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Dual Goals of Trunk Restriction and Stability are Prioritized by Individuals with Chronic Low Back Pain During a Volitional Movement

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Dual goals of trunk restriction and stability are prioritized by individuals with chronic low back pain during a volitional movement

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Abstract

Background.—Individuals with chronic low back pain demonstrate impaired responses to volitional and externally-generated postural perturbations that may impact stability whilst performing activities of daily living. Understanding how balance may be impaired by strategy selection is an important consideration during rehabilitation from low back pain to prevent future injurious balance loss.

Research Question.—This cross-sectional study explored the influence of an active pain episode on volitional movement patterns and stability during a sit-to-stand task in individuals with chronic low back pain compared to those with no low back pain history.

Methods.—Thirteen participants with low back pain who were in an active flare-up and 13 without pain sat on a height-adjusted chair and performed 5 sit-to-stand movements. Sagittal plane kinematics, kinetics, and surface electromyography were used to compute neuromuscular variables across Acceleration, Transition and Deceleration phases. Stability was assessed using times to contact of body centers of mass and pressure to base of support boundaries. Independent samples t-tests were used to examine group effects, and repeated measures analyses of variance assessed within-subjects effects across movement phases.

Results.—Individuals with low back pain tended to restrict proximal joint motions through heightened muscle activity while increasing distal joint movement and distal muscle contributions. Individuals with low back pain used a greater driving force, indicated by a longer time to contact of the center of pressure, to achieve comparable center of mass stability. Individuals with low back pain may prioritize trunk restriction and stability through the sit-to-stand movement, possibly related to fear of pain or movement.

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Conflict of Interest Statement

The authors have no conflicts of interest.

Significance.—The tendency for individuals with active low back pain to restrict trunk movements may require additional effort to maintain stability. Further research should examine whether trunk restriction is related to pain-related fear of movement and whether additional cognitive resources are required to maintain movement stability.

Keywords

low back pain; postural control; sit to stand; stability; time to contact

1. Introduction

Individuals with chronic low back pain (LBP) demonstrate movement patterns that differ from those of healthy controls, suggesting alterations in automatic[1], anticipatory[2] and volitional[3–6] postural control. Less is known about individuals experiencing acute LBP, particularly during volitional movements, yet these movements provide a window into higher level, task-related intentions that may shape movement patterns. For example, when walking at self-selected pace, individuals with LBP moved more slowly[5,3] despite having the capability to attain the higher speeds of non-LBP individuals[5]. Thus, quantifying movement patterns during volitional tasks may reveal motor control impairments associated with LBP impacted by higher level intentions.

The sit-to-stand (STS) task is a volitional activity of daily living that is often accomplished using large forward flexion of the trunk and/or hip. However, the STS can also be performed relying primarily on the knee with little contribution from the trunk/hip[4]—a strategy that may lead to a more posterior location of the body's center of mass (CM) at seatoff, when the base of support (BOS) is transferred to the feet only. In this position, there may be a greater potential for loss of stability during the movement because the more posterior position of the CM requires a higher forward CM velocity to successfully avoid a backwards fall, thereby increasing the risk of a forwards fall[7]. Thus, moving with a more posterior body CM position, as would likely result from using a knee strategy, may pose a greater postural threat than using a hip/trunk strategy. A strategy with the potential for stability loss may be avoided by individuals with LBP who could sustain further injury should a fall occur and who may also have compromised proprioception, potentially limiting accurate control of movement and detection of instability[8–11]. Indeed, Coghlin and McFadyen (1994) reported that individuals with LBP tended to move more slowly during the ascension phase of the STS, possibly to prevent loss of stability as the body CM moves to an upright position.

A second STS strategy (hip/trunk) involves greater trunk and hip flexion[12], resulting in a potentially more stable movement pattern that moves the CM close to the BOS of the feet prior to leaving the seat. Schultz et al. (1992) demonstrated that older, healthy individuals used larger trunk excursions to shift their ground reaction force more anteriorly during the STS, which they attribute to a desire to increase postural stability and decrease the likelihood of a backwards fall. However, this pattern may be undesirable for individuals experiencing LBP or have restricted lower back range of motion, who may wish to limit movement of and/or forces applied to the lumbar spine. Shum et al. (2005) reported that individuals with

chronic LBP restricted lumbar spine and hip excursions during the STS, and minimized torque applied about their lumbar spine[13] suggesting an intent to restrict trunk and hip motion and forces, perhaps at the expense of movement stability.

Presumably strategy selection can directly impact movement stability during the STS, but this has yet to be tested. To test this assumption the postural time to contact (TtC), which describes the time for the CM or center of pressure (COP) to contact the stability boundaries formed by the BOS[14], could be assessed. Postural TtC quantifies the predicted time of approach to stability boundaries based on the instantaneous position, velocity and acceleration of the CM or COP[15]. During a standing task, TtC has direct implications on the maintenance of upright balance because it indicates the amount of time available to respond before balance loss and can therefore be used to assess movement stability[14].

