden). Markers were placed on the C7 and T10 spinous processes; the sternal notch and xiphoid; and bilaterally across the acromion, anterior/posterior superior iliac spines, and iliac crests. Kinetic data were also collected (1200 Hz) using a force platform (AMTI, OR6-7-2000, Watertown, MA, USA) mounted beneath the unstable chair. Electromyographic (EMG) data were collected (1200 Hz, Motion Lab Systems, Baton Rouge, LA, USA) bilaterally from the thoracic erector spinae (TES) and lumbar erector spinae (LES) muscle groups. Bipolar electrodes (Ag/AgCl) were placed 4 cm lateral to T9 spinous process (TES), and 6 cm lateral to L2 spinous process (LES) [15]. Prior to electrode placement, the skin was shaved (if necessary), abraded, and cleaned with alcohol.

2.3. Data processing and dependent measures

All data were analyzed in Visual3D (C-motion, Germantown, MD, USA) and MATLAB (MathWorks, Natick, MA, USA). Trunk kinematic and COP data were low-pass filtered (4^{th} order bi-directional Butterworth, cut-off frequencies 6 and 10 Hz, respectively). Prior to analyses, the first and last 5 s of each trial were removed. Several "traditional" measures were derived from the COP time series: 95% confidence ellipse area (CEA), and mean velocity ($MVEL_{AP}$, $MVEL_{ML}$) in the anteroposterior (A–P) and mediolateral (M-L) directions [22]. Among these measures, higher values are interpreted as indicating deteriorated or inferior postural control [22,23]. To supplement these traditional measures, COP time series were also used to calculate nonlinear stabilogram diffusion analyses, given reported differences in measures obtained from this method between persons with vs. without LLA and persons with vs. without LBP [12,15]. Stabilogram diffusion analysis characterizes the time dependent behavior of a control system in both the short term (open-loop control) and long term (closed-loop control) [23,24]. The critical point (CP) represents where the system transitions from an open-loop to closed-loop control strategy. CP coordinates [amplitude (cm^2)] were calculated separately in the A–P and M–L directions [12].

EMG data were pre-processed as in prior reports [15]. Briefly, eight bandpass filters (50–400 Hz) were applied to the EMG time series followed by a 30 Hz high-pass filter; absolute Hilbert amplitudes were then calculated as the means of the time series. From these, ratios between LES and TES (lumbar/thoracic), and prosthetic and intact-side LES (P/I_{LES}) and TES (P/I_{TES}) activations were determined.

Three-dimensional trunk angles (relative to the pelvis) were calculated in Visual3D. Markers over C7, T10, and the sternum were used to model the trunk, while those over the bilateral anterior and posterior iliac spines were used to model the pelvis. From these, ranges of motion (ROM) were calculated as the differences between the maximum and minimum angles within each seated trial. Maximum short-term Lyapunov exponents (λ_s; [25]) were used to characterize the local dynamic stability of trunk motion, with larger positive values representing a decreased ability to resist local perturbations (i.e., decreased local dynamic stability) [8]. This measure was chosen as it characterizes how the postural control system is functioning over the time series, since it considers how the variability in motor behavior (i.e., trunk motion)

changes between time points [26]. Global false nearest neighbor and mutual average information analyses were respectively used to determine embedding dimensions (m = 6) and time delays (τ = 100 samples).

2.4. Statistical analyses

To address the study hypothesis, separate multivariate analyses of covariance (MANCOVAs) were used to compare COP-based measures, EMG, the trunk kinematics between participants with vs. without LBP. Traditional and non-linear parameters were included in the same MANCOVA. When a significant group effects was observed, subsequent univariate ANCOVAs were used separately for each dependent measure. Mass and stature were included as covariates in these analyses, since significant associations of these with COP-based measures (Pearson's *r* range: 0.6-0.8) were observed. Level of amputation was also included as a blocking variable given the potential for unique trunk mechanical differences observed between persons with transtibial and transfemoral LLA [4]. Of note, there were no differences ($p > 0.05$) between transtibial and transfemoral patients for any traditional or non-linear COP measure or EMG ratio. Data from one participant in the LBP group were excluded from these analyses due to technical errors during data collection. No violations of parametric model assumptions were evident. All statistical analyses were performed using SPSS (version 24.0, SPSS Inc, Chicago, IL) with statistical significance set at $p < 0.05$.

