Title: Trunk Muscle Forces and Spinal Loads while Walking in Persons with Lower Limb Amputation: Influences of Chronic Low Back Pain

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ABSTRACT

Persons with lower limb amputation (LLA) are at high risk for developing chronic low back pain (LBP), often with biomechanical factors considered as likely contributors. Here, trunk and pelvis kinematics, muscle forces, and resultant spinal loads were characterized in persons with LLA, with and without chronic LBP. Thirty-five persons with unilateral LLA – 19 with chronic LBP ("LLA-cLBP"), 16 without LBP ("LLA-nLBP") – and 15 (uninjured) persons without LBP ("CTR-nLBP") walked overground (1.3m/s) while thorax, pelvis, and lumbar kinematics were tracked (and ranges of motion [ROM] computed), and used as inputs for a non-linear finite element model of the spine to estimate global and local muscle forces, and resultant spinal loads. In the frontal and transverse planes, thorax ROM were up to 66.6% smaller in LLA-nLBP versus LLA-cLBP (*P*<0.001) and CTR-nLBP (*P*<0.001). In the sagittal plane, pelvis ROM was 50.4% smaller in LLA-nLBP versus LLA-cLBP (*P*=0.014). LLA-cLBP exhibited 45.5% and 34.2% greater peak local and global muscle forces, respectively, versus CTR-nLBP (*P*<0.011). Up to 48.1% greater spinal loads were observed in LLA-cLBP versus CTR-nLBP (*P*<0.013); peak compression and local muscle forces were respectively 20.2% and 41.0% larger in LLA-nLBP versus CTR-nLBP (*P*<0.005). Despite differences in trunk and pelvis kinematics between LLA-cLBP and LLA-nLBP, trunk muscle forces and spinal loads were similar (*P*>0.101) between these groups. Similar loading parameters regardless of LBP presence, while highly dependent on trunk muscle activation strategies, may mitigate further accumulation of mechanical fatigue. It remains important to understand the temporality of loading with respect to LBP onset following LLA.

Keywords: Extremity Trauma; Limb Loss; Finite Element Model; Gait; Biomechanics

INTRODUCTION

It has been projected that the number of persons living with amputation will more than double from 2005 to 2050 (Ziegler-Graham et al., 2008). Moreover, extremity trauma accounts for nearly half of all combat wounds during the Global War on Terror (Hoencamp et al., 2014), with more than 1700 Service members sustaining over 1900 lower limb amputations (LLA; Farrokhi et al., 2018). Low back pain (LBP) is a common musculoskeletal impairment following LLA – occurring much more frequently relative to the general population (52-89% vs. 12-45%) – and capable of substantially reducing longer-term function and quality of life (Devan et al., 2017; Taghipour et al., 2009). Although LBP is a multifactorial disorder secondary to LLA (Farrokhi et al. 2017), biomechanical factors have been suggested as important contributors to LBP due to repetitive exposures to abnormal motion patterns during activities of daily living (e.g., Coenen et al., 2012). Consistently, persons with LLA perceive these altered movements as primary contributors to LBP (Devan et al. 2015).

Altered trunk-pelvic motions with (vs. without) LLA are evident during activities of daily living, such as walking (Devan et al., 2017; Goujon-Pillet et al., 2008; Jaegers et al., 1995). In addition, persons with (vs. without) LLA also demonstrate earlier and prolonged activation of posterior trunk muscles during walking (Butowicz et al., 2018), as well as larger magnitudes and durations of trunk muscle activations during controlled trunk flexion (Hendershot and Nussbaum, 2014). During walking, these biomechanical alterations at the trunk and pelvis with LLA can impose up to 60% greater mechanical loads on spinal tissues compared to uninjured persons (Hendershot et al., 2018; Shojaei et al., 2016; Yoder et al., 2015). This in turn may accelerate fatigue failure of spinal tissues (Hendershot and Bazrgari, 2020), supporting a potential causal pathway for LBP onset and/or recurrence.

