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14. ABSTRACT

Low back pain (LBP) is an important secondary health condition following lower-extremity trauma, with an estimated prevalence as high as 52-89%, and reported as the condition most contributing to a reduced quality of life. During gait and activities of daily living, alterations in trunk motion following lower-extremity trauma likely impose distinct demands on trunk muscles to maintain equilibrium and stability of the spine that, with repeated exposure, may increase risk for LBP. The overall objective of this research was to characterize features of trunk (spine) motion with lower-extremity trauma, thereby elucidating the relationship(s) between trunk motion and LBP risk via changes in spine mechanics and spine health. Using a novel set of clinical, experimental, and computational methods, we have demonstrated that altered trunk motions with lower-extremity trauma increase spinal loads by 17-95% relative to uninjured individuals. Moreover, poor spine health/history of LBP was also related to greater self-reported functional limitations, and further moderated by psychosocial factors, thereby highlighting the interdependence of biopsychosocial factors in musculoskeletal pain following limb loss. Nevertheless, the positive association between elevated spinal loads and LBP support the need for trunk-specific rehabilitation procedures to reduce long-term incidence and recurrence of LBP.

15. SUBJECT TERMS

Low Back Pain; Intervertebral Disc; Inter-Segmental Motion; Spine Load; Finite Element Model; Biomechanics

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1. INTRODUCTION:

Linking lower-extremity trauma (i.e., amputation/injury) with low back pain (LBP) risk via biomechanical theory suggests that altered and asymmetric trunk motions and corresponding passive spinal tissue and trunk neuromuscular responses alter spine mechanics such that would, over time, adversely affect spine health. Therefore, the overall objective of this study was to investigate such relationships through cross-sectional evaluations of spine health and spine mechanics in persons with lower-extremity amputation/injury (with and without LBP) and uninjured controls. Traditional kinesiopathological models do not successfully describe initial diagnosis and subsequent treatment of chronic low back pain (LBP). As a result, support for a biopsychosocial model has increased, particularly in clinical populations with concurrent musculoskeletal disorders. However, biopsychosocial models are complex and dynamic, making them difficult to both research and implement in practice. Recently, the biopsychosocial model has been suggested as a means to elucidate the increased prevalence of chronic LBP among persons with lower limb loss. We therefore also explored the extent to which psychosocial factors mediated the LBP experience among persons with limb loss.

KEYWORDS: Low Back Pain; Intervertebral Disc; Inter-Segmental Motion; Spine Load; Finite Element Model

2. ACCOMPLISHMENTS:

What were the major goals of the project?

This study has three main aims, as indicated below:

Specific Aim 1: Quantify lumbar spinal alignment and inter-segmental vertebral motions with traumatic lower-extremity amputation.
Major Task 1: Obtain IRB and HRPO approvals.
Target Date: by April 2015
Actual Date: April 24, 2015 (IRB approval) / June 26, 2015 (HRPO approval)
Major Task 2: Complete biomechanical data collections, analysis, and interpretations.
Target Dates: Months 6-24 ***(~100% complete)***
Additional Milestones: One abstract presented and one manuscript submitted.

Specific Aim 2: Quantify alterations in spine mechanics (loading) with traumatic lower-extremity amputation.
Major Task 3: Estimate spinal loads using collected biomechanical data as inputs into the finite element model of the lumbar spine.
Target Dates: Months 6-24 ***(~100% complete)***
Additional Milestones: One abstract presented and one manuscript published.

Specific Aim 3: Determine associations between spine loading and current spine health with traumatic lower-extremity amputation.
Major Task 4: Conduct physical spinal examinations.
Target Dates: Months 6-24 ***(100% complete)***

Major Task 5: Obtain magnetic resonance images of the lumbar spine for quantitative evaluation of lumbar disc health.

Target Dates: Months 6-24 ***(75% complete- ongoing via retrospective chart reviews)***

Major Task 6: Author manuscript on entire study.

Target Dates: Months 30-36 ***(100% complete)***

Additional Milestones: One abstract presented and one manuscript submitted.

What was accomplished under these goals?

Prospective data collections included biomechanical, clinical, and self-reported assessments focused on the trunk and spine to identify potential relationships with low back pain risk factors following limb loss and extremity trauma. Biomechanical assessments involved instrumented movement analyses with a focus on kinematics of the trunk and spine, as well as trunk muscle activity recorded using surface EMG. In addition, we also captured a more comprehensive understanding of current/recent history of LBP and its impact on daily life and functional activities, including the NIH Task Force's LBP Questionnaire and a legacy LBP questionnaire (Oswestry Disability Index), as well as several psychosocial factors commonly associated with musculoskeletal pain and functional disability. The bullet points below highlight and summarize the most salient messages from our analyses to date, supplemented by the many original manuscripts/abstracts attached as appendices.

- The coordination / motions between the trunk and pelvis with vs. without LLA are associated with ~31-55, 41-83, and 3-14% larger external demands on the lower back in the sagittal, coronal, and transverse planes, respectively

- Joint contact forces within the spine are increased with LLA; notably, largest increases (up to ~65% relative to uninjured individuals) were found in joint compressive forces owing to a complex pattern and increased (6-80%) activation of trunk muscles

- Peak compressive, lateral, and anteroposterior shear loads generally increased with increasing walking speed. However, increases in compression and lateral shear with increasing walking speed were larger among the persons with vs. without LLA, particularly in lateral shear at the fastest speed. In contrast, peak anteroposterior shear decreased with increasing walking speed among persons with LLA.

- Evaluation of trunk muscle activities during gait identified differences in the motor control strategies underlying the observed trunk motion patterns. Specifically, persons with lower-extremity trauma demonstrated a second peak in erector spinae activation during mid-terminal swing (not observed in controls), and an overall longer duration of activation throughout the gait cycle (see Butowicz et al., 2018 in *Journal of Electromyography and Kinesiology*). Trunk neuromuscular control strategies secondary to lower-extremity trauma are seemingly driven by functional requirements to generate force proximally to help advance the (affected) lower limb during gait.

- Interestingly, spinal loads derived from our finite element simulations indicated differential increases with faster walking speeds among persons with vs. without lower-extremity trauma. At the fastest (vs. slowest) speed, increases in peak compressive and shear forces

were respectively 24-84% and 29-77% larger among persons with lower-extremity trauma vs. uninjured controls (see Hendershot et al., 2018 in *Journal of Biomechanics*). Over time, repeated exposures to these increased loads, particularly at faster walking speeds, may contribute to the elevated risk for LBP among persons with lower-extremity trauma.

- When evaluating the influences of LBP on spinal loads, despite larger motions in the frontal and transverse planes, spinal loads were similar between persons with lower-extremity trauma presenting both with and without (chronic) LBP; though these were generally still larger relative to uninjured controls (see Acasio et al., 2018 in *Proceedings of the American Society of Biomechanics*). Nevertheless, it is certainly plausible that the presence or history of LBP have concurrently altered features of trunk-pelvic motion, as previously observed among non-limb loss individuals with and without LBP.

- Preliminary (and prior) analyses using a legacy measure for LBP disability (Oswestry Disability Index; ODI) had identified minimal disability (43/58 reported less than 20% disability). However, categorization using the NIH Research Task Force (RTF) definitions for chronicity of LBP, which utilize both duration and frequency, told a different story (Table 1). Additional psychosocial outcomes and subcategories are also preliminarily reported below.

Table 1. Mean (standard deviation) classification/disability scores and individual psychosocial outcomes, by low back pain (LBP) status using NIH definition.

	No Current/Recent History of (chronic) LBP	(chronic) LBP
RTF Classification	10.6 (2.4)	16.9 (6.3)
ODI % Disability	2.0 (3.1)	22.8 (21.1)
Pain Intensity (7 days)	0.2 (0.1)	3.2 (1.5)
Pain Interference	1.1 (0.3)	3.4 (3.1)
Functional Impact	1.1 (0.3)	1.7 (0.7)
Anxiety and Depression	5.9 (5.2)	9.2 (7.0)
Pain Catastrophizing	2.5 (3.1)	22.8 (21.8)
Kinesiophobia	19.2 (3.6)	26.8 (4.3)

- Impairments in trunk postural control are evident among persons with LLA, both with and without LBP, relative to non-LLA controls (Table 2), as evidenced by increased confidence ellipse area ($t = -3.24$, $p = 0.004$), increased mean velocity ($t= -4.26$, $p=0.0004$), and increased mediolateral deviation in center of pressure ($t = -4.33$, $p = 0.0004$). These differences suggest LLA demonstrate less postural control when proprioceptive influence from the lower extremities is limited, particularly the intact limb.

Table 2. Mean (SD) trunk postural control variables between persons with LLA (with and without LBP), and uninjured controls, for reference.

	LBP	No Pain	Control	Effect Size (d)
95% Ellipse area (cm^2)	8.33 (5.24)	5.20 (3.12)	1.96 (1.36)	0.70
Mean velocity(cm/s)	2.07 (0.74)	1.66 (0.55)	1.02 (0.25)	0.61
RMS distance- ML (cm)	0.63 (0.25)	0.47 (0.14)	0.24 (0.09)	0.76
RMS distance- AP (cm)	0.66 (0.20)	0.56 (0.20)	0.42 (0.14)	0.50

- Additionally, to improve eventual clinical translation, we collected several clinic-based strength and endurance tests to supplement and connect the biomechanical and clinical evaluations. Briefly, these include hip abduction strength (isometric and eccentric), as well as bilateral hip bridge measurements. These metrics have been purported to play a more direct role in the altered trunk motions observed during gait and thus may be easily modifiable in future clinical efforts. For example, isometric hip abduction strength is smaller among persons with TTA and TFA (Figure 1).

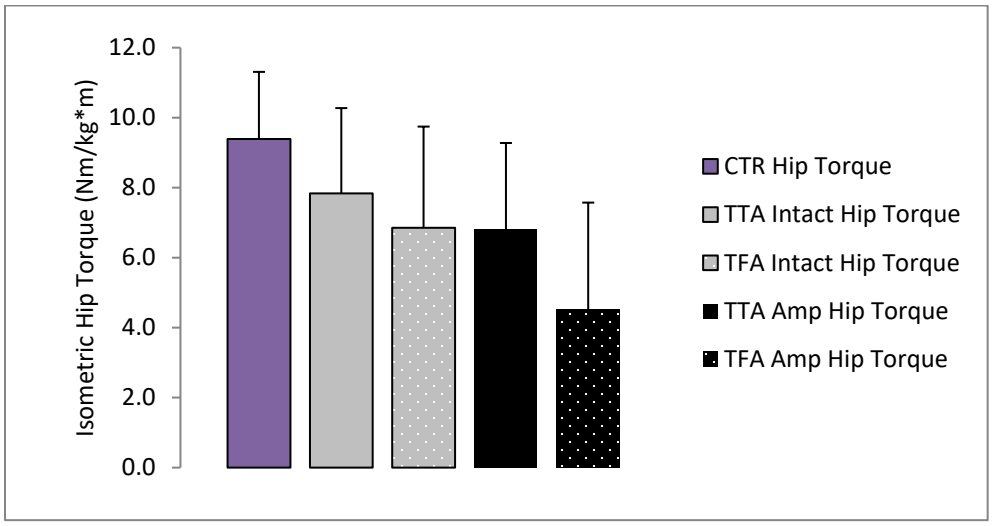

Figure 1. Isometric hip abduction strength.

What opportunities for training and professional development has the project provided?

Under the subaward to the University of Kentucky, Dr. Bazrgari and I provided mentorship to a PhD student, Iman Shojaei (now graduated). Beyond that, the project was not necessarily intended to provide training or professional development opportunities; however, the hiring of Dr. Butowicz as a post-doctoral researcher allowed additional training and mentorship opportunities as part of this project.

How were the results disseminated to communities of interest?

Throughout the reporting period, results were disseminated, thus far, via thirty-three knowledge products: *17 conference abstracts/presentations* and 16 *peer-reviewed scientific manuscripts* (See "Section 6: Products" for a list with citation details). The team also participated in 3 additional presentations wherein information was disseminated to the clinical and research communities

(Amputation System of Care Grand Rounds, State of the Science Symposium at USUHS, and AMSUS).

What do you plan to do during the next reporting period to accomplish the goals?

N/A (but there are several manuscripts under review that we expect to be published later this year).

4. **IMPACT:** Describe distinctive contributions, major accomplishments, innovations, successes, or any change in practice or behavior that has come about as a result of the project relative to:

What was the impact on the development of the principal discipline(s) of the project?

This study provides strong evidence suggesting the clinical/rehabilitation team should consider quality of trunk motion during walking to minimize future risk for low back pain. Moreover, psychosocial factors appear to augment/succeed biomechanical factors in the pathway(s) connecting lower-extremity amputation with low back pain, thereby expanding the comprehensive care model for this patient population in considering long-term health and quality of life.

What was the impact on other disciplines?

Nothing to report.

What was the impact on technology transfer?

Nothing to report.

What was the impact on society beyond science and technology?

Our results support a prevailing model that altered trunk (spinal) motions among persons with lower-extremity trauma increase risk for the onset and/or recurrence of LBP. As we continue building evidence for this model, there is likely to be a strong case for interventional approaches aimed at controlling trunk motions and spinal loads during (and beyond) rehabilitation. While that is specific to one patient population, these relationships may advance overall public knowledge regarding such a common and impactful musculoskeletal disorder. Over time, this will reduce the substantial economic costs associated with its treatment and promote enhancements in psychological health and overall quality of life.

5. **CHANGES/PROBLEMS:**

Changes in approach and reasons for change

N/A

Actual or anticipated problems or delays and actions or plans to resolve them

Nothing to report

Changes that had a significant impact on expenditures

Nothing to report.

Significant changes in use or care of human subjects

No significant changes to report. As noted above, IRB/HRPO approval dates:

IRB approval granted on April 1, 2015 (formal approval documents were uploaded to IRBnet on April 24)

HRPO approval for WRNMMC was granted on June 26, 2015 (A-18549.1)

HRPO approval for University Kentucky was granted on June 29, 2015 (A-18549.2)

Walter Reed IRB official start date (permission to begin study): August 4, 2015

Walter Reed IRB continuing review date: March 30, 2019 (just renewed until 2020 to complete data analyses).

6. PRODUCTS:

- **Publications, conference papers, and presentations**

  Journal publications:

1. Golyski, P.R., Hendershot, B.D. (2018) Trunk and Pelvic Dynamics during Transient Turns among Persons with Unilateral Lower Limb Amputation. *Human Movement Science* 58: 41-54. Federal Support Acknowledged.

2. Hendershot, B.D., Shojaei, I., Acasio, J.C., Dearth, C.L., Bazrgari, B. (2018) Walking Speed Differentially Alters Spinal Loads among Persons with Traumatic Lower Limb Amputation. *Journal of Biomechanics* 70(21): 249-254. Federal Support Acknowledged.

3. Acasio, J.C., Butowicz, C.M., Golyski, P.R., Nussbaum, M.A., and Hendershot, B.D. (2018) Associations between trunk postural control in walking and unstable sitting at various levels of task demand. *Journal of Biomechanics* 75: 181-185. Federal Support Acknowledged.

4. Butowicz, C.M., Acasio, J.A., Dearth, C.L., Hendershot, B.D. (2018) Trunk Muscle Activation Patterns among Persons with Lower Limb Loss: Influences of Walking Speed. *Journal of Electromyography and Kinesiology* 40: 48-55. Federal Support Acknowledged.

5. Highsmith, M.J., Goff, L.M., Lewandowski, A.L., Farrokhi, S., Hendershot, B.D., Hill, O.T., Rabago, C.A., Russell-Esposito, E., Orriola, J.J., Mayer, J.M. (2018) Low Back

Pain in Persons with Lower Extremity Amputation: A Systematic Review of the Literature. *The Spine Journal*, In Press. https://doi.org/10.1016/j.spinee.2018.08.011

6. Shojaei, I., Hendershot, B.D., Ballard, M., Acasio, J.C., Dearth, C.L., Bazrgari, B. Trunk Muscle Forces and Spinal Loads in Persons with Transfemoral Amputation during Sit-to-Stand and Stand-to-Sit Activities. *Clinical Biomechanics*, Under Review. Federal Support Acknowledged.

7. Butowicz, C.M., Krupenevich, R.L., Acasio, J.C., Dearth, C.L., Hendershot, B.D. Relationships between mediolateral trunk-pelvic motion, hip strength, and knee joint moments during gait among persons with lower limb loss. *Clinical Biomechanics*, Under Review. Federal Support Acknowledged.

8. Yoder, A., Silder, A., Farrokhi, S., Dearth, C.L., Hendershot, B.D. Lower Extremity Joint Contributions to Frontal Plane Trunk Dynamics in Persons with Transtibial Amputation. *Clinical Biomechanics*, Under Review. Federal Support Acknowledged.

9. Mahon, C.E., Butowicz, C.M., Dearth, C.L., Hendershot, B.D. Trunk-Pelvic Coordination with Lower-Limb Amputation: Longitudinal Changes in the First Year after Initial Ambulation. *Archives of Physical Medicine and Rehabilitation*, Under Review. Federal Support Acknowledged.

10. Butowicz, C.M., Dearth, C.L., and Hendershot, B.D. (2017) Impact of Traumatic Extremity Injuries beyond Acute Care: Implications for Resultant (Long-Term) Secondary Health Conditions. *Advances in Wound Care- Special Issue on Amputee Care and Rehabilitation* Federal Support Acknowledged.

11. Farrokhi, S, Mazzone, B, Schneider, M, Gombatto, S, Mayer, J, Highsmith, J, Hendershot, B. (2017) Biopsychosocial risk factors associated with chronic low back pain after lower limb amputation. *Medical Hypotheses* 108; 1-9.

12. Golyski, P.R. and Hendershot, B.D. Trunk-Pelvic Dynamics during Transient Turns in Persons with Unilateral Lower Limb Amputation. Human Movement Science, Under Revision. Federal Support Acknowledged.

13. Butowicz, C.M., Acasio, J.A., Hendershot, B.D. Trunk Neuromuscular Control Strategies among Persons with Lower Limb Amputation while Walking and Performing Concurrent Tasks. Gait and Posture, Under Review. Federal Support Acknowledged.

14. Hendershot, B.D., Acasio, J.A., Shojaie, I., Dearth, C.L., Bazrgari, B. Walking Speed Differentially Alters Spinal Loads in Persons with Traumatic Lower Limb Amputation. Journal of Biomechanics, Under Revision. Federal Support Acknowledged.

15. Shojaei, I, Arjmand, N, Meakin, J, Bazrgari, B (2017) A model-based approach for estimation of changes in lumbar segmental kinematics associated with alterations in trunk muscle forces. *Journal of Biomechanics* Federal Support Acknowledged.

Books or other non-periodical, one-time publications.

Pasquina, P.F., Hendershot, B.D., and Isaacson, B.M. (2016) Secondary Health Effects of Amputation (Chapter 24) Atlas of Amputations and Limb Deficiencies: Surgical, Prosthetic, and Rehabilitation Principles, 4th Edition. American Academy of Orthopaedic Surgeons: Rosemont, IL.

Other publications, conference papers, and presentations.

1. Dearth, C.L., Eskridge, S, Farrokhi, S., Hendershot, B.D., Russell Esposito, E. Living with Extremity Trauma and Limb Loss for a Lifetime: A Review of Efforts to Identify and Mitigate Risk Factors for Secondary Health Conditions.

2. Hendershot, B.D., Butowicz, C.B., Mahon, C.E., Schnall, B.L., Dearth, C.L. Longitudinal Changes in Mediolateral Trunk and Pelvic Motion Among Persons with Lower Limb Amputation during the First Year of Ambulation. 2017 Meeting of the American Society of Biomechanics (ASB), Boulder, CO.

3. Yoder, A.J., Farrokhi, S., Dearth, C.L., Hendershot, B.D. Lower Extremity Joint Contributions to Trunk Dynamics in Persons with Lower Extremity Amputation. 2017 Meeting of the American Society of Biomechanics (ASB), Boulder, CO.

4. Golyski, P.R., and Hendershot, B.D. Trunk-Pelvic Dynamics during Transient Turns in Persons with Unilateral Lower Limb Amputation. 2017 Military Health System Research Symposium (MHSRS), Kissimmee, FL, USA.

5. Butowicz, C.M., Acasio, J.C., and Hendershot, B.D. Trunk Neuromuscular Control Strategies among Persons with Lower Limb Amputation while Walking and Performing Concurrent Tasks. 2017 Meeting of the American Society of Biomechanics (ASB), Boulder, CO.

6. Butowicz, C.M., Acasio, J.C., Dearth, C.L., Hendershot, B.D. Trunk Muscle Activation Patterns during Walking among Persons with Lower Limb Loss. *World Congress of Biomechanics* (WCB), Dublin, Ireland. Federal Support Acknowledged.

7. Butowicz, C.M., Krupenevich, R.L., Acasio, J.C., Hendershot, B.D. Relationships among Trunk-Pelvic Motions, Hip Strength, and Knee Joint Moments during Gait among Persons with Lower Limb Loss. *World Congress of Biomechanics* (WCB), Dublin, Ireland. Federal Support Acknowledged.

8. Mazzone, B., Farrokhi, S., Hendershot B.D., Watrous, J.R., McCabe, C.T. (2018) Prevalence and Relationship of Low Back Pain and Psychosocial Factors after Lower Limb Amputation among Wounded Warrior Recovery Project Participants. *Military Health System Research Symposium* (MHSRS), Kissimmee, FL, USA. Federal Support Acknowledged.

9. Acasio, J.C., Butowicz, C.B., Dearth, C.L., Shojaei, I., Bazrgari, B., Hendershot, B.D. (2018) Trunk Muscle Activations, Motions, and Spinal Loads among Persons with Lower Limb Amputation: Influences of Chronic Low Back Pain. *Military Health System Research Symposium* (MHSRS), Kissimmee, FL, USA. Federal Support Acknowledged.

10. Acasio, J.C., Butowicz, C.B., Dearth, C.L., Shojaei, I., Bazrgari, B., Hendershot, B.D. (2018) Trunk Muscle Forces and Spinal Loads while Walking in Persons with Lower Limb Amputation both with and without Chronic Low Back Pain. *American Society of Biomechanics* (ASB), Rochester, MN, USA. Federal Support Acknowledged.

11. Acasio, J.C., Butowicz, C.B., Hendershot, B.D. (2018) Patterns of Erector Spinae Activation and Trunk-Pelvis Kinematics in Persons with Lower Limb Amputation: Influences of Low Back Pain. *American Society of Biomechanics* (ASB), Rochester, MN, USA. Federal Support Acknowledged.

12. Butowicz, C.B., Krupenevich, R.L., Acasio, J.C., Hendershot, B.D. (2018) Influences of Low Back Pain on the Energy Contributions of the Hip and Spine during Gait among Persons with Lower Limb Loss. *American Society of Biomechanics* (ASB), Rochester, MN, USA. Federal Support Acknowledged.

13. Hendershot, B.D., Butowicz, C.B., Krupenevich, R.L., Acasio, J.A., Pruziner, A.L., Miller, R.H., Goldman, S.G., Dearth, C.L. (2018) Toward Optimizing Long-Term Health after Limb Loss: Comprehensive Evaluations of Secondary Health Conditions. 10^{th} *Annual Joint National Capital Region Research Competition*, Bethesda, MD, USA. Federal Support Acknowledged.

14. Shojaei, I., Hendershot, B.D., Ballard, M., Acasio, J.C., Dearth, C.L., Bazrgari, B. (2018) Trunk Muscle Forces and Spinal Loads during Sit-to-Stand and Stand-to-Sit Activities: Differences between Persons with and without Unilateral Transfemoral Amputation. *15^{th} International Symposium on Computer Methods in Biomechanics and Biomedical Engineering* (CMBBE), Lisbon, Portugal. Federal Support Acknowledged.

15. Hendershot, B.D. (2016) Biomechanical risk factors for low back pain with extremity trauma. *The Military Health System Research Symposium (MHSRS), Kissimmee, FL, USA.*

16. Hendershot, B.D., Shojaei, I., and Bazrgari, B. Faster walking speeds differentially alter spinal loads among persons with traumatic lower limb amputation. *2^{nd} International Workshop on Spine Loading and Deformation.* Julius Wolff Institute, Berlin, Germany. May 18-20, 2017.

- **Website(s) or other Internet site(s)**

  Nothing to report.

- **Technologies or techniques**

Nothing to report.

- **Inventions, patent applications, and/or licenses**

  Nothing to report.

- **Other Products**

  Nothing to report.

7. PARTICIPANTS & OTHER COLLABORATING ORGANIZATIONS

What individuals have worked on the project?

Name:	Bradford Hendershot, PhD
Project Role:	Principal Investigator, EACE/WRNMMC
Nearest person month worked:	2
Contribution to Project:	Provided overall project direction, including: tracking resources, ensuring regulatory compliance, coordinating data collections / analyses, and generating reports.
Funding Support:	Federal Employee

Name:	Babak Bazrgari, PhD
Project Role:	Co-Investigator, Site PI at University of Kentucky
Nearest person month worked:	1
Contribution to Project:	Led the finite element modeling for all biomechanical data
Funding Support:	

Name:	Courtney Butowicz, PhD
Project Role:	Post-Doctoral Researcher, HJF/WRNMMC
Nearest person month worked:	12
Contribution to Project:	Led data collection, analysis, and interpretation with direction from the study PI.
Funding Support:	

Name:	Julian Acasio, MS
Project Role:	Research Engineer, HJF/WRNMMC
Nearest person month worked:	10
Contribution to Project:	Assisted with data collection, analysis, and interpretation

Funding Support:	

Has there been a change in the active other support of the PD/PI(s) or senior/key personnel since the last reporting period?

Nothing to report.

What other organizations were involved as partners?

Nothing to report.

8. SPECIAL REPORTING REQUIREMENTS

COLLABORATIVE AWARDS: N/A

QUAD CHARTS: N/A

9. APPENDICES: N/A

Increased and asymmetric trunk motion during level-ground walking is associated with larger spinal loads in persons with unilateral transfemoral amputation

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ABSTRACT

Background: During gait, alterations in trunk motion following lower limb amputation likely impose distinct demands on trunk muscles to maintain equilibrium and stability of the spine. However, trunk muscle responses to such changes in physical demands, and the resultant effects on spinal loads, have yet to be determined in this population.

Methods: Trunk and pelvic kinematics collected during level-ground walking from 40 males (20 with unilateral transfemoral amputation and 20 matched controls) were used as inputs to a kinematics-driven, nonlinear finite element model of the lower back to estimate forces in 10 global (attached to thorax) and 46 local (attached to lumbar vertebrae) trunk muscles, as well as compression, lateral, and antero-posterior shear forces at all spinal levels.

Findings: Trunk muscle force and spinal load maxima corresponded with heel strike and toe-off events, and were respectively 10-40% and 17-95% larger during intact vs. prosthetic stance in persons with amputation, as well as 6-80% and 26-60% larger during intact stance relative to controls.

Interpretation: In addition to larger individual muscle responses to overall increases and asymmetries in trunk motion during walking, co-activations of antagonistic muscles were needed to assure spine equilibrium in three-dimensional space, hence resulting in substantial increases in spinal loads. Knowledge of trunk neuromuscular adaptations to changes in task demands following amputation could inform rehabilitation procedures such to reduce long-term incidence or recurrence of low back pain.

Keywords: Amputation, Gait, Muscle forces, Spinal loads, Low back pain

HIGHLIGHTS:

- Persons with lower limb amputation walk with large and asymmetric trunk motion
- Spinal equilibrium and stability under such motions require large muscular response
- Larger trunk muscle forces contribute to increase compression and shear loads
- Repeated exposures to altered spinal loading may elevate low back pain risk

1. INTRODUCTION

The prevalence of low back pain (LBP) is considerably higher in persons with lower limb amputation (LLA) compared with able-bodied individuals (Friberg, 1984, Sherman, 1989, Sherman et al., 1997, Smith et al., 1999). As a secondary health-related concern, LBP is suggested to be the most important condition that adversely affects the physical performance and quality of life in persons with LLA (Ehde et al., 2001, Taghipour et al., 2009). Providing the projected increase in the number of people with LLA, it is important to investigate the underlying mechanism(s) responsible for the elevated prevalence of LBP in this cohort (Reiber et al., 2010, Devan et al., 2014).

Considering spine biomechanics, spinal loads are the resultant of interactions between internal tissue forces (primarily from muscles) and physical demands of a given activity on the lower back (Cholewicki and Mcgill, 1996, Calisse et al., 1999, Arjmand and Shirazi-Adl, 2005, Adams et al., 2007, Mcgill et al., 2014). During gait, increased and asymmetric trunk motion following LLA has been reported to impose higher physical demands on the lower back (Cappozzo and Gazzani, 1982, Hendershot and Wolf, 2014). Such an increase in physical demand of a common daily activity like walking would require larger responses from internal trunk tissues to assure equilibrium and stability of the spine, hence leading to larger spinal loads that would presumably increase the risk for LBP due to the repetitive nature of such activities (Adams et al., 2007).

There is limited information in the literature related to internal trunk tissue responses and resultant spinal loads during walking (Cappozzo et al., 1982, Cappozzo, 1983, Cappozzo, 1983, Khoo et al., 1995, Cheng et al., 1998, Callaghan et al., 1999, Yoder et al., 2015). All but two of these few earlier studies included relatively small sample sizes of able-bodied male participants and have reported spinal loads at either the L4-L5 or L5-S1 discs. The predicted pattern of spinal loads in these studies included symmetric local maxima occurring around heel strike and toe off within the gait cycle, with values ranging between 1.2 to 3.0 times body weight. The other two studies regarding internal tissue responses and resultant spinal loads during walking also include persons with LLA (Cappozzo and Gazzani, 1982, Yoder et al., 2015). Using kinematics data obtained from two subjects (one with transfemoral amputation and one with knee

ankylosis), Cappozzo and Gazzani (1982) used a rigid link-segment model of the whole body to obtain mechanical demands of walking on the lower back. A simple muscle model was then used to calculate internal tissue responses and the resultant spinal loads. Contrary to the patterns of spinal loads observed in able-bodied individuals, the occurrence of local maxima among persons with LLA did not have a symmetric pattern. Rather, the maximum compression forces were larger at the instance of prosthetic vs. intact toe off (2-3.0 vs. 1.0 times body weight). Similar differences in patterns of trunk muscular responses during walking, and the resultant effect on spinal loads (but at much lower magnitudes), between persons with and without transtibial LLA have been recently reported by Yoder et al. (2015). Although these earlier studies highlight the impact of altered and asymmetric gait on loads experienced in the lower back, they were limited to small samples and/or a very simple biomechanical model of the lower back.

Using a relatively large sample size, along with a biomechanical model of the lower back with more bio-fidelity, the objective of this study was to investigate the differences in internal tissue responses, specifically muscle forces, and resultant spinal loads during level-ground walking between individuals with (n=20) and without (n=20) unilateral LLA. Considering that alterations in trunk motion following amputation impose higher (and asymmetric) physical demands on the lower back (Cappozzo and Gazzani, 1982, Hendershot and Wolf, 2014), it was hypothesized that compared to able-bodied individuals, persons with LLA will require larger muscle forces in the lower back to overcome the physical demands of walking while maintaining spinal stability and equilibrium. Such increases in trunk muscle forces would, in turn, result in larger spinal loads. A better knowledge of lower back biomechanics (i.e., in terms of spinal loads) among individuals with LLA can inform future development of effective clinical programs aimed at modifying lower back biomechanics such to mitigate LBP risk.

2. METHODS

2.1 Experimental study: Kinematic data collected in an earlier study were used in these analyses (Hendershot and Wolf, 2014). Briefly, full-body kinematics from 20 males with transfemoral amputation and 20 male able-bodied controls were collected using a 23-camera motion capture system during level-ground walking across a 15 m level walkway at a self-

selected speed (mean ≈ 1.35 m/s; Table-1). Here, kinematic data of interest included three-dimensional pelvic and thorax motions that were collected by tracking markers positioned in the mid-sagittal plane over the S1, T10, and C7 spinous processes, sternal notch, and xiphoid; and bilaterally over the acromion, ASIS, and PSIS. All amputations were a consequence of traumatic injuries with a mean (standard deviation) duration of 3.1 (1.4) years since amputation. Main inclusion criteria were: (1) unilateral transfemoral amputation with no contralateral functional impairments, (2) daily use of a prosthetic device (≥1 year post-amputation), (3) no use of an upper-extremity assistive device (e.g., cane, crutches, walker), and (4) having no other musculoskeletal or neurologic problem, except amputation, that may affect gait results. Details of inclusion and exclusion criteria and other experimental methodology can be found in Hendershot and Wolf (2014). This retrospective study was approved by Institutional Review Boards of both University of Kentucky and Walter Reed National Military Medical Center.

Table-1 may be inserted here

2.2 Modeling study: The biomechanical model used to estimate trunk muscle responses and resultant spinal loads included a non-linear finite element (FE) model of the spine that estimated the required muscle forces to complete the activity using an optimization-based iterative procedure (Arjmand and Shirazi-Adl, 2005, Arjmand and Shirazi-Adl, 2006, Bazrgari et al., 2007, Bazrgari et al., 2008, Bazrgari et al., 2009, Arjmand et al., 2010). In this model, muscle forces are estimated such that equilibrium equations are satisfied across the entire lumbar spine. The finite element model included a sagittally symmetric thorax-pelvis model of the spine composed of six non-linear flexible beam elements and six rigid elements (Figure 1) (Arjmand and Shirazi-Adl, 2005, Bazrgari et al., 2008). The six rigid elements represented the thorax, and each of lumbar vertebrae from L1 to L5, while the six flexible beam elements characterized the nonlinear stiffness of each intervertebral disc between the T12 and S1 vertebrae. Intervertebral discs' stiffness were defined using nonlinear axial compression–strain relationships along with moment–rotation relationships in sagittal/coronal/transverse planes that were obtained from earlier numerical and experimental studies of lumbar spine motion segments (Yamamoto et al., 1989, Oxland et al., 1992, Shirazi-Adl et al., 2002). Upper-body mass and mass moments of inertia were distributed along the spine according to reported ratios (Zatsiorsky and Seluyanov, 1983, De Leva, 1996, Pearsall et al., 1996). Inter-segmental damping with properties defined based on earlier experimental studies were also considered using connector elements (Markolf,

1970, Kasra et al., 1992). The muscle architecture in the biomechanical model included 56 muscles (Fig. 1); 46 muscles connecting lumbar vertebrae to the pelvis (i.e., local muscles) and 10 muscles connecting thoracic spine/rib cage to the pelvis (i.e., global muscles) (Arjmand and Shirazi-Adl, 2005, Arjmand and Shirazi-Adl, 2006, Bazrgari et al., 2008, Bazrgari et al., 2008).

Figure 1 may be inserted here

To determine the required muscle forces for satisfaction of equilibrium across the entire lumbar spine, segmental kinematics in the lumbar region were required. Since only kinematics of the thorax and the pelvis were available from the experimental measurements, a heuristic optimization procedure (Figure 2) was used in the biomechanical model to determine a set of segmental kinematics in the lumbar region (i.e., from L1 to L5) such that the corresponding set of predicted muscles forces minimized a cost function (Shojaei and Bazrgari, 2014). The cost function used for this heuristic optimization procedure was the sum of squared muscle stress across all lower back muscles. Specifically, a set of possible segmental kinematics in the lumbar region that was within the reported range of motion of lumbar motion segments was initially prescribed on the FE model and the equations of motion were solved using an implicit integration algorithm inside an FE software (ABAQUS, Version 6.13, Dassault Systemes Simulia, Providence, RI). The outputs of equations of motion were three-dimensional moments at each spinal level, from T12 to L5, that were to be balanced by muscles attached to these same spinal levels. Because the number of attached muscles to these levels (i.e., 10 muscles in each level from T12 to the L4 and 6 muscles at L5) was more than the number of equilibrium equations (i.e., three at each vertebra), a local optimization problem was also solved for each level to obtain a set of muscle forces that minimize the aforementioned cost function only at that specific level (Arjmand and Shirazi-Adl, 2006). These local optimization procedures were performed using the Lagrange Multiplier Method. The above procedure was repeated inside the heuristic optimization for as many possible sets of segmental kinematics, determined using a genetic algorithm, until a set of segmental kinematics was obtained that meets the optimization criterion. The associated muscle forces with the optimal local kinematics were then used to estimate spinal loads at all lumbar levels. These spinal loads included compression forces, along with anterior-posterior and medio-lateral components of the shear forces, relative to the mid-plane of the intervertebral disc and at each lumbar level. The heuristic optimization procedure was developed in Matlab (The MathWorks Inc., Natick, MA, USA, version 7.13).

Figure 2 may be inserted here

2.3 Statistical analyses: Rather than comparing the predicted forces in all 56 muscles between the two groups, the summation of forces in global and local muscles were separately used for statistical analyses. Similarly, rather than comparing spinal loads at each level, levels with highest spinal loads (i.e., L4-L5 or L5-S1 for compression forces and L5-S1 for shear forces) were considered for subsequent statistical analyses. For each outcome measure, local maxima were extracted from the stance phase of each leg, resulting in the following values: 1) two peaks in the predicted global and local muscle forces (Fig. 3; Peak-1 at heel strike of the ipsilateral limb and Peak-2 at toe off the contralateral limb), 2) two peaks in the predicted compression forces (Fig. 4; Peak-1 at heel strike of the ipsilateral limb and Peak-2 at toe off the contralateral limb), and 3) one peak in each of the lateral (Fig. 5; at toe off of the contralateral limb), anterior (Fig. 5; at toe off of the contralateral limb), and posterior shear forces (Fig. 5; at heel strike of the ipsilateral limb). It is of note that the gait cycle was defined from right heel strike to subsequent right heel strike for controls, and from heel strike of the intact leg to next heel strike of the intact leg for persons with LLA. Prior to statistical analyses, all maxima were normalized with respect to total body mass. Furthermore, because there was no significant differences ($P>0.21$ from paired t-tests) in any of the aforementioned maxima between the right and left legs of controls, statistical analyses were performed using the mean values for the two legs of control group.

3. RESULTS

Mean sum of global and local muscle forces for both groups are depicted in Figure 3. Mean sum of maximum global muscle forces was 2.6 N/kg larger at heel strike of the intact vs. prosthetic limb among persons with LLA (Table 2); the sum of global muscle forces was only significantly larger at intact heel strike in persons with LLA than the corresponding value in controls. For local muscles at the instant of heel strike, there were no significant differences ($P>0.41$) within and between groups. At toe-off, the mean sum of maximum global muscle forces was 3.6 N/kg larger in intact vs. prosthetic limb stance among persons with LLA; this local maximum was also 5.6 N/kg larger in intact stance among persons with LLA than controls, but not significantly

different between prosthetic stance relative to controls. For local muscles at the instant of toe-off, while there were no significant differences between the values in the stance phase of intact and prosthetic legs of persons with LLA, they were, respectively, 2.5 N/kg and 1.5 N/kg larger than the corresponding values in controls.

Figure 3 may be inserted here

Table-2 may be inserted here

Mean compression forces were 3.4 N/kg larger at heel strike of the intact vs. prosthetic leg among persons with LLA; the compression force at heel strike of the intact leg was also 4.8 N/kg larger than the corresponding value in controls, while there were no significant differences between the prosthetic leg of persons with LLA and the corresponding value in controls (Table 2). Mean compression force at toe off of the contralateral limb was similar between stance of the intact and prosthetic legs among persons with LLA, but were 8.6 N/kg (4.7 N/kg) larger during intact (prosthetic) leg stance than the corresponding value in controls.

Figure 4 may be inserted here

In the lateral direction, maximum shear forces were 4.3 N/kg larger in the stance phase of the intact vs. prosthetic leg among persons with LLA (Table 2). These were also 3.3 N/kg larger in the stance phase of intact leg of persons with LLA than the corresponding value in controls; there were no significant differences between the stance phase of prosthetic leg and that of controls. In the posterior direction, maximum shear forces among controls were 1.3 and 1.8 N/kg larger than the corresponding values in intact and prosthetic stance among persons with LLA, respectively. Maximum posterior shear forces were not different between intact and prosthetic stance among persons with LLA. In the anterior direction, maximum shear forces were 1.4 N/kg larger in the stance phase of the intact vs. prosthetic leg among persons with LLA; these were also 1.8 N/kg larger in the stance phase of the intact leg than the corresponding value in controls.

Figure 5 may be inserted here

4. DISCUSSION

In this study, trunk muscle responses to walking demands and the resultant spinal loads were estimated in individuals with and without unilateral LLA. It was hypothesized that individuals with LLA would require larger muscle forces to overcome the physical demands of walking while maintaining spinal equilibrium and stability, which would in turn result in larger spinal loads compared to individuals without amputation. The results obtained through computational simulations and subsequent statistical analyses confirmed our hypothesis. Higher trunk muscle forces and larger spinal loads on the lower back of individuals with unilateral LLA during walking may be in part responsible for the reported higher prevalence of LBP among persons with vs. without LLA.

The local maxima for muscle forces and the resultant spinal loads occurred at the instants of heel strike and toe off within the gait cycle. These time points also happen to correspond with the instances of large axial twist of the trunk (i.e., heel strike) and asymmetric trunk posture (i.e. toe off where there were relatively large motions in all three planes (Hendershot and Wolf, 2014)). In addition to individual muscle responses, co-activations of antagonistic muscles were needed under such trunk motions to assure spine equilibrium in three-dimensional space. The effects of such an increased and asymmetric motion on muscle forces is more evident when comparing the kinematics and associated muscle forces in the stance phase of intact and prosthetic legs among individuals with LLA. The increases in trunk motion and its asymmetry at instances of heel strike and toe off were more pronounced during the stance phase of the intact leg of persons with LLA, particularly at heel strike of the ipsilateral limb (Hendershot and Wolf, 2014), that resulted in much larger muscle forces during the stance phase of intact than prosthetic leg. Such an effect may also be a result of proximal compensations (e.g., hip-hiking) to assist with toe clearance (Michaud et al., 2000), or simply because these individuals feel more confident during intact (vs. prosthetic) stance to advance their center of mass.

The sum of forces in global muscles during the gait cycle was comparable with the sum of forces in the local muscles (Fig. 3). It should be mentioned, however, global muscles were the primary responders to activity demands during the first iteration of muscle force calculations in

our model (i.e., the local loop in Fig. 2). As the effects of such global muscle forces were applied into the model, during the subsequent iterations, local muscles became activated to prevent buckling of the spine under the penalties of global muscle forces. If the summation of forces in global and local muscles is assumed to represent the required energy for respectively equilibrate and stabilize the spine, our results suggest that relatively equal amounts of energy were consumed to provide equilibrium and stability to the spine during walking. However with such an assumption, it seems that overcoming the equilibrium demands of walking impact the spinal loads of individuals with LLA more than overcoming its segmental stability demands when compared with able-bodied individuals. This observation is reflected in the sum of differences in mean global muscle forces (i.e., assumed to represent differences in equilibrium demands) between persons with and without LLA that was 955 N larger than the sum of differences in mean local muscle forces (i.e., assumed to represent differences in stability demands) between the same two groups. We should, however, emphasize that such interpretation is limited to assumptions made in our optimization-based method for estimation of muscle responses to activity demand and would require verification via measurement of muscle activity. A stabilizing response from local muscles as suggested here should occur sooner than equilibrating response from global muscles.

The predicted spinal loads for controls were in agreement (in terms of patterns and magnitudes) with those obtained in earlier studies (Cappozzo, 1983, Khoo et al., 1995, Cheng et al., 1998, Callaghan et al., 1999, Yoder et al., 2015). Depending on walking speed, the reported values of maximum compression force at the lower spinal level ranged between 1.0 to 2.95 times body weight for walking speeds ranging from 0.9 to 2.2 m/s (Table 3). The mean maximum compression force from these studies, along with average walking speed, were respectively ~ 1.94 times body weight at 1.4 m/s, which are comparable with our predictions of a maximum spinal load of ~ 1.85 times body weight for an average walking speed of ~1.35 m/s. Maxima in predicted compression forces in this study occurred around heel strike and toe off instances within the gait cycle, which are also consistent with reported timing of maximum compression forces in earlier studies: around toe off instants (Callaghan et al., 1999), within a short time interval around toe off (Cappozzo, 1983), right after the heel strike and before complete toe off (Cheng et al., 1998), and around 20% and 80% of walking cycle (Khoo et al., 1995).

Table-3 may be inserted here

The results obtained from individuals with unilateral LLA in this study were also consistent in pattern and magnitude with those reported by Cappozzo and Gazzani (Cappozzo and Gazzani, 1982). This earlier study reported spinal loads for two subjects (i.e., one with transfemoral amputation and one with knee ankylosis) during level-ground walking. The reported maxima of predicted compression forces for the person with LLA) ranged from 2 to 3 times body weight for walking speeds between 1.0 m/s and 1.5 m/s (Table 3), which is consistent with the range of maxima of predicted compression forces in this study (~ 2 to 2.6 body weight). In both studies, the maximum compression forces occurred during intact limb stance at the instance of prosthetic toe off. In a more recent study (Yoder et al 2015), much smaller maxima (i.e., ~ body weight) have been reported for maximum spinal loads among persons with transtibial LLA; though smaller maxima could be due, in part, to the relatively slower walking speed and/or more distal amputation.

The sample of persons with LLA in this study included young and physically fit members of the military with transfemoral amputations resulting from traumatic injuries. Thus, the results cannot be generalized to groups with other levels or etiologies of amputation. This cross sectional study also does not provide any information about lower back biomechanics in these individuals before the amputations, and history of LBP was not controlled in the participants, though those with current LBP were excluded from the study. Although we accounted for individual differences in trunk inertial properties in the non-linear FE model of spine, we used the same passive tissue properties for all subjects since we had no access to the subject-specific behavior of such tissues (i.e., ligaments, intervertebral discs, passive behavior of muscles and bony structures) for these participants. Furthermore, same heights were considered in the spine model for all subjects, though stature was not significantly different between groups.

5. CONCLUSION

Asymmetric and larger trunk motion of individuals with LLA during walking requires higher activation and co-activation of trunk muscles to assure equilibrium and stability of the spine, which in turn increase spinal loads. An elevated level of spinal loads during a basic activity of

daily living like walking may increase risk of developing LBP, in particular due to the repetitive nature of such activity. It is imperative to investigate whether those with LLA consistently experiencing higher levels of spinal loads during other important activities of daily living (e.g., ascending and descending ramps or stairs) as a result of an alteration in internal tissue responses to activity demands. Such knowledge can inform future development of effective clinical programs aimed at reducing the risk for developing LBP via management of spinal loads during daily activities.

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TABLE AND FIGURE CAPTIONS

Table-1: Participant characteristics for the control (CTL) and lower limb amputation (LLA) groups. (Hendershot and Wolf, 2014).

Table-2: Mean (SD) predicted maximum muscle forces and resultant spinal loads.

Table-3: Reported values of maximum compression force (*body weight) at the lower spinal level.

Figure 1. Sagittal view of the biomechanical model including FE model of the spine and 56 trunk muscles (dimensions in mm). ICPL: iliocostalislumborum pars lumborum, ICPT: iliocostalislumbroum pars thoracis, IP: iliopsoas, LGPL: longissimusthoracis pars lumborum, LGPT: longissimusthoracis pars thoracis, MF: multifidus, QL: quadratuslumborum, IO: internal oblique, EO: external oblique and RA: rectus abdominus.

Figure 2. The process used to estimate muscle forces and spinal loads. Each set of possible segmental kinematics is generated using a genetic algorithm subjected to measured kinematics of thorax and pelvis as well as the reported values of lumbar segments' range of motion. The convergence in the local and global loops are achieved when the changes in respectively sum of predicted muscle forces in two consecutive local iterations and the value of the cost function of the heuristic optimization procedure in two consecutive global iterations are less than 1%.

Figure 3. Mean sum of forces in global (i.e., muscles attached to the thoracic spine – top) and local (i.e., muscles attached to the lumbar spine – bottom) muscles. CTL: control group, LLA: group with lower limb amputation.

Figure 4. Mean compression forces at mid-plane of the L4-L5 (top) and L5-S1 (bottom) intervertebral discs. CTL: control group, LLA: group with lower limb amputation.

Figure 5. Mean shear forces at the mid-plane of the L5-S1 in lateral (top) and antero-posterior (bottom) directions. CTL: control group, LLA: group with lower limb amputation. Positive shear force in lateral direction indicates force toward the right (intact) leg for controls (LLA) and positive shear force in antero-posterior direction indicate anterior direction.

Table-1: Participant characteristics for the control (CTL) and lower limb amputation (LLA) groups. (Hendershot and Wolf, 2014).

Variable	CTL (n=20)	LLA (n=20)
Age (year)	28.1 (4.8)	29.20 (6.70)
Stature (cm)	181.00 (6.10)	176.20 (6.70)
Body mass (kg)	83.90 (8.60)	80.60 (12.20)

Table-2: Mean (SD) predicted maximum muscle forces and resultant spinal loads.

Variable	Control (n=20)	Transfemoral Amputation (n=20)	
		Intact Stance	Prosthetic Stance
MUSCLE FORCES			
Global (thorax) – Peak 1 (N/kg)	7.7 (2.5)	10.4 (5.0) *	7.8 (3.0) †
Global (thorax) – Peak 2 (N/kg)	7.0 (2.6)	12.6 (5.2) *	9.0 (4.1) †
Local (lumbar) – Peak 1 (N/kg)	8.4 (2.0)	8.9 (2.1)	8.1 (1.7)
Local (lumbar) – Peak 2 (N/kg)	7.8 (1.4)	10.3 (3.1) *	9.3 (2.3) *
SPINAL LOADS			
Compression – Peak 1 (N/kg)	18.2 (3.4)	23.0 (5.8) *	19.6 (4.1) †
Compression – Peak 2 (N/kg)	16.8 (3.3)	25.4 (7.0) *	21.5 (4.8) *
Lateral Shear (N/kg)	5.5 (1.1)	8.8 (1.6) *†	4.5 (1.2)
Posterior Shear (N/kg)	3.7 (0.8)	2.4 (0.8) *	1.9 (0.6) *
Anterior Shear (N/kg)	4.2 (1.0)	6.0 (1.1) *	4.6 (0.9) †

* Significant difference relative to control

† Significant difference between intact vs. prosthetic

Table-3: Reported values of maximum compression force (*body weight) at the lower spinal level.

Study		Walking Speed (m/s)						
		0.90	1.00	1.20	1.35	1.50	1.70	2.20
Typical walking	Current study				1.85			
	Cappozzo, 1983		1.20		1.50		1.90	2.50
	Cheng et al., 1998		2.28	2.53		2.95		
	Khoo et al., 1995			1.71				
	Yoder et al., 2015	1.0						
Atypical walking	Current study				2.60			
	(Cappozzo and Gazzani, 1982) (amputation)		2.00		2.70	3.00		
	(Cappozzo and Gazzani, 1982) (knee ankylosis)		1.80			2.10		
	Yoder et al., 2015	1.0						

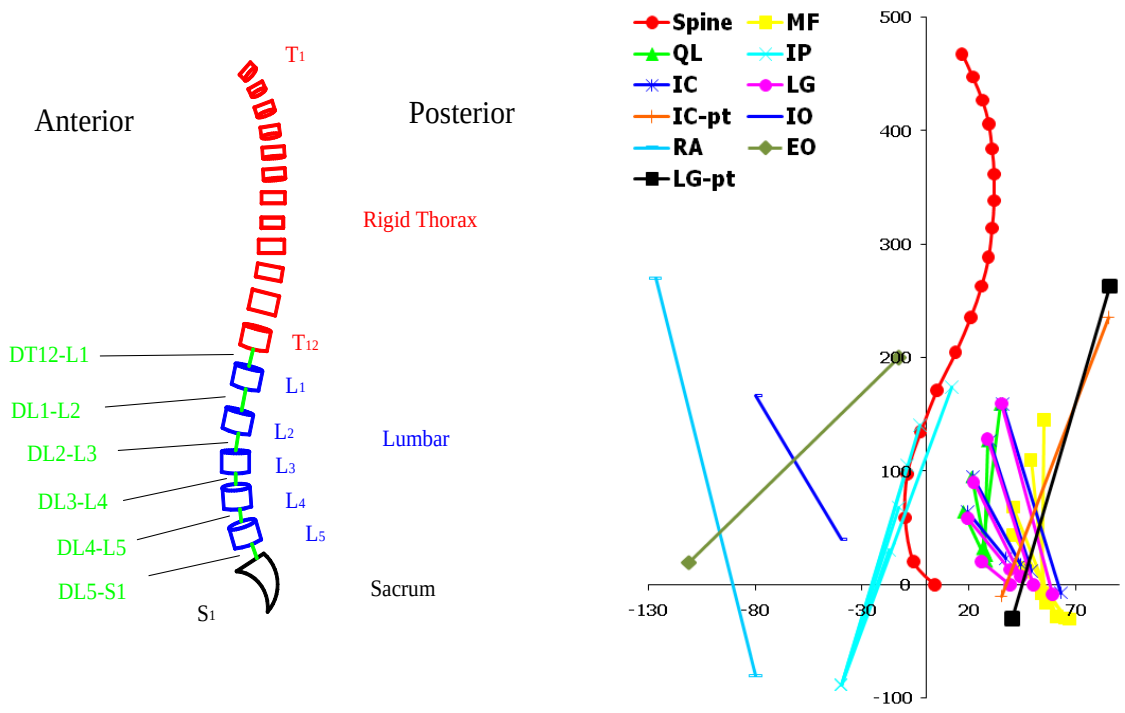

Figure 1. Sagittal view of the biomechanical model including FE model of the spine and 56 trunk muscles (dimensions in mm). ICPL: iliocostalislumborum pars lumborum, ICPT: iliocostalislumbroum pars thoracis, IP: iliopsoas, LGPL: longissimusthoracis pars lumborum, LGPT: longissimusthoracis pars thoracis, MF: multifidus, QL: quadratuslumborum, IO: internal oblique, EO: external oblique and RA: rectus abdominus.

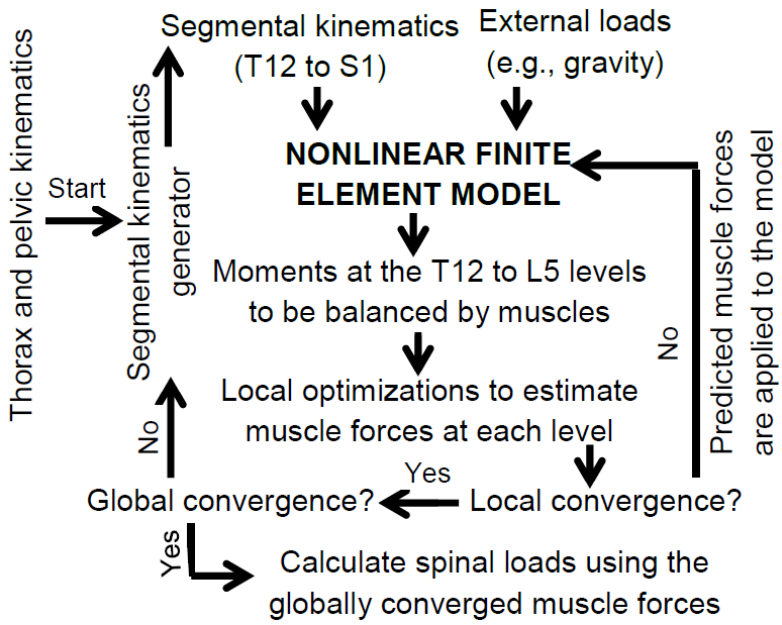

Figure 2. The process used to estimate muscle forces and spinal loads. Each set of possible segmental kinematics is generated using a genetic algorithm subjected to measured kinematics of thorax and pelvis as well as the reported values of lumbar segments' range of motion. The convergence in the local and global loops are achieved when the changes in respectively sum of predicted muscle forces in two consecutive local iterations and the value of the cost function of the heuristic optimization procedure in two consecutive global iterations are less than 1%.

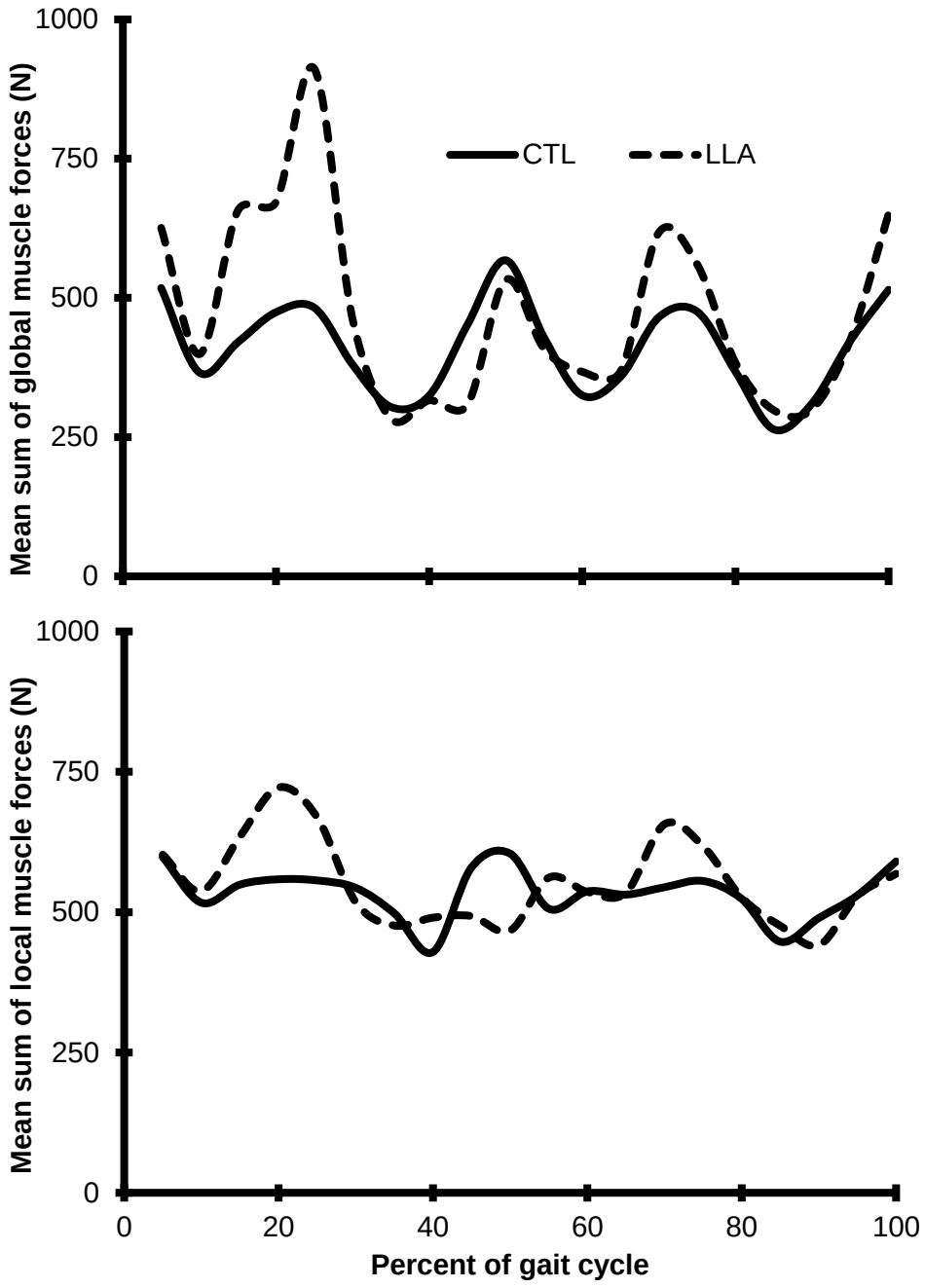

Figure 3. Mean sum of forces in global (i.e., muscles attached to the thoracic spine – top) and local (i.e., muscles attached to the lumbar spine – bottom) muscles. CTL: control group, LLA: group with lower limb amputation.

