TRUNK MUSCLE ACTIVATION PATTERNS ADAPT TO DEFICITS IN INDIVIDUAL SPINAL SYSTEMS

by

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Submitted in partial fulfilment of the requirements
for the degree of Doctor of Philosophy

at

Dalhousie University
Halifax, Nova Scotia
August 2019

To all of those who supported me, remember no gesture is too small. Thank you for all the guidance, advice and emotional support you provided during this process.

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ABSTRACT

In the early 1990's Panjabi proposed a theoretical model suggesting one risk of low back pain is a spinal instability event theorizing a deficit to any spinal system (active, passive and neural) could represent a risk. However, the interplay between systems allows for compensation in the event of a deficit. Despite this theory being used for the treatment of low back pain, there is limited supporting empirical evidence. The overarching purpose of this dissertation was to investigate this compensation theory. Study one compared two populations (older adults and those recovered from a low back injury) suspected of having deficits in all three spinal systems with a young asymptomatic control group. The objective was to determine whether both groups had different muscle activation patterns compared to controls. Both deficit groups had higher agonist and antagonist activation amplitudes and evidence of reduced responsiveness of trunk muscles to changing external moments. While the direction of adaptations observed in both deficit groups, suggests that these adaptations were in response to common spinal system deficits, the magnitude differed. Thus, it was unknown if adaptations were unique to a deficit in any singular spinal system or a result of recent pain. To investigate these limitations, three studies compared those with high or low function of the active, passive, and neural systems and whether recent low back pain modified these adaptations. These comparisons identified that lower function within any single spinal system had different trunk muscle activation patterns that interacted with recent pain. Adaptations included increased agonist and antagonist amplitudes and different (increased or decreased) responsiveness to changing external moments. Muscle adaptations were specific to the measured spinal system to produce higher relative muscle force (active) or increase the general stiffness of the spine (passive and neural). Collectively these findings were consistent with Panjabi's theoretical model that trunk muscle activation patterns adapt to dysfunction within spinal systems. While cross-sectional data cannot infer causation, a rigorous methodology was employed, contributing to a broader understanding of the interrelationships between individual spinal system function, neuromuscular control during dynamic tasks and the modifying effect of recent pain.

LIST OF ABBREVIATIONS

2MC	Second Metacarpal
5MC	Fifth Metacarpal
Ac	Acromion
ACS	Anatomical Coordinate System
A/D	Analog to Digital
Ag	Silver
AgCl	Silverchloride
ANOVA	Analysis of Variance
ASYM	Asymptomatic
ASIS	Anterior Superior Iliac Spine
BMI	Body Mass Index
BSIP	Body Segment Inertial Parameters
C7	Seventh Cervical Spinous Process
CSA	Cross Sectional Area
CoM	Center of Mass
CCW	Counter Clockwise
CW	Clockwise
EMG	Electromyography
EO1	External Oblique (Anterior Fibers)
EO2	External Oblique (Lateral Fibers)
EO3	External Oblique (Posterior Fibers)
FoB	Flock of Birds
FT	Forearm Triad
FRL	Fast Reflex Latency
GCS	Global Coordinate System
HT	Hand Transition
HTT	Horizontal Transfer Task
Hz	Hertz
IFFT	Inverse Fast Fourier Transfer
IC	Iliac Crest
IO	Internal Oblique
JC	Joint Centre
Kg	Kilogram
L	Left
L13	Superior Erector Spinae Longissimus Lumborum
L16	Superior Erector Spinae Iliocostalis Lumborum
L33	Inferior Erector Spinae Longissimus Lumborum
L36	Superior Erector Spinae Iliocostalis Lumborum
L48	Quadratus Lumborum
L52	Superficial Multifidus
L4/L5	Fourth and Fifth Lumbar Invertebral Disc
LBP	Low Back Pain
LBI	Low Back Injury
LHT	Left Hand Transfer

LHE	Lateral Humeral Epicondyle
LRA	Lower Rectus Abdominis
m	Meter
MHE	Medial Humeral Epicondyle
MVIC	Maximum Voluntary Isometric Contraction
N	Newton
Nm	Newton Meter
PC	Principal Component
PCA	Principal Component Analysis
PCS	Pain Catastrophizing Scale
PSIS	Posterior Superior Iliac Spine
PT	Pelvic Triad
R	Right
RHT	Right Hand Transfer
RMD	Roland Morris Disability
RMS	Root Mean Squared
RS	Radial Styloid
SD	Standard Deviation
SLR	Slow Latency Reflex
SRL	Slow Reflex Latency
SSN	Suprasternal Notch
TSK	Tampa Scale of Kinesiophobia
TST	Trunk Stability Task
TT	Thorax Triad
URA	Upper Rectus Abdominis
US	Ulnar Styloid
UT	Upperarm Triad
VAF	Variance Accounted For
VAS	Visual Analog Scale

ACKNOWLEDGEMENTS

I would like to thank my supervisor Dr. Cheryl Hubley-Kozey whose dedication, support, encouragement, and advice was essential to the completion of my studies. Thank you to my supervisory committee, Dr. Shaun Boe and Dr. Derek Rutherford for their time and advice for this and previous work. Special thanks to Dr. John Kozey a valued colleague and mentor who sparked my interest in academia introducing me to my supervisors. I must acknowledge Maj. Raymond Daniel Trudel and Capt. Eric Fagan for their involvement in the physiotherapy screens, and support from the Canadian Department of National Defense and Surgeon General Health Research Program in backing the participants time commitment to this study. I thank participants of the study for their interest and time commitment, without volunteers this work is not possible, I only hope I provided you with some knowledge and entertainment. Erin Barry, Lauren Baikie and periodically members of the Joint Action Research (JAR) lab all deserve credit for assistance with data collection. Without funding from the Natural Science and Engineering Research Council (NSERC), Nova Scotia Health Research Foundation (NSHRF), the School of Biomedical Engineering and the Nova Scotia Graduate Scholarship this research would not be possible. Thank you, Binnie Langley, you are the best man for the fabrication of many of the wonderful wooden structures for this study. Finally, I would like to extend a huge thanks to my lovely wife Melissa Quirk for emotional support, editing services for my comprehensive exams, and assistance with the highs and lows that accompanied this journey.

CHAPTER 1 INTRODUCTION

Musculoskeletal (bones, joints and muscles) disorders represent the second greatest contributor to societal disability worldwide and are associated with significant economic, individual and societal burden (March et al., 2014). Low back pain (LBP) is the most common musculoskeletal disorder, and reports indicate that 15-45% of the population at any given point in time experiences LBP, with prevalence increasing with age (Hoy et al. 2014). All activities of daily living such as walking, lifting etc., place loads on the lower back (McGill, Grenier, Kavcic, & Cholewicki, 2003a) which may explain why individuals with LBP experience difficulties performing many of these activities (Froud et al., 2014). Unfortunately, reducing activity levels can negatively influence overall health and wellbeing creating a plethora of chronic problems that can impact healthy aging. Furthermore, maintaining spinal joint function is necessary for physical function as the trunk is the largest segment of the body where control of this segment is essential for executing many tasks (walking, balancing, and lifting, etc.) in a safe and effective manner (Grabiner et al., 2007). Thus, understanding how LBP develops could be important to maintain spinal joint and overall health.

The problem is that the link between the pain, disability and a specific anatomical structure has been difficult for many LBP complaints, resulting in "non-specific LBP" as the most common classification. Non-specific LBP is believed to be the result of soft tissue injuries following excessive strain on spinal tissues causing inflammation (Queiroz et al., 2014; Solomonow, Zhou, Lu, & King, 2011), pain and disability (Panjabi, 2003; de Queiroz et al., 2015). In most individuals, pain associated with the initial low back injury (LBI) recovers spontaneously (Stanton et al., 2008) reflecting reduced inflammation (Sterling, Elliott, & Cabot, 2013) and tissue remodeling. However, in some, pain may persist beyond the duration of typical inflammation and tissue repair (3 months) and is defined as chronic non-specific LBP (Wasiak et al., 2009). The causes of non-specific LBP are not clear; however, theoretical and some empirical evidence support that spinal instability is a potential mechanism for LBI and subsequent inflammation and pain (McGill, Grenier, Kavcic, & Cholewicki, 2003a; Panjabi, 2003; Reeves, Cholewicki, van Dieën, Kawchuk, & Hodges, 2019).

Excessive vertebral body motion is one mechanism that can produce painful tissue strains and represents the clinical version of spinal instability (Panjabi, 1992a; 1992b; 2003). This clinical definition shares features with the mechanical definition of spinal stability where, if sufficient, potential energy within vertebral structures can resist excessive motion (Bergmark, 1989; Crisco & Panjabi, 1992; Crisco, Panjabi, Yamamoto, & Oxland, 1992; McGill, Grenier, Kavcic, & Cholewicki, 2003a). However, in the mechanical definition, instability is defined as catastrophic buckling, or an inability to restore equilibrium, rather than Panjabi's criteria of pain associated with excessive motion. According to Panjabi's model, all tissues that surround the spine control vertebral body motion. Tissues are categorized into three systems: the passive (osteoligamentous), active (musculotendinous), and neural controller (Panjabi, 1992a; 2003). It is thought that these systems work together to produce forces and moments to maintain spinal stability during the performance of fundamental tasks. Thus, in this theoretical work the interrelationship among systems is essential to maintain healthy spinal function and LBI prevention.

Given that all systems interact in an attempt to maintain spinal stability, Panjabi proposed that a deficit within an individual system can increase the risk of LBI (Panjabi, 1992a; 1992b; 2003). While Panjabi's model is primarily theoretical, there are two pieces of evidence that provide partial support: i) indirect evidence from cross-sectional studies in those with suspected deficits, and ii) a few studies showing that lower measured function in spinal systems are associated with injury risk.

The first piece of empirical evidence comes from studies showing that populations with spinal system deficits such as older adults (Bressler, Keyes, Rochon, & Badley, 1999; Cho et al., 2014; Gourmelen et al., 2007; Hoy et al., 2014) and those with a history of LBP (defined as a previous LBI that occurred at any time in one's life) (Hancock et al., 2015; Mitchell et al., 2010; Stanton et al., 2008; Taylor, Goode, George, & Cook, 2014) have both a higher prevalence of LBP and are at increased risk of experiencing future LBI. This higher risk of LBI is suspected to be related to cross-sectional and longitudinal evidence that osteoligamentous (Fujiwara et al., 2001; Galbusera et al., 2014; Hicks, Morone, & Weiner, 2009; Wang, Videman, & Battié, 2012; de Schepper et al., 2010), musculotendinous (Anderson & Quinn, 2015; Anderson

et al., 2012; D'hooge, Cagnie, et al., 2012a; Fortin, Videman, Gibbons, & Battié, 2013; Whittaker, Warner, & Stokes, 2012), and neural structures (Bednar, Orr, & Simon, 1995; Franchi, Zaccherotti, & Aglietti, 1995; Goble, Coxon, Wenderoth, Van Impe, & Swinnen, 2008; Ludwig, Mobargha, Okogbaa, Hagert, & Ladd, 2015; Shaffer & Harrison, 2007; Solomonow et al., 2011) are different in older adults or those with LBP (current or a history of) relative to young asymptomatic controls. These structural differences are accompanied by different spinal system functions as summarized in Figure 1.1. Older adults and those with LBP experience reduced trunk flexor and extensor muscle strength (Keller, Johansen, Hellesnes, & Brox, 1999; Newton, Thow, Somerville, Henderson, & Waddell, 1993; Sinaki, Nwaogwugwu, Phillips, & Mokri, 2001; D. K. A. Singh, Bailey, & Lee, 2013), impaired trunk proprioception (Brumagne, Cordo, & Verschueren, 2004; Claeys, Brumagne, Dankaerts, Kiers, & Janssens, 2010; Goldberg, Hernandez, & Alexander, 2005; Laird, Gilbert, Kent, & Keating, 2014; Tong, Mousavi, Kiers, & Ferreira, 2015), and delayed/ attenuated reflexes (Hwang, Lee, Park, & Kwon, 2007; Klass, Baudry, & Duchateau, 2011; Ramprasad, Shenoy, Singh, Sankara, & Joseley, 2010; Reeves, Cholewicki, & Milner, 2004; Shenoy, Balachander, & Sandhu, 2013) relative to asymptomatic controls. Furthermore, because of greater likelihood of severe to moderate disc degeneration in older adults and those with LBP (Fujiwara et al., 2001; Galbusera et al., 2014; Hicks et al., 2009; Wang et al., 2012; de Schepper et al., 2010) functionally these populations should have increased spinal joint laxity relative to young asymptomatic controls who experience less disc degeneration (Fujiwara et al., 2001; Galbusera et al., 2014; Sengupta & Fan, 2014). Despite content validity, two problems exist with using these cross-sectional studies to make inferences on relationships between spinal systems and the risk of LBI. The first problem is that without direct measures of individual spinal system function one can only conclude that the collective degeneration in multiple spinal systems, which occurs in older adults and those with a history of LBP, potentially explains increased injury risk. Thus, no association regarding how lower function in an individual spinal system contributes to injury risk is evident. The second problem is that the rate of tissue decline (changes in the structure and function of tissue systems) is heterogeneous, explained by genetic and environmental factors (Demoulin, Crielaard, & Vanderthommen, 2006; Fortin et al.,

2013; McGill, Seguin, & Bennett, 1994; Ribeiro & Oliveira, 2007; Seidler et al., 2009; Sinaki et al., 2001); hence these studies show, despite variability, that the signal measured in older adults and those with a history of LBP differ enough from asymptomatic controls to explain LBI risk (Figure 1.2). To address these problems, more compelling evidence is needed from studies showing a relationship between the structure or function of an individual spinal system and risk of LBI.

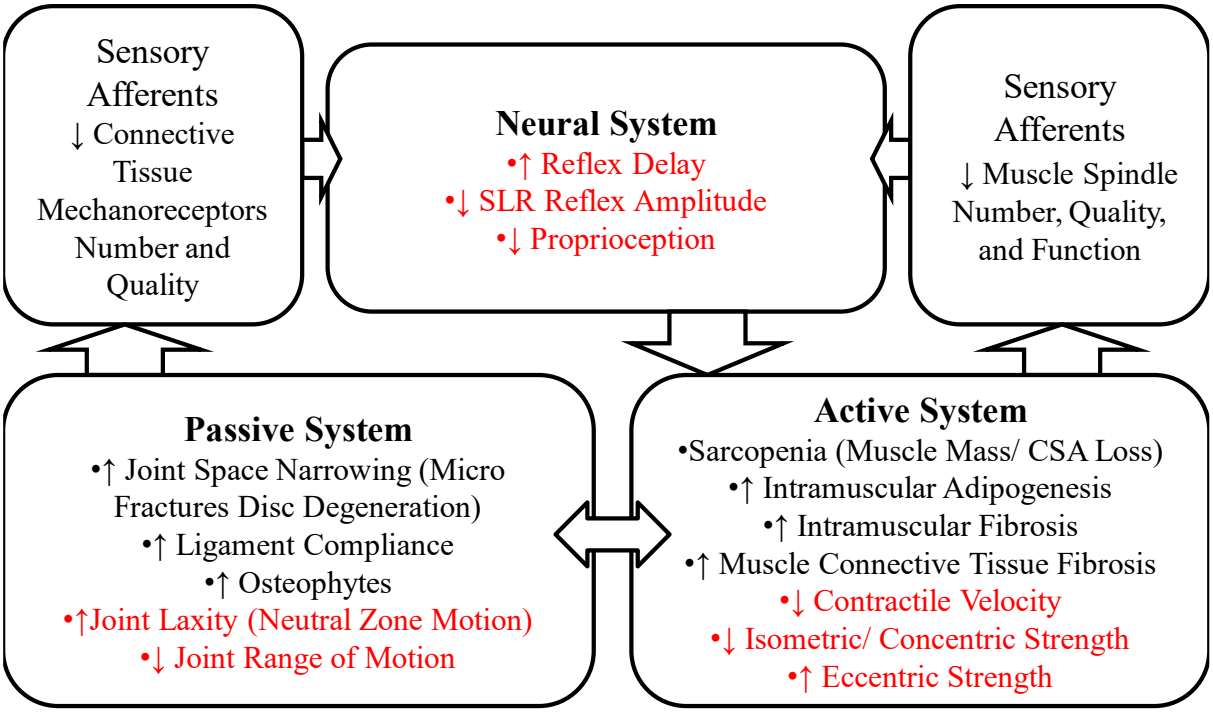

Figure 1.1: Modified version of Panjabi's model of spinal systems, showing general structural and functional differences of older adults and those with low back injuries relative to young asymptomatic controls. Spinal systems are depicted with bold lettering. Structural and functional differences are depicted in black and red lettering respectively. Abbreviations: short latency reflex (SLR), cross sectional area (CSA).

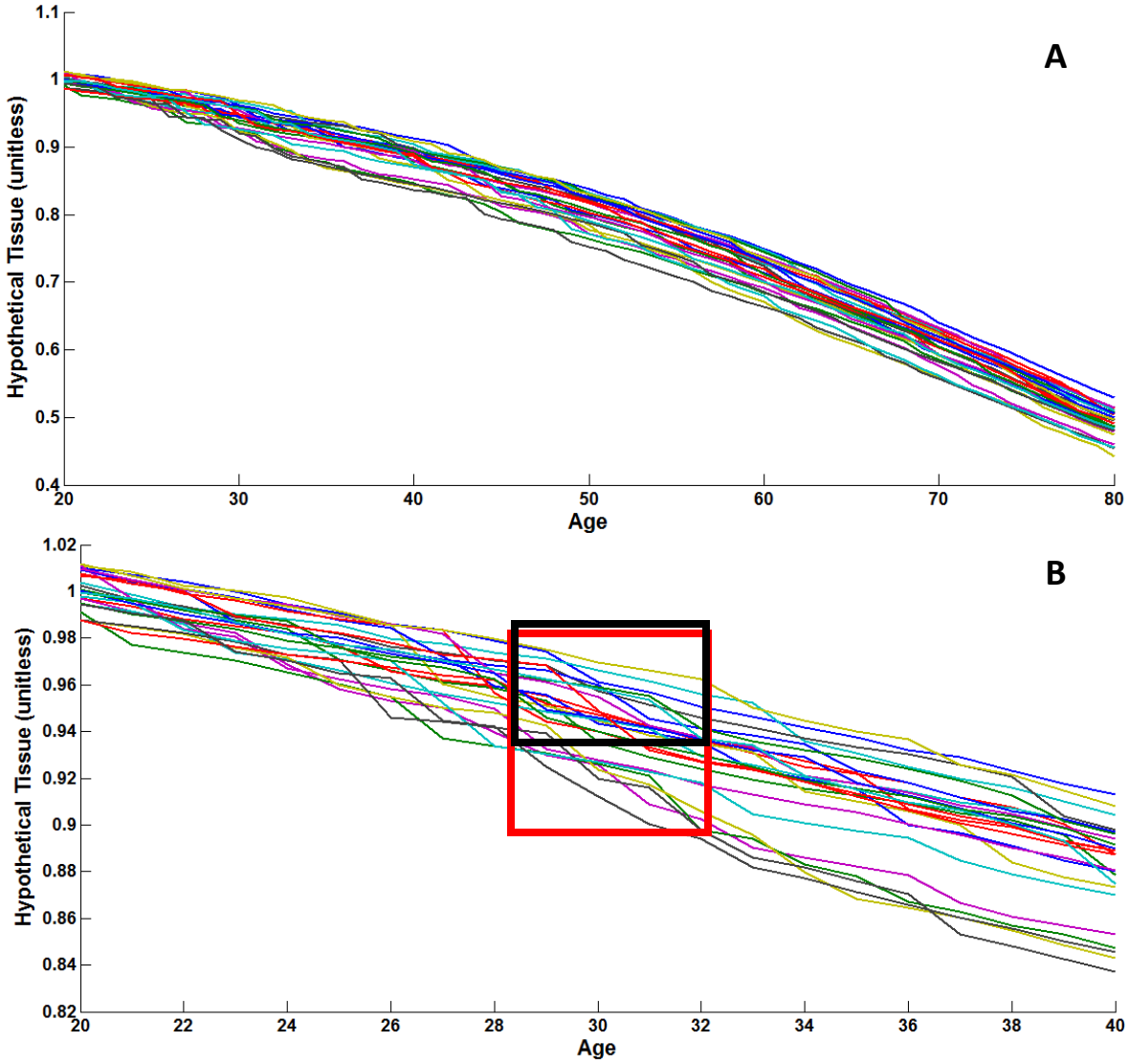

Figure 1.2: Hypothetical model of time-course variability in structural decline of spinal system tissues. A simple model was generated with an exponential decline in a possible spinal tissue score (y-axis), with increasing age (x-axis). Variability was introduced by changing baseline characteristics of tissues at age 20, the annual rate of decline was modified randomly to reflect tissue score improvements via physical activity, or tissue score decrements capturing an annual risk of a mild (10% chance / year) and severe (5% chance/year) low back injury (shown as sudden drops in Figure B). The result is increased variability in hypothetical tissue scores with increased age (A), and differences in the tissue score between those with (red box) or without (black box) low back injuries in a small age range (B).

The second and more compelling empirical evidence to support Panjabi's theory of the risk of LBI comes from studies that make direct comparisons between structural or functional measures of Panjabi's spinal systems and LBI risk. Prospective studies show

that individuals with disc degeneration (Hancock et al., 2015), lower trunk strength (Cho et al., 2014; Keller et al., 1999), delayed trunk muscle offset times (Cholewicki et al., 2005), and impaired trunk proprioception (Claeys et al., 2014) are at an increased risk of experiencing future LBI or re-injuries. These findings are controversial with an equally limited number of studies showing no relationship between measurements of structure or function of the passive (Boos et al., 2000), active (Reenen, Ariëns, Blatter, van Mechelen, & Bongers, 2007) and neural (proprioception) (Silfies, Cholewicki, Reeves, & Greene, 2007) spinal systems and LBI risk. Thus, these studies reporting direct measures provide equivocal evidence that a measurement of lower function in an individual spinal system could increase the risk of LBI, providing partial support of the link inferred through cross-sectional comparisons.

Despite limited empirical evidence that lower function in spinal systems increase the risk of LBI, controversial findings may be explained by a secondary theory proposed by Panjabi that the interaction between systems suggests that a deficit in any spinal system could be compensated for by another (Panjabi, 1992a; 2003). Around the spine (Ito et al., 2015), and other joints (Cammarata & Dhaher, 2012; Levin et al., 2014; Shultz, Carcia, & Perrin, 2004) there is evidence that degeneration in the passive, active, and neural structures might be interrelated. For example, reduced spinal muscle cross sectional area (Ito et al., 2015) and reduced osteoligamentous stiffness (the force required to produce displacement) around the knee and ankle have been weakly ($r=0.09-0.4$) associated with impaired proprioception and attenuated reflex amplitudes around their respective joint (Cammarata & Dhaher, 2012; Shultz et al., 2004). As such, these findings do not support the compensatory hypothesis. However, Panjabi's model referred to changes in neuromuscular patterns (Panjabi, 2005), defined as how the nervous system influences the time varying activation of trunk muscles. Indeed, computational modeling studies have shown that trunk muscles are the primary determinant in controlling spinal stability, particularly when the vertebral bodies are in a neutral alignment (Arjmand & Shirazi-Adl, 2006; Cholewicki & McGill, 1996; Kavcic, Grenier, & McGill, 2004; McGill, Grenier, Kavcic, & Cholewicki, 2003a). However, whether trunk muscles adapt to a deficit to individual spinal systems requires direct comparisons.

To date, much of the evidence supporting a link between spinal systems and neuromuscular patterns comes from cross-sectional studies comparing trunk muscle activation patterns, captured using electromyography (EMG), for both participants with LBP (chronic and history of) and older adults relative to young asymptomatic controls. These EMG studies often utilize dynamic tasks to challenge spinal stability by inducing a changing external load (including body weight) and monitoring how trunk muscles anticipate or respond when acting as both agonist and antagonist. In general, these studies show that older adults and those with LBP recruit trunk muscles with higher agonist and antagonist activation amplitudes (Hodges & Moseley, 2003; Hubley-Kozey, Moreside, & Quirk, 2013; McGill, Yingling, & Peach, 1999; Mehta, Cannella, Smith, & Silfies, 2010; Moreside, Quirk, & Hubley-Kozey, 2013; Quirk & Hubley-Kozey, 2014; van Dieën, Selen, & Cholewicki, 2003), have altered temporal recruitment of the musculature including delayed trunk muscle onset and offsets (Hodges & Richardson, 1999; Hwang et al., 2007; Jacobs, Henry, Jones, Hitt, & Bunn, 2011; Macdonald, Moseley, & Hodges, 2009; 2010; Reeves et al., 2004; Stokes, Fox, & Henry, 2005), and sustained muscle activation in response to changing external moments (Butler, Hubley-Kozey, & Kozey, 2012; D'hooge, Hodges, et al., 2012b; Hubley-Kozey & Vezina, 2002; Hubley-Kozey et al., 2013; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014) compared to the younger asymptomatic cohorts. In these studies, authors deduce that differences in muscle activation patterns and subsequent changes in joint kinetics and kinematics (Figure 1.3) are explained by the deficits in individual spinal systems within these populations relative to young asymptomatic controls (Figure 1.1). However, these cohort studies include confounders that can alter muscle activation patterns, limiting their ability to conclude that the health of an individual spinal system alone leads to muscle activation pattern adaptations.

