

MODELING LOW BACK BIOMECHANICS IN PEOPLE WITH A  
TRANSTIBIAL AMPUTATION

by

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## ABSTRACT

Low back pain is a debilitating and costly condition that affects the majority of people in their lifetime. Some populations, like people with a transtibial amputation, have a higher risk for developing low back pain, which may be explained by altered biomechanical loading. People with a transtibial amputation have altered movement strategies during walking that lead to changes in low back loading, but little is known about their low back biomechanics during the sit-to-stand motion. A musculoskeletal model with detail of the lumbar spine was developed and validated during trunk range of motion trials. This model was used to analyze eight people with a transtibial amputation and eight people without an amputation. People with an amputation had greater trunk angles compared to people without an amputation, which was associated with greater low back compressive loads. Identifying this movement strategy has potential to guide interventions aimed at reducing low back pain.

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## CHAPTER 1:

### GENERAL INTRODUCTION

Low back pain (LBP) is a condition that affects the large majority of people in their lifetime. Many people suffer from chronic LBP, which can be debilitating and expensive. \$86 billion is spent annually on spinal problems by Americans, and LBP remains one of the most common reasons for physician's visits. There are many suggested causes of LBP and a source of pain is typically difficult to identify via imaging, but intervertebral disc and facet joint damage are considered to be common mechanical sources of LBP. Intervertebral discs may rupture or tear under repetitive or large loading, and facet joints can be susceptible to osteoarthritis. Certain populations, such as people with a transtibial amputation (TTA), are more likely to develop LBP compared to people without an amputation. People with TTA have altered movement strategies during a variety of tasks, which can lead to different low back loads and muscle activity compared to able-bodied people. However, little is known about the low back biomechanics of people with a TTA during the sit-to-stand motion, which is a task that they will perform about 50 times/day. *In vivo* low back loads during the sit-to-stand motion may exceed those of most common tasks, such as walking or running. In addition, the lumbar region of the spine has a greater range of motion compared to other regions of the spine during sit-to-stand. Musculoskeletal modeling techniques have indicated that people with TTA have greater L4-L5 joint contact forces and asymmetric low back muscle activity during walking. This research investigated low back biomechanics in people with and without TTA during the sit-to-stand motion. The

results of this work have identified movement strategies that may help explain the greater risk for LBP development in people with TTA.

A literature review covering low back pain, lower limb amputation, the sit-to-stand motion, and methods in biomechanics follows in Chapter 2. This research encompasses two studies: 1) the development of a musculoskeletal model including detail of the lumbar spine with validation of L4-L5 loading predictions (Chapter 3), and 2) comparison of low back kinematics, loading and muscle forces between people with and without a unilateral transtibial amputation during sit-to-stand (Chapter 4). Chapter 5 summarizes overall conclusions of this work and recommendations for future work.

## CHAPTER 2: LITERATURE REVIEW

The following provides a review of literature on low back pain, methods in biomechanics, biomechanics of people with a lower-limb amputation, and the biomechanics of the sit-to-stand movement.

### **2.1 - Low Back Pain**

28% of adults in the United States report chronic low back pain (LBP) (Schiller, 2012), and spinal problems account for approximately \$85.9 billion annual national health expenditures (Martin et al., 2008). LBP is also the fifth most common reason for physician visits (Hart et al., 1995), with up to 84% of people expected to experience LBP in their lifetime (Balagué et al., 2012). Although LBP is the most common form of chronic pain, there are many potential interrelated factors that make it difficult to determine a single cause. These interrelated factors present challenges in prevention and treatment of LBP. Some of these factors include mechanical spinal loading, muscle fatigue, genetic disorders, or anatomical variation. Additional psychosocial factors such as monotonous work or limited possibilities for job development can affect LBP risk. Risk increases in people with less education, and mechanical demands in the workplace are the biggest contributor to this trend (Sterud et al., 2016), but social factors are difficult to assess (Froud et al., 2014). Regardless of the multifactorial nature of LBP, it is important to understand the spinal loading that occurs from the biomechanics of daily living. To inform an investigation of LBP, the following sections

provide an overview of spinal anatomy, the known causes of LBP, and the current treatments for LBP.

### **2.1.1 – Anatomy of the Spine**

A general understanding of the anatomy of the spine is necessary for describing potential biomechanical mechanisms that contribute to LBP. The spine, as part of the musculoskeletal system, consists of bones, muscles, tendons, nerves and other soft tissue, all of which have potential for damage. Although LBP may result in the absence of observable degeneration, spinal damage in the lumbar and sacral regions (L1-L5 & S1-S5, Figure 2.1a) commonly manifests as LBP. Due to the spine's function as a relay in the nervous system, lumbar damage can be perceived as pain elsewhere in the body. Innervation throughout the low back and the lower limbs is transmitted through spinal nerves in the lumbar spine and the sacrum (Figure 2.1b). LBP is generally assumed to be caused by neuromusculoskeletal abnormalities in the lumbosacral region of the spine.

The vertebral bodies of the spine are separated in the posterior articular processes by synovial facet joints and anteriorly by intervertebral discs (IVDs, Figure 2.2a). Intervertebral joints are named after the superior and inferior vertebral bodies, e.g., L4-L5 or L5-S1. Resting between superior and inferior cartilaginous vertebral endplates, IVDs consist of a semifluid center called the nucleus pulposus that allows the disc to deform under pressure. The nucleus pulposus is surrounded by the annulus fibrosus, which consists of 10-20 sheets of collagen fibers (Figure 2.2b; (Bogduk, 2005). IVDs provide vertebral bodies with cushioning and the freedom to articulate. However,

the incompressible nature of the nucleus pulposus may lead to substantial deformations of the IVD.

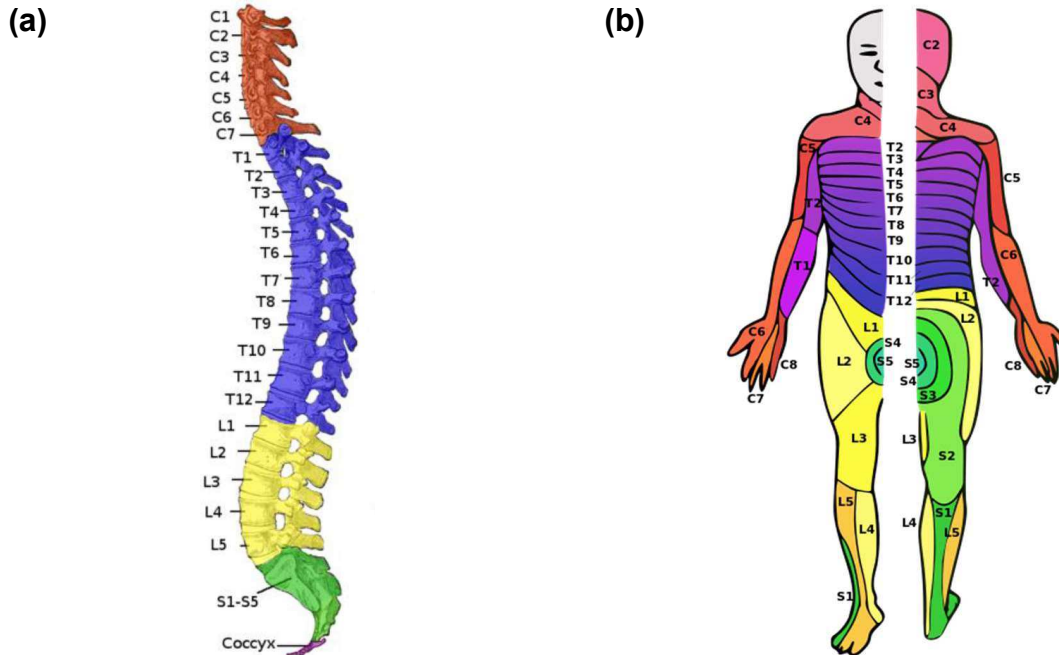


Figure 2.1: (a) Spine anatomy, C: cervical region, T: Thoracic region, L: Lumbar region, S: Sacrum. (b) Dermatome map showing the locations where respective spinal nerves may signal referred pain (Wikimedia Commons).

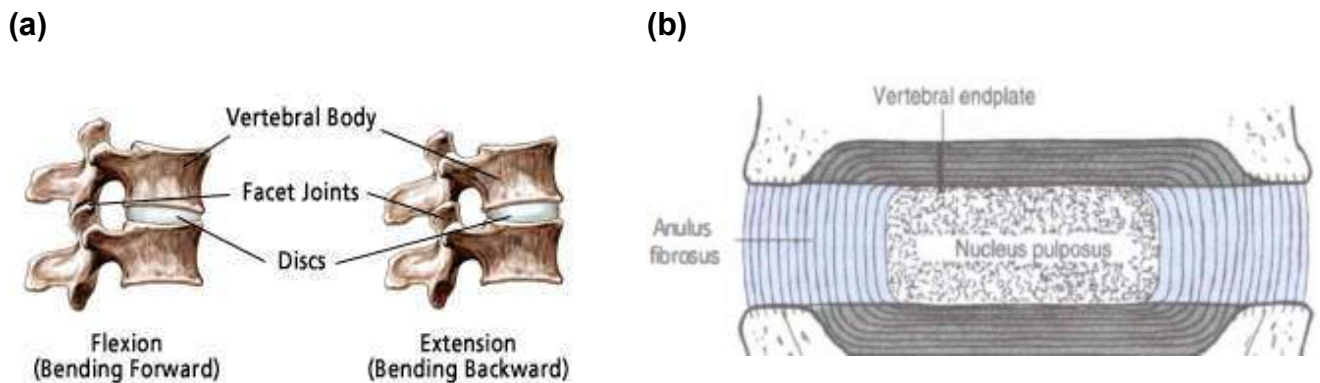


Figure 2.2: (a) Principal mechanical components of the lumbar spine during flexion and extension (Wikimedia Commons). (b) Components of intervertebral discs (Bogduk, 2005).

## 2.1.2 – Causes of Chronic Low Back Pain

The majority of LBP cases are considered to be mechanical in nature, relating to damage or impairment of the spine, muscles, IVDs or nerves of the lumbar spine. Physical damage, especially from lifting, may be the most common cause of mechanical damage (van Dieën et al., 1999). Chronic non-specific LBP leads to long-term pain and disability, but diagnosis is complicated by the difficulty of isolating a specific cause. There are pain receptors in every part of the spine, but the majority of chronic pain is caused by problems in the IVD joints, facet joints, sacroiliac joint or the sacroiliac ligament (Table 2.1). However, damage in each of these areas is closely related due to load sharing, and difficult to identify with medical imaging. Of these areas, IVD damage is the easiest to detect via imaging and it has thus survived the most scientific scrutiny (Bogduk, 2005). IVD degeneration can also lead to excessive facet joint loading and less resistance to axial rotation (An et al., 2006), which may lead to damage and pain development in other parts of the spine.

Table 2.1: Conditions that lead to chronic LBP with a known prevalence (Bogduk, 2005).

Condition	Mechanical Cause?	Chronic LBP prevalence
Iliac crest syndrome	Probable	30-50%
Sacroiliac joint pain	Probable	12% ( $\pm 7\%$ )
Facet joint pain	Probable	<10%, 32% (elderly)
Internal disc disruption	Yes	39% ( $\pm 10\%$ )

Among people with chronic LBP in the US, discogenic pain (pain from a damaged or degenerated IVD) has been reported with a prevalence between 25% and 65% (Finch, 2006), and internal disc disruption (IDD) may be the most well supported and well understood cause of LBP. IDD cannot be diagnosed with standard clinical tests, but computed tomography (CT) along with an injection technique called discography can identify the source of pain. These invasive measurements have been able to show that IDD has a 39% prevalence in LBP patients, most commonly at L4-L5 and L5-S1 (Schwarzer et al., 1995).

IDD is characterized by disruptions of the nuclear matrix of the nucleus pulposus via the presence of radial fissures. The disruptions may worsen progressively as fissures further penetrate the peripheral laminae of the annulus (Figure 2.3a). IDD is not necessarily equivalent to disc degeneration or disc herniation, but it may lead to disc herniation. If radial fissures reach the outer layers of the annulus, compressive loads can cause the nuclear material to herniate (Bogduk, 2005). Herniation may cause pain in the innervated outer annulus, or it may compress spinal nerves (Figure 2.3b).

Disc deterioration can come in multiple forms, and increased levels of deterioration have been linked to increased pain (Vanharanta et al., 1987). The etiology of different disc conditions is still unclear. IDD and other forms of disc deterioration could be caused by mechanical loading through compression, bending, or torsion. There are two clear phenotypes of disc degeneration with possible mechanical causes: vertebral endplate-driven degeneration and annulus-driven degeneration (Adams and Dolan, 2012).

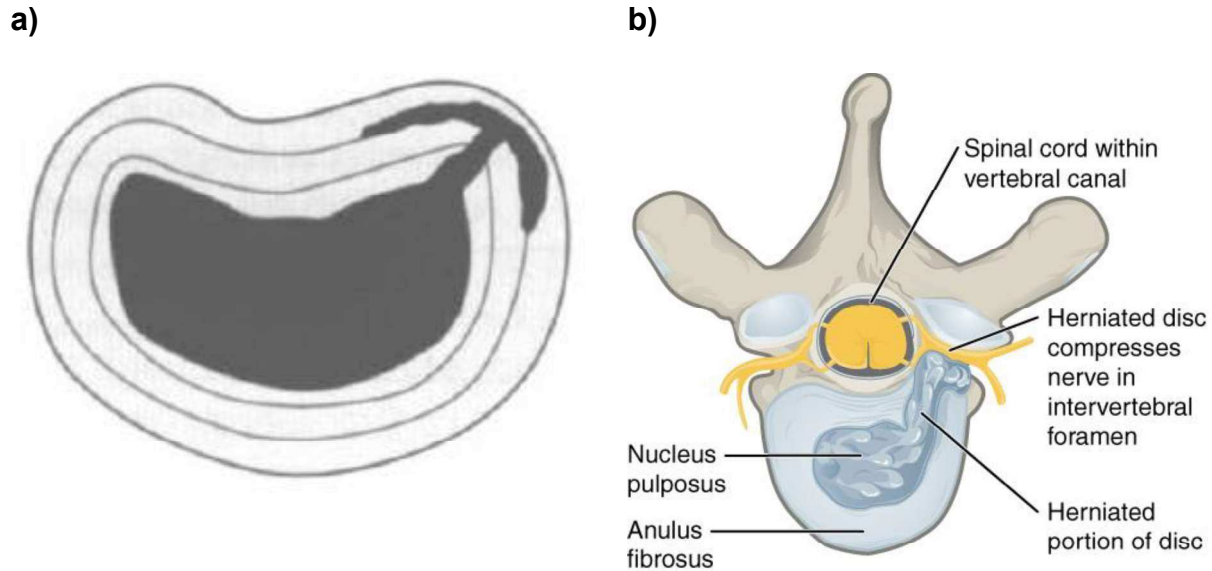


Figure 2.3: (a) Superior view of severe IDD with fissures nearing the outer posterolateral annulus fibrosus (Bogduk, 2005). (b) Superior view of posterolateral herniated lumbar disc with nerve compression (OpenStax).

Endplate-driven degeneration can be caused by fracture of the vertebral endplate from large compressive loading. With repeated loading, fracture could occur at forces as low as 3000 N, but it is more common to occur at around 10,000 N (Brinckmann et al., 1983). In some cases, endplate-driven degeneration may also be influenced by congenital risk factors (Williams et al., 2007).

Annulus-driven degeneration is related to tears or punctures of the annulus fibrosus, which includes IDD. The outer annulus is innervated and a relationship has been observed between LBP and leaking tears, or tears in the peripheral annulus. LBP has also been related to occupational loading, and it is commonly believed that repeated loading will lead to disc degeneration. Annular tears are found in adults of all ages, but leaking tears and tears in the outer annulus increase in prevalence with age (Videman and Nurminen, 2004).

Large compressive loads have been related to vertebral endplate damage, but a hydrostatic model does not fully explain annular loading. IVDs are deformable but they have non-linear stiffness properties with more rigid behavior under higher loading. Maximum strains and disc bulging appear at the posterolateral surface, where degenerative changes usually start. Larger strains and disc bulging are found during flexion or extension, where one side of the disc bulges under buckling and the opposite side is strained tangentially from the shifting nucleus pulposus (Figure 2.4, (Shah et al., 1978)). Maximum shear strains of the IVD are found during flexion and lateral bending (Costi et al., 2007).