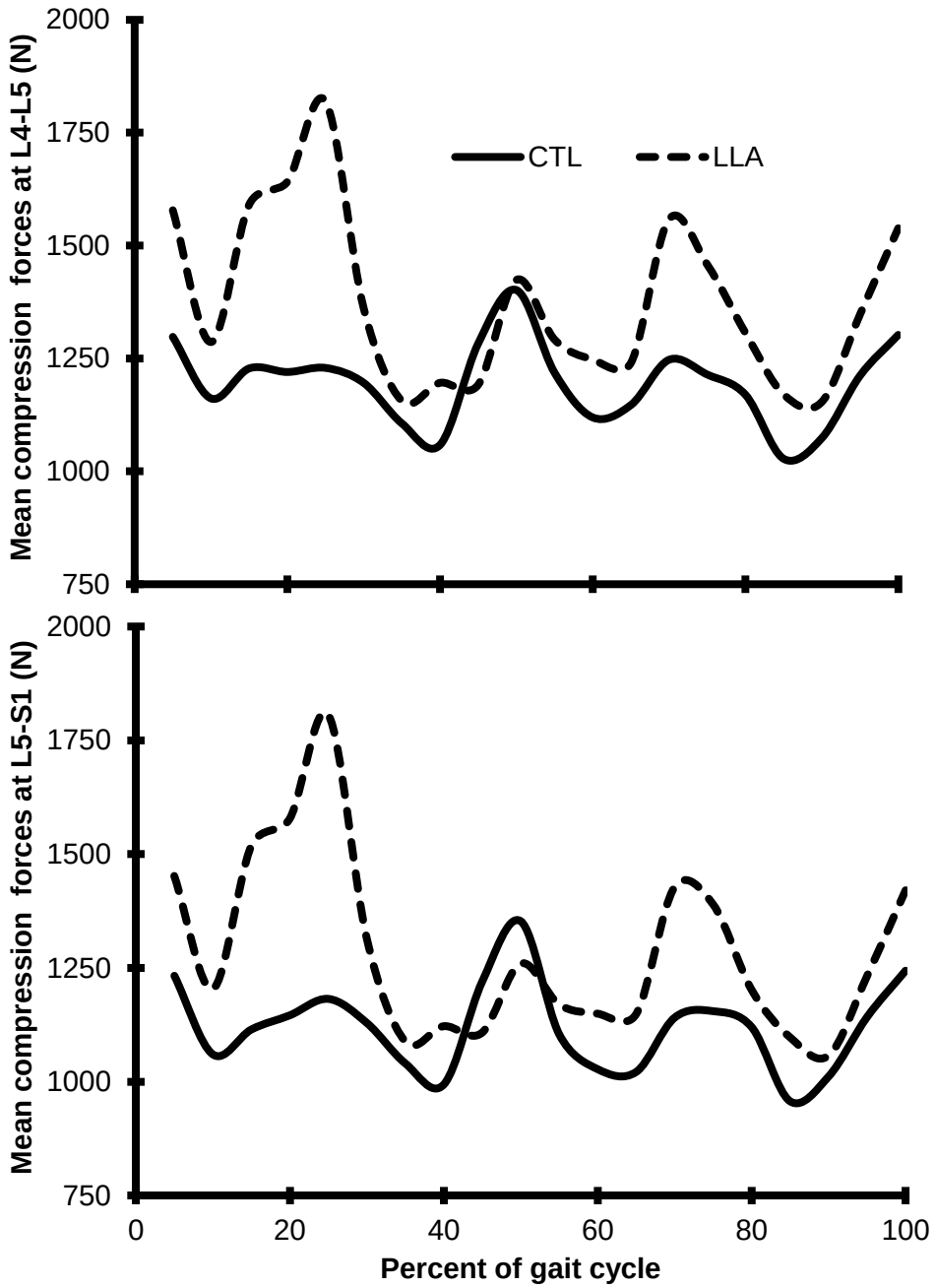

Figure 4. Mean compression forces at mid-plane of the L4-L5 (top) and L5-S1 (bottom) intervertebral discs. CTL: control group, LLA: group with lower limb amputation.

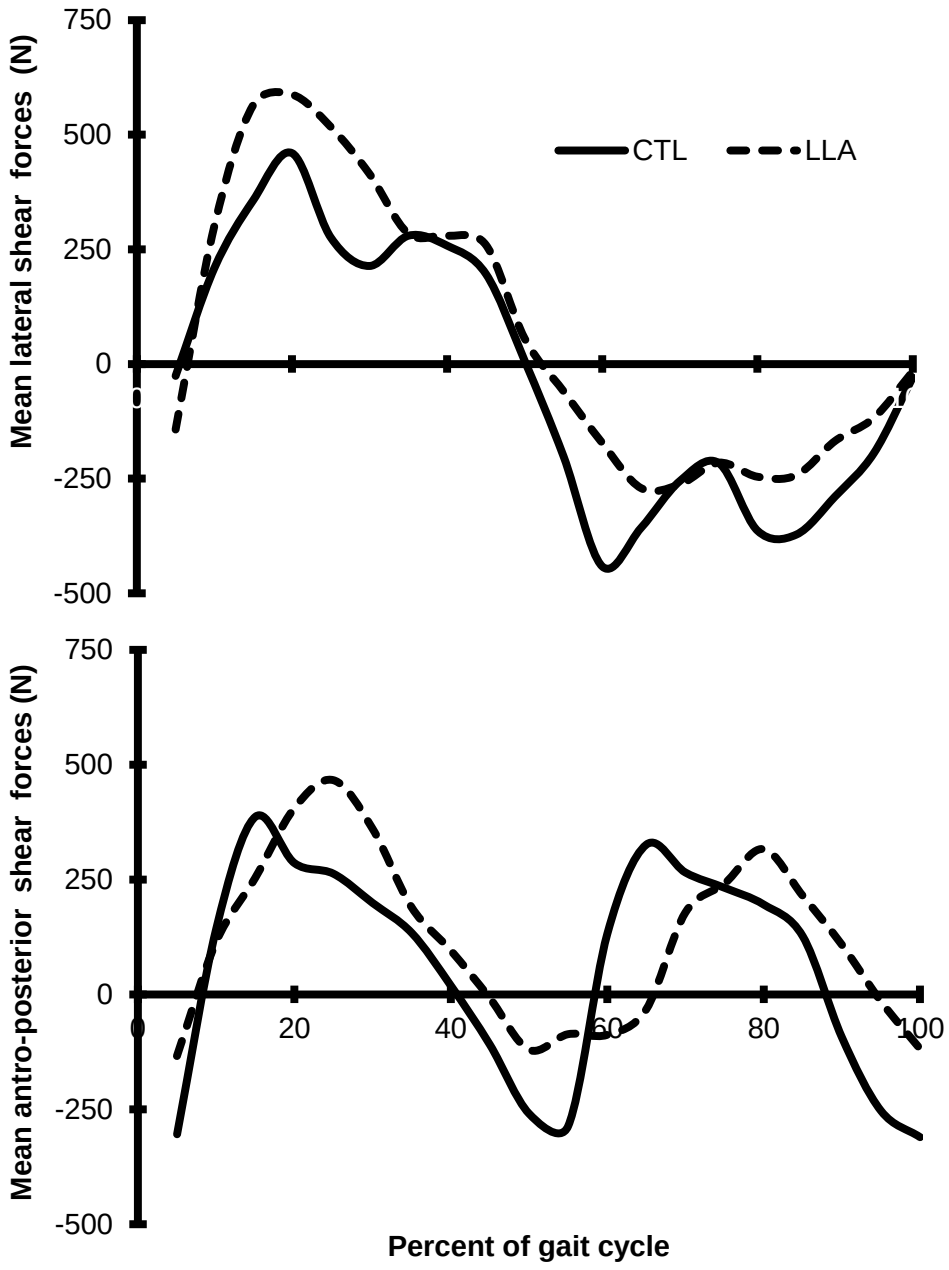

Figure 5. Mean shear forces at the mid-plane of the L5-S1 intervertebral disc in lateral (top) and antero-posterior (bottom) directions. CTL: control group, LLA: group with lower limb amputation. Positive shear force in lateral direction indicates force toward the right (intact) leg for controls (LLA) and positive shear force in antero-posterior direction indicate anterior direction.

Changes in Gait following Transfemoral Amputation Increase Spinal Loads

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Persons with transfemoral amputation (TFA) report a considerably higher prevalence of low back pain (LBP) compared to able-bodied individuals. Altered gait mechanics with TFA, particularly increased and asymmetric trunk motion, likely impose distinct demands on trunk muscles to maintain stability and equilibrium of the spine. Since alterations in trunk kinematics and muscle responses influence spinal loads, and spine loads are linked with LBP risk, the goal of this work was to demonstrate the effects of increased and asymmetric trunk kinematics with TFA on the relative contributions of external (i.e., gravity and inertia) and internal (i.e., muscle) forces to spine loads. Peak lumbar (i.e., thorax with respect to pelvis) lateral bending and forward lean obtained during gait from 20 persons with TFA (and 20 without), at 10 (4)° and 6 (3)°, respectively, were input into a kinematics-driven finite element model of the spine [1]. Total compressive and shear forces at the L5-S1 disc were computed, as well as relative contributions of internal and external forces. Influences of lumbar posture and mechanical properties of the passive ligamentous spine were also investigated.

Total compressive and shear forces at the L5-S1 disc were substantially larger among persons with (vs. without) TFA, at 1548 (785) and 429 (252) N, respectively. Given the comparable contributions from external forces between groups (≈ 350 N in compression and 150 N in shear), the main cause for such higher spinal loads is the internal muscle response to spinal equilibrium requirements; muscle force contributions among persons with (vs. without) TFA were 1201 (434) N in compression and 299 (114) N in shear. Additional simulations with altered lumbar postures and passive tissues properties among persons with TFA revealed minimal changes in spinal loads (<150 N), but again with larger contributions from muscle forces. Although obtained from static simulations (i.e., no inertia), these results clearly support our hypothesis of abnormal spine loading with altered trunk motion in persons with TFA, who here, exhibited substantially larger spinal loads compared to able-bodied controls. Due to the cyclic nature of gait, repeated exposures to increased spinal loads may accelerate degenerative changes in the spine and/or increase the risk for chronic LBP.

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Low Back Pain in Service Members with Traumatic Extremity Injuries: Implications of Biomechanical Risk Factors

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BACKGROUND: Low back pain (LBP) is far-reaching within the military and general population. While LBP generally has a multifactorial etiology and complex pathogenesis, biomechanical risk factors likely contribute more substantially among persons with traumatic extremity injuries, including lower-limb amputation (LLA). Specifically, persons with unilateral LLA walk and perform other activities of daily living in ways that often disproportionately rely on the intact (vs. prosthetic) limb. Such a compensational strategy is most notably associated with increased and/or asymmetric trunk movements as compared to able-bodied individuals; these movements are of particular concern given the biomechanical association between joint motions and musculoskeletal loads [1], and perceived by individuals with LLA as primary contributors toward LBP [2].

METHODS: Kinematic and kinetic data from 40 males with unilateral transtibial (n=20) and transfemoral (n=20) amputation, and 20 uninjured males, were obtained during level-ground walking at a self-selected pace. Net external demands (inverse dynamics) and bone-bone joint contact loads (finite element modeling) at L5-S1 are summarized in an effort to better understand relationships between altered trunk/pelvic motions with LLA on musculoskeletal loads within the lower back.

RESULTS: The coordination / motions between the trunk and pelvis with vs. without LLA are associated with ~31-55, 41-83, and 3-14% larger external demands on the lower back in the sagittal, coronal, and transverse planes, respectively. Similarly, joint contact forces within the spine are increased with LLA; notably, largest increases (up to ~65% relative to uninjured individuals) were found in joint compressive forces owing to a complex pattern and increased (6-80%) activation of trunk muscles. Also of note, increases were generally larger among individuals with more proximal amputations (transfemoral vs. transtibial), consistent with changes in trunk motions.

CONCLUSION: Though walking is generally not a mechanically demanding task for the low back (i.e., loads are well below reported injury thresholds), and sometimes even considered therapeutic for individuals with LBP, altered trunk-pelvic motions with LLA during gait are associated with larger external demands on the lower back and internal loads among tissues within the spine. Given the repetitive nature of gait, over time, even minimal increases in trunk motions and musculoskeletal loads may synergistically and progressively contribute toward LBP onset/recurrence and accelerate degenerative joint changes. However, to comprehensively characterize relative and accumulated risk profiles, additional efforts are needed to classify such relationships during other activities of daily living. In doing so, future work can begin to assess the ability of specific interventions (e.g., prosthetic devices, physical therapy) to mitigate injury risk.

1. Davis and Marras (2000) *Clinical Biomechanics*
2. Devan et al. (2015) *Disability and Rehabilitation*

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LEARNING OBJECTIVES:

1. Describe risk factors for low back pain with traumatic extremity injuries.
2. Define the impact of altered mechanics on the lower back and influence on low back pain risk.
3. Describe potential ways in which the elevated risk can be minimized, either clinically or with novel technologies.

COMPREHENSIVE INVITED REVIEW

Impact of Traumatic Lower Extremity Injuries Beyond Acute Care: Movement-Based Considerations for Resultant Longer Term Secondary Health Conditions

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Significance: Advances in field-based trauma care, surgical techniques, and protective equipment have collectively facilitated the survival of a historically large number of service members (SMs) following combat trauma, although many sustained significant composite tissue injuries to the extremities, including limb loss (LL) and limb salvage (LS). Beyond the acute surgical and rehabilitative efforts that focus primarily on wound care and restoring mobility, traumatic LL and LS are associated with several debilitating longer term secondary health conditions (*e.g.*, low back pain [LBP], osteoarthritis [OA], and cardiovascular disease [CVD]) that can adversely impact physical function and quality of life.
Recent Advances: Despite recent advancements in prosthetic and orthotic devices, altered movement and mechanical loading patterns have been identified among persons with LL and salvage, which are purported risk factors for the development of longer term secondary musculoskeletal conditions and may limit functional outcomes and/or concomitantly impact cardiovascular health.
Critical Issues: The increased prevalence of and risk for LBP, OA, and CVD among the relatively young cohort of SMs with LL and LS significantly impact physiological and psychological well-being, particularly over the next several decades of their lives.
Future Directions: Longitudinal studies are needed to characterize the onset, progression, and recurrence of health conditions secondary to LL and salvage. While not a focus of the current review, detailed characterization of physiological biomarkers throughout the rehabilitation process may provide additional insight into the current understanding of disease processes of the musculoskeletal and cardiovascular systems.

Keywords: amputation, biomechanics, cardiovascular disease, limb salvage, low back pain, osteoarthritis

SCOPE AND SIGNIFICANCE

Extremity trauma, including limb loss (LL) and limb salvage (LS), is commonly associated with an elevated risk for secondary health conditions (*e.g.*, low back pain [LBP], osteoarthritis [OA], cardiovascular disease [CVD]) that can significantly limit physical function, reduce quality of life (QoL), and life expectancy. This review

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provides an extensive commentary regarding resultant secondary health effects of extremity trauma in service members (SMs), with a particular focus on functional outcomes and quality of movement.

TRANSLATIONAL RELEVANCE

Physiologic biomarkers provide an opportunity to enhance translation in future work to examine the pathophysiology of the secondary health conditions associated with traumatic LL from a basic science perspective. While this approach is yet to be fully explored and thus was not a primary focus of this review, such biomarkers may augment traditional analyses and support more comprehensive risk characterization, thereby allowing clinicians and researchers to better mitigate disease onset or progression.

CLINICAL RELEVANCE

The increased prevalence of secondary health effects following traumatic extremity injuries places a significant physical and psychosocial burden on SMs with LL and LS. Altered movement patterns often result in mechanical loading of the spine and lower extremities, potentially increasing the risk of LBP and OA. Adopting a biopsychosocial model of treatment/care may allow clinicians to utilize a multifaceted approach to treat chronic pain and dysfunction associated with resultant health effects of LL.

BACKGROUND

Musculoskeletal disorders are the most prevalent source of disability in the United States.[1,2] As a result, the annual direct costs associated with treatment total a substantial $900 billion.[3] Among these, extremity amputation, or LL, is projected to affect an estimated 3.6 million people by the year 2050.[4] Approximately 185,000 individuals undergo either an upper or lower extremity amputation annually, primarily due to trauma, dysvascular disease, and/or osteosarcoma.[5–7] While the incidence of LL due to dysvascular etiologies has steadily risen among the civilian sector, trauma remains a leading source of LL within the Military Health System. However, prior estimates of the current/future impact of LL do not include SMs injured during combat nor do they consider individuals with LS; an alternative to amputation in which heroic measures are undertaken by the military surgical teams at all echelons of care to preserve as much form and function of the traumatically injured limb as possible. Despite these surgical efforts and advances in orthotic technology, many with LS are unable to achieve preinjury functional outcomes, much like those with LL.

The combat theaters of Operations: Enduring Freedom (OEF), Iraqi Freedom (OIF), New Dawn, Inherent Resolve, and Freedom's Sentinel were characterized by high-energy munitions and explosives. With advances in personal protective equipment, field-based trauma care, and surgical techniques, injuries sustained as a result of these often-improvised devices are now survivable at higher rates than conflicts past. However, traumatic extremity injuries, including LL and LS, remain a hallmark casualty of recent conflicts. Across all services, 52,351 military personnel have been wounded in action since 2001[8]; more than half of evacuated SMs have sustained extremity injuries and nearly a quarter of these are open fractures.[9] In addition, 1,703 SMs sustained injuries requiring major (or multiple) limb amputation (As of October 1, 2016; Data source: EACE-R). The decision to amputate a limb may be made in as few as 24 h post-trauma, during the first hospitalization as a secondary surgical intervention, or potentially years after LS (*i.e.*, delayed amputation).[10–13] Factors contributing to the decision include the extent and severity of injuries and resources available during the rehabilitation process.[14] Recent evidence suggests that SMs who undergo LS will typically experience more expansive complications than individuals who undergo amputation.[15–17] LS has been associated with significantly higher rates of rehospitalization, greater numbers of surgical procedures, and higher rates of surgical complications.[18,19]

Initial wound care and rehabilitation after LL and/or LS are critical to the recovery process. Such efforts are generally categorized by nine distinct phases, each with specific goals and objectives.[20] The complexity and interdependence between each phase elucidate the need for an efficient interdisciplinary approach within the overall rehabilitation paradigm. Despite these comprehensive and substantive efforts, persons with LL and LS are at an increased risk for acute secondary health conditions such as phantom limb pain, wounds/sores, vascular and nerve damage, infection, decreased physical function, and psychosocial issues. Furthermore, beyond these acute conditions, persons with LL and LS are also at an elevated risk for longer term complications including LBP, OA, and CVD, among others. Importantly, once the disease progression initiates, these longer term resultant conditions will plague these individuals for life, as SMs with extremity trauma are typically younger than 30 years at the time of

injury and thus will continue living with their injuries for several decades.[17]

The long-term economic burden of trauma-related LL and LS is significant. Edwards *et al.* predicted the long-term (40 year) cost of trauma repair, rehabilitation, and lifelong prosthetic support of British soldiers wounded in Afghanistan to be approximately $444 million.[21] In the United States, the estimated average lifetime cost of treatment for unilateral lower LL is $342,716 and $1.4 million for Vietnam and OIF/OEF veterans, respectively.[22] However, such estimates are likely conservative, not fully accounting for costs associated with novel technology/repairs or, perhaps exponentially more economically burdensome over the longer term, for the wide range of healthcare costs associated with the treatment of secondary health conditions. The ability to evaluate, predict, and ultimately treat these resultant health conditions would not only help reduce these costs but also, and most importantly, preserve and/or improve function and QoL for those with LL and LS.

The risk for secondary health conditions is often related to physiological adaptations to trauma or pervasive surgical complications, poor biomechanics, and/or the prosthetic (orthotic) device itself. For SMs, in particular, the young age at which these injuries occur likely presents a unique challenge over the longer term and further highlights the importance for understanding resultant health conditions secondary to extremity trauma. Notably, the cumulative effects of many years of functional adaptations during gait and movement with extended prosthetic/orthotic device use in otherwise young and active SMs remain unclear.[23,24] This is an important distinction from civilian populations as a majority of civilians with LL are over the age of 50, incurred LL as a result of vascular damage/complications, are likely less active, and may present with different resultant health conditions/outcomes for less time.[25] Thus, as a preliminary step toward addressing this knowledge gap, the purpose of this review is to provide a commentary regarding resultant health conditions associated with high-energy extremity trauma, with a primary focus on biomechanical features of movement and associated functional limitations. In particular, we highlight considerations for longitudinal care aimed at maximizing QoL, for those with both LL and LS.

DISCUSSION

Low back pain

The World Health Organization describes LBP as any pain or discomfort for a variable duration in the lumbar spine region.[26] The onset of pain may occur suddenly, coincident to a singular traumatic event, or develop over time with age or as the result of repeated microtrauma from a given (or set of) activity(ies). Often, LBP is considered idiopathic, as pain may be present without pathoanatomical evidence of disease or structural abnormality. LBP costs nearly $100 billion annually in the United States, with a majority of this cost associated with lost wages and decreased productivity.[27] While cross-sectional figures indicate that chronic LBP affects up to 33% of adults in the general population, the incidence in persons with LL who report LBP secondary to trauma is nearly double (52–76%).[28–31] Along with this significantly higher prevalence, nearly 50% of persons with LL have reported LBP as "more bothersome" than either residual or phantom limb pain and as having a significant reduction in overall QoL metrics.[28,30,32] While the exact etiology of LBP within this population is unclear, there is a growing body of evidence suggesting that altered lumbopelvic mechanics during the (repetitive) gait cycle likely influences such risk.

Persons with lower LL frequently develop altered movement patterns to maintain balance and achieve forward progression in walking. Movement patterns can be influenced by the following, either individually or in combination: socket fit/prosthetic alignment, general deconditioning, leg length discrepancies, complications within the residual limb, and muscular imbalances.[33,34] More specifically, altered movement patterns during gait affect trunk and pelvis mechanics and contribute, at least in part, to the increased incidence of LBP in persons with lower LL and may be dependent on the extent of injury or ultimate level of amputation.[35–38] These alterations and asymmetries may increase loads on the lumbar spine during gait which, when considering the repetitive gait cycle, over time may thus contribute to the occurrence or recurrence of LBP. For example, persons with transfemoral LL tend to exhibit 10° of anterior pelvic tilt, which is considered to be a compensatory mechanism to assist in the ability to achieve hip extension during gait. Increased anterior pelvic tilt is associated with increased lumbar lordosis, which is linked to an increased incidence of LBP in persons with LL.[28,39] Previous work has demonstrated that increased loads on the lumbar spine are a direct source of LBP in the general population.[40,41] Mechanical loading of the passive and active structures of the spine is affected by both internal and external loads, such as forces produced by muscular activation, ligamentous tension, gravity, and inertia.[42]

These loads can be significant, as potentially small alterations in trunk (which accounts for nearly 2/3 of the body's mass) movement may increase joint reaction loading due to increased muscular contractions of the surrounding musculature.[43] The increased demand on the active structures (muscles) may lead to increased forces and joint loading on the passive structures (discs and vertebrae). The accumulation of these altered loads over time has the potential to augment degenerative joint changes in the spine.[40]

Similar to uninjured individuals with LBP, persons with transfemoral LL exhibit irregular trunk–pelvis coordination and movement variability.[44] Specifically, persons with LL tend to walk with a large lateral trunk lean toward the affected side; a possible neuromuscular strategy/compensation to assist in forward progression during gait.[42] This frontal plane motion has been reported to increase peak joint reaction forces and moments asymmetrically in the lumbar spine (L5-S1 integration specifically) in this population. A recent report suggested this observed frontal plane motion as a possible mechanistic pathway through which recurring exposure to altered trunk motion and cumulative spinal loading may contribute to LBP in persons with lower LL.[42] Persons with transfemoral LL (with current LBP) exhibit larger axial trunk rotations when compared to those without LBP, which may subsequently affect vertebral disc degeneration and potentially contribute to LBP recurrence.[45,46] Previous evidence demonstrated degenerative changes in the lumbar spine via radiographic imaging in 76% of persons with LL, potentially supporting the role of increased trunk motion leading to degenerative changes in this population.[47]

While LBP is commonly cited as a secondary health effect of LL, persons with LS may also experience LBP as a result of altered movement patterns during gait and functional activities.[48] Persons with LS typically experience reduced ankle function, which is associated with altered gait mechanics and increased metabolic cost.[34,49,50] However, the influence of distal LS on proximal (trunk/pelvis) biomechanics remains unstudied to date. Currently, a paucity of evidence exists relative to the prevalence of LBP in the LS population. Therefore, further work is needed to elucidate the relationship between LS and the development of LBP.

In summary, LBP has been reported as the most important health-related physical condition contributing to a reduced QoL among veterans who had sustained a traumatic lower extremity amputation over 20 years prior.[32] Thus, identifying factors contributing to the development and recurrence of LBP, such as a widely prevalent and "bothersome" secondary health concern, is critical for improving long-term health. Abnormal mechanical loading of lumbar spine, altered trunk and pelvis coordination, and psychosocial factors may influence the prevalence of LBP in this population. Therapeutic interventions that address the underlying impairment(s) in trunk neuromuscular responses and/or motor control strategy may also contribute to reducing the prevalence and incidence of LBP among SMs with lower extremity trauma, thereby improving longer term functional outcomes by mitigating a significant secondary impairment with a substantial adverse impact on daily activities. Further evidence is needed to understand the relationship between these risk factors and the incidence of LBP in persons with LL. In particular, no studies to date have evaluated the influence of different prostheses or orthoses on the incidence of LBP in the traumatic LL and LS populations.

Osteoarthritis

The National Institute of Arthritis and Musculoskeletal and Skin Diseases describes OA as a joint disease affecting the cartilage, often characterized by pain and stiffness within a joint and limitations in physical function.[51] The primary pathology is articular cartilage deterioration, although evidence suggests that possible morphological changes of bone are reflective of disease onset. Within the joint, articular cartilage functions to dissipate forces sustained by the bony structures throughout motion. During activities such as walking or running, when the loading velocity and intensity of the structures are increased, the cartilage's ability to dissipate forces is reduced.[52] In the general population, mechanical loading of the knee joint during walking has been associated with the presence, severity, and progression of knee OA.[53–56] Persons with unilateral lower LL are 17 times more likely to suffer from knee OA in the intact limb when compared to able-bodied individuals.[57]

As previously noted, persons with LL frequently develop altered movement patterns during gait. Of particular importance here, those with unilateral LL preferentially utilize their intact limb, leading to increased and prolonged loading of the intact joints. Mechanical alterations in static and dynamic alignment of the knee joint may affect joint loading as increased forces are incurred through medial or lateral aspects of the joint. The external knee adduction moment (EKAM) is a vastly reported risk factor for knee OA based on its relationship with internal loading of the medial joint surface.[58] The size of the EKAM and its respective angular impulse

are associated with knee OA severity and progression.[53,55,59,60] During gait, individuals with lower LL asymmetrically load their intact limb to a greater extent than their involved limb, suggesting that mechanical factors play a role in the increased incidence of knee OA in this population.[36,61] For example, Lloyd *et al.* identified larger peak knee adduction moments in the intact relative to involved limb.[62] This increased mechanical loading may be explained by decreased push-off power and ground reaction forces demonstrated with conventional prosthetic feet.[61,63] Push-off power generated by the prosthetic foot instance may affect the ground reaction forces at heel strike in the intact limb as the velocity of an individual's center of mass changes from an anterior and inferior direction to an anterior and superior direction during gait.[64] The redirection of the center of mass is caused by the ground reaction impulse through the gait cycle, crudely relative to double-limb support.[64] If the prosthetic stance foot lacks adequate push-off power to propel the center of mass anteriorly, the intact limb must compensate by performing more work to move the center of mass anterior and superior, resulting in increased ground reaction forces and loading of the intact limb.[61] Morgenroth *et al.* suggested that by utilizing a prosthetic foot with increased push-off power, the peak EKAM of the intact limb may be reduced and therefore potentially decreasing the OA risk.[61] This was supported as a powered ankle–foot prosthetic was able to decrease the EKAM and vertical ground reaction force in persons with lower LL, however, the prosthetic used was unable to alter the knee joint loads of the intact limb.[65] Similar to LBP, the progression and severity of OA may be further amplified by psychosocial determinants; anxiety, depression, coping strategies, and stress have also been associated with increased pain in patients with OA.[66–68]

OA is not exclusive to the LL population as individuals with LS present with similar (sometimes larger) gait and movement deviations. As high as 95% of OA diagnoses among combat-wounded SMs are post-traumatic in origin.[69] Chronic pain, nerve damage, and volumetric muscle loss are common barriers to LS rehabilitation and may serve as confounding factors in the development of OA treatment plans.[70,71] Ankle–foot orthoses (AFOs) are commonly used to assist ankle function or offload painful structures.[72] Optional therapies that include sports medicine-based interventions utilizing a dynamic AFO (*e.g.*, the Intrepid Dynamic Exoskeletal Orthosis) are available to LS patients. Such devices are designed to improve functional performance on tasks such as walking, changing directions, sit-to-stand, and ascending stairs.[48] While dynamic AFOs are suggested to improve functional capabilities, evidence is inconclusive in its ability to positively alter gait parameters related to OA as well as the effects of long-term use.[34,73,74]

Treatment modalities focused on reducing symptoms and OA disease progression in persons with LL and LS are vital to improving QoL. The Osteoarthritis Research Society International recommends biomechanical interventions, intra-articular corticosteroids, exercise (land and water based), self-management and education, strength training, and weight management.[75] Autologous platelet-rich plasma (PRP) therapy is a therapeutic intervention that delivers high concentrations of growth factors to an area to stimulate healing.[76] Recent evidence suggests that PRP may provide relief of knee OA symptoms in younger patients within the early stages of cartilage degeneration.[77–79] Strength training (weight and body-weight training) and exercises such as t'ai chi have demonstrated the ability to improve overall function in decreasing pain in OA patients and may also serve to assist in weight management.[80,81] Weight reduction is considered a pragmatic therapy for knee OA as overweight individuals demonstrate a high prevalence of knee OA and the risk of severity progression increases 35% for every 5 kg of weight gain.[82] Strength training and weight management are considered integral aspects of the rehabilitation paradigm for persons with LL as deficits in strength and increases in weight influence gait, joint loading, movement efficiency, and cardiovascular health. Canes, knee braces, and foot orthotics are other potential treatment options to decrease movements at the knee, reduce pain, and improve function.[83–85]

In summary, biomechanical factors likely play a substantial role in the risk for OA secondary to extremity trauma, whether LL or LS. While the prevalence of OA in LL and LS populations may decrease as technological improvements in prostheses and orthoses are realized, further evidence is needed to determine the specific relationship between different classes or features of these devices and OA risk factors. Unfortunately, recent technological advancements in prosthetic devices have outpaced orthotic devices, the benefits of which are evident in the biomechanical characteristics of persons with LL versus LS. Nevertheless, LS typically presents with more complex neurovascular injuries and other unique challenges, which can negatively affect functional outcomes.

Cardiovascular disease

CVD is defined by a vast array of diseases affecting the heart and blood vessels.[86] CVD may present

as coronary artery disease, stroke, arrhythmias, cardiomyopathy, heart disease, peripheral artery disease, aneurysms, venous thrombosis, and/or carditis.[86,87] While CVD is largely preventable, it remains the leading cause of death worldwide, particularly in lower socioeconomic demographics.[86] The American Heart Association reports there are ~85 million individuals with CVD in the United States, causing a staggering 2,200 deaths each and every day.[88] This is accompanied by direct and indirect costs of nearly $315 billion.[89] Risk factors for CVD include, but are not limited to, family history and genetics, high cholesterol and lipids, high blood pressure, diabetes, metabolic syndrome, obesity, and kidney disease.[89] In addition, significant combat trauma may be a risk factor for the development of CVD.[90–92] For example, Hrubec and Ryder conducted a 30-year follow-up of World War II veterans with lower LL and demonstrated that the relative risk of CVD mortality was increased 2.4–4 times that of persons with LS.[90] Similarly, Modan *et al.* reported significantly higher mortality rates of persons with traumatic lower LL when compared to able-bodied controls, suggesting that CVD was the primary cause (21.9% vs. 12.1%, $p < 0.001$).[91]

The pathophysiology of increased mortality rates may be a result of systemic and/or regional hemodynamic effects of trauma.[91,93–97] Obesity and hypertension secondary to decreased overall activity levels may lead to insulin regulation complications in persons with LL.[97] When compared to uninjured controls with no difference in body mass index, blood pressure, or lipid levels, persons with LL exhibited significantly higher increased fasting plasma insulin levels as well as insulin resistance.[96] Increased plasma insulin levels and insulin resistance are risk factors for atherosclerosis and metabolic syndrome, considered precursors to CVD. The role of psychological stressors in the development of CVD is not well understood; however, psychosocial factors have demonstrated involvement in the pathogenesis of CVD.[98,99] Depression and post-traumatic stress disorder have been associated with increased incidence of CVD, while veterans with high levels of cynical distrust and anger demonstrate an accelerated progression of atherosclerosis, a risk factor for CVD.[100–102] Limited evidence precludes a definitive relationship between psychosocial factors and CVD risk in persons with LL, and therefore, future work should prospectively examine the relationship between psychosocial factors/stressors and the development of CVD.

Hemodynamically, proximal amputation increases the risk of CVD development based on alterations in proximal arterial flow. Pathogenic mechanisms may include early reflection pulse waves. Early return reflection pulse waves are produced at arterial occlusion sites and have been linked to a myriad of medical complications.[103] An early returned reflection pulse wave creates a second systolic peak, which results in an increase in aortic pressure. The increased aortic pressure generates an increased left ventricular load resulting in left ventricular hypertrophy, atherothrombosis, and ultimately cardiac death.[104] Vollmar *et al.* suggested that persons with traumatic LL above the knee were five times more likely to suffer from abdominal aortic aneurysms when compared to healthy controls.[94] A possible explanation may be that after amputation, blood flow is decreased by ~25% in the terminal aorta due to altered flow paths in the visceral and renal arteries, resulting in a disrupted flow pattern at the aortic bifurcation.[95] Altered flow patterns, paired with increased shear stress along the convex aspect of the aorta and decreased shear stress along the concave aspect, are theorized to damage aorto-iliac blood vessels by increasing hydraulic forces within the aorta.[95] Persons with transfemoral LL should have regular consultations with appropriate medical personnel to assess the risk of abdominal aortic aneurysm.[95]

While the hemodynamic effects of trauma appear to influence CVD risk, addressing modifiable risk factors may be an effective strategy to help decrease CVD risk. It is widely accepted that habitual exercise with activities such as running, walking, bicycling, rowing, and swimming increases aerobic capacity and decreases the risk of CVD. When joined with dietary modifications, regular exercise can effectively reduce excess body weight, another risk factor for CVD. Moreover, the increased risk of CVD in persons with LL highlights the importance of managing modifiable risk factors, engaging in preventative treatment strategies, and adopting an active lifestyle.

SUMMARY

Maintaining an active lifestyle is critically important for physiological health, psychological well-being, and overall QoL. Such guidance is no different for individuals with LL and LS. However, given the limited (but growing) body of evidence relating movement abnormalities to altered musculoskeletal demands that may lead to the development of longer term secondary conditions in this population, additional consideration for the quality of movement during recreational and daily activities is warranted. While the overwhelming focus of recent efforts has been on persons with LL, the aforementioned secondary health conditions are likely also major con-

cerns for those with LS. As such, we posit that an underlying focus of clinical care and future research, in both cohorts, should be toward mitigating concomitant risk for the development or recurrence of chronic pain.

While advances in trauma care and prosthetic/orthotic technologies may eventually mollify acute and subacute secondary health effects of extremity trauma, longitudinal tracking is urgently needed to better understand the mechanisms by which secondary health effects develop and progress in this population. Such efforts should encompass a transdisciplinary team, in which a comprehensive suite of evaluation metrics are employed; for example, traditional clinical evaluation and movement analysis supplemented with local and systemic physiological biomarker analyses and next-generation imaging modalities. In doing so, a better understanding of the specific pathways for the development of these secondary health effects can be realized, thus enabling clinicians to develop and prescribe appropriate treatment interventions. Ultimately, diminishing risk factors relative to the degeneration of joint and cardiovascular function will reduce the overall prevalence of secondary health conditions and improve QoL for our nation's injured SMs and veterans over the longer term.

TAKE HOME MESSAGES

- Living with LL and LS over time leads to increased morbidity and mortality from secondary medical and musculoskeletal problems. Awareness of the long-term health risks associated with LL and LS, as well as the physiologic and biomechanical origin of these risks, is critical to improving outcomes
- Understanding the pathogenesis of the secondary health conditions of traumatic LL and LS and salvage may help guide optimal management in acute, subacute, and chronic phases of care for these individuals
- Reducing modifiable risk factors through patient education, identifying appropriate support systems, encouraging proper gait mechanics, and utilizing the prescription of evolving technologies may help mitigate long-term health conditions

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Abbreviations and Acronyms

AFO = ankle–foot orthoses
CVD = cardiovascular disease
EACE = Extremity Trauma and Amputation Center of Excellence
EKAM = external knee adduction moment
LBP = low back pain
LL = limb loss
LS = limb salvage
OA = osteoarthritis
OEF = Operation Enduring Freedom
OIF = Operation Iraqi Freedom
PRP = platelet-rich plasma
QoL = quality of life
SM = service member
USUHS = Uniformed Services University of the Health Sciences
WRNMMC = Walter Reed National Military Medical Center

FASTER WALKING SPEEDS DIFFERENTIALLY ALTER SPINAL LOADS IN PERSONS WITH TRAUMATIC LOWER LIMB AMPUTATION

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Persons with lower limb amputation (LLA) commonly report low back pain and perceive altered trunk motions/postures during activities of daily living as primary contributors [1]. When walking at a self-selected pace, our prior work has demonstrated altered trunk motions among persons with vs. without LLA are associated with 26-60% increases in spinal loads [2]. Here, we expand these efforts by presenting preliminary data of a much larger sample[a] regarding the influence of walking speed on spinal loads in this population. Trunk and pelvic kinematics, collected during level-ground walking at 3 controlled speeds (~1.0, 1.3, and 1.6 m/s), were extracted for 1 male servicemember with unilateral transfemoral amputation (35 yr, 173.0 cm, 106.8 kg) and 1 male servicemember without amputation (27 yr, 179.0 cm, 72.0 kg). These kinematic data were input to a kinematics-driven, non-linear finite element model of the lower back to estimate the resultant compressive and lateral/anteroposterior shear loads at L5/S1 using an optimization-based iterative procedure [3]. Peak compressive, lateral, and anteroposterior shear loads generally increased with increasing walking speed. However, increases in compression and lateral shear with increasing walking speed were larger among the person with vs. without LLA, particularly in lateral shear at the fastest speed (Figure 1A-B). In contrast, peak anteroposterior shear decreased with increasing walking speed among the person with LLA (Figure 1C). Although walking is generally not a mechanically demanding task for the low back (i.e., loads are well below reported injury thresholds), walking faster for persons with LLA appear to differentially alter external demands on the lower back and internal loads among tissues within the spine. Thus, over time, repeated exposures to faster walking speeds may contribute to the elevated risk for low back pain after LLA, due to fatigue failure of spinal tissues, though further work to more completely characterize spinal loads during activities of daily living is warranted.

[a] Final results from n ≥ 20 in each group (with additional speeds and levels of amputation) will be presented at the workshop

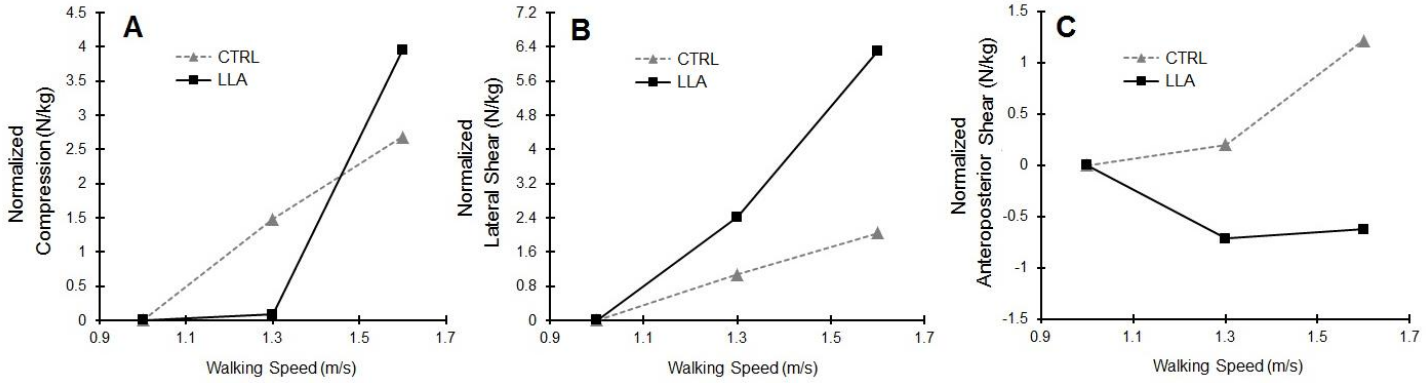

Figure 1. Normalized changes in (A) compression, (B) lateral shear, and (C) anteroposterior shear with increasing walking speed, for an individual with lower limb amputation (LLA) and an uninjured control (CTRL). To highlight the influences of walking speed, changes in spinal loads are shown with respect to values obtained in the 1.0 m/s walking speed and are normalized by body mass.

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The Biopsychosocial Correlates of Chronic Low Back Pain after Lower Limb Amputation

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ABSTRACT

Low back pain is a common secondary health condition after lower limb amputation with important implications related to functional capabilities and overall quality of life. Despite the high prevalence of low back pain after lower limb amputation, the underlying etiologies of the disorder remain unknown. This special communication summarizes evidence in support of the multifactorial, biopsychosocial model of the low back pain experience in the general population and after lower limb amputation for identification of potential risk factors and treatment targets. Key findings that link biological, psychological, and social factors and the experience of low back pain after lower limb amputation are discussed while highlighting gaps in our current state of knowledge to direct future research. Importantly, the aim of this special communication was not to propose a new model, but rather to organize data originating from prior work into a coherent conceptual framework to better understand the need for multifaceted and multidisciplinary intervention approaches for effective treatment of low back pain after lower limb amputation.

INTRODUCTION

Low back pain (LBP) is a common health condition worldwide, with 11-38% of the general population reporting symptoms over a one year period.[1, 2] LBP is currently considered the leading cause of disability globally, ahead of 290 other conditions, and is responsible for 83 million years lived with disability.[3] Additionally, LBP is a major source of activity limitation, work absenteeism, and increased cost of medical care throughout much of the world.[2, 4-6] LBP is also a common and perhaps more impactful, secondary health condition after lower limb amputation (LLA), with high estimated annual prevalence rates between 50-90%.[7-13] Individuals with LLA often report more LBP after amputation than before[8, 9] and in most cases directly attribute their LBP to their amputation.[10] Additionally, presence of LBP daily or several times per week has been associated with moderate to severe physical disability and limitations in performing daily activities in patients with LLA.[8, 9, 13-15] To this end, LBP is often rated by patients with LLA as more bothersome than phantom or residual limb pain,[11] suggesting LBP is an important secondary musculoskeletal condition associated with functional limitation and disability after LLA.

Despite the high prevalence of LBP after LLA, the exact etiologies of the disorder in this population remain unknown, thereby making its treatment exceptionally challenging. Importantly, there are currently no published guidelines specifically tailored toward the management of LBP for individuals with LLA. Therefore, there exists a clear need for comprehensive identification of contributing factors to the LBP experience after LLA that can serve as a basis for the development of targeted treatments and future research investigations. Here a new application of the multifactorial, biopsychosocial model for LBP, previously developed for the general population,[16-18] is proposed as a way of identifying risk factors and

potential treatment targets for treatment of LBP after LLA. The objective of this special communication was to organize data originating from prior studies of the biopsychosocial correlates of LBP after LLA into a coherent conceptual framework. We hypothesized that alterations in biological, psychological, and social factors with LLA are related to the development of LBP symptoms and disability after LLA that merit specific attention during the clinical decision making process and for future research efforts to improve patient-related outcomes.

The Biopsychosocial Model of Low Back Pain

Treatment of LBP has historically centered around the traditional biomedical model of illness, which assumes a direct relationship between regional pathoanatomy and the perception of pain.[18] As such, it was expected that once the anatomical source of LBP is identified, biochemical and/or mechanical treatments of underlying pathoanatomy would result in cessation of pain. Despite leading to successful treatment of many other disease processes, the outcomes of interventions based on the biomedical model have proven to be less than ideal for treatment of LBP.[18-20] One potential reason for the failure of the biomedical model to provide an effective treatment option for LBP is that no single underlying pathoanatomical lesion has been consistently identified,[18] with up to 85% of LBP patients left without a precise pathoanatomical diagnosis.[21] Additionally, determination of pathoanatomical sources of LBP frequently lacks interexaminer reliability and evidence for generalizability.[22] The often equivocal outcomes from many "lesion-specific" treatment options such as intra-articular corticosteroid injections[19] and spinal fusion surgeries,[20] along with the generally poor predictive value of diagnostic imaging for identification of pathoanatomical pain sources,[23] have led to a recent paradigm shift toward a "non-structural" approach for the management of LBP.[24]

A growing body of evidence now suggests that successful treatment of LBP should include biological, psychological, and social assessments to comprehensively address the patient's unique pain experience.[18] The so called "biopsychosocial model" of LBP suggests that the patient's perceptions and reactions to pain should also be considered as these factors often lead to unnecessary avoidance of physical activity and social interactions, work absenteeism, and high health care utilization.[16, 17] Whereas the pathoanatomy may initiate the pain process, the psychological and social factors appear to play an important role in exacerbating the biological component of LBP by influencing the perception of pain.[25] For example, it has been hypothesized that the presence of mechanical LBP can lead to a pain-generated stress response that could have a negative impact on the endocrine and immune systems, which in turn may negatively affect the cognitive assessment, emotional response, coping strategies and health practices of the individual.[26]

Proponents of the biopsychosocial model argue that the complex, multidimensional nature of LBP does not lend itself to the reductionist view of the biomedical model; instead, the patient's unique biologic, psychological, and social factors must equally be considered.[18] Therefore, the term biopsychosocial implies that the biological, psychological and social factors are interwoven within the context of the patient's overall LBP experience and should be directly and concurrently considered as a part of a comprehensive treatment program.[26] In support of this theory, multidisciplinary treatment approaches that include biopsychosocial components for treatment of LBP in adults have demonstrated positive effects on pain, disability, and health-related quality of life.[27, 28] It stands to reason that LLA likely amplifies and/or alters specific components within the multifactorial biopsychosocial model of LBP**,** previously suggested for the general population. Given that LLA may differentially affect the various components of this

model **(Figure 1)**, discrimination of clinically meaningful sub-groups of patients with LBP after LLA will most likely require assessments of biological, psychological and social domains,[22] which have not been previously evaluated in this patient population.

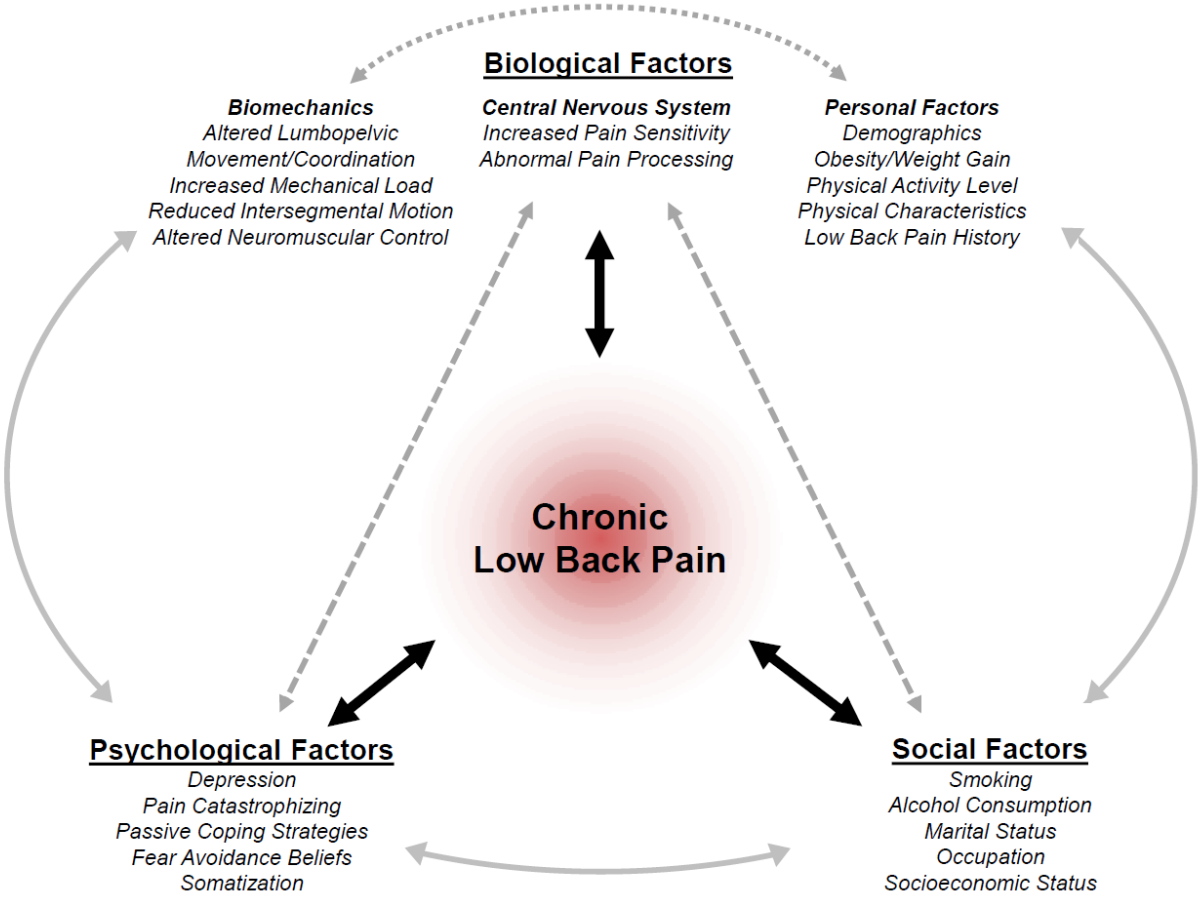

Figure 1. Individual components (and their potential associations) of the biopsychosocial model of low back pain likely influenced or amplified by lower limb amputation.

Biological Factors

Biomechanics

Altered mechanics of gait and movement have been historically proposed to play a causative role in the development and/or recurrence of LBP after LLA.[29] In fact, persons with LLA perceive "uneven postures and compensatory movements" affected by "fatigue" and

"prosthesis-related factors" during functional activities as the primary contributors to LBP.[30] Though at the expense of higher metabolic cost of transport,[31] compensatory movement strategies adopted after LLA typically involve adaptations to maintain the body's center of mass within the base of support (i.e., improve stability and balance), primarily with a preference for the intact limb, if applicable.[32] During gait, for example, the intact limb (relative to prosthetic limb) is characterized by a longer stance time, shorter step length, wider stride width, and larger vertical ground reaction forces.[33] As the trunk accounts for approximately two-thirds of total body mass,[34] altered motions of this segment play a substantial role in post-amputation movement strategies, thereby warranting more trunk-focused biomechanical investigations for assessing potential links with the development and persistence of LBP.

Altered trunk and pelvic movements in persons with LLA have been previously identified in all three cardinal planes, including larger forward trunk lean and flexion-extension range of motion, greater lateral trunk flexion (towards the prosthetic limb) and pelvic obliquity motion, as well as more axial rotations between the shoulders/pelvis or regional/intervertebral motion segments.[35, 36] The presence (and likely severity) of LBP further influences such motions, most notably increasing axial rotations within the lumbar region.[37] LBP has also been associated with more in-phase mediolateral coordination between the trunk and pelvis,[38] which is indicative of inter-segmental rigidity (i.e., "guarding behavior") previously reported in able-bodied individuals who are experiencing LBP.[39, 40] Additional evidence suggests that individuals with LLA employ an active mediolateral trunk movement strategy, inferred from increases in generation and absorption of energy between the trunk and pelvis.[41, 42] Although actively increasing mediolateral trunk sway is likely an attempt to improve joint stability within the lower extremity by altering lever arms of ab/adductor musculature,[43] most notably within the hip among patients with

transfemoral amputation,[7] such strategies have been associated with LBP/discomfort among able-bodied individuals performing gait training aimed at reducing knee joint loads via trunk lateral flexion.[44]

Abnormal mechanics of the spinal column, primarily larger mechanical loads and instability, are often considered risk factors for the development of LBP.[45] Of particular interest here, characteristics of trunk motion can directly influence musculoskeletal loading,[46] typically due to altered muscular response (i.e., coactivity).[45] Though walking is generally not a mechanically demanding task for the low back (i.e., loads are well below reported injury thresholds),[47] and sometimes even considered therapeutic for individuals with LBP,[48] altered trunk-pelvic motions with LLA during gait have recently been associated with large internal loads among tissues within the spine.[49-51] Notably, largest increases (up to ~65% relative to uninjured individuals) were found in joint compressive forces owing to a complex pattern of muscle responses.[50] Given the repetitive nature of gait, over time, even minimal increases in trunk motions and musculoskeletal loads may synergistically and progressively contribute toward LBP onset and/or recurrence and accelerate degenerative joint changes in the spine.

It is well accepted that the neuromuscular system plays a central role in supporting the upper body and maintaining mechanical equilibrium and stability of the spine.[52, 53] Irregular patterns of trunk muscle recruitment have been identified among the general population with recurrent LBP,[54] and impaired postural control has been associated with spinal instability and LBP.[55] Among persons with LLA without LBP, similar assessments have identified impairments in trunk postural control during an unstable seated balance task,[56] bilateral asymmetries in trunk mechanical and muscular responses to applied positional perturbations,[57] as well as altered load-sharing between active and passive trunk tissues during quasi-static trunk flexion/extension

movements.[58] Additionally, substantially greater fatigability has been reported for the low-back extensor musculature in patients with LLA with and without LBP,[15] that are more pronounced than healthy individuals with and without LBP.[59] Fatigue of the low back extensors may further contribute to increased intersegmental spinal motion and instability during prolonged functional activities.[60] Though the specific origin and functional impact of such alterations remain somewhat speculative, these data support the theory that repeated exposure to altered loading associated with LLA and repeated use of a prosthetic device may result in tissue and neuromuscular adaptations and increased risk for LBP in this population.

Central Nervous System

In addition to changes in trunk/pelvic biomechanics with LLA, central nervous system factors may also play an important biological role in the manifestation of LBP after LLA. Because of the trauma to peripheral nerves, amputation has the potential to influence the processing of pain signals in the peripheral and central nervous systems. Phantom limb pain has been long described as the perception of pain in the missing (amputated) limb,[61, 62] and may be indicative of alterations in the processing of pain signals. Although it is unknown how alterations in pain processing might influence the incidence and prevalence of secondary musculoskeletal pain problems, such as LBP, there are several plausible explanations.

In the general population, people with LBP display generalized hypersensitivity to pain that is reflective of central sensitization.[63] Central sensitization is the increased neuronal responsiveness to a stimulus due to prolonged or strong activity in the dorsal horn neurons that may be associated with an episode of pain or prolonged pain.[64] It is plausible that the pain stimulus associated with amputation could elicit central sensitization and increased pain

sensitivity, putting persons with amputation at risk for developing secondary pain conditions. Pain sensitivity is typically evaluated by assessing thresholds and tolerance to pain using a variety of modalities for stimuli, including: mechanical (pressure), electrical, and thermal (cold/heat).[63] Changes in pain sensitivity also can be measured after either an inhibitory stimulus (conditioned pain modulation), or a facilitory stimulus (temporal summation) to further elucidate central mechanisms of pain inhibition or facilitation. Specific alterations in pain processing that have been reported in people with LBP include local[65, 66] and widespread[65-70] hyperalgesia and enhanced temporal summation of pain signals.[71-74] Although people with chronic pain conditions such as osteoarthritis, fibromyalgia, and chronic fatigue syndrome also typically display decreased inhibition of nociceptive signals (conditioned pain modulation),[75-77] most studies report that people with chronic LBP display normal inhibition of pain signals.[78-80]

Pain sensitivity has been examined to a limited extent in persons with amputation. In a small sample, Li et al.[81] reported that persons with traumatic amputation and phantom limb pain displayed decreased thresholds for sensation and pain with electrical stimuli in the unaffected limb, suggesting central sensitization. Further, Vase et al.[82] reported that people with upper limb amputation and phantom limb pain display decreased thresholds for pressure and cold stimuli, and enhanced temporal summation of pain signals. Inhibition of nociceptive signals has not been explicitly examined in person with amputation, but it is plausible that those with phantom limb pain may display decreased inhibition of pain similar to people with other chronic pain conditions. Although the mechanisms of altered pain processing are similar in persons with amputation and people with LBP, to our knowledge, no investigators have examined the neurophysiology of pain in patients with amputation and secondary musculoskeletal pain

problems to determine whether central sensitization places them at greater risk for secondary pain conditions.

Alterations in pain-processing areas of the brain in persons with amputation are also consistent with changes reported in otherwise uninjured individuals with LBP. For example, thalamic structural variations and, more specifically, decreases in gray matter of the posterolateral thalamus have been reported in people with amputation.[83] These changes appear to be positively correlated with duration of time since amputation, suggesting that they may be related to reduced afferent input.[83] Further, Lotze et al.[84] reported shifts in motor and sensory cortical activation patterns during movement in patients with phantom limb pain compared to pain-free persons with amputation, while Makin et al.[85] reported cortical reorganization of the sensorimotor cortex following arm amputation regardless of phantom limb pain. Collectively, these data suggest that neuroplastic changes associated with chronic pain in persons with amputation may involve cortical reorganization.[84] Similar alterations in brain morphology, including reduced density of gray matter in the dorsolateral prefrontal cortex, the thalamus, and the middle cingulate cortex has been reported in patients with LBP without amputation.[86] Although similar neuroplastic changes have been observed in some people with limb amputation and in people with LBP,[87] whether the similarities in mechanism might be related to the development of LBP in persons with LLA requires further investigation. Identifying the contribution of altered pain processing to LBP in patients with amputation could inform the development of more targeted and individualized interventions

Personal Factors

The link between personal demographics and LBP has been well studied in the general population. Prevalence of LBP has been reported to increase with age (up to 65), [88-92] with onset

typically occurring in the third decade of life.[88, 92] Race and ethnicity have also been investigated and the data supports the observation that Caucasians, Western Europeans and North Africans are more likely to experience LBP than African Americans, Caribbeans and Latin Americans.[88, 93] However, reports of gender prevalence for LBP are vastly inconsistent.[88-90, 93, 94] Age, race, and gender have also been studied in persons with LLA. Traumatic amputations commonly occur in a younger population,[95] with as many as 63% of military service members with LLA being less than 30 years of age.[96] Non-traumatic LLA secondary to various pathological conditions such as diabetes mellitus and cancer are more frequently seen in individuals greater than 60 years of age.[95] In a study of 255 patients with traumatic and non-traumatic LLA between the ages of 19-86, age was shown to be modestly but significantly correlated ($r = .12$, $p = .05$) with whether participants experienced LBP.[97] Distinct gender and race features have also been reported in previous research with the majority of patients with traumatic and non-traumatic LLA being male[98, 99] and Caucasian.[99, 100] However, whether these demographic characteristics are associated with higher prevalence of LBP experience after LLA remains unexamined.