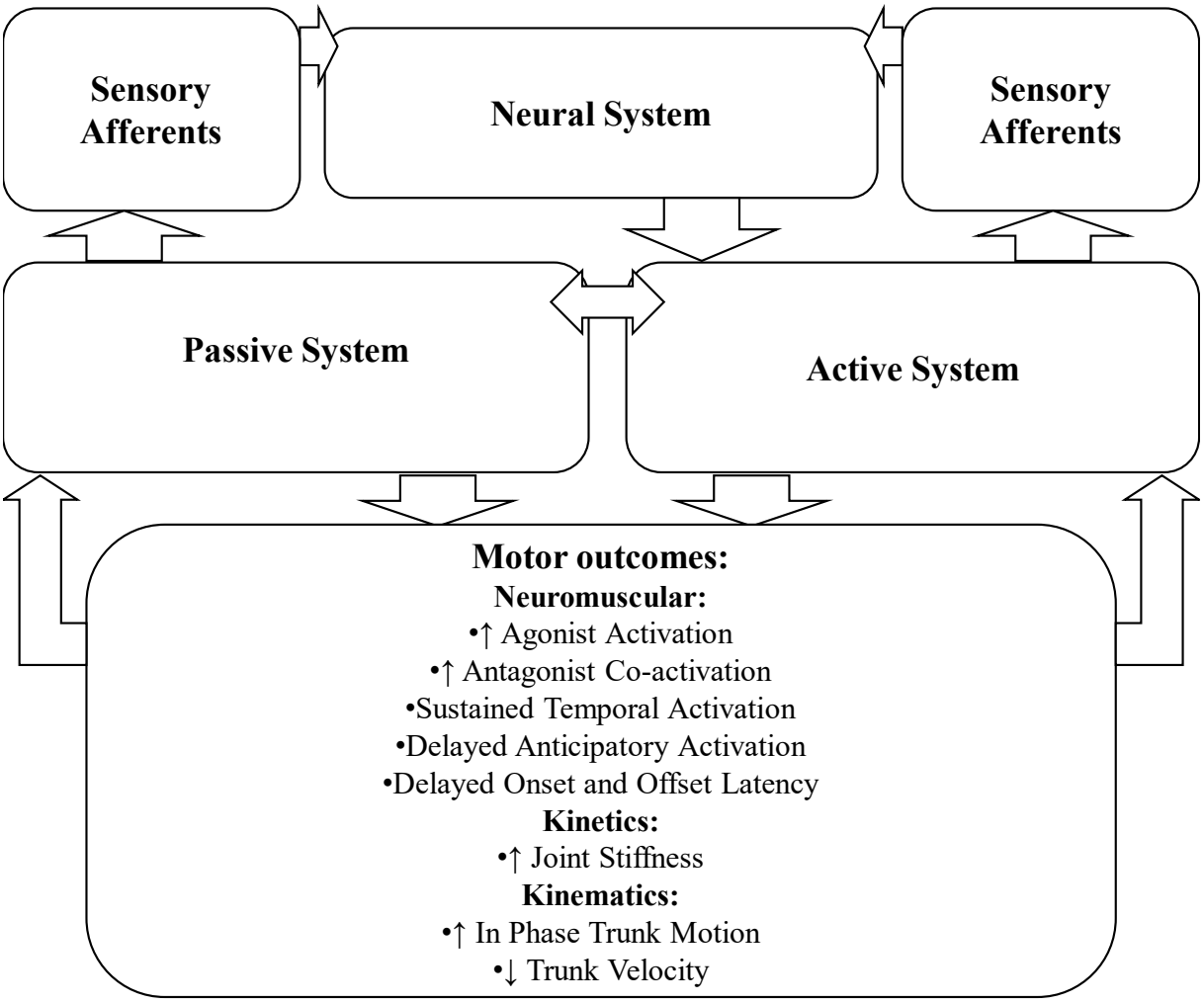

Figure 1.3: Neuromuscular and movement pattern changes identified in populations with suspected or known deficits within spinal systems.

All cross-sectional cohort studies contain potential variables that could explain differences in muscle activation patterns beyond Panjabi's spinal system theory. One confounder is using participants experiencing LBP at the time of testing. Experimental pain studies that temporarily induce pain without changing spinal structures, show that muscle activation patterns change immediately after pain is induced, and these patterns mimic the differences that exist between participants with chronic LBP and asymptomatic controls (Dubois, Piché, Cantin, & Descarreaux, 2011; Hodges, Coppieters, MacDonald, & Cholewicki, 2013; Hodges, Moseley, Gabrielsson, & Gandevia, 2003; Hug, Hodges, & Tucker, 2014; van den Hoorn, Hodges, van Dieën, & Hug, 2014). Thus, using participants that are in pain at the time of testing make isolating

the influence of the individual spinal systems on muscle activation patterns difficult. The first study of this dissertation acknowledges this "pain" confounder and attempted to determine whether trunk muscle activation patterns do indeed adapt to suspected spinal system deficits. Cross-sectional evidence suggests that older adults and those recently recovered from a (not presently in pain) LBI experience similar changes in spinal systems and as such, they should share similar muscle activation pattern adaptations. Indeed, relative to young asymptomatic controls, both groups have different trunk muscle activation patterns measured from EMG. However, no study to date has made a direct comparison of muscle activation patterns between these two groups. While on the surface trunk muscle activation patterns appear to be similar for both groups, there are several differences among methodologies (different tasks and data analysis techniques) that limit direct comparisons and interpretation among studies.

Study one of this dissertation performed a direct comparison of trunk muscle activation patterns used by older adults and those recovered from LBI to determine if a common adaptation exist for both groups supporting an indirect link between the function of spinal systems and muscle activation patterns. Preliminary data shows that the muscle activation patterns of older adults and those recovered from LBI were similar for some features such as higher activation amplitudes (abdominals and back muscles), and less responsiveness, defined as less differential in change in muscle activation pattern amplitudes as external moments change, relative to a control population (Quirk & Hubley-Kozey, 2015). Some of these features are explained by factors beyond lower strength in these populations as increasing the external load of dynamic tasks increases the responsiveness of trunk muscles (Butler, Hubley-Kozey, & Kozey, 2010; Quirk & Hubley-Kozey, 2014), whereas reduced responsiveness was observed in these weaker populations. These groups, however, differ from one another in that older adults had changes consistent with having less ability to modify the time-varying recruitment of trunk muscles, and those recently recovered from LBI had higher antagonistic co-activation than older adults. Common features between these groups support that a link potentially exist between spinal system deficits and muscle activation patterns. However, group differences suggest that the magnitude of deficits to the individual spinal system is

not comparable between these populations, and that specific muscle activation pattern adaptations are potentially linked to a deficit within a single spinal system.

Thus, these results led to the need to determine whether those with a greater deficit within any single spinal system would have specific differences in trunk muscle activation patterns. Only a few studies have tested the associations between measures of osteoligamentous, musculotendinous, and neural tissue function and muscle activation patterns, and this work has not been performed in the spine. Around the knee and ankle, Takai et al., have shown individuals with lower maximum voluntary moment production have higher joint specific (correlated with joint with the strength measure) muscle activation amplitudes to perform a variety of functional tasks (Takai, Sawai, Kanehisa, Kawakami, & Fukunaga, 2008). At the knee joint, Shultz et al., showed that women with increased anterior-posterior knee joint laxity have greater knee muscle activation to stand on a single leg in 30° of knee flexion while anticipating a perturbation (Shultz et al., 2004). They suggested that participants increased their muscle activation to compensate for deficient passive tissue stiffness. Fu et al., showed that male university athletes with lower ankle proprioception (higher reposition error) have increased ankle muscle co-activation in preparation for landing from a drop jump (Fu & Hui-Chan, 2007). These data provide evidence that measurements of lower function within the osteoligamentous, musculotendinous, and neural tissues are linked with muscle activation patterns adaptations as proposed by Panjabi's model. However, these studies are limited as they only established a link between the magnitude of functional measures of an individual joint system with overall muscle activation amplitudes. Cross-sectional comparative studies (see above) suggest that the time-varying recruitment of trunk muscles also adapt to degeneration within spinal systems. Thus, the second set of studies of this dissertation were designed to determine whether the measurement of an individual spinal system function (active, passive and neural) influences muscle activation patterns (including temporal features) in a predictable way.

In summary, while the ability to control spinal stability is paramount in performing activities without the risk of injury and pain, there is a lack of evidence showing the interrelationships between degeneration of individual spinal systems and muscle activation patterns to maintain stability. A theoretical model suggesting that the

muscle activation patterns are fundamental to ensure spinal stability has been proposed, but little empirical work has established how muscle activation patterns might adapt to deficits in individual spinal systems. To date, much of what is known about this relationship is inferred through cross-sectional comparisons between populations expected to have varying levels of spinal (and joint) systems deficits. However, cross-sectional comparisons can be confounded by pain or degeneration in multiple systems, and they do not provide evidence regarding how muscle activation patterns adapt to a deficit within an individual spinal system. Stronger evidence could be revealed by direct functional measurements of individual spinal systems and muscle activation pattern adaptations. However, there exists limited evidence, probing direct relationships between measurable differences within the function of an individual spinal systems and corresponding muscle activation pattern adaptations, and these studies did not consider how these differences impact temporal activation patterns.

The overall goal of this dissertation is to advance our understanding of the factors that contribute to trunk (both abdominal and back extensor) muscle activation patterns given their role in maintaining spinal stability during fundamental movements. Findings of this dissertation aim to advance our knowledge regarding how muscle activation patterns interact with deficit joint function to maintain joint stability. These findings have implications towards understanding how musculoskeletal injuries develop and may provide information on how they can be prevented and/or managed (in the workplace or recreational activities) to ensure joint health can be maintained while individuals age allowing them to maintain their physical function and remain physically active. Knowledge from this dissertation has direct application towards understanding spinal stability in hopes that it can provide empirical evidence for clinical practise regarding the treatment and prevention of lower back injuries.

1.1 PURPOSE

The overarching aim of this dissertation was to better understand the relationships between individual spinal system function consistent with the three spinal system model of Panjabi and trunk muscle activation patterns during fundamental tasks. The following

objectives used a series of cross-sectional comparative studies between those with suspected or measurable deficits within spinal systems to address the overarching aim.

1.2 OBJECTIVES

One (a): To directly test whether differences exist in trunk muscle activation patterns during the performance of highly controlled dynamic tasks designed to challenge spinal stability between older adults and those recently recovered from a low back injury, both of which are suspected to have deficits to multiple spinal systems.

One (b): To determine whether the differences in muscle activation pattern features between older adults and those recovered from a low back injury to a reference young asymptomatic control group are similar in pattern and magnitude.

Two: To determine whether individuals with direct measures of individual spinal system function that are consistent with a lower function based on risk of low back pain ((a) active, (b) passive, and (c) neural) have different trunk muscle activation patterns during a highly controlled task designed to challenge spinal stability than those who do not. A secondary purpose was to determine whether these observed adaptations would differ between those who have or have not recently recovered from a low back injury.

1.3 HYPOTHESIS

Objective one (a): No difference will exist in the muscle activation patterns used to complete highly controlled dynamic tasks designed to challenge spinal stability between older adults and those recently recovered from a low back injury.

Objective one (b): Older adults and those recently recovered from a low back injury will have muscle activation pattern differences compared to an asymptomatic younger control population.

Objective two: The primary hypothesis is that those with lower function within specific spinal systems will have specific adaptations to muscle activation patterns as indicated below and secondly these adaptations would be similar in both those who have or have not recently recovered from a low back injury.

a) Those with lower trunk strength (maximum isometric torque) will have higher agonist, antagonist activation amplitudes, and greater responsiveness to

changing external moments. Features known to change with increasing external task load.

b) Those with lower passive stiffness will have higher antagonist activation amplitudes, and evidence of reduced responsiveness of trunk muscles to changing external moments consistent with adaptations to increase active trunk stiffness.

c) Those with delayed trunk muscle reflexes will have trunk muscle activation patterns consistent with increasing intrinsic stiffness of the trunk including higher antagonist co-activation amplitudes and reduced responsiveness of trunk muscles to changing external moments.

1.4 DISSERTATION STRUCTURE

Chapter 1 introduces Panjabi's model of spinal stability, how deficits within individual spinal systems might increase the risk of instability events, and how trunk muscle activation patterns might compensate for these deficits. The overarching purpose of this dissertation and two objectives are stated along with the specific hypothesis.

Chapter 2 provides a comprehensive overview of the general methodology employed to carry out the objectives of the dissertation. Specifically, this section was divided into five sub-sections. Section 2.1 describes the general research design. Section 2.2 outlines participant recruitment for Objective 1 (Chapter 3) and Objective 2 (Chapter 4-6). Section 2.3 outlines electromyography setup (Chapters 3-6), a description of the spinal system tests (Chapters 4-6), the kinematic setup (Chapters 4-6), and a thorough description of the horizontal transfer task and normalization tasks (Chapters 3-6). Section 2.4 provides an in-depth review of data analysis used in this dissertation including a description of the pattern recognition technique (principal component analysis) used to aid in interpreting temporal patterns. Finally, Section 2.5 explains the generalized outline of the statistical analysis used in this dissertation.

Chapter 3 addresses the primary objective and first sub objective of the dissertation. This experiment titled "Do older adults and those recovered from low back injury share common muscle activation adaptations" was published in the Journal of Motor Behavior on April 2018: volume 51, issue 2, pages 1-17.

Chapter 4 addresses sub-objective 2a of this dissertation written as an independent manuscript to be submitted for scientific publication titled “Trunk muscle activation patterns differ between those with low and high back extensor strength during a controlled dynamic task”.

Chapter 5 addresses sub-objective 2b of this dissertation written as an independent manuscript to be submitted for scientific publication titled “Trunk muscle activation patterns adapt to impaired transverse plane trunk stiffness in unique ways for asymptomatic and recovered low back pain participants”.

Chapter 6 addresses sub-objective 2c of this dissertation written as an independent manuscript to be submitted for scientific publication titled “Trunk muscle activation patterns differ between those with delayed trunk muscle reflexes in asymptomatic and recovered low back pain participants”.

Chapter 7 contains the conclusion of this dissertation, providing a summary of key results and their implications. It identifies the limitations of these studies and gives directions for future studies.

Appendix A contains a focused analysis on the trunk kinetic model used in Chapters 4-6, including how the outcome measures of this analysis differed from a previously used hypothetical model of the changing trunk moments associated with the horizontal transfer task.

CHAPTER 2 GENERAL METHODOLOGY

2.1 General Research Design

For objective one a cross-sectional design compared data collected as part of an ongoing study aimed at determining if trunk muscle activation patterns differ between participants across a wide age range (18-80 years old) with and without low back pathology. To date, 155 participants have completed studies involving the characterization of trunk muscle activation patterns during the completion of controlled dynamic tasks in the Neuromuscular Function laboratory. These participants represent three cohorts, asymptomatic controls (18-64 years old), older adults (asymptomatic with an age >65 years old), and those recently recovered but in the sub-acute phase of a low back injury (LBI) (18-64 years old). Objective two also uses a cross-sectional design. However, while participants from this objective were recruited using a protocol(Section 2.2), underwent a similar electromyography experimental set-up and analysis (Section 2.3.1 & 2.4.2) and performed the same controlled horizontal transfer task (Section 2.3.4) to the Neuromuscular Function lab, these participants also underwent a series of new experimental tests to characterize individual spinal system function (Section 2.3.2 & 2.4.1). Given the overlap between studies, the common methodology is primarily consistent with the protocols for participants in objective 2, with divergence indicated within the document.

2.2 Participant Recruitment

2.2.1 Objective 1

To address objective one, three groups of participants (asymptomatic controls, recovered LBI (rLBI), and older adults) were identified within our database. Asymptomatic controls were defined as not having experienced a LBI one-year before the time of testing. Older adults are participants with an age exceeding 65 years old that have not experienced a LBI one-year before the time of testing. Recovered sub-acute LBI participants experienced a LBI 4-12 weeks before testing. With an injury being defined as

"pain between the ribs and gluteal fold causing [the participant] to seek medical attention, and/or limit activities of daily living (including time off work) for three days" (Ozguler, Leclerc, Landre, Pietri-Taleb, & Niedhammer, 2000). However, these participants self-report that they are recovered having resumed normal activities of daily living (including work)(de Vet et al., 2002), as well as having minimal pain and disability (Section 2.3). To maximize the effect size between age groups, young control and recovered sub-acute low back injured participants were restricted to an age range of 20-45 years old consistent with previous studies (Quirk & Hubley-Kozey, 2014). To minimize the influence of demographic confounders younger asymptomatic participants were matched to the smaller older adult cohort to ensure participants were selected with a similar height, mass, and sex (Hubley-Kozey, Butler, & Kozey, 2012). To minimize the confounding influence of age (Quirk & Hubley-Kozey, 2014) participants recently recovered from an LBI were age matched with the young asymptomatic controls.

2.2.2 Objective 2

To address objective two, two groups of participants (asymptomatic and rLBI) were recruited using the same definitions as Objective 1 (Section 2.2.1). For this objective, all participants were active members of the Canadian Armed Forces at the time of testing. Participant recruitment was different for the asymptomatic and rLBI. Asymptomatic participants expressed interest for the study in response to basewide e-mails and posters distributed to the Canadian Forces Bases (CFB) located in the Halifax Regional Municipality where they would contact the research team directly. Participants recovered from an LBI were recruited in collaboration with the military Hospital. If a forces member accessed medical treatment specific to their lower back they were asked if they would be interested in the study.

All interested parties were informed of the studies objectives and time commitment by a member of the research team using a standardized script. Consistent with objective 1 this script included a health screening questionnaire to exclude participants if they acknowledged having a previous abdominal (except appendectomy that does not affect abdominal muscle integrity) or back surgery, spinal fractures, spinal tumours, cardiorespiratory or neurological conditions. This questionnaire also included additional

questions to exclude: i) asymptomatic participants if they reported experiencing a LBI within the past 12-months, causing them to seek medical attention, and/or limit activities of daily living for three or more days (including time off work), and ii) participants with a recovered LBI if their most recent pain event has lasted beyond the sub-acute phase (>12 weeks) and if they have experienced a LBI three months prior to their most recent event, which would define the participant as having chronic or recurrent LBP respectively (Delitto et al., 2012).

2.3 Procedure (Protocol and Data Collection)

Participants met with members of the research team on two sessions. In the first session, participants were given a brief overview and signed the informed consent. The participant then underwent a physiotherapy assessment to ensure they met the eligibility of the study. To ensure participants had minimal pain and disability they completed a Visual Analog Scale (VAS) (Wewers & Lowe, 1990) and Roland Morris Disability (RMD) questionnaire (Roland & Morris, 1983). To be eligible for the study participants had to have minimal pain (VAS <2/10) and disability (RMD <8/24). The threshold for pain is consistent with the lower bounds of mild pain (Boonstra, Preuper, Balk, & Stewart, 2014; Jensen, Chen, & Brugger, 2003), and is the minimum clinically detectable change in pain using the VAS (Boonstra et al., 2014; Jensen et al., 2003; Lee, Hobden, Stiell, & Wells, 2003; Turner et al., 2004). The threshold for disability is consistent with the level of disability associated with minimal pain (Turner et al., 2004), and is the minimum score required to capture a clinically relevant change in disability (Stratford, Binkley, Riddle, & Guyatt, 1998). If eligible a registered physiotherapist conducted a series of clinical tests designed to screen for postural (scoliosis and kyphosis), and neurological (reflexes, myotomes, and dermatomes) impairments that were not reported by the participant. Identified postural abnormalities, dermatome and myotome test scores below 3/5, and abnormal reflexes would result in participant exclusion. However, no participant was excluded from the study during the first session. Once the clinical assessment was completed participants were informed of the upcoming experimental tasks that would occur in their second (data collection) session.

For the second data collection session when participants arrived at the laboratory, they filled out questionnaires regarding level of kinesiophobia (Tampa Scale of Kinesiophobia (TSK) (Kori, Miller, & Todd, 1990)), and pain catastrophizing (Pain Catastrophizing Scale (PCS) (Sullivan, Bishop, & Pivik, 1995)) in response to any type of musculoskeletal injury. Basic anthropometric measures including mass (kg) and height (m) were measured using a sliding balance scale and standiometer. Self-reported age (years), sex (man or woman), and handedness were collected. Physical activity was recorded using self-report levels of aerobic activity ((# sessions > 30 minutes / week, causing them to sweat or increase breathing rate)), participation in abdominal (core) training (sessions/ week), and strength training (sessions/ week). Finally, the participant was asked questions of their self-perceived occupational task demands to categorize participants as having a heavy job if they "frequently (2 or more hours of the day) lift objects weighing more than 23kg" (Seidler et al., 2009). These were collected as psychosocial perceptions of pain and fear of movement (Moseley & Hodges, 2006; Ross, Mavor, Brown, & Graham, 2015; Svendsen, Svarrer, Laessoe, Vollenbroek-Hutten, & Madeleine, 2012), level of physical activity (Carvalho, Vasconcelos, Gonçalves, Conceição, & Vilas-Boas, 2010; Granacher, Gollhofer, & Strass, 2006; Hortobágyi & Devita, 2006), and occupational loading (Marras et al., 2005; Seidler et al., 2009) have all been associated with changes in spinal structures and/or changes in muscle activation patterns, and could act as confounders to the studies outcome measures.

2.3.1 Electromyography Setup

Participants were then prepared for data collection starting with the setup of electromyography (EMG) equipment. For all objectives, standard skin preparation included shaving excess hair and light abrasion of the skin using alcohol swabs, to minimize skin/ surface electrode impedance. Single use disposable Ag/AgCl surface electrode pairs (10mm diameter, Red Dot, 3M, London, Ontario, Canada) were positioned by a trained researcher on standardized locations in line with the muscle fibres, in a bipolar configuration (interelectrode distance 30mm), over 12 bilateral trunk muscle sites (Figure 2.1) (Butler et al., 2010). Minor adjustments were made to accommodate anatomical differences confirmed by palpation and submaximal validation

exercises for specific muscle sites (Kendall, McCreary, & Kendall, 1983). Abdominal sites included: The lower (LRA- the midpoint between the pubic symphysis and umbilicus) and upper rectus abdominis (URA- the midpoint between the umbilicus and the sternum) (Gilleard & Brown, 1994); the anterior (EO1- over the eight rib at a 30° angle) (Ng, Kippers, & Richardson, 1998), lateral (EO2- approximately 15cm lateral to the umbilicus at a 45° angle) (McGill, 1991), and posterior (EO3- halfway between the iliac crest and the lower portion of the ribcage) (Nouwen, Van Akkerveeken, & Versloot, 1987) sites of the external oblique; and the internal obliques (IO- centered in the triangle formed by the inguinal ligaments, lateral border of the rectus sheath and the line between the two anterior superior iliac spine) (Ng et al., 1998). Back extensor sites included: the lumbar erector spinae spanning the lumbar 1 and lumbar 3 spinous processes positioned 3 and 6 cm lateral to capture the longissimus (L13, L33) and iliocostal (L16, L36) fibres of the erector spinae respectively (Vink, van der Velde, & Verbout, 1987); the superficial fibres of the multifidus (2 cm lateral to the 5th lumbar spinous process (L52)), and the superficial fibres of the posterior quadratus lumborum (8cm lateral to the 4th lumbar spinous process (L48)) (McGill, Juker, & Kropf, 1996).

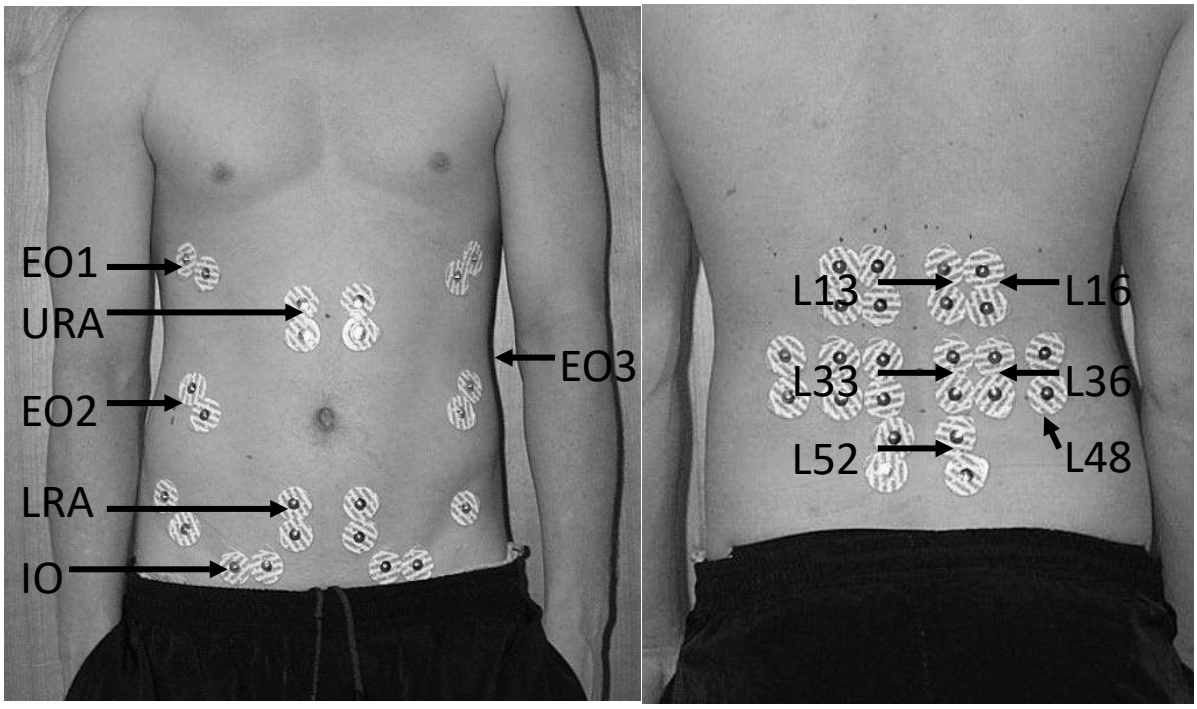

Figure 2.1: Bilateral surface electrode placement of abdominal (left) and back extensor sites (right). Surface electrode positions denoted by their abbreviated form.

EMG signals were pre-amplified (500x) and variably amplified using 3x AMT-8 EMG systems (Bortec^{TM}, Canada, Bandpass 10-1000Hz, CMRR=115dB, input impedance 10GΩ) to maximize EMG activity captured within the analog range (± 5V) of a 16 bit A/D card (NDI PCI-6259, 16 Bit, National Instruments, Austin, TX), without signal clipping. For all experimental tasks, analog signals were sampled at 2000Hz using a custom Labview program (National Instruments, Austin, TX). This program was designed to display EMG signals to validate electrode placement using specific exercise task to isolate muscles (Winter, Fuglevand, & Archer, 1994) and to ensure high signal quality (high visual signal to noise ratio). For each experimental trial, all data were stored on an internal hard drive for post processing.