IVD degeneration is strongly correlated with greater motion of the spinal segments, especially in axial rotation. Greater axial rotation is associated with greater IVD degeneration both *in vivo* and *in vitro* (Inoue and Espinoza Orias, 2011). Damage from torsional strain has been shown to lead to disc rupture, and it has been suggested that disc degeneration is primarily due to torsional strains (Farfan et al., 1970). Axial rotation of the spine is also primarily resisted by facet joints (Adams and Hutton, 1981), and IVD degeneration increases facet joint loading during axial rotation and lateral bending (Park et al., 2013). IVD degeneration is also related to greater motion of the spinal segments (especially axial rotation), which further increases facet joint loading (An et al., 2006). Although facet joint pain has a less clear pathology than IVD damage, lumbar facet arthrosis has been suggested as a cause of pain, and it is very common, especially in older people. Although it is generally assumed the IVD degeneration leads to facet arthrosis, in some cases, facet joint degeneration may even precede IVD degeneration (Eubanks et al., 2007).

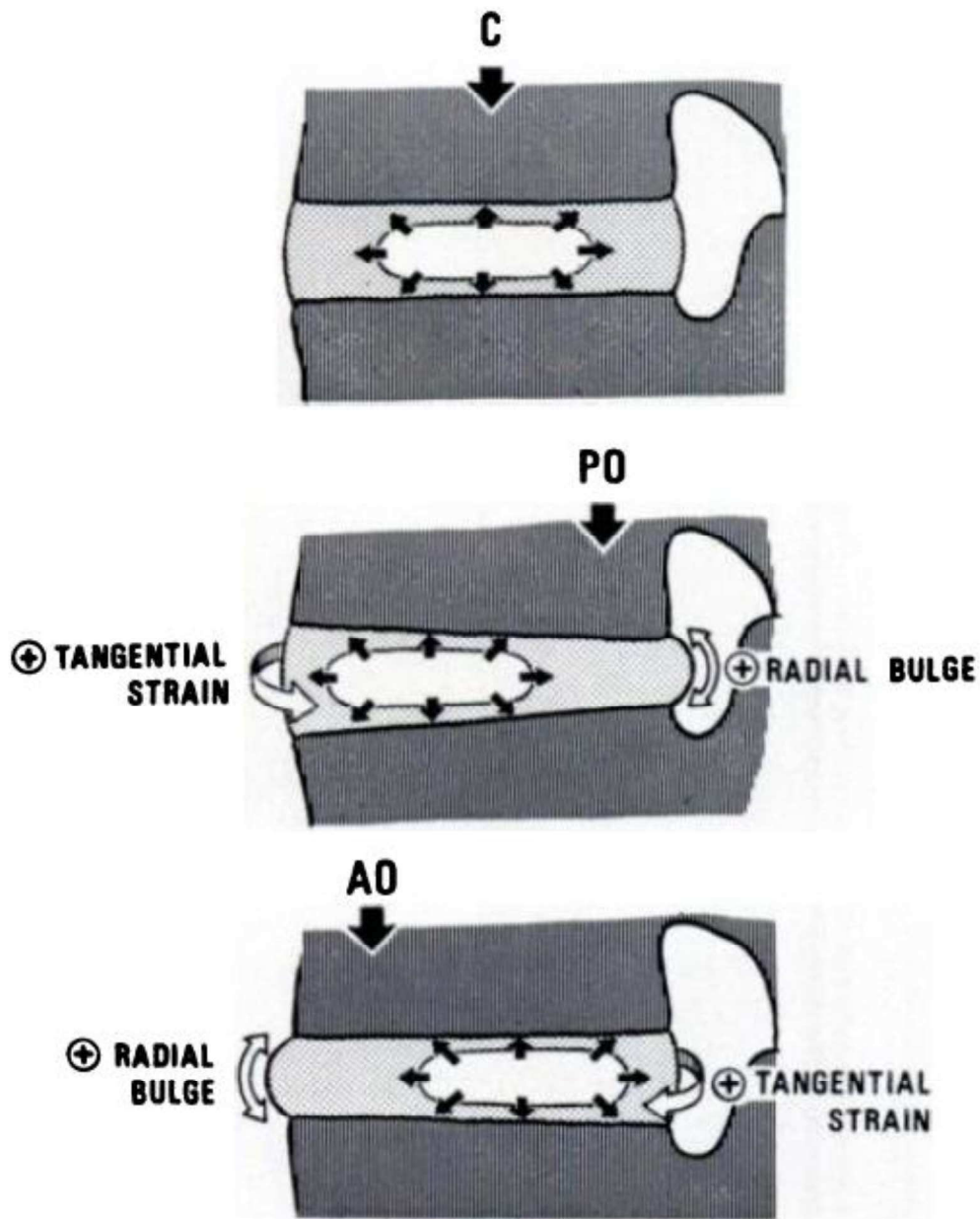


Figure 2.4: Behavior of lumbar IVDs under axial compression (C), extension (PO, posterior offset), and flexion (AO, anterior offset) (Shah et al., 1978).

IVD degeneration remains the most well researched condition that leads to LBP, but damage to the iliosacral ligament, sacroiliac joint or facet joints are also prevalent (Table 2.1), and the same mechanical pathways to IVD damage may be related to damage in these areas. For example, Iliac crest syndrome is a major source of LBP that could be caused by overuse and damage to the iliosacral ligament, which is responsible for restraining the sacroiliac joint and the lumbosacral joint (Pool-Goudzwaard et al., 2001).

### **2.1.3 – Treatment for Low Back Pain**

Assuming that LBP is caused by altered loading, treatment will be most successful if these loading patterns can be addressed and retrained. Disc degeneration leads to functional changes in the IVDs that could worsen pain or prevent recovery. Altered nucleus pulposus pressurization and damage to the annulus fibrosus are related to stress concentrations, decreased disc height, and torsional instability. Surgery can effectively reduce pain (Lurie et al., 2014), but surgical intervention is rare (Andersson, 1999), and its efficacy for treating LBP is questionable (Taher et al., 2012). Biomaterials and new surgical procedures offer potential for restoring spinal function, but non-surgical treatment remains the standard (Iatridis et al., 2013). A combination of physical training and cognitive therapy may even be as effective as surgery (Pedersen and Saltin, 2015). However, there is very little consensus on the appropriate methods for clinical intervention and management of LBP. A wide variety of therapies, educations, manipulations and activities are employed by clinics with varying degrees of effectiveness (Chou et al., 2007).

Primary care trials have shown that more complex and extensive LBP treatments including physical therapy sessions, spinal manipulation or cognitive behavioral therapy are more effective than standard care. However, minimal treatment can be effective for less severe cases of LBP (Hill et al., 2011), and general strengthening exercises are widely adopted and beneficial (Ishak et al., 2016). Core strengthening can be especially effective in reducing pain and improving posture (Ebrahimi et al., 2014), while practicing yoga can aid in recovery by improving physical and mental function (Chang et al., 2016). A wide variety of treatments are used to improve posture and movement capability, which may reduce the risk of re-injury. However, treatments have varying levels of efficacy, and understanding the mechanical risk factors for pain development may provide a basis for developing more targeted treatments.

## **2.2 – Lower Limb Amputation**

In 2005, approximately 1.6 million Americans were living with an amputation, with 185,000 new amputations being performed each year (Ziegler-Graham et al., 2008). This number has been growing, and is expected to exceed 3.2M by the year 2050. Most commonly, amputations result from dysvascular disease or trauma. From 1988 to 1996, transtibial amputation (TTA) and transfemoral amputation (TFA) were the most common major limb amputations, accounting for 287,295 and 266,465 U.S. limb loss cases respectively (Dillingham et al., 2002).

People with a lower limb amputation (LLA) are an important group to study in relation to LBP because up to 71% of people with LLA suffer from LBP (Ephraim et al., 2005). In addition, 60% of people with LLA develop LBP within the first two years of amputation (Kulkarni et al., 2005). Understanding the reason for increased prevalence

in this population has great potential to help improve their quality of life while also increasing understanding of LBP in the general population. The greater LBP prevalence in people with LLA could be attributed to mechanical, rather than biological or social causes, due to the difference between people with LLA and the general population being the mechanical change in lower limb function.

TTA is characterized by the functional loss of ankle plantarflexors. There have been efforts to address the loss of ankle function with the development and prescription of powered ankle-foot prostheses (e.g. (Au and Herr, 2008)), but passive prostheses remain the most commonly prescribed prosthetic devices. People with TTA also have greatly reduced thigh muscle strength in the residual leg (Isakov et al., 1996), and people with TFA have no biological knee function. Overall, LLA has been linked to changes in muscle strength, balance, walking speed and walking symmetry, which may be interrelated with the development of LBP. People who have LLA without LBP tend to have faster walking speeds (Kulkarni et al., 2005). People with TTA are more likely to be mobile one year after amputation than people with TFA (Basu et al., 2008), and they have a lower prevalence of LBP (Kulkarni et al., 2005). Lower-limb deficiencies are a challenge for people with LLA, which may necessitate altered movement strategies that can lead to anatomical changes. For example, people with TFA have psoas hypertrophy on their intact side and psoas atrophy on their prosthetic side. Instability and muscle asymmetries have been suggested as pathways to myofascial LBP development (Kulkarni et al., 2005).

During walking, the primary mechanical compensation for altered ankle and knee function in people with TTA is greater energy absorption and generation in the residual

leg hip joint compared to the intact leg (Soares et al., 2009). During walking, people with TTA favor their intact leg for body propulsion and rely more on their residual and intact leg hip extensors (Silverman et al., 2008) relative to people without an amputation. In addition, there is a notable reduction in ankle and knee moments in the residual leg compared to the intact leg (Sanderson and Martin, 1997). Altered walking mechanics have been observed in both legs, but walking speed and residual knee range of motion improve during rehabilitation from an amputation (Barnett et al., 2009).

Many kinematic and kinetic asymmetries have been attributed to reducing residual leg loading, which can result in larger prosthetic side trunk-pelvis range of motion (Rueda et al., 2013). During walking, greater sagittal and frontal plane trunk-pelvis range of motion in people with TTA has been shown to coincide with increased lumbosacral joint reaction forces and moments in those planes (Hendershot and Wolf, 2014). Larger ranges of lateral bending and axial rotation have also been related to increased and asymmetric low back muscle activity during walking (Yoder et al., 2015). An asymmetric and increased range of trunk motion in people with TTA may result in altered low back loading and muscle activity, which could lead to pain development.

### **2.3 – The Sit-to-Stand Motion**

Rising from a seated position is an important activity of daily living and like other tasks, requires coordination of the trunk and pelvis segments. For example, people with a dysvascular TTA perform the sit-to-stand motion (STS) more than 40 times each day and people with a traumatic TTA perform STS more than 50 times each day (Bussmann et al., 2008, 2004). STS can be described in four phases: flexion momentum,

momentum transfer, extension, and stabilization (Figure 2.5).

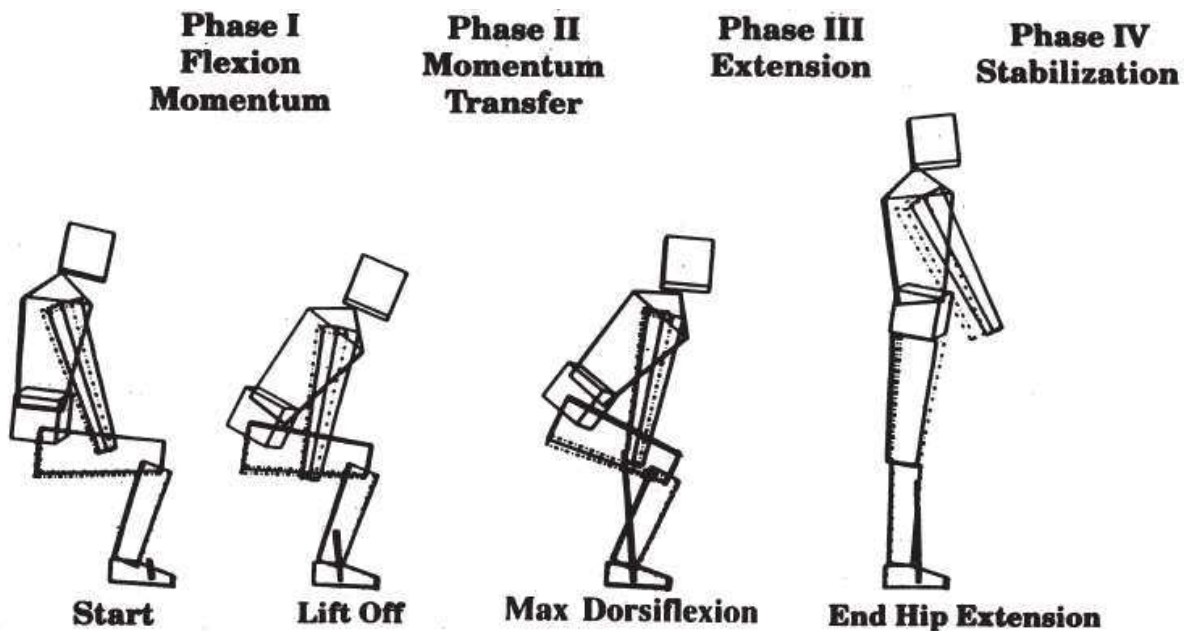


Figure 2.5: The four phases of STS. Phase I, flexion momentum: from the initiation of forward movement until immediately before lifting off from the chair. Phase II, momentum transfer: from lift-off until maximum ankle dorsal flexion. Phase III, extension: from maximum ankle dorsal flexion until full hip, leg and trunk extension. Phase IV, stabilization: from the end of hip extension until stabilization motion is complete (Schenkman et al., 1990).

Standing from a chair can be an indicator of functional mobility. Impaired populations and elderly people can have difficulty successfully standing, and they use a variety of movement strategies to complete this task. There are generally two strategies for standing from a chair: momentum transfer (generating enough momentum during the flexion momentum phase to carry out the motion) and stabilization (repositioning the body's center of mass (CoM) and rising with little momentum). Momentum transfer may reduce effort level but it sacrifices postural control and CoM stability. Often, a combination of both of these strategies is used (Hughes et al., 1994). Greater momentum transfer is often used to overcome the difficulty associated with lower seat

heights, but elderly people may also try to maintain slow stabilization, which can lead to a failure to stand (Hughes and Schenkman, 1996). Due to decreasing muscle strength, the STS task becomes more difficult with age and requires a higher demand from the knee and hip extensor muscles (Samuel et al., 2013), and knee extensor strength may be a limiting factor in standing. Healthy, unimpaired people generally favor a momentum transfer method with greater knee extensor moments, but under weighted conditions, they favor a stabilization method with greater hip extensor and ankle plantar flexor moments (Van der heijden et al., 2009). In addition, greater use of the hip extensors during STS is a primary compensation for knee muscle damage (Spyropoulos et al., 2013).

Hip and trunk kinetics and kinematics during STS are strong indicators of low back loading and LBP risk. Forward flexion, movement time, and hip moments all increase under weighted conditions (Savelberg et al., 2007). Compared to people without pain, people with LBP have decreased sagittal plane low back movement (Christe et al., 2016) and moments when standing, with increased moments in the other planes, especially trunk rotation (Shum et al., 2007).

People with LLA have different STS strategies due to their altered lower limb function. For example, people with TFA have greater trunk-pelvis range of motion in all three planes and greater lumbosacral loads than able-bodied controls during STS (Hendershot and Wolf, 2015). People with TTA favor their intact leg for vertical force generation when standing from a chair (Agrawal et al., 2011; Ferris et al., 2017; Özyürek et al., 2013). This GRF asymmetry is especially notable at liftoff and during momentum transfer and extension (Agrawal et al., 2011), and people with TTA

demonstrate greater postural sway throughout the STS motion (Özyürek et al., 2013). However, low back loads have not been estimated in people with TTA during STS, even though they may be an important factor in further understanding LBP in this population.

## **2.4 - Methods in Biomechanics**

Biomechanical measurements are used to quantify characteristics of movement to understand and treat movement disorders and pain. As applied to LBP, biomechanical analyses have identified dangerous activities such as lifting weights (Wilke et al., 1999), advised lifting strategies (van Dieën et al., 1999), and determined the efficacy of treating LBP with exercise (Ishak et al., 2016). Implantable sensors are capable of providing direct *in vivo* measurements, but they are rare, invasive, and may alter biomechanics. Experimental methods are capable of analyzing kinematics, muscle activity, and external forces in a laboratory, but they do not reveal internal loads. Musculoskeletal modeling provides an estimate of internal loads from experimentally-measured motions. A combination of these approaches should be used to fully understand biomechanical mechanisms that lead to altered loading during movement, which can provide insight into LBP.