In the presence of LBP, further alterations in trunk-pelvic motion have been identified among persons with LLA. Specifically, persons with transfemoral limb loss, with versus without LBP, tend to walk with more sustained lumbar rotation toward the prosthetic limb, perhaps due to fear of pain with trunk lateral flexion, which was associated with greater pelvic elevation/hip hiking (Devan et al., 2017). Other studies have illustrated either greater axial rotational excursion (Morgenroth et al., 2010) with LBP, or oppositional motion patterns in the sagittal and transverse planes (Fatone et al., 2015). However, numerous confounding factors have been discussed toward explaining these outcomes (e.g., modelling approaches, subject demographics, LBP categorization), thereby limiting their strength of evidence (Highsmith et al., 2018). Nevertheless, better understanding these biomechanical factors, specifically comparing persons with LLA, with and without LBP, remains an important knowledge gap for mitigating the long-term consequences of LBP following LLA (Sivapuratharasu et al., 2019).

Therefore, the purpose of this study was to evaluate trunk-pelvic motion, corresponding trunk muscle forces, and spinal loads among persons with LLA, and specifically compare between those with versus without chronic LBP (and relative to uninjured controls). Given prior work among persons without LLA demonstrating elevated muscle activity and spinal loading with LBP, we hypothesized that there would be distinct differences in trunk-pelvic motions that are associated with larger trunk muscle forces and spinal loads among persons with lower limb loss and LBP, supporting a pathway wherein repeated exposure to abnormal spine mechanics can adversely affect spine health.

METHODS

Participants

Thirty-five persons with traumatic, unilateral LLA – nineteen (12 transtibial / 7 transfemoral) with chronic LBP ("LLA-cLBP") and sixteen (12 transtibial / 4 transfemoral) without chronic LBP ("LLA-nLBP") – and fifteen uninjured controls without LBP ("CTR-nLNP") participated (Table 1). To categorize by LBP status, participants completed the National Institutes of Health minimal dataset for chronic LBP (40-item questionnaire; Deyo et al., 2014) which defines chronic LBP as lasting longer than 6 months and occurring at least half the days in the past 6 months. Among the LLA-cLBP group, most (n=11) reported LBP lasting greater than 5 years, with the rest reporting LBP lasting either 1-5 years (n=7) or 6-12 months (n=1). In addition, participants in the LLA-cLBP group reported experiencing LBP either every day or nearly every day in the most recent six months (n=13), or at least half the days in the most recent 6 months (n=6). Mean (standard deviation) pain in the past seven days = 3.4 (1.4).

[Insert Table 1 about here]

Experimental Design and Procedures

Participants walked overground across a 15m walkway at 1.3 m/s, with speed enforced by auditory feedback; a tone (beep) sounded when the fore-aft component of velocity was within ±5% of the desired speed. Multiple trials were completed to obtain ~20 (range: 16 - 27) complete gait cycles. Post-hoc analyses confirmed mean walking speed = 1.31±0.05 m/s across all trials.

During walking, an 18-camera motion capture system (Qualisys, Göteborg, Sweden) tracked (120Hz) trunk and pelvis kinematics via 10 reflective markers placed at the C7 and T10 spinal processes, sternal notch, xiphoid, and bilaterally across the acromion and anterior/posterior superior iliac spines. Marker trajectories were low-pass filtered (Butterworth, 6Hz).

Dependent Measures and Data Analyses

Tri-planar thorax (global), pelvis (global), and lumbar (i.e., trunk relative to pelvis) rotations, and pelvis center of mass displacements were calculated in Visual3D (C-motion, Germantown, MD, USA), time-normalized to stride, and subsequently averaged across strides to yield a singular representative gait cycle per subject. These data were input to a non-linear finite element model of the spine at the T12 and the S1 vertebrae (Shojaei et al., 2016). The model (Bazrgari et al., 2007; Bazrgari et al., 2008a; Bazrgari et al., 2008b) consists of six rigid elements representing the thorax (T1-T12) and each lumbar vertebra (L1-L5), and six flexible, non-linear beam elements characterizing the non-linear stiffness of each lumbar motion segment between T12 and S1 (Shirazi-Adl and Parnianpour 1993, Shirazi-Adl and Parnianpour 2000). Mass and inertial properties were distributed according to previously reported ratios (de Leva, 1996, Pearsall et al.,1996, Zatsiorsky and Seluyanov, 1983). In total, 56 muscles are represented in the model (Bazrgari et al., 2007) – 46 connecting individual lumbar vertebrae to the pelvis (i.e., local) and 10 connecting the thoracic spine/rib cage to the pelvis (i.e., global).