Obesity has also been identified as a strong risk factor for LBP in the general population.[89, 90, 101] In patients with LLA, obesity appears to be prevalent and dependent on the level of amputation, with 38% of persons with transtibial, 48% of persons with transfemoral, and 64% of persons with bilateral amputation presenting with noticeable clinical signs of obesity.[102] In support of the potential link between obesity and LBP, patients with LLA and LBP appear to have body mass index ratios above 50% of the recommended ratio compared to their counterparts without LBP.[13] The excess weight gain appears to be substantial and most common within the first two years after LLA,[103] which may be attributed to the sedentary lifestyle immediately after amputation.[104 13]

Maintaining a healthy weight is commonly a challenge for patients with LLA due to difficulties associated with participating in exercise and sports activities.[10] Given the previous reports of increased risk of chronic LBP development as a result of inactivity in the general population,[90, 92] the reported reductions in physical activity levels after LLA[105, 106] inherently increase the risk of LBP in this patient population. While participation in recreational or competitive sports has been reported in 32-60% of patients with LLA,[96, 107] there are fewer barriers in younger individuals who are more likely to achieve higher levels of physical performance due to accelerated rates of recovery and early fitting of running-specific prostheses.[108, 109] Conversely, up to 46% of older persons with LLA become non-ambulatory one year post-amputation, which may place them at a higher risk of developing chronic LBP.[110] Although clinicians often attribute functional difficulties in this population to problems with the amputation and the prosthesis, LBP can also independently restrict activity levels in patients with LLA and warrants further investigation.[111, 112]

A number of physical characteristics have also been identified as risk factors for non-specific LBP in the general population, such as altered muscle strength/endurance, leg length discrepancy, or previous history of LBP.[92, 113-117] In persons with LLA, greater iliopsoas muscle length but reduced back extensor strength and endurance have been associated with the presence of LBP.[15] Leg length discrepancy as a source of structural malalignment, including pelvic obliquity and functional scoliosis,[118] has also been suggested as a potential cause of LBP after LLA but with conflicting supporting evidence. For example, in a study of 113 Finnish war-disabled service members with amputation, those with unilateral LLA and LBP with mild and occasional symptoms had a mean leg length discrepancy of 6.1 mm as compared to a 21.7 mm discrepancy for those who reported severe and constant symptoms.[119] In other studies, however,

no correlations have been reported between LBP and leg length discrepancy in persons with LLA.[120, 121] Previous history of LBP in the general population has also been suggested to almost double the risk of future episodes of LBP.[116, 117] In patients with LLA and LBP, however, only less than 20% recall having LBP prior to their amputation,[8, 9] and in most cases directly attribute their LBP to their LLA.[10]

Psychological Factors

Beyond biologic factors, as an individual with LLA reintegrates within the community, additional psychological factors can affect the risk for LBP and its eventual chronicity. Presence of psychological risk factors in the general LBP population are suggested to affect the frequency and intensity of follow-up medical care and the choice of interventions; whereas in their absence the patient has enhanced potential for quick recovery.[122] Recent evidence further suggests that targeting psychological factors in patients with LBP, particularly when they are at high levels, does seem to lead to more consistently positive results than either ignoring them or providing omnibus interventions regardless of psychological risk factors.[123] In the general population, moderate to strong associations have been reported between onset and chronicity of LBP with various psychological conditions such as depression, pain catastrophizing, passive coping strategies, fear-avoidance beliefs and somatization.[122-126] However, the influence of these psychological factors on the experience of LBP after LLA has not been fully evaluated.

Depressive mood has been related to the onset of LBP, higher levels of LBP intensity, poorer treatment outcome and transition from acute to chronic LBP.[127, 128] To this end, depressive mood has also been reported as a significant predictor of the level of LBP intensity and bothersomeness in patients with LLA.[12] Given the much higher rates of depression in

patients with LLA as compared to the general population,[129, 130] presence of depressive mood may play an important role in the increased risk for chronic LBP in this patient population. There is also a growing recognition that particular kinds of coping mechanisms such as pain catastrophizing (defined as the tendency to focus on, ruminate, and magnify pain sensations) are correlated with the transition from acute to chronic LBP and may be associated with poor treatment outcomes in the general population.[128, 131] Prospective studies suggest that passive coping strategies, especially high levels of pain catastrophizing before an amputation, are associated with development and higher intensity of phantom limb pain and disability.[132-135] However, the extent to which passive coping strategies could influence the LBP experience after LLA remains unknown.

Fear of movement or injury (kinesiophobia) is another important predictor of LBP development and chronicity that could lead to severe disability in the general population.[136, 137] This fear of movement can impede the rehabilitation process and cause dysfunctional pain-avoidance movement patterns that may lead to the development of secondary LBP after LLA. To this end, patients with higher fear-avoidance scores are more likely to have worse outcomes at 3, 6, and 12 months.[122] Although, it stands to reason that patients with LLA may develop beliefs about their condition that may cause them to become fearful of moving and engaging in daily activities, evidence of kinesiophobia in patients with LLA has not been previously evaluated.

Similarly, somatization is another prevalent psychological condition in patients with LBP that includes increased reports of widespread muscle pain located along the whole spine as well as to the legs and the head.[138] Somatization may also be related to presence of sleep disorder, anxiety, and symptoms of depression.[138] Higher somatization scores have been previously correlated with higher intensity of pain and grater disability, failure to return to work

at 3 months and increased likelihood of a worse outcome at 1 year in patients with LBP.[122, 139] Evidence of somatization has also been previously reported in patients with traumatic LLA and neuropathic pain with the resulting abnormal sensory processing leading to locomotor dysfunction and body image disturbances.[140] However, a number of factors such as time since amputation, time since first prosthesis, duration of daily prosthesis use, and high prosthesis satisfaction have shown to be negatively correlated with somatization.[141] Given the evidence suggesting that psychosocial factors can influence the outcome of rehabilitation, more research efforts are warranted for developing clinical tools to identify when and how psychosocial factors could be utilized in clinical decision making to improve patient-related outcomes.[142]

Alterations in central pain processing are also influenced by psychosocial and cognitive factors such as pain catastrophizing, attention, stress, and expectation.[64] People with amputation have been reported to display more depressive symptoms, greater anxiety, lower quality of life, and emotional disturbances.[143] Further, neuropathic pain in persons with amputation has been associated with depression, post-traumatic stress disorder, and catastrophizing.[144] It has also been reported that alterations in pain sensitivity and temporal summation of pain, as well as cortical responses to painful stimuli, were modulated by pain catastrophizing.[82, 145] These psychosocial factors present in some patients with amputation and neuropathic pain, have also been associated with chronic-recurrent LBP and alterations in pain processing.[146, 147] Although no specific association was previously reported between presence of phantom limb pain and psychological symptoms in a small study,[143] strong evidence in support of the relationship between presence of psychosocial risk factors, alterations in central processing of pain, and LBP in patients with LLA remains scant.

Social Factors

The effects of social factors such as cigarette smoking, alcohol use, marital status, occupation, and income on the experience of LBP have been under extensive investigation in the general population. For example, findings from systematic reviews including cross-sectional and longitudinal studies have revealed that both current and former smokers have a higher prevalence and incidence of LBP than "never smokers", but the association is fairly modest.[148, 149] In military personnel with amputation, 21% report smoking cigarettes on a regular basis,[150] while other studies have found that 37-48% of males with amputation are current cigarette smokers.[100, 110] Although strong evidence linking cigarette smoking and LBP after LLA is lacking; one small study reported no difference in frequency of cigarette smoking between persons with transfemoral amputations with and without LBP.[120]Alcohol consumption has also been found to be greater in those with LBP in the general population.[92] In military personnel with amputation, alcohol consumption and substance abuse, along with probable alcohol addiction, is more prevalent than in their non-amputee counterparts.[151] However, research evidence in support of the association between alcohol consumption and LBP after LLA does not currently exist. Being married is another social factors associated with higher risk of developing LBP in the general population compared to those who are divorced or single.[90] Although most reports indicate that the majority of individuals with LLA are married,[96, 150, 152, 153] marital status in at least one cohort study was shown not to be associated with either the intensity or bothersomeness of LBP in patients with LLA.[12]

Individuals with occupations involving heavy lifting/pushing/pulling and driving have historically been identified to be more prone to development of LBP in the general population. [90,

[92, 154-156] As for the military population, predictors of LBP include jobs involving lifting and wearing body armor,[157] with a higher incidence seen in construction workers, auto mechanics, and law enforcement personnel.[158] However, both military and non-military individuals with LLA often return to employment in less physically demanding occupations,[150, 159, 160] which may decrease their risk of developing occupation-related LBP. Lower socioeconomic class and lower levels of education have also been found to correlate with LBP in the general population.[90] Enlisted rank and service in the Navy, Army or Air Force have been identified as risk factors for LBP in a military sample.[161] Education at or below a high school level has been reported in 27-60% of service members with amputation[96, 152] and 78% of those with amputations of dysvascular or diabetic aetiologies.[100] Of the service members with (traumatic) amputation, 31% were junior enlisted, 49% mid to senior enlisted, and 20% were officers.[96] In a sample of individuals with dysvascular or diabetic amputations, 44% reported an income of <$25,000, 37% between $25,001 and $50,000, and 19% an income >$50,000.[100] Further investigations are needed to determine the potential relationships between occupation, socioeconomic class, level of education and salary with LBP experience after LLA.

Conclusions

In the United States, an estimated 185,000 persons undergo limb amputation each year as a result of dysvascular disease (54%), trauma (45%), or cancer (1%), with the projected total number of people living with limb loss doubling to up to 3.6 million by the year 2050.[162] In general, most amputations are major LLA (excluding toes) with increasing prevalence rates due to dysvascular diseases such as diabetes mellitus.[162, 163] Despite the high prevalence of LBP after LLA, there currently exists a lack of understanding to identify any definite pathologic processes or anatomic sources of pain. A growing body of evidence from studies of LBP in the general

population suggests that it is no longer appropriate to try to subclassify LBP solely using a biomedical construct, and that a successful classification system must include biomedical, psychological, and social assessments.[25] Given the multifactorial nature of LBP after LLA, a more comprehensive understanding of how amputation influences these biopsychosocial risk factors will further allow effective stratification of care for LBP after LLA, where patients are screened and placed in interventions designed to target their specific biopsychosocial risk profiles. The aim of this special communication was to integrate evidence originating predominantly from prospective studies on biopsychosocial correlates of LBP after LLA into a coherent model that could help generate new research questions and improve our understanding of the LBP experience in this unique patient population.

The proposed biopsychosocial model could be useful in identifying risk factors for early identification of patients at risk for LBP and testing the effectiveness of different approaches aimed at reducing chronic LBP-related disability after LLA. Currently, the results from psychosocial interventions for LBP in the general population consistently show only small to moderate effects.[16, 164, 165] However, a multidisciplinary approach that addresses all three components of the biopsychosocial model of LBP may provide a more appropriate solution aimed at the multifaceted nature of the LBP experience after LLA.[16] A number of prospective studies have shown that psychosocial factors influence how patients respond to rehabilitative and surgical treatment, thus indicating the interaction between physical and psychological factors are important in determining the outcome of a given treatment for LBP after LLA.[17]

Another potential approach would be to implement a stratified care approach, where patients with LLA are screened for known biopsychosocial risk factors using reliable and valid tools, and then referred to interventions designed to target their specific problem and risk

profile.[16] To this end, use of a stratified approach, by use of prognostic screening with matched clinical pathways has shown promising results in management of LBP in primary care for the general population.[166] However, the current challenge to implementation of a stratified care approach is the identification and development of a validated risk factor profiles that could be used as a clinical guide to stratify patients with LLA into streams of care that optimize their chance of a good outcome for treatment of LBP. Given that some factors exert an influence on outcome regardless of treatment, whereas some only influence response to specific treatment,[16] additional clarity is needed to determine which predictors of outcome are prognostic factors and which are potential treatment effect modifiers to help guide best practice treatments and the prevention of disability.[167] Additional research and insight are needed to determine more effective approaches to mitigate or manage LBP after LLA.

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COMPREHENSIVE INVITED REVIEW

Impact of Traumatic Lower Extremity Injuries Beyond Acute Care: Movement-Based Considerations for Resultant Longer Term Secondary Health Conditions

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Significance: Advances in field-based trauma care, surgical techniques, and protective equipment have collectively facilitated the survival of a historically large number of service members (SMs) following combat trauma, although many sustained significant composite tissue injuries to the extremities, including limb loss (LL) and limb salvage (LS). Beyond the acute surgical and rehabilitative efforts that focus primarily on wound care and restoring mobility, traumatic LL and LS are associated with several debilitating longer term secondary health conditions (*e.g.*, low back pain [LBP], osteoarthritis [OA], and cardiovascular disease [CVD]) that can adversely impact physical function and quality of life.
Recent Advances: Despite recent advancements in prosthetic and orthotic devices, altered movement and mechanical loading patterns have been identified among persons with LL and salvage, which are purported risk factors for the development of longer term secondary musculoskeletal conditions and may limit functional outcomes and/or concomitantly impact cardiovascular health.
Critical Issues: The increased prevalence of and risk for LBP, OA, and CVD among the relatively young cohort of SMs with LL and LS significantly impact physiological and psychological well-being, particularly over the next several decades of their lives.
Future Directions: Longitudinal studies are needed to characterize the onset, progression, and recurrence of health conditions secondary to LL and salvage. While not a focus of the current review, detailed characterization of physiological biomarkers throughout the rehabilitation process may provide additional insight into the current understanding of disease processes of the musculoskeletal and cardiovascular systems.

Keywords: amputation, biomechanics, cardiovascular disease, limb salvage, low back pain, osteoarthritis

SCOPE AND SIGNIFICANCE

Extremity trauma, including limb loss (LL) and limb salvage (LS), is commonly associated with an elevated risk for secondary health conditions (*e.g.*, low back pain [LBP], osteoarthritis [OA], cardiovascular disease [CVD]) that can significantly limit physical function, reduce quality of life (QoL), and life expectancy. This review

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provides an extensive commentary regarding resultant secondary health effects of extremity trauma in service members (SMs), with a particular focus on functional outcomes and quality of movement.

TRANSLATIONAL RELEVANCE

Physiologic biomarkers provide an opportunity to enhance translation in future work to examine the pathophysiology of the secondary health conditions associated with traumatic LL from a basic science perspective. While this approach is yet to be fully explored and thus was not a primary focus of this review, such biomarkers may augment traditional analyses and support more comprehensive risk characterization, thereby allowing clinicians and researchers to better mitigate disease onset or progression.

CLINICAL RELEVANCE

The increased prevalence of secondary health effects following traumatic extremity injuries places a significant physical and psychosocial burden on SMs with LL and LS. Altered movement patterns often result in mechanical loading of the spine and lower extremities, potentially increasing the risk of LBP and OA. Adopting a biopsychosocial model of treatment/care may allow clinicians to utilize a multifaceted approach to treat chronic pain and dysfunction associated with resultant health effects of LL.

BACKGROUND

Musculoskeletal disorders are the most prevalent source of disability in the United States.[1,2] As a result, the annual direct costs associated with treatment total a substantial $900 billion.[3] Among these, extremity amputation, or LL, is projected to affect an estimated 3.6 million people by the year 2050.[4] Approximately 185,000 individuals undergo either an upper or lower extremity amputation annually, primarily due to trauma, dysvascular disease, and/or osteosarcoma.[5–7] While the incidence of LL due to dysvascular etiologies has steadily risen among the civilian sector, trauma remains a leading source of LL within the Military Health System. However, prior estimates of the current/future impact of LL do not include SMs injured during combat nor do they consider individuals with LS; an alternative to amputation in which heroic measures are undertaken by the military surgical teams at all echelons of care to preserve as much form and function of the traumatically injured limb as possible. Despite these surgical efforts and advances in orthotic technology, many with LS are unable to achieve preinjury functional outcomes, much like those with LL.

The combat theaters of Operations: Enduring Freedom (OEF), Iraqi Freedom (OIF), New Dawn, Inherent Resolve, and Freedom's Sentinel were characterized by high-energy munitions and explosives. With advances in personal protective equipment, field-based trauma care, and surgical techniques, injuries sustained as a result of these often-improvised devices are now survivable at higher rates than conflicts past. However, traumatic extremity injuries, including LL and LS, remain a hallmark casualty of recent conflicts. Across all services, 52,351 military personnel have been wounded in action since 2001[8]; more than half of evacuated SMs have sustained extremity injuries and nearly a quarter of these are open fractures.[9] In addition, 1,703 SMs sustained injuries requiring major (or multiple) limb amputation (As of October 1, 2016; Data source: EACE-R). The decision to amputate a limb may be made in as few as 24 h post-trauma, during the first hospitalization as a secondary surgical intervention, or potentially years after LS (*i.e.*, delayed amputation).[10–13] Factors contributing to the decision include the extent and severity of injuries and resources available during the rehabilitation process.[14] Recent evidence suggests that SMs who undergo LS will typically experience more expansive complications than individuals who undergo amputation.[15–17] LS has been associated with significantly higher rates of rehospitalization, greater numbers of surgical procedures, and higher rates of surgical complications.[18,19]

Initial wound care and rehabilitation after LL and/or LS are critical to the recovery process. Such efforts are generally categorized by nine distinct phases, each with specific goals and objectives.[20] The complexity and interdependence between each phase elucidate the need for an efficient interdisciplinary approach within the overall rehabilitation paradigm. Despite these comprehensive and substantive efforts, persons with LL and LS are at an increased risk for acute secondary health conditions such as phantom limb pain, wounds/sores, vascular and nerve damage, infection, decreased physical function, and psychosocial issues. Furthermore, beyond these acute conditions, persons with LL and LS are also at an elevated risk for longer term complications including LBP, OA, and CVD, among others. Importantly, once the disease progression initiates, these longer term resultant conditions will plague these individuals for life, as SMs with extremity trauma are typically younger than 30 years at the time of

injury and thus will continue living with their injuries for several decades.[17]

The long-term economic burden of trauma-related LL and LS is significant. Edwards *et al.* predicted the long-term (40 year) cost of trauma repair, rehabilitation, and lifelong prosthetic support of British soldiers wounded in Afghanistan to be approximately $444 million.[21] In the United States, the estimated average lifetime cost of treatment for unilateral lower LL is $342,716 and $1.4 million for Vietnam and OIF/OEF veterans, respectively.[22] However, such estimates are likely conservative, not fully accounting for costs associated with novel technology/repairs or, perhaps exponentially more economically burdensome over the longer term, for the wide range of healthcare costs associated with the treatment of secondary health conditions. The ability to evaluate, predict, and ultimately treat these resultant health conditions would not only help reduce these costs but also, and most importantly, preserve and/or improve function and QoL for those with LL and LS.

The risk for secondary health conditions is often related to physiological adaptations to trauma or pervasive surgical complications, poor biomechanics, and/or the prosthetic (orthotic) device itself. For SMs, in particular, the young age at which these injuries occur likely presents a unique challenge over the longer term and further highlights the importance for understanding resultant health conditions secondary to extremity trauma. Notably, the cumulative effects of many years of functional adaptations during gait and movement with extended prosthetic/orthotic device use in otherwise young and active SMs remain unclear.[23,24] This is an important distinction from civilian populations as a majority of civilians with LL are over the age of 50, incurred LL as a result of vascular damage/complications, are likely less active, and may present with different resultant health conditions/outcomes for less time.[25] Thus, as a preliminary step toward addressing this knowledge gap, the purpose of this review is to provide a commentary regarding resultant health conditions associated with high-energy extremity trauma, with a primary focus on biomechanical features of movement and associated functional limitations. In particular, we highlight considerations for longitudinal care aimed at maximizing QoL, for those with both LL and LS.

DISCUSSION

Low back pain

The World Health Organization describes LBP as any pain or discomfort for a variable duration in the lumbar spine region.[26] The onset of pain may occur suddenly, coincident to a singular traumatic event, or develop over time with age or as the result of repeated microtrauma from a given (or set of) activity(ies). Often, LBP is considered idiopathic, as pain may be present without pathoanatomical evidence of disease or structural abnormality. LBP costs nearly $100 billion annually in the United States, with a majority of this cost associated with lost wages and decreased productivity.[27] While cross-sectional figures indicate that chronic LBP affects up to 33% of adults in the general population, the incidence in persons with LL who report LBP secondary to trauma is nearly double (52–76%).[28–31] Along with this significantly higher prevalence, nearly 50% of persons with LL have reported LBP as "more bothersome" than either residual or phantom limb pain and as having a significant reduction in overall QoL metrics.[28,30,32] While the exact etiology of LBP within this population is unclear, there is a growing body of evidence suggesting that altered lumbopelvic mechanics during the (repetitive) gait cycle likely influences such risk.

Persons with lower LL frequently develop altered movement patterns to maintain balance and achieve forward progression in walking. Movement patterns can be influenced by the following, either individually or in combination: socket fit/prosthetic alignment, general deconditioning, leg length discrepancies, complications within the residual limb, and muscular imbalances.[33,34] More specifically, altered movement patterns during gait affect trunk and pelvis mechanics and contribute, at least in part, to the increased incidence of LBP in persons with lower LL and may be dependent on the extent of injury or ultimate level of amputation.[35–38] These alterations and asymmetries may increase loads on the lumbar spine during gait which, when considering the repetitive gait cycle, over time may thus contribute to the occurrence or recurrence of LBP. For example, persons with transfemoral LL tend to exhibit 10° of anterior pelvic tilt, which is considered to be a compensatory mechanism to assist in the ability to achieve hip extension during gait. Increased anterior pelvic tilt is associated with increased lumbar lordosis, which is linked to an increased incidence of LBP in persons with LL.[28,39] Previous work has demonstrated that increased loads on the lumbar spine are a direct source of LBP in the general population.[40,41] Mechanical loading of the passive and active structures of the spine is affected by both internal and external loads, such as forces produced by muscular activation, ligamentous tension, gravity, and inertia.[42]

These loads can be significant, as potentially small alterations in trunk (which accounts for nearly 2/3 of the body's mass) movement may increase joint reaction loading due to increased muscular contractions of the surrounding musculature.[43] The increased demand on the active structures (muscles) may lead to increased forces and joint loading on the passive structures (discs and vertebrae). The accumulation of these altered loads over time has the potential to augment degenerative joint changes in the spine.[40]

Similar to uninjured individuals with LBP, persons with transfemoral LL exhibit irregular trunk–pelvis coordination and movement variability.[44] Specifically, persons with LL tend to walk with a large lateral trunk lean toward the affected side; a possible neuromuscular strategy/compensation to assist in forward progression during gait.[42] This frontal plane motion has been reported to increase peak joint reaction forces and moments asymmetrically in the lumbar spine (L5-S1 integration specifically) in this population. A recent report suggested this observed frontal plane motion as a possible mechanistic pathway through which recurring exposure to altered trunk motion and cumulative spinal loading may contribute to LBP in persons with lower LL.[42] Persons with transfemoral LL (with current LBP) exhibit larger axial trunk rotations when compared to those without LBP, which may subsequently affect vertebral disc degeneration and potentially contribute to LBP recurrence.[45,46] Previous evidence demonstrated degenerative changes in the lumbar spine via radiographic imaging in 76% of persons with LL, potentially supporting the role of increased trunk motion leading to degenerative changes in this population.[47]

While LBP is commonly cited as a secondary health effect of LL, persons with LS may also experience LBP as a result of altered movement patterns during gait and functional activities.[48] Persons with LS typically experience reduced ankle function, which is associated with altered gait mechanics and increased metabolic cost.[34,49,50] However, the influence of distal LS on proximal (trunk/pelvis) biomechanics remains unstudied to date. Currently, a paucity of evidence exists relative to the prevalence of LBP in the LS population. Therefore, further work is needed to elucidate the relationship between LS and the development of LBP.

In summary, LBP has been reported as the most important health-related physical condition contributing to a reduced QoL among veterans who had sustained a traumatic lower extremity amputation over 20 years prior.[32] Thus, identifying factors contributing to the development and recurrence of LBP, such as a widely prevalent and "bothersome" secondary health concern, is critical for improving long-term health. Abnormal mechanical loading of lumbar spine, altered trunk and pelvis coordination, and psychosocial factors may influence the prevalence of LBP in this population. Therapeutic interventions that address the underlying impairment(s) in trunk neuromuscular responses and/or motor control strategy may also contribute to reducing the prevalence and incidence of LBP among SMs with lower extremity trauma, thereby improving longer term functional outcomes by mitigating a significant secondary impairment with a substantial adverse impact on daily activities. Further evidence is needed to understand the relationship between these risk factors and the incidence of LBP in persons with LL. In particular, no studies to date have evaluated the influence of different prostheses or orthoses on the incidence of LBP in the traumatic LL and LS populations.

Osteoarthritis

The National Institute of Arthritis and Musculoskeletal and Skin Diseases describes OA as a joint disease affecting the cartilage, often characterized by pain and stiffness within a joint and limitations in physical function.[51] The primary pathology is articular cartilage deterioration, although evidence suggests that possible morphological changes of bone are reflective of disease onset. Within the joint, articular cartilage functions to dissipate forces sustained by the bony structures throughout motion. During activities such as walking or running, when the loading velocity and intensity of the structures are increased, the cartilage's ability to dissipate forces is reduced.[52] In the general population, mechanical loading of the knee joint during walking has been associated with the presence, severity, and progression of knee OA.[53–56] Persons with unilateral lower LL are 17 times more likely to suffer from knee OA in the intact limb when compared to able-bodied individuals.[57]

As previously noted, persons with LL frequently develop altered movement patterns during gait. Of particular importance here, those with unilateral LL preferentially utilize their intact limb, leading to increased and prolonged loading of the intact joints. Mechanical alterations in static and dynamic alignment of the knee joint may affect joint loading as increased forces are incurred through medial or lateral aspects of the joint. The external knee adduction moment (EKAM) is a vastly reported risk factor for knee OA based on its relationship with internal loading of the medial joint surface.[58] The size of the EKAM and its respective angular impulse

are associated with knee OA severity and progression.[53,55,59,60] During gait, individuals with lower LL asymmetrically load their intact limb to a greater extent than their involved limb, suggesting that mechanical factors play a role in the increased incidence of knee OA in this population.[36,61] For example, Lloyd *et al.* identified larger peak knee adduction moments in the intact relative to involved limb.[62] This increased mechanical loading may be explained by decreased push-off power and ground reaction forces demonstrated with conventional prosthetic feet.[61,63] Push-off power generated by the prosthetic foot instance may affect the ground reaction forces at heel strike in the intact limb as the velocity of an individual's center of mass changes from an anterior and inferior direction to an anterior and superior direction during gait.[64] The redirection of the center of mass is caused by the ground reaction impulse through the gait cycle, crudely relative to double-limb support.[64] If the prosthetic stance foot lacks adequate push-off power to propel the center of mass anteriorly, the intact limb must compensate by performing more work to move the center of mass anterior and superior, resulting in increased ground reaction forces and loading of the intact limb.[61] Morgenroth *et al.* suggested that by utilizing a prosthetic foot with increased push-off power, the peak EKAM of the intact limb may be reduced and therefore potentially decreasing the OA risk.[61] This was supported as a powered ankle–foot prosthetic was able to decrease the EKAM and vertical ground reaction force in persons with lower LL, however, the prosthetic used was unable to alter the knee joint loads of the intact limb.[65] Similar to LBP, the progression and severity of OA may be further amplified by psychosocial determinants; anxiety, depression, coping strategies, and stress have also been associated with increased pain in patients with OA.[66–68]

OA is not exclusive to the LL population as individuals with LS present with similar (sometimes larger) gait and movement deviations. As high as 95% of OA diagnoses among combat-wounded SMs are post-traumatic in origin.[69] Chronic pain, nerve damage, and volumetric muscle loss are common barriers to LS rehabilitation and may serve as confounding factors in the development of OA treatment plans.[70,71] Ankle–foot orthoses (AFOs) are commonly used to assist ankle function or offload painful structures.[72] Optional therapies that include sports medicine-based interventions utilizing a dynamic AFO (*e.g.*, the Intrepid Dynamic Exoskeletal Orthosis) are available to LS patients. Such devices are designed to improve functional performance on tasks such as walking, changing directions, sit-to-stand, and ascending stairs.[48] While dynamic AFOs are suggested to improve functional capabilities, evidence is inconclusive in its ability to positively alter gait parameters related to OA as well as the effects of long-term use.[34,73,74]

Treatment modalities focused on reducing symptoms and OA disease progression in persons with LL and LS are vital to improving QoL. The Osteoarthritis Research Society International recommends biomechanical interventions, intra-articular corticosteroids, exercise (land and water based), self-management and education, strength training, and weight management.[75] Autologous platelet-rich plasma (PRP) therapy is a therapeutic intervention that delivers high concentrations of growth factors to an area to stimulate healing.[76] Recent evidence suggests that PRP may provide relief of knee OA symptoms in younger patients within the early stages of cartilage degeneration.[77–79] Strength training (weight and body-weight training) and exercises such as t'ai chi have demonstrated the ability to improve overall function in decreasing pain in OA patients and may also serve to assist in weight management.[80,81] Weight reduction is considered a pragmatic therapy for knee OA as overweight individuals demonstrate a high prevalence of knee OA and the risk of severity progression increases 35% for every 5 kg of weight gain.[82] Strength training and weight management are considered integral aspects of the rehabilitation paradigm for persons with LL as deficits in strength and increases in weight influence gait, joint loading, movement efficiency, and cardiovascular health. Canes, knee braces, and foot orthotics are other potential treatment options to decrease movements at the knee, reduce pain, and improve function.[83–85]

In summary, biomechanical factors likely play a substantial role in the risk for OA secondary to extremity trauma, whether LL or LS. While the prevalence of OA in LL and LS populations may decrease as technological improvements in prostheses and orthoses are realized, further evidence is needed to determine the specific relationship between different classes or features of these devices and OA risk factors. Unfortunately, recent technological advancements in prosthetic devices have outpaced orthotic devices, the benefits of which are evident in the biomechanical characteristics of persons with LL versus LS. Nevertheless, LS typically presents with more complex neurovascular injuries and other unique challenges, which can negatively affect functional outcomes.

Cardiovascular disease

CVD is defined by a vast array of diseases affecting the heart and blood vessels.[86] CVD may present

as coronary artery disease, stroke, arrhythmias, cardiomyopathy, heart disease, peripheral artery disease, aneurysms, venous thrombosis, and/or carditis.[86,87] While CVD is largely preventable, it remains the leading cause of death worldwide, particularly in lower socioeconomic demographics.[86] The American Heart Association reports there are ~85 million individuals with CVD in the United States, causing a staggering 2,200 deaths each and every day.[88] This is accompanied by direct and indirect costs of nearly $315 billion.[89] Risk factors for CVD include, but are not limited to, family history and genetics, high cholesterol and lipids, high blood pressure, diabetes, metabolic syndrome, obesity, and kidney disease.[89] In addition, significant combat trauma may be a risk factor for the development of CVD.[90–92] For example, Hrubec and Ryder conducted a 30-year follow-up of World War II veterans with lower LL and demonstrated that the relative risk of CVD mortality was increased 2.4–4 times that of persons with LS.[90] Similarly, Modan *et al.* reported significantly higher mortality rates of persons with traumatic lower LL when compared to able-bodied controls, suggesting that CVD was the primary cause (21.9% vs. 12.1%, $p < 0.001$).[91]

The pathophysiology of increased mortality rates may be a result of systemic and/or regional hemodynamic effects of trauma.[91,93–97] Obesity and hypertension secondary to decreased overall activity levels may lead to insulin regulation complications in persons with LL.[97] When compared to uninjured controls with no difference in body mass index, blood pressure, or lipid levels, persons with LL exhibited significantly higher increased fasting plasma insulin levels as well as insulin resistance.[96] Increased plasma insulin levels and insulin resistance are risk factors for atherosclerosis and metabolic syndrome, considered precursors to CVD. The role of psychological stressors in the development of CVD is not well understood; however, psychosocial factors have demonstrated involvement in the pathogenesis of CVD.[98,99] Depression and post-traumatic stress disorder have been associated with increased incidence of CVD, while veterans with high levels of cynical distrust and anger demonstrate an accelerated progression of atherosclerosis, a risk factor for CVD.[100–102] Limited evidence precludes a definitive relationship between psychosocial factors and CVD risk in persons with LL, and therefore, future work should prospectively examine the relationship between psychosocial factors/stressors and the development of CVD.

Hemodynamically, proximal amputation increases the risk of CVD development based on alterations in proximal arterial flow. Pathogenic mechanisms may include early reflection pulse waves. Early return reflection pulse waves are produced at arterial occlusion sites and have been linked to a myriad of medical complications.[103] An early returned reflection pulse wave creates a second systolic peak, which results in an increase in aortic pressure. The increased aortic pressure generates an increased left ventricular load resulting in left ventricular hypertrophy, atherothrombosis, and ultimately cardiac death.[104] Vollmar *et al.* suggested that persons with traumatic LL above the knee were five times more likely to suffer from abdominal aortic aneurysms when compared to healthy controls.[94] A possible explanation may be that after amputation, blood flow is decreased by ~25% in the terminal aorta due to altered flow paths in the visceral and renal arteries, resulting in a disrupted flow pattern at the aortic bifurcation.[95] Altered flow patterns, paired with increased shear stress along the convex aspect of the aorta and decreased shear stress along the concave aspect, are theorized to damage aorto-iliac blood vessels by increasing hydraulic forces within the aorta.[95] Persons with transfemoral LL should have regular consultations with appropriate medical personnel to assess the risk of abdominal aortic aneurysm.[95]

While the hemodynamic effects of trauma appear to influence CVD risk, addressing modifiable risk factors may be an effective strategy to help decrease CVD risk. It is widely accepted that habitual exercise with activities such as running, walking, bicycling, rowing, and swimming increases aerobic capacity and decreases the risk of CVD. When joined with dietary modifications, regular exercise can effectively reduce excess body weight, another risk factor for CVD. Moreover, the increased risk of CVD in persons with LL highlights the importance of managing modifiable risk factors, engaging in preventative treatment strategies, and adopting an active lifestyle.

SUMMARY

Maintaining an active lifestyle is critically important for physiological health, psychological well-being, and overall QoL. Such guidance is no different for individuals with LL and LS. However, given the limited (but growing) body of evidence relating movement abnormalities to altered musculoskeletal demands that may lead to the development of longer term secondary conditions in this population, additional consideration for the quality of movement during recreational and daily activities is warranted. While the overwhelming focus of recent efforts has been on persons with LL, the aforementioned secondary health conditions are likely also major con-

cerns for those with LS. As such, we posit that an underlying focus of clinical care and future research, in both cohorts, should be toward mitigating concomitant risk for the development or recurrence of chronic pain.

While advances in trauma care and prosthetic/orthotic technologies may eventually mollify acute and subacute secondary health effects of extremity trauma, longitudinal tracking is urgently needed to better understand the mechanisms by which secondary health effects develop and progress in this population. Such efforts should encompass a transdisciplinary team, in which a comprehensive suite of evaluation metrics are employed; for example, traditional clinical evaluation and movement analysis supplemented with local and systemic physiological biomarker analyses and next-generation imaging modalities. In doing so, a better understanding of the specific pathways for the development of these secondary health effects can be realized, thus enabling clinicians to develop and prescribe appropriate treatment interventions. Ultimately, diminishing risk factors relative to the degeneration of joint and cardiovascular function will reduce the overall prevalence of secondary health conditions and improve QoL for our nation's injured SMs and veterans over the longer term.

TAKE HOME MESSAGES

- Living with LL and LS over time leads to increased morbidity and mortality from secondary medical and musculoskeletal problems. Awareness of the long-term health risks associated with LL and LS, as well as the physiologic and biomechanical origin of these risks, is critical to improving outcomes
- Understanding the pathogenesis of the secondary health conditions of traumatic LL and LS and salvage may help guide optimal management in acute, subacute, and chronic phases of care for these individuals
- Reducing modifiable risk factors through patient education, identifying appropriate support systems, encouraging proper gait mechanics, and utilizing the prescription of evolving technologies may help mitigate long-term health conditions

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No competing financial interests exist. The content of this article was expressly written by the authors listed. No ghostwriters were used to write this article. The views expressed in this article are those of the authors and do not necessarily reflect the official policy of the Departments of the Army, Navy, Defense, nor the United States Government.

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Abbreviations and Acronyms

AFO = ankle–foot orthoses
CVD = cardiovascular disease
EACE = Extremity Trauma and Amputation Center of Excellence
EKAM = external knee adduction moment
LBP = low back pain
LL = limb loss
LS = limb salvage
OA = osteoarthritis
OEF = Operation Enduring Freedom
OIF = Operation Iraqi Freedom
PRP = platelet-rich plasma
QoL = quality of life
SM = service member
USUHS = Uniformed Services University of the Health Sciences
WRNMMC = Walter Reed National Military Medical Center

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Biopsychosocial risk factors associated with chronic low back pain after lower limb amputation

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ABSTRACT

Low back pain is a common secondary health condition after lower limb amputation with important implications related to functional capabilities and overall quality of life. Despite the high prevalence of low back pain after lower limb amputation, the underlying etiologies of the disorder remain unknown. This hypothesis-driven communication provides evidence in support of using the multifactorial, biopsychosocial model of low back pain experience in the general population for identification of potential risk factors and rehabilitation targets for low back pain after lower limb amputation. Key findings that link biological, psychological, and social factors and the experience of low back pain in the general patient population with LBP are discussed while highlighting gaps in our current state of knowledge related to the association of these factor and presence of low back pain after lower limb amputation. Importantly, the aim of this communication was not to propose a new model, but rather to organize data originating from prior work into a coherent hypothesis-driven conceptual framework to better understand the need for multifaceted and multidisciplinary intervention approaches for effective treatment of low back pain after lower limb amputation.

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Introduction

Low back pain (LBP) is a common health condition worldwide, with 11–38% of the general population reporting symptoms over a one year period [1,2]. LBP is currently considered the leading cause of disability globally, ahead of 290 other conditions, and is responsible for 83 million years lived with disability [3]. Additionally, LBP is a major source of activity limitation, work absenteeism, and increased cost of medical care throughout much of the world [2,4–6]. LBP is also a common and perhaps more impactful, secondary health condition after lower limb amputation (LLA), with high estimated annual prevalence rates between 50–90% [7–13]. Individuals with LLA often report more LBP after amputation than before [8,9] and in most cases directly attribute their LBP to their amputation [10]. Additionally, presence of LBP daily or several times per week has been associated with moderate to severe physical disability and limitations in performing daily activities in patients with LLA [8,9,13–15]. To this end, LBP is often rated by patients with LLA as more bothersome than phantom or residual limb pain [11], suggesting LBP is an important secondary musculoskeletal condition associated with functional limitation and disability after LLA.

Despite the high prevalence of LBP after LLA, the exact etiologies of the disorder in this population remain unknown, thereby making its treatment exceptionally challenging. Importantly, there are currently no published randomized clinical trials or clinical practice guidelines specifically tailored toward the management of LBP for individuals with LLA. Therefore, there exists a clear need for comprehensive identification of contributing factors to the LBP

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experience after LLA that can serve as a basis for the development of targeted rehabilitation strategies and future research investigations. Here a new application of the multifactorial, biopsychosocial model for LBP, previously developed for the general population [16–18], is proposed as a way of identifying risk factors and potential intervention targets for treatment of LBP after LLA. The objective of this hypothesis-driven communication was to organize data originating from prior studies of the biopsychosocial correlates of LBP after LLA into a coherent conceptual framework. We hypothesized that alterations in biological, psychological, and social factors are related to the development of LBP symptoms and disability after LLA that merit specific attention during the clinical decision making process and for future research efforts to improve patient-related outcomes.

The biopsychosocial model of low back pain

Treatment of LBP has historically centered around the traditional biomedical model of illness, which assumes a direct relationship between regional pathoanatomy and the perception of pain [18]. As such, it was expected that once the anatomical source of LBP is identified, biochemical and/or mechanical treatments of underlying pathoanatomy would result in cessation of pain. Despite leading to successful treatment of many other disease processes, the outcomes of interventions based on the biomedical model have proven to be less than ideal for treatment of LBP [18–20]. One potential reason for the failure of the biomedical model to provide an effective treatment option for LBP is that no single underlying pathoanatomical lesion has been consistently identified [18], with up to 85% of LBP patients left without a precise pathoanatomical diagnosis [21]. Additionally, determining the pathoanatomical sources of LBP frequently lacks interexaminer reliability and adequate evidence for generalizability [22]. The often equivocal outcomes from many "lesion-specific" treatment options such as intra-articular corticosteroid injections [19] and spinal fusion surgeries [20], along with the generally poor predictive value of diagnostic imaging to identify pathoanatomical sources of pain [23], have led to a recent paradigm shift toward a "non-structural" approach for the management of LBP [24].

A growing body of evidence now suggests that successful treatment of LBP should include biological, psychological, and social assessments to comprehensively address the patient's unique pain experience [18]. The so called "biopsychosocial model" of LBP suggests that the patient's perceptions and reactions to pain should also be considered as these factors often lead to unnecessary avoidance of physical activity and social interactions, work absenteeism, and high health care utilization [16,17]. Whereas the pathoanatomy may initiate the pain process, the psychological and social factors appear to play an important role in exacerbating the biological component of LBP by influencing the perception of pain [25]. For example, it has been hypothesized that the presence of mechanical LBP can lead to a pain-generated stress response that could have a negative impact on the endocrine and immune systems, which in turn may negatively affect the cognitive assessment, emotional response, coping strategies and health practices of the individual [26].

The hypothesis

Proponents of the biopsychosocial model argue that the complex, multidimensional nature of LBP does not lend itself to the reductionist view of the biomedical model; instead, the patient's unique biologic, psychological, and social factors must equally be considered [18]. Therefore, the term biopsychosocial implies that the biological, psychological and social factors are interwoven within the context of the patient's overall LBP experience and should be directly and concurrently considered as a part of a comprehensive treatment program [26]. In support of this theory, multidisciplinary treatment approaches that include biopsychosocial components for treatment of LBP in adults have demonstrated positive effects on pain, disability, and health-related quality of life [27,28]. It stands to reason that LLA likely amplifies and/or alters specific components within the multifactorial biopsychosocial model of LBP, previously suggested for the general population. Given that LLA may differentially affect the various components of this model (Fig. 1), we hypothesize that discriminating clinically meaningful sub-groups of patients with LBP after LLA will most likely require assessments of biological, psychological and social domains [22].

Biological factors

Biomechanics

Altered mechanics of gait and movement have been historically proposed to play a causative role in the development and/or recurrence of LBP after LLA [29]. In fact, persons with LLA perceive "uneven postures and compensatory movements" affected by "fatigue" and "prosthesis-related factors" during functional activities as the primary contributors to LBP [30]. Though at the expense of higher metabolic cost of transport [31], compensatory movement strategies adopted after LLA typically involve adaptations to maintain the body's center of mass within the base of support (i.e., improve stability and balance), primarily with a preference for the intact limb, if applicable [32]. During gait, for example, the intact limb (relative to prosthetic limb) is characterized by a longer stance time, shorter step length, wider stride width, and larger vertical ground reaction forces [33]. As the trunk accounts for approximately two-thirds of total body mass [34], altered motions of this segment play a substantial role in post-amputation movement strategies, thereby warranting more trunk-focused biomechanical investigations for assessing potential links with the development and persistence of LBP.

Altered trunk and pelvic movements in persons with LLA have been previously identified in all three cardinal planes, including larger forward trunk lean and flexion-extension range of motion, greater lateral trunk flexion (towards the prosthetic limb) and pelvic obliquity motion, as well as more axial rotations between the shoulders/pelvis or regional/intervertebral motion segments [35,36]. The presence (and likely severity) of LBP further influences such trunk and pelvic movements [37]. For instance, it has been reported that patients with transfemoral amputation and LBP elevate their pelvis on the intact side, minimize their lumbar lateral flexion, and keep their lumbar spine rotated toward the prosthetic limb throughout the gait cycle as compared to patients with transfemoral amputation without LBP [38].

LBP has also been associated with more in-phase mediolateral coordination between the trunk and pelvis [39], which is indicative of inter-segmental rigidity (i.e., "guarding behavior") previously reported in able-bodied individuals who are experiencing LBP [40,41]. Additional evidence suggests that individuals with LLA employ an active mediolateral trunk movement strategy, inferred from increases in generation and absorption of energy between the trunk and pelvis [42,43]. Although actively increasing mediolateral trunk sway is likely an attempt to improve joint stability within the lower extremity by altering lever arms of ab/adductor musculature [44], most notably within the hip among patients with transfemoral amputation [7], such strategies have been associated with LBP/discomfort among able-bodied individuals performing gait training aimed at reducing knee joint loads via trunk lateral flexion [45].

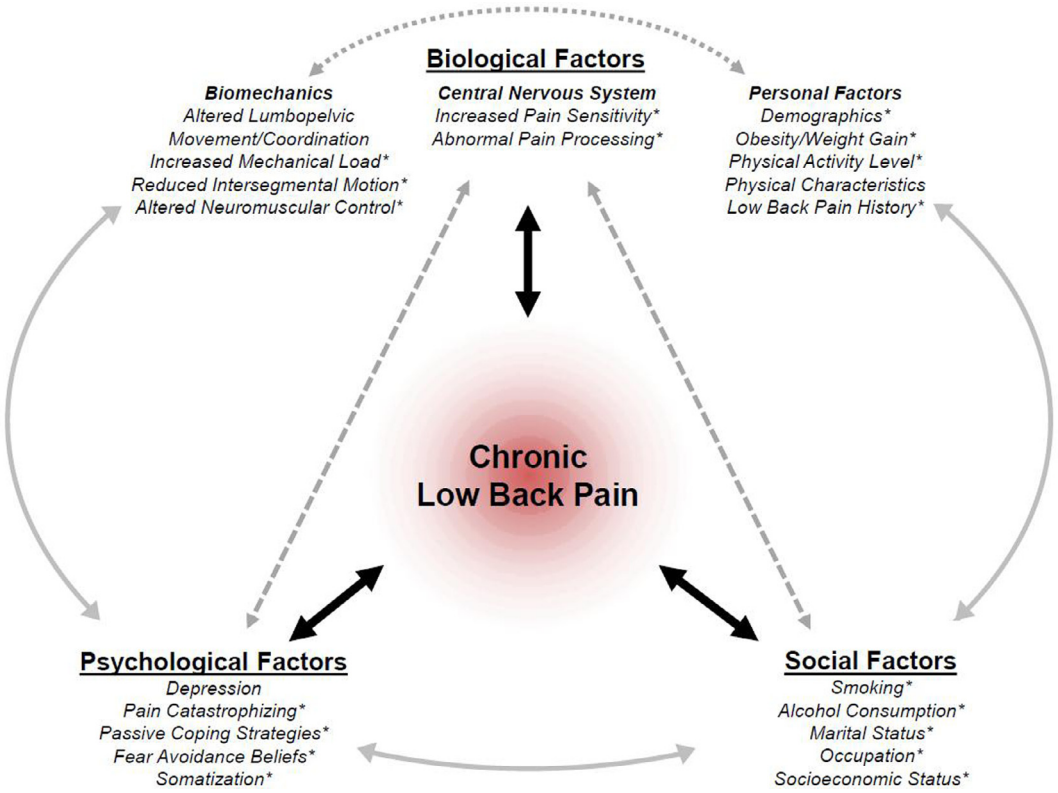

Fig. 1. Individual components (and their potential associations) of the biopsychosocial model of low back pain likely influenced or amplified by lower limb amputation. The * symbol identifies the components of the model that are supported in the literature for the general low back pain patient population but lack validation by studies performed in patients with lower limb amputation and low back pain.

Abnormal mechanics of the spinal column, primarily larger mechanical loads and instability, are often considered risk factors for the development of LBP [46]. Of particular interest here, characteristics of trunk motion can directly influence musculoskeletal loading [47], typically due to altered muscular response (i.e., coactivity) [46]. Though walking is generally not a mechanically demanding task for the low back (i.e., loads are well below reported injury thresholds) [48], and sometimes even considered therapeutic for individuals with LBP [49], altered trunk-pelvic motions with LLA during gait have recently been associated with large internal loads among tissues within the spine [50–52]. Notably, largest increases (up to ~65% relative to uninjured individuals) were found in joint compressive forces owing to a complex pattern of muscle responses [51]. Given the repetitive nature of gait, over time, even minimal increases in trunk motions and musculoskeletal loads may synergistically and progressively contribute toward LBP onset and/or recurrence and accelerate degenerative joint changes in the spine. To date, however, altered spinal loads after LLA has not been directly associated with development and/or presence of LBP in published studies.

It is well accepted that the neuromuscular system plays a central role in supporting the upper body and maintaining mechanical equilibrium and stability of the spine [53,54]. Irregular patterns of trunk muscle recruitment have been identified among the general population with recurrent LBP[55], and impaired postural control has been associated with spinal instability and LBP [56]. Among persons with LLA without LBP, similar assessments have identified impairments in trunk postural control during an unstable seated balance task [57], bilateral asymmetries in trunk mechanical and muscular responses to applied positional perturbations [58], as well as altered load-sharing between active and passive trunk tissues during quasi-static trunk flexion/extension movements [59]. Additionally, substantially greater fatigability has been reported for the low-back extensor musculature in patients with LLA with and without LBP [15], that are more pronounced than healthy individuals with and without LBP [60]. Fatigue of the low back extensors may further contribute to increased intersegmental spinal motion and instability during prolonged functional activities [61]. Though the specific origin and functional impact of such alterations remain somewhat speculative, these data support the theory that repeated exposure to altered loading associated with LLA and repeated use of a prosthetic device may result in tissue and neuromuscular adaptations and increased risk for LBP in this population.

Central nervous system

In addition to changes in trunk/pelvis biomechanics with LLA, central nervous system factors may also play an important biological role in the manifestation of LBP after LLA. Because of the trauma to peripheral nerves, amputation has the potential to influence the processing of pain signals in the peripheral and central nervous systems. Phantom limb pain has been long described as the perception of pain in the missing (amputated) limb [62,63], and may be indicative of altered pain signal processing. Although it is unknown how alterations in pain processing might influence the incidence and prevalence of secondary musculoskeletal pain problems, such as LBP, there are several plausible explanations.

In the general population, people with LBP display generalized hypersensitivity to pain that is reflective of central sensitization [64]. Central sensitization is the increased neuronal responsiveness to a stimulus due to prolonged or strong activity in the dorsal horn neurons that may be associated with an episode of pain or prolonged pain [65]. It is plausible that the pain stimulus associated with limb amputation could elicit central sensitization and

increased pain sensitivity, putting persons with amputation at risk for developing secondary pain conditions. Pain sensitivity is typically evaluated by assessing thresholds and tolerance to pain using a variety of modalities for stimuli, including: mechanical (pressure), electrical, and thermal (cold/heat) [64].

Changes in pain sensitivity also can be measured after either an inhibitory stimulus (conditioned pain modulation), or a facilitory stimulus (temporal summation) to further elucidate central mechanisms of pain inhibition or facilitation. Specific alterations in pain processing that have been reported in people with LBP include local [66,67] and widespread [66–71] hyperalgesia and enhanced temporal summation of pain signals [72–75]. Although people with chronic pain conditions such as osteoarthritis, fibromyalgia, and chronic fatigue syndrome also typically display decreased inhibition of nociceptive signals (conditioned pain modulation) [76–78], most studies report that people with chronic LBP display normal inhibition of pain signals [79–81].

Pain sensitivity has been examined to a limited extent in persons with limb amputation. In a small sample, Li et al. [82] reported that persons with traumatic amputation and phantom limb pain displayed decreased thresholds for sensation and pain with electrical stimuli in the unaffected limb, suggesting central sensitization. Further, Vase et al. [83] reported that people with upper limb amputation and phantom limb pain display decreased thresholds for pressure and cold stimuli, and enhanced temporal summation of pain signals. Inhibition of nociceptive signals has not been explicitly examined in person with amputation, but it is plausible that those with phantom limb pain may display decreased inhibition of pain similar to people with other chronic pain conditions. Although the mechanisms of altered pain processing are similar in persons with amputation and people with LBP, to our knowledge, no prior investigations have examined the neurophysiology of pain in patients with amputation and secondary musculoskeletal pain problems such as LBP to determine whether central sensitization places them at greater risk for secondary pain conditions.

Alterations in pain-processing areas of the brain in persons with amputation are also consistent with changes reported in otherwise uninjured individuals with LBP. For example, thalamic structural variations and, more specifically, decreases in gray matter of the posterolateral thalamus have been reported in people with amputation [84]. These changes appear to be positively correlated with duration of time since amputation, suggesting that they may be related to reduced afferent input [84]. Further, Lotze et al. [85] reported shifts in motor and sensory cortical activation patterns during movement in patients with phantom limb pain compared to pain-free persons with amputation, while Makin et al. [86] reported cortical reorganization of the sensorimotor cortex following arm amputation regardless of phantom limb pain. Collectively, these data suggest that neuroplastic changes associated with chronic pain in persons with amputation may involve cortical reorganization [85]. Similar alterations in brain morphology, including reduced density of gray matter in the dorsolateral prefrontal cortex, the thalamus, and the middle cingulate cortex has been reported in patients with LBP without amputation [87]. Although similar neuroplastic changes have been observed in some people with limb amputation and in people with LBP [88], whether the similarities in mechanism might be related to the development of LBP in persons with LLA requires further investigation. Identifying the contribution of altered pain processing to LBP in patients with amputation could inform the development of more targeted and individualized interventions.

Personal factors

The link between personal demographics and LBP has been well studied in the general population. Prevalence of LBP has been reported to increase with age (up to 65), with onset typically occurring in the third decade of life [89–93]. Race and ethnicity have also been investigated and the data supports the observation that Caucasians, Western Europeans and North Africans are more likely to experience LBP than African Americans, Caribbeans and Latin Americans [89,94]. However, reports of gender prevalence for LBP are vastly inconsistent [89–91,94,95]. Age, race, and gender have also been studied in persons with LLA. Traumatic amputations commonly occur in a younger population [96], with as many as 63% of military service members with LLA being less than 30 years of age [97]. Non-traumatic LLA secondary to various pathological conditions such as diabetes mellitus and cancer are more frequently seen in individuals greater than 60 years of age [96]. In a study of 255 patients with traumatic and non-traumatic LLA between the ages of 19–86, age was shown to be modestly but significantly correlated ($r = 0.12$, $p = 0.05$) with whether participants experienced LBP [98]. Distinct gender and race features have also been reported in previous research with the majority of patients with traumatic and non-traumatic LLA being male [99,100] and Caucasian [100,101]. However, whether demographic characteristics are associated with higher prevalence of LBP experience after LLA remains unexamined.

Obesity has also been identified as a strong risk factor for LBP in the general population [90,91,102]. In patients with LLA, obesity appears to be prevalent and dependent on the level of amputation, with 38% of persons with transtibial, 48% of persons with transfemoral, and 64% of persons with bilateral amputation presenting with noticeable clinical signs of obesity [103]. In support of the potential link between obesity and LBP, patients with LLA and LBP appear to have body mass index ratios above 50% of the recommended ratio compared to their counterparts without LBP [13]. The excess weight gain appears to be substantial and most common within the first two years after LLA [104], which may be attributed to the sedentary lifestyle immediately after amputation [13,105] .

Maintaining a healthy weight is commonly a challenge for patients with LLA due to difficulties associated with participating in exercise and sports activities [10]. Given the previous reports of increased risk of chronic LBP development as a result of inactivity in the general population [91,93], the reported reductions in physical activity levels after LLA [106,107] inherently increase the risk of LBP in this patient population. While participation in recreational or competitive sports has been reported in 32–60% of patients with LLA [97,108], there are fewer barriers in younger individuals who are more likely to achieve higher levels of physical performance due to accelerated rates of recovery and early fitting of running-specific prostheses [109,110]. Conversely, up to 46% of older persons with LLA become non-ambulatory one year post-amputation, which may place them at a higher risk of developing chronic LBP [111]. Although clinicians often attribute functional difficulties in this population to problems with the amputation and the prosthesis, LBP can also independently restrict activity levels in patients with LLA and warrants further investigation [112,113].

A number of physical characteristics have also been identified as risk factors for non-specific LBP in the general population, such as altered muscle strength/endurance, leg length discrepancy, or previous history of LBP [93,114–118]. In persons with LLA, greater iliopsoas muscle length but reduced back extensor strength and endurance have been associated with the presence of LBP [15]. Leg length discrepancy as a source of structural malalignment, including pelvic obliquity and functional scoliosis [119], has also been suggested as a potential cause of LBP after LLA but with conflicting supporting evidence. For example, in a study of 113 Finnish war-disabled service members with amputation, those with unilateral LLA and LBP with mild and occasional symptoms had a mean

leg length discrepancy of 6.1 mm as compared to a 21.7 mm discrepancy for those who reported severe and constant symptoms [120]. In other studies, however, no correlations have been reported between LBP and leg length discrepancy in persons with LLA [121,122]. Previous history of LBP in the general population has also been suggested to almost double the risk of future episodes of LBP [117,118]. In patients with LLA and LBP, however, only less than 20% recall having LBP prior to their amputation [8,9], and in most cases directly attribute their LBP to their LLA [10].

Psychological factors

Beyond biologic factors, as an individual with LLA reintegrates within the community, additional psychological factors can affect the risk for LBP and its eventual chronicity. Presence of psychological risk factors in the general LBP population are suggested to affect the frequency and intensity of follow-up medical care and the choice of interventions; whereas in their absence the patient has enhanced potential for quick recovery [123]. Recent evidence further suggests that targeting psychological factors in patients with LBP, particularly when they are at high levels, does seem to lead to more consistently positive results than either ignoring them or providing omnibus interventions regardless of psychological risk factors [124]. In the general population, moderate to strong associations have been reported between onset and chronicity of LBP with various psychological conditions such as depression, pain catastrophizing, passive coping strategies, fear-avoidance beliefs and somatization [123–127]. However, the influence of these psychological factors on the experience of LBP after LLA has not been fully evaluated.

Depressive mood has been related to the onset of LBP, higher levels of LBP intensity, poorer treatment outcome and transition from acute to chronic LBP [128,129]. To this end, depressive mood has also been reported as a significant predictor of the level of LBP intensity and bothersomeness in patients with LLA [12]. Given the much higher rates of depression in patients with LLA as compared to the general population [130,131], presence of depressive mood may play an important role in the increased risk for chronic LBP in this patient population. There is also a growing recognition that particular kinds of coping mechanisms such as pain catastrophizing (defined as the tendency to focus on, ruminate, and magnify pain sensations) are correlated with the transition from acute to chronic LBP and may be associated with poor treatment outcomes in the general population [129,132]. Prospective studies suggest that passive coping strategies, especially high levels of pain catastrophizing before an amputation, are associated with development and higher intensity of phantom limb pain and disability [133–136]. However, the extent to which passive coping strategies could influence the LBP experience after LLA remains unknown.

Fear of movement or injury (kinesiophobia) is another important predictor of LBP development and chronicity that could lead to severe disability in the general population [137,138]. This fear of movement can impede the rehabilitation process and cause dysfunctional pain-avoidance movement patterns that may lead to the development of secondary LBP after LLA. To this end, patients with higher fear-avoidance scores are more likely to have worse outcomes at 3, 6, and 12 months [123]. Although, it stands to reason that patients with LLA may develop beliefs about their condition that may cause them to become fearful of moving and engaging in daily activities, evidence of kinesiophobia in patients with LLA and LBP has not been previously evaluated.