### **2.4.1 – Implantable Sensors**

The internal stresses and strains of the human body are strongly related to the health of bones, muscles, musculoskeletal tissues, and orthopedic implants. Although *in vitro* measurements can reveal postural loading behavior, muscle forces largely contribute to loading, and in a few cases, *in vivo* loads have been estimated from strain gauges or pressure transducers implanted in locations such as the hip, shoulder, knee, or spine. Although instrumented implants are more rare and costly than standard

implants, they are able to provide *in vivo* internal loading information in people who have undergone orthopedic procedures. This information can be used predict implant lifespan, improve implant design, improve physical therapy practices, improve the design of orthoses or prostheses, validate modeling estimates and understand the biomechanical loads on the musculoskeletal systems of people with and without orthopedic implants.

Spinal loads during *in vivo* studies have primarily been measured using three types of instrumented devices: internal spinal fixators, vertebral body replacements, and pressure transducers implanted in the intervertebral disc (Figure 2.6).

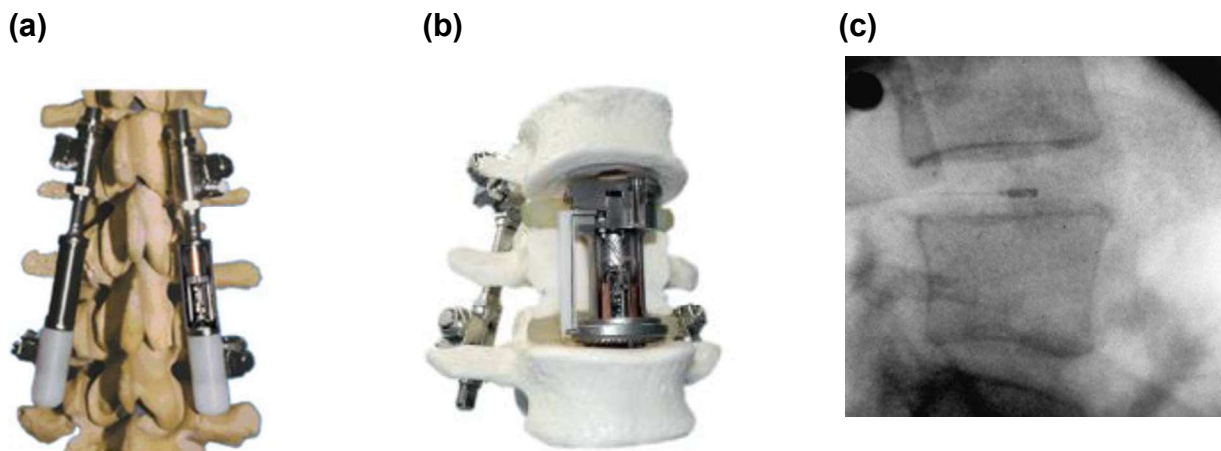


Figure 2.6: Devices for measuring *in vivo* spinal loads: (a) internal spinal fixators, (b) vertebral body replacement (orthoload.com), (c) pressure transducer implanted in the intervertebral disc (Wilke et al., 1999).

Measurements from internal spinal fixators have demonstrated that *in vitro* lumbar spine loads in cadaver samples differ dramatically from *in vivo* loads in patients before and after anterior interbody fusion procedures. The primary difference between cadaver spines and the spines of patients in various postures is the contribution of

muscle forces, which have a large influence on the magnitude and direction of the load. The *in vivo* axial spinal loads are much larger and often different directions than from *in vitro* loads during standing, flexion, extension and lateral bending (Rohlmann et al., 1997).

Posture can have a significant influence on spinal loading, which has been shown with vertebral body replacements (VBRs) that measure partial loads directly between vertebrae *in vivo*. VBRs have been used to quantify variations in spinal load from sitting in a variety of chairs with different postures. In general, shifting the center of mass of the upper body anterior to the lumbar spine increases VBR loads, while leaning against a backrest reduces loads (Rohlmann et al., 2011). Holding dumbbells was shown to cause a larger increase in axial spinal force than the weight of the dumbbells and the effects were amplified by holding the weights away from the body (Zander et al., 2015). Analysis of a database of VBR loads (orthoload.com) support the conclusion that the activities that involve shifting the upper body center of mass (CoM) anteriorly lead to the greatest resultant loads (Rohlmann et al., 2014b). These activities increase the required stabilizing muscle forces in the back, which will cause larger spinal loads. When lifting objects or standing, reducing the upper body's moment arm relative to the lumbar spine by holding objects closer to the CoM or shifting the CoM posteriorly will reduce low back loads. Providing additional support for the upper body and following physiotherapeutic guidelines for movement can also reduce back loading (Rohlmann et al., 2014a).

Pressure sensors within the nucleus pulposus of the intervertebral disc may provide the most direct spinal loading measurements. With pressure sensors, *in vitro*

measurements of the stress distribution in various intervertebral discs have shown that the location of maximum stress can depend on spinal posture and the level of degeneration of the disc. Differing mechanical behavior between the nucleus pulposus and the annulus fibrosus also contributes to the complexity of disc loading and failure risk (McNally and Adams, 1992). The effects of combined loading and pelvic obliquity on lumbar loads were examined using needle pressure sensors in the L4-L5 (between the fourth and fifth lumbar vertebrae) intervertebral disc (IVD) and the L4-L5 facet joints of cadaver spines. Isolated lateral bending led to the greatest intradiscal pressure, followed by spinal flexion. Axial rotation reduced intradiscal pressure from lateral bending, but it greatly increased facet joint loads. A combination of axial rotation and lateral bending led to the greatest loads on the facet joints, and isolated flexion caused the lowest facet joint loads (Popovich et al., 2013).

Studies since the 1960s have shown relatively consistent intradiscal pressures during standing in the area of ~0.3 MPa at L3-L4, and ~0.5 MPa at L4-L5. Bending forward and lifting weights show the largest pressures compared to other activities of daily living (Dreischarf et al., 2016b). Three modern *in vivo* studies have collected intradiscal pressure readings in the L4-L5 IVD during various activities and spinal postures:

1. In a comparison of L4-L5 loads between healthy volunteers and people with LBP or sciatica, average disc pressures across all subjects during forward spinal flexion were more than double the value during upright standing, and the pressure also increased from overextension. Overall disc pressure was found to decrease with increasing levels of disc degeneration (Sato et al., 1999).

2. L4-L5 intradiscal pressure in a healthy 45 year old man during many activities showed that standing from a chair (1.10 MPa) caused greater loading than walking (0.53-0.65 MPa), or jogging (0.35-0.85 MPa), and only weighted activities consistently caused greater loading than STS (Wilke et al., 1999).
3. In three healthy men, intradiscal pressure along with erector spinae and rectus abdominus muscle activities were measured continuously during forward bending. Erector spinae muscles were more active at greater degrees of bending. The resulting muscle forces were part of the reason why disc loads at 30° of bending were 360% as large as those during upright standing. The results were amplified by adding external weight (Takahashi et al., 2006).

Although *in vivo* spinal measurements are rare and invasive, they have demonstrated the importance of posture and muscle activity on spinal loading. Direct measurements also show the variation of low back loads between people, and they are necessary for validating musculoskeletal models.

#### **2.4.2 - Non-invasive Experimental Measures**

The analysis of human biomechanics through non-invasive experimental methods is capable of providing measures of skeletal kinematics, external loads, and muscle excitation patterns during any range of activities. Experimental motion capture utilizes kinematic markers placed on participants to provide an estimation of skeletal positions. Markers are subject to tracking errors from soft tissue artifact during certain motions, but measurements are repeatable, which can help to formulate a standard experimental error (Benoit et al., 2006). The addition of force plates and

electromyographic (EMG) sensors can provide measurement of external loads and muscle excitation patterns during activity.

Quantifying spinal motion has been addressed with various levels of tracking complexity, with the least complex being a standard minimal back marker set (Figure 2.7b). Many marker sets are capable of accurately tracking changes in spinal curvature, but the increased number and density of markers and segments can lead to difficulties during data collection and processing. Consistent tracking of absolute spinal curvature can be difficult, even when tracking each vertebra ((Zemp et al., 2014), Figure 2.7a). An over-defined marker set may lead to excessive processing time, and it still may not be as effective as using fewer markers along with an estimation of neutral postures (Rast et al., 2016).

The choice of a marker set depends on the activity and quantities being examined. While spinal posture is difficult to assess, changes in posture and range of motion can be consistently estimated with motion capture. A comparison of eight standard trunk marker sets during STS and stand-to-sit resulted in similar RoM measurements in the frontal and transverse planes for all marker sets. Sagittal plane RoM differed between marker sets with acromion tracking and those with markers along the spine and low back, which indicates a tendency for sagittal plane shoulder motion during STS (Leardini et al., 2009). A clinical snapshot of lumbar lordosis could vary substantially from what occurs in daily life, and it is important to understand the context and error associated with experimental results (Dreischarf et al., 2016a).

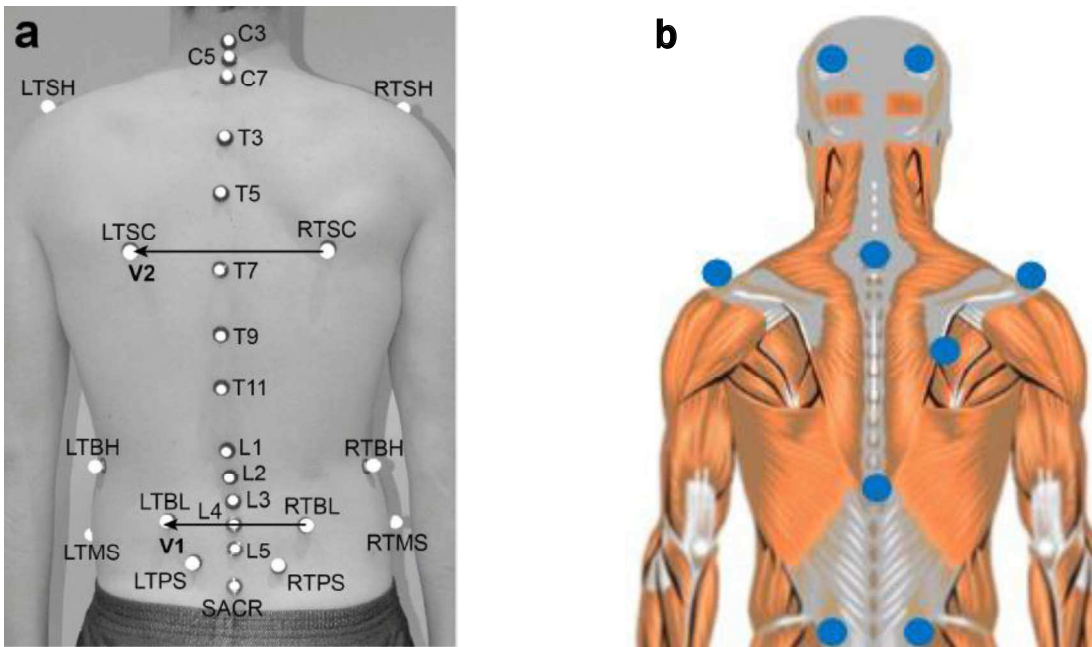


Figure 2.7: (a) A comprehensive spinal marker set with closely spaced markers and tracking of spinal curvature (Zemp et al., 2014). (b) Standard minimal back marker set with minimal tracking of spinal curvature. Markers are placed on seventh cervical vertebra, tenth thoracic vertebra, right back near the scapula, and bilaterally on the back of the head, acromion and the posterior superior iliac spine.

Experimental tracking of the RoM of a five-segment spine during STS has shown that the majority of sagittal plane motion occurs in the lumbar spine, and frontal plane motion is more uniform (Leardini et al., 2011). Two-segment tracking of the lumbar and thoracic regions of the spine has captured highly repeatable kinematics (Fernandes et al., 2016). Tracking of a single segment for the lumbar spine has shown greater transverse plane RoM during walking in people with TFA and LBP compared to people with TFA and no LBP (Morgenroth et al., 2010).

There are inherent errors in kinematic data between trials, days, and observers, but these errors may be acceptable if they are interpreted correctly (Schwartz et al., 2004). Quantifying experimental uncertainty with error estimation can allow for rigorous

clinical interpretations. Inter-session and inter-assessor reliability analyses of lower body joint kinematics in several studies have shown that errors of 2° to 5° are common for most joints. However, certain joint degrees of freedom are more difficult to measure, such as hip rotation, having errors of 6° to 8°. In most cases, errors are considered to be acceptable as long as they are minimized and considered during interpretation (Mcginley et al., 2009).

Regardless of the marker set, it is possible to make reliable comparisons between subject groups when ensuring highly-repeatable results while understanding experimental error. Accurate experimental results are important for further analysis in a modeling framework.

### **2.4.3 – Musculoskeletal Modeling**

Experimentally determining internal loads or muscle forces cannot be accomplished without rare and invasive implantable sensors. However, musculoskeletal modeling and simulation provides an alternative for estimating internal loads. Newton-Euler inverse dynamics assumes rigid body segments to reveal joint moments and powers, but the body is a kinematic chain with hundreds of muscles driving any given motion. One method to determine muscle forces is to use joint moments from inverse dynamics as input into a static optimization framework that minimizes a cost function at each time step, such as muscle fatigue or stress. Often, simulated muscle activations are later compared to experimental EMG readings for validation (Zajac et al., 2002). In other cases, simulations driven by EMG readings have estimated L4-L5 compressive loads exceeding 4 kN during trunk extensor exercises (Callaghan et al., 1998), or 300% body weight at fast walking speeds (Callaghan, 1999).

Inverse dynamics analyses indicate greater lumbosacral joint moments during STS in people with TFA compared to people without an amputation (Hendershot and Wolf, 2015), and greater axial rotation moments in people with LBP compared to people without LBP (Shum et al., 2007). Musculoskeletal models have also been used to estimate altered *in vivo* muscle forces and joint loading in people with an amputation. For example, musculoskeletal modeling has shown greater peak contact forces in the intact knee of people with TTA compared to the residual knee and people without TTA (Silverman and Neptune, 2014). During walking, people with TTA were also found to have greater L4-L5 joint contact forces, and increased and asymmetric muscle forces in the erector spinae and obliques compared to people without TTA (Yoder et al., 2015).

OpenSim is one commonly used musculoskeletal modeling platform capable of performing simulations driven by muscles to match experimental kinematics (Delp et al., 2007). Two upper-body models with articulated segments and detail of the lumbar spine have been developed in OpenSim and made publicly available (Delp et al., 2007). A model with hundreds of muscles and full articulation in the thoracic and lumbar spine has been validated during trunk flexion with intradiscal pressure measurements, VBR implants, and predicted muscle tension from EMG readings (Bruno et al., 2015). Another model has been developed with 238 muscles, articulation of the lumbar spine, and an accurate physiological representation of the 8 muscle groups of the lumbar spine, but it has not been validated with *in vivo* spinal loading data (Christophy et al., 2012). Building upon these trunk models, another model has combined a lower limb model (Anderson and Pandy, 1999; Anderson and Pandy, 2001; Delp et al., 1990; Yamaguchi and Zajac, 1989) with an upper body lumbar spine model (Christophy et al.,

2012) along with actuated arms. This model is intended for running simulations and its predictions of internal loading have not been compared to *in vivo* measurements from the literature (Raabe and Chaudhari, 2016). In addition, a full-body model with 194 muscles and a detailed lumbar spine has shown that the gluteus maximus is the greatest force contributor to the sit-to-stand motion in healthy participants (Caruthers et al., 2016), although this model did not estimate low back loads. A model with a detailed lumbar spine in the commercial AnyBody modeling software applied to one participant (Bassani et al., 2017), and a custom 56 muscle multiscale model with a micro-scale L4-L5 component (Azari et al., 2017) have both been validated for L4-L5 loading during trunk range of motion trials. However, there remains a need for a model with appropriate detail of the lumbar spine, with validated L4-L5 loading predictions, that can be applied to many activities of daily living, such as sit-to-stand.