Angular kinematics of lumbar vertebrae, required for estimation of local muscle forces, were estimated in an iterative mixed inverse and forward dynamic approach (Figure 1). Specifically, the inverse dynamic aspect of the approach involved the prescription of measured kinematics to T12 and S1 to obtain the mechanical demand of the task on the global muscles (i.e., tri-planar moment at T12). The forward dynamic aspect, on the other hand, involved the prescription of tri-planar moments at each lumbar vertebrae to obtain the tri-planar angular kinematics of lumbar vertebrae. For each iteration, these prescribed moments were determined by multiplying the T12 moment from the previous inverse dynamic phase with the ratio of the sum of physiological cross-sectional area of muscles attached to that lumbar vertebra over the sum of physiological cross-sectional areas of global muscles. The convergence of this iterative approach was assumed when the changes in the vector norm of the T12 moment became less than 5%. Once the angular kinematics of lumbar vertebrae were determined, trunk muscle forces and spinal loads at all lumbar levels were calculated as in our previous studies (Bazrgari et al., 2007; Bazrgari et al., 2008a; Bazrgari et al., 2008b). Rather than report individual muscle forces, summations across the 46 local and 10 global muscles were obtained (hereby referred to as “local” and “global” muscle forces, respectively). Spinal loads were compiled from the intervertebral level at which maximum spinal loads occurred (i.e., L5/S1). Peak spinal loads and muscle forces were determined and normalized to body mass. Thorax, pelvic, and lumbar ranges of motion (ROM) were also determined for each gait cycle and averaged across strides.

[Insert Figure 1 about here]

Statistical analyses were performed in R (Vienna, Austria). Separate one-way repeated measures ANCOVAs assessed the effect of group (LLA-P, LLA-NP, CTR) on all outcomes ($P<0.05$) while controlling for injury level (transtibial, transfemoral, uninjured). Bonferroni-corrected t-tests ($P<0.0167$) assessed pairwise differences when main effects were observed. Partial eta-squared (η^2) and Cohen's *d* (d) assessed effect sizes for ANCOVAs and t-tests, respectively and evaluated according to prior reports (η^2: small=0.01, medium=0.06, large=0.14; d: small=0.2, medium=0.5, large=0.8; Cohen 1988).

RESULTS

Main effects of group were observed in sagittal plane pelvic ROM ($P=0.018$, $\eta^2=0.147$), frontal ($P<0.001$, $\eta^2=0.327$) and transverse ($P<0.001$, $\eta^2=0.368$) plane thorax ROM, and sagittal ($P=0.021$, $\eta^2=0.086$) and frontal ($P=0.036$, $\eta^2=0.327$) plane lumbar ROM (Table 2). In the sagittal plane, pelvic ROM was smaller in LLA-nLBP vs. LLA-cLBP (3.4 [1.4]° vs. 5.0 [1.7]°, $P=0.014$, d=1.00); similarly, lumbar ROM was lesser in LLA-nLBP vs.