The STS, therefore, represents a volitional movement that may present a conflict for the individual with LBP; that is, choosing a movement strategy that requires either significant trunk/pelvis excursion or one that may result in compromised postural stability. Therefore, the purpose of this study was to compare the movement patterns and stability of individuals with LBP actively seeking treatment for an acute flare-up, with those of individuals with no history of LBP, whilst performing STS movements. We hypothesized that individuals with LBP would demonstrate reduced trunk motion and postural stability during the STS compared to those without LBP.

2. Methodology

2.1 Participants

A sample size estimation was performed using pilot data from individuals with aLBP and those without LBP. Altered activation of the internal oblique (IO) muscle (a trunk flexor) during volitional movements in individuals with chronic LBP has been well documented [16–19], therefore myoelectric responses of the IO were used to estimate effect size using Cohen's d [20]. Based on pilot data collection, a mean difference between individuals with and without LBP of 12.2 % of maximal IO activation (%max) and a pooled standard deviation between the two groups of 9.2 %max was determined, yielding a Cohen effect size of 1.33. Testing at an alpha level of 0.05, a power > 0.80 was obtained when the sample size was set at 10 per group, while a power of greater than 0.90 was obtained when the sample size was set at 13 per group [21].

Thirteen participants with chronic, recurrent LBP (aLBP) were recruited from local physical therapy clinics (Table 1). Participants with LBP were excluded by clinical exam if they had: neurological signs consistent with disc herniation; spinal or lower extremity disease, conditions or surgery; balance or cardiovascular disorders; current pregnancy; involvement in litigation because of LBP; or received worker's compensation for their LBP. Participants were tested when seeking treatment for LBP and reported average Numeric Pain Rating Scale (NPRS) values of 3 (0–7) [median (range)] on the day of testing.

Thirteen participants with no history of LBP (NLBP) were recruited from the local community through posted advertisements and word of mouth (Table 1). NLBP participants

were excluded if they had a neurological disease or balance disorder, uncorrected vision problems, cardiovascular disorders, severe musculoskeletal injuries, or back pain during the prior 12 months that required medical attention or resulted in missed work. All participants were participating fully in their usual role (e.g. employed, full-time student, homemaker) and signed an informed consent document in accordance with University of Vermont Institutional Review Board policy.

2.2 Procedures

Participants sat on an instrumented chair (force plate under the buttocks) with no back or arm rests, adjusted to 100% of knee height while both feet were placed at a self-selected width on a second force plate (Figure 1A). Mid-heel distance and average toe-out angle (from mid-sagittal plane to line connecting mid-heel and 2nd toe) were measured and marked to maintain foot position. Participants were not given instructions about how to sit but a 12 cm semi-flexible support was attached to the seat, behind the subject's sacrum, to aid return to original seated position. Participants performed 7 STS movements (2 practice and 5 recorded trials) at self-selected pace with arms crossed, and two quiet sitting and standing trials (5 s).

2.3 Data recordings

A 3-camera passive marker system (BTS, Milan, Italy) was used to collect 3-dimensional spatial coordinate data. Kinematic data were sampled at 50 Hz and low-pass filtered (dual pass, 2nd order Butterworth filter, 5 Hz). Retro-reflective markers were placed on bony landmarks, including the acromion process, superior aspect of the iliac crest, greater trochanter, lateral epicondyle of the femur, lateral malleolus, head of 5th metatarsal, which delineated trunk, pelvis, thigh, leg and foot segments in the sagittal plane (Figure 1A), as well as both force plates. In addition, the BOS boundary was demarcated by the rearward and forward positions of the heel and second toe, respectively, to determine the anterior and posterior BOS boundaries. First and second derivatives of marker coordinates were computed using a central difference technique to calculate linear and angular velocity and acceleration data.

Kinetic data measured using the force plates (AMTI, Watertown, MA, USA), representing the vertical and anterior-posterior ground reaction forces (GRFs) were amplified (4000x), low-pass filtered at 1050 Hz and sampled at 1000 Hz. Kinetic data were subsequently low-pass filtered (2nd order, dual pass, Butterworth filter) at 10 Hz and re-sampled at 50 Hz in synchrony with kinematic data using a custom program written using Matlab software (The Mathworks Inc., Natick, MA).