## **2.5 - Summary**

LBP is a costly and debilitating condition, and the cause, while multifactorial, is often related to altered biomechanics. People with TTA have an increased prevalence of LBP relative to the general population, and have altered biomechanics that likely contribute to this higher prevalence. An understanding of the movement strategies used by people with TTA will provide insight into potential mechanisms of LBP development. The sit-to-stand motion is an important activity of daily living, and it involves large low back loads and trunk-pelvis range of motion. However, little is known about the low back biomechanics of people with TTA during sit-to-stand.

Experimental analysis combined with musculoskeletal modeling and simulation has the potential to reveal risk factors for LBP development in people with a TTA during

sit-to-stand. People with TTA have shown weight distribution asymmetry during sit-to-stand, and people with a transfemoral amputation have utilized greater trunk motion during sit-to-stand, but low back muscle activity and joint contact forces have not been examined in people with an amputation. Discovering sit-to-stand strategies that lead to increased or asymmetric low back muscle activity and loading could identify strategies that should be avoided. A better understanding of the underlying contributions to LBP development could advise physical therapy practices that will prevent and mitigate LBP.

Thus, the purpose of this research is to identify and understand low back biomechanics in people with unilateral TTA during the sit-to-stand movement. Based on prior work during walking, it is expected that people with TTA will have asymmetric and/or greater low back muscle forces and greater low back loads relative to healthy control participants. These quantities are important to identify because they are risk factors for LBP development. Identifying high-risk strategies and the time points during the motion where they occur has the potential to mitigate risk of LBP development through physical therapy, exercise or device interventions.

A validated musculoskeletal model with detail in the low back will be necessary to accurately estimate low back loads and muscle forces. Simulations may reveal risk factors for LBP development such as increases or asymmetries in range of motion, muscle forces, and joint loading in the low back. Trunk shifting and stabilization may lead to some muscles being over-active on one side of the body. Greater range of motion, greater muscle forces, and greater or asymmetric joint loading are all closely related, so it is important to consider each as a risk factor for LBP development.

CHAPTER 3:  
VALIDATION OF LUMBAR SPINE LOADING FROM A MUSCULOSKELETAL MODEL  
INCLUDING THE LOWER LIMBS AND LUMBAR SPINE

A paper submitted to the *Journal of Biomechanics*

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### 3.1 – Abstract

Low back pain is a common condition with a higher prevalence among certain populations, like people with a lower limb amputation. Knowledge of how spinal loading contributes to low back pain can be gained through validated estimates of *in vivo* spinal loading and muscle forces. Motion capture, ground reaction force and electromyographic data were collected from four participants with a unilateral transtibial amputation (three male/one female) and ten participants without an amputation (five male/five female) during trunk-pelvis range of motion trials in flexion/extension, lateral bending and axial

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rotation. A musculoskeletal model with a detailed lumbar spine and 294 muscles was used to predict L4-L5 loading estimates and muscle activations with a static optimization framework. Model estimates of L4-L5 intervertebral joint loading were compared to measured intradiscal pressures from the literature and muscle activations were compared to electromyographic signals. Model loading estimates were only significantly different from experimental measurements during trunk extension, which involves greater facet joint loading that is not measured with intradiscal pressure transducers. Pressure estimates between the model and previous work were not significantly different for flexion, lateral bending or axial rotation. Timing of model-estimated muscle activations compared well with EMG activity of sensors placed on the lumbar paraspinals and upper erector spinae. Predicted spinal loads and trunk-pelvis kinematics were similar between people with and without an amputation. Women had greater lateral bending range of motion compared to men. Validated estimates of low back loading can increase the applicability of musculoskeletal models to clinical diagnosis and treatment.

### **3.2 - Introduction**

Low back pain (LBP) is debilitating (Hoy et al., 2010) and affects people with a lower-limb amputation disproportionately (Ehde et al., 2001; Ephraim et al., 2005; Friel et al., 2005; Kulkarni et al., 2005). Asymmetric kinematics and loading occur following amputation and are linked with the higher prevalence of LBP (Devan et al., 2014). However, knowledge of *in vivo* spinal loading is limited by invasive measurement (Nachemson, 1965; Sato et al., 1999; Takahashi et al., 2006; Wilke et al., 2001). Musculoskeletal modeling techniques can be used to estimate *in vivo* muscle and joint

contact forces (Yoder et al., 2015), which may suggest biomechanical mechanisms that contribute to LBP development (Arendt-Nielsen et al., 1996).

Existing musculoskeletal models of the lower limbs often do not incorporate degrees of freedom at intervertebral joints or muscles in the low back, which are critical to quantify spinal loading. Musculoskeletal models of the lumbar spine have also been developed (Bruno et al., 2015; Christophy et al., 2012; de Zee et al., 2007; Ignasiak et al., 2016), but they lack legs, which are needed in simulating many activities and in modeling populations with musculoskeletal deficits in the legs. Loads from a musculoskeletal model of the lumbar spine have been compared to *in vivo* intradiscal pressures at static poses (Bruno et al., 2015), and from one person during movement (Bassani et al., 2017). Validation of the metrics of interest in a musculoskeletal model applied across many participants is important for increasing confidence in the clinical application of such a model.

The purpose of this study was to develop a whole-body musculoskeletal model with the lumbar spine, and validate predicted lumbar spine loads by comparing to previously reported intradiscal pressure data (Nachemson, 1965; Sato et al., 1999; Wilke et al., 2001). We performed these comparisons for people with and without transtibial amputations (TTA), as people with TTA have differences in musculoskeletal structure of the legs and are susceptible to LBP.

### **3.3 – Methods**

#### **3.3.1 - Musculoskeletal Model**

A musculoskeletal model of the trunk and lower limbs with detail of the lumbar spine was developed in OpenSim 3.3 (simtk.org), including the lower body from (Delp et al., 1990; Yamaguchi et al., 1989; Anderson et al., 1999; Anderson et al., 2001; Carhart et al., 2000), the lumbar spine and torso from (Christophy et al., 2012), muscle strengths from (Bruno et al., 2015), and body mass distributions from (Winter, 2009). 294 Hill-type musculotendon actuators with force-length-velocity properties (Zajac, 1989), 18 body segments (torso, five lumbar vertebrae, sacrum, pelvis as well as the left and right thigh, shank, calcaneus, talus and toes), and 19 degrees of freedom (subtalar and metatarsophalangeal joints locked) with the motion of the five lumbar intervertebral joints defined with a linear function based on overall trunk-pelvis motion (Christophy et al., 2012) were included. Modifications to represent a person with TTA included removing twelve muscles crossing the affected leg ankle joint, reducing the residual shank mass, and shifting residual shank center-of-mass location proximally (Silverman and Neptune, 2012).

#### **3.3.2 - Experimental Protocol**

Four participants with a unilateral TTA of the left leg (3M/1F), five men without TTA, and five women without TTA provided informed consent to participate in the IRB-approved protocol. Participants performed a series of four planar trunk-pelvis range of motion (ROM) trials (Table 3.1). Each trial began with participants standing in a neutral posture with their arms hanging at their sides. They then performed trunk flexion and

extension, lateral bending to the right, and axial rotation to the left. Participants were instructed to maintain a rigid pelvis while using the spine to perform each motion and to pause (~2-5 seconds) at the terminal position of each motion. In addition, participants were asked to pause momentarily approximately halfway through their flexion ROM. Although participants chose terminal position for all motions based on comfort-level, trunk flexion was only performed as a forward lean as in (Nachemson, 1965; Sato et al., 1999; Wilke et al., 2001), and not full flexion of the spine where flexion relaxation occurs as in (Takahashi et al., 2006; Watson et al., 1997).

Marker positions were collected at 120 Hz with a 20-camera motion capture system (Motion Analysis Corp., Santa Rosa, CA) and a full-body marker set with 55 total markers and trunk and pelvis markers placed at C7, T10, xyphoid process, sternal notch, and bilaterally at the acromion, T8, posterior superior iliac spine (PSIS), anterior superior iliac spine (ASIS), iliac crest, and greater trochanter. Surface electromyography (EMG) sensors (1200 Hz, Delsys, Inc., Boston, MA) were placed bilaterally on the upper erector spinae and lumbar paraspinal muscles (Konrad, 2005). Participants performed trials with each foot on a separate force plate (1200 Hz, AMTI Inc., Watertown, MA).

### **3.3.3 - Data Processing & Simulations**

Kinematics and ground reaction forces (GRFs) for the ROM trials were low-pass filtered with a bi-directional 4<sup>th</sup>-order Butterworth filter with cutoff frequencies of 6 Hz and 10 Hz, respectively. An eight-segment model (feet, shanks, thighs, pelvis, and torso) was used to determine an inverse kinematics solution (Lu and O'Connor, 1999) and to scale models to each participant in Visual3D (C-Motion, Inc., Germantown, MD).

A residual reduction algorithm was performed in OpenSim to improve dynamic consistency and reduce residual forces and moments at the pelvis. Then, a static optimization algorithm solved the muscle recruitment problem at each time step by minimizing an objective function of the sum of muscle activations squared. Net joint contact loads were calculated at L4-L5, low-pass filtered at 1 Hz and converted to intradiscal pressure (IDP) (Bruno et al., 2015), with average L4-L5 intervertebral disc cross-sectional areas (CSA) taken from (Sato et al., 1999; Takahashi et al., 2006; Wilke et al., 2001):

$$IDP = \frac{\text{Compressive Force}}{CSA * 0.66}$$

### 3.3.4 - Data Analysis

The sum of muscle activations from the multifidus, quadratus lumborum, longissimus pars lumborum, and iliocostalis pars lumborum in the model was compared to the EMG signal from the lumbar paraspinals for both the left and right sides. Similarly, the sum of the longissimus pars thoracis and iliocostalis pars thoracis was compared to the upper erector spinae. Model activations were low-pass filtered at 2.5 Hz. EMG data were demeaned, band pass filtered between 30 and 500 Hz, rectified, and low-pass filtered at 2.5 Hz (Brereton and McGill, 1998; Drake and Callaghan, 2006). Both model activations and EMG signals were normalized to their peak filtered value for each trial for each participant. Agreement was quantified as the percentage of each trial during which both EMG activity and model muscle activations were either above or below a threshold of 0.5 at the same time. Estimated IDP, normalized to standing, from the male, female, and TTA participant groups were compared to experimentally measured

L4-L5 IDP from previous studies using t-tests ( $\alpha=0.05$ , Table 1). Trunk-pelvis kinematics were also statistically compared between TTA, male, and female groups.

### 3.4 – Results

The only significant difference between experimentally-measured IDP and model-estimated IDP was found at terminal extension (Figure 3.1). Here, the model-estimated IDP was greater for men ( $p = 0.002$ ), women ( $p = 0.031$ ), and participants with TTA ( $p = 0.01$ ) compared to experimentally measured values in previous research (Nachemson, 1965; Sato et al., 1999; Wilke et al., 2001).

Participants generally had greater L4-L5 loading as trunk-pelvis angle increased, similar to (Wilke et al., 2001), especially during flexion and lateral bending (Figure 3.2, page 49). IDP measurements from (Nachemson, 1965; Sato et al., 1999; Wilke et al., 2001) were within the range of loads estimated by the model, except for the terminal period of lateral bending (20-40°). The decrease in disc pressure after 20° of lateral bending as seen in (Wilke et al., 2001) was also seen in model-estimated IDPs, but to a lesser extent. Female participants had significantly larger average peak lateral bending angles than males (36° vs. 25°,  $p = 0.02$ ), whereas their average peak flexion (41° vs. 33°,  $p = 0.3$ ) and axial rotation angles (33° vs. 28°,  $p = 0.24$ ) were not significantly different.

Model muscle activations agreed well with measured EMG activity (Figures 3.3-3.5, pages 50-52). Model and EMG agreement was high for all participant groups during flexion (>70% agreement). The lumbar paraspinal EMG compared well with model muscle activations with only left-side activity for the TTA group during axial rotation and

for the female group during extension having <60% agreement. Generally, the worst agreement was found in the thoracic paraspinals during extension (13-75% agreement).

Table 3.1: Methods from previous L4-L5 intradiscal pressure measurement studies compared with the current simulation study. Mean values for participant characteristics are given as well as the standard deviation across participants in parenthesis.\*Continuous pressure data available. \*\*Only discrete data points are available.

	<b>[1] Wilke et al., 2001*</b>	<b>[2] Sato et al., 1999**</b>	<b>[3] Nachemson, 1965**</b>	<b>Current Study*</b>
<b>L4-L5 Load Estimate Method</b>	Implanted IDP Sensor	Implanted IDP Sensor	Implanted IDP Sensor	Simulation
<b>Participants</b>	<b>1 Male</b> 45 yrs 1.74 m 70 kg	<b>8 Males</b> 25 (2) yrs 1.73 (0.06) m 73 (11) kg	<b>1 Male</b> 43 yrs 1.76 m 74 kg	<b>5 Males w/o TTA</b> 30 (9) yrs, 1.82 (0.06) m, 88 (18)  <b>5 Females w/o TTA</b> 22 (3) yrs, 1.67 (0.06) m, 68 (10) kg  <b>3M/1F w/ TTA</b> 42 (15) yrs, 1.77 (0.09) m, 85 (14) kg
<b>Trunk Motions</b>	Flexion, Extension, Axial Rotation, Lateral Bending	Flexion, Extension	Flexion	Flexion, Extension, Axial Rotation, Lateral Bending

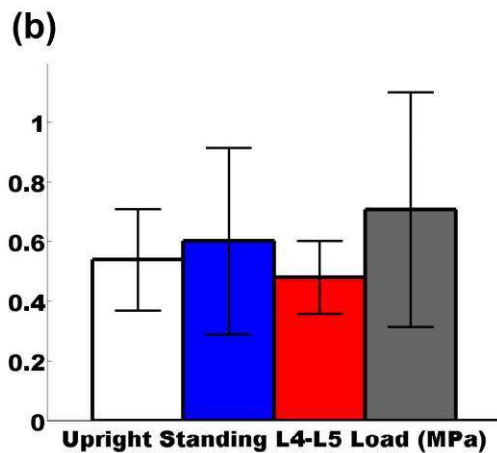
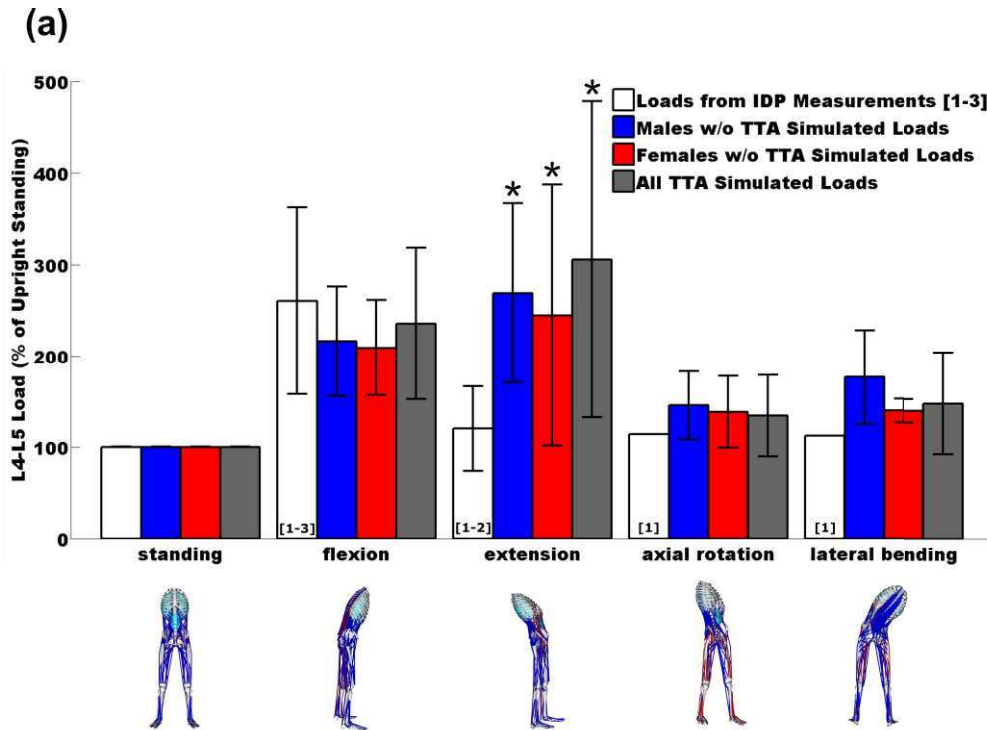


Figure 3.1: (a) Mean  $\pm$  standard deviation of intradiscal pressure (IDP) estimated from the simulation in male, female and TTA participants compared to IDP measurements from [1] (Wilke et al., 2001), [2] (Sato et al., 1999), and [3] (Nachemson, 1965). All pressures were normalized to those found during upright standing for each participant. For flexion and extension, average measurements across [1-3] and the standard deviation across all participants are shown. Pressures were taken from terminal flexion, extension, and axial rotation, and from 20° of lateral bending (2 males w/o TTA and 2 males w/ TTA did not reach 20° of lateral bending and were not included for statistical testing). \* indicates a significant difference between the model estimates for the corresponding participant group and experimental values from the literature ( $p \leq 0.05$ ). (b) Pressure estimates during upright standing.