LLA-cLBP (3.7 [1.3]° vs. 5.8 [2.0]°, *P*=0.005, d=1.23). In the frontal plane, thorax ROM were smaller in LLA-nLBP vs. LLA-cLBP (3.6 [1.4]° vs. 7.3 [2.9]°, *P*<0.001, d=1.60) and CTR-nLBP (3.6 [1.4]° vs. 7.5 [2.0]°, *P*<0.001, d=2.19); lumbar ROM was smaller in LLA-nLBP vs LLA-cLBP (10.5 [2.8]° vs. 14.6 [4.4]°, *P*=0.003, d=1.10). In the transverse plane, thorax ROM were smaller in LLA-nLBP vs. both LLA-cLBP (5.3 [1.1]° vs. 8.6 [2.5]°, *P*<0.001, d=1.70) and CTR-nLBP (5.3 [1.1]° vs. 8.7 [1.9]°, *P*<0.001, d=2.12).

[Insert Table 2 about here]

Main effects of group were also observed in peak anterior-posterior shear (*P*=0.038, η^2=0.008), mediolateral shear (*P*=0.004, η^2=0.021), and compression (*P*<0.001, η^2=0.058). Similarly, main effects of group were observed in both local (*P*<0.001, η^2=0.008) and global (*P*=0.033, η^2=0.007) muscle forces. Greater peak spinal loads were observed in LLA-cLBP versus CTR-nLBP (Figure 2a) in the anterior-posterior (11.9 [2.7] vs. 9.4 [2.1] N/kg, *P*=0.013, d=1.00), mediolateral (3.7 [1.2] vs. 2.5 [0.4] N/kg, *P*=0.009, d=1.29), and compressive (23.5 [3.4] vs. 17.9 [2.6] N/kg, *P*<0.001, d=1.86) directions. In LLA-nLBP versus CTR-nLBP, larger peak compression forces (21.5 [4.1] vs. 17.9 [2.6] N/kg, *P*=0.005, d=1.06) were observed (Figure 2a). Similarly, in LLA-cLBP versus CTR-nLBP, there were greater peak local (10.1 [3.0] vs. 7.0 [1.9] N/kg, *P*<0.001, d=1.25) and global muscle forces (16.3 [4.2] vs. 12.1 [2.9] N/kg, *P*=0.011, d=1.15; Figure 2b). Larger peak local muscle forces were also observed in LLA-nLBP compared to CTR-nLBP (9.8 [2.3] vs. 7.0 [1.9] N/kg, *P*=0.003, d=1.35; Figure 2b). However, no pairwise differences were observed between LLA-cLBP and LLA-nLBP (*P*>0.101).

[Insert Figure 2 about here]

DISCUSSION

This study aimed to characterize trunk-pelvic kinematics and estimate corresponding trunk muscle forces and spinal loads among persons with LLA, specifically comparing those with and without chronic LBP. In partial support of our hypothesis, altered thorax, pelvis, and lumbar ROM were observed in LLA-cLBP vs. LLA-nLBP; however, these motions were similar between LLA-cLBP and CTR-nLBP. Greater transverse-plane trunk and pelvis motions in the presence of LBP have been previously reported in cohorts with LLA (Morgenroth et al., 2010). However, among persons without LLA, prior work has generally observed lesser or unchanged trunk and pelvis rotations with vs. without LBP (Laird et al., 2014, Muller et al., 2015, Seay et al., 2011). Among persons without LLA, smaller and less variable trunk and pelvis motions in the presence of LBP have been proposed as a guarding mechanism to avoid large and/or aberrant motions that may result in injury (van der Hulst et al., 2010). Similarly, in the absence of LBP, persons with vs. without LLA exhibit more in-phase and less variable trunk and pelvic motion, again posited as a guarding mechanism (Butowicz et al., 2018); the presence of LBP had little effect on trunk-pelvic coordination (Esposito and Wilken, 2014).

These movement patterns are reflected in the trunk muscle activation patterns among persons with LBP (without LLA) and LLA (without LBP). Larger trunk muscle activations, relative to uninjured controls, have been observed in both persons with LBP (Ghamkhar and Kahlaee, 2015) and persons with LLA (Butowicz et al., 2018). During gait, trunk musculature serve to both stabilize the spine (Granata and Oroshimo, 2001) and assist in propulsion (Ceccato et al., 2009). Thus, these increases are likely related to the observed guarded gait, and in the case of LLA, may be a compensatory measure for propulsion to account for the loss of lower limb musculature. In addition, prior work has identified deficiencies in trunk musculature, as a risk factor for LBP (Hodges et al., 1996; Lee et al., 1999). It is therefore possible the LLA-cLBP cohort was unable or unwilling to use a guarded gait due to such deficiencies as it could result in pain and/or injury. While the present results, taken together with prior reports, may imply a guarded gait is beneficial for avoiding cLBP with LLA, it is important to note that the causal relationship between the two (if any) is unknown; thus, there remains a need for longitudinal tracking of these outcomes in LLA populations.