3. Results

There was not a main effect of group on the set of COP-based measures (Wilks' $\Lambda = 0.84$, $F_{(10,18)} = 0.74$, $p = 0.60$, $\eta^2 = 0.16$; Table 2). A significant effect of group was found on trunk kinematics (Wilks' $\Lambda = 0.46$, $F_{(6,19)} = 3.52$, p = 0.02, $\eta^2 = 0.54$; Fig. 2) and trunk muscle activity (Wilks' $\Lambda = 0.69$, $F_{(4,15)} = 3.46$, $p = 0.03$, $\eta^2 = 0.31$; Fig. 1). Subsequent univariate analyses revealed that A–P trunk motion was larger in the LBP group ($F_{(2,1)} = 5.24$, $p = 0.03$, $\eta^2 = 0.19$), but that group-level differences were not significant in the M–L ($p = 0.08$) or axial directions ($p = 0.18$). Group differences were also significant for local dynamic stability of the trunk in both the A–P ($F_{(2,1)} = 5.58$, $p = 0.03$, $\eta^2 = 0.20$) and axial $F_{(2,1)} = 9.77$, $p = 0.005$, $\eta^2 = 0.30$) directions, with the LBP group demonstrating larger λ_s. There were no univariate differences between groups for trunk motion in the M–L ($F_{(2,1)} = 3.32$, $p = 0.08$, $\eta^2 = 0.12$) or axial directions ($F_{(2,1)} = 1.95$, $p = 0.18$, $\eta^2 = 0.08$), or local dynamic stability in the M–L direction ($F_{(2,1)} = 3.06$, $p = 0.09$, $\eta^2 = 0.12$).

4. Discussion

We hypothesized that individuals with LBP would demonstrate impaired trunk postural control compared to those with no pain, in the context of an unstable sitting task. COP-based measures and trunk muscle activation ratios were similar between the current groups, contradicting part of our hypothesis. Partially supporting the study hypothesis, however,

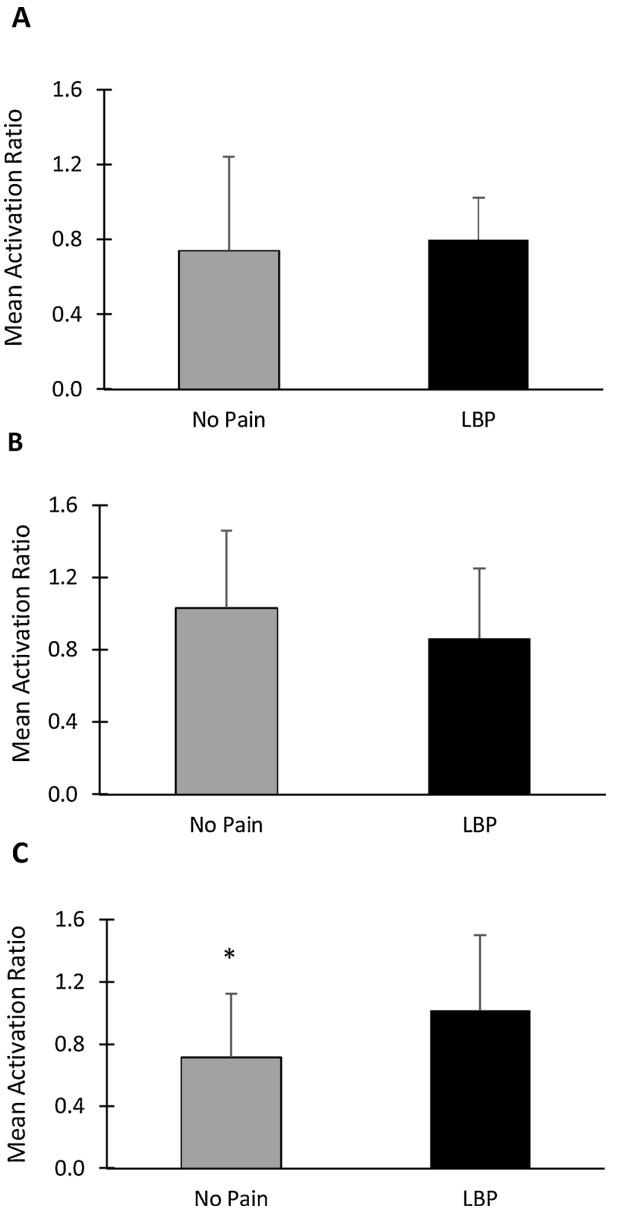

Fig. 1. Mean trunk muscle activation ratios of A) lumbar/thoracic activation, B) prosthetic/intact thoracic erector spinae activation, and C) prosthetic/intact lumbar erector spinae activation during unstable sitting in persons with lower limb amputation without (No Pain) and with low back pain (LBP). Asterisk indicates a significant difference between groups ($p < 0.05$).