### 3.5 – Discussion

Loading estimates from the model during trunk-pelvis ROM trials agreed well with published values. The greatest discrepancies between model-estimated and measured IDP were at terminal extension (Figure 3.1a) and in lateral bending beyond 20° (Figure 3.2). Higher loading may suggest a greater proportion of the intervertebral joint loading being transferred to the facet joints. In the model, facet and disc loading are both included in the net contact force, while facet joint loading is not measured by intradiscal pressure transducers. Increased facet joint loading is consistent with model IDP estimates being larger than measurements from the literature for extension, lateral bending, and axial rotation. In addition, lateral bending and axial rotation are known to increase facet joint loading in cadaver studies (Popovich et al., 2013).

Predicted loads and trunk-pelvis kinematics among people with TTA were similar to those for people without TTA. Women had a significantly greater range of trunk-pelvis motion than men in lateral bending. While IDP estimates were similar for men and women, the intervertebral disc cross-sectional area used to calculate IDP was taken from MRIs of men. Women have smaller vertebral endplate cross-sectional area (Zhou et al., 2000), which may suggest smaller intervertebral disc cross-sectional area and greater IDP for a given load. Variations in motion and curvature of the spine between men and women have been previously reported (Dvorak et al., 1992; Fon et al., 1980; Sullivan and Dickinson, CE, Troup, 1994), and women are more susceptible to LBP (Wong et al., 2017). Thus, there may be justification for developing sex-specific models to adequately account for anatomical and kinematic differences, particularly at extreme ranges of motion.

Model muscle activations compared well with measured EMG data with similar timing in high and low activity levels. Agreement was high for the lumbar paraspinals and flexion motion. However, the thoracic paraspinals exhibited poor agreement during extension for female and TTA participants. There was inconsistent agreement for extension, axial rotation, and lateral bending motions, which may be attributed to poor model characterization of co-contraction. In addition, the sum of muscle activations from multiple muscles in the model may also have over or under-predicted the muscle activity that was measured by EMG (also from multiple muscles). Agreement between modeled and measured muscle excitations can also be sensitive to methods of processing and normalization.

Other models with detail of the lumbar spine have accurately estimated loading throughout the spine during static poses (Bruno et al., 2015). The current study builds upon this prior work by accurately estimating lumbar loads during dynamic ROM trials. Kinematic motion capture data measured with a skin-mounted marker set that includes tracking of the trunk, pelvis and lower limbs can be used with this model to provide a greater understanding of low back biomechanics during movement. Despite the inherent complexity involved in tracking the trunk (Dreischarf et al., 2016a; Leardini et al., 2009; Zemp et al., 2014), this model accurately represents spinal loading using tracking of a three degree-of-freedom trunk segment, while revealing differences between men and women, and performing well for people with TTA. We feel the model can reasonably be extended to contexts of use beyond ROM simulation, such as walking or sit-to-stand. The current model can also be extended to include arms, head, and neck if tracking data are available for those segments (Hamner et al., 2010; Raabe and Chaudhari,

2016). Analyses requiring precise full-spine curvature are not advisable with this model as skin-mounted markers cannot fully capture intervertebral motion.

### **3.6 – Conclusions**

Estimates of lumbar spine loads during trunk ROM trials in this study were consistent with published *in vivo* measurements. Timing of predicted lumbar paraspinal and upper erector spinae muscle activity agreed well with EMG measurements. Validated estimates of low back biomechanics can assist in identifying mechanisms for low back pain development.

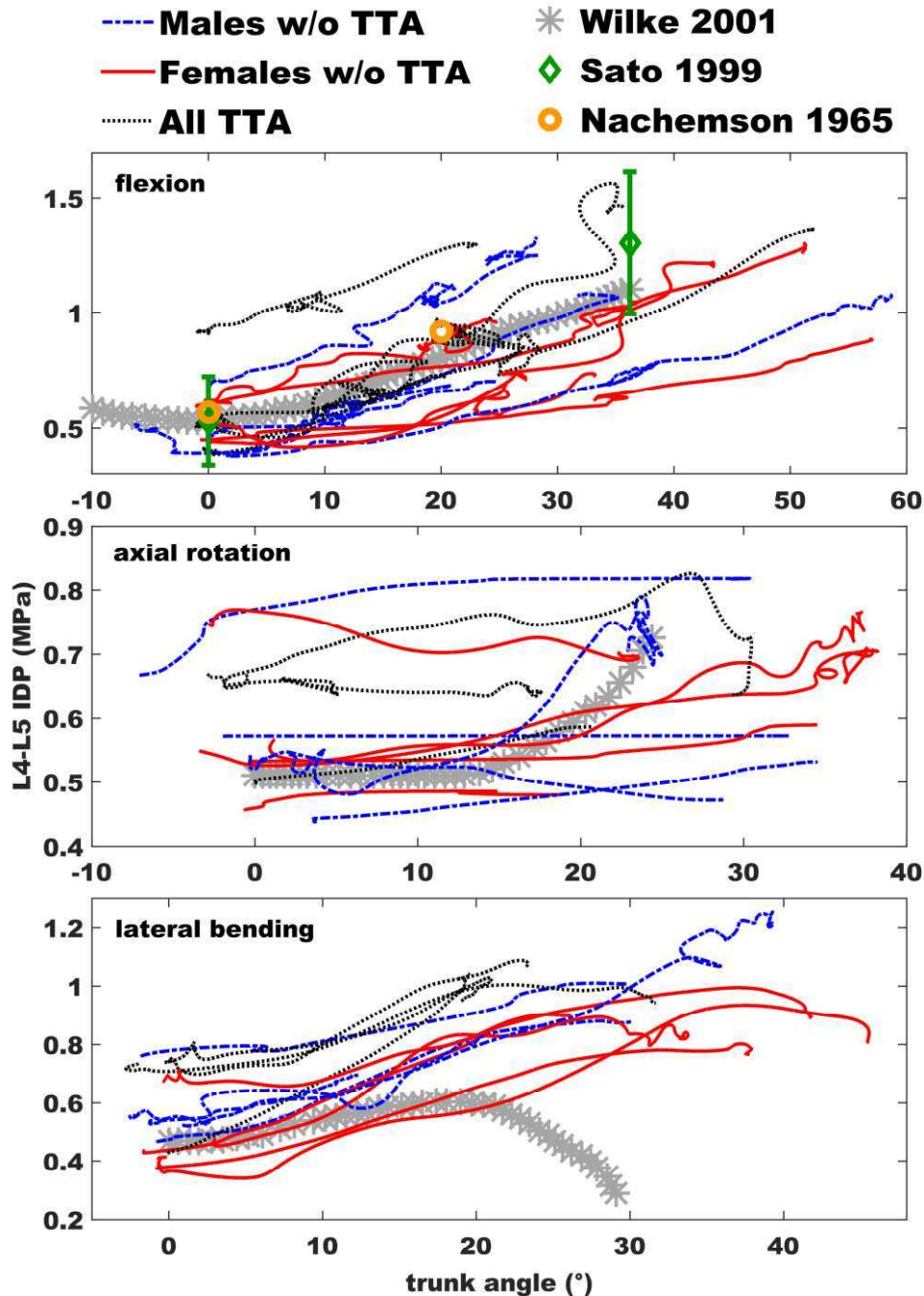


Figure 3.2: Trunk-pelvis angle vs. L4-L5 IDP during flexion, lateral bending and axial rotation trials for all participants compared to IDP from the literature (Nachemson, 1965; Sato et al., 1999; Wilke et al., 2001). All IDPs were normalized to the 70-kg mass of the experimental participant in Wilke et al (2001) by multiplying IDP by a unit-less constant (70 kg/ participant mass in kg). Male participants without TTA are shown in blue (dash-dot), female participants without TTA are shown in red (solid) and participants with an amputation are shown in black (dotted). The terminal flexion angle from Wilke et al. (grey \*) was used to approximate the terminal flexion angle from Sato et al. (green diamond (IDP  $\pm$ SD)).

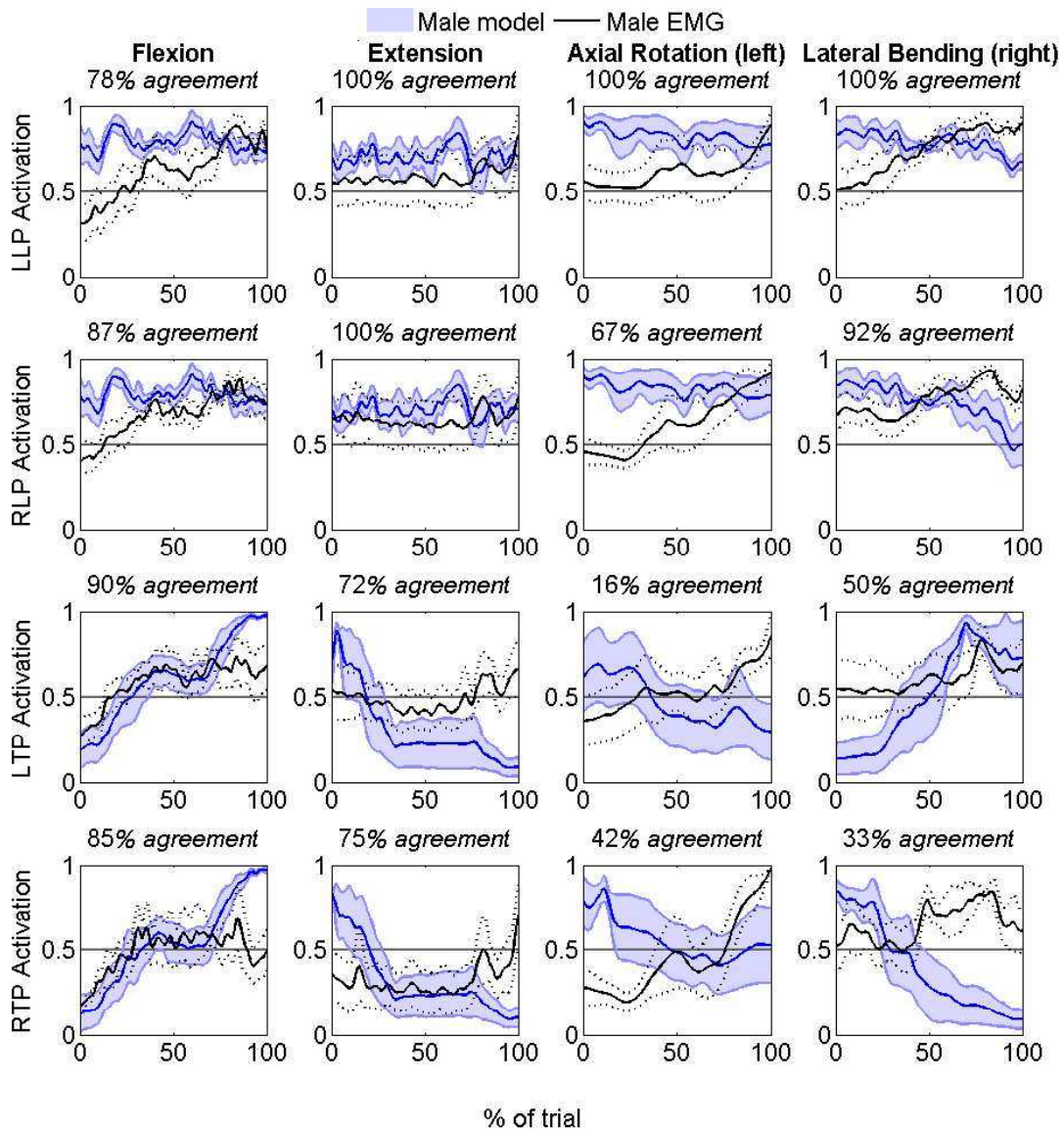


Figure 3.3: Average model activations (blue shaded) versus measured EMG activity (black). Mean values are shown  $\pm 0.5 \cdot SD$  for the left lumbar paraspinals (LLP), right lumbar paraspinals (RLP), left thoracic paraspinals (LTP), and right thoracic paraspinals (RTP) for all male participants without TTA. Agreement between model activations and measured EMG activity, quantified as the percentage of each trial during which both EMG activity and model muscle activations were on the same side of (above or below) a threshold of 0.5 at the same time, are shown above each plot.

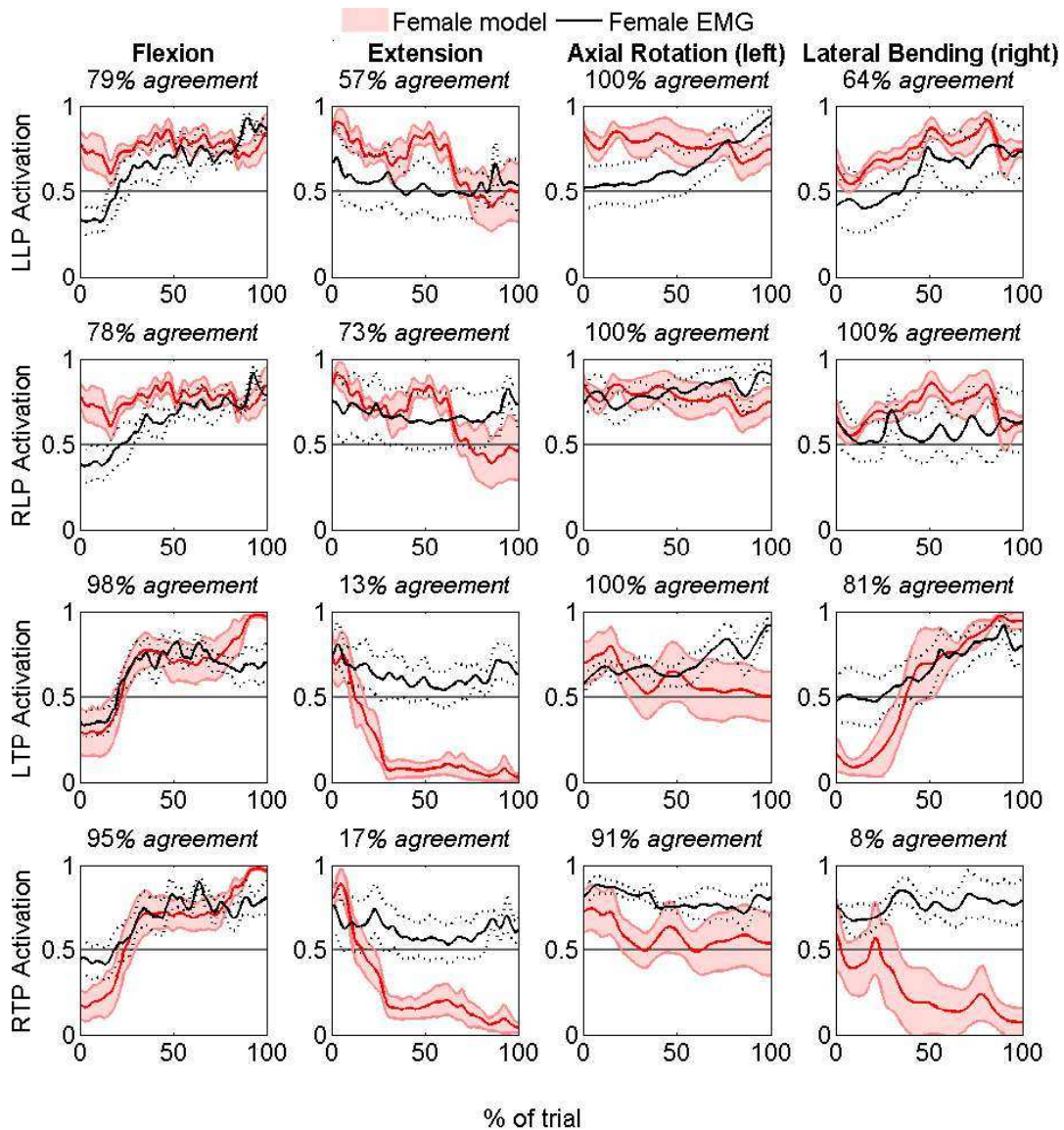


Figure 3.4: Average model activations (red shaded) versus measured EMG activity (black). Mean values are shown  $\pm 0.5 \cdot SD$  of the left lumbar paraspinals (LLP), right lumbar paraspinals (RLP), left thoracic paraspinals (LTP), and right thoracic paraspinals (RTP) for all female participants without TTA. Agreement between model activations and measured EMG activity, quantified as the percentage of each trial during which both EMG activity and model muscle activations were on the same side of (above or below) a threshold of 0.5 at the same time, are shown above each plot.