2.3.2 Spinal System Tests (Objective 2)

The following outlines the spinal system tests unique to objective two that were collected in a single data collection session. The tests are presented in the order participants were exposed to each.

2.3.2.1 Transverse Plane (Passive) Stiffness Test

To measure passive stiffness of the lumbar spine, the participant was secured to two rigid bodies designed such that the lower rigid body would rotate relative to a fixed upper rigid body (Drake & Callaghan, 2008; McGill et al., 1994). To adjust the equipment to the participant's anthropometrics the participant was asked to stand on the lower rigid body platform. The height of the lower platform was then raised or lowered such that the participant's posterior superior iliac spine was leveled with a rigid body covered in 1" of dense foam (Figure 2.2). This design ensured pulley rotation occurred 8cm anterior to the posterior superior iliac spine (cross bar centroid) approximating the L4/L5 transverse plane center of rotation (Dumas, Chèze, & Verriest, 2006; Reed, Manary, & Schneider, 1999). Next, the lower rigid body was aligned to the upper rigid body moving it anteriorly or posteriorly until the participant felt they were in a comfortable standing position when they rested their back on the upper rigid body. Once in position nylon straps secured the participant to the: lower rigid body below the iliac

crest, and the upper rigid body by crossing lower straps located approximately at the bottom of the 12th rib to the participant's contralateral shoulder (Figure 2.2). These straps were designed to be tight, with additional pressure to the upper rigid body straps applied by the participant's hands to minimize the risk of motion artifact within the straps to aid in the assumption that motion of the lower rigid body approximated pelvis motion.

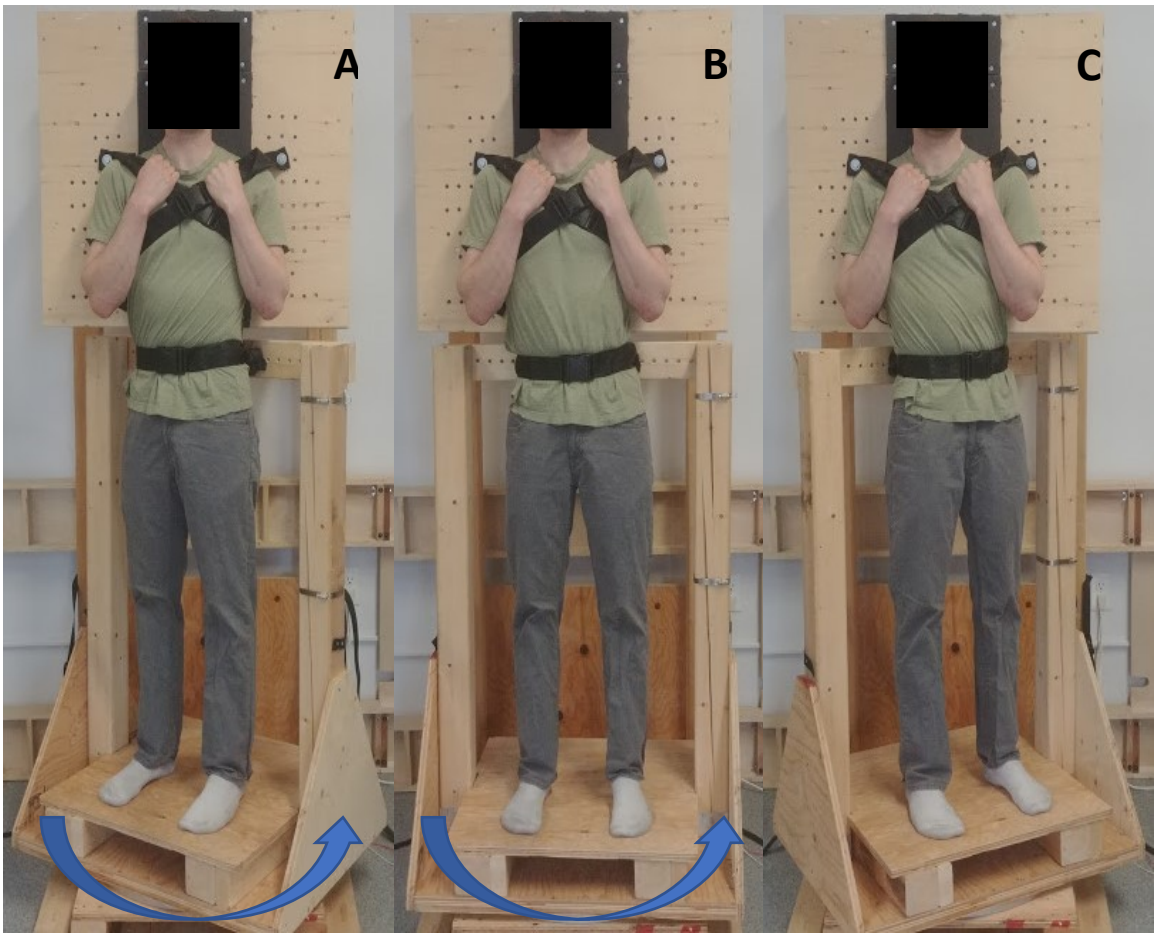

Figure 2.2: Visualization of the passive stiffness test. Height of lower rigid body was adjusted by raising the platform. The participant was secured to both the upper and lower rigid body using nylon straps. Using a pulley system under the lower rigid body all trials included the participant being rotated from one maximum comfortable rotation position to another (A→C or C→A), passing through their neutral spine (B) position.

With the participant secure, they were requested to actively rotate to the left and right until they feel they could comfortably go no further. Using a nylon strap, a hard stop was set to prevent rotation beyond this point ensuring the participant felt safe. The participant was requested to actively rotate to their "natural and comfortable forward-

facing standing position" defined as their "neutral spine." A single trial recorded the participant's neutral position as they would "stand as relaxed as possible" for 3 seconds. This step both determined the participant's neutral spine bias to express trunk rotation and acted as a measure of resting EMG activity to be used in a custom biofeedback program.

Using Labview™ software, resting baseline EMG from this trial was used to calculate the mean and standard deviation of the root mean squared signal from 4 bilateral muscle sites (IO, EO1, L52, L16). This information was used to set a threshold within an online biofeedback program designed to notify the participant and researcher if EMG activity exceeds a resting threshold. For all passive stiffness trials, the biofeedback program constantly analyzed 200 ms blocks of EMG activity. If EMG activation of any muscle exceeded the baseline threshold (mean+ three standard deviations (SD)) an auditory signal was emitted and the participant was encouraged to relax while the trial was repeated. This biofeedback program ensured muscle activation was minimal during the passive rotation trials to reduce the stiffness contributed by the muscular system (Arjmand & Shirazi-Adl, 2006; Brown & McGill, 2010).

The participant experienced ten passive rotation trials (five left and five right). Starting in either maximum right or left rotation participants were passively rotated to the opposite position using a (10 cm radius) pulley located underneath the lower rigid body, fixed to a steel cable, while they were encouraged to remain "as relaxed as possible" (Figure 2.2A→C). An angular potentiometer (3547S, Bourns Technology, Riverside CA) measured angular displacement of the lower rigid body. A linear load cell (FTD-IU-200, Schaevitz Sensors, Hampton VA) measure tension in the steel cable. Using an auditory cue emitted from a Labview program a trained researcher pulled the steel cable approximately 1.2cm every second (tone interval) to rotate the lower rigid body at a nearly constant rate (~ 6.9°/s). For each trial, EMG, load cell, and potentiometer data were sampled at 2000Hz using the common A/D (Section 2.3.1) and stored for post processing.

2.3.2.2 Reflex (Neural) Test

Reflex testing was performed using a HUMAC Norm dynamometer (Model 770, CSMI Solutions, Stoughton, MA) aligned to a custom-made lower body restraint (Figure

2.3). For this test, the participant was asked to position themselves with respect to the lower body restraint (vertically aligned flush with a horizontal bar positioned 8cm posterior to HUMAC dynamometer center of rotation (approximately the L4/L5 center of rotation (Dumas et al., 2006; Reed et al., 1999))). By adjusting platform height, the participant posterior superior iliac spine was positioned to an 8x4" block covered in 2 cm of dense foam. Using nylon straps the participant was secured to the lower rigid body. A steel arm covered with 2 cm of dense foam, attached to the dynamometer, was positioned 2cm inferior to the participant's scapular spine. Nylon straps secured the participant to the steel arm (Figure 2.3).

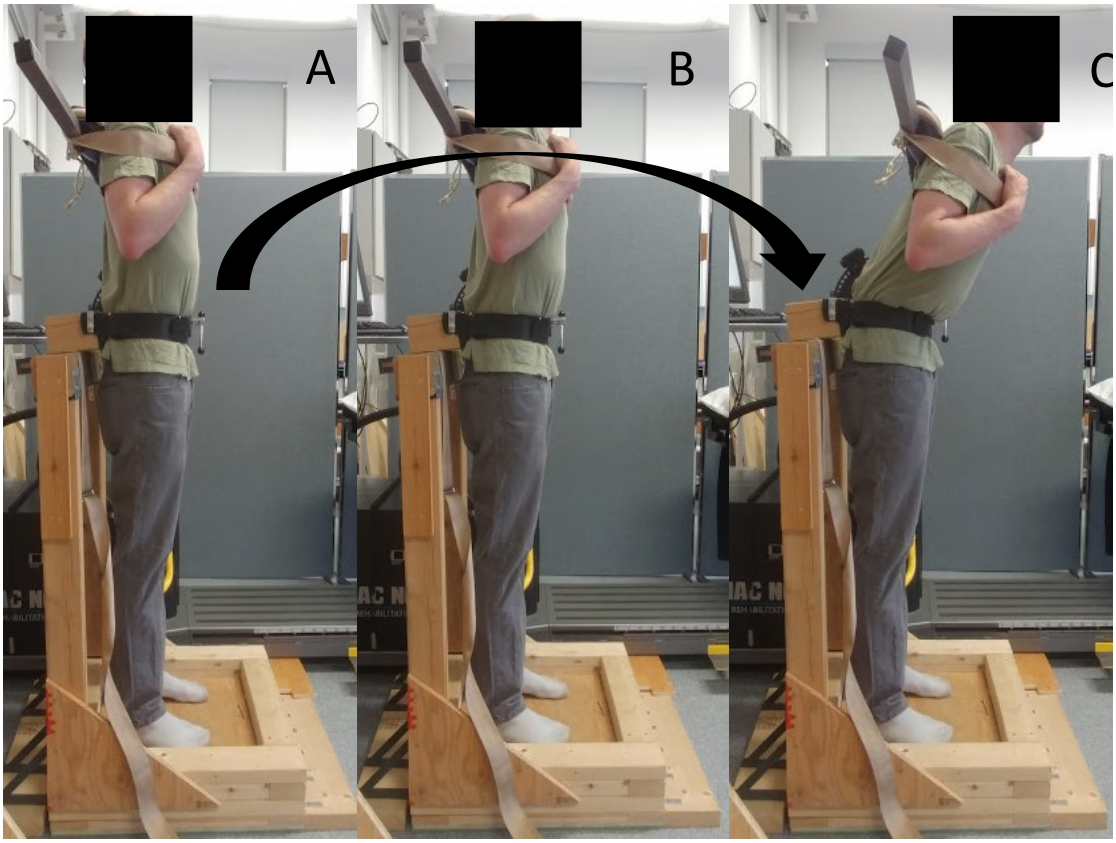

Figure 2.3: Visualization of the reflex test. Height of the lower rigid body was adjusted by raising the platform. The participant was secured to the HUMAC dynamometer using a steel bar attached to nylon straps. Using the HUMAC software in isokinetic mode the participant was perturbed from 3˚ extension (A) to 12˚ trunk flexion (C) or visa-versa passing through their comfortable neutral standing position (B) for each trial.

Once secured the participant was requested to assume a comfortable standing position, defined as neutral standing (0°) (Figure 2.3B). Electronic stops were set at 3˚ extension and 12˚ flexion (Figure 2.3A and C respectively) using the HUMAC software.

This range of motion represents 20% of standard lumbar spine range of motion in flexion and extension (Van Herp, 2000), reducing the risk of injury caused by excessive trunk motion. Participants were instructed, “for this test they would be flexed forwards (Figure 2.3A→C) or extended backwards seven times in each direction with an unexpected time between these motions”. To limit the confounding influence of visual reflexes (Goodworth & Peterka, 2009) or background muscle activation (Larivière, Forget, Vadeboncoeur, Bilodeau, & Mecheri, 2010; Lee, Cholewicki, & Reeves, 2007a), participants were instructed to “keep their eyes closed and relax their trunk muscles during the entire test”. However, participants were also requested to “keep tension in their neck muscles to prevent unwanted head motion”. Using the isokinetic setting 7 flexion and extension perturbations were produced with a maximum velocity of 120˚/s every 7 seconds (Sánchez-Zuriaga, Adams, & Dolan, 2010). A single (110s) trial captured all perturbation events while EMG and HUMAC (position and moment) signals were simultaneously sampled at 2000Hz using the common A/D (Section 2.3.1) and stored for post processing.

2.3.2.3 Strength Test (Active)

Following normalization task (Section 2.3.5), EMG surface electrodes were removed and the participant's trunk strength (flexor and extensor moment production) was measured during two maximum voluntary isometric contraction (MVIC) exercises. Participants were positioned in a prone or supine crook lying position with the HUMAC Norm Dynamometer (Computer Science Medicine Inc, Strongton, MA, USA) arm positioned anterior and inferior to the clavicle for trunk flexion, or superior and posterior to the spine of the scapula for trunk extension (Hasue, Fujiwara, & Kikuchi, 1980). The HUMAC centroid was positioned approximately 5cm anterior to the posterior superior iliac spine, in line with the iliac crest, and non-elastic straps were secured to anchor the pelvis and shank. Following gravity correction, participants performed two trials for these contractions (flexion and extension). For these trials, the participants were instructed to “maximally produce their strongest effort and then hold the contraction for three seconds”. HUMAC (position and moment) signals were simultaneously sampled at 2000Hz using the common A/D (Section 2.3.1) and stored for post processing.

2.3.3 Kinematic Setup (Objective 2)

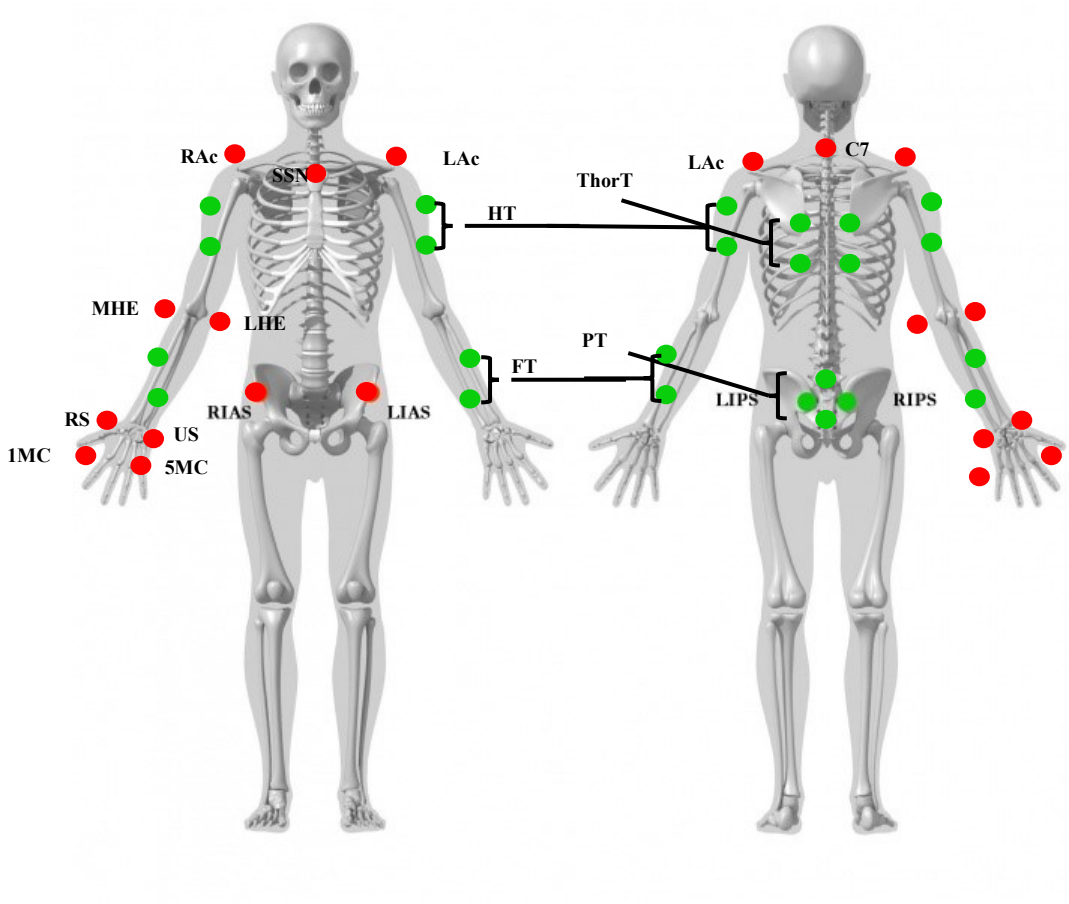

Figure 2.4: Experimental kinematic set up. Showing abbreviated labels for individual passive reflective markers (in red), and rigid body triads (in green).

Following the reflex test (2.3.2.2) passive reflective markers were positioned on the suprasternal notch (SSN) and 7th cervical spinous (C7) process along with 16 bilateral landmarks on the limbs: 5th (5MC) and 2nd metacarpal (2MC), radial styloid (RS), ulnar styloid (US), medial (MHE) and lateral humeral epicondyle (LHE), the mid acromion clavicular (Ac) joint, and the anterior superior iliac spine (ASIS). Rigid body clusters (4 markers) were affixed to the thorax (TT) and pelvis (PT), along with bilateral clusters on the forearm (FT) and upper arm (UT) (Figure 2.4). Following marker setup, a single standing calibration trial captured the marker position relative to the rigid bodies using six infrared emitting cameras (ProReflex 240, Qualisys^{TM}, Goteborg, Sweden) sampled at 100Hz using Qualisys Track Manager Software (Version 2.10, Qualisys^{TM}, Goteborg, Sweden). Following this trial, all single passive reflective markers were removed leaving

only the rigid body clusters. For synchronization, the Labview program collecting analog-to-digital data triggered the motion capture system where kinematic data were stored for post processing.

2.3.4 Controlled Horizontal Transfer Task (HTT)

The right-to-left horizontal transfer task is an experimental task designed to produce a highly controlled and predictable changing external moment to the lumbar spine. For this task, the participant lifted a mass (3 kg) with the handle vertically aligned to the participant's standing elbow height. To ensure minimal movement, tactile feedback was delivered to the participant's thoracic spinous process using a vertical jig (Figure 2.5E). By using both a low mass (3kg) and setting the lifting position at standing elbow height, this task was well below ergonomic guidelines ensuring the lift is safe for most participants.

The basic outline of this task involved transferring a mass from the right side of the participant's body (located 60° from their body midline) and returning it to the left side (60° from the midline) of their body (Figure 2.5A-C). To control this task the participant was requested to perform the movement to a 5-second external count/ pace, where the load must be in a unique position every second (Butler et al., 2010; Hubley-Kozey et al., 2012). This motion sequence was designed to produce a constantly changing lateral flexion and flexion moment around the lumbar spine (Figure 2.5D). On right-hand lift, a pressure sensor located under the mass identified task onset (mass lift) and offset (mass lower). The period of hand transition was identified using an optoelectric switch positioned at the participant's midline (Figure 2.5B). While the participants performed this motion the researcher observed that the participant held the mass in maximum reach with the elbow fully extended (Figure 2.5E) (Butler et al., 2010). For this task, the participant completed three trials.

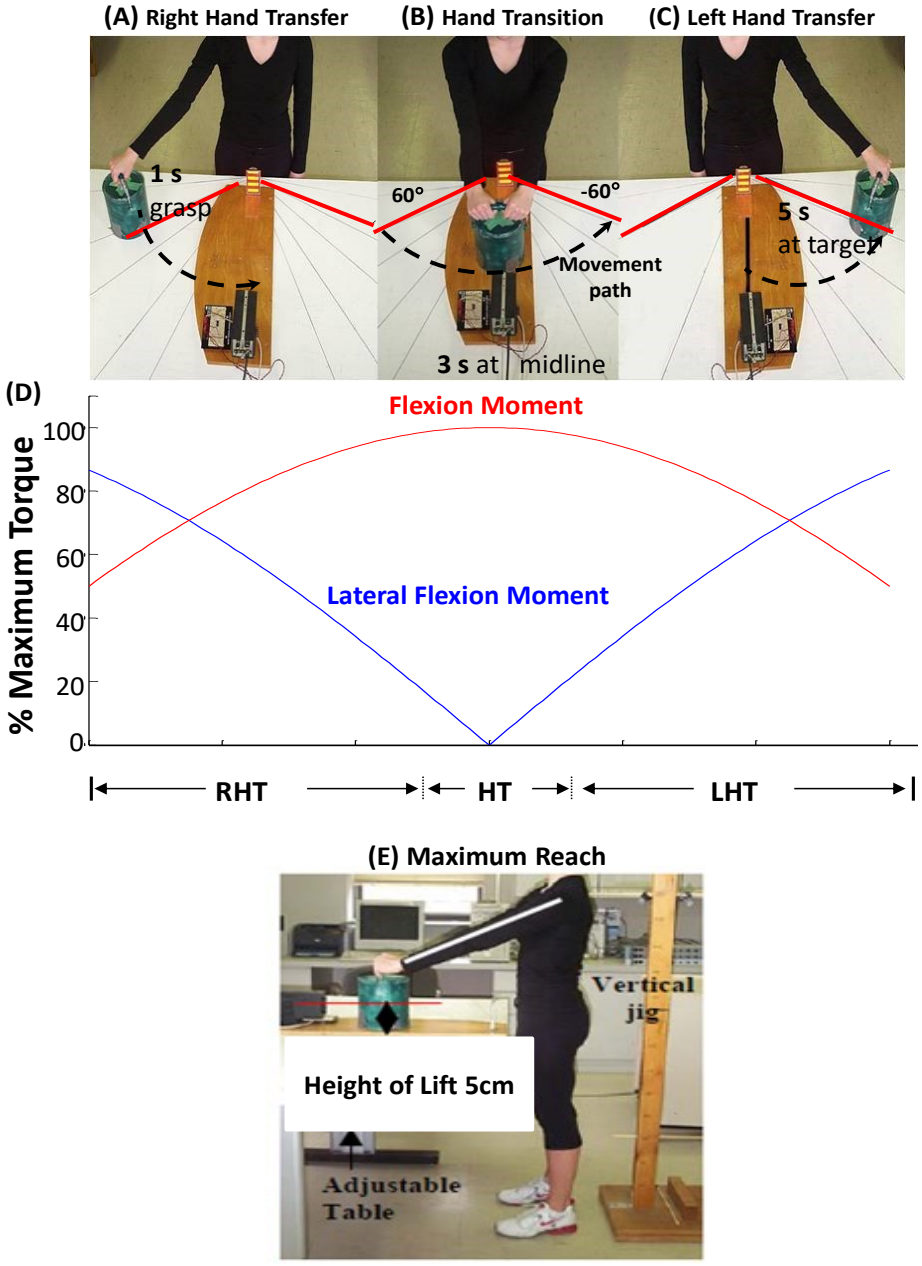

Figure 2.5: Experimental setup for the controlled dynamic transfer task, adapted from Butler et al. 2012 and 2010. As the participants transfer the mass from right to left (A-C) to a controlled count, the theoretical external moment generated around the spine changes from right lateral flexion moment (RHT), to a maximum flexion moment (HT), towards a left lateral flexion moment (LHT) (D). To aid in minimizing trunk motion tactile feedback was provided by a vertical jig positioned to contact the upper thoracic spine (E).

2.3.5 Normalization Test

At the end of the experimental tasks, the participant performed a series of trials for EMG normalization. First, the participant assumed a relaxed supine position; during this time baseline muscle activity (subject bias) was recorded for 3 seconds. Second, for amplitude normalization, the participant performed a series of standardized exercises that require brief (3s) maximum voluntary isometric contractions (MVIC). For these exercises, the participant was positioned by a trained researcher and secured to rigid structures using non-elastic straps during these contractions to ensure their safety. During normalization exercises, the participant was instructed how to produce the desired movement and then to apply force against the non-elastic strap "as hard as possible and hold it for 3 seconds" while the researcher provided the participant with standard verbal encouragement to increase the likelihood of achieving MVIC (Ng, Kippers, Parnianpour, & Richardson, 2002). Multiple MVIC exercises were performed including i) a supine sit-up; ii) side-lying lateral flexion (right and left); iii) prone position back extension; and iv) prone positioned back extension coupled with axial rotation (right and left) (Butler et al., 2010). Each contraction was repeated twice with a minute rest between trials resulting in a total of 12 trials.

2.4 Data Analysis

This section provides a comprehensive description of the data analysis. When possible for each section or sub-section ends with the primary outcome measure that was used for comparative purposes within this dissertation.

2.4.1 Spinal System Tests

Processing for each spinal system test is discussed prior to general EMG analysis as events measured from the spinal system test are used to provide time-points for EMG epoch analysis.