Similarly, somatization is another prevalent psychological condition in patients with LBP that includes increased reports of widespread muscle pain located along the whole spine as well as to the legs and the head [139]. Somatization may also be related to presence of sleep disorder, anxiety, and symptoms of depression [139]. Higher somatization scores have been previously correlated with higher intensity of pain and grater disability, failure to return to work at 3 months and increased likelihood of a worse outcome at 1 year in patients with LBP [123,140]. Evidence of somatization has also been previously reported in patients with traumatic LLA and neuropathic pain with the resulting abnormal sensory processing leading to locomotor dysfunction and body image disturbances [141]. However, a number of factors such as time since amputation, time since first prosthesis, duration of daily prosthesis use, and high prosthesis satisfaction have shown to be negatively correlated with somatization [142]. Given the evidence suggesting that psychosocial factors can influence the outcome of rehabilitation, more research efforts are warranted for developing clinical tools to identify when and how psychosocial factors could be utilized in clinical decision making to improve outcomes in patients with LLA and LBP [143].

Alterations in central pain processing are also influenced by psychosocial and cognitive factors such as pain catastrophizing, attention, stress, and expectation [65]. People with amputation have been reported to display more depressive symptoms, greater anxiety, lower quality of life, and emotional disturbances [144]. Further, neuropathic pain in persons with amputation has been associated with depression, post-traumatic stress disorder, and catastrophizing [145]. It has also been reported that alterations in pain sensitivity and temporal summation of pain, as well as cortical responses to painful stimuli, were modulated by pain catastrophizing [83,146]. These psychosocial factors present in some patients with amputation and neuropathic pain, have also been associated with chronic-recurrent LBP and alterations in pain processing [147,148]. Although no specific association was previously reported between presence of phantom limb pain and psychological symptoms in a small study [144], strong evidence in support of the relationship between presence of psychosocial risk factors, alterations in central processing of pain, and LBP in patients with LLA remains scant.

Social factors

The effects of social factors such as cigarette smoking, alcohol use, marital status, occupation, and income on the experience of LBP have been under extensive investigation in the general population. For example, findings from systematic reviews including cross-sectional and longitudinal studies have revealed that both current and former smokers have a higher prevalence and incidence of LBP than "never smokers", but the association is fairly modest [149,150]. In military personnel with amputation, 21% report smoking cigarettes on a regular basis [151], while other studies have found that 37–48% of males with amputation are current cigarette smokers [101,111]. Although strong evidence linking cigarette smoking and LBP after LLA is lacking; one small study reported no difference in frequency of cigarette smoking between persons with transfemoral amputations with and without LBP [121]. Alcohol consumption has also been found to be greater in those with LBP in the general population [93]. In military personnel with amputation, alcohol consumption and substance abuse, along with probable alcohol addiction is more prevalent than in their non-amputee counterparts [152]. However, research evidence in support of the association between alcohol consumption and LBP after LLA does not currently exist. Being married is another social factors associated with higher risk of developing LBP in the general population compared to those who are divorced or single [91]. Although most reports indicate that the majority of individuals with LLA are married [97,151,153,154], marital status in at least one cohort study was shown not to be associated with either the intensity or bothersomeness of LBP in patients with LLA [12].

Individuals with occupations involving heavy lifting/pushing/pulling and driving have historically been identified to be more prone to development of LBP in the general population [91,93,155–157]. As for the military population, predictors of LBP include jobs involving lifting and wearing body armor [158], with a higher incidence seen in construction workers, auto mechanics, and law enforcement personnel [159]. However, both military and non-military individuals with LLA often return to employment in less physically demanding occupations [151,160,161], which may decrease their risk of developing occupation-related LBP. Lower socioeconomic class and lower levels of education have also been found to correlate with LBP in the general population [91]. Enlisted rank and service in the Navy, Army or Air Force have been identified as risk factors for LBP in a military sample [162]. Education at or below a high school level has been reported in 27–60% of service members with amputation [97,153] and 78% of those with amputations of dysvascular or diabetic aetiologies [101]. Of the service members with (traumatic) amputation, 31% were junior enlisted, 49% mid to senior enlisted, and 20% were officers [97]. In a sample of individuals with dysvascular or diabetic amputations, 44% reported an income of <$25,000, 37% between $25,001 and $50,000, and 19% an income >$50,000 [101]. Further investigations are needed to determine the potential relationships between occupation, socioeconomic class, education level and salary with LBP after LLA.

Consequences of the hypothesis and discussion

In the United States, an estimated 185,000 persons undergo limb amputation each year as a result of dysvascular disease (54%), trauma (45%), or cancer (1%), with the projected total number of people living with limb loss doubling to up to 3.6 million by the year 2050 [163]. In general, most amputations are major LLA (excluding toes) with increasing prevalence rates due to dysvascular diseases such as diabetes mellitus [163,164]. Despite the high prevalence of LBP after LLA, there currently exists a lack of understanding to identify any definite pathologic processes or anatomic sources of pain. A growing body of evidence from studies of LBP in the general population suggests that it is no longer appropriate to subclassify LBP solely using a biomedical construct, and that a successful classification system must include biomedical, psychological, and social assessments [25]. Given the multifactorial nature of LBP after LLA, a more comprehensive understanding of how amputation influences these biopsychosocial risk factors will further allow effective stratification of care for LBP after LLA, where patients are screened and placed in interventions designed to target their specific biopsychosocial risk profiles. The aim of this special communication was to integrate evidence originating predominantly from prospective studies on biopsychosocial correlates of LBP in the general patient population with LBP into a coherent hypothesis-driven model that could help generate new research questions and improve our understanding of the LBP experience after LLA.

The proposed biopsychosocial model could be useful in identifying risk factors for early identification of patients at risk for LBP and testing the effectiveness of different approaches aimed at reducing chronic LBP-related disability after LLA. Currently, the results from psychosocial interventions for LBP in the general population consistently show only small to moderate effects [16,165,166]. However, a multidisciplinary approach that addresses all three components of the biopsychosocial model of LBP may provide a more appropriate solution aimed at the multifaceted nature of the LBP experience after LLA [16]. A number of prospective studies have shown that psychosocial factors influence how patients respond to rehabilitative and surgical treatment, thus indicating the interaction between physical and psychological factors are important in determining the outcome of a given treatment for LBP after LLA [17].

Another potential approach would be to implement a stratified care approach, where patients with LLA are screened for known biopsychosocial risk factors using reliable and valid tools, and then referred to interventions designed to target their specific problem and risk profile [16]. To this end, use of a stratified approach, by use of prognostic screening with matched clinical pathways has shown promising results in management of LBP in primary care for the general population [167]. However, the current challenge to implementation of a stratified care approach is the identification and development of a validated risk factor profiles that could be used as a clinical guide to stratify patients with LLA into streams of care that optimize their chance of a good outcome for treatment of LBP. Given that some factors exert an influence on outcome regardless of treatment, whereas some only influence response to specific treatment [16], additional clarity is needed to determine which predictors of outcome are prognostic factors and which are potential treatment effect modifiers to help guide best practice treatments and the prevention of disability [168]. Additional research and insight are needed to determine more effective approaches to mitigate or manage LBP after LLA.

Disclaimer

The views expressed herein are those of the author(s) and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.

Conflict of interest statement

The authors declare that they have no competing interests.

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Short communication

A model-based approach for estimation of changes in lumbar segmental kinematics associated with alterations in trunk muscle forces

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ABSTRACT

The kinematics information from imaging, if combined with optimization-based biomechanical models, may provide a unique platform for personalized assessment of trunk muscle forces (TMFs). Such a method, however, is feasible only if differences in lumbar spine kinematics due to differences in TMFs can be captured by the current imaging techniques. A finite element model of the spine within an optimization procedure was used to estimate segmental kinematics of lumbar spine associated with five different sets of TMFs. Each set of TMFs was associated with a hypothetical trunk neuromuscular strategy that optimized one aspect of lower back biomechanics. For each set of TMFs, the segmental kinematics of lumbar spine was estimated for a single static trunk flexed posture involving, respectively, 40° and 10° of thoracic and pelvic rotations. Minimum changes in the angular and translational deformations of a motion segment with alterations in TMFs ranged from 0° to 0.7° and 0 mm to 0.04 mm, respectively. Maximum changes in the angular and translational deformations of a motion segment with alterations in TMFs ranged from 2.4° to 7.6° and 0.11 mm to 0.39 mm, respectively. The differences in kinematics of lumbar segments between each combination of two sets of TMFs in 97% of cases for angular deformation and 55% of cases for translational deformation were within the reported accuracy of current imaging techniques. Therefore, it might be possible to use image-based kinematics of lumbar segments along with computational modeling for personalized assessment of TMFs.

1. Introduction

Neuromuscular control of spinal equilibrium and stability changes in the presence of pain or following exposure to known risk factors for low back pain (LBP) (Muslim et al., 2013; Radebold et al., 2000, 2001; Toosizadeh et al., 2013). Such alterations may cause deformations and/or forces in lower back tissues such that exceed injury/pain thresholds instantaneously or cumulatively (Adams et al., 2013; Coenen et al., 2014; Marras et al., 2001; Panjabi, 1992a,b). Despite such a significant role, the current methods for personalized assessment of trunk muscle forces (TMFs) are limited. Kinematic measures of lumbo-pelvic coordination, though capable of distinguishing patients with LBP from controls (Vazirian et al., 2016), do not provide much information about individual muscle forces. Specifically, neuromuscular redundancy in control of lumbo-pelvic motion as well as individual variability in mechanical behavior of passive lumbar tissues hinder relating measured kinematics data to TMFs. The commonly used surface electromyography (EMG)-based methods for the assessment of TMFs, on the other hand, can only provide information about the activity of superficial trunk muscles. Further, the relationship between EMG measures of muscle activity and actual muscle force is still unclear (Staudenmann et al., 2010). Finite element and multi-joint biomechanical models of the spine with detailed musculature have also been developed and used for general assessment of TMFs (Arjmand and Shirazi-Adl, 2006a,b; Dreischarf et al., 2014; Ezquerro et al., 2004; Hughes, 2000; Stokes and Gardner-Morse, 2001). These models often implemented optimization procedures to estimate TMFs (Arjmand and Shirazi-Adl, 2006b; Daniel, 2011; Hughes, 2000; Stokes and Gardner-Morse, 2001) and are not suitable for personalized assessment of TMFs due to assumptions made related to lumbar segmental rotations and the requirement for a priori knowledge of trunk neuromuscular strategy (e.g., a strategy that minimizes stress in muscles).

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Currently, imaging is used to detect structural and geometrical/kinematics abnormalities in the lumbar spine (Fujii et al., 2007; Iwata et al., 2013; Keller et al., 2003; Kjaer et al., 2005; Ochia et al., 2006). The image-based geometrical/kinematics information have also been used for development of geometrically personalized biomechanical models of normal and scoliotic spine (Eskandari et al., 2017; Ghezelbash et al., 2016; Lafon et al., 2010; Petit et al., 2004), biomechanical comparison of healthy and metastatically involved vertebrae (O'Reilly and Whyne, 2008), material sensitivity analysis of intervertebral disc (Fagan et al., 2002), indirect estimation of spinal loads (Shymon et al., 2014), and estimation of elastic modulus of cancellous bone (Diamant et al., 2005). The geometrical information from imaging if combined with optimization-based biomechanical models may provide a unique platform for personalized assessment of TMFs. Particularly, it will be possible to use an optimization-based biomechanical model to search for a set of muscle forces that results in lumbar kinematics similar to those obtained from imaging. Such a method, however, is reliable only if differences in lumbar spine kinematics due to differences in TMFs can be captured by the current imaging techniques.

Recently, we have used our finite element model of the spine within an optimization procedure to estimate TMFs and kinematics of lumbar segments resulting from a trunk neuromuscular strategy that minimized sum of squared stress across all trunk muscles (Shojaei et al., 2015). The resultant kinematics were consistent with image-based reports of lumbar spine kinematics of asymptomatic individuals. Using the proposed algorithm, estimation of TMFs and lumbar segmental kinematics for other hypothetical trunk neuromuscular strategies that optimize other aspects of lower back biomechanics is possible. As a first step toward testing the feasibility of using image-based kinematics of lumbar segments for personalized assessment of TMFs, therefore, the objectives of this short communication are to determine changes in lumbar segmental kinematics due to alterations in trunk neuromuscular strategy and the associated TMFs and to verify if such changes are within the reported precision of current imaging techniques.

2. Methods

To address our research questions, TMFs and lumbar segmental kinematics were estimated for five different trunk neuromuscular strategies. In our approach each neuromuscular strategy was represented by a distinct cost function for the optimization procedure and assumed to either represent the trunk neuromuscular strategy of asymptomatic persons or a neuromuscular abnormality that minimizes loading on a specific aspect of lower back tissues (i.e., muscles, ligaments, intervertebral discs, and facet joints). As noted earlier, a neuromuscular strategy associated with the minimum value of sum of squared muscle stresses across the entire trunk muscles resulted in lumbar segmental kinematics consistent with image-based reports of lumbar spine kinematics of asymptomatic individuals, hence, was regarded to represent a normal trunk neuromuscular strategy (Shojaei et al., 2015). On the other hand, abnormal neuromuscular strategies that minimize loads in muscles, ligaments, intervertebral discs, and facet joints were represented by cost functions that respectively minimize sum of squared muscle forces across the entire trunk muscles, passive moment, compression, and shearing force at the L5-S1 intervertebral disc. For each neuromuscular strategy, the associated TMFs and lumbar segmental kinematics for a single static trunk flexed posture involving, respectively, 40° and 10° of thoracic and pelvic rotations (i.e., equal to a total lumbar flexion of 40–10° = 30°) in the sagittal plane were estimated using our kinematics-driven finite element approach. Specifically, the changes in distance between centers of two vertebrae of each motion segment (i.e., translational deformation) as well as changes in their relative angular orientations with respect to each other (i.e., angular deformation) with alterations in TMFs were considered as changes in lumbar segmental kinematics. Forward trunk bending is a common posture used for X-ray imaging of patients with LBP and the specific thoracic and pelvic rotations considered here are the same rotations we used in a recent study for validation of our method (Shojaei et al., 2015).

In our approach, rather than implementing a force-driven approach for estimation of lumbar segmental kinematics resulting from TMFs that are associated with a given neuromuscular strategy, we used our kinematics-driven methods. Such a methodological choice was mainly because of the lower computational cost of kinematics-driven approach. Specifically, the potential TMFs that are searched in the optimization procedure, where a kinematics-driven approach is used, readily satisfy spine equilibrium. Hence, the solution space that is searched by the optimization search engine is much smaller than the case when a force-driven approach is implemented. Therefore, in our approach, from all possible sets of lumbar segmental kinematics that can be distributed across lumbar vertebrae and generate the total 30° lumbar flexion, we will search (i.e., through optimization procedures) for a set of lumbar segmental rotations where the associated biomechanical outcomes from the kinematics-driven approach minimize the desired cost function. Such a methodological choice (i.e., kinematics- versus force-driven), however, does not affect the outcomes. In the following subsections, we first elaborate on the kinematics-driven approach that uses lumbar segmental kinematics to estimate TMFs and other biomechanical outcomes (e.g., the L5-S1 passive moment) and subsequently present the structure of the optimization algorithm that finds the lumbar segmental rotations that optimize its cost function (i.e., representing a given neuromuscular strategy).

2.1. Estimating trunk muscle forces using the kinematics-driven approach

A nonlinear finite element (FE) model of spine, developed in the ABAQUS software (Version 6.13, Dassault Systémes Simulia, Providence, RI), is used in the kinematics-driven approach to estimate the moment at each lumbar vertebra to be balanced by muscles attached to that same vertebra (Arjmand et al., 2009; Bazrgari et al., 2007). In the FE model of spine, the thoracic region and lumbar spine vertebrae are simulated by rigid elements and intervertebral discs are simulated by nonlinear flexible beam elements (Fig. 1). Inputs to the FE model include sagittal plane rotational boundary conditions at the T12 to the S1 spinal levels along with the ~50% of total body weight distributed across the entire spine (Arjmand and Shirazi-Adl, 2006b). A muscle architecture including 56 muscles attached to the spine from lumbar and thorax to pelvis is considered for estimation of TMFs required to balance moments at lumbar vertebrae. Since the attached muscles to each level (i.e., 10 muscles in each level from T12 to L4 and 6 muscles in the level L5) outnumbers the moment equilibrium equations, an optimization procedure, hereafter called force optimization procedure, is used to estimate muscle forces at each level as follows:

$$\begin{cases} Var\ \boldsymbol{F} \\ Cost\ function = g(\boldsymbol{F}) \\ \text{Minimize } (cost\ function) \\ \text{Subject to } \sum_{i=1}^{m} r_i \times F_i = M \end{cases} \quad (1)$$

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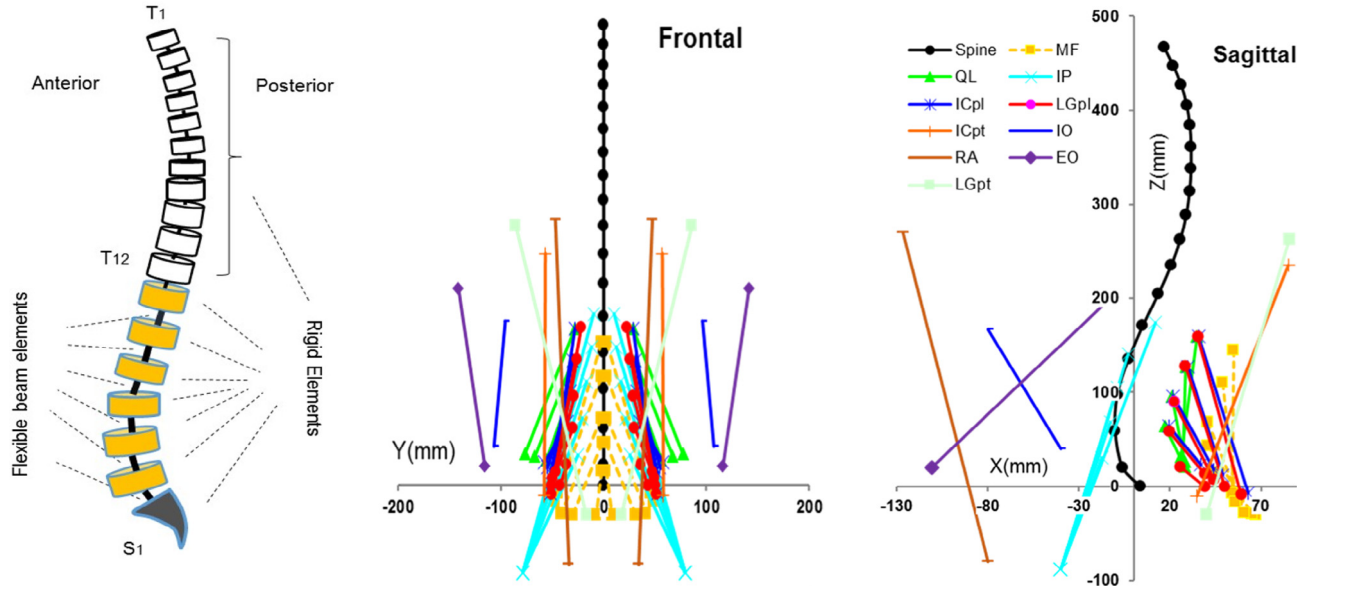

Fig. 1. A schematic model of the spine and its components (left), the musculatures in the sagittal (right) and frontal (middle) planes in upright posture. ICpl: iliocostalislumborum pars lumborum, ICpt: iliocostalislumbroum pars thoracis, IP: iliopsoas, LGpl: longissimusthoracis pars lumborum, LGpt: longissimusthoracis pars thoracis, MF: multifidus, QL: quadratuslumborum, IO: internal oblique, EO: external oblique and RA: rectus abdominus.

where F_i and r_i denote the force and the moment arm of the ith muscle, respectively and m is the number of muscles attached to that level and M is the output (reaction) moment. Where applicable, the cost function $g(\boldsymbol{F})$ was set to be the same as the cost function representing trunk neuromuscular strategy (see the following section). However, if such a selected cost function is independent of muscle forces in force optimization (i.e., only when neuromuscular strategy minimizes the passive moment at the L5-S1), $g(\boldsymbol{F})$ is set to minimize the sum of squared muscle stress at that level. A classic optimization technique (i.e., Lagrange Multiplier Method) is used to solve the force optimizations. Given the nonlinearity of FE model, the impact of estimated TMFs on mechanical response of the model is also considered by application of the estimated TMFs to the model as external loads and accounting for any residual moment estimated at each lumbar level in calculation of TMFs. Such iterative procedure is stopped when the residual moments estimated at each lumbar level become negligible (i.e., <0.1 N m).

2.2. Finding the lumbar segmental rotations that is associated with a given neuromuscular strategy

An optimization procedure (hereafter called neuromuscular optimization) was developed to minimize values of cost functions representing the trunk neuromuscular strategies (Shojaei et al., 2015). The decision variables of the optimization procedure were angular kinematics of lumbar spine that were input to the kinematics-driven model. Predictions of the kinematics-driven model, including TMFs, were then used to calculate the cost function of the optimization procedure. The optimization procedure uses a genetic algorithm that involves 100 generations and 30 individuals in each generation (i.e., a total number of 3000 individuals/iterations), and the stop criterion is considered as the tolerance of 10^{-3} for both values of decision variables and cost function. The neuromuscular optimization procedure was formulated as:

$$\begin{cases} Var\boldsymbol{\theta} = [\theta_{L_1}\theta_{L_2}\theta_{L_3}\theta_{L_4}\theta_{L_5}] \\ Cost\ function = g(\boldsymbol{F})\left(1+\alpha\sum_{i=1}^{n=62} max[0,k]\right) \\ \text{Minimize } (cost\ function) \\ \text{Subject to} \\ 0 \leqslant F_i \leqslant \sigma_{max} \times PCSA_i \\ -9.6^\circ \leqslant \theta_{T_{12}} - \theta_{L_1} \leqslant 6^\circ \\ -9.6^\circ \leqslant \theta_{L_1} - \theta_{L_2} \leqslant 6^\circ \\ -12^\circ \leqslant \theta_{L_2} - \theta_{L_3} \leqslant 3.6^\circ \\ -14.4^\circ \leqslant \theta_{L_3} - \theta_{L_4} \leqslant 1.2^\circ \\ -15.6^\circ \leqslant \theta_{L_4} - \theta_{L_5} \leqslant 2.4^\circ \\ -10.8^\circ \leqslant \theta_{L_5} - \theta_{S_1} \leqslant 6^\circ \end{cases} \quad (2)$$

where θ_{L1} to θ_{L5} are vertebral kinematics from the L1 to the L5 respectively and are decision variables of the neuromuscular optimization procedure. n = 62 denotes the number of optimization constraints including 56 constraints for muscle forces and 6 rotational constraints. F_i and $PCSA_i$ denote the force and the physiological cross section area of ith trunk muscle respectively, k is the number of estimated muscle forces that exceed the muscle force boundaries plus the number of violated rotational constraints, α is a penalizing value, and σ_{max} is the maximum allowable stress in the muscle (i.e., assumed to be 1.0 MPa). θ_{T12} and θ_{S1} are inputs of the neuromuscular optimization representing the rotations of the T12 and the S1 vertebrae. The rotational inequality constraints denote modified sagittal plane range of motion of lumbar motion segments with negative sign denoting flexion. These were obtained by adding a 20% increase to the mean reported values in Adams et al. (2013) to account for individuals' variability.

The flowchart of the procedure for finding the lumbar segmental kinematics and TMFs that are associated with a given trunk neuromuscular strategy is presented in Fig. 2.

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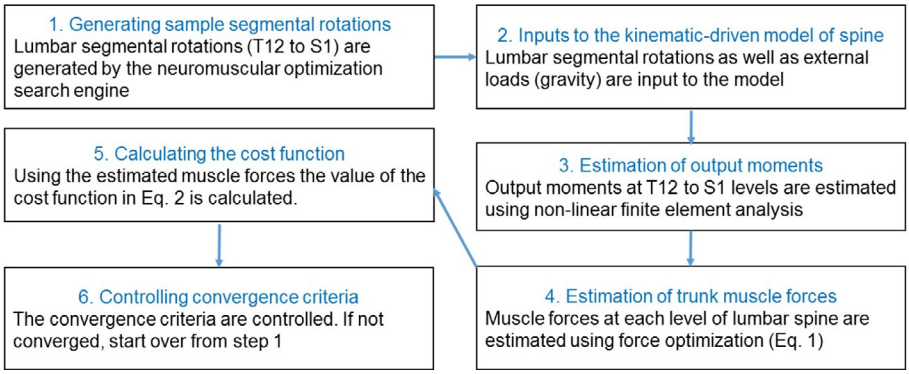

Fig. 2. The algorithm used for finding a set of lumbar segmental rotations whose associated biomechanical predictions from the kinematics-driven approach minimizes a cost function.

3. Results

The estimated angular and translational deformations of lumbar motion segments in the sagittal plan under TMFs associated with the five trunk neuromuscular strategies studied here are presented in the Table 1. Minimum changes in the angular and translational deformations of a motion segment with alterations in TMFs ranged from 0° (L2-L3 segment) to 0.7° (L4-L5 segment) and from 0 mm (L1-L2 and L2-L3) to 0.04 mm (L4-L5), respectively (Table 1). Similarly, maximum changes in the angular and translational deformations of a motion segment with alterations in TMFs ranged from 2.4° (L2-L3 segment) to 7.6° (L5-S1 segment) and from 0.11 mm (L2-L3) to 0.39 mm (L3-L4), respectively (Table 1). For each set of TMFs, the values of cost functions of other neuromuscular strategies were also calculated using the biomechanical predictions of the kinematics-driven approach (Table 2). As expected, the minimum value of a cost function was associated with predictions of kinematics-driven approach that were estimated to minimize that cost function.

4. Discussion

Lumbar segmental kinematics and TMFs resulting from neuromuscular strategies that optimize specific aspects of lower back biomechanics were calculated using a finite element model of the spine within an optimization procedure. The precision of current imaging techniques (e.g., computed tomography, magnetic resonance, fluoroscopy) have been reported to be ~0.1 mm and ~0.1° (Iwata et al., 2013; Keller et al., 2003; Ochia et al., 2006; Shymon et al., 2014; Breen and Breen, 2016; Zanjani-Pour et al., 2016) with repeatability errors of up to ~0.7 mm and ~1.3° (Breen et al., 2012). The differences in at least five (out of twelve: i.e., six angular and six translational deformations) kinematics outcome measures between each two sets of TMFs appear to be within the reported accuracy of current imaging techniques. Particularly, the differences in kinematics of lumbar segments between each combination of two different sets of TMFs (10 possible combinations) are detectable in 97% of cases for angular deformation and 55% of cases for translational deformation. Therefore, it might be possible to use image-based kinematics of lumbar segments along with computational modeling for personalized assessment of TMFs.

While image-based information have been used for development of subject-specific mechanical models of spine (Diamant et al., 2005; Eskandari et al., 2017; Fagan et al., 2002; Ghezelbash et al., 2016; Lafon et al., 2010; O'Reilly and Whyne, 2008; Petit et al., 2004; Shymon et al., 2014), previous studies have primarily used image-based information to personalize geometry (e.g., vertebra/disc dimensions, muscles cross-sectional areas and insertion points) and/or mechanical property of spine models (Diamant et al., 2005; Eskandari et al., 2017; Fagan et al., 2002; Ghezelbash et al., 2016; Lafon et al., 2010; O'Reilly and Whyne, 2008; Petit et al., 2004). Furthermore, some of these studies have been conducted in tissue level (Diamant et al., 2005; Fagan et al., 2002), have been designed for specific group of patients (Lafon et al., 2010; Petit et al., 2004), and have oversimplified the spine model by disregarding the effects of muscle forces when calibrating using experimental measures (Lafon et al., 2010; Petit et al., 2004). Although potentially feasible according to the results of current study, the personalized assessment of TMFs using geometrical information from imaging combined with optimization-based modeling, to the best of our knowledge, has not yet been reported.

Table 1
The estimated angular (°) and translational (mm) deformations of lumbar motion segments in the sagittal plane under muscle forces of trunk neuromuscular strategies that minimize (1) sum of squared muscle stresses, (2) sum of squared muscles forces, (3) L5-S1 compression force, (4) L5-S1 anterior-posterior shearing force, and (5) L5-S1 passive moment.

	Angular deformations						Translational deformations					
	T12-L1	L1-2	L2-3	L3-4	L4-5	L5-S1	T12-L1	L1-L2	L2-L3	L3-L4	L4-L5	L5-S1
$\sum Stress^2$	3.1	5.2	4.8	3.6	5.7	7.6	0.70	1.10	1.22	1.24	1.48	0.75
$\sum Force^2$	7.8	7.6	4.8	1.6	2.5	5.7	0.97	1.19	1.22	1.09	1.20	0.70
Compression force	7.9	6.8	5.9	2.1	1.8	5.5	0.97	1.10	1.24	1.07	1.15	0.69
Shearing force	5.6	3.3	5.0	7.5	7.7	0.9	0.89	0.96	1.21	1.37	1.52	0.81
L5-S1 passive moment	4.2	6.6	7.2	7.8	4.2	0.0	0.87	1.18	1.32	1.46	1.44	0.86
Minimum change	0.1	0.2	0.0	0.3	0.7	0.2	0.02	0.0	0.0	0.02	0.04	0.01
Maximum change	4.8	4.3	2.4	6.2	5.9	7.6	0.27	0.23	0.11	0.39	0.37	0.17

Minimum and maximum changes in each column were, respectively, the smallest and largest value of difference between the deformations of any two trunk neuromuscular strategies.

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Table 2
The value of cost functions (horizontal top) under the five sets of muscle forces associated with neuromuscular strategies (vertical left) studied here.

	$\sum Stress^2$	$\sum Force^2$	Compression force (N)	Shearing force (N)	L5-S1 passive moment (Nm)
$\sum Stress^2$	8.39e+11	1.53e+05	1.49e+03	622.9	16.0
$\sum Force^2$	2.28e+12	6.37e+04	1.34e+03	626.2	12.1
Compression force	3.02e+12	7.02e+04	1.32e+03	601.8	12.1
Shearing force	7.10e+12	1.73e+05	1.84e+03	518.0	2.7
L5-S1 passive moment	2.86e+12	1.92e+5	1.78e+03	610.5	0.0

The value of cost function of each neuromuscular strategy, as expected, increased when calculated using predictions of the kinematics-driven approach associated with the other cost functions (Table 2). However, what is notable in results presented in Table 2 is that alterations in TMFs, for example due to an abnormal trunk neuromuscular strategy, could result in loads and/or deformations in some areas of lower back that are larger than what is normally resisted by those areas. For instance, TMFs associated with the hypothetical neuromuscular strategy that minimized shearing force at the L5-S1 intervertebral disc resulted in an increase of ~350 N in compression force when compared to TMFs that were associated with the ~~a~~ strategy that was considered normal in this study (i.e., the strategy that minimizes sum of squared muscle stresses). Similarly, a strategy that minimized compression force or muscle forces, compared to the normal strategy, led to large muscle stresses. Although the short term effect of a specific trunk neuromuscular strategy can be beneficial, for instance by protecting the injured tissues, the long term consequences of altered trunk neuromuscular strategy could be an injury to other lumbar tissues due to compensatory resisted larger than normal loads (Hodges and Smeets, 2015).

In the present study, we postulated that trunk neuromuscular strategies optimize some aspects of lower back biomechanics. Though alterations in neuromuscular strategy have been reported in the literature, our assumption might not be accurate and was merely made for the purpose of this feasibility study (i.e., to demonstrate changes in lumbar segmental kinematics with alterations in TMFs are within the reported accuracy of current imaging techniques). Furthermore, in all cases, the abnormal neuromuscular strategy that minimized loads in a tissue was represented by a single-force cost function which was a simplified assumption. For example, minimizing the loads on the facet joint involves reducing both shearing and compression forces, though shearing is the dominant force in characterizing facet joint environment. Whether there are one-on-one relationships between sets of TMFs (or the resultant kinematics) and neuromuscular strategies or whether all differences in trunk neuromuscular strategies result in detectable change in TMFs remains to be investigated in future. The availability of personalized assessment of TMFs, as proposed in this short communication, should, however, facilitate such future research efforts.

In conclusion, results of this feasibility study, support the idea of image-based personalized assessment of TMFs using computational models. Specifically, a geometrically and materially subject-specified model of the spine can be used in future to obtain a set of TMFs, as individualized TMFs, that generates the closest lumbar kinematics to those measured from imaging. The accuracy of such assessment strategy can further be improved by implementing dynamic rather than static assessment tasks. However, immediate research question to be addressed will be the reliability and validity of such an image-based method for personalized assessment of TMFs.

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Conflict of interest statement

We declare that all authors have no financial or personal relationships with other persons or organizations that might inappropriately influence our work presented therein.

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Title: Living with Extremity Trauma and Limb Loss for a Lifetime: A Review of Efforts to Identify and Mitigate Risk Factors for Secondary Health Complications

Objectives:

1. Identify and describe key risk factors for musculoskeletal conditions secondary to extremity trauma / limb loss

2. Identify and describe key risk factors for cardiovascular disease secondary to extremity trauma / limb loss

3. Identify and describe effective approaches to mitigate onset and progression of these secondary health conditions

Type of Presentation: Panel Discussion (45min)

Target Audience: all clinicians (and researchers) involved with the care of Servicemembers and Veterans with extremity trauma and amputation

Presentation Summary: Extremity trauma, including limb loss, is commonly associated with an increased prevalence of and risk for developing deleterious secondary health conditions, such as musculoskeletal and cardiovascular disease [1,2], among others. These secondary health conditions significantly impact functional outcomes and quality of life over the longer term and, thus, are particularly concerning given the relatively young age of Servicemembers with extremity trauma and potential for cumulative, lifelong disability. Here, we focus on synthesizing recent efforts at each Military Treatment Facility (WRNMMC, SAMMC, NMCSD) and NHRC; specifically, those toward identifying and tracking risk factors for these complications secondary to extremity trauma and limb loss.

The Extremity Trauma and Amputation Center of Excellence is composed of clinicians and researchers who are actively conducting numerous efforts with the collective aim to comprehensively understand the specific factors (e.g., biomechanical, personal, physiological, psychological) underpinning the development and progression of these deleterious secondary health conditions. These include epidemiological efforts to characterize this patient population and their health over time, biomechanical calculations of joint loads during activity, local/systemic biomarkers (e.g., blood/synovial fluid, respectively), changes in patient-level risk factors, and self-reported quality of life metrics.

We have identified several instances where altered gait mechanics are associated with abnormal joint loading parameters as likely contributors to the high prevalence and incidence of low back and knee pain among persons with limb loss. Specifically, increased trunk motions in persons with vs. without limb loss are associated with greater external demands on the lower back [3] and up to a 65% increase in joint compressive forces [4], and closely mimic the movement patterns seen in individuals who have low back pain [5]. Peak joint contact forces and loading rates within the intact

knee are also respectively 17 and 24% larger among persons with vs. without unilateral limb loss [6]. However, level ground walking is likely not the only contributor to the risk of knee osteoarthritis [7] as other common movements, such as hopping when moving without a prosthesis, place the knee at even greater risk [8]. Advancements in prosthetic designs offering powered push-off can also mitigate risk factors by reducing knee joint loads [7]. Alterations in health habits (e.g. tobacco/alcohol use) and ensuing physical inactivity and weight gain [9], which are commonly observed after lower limb trauma and amputation, can increase the risk for musculoskeletal and cardiovascular disease.

A more holistic understanding of the specific pathways by which these secondary conditions develop is urgently needed, particularly for clinicians to prescribe effective treatment interventions which decrease concomitant risk. Similarly, it is important for individuals with limb loss to be cognizant of the multi-factorial contributors to these conditions. Identifying and mitigating the risk of health effects secondary to amputation is a positive step towards setting patients up for lifelong health. Knowledge of adverse movement mechanics, practical metrics and tests for evaluation, and the effectiveness of different prosthetic designs will provide information useful for treating individuals with limb loss. This session will deliver this information through a summary of the clinically-applicable research efforts at the DoD's Military Treatment Facilities to assist clinicians in their efforts to serve the men and women who have served our country.

References: [1] Gailey et al. 2008 [2] Butowicz et al. 2017 [3] Hendershot et al. 2014 [4] Shojaie et al. 2016 [5] Russell Esposito 2014 [6] Miller et al. 2017 [7] Russell Esposito 2015 [8] Krupenovich et al. 2017 [9] Eckard and Pruziner 2016

Biosketches:

Christopher Dearth, PhD, concurrently serves as the Facility Research Director for the Extremity Trauma & Amputation Center of Excellence (EACE), Director of the Research & Development Section within the Department of Rehabilitation at WRNMMC, and the Founding Director of the Regenerative Biosciences Laboratory at the Uniformed Services University of the Health Sciences (USUHS). In these roles, Dr. Dearth leads a multidisciplinary team of clinicians and researchers whose collective focus is on the mitigation, treatment and rehabilitation of traumatic extremity injuries and amputations by implementation of clinically relevant research aimed at optimizing the quality of life of service members and veterans. Dr. Dearth's preclinical and clinical research interests include: development of innovative regenerative medicine & tissue engineering constructs, utilization of next generation imaging modalities for extremity trauma applications, evaluation of advanced (i.e., powered and intuitively controlled) prosthetics for Service Members and Veterans with limb loss, and championing a synergy of efforts between the fields of rehabilitative and regenerative medicine.

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Brad Hendershot, PhD, is a Research Biomedical Engineer with the EACE, stationed at WRNMMC. In addition to this role, he directs activities within the Biomechanics and Virtual Reality Laboratories within the Department of Rehabilitation. Dr. Hendershot also holds an appointment, as Assistant Professor, within the Department of Rehabilitation Medicine at USUHS. He received his BS in Bioengineering from Penn State and PhD in Biomedical Engineering from Virginia Tech. Dr. Hendershot's research is primarily focused on the mechanics and neuromuscular control of human movement, with a particular emphasis on the trunk/spine in both uninjured and patient populations. Recently, his research aims to comprehensively characterize factors underlying the high prevalence of musculoskeletal complications secondary to extremity trauma, to mitigate development/recurrence over the longer term and ultimately improve quality of life for Service Members and Veterans with limb loss.

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dynamical systems and movement coordination analyses, and musculoskeletal modeling to determine how to concurrently improve performance while establishing strategies to reduce the risk of injury over time. Dr. Russell Esposito also contributes to the scientific community as a reviewer for over a dozen scientific journals and serves roles within numerous professional organizations.

LOWER EXTREMITY JOINT CONTRIBUTIONS TO FRONTAL PLANE TRUNK DYNAMICS IN PERSONS WITH LOWER EXTREMITY AMPUTATION

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INTRODUCTION

Persons with unilateral lower extremity amputation (LEA) have altered trunk-pelvis kinematics and joint\muscle forces during walking relative to able-bodied (AB) persons [1,2]. These altered mechanics are believed to be associated with the increased prevalence of and risk for both low back pain (LBP) and falls in persons with LEA. Recent work has demonstrated that in AB walking, all joint moments in the body can contribute to net trunk dynamics, which may include complex inter-planar couplings; for example, the stance limb sagittal ankle moment can induce frontal plane trunk angular accelerations that tend to tilt the trunk ipsilaterally [3,4]. In persons with vs. without LEA, elevated ipsilateral trunk lean during prosthetic limb stance is a commonly reported deviation [1,2]. Therefore, the aim of this exploratory study was to identify the primary contributors to frontal plane angular trunk dynamics during walking of persons with LEA, in comparison to AB walking.

METHODS

One subject with unilateral transfemoral LEA (TFA), one with unilateral transtibial LEA (TTA), and two AB controls (AB1, AB2), were retrospectively identified from records of the Biomechanics Lab at Walter Reed National Military Medical Center. Subjects were selected such that self-selected walking velocity (SSWV) fell within +/-5%, and no participants self-reported current LBP at time of data collection (Table 1).

Subjects walked overground along a 15m walkway at SSWV, with full-body kinematics (f_s=120Hz) and ground reaction forces (f_s=1200Hz) respectively measured via a 27-camera motion capture system and six floor-embedded force platforms. One representative prosthetic(right) stance phase was extracted for each subject, kinematic\kinetic data were filtered at 6\25Hz, and an inverse kinematic solution was computed using Visual3D, applying the Gait2392 model definition of the OpenSim software[5]. The Residual Reduction Algorithm was used to compute a set of joint moments that best tracked experimental kinematics while also adjusting trunk center-of-mass location to reduce dynamic inconsistencies. Joint moments and kinematics were input to an induced acceleration analysis, applying a rolling-without-slipping contact constraint at each foot in place of measured forces. For each system force, mean induced angular accelerations were computed using separate positive and negative mean integrals during gait phases of interest [4]. As early stance is generally where elevated lateral lean occurs in persons with LEA, analyzed phases were limited to initial double-limb support (IDS) and early single-limb support (ESS).

Table 1: Demographic characteristics and self-selected walking velocity (SSWV) for the able-bodied (AB), transtibial (TTA), and transfemoral (TFA) participants.

	Gender	Age (yr)	Stature (cm)	Mass (kg)	Time Since Amp. (yr)	Prosthetic Componentry	SSWV (m/s, Fr)
TTA	Male	34	177	93	1.29	Renegade® (Freedom Innovations)	1.24 (0.17)
TFA	Male	23	174	70	1.25	Ceterus™, Power Knee™ (Ossur)	1.22 (0.17)
AB1	Male	27	176	76	-	-	1.32 (0.20)
AB2	Male	27	172	81	-	-	1.42 (0.23)

RESULTS AND DISCUSSION

Simulations were successfully generated for each subject, with quality verified against suggested standards [5]. Comparing simulated trunk kinematics (Fig. 1, left) mean trunk lean during ESS was greatest in TTA (5.0º) followed by TFA (3.6º), and AB1\AB2 (0.5º \ 0.1º).

During both IDS and ESS, inter-planar coupling was observed across all subjects. Sagittal ankle and hip moments induced notable contributions to net frontal trunk accelerations throughout stance - in agreement with prior work in AB subjects [3,4]. Contributions from the frontal lumbar moment differed between subjects (Fig 1, right): for TTA a prosthetic(right)-tending acceleration was induced, while for AB1, AB2, and TFA accelerations were intact(left)-tending, although considerably larger in TFA. Comparing AB1 and AB2, AB2 had a larger contribution from stance(right)-side knee Flx\Ext during IDS relative to AB1, while AB1 instead had a larger contribution from trailing(left)-side hip Ab\Ad. This suggests that able-bodied persons may apply notably different underlying mechanics to compose similar net frontal plane trunk accelerations. To facilitate comparison of LEA subjects with AB, the following reasoning was applied; a moment that contributes elevated prosthetic-tending, or reduced intact-tending, acceleration to the cumulative net over IDS (relative to AB1 and AB2), has potential to be associated with subsequent, elevated trunk lateral lean towards the prosthesis in later stance. For TFA, this highlighted prosthetic-side ankle Flx\Ext and prosthetic-side hip Ab\Ad. In contrast for TTA, this highlighted lumbar frontal moment, followed by prosthetic-side knee Flx\Ext, and prosthetic-side ankle Flx\Ext. The prosthetic ankle contribution was elevated in both TTA and TFA, suggesting adjustment of prosthetic ankle componentry, or distally-targeted gait re-training, may warrant investigation as means to affect elevated lateral trunk lean in LEA patients. However, these simulation-based observations require empirical validation prior to clinical translation.

CONCLUSIONS

This exploratory study highlights that frontal plane trunk dynamics during level walking in persons with LEA and no LBP are composed of a whole-body balance of system forces, wherein distal prosthetic ankle function may play a role. A larger cross-sectional analysis is needed to ascertain if features observed in these case subjects are characteristic of the broader LEA population, and also if those with versus without LBP differ.

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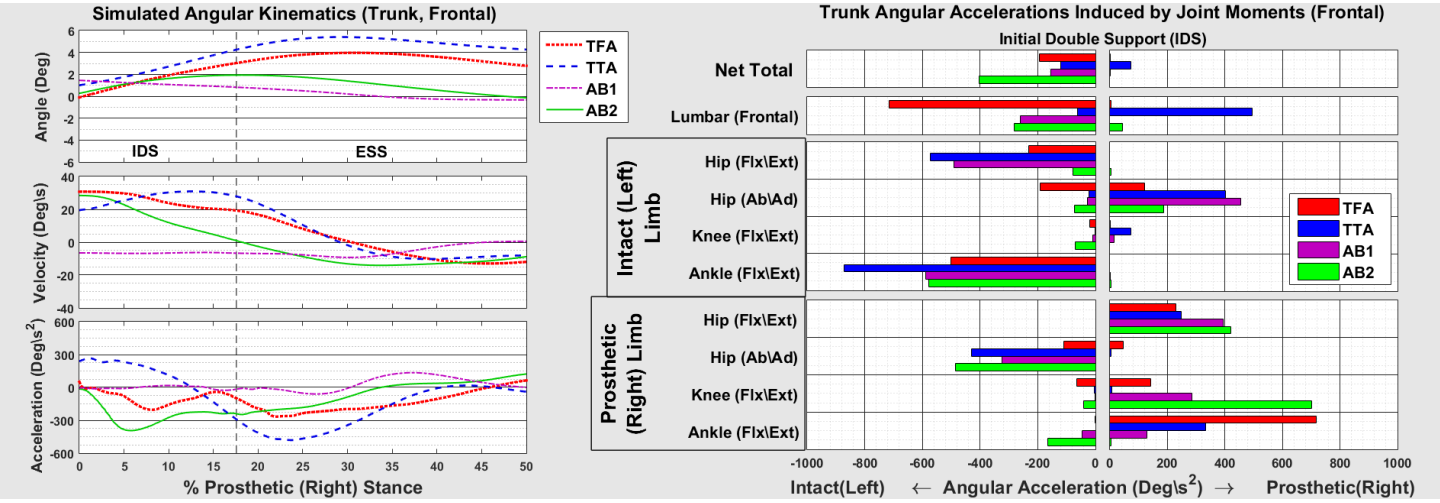

Figure 1: Simulated trunk angular kinematics relative to global during initial double support (IDS) and early single support (ESS) (left). Mean, angular accelerations induced on the trunk in the frontal plane by lower-extremity joint moments during prosthetic IDS (right).

Trunk-Pelvic Dynamics during Transient Turns in Persons with Unilateral Lower Limb Amputation

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Background: Persons with lower limb amputation (LLA) often walk with compensatory movement strategies involving a prominent reliance on the trunk and pelvis. Altered kinematic features and coordination of these two segments have been associated with elevated demands on the low back [1], increased inter-segmental rigidity [2], and larger trunk muscular forces and spinal loads [3-4]. Consequently, biomechanical factors are considered to play an important role in the development or recurrence of longer-term musculoskeletal complications after LLA (e.g., LBP; [5,6]); however, evaluations of other common ambulatory tasks are limited. Turning is ubiquitous in daily life and can be biomechanically challenging for individuals with musculoskeletal impairments [7]. Thus, the purpose of this study was to characterize proximal compensations using inter-segmental momenta and coordination during transient (90-degree) turning tasks among persons with vs. without LLA. We hypothesized persons with vs. without LLA execute turns with altered trunk-pelvic segmental coordination and larger ranges in segmental momenta, to overcome challenges associated with altered or impaired ankle function (a key joint for executing turns).

Methods: Three-dimensional trunk and pelvic kinematics, momenta, and coordination phase/variability were calculated among 8 persons with LLA (n=4 with transtibial and n=4 with transfemoral amputation), and 5 uninjured controls, performing 90-degree turns to the left (n=10) and right (n=10). Participants walked at their self-selected speed along a 12-foot straight path and were verbally cued to turn left or right at a specified and consistent point, self-selecting the turning strategy (i.e., step vs. spin) and pivot limb.

Results: There were no differences in the frequency of intersegmental coordination phase (or its variability) between persons with and without LLA. However, ranges of motion and segmental momenta differed between persons with and without LLA, depending on the plane and turn strategy employed. In the sagittal plane, ranges of motion and momenta were respectively larger and smaller in step turns among persons with vs. without LLA. In the frontal plane, ranges of motion and momenta were respectively smaller and larger among persons with LLA. In the transverse plane, ranges in pelvic momenta were larger/smaller during spin/step turns, respectively, among persons with vs. without LLA.

Conclusions: Although no differences in the frequency or variability of inter-segmental coordination were observed, altered ranges of motion and momenta among persons with LLA indicate similar reliance on proximal segments during turning as in walking. Such altered and task-dependent modulation of trunk-pelvic dynamics among persons with LLA provides additional support for a hypothesis linking repeated exposures to altered trunk-pelvic dynamics with elevated LBP risk over the long term.

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LONGITUDINAL CHANGES IN MEDIOLATERAL TRUNK AND PELVIC MOTION AMONG PERSONS WITH LOWER LIMB AMPUTATION DURING THE FIRST YEAR OF AMBULATION

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INTRODUCTION

Persons with lower limb amputation (LLA) often walk with compensational movement strategies involving a prominent reliance on the trunk and pelvis [1]. Resulting increases in trunk and pelvic motions compared to able-bodied individuals have been associated with an elevated risk for low back pain (LBP; [2,3]). In the frontal plane, larger trunk range of motion (ROM) and peak lateral flexion (on the prosthetic side, specifically) among persons with vs. without LLA are often assumed to be a result of an active/learned neuromuscular movement strategy to compensate for weak (or missing) musculature in the residual limb [4]. Identifying the extent to which trunk and pelvic motions change or evolve over time following amputation, particularly beginning at time of initial ambulation, could provide additional insight into contributing factors and, ultimately, guidance for rehabilitation strategies to mitigate deleterious consequences of such motions. Thus, the purpose of this study was to characterize features of trunk and pelvic motion in the frontal plane among persons with LLA as a function of time after amputation. We hypothesized the overall magnitude of mediolateral trunk and pelvic motion would increase with increasing time of ambulation, suggesting a learned proximal movement strategy.

METHODS

Thirty-two males with unilateral LLA (Table 1) completed gait analyses at 5 distinct time points: 0, 2, 4, 6, and 12 months after initial ambulation (defined as able to walk 50ft independently without assistive devices). Participants walked overground across a 15m level walkway at 3 controlled speeds (completed in a randomized order): Froude2 (mean=0.96 m/s), Froude3 (mean=1.20 m/s), and Froude4 (mean=1.44 m/s). Full-body kinematics were recorded by tracking (120Hz) reflective markers using a 23-camera motion capture system (Vicon, Oxford, UK). All participants provided informed consent to procedures approved by the Walter Reed National Military Medical Center Institutional Review Board. Note, participants were not required to wear the same prosthetic components throughout, though only 3 of the 32 changed between similar passive energy storage and return feet (all TF wore microprocessor knees).

Table 1. Mean (SD) participant demographics, obtained at visit t=0, for persons with unilateral transtibial (TT) and transfemoral (TF) amputation. All amputations were the result of traumatic injuries.

	TT (n=22)	**TF** (n=10)
Age (yr)	27.1 (6.0)	28.5 (6.2)
Stature (cm)	178.5 (4.0)	177.9 (7.8)
Body Mass (kg)	84.2 (11.1)	83.9 (10.8)

Mediolateral trunk and pelvic ROM, and peak trunk lateral flexion in prosthetic limb stance, were computed for each walking speed within Visual3D (C-Motion, Germantown, MD, USA). The trunk was modeled as a single rigid segment, defined proximally by the acromia and C7 vertebrae, and distally at the T10 spinal level. All segmental kinematics were defined in the global coordinate system (relative to vertical). Because not all participants completed all five time points (mean=3, range 2-5), mixed-model repeated measures analyses of variance (ANOVA) were used to compare trunk ROM and peak lateral flexion by group (TT vs. TF) and time, and their first-order interaction. All statistical analyses were performed using SPSS (Version 21, IBM Corp., Armonk, NY, USA), with statistical significance determined when $p<0.05$.

RESULTS AND DISCUSSION

There were no differences in pelvis ROM by time (p>0.09) or between groups (p>0.56) at any speed. However, both trunk ROM and peak lateral flexion in prosthetic limb stance decreased (all p<0.024) over time (Figure 1), wherein the 0 and 12 month times points were significantly different. At Froude2 and Froude4, these did not differ between groups (p>0.28). At Froude3, however, there was a significant interaction between visit and group (p=0.014); trunk ROM and peak lateral flexion decreased among persons with TF but not TT amputation, primarily due to the larger values at t=0 among persons with TF amputation.

In contrast to our hypothesis, mediolateral trunk ROM and peak lateral flexion in prosthetic limb stance decreased with increasing time after initial ambulation, regardless of walking speed, most notably within the first 2 months of ambulation among persons with TF amputation. Such a trend suggests mediolateral movements of the trunk and pelvis may be more of a near-term reaction to walking with a prosthesis than a proactive movement strategy learned over time. However, these individuals were also participating in extensive rehabilitation throughout this one-year period and, thus, these changes may also reflect physical therapy and other gait training techniques. Nevertheless, the magnitudes of these motions remain larger than persons without LLA (see gray shaded areas in Figure 1), as well as compared to values reported elsewhere from individuals with LLA who are generally evaluated greater than 12 months post amputation [1,5]; suggesting these motions may continue to change over time. Future work should therefore longitudinally evaluate trunk and pelvic motions for extended durations to capture longer-term changes, particularly when these individuals are no longer receiving frequent rehabilitative care. In summary, data reported here identifies, for the first time, temporal relationships of mediolateral trunk motions within the first year of ambulation among persons with LLA. These data have longer-term implications for the surveillance of LBP onset and recurrence, and may help identify important biomechanical factors in its causation.

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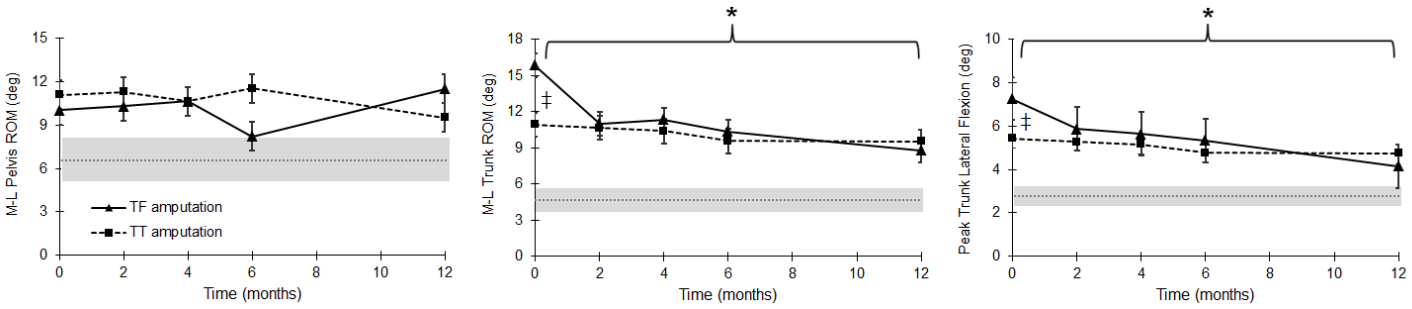

Figure 1. Froude3 walking speed. Mean mediolateral (M-L) pelvis range of motion (ROM; left), trunk ROM (middle), and peak trunk lateral flexion in prosthetic limb stance (right) among persons with transtibial (TT) and transfemoral (TF) amputation with increasing time after initial ambulation. Error bars represent standard errors. Grey dotted lines (shaded areas) respectively represent mean (standard deviations) for 6 males without amputation walking at a similar speed. Asterisks indicate significant differences in mean responses between the first (0 month) and last (12 month) time points; group differences within a given time point are also indicated (‡).

TRUNK NEUROMUSCULAR CONTROL STRATEGIES AMONG PERSONS WITH LOWER LIMB AMPUTATION WHILE WALKING AND PERFORMING CONCURRENT TASKS

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INTRODUCTION

Altered trunk-pelvic motions and inter-segmental coordination in persons with unilateral lower limb amputation (ULLA) have been associated with increased risk for secondary health conditions (e.g., low back pain) [1-3]. Kinematic adaptations within these proximal segments may be a result of adopted neuromuscular strategies to account for absent sensory information and/or altered musculoskeletal tissues within the lower extremity due to trauma. In contrast, these adaptations may also indicate impaired trunk neuromuscular control, in that the neuro-musculoskeletal system cannot successfully govern kinematic variability (i.e., mechanical disturbances) and control errors in order to maintain stability. Non-linear analyses quantify the structure of variability over time using stochastic dynamics and may posit how the neuromuscular system controls the trunk in persons with varying levels of limb loss while walking. Thus, the objectives of this study are to: (1) characterize trunk local stability (as an indication of neuromuscular control) in persons with varying levels of ULLA during gait (vs. controls), and (2) evaluate the influences of concurrent cognitive challenges, as the ability to control the trunk during gait requires attentional resources, potentially further influencing trunk stability [4]. We hypothesize that persons with transfemoral ULLA will demonstrate decreased trunk local stability (greater lyapunov exponent) compared to persons with transtibial ULLA and able-bodied controls. Secondarily, persons with transfemoral ULLA will demonstrate larger decreases in local stability with increased cognitive challenge compared to transtibial ULLA and able-bodied controls.

METHODS

Sixteen males with traumatic ULLA [8 transfemoral (TFA; mean age: 35.4 yrs) and 8 transtibial (TTA; mean age: 33.5 yrs)] and eight controls (CTR; mean age: 27.1 yrs) provided written informed consent, and all study procedures were approved by the local IRB. Participants completed a repeated measures design with three distinct conditions (in random order). For each, participants walked for 8 minutes within the Computer Assisted Rehabilitation Environment (CAREN; Motek Medical BV, Netherlands) at their self-selected speed (mean=1.1±0.2 m/s) while performing a secondary cognitive task ("easy" and "hard") – each involving distinct attentional demands – or no ("none") secondary task. For the "easy" task, a single object was displayed on a screen within direct line-of-sight with changing shapes. Participants were asked to press a button whenever a square appeared. For the "hard" task, two objects were displayed simultaneously, with individually changing shapes and colors. Participants were asked to press a button when both objects were either the same shape or same color.

Three-dimensional trunk positions were tracked (120Hz) using a 12-camera motion capture system (Vicon; Oxford, UK). Accelerations were derived using model-based computations in Visual3D (C-motion; Germantown, MD). Local stability of trunk movements was quantified from trunk accelerations using short-term maximum lyapunov exponents (λ) [5]. Trunk ranges of motion (ROM) were also calculated in each plane as the angular deviation relative to the global coordinate system. A two-way ANOVA compared group (CTR, TTA, TFA) and task (none, easy, hard) for each plane of movement ($p<0.05$). Eta squared for main effects of group are presented as an index of the power of the effect.

RESULTS AND DISCUSSION

There were no significant interactions between the effects of levels of ULLA and task condition on trunk local stability; however, there were significant main effects of group in the mediolateral, $F(2,62) = 20.58$, $p < 0.001$, $\eta^2 = 0.39$, and axial, $F(2,62) = 28.30$, $p < 0.001$, $\eta^2 = 0.47$ planes. In general, CTR demonstrated less trunk local stability ($p<0.001$) and smaller trunk ROM ($p<0.001$) compared to persons with ULLA (Figure 1). However, there were no group differences in anteroposterior plane stability ($p=0.34$). In the mediolateral and axial planes, TFA demonstrated more trunk local stability, with no differences between cognitive tasks. While the latter is contrary to prior work [6], here we used a different secondary task and a more homogenous sample with respect to age, time since amputation, and walking speed. The concomitant increases in trunk motion and local stability in the TFA group suggest these individuals may employ larger global movements proximally to account for disrupted proprioception and motor function distally. Within these increased global movements, however, this subset of TFA appear to have developed a trunk neuromuscular control strategy that is better able to accommodate increased kinematic variability within the system. This is potentially a compensatory mechanism to prevent balance impairments that can accompany trunk movements outside the base of support. Clinically, rehabilitation goals should aim to increase trunk neuromuscular control via targeted trunk rehabilitation incorporating both anticipatory feed-forward (e.g., active muscle stiffness) and reactive feed-back (e.g., postural adjustments through proprioceptive mechanisms), resulting in increased local stability and effective control of local mechanical disturbances.