After appropriate skin preparation, EMG of the left lower limb and dorsal and ventral trunk, bilaterally, were recorded (BTS, Milan, Italy) using silver-silver chloride surface electrodes (Norotrode 20 bipolar, Myotronics, Kent, WA, USA) placed over the bellies of 16 muscles: Tibialis Anterior (TA), Medial Gastrocnemius (GA), Rectus Femoris (RF), Medial Hamstrings (HA), Rectus Abdominus (LRA and RRA: left and right RA respectively), Internal Oblique (LIO and RIO), External Oblique (LEO and REO), Erector Spinae at the third (LES3 and RES3), and first lumbar segments (LES1 and RES1), as reported

previously[22]. EMG signals were sampled at 1000 Hz in synchrony with the force plates, amplified (2000–10000x), band-pass filtered from 35–200 Hz and full-wave rectified.

2.4 Data analysis

A four-segment, anthropometric model (foot, lower leg, thigh and HAT; head/arms/trunk) [23] (Figure 1A) was used to determine segment mass and inertial parameters, and used to calculate the body CM location based on participant weight and anatomical landmarks to determine segment lengths. A five-segment model (foot, leg, thigh, pelvis, and a single segment comprised of the head/arms/trunk but excluding the pelvis)[24] was used in conjunction with kinetic and kinematic data to determine the net joint torques at the trunk, hip, knee and ankle joints, using inverse dynamics[23]. Segment angles of the body segments were computed relative to the horizontal situated at the distal end of the joint per the method reported by Winter[23], although with the addition of the pelvis segment. Segment angles were used to compute trunk (trunk re pelvis), hip (pelvis re thigh), knee (thigh re shank) and ankle (shank re foot) joint angles to reflect extension (+) and flexion (−) relative to anatomical position. The initial seated position of the pelvis angle is also reported relative to the horizontal placed at the distal end of the segment.

The STS was characterized by the total movement duration, relative proportions of the Acceleration, Transition and Deceleration phases[25], and time of Seatoff. These phases (Figure 1B) were defined, using whole body CM velocity traces as: Acceleration, onset of initial movement to time of peak horizontal CM velocity; Transition, peak horizontal CM velocity to peak vertical CM velocity; Deceleration, peak vertical CM velocity to the end of movement; and Seatoff, the time when seat force fell below 10N. Spatial and temporal variables quantified kinematic and kinetic data including: peak (relative to movement initiation detected by net anterior-posterior force) of the net COP, body CM positions and velocities (horizontal and vertical components), segment and joint angles; average forces (GRF), net joint torques and EMG computed as integrals for each of the acceleration, transition and deceleration phases divided by the length of each phase. Muscle activation profiles were normalized to the maximum average activity obtained over a 2000 ms rolling window during maximal, voluntary standardized contractions. Average baseline activity for each muscle was determined across an epoch from 500 to 250 ms prior to movement initiation. The average TtC[15] of the CM and COP in the anterior/posterior (ap) direction were computed across Transition ($Transition_{ttc}$) and Deceleration ($Deceleration_{ttc}$) phases to provide measures of postural stability relative to the BOS boundary. $Transition_{ttc}$ was truncated to start at seatoff until the end of Transition to restrict computation to when the BOS is defined solely by the boundaries of the feet.

All temporal and spatial variables were compared using independent samples T-tests to detect group differences and Cohen's d was used to compute effect size. GRF, joint torque and myoelectric averages across the three movement phases were compared using repeated measures analyses of variance, with movement phase as the repeated factor (Acceleration, Transition and Deceleration for all variables except EMG which also included Baseline) and aLBP vs. NLBP as the grouping factor. Partial eta squared (η^2) was used to assess effect size for these comparisons. An alpha level of $P<0.05$ was considered significant for statistical

comparisons and effect size thresholds were set at 0.2, 0,5 and 0.8 to denote small, medium and large effects, respectively.

3. Results

Individuals with aLBP, compared to NLBP, demonstrated altered STS movements reflected in some timing changes of the Acceleration phase and Seatoff time, restricted trunk movement as well as increased ankle dorsiflexion range of motion, posterior GRF magnitude, knee joint torque, COP TtC and co-activation of proximal muscles.

3.1 Demographics

aLBP and NLBP groups did not differ by age, height, weight, BMI, stance width or toe-out angle (P-values ranged from 0.180 to 0.900, Table 1), although there were moderate effects of height and stance width (Table 1). However, individuals with aLBP demonstrated significantly higher pain ratings (NPRS, P<0.001, d=0.89; McGill Number of Words Score, P<0.001, d=0.98; Table 1). Eleven of 13 individuals with aLBP reported current bilateral pain, while one reported unilateral pain (left), and one reported no pain on the day of testing.

3.2 Temporal parameters

Individuals with aLBP did not differ from the NLBP group on STS movement time (P=0.57, d=0.23) (Figure 1B), nor in the proportions of the Transition (P=0.13, d=0.62) or Deceleration phases (P=0.51, d=0.27). However, individuals with aLBP demonstrated a trend towards a shorter Acceleration (P=0.067, d=0.75) and had an earlier Seatoff (P=0.036, d=0.87).