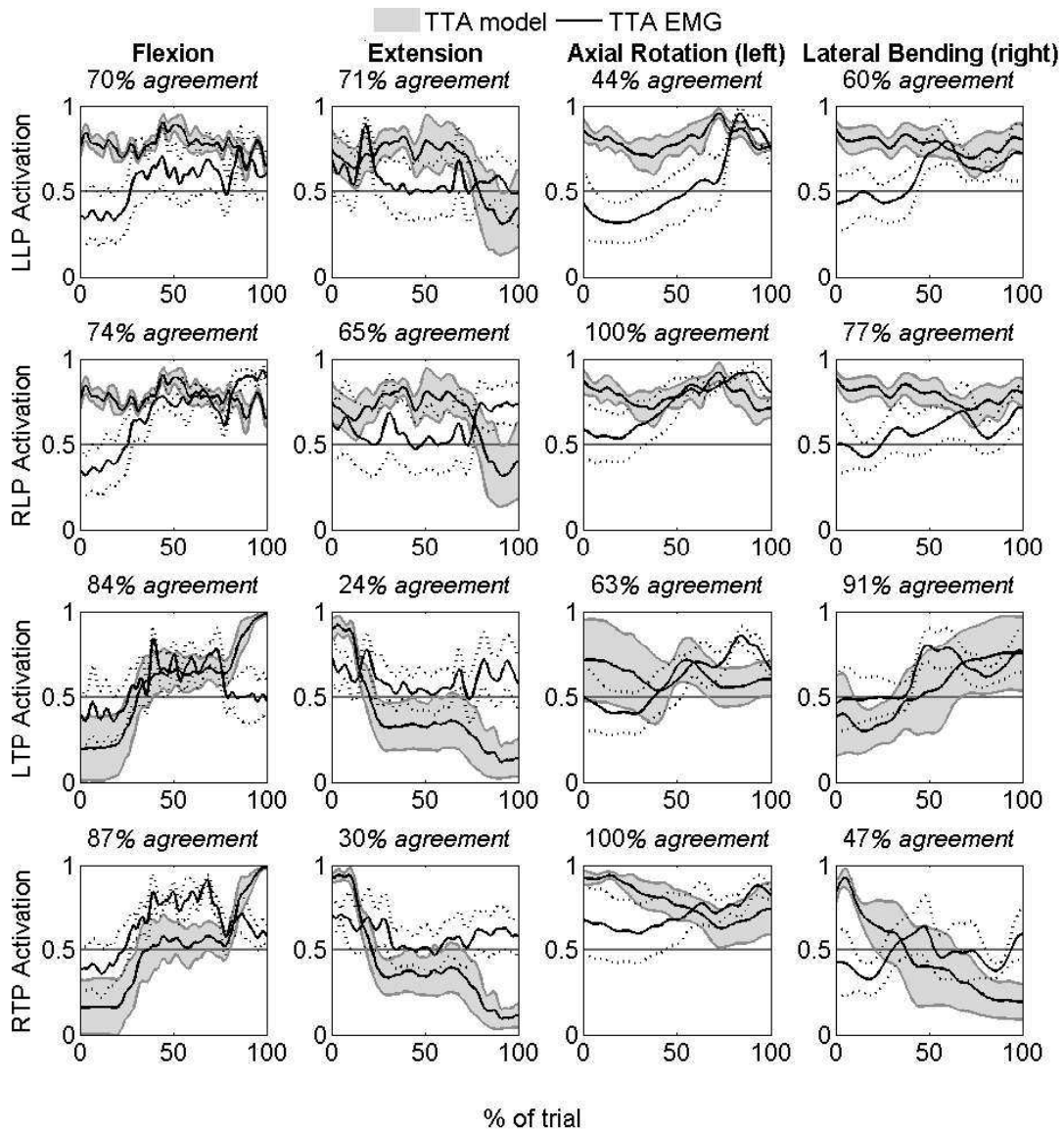


Figure 3.5: Average model activations (grey shaded) versus measured EMG activity (black) for participants with an amputation (three males, one female). Mean values are shown  $\pm 0.5 \cdot \text{SD}$  of the left lumbar paraspinals (LLP), right lumbar paraspinals (RLP), left thoracic paraspinals (LTP), and right thoracic paraspinals (RTP) for all participants with TTA. Agreement between model activations and measured EMG activity, quantified as the percentage of each trial during which both EMG activity and model muscle activations were on the same side of (above or below) a threshold of 0.5 at the same time, are shown above each plot.

CHAPTER 4:  
LOW BACK BIOMECHANICS OF PEOPLE WITH A TRANSTIBIAL AMPUTATION  
DURING SIT-TO-STAND

To be submitted to the *Journal of Biomechanics*

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#### 4.1 – Abstract

People with a transtibial amputation are characterized by numerous secondary health conditions, including an increased prevalence of low back pain. This increased prevalence may be partially explained by altered low back biomechanics during movement. The purpose of this study was to compare the low back biomechanics of people with and without a transtibial amputation during the sit-to-stand motion. Motion capture, ground reaction force and electromyographic data were collected from eight people with a unilateral transtibial amputation and eight people without an amputation during five self-paced sit-to-stand motions. A musculoskeletal model of the torso, lumbar spine, pelvis, lower limbs, and 294 muscles was used in a static optimization

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framework to quantify low back loading, low back muscle forces, and trunk kinematics. Participants with an amputation had greater peak and average L4-L5 intervertebral joint compressive loading compared to control participants, with peak loading occurring shortly after liftoff from the seat. At the instant of peak loading, participants with an amputation were in a position with significantly greater trunk-room (i.e., trunk orientation relative to a global coordinate system) lateral bending and trunk-pelvis axial rotation toward the intact side, and significantly greater trunk-room axial rotation toward the prosthetic side compared to control participants. Participants with an amputation also had greater peak frontal plane and transverse plane trunk-room angular velocity. The movement strategy used by participants with a transtibial amputation was associated with greater L4-L5 loading and greater axial rotation of the L4-L5 intervertebral joint, which suggests an increased risk for injury and pain development.

## **4.2 - Introduction**

28% of US adults suffer from chronic low back pain (LBP) (Schiller, 2012), but chronic LBP is even more common among people with a lower limb amputation (LLA). The prevalence of LBP among people with LLA is nearly 70%, with 60% developing LBP within the first two years of amputation (Kulkarni et al., 2005). In addition to an increased prevalence of LBP, people with a transtibial amputation (TTA) have reduced activity levels (e.g., Bussmann et al., 2008), kinematic and kinetic asymmetry (e.g., Sagawa et al., 2011), and pain in the lower-limb joints (Struyf et al., 2009). Identifying the biomechanical risk factors for the increased prevalence of chronic LBP among people with TTA has potential to help mitigate LBP development in people with or without an amputation.

Interrelated social, biological and psychological factors make LBP difficult to diagnose and treat, but mechanical damage is one of the greatest contributors to LBP development (Bogduk, 2005; Sterud et al., 2016; van Dieën et al., 1999). Commonly diagnosed sources of chronic pain include degeneration to the facet joints and intervertebral discs in the lumbar spine, with discogenic pain accounting for approximately 40% of LBP cases (Bogduk, 2005; Finch, 2006; Schwarzer et al., 1995). Some mechanical risk factors for intervertebral joint damage may include excessive loading (Dreischarf et al., 2016b; Kumar, 2001; van Dieën et al., 1999), excessive tangential strain from axial rotation combined with deformation of the intervertebral disc (Costi et al., 2007; Farfan et al., 1970; Inoue and Espinoza Orias, 2011), or increased facet joint load sharing from excessive lateral bending and/or axial rotation (Adams and Hutton, 1981; An et al., 2006; Eubanks et al., 2007; Park et al., 2013). Altered biomechanics in the low back are suspected as one of the main contributing factors to LBP in people with LLA (Hendershot and Wolf, 2014).

People with TTA have greater trunk-pelvis ranges of motion and greater lateral bending toward the prosthetic leg during walking (Hendershot and Wolf, 2014; Rueda et al., 2013; Yoder et al., 2015), with associated increases L4-L5 joint contact forces (Yoder et al., 2015). Sit-to-stand (STS) is another important activity of daily living that is performed by people with TTA approximately 50 times/day (Bussmann et al., 2008, 2004) and has greater L4-L5 loading than walking or running (Wilke et al., 1999). Further, people with a transfemoral amputation have greater peak lumbosacral joint moments and angles in the frontal, transverse, and sagittal planes during STS (Hendershot and Wolf, 2015), which may also apply to people with TTA. Characterizing

the *in vivo* low back biomechanics of people with TTA could reveal risk factors for LBP development, although obtaining *in vivo* measurements remains challenging. For example, measurement techniques including the use of intradiscal pressure transducers in the L4-L5 intervertebral disc are invasive, while only providing pressure data for a specific location within the disc (Dreischarf et al., 2016b). In contrast, musculoskeletal modeling and simulation can be used to provide estimates of muscle and joint mechanics in the low back non-invasively, which can help identify risk factors for pain development (e.g., Yoder et al., 2015).

Therefore, the purpose of this study was to characterize the low back biomechanics of people with and without unilateral TTA during STS using musculoskeletal modeling. Participant-specific musculoskeletal models with detail of the lumbar spine were used to identify L4-L5 contact forces, muscle forces, and kinematics that may help explain the elevated risk for LBP development. We expected that people with TTA would have an altered movement strategy during sit-to-stand compared to people without TTA because of their asymmetric GRFs during STS (e.g., Agrawal et al., 2011), and larger low back loads and muscle forces during walking (Yoder et al., 2015). Specifically, we hypothesized that people with TTA would have greater L4-L5 intervertebral joint loading, larger trunk-room (i.e., trunk angle relative to a global coordinate system) and trunk-pelvis angles at the instant of peak loading, greater trunk-room peak angles and angular velocities, greater trunk-pelvis peak angles, and greater low back muscle forces compared to people without TTA.

## 4.3 - Methods

### 4.3.1 - Participants

Eight participants with a unilateral TTA wearing their clinically prescribed dynamic response prostheses (TTA group, 7M/1F), and eight participants without TTA (Control group, 4M/4F) were analyzed during five self-paced STS trials (Table 4.1). TTA participants had been using their prosthesis for at least 6 months and were classified as K3 or greater on the Medicare Functional Classification Scale. All participants provided informed consent to the protocol that was approved by the Institutional Review Board. All participants completed a Modified Oswestry Low Back Pain Questionnaire (Fairbank and Pynsent, 2000). Limb dominance was determined as the leg used to kick a ball.

Table 4.1: Mean (SD) participant demographics for TTA and Control groups.

	<b>Age (years)</b>	<b>Height (m)</b>	<b>Mass (kg)</b>	<b>Modified Oswestry (%)</b>	<b>Cause of Amputation</b>	<b>Years Since Amputation</b>
<b>Control</b> (4M/4F)	28 (8)	1.74 (0.1)	80 (19)	1.3 (1.7)	N/A	N/A
<b>TTA</b> (7M/1F)	42 (15)	1.8 (0.1)	90 (16)	7.5 (9.8)	Traumatic (x5), Dysvascular (x2), Cancer (x1)	11.5(12.2)

Each participant completed a static, standing calibration trial prior to the dynamic trials. Trials began with participants seated on an adjustable-height backless chair, with their knees and hips at 90°, and each ankle hip-width apart on a separate force plate (AMTI Inc., Watertown, MA). The chair was in contact with a third force plate for 13 of

the 16 participants. The five STS trials were performed in succession with pauses while standing and sitting between each trial.

Participants were instrumented with 42 kinematic markers for tracking the feet, shanks, thighs, pelvis and trunk. Markers were placed bilaterally on the acromion, iliac crest, anterior superior iliac spine, posterior superior iliac spine, greater trochanter, 4-marker thigh cluster, medial/lateral femoral condyles, 4-marker shank cluster, medial/lateral malleoli, heel, first metatarsal head and 5<sup>th</sup> metatarsal head. Markers were also placed on the sternal notch, xyphoid process, C7, and T8. Electromyography (EMG) sensors (Delsys, Inc., Boston, MA) were placed bilaterally on the upper and lower erector spinae, gluteus medius, biceps femoris long head, rectus femoris, vastus lateralis, medial gastrocnemius, and tibialis anterior. Marker kinematics were collected at 120 Hz with a 20-camera motion capture system (Motion Analysis Corp., Santa Rosa, CA). EMG and ground reaction force (GRF) data were collected at 1200 Hz.

#### **4.3.2 - Data Processing**

Kinematic, GRF, and EMG data during STS trials were processed with a bi-directional 4<sup>th</sup>-order Butterworth filter. Kinematics and GRFs were low-pass filtered with cutoff frequencies of 6 Hz and 10 Hz, respectively. EMG data were demeaned, band-pass filtered between 30 and 500 Hz, rectified, and low-pass filtered at 2.5 Hz for validation of model muscle activations (Brereton and McGill, 1998; Drake and Callaghan, 2006). EMG signals were then normalized to their peak value during each STS trial. The inverse kinematics solution for each STS trial was computed in Visual3D (C-Motion, Inc., Germantown, MD) using an eight-segment model (feet, shanks, thighs,

pelvis, and trunk) (Lu and O'Connor, 1999) with 19 degrees of freedom (DOFs, 1 DOF for each ankle and knee, 3 DOFs for hips and trunk, 6 DOFs for pelvis). For each participant, the standing portion of the STS trials was used to prescribe a default standing spinal posture (Rast et al., 2016; Zemp et al., 2014).

### **4.3.3 - Musculoskeletal Modeling**

A musculoskeletal model was developed in OpenSim 3.3 (Delp et al., 2007; simtk.org) including: 294 Hill-type musculotendon actuators with force-length-velocity properties (Zajac, 1989), the lower limbs from (Anderson and Pandy, 1999; F C Anderson and Pandy, 2001; Delp et al., 1990; Gary T. Yamaguchi and Zajac, 1989), the lumbar spine and torso from (Christophy et al., 2012), muscle strengths from (Bruno et al., 2015), and body mass distributions from (Winter, 2009). The motion of individual lumbar vertebrae was prescribed by overall trunk-pelvis angles. A version of the model for people with unilateral TTA was created by removing 12 ankle muscles of the prosthetic leg, reducing residual shank mass, and shifting residual shank center of mass proximally as in (Silverman and Neptune, 2012). The model was scaled to each participant based on the static, standing trial. For each STS trial, the inverse kinematics solution was determined from the initiation until the termination of trunk center of mass velocity. The inverse kinematics solution, GRF data, and scaled models were then used as inputs to a residual reduction algorithm to improve dynamic consistency. Due to the lack of force measurement underneath the chair for 3 participants, the instant when the vertical residual force applied to the pelvis reached zero was used as an estimate of the instant of liftoff. The residual reduction algorithm was then completed again beginning 30 ms prior to this instant until the termination of trunk center of mass velocity. A static

optimization algorithm was then performed to solve for muscle activations by minimizing the sum of all muscle activations squared at each instant in time for this duration. Joint contact forces were calculated between the L4 and L5 vertebrae and expressed in the L5 reference frame. Loading at the L4-L5 intervertebral joint was normalized to body weight for comparisons between participants.