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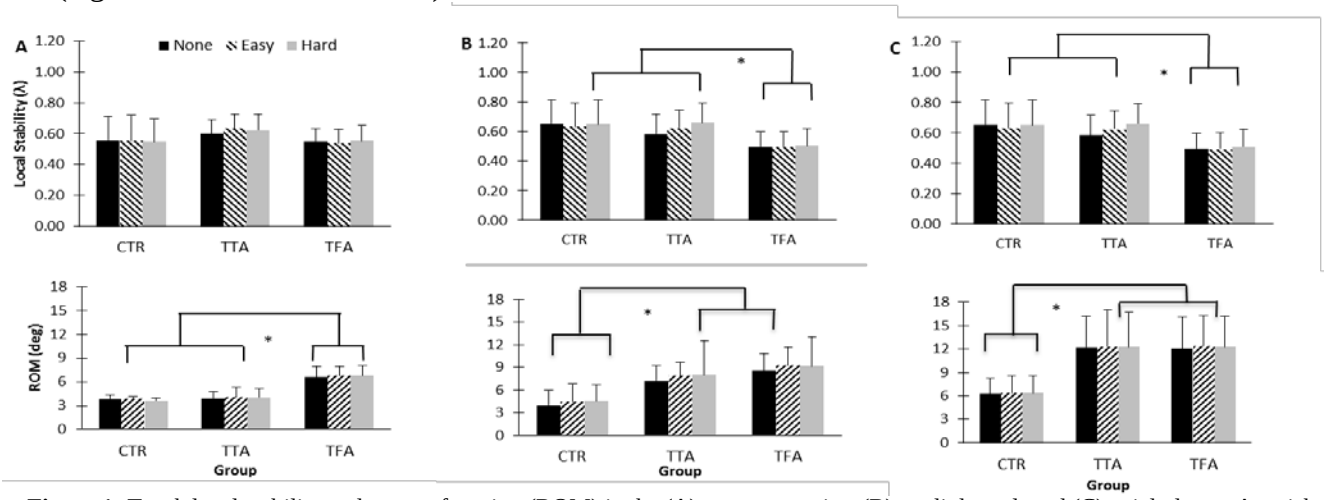

Figure 1: Trunk local stability and range of motion (ROM) in the (A) anteroposterior, (B) mediolateral, and (C) axial planes. Asterisks indicate significant ($p<0.001$) differences between groups.

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Full Length Article

Trunk and pelvic dynamics during transient turns among individuals with unilateral traumatic lower limb amputation

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Biomechanics

ABSTRACT

Prior work has identified alterations in trunk-pelvic dynamics with lower limb amputation (LLA) during in-line walking; however, evaluations of other ambulatory tasks are limited. Turns are ubiquitous in daily life but can be challenging for individuals with LLA, prompting additional or unique proximal compensations when changing direction, which over time may lead to development of low back pain. We hypothesized such proximal kinematic differences between persons with and without LLA would exist in the sagittal and frontal planes. Three-dimensional trunk and pelvic kinematics, translational and rotational momenta, and coordination phase/variability were compared among eight persons with unilateral LLA (4 with transfemoral amputation and 4 with transtibial amputation), and five uninjured controls, who performed 90-degree turns to the left (n = 10) and right (n = 10). Participants self-selected the turn strategy (i.e., step vs. spin) and pivot limb in response to verbal cues regarding when and which direction to turn. Coordination variability and translational angular momenta did not differ between groups in either turn type. During spin turns, frontal rotational angular momenta were larger and frontal trunk-pelvis range of motion was smaller among persons with vs. without LLA. During step turns, pelvis leading transverse coordination was more frequent, frontal trunk rotational angular momentum was smaller, and sagittal pelvis range of motion was larger among persons with vs. without LLA. Altered and task-dependent modulation of trunk-pelvic dynamics among persons with LLA provides additional support for a potential link between repeated exposures to altered trunk-pelvic dynamics with elevated low back pain risk.

1. Introduction

Persons with lower limb amputation (LLA) often walk with compensatory movement strategies involving a prominent reliance on the trunk and pelvis (Goujon-Pillet, Sapin, Fodé, & Lavaste, 2008). Altered kinematic features and coordination of these two segments have been associated with elevated demands on the low back (Hendershot & Wolf, 2014), increased inter-segmental rigidity (Russell Esposito & Wilken, 2014), and larger trunk muscular forces and spinal loads (Shojaie, Hendershot, Wolf, & Bazrgari, 2016; Yoder, Petrella, & Silverman, 2015). These altered loads and asymmetric trunk-pelvis kinematics among persons with LLA have been suggested as key factors in disc degeneration and passive ligamentous strain potentially leading to development of low back pain (LBP; Devan, Hendrick, Ribeiro, Hale, & Carman, 2014; Gailey, Allen, Castles, Kucharik, & Roeder, 2008). As such, differences in trunk/pelvis kinematics between persons with and without LLA have been characterized during in-line walking (Goujon-Pillet et al.,

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2008; Hendershot & Wolf, 2014; Morgenroth et al., 2010). Yet, in-line walking is but one movement among many required for functional independence. Thus, characterizing the extent to which persons with LLA utilize proximal compensations during other (perhaps more demanding) tasks/activities of daily living would facilitate a more comprehensive understanding of biomechanical contributors to LBP risk.

Transient (i.e., non-steady-state) tasks embedded within in-line walking are ubiquitous and often necessary to adequately navigate an environment. Turns, in particular, account for approximately half of daily steps (Glaister, Bernatz, Klute, & Orendurff, 2007; Sedgeman, Goldie, & Iansek, 1994). Biomechanically, turns require a redirection of the body's center of mass, typically as a change in direction between 76 and 120 degrees (Sedgeman et al., 1994) executed using either a step (turn direction is contralateral to pivot leg) or spin strategy (turn direction is ipsilateral to pivot leg; Taylor, Dabnichki, & Strike, 2005). Among persons with LLA, compromised ankle function alters control of braking/propulsive and mediolateral forces during a turn (albeit along a circular vs. orthogonal path; Segal, Orendurff, Czerniecki, Shofer, & Klute, 2008; Ventura, Segal, Klute, & Neptune, 2011), thereby likely necessitating proximal adaptations of the trunk/pelvis to adequately redirect the body's center of mass. Furthermore, proximal compensations during turns may also exist to minimize discomfort within the residual limb-socket interface, particularly as it relates to torsion/shear (Heitzmann et al., 2015).

Inter-segmental coordination and momentum have been used for identification of compensational movement strategies during ambulation. For example, persons with unilateral LLA generate and arrest larger trunk and pelvic segmental momenta during walking (Gaffney, Murray, Christiansen, & Davidson, 2016), as well as alter segmental coordination strategies dependent on the presence of current LBP (Russell Esposito & Wilken, 2014). While recent efforts have similarly identified altered trunk-pelvic coordination strategies in able-bodied individuals (with and without LBP) executing turns (Smith & Kulig, 2016), there exist no studies specifically focused on trunk and pelvic compensations during turns among persons with LLA. Thus, the primary purpose of this study was to characterize proximal compensations using inter-segmental momenta and coordination during transient (90-degree) turns among persons with LLA. Although turns are predominantly associated with movement in the transverse plane, it was hypothesized that persons with vs. without LLA execute turns with altered trunk-pelvic segmental coordination, particularly in the sagittal and frontal planes, to overcome the aforementioned challenges associated with modulating braking/propulsive and mediolateral forces with altered ankle function. Secondarily, we hypothesized that such alterations in trunk-pelvic coordination would also be associated with larger ranges of segmental momenta among persons with vs. without LLA.

2. Methods

2.1. Participants

Eight persons with unilateral LLA of traumatic etiology (four with transtibial amputation [TTA], three with transfemoral amputation, and one with knee disarticulation [TFA]) and five persons without LLA (uninjured controls; CTRL) completed this study (Table 1). All participants provided informed consent approved by the Walter Reed National Military Medical Center Institutional Review Board. All participants were free of neurological and orthopaedic injury aside from lower limb amputation, were able to ambulate over even terrain without an assistive device, and were not experiencing any moderate or severe discomfort/pain, regardless of cause, at any point during data collection, as measured by overall pain scores less than 4 cm on a 10 cm Visual Analog Scale (Jensen, Chen, & Brugger, 2003). Of the persons with TTA, 2 wore the RUSH and 2 wore the Vari-Flex XC foot. Of the persons with TFA or knee disarticulation, 2 wore the X3 microprocessor knee and Vari-Flex XC foot, 1 wore the X2 microprocessor knee and Vari-Flex XC foot, and 1 wore the Total Knee 2100 mechanical knee and Vari-Flex XC foot.

Table 1
Demographic information by participant category (CTRL = uninjured controls, TTA = persons with transtibial amputation, and TFA = persons with transfemoral amputation or knee disarticulation). Note, there were no significant differences in demographic information or walking speeds (all $P > .167$).

	Age (yr)	Months Since Amputation	Height (m)	Mass (kg)	In-line Walking Speed (m/s)
CTRL	20		1.8	61.5	1.4
	28		1.7	88.4	1.4
	31		1.9	105.7	1.4
	28		1.9	72.6	1.3
	29		1.8	83.5	1.3
TTA	24	5.5	1.8	90.9	1.4
	27	47.8	1.8	106.9	1.4
	34	133.3	1.9	89.9	1.5
	45	17.7	1.8	135.6	1.5
TFA	34	59.7	1.7	71.4	1.1
	23	15.8	1.9	96.2	1.4
	26	59.0	1.7	74.9	1.4
	25	32.9	1.7	101.2	1.2

2.2. Experimental procedures

Each participant performed 20 turns involving a 90-degree change in direction to the left (n = 10) and right (n = 10). Participants walked at their self-selected speed along a 12-foot straight path and were verbally cued to turn left or right at a specified and consistent location (approximately 6 feet away from the turning point, allowing the participant to ultimately self-select the pivot limb). Turn direction was randomized, and no specific guidance was provided for which foot or type of turn (i.e., step vs. spin) to employ. Full-body kinematics were collected by tracking (120 Hz) 70 reflective markers with a 27-camera motion capture system (Vicon, Oxford, UK). Markers were placed on the C7 and T10 spinous processes, sternal notch, xiphoid process, and bilaterally on the acromia, ASIS, and PSIS. Lower and upper extremities were tracked as 6 DOF segments, with markers placed accordingly (Collins, Ghoussayni, Ewins, & Kent, 2009). All kinematic data were filtered at 6 Hz using a 5th order Butterworth filter.

2.3. Dependent measures and data analyses

The pivot foot and type of turn (step or spin) were first determined using a previously described, automated method (*cf.* Golyski & Hendershot, 2017), and heel strike/toe-off events were calculated using the position of the feet relative to the pelvis (Zeni, Richards, & Higginson, 2008). Step lengths were calculated for the step leading into pivot and the step after pivot as the absolute distance between the positions of heel strikes of each respective step relative to pivot. Stride widths were evaluated using the heel strike positions of the steps before, during, and after the turn (Huxham, Gong, Baker, Morris, & Iansek, 2006).

Three-dimensional trunk segmental kinematics were computed, relative to the pelvis, using Visual3D (Version 5.02.27, C-Motion Inc., Germantown, MD, USA), with local coordinate systems defined by a static calibration trial. Trunk-pelvis range of motion was calculated for each plane over the period from heel strike of the step before pivot to the toe-off of the step after pivot. Individual trunk and pelvic segmental trajectories were also computed and exported to MATLAB (Release 2015a, The MathWorks, Inc. Natick, MA, USA). Using these, tri-planar translational (Eq. (1)) and rotational (Eq. (2)) angular momenta of the trunk and pelvis segments were calculated as described by Gaffney et al. (2016), and normalized by each participant's body mass, height, and self-selected in-line walking speed (Herr & Popovic, 2008). Translational angular momentum (TAM) for the trunk and pelvis segments was calculated as:

$$\boldsymbol{h}_{i/Foot} = (\boldsymbol{r}_i - \boldsymbol{r}_{Foot}) \times m_i(\boldsymbol{v}_i - \boldsymbol{v}_{Foot}) \tag{1}$$

where $\boldsymbol{r}_i$ is the position vector of the segment's center of mass, $\boldsymbol{r}_{Foot}$ is the position vector of the pivot foot, m_i is the mass of the segment, $\boldsymbol{v}_i$ is the velocity vector of the segment's center of mass, and $\boldsymbol{v}_{Foot}$ is the velocity vector of the pivot foot. TAM was evaluated only during the period from heel strike before the turn to toe-off after the turn (i.e. pivot stance). Rotational angular momentum (RAM) for the trunk and pelvis segments was calculated as:

$$\boldsymbol{h}_i = \boldsymbol{I}_i \cdot \boldsymbol{w}_i \tag{2}$$

where $\boldsymbol{I}_i$ is the moment of inertia tensor for the segment of interest and $\boldsymbol{w}_i$ is the segment's angular velocity vector. RAM was evaluated during the same period as trunk-pelvis range of motion. Both TAM and RAM were resolved in the three planes of motion, defined using center of mass velocity (to define a forward direction), gravity, and the resulting cross product; TAM and RAM ranges (i.e., max-min) were extracted within each plane for subsequent analyses.

Finally, inter-segmental coordination of the trunk and pelvis in each plane of movement was calculated using a vector coding method described by Needham, Naemi, and Chockalingam (2014). For this, each turn was subsequently divided into two phases: (1) pivot stance; defined as the period from heel strike to toe-off of the foot in stance during the apex of the turn, and (2) pivot swing; defined as the period from pivot foot toe-off to subsequent ipsilateral heel strike. Time-series trajectories of the trunk and pelvic angle defined a 0–360° relative coupling angle, which at each time point is separated into one of eight 45° bins to evaluate the frequency of a given coordination mode (in-phase, anti-phase, trunk-phase, and pelvic-phase) in both pivot stance and pivot swing; circular statistics were used to define the mean and variability of each coupling angle/phase while preserving directionality of the trunk-pelvis relative coupling angle (Hamill, Haddad, & McDermott, 2000; Needham et al., 2014; Watson & Batschelet, 1982). Note, a common alternative method for assessing segmental coordination is continuous relative phase, but this method does not explicitly quantify trunk- and pelvic-phase coordination modes (i.e., dominance of a given segment).

2.4. Statistical analyses

Given that turn type was not controlled as part of the experimental design (i.e., the pivot foot was selected by the participant), and no *a priori* hypotheses were formulated as to how turn type would influence the dependent variables, no explicit comparisons were made between turn strategies. Instead, Mann-Whitney *U* tests were used to compare all dependent measures between persons with LLA vs. CTRL, separately within each turn type; statistical significance was concluded at $P < .050$. All statistical analyses were performed in SPSS (version 21.0; IBM SPSS Inc., Chicago, IL). Unless otherwise specified, data are reported as medians (interquartile ranges). In total, 60 (of 80) trials/turns from persons with TFA, 71 (of 80) from persons with TTA, and 77 (of 100) from persons without LLA were included as part of subsequent analyses due to marker drop out and/or in-line walking periods of insufficient length before and after the turn.

3. Results

3.1. Turn type and temporal-spatial parameters

Persons with TFA performed 32 step turns (13/19 on the intact/prosthetic limb, respectively) and 28 spin turns (9/19 on the intact/prosthetic limb). Persons with TTA performed 51 step turns (34/17 on the intact/prosthetic limb) and 20 spin turns (19/1 on the intact/prosthetic limb). CTRL performed 51 step and 26 spin turns. During spin turns, no significant differences were observed between persons with or without amputation in step lengths before pivot [LLA: 58.9 (11.2), CTRL: 64.0 (7.9) cm; $P = .343$], step lengths after pivot [LLA: 60.8 (27.0), CTRL: 60.1 (9.5) cm; $P = .734$], and stride widths over the pivot [LLA: 13.8 (11.0), CTRL: 17.6 (7.6) cm; $P = .427$]. Similarly, during step turns no significant differences were observed between persons with and without amputation in step lengths before pivot [LLA: 62.3 (9.2), CTRL: 67.2 (16.8) cm; $P = .310$], step lengths after pivot [LLA: 67.3 (15.6), CTRL: 68.0 (8.8) cm; $P = .586$], and stride widths over the pivot [LLA: 45.0 (6.2), CTRL: 46.4 (5.0) cm; $P = .363$].

3.2. Trunk and pelvic kinematics

During spin turns, sagittal plane range of motion was similar between individuals with vs. without LLA [LLA: 8.5 (3.1), CTRL: 6.9 (5.9)°; $P = 1.000$]. Conversely, frontal plane trunk-pelvis range of motion was significantly smaller in the LLA group than the CTRL group [LLA: 11.4 (3.5), CTRL: 15.3 (6.3)°; $P = .004$]. Transverse plane trunk-pelvis range of motion was not significantly different between groups [LLA: 19.2 (8.4), CTRL: 16.5 (5.0)°; $P = .384$]. During step turns, trunk-pelvis range of motion was larger in the LLA vs. CTRL groups in the sagittal plane [LLA: 8.9 (2.6), CTRL: 6.5 (3.9)°; $P = .047$], but no significant differences between groups were observed in the frontal plane [LLA: 11.7 (5.0), CTRL: 17.5 (7.1)°; $P = .201$] or transverse plane [LLA: 15.4 (5.0), CTRL: 14.6 (2.1)°; $P = .586$].

3.3. Trunk and pelvic angular momenta

3.3.1. Translational angular momentum

During both spin ($P > .157$) and step turns ($P > .087$), group was not a significant main effect for TAM of the trunk or pelvis in any plane (Fig. 1/Table 2).

3.3.2. Rotational angular momentum

During spin turns, trunk and pelvis RAM in the sagittal plane were not significantly different by level of amputation ($P > .115$). However, frontal plane trunk RAM ($P < .001$), and pelvis RAM ($P = .047$) were larger in individuals with vs. without LLA. Additionally, trunk and pelvis RAM in the transverse plane were not significantly different by group ($P > .678$; see Fig. 2). During step turns, trunk and pelvis RAM in the sagittal plane were not significantly different by level of amputation ($P > .698$). Frontal plane trunk RAM was larger among individuals with vs. without LLA ($P < .001$), while frontal plane pelvis RAM was not ($P = .310$). No significant differences were observed in the transverse plane between groups in either trunk or pelvis RAM ($P > .391$; see Fig. 2/Table 2).

3.4. Trunk and pelvic coordination

No significant differences by group were observed in trunk-pelvis coordination angle variability between persons with vs. without LLA ($P > .098$; Table 3). During spin turns, there were no significant differences in the frequency of any coordination mode in either stance or swing phase ($P > .082$). During step turns, transverse plane pelvis-phase coordination was significantly more frequent in individuals with vs. without LLA ($P = .036$), with no other coordination mode exhibiting significant differences between populations ($P > .068$; Fig. 3).

4. Discussion

The aim of this study was to characterize compensatory movements of the trunk and pelvis during transient 90 degree turns in persons with vs. without LLA. We hypothesized that differences in coordination would exist principally in the sagittal and frontal planes among persons with LLA, concurrent to increases in segmental momenta, to overcome limitations associated with altered ankle function. In support of our hypotheses, ranges of motion, segmental rotational momenta, and frequency of coordination modes differed between individuals with and without LLA, depending on the plane and type of turn employed.

4.1. Trunk-pelvis coordination

Coordinated movements of the trunk and pelvis are important for efficient and steady ambulation, and alterations in trunk-pelvic coordination strategy (or its variability) have been associated with current or future risk for LBP (Hamill, Van Emmerik, Heiderscheit, & Li, 1999; Seay, Van Emmerik, & Hamill, 2011). Though more frequent in-phase coordination has been associated with LBP (Seay et al., 2011) and may decrease relative motion of the trunk to the pelvis as a guarding strategy (Russell Esposito & Wilken, 2014; van der Hulst, Vollenbroek-Hutten, Rietman, & Hermens, 2010) by preventing strain on anatomical structures of the low back, in the

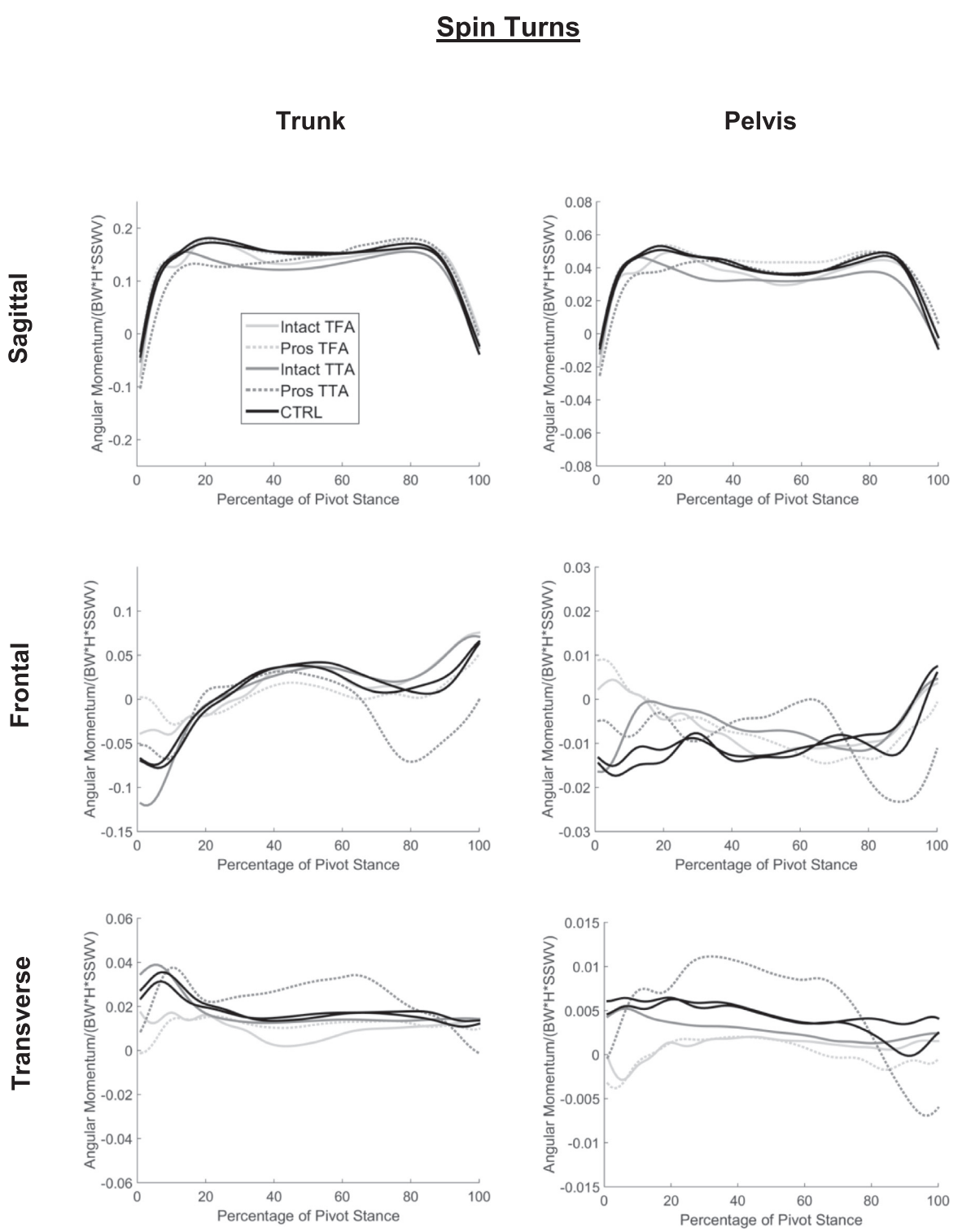

Fig. 1. Ensemble averages of trunk and pelvis translational angular momentum (TAM) in the sagittal, frontal, and transverse planes. Data are normalized by body weight (BW), height (H), and self-selected (in-line) walking velocity (SSWV). The two traces for each turn strategy executed by controls represent turns performed on the right and left feet and are provided as an indicator of healthy variability in angular momenta in each plane. For visualization purposes, frontal and transverse TAM for both the trunk and pelvis were negated for left turns.

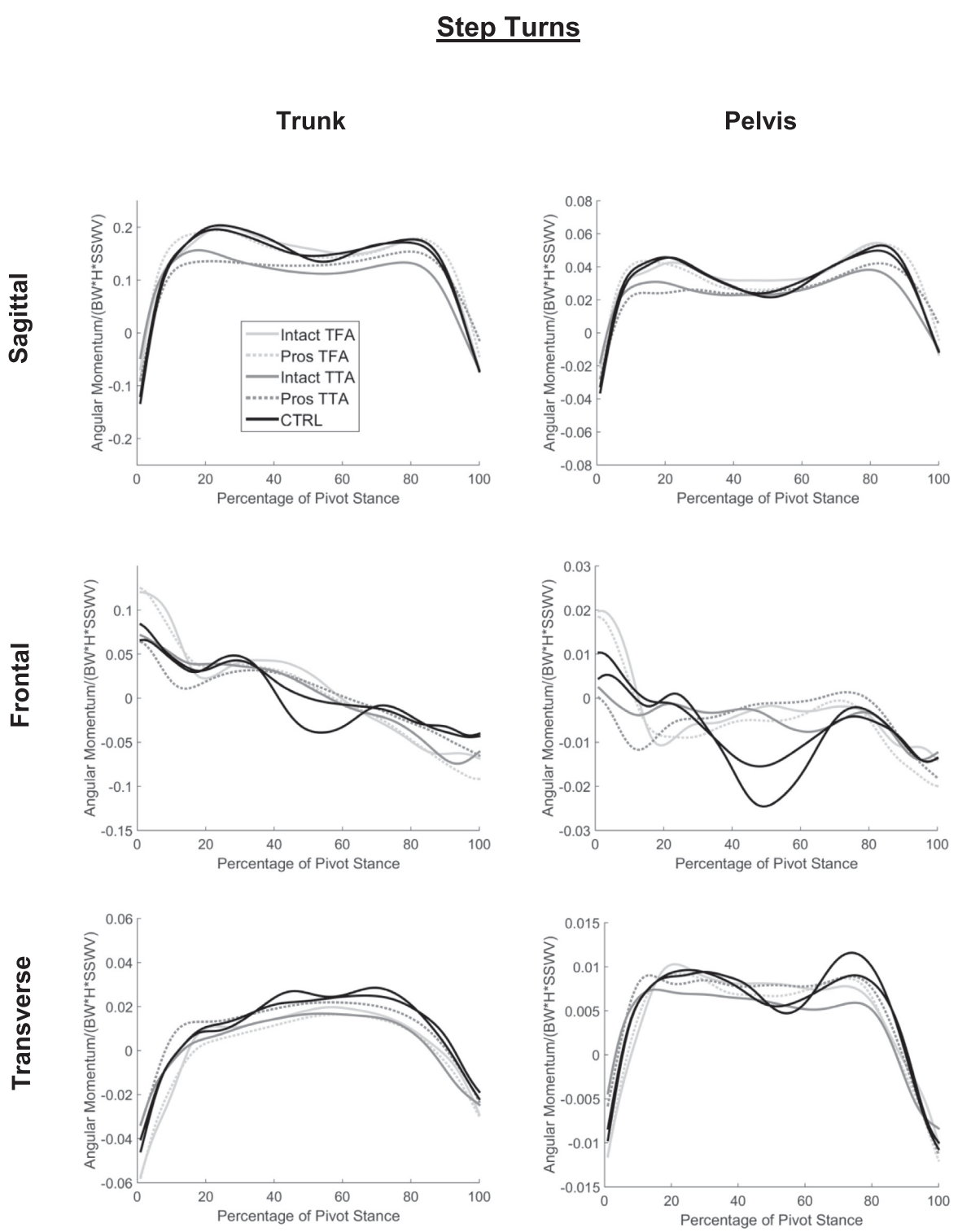

Fig. 1. (*continued*)

transverse plane individuals with vs. without LLA exhibited a lesser (though not significant) frequency of in-phase coordination compared to uninjured controls. Such a decrease in in-phase coordination could be a compensatory mechanism for reduced ankle function, but may indicate an increased risk of repetitive injury. Though no participants reported acute pain during collection, a limitation of the present study was that LBP history was not collected.

To the authors' knowledge, the only previous study of trunk-pelvis coordination during turns evaluated differences in transverse

Table 2
Median (interquartile range) ranges in trunk and pelvic translational angular momenta (TAM) and rotational angular momenta (RAM) for individuals with unilateral lower limb amputation (LLA) and uninjured controls (CTRL), during spin/step turns. TAM was calculated during pivot stance. RAM was calculated during the period from heel strike of the step before the pivot step to toe-off of the step after the pivot step, respectively. For metrics marked by * and **, groups were significantly different at the $\alpha = 0.05$ and $\alpha = 0.001$ levels, respectively. All momenta are normalized by body weight, height, and self-selected (in-line) walking speed.

		Spin Turns	
		LLA	CTRL
Sagittal	Trunk TAM Range	0.2781 (0.0824)	0.2645 (0.0608)
	Pelvis TAM Range	0.0733 (0.0206)	0.0695 (0.0172)
	Trunk RAM Range	0.0022 (0.0011)	0.0021 (0.0008)
	Pelvis RAM Range	0.0004 (0.0002)	0.0003 (0.0001)
Frontal	Trunk TAM Range	0.1420 (0.1087)	0.1474 (0.0927)
	Pelvis TAM Range	0.0337 (0.0179)	0.0320 (0.0213)
	Trunk RAM Range**	0.0055 (0.0022)	0.0038 (0.0011)
	Pelvis RAM Range*	0.0006 (0.0004)	0.0005 (0.0001)
Transverse	Trunk TAM Range	0.0313 (0.0158)	0.0278 (0.0189)
	Pelvis TAM Range	0.0135 (0.0076)	0.0084 (0.0058)
	Trunk RAM Range	0.0059 (0.0023)	0.0056 (0.0021)
	Pelvis RAM Range	0.0014 (0.0010)	0.0017 (0.0007)
		Step Turns	
		LLA	CTRL
Sagittal	Trunk TAM Range	0.2931 (0.0506)	0.3095 (0.0591)
	Pelvis TAM Range	0.0808 (0.0221)	0.0835 (0.0162)
	Trunk RAM Range	0.0023 (0.0013)	0.0024 (0.0008)
	Pelvis RAM Range	0.0004 (0.0003)	0.0003 (0.0001)
Frontal	Trunk TAM Range	0.1817 (0.1046)	0.1580 (0.0539)
	Pelvis TAM Range	0.0326 (0.0204)	0.0385 (0.0215)
	Trunk RAM Range**	0.0046 (0.0021)	0.0030 (0.0005)
	Pelvis RAM Range	0.0006 (0.0004)	0.0005 (0.0002)
Transverse	Trunk TAM Range	0.0683 (0.0182)	0.0717 (0.0210)
	Pelvis TAM Range	0.0235 (0.0090)	0.0270 (0.0082)
	Trunk RAM Range	0.0058 (0.0034)	0.0070 (0.0030)
	Pelvis RAM Range	0.0015 (0.0011)	0.0014 (0.0006)

Units: Angular Momentum/(Body Weight*Height*Self Selected Walking Velocity).

plane coordination only, in persons with and without LBP during spin turns (Smith & Kulig, 2016). The dominant in-phase coordination in the transverse plane during stance was consistent with this work, though no significant differences were found between groups.

Characterization of trunk-pelvis coordination during in-line walking (Russell Esposito & Wilken, 2014) found higher frequencies of anti-phase coordination in individuals with TFA relative to uninjured controls in the sagittal and frontal planes. In contrast, we only observed a significant increase in transverse plane pelvic-phase coordination in persons with vs. without LLA during step turns, which are more biomechanically similar to in-line walking than spin turns (Taylor et al., 2005). In contrast to the hypothesized changes in sagittal and frontal coordination, the only significant difference between groups was in the transverse plane during step turns, which nonetheless suggests alternative proximal movement strategies within the LLA population. In support of our hypothesis, differences between populations were observed in trunk-pelvis range of motion and angular momenta in the sagittal and frontal planes.

4.2. Sagittal plane

Significantly larger sagittal trunk-pelvis range of motion during step turns among persons with vs. without LLA is consistent with previous observations of trunk-pelvis kinematics during in-line walking (Goujon-Pillet et al., 2008). Such larger trunk flexion angles may be a compensation to facilitate hip extension, which is hampered by hip flexion contractures (Gailey et al., 2008), but this motion may also increase demand on trunk extensors (Hendershot & Wolf, 2014). Moreover, this more extreme sagittal trunk-pelvic movement is also consistent with the larger (though not significant) observed pelvis RAM in persons with vs. without LLA, and in agreement with a previous study of in-line walking (Gaffney et al., 2016).

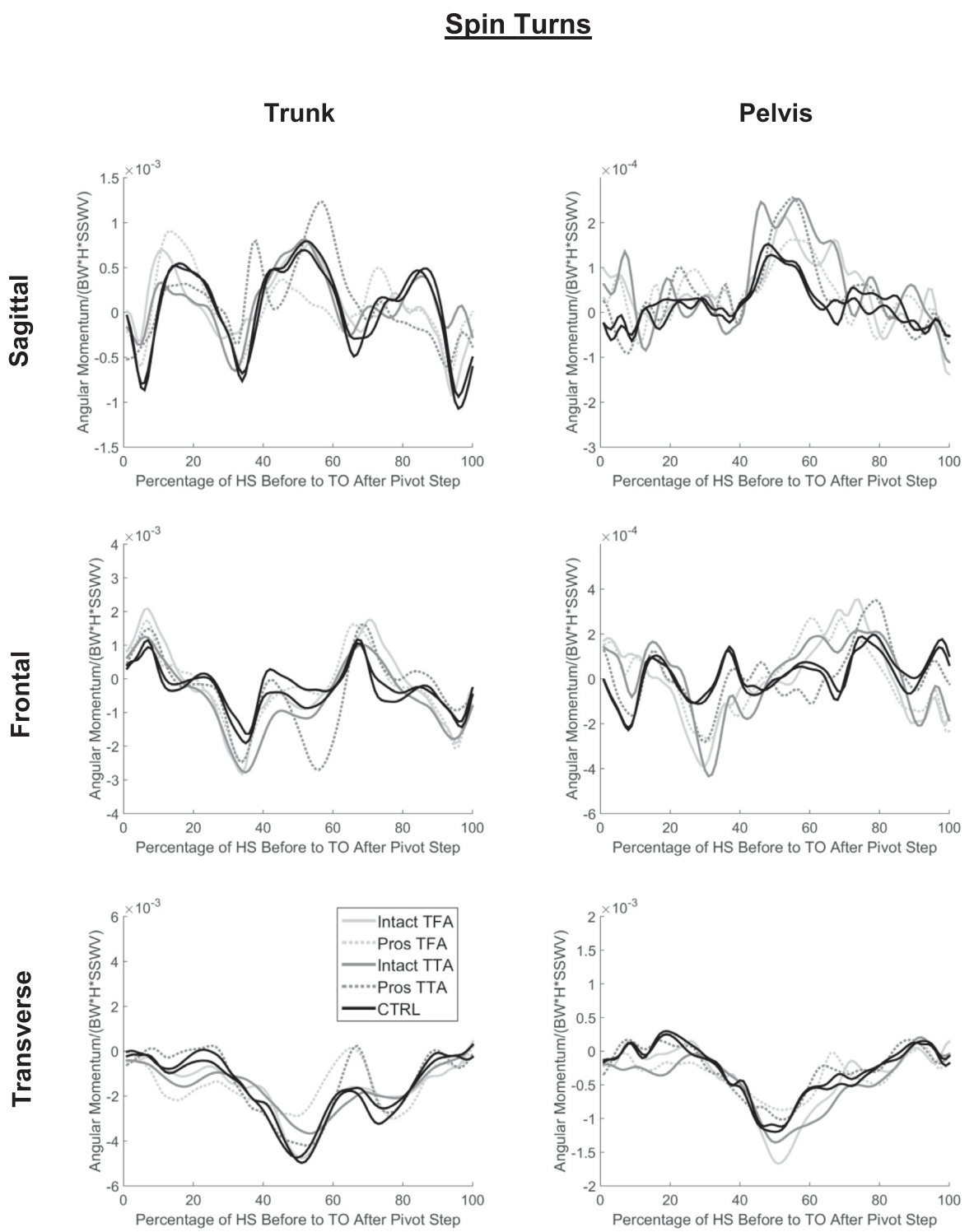

Fig. 2. Ensemble averages of trunk and pelvis rotational angular momentum (RAM) in the sagittal, frontal, and transverse planes during spin/step turns. Data are normalized by body weight (BW), height (H), and self-selected (in-line) walking velocity (SSWV). The two traces for each turn strategy executed by controls represent turns performed on the right and left feet and are provided as an indicator of healthy variability in angular momenta in each plane. For visualization purposes, frontal and transverse RAM for both the trunk and pelvis were negated for left turns.

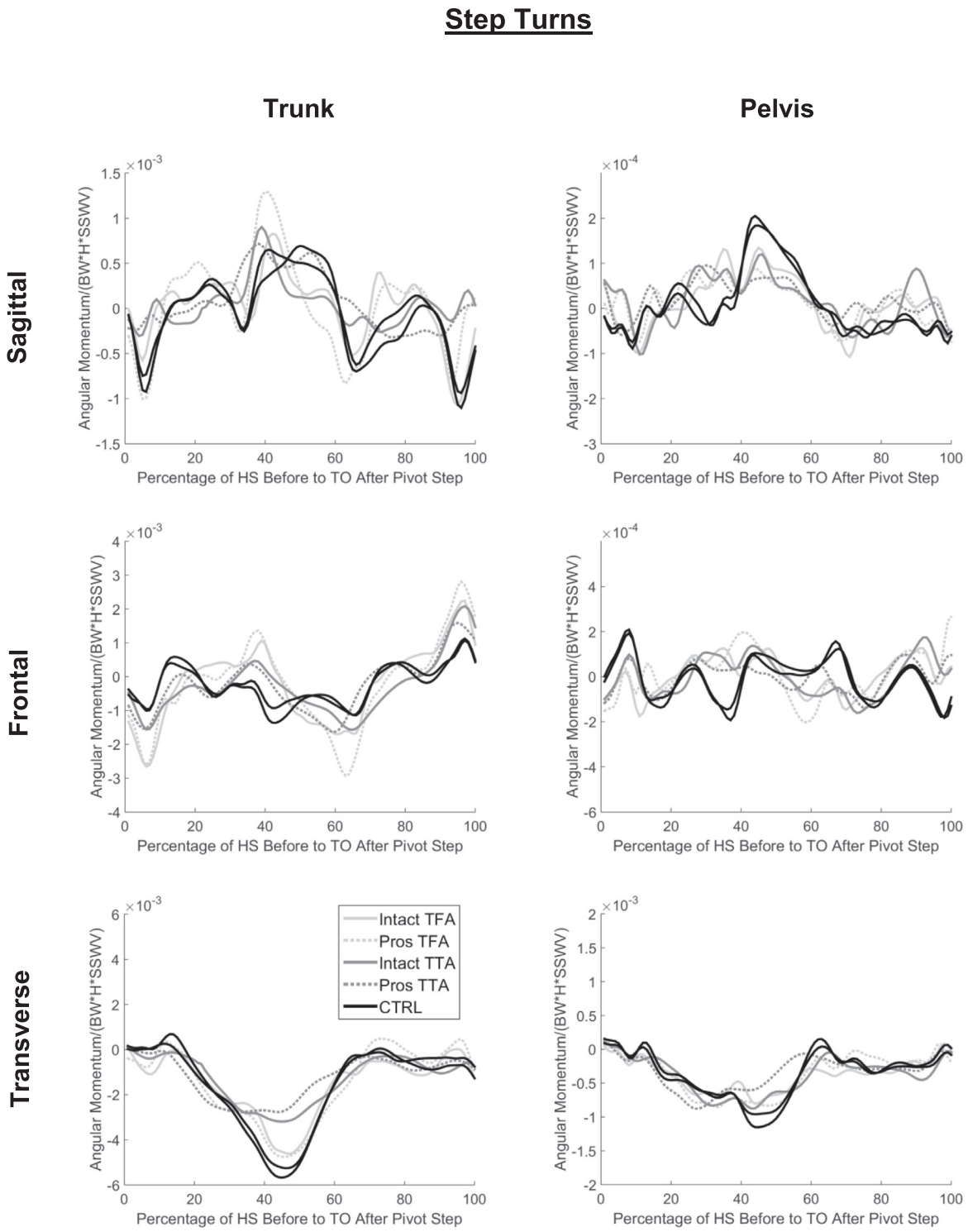

Fig. 2. (*continued*)

4.3. Frontal plane

In contrast to increases in trunk-pelvis range of motion among persons with vs. without LLA during in-line walking (Goujon-Pillet et al., 2008; Yoder et al., 2015), frontal plane range of motion during spin turns was smaller in the LLA than CTRL group (no difference between groups during step turns). During in-line walking, a larger range of motion is primarily due to increased lateral

Table 3
Median (interquartile range) variability of trunk-pelvis coupling angle during spin/step turns in stance/swing of the pivot limb for persons with unilateral lower limb amputation (LLA), and uninjured controls (CTRL). No significant differences were found between groups at the $\alpha = 0.05$ level.

		Spin Turns	
		LLA	CTRL
Sagittal	Pivot Stance (°)	21.7 (33.9)	23.2 (17.6)
	Pivot Swing (°)	8.7 (20.2)	19.6 (15.0)
Frontal	Pivot Stance (°)	10.8 (27.1)	14.7 (18.7)
	Pivot Swing (°)	20.1 (33.5)	27.5 (25.6)
Transverse	Pivot Stance (°)	12.1 (21.1)	8.8 (6.2)
	Pivot Swing (°)	18.0 (43.0)	26.0 (20.1)
		Step Turns	
		LLA	CTRL
Sagittal	Pivot Stance (°)	23.2 (7.5)	22.7 (17.1)
	Pivot Swing (°)	22.2 (31.2)	13.8 (19.9)
Frontal	Pivot Stance (°)	15.9 (9.1)	13.5 (8.0)
	Pivot Swing (°)	28.2 (31.9)	28.1 (20.9)
Transverse	Pivot Stance (°)	14.7 (6.0)	12.3 (10.4)
	Pivot Swing (°)	23.5 (26.6)	24.9 (15.7)

trunk lean over the prosthetic limb and is considered a compensation, at least in part, for reduced residual limb function (Hendershot & Wolf, 2014; Rueda et al., 2013). Future studies exploring turns on the intact vs. prosthetic side may elucidate the basis for reduced frontal plane range of motion, though we speculate the relative decrease in lateral trunk lean throughout turns may be a result of the more proximal (i.e., hip vs. ankle) strategy and generally not leaning into/away from the turn to minimize excursions of the body center of mass and improve stability (Ventura et al., 2011). Despite the trends in frontal plane trunk-pelvis range of motion being inconsistent with those of existing literature, differences in frontal plane trunk RAM (which is dependent on segmental angular velocity) between groups during both turn types were apparent. Such differences are consistent with our hypothesis and previous work identifying larger ranges in whole body frontal plane angular momentum in persons with LLA (albeit during in-line walking; Silverman & Neptune, 2011). Large changes in whole-body angular momentum in the frontal plane have also been correlated with poorer clinical balance outcomes post-stroke (Nott, Neptune, & Kautz, 2014). Moreover, such deviations in trunk and pelvis angular momentum in the frontal plane are of particular interest since these segments are the principal contributors to whole body angular momentum in the frontal plane (Herr & Popovic, 2008). During spin turns the more extreme frontal trunk angular velocity coupled with smaller trunk-pelvic range of motion could suggest a trunk-stiffening strategy (Arendt-Nielsen, Graven-Nielsen, Svarrer, & Svensson, 1996; Lamoth et al., 2002), similar to the segmental rigidity identified among persons with TFA during in-line walking (Russell Esposito & Wilken, 2014); however, such a stiffening strategy would likely be associated with increased in-phase coordination (Wu et al., 2014) – a trend we did not observe here with the vector coding method.

4.4. Transverse plane

Larger axial trunk rotations have been observed in persons with TFA during in-line walking (Goujon-Pillet et al., 2008), which are concerning given the association of such rotations with LBP (Fujiwara et al., 2000; Morgenroth, Medverd, Seyedali, & Czerniecki, 2014). We did not observe differences in transverse plane trunk-pelvis range of motion, though this could be attributed to turns requiring more control over transverse plane angular displacements. However, during step turns, range in transverse trunk RAM was smaller, albeit not significantly, in persons with LLA compared to uninjured controls. As illustrated in Fig. 2, at approximately 50% of the turn the trunk RAM was smaller for turns on both the prosthetic and intact limbs in persons with TFA and TTA vs. controls, indicating a smaller peak trunk angular velocity in the LLA group. This contradicts previous preliminary findings which suggested persons with unilateral TTA execute step turns with larger transverse trunk angular velocities than uninjured controls (Taylor & Strike, 2009).

Though the host of kinematic differences between turns and in-line walking (Taylor et al., 2005) precludes direct comparison of angular momentum components to previous work, qualitatively, transverse trunk and pelvis RAM were the most different in shape between the two ambulation tasks (c.f. Gaffney et al., 2016), stemming from the seemingly necessary peak in transverse angular velocity. Moreover, differences in the range of TAM/RAM between in-line walking and turns were most pronounced in the transverse plane, and were larger during transient turns by factors of 2 and 3 for TAM and RAM, respectively.

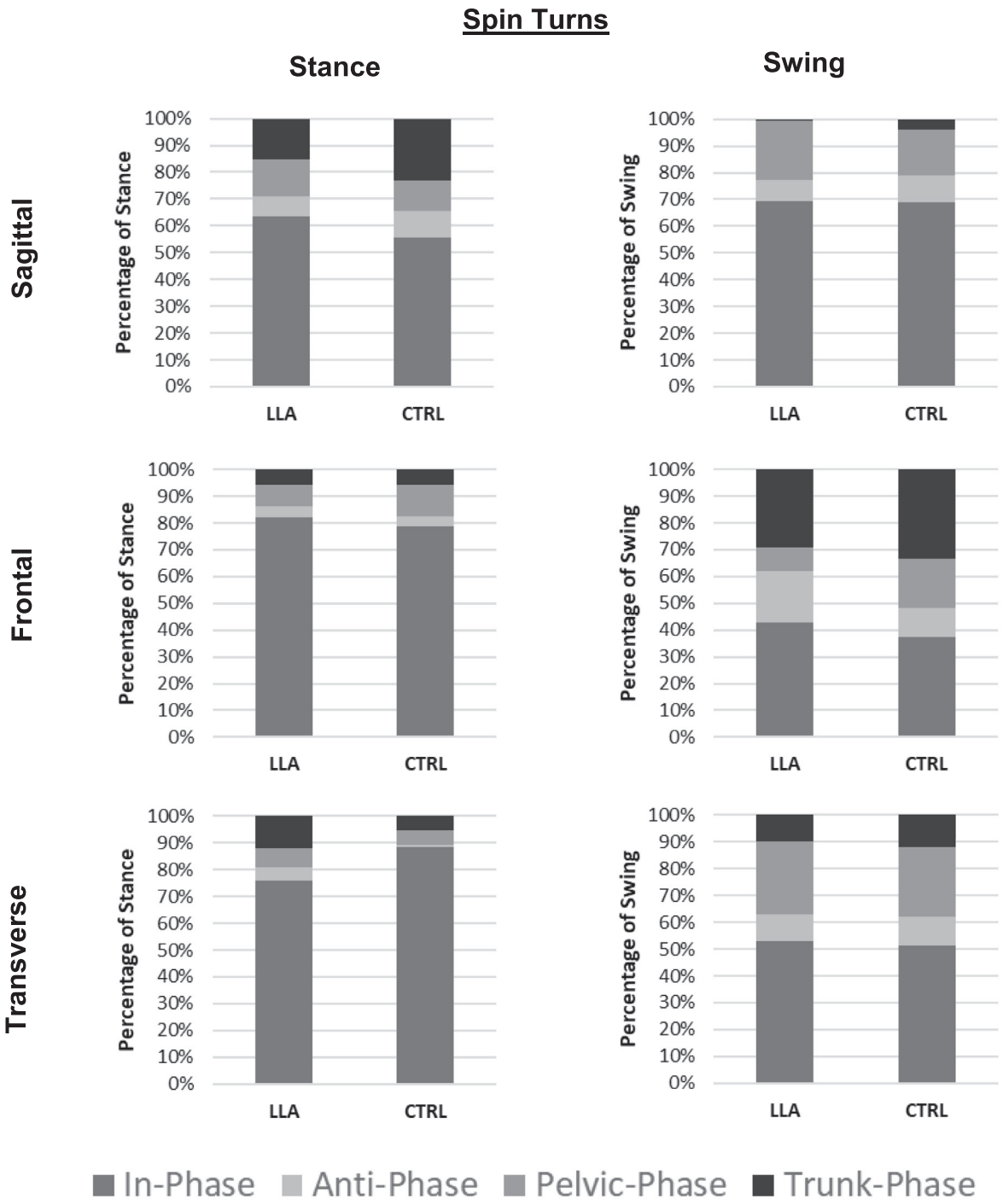

Fig. 3. Proportions of trunk-pelvis coordination modes during pivot stance and swing in each plane among persons with unilateral lower limb amputation (LLA) and uninjured controls (CTRL), by spin and step turns. Significant comparisons (*) between groups were at the $\alpha = 0.05$ level only.

4.5. Limitations

Several limitations require attention when interpreting results of the current study. First, the generalizability of findings may be limited given persons with LLA were young, healthy, and otherwise uninjured members of the military who had sustained traumatic lower limb amputations. Second, the small sample sizes, combination of individuals with different levels of amputation into the LLA group, and many inherent levels of potential comparisons precluded additional analyses between pivot legs (i.e., prosthetic and intact). The five-person control group also may not provide an accurate statistical representation of the healthy able-bodied

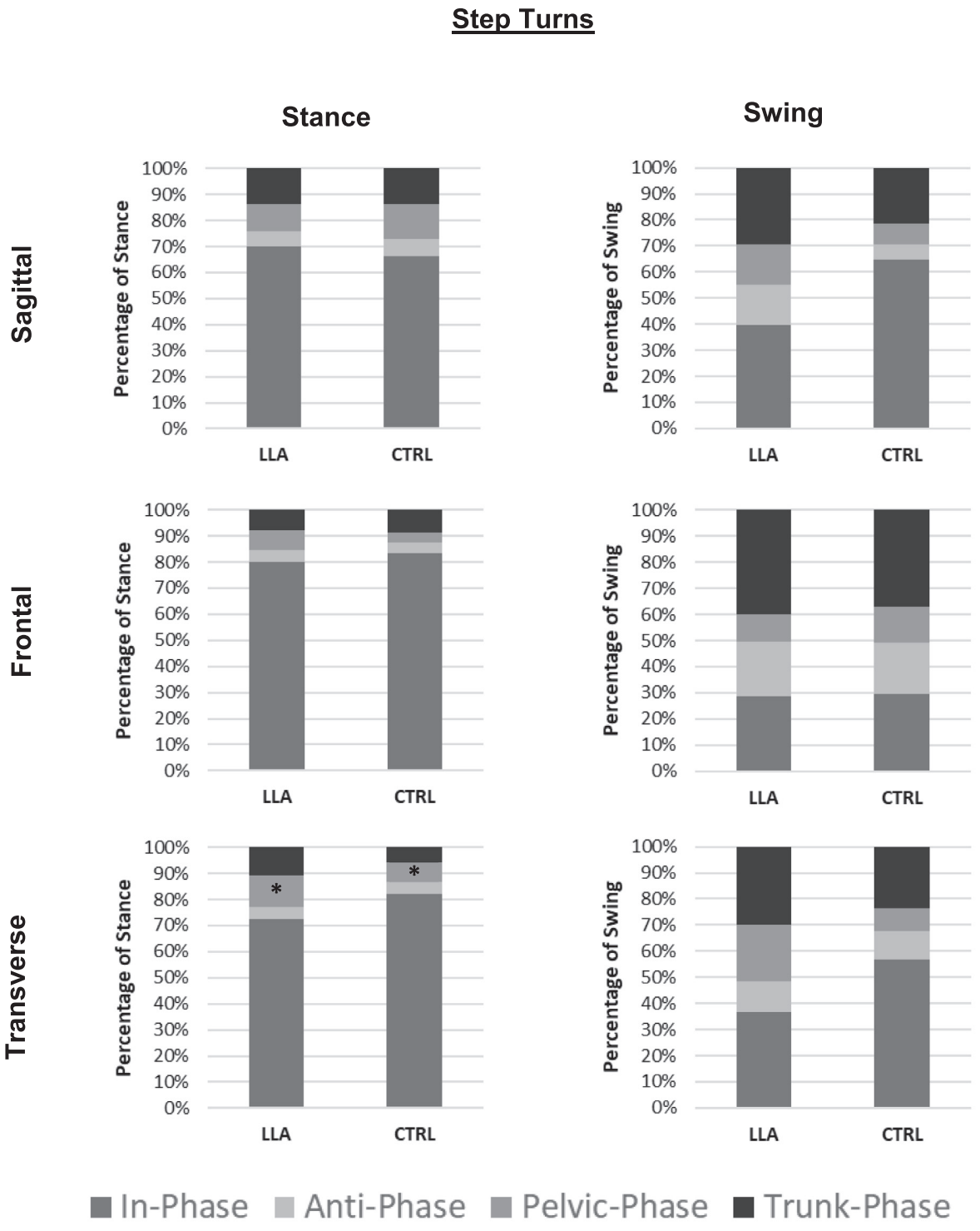

Fig. 3. (*continued*)

population at large, and future studies with larger sample sizes are warranted. Third, although we suggest that observed differences in trunk-pelvis movement patterns between persons with and without LLA may be associated with elevated risk of LBP onset or re-currence, we did not specifically control for its presence or prior/recent history, though no participants reported acute LBP during testing. Fourth, we did not specifically evaluate the influences of arm motion. While most likely to affect angular momentum in the transverse plane (Collins, Adamczyk, & Kuo, 2009; Herr & Popovic, 2008), general qualitative differences in arm swing strategies

between groups were not observed. Finally, the turn cueing paradigm used was intended to represent transient changes in direction encountered in daily life, though the somewhat unpredictable, verbally-cued direction may have resulted in events that are more difficult to reproduce than turns in other studies wherein participants walked along a more consistent circular path (Segal et al., 2008; Ventura et al., 2011). Future work can control for such variability with alternative cueing methods (e.g., visual, compared to our auditory cues; Heitzmann et al., 2015), thereby also supporting explicit comparisons between step vs. spin turns, and potential interactions with the chosen pivot limb (i.e., prosthetic vs. intact).

4.6. Summary

We compared features of trunk-pelvic segmental motion and coordination between persons with and without LLA during 90-degree turns executed using self-selected step and spin strategies. We observed differences in the frequencies of inter-segmental coordination, trunk-pelvis ranges of motion, and segmental momenta across levels of amputation, depending on the plane and method of turn employed. Nevertheless, the identified compensatory adaptations used by persons with unilateral LLA to execute this common, but biomechanically challenging, task may be “maladaptive” and thus predispose these individuals to developing LBP (or its recurrence) with repeated exposure over the longer term.

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Walking speed differentially alters spinal loads in persons with traumatic lower limb amputation

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ABSTRACT

Persons with lower limb amputation (LLA) perceive altered motions of the trunk/pelvis during activities of daily living as contributing factors for low back pain. When walking (at a singular speed), larger trunk motions among persons with vs. without LLA are associated with larger spinal loads; however, modulating walking speed is necessary in daily life and thus understanding the influences of walking speed on spinal loads in persons with LLA is of particular interest here. Three-dimensional trunk-pelvic kinematics, collected during level-ground walking at self-selected (SSW) and two controlled speeds (~1.0 and ~1.4 m/s), were obtained for seventy-eight participants: 26 with transfemoral and 26 with transtibial amputation, and 26 uninjured controls (CTR). Using a kinematics-driven, non-linear finite element model of the lower back, the resultant compressive and mediolateral/anteroposterior shear loads at the L5/S1 spinal level were estimated. Peak values were extracted and compiled. Despite walking slower at SSW speeds (~0.21 m/s), spinal loads were 8–14% larger among persons with transfemoral amputation vs. CTR. Across all participants, peak compressive, mediolateral, and anteroposterior shear loads increased with increasing walking speed. At the fastest (vs. slowest) controlled speed, these increases were respectively 24–84% and 29–77% larger among persons with LLA relative to CTR. Over time, repeated exposures to these increased spinal loads, particularly at faster walking speeds, may contribute to the elevated risk for low back pain among persons with LLA. Future work should more completely characterize relative risk in daily life between persons with vs. without LLA by analyzing additional activities and tissue-level responses.

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1. Introduction

Persons with unilateral lower limb amputation (LLA) – both above and below the knee – commonly report low back pain (Hammarlund et al., 2011; Kulkarni et al., 2005) and perceive altered trunk motions/postures during activities of daily living as primary contributors to its onset and recurrence (Devan et al., 2015). Indeed, altered trunk motion can adversely influence the mechanical environment among spinal structures and tissues within the lower back, especially when the motion occurs in multiple planes simultaneously (Davis and Marras, 2000). Such alterations in the mechanical environment of the lower back may lead to pain if the associated changes in force and/or deformation experienced in lower back tissues, instantaneously or cumulatively, exceed tolerances (Coenen et al., 2014; Kumar, 2001). The latter is of particular interest here given that many activities of daily living are highly repetitive and thus warrant consideration when assessing cumulative injury risk among persons with LLA.

Walking is a critically important activity of daily living. While not overly demanding on the lower back, walking nevertheless exposes the spine to a large number of loading cycles. For example, healthy adults with a moderately active lifestyle take approximately seven to thirteen thousand steps per day (Tudor-Locke et al., 2011). Although persons with LLA often take fewer steps (~half, though dependent on functional classification level; Halsne et al., 2013; Stepien et al., 2007), prior work has reported increases and asymmetries in trunk-pelvic motions during walking among persons with vs. without LLA (Goujon-Pillet et al., 2008; Jaegers et al., 1995). Recently, these differences were associated with larger mechanical demands on the lower back as well as lar-

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ger internal trunk muscle responses and resultant spinal loads (Hendershot and Wolf, 2014; Shojaei et al., 2016; Yoder et al., 2015). Repeated exposures to these elevated demands and loads may thus contribute to the higher prevalence and recurrence of low back pain among persons with LLA. However, these prior studies have predominantly focused on a singular (often self-selected) walking speed. Given that the amplitudes of trunk motion and acceleration increase among uninjured individuals with increasing walking speed (Kavanagh, 2009; Thorstensson et al., 1984), it is important to understand the influences of walking speed on trunk motions and spinal loads in persons with LLA.

Although the selection of an optimal walking speed is often governed by minimizing metabolic costs of transport (e.g., Ralston, 1958), the ability to increase/decrease walking speed remains important for many aspects of daily living (e.g., community ambulation and recreational activities). Modulation of walking speed can be achieved through a variety of temporal-spatial, kinematic, and kinetic mechanisms (Neptune et al., 2008), which are achieved primarily via the ankle plantarflexors during step-step transitions (Jonkers et al., 2009; Requiao et al., 2005). Although persons with LLA lack active ankle function (on the prosthetic side), these individuals can typically compensate via other joints within the lower extremity (e.g., the knee or hip; Fey et al., 2010; Silverman et al., 2008). Of particular interest here, persons with LLA also employ a seemingly active trunk movement strategy (Hendershot and Wolf, 2015) that, given its relatively large mass, may differentially alter inertial demands of walking on the lower back and surrounding musculature with changing walking speed. Among uninjured individuals, increases in trunk motion at faster walking speeds have been associated with elevated demands/loads on the low back, albeit modest (Callaghan et al., 1999; Cheng et al., 1998); however, such a relationship has not been evaluated among persons with LLA, wherein there is an increased reliance on these proximal segments. The purpose of this study was therefore to quantify and compare trunk muscle responses and resultant spinal loads among persons with and without LLA across multiple walking speeds. It was hypothesized that, with increasing walking speed, persons with vs. without LLA increase their trunk muscle forces more, hence experiencing larger increases in spinal loads; secondarily, these increases would be largest among persons with more proximal levels of LLA (i.e., transfemoral).