The similar spinal loads and trunk muscle forces observed between LLA-cLBP and LLA-nLBP cohorts, while contrary to our hypothesis, may support the notion of a guarded gait. Among persons without LLA, the presence of LBP has been linked to elevated muscle activity (van der Hulst et al., 2010) and increases in spinal loading (Marras et al., 2001). Similarly, independent of LBP, LLA has been associated with larger trunk muscle forces and spinal loads compared to uninjured controls (Shojaei et al., 2016, Hendershot et al., 2018). While it follows that there would be compounding effects on trunk muscle forces and spinal loads in the presence of both LLA and LBP, insufficient activation of trunk musculature may have prevented the use of a guarded gait, resulting in lesser spinal loads than would have otherwise been present. While this motor control strategy mitigates injury risk due to mechanical fatigue/overuse, it puts the LLA-cLBP cohort at a higher risk for acute injury due to large/aberrant motions at the low back.

Several limitations are worth noting when interpreting results from the current study. First, the LLA-cLBP cohort was largely asymptomatic at time of testing (mean pain = 1.7±1.5). Prior work in persons with chronic LBP observed more in-phase coordination during active bouts of LBP compared to when LBP is in remission (Shih et al., 2021). Additionally, the definition of LBP used here only takes into account duration and frequency of LBP and does not consider other factors which may contribute to LBP/LBP avoidance (e.g., fear of pain); though induced pain and fear of pain minimally affect trunk muscle activities and kinematics in the near term (Lamoth et al., 2004). Second, we lacked a cohort of non-limb loss persons with LBP, to provide a full-factorial experimental design (i.e., with and without LLA, with and without LBP) which would allow us to independently evaluate the effects of LLA and LBP on trunk biomechanics. Notably, effect sizes of group for spinal loads and trunk muscle forces were relatively small ($\eta^2<0.058$) suggesting that, despite statistical significance, the observed differences may not be clinically meaningful based on established norms (Cohen 1988); however, due to the repetitive nature of walking, even small increases in these measures can result in cumulative fatigue and failure (Hendershot and Bazrgari, 2020). Additionally, although differences in ROM were identified between LLA-cLBP and LLA-nLBP, we acknowledge there exists inconsistent evidence for using kinematic and kinetic parameters to evaluate LBP, albeit among a non-limb loss population, and largely limited by sample size and heterogeneity (Papi et al., 2018). Further, lumbar segmental kinematics were estimated by tracking thoracic motion and the assumption that the trunk is a rigid body, likely leading to over estimations of lumbar motion (Papi et al., 2019). However, while computation of thorax relative to pelvis ROM revealed similar trends, any overestimation in segmental kinematics within the model applies similarly to all participants, regardless of group. Tasks other than walking (or walking at faster speeds) might also be more challenging/provocative and thus better differentiate by LBP status.