3.3 CM, COP and TtC

Height-normalized, peak CM velocities did not differ between groups (Horizontal, P=0.510, d=0.26; Vertical, P=0.510, d=0.26), although the aLBP cohort attained the lowest CM position earlier than the NLBP group (P=0.037, d=0.86). Average TtC of the CM did not differ between groups (P=0.26, η^2=0.054 for $Transition_{ttc}$ and P=0.30, η^2=0.045 for Deceleration). Although there were no group differences in onset, peak or timing of peak of the COP (Figure 2), the average COP TtC was increased in the aLBP group during $Transition_{ttc}$ (P<0.0001, η^2=0.50) but not Deceleration (P=0.196, η^2=0.069).

3.4 Angular kinematics

Participants with aLBP demonstrated altered proximal and distal joint movements compared to the NLBP group (Figure 3A). Those with aLBP demonstrated a trend to reduced peak trunk flexion (P=0.078, d=0.72) and increased peak ankle dorsiflexion (P=0.046, d=0.83). Individuals with aLBP also demonstrated a trend towards a greater trunk angle (P=0.094, d=0.68) and reduced hip angle (P=0.037, d=0.87) for their seated position, likely due to a posterior pelvic tilt, indicated by a trend to a greater pelvis segment angle (P=0.070, d=0.74).

3.5 Kinetics

The aLBP group demonstrated similar lateral (P=0.64, η^2=0.009) and vertical GRF (P=0.29, η^2=0.047) patterns compared to NLBP (Figure 3B). However, individuals with aLBP demonstrated increased braking forces (P=0.014, η^2=0.23), that appear to be driven by the Transition (P=0.01, η^2=0.25) and Deceleration phases (P=0.044, η^2=0.16).

Both groups demonstrated similar joint torque patterns dominated by the hip/trunk during Acceleration, the hip and knee during Transition and the ankle during Deceleration (Figure 4). However, individuals with aLBP used greater average knee torque (Group X Phase, P=0.023, η^2=0.146), evident during the Transition phase (P<0.0001, η^2=0.45; trend during Deceleration, P=0.06, η^2=0.14).

3.6 Muscular activation

Similar muscle activation patterns were evident for both groups (Figure 5). During Acceleration, the movement was largely governed by TA and GA distally, and RA and EO proximally. During Transition, TA activity peaked, with HA and RF also contributing, while ES at L1 and L3 slowed trunk flexion and initiated trunk extension. During Deceleration, HA distally and IO proximally drove the movement, although EO and ES3 contributed bilaterally, albeit to a lesser degree than during Transition.

Group differences were evident largely at distal muscles (Figure 5). Individuals with aLBP demonstrated greater TA (P=0.048, η^2=0.15) and HA (P=0.049, η^2=0.31) activation throughout the movement, although TA differences were greatest during Acceleration and Transition, and for HA during Transition and Deceleration. Individuals with aLBP also demonstrated greater RF activity during Transition and Deceleration (Group X Phase: P=0.003, η^2=0.32). Proximally, the aLBP group demonstrated increased IO activity (Left, P=0.019, η^2=0.21; Right, P=0.046, η^2=0.16) across phases, indicating greater abdominal muscle involvement. The aLBP group demonstrated increased right EO activation (P=0.034, η^2=0.17), that was not evident on the left side (P=0.64, η^2=0.009). A similar asymmetry was detected for the right ES1, with increased activity in the aLBP group during Transition (P=0.004, η^2=0.17) but not the left (P=0.97; η^2<0.0001).

4. Discussion

Individuals with aLBP performed the STS with an initial posterior pelvic tilt, restricted trunk motion, increased ankle motion and greater knee torque throughout Transition and Deceleration compared to the NLBP cohort. These alterations were likely accomplished through increased activity of distal and abdominal muscles. Individuals with aLBP also utilized a greater driving force of the COP to achieve the same CM stability as those in the NLBP, as indicated by longer COP TtC values.

4.1 Individuals with aLBP favour a knee strategy yet maintain movement stability

The hip/trunk and knee strategies anchor a spectrum of movement strategies to accomplish the STS. On one hand, the hip/trunk strategy is thought to be more stable as it positions the body CM close to the BOS of the feet, prior to seatoff[4,26], while a knee strategy, typified

by high knee torque relative to other joints, is thought to be less stable because the CM is behind the feet BOS at seatoff and requires control of forward momentum[4,26]. Although the knee strategy could be ideal for individuals with LBP who may prefer to restrict trunk motion, this has not been shown. One patient cohort used equivalent torque distribution among joints[4] and while a second cohort demonstrated reduced lumbar and hip extensor torques, distal joint contributions were not assessed making it unclear whether there was overall reduction in joint torques or increased distal torques indicative of a knee strategy[13]. Consistent with our hypothesis, individuals in the current aLBP cohort favoured a greater knee contribution, demonstrated by increased knee torque and reduction of trunk torque, particularly during Transition.