2. Methods

2.1. Experimental procedures

This study retrospectively evaluated biomechanical data from seventy-eight male participants (Table 1) – 26 with unilateral transtibial (TTA), 26 with unilateral transfemoral (TFA) amputation, and 26 uninjured controls (CTR) – walking overground along a 15 m level walkway at one self-selected (SSW) and two additional (controlled) speeds (~1.0 and 1.4 m/s). All persons with LLA were independently ambulatory without the use of assistive devices (e.g., canes, walkers). Additionally, all amputations were the result of traumatic injuries, and the participants reported no additional underlying musculoskeletal conditions. This retrospective study was approved by Institutional Review Boards of both the Walter Reed National Military Medical Center and University of Kentucky.

Three-dimensional kinematic data of the pelvis and thorax were collected by tracking (120 Hz) reflective markers positioned in the mid-sagittal plane over the S1, T10, and C7 spinous processes, sternal notch, and xiphoid; and bilaterally over the acromion, and the anterior/posterior superior iliac spines. All kinematic data (marker trajectories) were low-pass filtered using a fourth-order, bidirectional filter (cut-off frequency = 6 Hz). Controlled speeds were dictated using an auditory tone ("beep") that sounded when the horizontal component of the velocity of the sternal notch marker was within 5% of the intended speed. Multiple passes were performed at each speed such that ~10 complete gait cycles could be obtained.

Table 1
Mean (standard deviation) participant demographics by group: uninjured controls (CTR), persons with unilateral transtibial amputation (TTA), and persons with unilateral transfemoral amputation (TFA). The duration of time elapsed between injury and biomechanical testing is also indicated. Note, there were no significant ($P > .27$) group-level differences in these measures.

	CTR (n = 26)	TTA (n = 26)	TFA (n = 26)
Age (yr)	28.0 (4.7)	28.2 (6.6)	32.3 (8.8)
Stature (cm)	167.8 (6.6)	177.9 (6.1)	176.5 (6.5)
Body mass (kg)	85.7 (12.7)	88.7 (11.2)	84.0 (13.2)
Time (months)	N/A	13.6 (16.9)	36.0 (78.7)

2.2. Dependent measures and analyses

Kinematic data was calculated and analyzed using Visual3D (C-Motion, Germantown, MD, USA) and custom MATLAB (Mathworks, Inc., Natick, MA, USA) scripts. Global trunk and pelvis angles, as well as pelvis center of mass position, were normalized and averaged over each stride. Relative trunk-pelvic angles were similarly calculated. Trunk-pelvic ranges of motion (ROM) were calculated as the difference between the maximum and minimum relative trunk-pelvic angles in all three planes.

To estimate trunk muscle responses and resultant spinal loads, these kinematic data were used as inputs to a non-linear finite element model of the spine with an optimization-based iterative procedure (Bazrgari et al., 2007), previously validated in a variety of dynamics tasks (Bazrgari et al., 2008a, 2008b, and 2009), covering a range of trunk motions and postures. The sagitally symmetric model is composed of six rigid elements representing the thorax and each lumbar vertebrae (L1-L5) along with six non-linear flexible beam elements representing the intervertebral discs/ligaments between T12 and S1. Mass and inertial properties were distributed along the spine according to reported ratios. Fifty-six muscles were represented in the model: 46 muscles connecting the individual lumbar vertebrae to the pelvis (i.e., local) and 10 muscles connecting the thoracic spine/rib cage to the pelvis (i.e., global).

Muscle forces are estimated via a heuristic optimization of equilibrium across the lumbar spine (via changing lumbar segmental kinematics) to satisfy a cost function that minimizes the sum of squared muscle stresses across all 56 muscles. A custom MATLAB (Mathworks, Inc., Natick, MA, USA) script was used to control the optimization procedure whereas a finite element software package (ABAQUS; version 6.13, Dassault Systemes Simulia, Providence, RI, USA) was used to estimate muscle forces and associated spinal loads within the non-linear FE model.

Rather than comparing the individual forces in each of the 56 muscles, the summation forces in all local and global muscles were calculated, hereby referred to as "local" and "global" muscle force. Similarly, rather than comparing spinal loads for all lumbar levels, loads (i.e., compression, as well as anteroposterior [A-P] and mediolateral shear [M-L]) were compiled from the L5/S1 spinal level (i.e., the level that usually experiences the maximum spinal loads). From all outcomes, peak values were extracted and evaluated using a linear mixed-model analysis of variance (between factor = group; within factor = speed). Participants were considered random effects with the correlation among repeated measures assumed to follow a compound symmetry model. Statistical signif-

icance was concluded at P < .05. All data are reported as means (standard deviations).

3. Results

Controlled walking speeds were not different (P = .91) between all three groups at 0.99 (0.05) m/s and 1.42 (0.09) m/s for the "slow" and "fast" conditions, respectively. However, SSW speeds differed between groups; CTR (1.41 (0.15) m/s) and persons with TTA (1.35 (0.14) m/s) were faster (P < .001) than persons with TFA (1.24 (0.14) m/s).

Overall, trunk-pelvic ROM were larger (P < .001) among persons with TFA and TTA vs. CTR (Table 2). With increasing speed, trunk ROM among persons with TFA increased (P = .004) in the sagittal plane; increases in the frontal and transverse planes were not different (P > .27) between groups.

Peak global muscle forces tended (P = .07) to increase with increasing speed, but these were not different (P > .22) between groups at each speed. However, there was a significant (P = .035) group × speed interaction on peak local muscle forces; specifically, peak local muscle forces were larger among persons with TFA vs. TTA and CTR only at the fastest speed (Table 3/Fig. 1).

Peak A-P and M-L shear, as well as peak compression, all increased (P < .001) with increasing walking speed. There was a significant group × speed interaction on both A-P (P = .02) and M-L (P = .002) shear forces; at the fastest speed, these were larger among persons with TFA and TTA vs. CTR. Similarly, there was a significant (P = .003) group × speed interaction on peak compression; at the fastest speed, compression forces were larger among persons with TFA and TTA vs. CTR (Table 3/Fig. 1).

4. Discussion

This study assessed the influences of walking speed on trunk muscle responses and spinal loads in persons with and without LLA. As expected, both trunk muscle forces and spinal loads increased with increasing walking speed; however, these increases were generally larger among persons with LLA vs. CTR (supporting our primary hypothesis). Additionally, with the exception of lateral shear, spinal loads were larger among persons with TFA vs. TTA (partially supporting our secondary hypothesis).

Altered trunk muscle recruitment has been related to subjective (internal) factors, such as the presence of pain (Lamoth et al., 2006; van der Hulst et al., 2010), as well as changes in external demands, such as increasing walking speed (Anders et al., 2007). In activities involving trunk motion around neutral postures, such as during walking, trunk muscle forces contribute substantially to spinal loads (due to minimal passive tissue contributions; Panjabi, 2003). The amplitudes of trunk motions and accelerations increase with increasing walking speed (Kavanagh, 2009; Thorstensson et al., 1984); associated alterations in inter-planar coupling suggest the importance of efficient neuromuscular control of global trunk motions. At faster speeds, trunk motions tend to become larger/-faster and thus the demands on and resultant responses from trunk muscles generally increase as well (4.4–8.3% across speeds ranging from 0.4 to 1.5 m/s; Anders et al., 2007, Callaghan et al., 1999, van der Hulst et al., 2010). Moreover, these responses tend to differ slightly depending on the specific muscle of interest (i.e., global vs local stabilizer), whereby the local (vs. global) stabilizers are much lower in activation magnitude at slower speeds but increase more substantially at faster speeds (Anders et al., 2007). Although not different between groups, the global muscle forces reported herein tended to increase with increasing walking speed. However, increases in local muscle forces at the faster walking speeds among persons with TFA suggest a larger stabilizing response. Considering their respective anatomical and biomechanical differences, global trunk muscles (i.e., spanning the thorax and pelvis) best contribute to spine equilibrium (in response to external task demands) whereas local trunk muscles (i.e., spanning the lumbar vertebrae and pelvis) are better positioned to provide spine (segmental) stability. The similarities among global muscle forces with alterations in walking speed between person with and without LLA may be an indication of similar speed-related changes in spine equilibrium between the groups; larger increases in local muscle forces in person with LLA (TFA, specifically) at faster speeds suggest a larger stabilizing response.

The largest increases in spinal loads with increasing walking speed among persons with LLA were observed in the A-P direction. In the fastest (vs. slowest) controlled speed, A-P shear forces were respectively 77.1 (31.8), 84.8 (34.5), and 42.1 (24.3)% larger among persons with TFA, TTA, and CTR. Notwithstanding the often complex muscle responses that make direct associations between motion and spinal loads somewhat challenging, these larger increases among persons with LLA are likely due to an altered trunk flexion-extension movement pattern, particularly among persons with TFA (Table 2). This movement pattern likely assists with altering walking speed in the presence of altered lower limb anatomy and function. Such an observation is also consistent with more out-of-phase trunk-pelvic coordination in the sagittal plane as walking speed increases among persons with TFA (Russell Esposito and Wilken, 2014). Moreover, this altered movement pattern likely contributes to larger whole-body angular momentum commonly observed in persons with vs. without LLA at faster walking speeds (Silverman and Neptune, 2011). Previous work has suggested leg motion is the primary contributor to whole-body angular momentum (~60%) while trunk movement contributes little (<10%; Bruijn et al., 2008) in uninjured individuals. However, persons with LLA reduce propulsive forces from the prosthetic limb (Silverman and Neptune, 2011); they are thus unlikely to receive the same contribution to whole-body angular momentum from their legs as an uninjured individual and may have to rely on trunk motion to compensate. While this increased contribution of the trunk may help to regulate whole-body angular momentum and assist in fall prevention, the results herein suggest it may also be contributing to increased injury risk at the lower back.

Persons with LLA tend to self-select walking speeds that are slower than uninjured controls. Given the influences of walking speed on common biomechanical parameters, this presents challenges when designing a study or interpreting its results, particularly as it relates to ecological validity and clinical significance (Astephen Wilson, 2012). Our prior work specifically selected par-

Table 2
Mean (standard deviation) trunk-pelvis range of motion (ROM) by group and walking speed (SSW = self-selected walking speed, speed 1 ≈ 1.0 m/s, speed 2 ≈ 1.4 m/s). # = significant difference between controlled speed 1 and 2; * = significant difference relative to CTR (in the same speed condition).

	Trunk-Pelvis ROM (degrees)		
	Sagittal	Frontal	Transverse
CTR			
SSW (1.41 m/s)	2.9 (0.9)	11.9 (3.5)	14.4 (3.9)
Controlled Speed 1	2.7 (0.8)	8.7 (2.9)	11.9 (2.7)
Controlled Speed 2	2.8 (0.9)	12.1 (3.3)#	15.3 (4.3)#
TTA			
SSW (1.35 m/s)	4.2 (1.4)	10.7 (3.2)	15.5 (2.8)
Controlled Speed 1	3.9 (1.3)*	7.8 (2.3)	12.1 (3.0)
Controlled Speed 2	4.4 (1.4)*	11.4 (3.1)#	16.7 (4.3)#
TFA			
SSW (1.24 m/s)	8.7 (2.9)*	9.3 (2.5)	15.3 (4.4)
Controlled Speed 1	7.1 (2.6)*	7.5 (2.8)	12.9 (3.8)
Controlled Speed 2	10.0 (3.1)#*	10.1 (3.6)#	16.4 (3.7)#

Table 3
Mean (standard deviation) muscle forces and spinal loads by group and walking speed (SSW = self-selected walking speed, speed1 ≈ 1.0 m/s, speed2 ≈ 1.4 m/s). All outcomes are normalized by total body mass (N/kg). # = significant interaction effect between group × walking speed.

	Peak local muscle force	Peak global muscle force	Peak A-P shear force	Peak M-L shear force	Peak compression
CTR					
SSW (1.41 m/s)	9.3 (1.9)	11.4 (3.7)	5.1 (3.5)	8.8 (3.6)	23.6 (5.9)
Controlled Speed 1	8.2 (1.4)	8.7 (2.9)	3.4 (1.8)	5.2 (2.3)	19.2 (3.7)
Controlled Speed 2	9.7 (2.0)	11.8 (4.1)	4.9 (3.1)	8.7 (3.7)	23.5 (5.3)
TTA					
SSW (1.35 m/s)	9.2 (1.6)	11.9 (3.2)	5.0 (2.3)	8.9 (3.4)	23.7 (5.3)
Controlled Speed 1	8.2 (1.7)	9.2 (4.2)	3.1 (1.4)	6.2 (3.3)	19.9 (5.1)
Controlled Speed 2	10.3 (2.7)	13.9 (5.1)	5.7 (3.3)#	10.3 (5.2)	26.5 (7.6)#
TFA					
SSW (1.24 m/s)	10.0 (2.7)	13.2 (4.3)	5.7 (2.0)	9.5 (4.1)	25.5 (6.0)
Controlled Speed 1	8.9 (2.6)	11.2 (4.8)	3.7 (1.8)	7.9 (4.3)	22.7 (6.1)
Controlled Speed 2	12.1 (3.1)#	14.6 (4.9)	6.6 (4.0)#	9.6 (3.8)	29.1 (7.1)#

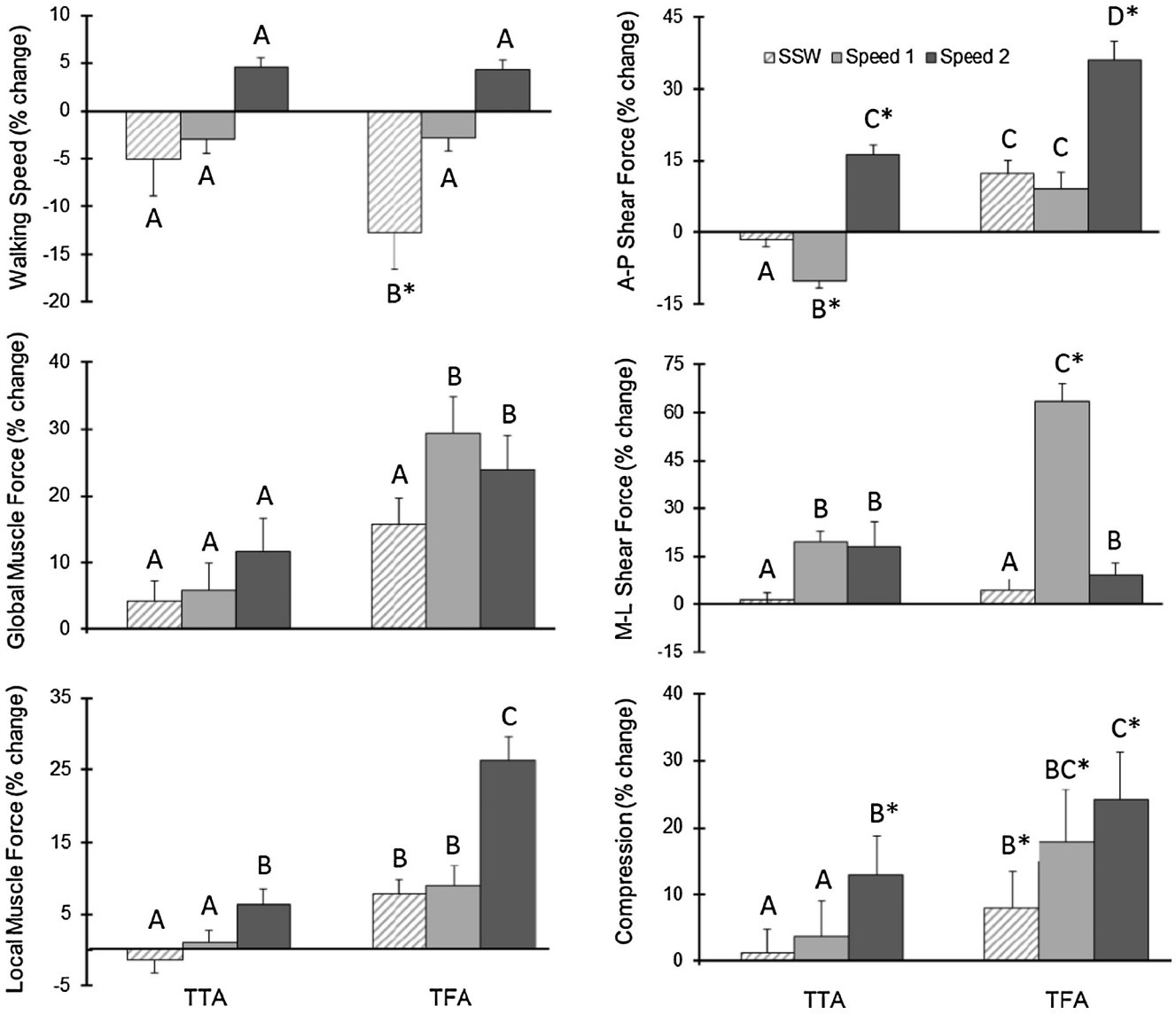

Fig. 1. Mean (standard deviation) percent change in each outcome for both the transtibial (TTA) and transfemoral (TFA) groups with respect to controls at self-selected (SSW) and controlled speeds ("Speed 1" = 1.0 m/s and "Speed 2" = 1.4 m/s). Letters indicate post hoc comparisons and asterisks indicate significant differences relative to controls.

ticipants by matching SSW speeds post hoc (within 5%; mean ≈ 1.35 m/s), and identified 39–60% larger spinal loads in persons with TFA vs. uninjured CTR (Shojaei et al., 2016). In the current study, SSW speeds among persons with TFA were 0.21 (0.14) m/s slower than uninjured CTR, suggesting smaller trunk inertial contributions to spinal loads in this group. However, larger spinal loads were observed in persons with TFA, despite slower self-selected walking speeds. This highlights the increased contribution of gravitational

demand to spinal loads among persons with TFA due to larger and more asymmetric trunk ROM. One might also presume the relative differences in the slow and fast vs. SSW speeds within each group may require more or less "effort" and thereby differentially affect the relationships reported herein; however, a sensitivity analysis revealed these differences in SSW did not influence any of the dependent measures.

Several limitations should be considered when interpreting the current results. Persons with LLA were young and generally active military personnel with injuries sustained due to trauma. Thus, the results may not be generalizable to all etiologies of amputation (e.g., older or as a result of dysvascular conditions). This study was cross-sectional and the durations of time since injury among persons with LLA were relatively short and highly variable (median = 23 months, range = 6–408 months). As such, it is possible that gait patterns may change (improve or decline) over time and the associated influences on spinal loads with changing walking speed may differ if assessed longitudinally. Moreover, the retrospective nature of this study limited the range of available walking speeds. Additional analyses at slower (i.e. <1.0 m/s) and faster (i.e. >1.4 m/s) walking speeds, or speeds more consistently spaced relative to each participant's SSW, may elucidate additional relationships among spinal loads and walking speed in persons with LLA. Although estimates of these model simulations are highly dependent on the accuracy and reliability of kinematic data, prior work suggests high intra-lab reliability and low standard errors of measurement (Kaufman et al., 2016). Additionally, trunk muscle responses and segmental kinematics were estimated using an optimization procedure assuming similar responses between persons with and without LLA; however, future work is needed with electromyography, imaging modalities, or other modeling techniques to understand these more directly. Such efforts would also support future tissue-level analyses incorporating physiological properties and biological responses (e.g., Lotz et al., 2013). Finally, we did not explicitly include contributions of arm swing in the model (Angelini et al., 2016), though participants were not instructed to alter arm swing and full body kinematics were collected and could be evaluated in subsequent analyses.

Walking is generally not a mechanically demanding activity for the lower back. For example, prior work in uninjured individuals has found peak compressive loads ranging from one to three times body weight when walking over level ground at varying speeds (Callaghan et al., 1999; Cheng et al., 1998). These magnitudes are substantially lower than during other activities (e.g., manual material handling or lifting tasks) and below injury thresholds. However, walking is a highly repetitive task with estimates of 1.5–4 million cycles per year depending activity level. Thus, we posit that increases in spinal loads among persons with vs. without LLA warrant consideration when assessing injury risk. While tasks involving high physical demands on the lower back or which have a high rate/repetition have been traditionally considered high risk for low back pain (Putz-Anderson et al., 1997), a recent review paper suggests that repetition of low-force tasks seems to result in modest increases in risk; however, surprisingly rapid increases in risk are subsequently observed under high-force tasks (Gallagher and Heberger, 2013). Although not reported here, mean values of each component of spinal load across the entire gait cycle were similarly larger among persons with vs. without LLA, and also tended to increase more with increasing walking speed among persons with LLA, suggesting not just peak loads but the overall mechanical environment is elevated throughout.

In summary, the results presented herein indicate walking speed differentially alters trunk muscle responses and spinal loads among persons with vs. without LLA. Walking faster for persons with LLA was associated with larger increases in the estimated loads among tissues within the spine, regardless of SSW speed. Over time, repeated exposure to these larger spinal loads during such a common and important activity of daily living may contribute to the elevated risk for low back pain after LLA, particularly due to fatigue failure of spinal tissues. Further work to more completely characterize spinal loads during other activities of daily living is warranted, thereby supporting future clinical recommendations for controlling risk over the longer term.

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Conflict of interest

All authors have no financial or personal relationships with other persons or organizations that might inappropriately influence our work presented therein.

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Short communication

Associations between trunk postural control in walking and unstable sitting at various levels of task demand

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ABSTRACT

Trunk postural control (TPC) has been investigated in several populations and tasks. Previous work observed targeted training of TPC via isolated trunk control tasks may improve performance in other activities (e.g., walking). However, the nature of this relationship remains unknown. We therefore investigated the relationship between TPC, at both the global (i.e., response to finite perturbations) and local (i.e., resistance to continuous perturbations) levels, during walking and unstable sitting, both at varying levels of task demand. Thirteen individuals (11 Male, 2 Female) with no recent history (past 12 months) of illness, injury, or musculoskeletal disorders walked on a dual-belt treadmill at four speeds (−20%, −10%, +10%, and + 20% of self-selected walking speed) and completed an unstable sitting task at four levels of chair instability (100, 75, 60, and 45% of an individual's "neutral" stability as defined by the gravitational gradient). Three-dimensional trunk and pelvic kinematics were collected. Tri-planar Lyapunov exponents and sample entropy characterized local TPC. Global TPC was characterized by ranges of motion and, for seated trials, metrics derived from center-of-pressure time series (i.e., path length, 95% confidence ellipse area, mean velocity, and RMS position). No strong or significant correlations ($-0.057 < \rho < 0.206$) were observed between local TPC during walking and unstable sitting tasks. However, global TPC declined in both walking and unstable sitting as task demand increased, with a moderate inter-task relationship ($0.336 < \rho < 0.544$). While the mechanisms regulating local TPC are inherently different, global TPC may be similarly regulated across both tasks, supporting future translation of improvements in TPC between tasks.

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1. Introduction

Physical pathologies including stroke (Verheyden et al., 2006), lower limb loss (Hendershot and Nussbaum, 2013), and low back pain (Lamoth et al., 2006) can adversely influence trunk postural control (TPC). While TPC has been studied extensively, reported measures vary between tasks and specific features of dynamic systems (i.e., global and local). Here, we consider global TPC as the ability of a system to respond to finite ("global") perturbations (e.g., slip or trip), while local TPC is the ability to resist infinitesimal ("local") perturbations (e.g., natural gait fluctuations). During gait, global TPC has been indirectly quantified by characterizing segmental motions, such as trunk position variability (Dingwell and Marin, 2006) and ranges of motion (ROM). Meanwhile, nonlinear measures, including Lyapunov exponents (Asgari et al., 2015, Dingwell and Marin, 2006) and sample entropy (SampEn; Lamoth et al., 2010), have characterized local TPC. During unstable sitting, global TPC is often characterized using metrics derived from center-of-pressure (CoP) time series (Hendershot and Nussbaum, 2013; Radebold et al., 2001) and ROM (Larivière et al., 2015); while local TPC has also been characterized by nonlinear analyses of CoP (Larivière et al., 2015; Van Dieën et al., 2010). In both walking and unstable sitting, TPC generally declines with increasing task demand as evidenced by larger values of TPC measures described previously (Dingwell and Marin, 2006; Radebold et al., 2001).

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Altered TPC can adversely influence performance in functional activities (e.g., walking), particularly given the relative mass and position of the trunk. Indeed, TPC deficits are associated with an increased risk of falls (Grimbergen et al., 2008, Tinetti et al., 1988) and musculoskeletal injury (Zazulak et al., 2007). Trunk-specific exercise regimens are therefore often proposed or utilized to help mitigate these risks and, in populations with impaired TPC, incorporated into rehabilitation efforts (e.g., Karthikbabu et al., 2011). Such isolated TPC tasks have been shown to reduce pain and functional disability scores in individuals with LBP (O'Sullivan et al., 1997, Carpes et al., 2008) and improve gait parameters in patients after stroke (Karthikbabu et al., 2011). These observations suggest that improvements to TPC may translate between tasks, but there remains a limited understanding of the effectiveness of such rehabilitation paradigms since the relationship between TPC mechanisms in isolated (e.g., unstable sitting) and functional (e.g., walking) activities has not been investigated thoroughly. Evidence comparing local TPC in two upright tasks (standing and walking) observed little-to-no correlation between them (Kang and Dingwell, 2006). However, only a single level of demand was investigated, and TPC during an isolated task (i.e., unstable sitting) was not determined. We thus explored the relationships between TPC during two distinct tasks, walking and unstable sitting, when both are performed at varying levels of task demand. As TPC has been observed to decrease with increasing demand in both tasks, we hypothesized that increases in respective task demands of walking and unstable sitting would be similarly reflected in decrements to TPC, as evidenced by strong inter-task correlations among TPC measures at each level of demand.

2. Methods

2.1. Study design and procedures

Thirteen participants with no current or recent history of illness, injury, or musculoskeletal disorders within the past 12 months (Table 1) completed walking and unstable sitting trials at varying demand levels. For walking trials, participants walked on an instrumented dual-belt treadmill (Bertec, Columbus, OH) at four speeds relative to self-selected walking speed (SSWS; Table 1), determined from the mean velocity of five over-ground trials across a 15 m walkway: −20%, −10%, +10%, and +20% SSWS. Relative (vs. absolute) speeds were chosen to better normalize task demand across participants, with the expectation that faster speeds increase demand (Dingwell and Marin, 2006). At each speed, a 30-s acclimation period was provided before two minutes of data collection. For seated trials, participants sat on an unstable chair (Hendershot and Nussbaum, 2013) with eyes open at four levels of instability, relative to an individual's gravitational gradient (∇G): 100, 75, 60, and 45% ∇G (with instability increasing as %∇G decreased). ∇G was calculated using previously established methods (Slota et al., 2008) and determined neutral seated stability. Participants completed four 60-s trials per condition. However, only the final (i.e., fourth) trial was used for data analyses; the prior three practice trials were used to attenuate learning effects (Van Daele et al., 2007). By the final trial, all participants successfully completed the unstable sitting task (i.e., the seat did not contact the base of support). Participants were asked to keep the chair level and arms crossed throughout trials.

An 18-camera motion capture system (Qualisys, Göteborg, Sweden) collected (120 Hz) 10 surface-marker locations to estimate three-dimensional trunk and pelvic kinematics for all tasks. Markers were placed over the T10 and C7 spinous processes, sternal notch, xiphoid, and bilaterally over the acromion, ASIS, and PSIS. During seated trials, kinetic data were collected (1200 Hz) using a force platform (AMTI, OR6-7-2000, Watertown, MA) mounted beneath the chair. Task and condition order were randomized and counterbalanced, respectively, with 60-s rests provided between trials. Prior to data collection, participants gave informed consent to protocols approved by the local Institutional Review Board.

2.2. Pre-processing

Data were analyzed using Visual3D (C-motion, Germantown, MD) and MATLAB (Mathworks, Natick, MA). Kinematic and kinetic data were low-pass filtered (Butterworth, 4th order, cut-off frequencies 6 and 10 Hz, respectively). Three-dimensional trunk angles (relative to pelvis) were determined using 6DOF inverse dynamics in Visual3D. For each walking trial, 75 strides of data were analyzed and resampled to 101 points per stride (i.e., 0–100% gait cycle). For unstable sitting trials, the first and last five seconds of data were removed to account for initial and anticipatory adjustments respectively.

2.3. Global TPC analyses

For both tasks, tri-planar trunk-pelvic ROM were determined. Though ROM does not directly quantify global TPC (i.e., response to a perturbation), increases in trunk ROM have been observed in populations with impaired TPC such as fall-prone populations (Tinetti et al., 1988, Grimbergen et al., 2008). Thus, though participants were not perturbed in the current protocol, ROM provided an indirect characterization of global TPC. For seated trials CoP path length, mean velocity, 95% confidence ellipse area (CEA), and RMS positions in the anteroposterior and mediolateral directions were also determined (Prieto et al., 1996).

2.4. Local TPC analysis

Maximum short-term Lyapunov exponents (λ_s; Rosenstein et al., 1993) and SampEn (Richman and Moorman, 2000) were used to characterize local stability of trunk-pelvic angles. λ_s quantifies the rate of convergence/divergence of initially neighboring trajectories. Negative and positive λ_s values respectively indicate convergence (i.e., stability) and divergence (i.e., instability); larger positive values represent a decreased ability to resist local perturbations (i.e., decreased local TPC). Here, tri-planar λ_s were calculated via state spaces reconstructed from trunk-pelvic angles and their time-delayed copies (Dingwell et al., 2001). Global false nearest neighbor and mutual average information analyses respectively determined embedding dimensions ($m = 6$) and time delays ($\tau = 10$ and $\tau = 100$ samples for walking and seated conditions, respectively).

Unlike λ_s, SampEn does not directly characterize the response to local perturbations. Rather, it characterizes the prevalence of local perturbations within the system by quantifying its regularity (Richman and Moorman, 2000). Larger values of SampEn indicate

Table 1
Mean (standard deviation) participant demographic information and self-selected walking speeds (SSWS).

N	Age (years)	Stature (cm)	Mass (kg)	SSWS (m/s)
13 (11 M, 2 F)	28.7 (7.2)	177.1 (6.3)	74.6 (11.4)	1.46 (0.18)

low regularity (i.e., high prevalence of local perturbations) while lower values indicate high regularity (i.e., low prevalence of local perturbations). Similar to λ_s, SampEn was determined via state-spaces reconstructed from trunk-pelvic angles. For SampEn calculations, state-spaces were reconstructed with m = 2 (Yentes et al., 2013).

2.5. Statistical analyses

Single-factor, repeated-measures ANOVAs (SPSS Inc., Chicago, IL) assessed the effect of task demand (i.e., speed or %∇G) on each outcome measure, with significance concluded when $P < 0.05$. Linear correlation analyses related local and global TPC measures between tasks (e.g., m $\lambda_{s,\ walking}$ vs. $\lambda_{s,\ seated}$) using Spearman's rho (ρ) as data were not normally distributed. Correlation strength was assessed qualitatively (Portney and Watkins, 2009): 0–0.25 (little or no relationship), 0.25–0.50 (weak-moderate), 0.50–0.75 (moderate-strong), and >0.75 (strong-excellent).

3. Results

3.1. Walking

λ_s increased with increasing walking speed in all planes (Table 2). SampEn increased with speed in the sagittal and transverse planes. Although only approaching significance, SampEn also increased in the frontal plane. Sagittal and frontal plane trunk-pelvic ROM were similar between speeds, but transverse plane ROM increased with walking speed.

3.2. Unstable sitting

All CoP-based metrics were inversely related with %∇G. In all planes, λ_s remained similar across %∇G levels. While not statistically significant, SampEn tended to decrease with %∇G in the transverse plane. Decreasing %∇G led to increased sagittal and frontal plane ROM (Table 2).

3.3. Correlation analyses

No strong or significant inter-task correlations were observed in local TPC measures (i.e., SampEn and λ_s). However, measures of global TPC were weakly-to-moderately correlated (Fig. 1). Transverse plane ROM while walking was correlated with sagittal ($\rho = 0.424$, $P = 0.002$) and frontal plane ($\rho = 0.433$, $P = 0.001$) ROM, CEA ($\rho = 0.527$, $P < 0.001$), and both anteroposterior ($\rho = 0.470$, $P < 0.001$) and mediolateral ($\rho = 0.544$, $P < 0.001$) RMS positions while seated. Frontal plane ROM while walking was correlated with frontal plane ROM ($\rho = 0.345$, $P = 0.012$), CEA ($\rho = 0.336$, $P = 0.015$) and mediolateral RMS position ($\rho = 0.417$, $P = 0.002$) while seated. Although sagittal plane ROM while walking was not correlated with seated ROM in any plane, it was weakly correlated with mediolateral RMS position ($\rho = 0.382$, $P = 0.005$) while seated.

4. Discussion

Increases in λ_s, SampEn, and transverse plane trunk ROM with increased walking speed are consistent with previous work (Asgari et al., 2015, Dingwell and Marin, 2006, Lamoth et al., 2010, Van Emmerik et al., 2005), and suggest both local and global TPC declines with increasing task demand. Specifically, the increases in λ_s and SampEn suggest that as walking speed increased, participants became less able to resist local perturbations while simultaneously experiencing more of these perturbations. During unstable sitting trials, the increases in CoP-based measures with decreased chair stability are also consistent with prior reports (e.g., Radebold et al., 2001) and suggest that global TPC declines with increasing task demand during unstable sitting.

Table 2
Mean (standard deviation) ranges of motion (ROM), maximum short-term Lyapunov exponents (λ_s), sample entropy (SampEn), and CoP-based metrics for walking and unstable sitting conditions (SSWS = self-selected walking speed; ∇G = gravitational gradient, AP = anteroposterior, ML = mediolateral, VT = vertical). Asterisks (*) indicate a significant effect of task demand ($P < 0.05$).

Walking							
	−20% SSW	−10% SSW	+10% SSW	+20% SSW	$F_{(3,48)}$	P	η^2
ROM AP (degrees)	10.6 (4.5)	10.23 (3.5)	10.7 (3.1)	11.0 (3.4)	0.174	0.914	0.011
ROM ML (degrees)	16.3 (4.3)	16.82 (4.5)	18.8 (4.8)	18.7 (4.2)	1.448	0.241	0.083
ROM VT (degrees)	16.4 (4.5)	17.63 (5.8)	20.3 (5.4)	22.5 (7.9)	5.057	0.004*	0.240
λ_s AP	1.27 (0.09)	1.31 (0.10)	1.37 (0.15)	1.44 (0.09)	5.333	0.003*	0.250
λs ML	1.04 (0.11)	1.10 (0.13)	1.18 (0.16)	1.28 (0.20)	6.116	0.001*	0.278
λs VT	1.17 (0.15)	1.27 (0.15)	1.30 (0.12)	1.38 (0.14)	4.880	0.005*	0.234
SampEn AP	0.27 (0.06)	0.28 (0.06)	0.33 (0.08)	0.35 (0.08)	4.401	0.008*	0.216
SampEn ML	0.22 (0.04)	0.23 (0.04)	0.24 (0.04)	0.26 (0.04)	2.708	0.056	0.145
SampEn VT	0.17 (0.03)	0.18 (0.04)	0.21 (0.04)	0.23 (0.04)	7.349	<0.001*	0.315
Unstable Sitting							
	100% ∇G	75% ∇G	60% ∇G	45% ∇G	$F_{(3,48)}$	P	η^2
ROM AP (degrees)	3.8 (2.7)	5.5 (3.20)	5.6 (2.6)	8.4 (3.2)	5.127	0.004*	0.243
ROM ML (degrees)	1.8 (1.1)	2.0 (0.7)	2.4 (1.2)	4.5 (1.0)	19.457	<0.001*	0.549
ROM VT (degrees)	2.8 (1.9)	2.5 (0.6)	2.6 (1.0)	3.3 (1.3)	0.993	0.404	0.058
λ_s AP	0.12 (0.06)	0.10 (0.04)	0.10 (0.02)	0.09 (0.02)	1.235	0.307	0.072
λs ML	0.11 (0.04)	0.09 (0.04)	0.11 (0.04)	0.10 (0.03)	0.657	0.583	0.039
λs VT	0.13 (0.03)	0.12 (0.04)	0.10 (0.04)	0.11 (0.02)	1.987	0.128	0.110
SampEn AP	0.05 (0.03)	0.05 (0.02)	0.06 (0.02)	0.06 (0.04)	0.656	0.583	0.039
SampEn ML	0.04 (0.02)	0.04 (0.02)	0.05 (0.02)	0.04 (0.02)	0.783	0.784	0.220
SampEn VT	0.04 (0.02)	0.07 (0.02)	0.06 (0.04)	0.04 (0.02)	2.276	0.092	0.124
Path Length (cm)	43.66 (12.27)	45.49 (11.13)	61.90 (19.72)	84.74 (17.79)	15.498	<0.001*	0.569
Mean Velocity (cm/s)	0.84 (0.48)	1.21 (0.59)	2.84 (1.95)	5.26 (2.77)	9.051	<0.001*	0.492
95 %CEA (cm^2)	0.84 (0.26)	0.87 (0.19)	1.17 (0.35)	1.62 (0.34)	18.221	<0.001*	0.361
RMS AP (cm)	0.26 (0.07)	0.34 (0.12)	0.47 (0.15)	0.61 (0.14)	18.221	<0.001*	0.532
RMS ML (cm)	0.18 (0.09)	0.20 (0.06)	0.31 (0.11)	0.46 (0.15)	21.614	<0.001*	0.575

η^2: small = 0.01, medium = 0.06, large = 0.14 (Cohen 1988).

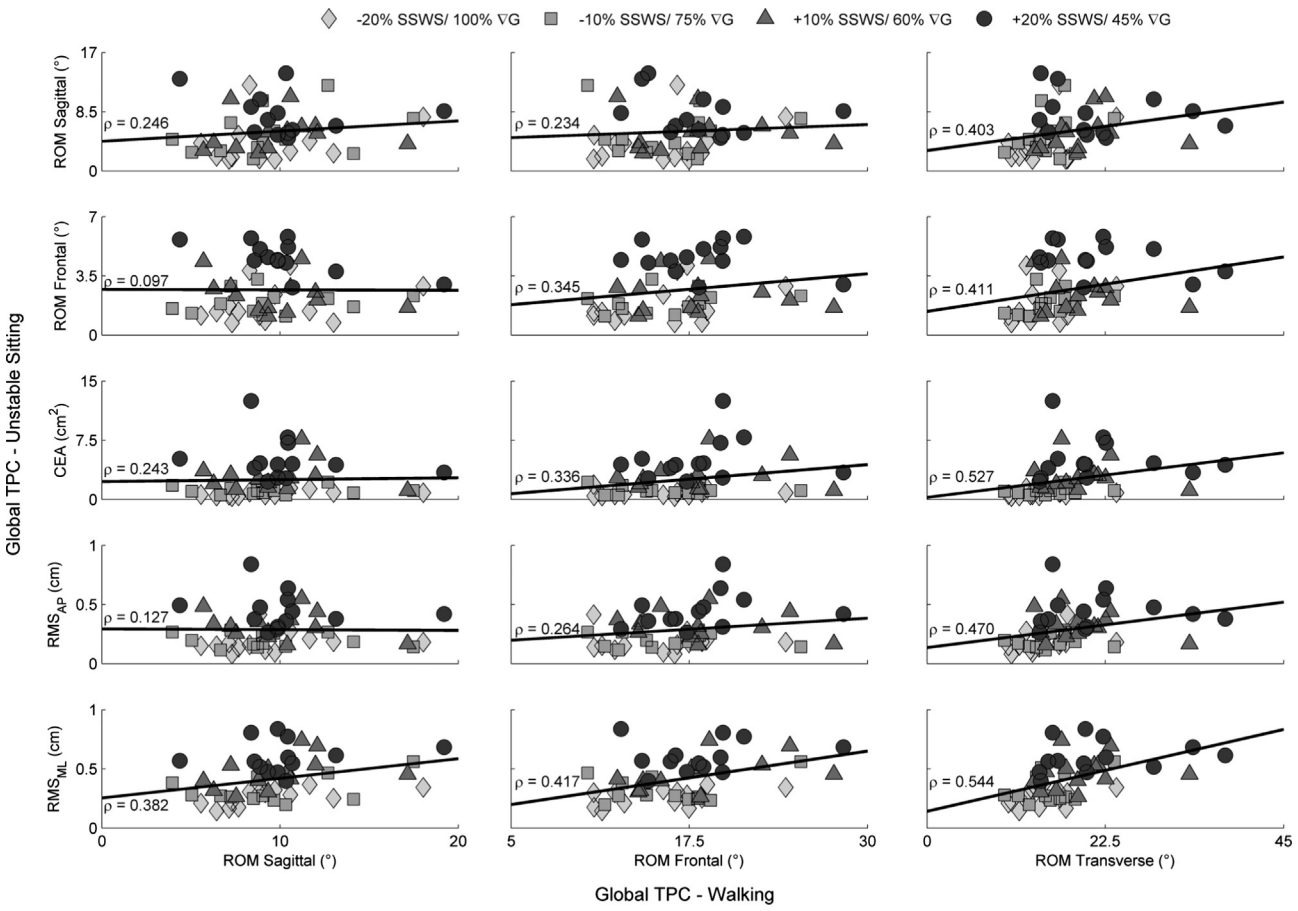

Fig. 1. Trunk-pelvic ranges of motion (ROM), 95% confidence ellipse area (CEA), and RMS positions for unstable sitting plotted against trunk-pelvic ROM while walking. Linear fits and corresponding correlation coefficients (ρ) are displayed. (SSWS = self-selected walking speed; ∇G = gravitational gradient, AP = anteroposterior, ML = mediolateral).

However, no significant differences were observed in non-linear metrics between levels of instability in seated conditions suggesting local TPC was not affected by increases in task demand. Moreover, and contrary to our hypothesis, no strong correlations were observed between non-linear TPC measures of walking and unstable sitting, suggesting that local TPC mechanisms differ between seated and walking tasks. This is likely due to the relatively static nature of sitting (vs. walking), evidenced by smaller ROM. Furthermore, while the unstable sitting task required dynamic movements to correct for global perturbations, local perturbations and fluctuations of movement were less prominent given the ultimate goal to remain "still", likely leading to increased local TPC (i.e., smaller λ_s and SampEn) regardless of demand (Table 2). Prior work observed similar results when comparing local stability in static and dynamic tasks (Kang and Dingwell, 2006).

Notably, non-linear metrics exhibited higher variance in seated versus walking tasks. Coefficients of variation for these metrics while walking were 6–24%, and in sitting were 23–64%; high inter-subject variability in the latter was perhaps due to task novelty. Participants may thus have adopted different strategies while adapting to the unstable sitting task, possibly contributing to poor inter-task correlations. Additionally, treadmill (vs. overground) walking can artificially reduce λ_s (Dingwell et al., 2001). Changes in gait parameters also persist for five minutes while acclimating to a dual-belt treadmill (Zeni and Higginson, 2010). Our relatively short acclimation period may therefore have influenced trunk kinematics, though all trials were performed under the same conditions and no order effects were observed ($P > 0.301$).

While transverse plane ROM during unstable sitting remained similar across task demands, this may be a result of the unstable chair design. The springs mounted beneath the chair, while allowing for the control of instability level, also limit rotations about the vertical axis. Future work could therefore consider using an apparatus that allows for tri-axial rotations (Van Daele et al., 2009). Additionally, although moderate inter-task correlations were observed, future work could also investigate more "extreme" levels (or spacing) of task demand to further assess this relationship.

Despite little evidence relating local TPC in walking and unstable sitting, recent work suggests that a relationship between global TPC mechanisms exists between tasks. Persons with LBP reported decreased pain and functional disability scores after targeted TPC training (Carpes et al., 2008, O'Sullivan et al., 1997) with changes persisting in a 30-week follow-up (O'Sullivan et al., 1997). Trunk-specific training has improved gait parameters (e.g., gait speed, symmetry, etc.) and functional outcomes in patients post-stroke (Karthikbabu et al., 2011), with more pronounced improvements when trunk-specific exercises were performed on an unstable (versus stable) surface (Karthikbabu et al., 2011, Jung et al., 2016). These results, along with the positive correlations among global TPC measures in the present study, establish a tentative relationship by which improvements in TPC via unstable sitting may translate to other functional activities, though it is presently unclear if this relationship persists among individuals with impaired TPC.

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Conflict of Interest

The authors have no financial or personal relationships with other persons or organizations that might inappropriately influence our work presented herein.

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Trunk muscle activation patterns during walking among persons with lower limb loss: Influences of walking speed

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ABSTRACT

Persons with lower limb amputation (LLA) walk with altered trunk-pelvic motions. The underlying trunk muscle activation patterns associated with these motions may provide insight into neuromuscular control strategies post LLA and the increased incidence of low back pain (LBP). Eight males with unilateral LLA and ten able-bodied controls (CTR) walked over ground at 1.0 m/s, 1.3 m/s, 1.6 m/s, and self-selected speeds. Trunk muscle onsets/offsets were determined from electromyographic activity of bilateral thoracic (TES) and lumbar (LES) erector spinae. Trunk-pelvic kinematics were simultaneously recorded. There were no differences in TES onset times between groups; however, LLA demonstrated a second TES onset during mid-to-terminal swing (not seen in CTR), and activation for a larger percentage of the gait cycle. LLA (vs. CTR) demonstrated an earlier onset of LES and activation for a larger percentage of the gait cycle at most speeds. LLA walked with increased frontal plane trunk ROM, and a more in-phase inter-segmental coordination at all speeds. These data collectively suggest that trunk neuromuscular control strategies secondary to LLA are driven by functional needs to generate torque proximally to advance the affected limb during gait, though this strategy may have unintended deleterious consequences such as increasing LBP risk over time.

1. Introduction

Low back pain (LBP) is a common deleterious health condition secondary to lower limb amputation (LLA), with prevalence rates as high as 52–89% (Ehde et al., 2001; Ephraim et al., 2005; Kušljugić et al., 2006). Frequent incidences of LBP are linked to severe physical disability and performance limitations of activities of daily living (Ehde et al., 2001; Kulkarni et al., 2005). The etiology of LBP is typically multifactorial, with physical (i.e., biomechanical), psychological (i.e., anxiety) and social (i.e., support structure) risk factors considered in the holistic approach to understanding the disorder in both able-bodied and individuals with LLA (Farrokhi et al., 2017). Biomechanical factors, specifically, such as altered trunk-pelvic motion and coordination during repetitive/cyclical tasks (i.e., walking), are commonly posited to play a predominant role in LBP risk among persons with LLA (Devan et al., 2014; Esposito and Wilken, 2014; Hendershot and Wolf, 2014).

To assist balance and forward progression during walking, particularly as speed increases, persons with LLA laterally flex the trunk toward the prosthetic limb during ipsilateral stance and minimize relative motion between the trunk and pelvis in the axial plane (Esposito and Wilken, 2014; Goujon-Pillet et al., 2008; Hendershot and Wolf, 2014; Jaegers et al., 1995). Able-bodied individuals demonstrate increases in trunk motion and muscle activity as walking speed increases (Anders et al., 2007; Callaghan et al., 1999; Saunders et al., 2005). Similarly, increased trunk motion as a function of walking speed is also observed in persons with LLA (Jaegers et al., 1995). As speed increases, axial trunk-pelvic coordination evolves from a synchronous in-phase pattern (i.e., rotations in the same direction) to a more asynchronous anti-phase pattern (i.e., rotation in opposite directions) (Lamoth et al., 2006a). This is comparable between persons with LLA (with and without LBP) and able-bodied individuals; however, persons with LLA demonstrate a more anti-phase coordination pattern in the sagittal plane and a more in-phase coordination pattern in the frontal plane (Esposito and Wilken, 2014). The frontal plane (in-phase) coordination pattern is suggested as a protective "guarding" of the trunk (Lamoth et al., 2002); a compensatory mechanism to increase stability (Esposito and Wilken, 2014). However, the trunk muscle activation patterns driving kinematic outcomes remain unknown.

Coordinated trunk muscle responses maintain equilibrium, maximize energy efficiency, and govern unexpected disturbances

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characteristic of normal environmental conditions (i.e., sudden changes in walking speed) (Lamoth et al., 2006b). For example, the thoracic erector spinae (contralateral to the stance limb) concentrically contract prior to lumbar thoracic spinae, thereby inverting the curvature of the spine toward the swing limb and moving the upper trunk. The subsequent contraction of the lumbar erector spinae then eccentrically controls the trunk while aiding pelvis and swing leg elevation, with the upper aspect of the trunk as the inertial reference (Anderson et al., 2003; Ceccato et al., 2009; Shiavi, 1990). Impaired trunk neuromuscular control may manifest as altered timing and activation of trunk musculature, which is associated with LBP (Arendt-Nielsen et al., 2010; van der Hulst et al., 2010; Vogt et al., 2003). For instance, during the swing phase of gait able-bodied individuals with vs. without LBP demonstrate increased lumbar and thoracic erector spinae activation, earlier onsets of lumbar erector spinae (LES), and increased co-contraction of trunk flexors and extensors (Arendt-Nielsen et al., 2010; Lamoth et al., 2006a; van der Hulst et al., 2010; Vogt et al., 2003). This increased and prolonged activation during swing, when paraspinal muscles are typically silent, suggests a protective mechanism to increase spinal stability. Moreover, increased activation of LES during swing is noted in LBP patients (vs. controls) when walking at faster speeds, suggesting an attempt to increase stiffness and thus spinal stability during speed-dependent perturbations (Lamoth et al., 2006b). While aberrant activation timing and magnitude of trunk musculature during gait are characteristic of persons with LBP, it is unknown whether persons with LLA exhibit similar changes.

Therefore, the first objective of this study was to determine trunk muscle activation patterns and corresponding trunk-pelvic segmental coordination in persons with LLA. We hypothesized that persons with LLA would demonstrate similar muscle activation and segmental coordination patterns to able-bodied individuals with LBP (e.g., earlier, prolonged activation and more in-phase segmental coordination) (Arendt-Nielsen et al., 2010; Lamoth et al., 2006a; van der Hulst et al., 2010; Vogt et al., 2003). The second objective was to determine how these patterns modulate with walking speed, hypothesizing that persons with LLA would demonstrate increased axial and frontal plane segmental motion with corresponding muscular activation patterns as speeds increase.

2. Methods

2.1. Participants

Eight males with unilateral LLA (three transfemoral, five transtibial) and ten able-bodied controls (CTR) participated in this study (Table 1). LLA participants wore energy storage and return feet, microprocessor knees (as relevant), and their prosthesis for 15 h per day (on average via self-report). LLA participants were at least one-year post traumatic injury and could ambulate without the use of an assistive device. LLA participants were excluded if they presented with any of the following: pre-existing spinal pathology or chronic LBP prior to traumatic amputation or injury, co-existing spinal trauma which occurred at the time of the traumatic amputation or injury, diagnosed neurologic deficit(s), including traumatic brain injury, any underlying musculoskeletal disorders (not including amputation) resulting in functional impairment, upper-extremity amputations above the wrist, and/or pain or discomfort, regardless of cause ($>$ 3/10 on a VAS for pain), with 100% weight bearing in socket or which interferes with performance of functional activities. All participants gave written informed consent to procedures approved by the local Institutional Review Board.

Table 1
Mean (standard deviation) participant demographics. * Indicates significant difference ($p < 0.05$) between groups. Abbreviations: ODI: Oswestry Disability Index; CTR: able-bodied controls; LLA: persons with lower limb amputation.

Group	Age (yrs)	Stature (cm)	Mass (kg)	ODI	Time since injury (months)
CTR	29.1 (7.8)*	176.9 (7.0)	74.8 (14.9)	2.4 (4.9)	NA
LLA	37.9 (8.6)	177.9 (8.4)	88.2 (9.3)*	7.3 (11.9)	95.6 (51.4)

2.2. Experimental procedures

Participants walked along a 15 m walkway at four speeds (5 trials in each): 1.0, 1.3, 1.6 m/s, and self-selected walking (SSW) speeds. Non-SSW speeds were enforced within 5% of desired speed via auditory feedback using a custom LabVIEW VI (National Instruments, Austin, TX). Full-body kinematics were recorded by tracking (120 Hz) the locations of 51 surface-markers using an 18-camera motion capture system (Qualisys, Göteborg, Sweden). Electromyographic (EMG) activities of the erector spinae were simultaneously recorded (1200 Hz, Motion Lab Systems, Baton Rouge, LA), pre-amplified per channel with a 500 Hz anti-alias low pass filter, using rectangular bipolar Ag/AgCl surface electrodes. Electrodes were placed bilaterally at the thoracic longissimus (TES, 4 cm lateral to T9) and lumbar iliocostalis (LES, 6 cm lateral to L2) (Willigenburg et al., 2013), with reference electrode placed on the ulnar head. Prior to electrode application, skin was shaved, abraded, and cleaned with alcohol.

2.3. Data analysis

All data were analyzed using Visual3D (C-motion, Germantown, MD) and MATLAB (Mathworks, Natick, MA). Kinematic data were low-pass filtered (Butterworth, 6 Hz). Gait events were determined using previously published methods (Zeni et al., 2008). Three-dimensional trunk and pelvis angles, corresponding ranges of motion (ROM; relative to lab), and angular velocities were calculated. Tri-planar continuous relative phase (CRP) was calculated from angles and angular velocities during each stride (right heel strike to right heel strike) (Hamill et al., 1999). EMG data were normalized to the respective pre-amplification gains, high-pass filtered (Butterworth, cut-off frequency 20 Hz), and full-wave rectified. A root mean square envelope was then calculated using a 50 ms smoothing window (Anders et al., 2007). EMG signals were resampled to 1201 samples per stride and averaged across all strides and participants within each group. For all analyses, the right limb of CTR was used for comparison against the intact and affected limbs of LLA, as there were no differences ($p > 0.05$) between limbs among CTR.

EMG onsets and offsets were determined by visual inspection (Hodges and Bui, 1996a; Saunders et al., 2005); EMG onset was defined as the first upward deviation in EMG amplitude above baseline levels of activity; EMG offset was determined when the level of EMG activity returned to baseline and remained there for $>$ 5% of the gait cycle (Hodges and Richardson et al., 1999; Saunders et al., 2005). Four reviewers independently analyzed each EMG signal and identified all perceived onsets and offsets of muscle activity within each time series. All occurrences of onset/offset were determined using the same criteria, and named sequentially (i.e., first/second) within the software once identified by each rater. A total of 32 EMG signals (four muscles $\times$ four speeds $\times$ two groups) were analyzed in a random order. Reviewers completed this analysis twice with at least 24 h between analyses and were blinded to analysis results to reduce rater bias (Portney and Watkins et al., 2009; Tenan et al., 2017). Intraclass Correlation Coefficients ($ICC_{3,1}$) were used to determine intra-rater reliability between analyses with values ranging from $ICC_{3,1}$ = 0.86–0.98. Analyses with ICC values greater than 0.75 are considered to have "good" reliability (Portney and Watkins, 2009); thus all data used met this criteria and were consistent with prior work (Tenan et al., 2017). The mean of the eight visual detections (two per reviewer) was used to evaluate onset and offset (Hodges and Bui, 1996a; Solnik et al., 2010; Tenan et al.,

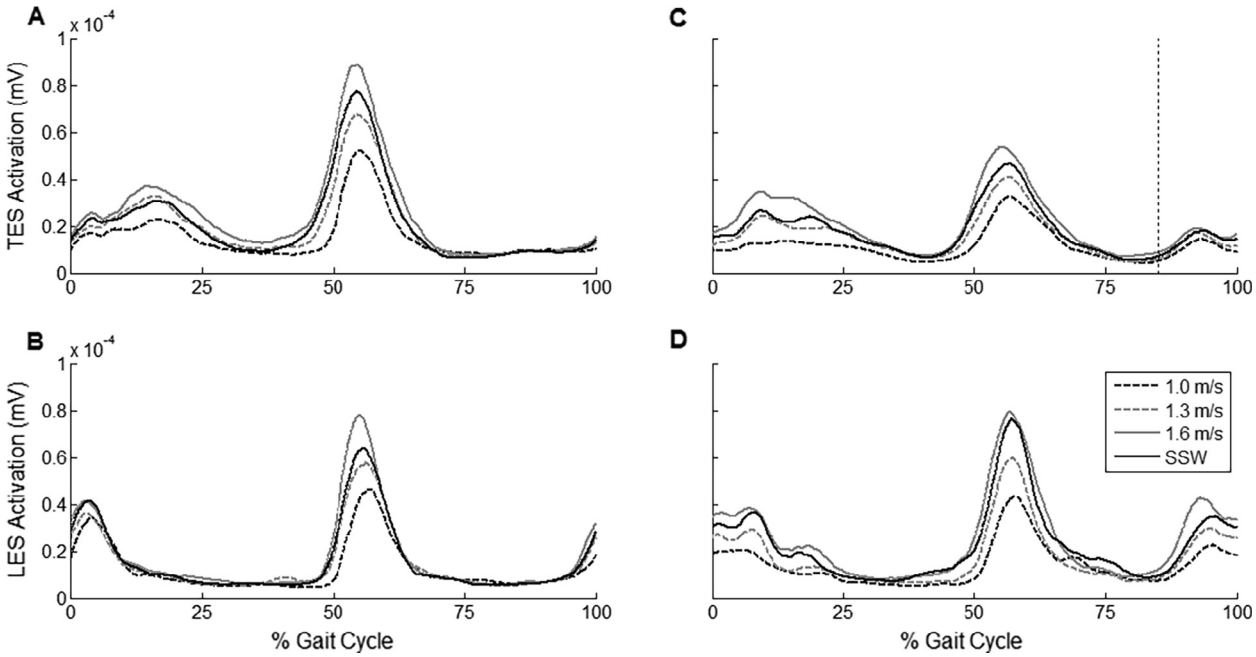

Fig. 1. (A) CTR thoracic erector spinae (TES), (B) CTR lumbar erector spinae (LES), (C) LLA TES, (D) LLA LES at 1.0 m/s, 1.3 m/s, 1.6 m/s, and SSW conditions. Displayed are raw EMG signals of group ensemble means. Vertical dashed line indicates second onset detected in LLA TES that was not demonstrated in CTR TES.

2017). Duration of muscle activation is reported as a percentage of the gait cycle between onset and offset.

Two-factor ANOVAs assessed the effects of group and speed on all outcome measures, with significance set at $p < 0.05$. When a main effect of group was observed, *t*-tests assessed differences between groups at each speed. Significance was adjusted to account for multiple comparisons ($p < 0.0125$). When a main effect of speed was observed, single-factor ANOVAs were used to assess the effect of speed within each group. If the main effect persisted, *t*-tests ($p < 0.008$) assessed speed-related differences within a group, with significance adjusted to account for multiple comparisons.

3. Results

3.1. Trunk muscle activation and speed dependency

There were no differences in the first onset of TES between LLA vs. CTR (46% vs. 45% gait cycle) during intact stance at any speed; however, LLA demonstrated a second onset during mid-terminal swing that was not observed in CTR (Fig. 1). A main effect of group was observed in TES activation (as a percentage of the gait cycle) in both intact and affected comparisons. During intact stance, LLA activated TES for a larger percentage of the gait cycle, compared to CTR ($F_{(1,56)} = 103.34$, $p < 0.0001$, eta = 0.81), with pairwise differences ($p < 0.0125$) at all speeds (Table 2). During affected stance, there was a main effect of group for initial TES onset ($F_{(1,56)} = 20.27$, $p < 0.0001$, eta = 0.32); however, LLA onsets were only earlier than CTR in the 1.3 m/s condition ($t = 6.00$, $p < 0.0001$, $d = 3.00$) (Fig. 2). In affected stance, LLA demonstrated TES activation for a larger percentage of the gait cycle ($F_{(1,56)} = 11.98$, $p = 0.001$, eta = 0.47) (Table 3). A main effect of group ($F_{(1,56} = 34.83$, $p < .0001$, eta = 0.68) was observed during intact stance, with LLA delaying ($p < 0.0125$) the first TES offset during mid stance in all conditions (except SSW). Similar results were noted during affected stance ($F_{(1,56)} = 30.73$, $p < .0001$, eta = 0.66), with LLA exhibiting earlier ($p < 0.0125$) offsets at 1.0 m/s and 1.6 m/s compared to CTR during early stance. However, there were no group differences in the second offset of TES in this comparison.