There are several limitations with regards to the model used as well. Though the model has been extensively validated in back-healthy populations (Bazrgari et al., 2008a; Bazrgari et al., 2009; Bazrgari et al., 2011), it has yet to be formally validated in individuals with LBP and/or LLA. Thus, consideration of physiological and neuromuscular changes in trunk musculature associated with these populations is warranted. First, the model does not account for changes in physiological cross-sectional areas (PCSA) of trunk muscles often associated with LBP (Fortin and Macedo, 2013) and LLA (Kulkarni et al., 2005). The ratio of local-to-global muscle PCSA influences segmental kinematics during the forward dynamics portion of the simulation as well as the distribution of task demand across muscles during the optimization procedure. However, prior work has reported similar reduction in PCSA of local and global muscles with LBP (Fortin and Macedo, 2013). As such, alterations in PCSA are unlikely to affect these results, though further research is warranted. Further, changes in mechanical properties (e.g., stiffness) of the spine (Hendershot et al., 2013) are not replicated in the model; note, while prior work has observed that decreases in stiffness can result in significant increases in spinal loads during lifting tasks (Bazrgari and Shirazi-Adl, 2007), the same is likely not true for walking. During lifting, passive spine ligaments contribute greatly to spine equilibrium due to the forward bending posture. During walking, however, lumbar range of motion likely falls within spinal neutral zones wherein active muscle forces are the main contributor to spine equilibrium and stability (Panjabi, 1992); therefore, alterations in stiffness are unlikely to alter simulation results. In addition to muscle PCSA and spine stiffness, further personalization of the model (e.g., body size/mass distribution, radiographic measures, spine curvature) would likely lead to more accurate results. Additionally, the kinematics-driven model used here did not individually account for altered muscle recruitment strategies with LBP (e.g., co-activation; Van Dieen et al., 2003); thus, the reported spinal loads among the LLA-cLBP group are likely underestimates given common increases in spinal loads with co-activation (Marras et al., 2001). We also implemented a new method for determination of lumbar vertebrae kinematics in this study because the angular kinematics of lumbar vertebrae, calculated using the reported ratios of lumbar segmental contribution to total lumbar range of motion, would not often yield reaction moments at each lumbar vertebra that could be balanced by muscles attached to that specific level. This issue is likely due to the facts that such contribution ratios were obtained under lumbar motion in one anatomical plane (Adams et al., 2006) and did not represent the actual contribution of lumbar segments to the complex three-dimensional movement of the lumbar spine during walking. In our new approach, we assume that the local muscles contribute to spine equilibrium proportional to their moment-generating capacity as compared to the global muscles. In an earlier study, we have shown that variations in angular kinematics of lumbar vertebrae can alter spinal loads up to 15% (Shojaei et al., 2015).

In summary, although prior work has identified larger spinal loads in persons with vs. without LLA, the current results do not necessarily support the notion that larger spinal loads during walking influence the persistence of LBP. It is, however, possible that persons in the LLA-cLBP cohort modulate their gait due to deficiencies in trunk musculature in order to reduce demand at the low back and thus mitigate the risk of overuse and injury. Although it is unclear whether this is an effect of LBP or a mechanism which led to it. As noted previously, future work is still needed to longitudinally characterize the temporal relationships in these outcomes, ideally with time since LLA, to better elucidate the causal relationships.

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Table 1. Mean (standard deviation) participant demographics among persons with lower limb amputation (LLA), with (cLBP) and without (nLBP) low back pain, and without LLA (controls; CTR-nLBP). TT = transtibial; TF = transfemoral. Asterisks (*) indicate main effects of group (*P*<0.05); + indicates significant difference from CTR-nLBP (*P*<0.0167)

	LLA-cLBP	**LLA-nLBP**	**CTR-nLBP**
Age (yr)	35.8 (7.3)	33.9 (7.2)	29.2 (8.9)
Stature (cm)	179.5 (7.4)	178.4 (4.8)	168.5 (27.5)
Mass (kg)*	90.4 (16.5)$^{+}$	88.9 (13.9)$^{+}$	72.8 (12.6)
Time since injury (mo)	100.4 (47.4)	89.5 (54.5)	NA
Level of injury (TT/TF)	12/7	12/4	NA
LBP, at testing (0-10)	1.7 (1.5)	0.3 (0.6)	0.1 (0.3)
LBP, last 7 days (0-10)	3.4 (1.4)	1.5 (1.1)	1.2 (0.4)