2.4.1.1 Transverse Stiffness (Passive)

Custom Matlab code filtered potentiometer and load cell data using a 2Hz second order zero-lag low pass Butterworth filter. Potentiometer and load cell voltage data were converted to angle (Θ) and force (newtons (N)) using calibration equations (Equation 1 and 2 respectively). These linear equations could fit experimental data with an R^2=1.0 and a % root mean square error between measured and fitted data of 0.9% and 0.45% respectively for each equation. Force data were converted to moments by calculating the product of the time-varying force (N) and the radius of the pully (0.1m) to express data in Newton-meters (Nm). Angle data were expressed relative to the standing neutral position by subtracting the bias position.

$$Angle(\theta(t)) = 1.00\theta + (Voltage(v(t)) * -156.05(\theta/v))$$

Equation 2.1: Measured angle (θ(t)) via time varying change in potentiometer voltage (v(t))

$$Force(N(t)) = [51.76lbs + (Voltage(v(t)) * -38.04(lbs/v))] * 4.45N/lbs$$

Equation 2.2: Measured force (N(t)) via time varying change in load cell voltage (v(t))

For each trial, the average moment measured at -0.5 to 0.5˚ relative to the participant's neutral position (0˚) was subtracted to remove bias (Drake & Callaghan, 2008; McGill et al., 1994). The time-varying angle was measured from 0-±15˚ in the direction desired for the trial. Simultaneously collected moment data was cut for the same period. For each participant, the moment-angle profile for all trials developed a data matrix. Using the Polyfit function in Matlab a 4th degree polynomial modeled the external moment ($\overline{Nm}$) for each angle from -15 to 15˚ (Equation 3) (Figure 2.6) (Scannell & McGill, 2003).

$$\overline{Nm} = c + a\theta + a_2\theta^2 + a_3\theta^3 + a_4\theta^4$$

Equation 2.3: Forth degree polynomial equation used to calculate external moment (Nm) from a known angle (θ) using predicted constants (c) and coefficients (a_n)

Stiffness (Nm/˚) was calculated from the modeled moment-angle curve as the quotient between the derivative of moment ($\dot{Nm}$) and angle ($\dot{\theta}$) (Equation 4). Using this stiffness profile two outcome measures were calculated. The primary outcome measure was the average neutral zone stiffness. This measure was derived by calculating the average stiffness within the 75th percentile of the measured neutral zone (ΔΘ, to be discussed) in both the CCW and CW direction. For this measure, the average stiffness

was calculated from 0-3˚ in the CW or CCW direction relative to (0˚) the minimum of the absolute value stiffness. Because spinal stiffness is known to increase with increasing compressive forces (Noguchi, Gooyers, & Callaghan, 2015; Zirbel, Stolworthy, Howell, & Bowden, 2013), average stiffness was normalized to the participant's mass as Nm/kg˚. The second and somewhat experimental outcome measure was in vivo neutral zone calculated in two steps. First, the angle at which the stiffness in the CW (Θ^1) or CCW (Θ^2) direction exceeded the threshold of 0.1Nm/˚ was calculated (Scannell & McGill, 2003), then neutral zone width ($\Delta\Theta$) was calculated as the difference between the CCW and CW angle (Figure 2.6).

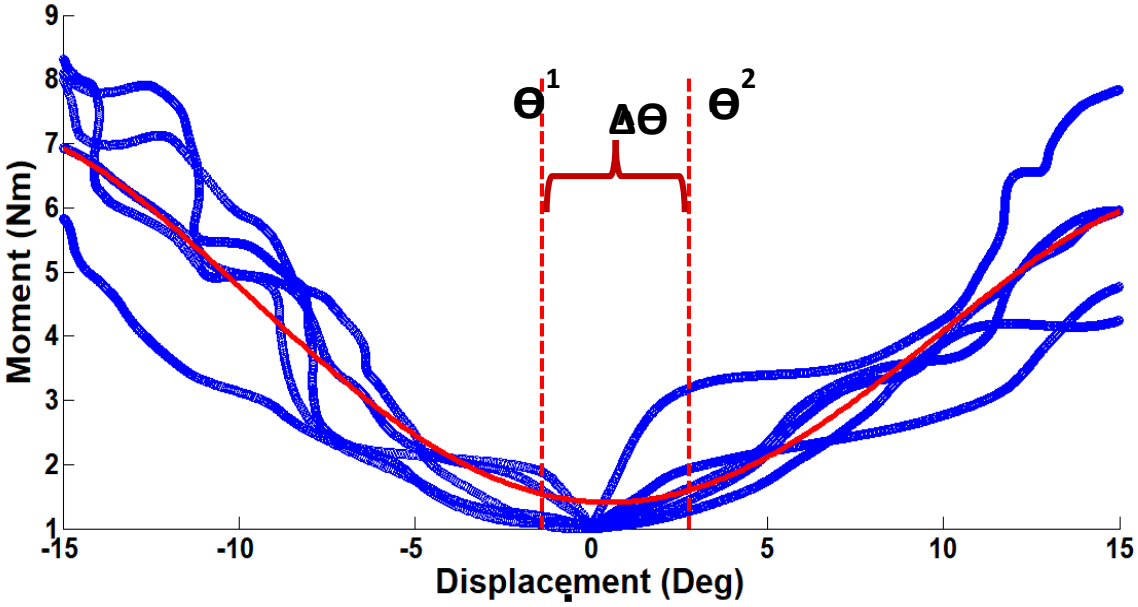

Figure 2.6: Visualization of stiffness test data processing. Representative moment-displacement curve for one participant from five trials in both the clockwise (CW) and counter CW direction (blue). Modeled fourth degree polynomial shown in red. Stiffness was measured as the derivative of the modeled moment-displacement curve. In both the CW and CCW direction the angle at which absolute stiffness exceeded 0.1Nm was calculated as Θ^1 and Θ^2. Neutral zone range ($\Delta\Theta$) was calculated as the difference between Θ^2 and Θ^1.

2.4.1.2 Reflex Test (Neural)

Custom Matlab code filtered the HUMAC data using a 10Hz second order zero-lag low pass Butterworth filter and converted it to angle (˚) according to equations provided by HUMAC. The first derivative of the position data was calculated to

determine perturbation velocity. The velocity signal was monitored for event detection, as analog data was collected across the entire reflex experiment. Perturbation onset was calculated as the sample at which the velocity exceeded a threshold of -110˚/s or 110˚/s for flexion and extension perturbations respectively. The outcome measure of time points calculated for each event detection was needed for subsequent analysis of EMG signals for muscle onset detection (Section 2.4.3.1).

2.4.1.3 Strength Test (Active)

Using custom Matlab code HUMAC moment signals were filtered using a 6Hz second order zero-lag low pass Butterworth filter, and converted to a moment (Nm) according to equations provided by HUMAC. The for this test maximum isometric moment for flexors and extensors was calculated as the highest average moment produced within a 500ms window. As an outcome measure, the highest moment from the two trials (flexion and extension) was calculated and then normalized to body mass to compensate for anthropometric differences between participants (Smith, Mayer, Gatchel, & Becker, 1985).

2.4.2 Kinematics and Kinetics Analysis

Marker data were processed in Qualisys Track Manager, to label marker's coordinates and entered into a custom Matlab™ script for quadratic interpolation of missing data points, and low pass filtered at 4Hz using a fourth order, zero-lag Butterworth filter (Wicks, 2017).

For the standing calibration trial the bony landmark marker set was used in a variety of regression equations to determine joint (wrist, elbow, shoulder, trunk) center of rotation (Campbell, Lloyd, Alderson, & Elliott, 2009; Dumas et al., 2006; Reed et al., 1999), and the location of each body segments (hand, forearm, upper arm, and torso's) center of mass (CoM) (Dumas et al., 2006). In addition, these markers were used to determine the anatomical coordinate system (ACS) around each segments CoM using the recommendations set forth by the International Society of Biomechanics (Wu et al., 2002; 2005).

The ACS of the torso and pelvis were tracked for the horizontal transfer task. These data were processed using Euler angles in a Z-Y-X (lateral flexion, axial rotation, flexion-extension) rotation sequence to determine segment angles. To quantify the motion during the horizontal transfer task segment angles were filtered using a 1Hz second order, zero-lag low pass Butterworth filter (Butler et al., 2010; Wicks, 2017). Epochs or the filtered data were determined from the beginning and end of the horizontal transfer task using event markers determined from the pressure sensor located under the mass (Section 2.3.4). For each trial, the total displacement (maximum – minimum) of the torso and pelvis segment was calculated in lateral flexion, flexion extension, and axial rotation was calculated by subtracting the minimum by the maximum angle in each plane. These displacements were averaged over the three trials for each participant.

Kinematic data were also used to estimate the forces and moments around the trunk (L4/L5) during the horizontal transfer task using a static top-down inverse dynamic model (Callaghan, Keown, & Andrews, 2005; Iino & Kojima, 2011) (Appendix A). The relative magnitude of each segments mass was estimated as a proportion of total body mass using anthropometrically derived regression equations (Dumas et al., 2006). For this model, necessary inputs were the magnitude (mass) and position (CoM) of all external and estimated segment masses along with each joint center of rotation. As only the mass of the external object was known (3kg) and its direct position was not measured it was assumed the center of mass of the lifted object was a weight vector that acted at the middle of the 2nd and 5th metacarpal of the right hand for the beginning of the lift (3kg right hand), both hands during hand transition (1.5 kg each hand), and the left hand following hand transition (3kg left hand), as determined by event markers for the pressure sensor and movement through the optoelectric switch.

Starting at the most distal and open-ended hand segment, forces and moments were calculated in the global coordinate system and propagated from one segment to the next using a system of Newton equations. As this model was static all linear and angular acceleration terms were assumed to be zero. While using this static model can attenuate the peak external moments recorded by a dynamic task these differences become negligible for slow and controlled movements (Bernard, Ayoub, & Lin, 1999; De Looze, Kingma, Thunnissen, van Wijk, & Toussaint, 1994) such as our highly controlled

horizontal transfer task. Assuming the human body acted as a series of linked frictionless rigid body segments an open kinetic model approach was used where the predicted forces and moments calculated at the proximal end of a body segment were propagated, assumed to be equal and opposite, to the distal end of the subsequent body segment with respect to Newton's third law until they converged on the lumbar spine (Winter, 2009). Following this calculation, the peak lateral flexion and flexion moment around the lumbar spine (L4/L5) was calculated for each trial and then averaged across trials for each participant as the primary outcome measure.

2.4.3 Electromyography

Custom Matlab (Math Works, Natick, MA) code corrected EMG signals for participant bias, electrocardiogram artifact (high pass zero-lag 30Hz filtered) (Butler, Newell, Hubley-Kozey, & Kozey, 2007) and noise from electromagnetic sources (inverse fast-Fourier filtered removing frequency spikes with a power 3 times greater than their nearest frequency (+/-15Hz) neighbours). Data were then full wave rectified and processed separately for the reflex and horizontal transfer task/ normalization test.

2.4.3.1 Reflex Test Analysis

For the reflex test, EMG data were rectified and low pass filtered at 50Hz using a second order zero-lag Butterworth filter, to produce a linear envelope to determine reflex onset (Hodges & Bui, 1996). For each muscle, filtered data were normalized to the MVIC measured for that particular muscle regardless what task evoked the maximum voluntary isometric contraction (MVIC) (Vera-Garcia, Moreside, & McGill, 2009; Vezina & Hubley-Kozey, 2000) (Section 2.4.3.1). Using the event signals from the HUMAC (see Section 2.4.1.2), EMG data were windowed -500ms before and 1000ms following the perturbation event (Figure 2.7). Primary outcome measures were calculated for the agonist (stretched) muscles only, the back extensor or abdominal sites, depending on whether the perturbation was in the flexion or extension direction respectively. Three outcome measures were calculated i) muscle onset, defined as the period at which the EMG signal exceeded the mean and 3x the SD of EMG muscle activation measured -500 to -400ms prior to the perturbation onset (time 0) for at least 25ms (Hodges & Bui, 1996)

in a period of 25-150ms following the perturbation, suggesting the source was from involuntary reflex (Radebold, Cholewicki, Panjabi, & Patel, 2000; I. A. Stokes, Gardner-Morse, Henry, & Badger, 2000; Taube et al., 2006). Each muscle onset was confirmed using visual inspection (Figure 2.7). If muscle activation reached threshold 25-150ms following the perturbation the reflex was defined as ii) responsive, otherwise, it was not responsive. Finally iii) the short latency reflex amplitude, quantified as the average linear-envelope amplitude from the time of muscle onset and the 30ms following reflex onset, was calculated (Klass et al., 2011; Obata, Kawashima, Akai, Nakazawa, & Ohtsuki, 2009; Obata, Kawashima, Ohtsuki, & Nakazawa, 2011).

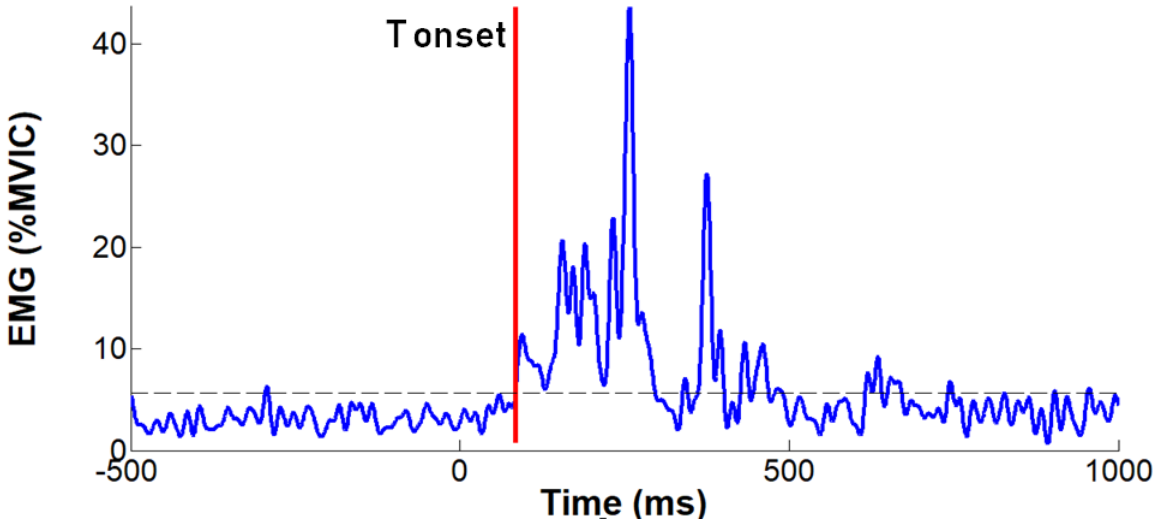

Figure 2.7: Reflex onset determination. Representative trial of an individual muscle sites reflex response. For each perturbation (EMG) data (blue line) were windowed from 500ms prior to and 1000ms following a perturbation (time 0ms). If a muscle was responsive, reflex onset (red line) was measured at the time (T onset) EMG activation exceeded a mean + 3 SD (black dashed line).

2.4.3.2 Normalization Task

For the normalization and horizontal transfer task (Section 2.4.3.3) rectified EMG data were low pass filtered at 6Hz using a second order zero-lag Butterworth filter, to produce a linear envelope. For each normalization trial, the maximum activity was measured for a 500ms moving average, for each muscle site. The maximum activity for each muscle was then calculated regardless of what task evoked the MVIC (Vera-Garcia et al., 2009; Vezina & Hubley-Kozey, 2000). This normalization protocol was used to express EMG data from the experimental trials as a percent of maximal voluntary

isometric contraction (% MVIC) allowing for comparisons between participants, and muscle sites (Burden, 2010).

2.4.3.3 Horizontal Transfer Task

The low pass filtered EMG signals (Section 2.4.3.2) from the horizontal transfer task was time normalized from 0-100% of the total task time (from the event markers (Section 2.3.4)) using a quadratic interpolation algorithm. Each muscle site was maximum normalized to express EMG activity as a % MVIC. For each muscle and participant ensemble average waveforms were calculated from the three trials.

2.4.3.4 Principal Component Analysis

The EMG ensemble-average waveforms were used in a pattern recognition technique, principal component analysis model (Hubley-Kozey & Vezina, 2002). For this technique, a data matrix was developed including all abdominal or back extensor time-normalized (101 time points) ensemble average EMG waveforms for all participants (X= n*12 (muscles)) [X * 101]. The grand mean was removed from this data matrix and the data were transformed into an orthogonal co-variance matrix (C). This co-variance matrix underwent an eigenvector decomposition to create a transformation matrix (T) the rows of which contained eigenvectors (principal components PCn) that define a new coordinate system. Each principal component PC_n is a vector space designed to represent a pattern that would capture the maximum variance within the original data matrix. Each subsequent principal component (PC_{n+1}) capture the variance of the residual vector space after the previous principal component was removed (Figure 2.8 (PC2)). The process was repeated until an orthogonal transformation matrix of the same dimensions of the covariance matrix was constructed. For each PC, a singular scalar weighting coefficient or PC score was calculated by transforming the original data on the multivariable coordinate system defined by the PC. Positive PC scores identify that the PC represents the waveform, whereas negative PC scores indicated a negative correlation with the PC waveform. These PC scores allowed for comparisons between participants', groups, and muscle sites as any factor with similar PC coefficients would share a common feature in EMG waveforms in both shape and magnitude. Thus, PC analysis allows for the

interpretation of muscle synergies as muscle sites with similar PC coefficients would share similar time varying patterns.

Consistent with previous work the number of principal components analyzed were determined using two criteria: i) the total explained variance of the waveforms must exceed 90% , and ii) the explained variance of an individual PC must be greater than 1% (Hubley-Kozey & Vezina, 2002). The primary outcome measure was the PC scores generated from this technique that were used for follow-up statistical analysis specific for each objective within this dissertation.

2.5 Statistical Analysis

The focus of the common and primary objectives of this dissertation was to determine whether muscle activation patterns adapted for suspected or measured deficits within individual spinal systems. PC scores from a controlled dynamic task were the primary outcome measure to compare muscle activation patterns, with other outcome measures compared to determine whether there were potential confounders between populations. Normality of PC scores was assessed using a Kolmogorov-Smirnov test, with non-normal data transformed using a Johnson transformation. For objective 1 three groups (controls, older adults, and rLBI) of participants were compared. For this analysis abdominals and back extensors were analyzed separately and were compared using a two-way mixed model analysis of variance (ANOVA) to test for differences among groups and muscles sites for main effects and interactions. For objective 2 (a-c) controls and rLBI were separated as either having a high or low function for a specific spinal system test (active, passive, or neural) using a median split approach. A three-way ANOVA compared between spinal system (active (strength), passive (stiffness), or neural (muscle onset)), group (control and rLBI) and muscle sites, to determine if specific muscle activation patterns were observed in those with lower measures of spinal system function and whether these would interact with the participants unique history of recent LBP. Tukey simultaneous tests compared pairwise differences when significant. Statistical analyses were performed in Minitab (version 17, State Collage, PA). Alpha was set at 0.05 and Bonferroni corrected for multiple comparisons.

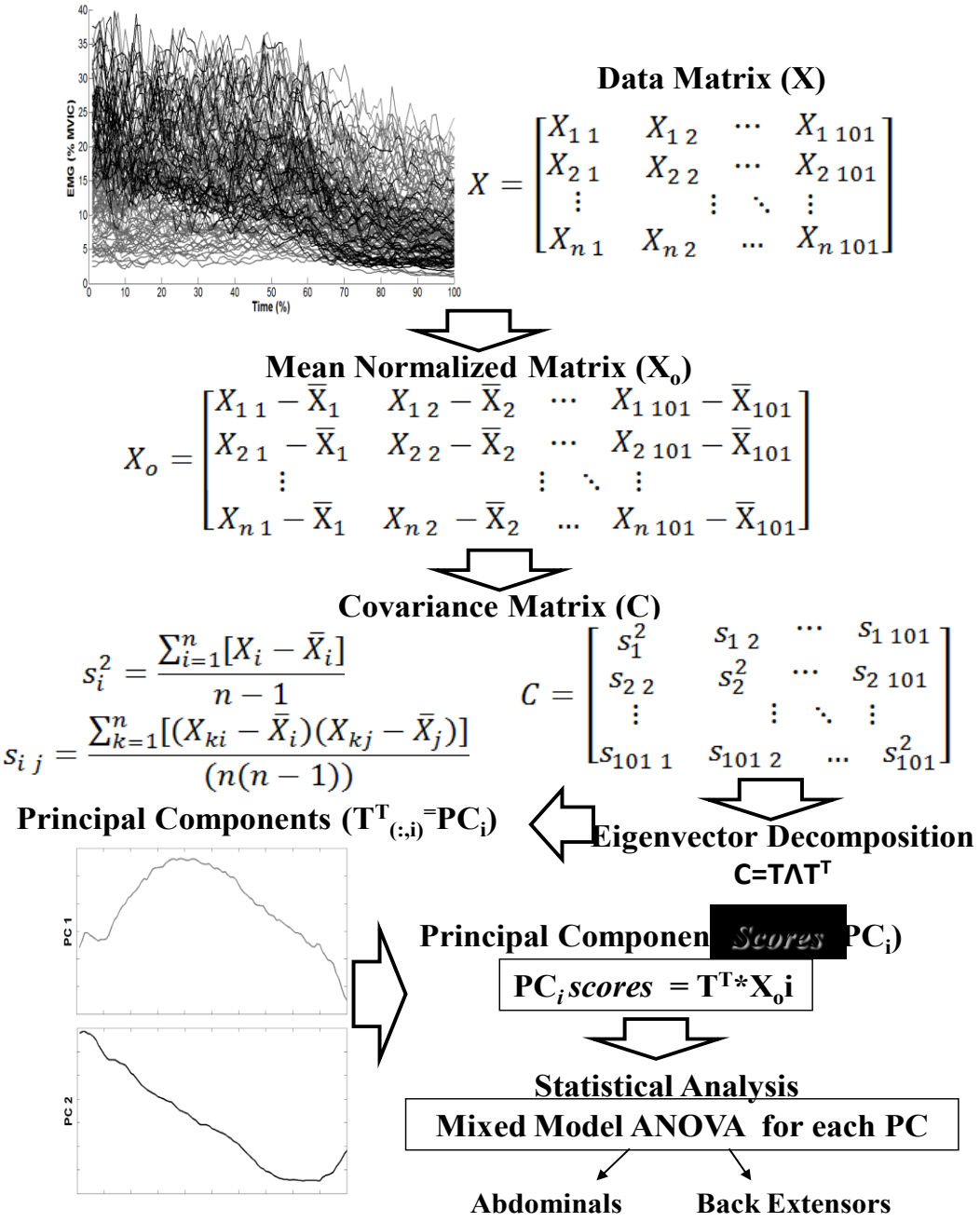

Figure 2.8: Visual display of PCA analysis. A covariance matrix (C) was generated from a data matrix (X) including all ensemble average waveforms using the equations for variance and covariance outlined on the left. The covariance matrix (C) was used in an eigenvector decomposition generating an orthogonal transformation matrix (T). The rows of this matrix describe a vector known as the principal component (PCi). For each PC all individual waveforms are transformed on the new coordinate system providing a PC score based on their similarity to the principal component. These scores were used for subsequent statistical models.

CHAPTER 3
DO OLDER ADULTS AND THOSE RECOVERED FROM LOW BACK INJURY SHARE COMMON MUSCLE ACTIVATION ADAPTATIONS

This is an **Accepted Manuscript** of an article published by Taylor & Francis in THE JOURNAL OF MOTOR BEHAVIOR on April 2019, available online at 10.1080/00222895.2018.1458280

3.1 INTRODUCTION

Low back pain (LBP) prevalence ranges from 15-45% of the population making it the most common musculoskeletal disorder (Hoy et al., 2014). A majority of those with LBP have no pathoanatomical diagnosis (Manusov, 2012), and are categorized as non-specific LBP thought to be the result of soft tissue injuries (Ebenbichler, Oddsson, Kollmitzer, & Erim, 2001). While risk factors for developing non-specific LBP are variable; increased age (Cho et al., 2014; Hoy et al., 2014) and history of LBP (Stanton et al., 2008; Taylor et al., 2014) are established risks. Panjabi's spinal stability model provides a framework for how pain can result from excessive tissue strain defined as an instability event. The model includes three spinal systems (passive, active, and neural) working in synchrony to prevent an instability event, where an impairment within any system will increase the likelihood of spinal instability and subsequent pain (Panjabi, 1992a; 2005). Cross-sectional and longitudinal studies confirm that differences in specific elements of all three systems exist between those with LBP and older adults compared to young asymptomatic controls.

There are passive osteoligamentous system changes in older adults and those with LBP. In a large (n>2800) epidemiological study, LBP was associated with increased risk of disc degeneration, which remained after adjusting for the risk associated with increased age (de Schepper et al., 2010). This moderate disc degeneration, common in both populations, is associated with increased vertebral body motion (neutral zone and total range) (Galbusera et al., 2014; Sengupta & Fan, 2014). Common active muscle system changes in older adults (Anderson et al., 2012; Anderson, D'Agostino, Bruno, Manoharan, & Bouxsein, 2011; Fortin et al., 2013; Kragstrup, Kjaer, & Mackey, 2011) and those with LBP (D'hooge, Cagnie, et al., 2012a; Hides, Richardson, & Jull, 1996; P. Kjaer, Bendix, Sorensen, Korsholm, & Leboeuf-Yde, 2007; Langevin et al., 2009;

Whittaker et al., 2012) include reduced trunk muscle cross-sectional area, increased intramuscular fat and fibrosis. Collectively these changes reduce the maximum torque (muscle strength) these populations can produce compared to healthy young controls (Hasue et al., 1980; Keller et al., 1999; Newton et al., 1993), causing them to work at a higher percentage of their maximum muscle capacity for a given task. Neural system changes with age (Goble et al., 2008) and joint injuries (Solomonow, 2006) have been associated with degeneration to sensory organelles embedded within osteoligamentous and musculoskeletal tissue. Sensory motor transducer degeneration in combination with changes in musculotendinous (Ito et al., 2015) and connective tissue (Cammarata & Dhaher, 2012; Levin et al., 2014; Sánchez-Zuriaga et al., 2010) potentially explains impaired proprioception and lower short latency reflex amplitudes measured in older adults (Brumagne et al., 2004; Goble et al., 2008; Klass et al., 2011) and those with LBP (Osthoff et al., 2015; Ramprasad et al., 2010) compared to young asymptomatic controls.