#### **4.3.4 - Data Analysis**

Individual muscles in the low back were grouped according to anatomical location (Table 4.2) and their activations and forces were summed for interpretation. Muscle activations and EMG signals were compared for validation of model muscle results. For comparison to EMG signals, muscle group activations from the model were similarly low-pass filtered at 2.5 Hz and normalized to their peak value during each trial. Muscle group forces were also low-pass filtered at 2.5 Hz. Muscle forces and L4-L5 loading were normalized to each participant's bodyweight (BW). Kinematic, kinetic and muscle data were averaged across five trials for each participant. Differences between participant groups in peak and average L4-L5 compressive loading; minimum, maximum, and value at peak loading for trunk-room and trunk-pelvis angles; and maximum, average, and value at peak loading for muscle force groups on the intact/dominant and prosthetic/non-dominant sides were assessed using t-tests ( $\alpha=0.05$ ). Differences were defined as approaching significance if  $0.05 < p \leq 0.10$ .

## 4.4 - Results

Only one participant (male with TTA) had more than minimal LBP (30% Oswestry score). All participants successfully completed the STS trials, with no significant difference in average trial length between control (1.16 (0.17) seconds) and TTA (1.32 (0.26) seconds) participants. EMG signals were available for all participants without an amputation and five participants with TTA.

### 4.4.1 – Model Quality

Average root-mean-squared (RMS) residual forces and moments in the model across all trials and participants were low: 2.55, 1.53, and 0.09 %BW forces in the anterior/posterior, vertical, and medial/lateral directions, and 1.17, 0.39, and 2.44 %BW-m moments in the frontal, transverse and sagittal planes. Average RMS kinematic adjustments across all trials during the residual reduction algorithm were less than 10 mm for pelvis position and less than  $0.05^\circ$  for all joint angles except for trunk-pelvis flexion ( $0.43^\circ$  average). Two of the five trials from one female TTA participant were excluded from analysis due to peak residual forces approaching 20% BW. There was agreement between average muscle activations and EMG activity, particularly in the low back musculature most relevant to the analysis, for all participants (Figure 4.1). TTA participants had very little EMG activity for prosthetic side vastus lateralis and rectus femoris, and although the model activations for these muscles was also low, there was more variation in the model activations. Thus, the disagreement between the EMG signals and modeled muscle activations is largely a result of normalization technique. For trials when the chair was in contact with a force plate, the period when force on the chair was decreasing rapidly prior to final liftoff lasted between 200-400ms, and the

instant when GRFs on the chair reached zero was on average 38 ms after the instant when the vertical residual force applied to the pelvis reached zero (68 ms after the initial time of simulations). Thus, we are confident that the simulation captured the duration of the motion from liftoff until standing.

#### **4.4.2 - L4-L5 Loading**

Participants with TTA had 17% greater peak and 21% greater average L4-L5 compressive loading compared to control participants (Figure 4.2, Table 4.3). Peak L4-L5 compressive loading was 3.1x the standing load for control participants, and 3.32x the standing load for TTA participants. The only other L4-L5 loading component with peak forces/moments greater than 0.1 BW/BW-m was the posterior force (TTA peak = 0.20 BW, Control peak = 0.18 BW,  $p=0.818$ ). On average, peak compressive loading occurred 95 ms after the beginning of trials (27 ms after the GRF underneath the chair reached zero ( $n=13$ )), and 65 ms after vertical residuals applied to the pelvis reached zero).

#### **4.4.3 - Trunk Kinematics**

At the instant when peak loading occurred, participants with TTA were in a position with greater trunk-room and trunk-pelvis angles, including significantly greater trunk-room lateral bending toward the intact leg, trunk-room axial rotation toward the prosthetic leg, and trunk-pelvis axial rotation toward the intact leg, along with greater trunk-room flexion that approached significance (Figure 4.3, Table 4.3). Also, participants with TTA had greater peak angles during the STS motion, including trunk-room flexion, trunk-room lateral bending toward the intact leg, trunk-room axial rotation

toward the prosthetic leg, and trunk-pelvis axial rotation toward the intact leg. Control participants had larger peak trunk-room axial rotation toward the dominant leg and larger trunk-pelvis axial rotation toward the non-dominant leg. Participants with TTA also had greater trunk-room lateral bending and axial rotation angular velocities.

Table 4.2: Grouping of musculotendon actuators within the model. Muscle group activations were compared to available electromyographic signals (indicated by <sup>E</sup>).

<b>Muscle Group</b>	<b>Abbreviation</b>	<b>Muscles in Model</b>	<b># of Modeled Musculotendon Actuators (per side)</b>
Upper Erector Spinae <sup>E</sup>	UES	Iliocostalis pars thoracis, longissimus pars lumborum	29
Lower Erector Spinae <sup>E</sup>	LES	Iliocostalis pars lumborum, longissimus pars lumborum	9
Multifidus	Multifidus	Multifidus	25
Obliques	Obliques	Internal obliques, external obliques	10
Gluteus Medius <sup>E</sup>	GM	Gluteus Medius (three compartments) Gluteus Minimus (three compartments)	6
Biceps Femoris <sup>E</sup>	BF	Biceps femoris long head, semimembranosus, semitendinosus, gracilis	4
Vastus Lateralis <sup>E</sup>	VL	Vastus lateralis, vastus intermedius, vastus medialis	3
Rectus Femoris <sup>E</sup>	RF	Rectus femoris	1
Medial Gastrocnemius <sup>E</sup>	MG	Medial gastrocnemius, lateral gastrocnemius	2
Tibialis Anterior <sup>E</sup>	TA	Tibialis anterior, extensor digitorum longus, extensor hallucis longus, peroneus tertius	4

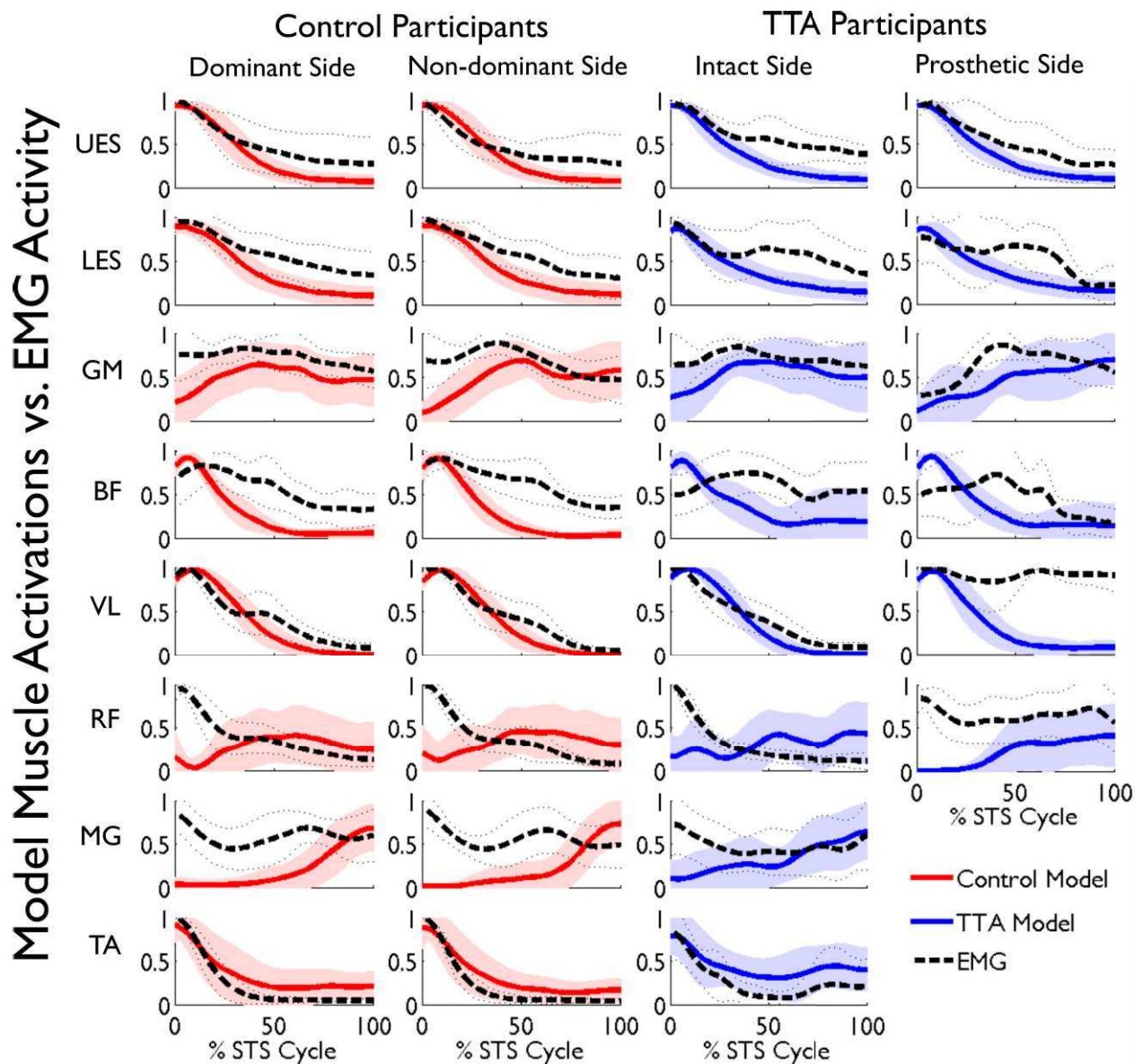


Figure 4.1: Comparison of average ( $\pm$  standard deviation) EMG activity and model muscle activations (low-pass filtered at 2.5 Hz) for all control participants and the five TTA participants (who had all EMG sensors). All 16 bilateral sensors are included for control participants (UES = upper erector spinae, LES = lower erector spinae, GM = gluteus medius, BF = biceps femoris, VL = vastus lateralis, RF = rectus femoris, MG = medial gastrocnemius, TA = tibialis anterior). TTA participants did not have data for prosthetic side MG and TA, and one control participant did not have EMG data for the right medial gastrocnemius.

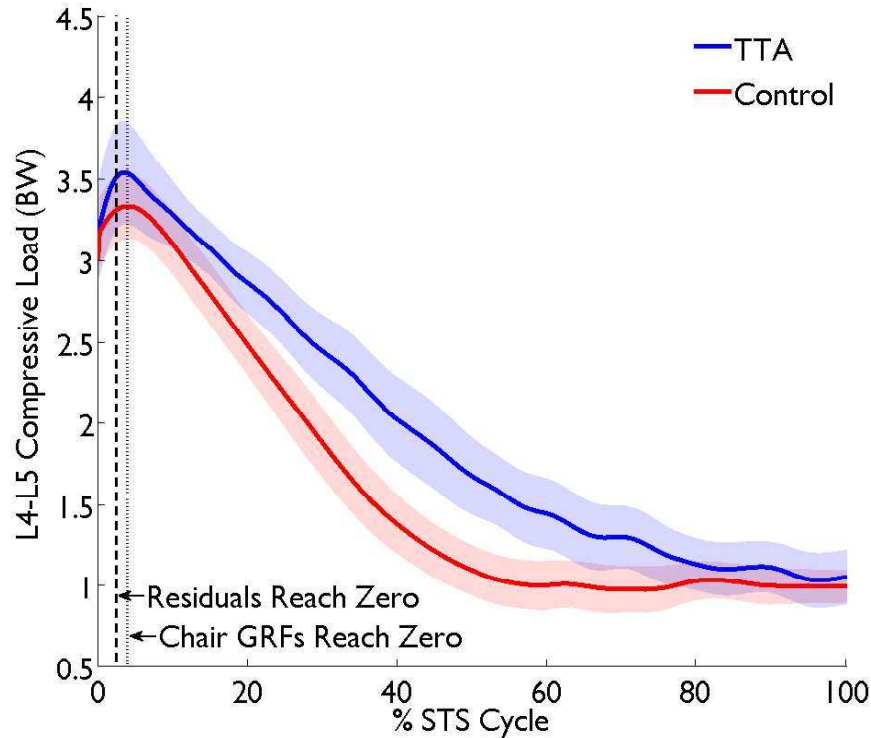


Figure 4.2: Average ( $\pm 0.5$ \*standard deviation) L4-L5 compressive load across all trials for TTA and Control groups with the STS cycle from 30 ms prior to the instant when residuals applied to the pelvis reached zero (dashed line) until the termination of forward trunk center of mass velocity. Average estimate of when GRFs applied to the chair for 13 participants is marked with a vertical dotted line.

#### 4.4.4 - Low Back Muscle Forces

Muscle forces were not significantly different between groups. However, some comparisons approached significance ( $0.05 < p \leq 0.10$ ). For example, at the instant of peak loading, participants with TTA had nearly significantly greater upper and lower erector spinae muscle forces (Figure 4.4). On the prosthetic/non-dominant side, people with TTA also had nearly significantly greater average oblique and multifidus muscle forces, and control participants had nearly significantly greater peak oblique muscle forces.

Table 4.3: Summary of mean (SD) metrics that were significantly different ( $p \leq 0.05$ , \*) or approached significance ( $0.05 < p \leq 0.10$ ) between groups: MAX = maximum, MIN = minimum, VaPL = value at peak loading, AVG = average (only tested for L4-L5 loading and muscle forces), absMAX = absolute value maximum (only tested for velocities). I/D = Intact/Dominant Side, P/nD = Prosthetic/non-Dominant Side.

		Measure	Control	TTA	p-value
<b>L4-L5 Loading</b>	<b>Compression</b>	MAX	3.41 (.43)BW	3.98 (.61)BW	0.047*
		AVG	-1.82 (.24)BW	-2.20 (.26)BW	0.010*
<b>Trunk-Room Angle</b>	<b>Flexion</b>	VaPL	-43.98 (5.58)°	-48.90 (4.22)°	0.067
		MIN	-45.29 (5.95)°	-51.27 (5.77)°	0.061
	<b>Lateral Bending</b>	VaPL	.47 (2.48)°	4.50 (4.43)°	0.042*
		MAX (I/D)	2.20 (1.55)°	6.33 (5.14)°	0.047*
	<b>Axial Rotation</b>	VaPL	-1.39 (2.63)°	2.26 (2.57)°	0.014*
		MIN (I/D)	-3.05 (1.98)°	-.94 (1.85)°	0.045*
		MAX (P/nD)	1.58 (.90)°	5.91 (4.25)°	0.014*
<b>Trunk-Room Angular Velocity</b>	<b>Lateral Bending</b>	absMAX	11.44 (2.88)°/s	32.19 (26.03)°/s	0.042*
	<b>Axial Rotation</b>	absMAX	12.41 (2.97)°/s	24.64 (8.75)°/s	0.002*
<b>Trunk-Pelvis Angle</b>	<b>Axial Rotation</b>	VaPL	.42 (3.45)°	-5.10 (2.86)°	0.004*
		MIN (I/D)	-2.08 (2.99)°	-6.22 (3.10)°	0.017*
		MAX (P/nD)	2.65 (2.02)°	.77 (1.27)°	0.043*
<b>Muscle Forces</b>	<b>Prosthetic/ Non-Dominant Side</b>				
	<i>Obliques</i>	MAX	.37 (.17) BW	.34 (.18) BW	0.095
		AVG	.12 (.05) BW	.13 (.08) BW	0.065
	<i>Multifidus</i>	AVG	.12 (.05) BW	.16 (.05) BW	0.052
	<b>Intact/ Dominant Side</b>				
	<i>Lower Erector Spinae</i>	VaPL	.09 (.03) BW	.12 (.04) BW	0.095
		<i>Upper Erector Spinae</i>	VaPL	.97 (.12) BW	1.13 (.23) BW