Table 2
Mean (SD) center of pressure sway measures of seated balance among persons with lower limb amputation (LLA), with and without low back pain (LBP). Cohen's *d* represents the magnitude of the effect of the difference.

	LBP	No Pain	Effect Size (*d*)
95% ellipse area (cm^2)	8.33 (5.24)	5.20 (3.12)	0.70
Mean Velocity Mediolateral (cm/s)	1.37 (0.50)	1.03 (0.32)	0.81
Mean Velocity Anteroposterior (cm/s)	1.30 (0.46)	0.97 (0.32)	0.83
CP amplitude Mediolateral (cm^2)	0.92 (0.76)	0.45 (0.29)	0.82
CP amplitude Anteroposterior (cm^2)	0.79 (0.55)	0.52 (0.49)	0.52

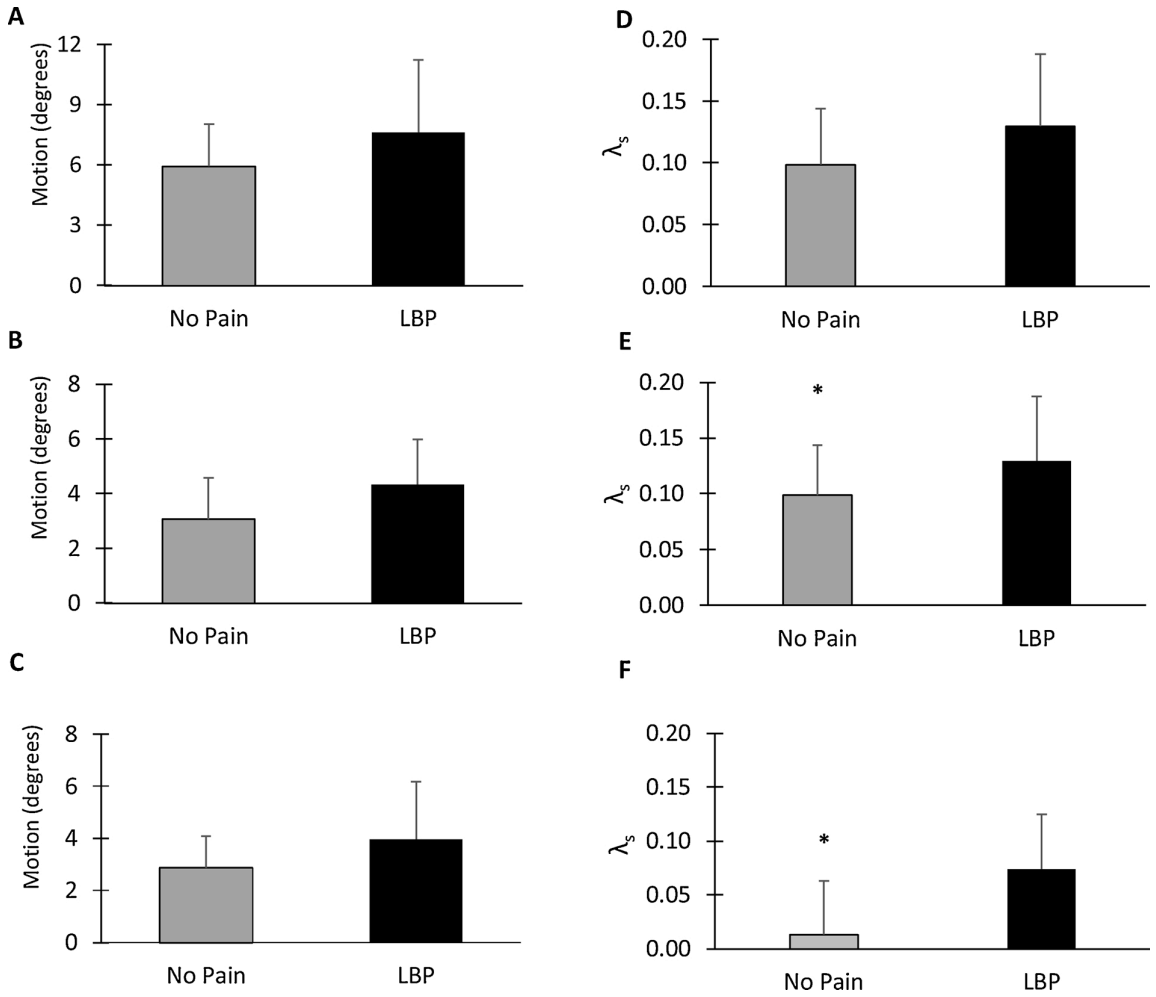

Fig. 2. Mean traditional and non-linear estimates of trunk motion during unstable sitting in persons with lower limb amputation with (LBP) vs. without (No Pain) low back pain. A) mediolateral trunk motion, B) anteroposterior trunk motion, C) axial trunk motion, D) short-term Lyapunov exponent (λ_s) of trunk motion in the mediolateral direction, E) short-term Lyapunov exponent of trunk motion in anteroposterior direction, F) short-term Lyapunov exponent of trunk motion in axial direction. Asterisks indicate significant difference between groups ($p < 0.05$).

and suggesting an association between reduced trunk postural control and LBP in this cohort, the LBP group exhibited larger A–P trunk motion and reduced local dynamic trunk stability in the rotational direction.

4.1. COP-based measures

Though COP-based measures did not differ significantly between groups, there was a large effect size for the difference in CP amplitudes ($d = 0.82$), as well as for mean velocity in both the M–L ($d = 0.81$) and A–P ($d = 0.83$) directions. These suggest potentially meaningful differences, though perhaps a large inter-individual variability precluded detecting statistical significance. The current LBP group exhibited larger CP amplitudes and mean velocity (in the M–L direction; Table 2), while the no pain group exhibited similar values to those previously reported among persons with LLA but without pain [12]. Further, the current LBP group demonstrated substantially larger values for all COP-based measures than previously reported for individuals without LLA and LBP [12,13]. Together, these results imply that persons with both LLA and LBP have a delayed ability to generate corrective responses to postural perturbations, specifically the transition from open-loop to closed-loop control [12]. This finding in the M–L, but not the