Computational modeling shows that the time-varying recruitment of trunk musculature has the greatest contribution to spinal stability, particularly when the spine is in a neutral position (Cholewicki, Panjabi, & Khachatryan, 1997; Kavcic et al., 2004; McGill, Grenier, Kavcic, & Cholewicki, 2003a). Consistent with Panjabi's theoretical model, populations with spinal system impairments could compensate by modifying the spatial-temporal recruitment of trunk muscles to stabilize the spine. There is indirect evidence from cross-sectional studies reporting that trunk muscle activation in older adults (Claudino, Santos, & Santos, 2013; Hubley-Kozey, Hanada, Gordon, Kozey, & McKeon, 2009; Lee, Chen, & Aruin, 2015; McGill et al., 1999; Quirk & Hubley-Kozey, 2014) and those recovered from low back pain (Butler et al., 2012; D'hooge, Hodges, et al., 2012b; Hubley-Kozey et al., 2013; Jones, Henry, Raasch, Hitt, & Bunn, 2011; Moreside et al., 2013), have higher amplitudes, are less responsive to changing external moments and have different spatial recruitment strategies compared to young asymptomatic controls. These different spatial strategies capture modified trunk muscular synergies defined as a pattern of activation across multiple muscles that are combined with time or task dependent features to produce complex muscle activation patterns (Cheung, Samartzis, Karppinen, & Luk, 2012a; Torres-Oviedo & Ting, 2010). In general, these activation patterns have the potential to alter spinal stability via increased active

stiffness (Brown, Vera-Garcia, & McGill, 2006; Granata & Marras, 2000; Kavcic et al., 2004).

A gap exist however in our understanding of how older adult and LBP groups can compensate for specific spinal system impairments and if the compensations are similar between groups. To our knowledge, no study has directly compared trunk muscle activation of older adults and those with a history of LBP, to determine whether differences exist in their muscle activation patterns and if the magnitude of alterations are similar for both groups compared to asymptomatic controls. Extrapolating from cross-sectional studies of each group independently could be confounded. First, pain can alter muscle activation patterns independent of spinal system pathology, with current theories suggesting these modifications persist despite the resolution of pain (Hodges et al., 2013). Thus, those with a history of LBP may modify muscle activation patterns independent of spinal system impairments. Second, the tasks compared and variables used to assess the trunk muscle activation patterns differed among studies making comparisons and common conclusions among these studies difficult. Collectively the work to date leads to question whether older adults and those with a history of LBP do share common structural and functional changes in spinal structures that manifest as specific muscle activation pattern changes. Examining this relationship would provide empirical evidence consistent with Panjabi's theory of spinal system compensation (Panjabi, 2005), improving our understanding of spinal system function and dysfunction in these clinical populations that could potentially inform preventative intervention strategies.

The purpose of this study was to compare trunk muscle activation patterns during controlled dynamic tasks that challenge the stabilizing function of the abdominal and back extensor muscles between older adults and those recovered from a low back injury who report minimal spinal pain and dysfunction. The central hypothesis was that regardless of the task, muscle activation patterns will not differ between older adult and the recovered low back injury groups and the secondary hypothesis was that both groups will have similar alterations compared to young asymptotic controls.

3.2 METHODS

3.2.1 Participants

Three groups were identified from a cohort of participants in a larger trial including twelve older adults (65+ years), 16 younger (20-45 years) participants recovered from low back injury (rLBI) and 19 younger asymptomatic control participants based on groups having similar sex distribution, mass, and height. Exclusion criteria for all participants were previous abdominal or back surgery, spinal fractures, cardiovascular, respiratory, neurological, or current musculoskeletal conditions. All participants were recruited through local advertising. Older adults and controls had no self-report LBP within one-year prior to or at time of testing. Those in the rLBI group experienced non-specific LBP (between ribs and gluteal folds) 4-12 weeks (mean = 5±3 weeks) before testing resulting in participants seeking medical attention or reducing activities of daily living for at least three days (Ozguler et al., 2000). At the time of testing rLBI were deemed recovered reporting minimal pain (Visual Analogue Score (VAS) <30, mean= 9±12/100mm) (Wewers & Lowe, 1990), disability (Roland Morris Disability <9, mean=3±4/24) (Roland & Morris, 1983) and the resumption of regular activities upon the day of data collection. Before testing, all participants signed an informed consent approved by the Institution's Research Ethics Board.

3.2.2 Protocol

Participants attended two sessions. During the first session, one week before testing, basic demographic information was collected. Participants then practiced all experimental tasks, and were instructed to practice the trunk stability task (TST) at least three times (mean=3.5±1.3) before data collection (session two). At session two, self-report pain and disability measures were collected for rLBI confirming recovery status. All participants were prepared for data collection (described below) and then performed a series of highly controlled lifting (Butler et al., 2010) and leg loading (TST) (Hubley-Kozey & Vezina, 2002) tasks presented in random order. Timing of all tasks was controlled to an external count with event markers detecting movement onset and offset to synchronize motion and electromyography (EMG) data. For each task, participants attempted to minimize trunk and pelvis motion and adhere to the external count. The tasks were repeated until three successful trials were recorded for each condition based

on correct timing and minimal observable motion, confirmed by inspecting recorded event marker and motion traces (described below).

3.2.2.1 Trunk Stability Task

A leg lifting exercise task (Hubley-Kozey & Vezina, 2002) was performed from a supine crook lying position to an external count ("ready-set-go-1-2-3-4"). Participants were instructed to perform an abdominal hollowing maneuver on "set", pulling their navel inwards towards their spine and upwards towards their rib cage. Participants then performed a series of leg movements aimed to produce a controlled dynamic stability challenge (Figure 3.1). Knee angle was to be maintained at 90° flexion. Using metallic tape positioned on the participants' right heel and thigh, a simple circuit identified movement onset/offset and 90° hip flexion (Figure 3.1).

3.2.2.2 Right to Left Horizontal Transfer Task

For the HTT participants were instructed to lift and transfer a 2.9kg mass (on average less than 4% of total body mass) positioned 60° to the right of their midline, transition the mass between their hands at the midline of their body (hand transition) and lower the mass 60° to the left (Butler et al., 2010). This task was timed to a 5-second count with each count corresponding to a specific mass location. Mass lift (onset) and lower (offset) were recorded using pressure sensors located at the base of the mass, and hand transition was determined from an optoelectric switch. A height adjusted table ensured that the mass was lifted at standing elbow height, the optoelectric switch confirmed the mass was lifted no higher than 5cm and visual inspection ensured the elbow remained in full extension (Butler et al., 2010). Tactile feedback applied to the upper thoracic spine aided in minimizing trunk motion.

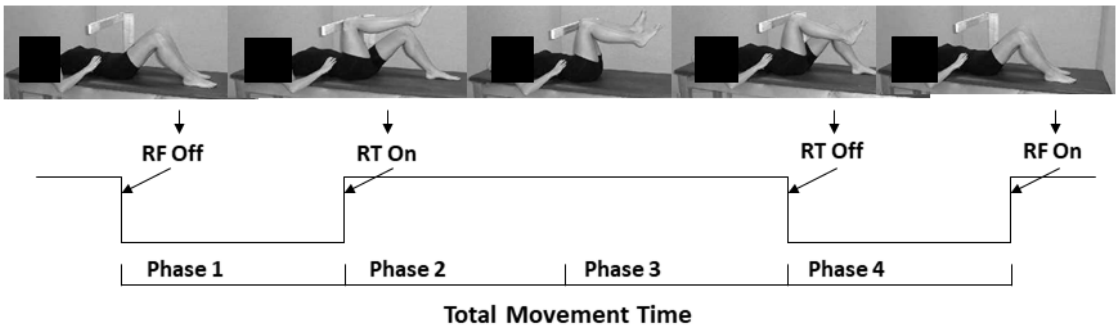

Figure 3.1: Trunk Stability Task: From a starting position the participant lift their right foot (RF Off), when their right thigh touched the bar (RT On) they lift their left leg to the bar. The participant would then lower their left leg to the bed and begin to lower their right leg (RT off) until their right foot came to rest on the bed (RF on). Contact of the right foot and thigh completed an electric circuit to break the task into phases. During right thigh on to right thigh off (phase 2 and 3) the participant would have neither leg on the bed representing a period of no-leg-support.

3.2.2.3 Normalization Task

Participants performed a series of 6 exercises to elicit maximum voluntary isometric contractions (MVIC) for EMG normalization including two trials of: supine sit-up, side laying lateral flexion (left and right), prone back extension, and prone back extension coupled with rotation (right and left) (Butler et al., 2010). Participants were instructed to maintain a constant level maximal effort for 3 seconds for each exercise, with 2 minutes rest between exercises.

3.2.3 Data Collection and Analysis

3.2.3.1 Motion

A Flock of Birds motion capture system (Ascension Technology Corp, Shelburne, VT) recorded motion of sensors affixed to the pelvis (left iliac crest) and thoracic spine (eight spinous process) for the HTT and only the pelvis marker for the TST. The angular displacement of each sensor was calculated in the global coordinate system with participants aligned to approximate motion within an anatomical reference (Butler et al., 2010).

Motion data and event markers (collected using a 12-bit analog-to-digital board (DAQPad-6020E, National Instruments, Austin, TX)) were sampled at 50 Hz using

Labview™ software (Version 7). Using custom Matlab™ code (Math Works, Natick, Massachusetts), angular motion data were low-pass filtered with a 2 Hz second order zero-lag Butterworth filter. Maximum angular displacements were calculated for the entire movement task.

3.2.3.2 Surface Electromyography

Surface electrodes (Ag/AgCl, 10mm diameter, Kendall, Chicopee, MA) were placed in a bipolar configuration (30 mm interelectrode distance) over 12 bilateral muscle sites using standardized guidelines (Butler et al., 2010). Abdominal sites included upper and lower rectus abdominis (URA& LRA), internal oblique (IO) and 3 external oblique sites (EO1-3) representing anterior, lateral and posterior fibres, respectively. Back sites included; the quadratus lumborum at lumbar level 4 and 8 cm laterally from the 4th spinous process (L48), the multifidus at lumbar level 5 and 2 cm laterally from the 5th spinous process (L52), and erector spinae at the level of the lumbar first and third spinous processes, 3 and 6 cm horizontal to the midline, representing longissimus (L13& L33) and iliocostalis (L16& L36) sites respectively.

EMG signals were pre-amplified (500x) and further amplified using three AMT-8 amplifiers (band-pass 10-1000Hz, CMRR=115 dB, input impedance 10GΩ; Bortec Inc., Calgary, AB). EMG signals and event markers were digitized at 2000Hz using a 16-bit analog-to-digital board (PCI-6033E, National Instruments) and Labview™ software. Custom Matlab™ code corrected EMG signals for subject bias, electrocardiogram artifact (high-pass zero-lag filtered 30Hz) (Butler et al., 2007), and noise from the electromagnetic source (inverse fast-Fourier filtered). Corrected signals were full wave rectified and low-pass filtered at 6Hz using a second order zero-lag Butterworth filter, to produce a linear envelope.

EMG signals were processed in two ways. First, to capture overall neural drive, average root mean squared (RMS) amplitudes were calculated for the entire task (onset to offset). These data were amplitude normalized to the maximum average (500ms moving window) RMS voltage regardless of which task elicited MVIC (Vera-Garcia et al., 2009; Vezina & Hubley-Kozey, 2000). For each muscle site, the average of three trials was calculated. For descriptive purposes, the overall activation for all abdominals and all back

extensor sites for each task were averaged and expressed as an activation ratio relative to the young asymptomatic control group.

Second, to capture temporal features, linear envelope EMG signals were amplitude normalized to average activation over the entire task, and time normalized with 100% being the total movement time using a quadratic interpolation algorithm. For each condition, the ensemble average was calculated from three trials. Principal component analysis (Butler et al., 2010; Hubley-Kozey et al., 2012; Hubley-Kozey & Vezina, 2002; Ivanenko, Poppele, & Lacquaniti, 2004) is a data reduction method where a muscle activation pattern can be decomposed into a lower dimensional subspace (Cheung et al., 2012b). This mathematical operation can be performed through decomposition of the original data in either a spatial or temporal domain producing a spatial (muscle synergy) or temporal (temporal pattern) vector respectively (Delis, Panzeri, Pozzo, & Berret, 2013). Since we were interested in the time-varying recruitment to look at responsiveness, we reduced our dimensionality within the temporal domain by arranging the ensemble average waveforms for each participant (47) and muscle (12) created a data matrix (564 x 101) for each condition (HTT and TST) for the abdominals and back separately. These data matrices were entered into PCA models (Hubley-Kozey & Vezina, 2002; Jackson, 2003). Briefly, a covariance matrix was calculated, eigenvector decomposition identified vectors (PCs) that explained patterns of variation in the EMG waveforms. For each EMG waveform, a weighting coefficient (PC score) quantified how much variance of the original EMG waveform was captured by the PC.

For each PC the variance accounted for (VAF) was determined by calculating the RMS error between the reconstructed and true EMG waveform (van den Hoorn et al., 2014). The number of PC's retained was determined to maximize the explained variance such that either 1) the total VAF was >90% or 2) the inclusion of a PC improved the VAF by more than 1%.

3.2.4 Statistical Analysis

One-way analysis of variance (ANOVA) tested for differences in demographic, anthropometric, motion and timing data. Sex distribution was compared using a Chi-square test for independence. Two-factor (group, muscle) mixed model ANOVA, with

separate models created for the abdominals and back extensors tested for main effects and interactions for PC scores and RMS amplitudes. This analysis allowed for comparisons in the spatial domain where non-significant muscle differences would indicate muscle synergies (Hubley-Kozey et al., 2012), showing similar activation amongst muscle sites in response to a temporal (PC) or task (average RMS amplitude) dependent feature (Cheung et al., 2012b). Alternatively significant muscle differences would indicate spatial complexities, with group by muscle interaction capturing modifications in spatial synergies among groups. Tukey simultaneous tests compared pairwise differences when necessary. Normality was confirmed from a Kolmogorov-Smirnov test, with non-normal data transformed using a Johnson transformation. All statistical analyses were performed in Minitab (version 17, State Collage, PA). Alpha was set at 0.05 and Bonferroni corrected.

3.3 RESULTS

3.3.1 Demographic, Timing, and Motion Data

Participant's demographic data are in Table 3.1. Other than age ($F(2,44)=184.3$, $p<0.001$) there were no significant differences among groups. The only significant group differences in timing and motion variables (Table 3.2) were that older adults had greater pelvis axial rotation than rLBI for the TST ($F(2,44)=6.83$, $p=0.006$) and both older adults and rLBI had greater trunk axial rotation than controls for the HTT ($F(2,44)=8.48$, $p<0.001$) (Table 3.2).

Table 3.1: Participant demographics and anthropometric measures

Variable	rLBI	Older	Control
Age (Years old)	28.9±7 *	68.8±3	29.8±6 *
Sex (% Female)	40%	42%	42%
Mass (Kg)	77.4±17	78.7±15	74.3±15
Height (m)	170.4±9	170.0±9	171.5±9
BMI (Kgm^2)	26.4±4	27.1±4	24.8±3

Body mass index (BMI) is calculated as the product of mass in kilograms (kg) and height in meters (m) squared. Significant post hoc difference shown as * to denote group differences relative to older adults

Table 3.2: Means and standard deviations of timing and motion data

	rLBI	Older	Control
Trunk Stability Task (TST)			
Time (seconds)	3.8±0.3	3.6±0.3	3.6±0.3
Pelvis Lat. Flex. °	3.5±2	5.0±4	5.2±3
Pelvis Flex.-Ext. °	2.7±2	4.3±2	5.1±3
Pelvis Axial Rot. °	2.9±2 *	5.5±2	3.7±2
Horizontal Transfer Task (HTT)			
Time (seconds)	4.2±0.4	4.2±0.5	4.0±0.3
Trunk Lat. Flex. °	2.7±2	2.5±1	2.0±1
Trunk Flex.-Ext. °	3.5±2	1.9±1	3.0±2
Trunk Axial Rot. °	5.9±3	5.1±4	4.7±3
Pelvis Lat. Flex. °	1.9±1	3.3±3	1.6±1
Pelvis Flex.-Ext. °	1.4±1	2.6±2	1.2±1
Pelvis Axial Rot. °	4.3±2	4.1±2	2.2±2 *‡

Motion data reported in degrees (°) for flexion-extension (Flex.-Ext), lateral flexion (Lat. Flex) and axial rotation (Rot.). Significant post-hoc differences shown as * and ‡ to denote differences relative to older adults and rLBI respectively.

3.3.2 Surface Electromyograms

The EMG waveforms for the TST and the HTT are in Figures 3.2 and 3.3, with the PC features found in Figure 3.4 and 3.5. Descriptive statistics for the group main effects are in Table 3.3, abdominal and back extensor muscle main effects and group by muscle interactions are in Tables 3.4 and 3.5 respectively. Ratio of older adults and rLBI activation amplitudes relative to young controls are in Table 3.6.

3.3.2.1 Trunk Stability Task (Amplitude)

Post-hoc results for the group main effect ($F(2,484)=17.63$, $p<0.001$) showed that older adults had higher RMS amplitude than controls and rLBI group for the abdominal sites (Table 3.3). The pairwise differences for the significant group by muscle interaction ($F(22,484)=2.72$, $p<0.001$) for back extensor RMS amplitude found that rLBI had higher activation than controls for all sites (12/12), whereas older adults had higher activation than controls for 4/12 sites (Table 3.5). Post-hoc findings also identified within group muscle differences, showing that inferior sites had higher activation (RMS) than superior sites for controls and rLBI, whereas older adults had synergistic recruitment with only one significant muscle site difference (Table 3.5).

3.3.2.2 Horizontal Transfer Task (Amplitude)

Post-hoc findings for the abdominal site group main effect ($F(2,484)=106.5$, $p<0.001$) captured that rLBI had higher RMS amplitudes than controls and that older adults were higher than both rLBI and controls (Table 3.3). For the back extensors, the group main effect ($F(2,484)=212.0$, $p<0.001$) captured that both older adults and rLBI had higher RMS amplitudes than controls (Table 3.3).

3.3.2.3 Trunk Stability Task (Temporal)

Three PCs explained 84±6% VAF for the abdominal sites, PC1 captured a differential of activation between right leg lift (0-20% of the task) and no-leg-support (30-80%) (Figure 3.4a). PC1 had a group main effect ($F(2,484)=16.8$, $p<0.001$), with older adults having higher PC1 score (Table 3.3), capturing less responsiveness (differential), higher activation before left leg lift compared to no-leg support, relative to rLBI and controls (Figure 3.2a).

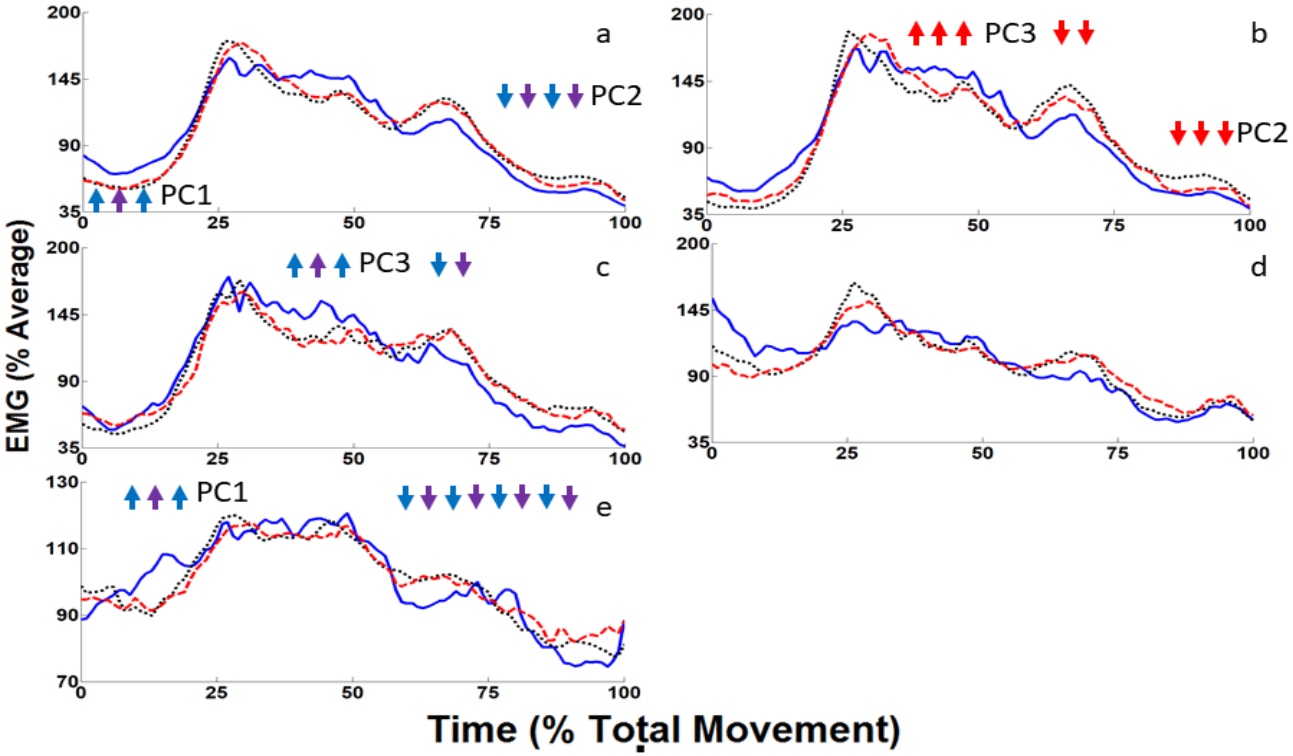

Figure 3.2: Normalized to average (% AVG) ensemble average trunk electromyograms for asymptomatic controls (black dot), older adults (blue solid) and recovered low back injured (red dashed) participants during the trunk stability task. Waveforms were selected to illustrate group and group by muscle differences. The group main effect is shown as differences between the a) ensemble average of all abdominal muscle sites combined. Group by muscle interactions in abdominal sites are illustrated by group differences within the b) ensemble average of all rectus abdominus sites (RA), c) right posterior external obliques (REO3), and d) right internal obliques (RIO). For the back extensors the group main effect is shown as the e) ensemble average of all back extensors sites combined. Significant group differences in the time-varying recruitment of musculature are illustrated by coloured arrows showing differences between older adults (blue) and rLBI (red) compared to the control population. Purple arrows illustrate differences between older adults and rLBI. With the corresponding principal component (PC) number shown to the right or in between the group difference arrows.

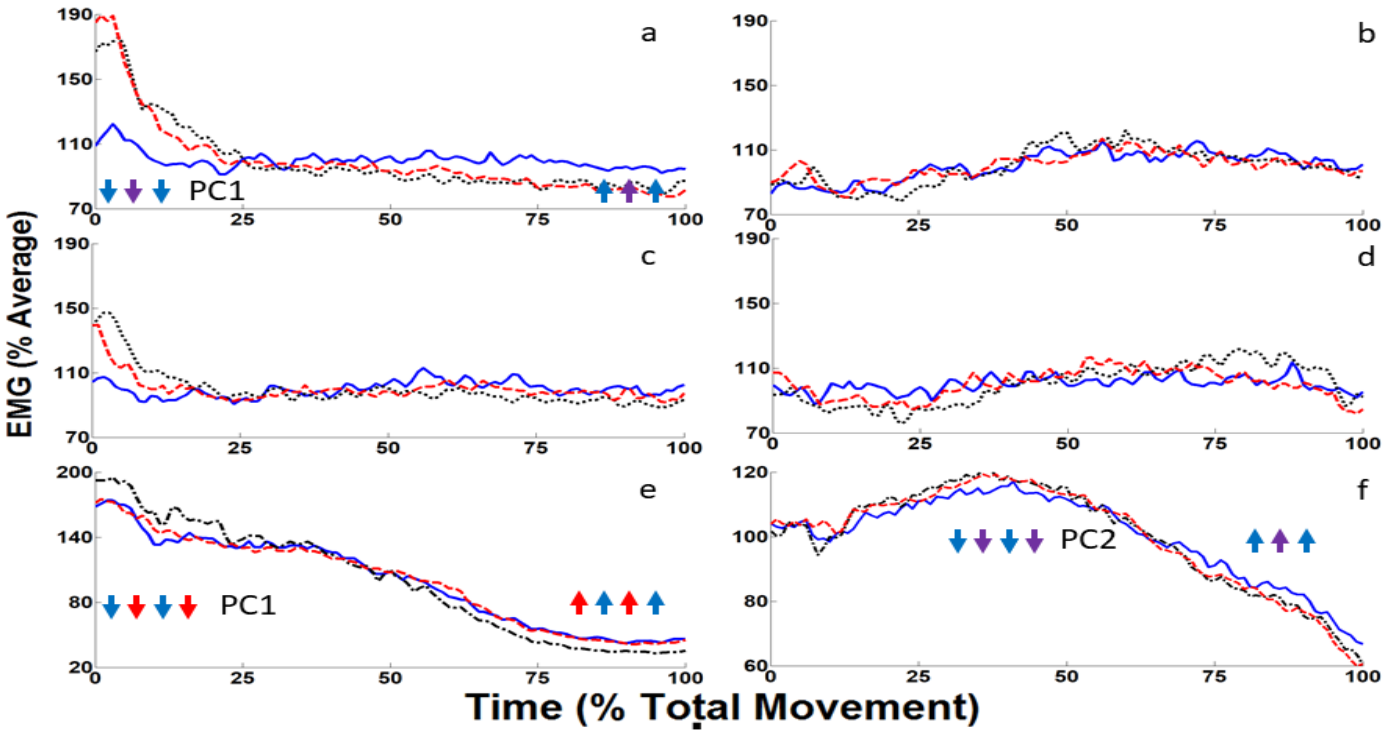

Figure 3.3: Normalized to average (% AVG) ensemble average trunk electromyograms for asymptomatic controls (black dot), older adults (blue solid) and recovered low back injured (red dashed) participants during the horizontal transfer task. Waveforms were selected to illustrate group and group by muscle differences. Group by muscle interactions in abdominal sites are illustrated by group differences within the a) left posterior external obliques (LEO3), b) left internal obliques (LIO), c) left middle external obliques (LEO2), and d) left anterior external obliques (LEO1) and altered synergies (a vs c & b vs d) and differences (a vs b & c vs d) that occur between groups. Group main effects for the back extensor sites are illustrated by e) group differences between the left superior iliocostalis site (LL16) and f) group differences between all back extensor sites combined. Significant group differences in the time-varying recruitment of musculature are illustrated by coloured arrows as described in Figure 2 with PC numbers indicated the right or in between group difference arrows.