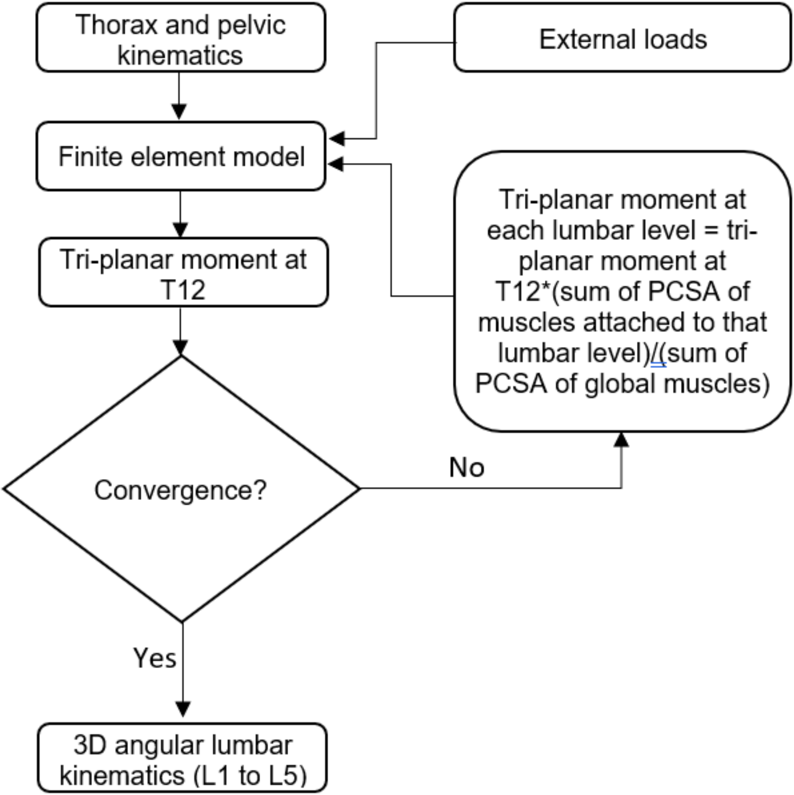
Table 2. Mean (standard deviation) thorax, pelvis, and lumbar ROM among persons with lower limb amputation (LLA), with (cLBP) and without low back pain (nLBP), and without LLA (controls; CTR-nLBP). Asterisks (*) indicate main effects of group (*P*<0.05); + and ^ indicate significant differences from CTR-nLBP and LLA-cLBP, respectively (*P*<0.0167)