Although it seems logical that the knee strategy would lead to reduced movement stability, the aLBP cohort achieved the same level of stability (indicated by similar CM TtC) using a knee strategy as the NLBP group which, in contrast, relied upon greater trunk motion. How did they achieve this? The aLBP group appears to have used a greater driving force, or “thrust”[27] indicated by a longer TtC of the COP. In other words, the aLBP cohort may have maintained a larger margin of stability (or “safety margin”) to control motion of the body CM[1,28]. Thus, individuals with LBP may prioritize stability through the STS movement but can do so even while restricting trunk movement.

This knee dominant movement strategy also requires control of forward momentum, largely by the hip and trunk musculature to prevent balance loss during the STS[4,26]. In the current aLBP cohort, forward momentum was controlled through a larger braking force during Transition and Deceleration, consistent with previous findings[25]. It appears that this braking force was not mediated by trunk extensor muscle activity, but rather by increased contribution of the RF and HA muscles, through their biarticular muscle action[25]. As a result, decreased reliance on the trunk and sufficient movement stability were achieved through an increased reliance on more distal musculature.

4.2 Restricted trunk movement: an active or passive phenomenon?

The aLBP cohort under study restricted trunk flexion during the STS (Figure 3) and demonstrated a reluctance to extend their trunk, indicated by a tendency toward posterior pelvic tilt at movement initiation and increased bilateral IO activity, to slow trunk extension during Deceleration. Individuals with LBP have demonstrated reduced trunk flexion and extension ranges of motion (ROM) during clinical testing[29], as well as slower lumbar flexion and extension velocities during the STS[6] that may indicate reluctance or inability to move through a normal ROM. Our current aLBP cohort demonstrated reduced lumbar flexion ROM [mean (sd): 42.7 (18.4)°] compared to normative values[30], which could be mediated by either passive or active lumbar ROM restrictions.

Passive tissue alterations that result in difficult or painful trunk movement could explain the trunk restrictions demonstrated in this cohort. A similar cohort of individuals with chronic LBP demonstrated increased perimuscular tissue thickness and connective tissue heterogeneity[31]— alterations that could lead to impaired movement. Individuals with LBP also demonstrated increased side-specific asymmetry of passive muscle elastic energy,

suggesting that there may be increased passive tissue resistance in this population[32], which could explain the tendency to trunk restriction in our cohort.

Reluctance of trunk movement has been previously reported during STS[6], bending to put on a sock[33] as well as following self-generated balance perturbations[34] that could be mediated through active modification of movement patterns, perhaps secondary to pain-related fear. Although we did not assess fear of pain or movement, individuals with LBP have previously demonstrated reduced movement speed[3], increased inhibition of muscle activity[35] and lumbar flexion restrictions[36] that were related more strongly to pain-related fear than reported pain levels. Therefore, it is conceivable that individuals with LBP have adapted their movements to restrict trunk motion as a consequence of pain-related fear of movement. Given that restricted trunk movement is apparent during volitional movements and anticipatory postural adjustments[34], as well as during dual task walking with increased attentional demands[37], it is plausible that trunk restriction in this population represents a higher-level task-related intention (i.e., central set) as opposed to solely a volitional movement adaptation.

5. Conclusions

Individuals with active LBP demonstrated movement patterns consistent with the dual purposes of trunk restriction and movement stability when performing STS movements. These seemingly contradictory goals were achieved by compensating for reduced trunk motion through increased distal joint motions and muscle activation. Similar trunk restriction with enhanced distal contributions have also been elicited by unexpected postural perturbations[22], although in a cohort with a history of LBP but no current pain, providing support for centrally-mediated movement alterations related to LBP that persist beyond the pain episode. Whether this proximal to distal reorganization is acquired across modes of postural coordination (volitional, anticipatory, automatic) concurrently or are sequentially acquired through volitional movement patterns reinforced over time, perhaps secondary to pain-related fear of movement, warrants further study.