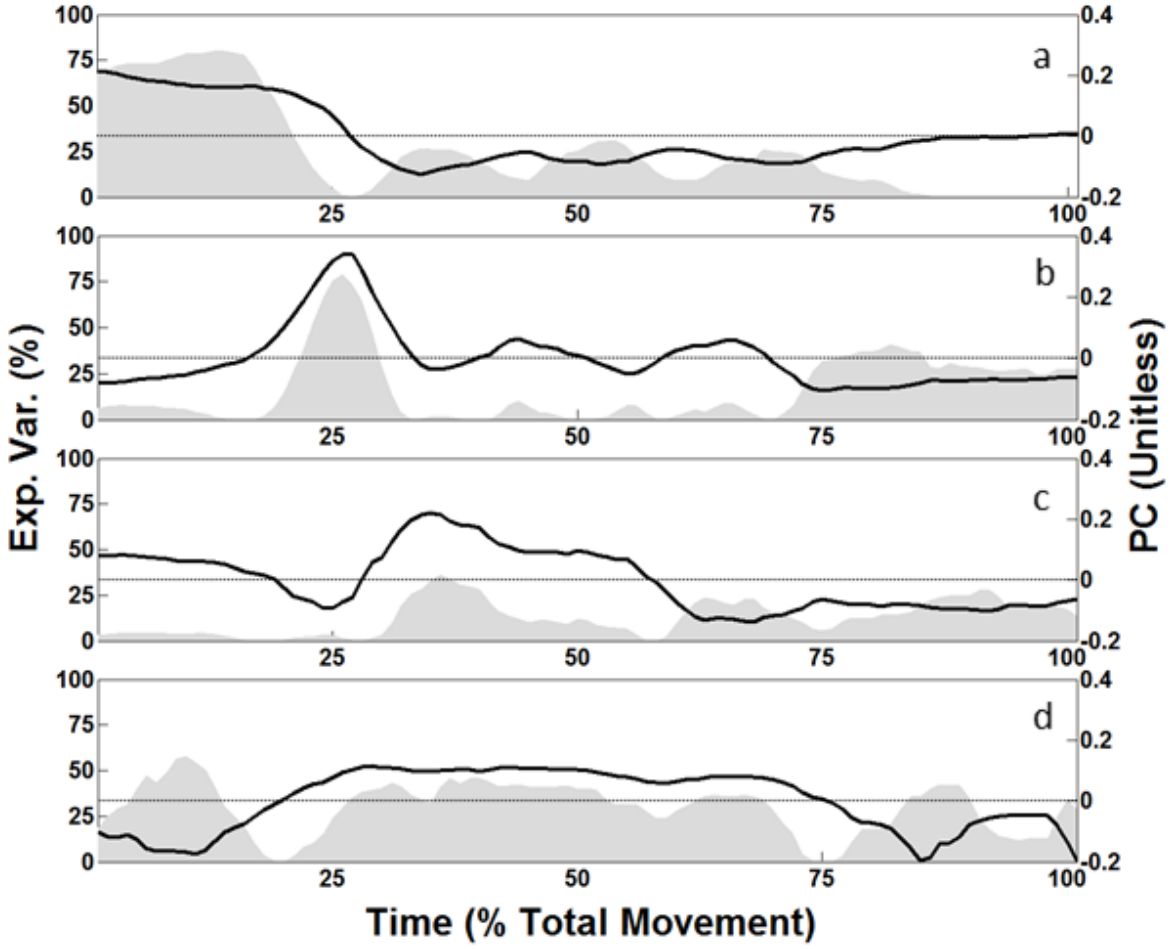

Figure 3.4: Principal component waveforms (black solid line, eigenvectors) from the trunk stability task for the abdominals (PC 1-3, a-c respectively) and back extensors (d). Grey shading represents explained variance for the respective principal component at periods throughout the movement time.

Table 3.3: Means and standard deviations of amplitude (RMS) and temporal (PC) features with group main effects.

Variable	rLBI	Older	Control
Trunk Stability Task (TST)			
Abdominal RMS (% MVIC)	14.3±8.3 *	17.2±8.3	14.1±8.1 *
Abdominal PC1	-9±164 *	35±167	-26±151 *
Abdominal PC2	9±147 *	39±153	-9±135 *‡
Back Extensor PC1	10±103 *	-33±153	9±83 *
Horizontal Transfer Task (HTT)			
Abdominal RMS (% MVIC)	5.5±3.2 *	6.7±4.0	3.8±3.1 *‡
Back Extensor RMS (% MVIC)	17.7±6.8	19.8±10.0	11.6±6.2 *‡
Back Extensor |PC1|§	237±129	247±125	290±152 *‡
Back Extensor PC2	8±78 *	-28±80	13±93 *

Electromyographic amplitudes are reported as root mean square (RMS) normalized to maximum voluntary isometric contractions (%MVIC). Temporal patterns quantified as principal component (PC) scores. § denotes absolute value PC scores data were reported. Significant post-hoc group differences denoted by symbols to show differences relative to older adults (*) and rLBI (‡)

PC2 captured a differential of activation between the initiation of left leg lift (20-30%) and right leg lowering (75-100%) (Figure 3.4b). Post-hoc analysis of the group main effect ($F(2,484)=22.1$, $p<0.001$), found higher PC2 scores (Table 3.3) for older adults capturing a greater relative decrease in activation during right leg lowering compared to both rLBI and controls (Figure 3.2a). The rLBI had higher scores than controls (Table 3.3) capturing a similar trend toward a decrease in activation during right leg lowering (Figure 3.2b).

PC3 captured a differential of activation during no-leg-support (30-55%) and left and right leg lowering (60-100%) (Figure 3.4c). PC3 had a group by muscle interaction ($F(22,484)=2.6$, $p<0.001$), with older adults having higher scores for 8/12 muscle sites than controls and 4/12 (right oblique) sites than rLBI (Table 3.4), capturing that older adults had less responsiveness between no-leg-support and left leg lowering, relative to both controls and rLBI (Figure 3.2c). The rLBI group had higher PC3 scores than controls in 4/12 (RA) sites (Table 3.4) also capturing reduced responsiveness (Figure 3.2b). Within participant groups, muscle synergies were different as illustrated in Table

3.4. Within controls, EO3 had lower scores relative to IO (Figure 3.2c vs. d). Within older adults, the left IO differed from EO1 (Table 3.4).

For the back extensors, one PC explained 89±6% VAF. PC1 captured a differential of activation during no-leg-support (30-80%) relative to right leg lift (0-20%) and lower (80-100%) (Figure 3.4d). PC1 had a group main effect ($F(2,484)=13.2$, $p<0.001$), with older adults having lower PC1 scores (Table 3.5), capturing sustained activation during right leg lift, compared to rLBI and controls (Figure 3.2e).

3.3.2.4 Horizontal Transfer Task (Temporal)

One PC explained 90±7% VAF for the abdominal sites, PC1 captured a differential of activation during right-hand (0-35%) and left-hand transfer (60-100%) (Figure 3.5a), capturing responsiveness to the changing lateral flexion moment. A group by muscle interaction ($F(22,484)=2.96$, $p<0.001$), showed that controls had higher absolute magnitude of PC1 scores for EO3 sites compared to older adults (Table 3.4), and that rLBI had higher left EO3 PC scores than older adults (Table 3.4) capturing that both groups had greater responsiveness to the lateral flexion moment than older adults (Figure 3.3a). Within participant groups, muscle synergies were different. First, groups differed in the number of abdominal sites with left versus right muscle site asymmetries. Controls had asymmetries for all oblique sites, rLBI had side differences for two oblique sites and older adults had no side differences (Table 3.4). Second, groups differed in synergistic relationship between ipsilateral muscle sites (Table 3.4). PC1 scores captured temporal activation differences between horizontally and laterally orientated fibres in controls (Figure 3.3a&c vs. b&d) i.e. EO1 and IO both differed from EO2 and EO3, rLBI had differences between IO and EO3 only (Figure 3.3a vs. b) whereas older adults had no site differences.

Table 3.4: Means and standard deviations of abdominal muscles amplitude (RMS) and temporal (PC) features with muscle main effects or group by muscle interactions

	Group	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO
Trunk Stability Task (TST)													
RMS	Comb	11.1	11.1	12.0	11.2	17.8^{ab}	17.7^{ab}	17.0^{ab}	17.7^{ab}	17.4^{ab}	16.0^{ab}	15.6^{a}	15.1
(%MVIC)		±4.7	±4.4	±6.0	±5.1	±10.8	±9.8	±8.9	±9.3	±7.9	±8.4	±8.4	±8.4
PC1	Comb	-49	-77	-59	-96	15^{ab}	23^{b}	4	-32	-16	-34	194^{abcde}	90^{abcde}
		±121	±136	±141	±132	±157	±157	±122	±122	±116	±97	±154	±235
PC2	Comb	24	47	-14	7	38	21	25	17	9	-1	-53^{abcde}	6
		±138	±143	±134	±142	±186	±148	±149	±141	±157	±127	±119	±142
PC3	rLBI	33	57	4	31	-6	22	-23	26	-34	7	-6	74
		±119	±149	±117	±122	±84 *	±91	±104 *	±105	±88 *	±102	±87 *	±108
	Older	73	94	44	78	92	40^{f}	59	63	77	68	70	131
		±108	±97	±101	±126	±90	±52	±57	±86	±71	±87	±69	±77
	Con	-47	-26^{f}	-52^{f}	-39^{f}	-8	9	-27	-13	-51^{f}	-30^{f}	11	43
		±65 *‡	±65 *‡	±83 ‡	±83 *‡	±67 *	±81	±66 *	±71 *	±73 *	±75	±71	±81 *
Horizontal Transfer Task (HTT)													
RMS	Comb	3.1^{cdef}	3.3^{cdef}	3.5^{cdef}	3.5^{cdef}	5.3^{f}	4.7^{f}	4.7^{f}	5.0^{f}	5.4^{f}	4.9^{f}	8.0	9.4
(%MVIC)		±2.2	±2.3	±2.5	±2.8	±2.9	±2.3	±2.8	±4.1	±2.6	±2.9	±3.7	±4.9
PC1	rLBI	18	3	8	-4	93	-35	-20	34	**-137**	**195**	**100^{e}**	**-49^{e}**
		±26	±21	±23	±26	±129	±146	±83	±110	**±145**	**±157 ***	**±98**	**±87**
	Older	6	-7	8	-1	17	-27	-10	-16	-63	24	35	-75
		±9	±15	±19	±15	±145	±99	±87	±46	±65	±44	±72	±76
	Con	0 ^{e}	1 ^{e}	17 ^{e}	0 ^{e}	**61de**	**-101de**	**-151**	**81**	**-218**	**192**	**67de**	**-79de**
		±26	±43	±32	±29	**±180**	**±158**	**±142**	**±153**	**±191 ***	**±179 ***	**±84**	**±94**

Electromyographic amplitudes are reported as root mean square (RMS) normalized to maximum voluntary isometric contractions (%MVIC). Temporal patterns are quantified as principal component (PC) scores. Significant post-hoc group differences denoted by symbols to show differences relative to older adults (*) and rLBI (‡). Muscle differences from muscle main effects (groups combined = Comb.) and group by muscle interactions are denoted by: bold lettering to represent asymmetry (differences) between right and left sites, and superscript letters to show differences between a=LRA, b=URA, c=EO1, d=EO2, e=EO3, f=IO for ipsilateral muscle sites.

Table 3.5: Means and standard deviations of back extensor muscles amplitude (RMS) and temporal (PC) features with muscle main effects or group by muscle interactions

	Group	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52
Trunk Stability Task (TST)													
RMS	rLBI	3.8^{def}	3.5^{ef}	5.9	4.1	5.0^{ef}	4.3^{ef}	6.3	4.8^{ef}	8.8	7.2	8.7	7.3
(%MVIC)		±1.7	±2.0	±2.5	±2.1	±2.3	±1.6	±2.7	±1.7	±4.0	±2.6	±4.4	±3.3
	Older	4.8	4.4	6.6	5.6	4.3	3.6	4.4	3.9^{f}	6.5	5.5	6.2	6.1
		±1.8	±1.4	±2.1	±2.1	±1.2	±1.3	±2.0	±1.5	±2.1	±3.4	±2.6	±2.4
	Con	2.2^{ef}	1.9^{ef}	3.5^{ef}	2.4^{f}	3.2^{ef}	3.0^{f}	2.9^{ef}	3.6	4.9	3.8	5.1	4.7
		±1.1	±0.8	±1.7*‡	±0.9	±3.0 ‡	±2.2 ‡	±1.8 ‡	±4.0 ‡	±3.2 ‡	±2.1 ‡	±3.8 ‡	±3.2 ‡
		*‡	*‡		*‡								
PC1	Comb	-68	-30	**-102**	**14** ^{a}	-8 ^{ab}	10	5^{ab}	24^{a}	41^{ab}	43^{a}	21^{ab}	23^{a}
		±128	±125	**±135**	**±153**	±106	±105	±8	±86	±72	±82	±101	±111
Horizontal Transfer Task (HTT)													
RMS	Comb	17.7	16.8	17.7	15.6	16.7^{f}	15.6	13.7^{abcf}	12.5^{abcf}	12.6^{abcf}	11.3^{abcf}	19.7	17.9
(%MVIC)		±8.7	±7.4	±8.9	±6.6	±8.3	±7.8	±8.5	±6.9	±8.4	±6.8	±8.6	±8.0
PC1	rLBI	**-227**	**305**	**-316**	**413**	**-197**	**240**	**-245**	**303**	**-181^{b}**	**208^{b}**	**-88^{b}**	**90^{abd}**
		±105	**±95**	**±129**	**±126**	**±109**	**±81**	**±85**	**±101**	**±76**	**±94**	**±93**	**±97**
	Older	**-216**	**269**	**-321**	**366**	**-201^{b}**	**201**	**-300**	**314**	**-301**	**312**	**-117^{b}**	**43^{abcde}**
		±69	**±102**	**±58**	**±94**	**±61**	**±95**	**±60**	**±115**	**±92**	**±163**	**±64**	**±60**
	Con	**-296^{b}**	**339^{b}**	**-407**	**488**	**-264^{b}**	**250^{b}**	**-348^{b}**	**340^{b}**	**-280^{b}**	**230^{b}**	**-145^{abcde}**	**73^{abcde}**
		±88	**±125**	**±97**	**±140**	**±95**	**±117**	**±114**	**±153**	**±109**	**±128**	**±98**	**±80**
PC2	Comb	41	41	-21^{acf}	-43^{acf}	47	59	-10^{acf}	-27^{acf}	-66^{abcdf}	-99^{abcdf}	47	54
		±67	±64	±74	±86	±72	±66	±68	±71	±67	±63	±65	±78

Electromyographic amplitudes are reported as root mean square (RMS) normalized to maximum voluntary isometric contractions (%MVIC). Temporal patterns are quantified as principal component (PC) scores. Significant post-hoc group differences denoted by symbols to show differences relative to older adults (*) and rLBI (‡). Muscle differences from muscle main effects (groups combined = Comb.) and group by muscle interactions are denoted by: bold lettering to represent asymmetry (differences) between right and left sites, and superscript letters to show differences between a=L13, b=L16, c=L33, d=L36, e=L48, f=L52 for ipsilateral muscle site.

Two PCs explained 90±4% VAF for back extensor sites. PC1 captured a differential of activation during right-hand transfer (0-35%) and left-hand transfer (60-100%) (Figure 3.5b). A group by muscle interaction ($F(22,484)=3.36$, $p<0.001$) showed muscle site differences within groups. In controls, L16 had higher absolute magnitude scores than all other muscle sites, capturing greater responsiveness to the lateral flexion moment, similarly other sites had higher scores than the multifidus (L52) (Table 3.5). Older adults and rLBI had fewer between muscle site differences compared to controls, indicative of more synergistic activation (Table 3.5). No between group difference were identified. However, since PC1 captured the directionality of responsiveness to the lateral flexion moment, positive scores (left sites with high to low activity) (Figure 3.3e) and negative scores (right sites with low to high activity) canceled each other (Figure 3.3f). To quantify responsiveness to the lateral flexion moment regardless of direction PC1 scores were transformed (absolute value) and compared in a mixed-model ANOVA (Group by Muscle). A group main effect ($F(2,484)=19.6$, $p<0.001$) showed that rLBI and older adults were less responsive, lower absolute scores (Table 3.3), than controls (Figure 3.3e).

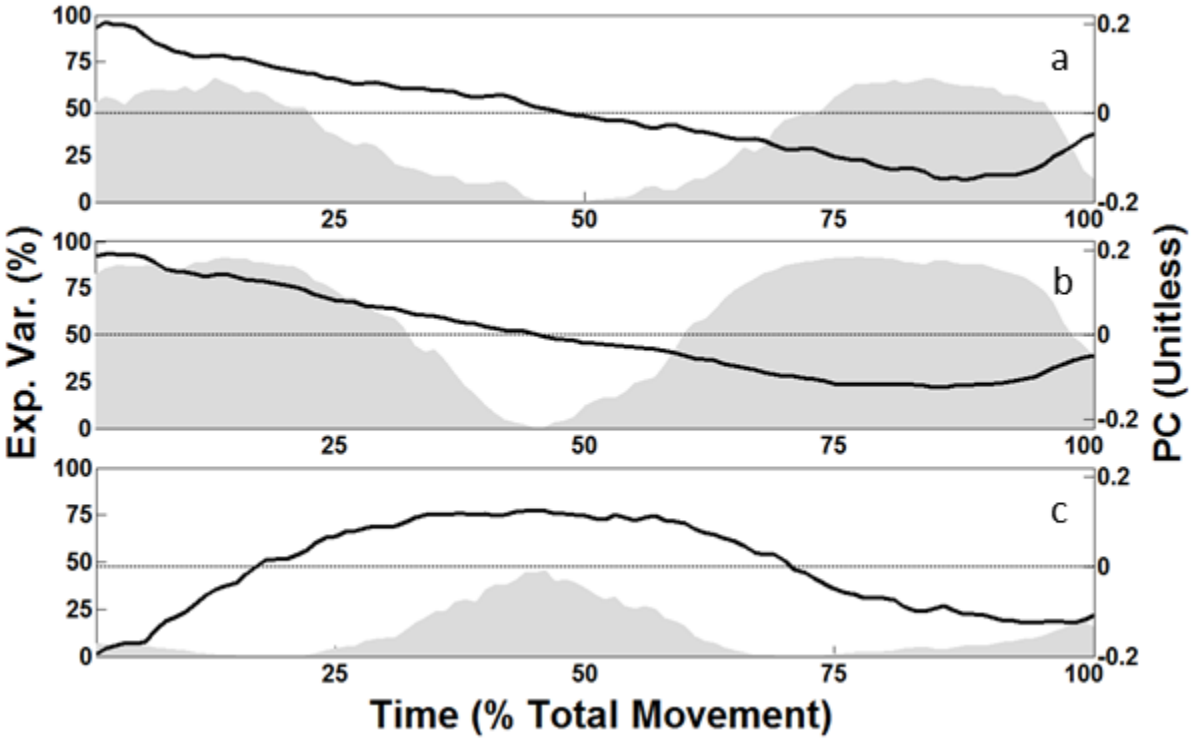

Figure 3.5: Principal component waveforms (black solid line, eigenvectors) from the horizontal transfer task for the abdominals (a) and back extensors (PC 1&2 as b & c). Grey shading represents explained variance for the respective principal component at periods throughout the movement time.

PC2 captured a differential of higher activation during hand transition (45-60%) relative to activation at the beginning (0-15%) and end (85-100%) of the task (Figure 3.5c). A group main effect ($p<0.001$) showed that older adults had lower PC2 scores (Table 3.5), capturing less responsiveness to the flexion moment, than rLBI and controls (Figure 3.3f).

3.3.3 Muscle Main Effects

While not central to this study, as we were interested in group differences and interactions, several RMS and PC scores had significant muscle main effects only ($F(11,484)>7.5$, $p<0.001$) capturing the synergistic strategies amongst trunk muscle sites. The post-hoc results are presented in Table 3.4 and 3.5 for the abdominals and back respectively for completeness with RMS amplitudes and PC scores averaged across all participants (combined) for muscle main effects.

Table 3.6: Means and standard deviations of average abdominal and back extensor muscle activation amplitudes (RMS) expressed as a percent difference to the young control population.

	rLBI (% Difference)	Older (% Difference)
Abdominals		
TST (agonist)	101	122
HTT (antagonist)	145	176
Back Extensors		
HTT (agonist)	153	171
TST (antagonist)	171	150

Data is presented as agonist or antagonist with respect to the movement task. Abdominals represent the agonist for the trunk stability task (TST) and back extensors are the agonist for the horizontal transfer task (HTT)

3.4 Discussion

Given that similar structural and functional impairments have been reported for each group compared to controls in independent studies, this study questioned whether older adults and those with a recovered LBI would have similar neuromuscular compensations. Controls were included to minimize the effect of variables that could alter muscle

activation patterns independent of group assignment. First no individual characteristics such as mass, height and sex distribution were different among groups. This was important to interpretation as the former two can influence the moment produced around the spine whereas sex distribution could influence muscle strength and muscle activation patterns (Hubley-Kozey et al., 2012). Second, timing and motion were not different among groups, except for the greater (<3°) pelvis axial rotation during the TST in older adults compared to rLBI. In addition, both older adults and rLBI had greater trunk axial rotation (<2°) during the HTT than controls. This suggests a reduced ability to minimizing motion, however for both tasks, changes in axial rotation were small and would have minimal effect on the external moments produced, which were primarily due to the external load (Hubley-Kozey et al., 2012). These similarities in motion and timing support that the forces the muscles responded to had a similar pattern for each group and would not explain the differences in patterns among groups. Thirdly, pain levels reported in rLBI (Table 3.1) were lower than the VAS threshold reported with modified function (Boonstra et al., 2014). Indeed, the majority of rLBI (9/16) reported no pain (VAS <4 /100mm) (Jensen et al., 2003) and only 3 reported mild pain (VAS >20, 20 & 27 /100mm) (Rainville, Feine, Bushnell, & Duncan, 1992). Removing these 3 participants from the rLBI group did not affect the key results of this study based on a sensitivity analysis. Thus, the differences observed in muscle activation patterns between the older adult and rLBI groups and with respect to the asymptomatic control group were not influenced by pain reported at time of testing.

The results of this study do not support hypothesis 1, as older adults and rLBI differed from one another on certain muscle activation features. Consistent with the secondary hypothesis older adults and rLBI had different muscle activation patterns than controls for specific muscle activation features. While the direction for the adaptation in many cases was the same, the magnitude of these adaptations was different for the older adults and rLBI relative to the controls. Hence, the results do not support the secondary hypothesis. The discussion attempts to explain these findings related to muscle activation amplitudes, temporal patterns, and synergies based on current literature on spinal system changes and Panjabi's theoretical model of spinal stability.

3.4.1 Neuromuscular Control Differs from Controls

3.4.1.1 Amplitude Differences

We evaluated two tasks (TST & HTT) designed to challenge the abdominals or back extensors respectively. During the TST, the abdominals function as agonists to prevent anterior pelvic tilt. Older adults had higher abdominal activation amplitudes (Table 3.3) whereas the rLBI group was similar to controls. This suggests a compensation for reduced trunk flexor strength which has been reported between older adults and controls (Hasue et al., 1980). No difference between rLBI and controls is consistent with reports of similar trunk flexor strength between those with a history of LBP and age matched controls (Hultman, Nordin, Saraste, & Ohlsèn, 1993; McGill, Grenier, Bluhm, Preuss, Brown, & Russell, 2003b). During the HTT the back extensors function as agonists and both rLBI and older adults had higher activation amplitudes than controls (Table 3.3), consistent with cross-sectional evidence that back extensor strength is reduced in both populations (Hasue et al., 1980; Newton et al., 1993) compared to young controls. The higher abdominal RMS amplitudes for older adults support lower abdominal strength compared to the younger participants recovered from an LBI (Table 3.3), whereas the back extensor RMS amplitudes do not clearly support a similar deficit in back extensor strength (Table 3.3).

3.4.1.2 Muscle activation amplitudes when muscles functioned as antagonist

For the HTT, older adults and rLBI had higher abdominal antagonist activation amplitudes than controls (Table 3.3), with older adults having higher activation than rLBI. To understand how activation amplitudes varied between tasks this study calculated activation ratios relative to the asymptomatic control population. The premise behind this measure was to determine whether changes in activation amplitudes were a consequence of strength deficits. If increases in activation were explained by strength deficits only, the activation ratios would be similar between tasks. Differences between tasks suggest activation gain favoured muscles when they functioned as agonist or antagonist. Comparing activation ratios, shows that abdominal muscles had greater gains when they

functioned, as antagonist (HTT) over agonist (TST) for both older adults and rLBI (Table 3.6), suggesting increased activation was not explained by strength deficits alone.

The rLBI group had higher activation for all back extensor sites during the TST whereas the older adults had higher activity compared to controls for the four upper level sites only (Table 3.5). Thus, the rLBI group increased antagonist activation amplitudes consistently across all back sites, which was not the case in the older adults. The activation ratios shows rLBI increased back extensor activation gain when acting as antagonist (TST) than when they functioned as agonists (HTT) (Table 3.6). In contrast, older adult ratios were similar between tasks (Table 3.6) suggesting increased back extensor activation likely compensated for strength deficits alone.

Higher antagonist activation observed in older adults and rLBI can function to increase active spinal stiffness (Brown et al., 2006; Granata & Marras, 2000; Kavcic et al., 2004) to compensate for decreased passive stiffness, as a consequence of disc degeneration or other changes to the osteoligamentous structures (Galbusera et al., 2014; Sengupta & Fan, 2014). Higher muscle activation comes with a cost such as increased risk of muscular fatigue (van Dieën, Putten, Kingma, & de Looze, 2008) hindering the capacity of trunk musculature to restore stability (van Dieën, Luger, & van der Eb, 2011). It is important to note while EMG differences were small (2-3% MVIC), they are within a range that can impact both spinal stability (Cholewicki et al., 1997) and the rate of muscular fatigue (van Dieën et al., 2008), suggesting these differences have clinical implications.