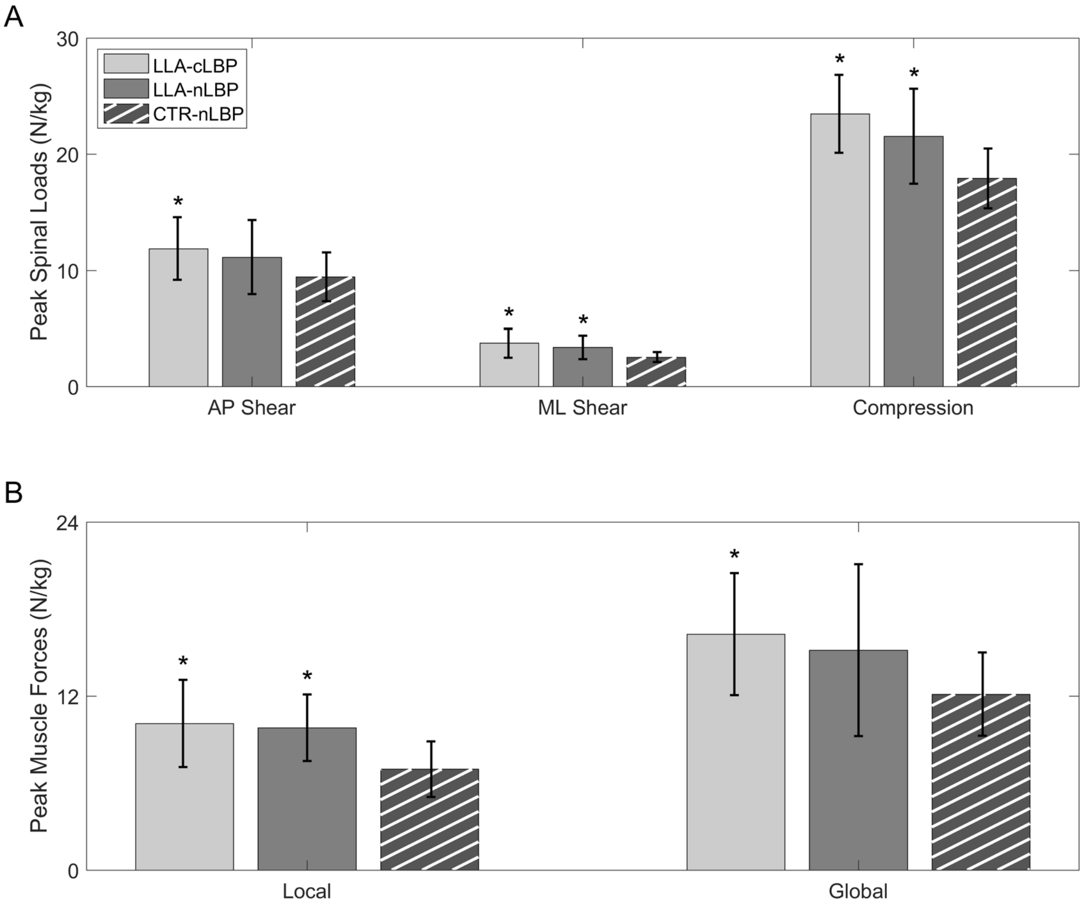
		LLA-cLBP	**LLA-nLBP**	**CTR-nLBP**
Thorax ROM (°)	**Sagittal**	3.7 (1.1)	2.9 (0.5)	3.8 (1.7)
	Frontal*	7.3 (2.9)	3.6 (1.4)$^{+\wedge}$	7.5 (2.0)
	Transverse*	8.6 (2.5)	5.3 (1.1)$^{+\wedge}$	8.7 (1.9)
Pelvis ROM (°)	**Sagittal***	5.0 (1.7)	3.4 (1.4)^	4.0 (1.5)
	Frontal	7.9 (2.5)	7.5 (2.3)	6.5 (1.3)
	Transverse	10.2 (3.7)	10.1 (3.8)	8.5 (1.9)
Lumbar ROM (°)	**Sagittal***	5.8 (2.0)	3.8 (1.3)^	5.2 (2.1)
	Frontal*	14.6 (4.4)	10.5 (2.8)^	12.7 (2.6)
	Transverse	12.9 (3.2)	11.6 (4.5)	12.6 (3.0)

Figure Legends

Figure 1: Kinematics of individual lumbar vertebrae were estimated by allocating portions of the tri-planar moment at T12, calculated using measured kinematics of the thorax and pelvis, to lumbar vertebrae. This was done through an iterative procedure and the convergence was assumed when the change in predicted tri-planar moment at T12 was less than 5% in two consecutive iterations.

Figure 2: A) Peak anterior-posterior (AP) shear, mediolateral (ML) shear, and compression spinal loads and B) local and global muscle forces in persons with lower limb amputation with and without chronic low back pain (LLA-cLBP and LLA-nLBP, respectively), as well as controls without chronic low back pain (CTR-nLBP). Asterisks (*) indicate significant differences ($P<0.0167$) from CTR-nLBP.


Thorax and pelvic kinematics
External loads
Finite element model
Tri-planar moment at T12
Tri-planar moment at each lumbar level = tri-planar moment at T12*(sum of PCSA of muscles attached to that lumbar level)/(sum of PCSA of global muscles)
Convergence?
No
Yes
3D angular lumbar kinematics (L1 to L5)


A
B
Peak Spinal Loads (N/kg)
30
20
10
0
LLA-cLBP
LLA-nLBP
CTR-nLBP
*
AP Shear
ML Shear
Compression
Peak Muscle Forces (N/kg)
24
12
0
Local
Global