A–P, direction is in contrast with previous work, where A–P measures were found to discriminate deficits in trunk postural control between non-LLA individual with vs. without LBP [10]. CP amplitudes in the M–L direction in the LBP group were nearly twice the magnitude of those in the no pain group ($d = 0.82$), suggesting that the thresholds for closed-loop corrective mechanisms are larger among persons with LLA and LBP [23,24]. The larger CP amplitudes may also indicate larger spinal neutral zones, where passive structures do not to provide resistance to movement, potentially increasing spinal instability and ultimately leading to LBP [16].

4.2. Trunk muscle activation

We expected trunk muscle activation ratios in persons with LLA and LBP to be larger compared to those without LBP. The LBP group demonstrated greater activation of the prosthetic-side LES compared to the intact-side LES, and compared to the no pain group. This suggests that persons with LLA and LBP perhaps develop an asymmetric preference or utilization of lumbar musculature compared to persons with LLA without LBP. A possible explanation for this may be that asymmetries develop as a consequence of habitual unilateral prosthesis use. Our findings with respect to LES/TES ratios are consistent with Willigenburg et al. [15], using a similar unstable sitting paradigm, who

reported ratios of lumbar/thoracic muscle activation that were also similar between persons with and without LBP. We speculate that the LBP group here adopted a co-contraction strategy, wherein increased activation of the anterior trunk musculature (i.e., rectus abdominus, external oblique) was used to increase spinal stability in the presence of larger neutral zones (as suggested above). While the lumbar and thoracic erector spinae muscles perform global trunk movements, other posterior muscles more specific to lumbar stabilization (e.g., lumbar multifidus) may exhibit muscular impairments specific to the lumbar segment and LBP. Ratios of lumbar/thoracic activation in persons with LLA (both LBP and no pain) found here are approximately 30% larger than those reported in non-LLA cohorts of persons with and without LBP [15]. While the activation ratio differences between the current groups did not reach statistical significance, the large effect size ($\eta^2 = 0.65$) warrants continued exploration of muscle pattern differences driving the changes in trunk and pelvis motion and coordination.