3.4.1.3 Temporal Differences

The temporal differences observed were consistent with previous work showing rLBI and older adults are less responsive to changing external moments than controls (Claudino et al., 2013; D'hooge, Hodges, et al., 2012b; Hubley-Kozey et al., 2009; Jones et al., 2011; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014). During the TST, PC3 differences (Table 3.4) showed both rLBI and older adults had higher relative abdominal activation during 90° hip flexion compared to controls (Figure 3.2b&c), despite a reduction in the sagittal plane moment (Figure 3.1). For the HTT, older adults and rLBI were less responsiveness to the lateral flexion moment captured by fewer differences between paired left and right oblique sites i.e. synergistic recruitment or greater co-

activation (Table 3.4, PC1) (Arjmand, Shirazi-Adl, & Parnianpour, 2008b; Brown & Potvin, 2007) and a lower absolute magnitude for back extensor muscles (Table 3.3, |PC1|) (Figure 3.3e). Compared to rLBI, older adults were less responsive to changing external moments as evidenced by fewer differences between paired left and right abdominal sites during the HTT (Figure 3.3a vs. b) (Table 3.4, PC1) and higher PC1 & 3 scores in abdominal sites during the TST (Table 3.3 & 3.4). Furthermore, the older adult back extensors were less responsive to the flexion moment produced during hand transition (Table 3.3, PC2) for the HTT relative to both rLBI and controls (Figure 3.3f). Decreased responsiveness may reflect a reduced ability to utilize feedback control to modulate muscle activation (Reeves, Narendra, & Cholewicki, 2011) consistent with the proprioceptive (Brumagne et al., 2004; Goble et al., 2008; Osthoff et al., 2015) and reflexive deficits (Klass et al., 2011; Ramprasad et al., 2010) reported for older adults and those with LBP compared to reference controls.

Most temporal differences support that rLBI and older adults are less responsive to changing external moments, except one group difference (PC2) during the TST. This feature captured that the abdominal sites of older adults were more responsive (Table 3.3), experienced a greater reduction in activation during right leg lowering compared to controls and rLBI (Figure 3.2a), with rLBI having greater responsiveness than controls (Figure 3.2b). This finding suggests greater feedforward control where participants anticipated the changes in lumbar loading associated with transitioning between single leg support and no-leg-support (Hubley-Kozey et al., 2009; Moreside et al., 2013). Increased anticipation is supported by electroencephalography findings showing that older adults and LBP participants are more likely to recruit frontal cortical structures earlier and with a greater magnitude when initiating movement compared to controls (Berchicci, Lucci, Pesce, Spinelli, & Di Russo, 2012; Inuggi et al., 2009; Sadeghi, Talebian, Olyaei, & Moghadam, 2016). Increased feedforward activation may also explain other group differences including the decreased temporal responsiveness and general increased antagonist activation as a strategy to compensate for impaired feedback control.

3.4.2 Older adults and rLBI have Less Motor Complexity Reflected by Altered Muscle Synergies.

Within this study, all amplitude and temporal characteristics for the control and combined data resulted in muscle site differences consistent with previous work showing trunk muscles are activated relative to their mechanical advantage (Arjmand, Shirazi-Adl, & Parnianpour, 2008b; Brown & McGill, 2007a; 2005; Butler et al., 2010; Hubley-Kozey et al., 2013; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014; Rashedi, Khalaf, Nassajian, Nasseroleslami, & Parnianpour, 2009). Thus, in young healthy controls, trunk muscles sharing mechanical properties have spatial-temporal synergies, whereas unique trunk muscle sites exhibit spatial complexities (differences between sites) (Butler et al., 2010; Hubley-Kozey et al., 2012; Sedaghat-Nejad et al., 2015). Of specific interest to the main study objectives, two comparisons for the TST and three for the HTT had group by muscle interactions, capturing differences in the synergistic relationship between muscle sites among the three groups. In general non-synergistic muscle activation, captured by left-right differences and differences between ipsilateral muscle sites, show controls were more likely to have spatial complexity, in both overall activation amplitudes (Table 3.5, TST(RMS)) and temporal recruitment (Table 3.4, TST(PC3) & HTT(PC1)) (Table 3.5, HTT(PC1)) than older adults and rLBI indicative of independent recruitment of specific trunk muscle sites. Furthermore, the younger rLBI had greater motor complexity than older adults for temporal recruitment of both abdominal and back sites during the HTT and for overall back site activation amplitudes during the TST (Table 3.4, HTT(PC1)) (Table 3.5, TST(RMS) & HTT(PC1)).

Reduced motor complexity could be explained by multiple phenomena. First according to Panjabi's model, if rLBI and older adults experience corruption to somatosensory feedback (Panjabi, 2005), increased afferent noise would interfere with the ability to recruit individual muscle sites, resulting in more synergistic activity (Giszter, Hart, & Silfies, 2009; Reeves et al., 2011). Second, the explanation may reside in the central nervous system where older adults and those with LBP experience reorganization within the motor (Papegaaij, Taube, Baudry, Otten, & Hortobágyi, 2014; Reuter, Behrens, & Zschorlich, 2015; Schabrun, Burns, & Hodges, 2015a; Schabrun, Elgueta-Cancino, & Hodges, 2015b; Tsao, Danneels, & Hodges, 2011; Tsao, Galea, &

Hodges, 2010) and somatosensory cortex (Flor, Braun, Elbert, & Birbaumer, 1997; Goble et al., 2011; Schabrun, Burns, & Hodges, 2015a). Less focal recruitment of muscles (Reuter et al., 2015; Schabrun, Elgueta-Cancino, & Hodges, 2015b; Tsao et al., 2011), consistent with reorganization, could explain greater synchrony of the time-varying recruitment of ipsilateral muscle sites that have cortical overlap (Meier, Aflalo, Kastner, & Graziano, 2008).

This study compared overall (RMS) amplitude or magnitude of PC scores among muscle sites to assess muscle synergies. While this approach can quantify muscular synergies it differs from studies that have compared muscle sites in the spatial domain to produce muscle synergy vectors (Butler et al., 2010; 2012; Gizzi, Muceli, Petzke, & Falla, 2015; Sedaghat-Nejad et al., 2015). Thus, the methodology of the current study is limited in characterizing spatial synergies that may be captured if decomposition were performed to produce a muscle synergy vector. However, if the mechanical features of a task remain constant dimensionality can be reduced in the temporal domain, where each participant-muscle waveform is given a unique score (coefficient) showing how to scale each waveform to fit a temporal feature (Hubley-Kozey et al., 2012; Ivanenko et al., 2004; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014). The strength of this approach is muscle sites are compared directly by considering the magnitude of the unique scores, rather than a qualitative description that muscles should be linked based on similarities/ differences conveyed in the muscle synergy vector, while still capturing similar muscle synergies that have been reported using the muscle synergy vector approach to analyze the HTT (Butler et al., 2010).

3.4.3 Older adults and rLBI have Unique Neuromuscular Adaptations to Spinal System Impairments.

This study found differences in muscle activation patterns between rLBI and older adults. Thus, some findings do not support the hypothesis that similar spinal system impairments would result in similar neuromuscular adaptations in both groups. This could in part be explained as older adults are more likely to experience greater spinal system impairments than those younger but recovered from an LBI as a consequence of cumulative exposure to joint loading and injuries (Seidler et al., 2009). Consistent with

this theory both older adults and rLBI differed in the same way (higher agonist amplitudes, reduced responsiveness, and less motor complexity (see above)) relative to controls. However, mean data (RMS and PC Scores) consistently shows older adults had the largest difference compared to controls, whereas means data of rLBI were somewhere between these two groups (Table 3.3-5). Thus, older adults required more muscle activation pattern adaptations than rLBI to compensate for greater spinal system impairments.

The one exception to this systematic group difference in muscle activation patterns was that rLBI had higher antagonist activation amplitudes than older adults (Table 3.3, 3.5 & 3.6). This could be interpreted that rLBI utilize higher antagonist activation to increase the stiffness of the spine to compensate for decreased passive stiffness (Cholewicki et al., 1997) or delays in trunk feedback control (Brown & McGill, 2008; Oomen, Reeves, Priess, & van Dieën, 2015) relative to older adults. Alternatively, this group difference may capture a feature unique to rLBI, i.e. the recent experience of pain. While the participants' in the rLBI measures of pain during testing was low and should not modify function, their pain in the recent past could explain alterations in muscle activation patterns according to the motor adaptation to pain model. This model suggests that when individuals experience pain they modify muscle activation to increase joint stiffness (Hodges et al., 2013; Wong et al., 2016) and it is proposed that in some individuals these muscle activation patterns may persist post pain (Hodges & Tucker, 2010). The present study shows higher antagonist co-activation in rLBI despite the resolution of pain, providing indirect evidence supporting Hodges's model. These results provide evidence that older adults experience greater spinal system impairments, but that pain adaptation contributes to muscle activation patterns independent of the impairments in individual spinal systems. Collectively these results support that muscle pattern adaptations may depend dependent on magnitude of spinal system impairment with some adaptations influenced by motor learning to recent pain. However, for a more complete understanding, direct evidence can determine the extent that changes in muscle activation patterns relate to magnitude of changes in a spinal system and motor learning to pain.

3.5 CONCLUSION

In conclusion, the abdominal and back extensor muscle activation patterns in both older adults and those recovered from LBI shared common differences compared to healthy young asymptomatic controls. This included higher activation amplitudes, less responsiveness to changing external moments, and less motor complexity supporting the existence of alterations that result from, or aim to improve spinal stability in the presence of, spinal system impairments. In addition, direct comparisons between the older adult and rLBI groups showed that differences existed in muscle activation patterns between these two groups. In most instances, compared to controls, the older adults had greater changes in activation amplitudes of agonist, temporal responsiveness, and motor complexity suggesting greater sub-system impairments compared to the recovered LBI group. However, the higher antagonist activation amplitudes for the rLBI relative to older adults suggest either rLBI have unique spinal system deficits or that their recent experience of pain has an independent influence on muscle activation patterns.

CHAPTER 4
TRUNK MUSCLE ACTIVATION PATTERNS DIFFER BETWEEN THOSE WITH LOW AND HIGH BACK EXTENSOR STRENGTH DURING A CONTROLLED DYNAMIC TASK

4.1 INTRODUCTION

The osteoligamentous spine is inherently unstable, unable to return to equilibrium under even low (80-90N) compressive loads (Crisco et al., 1992). To explain how the in vivo spine remains stable under higher loads Panjabi proposed a theoretical model based on the premise that the tissues surrounding the spine can prevent excessive vertebral body motion, or a clinical instability event, resulting in tissue damage thought to be one cause of incapacitating pain (Panjabi, 2003). This model categorizes tissues into three functional systems; passive, active and neural (Panjabi, 2003). Regarding the risk of instability, this model has two key postulations. First, is that a deficit to any one spinal system can increase the risk of an instability event, and second, as no one system alone contributes to spinal stability, other systems can modify their function to compensate for a deficit (Panjabi, 2003). There is however a lack of empirical evidence on whether deficits in individual spinal systems change the function of the other systems.

This paper focused on active spinal system. Animal (Hodges et al., 2015) and human studies (D'hooge, Cagnie, et al., 2012a; Kjaer et al., 2007) show musculotendinous tissues are different in those with low back injuries (LBI) compared to healthy controls including: reduced muscle cross sectional area and increased intramuscular fat. While these features appear consistently in individuals with chronic LBP (Steele, Bruce-Low, & Smith, 2014b), evidence is inconclusive whether individuals in the acute phase of a LBI or those who experience recurrent LBP have similar musculotendinous changes (Goubert, Van Oosterwijck, Meeus, & Danneels, 2016). Functionally, the active system generates forces through active muscle contractions that are transmitted to bones. Trunk muscle strength, defined as the maximal force / moment produced under volitional effort (Larivière et al., 2002), can be predicted by knowing the size (Guilhem, Giroux, Couturier, & Maffiuletti, 2014; Hultman et al., 1993) and composition (intramuscular fat content) of trunk muscles (Anderson, Bean, Holt, Keel, & Bouxsein, 2014). Consistent

with the structural changes, participants with chronic LBP have reduced trunk strength (Hasue et al., 1980; Newton et al., 1993) compared to asymptomatic controls (for review see (Steele, Bruce-Low, & Smith, 2014a)) whereas similar reductions in strength was not found in those with a history of LBP compared to pain free controls (Hultman et al., 1993; McGill, Grenier, Bluhm, Preuss, Brown, & Russell, 2003b).

Whether changes in the active system are associated with an increased risk of instability is equivocal. A recent meta-analysis found no conclusive evidence that either the size or composition of trunk muscles is predictive of risk of future LBP (Suri, Fry, & Gellhorn, 2015). Earlier studies report no relationship between baseline measures of back extensor strength and the risk of future LBP (Lee et al., 1999; Newton et al., 1993) and a more recent review inconclusive on the predictive ability of baseline measures of strength (Reenen et al., 2007). Despite this some studies have shown participants with lower back extensor strength (Cho et al. 2014) or lower extensor to flexor strength ratios (Lee et al. 1999) are at an increased risk of experiencing a low back injury in a 2 and 5 year follow up respectively. Furthermore, strength training is shown to reduce the risk of workplace LBI (Carpenter & Nelson, 1999), with a recent meta-analysis suggesting exercise alone or in combination with education is one of the most consistent methods to prevent LBP (Steffens et al., 2016).

The above studies suggest the active system may be important for maintaining spinal stability, and yet the effect of active system deficits, characterized as reduced trunk strength, alone have limited influence on LBP risk. According to Panjabi's theory of spinal system compensation, a deficit in the active system, should result in adaptations to the other systems to enable an individual to complete a functional task without experiencing an instability event. The most immediate way to change the stability of the spine is to adjust the time-varying activation of the muscles (active system) by modifying the function of the neural system (neuromuscular control) (McGill, Grenier, Kavcic, & Cholewicki, 2003a). Neuromuscular control can be measured by evaluating electromyography (EMG) where the amplitude of the EMG signal has been related to changes in muscle force through both linear (Brown & McGill, 2007a; Larivière et al., 2002) or curvilinear (Larivière et al., 2002) relationships. Thus, in agonist muscles, after considering activation necessary to overcome increased antagonist activation amplitudes

(Brown & McGill, 2007a), individuals who recruit greater EMG amplitudes, normalized to maximum voluntary contractions, to perform a task likely have reduced strength (Quirk & Hubley-Kozey, 2018).

Although the general EMG to force relationship is accepted, understanding how maximum force production relates to the EMG activation during functional tasks is less well understood. Studies show that individuals who have less lower limb muscle strength require higher activation amplitudes of agonist muscles during a variety of tasks involving the knee (Mizelle, Beam, & DeVita, 2003; Takai et al., 2008) and ankle joints (Takai et al., 2008). These relationships suggest stronger individuals require less EMG activation amplitudes of agonist muscles but not whether changes in strength influence the time-varying activation of muscles during a dynamic task. Studies comparing the spatial-temporal response of trunk muscles show that making a task more challenging not only increases agonist and antagonist activation amplitudes but also increases the responsiveness (relative increase in muscle activation amplitudes) of trunk muscles to changing external moments (Butler et al., 2010; Quirk & Hubley-Kozey, 2014). Thus, weaker individuals should have modified temporal responses of trunk muscles compared to stronger individuals.

Finally, while trunk muscle activation may differ between stronger and weaker individuals, there is evidence that pain can independently modify how individuals recruit their trunk muscles. Inducing experimental pain, increases activation amplitudes (Hodges et al., 2013) and delays muscle onset and offset (Hodges et al., 2003) ultimately modifying the spatial-temporal recruitment of trunk muscles during complex tasks (van den Hoorn et al., 2014). It is theorized that these changes persist following the resolution of pain (Hodges et al., 2013) and may explain why the recent experience of pain is related to changes in trunk muscle activation during functional tasks (Quirk & Hubley-Kozey, 2018).

The aim of this study was to probe the spinal system compensation theory by assessing the interaction between active spinal system function and neuromuscular control of the trunk muscles during a dynamic task. This experiment tested for a difference in abdominal and back extensor muscle activation amplitudes and patterns during a controlled dynamic lifting task between those who produce high (STRONG)

versus low (WEAK) back extensor moments as an assessment of active spinal system function. The hypothesis was that the WEAK group would recruit higher EMG activation amplitudes and have greater responsiveness to changing external moments during the task than STRONG. A secondary purpose was to determine whether the recent experience of a low back injury modifies the trunk muscle activation differences between those with high and low back extensor strength.

4.2 METHODS

4.2.1 Participants

Participants for this study were recruited from the Canadian Armed Forces. Asymptomatic participants volunteered by responding to on base posters and base-wide recruitment e-mails. Recovered LBI (rLBI) participants were identified if they reported a back related issue resulting in altered activities of daily living for at least 3 days (Ozguler et al., 2000) to the Canadian Forces Health Services Center and were contacted to see if they were interested in participating in the study. For both groups a self-report questionnaire screened for the following exclusion criteria: previous abdominal or back surgery, cardiovascular, respiratory or neurological conditions that place them at risk for participating in the study. For participant in the rLBI group questions determined that their recent LBP occurred 4-12 weeks prior to their data collection, was not chronic lasting longer than 12 weeks, and not recurrent where a previous injury occurred within 12 weeks prior to their most recent episode (Delitto et al., 2012). Asymptomatic participants were screened to determine that they had not experienced an activity limiting LBP (Ozguler et al., 2000) in the last year. All rLBI were deemed recovered at the time of testing reporting minimal pain (Visual Analog Scale (VAS) <30/100mm (Boonstra et al., 2014)), minimal disability (Roland Morris Disability (RMD) <9/24 (Roland & Morris, 1983; Stratford et al., 1998)), and the resumption of normal activities of daily living at the time of testing. Before testing all participants signed an informed consent approved by the Institutes Research Ethics Board.

4.2.2 Protocol

Participants attended two sessions. During session one all participants were screened by a registered physiotherapist to determine whether they met the minimal pain and disability criteria and confirmed the absence of neurological conditions. During session two participant's pain, demographic and anthropometric data were measured. Self-reports of weekly engagement in physical activity (aerobic sessions >30 minutes, core/ abdominal and strength training) and if they frequently lifted objects weighing over 23kg for their work, defined as a heavy job (Seidler et al., 2009) were recorded. Participants also filled out a Tampa Kinesiophobia (Kori et al., 1990) and Pain Catastrophizing Scale (Sullivan et al., 1995) to characterize their beliefs towards pain.

Participants were prepared for surface EMG (Section 4.2.3.1) and motion capture (Section 4.2.3.2) data collection. Participants then performed three trials of a highly controlled standardized right-to-left horizontal transfer task (Butler et al., 2010). Briefly, to a five-second count, participants were instructed to on "1" lift a 3kg mass orientated 60˚ to the right of the midline of their body with their right hand, on "3" transferring the mass to their left hand at the midline of their body, and then on "5" lower the mass 60˚ to the left of their midline. Mass lift and lower were recorded using a pressure sensor located at bottom of the mass, and hand transition was determined using an optoelectric switch. A height-adjustable table was used to ensure the mass was lifted just below elbow height, and the optoeletric switch ensured the mass was lifted no higher than 5cm. During the task, a researcher ensured the participant lifted the mass with the arm in full extension. Participants were asked to minimize trunk motion aided by tactile feedback provided to the upper thoracic spinous process.

Participants then performed a series of maximum effort voluntary isometric contractions (MVIC) for EMG amplitude normalization including: i) supine sit-up, ii) side lying lateral flexion (left and right), iii) prone back extension, and iv) prone back extension coupled with back extension (right and left) (Butler et al., 2010) with each exercise repeated (two trials). Participants were encouraged to maintain maximal effort for 3 seconds against non-elastic straps, with a one-minute rest between contractions to minimize fatigue.

Trunk strength (flexor and extensor moment production) was measured during two MVIC exercises. Participants were positioned in a prone or supine crook lying position with the HUMAC Norm Dynamometer (Computer Science Medicine Inc, Strongton, MA, USA) arm positioned anterior and inferior to the clavicle for trunk flexion, or superior and posterior to the spine of the scapula for trunk extension (Hasue et al., 1980). The HUMAC centroid was positioned approximately 5cm anterior to the posterior superior iliac spine, in line with the iliac crest, and non-elastic straps were secured to anchor the pelvis and shank. Following gravity correction, participants performed two trials with instructions and procedures consistent with the normalization tasks.

4.2.3 Data Collection and Analysis

4.2.3.1 Surface Electromyography

Surface electrodes (Ag/AgCl, 10mm diameter, Red dot, 3M, Maplewood, MN) were placed in a bipolar configuration (30mm interelectrode distance) over 12 bilateral muscle sites using a standard protocol (Butler et al., 2010). Abdominal sites included the upper and lower rectus abdominus (URA & LRA), the internal (IO), and external (EO1-3) oblique sites representing the anterior, lateral and posterior fibres respectively. Back extensor sites included the superficial quadratus lumborum (L48) and multifidus (L52) along with the erector spinae at level of the 1st and 3rd lumbar spinous approximately 3 and 6 cm horizontal to the midline to capture the longissimus (L13 & L33) and iliocostalis (L33 & L36) fibres respectively (Butler et al., 2010).

EMG signals were pre-amplified (500x) and further amplified using three AMT-8 amplifiers (band-pass 10-1000Hz, CMRR=115 dB, input impedance 10GΩ; Bortec Inc., Calgary, AB). EMG signals and event markers were digitized at 2000Hz using a 16-bit (±5V) analog-to-digital board (PCI-6033E, National Instruments, Austin, TX) and Labview^{TM} (Version 2017, National Instruments). Custom Matlab (Math Works, Natick, MA) code corrected EMG signals for participant bias, electrocardiogram artifact (high pass zero-lag 30Hz filtered) (Butler et al., 2007) and noise from electromagnetic sources (inverse fast-Fourier filtered removing frequency spikes with a power 3 times greater than their nearest frequency (+/-15Hz) neighbours). Corrected filtered data were rectified

and low pass filtered at 6Hz using a second order zero-lag Butterworth filter, to produce a linear envelope.

EMG amplitudes were normalized to the maximum average 500ms moving window linear envelope regardless of what task evoked the MVIC (Vera-Garcia et al., 2009), and time normalized from 0-100% of the total task time (event markers) using a quadratic interpolation algorithm. Ensemble average waveforms were calculated from the three trials. To characterize overall neural drive the average (% MVIC) activation was calculated for the waveform for the entire task. To characterize spatial temporal characteristics, ensemble average waveforms for each participant (N) and muscle site (12) created two data matrices ((N*12) x 101) for the abdominals and back extensors separately. Each data matrix was entered into a principal component (PC) analysis model (Hubley-Kozey & Vezina, 2002). Briefly, each data matrix was transformed into a covariance matrix and underwent an eigenvector decomposition to identify eigenvectors (PCs) which explained patterns of maximum variation within the waveforms. For each EMG waveform, a coefficient (PC score) was calculated to fit the PC to the original waveform. The number of PCs extracted was determined such that the total explained variance explained by the combinations of PCs reached 90%, and that each PC explained at least 1% variance (Hubley-Kozey & Vezina, 2002).

4.2.3.2 Kinematic and Kinetic Analysis

Passive reflective markers were positioned on the suprasternal notch and 7th cervical spinous process along with 16 bilateral landmarks on the limbs: 5th and 2nd metacarpal, radial styloid, ulnar styloid, medial and lateral humeral epicondyle, the mid acromion clavicular joint, and the anterior superior iliac spine. Four marker rigid body clusters were affixed to the thorax and pelvis, along with bilateral clusters on the forearm and upperarm to capture motion. Following marker setup, a single standing calibration trial captured the marker position relative to the rigid bodies using six infrared emitting cameras (ProReflex 240, Qualisys^{TM}, Goteborg, Sweden) sampled at 100Hz using Qualisys Track Manager Software (Version 2.10, Qualisys^{TM}, Goteborg, Sweden). For synchronization, the Labview program collecting analog-to-digital data triggered the motion capture system.

Marker data were processed in Qualisys Track Manager, to label marker's coordinates. Marker coordinate data were entered into a custom Matlab™ script for quadratic interpolation of missing data points, and low pass filtered at 4Hz using a fourth order, zero-lag Butterworth filter. Kinematic data were processed in accordance with the International Society of Biomechanics recommendation (Wu et al., 2002; 2005). Within each joint, an anatomical coordinate system was defined using bony landmarks, and joint centers as calculated using regression equations (Dumas et al., 2006). Segment angles were calculated using Euler angles in a Z, Y, X (lateral flexion, axial rotation, flexion-extension) rotation sequence. To quantify the motion during the horizontal transfer task segment angles were low pass filtered using a 1Hz second order, zero-lag Butterworth filter (Butler et al., 2010). For each trial, the total displacement (maximum – minimum) of the torso and pelvis was calculated in lateral flexion, flexion extension, and axial rotation and averaged over the three trials for each participant.

Kinematic data were used to calculate the moments of force around the trunk using a top-down static inverse dynamics approach. Known external mass and segment mass, estimated using anthropometrically derived regression equations (Dumas et al., 2006), were inputs for the joint force and moment calculations using a system of Newton-Euler equations (Winter, 2009). An open kinetic model including joint forces and moments from distal joints were used to determine forces and moments occurring at the proximal joint. The mass of the lifted object was assumed to apply a weight vector at the middle of the 2nd and 5th metacarpal of the right-hand for the beginning of the lift, both hands during hand transition, and the left-hand following hand transition. Following this calculation, the peak lateral flexion and flexion moment was calculated for each trial and then averaged across trials for each participant.

4.2.4 Strength Analysis

For strength testing, gravity corrected external moments sampled from the HUMAC, were digitized at 2000Hz using the same analog-to-digital converter for EMG acquisition (Section 4.2.3.1). Custom Matlab code filtered these data using a 6Hz second order zero-lag low pass Butterworth filter, and converted them to moment (Nm) according to equations provided by HUMAC. The maximum isometric moment for

flexors and extensors was calculated as the highest average moment over a 500ms window which was normalized to body mass to compensate for anthropometric differences between participants (Smith et al., 1985).