There was an interaction ($F_{(1,56)} = 4.66$, $p = 0.006$, eta = 0.45) for the second LES onset during intact stance; simple effects contrast revealed LLA demonstrated an earlier second onset of LES at all speeds. LES was active for a larger percentage of the gait cycle ($F_{(1,56)} = 69.54$, $p < 0.0001$, eta = 0.74) in LLA during intact stance (Table 2). Relative to affected stance, a main effect of group was observed for the first onset of LES ($F_{(1,56)} = 130.29$, $p < 0.0001$, eta = 0.84), with LLA demonstrating an earlier onset of LES compared to CTR at all speeds ($p < 0.0125$). LLA maintained LES activation for a larger percentage of the gait cycle compared to CTR in the affected comparison ($F_{(1,56)} = 38.40$, $p < 0.0001$, eta = 0.64) (Table 3). A main effect of group ($F_{(1,56)} = 20.20$, $p < 0.0001$, eta = 0.52) was noted in the first offset of LES in the intact comparison, with main effects of speed ($F_{(1,56)} = 7.15$, $p < 0.0001$, eta = 0.52) and group ($F_{(1,56)} = 36.45$, $p < 0.0001$, eta = 0.63) for the second offset. Pairwise within group differences ($p < 0.008$) were observed between 1.0 m/s and SSW conditions in CTR and 1.0 m/s vs. 1.6 m/s and 1.0 m/s vs. SSW conditions in LLA. Second offset group differences ($p < 0.0125$) were noted in 1.0 m/s and SSW conditions (Fig. 2). There were main effects of

Table 2
Mean (standard deviation) activation as a percentage of intact/right gait cycle. Abbreviations: CTR: controls; LLA: persons with lower limb amputation; TES: thoracic erector spinae; LES: lumbar erector spinae.

Group	Muscle	Speed	Activation (% Gait)	P-value
CTR	TES	1.0	40.8 ± 15.9	0.0001
LLA		1.0	87.0 ± 20.7	
CTR		1.3	48.2 ± 14.8	0.0007
LLA		1.3	86.3 ± 20.2	
CTR		1.6	48.5 ± 18.2	0.0006
LLA		1.6	79.3 ± 7.3	
CTR		SSW	35.5 ± 12.0	< 0.0001
LLA		SSW	79.4 ± 11.1	
CTR	LES	1.0	39.8 ± 4.5	< 0.0001
LLA		1.0	67.5 ± 4.7	
CTR		1.3	44.4 ± 10.1	0.013
LLA		1.3	64.3 ± 17.1	
CTR		1.6	44.8 ± 12.9	0.002
LLA		1.6	65.1 ± 9.1	
CTR		SSW	41.4 ± 8.0	0.001
LLA		SSW	60.1 ± 10.7	

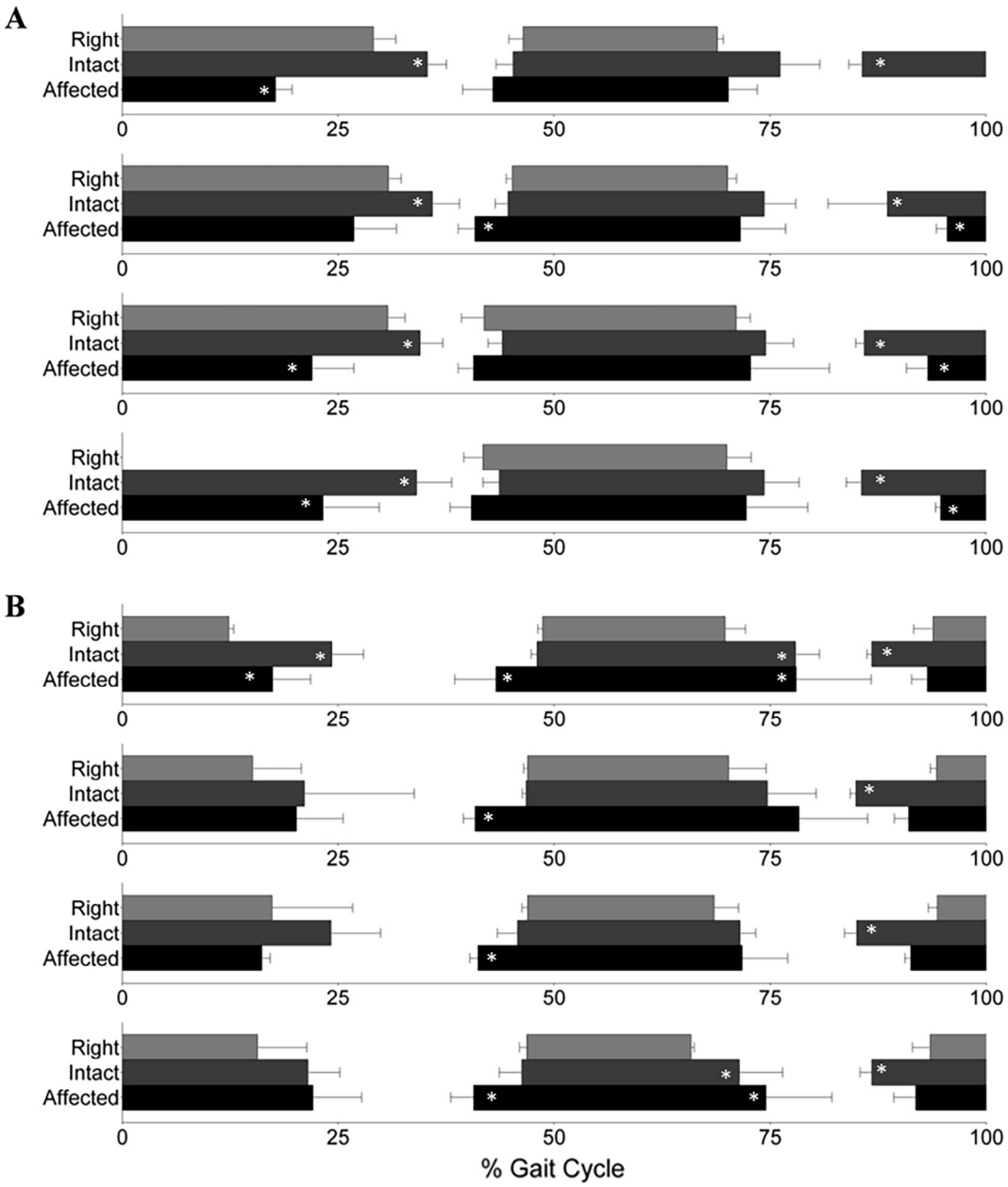

Fig. 2. (A) Thoracic erector spinae activation and (B) lumbar erector spinae activation in controls (right stance) and limb loss group (intact and affected stance). Asterisks (*) indicate significant differences ($p < 0.0125$) between LLA intact vs. CTR and LLA affected vs. CTR.

group for both the first ($F_{(1,56)} = 8.25$, $p = 0.006$, eta = 0.36) and second ($F_{(1,56)} = 24.88$, $p < 0.0001$, eta = 0.56) offset of LES during affected stance, with pairwise differences in the 1.0 m/s condition for the first offset and SSW condition for the second offset.

3.2. Trunk-pelvic motion and segmental coordination

Main effects of group were observed for global trunk ROM in sagittal ($F_{(1,64)} = 5.15$, $p = 0.027$, eta = 0.27), frontal ($F_{(1,64)} = 62.97$, $p < 0.0001$, eta = 0.70), and axial ($F_{(1,64)} = 26.63$, $p < 0.0001$, eta = 0.54) planes. Frontal plane trunk ROM was greater ($p < 0.0125$) among LLA vs. CTR across all speeds (Fig. 3). Axial plane trunk ROM was greater ($p < 0.0125$) in LLA vs. CTR during 1.3 m/s (13.0° vs. 9.6°) and SSW (11.9° vs. 8.5°) conditions (Fig. 3). There were no pairwise differences in the sagittal plane. While there was a main effect of group in the frontal plane ($F_{(1,56)} = 15.73$, $p < 0.0001$, eta = 0.44) , there were no pairwise differences in segmental coordination (Fig. 4). Although not statistically significant, LLA demonstrated a more in-phase coordination pattern than CTR in the frontal plane at all speeds.

4. Discussion

This study aimed to determine trunk muscle activation patterns, corresponding trunk-pelvic inter-segmental coordination, and speed-dependent pattern modulations in persons with LLA. In general, there were no differences in TES onset times between groups; however, LLA demonstrated a second TES onset (not seen in CTR) during mid-to-terminal swing, and TES activation for a larger percentage of the gait cycle. Also, LLA demonstrated an earlier onset of LES and activation for a larger percentage of the gait cycle at most speeds. As expected, persons with LLA consistently walked with increased frontal plane trunk ROM compared to CTR at all speeds. Corresponding CRP in the frontal plane was more in-phase in LLA (vs. CTR), supporting our hypothesis, and consistent with LBP patients and previous work (Esposito and

Table 3
Mean (standard deviation) activation as a percentage of affected/right gait cycle. Abbreviations: CTR: controls; LLA: persons with lower limb amputation; TES: thoracic erector spinae; LES: lumbar erector spinae.

Group	Muscle	Speed	Activation (% Gait)	P-value
CTR	TES	1.0	40.8 ± 15.9	0.771
LLA		1.0	38.8 ± 9.8	
CTR		1.3	48.2 ± 14.8	0.050
LLA		1.3	62.2 ± 11.3	
CTR		1.6	48.5 ± 18.2	0.167
LLA		1.6	61.1 ± 16.5	
CTR		SSW	35.5 ± 12.0	0.002
LLA		SSW	60.6 ± 14.8	
CTR	LES	1.0	39.8 ± 4.5	0.007
LLA		1.0	59.2 ± 16.9	
CTR		1.3	44.4 ± 10.1	0.003
LLA		1.3	66.8 ± 14.3	
CTR		1.6	44.8 ± 12.9	0.047
LLA		1.6	55.7 ± 5.8	
CTR		SSW	41.4 ± 8.0	0.005
LLA		SSW	64.2 ± 17.6	

Wilken, 2014; Seay et al., 2011).

4.1. Trunk muscle activation, motion, and speed dependency

Trunk muscles provide segmental stability while controlling trunk motion. As walking speed increases, activation magnitudes of the LES and TES respectively increase at heel strike and/or toe-off (Anders et al., 2007), which increase lumbar stability against braking forces at heel strike and eccentrically control the trunk prior to single-limb stance. In both CTR and LLA groups here, activation patterns were generally similar with increasing walking speed (Fig. 1), consistent with prior work in able-bodied controls (Anders et al., 2007).

Altered trunk muscular activation patterns were also observed in LLA vs. CTR, particularly during intact stance. Here, TES and LES were active at initial contact, remained active longer through early stance in LLA vs. CTR, and corresponded to increased lateral trunk flexion among persons with LLA during this phase of gait. Moving through terminal stance to mid swing, LLA significantly delayed the deactivation of TES compared to CTR; this delayed deactivation was coupled with an increase in lateral trunk flexion toward the affected (opposite) limb and axial rotation (shoulder opposite the intact limb is more forward than CTR and subsequently rotates backwards through stance). In preparation for the next intact heel strike, LLA then reactivate TES (i.e., second onset) during terminal swing, moving the center of mass back toward the intact limb. Of note, although temporal aspects of both stance and swing phases could influence trunk muscle activation patterns and kinematics, there were no significant differences observed between LLA and CTR groups in the current study, thereby mitigating this potential confounder. Contrary to our hypothesis, trunk ROM remained similar across walking speeds. The general invariance of trunk ROM across walking speeds in persons with LLA is not surprising considering global muscle (i.e., TES) activation was not different between speeds (Hendershot et al., 2018). Therefore, as the lumbar iliocostalis stabilizes the spine and thoracic longissimus laterally flexes the trunk, the lack of speed-dependent changes in activation would produce similar lateral trunk motions across speeds. The observed differences in trunk motion between groups are greater than reported minimal detectable change values previously reported for these variables (Wilken et al., 2012), with secondary analyses identifying a significant ($p = 0.01$) difference in step width between LLA and CTR at all speeds. The larger step width in LLA vs. CTR may be an adaptive control strategy to counteract greater lateral trunk motion, thereby increasing lateral stability. The increased activation of global trunk muscles (i.e., larger trunk muscles that span multiple segments to control trunk movement), paired with increased lateral trunk flexion and axial rotation, is therefore potentially an adopted control strategy driven by generation of torque proximally to advance the affected limb.

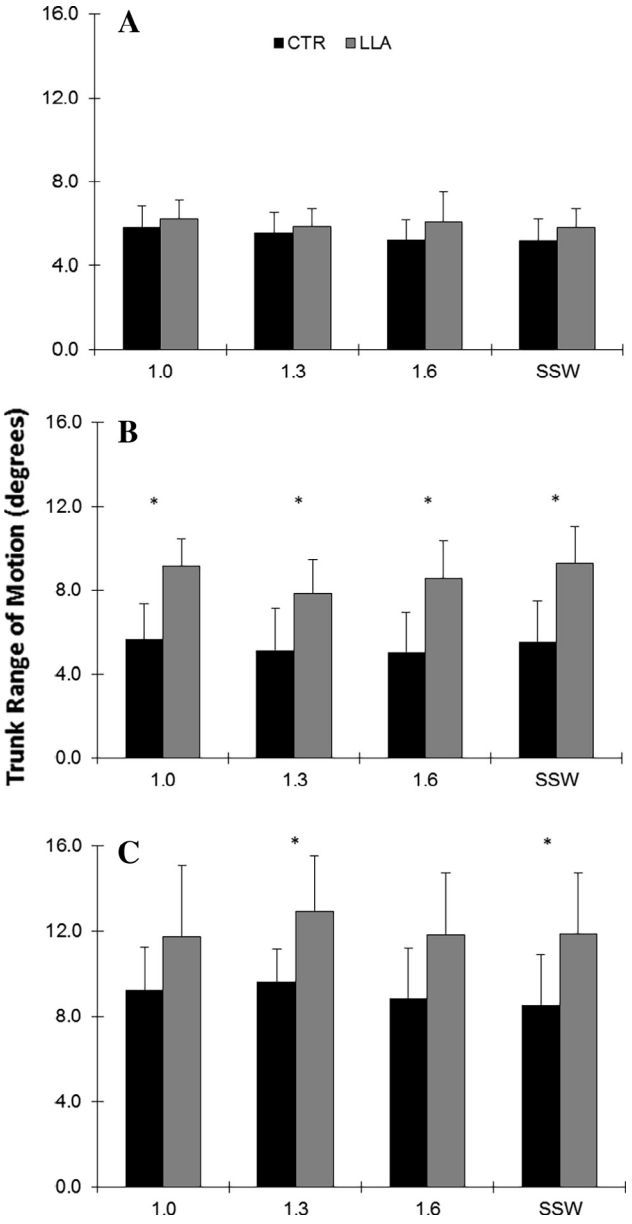

Fig. 3. Sagittal (A), frontal (B), and axial (C) trunk ranges of motion (ROM) at each speed. Compared to CTR, LLA walked with significantly larger trunk ROM at all speeds in the frontal plane, and at 1.3 m/s and 1.6 m/s in the axial plane. Asterisks (*) indicate significant differences ($p < 0.0125$) between groups.

During affected stance, LLA activated LES earlier than CTR at all speeds and maintained activation for a larger percentage of the gait cycle in all but the 1.6 m/s walking speed. The lack of a difference at 1.6 m/s may be due to an increased reliance on momentum to propel the body forward at a speed faster than their normal comfortable pace. These results are consistent with typical lumbar activation patterns in patients with LBP (Lamoth et al., 2006a; Vogt et al., 2003) and characterize the asymmetric gait mechanics and altered control strategies utilized by LLA. These activation patterns support the suggestion that LLA utilize greater TES activation than CTR during intact stance as a compensatory strategy to generate proximally generate torque to account for absent or altered torque generating capabilities distally, which is consistent with previous work (Hendershot and Wolf, 2014). Furthermore, the combination of increased ratio of LES to TES and

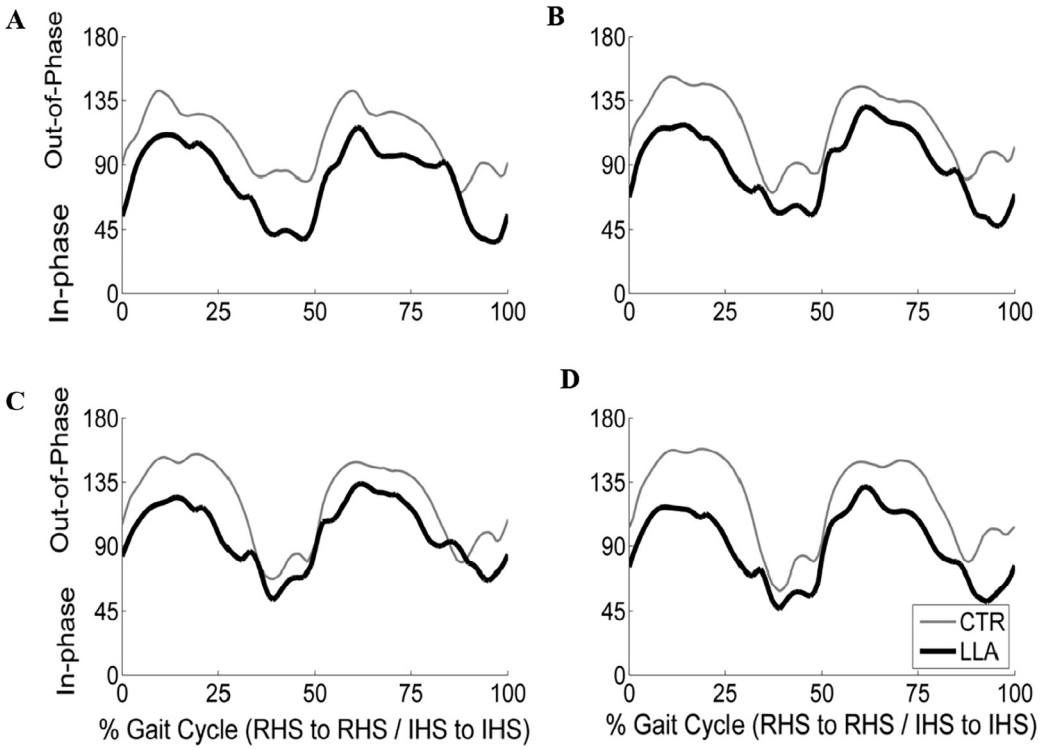

Fig. 4. Frontal plane segmental coordination among LLA (intact-intact foot strike) and CTR (right- right foot strike). (A) 1.0 m/s condition; (B) 1.3 m/s condition; (C) 1.6 m/s condition; (D) Self-selected walking velocity condition. Curves represent ensemble group averages.

overall occurrences of TES activations that are demonstrated by LLA and not CTR explain the more in-phase segmental coordination observed in LLA. Thus, it appears that LLA recruit global trunk musculature, increase global trunk ROM, and adopt a more rigid trunk-pelvic coordination pattern to aid forward propulsion of the affected limb. This strategy generates an increased rotational torque about the lumbar spine that, with repeated exposure, may offer a mechanistic explanation to LBP among persons with LLA.

4.2. Relationship to LBP

This is the first study to characterize trunk muscle activation patterns and corresponding trunk-pelvic motions in LLA during walking. These results suggest that these individuals adopt muscular activation patterns similar to able-bodied patients with LBP. The earlier activation of LES may stabilize the lumbar spine prior to affected limb heel contact and prepare the trunk to move towards the affected limb. As speed-related differences in LES patterns among LLA were most prominent when compared to the 1.0 m/s speed, the pattern observed at 1.0 m/s may be due to decreased muscular demand to advance the affected limb at slower (vs. faster) speeds.

During intact stance, there were no differences between groups in the second activation of TES; however, LLA activated TES significantly earlier than CTR during affected stance in the 1.3 m/s speed. While this is the first study to examine TES at the T9 vertebral level, the results are similar to previous work which found no thoracolumbar muscle activation (T12 vertebral level) of able-bodied controls during walking at a self-selected velocity (Vogt et al., 2003). Interestingly, these participants with chronic LBP demonstrated almost identical thoracolumbar activation patterns to controls (Vogt et al., 2003), which is inconsistent with the activation of TES presented here. These differences in activation patterns during gait could be attributed to the level of erector spinae analyzed, signal analysis differences (i.e., onset determined via computer-based algorithm vs. visual inspection), sample-specific characteristics, and/or functional walking demands of persons with LLA. Thus, it is possible that persons with LLA adopt unique neuromuscular control strategies that are mediated by functional requirements of locomotion and not by LBP (no/minimal disability; Table 1).

Trunk muscle activation and cyclical motions of the trunk and pelvis during the gait cycle generate loads on the lumbar spine. As walking speed increases, trunk muscle activation, lumbar motion, shear joint reaction forces and moments at the L4/L5 joint increase (Callaghan and McGill et al., 2001). Activation of trunk musculature increases stiffness and joint forces (McGill et al., 2003) and, when coupled with increases in trunk ROM and more in-phase segmental coordination patterns characteristic of LBP and LLA patient populations (Esposito and Wilken, 2014; Lamoth et al., 2006a) may elucidate pathways for LBP development (Hendershot et al., 2018). Of note, muscle activation magnitude was not an objective of the current study; therefore, EMG signals were not normalized to a reference signal (i.e., maximal voluntary contraction) as normalization is not required for temporal-based analyses of EMG data (Di Fabio, 1987; Hodges and Richardson, 1996b). However, the increased relative activation of LES to TES musculature, corresponding trunk “stiffening” strategy, and increased motion in the current study may be associated with an increase in intervertebral joint loads in the lumbar spine among persons with LLA. This control strategy could provide a mechanistic explanation for LBP development among persons with LLA.

4.3. Methodological considerations

The use of both persons with (traumatic) transtibial and/or transfemoral LLA is novel and allows for the generalization of the results to both of these populations; although caution is needed as the results of the current study may not be generalizable to individuals with LLA due to other causes. Previous reports suggest gait mechanics differ between persons with transfemoral and transtibial LLA; however, there were no statistically significant trunk-pelvic kinematic differences between individuals in the current study. While visual inspection is accepted as the “gold standard” of EMG onset detection (Hodges and Bui, 1996a; Solnik

et al., 2010; Tenan et al., 2017), this method is inherently variable and susceptible to human error. Reviewers in the current study demonstrated good test-retest reliability that is consistent with previous work, mitigating this concern. The general lack of significant post hoc differences between groups at each speed may be a function of small sample size and thus type II error. Nevertheless, the large effect sizes observed as group main effects support our hypotheses as well as confidence in the presence of group differences in trunk muscle activation patterns. Future work should also consider assessing anterior trunk muscular activation to determine flexor/extensor co-activation strategies as well as a reference criterion that allows for the comparison of activation magnitudes between groups.

4.4. Conclusions

Persons with LLA demonstrate altered activation of posterior trunk muscles (i.e., earlier onsets and delayed offsets) compared to able-bodied controls during walking. While prior work in able-bodied individuals with LBP has suggested that altered LES activation patterns are a function of poor neuromuscular control and efforts to increase lumbar stability, it appears persons with LLA adopt proximal strategies to advance the affected limb during over-ground walking. However, the differential patterns of muscular activation and trunk-pelvic motions may influence spinal loads and subsequently increase LBP risk. Further work is needed to explicitly relate muscular activation patterns (and magnitudes) with spinal loading during walking in persons with LLA.

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Conflict of interest

The authors declare that there is no conflict of interest.

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Review Article

Low back pain in persons with lower extremity amputation: a systematic review of the literature

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Abstract

BACKGROUND CONTEXT: Lower extremity amputation (LEA) is associated with an elevated risk for development and progression of secondary health conditions. Low back pain (LBP) is one such condition adversely affecting function, independence, and quality of life.

PURPOSE: The purpose of this study was to systematically review the literature to determine the strength of evidence relating the presence and severity of LBP secondary to LEA, thereby supporting the formulation of empirical evidence statements (EESs) to guide practice and future research.

STUDY DESIGN/SETTING: Systematic review of the literature.

METHODS: A systematic review of five databases was conducted followed by evaluation of evidence and synthesis of EESs.

FDA device/drug status: Not applicable

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RESULTS: Seventeen manuscripts were included. From these, eight EESs were synthesized within the following categories: epidemiology, amputation level, function, disability, leg length, posture, spinal kinematics, and osseointegrated prostheses. Only the EES on epidemiology was supported by evidence at the moderate confidence level given support by eight moderate quality studies. The four EESs for amputation level, leg length, posture, and spinal kinematics were supported by evidence at the low confidence level given that each of these statements had some evidence not supporting the statement but ultimately more evidence (and of higher quality) currently supporting the statement. The remaining three EESs that addressed function, disability and osseointegrated prosthetic use were all supported by single studies or had comparable evidence that disagreed with study findings rendering insufficient evidence to support the respective EES.

CONCLUSIONS: Based on the state of the current evidence, appropriate preventative and, particularly, treatment strategies to manage LBP in persons with LEA remain a knowledge gap and an area of future study.

Keywords: Amputee; Limb loss; Lumbago; Rehabilitation; Spinal pain; Transfemoral; Transtibial.

Introduction

Common musculoskeletal derangements of the spine that contribute to low back pain (LBP) include discogenic dysfunction, facet joint syndrome, sacroiliac joint syndrome, spinal instability, and postural syndrome [1]. There are many factors related to spinal derangements including behavioral, congenital, traumatic, disease processes, and others. These derangements and factors can co-exist, leading to varying levels of disability attributed to LBP. Severe lower extremity trauma, including lower extremity amputation (LEA), can further confound and complicate the clinical presentation and management of LBP [2].

The prevalence of LBP is much higher (52%–89%) among persons with LEA as compared to the general non-amputee population (12%–45%) [3–6]. Low back pain has been considered more bothersome than residual and phantom limb pain [4]. In a cross-sectional survey of persons with LEA (n = 255), 52% rated their pain as persistent and 25% described their pain as frequent and severely interfering with daily activities [6]. Performance of daily activities with altered anatomy and biomechanics may be related to the development of LBP following LEA [7–10]. Persons with LEA present unique challenges to rehabilitation clinicians managing their LBP. Clinical practice guidelines highlighting efficacious interventions to manage LBP in this group are not available. However, sparse evidence regarding the underlying mechanisms, prevalence, intensity, and management of LBP among those with LEA is available. A systematic review and synthesis of evidence in these areas may inform the development of targeted inventions and lead to improved rehabilitation in this population. Therefore, the purpose of this project was to systematically review and evaluatethe literature, and to formulate empirical evidence statements (EESs) regarding the etiology, epidemiology, and management of patients with LEA and LBP.

Materials and methods

Search strategy

A search strategy used in several previous prosthesis- and amputation-related systematic reviews was implemented[11,12]. Five medical literature databases (Medline/Pubmed, CINAHL, EMBASE Elsevier, Web of Science, and Cochrane Clinical Trials Register) were searched on January 1, 2016 based on the following terms (Table 1):

- Primary search terms (target population): *transtibial, transfemoral, lower extremity*, and *amputee*.
- Secondary search terms (target comorbidity): *low back pain, sciatica, lumbago*, back pain, *back disorder, spinal disease*, and *backache*.

Searches were prelimited using the following criteria: English language, abstract available, and peer reviewed. A manual search of included articles' reference lists was also conducted in the event very recent publications or keywords missed important publications in the electronic automated search.

Screening

Resulting references were exported to EndNote (vX7, Thompson, CA, USA) bibliographic citation software. Two reviewers independently screened resulting references' titles, then abstracts, and finally full text articles according to inclusion/exclusion criteria (listed below). Articles were then classified as either (i) pertinent, (ii) not pertinent, or (iii) uncertain pertinence. Full-text articles were then reviewed for all manuscripts classified as pertinent or uncertain pertinence. Disagreements regarding citations of uncertain pertinence were resolved by having a third reviewer independently review full-text articles, discuss, and reach agreement on ultimate inclusion or exclusion.

Inclusion criteria were as follows: (1) peer-reviewed publication; (2) English language; (3) published within the previous 10 years (2006–2016); and (4) study

Table 1
Selected sample search term sets and from the Medline and CINAHL databases

Database	Medline	CINAHL
General search term set	Dorsalgia[tiab] OR exp Back Pain OR backache[tiab] OR (lumbar pain)[tiab] OR coccyx[tiab] OR coccydynia[tiab] OR sciatica[tiab] OR sciatic neuropathy/ OR spondylosis [tiab] OR lumbago[tiab]	"lumbago" OR (MH "Spondylolisthesis") OR (MH "Spondylolysis") OR (MH "Thoracic Vertebrae") OR lumbar N2 vertebra OR (MH "Lumbar Vertebrae") OR "coccydynia" OR "coccyx" OR "sciatica" OR (MH "Sciatica") OR (MH "Coccyx") OR lumbar N5 pain OR lumbar W1 pain OR "backache" OR (MH "Low Back Pain") OR (MH "Back Pain+") OR "dorsalgia"
Amput* string	((((("Lower Extremity"[Mesh] OR lower extrem*[TIAB] OR lower extrem*[OT] OR lower limb*[TIAB] OR lower limb*[OT] OR leg[TIAB] OR leg[OT] OR legs[TIAB] OR legs [OT] OR hip[TIAB] OR hip[OT] OR hips[TIAB] OR hips [OT] OR thigh*[TIAB] OR thigh*[OT] OR foot[TIAB] OR foot[OT] OR feet[TIAB] OR feet[OT] OR "Knee Joint"[Mesh] OR knee[TIAB] OR knee[OT] OR knees[TIAB] OR knees[OT] OR "Ankle Joint"[Mesh] OR ankle*[TIAB] OR ankle*[OT] OR "Femur"[Mesh] OR femur*[TIAB] OR femur*[OT] OR transfemoral[TIAB] OR transfemoral[OT] OR trans-femoral[TIAB] OR trans-femoral[OT] OR "Tibia"[Mesh] OR tibia*[TIAB] OR tibia*[OT] OR transtibial[TIAB] OR transtibial[OT] OR trans-tibial[TIAB] OR trans-tibial [OT] OR transpelvic[TIAB] OR transpelvic[OT] OR trans-pelvic[TIAB] OR trans-pelvic[OT] OR syme's[TIAB] OR syme's[OT] OR symes[TIAB] OR symes[OT]))) AND (("Amputation"[Mesh] OR amput*[TIAB] OR amput*[OT] OR disarticulat*[TIAB] OR disarticulat*[OT] OR hemipelvectom*[TIAB] OR hemipelvectom*[OT] OR "Amputees"[Mesh] OR "Amputation Stumps"[Mesh] OR "Artificial Limbs"[Mesh] OR artificial limb*[TIAB] OR artificial limb*[OT] OR "Amputation, Traumatic"[Mesh] OR "Prostheses and Implants"[Mesh:noexp] OR residual limb*[TIAB] OR residual limb*[OT] OR limb loss*[TIAB] OR limb loss*[OT] OR prosthe*[TIAB] OR prosthe*[OT] OR stump*[TIAB] OR stump*[OT]))))	((MH "Lower Extremity+") OR (TI lower extrem* OR AB lower extrem*) OR (TI lower limb* OR AB lower limb*) OR (TI leg OR AB leg) OR (TI legs OR AB legs) OR (TI hip OR AB hip) OR (TI hips OR AB hips) OR (TI foot OR AB foot) OR (TI feet OR AB feet) OR (MH "Knee Joint+") OR (TI knee OR AB knee) OR (TI knees OR AB knees) OR (MH "Ankle Joint") OR (TI ankle* OR AB ankle*) OR (MH "Femur+") OR (TI femur* OR AB femur*) OR (TI transfemoral OR AB transfemoral) OR (TI trans-femoral OR AB trans-femoral) OR (MH "Tibia") OR (TI tibia* OR AB tibia*) OR (TI transtibial OR AB transtibial) OR (TI trans-tibial OR AB trans-tibial) OR (TI transpelvic OR AB transpelvic) OR (TI trans-pelvic OR AB trans-pelvic) OR (TI syme's OR AB syme's) OR (TI symes OR AB symes) OR (TI thigh* OR AB thigh*)) AND ((MH "Amputation+") OR (TI amput* OR AB amput*) OR (TI disarticulat* OR AB disarticulat*) OR (TI hemipelvectom* OR AB hemipelvectom*) OR (MH "Amputees") OR (MH "Amputation, Traumatic") OR (MH "Limb Prosthesis") OR (TI prosthe* OR AB prosthe*) OR (TI artificial limb* OR AB artificial limb*) OR (TI limb loss OR AB limb loss) OR (TI residual limb* OR AB residual limb*) OR (TI stump* OR AB stump*) OR (MH "Prostheses and Implants"))

included subjects with both lower extremity amputation and low back pain;

Exclusion criteria were as follows: publication date outside of the 10-year search window; nonhuman subject research; non-English language; pediatric studies; studies of patients with bilateral lower extremity amputations; case report or case series methodology; studies of digit or partial foot amputation; hypothesis, editorial, classification, or taxonomy papers; thesis, dissertation, and preliminary or pilot level research; and duplicate publication.

Study data

Data from each article including demographic, anthropometric, dependent and independent variables, quantifiable outcomes, and conclusions were entered into an Excel database (Microsoft Corporation. Redmond, WA, USA). These data were verified by a multidisciplinary team (ie, physical therapists, prosthetists, chiropractors, and biomechanists) for completeness and accuracy. Data were assessed for the ability to aggregate for descriptive characteristics (ie, anthropometrics) as well as outcomes (ie kinematic data and pain) and to calculate effect sizes (Cohen *D*) [13]. To prevent double counting of subject data, data from systematic reviews were not included in the extraction and aggregation.

Quality assessment

The study design and methodologic quality of those publications meeting eligibility criteria were independently assessed by two reviewers according to the American Academy of Orthotists and Prosthetists (AAOP) State-of-the-Science Evidence Report Guideline Protocol [14]. Prior to assessment, the two raters participated in a prelaunch reliability procedure. Test articles were assigned to the two reviewers for assessment. The process was repeated until 90% agreement was attained regarding use of the AAOP rating tool as scored by a third rater. Reviewers discussed pertinent issues until consensus on study design and methodological quality was obtained for the included publications. Each reviewer rated each study according to the AAOP Study Design Classification Scale that describes the type of study design [14]. The State of the Science Conference Quality Assessment Form [14] was used to rate

methodologic quality of studies classified as experimental (E1–E5) or observational (O1–O6). The form identifies 18 potential threats to internal validity and eight potential threats to external validity. In accordance with the guidelines, examples of criteria are provided and described as not applicable for certain study designs; however, guidelines indicate that provided examples are not exhaustive and that reviewers should use their judgment in determining which criteria are not applicable for certain study designs [14]. Threats were evaluated and tabulated.

The internal and external validity of each study was then subjectively rated as "high," "moderate," or "low" based on the quantity and importance of threats present. As a guide for rating the internal and external validity separately, studies achieving $\geq 80\%$ of applicable criteria were classified as "high." If studies achieved $<80\%$ but $>50\%$ of applicable criteria, they were classified as "moderate." Studies achieving $\leq 50\%$ of applicable criteria received a "low" classification. Each study was then given an overall quality of evidence rating of either "high," "moderate," or "low" by combining the ratings of internal and external validity as outlined by the AAOP State-of-the-Science Evidence Report Guidelines [14]. The overall ratings from the AAOP State-of-the-Science Evidence Report Guidelines were used in assigning confidence to the developed EESs described in the *Results* section.

Empirical evidence statements

Based on results from the included publications, EESs were developed describing collective findings from included research regarding LBP in persons with LEA. Reviewers rated the level of confidence of each EES as "high," "moderate," "low," or "insufficient," based on the number of publications contributing to the statement; the methodologic quality of those studies and whether the contributing findings were confirmatory or conflicting [14].

Results

In total, 302 articles were identified from the search (Fig. 1). Ten articles required eligibility determination by the third rater. In most cases, articles requiring the additional review were studies of the spine in a sample of individuals with amputation but the subjects did not have a history of LBP and thus were excluded.

Ultimately, 17 of the original 302 articles met inclusion criteria. Four articles were published in 2009. Between 2006 and 2016, the mean (standard deviation [SD]) number of articles published per year on the subject of LBP in LEA was 1.5 (1.1) (Fig. 2). Study designs included 13 cross-sectional studies, one controlled trial, and three systematic reviews (Table 2). Manuscripts were published predominantly in physical medicine, rehabilitative, and

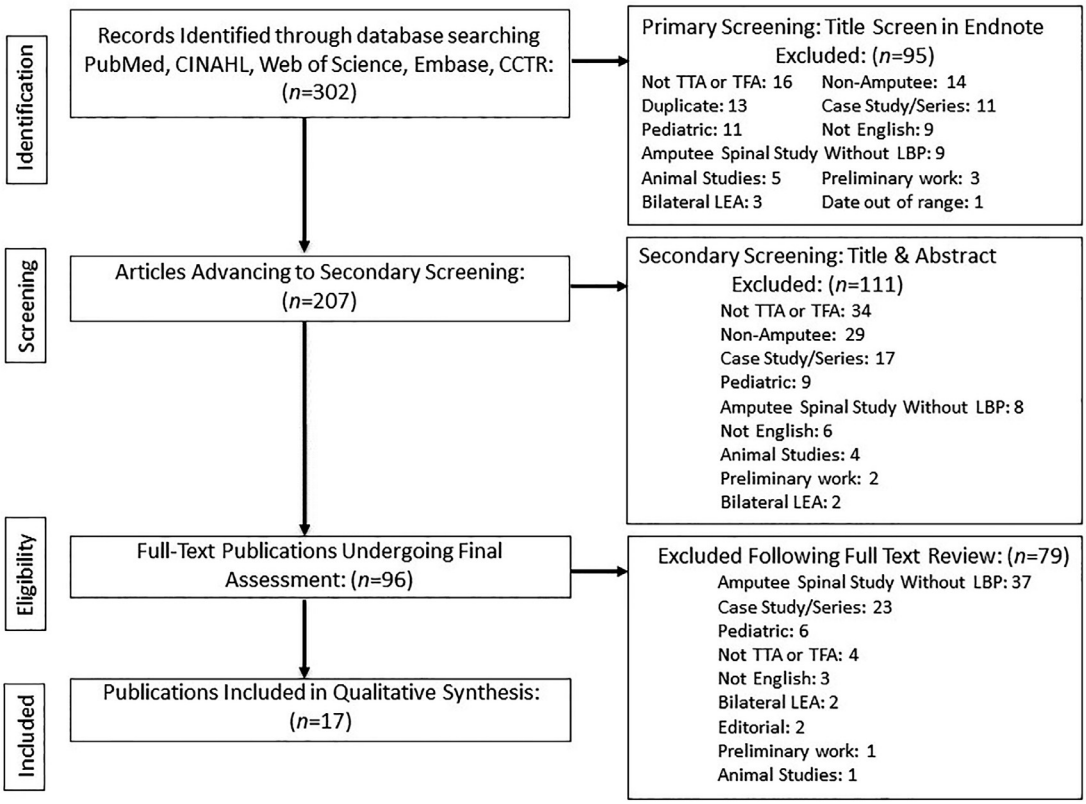

Fig. 1. Results of the literature search and application. TTA, transtibial amputee; TFA, transfemoral amputee; LBP, low back pain; LEA, lower extremity amputation.

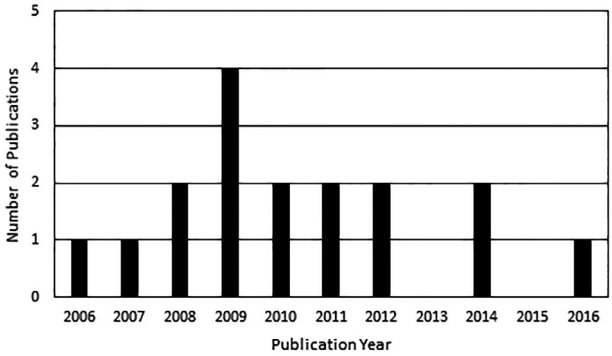

Fig. 2. Included studies by publication year.

Table 2
Distribution of included studies by study design

Study design	Number of publications
Meta-analysis (S_1)	0
Systematic review (S_2)	3
Randomized control trial (E_1)	0
Controlled trial (E_2)	1
Interrupted time series trial (E_3)	0
Single subject trial (E_4)	0
Controlled before and after trial (E_5)	0
Cohort study (O_1)	0
Case-control study (O_2)	0
Cross-sectional study (O_3)	13
Qualitative study (O_4)	0
Case series (O_5)	0
Case study (O_6)	0
Group consensus (X_1)	0
Expert opinion (X_2)	0
Total	17

Table 3
Distribution of the studies per journal

Journal	Number of publications
American Journal of Physical Medicine & Rehabilitation	3
Archives of Physical Medicine & Rehabilitation	1
Bosnian Journal of Basic Medical Sciences	1
British Journal of Surgery	1
Disability & Rehabilitation	1
Gait and Posture	1
Irish Journal of Medical Science	1
Journal of Foot & Ankle Surgery	1
Journal of Orthopaedic Trauma	1
Journal of Rehabilitation Research & Development	4
Military Medicine	1
Prosthetics Orthotics International	1
Total	17

biomechanical journals (Table 3). Due to heterogeneity in sample size and demography, methods, accommodation periods, outcome measures and design, the calculation of effect sizes and meta-analyses was not possible (Table 4).

Subjects

The clinical, patient-oriented studies included a total of 1,260 experimental subjects with a mean (SD; range) sample size of n = 79 (94;8–298). These were subjects with the combination of LEA and LBP. The interquartile mean (IQM) (interquartile range [IQR]) age for experimental study subjects (ie, those with LEA and BP) included in the clinical, patient-oriented studies with adequate data to aggregate was 47.2 years (8.2). The absolute age range of experimental subjects was 16 to 93 years. The height and weight of subjects were only reported in one of the 17 studies. Body mass index was reported or could be calculated in three [15–17] of the 17 studies with an IQM (IQR) of 27.1 m/kg^2 (0.4), which is considered "overweight" according to the Centers for Disease Control and Prevention.

Of studies sufficiently describing subjects for analysis of amputation level and etiology, the majority of amputee subjects (48.3%) were transfemoral level and 36.7% were transtibial level. The remaining 15% included partial foot amputation and disarticulations of the ankle, knee, and hip. In terms of amputation etiology, when sufficiently described for detailed analysis, the majority of amputations (89.5%) were caused by trauma. Malignancy, vascular disease, illness, and congenital limb difference were the causes for limb loss in the remaining cases.

Internal validity

Prior to rating, the prelaunch reliability procedure required three test ratings for the two raters to achieve <90% agreement. The most prevalent threats to internal validity in this body of literature include a lack of blinding, lack of use of a control group, no reported consideration for fatigue, learning, accommodation and washout, no reporting of effect size, and lack of random allocation (Table 5). Considering all included studies, the overall assessment favored moderate level internal validity (13/17 studies). Two of 17 had high internal validity [15,18] and two [19,20] had low internal validity. Additionally, seven studies had attrition greater than 20%.

External validity

The greatest threat to external validity was inadequate descriptions of the study samples. Specifically, amputation levels, sociodemography, and anthropometry were not clearly described. Thus, it is difficult to know whether findings are generalizable to the larger population of persons with LEA and LBP. Nevertheless, the majority of the studies (12/17) had high external validity and five had moderate external validity (Table 5).

Table 4
Participant characteristics, primary outcomes and conclusions extracted from included studies

Author (year)	Population (etiology)	Amputation level (sample size)	Mean (range/SD) age (y)	Mean (range/SD) time since amputation (y)	Primary outcome measure(s)	Conclusions
Kusljugic (2006)	Civilian/military (traumatic)	LEA (37)	46 (11)	Not reported	Pain prevalence, psychosocial factors	89% report chronic LBP. Higher levels of social function among civilian versus military
Ebrahimzadeh (2007)	Military (traumatic)	LEA (27)	21 (16–54)*	17 (15–22)	Pain prevalence, psychosocial factors	74% report long-term pain and discomfort
Smith (2008)	Civilian (trauma, PVD, cong, tumor)	TTA (57), TFA (32) KD (4), HD (2), BLEA (10), AD (2)	51 (16–83)	17 (15)	LBP, RLP (periodicity, Frequency, intensity, ADL interference)	48% had LBP w/ 5/10 intensity and reported activity interference of 3.4-3.8/10
Morgenroth (2009)	Civilian/veterans (traumatic)	TFA w/ (9) and w/out (9) LBP	51 (12)	23 (15)	Static and dynamic leg length in single- and double-limb support	Static and dynamic leg length discrepancy not different b/t groups
Taghipour (2009)	Military (traumatic)	LEA (141)	45 (36–63)	22 (20–27)	Pain prevalence, health-related quality of life	LBP most impactful physical condition reducing quality of life
Ebrahimzadeh (2009)	Military (traumatic)	TTA (200)	23 (14–60)*	17 (15–22)	Pain prevalence, psychosocial factors	At long-term follow-up (~17 y), 44% reported LBP and 54% had persistent psychological problems
Morgenroth (2010)	Civilian/veterans (traumatic)	TFA w/ (9) and w/out (8) LBP, CTR (6)	50 (30–77)	23 (3–57)	Lumbar spine kinematics	Larger transverse rotations among LBP group
Reiber (2010)	Military (traumatic)	Vietnam (298), OIF/OEF (283)	61/29	39 (4)/3 (1)	Pain prevalence and psychosocial factors	36%–42% report chronic LBP, 37%–59% w/ PTSD symptoms
Behr (2011)	(Traumatic, vascular)	TFA (14), KD (14), TTA (14)	55 (36–85)	12 (0.6–56)	Pain prevalence and activity level	57% reported LBP that was "troublesome"
Hammarlund (2011)	Not specified (traumatic/tumor)	TFA (19), KD (9), TTA (18)	48 (19–79)	23 (3–58)	Pain prevalence, health-related quality of life (RMDQ, SF36)	87% reported LBP after amputation (vs 20% before); not different by amputation level. Lower quality of life versus normative data
Devan (2012)	Civilian (traumatic)	TFA (145)	57 (18–93)	27 (1–66)	LBP prevalence, physical activity questionnaires	64% reported LBP and 39% reported activity restriction due to LBP
Esposito (2014)	Military (traumatic)	TFA w/ (9) and w/out (7) LBP, CTR (12)	28 (22–39)	2.7 (0.4–5.9)	Trunk-pelvic segmental coordination	Increased coronal in-phase coordination (segmental rigidity) w/ LBP
Hagberg (2014)	Not specified (trauma, tumor, other)	TFA (39)	44 (12)	Not reported	Health-related quality of life (Q-TFA, SF36)	Improved quality of life, prosthesis use, and physical activity 2 y after OI
Fatone (2016)	civilian	TFA w/ (12) and w/out (11) LBP	47 (20–67)	16 (2–41)	Pelvic and spinal kinematics	Reversal of motion pattern in sagittal/transverse plane w/ and w/out LBP

W/, with; w/out, without; Y, year(s); SD, standard deviation; LEA, lower extremity amputation; TTA, transtibial amputation; TFA, transfemoral amputation; LBP, low back pain; OEF, Operation Enduring Freedom; OIF, Operation Iraqi Freedom; RLP, residual limb pain; ADL, activity(ies) of daily living; AD, ankle disarticulation; KD, knee disarticulation; HD, hip disarticulation; BLEA, bilateral lower extremity amputee; OI, osseointegration; CTR, control (subjects); RMDQ, Roland Morris disability questionnaire; SF36, short form 36 health survey; PTSD, post-traumatic stress disorder; Q-TFA, Questionnaire for persons with transfemoral amputation.

*At the time of injury.

Table 5
Internal and external validity of included studies

Author (year)	Study design	Internal validity																		External validity								
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	Total	1	2	3	4	5	6	7	8	Total
Kusljugic (2006)[1]	O3	n/a	n/a	n/a	n/a	n/a				n/a	n/a	•	n/a	•	•		•	•	•	Mod			•	•	•	•	•	•	Mod
Ebrahimzadeh (2007)[2]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a		n/a	•						Low	•	•	•	•	•	•	•	•	High
Smith (2008)[3]	O3	n/a	n/a	n/a	n/a	n/a		•		n/a	n/a	•	n/a	•	•		•	•	•	Mod	•		•	•	•	•	•	•	High
Gailey (2008)[4]	S2	n/a	n/a	n/a	n/a	n/a	n/a	•	•	n/a	n/a	n/a	n/a	n/a	n/a		n/a	n/a	•	Mod	•	•	n/a	n/a	n/a	•	•	•	High
Morgenroth (2009)[5]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a	•	•	•	•		•	•	•	High	•		•	n/a	•	•	•	•	High
Robbins (2009)[6]	S2	n/a	n/a	n/a	n/a	n/a	n/a	•		n/a	n/a	n/a	n/a	n/a	n/a		n/a	n/a	•	Low	•	•	n/a	n/a	n/a	•	•	•	High
Taghipour (2009)[7]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a		n/a	•	•		•	•	•	Mod	•	•	•	•	•	•	•	•	High
Ebrahimzadeh (2009)[8]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a		n/a	•	•		•	•	•	Mod	•	•	•	•	•	•	•	•	High
Morgenroth (2010)[9]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a	•	n/a	•	•	•	•	•	•	High			•	•	•	•	•	•	Mod
Reiber (2010)[10]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a		•	•	•		•	•	•	Mod	•	•		•	•		•	•	High
Behr (2011)[11]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a	•	•		•	•	•		•	Mod			•	•		•	•	•	Mod
Hammarlund (2011)[12]	O3	n/a	n/a	n/a	n/a	n/a		•	•		•		•	•	•		•	•	•	Mod	•		•	•	•	•	•	•	High
Perkins (2012)[13]	S2	n/a	n/a	n/a	n/a	n/a	n/a	•	•	n/a	n/a	n/a	n/a	n/a	n/a		n/a	n/a	•	Mod			n/a	n/a	n/a	•	•	•	Mod
Devan (2012)[14]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a		n/a	•	•		•		•	Mod	•		•	•		•	•	•	High
Russell Esposito (2014)[15]	O3	n/a	n/a	n/a	n/a	n/a				n/a	n/a	•	•	•	•		•	•	•	Mod		•	•	•	•	•	•	•	High
Hagberg (2014)[16]	E2			•	•	n/a		•	•			•		•	•		•	•	•	Mod	•	•	•	•	•	•	•	•	High
Fatone (2016)[17]	O3	n/a	n/a	n/a	n/a	n/a		•	•	n/a	n/a			•	•	•	•	•	•	Mod	•	•	•	•			•		Mod

Scoring: a dot (•) indicates that the criterion was met. A blank space indicates that the criterion was not met. n/a means the item was not applicable in that particular study design. See Table 1 for study design key.

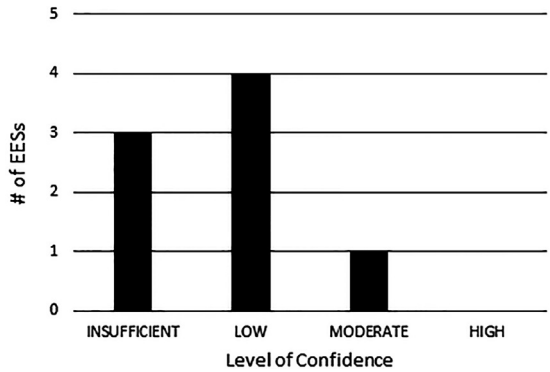

Fig. 3. Distribution of level of confidence for empirical evidence statements (EESs).

Funding analysis

Funding was declared in seven of 17 manuscripts (41%). One manuscript indicated private foundation support, two indicated funding through an academic institution, and four manuscripts indicated funding by way of a governmental sponsor. Within the four US government funded studies, three were supported by the US Department of Veterans Affairs and one by the National Institutes of Health. Three of the studies were funded outside of the United States. Support was not declared in nine manuscripts (53%), whereas one manuscript specifically declared that it was unfunded.

Empirical evidence statements

Eight empirical evidence statements were synthesized. The rate of EES production in this body of evidence was eight EES's per 10 years or a crude rate of 0.8 EES/y. Lack of evidence resulted in insufficient confidence in three of the statements. Evidence supported low confidence in four of the statements and moderate confidence in one EES (Fig. 3). The topical areas covered included epidemiology, amputation level, function, disability, leg length, posture, spinal kinematics, and osseointegrated prosthetic use. Only the epidemiology EES was supported by evidence at the moderate confidence level given that it was supported by eight moderate quality studies. The four EESs for amputation level, leg length, posture, and spinal kinematics were supported by evidence at the low confidence level given that each of these statements had some evidence not supporting the statement but ultimately more evidence (and of higher quality) supporting the statement at the present time. Finally, the remaining three EESs that addressed function, disability, and osseointegrated prosthetic use were all supported by single studies or had comparable evidence (quantity and quality) that disagreed with study findings rendering insufficient evidence to support the respective EES (Table 6).

Discussion

With regard to study design, 13 of the included studies were observational, one was experimental, and three were

Table 6
Empirical evidence statements, indicating level of confidence and category

Empirical evidence statement (EES)		Supporting studies	Level of confidence	Category
1	Back pain increases following lower extremity amputation	8 × Mod*[1, 2, 6, 8, 10, 12-14]	Moderate	Epidemiology
2	Back pain is affected by level of amputation	Support: 4 × Mod*[4, 6, 7, 13] Does not support: 3 × Mod[3, 11, 12]	Low	Amputation level
3	In persons with lower extremity amputation, function is affected by back pain	Support: 1 × Mod[12] Does not support: 1 × Mod[14]	Insufficient	Function
4	Frequent bouts of back pain in persons with lower extremity amputation are associated with increased disability	1 × Mod[12]	Insufficient	Disability
5	Leg length discrepancy is associated with back pain in persons with lower extremity amputation	Support: 1 × Mod*[4] Does not support: 1 × High[5]	Low	Leg length
6	Postural asymmetries and postural control issues are associated with back pain in patients with lower extremity amputation	Support: 1 × Mod*[4] Does not support: 1 × High[5]	Low	Posture
7	Spinal and pelvic kinematics are influenced by low back pain in persons with lower extremity amputation	Support: 2 × Mod[9, 15] Does not support: 1 × Mod[17]	Low	Spinal kinematics
8	Back pain is not affected by the use of osseointegrated prosthesis	1 × Mod[16]	Insufficient	Osseointegrated prosthetic use

*Indicates that the supporting reference is or includes a systematic review.

systematic reviews. Although this is a somewhat heterogeneous blend of study designs, a more optimal body of literature inclusive of prospective, randomized controlled intervention trials may have enabled meta-analyses. Internal validity could have been strengthened in the included studies with minor reporting changes as described by standardized criteria [21,22]. For instance, had the included samples been better described (ie, more uniform reporting of anthropometry and demography), effect sizes been reported, and learning/accommodation and fatigue reported, more of the studies would have likely improved their internal validity ratings from low to moderate or moderate to high. Conversely, external validity was generally high in the selected studies that provide confidence that results have clinical importance despite some methodological weaknesses (ie, threats to internal validity).

In this study, the rate of EES production regarding subjects having LEA and LBP was eight EES's per 10 years (crude rate of 0.8 EES/y). This rate of EES production is considerably low compared to other areas of prosthetic literature. For example, in a previous study of lower extremity prosthetic componentry for persons with transtibial amputation [23], the EES production rate was 1.4 EES/y. More problematic is that in the componentry review, this EES production rate was based upon the use of high-quality evidence, whereas the present review of LBP in LEA is low but is based upon all available quality of evidence. Further, although key sponsors, such as NIH, were notably absent as research supporters in the componentry review, all of the studies included were funded (ie, industry, other governmental departments, nonprofit sponsors, etc.). In the present review, the majority of research available, 53%, was unfunded. This identifies numerous potential issues. For instance, high-quality research can become more difficult to accomplish without adequate funding, which could also decrease interest among researchers in this area. More funding from key research sponsors is needed in this area if the quality and quantity of available research are to become available to fill knowledge gaps related to the care of persons with LEA who suffer from LBP.

Because the majority of this body of evidence was observational by study design, the EESs tended to describe factors that affect or are affected by LBP in persons with LEA. For example, EESs described LBP as increasing following LEA, differences by amputation level, decreased function, increased disability, and altered gait mechanics associated with LBP in persons with LEA. Again, these EESs are predominated by descriptions of LBP and its effects in persons with LEA. Thus, the number of experimental studies was limited to one, minimizing the ability to determine optimal therapeutic intervention choices or their effects in managing persons with LEA who have LBP. Therefore, efficacy of interventions to manage LBP in persons with LEA remains a considerable knowledge gap and an area of future study.

The first EES indicates that LBP increases following lower extremity amputation. Eight moderate quality studies support the statement ultimately providing moderate confidence in the EES [3,5,19,20,24–27]. The reported prevalence of LBP in the included studies ranged broadly from a minimum of 36% to a maximum of 89% with an interquartile range of 34% [3,25]. The interquartile mean(SD) of the reported prevalence was 62(19)%. These minimum and mean prevalence rates of LBP in persons with LEA are considerably higher than the 15% to 25% prevalence values of LBP reported in the nonamputee general population [25].

One study reported significantly increased LBP after amputation as opposed to before amputation [5]. Furthermore, the characteristics and consequences of this pain in persons with LEA have been described as progressive, disabling, and contributing to limitations in occupation, recreation, and socialization [19,20]. With regard to function, LBP in LEAs has been associated with problems sitting, sleeping, and traveling [3]. Finally, LBP in this population has been associated with decreased health-related quality of life [5].

The second EES states that back pain is affected by level of amputation. Four moderate quality studies support the statement whereas three moderate quality studies do not support it. Ultimately, this yields a low level of confidence in the statement. Importantly, two of the studies supporting the statement were systematic literature reviews [20,28]. Both concluded that persons with transfemoral level amputation reported LBP with a higher prevalence than their transtibial counterparts, which was consistent with two additional clinical studies [27,29]. Perkins et al. suggest that the increased susceptibility to LBP at the higher amputation level may in part be the result of myofascial changes following transfemoral amputation along with gait pattern alterations [27]. Confirming these proposed causes for LBP following LEA through further research could lead to improvements in prevention and management.

Adverse effects of function related to LBP are the subject of the third EES. Hammarlund et al. used the Roland Morris Disability Questionnaire (RMDQ), a valid and reliable measure of functional capacity relative to perceived back pain in a sample of 46 nondysvascular lower extremity amputees [5]. They concluded that nearly all participants with LBP daily or several times per week reported severe or moderate disability on the RMDQ. Devan et al. assessed the relationship of back pain on function in terms of physical activity [26]. Overall, they concluded that there was no relationship between physical activity of LEAs with or without LBP and that there was an equal distribution of persons with LBP in low, medium, and high physical activity groups. They did however find that those reporting activity limitations due to LBP had lower physical activity scores than those with LBP who did not have physical activity limitations. It is important to note that this difference in function related to LBP is potentially confounded by the use of two different outcome approaches and further by the fact that Devan et al. studied those with traumatic transfemoral amputation, whereas the Hammarlund et al. sample was more heterogeneous by amputation level [5,26]. Nonetheless, further evidence is needed to understand which elements of function may potentially be impaired by LBP in persons with LEA.