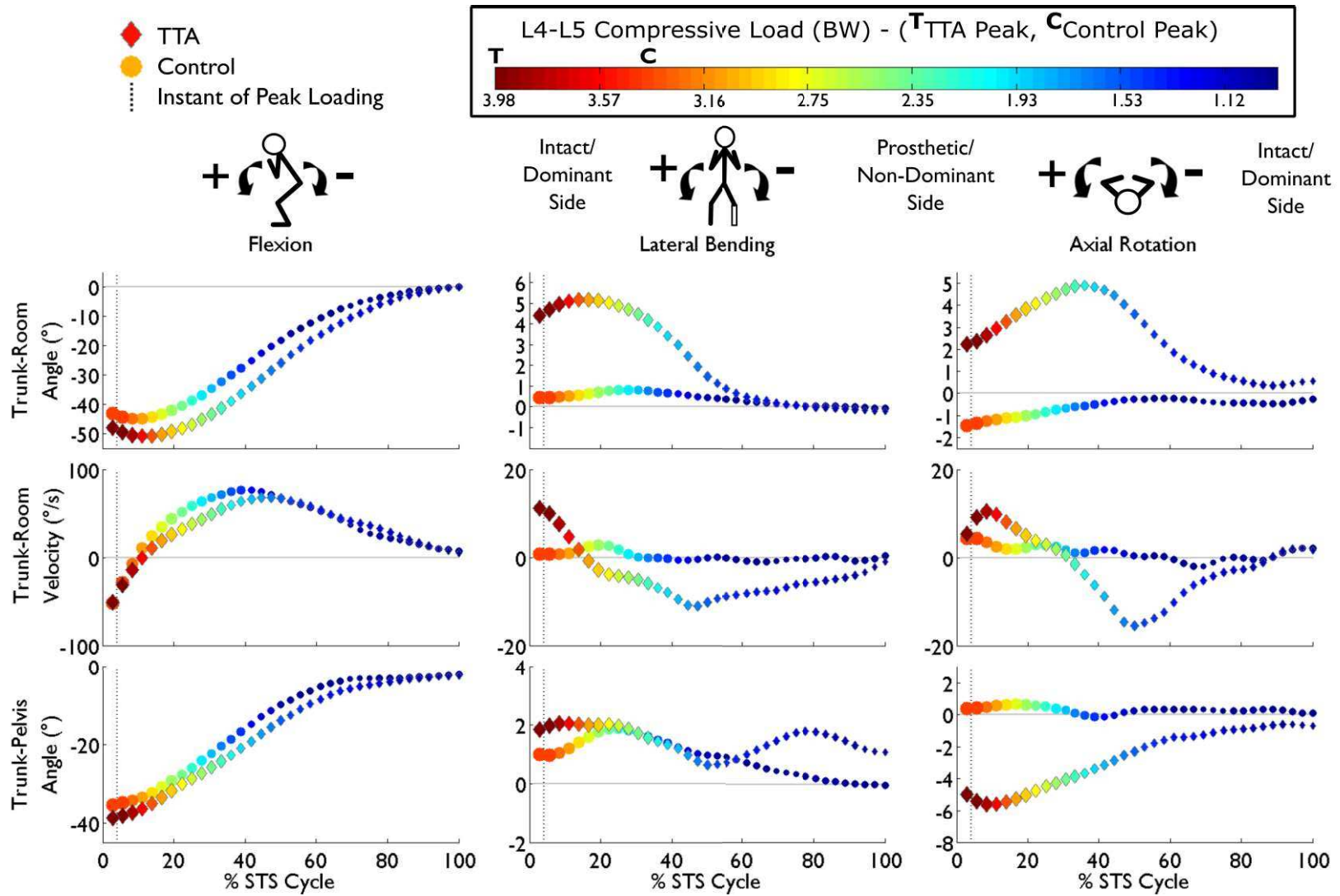


Figure 4.3: Average trunk-room angles (top row), trunk-room angular velocities (middle row), and trunk-pelvis angles (bottom row) for the Control (circles) and TTA (diamonds) groups. L4-L5 compressive load normalized by body weight is indicated by the color, with warmer colors indicating a larger load.

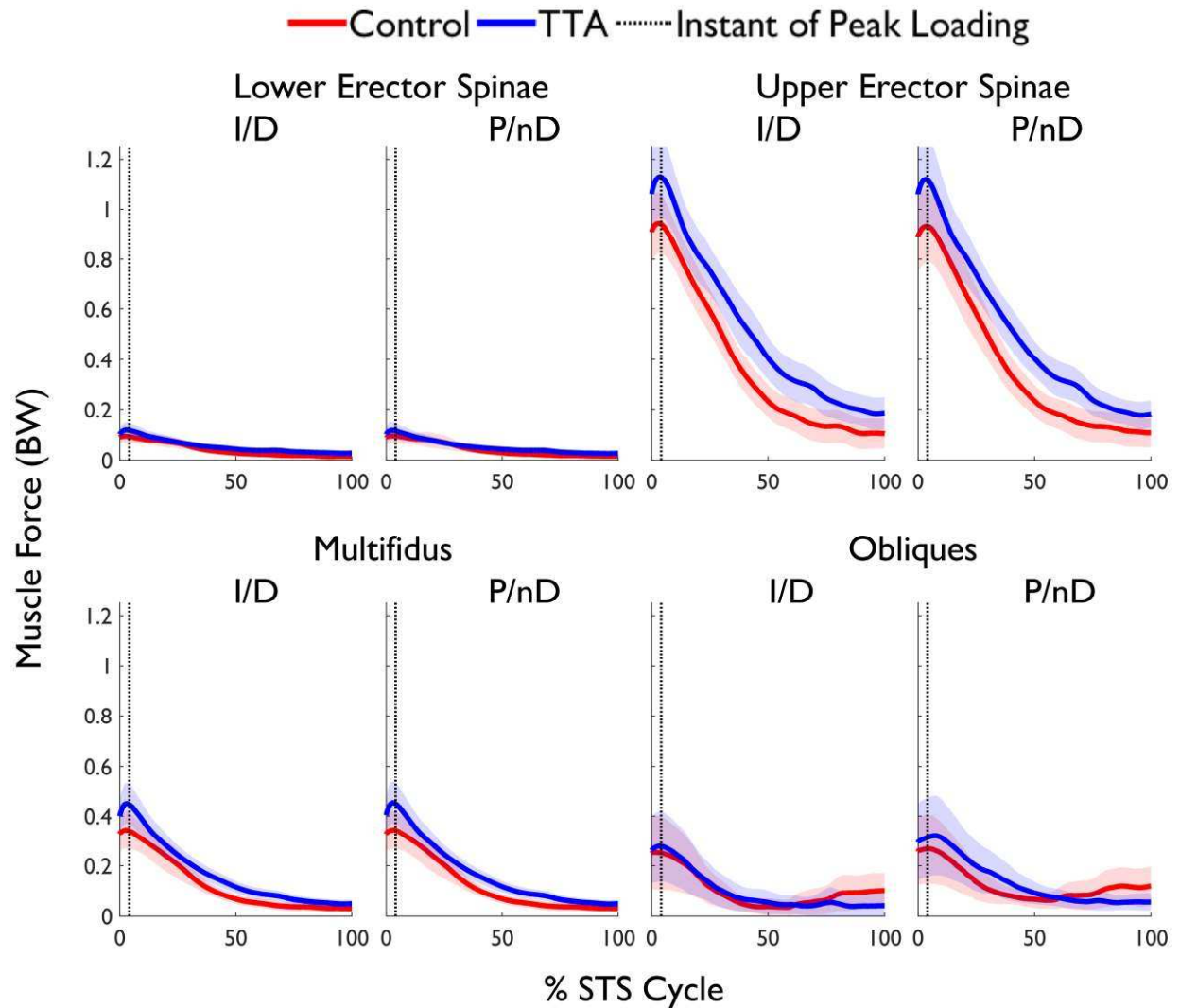


Figure 4.4: Average bodyweight normalized Intact/Dominant (I/D) and Prosthetic/non-Dominant (P/nD) side summed muscle forces ( $\pm 0.5$ \*standard deviation) from: lower erector spinae (LES), upper erector spinae (UES), multifidus, and obliques during the STS motion for control and TTA participants. Vertical (dotted) lined indicates the average instant of peak L4-L5 compressive loading.

## 4.5 - Discussion

The purpose of this study was to characterize low back loading and kinematics in people with TTA during STS. Our hypothesis that people with TTA would have greater L4-L5 intervertebral joint loading compared to people without TTA was well-supported, with greater peak and average compressive loads. Peak loading occurred shortly after liftoff for all participants, which has previously been identified as the instant of greatest GRF symmetry for people without an amputation, and the instant of greatest GRF asymmetry for people with TTA (Agrawal et al., 2011). GRF asymmetry and greater low back loading in people with TTA compared to control participants near liftoff suggest that this period is of key importance for identifying risks for LBP development, joint degeneration and/or injury.

Our hypothesis that people with TTA would have greater trunk-room and trunk-pelvis angles at the instant of peak loading was also largely supported, as people with TTA were in a position of greater trunk-room lateral bending, trunk-room axial rotation, and trunk-pelvis axial rotation at this instant. These larger angles coincided with greater intact side upper and lower erector spinae muscle forces compared to the dominant side in control participants, which contributed to greater L4-L5 loading in people with TTA. Greater displacement of the trunk center of mass away from its neutral position is one of the strongest factors in increasing low back muscle forces and loading (Dreischarf et al., 2015). In addition, the greater degree of trunk-pelvis axial rotation in participants with TTA indicates greater vertebral axial rotation, which increases tangential strains on the intervertebral disc (Costi et al., 2007) and facet joint loading

(An 2006). In combination with the greater peak compressive contact force, these kinematics may contribute to an elevated risk of spinal injury in people with TTA.

Greater axial rotation of the vertebrae has been correlated with increased disc degeneration (Inoue 2011). Our hypothesis that people with TTA would have greater trunk-pelvis angles was only supported for axial rotation toward the intact leg. Greater trunk-pelvis axial rotation has also been seen in people with a transfemoral amputation (Hendershot and Wolf, 2015) and in people with LBP (Shum et al., 2007). TTA participants maintained a more consistent axial rotation toward their intact leg while control participants remained near 0° of axial rotation with a greater peak rotation toward the non-dominant side. We verified that absolute maximum axial rotation, regardless of direction, was significantly greater in TTA participants with a secondary analysis (6.22° vs. 2.65°,  $p = 0.017$ ).

We also hypothesized that people with TTA would have greater trunk-room angles and angular velocities during STS, which was also supported. People with TTA had greater lateral bending toward the intact leg, greater axial rotation toward the prosthetic leg, and greater trunk-room velocity in both axial rotation and lateral bending relative to control participants. Greater lateral bending and greater axial rotation are consistent with greater weight distribution asymmetry (Agrawal et al., 2011), and greater trunk-room velocities are consistent with greater postural sway in people with TTA (Özyürek et al., 2013).

The hypothesis that people with TTA would have greater low back muscle forces was not supported, with no significant differences between groups. However, people

with TTA had nearly significantly greater average muscle forces for the obliques and multifidus ( $p = 0.065$ ,  $p = 0.052$ ), along with nearly significantly greater lower and upper erector spinae muscle forces ( $p = 0.095$ ,  $p = 0.090$ ). Despite the lack of significant differences between individual muscle groups, increased force from trunk muscles is one of the most important factors in increasing low back loading (Rohlmann et al., 1997). The net effect of muscle force differences in the 210 modeled trunk musculotendon actuators resulted in the greater compressive L4-L5 loading for participants with TTA.

Although six of the participants with TTA had been amputated for more than two years, and 60% of people with LLA develop LBP within two years of amputation (Kulkarni et al., 2005), only one of them reported more than minimal LBP. The movement strategy used by TTA participants in this study may contribute to elevated risk for LBP development, but psycho-social and work-related mechanical factors are also important. The participants in this study may not develop chronic LBP. However, we observed a consistent movement strategy across participants that was characterized by greater trunk-room lateral bending and trunk-pelvis axial rotation toward the intact leg, and vertebral axial rotation has been previously associated with disc and facet joint degeneration (Farfan et al., 1970; Inoue and Espinoza Orias, 2011). In addition, the kinematic results are consistent with favoring the intact leg for GRF generation relative to the prosthetic leg, which has been shown across multiple studies of people with TTA (Agrawal et al., 2011; Ferris et al., 2017; Özyürek et al., 2013). An associated increase in trunk-room axial rotation toward the prosthetic leg despite trunk-pelvis axial rotation

toward the intact leg was also apparent, which indicates that the rotation toward the prosthetic side is due to pelvis rotation and asymmetric motion of the lower limbs.

Predicted peak loads from the models during STS compared to loads at upright standing (3.1x upright standing for controls, 3.32x upright standing for people with TTA) were greater than prior *in vivo* measurements of 2.44x upright standing (Wilke et al., 1999). The greater loading estimates from the model compared to Wilke et al. (1999) may be attributed to loading in the facet joints. The model reports to the total load from both the disk and the facets, but *in vivo* measurements from intradiscal pressure transducers do not incorporate facet joint loading. In addition, prior data from (Wilke et al., 1999) is representative of one trial from one 70-kg male, and loading variation is expected across participants. Even with discrepancies in magnitude, the relative comparisons between people with and without TTA support our conclusions. Our modeling approach was consistent across groups and estimated loading patterns were consistent among all participants with a large peak immediately after liftoff followed by a steady decrease approaching the standing load.

A potential limitation to this study is that kinematic tracking of a single trunk segment is likely insufficient to provide precise estimates of spinal posture (Leardini et al., 2011; Rast et al., 2016; Zemp et al., 2014), and tracking errors are inherent in motion capture (Mcginley et al., 2009). Our approach of offsetting kinematics based on standing posture resulted in low residuals and minimal kinematic adjustments, providing confidence in trunk kinematics. However, spinal curvature was prescribed in the model as a function of trunk-pelvis motion, which cannot capture the variability in spinal curvature (Leardini et al., 2011). Higher resolution spinal tracking technology (e.g.,

Dreischarf et al., 2016a) would be valuable for better modeling vertebral kinematics (Azari et al., 2017; Bruno et al., 2015). Higher resolution modeling may also help in quantifying load sharing between the facet joints and intervertebral disc. Another potential limitation is that participants were not matched by age or sex between control or TTA groups. However, the strategy used by participants with TTA was not consistent with the strategy used in populations with decreased strength or increased age (Savelberg et al., 2007). Despite the potential limitations in this study, there was a consistent movement strategy in participants with TTA, resulting in greater L4-L5 compressive loading.

#### **4.6 - Conclusions**

Participants with TTA had greater L4-L5 compressive loading and trunk-pelvis peak axial rotation relative to participants without TTA during STS. These differences may be reduced with improved prosthetic technology and/or physical therapy targeting more neutral spinal posture and increased GRF symmetry between and legs, which may decrease risk for low back pain development.

## CHAPTER 5: GENERAL CONCLUSIONS

The goal of this research was to use musculoskeletal modeling to understand sit-to-stand movement strategies of people with a unilateral transtibial amputation that may increase risk for low back pain development. The musculoskeletal model that we developed for this investigation provided accurate estimates of lumbar spine loading during trunk-pelvis range of motion trials and during upright standing for 14 participants when compared to *in vivo* intradiscal pressure measurements from the L4-L5 intervertebral disc in previous research. The validity of these loading estimates allows meaningful conclusions to be drawn from model estimates of internal loading during the sit-to-stand motion. Applying the model to eight people with a unilateral transtibial amputation and eight people without an amputation during the sit-to-stand motion revealed greater L4-L5 intervertebral joint loading in participants with an amputation. People with a transtibial amputation had greater trunk-room lateral bending and trunk-pelvis axial rotation towards the intact leg, greater trunk-room axial rotation away from the intact leg, and greater frontal and transverse plane trunk-room angular velocity compared to control participants.

The sit-to-stand strategy used by participants with a transtibial amputation was consistent with favoring the intact leg for ground reaction force generation and greater postural sway, as seen in previous work with people with a transtibial amputation during

sit-to-stand. This strategy also resulted in greater low back loading and greater vertebral axial rotation at the instant of peak loading, which are risk factors for injury and pain development in the low back.

These findings may motivate targeted physical therapy for people with a lower limb amputation to improve their mobility and reduce their risk for developing chronic pain. The period immediately after liftoff may be of particular interest to physical therapists. During this period, people with a transtibial amputation have greater ground reaction force asymmetry, greater trunk displacements, and greater trunk-pelvis axial rotation, coinciding with greater low back loading, compared to control participants. Increasing ground reaction force symmetry in combination with reducing trunk displacements has potential reduce low back loading. In addition, decreasing trunk-pelvis axial rotation may reduce intervertebral disc strains and facet joint loading. Other approaches to encourage load bearing on the prosthetic leg and therefore symmetry between legs may include new surgical techniques (Ferris et al., 2017), or improved prosthetic foot design (Au and Herr, 2008), socket interface design (Mak et al., 2001), and/or optimized prosthetic alignment (Kobayashi et al., 2014).

The model used in this research is appropriate as a macro-scale component of a multi-scale modeling framework, in combination with tissue-level models of the spine. For example, muscle force results from this model can be used as inputs to a tissue-level model to provide insights into load sharing between the intervertebral disc and the facet joints or intervertebral disc stresses. These more-detailed quantities of the vertebral joints may help identify biomechanical mechanisms for damage. A multi-scale framework would benefit from a more accurate and higher resolution method for

tracking spinal posture, which would likely improve loading estimates. Future work on this model may also include adding the actuated arms and head, and increasing the number of trunk degrees of freedom.

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