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Highlights

- Individuals with active low back pain restrict trunk flexion during sit-to-stand
- Co-contraction of erector spinae and oblique muscles may limit trunk motion
- Increased ankle flexion and knee torque may compensate for reduced trunk motion
- Center of pressure time to contact is longer in pain group vs. control group
- Center of mass time to contact is equivalent implying similar movement stabilities

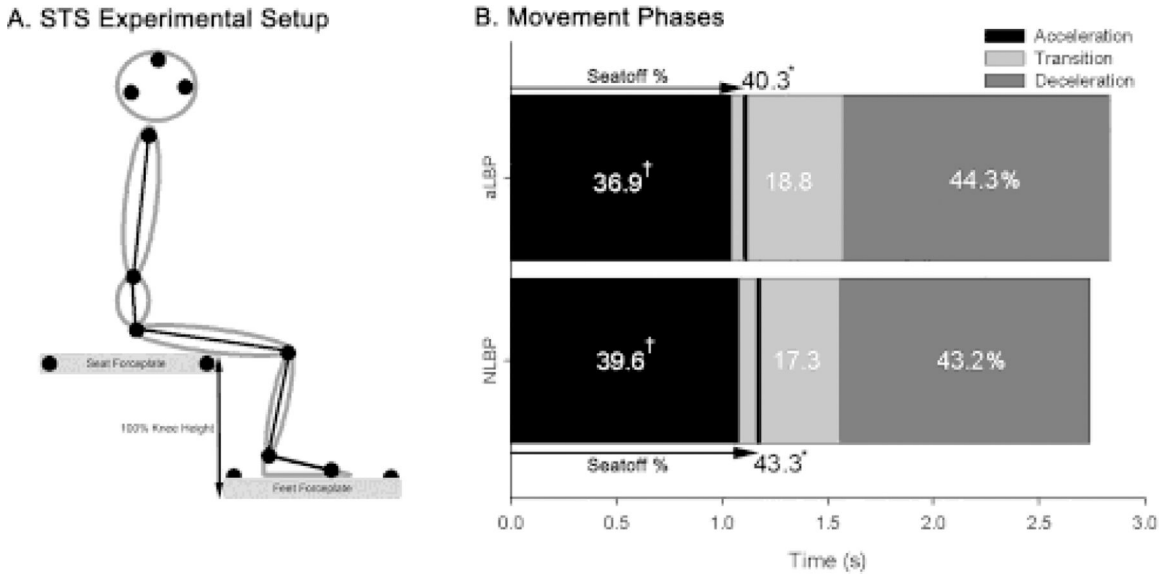

Figure 1:

A. Experimental setup. Black dots represent reflective marker placements and the grey bars represent two force plates, one embedded in the surface of a chair (chair not shown) and the second under the feet. Schematic figure does not depict the individual's arms, which were crossed across the chest, or the semi-flexible pelvic support used for repositioning between trials. B. Ensemble averages of the total movement time, and the Acceleration (black bar), Transition (light grey bar) and Deceleration (dark grey bar) phases of the STS movement. Relative contributions of each phase are reported in white (% of total movement time) and the relative times of Seatoff are denoted by the vertical black lines and corresponding black numbers (% total movement time). Bar plots depict group means comparing individuals with LBP (aLBP; top bar) to those without LBP (NLBP; bottom bar). Significant group effects are denoted by * ($P<0.040$) and non-significant between-group trends ($P<0.070$) are denoted by †.

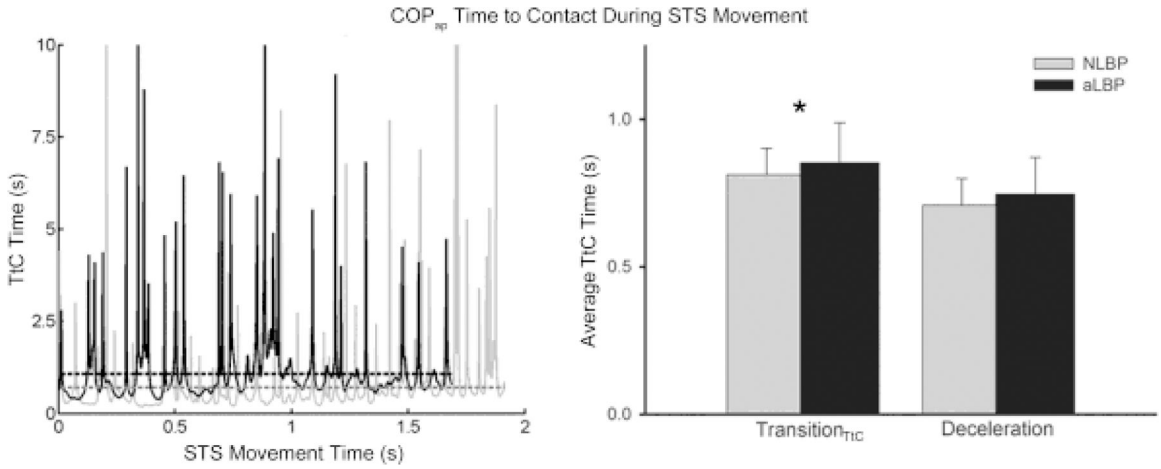

Figure 2:

Left panel depicts exemplar traces of the TtC of the COPap for the NLBP (grey) and aLBP (black) groups, dashed lines depict the average TtC values across the total movement time. Right panel depicts group means plus SD of the average TtC of the COP for the Transition (computed from seatoff) and Deceleration phases, respectively. Significant group effects are denoted by * ($P<0.0001$).