4.3. Trunk and pelvis kinematics

The observed differences in trunk and pelvis kinematics suggest that persons with both LLA and LBP adopt a distinct motor strategy. Trunk motion (relative to pelvis) in the A–P direction was larger in persons with LLA and LBP compared to those with LLA and no pain (Fig. 2), consistent with our study hypothesis and previous work [15]. The LBP group had reduced trunk local stability (evidenced by the increased Lyapunov exponent), in the M–L and rotational directions, which suggests impaired trunk postural control. Such an impairment has, in turn, been posited as a mechanism leading to altered trunk and pelvis coordination during gait in persons with LLA and LBP [17]. Similar to the arguments of Esposito and Wilken [17], persons with LLA and LBP may have difficulty achieving multi-directional/tri-planar control of the trunk and pelvis, and therefore may limit degrees-of-freedom in two planes (i.e., the frontal and transverse) in an effort to achieve stability using mainly sagittal-plane control mechanisms. Together, the increased A–P trunk motion, coupled with decreased local stability (evidenced by larger λ_s), suggest that persons with LLA and LBP may have difficulty stabilizing their trunk and pelvis independent of the lower extremities, particularly in situations that demand multi-planar control. This suggests a pathway to elucidate the altered trunk and pelvis mechanics exhibited during gait among LLA with vs. without LBP [17–19].

4.4. Limitations

A priori sample-size estimates indicated 11 participants per group were sufficient to detect statistically significant ($\alpha = 0.05$, $\beta = 0.20$) differences in traditional COP-based measures, with effect sizes of at least $d = 1.15$. These estimates used data from a prior study comparing unstable sitting performance in persons with vs. without LLA [12]. Despite the lack of statistically significantly group differences here, large effect sizes for the COP-based metrics and trunk muscle activation ratios suggest the study could have been underpowered for these variables despite the noted sample size estimates. Nevertheless, this population can be particularly difficult to study in large numbers.

The inability of traditional COP-based metrics to discriminate between groups may be due to insufficient precision of the testing paradigm, and it is possible that persons with LLA and LBP preferentially utilized visual feedback over trunk and pelvis proprioception to achieve seated balance. Notably, the magnitude of differences between groups for the traditional COP-based measure of CEA was less than the reported 95% minimal detectable change of this variable, suggesting that group differences did not exceed measurement error [11]. Therefore, future work should consider more challenging tasks capable of removing a potential reliance on the visual feedback system (e.g., eyes closed vs. open) [10,15]. Moreover, investigation of other trunk musculature, particularly deeper posterior and anterior musculature, is warranted to fully understand the neuromuscular control strategies associated with chronic LBP among persons with LLA.

Most participants in the LBP group were asymptomatic at the time of testing (i.e., pain levels < 3 on a visual analog scale 0–10), which is another potential explanation for the similar COP results between groups. We anticipate the present results to generalize to similar cohorts of individuals (e.g., males, relatively young and active), although caution should be used when generalizing to other populations with LLA (e.g., older adults with dysvascular limb loss). This study aimed to investigate the relationship between postural control and chronic LBP using the recently developed criterion for chronic LBP set forth in the NIH recommendations. Under these criteria, chronic LBP is defined as pain that persisted for at least three months and with pain on at least half the days in the past six months. Thus, it is possible that these individuals were not experiencing acute pain on the day of testing but have experienced pain for the duration and frequency aligned with the NIH criterion. This approach was used to capture postural control without acute pain confounding the results. Further, the ODI scores suggest that the chronic LBP group was significantly more affected by their LBP compared to the no pain group in functional outcomes.

Our participant groups included persons with both transtibial and transfemoral LLA; however, there were more persons with transfemoral LLA in the LBP group compared to the no pain group. The larger number of persons with transfemoral LLA in the LBP group compared to the no pain group could have impacted findings, since persons with transfemoral LLA have to account for more uneven mass distribution. Finally, while mass and stature were controlled for during statistical analyses, future work should consider normalizing chair difficulty to individual participant anthropometrics to provide enhanced sensitivity to group-level differences.

5. Conclusions

Persons with both LLA and LBP demonstrated impaired trunk postural control compared to those without pain, as evidenced by reduced local dynamic trunk stability and greater trunk motion during unstable sitting. It is possible that the traditional COP-based measures of trunk postural control provided a global measure of trunk postural control system output (i.e., observed sway), whereas non-linear measures of trunk and pelvis motion characterized how the trunk postural control system functioned to maintain stability during the task (i.e., response to continuous perturbations). The persistence of LBP among persons with LLA may be the result of neuromuscular adaptations in proximal structures driven by functional demands and aberrant motor behavior patterns that are often observed during activities of daily living. Future work should address the longitudinal development of LBP, as well as the relationship between trunk postural control and activities of daily living (e.g., gait, sit-to-stand), among persons with LLA.

Declaration of Competing Interest

The authors have no conflicts of interest to report.

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