Increasing frequency of LBP episodes and associated disability is the subject of EES four. A single, moderate-quality study supports EES four with a significant association ($p = .003$) between LEAs who reported LBP daily or several times per week and those reporting moderate or severe disability [5]. Devan et al. studied the relationship between LBP and physical activity [26]. Their findings create further ambiguity in understanding disability as it relates to LBP in LEAs. That is, they found no association between physical activity in LEAs with or without LBP. One additional systematic review concluded that the majority of LEAs with LBP report minimal to no impact on social, recreational or work activities [20]. Conversely, approximately 25% described their LBP as severely interfering with these activities. These are important findings but do not directly relate to the issue of bout frequency of LBP. Ultimately, although the association identified by Hammarlund et al. was significant, the fact that only a single study supports the conclusion is presently insufficient to confidently support the statement at this time [5].

In EES five, association is made between leg length discrepancy and the presence of LBP in persons with LEA. One clinical study used the RMDQ to identify LEAs with LBP and those without [15]. Motion analysis was then used to determine leg length differences during static standing, dynamically during single and double limb support in gait and with either the prosthetic or sound foot leading. This single, high-quality clinical study did not find a relationship between leg length discrepancy and LBP. Conversely, a systematic literature review [28] indicates that leg length discrepancy among lower extremity prosthetic users is among many contributors to LBP. Further, the review states that those using prostheses that are of the same length as the sound limb have significantly fewer pain symptoms compared to those with length asymmetries between the intact and prosthetic limbs. Postural asymmetries reportedly result from these disparities. For instance, leg length differences of 12.5 mm have been associated with as much as 4° of lateral sacral tilt. It has been further reported that only 15% of LEAs use prostheses of equal length to the sound limb, whereas 34% of prosthesis users have prosthetic leg length differences greater than 20 mm, and that in 79% of cases, the prosthesis is the shorter limb. Given this disagreement between a single clinical study [15] and a systematic literature review [28], there is low confidence in the evidence supporting EES five. This statement indicates an association between leg length discrepancy and back pain in persons with lower extremity amputation. One additional clarifying point is that Morgenroth et al. studied LEAs with chronic LBP as opposed to acute onset cases. Thus, it is not currently possible to determine causation of LBP as a result of leg length discrepancy using these findings. Rather, their study is more useful in assisting to determine whether leg length discrepancy has a role in altering symptoms in chronic LBP cases among those with LEA [15].

Empirical evidence statement six is somewhat related to EES five. Though EES five directly addresses leg length discrepancy, EES six indicates that postural asymmetries and postural control issues are associated with LBP in patients with LEA. Gailey et al. report that persons with LEA tend to stand with increased sway and with increased weight bearing on the sound limb and that this may be

related to the lack of proprioception from the prosthesis [28]. Postural abnormalities observed in those with LEA are numerous including coronal and sagittal compensatory pelvic tilt, increased lumbar lordosis, involved-side hip flexion contracture, lateral trunk asymmetry and more [28]. Morgenroth et al. state that LBP is a common secondary disabling condition affecting TFA and that it is common clinical practice to assess for and correct postural asymmetry in the form of leg length discrepancy [15]. In their sample of subjects with longstanding transfemoral amputation and moderate, persistent LBP, leg length, discrepancies were not different relative to a similar population without LBP. Morgenroth et al. concluded that in longstanding transfemoral amputees with chronic symptoms, their LBP was unlikely to be related to their postural asymmetry [15]. Confidence in EES six is low given support from a systematic review but a lack of support from one clinical study, both of moderate quality.

Relative to EES seven, there is limited (n = 3 studies) evidence reporting alterations in trunk, spinal, and pelvic motions among persons with LEA with LBP (EES seven). Although LBP is multifactorial, repeated exposures to altered trunk-pelvic motions is a purported risk factor for the onset or recurrence of LBP secondary to LEA [7,9,30–33]. The presence of LBP among persons with (transfemoral) LEA is associated with larger axial rotations of the lumbar spine [18], more rigid (in-phase) trunk-pelvic coordination strategies [16], and an apparent (albeit underpowered) trend toward a reversal in patterns of trunk-pelvic motion in the sagittal and transverse planes [17]. Such findings begin suggesting linkage of specific trunk/spinal and pelvic kinematic patterns with LBP secondary to LEA. However, the presence and magnitude of LBP has been inconsistently characterized using a variety of approaches, including binary yes/no, visual analog scale (0–10), question(s) within the Prosthesis Evaluation Questionnaire, and the Grade Questionnaire. Thus, there is a clear need for more consistent and comprehensive quantification of LBP in future work. For example, using the NIH task force for chronic LBP questionnaire that aims to classify LBP by its impact (ie, intensity, interference, and physical function), or using a minimal dataset to describe participants and reporting responder analyses in addition to mean outcomes could be useful [34]. Moreover, considerable prior work among non-amputation individuals have identified substantial influences of LBP on trunk and pelvic motions[35–37], begging the question of the relative contributions of LBP and LEA on the observed movement patterns. To that end, additional biomechanical metrics are needed to understand the underlying factors driving the movement patterns.

A single moderate quality study of 39 subjects supports the final EES [38]. This statement indicates that back pain is not affected by use of an osseointegrated prosthesis. Interfacing the prosthesis with a socket has been associated with adverse effects to skin, comfort, and function [23]. For example, skin erosions of varying degrees, pain, and unreliable suspension can all potentially emerge related to socket use [39]. Anchoring the prosthesis to the residual limb via osseointegration purportedly mitigates some of the aforementioned complications. Another issue potentially associated with socket use is that gait pattern alterations due to pain or instability could also lead to LBP. Hagberg et al. surveyed LBP in a single item from the Questionnaire for Persons with Transfemoral Amputation [38] finding that at 2 years following osseointegration, approximately 40% of subjects reported reduced LBP, nearly 40% were unchanged, and nearly 20% reported an increase in their LBP symptoms. Compared with baseline, these differences were not statistically significant. Of note, the authors indicated the small sample size and reliance solely upon subjective outcomes limited the strength of evidence. Findings were also confounded by the fact that prosthetic components were changed throughout the 2-year follow-up period. More research is needed to identify and characterize the relationships between osseointegrated prosthetic use and LBP.

Clinical practice guidelines (CPGs) for primary care management of LBP in the general population usually recommend focused history and examination, limited use of diagnostic imaging, self-care, brief education, nonsteroidal anti-inflammatory drugs, manual therapy, and exercise [40]. The US Department of Defense (DoD) and the US Department of Veterans Affairs (VA) similarly have CPGs for persons with LEA and for those with LBP [41,42]. None of the articles uncovered in this systematic review assessed the appropriateness of these recommendations for patients with LEA suffering from LBP. Whether the LBP CPGs can be applied to LEAs or if modifications in the treatment approach are needed is unknown. Further, the recommendations in the VA/DoD include measuring the intensity of LBP but also to initiate a strengthening program for the upper and lower extremities as well as the core to prevent the development of LBP [42]. These recommendations in the second version of the VA/DoD CPG were forwarded from the original CPG and were largely based in expert opinion. These recommendations remain untested in this population. Future research is needed to clarify clinical decision-making processes for management of LBP in lower extremity amputees.

Limitations

This body of literature only included a single experimental study [38] and a single study with high internal and external validity [15]. The majority of the included studies were observational, of moderate overall quality, and unfunded. Viewed in aggregate, the subjects studied were somewhat heterogeneous with regard to age, LEA etiology, time since LEA, and included both military and civilian sectors; the methodological quality could be improved with standardized reporting in most cases [21,22]. An example

may include more thorough sample descriptions. Additionally, incorporating blinding (ie, raters, statisticians) would also improve internal validity. Further, important factors are missed due to reporting omissions in many cases such as gender, race or ethnic considerations. Finally, this review uncovered three potential etiologies of LBP in LEA, namely, leg length discrepancy (ESS five), postural asymmetries and control issues (ESS six), and altered spinal kinematics. However, a causal relationship between these potential etiologies and LBP in LEA has not been established and requires further research.

Conclusions

Because the majority of this body of evidence was observational instead of experimental, the EESs produced tended to describe factors affecting or that are affected by LBP in persons with LEA. More specifically, the EESs supported observationally have concluded that back pain in LEAs has relationships with the following phenomena: increased experiences, level of LEA, leg length differences, postural issues as well as spinal and pelvic kinematics. With only a single experimental study, the ability to determine optimal therapeutic intervention choices or their effects in managing LEAs who have LBP is greatly limited. Therefore, efficacy of interventions to manage LBP in persons with LEA remains a considerable knowledge gap and an area of future study.

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Abstract

Background: Alterations and asymmetries in trunk motions during activities of daily living are suggested to cause higher spinal loads in persons with unilateral lower limb amputation (LLA). Given the repetitive nature of most activities of daily living, knowledge of the amount of increase in spinal loads among persons with LLA is important for designing interventions aimed at prevention of secondary low back pain due to potential fatigue failure of spinal tissues. The objective of this study was to determine differences in trunk muscle forces and spinal loads between persons with and without LLA when performing a common activity of daily living, sit-to-stand and stand-to-sit tasks.

Methods: Three-dimensional kinematics of the pelvis and thorax, obtained from ten males with unilateral (transfemoral) LLA and 10 male uninjured controls when performing five repetitions of sit-to-stand and stand-to-sit activities, were used within a non-linear finite element model of the spine to estimate trunk muscle forces and resultant spinal loads.

Findings: The peak compression force, medio-lateral (only during stand-to-sit), and antero-posterior shear forces were respectively 348N, 269N, and 217N larger in person with vs. without LLA. Persons with LLA also experienced on average 171N and 53N larger mean compression force and medio-lateral shear force, respectively.

Interpretation: The spinal loads for both groups were generally smaller than the reported threshold of spinal tissues injury. However, tasks like sit-to-stand and stand-to-sit, with a peak compression force of ~ 2.6kN in persons with LLA, if performed following a highly repetitive activity like walking will impose >50% risk of fatigue failure for spinal tissues.

Keywords: Rising and sitting; Trunk muscle forces; Spinal loads; Low back pain; Limb loss; Biomechanics

Abstract

Background: Repeated exposures to larger lateral trunk-pelvic motion and features of knee joint loading likely influence the onset of low back pain and knee osteoarthritis among persons with lower-limb amputation. Decreased hip abductor strength can also influence frontal plane trunk-pelvic motion and knee moments; however, it is unclear how these are inter-related post-amputation.

Methods: Twenty-four participants with unilateral lower-limb amputation (14 transtibial; 10 transfemoral) and eight uninjured controls walked at 1.3 m/s while full-body biomechanical data were captured. Multiple linear regression and Cohen's f^2 predicted ($P<0.05$) the influences of mediolateral trunk and pelvic ranges of motion, angular accelerations, and bilateral isometric hip abductor strength on peak (intact) knee adduction moment and loading rate.

Findings: There were no group differences in hip strength, peak knee adduction moment or pelvis acceleration ($p>0.06$). The combination of hip strength, and mediolateral trunk and pelvic motion did not predict ($F_{(5,29)}=2.53$, $p=0.06$, adjusted $R^2=0.27$, $f^2=0.08$) peak knee adduction moment. However, the combination of hip strength and trunk and pelvis acceleration predicted knee adduction moment loading rate ($F_{(7,29)}=3.59$, $p=0.008$, adjusted $R^2=0.45$, $f^2=0.25$), with peak trunk acceleration ($\beta=0.72$, $p=0.008$) and intact hip strength ($\beta=0.78$, $p=0.008$) significantly contributing to the model.

Interpretation: These data suggest increased hip abductor strength counteracts increased lateral trunk acceleration, concomitantly influencing the rate at which the ground reaction force vector loads the intact knee joint. Persons with lower-limb amputation perhaps compensate for increased intact limb loading by increasing trunk motion, increasing demand on hip abductors to attenuate this preferential loading.

1. Abstract

Background: Persons with a unilateral, transtibial amputation (TTA) often exhibit abnormal trunk movement deviations during walking relative to uninjured persons. Prior work has shown that kinetics throughout the whole body have potential to contribute to trunk control. The aim of this study was to characterize how gait compensations of persons with a unilateral TTA contribute to altered, angular trunk dynamics at a whole-body level during walking.

Methods: Overground motion capture data were collected for 10 persons with a unilateral TTA and 10 uninjured persons walking at a self-selected speed. An induced acceleration analysis was used to decompose experimentally measured trunk angular accelerations into constituent accelerations caused by actions of all net joint moments in the body.

Findings: Several deviations in joint moments were found to correspond with altered trunk accelerations for the TTA group. The primary finding was that the prosthetic ankle plantarflexor moment and affected limb knee extensor moment imparted different accelerations on the trunk in both the frontal and sagittal planes. Knee-induced differences appeared to a correspond with deficits in knee moment magnitude, while ankle-induced differences appeared associated body postural factors.

Interpretation: Our findings highlighted that maladaptive mechanical compensations throughout the body may contribute to abnormal trunk angular movements in persons with a unilateral TTA. Interventional strategies such as movement training to alter foot placement or adjusting prosthetic device mechanical properties may be a useful supplement to traditional treatment methods to correct faulty trunk motion.

Keywords (max 6)

Human locomotion, below knee amputation, induced acceleration analysis, musculoskeletal modeling, movement re-training

ABSTRACT

Objective: To investigate trunk-pelvic kinematic outcomes with time from initial ambulation with a prosthesis, and amputation level, among persons with unilateral lower limb loss. It was hypothesized the magnitudes of trunk-pelvic range of motion (ROM) will increase and pelvic-trunk coordination will increase (become more out-of-phase) with increasing time of ambulation. Secondarily, persons with more proximal limb loss will initially exhibit less trunk and pelvic ROM, and more in-phase trunk-pelvic coordination.

Design: Inception cohort with up to five repeated biomechanical evaluations during a one-year period (0, 2, 4, 6, and 12 months) after initial ambulation with a prosthesis.

Setting: Biomechanics laboratory within Military Treatment Facility

Participants: Thirty-two males with unilateral lower limb loss (twenty-two with transtibial limb loss and ten with transfemoral limb loss).

Interventions: Not applicable.

Main Outcome Measures: Triplanar trunk-pelvic ROM, and intersegmental coordination (continuous relative phase; CRP), were computed as participants walked overground at a self-selected (~1.30 m/s) and controlled (~1.20 m/s) speed.

Results: With increasing time after initial ambulation, trunk ROM generally decreased, most notably for persons with transfemoral limb loss, while pelvic ROM generally remained consistent. Mean CRP became more out-of-phase over time, and frontal CRP was more in-phase for persons with transfemoral vs. transtibial limb loss.

Conclusions: Temporal relationships in the features of trunk-pelvic motions within the first year of ambulation after limb loss have longer-term implications for the surveillance of LBP onset and recurrence, and may help identify important biomechanical factors in its causation. Future work should therefore continue longitudinal evaluations of trunk-pelvic motions, as well as injury rate and pain level.

Key Words: Extremity Trauma; Extremities; Wounds and Injuries; Biomechanics; Locomotion; Rehabilitation; Torso

Trunk muscle activation patterns during walking among persons with lower limb loss

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Introduction: Persons with vs. without unilateral lower limb amputation (LLA) walk with larger trunk and pelvis motions, presumably to assist with balance and forward progression; however, these potentially play a role in the elevated risk for low back pain (LBP) [1]. Uninjured individuals with vs. without LBP increase lumbar muscle activation when walking, often posited as a means to increase spinal stiffness and stability to avoid pain [2]. While aberrant activation magnitude of trunk musculature during walking is characteristic of persons with LBP, it is unknown whether similar alterations are observed in persons with LLA, and may provide insight into neuromuscular control strategies after LLA. Thus, the objective of this study is to determine trunk muscle activation patterns among persons with LLA during walking.

Methods: Fifteen participants with unilateral LLA [5 transfemoral (TFA; 38.4±6.3yrs, 174.3±4.6cm, 78.0±3.6kg); 10 transtibial (TTA; 33.3±8.4yrs, 178.9±8.1cm, 91.7±15.4kg)] and eleven uninjured controls (CTR; 30.6±8.9yrs, 176.8±8.7cm, 75.1±14.2kg) walked along a 15m walkway at 1.3m/s. Trunk electromyographic (EMG) data were obtained bilaterally from thoracic (TES) and lumbar (LES) erector spinae, high-pass filtered (Butterworth, cut-off frequency=20 Hz), and full-wave rectified. A root mean square envelope was calculated using a 50ms smoothing window. EMG activation magnitudes for each participant were normalized to the ensemble mean amplitude of each stride [3], maximum ("peak") activations (as a percentage of mean activity) extracted during the gait cycle, and activation onset/offsets (relative to % gait cycle) determined via visual inspection. Single-factor ANOVAs ($p<0.05$) assessed the effects of group on peak activation and onset/offset of peak activation, with post hoc t-tests, and Cohen's *d* assessing differences between groups ($p<0.0125$).

Results: While there were no differences in peak TES ($F_{(2,23)}=2.83$, $p=0.08$, eta=0.44) and LES ($F_{(2,23)}=2.87$, $p=0.077$, eta=0.45) between groups; TFA (vs. TTA, CTR) activated TES and LES earlier and maintained activation for longer durations (Figure 1).

Discussion: Earlier onset and delayed offset of trunk musculature among persons with TFA suggest these individuals adopt functional strategies to generate force to advance the affected limb. Among persons with TFA, reduced activations of global musculature, in the presence of larger motions, may thereby increase demands of local musculature to support and control motions of the spine. These patterns potentially elucidate altered neuromuscular control strategies associated with LBP development among persons with TFA, and therefore may guide trunk-specific motor control training paradigms.

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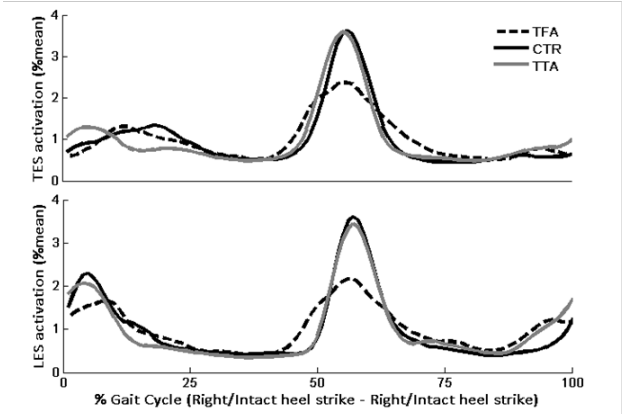

Group	EMG	TES	Effect Size *d*	LES	Effect Size *d*
CTR	Onset	43.7(1.0)*	** 3.50*	92.0(3.4)*^	** 2.81*
TTA	Onset	43.0 (1.0) †		87.5(0.4) †^	*^ 1.85*
TFA	Onset	40.2(1.0)*†	*† 2.80*	84.9(1.1)*†	*† 3.14*
CTR	Peak	3.8 (1.1)*	** 1.24*	3.9 (1.7)*	** 1.15*
TTA	Peak	3.7 (0.8)†		3.5 (0.6)†	
TFA	Peak	2.7 (0.6)*†	*† 1.41*	2.4 (0.7)*†	*† 1.69*
CTR	Offset	68.5(3.8)*^	** 2.52*	71.0(2.3)*	** 2.95*
TTA	Offset	65.5(1.6) †^	*^ 1.04*	71.1(4.8) †	
TFA	Offset	76.7(2.6)*†	*† 5.27*	77.5(2.1)*†	*† 1.73*

Figure 1. A) TES and LES activation as a percentage of the mean activation; B) Group and speed means (standard deviations) and effect sizes (*d*) for post hoc comparisons. *Indicate effect sizes of differences between CTR and TFA. †Indicate effect sizes of differences between TTA and TFA. ^Indicate effect sizes of differences between CTR and TTA.

Relationships among trunk-pelvic motion, hip strength, and knee joint moments during gait among persons with limb loss

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Introduction: Altered gait mechanics in persons with unilateral lower limb amputation (LLA) are associated with increased risk for low back pain (LBP) and knee osteoarthritis (OA) [1,2]. Specifically, repeated exposures to larger lateral trunk-pelvic motion and features of knee joint loading (e.g., rate of the knee adduction moment [KAM]) compared to uninjured controls likely contribute to such conditions over time. Decreased hip abductor strength can also influence frontal plane trunk-pelvic motion and knee moments; however, it is unclear how these are inter-related post-LLA [3]. Here, we report the relationships among features of trunk-pelvic motion, hip abductor strength, and frontal plane joint moment of the intact limb during gait among persons with LLA.

Methods: Fifteen participants with LLA [6 transfemoral (38.7±6.7yrs, 176.0±3.6cm, 80.8±9.8kg) and 9 transtibial (32.7±7.1yrs, 179.3±8.6cm, 92.3±16.7kg)] and six uninjured controls (25.5±4.2yrs, 175.8±9.6cm, 67.7±7.8kg) walked along a 15m walkway at 1.3(±10%) m/s. Full-body biomechanical data were captured using an 18-camera system and six force platforms embedded within the walkway. KAM was calculated by inverse dynamics using Visual3D; KAM_LR represents the slope between 20-80% of the period from minimum to first peak. Trunk and pelvic ranges of motion (ROM) were calculated with respect to the global coordinate system; peak trunk relative to pelvis angles (Trunk_Rel) were also computed. Trunk (TrunkAccel) and pelvis (PelvisAccel) angular accelerations were calculated as the slope of the angular velocity during the same period as KAM_LR. Intact/right limb eccentric hip abductor strength (HIP; (%BW*Ht)) was measured using a hand-held dynamometer. Multiple linear regression and Cohen's *d* were calculated to determine frontal plane predictors of KAM and KAM_LR ($p<0.10$).

Results: After controlling for participant groups, neither HIP, trunk-pelvic ROM, Trunk_Rel, or Trunk/PelvisAccel significantly predicted peak KAM. However, the combination of HIP, PelvisAccel, TrunkAccel, trunk ROM, pelvis ROM, and Trunk_Rel significantly predicted KAM_LR ($F_{(4,13)}=4.077$, $p=0.014$, $R^2=0.69$) with HIP ($p=0.02$, $d=6.49$), PelvisAccel ($p=0.03$, $d=1.35$), and TrunkAccel ($p=0.091$, $d=1.30$) significantly contributing to the model.

Discussion: These data suggest that increased HIP allows for faster correction of contralateral pelvic drop during stance; however, faster and larger trunk rotations among persons with LLA compensate for decreased HIP, concomitantly influencing the rate at which the ground reaction force vector loads the knee joint (i.e., increasing KAM_LR). As such, considering interactions between proximal and distal segments is likely important to comprehensively characterizing mechanistic pathways for LBP and OA in persons with LLA.

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Table 1. Mean±SD for dependent and independent variables

	KAM_LR (%BW*Ht)	HIP (%BW*Ht)	PelvisAccel (deg/s)	TrunkAccel (deg/s)	Pelvis ROM (deg)	Trunk ROM (deg)	Trunk_Rel (deg)
Control (n=6)	37.4±12.8	14.5±2.0	652.0±282.3	217.2±69.7	7.5±2.7	3.5±1.1	6.0±2.9
Transtibial (n=9)	26.9±9.4	9.45±1.7*	527.5±170.7	318.2±118.2	5.2±1.7	7.0±1.6*	7.7±3.4
Transfemoral (n=6)	38.7±9.1	8.3±1.3*	576.1±541.0	554.2±285.0*	6.1±2.3	9.2±2.9*	5.2±4.0

*different from controls, < 0.05

Prevalence and Relationship of Low Back Pain and Psychosocial Factors after Lower Limb Amputation among Wounded Warrior Recovery Project Participants

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Background: Low back pain (LBP) is a common secondary health condition after limb amputation with important implications related to functional capabilities and quality of life. To date, however, the majority of data regarding the prevalence of LBP after amputation have come from studies of older military veterans or civilians with limb loss. As such, there is limited information on the prevalence of LBP after limb amputation in younger Service members from recent conflicts. Additionally, a growing body of evidence suggests that psychosocial factors, such as depression symptoms, significantly influence the experience of LBP in patients without amputation. However, there is currently a dearth of information available regarding the association of psychosocial factors and LBP after limb amputation. The purpose of this study was to assess the prevalence and potential association of LBP with psychosocial factors in Service members with deployment-related lower limb amputations.

Methods: Data on psychosocial factors, including quality of life, post-traumatic stress disorder (PTSD), and depression, comes from the Wounded Warrior Recovery Project (WWRP). The WWRP is an ongoing, web-based, longitudinal study that aims to gather patient-reported outcomes of deployment-related injured Service members. The Military Health System Data Repository was utilized to extract medical record data. Diagnostic codes were queried for at least one instance of coding related to LBP. The population of interest was individuals with deployment-related amputations. Of the current WWRP sample of 4,974 individuals who were injured on deployment between June 2004 and May 2013 and completed a baseline WWRP assessment between 2012 and 2017, 81individuals had lower limb amputations. The majority of the sample of Service members with amputations were male (99%), enlisted (79%), Army (78%), and blast-related injuries (95%). General linear models were utilized to analyze associations between LBP and psychosocial factors, while controlling for injury severity and time since amputation.

Results: In this sample, 58% of individuals with amputations had been diagnosed with LBP by a medical provider; 31% screened positive for PTSD using the PTSD Checklist and 32% screened positive for depression using the Center for Epidemiological Studies Depression Scale. Among individuals with amputations, those with LBP reported lower quality of life (0.415, standard error [*SE*] = .014) compared to those with amputations without LBP (0.470, *SE* = .016) (*B* = .055, *p* =.01; η^2 = .085). Similarly, individuals with amputations and LBP reported higher PTSD scores (40.95, *SE* = 2.11) compared to those without LBP (33.91, *SE* = 2.43) (*B* = -7.039, *p*

=.033; η^2 = .059). There was no significant difference between depression scores in individuals with amputations with (15.17, SE = 1.50) or without LBP (11.51, SE = 1.72) (p =.113).

Conclusions: Presence of LBP after limb amputation appears to be associated with greater PTSD symptoms and lower quality of life. Given the cross-sectional nature of the current data, determination of a cause-and-effect relationship was not possible. Further research is needed to assess the efficacy of addressing psychosocial factors as part of a multi-disciplinary approach for improving pain and function in Service members with amputations and LBP.

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Trunk Muscle Activations, Motions, and Spinal Loads among Persons with Lower Limb Amputation: Influences of Chronic Low Back Pain

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INTRODUCTION: Low back pain (LBP) is a prevalent and costly musculoskeletal disability, particularly within the military, where LBP is among the leading causes of medical visits and lost duty days [1]. Although most LBP remains idiopathic, physical (biomechanical) factors likely contribute more substantially in certain populations (e.g., persons with lower limb amputation; LLA) [2]. During activities of daily living, altered trunk-pelvic motion in persons with (vs. without) LLA impose greater mechanical loads on spinal tissues, and thus have been suggested as a risk factor for the development of LBP [3]. Moreover, in the presence of LBP, further alterations in trunk-pelvic motion have been characterized [4-6]; yet, features of trunk muscle activations underlying these altered motions with LBP and effects on spinal loads remain unclear. The purpose of this study was therefore to evaluate trunk-pelvic motion, muscle activities, and spinal loads among persons with LLA both with and without LBP (and a control group without LBP for reference). Among persons with (vs. without) LLA, we hypothesized trunk muscle activations and corresponding trunk-pelvic motions with vs. without LBP would be associated with larger spinal loads, supporting a pathway wherein repeated exposure to abnormal lumbopelvic mechanics can adversely affect spine health.

METHODS: Eighteen persons with LLA – 8 with LBP ("LLA-P") and 10 without LBP ("LLA-NP") – and 10 uninjured controls without LBP ("CTR-NP") participated in this cross-sectional, IRB-approved study. The LLA-P group reported chronic LBP (n=7 every day or nearly every day in the most recent 6 months; n=1 at least half of days in the most recent 6 months [7]. Mean (standard deviation) pain in the past seven days = 3.8 (1.3). Participants walked overground across a 15m walkway at 1.3m/s while an 18-camera motion capture system (Qualisys, Göteborg, Sweden) tracked (120Hz) trunk and pelvis kinematics. Simultaneously, electromyographic (EMG) activities were sampled (1200Hz) bilaterally at two levels of the erector spinae (T9 [TES] and L2 [LES]). Kinematic data were low-pass filtered (Butterworth, 6 Hz), EMG data were high-pass filtered (Butterworth, 20 Hz), full-wave rectified, and smoothed with a 50ms RMS window. Tri-planar trunk and pelvis angles and angular velocities were calculated in Visual3D (C-motion, Germantown, MD). Global trunk and pelvic ranges of motion (ROM) were determined and trunk-pelvic continuous relative phases (CRP) were calculated in MATLAB (Mathworks, Natick, MA). EMG data were normalized to the signal mean and down-sampled to match kinematic capture rate, and processed bilaterally for individuals with LLA (i.e., on both the intact and affected side) and for the right side of CTR-NP as no differences were observed between left and right sides. Cross-correlations related EMG and trunk and pelvic rotation time series during both intact and affected strides. Finally, trunk-pelvic kinematic data were input to a non-linear finite element model of the spine [8], wherein a heuristic optimization procedure estimated trunk muscle forces and spinal loads by minimizing the sum of squared muscle stress (i.e., the cost function) across 56 muscles. Individual muscle forces were summed across local (i.e., connecting individual lumbar vertebrae to the pelvis) and global muscles (i.e., connecting the thorax/rib cage to the pelvis). 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. Separate one-way repeated-measure ANOVAs assessed the effect of group (LLA-P, LLA-NP, CTR-NP) on ROM, mean CRP, magnitudes

of cross-correlation coefficients (R), and spinal loads (P<0.05). Bonferroni-corrected t-tests assessed pairwise differences (P<0.0167).

RESULTS: Larger frontal plane trunk ROM were observed in both LLA-P and LLA-NP vs. CTR-NP (P<0.008). Transverse plane trunk ROM were larger in LLA-P vs. CTR-NP (P<0.001) but similar to LLA-NP (P=0.032). A main effect was observed in frontal plane mean CRP, with LLA-NP exhibiting smaller (i.e., more in-phase) CRP than CTR-NP (P=0.013). Main effects were observed in the R-values of both intact and affected side LES and sagittal trunk angles. Affected-side LES activations more strongly correlated with sagittal trunk angles in LLA-P and LLA-NP vs. CTR (P<0.003). Intact-side LES more strongly correlated with sagittal trunk angles in LLA-NP vs. CTR-NP (P=0.005), and with transverse trunk angles in LLA-NP vs. LLA-P (P=0.010). R-values of affected -side LES and sagittal pelvis angles were greater in LLA-NP vs. CTR-NP (P=0.011) but similar between LLA-P vs. CTR-NP (P=0.023). R-values of trunk and pelvis angles and TES did not differ between groups. No main effects were observed in peak spinal loads (P>0.078) or global muscle forces (P=0.076). However, peak local muscle forces differed between groups (P=0.017); local muscle forces were larger in CTR-NP vs. both LLA-P (P=0.012) and LLA-NP (P=0.016).

CONCLUSION: Though sagittal plane kinematics were similar between groups, R-values of LES activation patterns and sagittal plane trunk and pelvis angles were larger in both LLA-P and LLA-NP vs. CTR, suggesting an active LES control strategy that may be especially important in the LLA-P group as they tended to walk with more anterior trunk lean than CTR. In the transverse plane, smaller trunk ROM and stronger correlations between intact LES and trunk angles were observed in LLA-NP vs. LLA-P, suggesting LLA-NP are better able to control transverse plane trunk rotations. Despite larger trunk ROM in both the frontal and transverse planes, a lack of differences in spinal loads among LLA-P and LLA-NP vs. CTR are contrary to both our hypotheses (LLA-P ≠ LLA-NP) and prior work [LLA-NP > CTR; 9]. Nevertheless, larger transverse motions with vs. without LBP are consistent with prior work in persons with (transfemoral) LLA [4]. Interestingly, all persons in the LLA-P group had transtibial LLA while those in the LLA-NP group comprised a combination of both transtibial (n=7) and transfemoral (n=3) LLA. In the absence of LBP, alterations in the characteristics of trunk-pelvic motion are typically larger in persons with transfemoral vs. transtibial LLA [2]. It is therefore possible that presence of chronic LBP has concurrently increased trunk-pelvic motions in a group that is otherwise more similar to uninjured CTR. Also, while we identified group differences in local muscle forces, further consideration may be warranted for the model/optimization assumptions regarding muscle recruitment strategies with vs. without LBP [10]. Of note, despite categorization of chronic LBP, participants in the LLA-P group at the time of testing reported mean (standard deviation) numerical pain scores of 2.2 (1.3).

In summary, although prior work has identified larger spinal loads in persons with vs. without LLA, the current (cross-sectional) results do not necessarily support the notion that larger spinal loads during walking influence the persistence of LBP. It is however possible that individuals in the LLA-P group experienced larger spinal loads at some point prior to developing LBP and, thus, future work is needed to longitudinally characterize the temporal relationships in these outcomes with time since LLA to better elucidate the causal relationships.

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Learning Objectives:

1. Describe risk factors for low back pain secondary to LLA

2. Describe differences in trunk-specific outcomes between those with LLA with and without low back pain

3. Describe clinical considerations to mitigate the impact of LBP secondary to LLA

TRUNK MUSCLE FORCES AND SPINAL LOADS WHILE WALKING IN PERSONS WITH LOWER LIMB AMPUTATION BOTH WITH AND WITHOUT CHRONIC LOW BACK PAIN

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INTRODUCTION

Low back pain (LBP) is a common musculoskeletal impairment among persons with lower limb amputation (LLA), capable of substantially reducing longer-term quality of life [1]. During activities of daily living, such as walking, altered trunk-pelvic motion with (vs. without) LLA impose greater mechanical loads on spinal tissues, and thus have been suggested as a risk factor for the development of LBP [2]. Moreover, in the presence of LBP, further alterations in trunk-pelvic motion have been identified [3-5], yet the effects of these altered motions with LBP on spinal loads remain unclear. The purpose of this study was to evaluate the influences of LBP on trunk-pelvic motion and spinal loads among persons with LLA. We hypothesized that there are differences in trunk-pelvic motions that are associated with larger spinal loads between persons with LLA with and without LBP, supporting a pathway wherein repeated exposure to abnormal spine mechanics can adversely affect spine health.

METHODS

Eighteen persons with LLA – 8 with LBP ("LLA-P") and 10 without LBP ("LLA-NP") – and 10 uninjured controls ("CTR"; without LBP) participated (Table 1). The LLA-P group reported chronic LBP (n=7; every day or nearly every day in the most recent 6 months, n=1; at least half of days in the most recent 6 months) [6]. Mean (standard deviation) pain in the past seven days = 3.8 (1.3). Participants walked overground across a 15m walkway at 1.3 m/s, with speed enforced by auditory feedback. An 18-camera motion capture system (Qualisys, Göteborg, Sweden) tracked (120Hz) trunk and pelvis kinematics via 10 reflective markers. Marker trajectories were low-pass filtered (Butterworth, 6Hz).

Table 1. Mean (SD) participant demographics.

	LLA-P	LLA-NP	CTR
Age (yr)	35.1 (8.7)	36.4 (6.8)	29.7 (8.9)
Stature (cm)	177.5 (8.0)	179.3 (5.9)	176.0 (6.3)
Mass (kg)	86.8 (11.5)	91.6 (14.6)	73.2 (13.4)
Time (yr)	5.2 (2.6)	10.5 (3.1)	N/A

Tri-planar (global) trunk and pelvis angles, and pelvis center of mass position were calculated in Visual3D (C-motion, Germantown, MD, USA), time-normalized to stride, and subsequently input to a non-linear finite element model of the spine [8]. A heuristic optimization procedure, controlled via MATLAB (Mathworks, Natick, MA, USA), estimated trunk muscle forces and spinal loads by minimizing the sum of squared muscle stress (i.e., the cost function) across 56 muscles. Individual muscle forces were summed across local (i.e., connecting individual lumbar vertebrae to the pelvis) and global muscles (i.e., connecting the thorax/rib cage to the pelvis). 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. Trunk ranges of motion (ROM) were also determined. Separate one-way repeated-measure ANOVAs assessed the effect of group (LLA-P, LLA-NP, CTR) on all outcomes (P<0.05). Bonferroni-corrected t-tests (P<0.0167) assessed pairwise differences when main effects were observed.

RESULTS AND DISCUSSION

Main effects were observed in frontal (P=0.003) and transverse (P<0.001) plane trunk ROM (Table

1). In the frontal plane, trunk ROM were larger in LLA-P (P=0.001) and LLA-NP (P=0.011) vs. CTR. In the transverse plane, trunk ROM were larger in LLA-P versus both LLA-NP (P=0.015) and CTR (P<0.001), but were similar between LLA-NP versus CTR (P=0.022). No main effects were observed in peak spinal loads (P>0.078) or global muscle forces (P=0.076; Table 1). However, peak local muscle forces differed between groups (P=0.017); local muscle forces were larger in CTR vs. both LLA-P (P=0.012) and LLA-NP (P=0.016).

Despite larger trunk ROM in both the frontal and transverse planes, a lack of differences in spinal loads among LLA-P and LLA-NP vs. CTR are contrary to both our hypotheses (LLA-P ≠ LLA-NP) and prior work [LLA-NP > CTR; 8]. Nevertheless, larger transverse motions with vs. without LBP are consistent with prior work in persons with (transfemoral) LLA [4]. Interestingly, all persons in the LLA-P group had transtibial LLA while those in the LLA-NP group comprised a combination of both transtibial (n=7) and transfemoral (n=3) LLA. In the absence of LBP, alterations in the characteristics of trunk-pelvic motion are typically larger in persons with transfemoral vs. transtibial LLA [2]. It is therefore possible that presence of chronic LBP has concurrently increased trunk-pelvic motions in a group that is otherwise more similar to uninjured CTR. Also, while we identified group differences in local muscle forces, further consideration may be warranted for the model/optimization assumptions regarding muscle recruitment strategies with vs. without LBP [9]. Of note, despite categorization of chronic LBP, participants in the LLA-P group at the time of testing reported mean (standard deviation) numerical pain scores of 2.2 (1.3).

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 individuals in the LLA-P group experienced larger spinal loads at some point prior to developing LBP and, thus, future work is needed to longitudinally characterize the temporal relationships in these outcomes with time since LLA to better elucidate the causal relationships.

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Table 1: Mean (SD) peak anteroposterior (AP) and mediolateral (ML) shear forces, compression forces, and global and local muscle forces, and tri-planar trunk ranges of motion (ROM) for LLA-P, LLA-NP, and CTR. * indicate statistically different than CTR , # indicate statistically different from LLA-NP.

	Spinal Loads (N/kg)			**Muscle Forces (N/kg)**		**Trunk ROM (°)**		
	AP Shear	ML Shear	Compression	Global	Local	Sagittal	Frontal	Transverse
LLA-P	5.4 (1.6)	8.7 (2.1)	22.3 (3.5)	11.0 (2.0)	8.6 (0.9)*	2.9 (1.1)	7.4 (1.5)*	9.5 (1.9)*#
LLA-NP	5.3 (3.4)	10.4 (5.8)	23.1 (3.6)	10.9 (2.6)	8.8 (1.0)*	2.8 (0.8)	6.3 (3.0)*	7.2 (2.3)
CTR	4.4 (0.8)	6.3 (2.3)	22.5 (2.8)	13.5 (3.1)	10.6 (2.3)	2.2 (0.7)	3.6 (1.6)	5.2 (1.2)

PATTERNS OF ERECTOR SPINAE ACTIVATION AND TRUNK-PELVIS KINEMATICS IN PERSONS WITH LOWER LIMB AMPUTATION: INFLUENCES OF LOW BACK PAIN

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INTRODUCTION

A higher prevalence of low back pain (LBP) is reported in persons with lower limb amputation (LLA) vs. uninjured individuals; moreover, persons with LLA often report that LBP negatively impacts quality of life [1]. In uninjured persons, movement impairments at the trunk and pelvis during gait, as well as altered trunk muscle activities, have been associated with increased risk for LBP [2]. However, there has yet to be a comprehensive study which examines both trunk kinematic and electromyographic (EMG) data while walking in persons with LLA, and more specifically, compares the influences of spine health (i.e., presence/severity of LBP). Therefore, the purpose of this study is to examine the relationship between patterns of trunk/pelvis kinematics and trunk muscle activations while walking, specifically comparing individuals with LLA with and without LBP.

METHODS

Seventeen persons with traumatic unilateral LLA – 9 with LBP (BP; 8 transtibial (TT), 1 transfemoral (TF), mean±standard deviation age (yrs): 34.7±7.8, stature (cm): 176.8±7.8, mass (kg): 85.4±11.6, and time since injury (yrs): 5.1±2.5) and 8 without LBP (NP; 5 TT, 3 TF, age: 36.8±7.6, stature: 179.9±5.4, mass: 91.8±15.8, and time since injury: 10.0±3.2) – and 6 uninjured controls (CTR, age: 28.2±9.0, stature: 174.5±4.5, and mass: 68.9±7.8) participated in this study. LBP was characterized via the NIH recommended minimal dataset [3]. Individuals walked across a 15m overground walkway at 1.3 m/s, with speed enforced by auditory feedback. An 18-camera motion capture system (Qualisys, Göteborg, Sweden) tracked (120Hz) trunk and pelvis kinematics via 10 reflective markers. Kinematic data were low-pass filtered (Butterworth, 6 Hz). Tri-planar trunk and pelvis angles and angular velocities were calculated in Visual3D (C-motion, Germantown, MD). Global trunk and pelvic ranges of motion (ROM) were determined and trunk-pelvic continuous relative phases (CRP) were calculated in MATLAB (Mathworks, Natick, MA). EMG data were collected (1200 Hz, Motion Lab Systems, Baton Rouge, LA) bilaterally at two levels of the erector spinae (T9 (TES) and L2 (LES)), high-pass filtered (Butterworth, 20 Hz), and full-wave rectified. A 50ms RMS smoothing window was then applied. EMG data were normalized to the signal mean and down-sampled to match kinematic capture rate. EMG data were processed bilaterally for individuals with LLA (i.e., on both the intact and affected side) and for the right side of CTR as no differences were observed between left and right sides. Both kinematic and EMG data were time-normalized to stride. Cross-correlations related EMG and trunk and pelvic rotation time series during both intact and affected strides. Separate one-way repeated measures ANOVAs assessed the effect of group (CTR, BP, NP) on ROM, mean CRP, and magnitudes of cross-correlation coefficients (R), with significance concluded at $P<0.05$. Bonferroni-corrected t-tests assessed pairwise differences ($P<0.0167$).

RESULTS and DISCUSSION

Larger frontal plane trunk ROM were observed in both BP and NP vs. CTR ($P<0.008$). Transverse plane trunk ROM were larger in BP than CTR ($P<0.001$) and tended to be larger than NP ($P=0.032$). A main effect was observed in frontal plane mean CRP, with NP exhibiting smaller (i.e., more in-phase) CRP than CTR ($P=0.013$). Main effects were observed in the R-values of both intact and affected side LES and sagittal trunk angles (Figure 1). Affected-side LES activations more strongly correlated with sagittal trunk angles in BP

and NP vs. CTR (P<0.003). Intact-side LES more strongly correlated with sagittal trunk angles in NP vs. CTR (P=0.005), and with transverse trunk angles in NP vs. BP (P=0.010). R-values of affected -side LES and sagittal pelvis angles were greater in NP vs. CTR (P=0.011) and tended to be larger in BP vs. CTR (P=0.023). R-values of trunk and pelvis angles and TES did not differ between groups.

The observed changes in frontal plane CRP are consistent with prior work and posited to be a trunk stiffening strategy to prevent injury and/or mitigate pain [4]. Though sagittal plane kinematics were similar between groups, R-values of LES activation patterns and sagittal plane trunk and pelvis angles were larger in both BP and NP vs. CTR. This suggests persons with LLA utilize LES to control sagittal plane trunk and pelvis motion during walking. Such an active control strategy may be especially important in the BP group as they tended to walk with more anterior trunk lean than CTR (P=0.023); this anterior shift in center of mass has been associated with an increased risk of falls [5]. While increased LES contributions may compensate for decreases in passive stability, the increased demand on the muscles may contribute to LBP development. NP may minimize this risk by using LES bilaterally to regulate sagittal trunk motions, and distributing the associated demand across both sides. BP, meanwhile, seems to rely more heavily on affected-side LES, which may increase the risk of injury on that side. In the transverse plane, smaller trunk ROM and stronger correlations between intact LES and trunk angles were observed in NP vs. BP. This suggests NP individuals are better able to control transverse plane trunk rotations, likely using LES to limit axial rotations. As increases in trunk and pelvic rotations are associated with increased spinal loads [6], the observed reductions in transverse plane trunk ROM may help mitigate the risk of LBP in the NP group. Thus, interventions training low-back musculature and enhancing trunk postural control strategies in persons with LLA are likely warranted.

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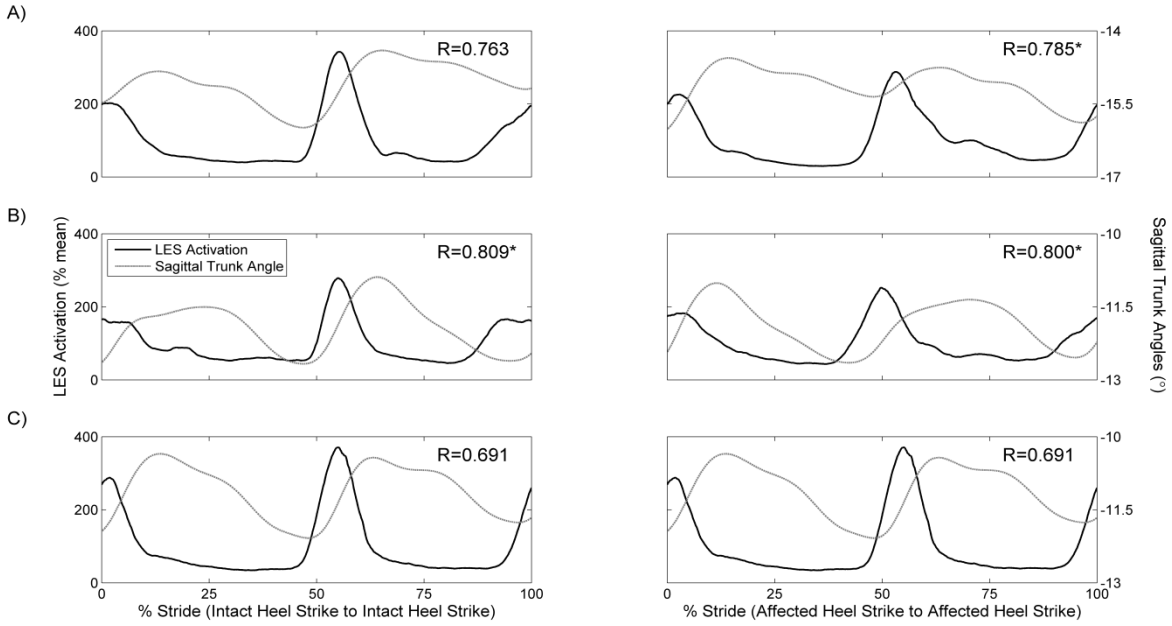

Figure 1: LES activation patterns (black), sagittal trunk angles (gray) and corresponding cross-correlation coefficients (R) for LBP (A), NP (B), and CTR (C) on the intact (left) and affected (right) sides. Asterisks (*) indicate statistically different from CTR.

INFLUENCES OF LOW BACK PAIN ON THE ENERGY CONTRIBUTIONS OF THE HIP AND SPINE DURING GAIT AMONG PERSONS WITH LOWER LIMB LOSS

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INTRODUCTION

Persons with vs. without unilateral lower limb loss (LL) walk with altered trunk-pelvic mechanics that, with repeated exposure, presumably represent a mechanistic pathway for low back pain (LBP) [1,2]. Specifically, persons with LL walk with increased lateral trunk motion over the stance limb, posited as an adaptive strategy to compensate for absent or weak musculature in the lower extremity. Moreover, hip ab/adduction moments may compensate for increased lateral trunk motion over the stance limb, suggesting an altered motor pattern that redistributes energy/power during gait [3]. Among uninjured individuals, impaired hip abductor strength is associated with LBP, suggesting impaired load/energy transfer between the lower extremity and lumbar spine. While persons with vs. without LL demonstrate increased positive phases of joint powers at L5/S1 in the frontal plane, the contributions of the unaffected hip powers to lumbar spine mechanics are unknown. Further, to date, no study has compared frontal plane low back and hip movement strategies among persons with limb loss and varying degrees of disability associated with LBP. Thus, the objective of the current study was to determine the contributions of hip and low back joint powers (L5/S1) to LBP-related disability among persons with limb loss, hypothesizing that persons with greater LBP disability will demonstrate larger power generation through the unaffected hip and low back.

METHODS

Nineteen persons with traumatic unilateral lower LL (n = 7 transfemoral, n = 12 transtibial; mean ± standard deviation age: 31.9 ± 12.5 yrs, stature: 1.8 ± 0.1 m, body mass: 89.1 ± 13.7 kg, and time since injury: 8.6 ± 7.0 yrs) participated in this cross-sectional study after providing written informed consent to study procedures approved by the local IRB. Acute LBP was characterized using a Visual Analog Scale. The presence of chronic LBP was determined via self-report using the Oswestry Disability Index (ODI; "I have 'chronic pain' or pain that has bothered me for 3 months or more"), and was further quantified using ODI percent disability. Participants walked at 1.3 m/s (±10%) along a 15m walkway with full-body biomechanical data captured using an 18-camera system (Qualisys, Göteborg, Sweden) and six force platforms (AMTI, Watertown, MA) embedded within the walkway. Marker positions and ground reaction forces (GRF) were smoothed using a fourth-order dual-pass Butterworth filter with cutoff frequencies of 6 Hz and 45 Hz, respectively. L5/S1 and hip joint powers were calculated as the product of joint moment and angular velocity using 6DOF inverse dynamics in Visual3D (C-motion, Germantown, MD) and normalized to body mass. Positive/negative work at the L5/S1 and hip joints, calculated as the total areas under the joint power curves, respectively indicate mechanical energy generation/absorption.

Multiple regression was used to determine the influences of frontal plane L5/S1 and hip joint powers on ODI percent disability among persons with LL. Independent t-tests were used to determine differences in work between persons with and without chronic LBP at L5/S1 and hip joints throughout the gait cycle ($P < 0.05$).

RESULTS AND DISCUSSION

After controlling for time since amputation and stride width, the total positive and negative powers for both L5/S1 and hip joints significantly predicted LBP-related disability ($F_{(6,18)} = 5.11$, $P = 0.008$), with all

but positive hip power significantly contributing to the prediction. Total positive and negative work through the unaffected hip and low back explain 58% of the variance in the model. While there were no significant ($P > 0.11$) differences in the total work at L5/S1 or hip joints between persons with LL whom identified themselves as having chronic LBP (acute pain = 1.4 ± 1.7, ODI percent disability = 26.5 ± 24.7) and those who did not (acute pain = 0.1 ± 0.3, ODI percent disability = 11.3 ± 12.4), there were distinct differences in joint power waveform characteristics throughout the gait cycle. At L5/S1, persons with LL and chronic LBP demonstrate greater energy absorption during loading response, whereas those without LBP demonstrate greater energy absorption just prior to toe-off (Figure 1). Persons with LL and chronic LBP walk with greater trunk motion during early stance yet demonstrate larger energy absorption at the hip. Although not reported here, this is likely the result of greater hip joint angular velocity, counteracting larger trunk lateral flexion and contralateral pelvic drop during early stance, as a means to maintain mediolateral balance. Such a hip dominant strategy could also have implications for the increased joint loading and prevalence of hip osteoarthritis among persons with LL [5]. The two distinct negative power phases at L5/S1 among persons with LL without chronic LBP are similar to previous reports [4]; in contrast, persons with LL and chronic LBP demonstrate a smaller L5/S1 negative peak power at toe-off that is coupled with a larger positive peak power at the hip. The larger power generation at the hip suggests an active hip strategy to control the mediolateral movement of the center of mass as it moves from peak lateral flexion over the stance limb towards the subsequent heel-strike of the affected limb. Thus, persons with limb loss and LBP may adopt a compensatory strategy to avoid pain and/or to account for impaired neuromuscular control of the trunk. Future research should focus on developing interventions geared towards improving neuromuscular control strategies of the trunk-pelvic-hip complex, thereby reducing possible mechanisms of LBP-related disability.

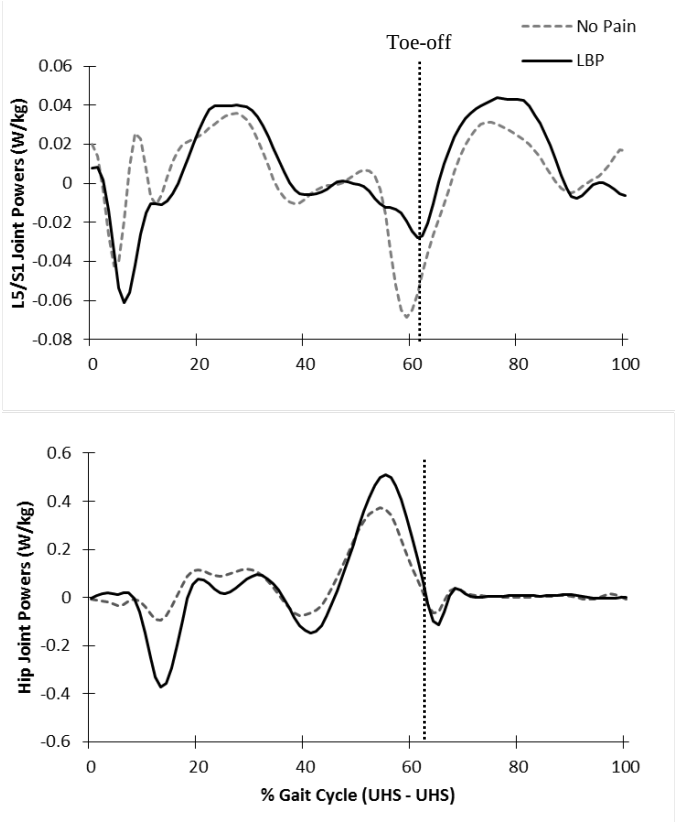

Figure 1: L5/S1 (top) and unaffected hip joint (bottom) powers during the gait cycle (unaffected heel strike (UHS) to unaffected heel strike (UHS)) among persons with limb loss with vs. without self-identified chronic LBP.

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Toward Optimizing Long-term Health after Limb Loss: Comprehensive Evaluations of Secondary Health Conditions

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Background: Extremity trauma, including limb loss, is commonly associated with an increased prevalence of and risk for developing secondary health conditions (e.g., low back pain and osteoarthritis) [1,2]. While the underlying etiologies of these disorders after limb loss remain unclear, there is growing support for the biopsychosocial model toward identifying the multifactorial contributors to their onset and progression [3,4]. Importantly, such an approach requires comprehensive and concurrent evaluation of several domains to effectively characterize risk. The objective of this meta-analysis is therefore to explore and describe relationships among biological and psychosocial outcomes associated with low back pain and (contralateral) knee joint health among individuals with unilateral lower limb loss.

Design/Methods: Eighteen males with traumatic, unilateral lower limb loss (10 transtibial, 8 transfemoral) completed a comprehensive evaluation consisting of biomechanical, biochemical, and psycho-social assessments. Estimates of mechanical loads at the contralateral knee (peak adduction moment) and low back (peak L5/S1 lateral moment) were calculated from full-body biomechanical data collected as participants walked along a 15m walkway. Biochemical data were obtained via blood draws to quantify serum levels of hyaluronan, stromelysin-1, and cartilage oligomeric matrix protein by ELISA as markers of cartilage degradation. Psychosocial outcomes were also obtained using validated, patient-administered instruments for fear of movement, pain catastrophizing, anxiety, and depression. These outcomes were collectively associated with low back pain disability [5] and knee-related quality of life [6] scores via regression analyses, controlling for level of amputation and time since injury (mean [SD] = 128 [86] months). All participants provided informed consent to procedures approved by the Walter Reed National Military Medical Center Institutional Review Board.

Results: With regard to low back pain disability (mean [SD]= 11.5 [18.0]), the collective suite of biopsychosocial outcomes accounted for 61.1% of variance within the model (vs. 13.5% when only controlling for level of amputation or time since injury exclusively); greater fear of movement was a significant predictor (p=0.016) of low back pain disability. With regard to knee-related quality of life (mean [SD]= 82.9 [17.5]), the comprehensive model accounted for 62.3% of variance (vs. 31.5%); greater anxiety/depression was a significant predictor (p=0.042) of lower knee-related quality of life.

Conclusions: Although risk for pain and joint degeneration within the low back and contralateral knee after unilateral limb loss is largely theorized as a biological/mechanical process [e.g., 7,8], psychosocial factors most influenced these outcomes in the current (cross-sectional) dataset. Additional participants and follow-up evaluations will be important to characterize biopsychosocial correlates (and their relative timing with respect to onset) of these secondary health conditions. Given the relatively young age of Service Members with extremity trauma and thus potential for cumulative, lifelong disability, such an approach is critical for optimizing long-term outcomes and quality of life.

References: [1] Gailey et al., 2008 *J Rehabil Res & Devel* **[2]** Butowicz et al., 2017 *Adv Wound Care* **[3]** Farrokhi et al., 2017 *Medical Hypotheses* **[4]** Farrokhi et al., 2016 *Military Medicine* **[5]** Fairbank et al., 1980 *Physiotherapy* **[6]** Roos et al., 1999 *Scan J Rheumatology* **[7]** Miller et al., 2017 *Peer J* **[8]** Hendershot et al., 2014 *Clin Biomech*

TRUNK MUSCLE FORCES AND SPINAL LOADS DURING SIT-TO-STAND AND STAND-TO-SIT ACTIVITIES: DIFFERENCES BETWEEN PERSONS WITH AND WITHOUT UNILATERAL TRANSFEMORAL AMPUTATION

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Low back pain (LBP) is a significant secondary health problem in persons with unilateral lower limb amputation. In particular, persons with versus without transfemoral amputation (TFA) often adopt different trunk postures/motions when performing activities of daily living to overcome the physical limitation(s) imposed by amputation. Such differences in trunk postures/motions, if associated with even moderate increases in spinal loads across all activities of daily living, can lead to LBP via cumulative damages in spinal tissues. The objective of this study was to compare spinal loads between persons with (n=10) and without (n=10) TFA when performing sit-to-stand and stand-to-sit activities. A non-linear finite element model of the lumbar spine and trunk muscles, adjusted for participant height and weight, was used to calculate trunk muscle forces and the resultant spinal loads. Model inputs were kinematics of thorax and pelvis measured when participants performed sit-to-stand and stand-to-sit activities. Forces within superficial muscles (attached between pelvis and thorax spine) were 145 N larger* in person with versus without TFA, while forces within deeper muscles (attached between pelvis and lumbar spine) were 57 N larger during stand-to-sit versus sit-to-stand. The resultant mean and peak values of compression force at L5-S1 were respectively 171 N (~12%) and 348 N (~16%) larger in persons with TFA. The maximum value of anterior-posterior shear force at L5-S1 was also 217 N (~24%) larger in persons with TFA. Finally, in persons with TFA the mean and maximum values of lateral shear force at L5-S1 were respectively 68 N (~92%) and 215 N (~81%) larger during stand-to-sit versus sit-to-stand. The peak value of shear force experienced at L5-S1 (~1.1 kN) among persons with TFA during sit-to-stand was within the reported range of threshold of injury (i.e., 1-2 kN) for lumbar spine motion segments. Considering we have recently reported persons with versus without TFA experience larger spinal loads during walking, characterization of these loads during (other) activities of daily living further highlights their potential role in LBP after TFA, and may assist with the development of trunk-specific movement retraining or other preventative therapies.

*$p<0.05$ in all reported results