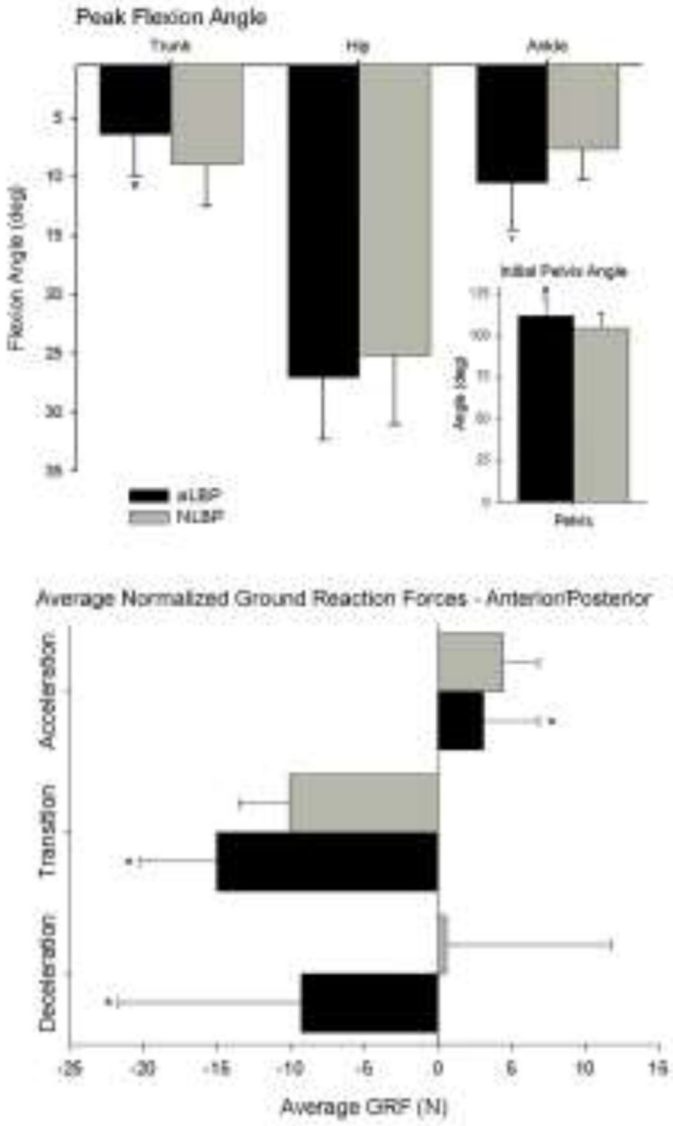

Figure 3:

Bar plots depict group means (±SD) comparing individuals with LBP (aLBP; black bars) to those without LBP (NLBP; grey bars). Top - Peak flexion angles of the trunk, hip and ankle during the STS movement. The inset depicts the angle of the pelvis relative to the right horizontal. Significant group effects ($P<0.05$) are denoted by *, whereas # denotes non-significant trends ($P<0.08$). Bottom - Average normalized anterior/posterior ground reaction forces across the Acceleration, Transition and Deceleration phases.

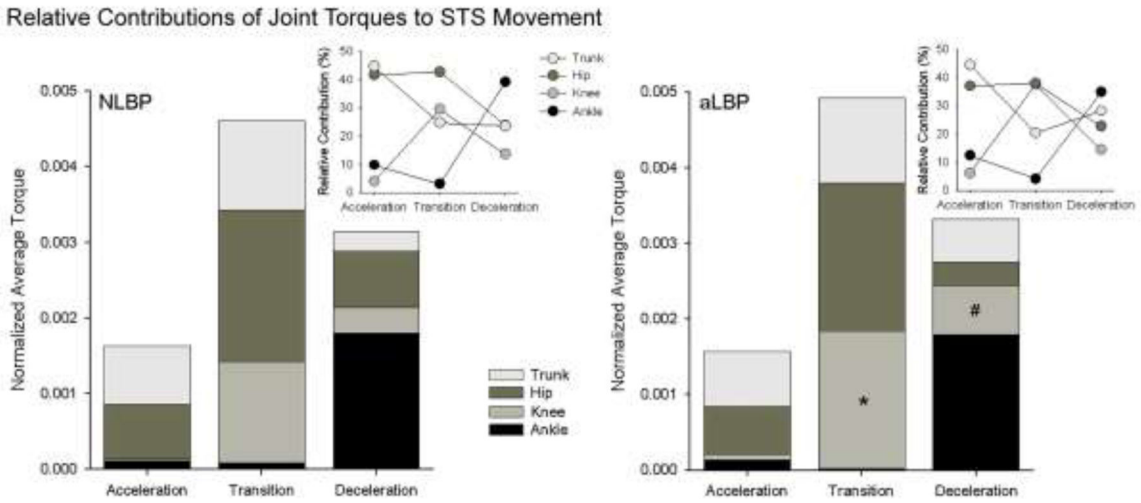

Figure 4:

Relative contributions of trunk, hip, knee and ankle torques to the STS movement across the Acceleration, Transition and Deceleration phases. Stacked bar plots depict the group means of each joint torque contribution to the total torque across each phase comparing individuals with LBP (aLBP; right panel) to those without LBP (NLBP; left panel). Significant group by phase interaction effects ($P<0.025$) are denoted by *, whereas # denotes a non-significant between groups post-hoc trend ($P<0.06$). Inset diagrams depict the relative contribution of each joint torque (% total torque) to the total summed torque across each STS phase.

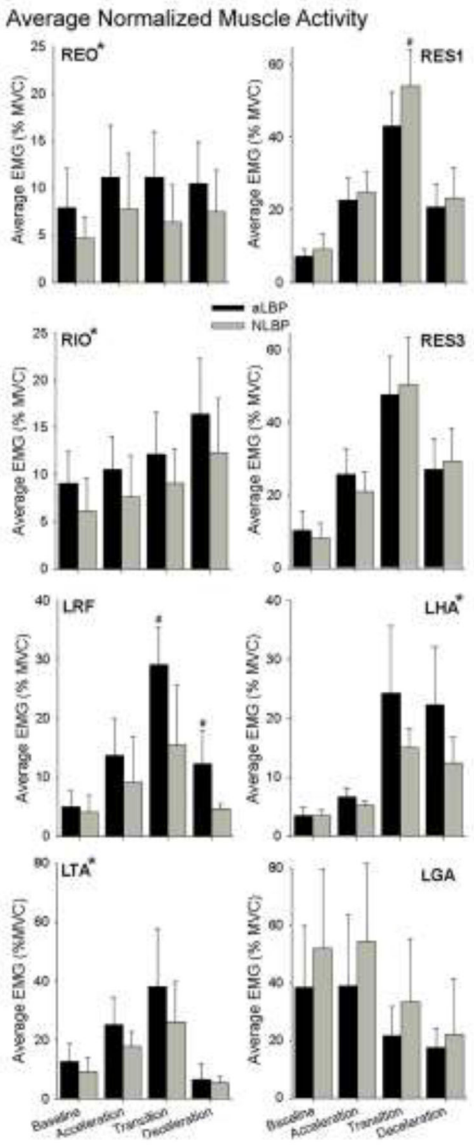

Figure 5:

Average normalized muscle activities (% MVC) for the right trunk and left leg muscles across the Baseline, Acceleration, Transition and Deceleration phases. Bar plots depict group means (±SD) comparing individuals with LBP (aLBP; black bars) to those without LBP (NLBP; grey bars). Significant group effects ($P<0.05$) are denoted by *, whereas significant group by phase interactions ($P<0.004$) are denoted by # at the phases with significant post-hoc differences.

Table 1:

Subject demographic information

Parameter [Mean (SD)]	aLBP (n = 13)	NLBP (n = 13)	P-value	Effect Size*
Age (years)	34.6 (6.4)	33.0 (5.9)	0.51	0.26
Height (m)	1.75 (0.09)	1.71 (0.07)	0.21	0.51
Weight (kg)	74.0 (9.1)	69.2 (13.3)	0.29	0.42
BMI (kg/m^2)	24.0 (2.3)	23.3 (2.8)	0.49	0.27
Sex (# Male/ # Female)	7 / 6	7 / 6	N/A	N/A
Stance Width (cm)	20.2 (3.9)	22.2 (3.3)	0.18	0.55
Toe-out Angle (°)	6.8 (6.0)	6.5 (4.3)	0.90	0.05
McGill Pain Questionnaire (# of Words Score) [Median (range)]	5 (2–11)	0 (0–3)	<0.001	0.98
Numeric Pain Rating Scale (/10) [Median (range)]	3 (0–7)	0 (0–1)	<0.001	0.89
Oswestry Disability Index (/100)	19.4 (9.1)	N/A	N/A	N/A
Lumbar Flexion Range (°)	42.7 (18.4)	N/A	N/A	N/A
Duration of Symptoms (yrs)	8.2 (8.0)	N/A	N/A	N/A

*Effect size is Cohen's d for parametic data; Rank biserial correlation for nonparametric data