4.2.5 Statistical Analysis

To test whether there were differences in muscle activation amplitudes and patterns between those with higher (STRONG) or lower strength (WEAK), a median split approach was applied to the mass normalized back moments, within the rLBI and asymptomatic group. Two-way analysis of variance models (ANOVA) (group and strength) tested for differences in demographics, anthropometrics, maximum flexor and extensor moment, motion, lifting moments, and timing data. Categorical data were compared using a Chi-square test for independence. These data determine if confounders should be included as co-variates when testing for muscle activation (PC) differences. Normality of PC scores was assessed using a Kolmogorov-Smirnov test, with non-normal data transformed using a Johnson transformation. PC scores were analyzed using three-factor (group, strength and muscle) mixed model ANOVAs for the abdominals and back extensors separately. Tukey simultaneous tests compared pairwise differences when significant. Statistical analyses were performed in Minitab (version 17, State Collage, PA). Alpha was set at 0.05 and Bonferroni corrected.

4.3 RESULTS

4.3.1 Anthropometrics, Demographics, Strength and Task Performance

Sixty-nine participants volunteered for the study and of those nine were women. Only two women were in the rLBI group. Given sex modifies muscle activation patterns (Hubley-Kozey et al., 2012) and only 2 women were in the rLBI group, the analyses were performed on the 60 men (30 rLBI reported LBI occurred 9±2 weeks from the day of testing). For the STRONG and WEAK categories, the median back extensor moment threshold was 2.46 and 2.51 Nm/kg for the ASYM and rLBI respectively. Characteristics of the separate participant groups are shown in Table 4.1. No group by strength interactions were identified. Significant group main effects found that the rLBI group had

higher mass (p=0.001), pain (VAS (p=0.001)), disability (RMD (p=0.003)), pain catastrophizing (PCS (p=0.004)) and kinesiophobia (TSK (p<0.001)) scores than ASYM. Non-normalized extensor strength was greater in the rLBI group (p<0.001) but this difference was not significant when strength was normalized to body mass (Table 4.1). Differences between the STRONG and WEAK groups were identified between both the non-normalized (p<0.008), and normalized maximum moment produced for both trunk flexors and extensors (p<0.001) (Table 4.1).

Table 4.1: Demographics, anthropometrics, pain characteristics, self reported physical activity, occupational loading and trunk strength

Strength (n)	STRONG (30)		WEAK (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Age (years)	35.4 (10.4)	34.6 (10.7)	34.5 (8.4)	42.1 (7.2)
Mass (kg)	78.0 (9.5)*	86.5 (13.8)	82.5 (14.0)*	91.8 (15.8)
Height (m)	173.1 (7.6)	178.2 (6.5)	177.4 (8.1)	181.7 (7.2)
BMI (kg/m^2)	26.0 (2.7)	27.3 (4.6)	26.2 (4.1)	27.8 (3.9)
Heavy Job (n (%))	4 (27%)	5 (33%)	4 (27%)	5 (33%)
L. Hand (n (%))	4 (27%)	2 (13%)	2 (13%)	1 (7%)
VAS (mm)	0.9 (2.0)*	5.1 (8.5)	0.3 (1.0)*	7.1 (8.4)
PCS	6.3 (7.7)*	10.9 (8.1)	7.9 (6.9)*	16.9 (11.2)
TSK	30.1 (7.6)*	35.6 (5.2)	29.7 (6.5)*	37.2 (7.0)
Aerobic Training (/ week)	3.7 (1.6)	3.8 (3.2)	5.1 (3.1)	2.8 (2.8)
Strength Training (/ week)	2.6 (1.9)	2.9 (2.7)	2.2 (2.1)	2.1 (2.6)
Core Training (/ week)	1.8 (1.6)	1.5 (1.7)	2.4 (2.2)	2.8 (2.3)
RMD	0.1 (0.3)*	2.4 (2.9)	0.3 (0.8)*	2.2 (2.1)
Flexor Mo (Nm)	139.2 (35.7)	162.3 (34.6)	118.5 (20.0) †	122.0 (31.1) †
Extensor Mo (Nm)	225.3 (25.8)*	263.5 (34.6)	166.8 (39.9)* †	193.7 (42.2) †
Norm Flexor Mo (Nm/kg)	1.8 (0.5)	1.9 (0.3)	1.5 (0.3) †	1.3 (0.3) †
Norm Ext. Mo (Nm/kg)	2.9 (0.3)	3.1 (0.4)	2.0 (0.3) †	2.1 (0.3) †

Significant differences (p<0.05) represented by * to indicate the difference between the ASYM and rLBI group, and † represent a difference between the STRONG and WEAK group. Abbreviations: asymptomatic (ASYM), recovered low back injury (rLBI), body mass index (BMI), left handed (L. Hand), visual analog scale (VAS), pain catastrophizing scale (PCS), tampa scale of kinesiophobia (TSK), roland morris disability score (RMD), moment (Mo), normalized (Norm) and extensor (Ext.).

Table 4.2: Horizontal transfer task performance data, timing, motion and external moments

Strength (n)	STRONG (30)		WEAK (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Time (s)	4.3 (0.3)	4.2 (0.3)	4.2 (0.3)	4.3 (0.3)
Torso Flex/Ext (°)	3.2 (1.6)	5.1 (2.8)	4.1 (1.7)	3.8 (1.5)
Torso Lat. Flex (°)	3.2 (1.2)	2.4 (1.0)	2.7 (1.2)	2.5 (1.6)
Torso Ax Rot (°)	5.6 (2.0)	5.3 (2.8)	4.5 (2.1)	4.3 (1.8)
Pelvis Flex/Ext (°)	1.5 (1.3)	1.7 (1.0)	1.7 (0.9)	1.2 (0.8)
Pelvis Lat. Flex (°)	1.3 (0.8)	1.4 (1.0)	1.4 (0.9)	1.4 (0.9)
Pelvis Ax Rot (°)	2.5 (1.4)	2.6 (1.1)	2.4 (1.3)	2.1 (0.9)
Peak Flexion (Nm)	26.2 (2.6)	28.8 (3.9)	28.2 (3.9)	29.1 (3.4)
Peak Lateral Flexion (Nm)	11.6 (2.5)	12.3 (3.2)	11.9 (2.4)	11.5 (1.3)
Norm Peak Flex (Nm/kg)	0.34 (0.03)	0.33 (0.03)	0.34 (0.02)	0.32 (0.02)
Norm Peak Lat. Flex (Nm/kg)	0.15 (0.04)	0.14 (0.04)	0.15 (0.03)	0.13 (0.03)

No significant group or strength main effects or interactions ($p>0.05$). Abbreviations: flexion (Flex), extension (Ext), lateral (Lat.), axial (Ax), rotation (Rot), normalized (Norm), asymptomatic (ASYM), recovered low back injured (rLBI).

Time to complete the task, trunk and pelvis motion, and peak external moments for the horizontal transfer task are shown in Table 4.2. There were no group or strength main effects or interactions ($p> 0.05$) supporting that the controlled task parameters of timing (approximately 4 seconds), minimizing trunk and pelvis motion and task demand of the external moment was consistent among subgroups.

4.3.2 Trunk Muscle Activation Patterns

To address the primary objective, strength main effects and interactions are first presented along with any group or muscle main effects to fully describe the data. Representative ensemble average profiles for the abdominals, and back extensors are shown in Figure 4.1A and B-D respectively to depict group by strength interactions.

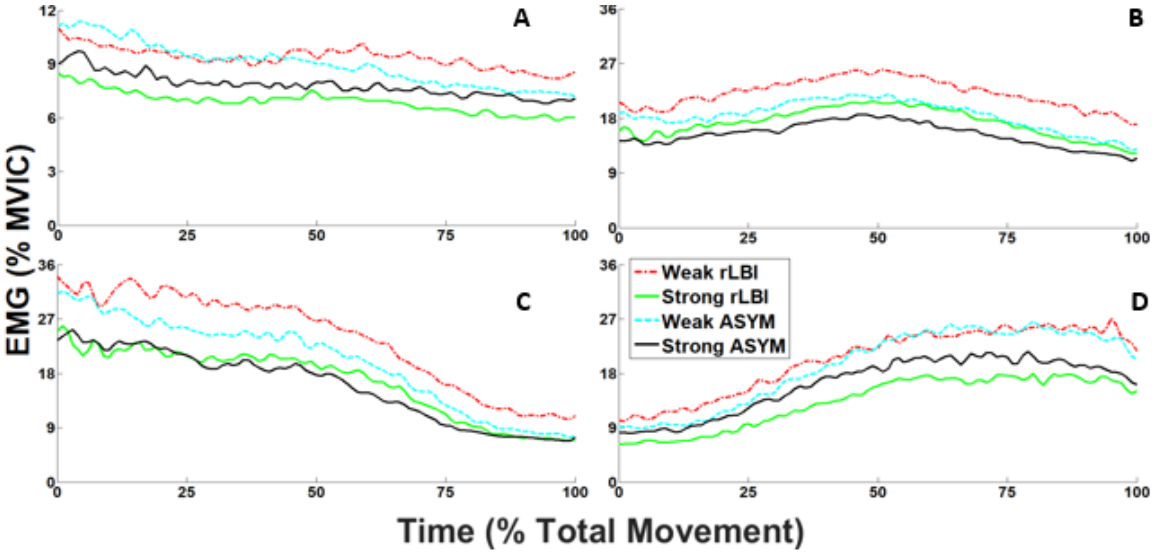

Figure 4.1: Normalized to maximum voluntary isometric contraction (%MVIC) ensemble average trunk electromyograms (EMG) for recovered low back injury (rLBI) and asymptomatic (ASYM) strong and weak subgroups. For the abdominals group by strength interactions are depicted by the contrast between a) the ensemble average of right anterior external (REO1) and internal obliques (RIO) and the left middle (LEO2) and lateral external oblique sites (LEO3). For the back extensors group by strength interactions are depicted by b) the ensemble average of both the right and left medial back extensor sites (L13, L33, & L52) and the c) left and d) right lateral back extensor sites (L16 & L36).

4.3.2.1 Abdominal Muscle Activation Patterns

Two PC's explained 96.5% of the total abdominal muscle activation waveform variance. For both the abdominals and back extensors PC1 captured the overall shape and amplitude of abdominal muscles (Figure 4.2A&E) where high scores captured higher activation amplitudes (Figure 4.2B&F). PC1 scores were highly correlated (r=1) with the average amplitude of muscle sites across the entire task so the average activation amplitudes in %MVIC are also provided to give context to PCs unitless score. Overall the

average amplitudes were less than 8% MVIC for the abdominals (Table 4.3), with the IO sites having the highest value but still less than 12% MVIC (Table 4.4). There was a significant group by strength interaction (p=0.046) where both ASYM and rLBI WEAK groups had higher activation amplitudes than their respective STRONG group (Table 4.3 & Figure 4.1A). In addition, a muscle main effect captured differences in the activation amplitudes between muscle sites showing IO sites had the highest activation, followed by EO sites and finally the RA sites (Table 4.4).

For both the abdominals and back extensors, PC2 captured the muscle activation response to the lateral flexion moment (Figure 4.2C&G). High scores corresponded with muscle sites having higher initial activation and lower terminal activation, whereas low (negative) scores corresponded to the opposite with lower initial activation (Figure 4.2D&H). A group by muscle interaction (p=0.004) found that the ASYM group had higher responsiveness of the right EO1 compared to the rLBI group (Table 4.4). This interaction also captured differences in the synergistic relationship among muscle sites where both groups had asymmetries in the EO1, EO3 and IO. Finally, the ASYM group had more differences among ipsilateral muscle sites than the rLBI which only had differences between IO and EO3 (Table 4.4).

Given PC2 captured both the directionality (positive or negative Table 4.4) and magnitude (coefficient) of the responsiveness to the lateral flexion moment, data were transformed to absolute values to explore whether the magnitude of responsiveness, regardless of direction, differed between strength groups (Quirk & Hubley-Kozey, 2018) but there were no differences (Table 4.3).

4.3.2.2 Back Extensor Muscle Activation Patterns

Two PC's explained 93.9% of the total waveform variance for the back-extensor muscle activation patterns. For PC1 there was a group by strength interaction (p=0.047) where within both the rLBI and ASYM groups, WEAK had higher activation amplitudes than STRONG (Table 4.3 & Figure 4.1B-D). This interaction also found that the WEAK rLBI subgroup had higher activation amplitudes than all other subgroups, and WEAK ASYM subgroup had higher activation than the STRONG rLBI subgroup (Table 4.3). Differences in muscle activation amplitudes were captured by a group by muscle

interaction (p=0.031) showing that there were differences in muscle synergies (Table 4.5). Specifically, the rLBI group had higher activation amplitudes of the L52 site between more ipsilateral sites (5/12 comparisons), than the ASYM group (3/12 comparisons) (Table 4.5).

Table 4.3: Strength main effects and group by strength interactions for abdominals and back extensor PC scores

Strength (n)	STRONG (30)		WEAK (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Abs. AVG	6.5	6.1	7.0	7.5
(% MVIC)	±5.5	±4.5	±5.3	±6.0
Abs. PC1	65.5‖	61.7¶	70.5	75.5
(unitless)	±55.7	±45.5	±53.4	±60.1
Abs |PC2|		5.5		
(unitless)		±9.3		
Back AVG	15.6	16.2	19.3	22.3
(% MVIC)	±7.6	±8.3	±8.0	±10.4
Back PC1	157.5¶‖	163.8¶‖	194.7‖	225.4
(unitless)	±77.1	±84.3	±81.8	±105.7
Back |PC2|	37.6†		47.6	
(unitless)	±27.4		±30.4	

If no main effect or interaction is identified the combined sample mean ± standard deviation is indicated in the center of the table or subgroup. Comparisons for PC2 are performed on the absolute value |PC2| scores. Significant differences ($p<0.05$) for strength main effects are indicated by †. Significant group by stiffness interactions are indicated by a specific symbol to show difference relative to the: WEAK rLBI (‖) and WEAK ASYM (¶) subgroup. Abbreviations: asymptomatic (ASYM), recovered low back injury (rLBI), average activation amplitudes (AVG), maximum voluntary isometric contraction (% MVIC), principal component (PC), abdominals (Abs), and back extensors (Back).

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Table 4.4: Muscle main effects and interactions for abdominal sites

	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO
AVG	3.7	3.7	3.8	3.3	6.9	7.8	6.8	6.9	7.5	8.2	11.1	11.6
(%MVIC)	±2.6	±2.4	±3.2	±2.7	±4.7	±6.3	±4.4	±5.5	±4.8	±4.9	±6.0	±6.4
PC1	36.8^{cdef}	37.1^{cdef}	38.5^{cdef}	33.5^{cdef}	69.6^{f}	78.0^{f}	67.9^{f}	69.8^{f}	75.7^{f}	82.2^{f}	111.4	117.1
	±25.9	±24.3	±31.7	±26.9	±47.5	±63.8	±44.1	±55.2	±48.6	±48.8	±60.1	±65.1
PC2	0.1^{c}	0.0^{c}	0.4^{c}	-1.0^{c}	**10.3***	**-13.4**	-2.1^{c}	4.9^{cf}	**-5.7^{cf}**	**14.7^{cf}**	**3.1**	**-4.0**
ASYM	±0.6	±0.7	±1.3	±1.4	**±15.5**	**±16.4**	±4.9	±12.1	**±11.8**	**±18.1**	**±7.7**	**±7.0**
PC2	-0.1	-0.5	-0.1	-0.7	**1.6***	**-6.5**	-2.8	0.7^{f}	**-6.4^{f}**	**10.9^{cf}**	**3.5**	**-6.7**
LBI	±1.1	±1.4	±2.0	±1.8	**±6.2**	**±17.0**	±4.7	±6.4	**±9.0**	**±11.4**	**±8.5**	**±11.2**

All values are mean ± standard deviation and unitless unless otherwise indicated. If only a muscle main effect exists the combined average all groups is shown, otherwise for interactions the respective group (LBI vs ASYM) is indicated by the row title. Significant ($p<0.05$) differences between muscle sites are indicated by **bold** lettering to show an asymmetry between left and right sites, and between muscle site differences amongst ipsilateral sites are indicated by superscript letters indicating a difference between the indicated muscle site and: a) LRA, b) URA, c) EO1, d) EO2, e) EO3, f) IO. Interaction effects also include the symbols * to indicate significant ($p<0.05$) differences between the LBI and ASYM group within a specific muscle site. Abbreviations: average activation amplitudes (AVG), maximum voluntary isometric contractions (% MVIC), principal component (PC), asymptomatic (ASYM), recovered low back injured (rLBI).

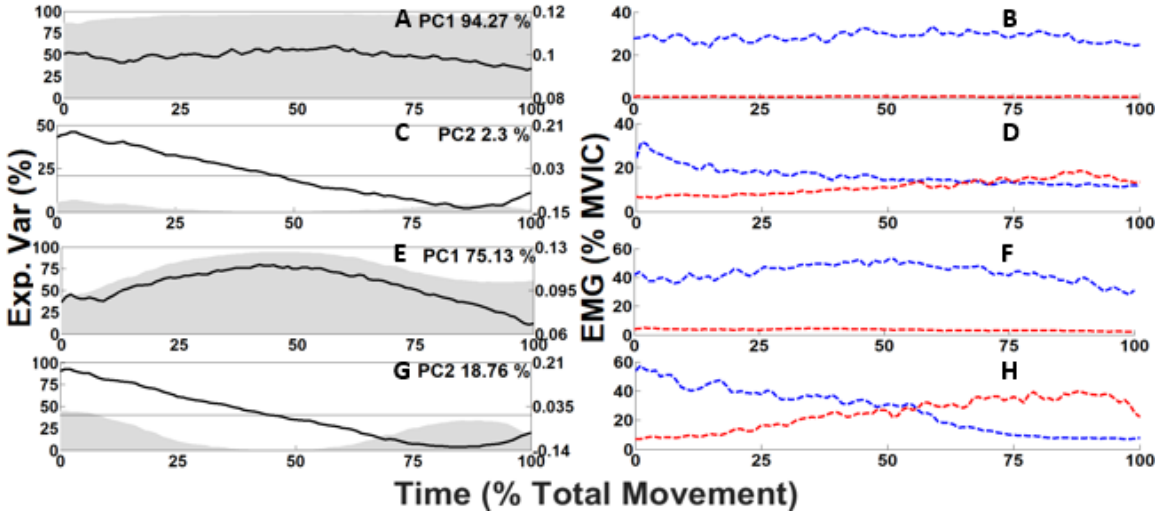

Figure 4.2: Visual representation of abdominal (A,C) and back extensor (E,G) principal component (PC) waveforms (black line) and ensemble average representations of high (blue) and low (red) electromyogram waveforms corresponding to each PC waveform (right) (B,D,F,H). For PC waveform plots grey shading depicts the time-varying explained variance with the collective total displayed on the top right of the sub-plot.

For PC2 there was a strength by muscle interaction ($p<0.001$) showing that WEAK was more responsive (higher PC2 scores) than STRONG for the LL16 site (Table 4.5). There were asymmetries between right and left sites for both strength groups. However, WEAK had more between muscle site differences than STRONG (Table 4.5). Transforming PC2 scores to absolute values found a strength main effect ($p<0.001$) capturing WEAK was more responsive (higher scores) to the changing lateral flexion moment than STRONG (Table 4.3 Figure 4.1C&D).

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Table 4.5: Muscle main effects and interactions for back extensor sites

	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52
AVG ASYM (%MVIC)	17.6 ±7.0	19.2 ±8.2	17.9 ±6.9	19.2 ±8.1	16.4 ±7.5	16.7 ±7.7	14.0 ±6.9	15.6 ±8.2	16.6 ±9.7	17.0 ±8.6	19.9 ±8.2	18.9 ±8.2
AVG rLBI (%MVIC)	17.3 ±8.5	21.7 ±10.1	16.4 ±7.1	20.9 ±9.3	18.2 ±8.9	19.8 ±10.2	15.6 ±10.3	18.3 ±9.9	18.4 ±10.3	18.6 ±11.1	22.0 ±8.9	24.4 ±10.0
PC1 ASYM	176.9 ±70.6	196.8 ±84.3	178.2 ±69.3	195.5 ±83.6	164.8[f] ±75.7	171.1 ±79.0	139.2[f] ±68.8	158.9 ±83.4	166.5[f] ±97.6	171.4 ±86.7	201.6 ±83.1	191.6 ±8.2
PC1 rLBI	172.7[f] ±85.0	222.2 ±102.8	**162.8[f]** **±70.5**	**214.0** **±94.7**	181.8 ±88.8	203.0 ±103.1	155.4[f] ±103.0	186.8[f] ±100.5	183.9 ±103.5	187.9[f] ±112.3	221.4 ±88.9	247.7 ±100.8
PC2 STRONG	**-48.4** **±23.2**	**45.6†** **±37.4**	**-48.8** **±27.7**	**52.5†** **±37.6**	**-45.1** **±22.4**	**31.9** **±25.7**	**-35.4** **±17.4**	**36.3** **±30.3**	**-29.1** **±18.7**	**20.6[ab]** **±20.6**	**-32.7** **±19.3**	**12.6[ab]** **±30.0**
PC2 WEAK	**-65.9** **±32.4**	**57.6** **±27.7**	**-64.4** **±30.6**	**74.9** **±29.4**	**-49.8** **±32.3**	**41.8[b]** **±23.6**	**-47.4** **±27.9**	**48.5[b]** **±28.7**	**-33.4[ab]** **±20.7**	**31.3[ab]** **±17.5**	**-36.9[ab]** **±26.1**	**12.6[ab]** **±22.2**

All values are mean ± standard deviation and unitless unless otherwise indicated. If only a muscle main effect exists the combined average of all groups is shown, otherwise for interactions the respective group (LBI vs ASYM, STRONG vs WEAK) is indicated by the row title. Significant ($p<0.05$) differences between muscle sites are indicated by **bold** lettering to show an asymmetry between left and right sites, and between muscle site differences amongst ipsilateral sites are indicated by superscript letters indicating a difference between the indicated muscle site and: a) L13 b) L16, c) L33, d) L36, e) L48, f) L52. Interaction effects also include the symbols † to indicate significant ($p<0.05$) differences between the STRONG and WEAK group within a specific muscle site. Abbreviations: average activation amplitudes (AVG), maximum voluntary isometric contractions (% MVIC), principal component (PC), asymptomatic (ASYM), recovered low back injured (rLBI).

4.4 DISCUSSION

This study explored the spinal system compensation theory by examining whether lower back extensor strength, indicative of a deficit in active spinal system function, resulted in adaptations to trunk muscle activation patterns during a dynamic task. Secondly, it examined whether the presence of a recent lower back injury modified that relationship. Fundamental was that all demographic variables and task performance variables were not different between the STRONG and WEAK groups with the only difference being muscle strength for both abdominals and back extensors.

Despite no differences in task performance, there were differences in the muscle activation amplitudes and patterns for abdominal and back extensor muscles between the STRONG and WEAK group. As hypothesized, relative to STRONG, the WEAK group had higher overall activation amplitudes of both their abdominal and back extensor sites and the back extensor sites were more responsive to the lateral flexion moment generated during task initiation and termination.

To understand whether the experience of recent LBP modified the relationship between strength and muscle activation, the group by strength interactions suggests that both groups responded to strength differences in a similar way but lower back extensor strength did have more influence on muscle activation pattern adaptations in the rLBI group. Thus, recent pain did modify these differences. Below these effects are compared to current literature.

4.4.1 Influence of strength on muscle activation patterns

Three of the four PCs examined had a significant strength main effects or group by strength interaction. Consistent with the EMG to force relationship (Brown & McGill, 2007a), WEAK recruited more motor units, reflected by higher EMG amplitudes of the primary agonist back extensor muscles (Butler et al., 2010). This finding is consistent with studies of lower limb muscles showing for a variety of tasks (calf raise, walking, sit-to-stand, stair ascent and descent) maximum muscle specific strength is negatively

correlated with agonist EMG amplitudes of the knee extensors (r=-0.3-0.7) and ankle plantar flexors (r=-0.4-0.5) (Takai et al., 2008).

The current study also found that participants with weaker back extensors recruited higher antagonist abdominal activation. If muscle recruitment is designed to minimize antagonist activation (Brown & Potvin, 2005) one might question why higher abdominal activation was observed. There are three plausible factors that explain this finding. First, the line of action of the abdominal oblique sites, specifically the lateral sites have been shown to contribute to generating lateral flexor moments (Brown & Potvin, 2007). These lateral obliques would contribute as an agonist to the lateral flexion moments generated at the beginning and end of the lifting task. Second the EO1 and IO sites (Arjmand, Shirazi-Adl, & Parnianpour, 2008a), would balance axial rotation moments produced by asymmetric activation of the back extensors (Table 4.5 PC2) and the more laterally orientated oblique muscles in the response to the changing lateral flexion moment (Arjmand, Shirazi-Adl, & Parnianpour, 2008b; Brown & Potvin, 2007). These two features provide a plausible explanation why the oblique sites had higher activation amplitudes than rectus abdominus sites which are designed to produce flexor moments (Table 4.4) (Arjmand, Shirazi-Adl, & Parnianpour, 2008b; Brown & Potvin, 2007). A third factor could relate to the stability demands of the lifting task. Computational modeling suggests the spine requires antagonist force production to maintain stability (Brown & Potvin, 2005). Whether there is a standard moment that must be produced by the antagonist to ensure joint stability is unknown. If a threshold did exist, weaker individuals would require higher activation amplitudes to meet this threshold. Our data showed that participants in the WEAK group also had significantly lower trunk flexor strength than STRONG (Table 4.1), supporting that the abdominal muscles compensated for this weakness to fulfill the above-mentioned roles. This finding is consistent with previous work showing individuals with lower knee extensor strength had higher knee flexor EMG activation amplitudes for fundamental tasks such as sit-to-stand and stair ascent/descent (Mizelle et al., 2003).

Novel to this study was the changes in the relative responsiveness of trunk muscles to the dynamic changing moments. For the back extensor sites, WEAK participants were more responsive to changes in the lateral flexion moment produced at

the beginning and end of the task (PC2) (Table 4.3) than STRONG. While not directly measured, sagittal plane trunk strength is related to frontal (r=0.81) and transverse plane (r=0.91) strength (Kocjan & Sarabon, 2014). Thus, weaker individuals should require higher activation of lateral flexor sites to meet the frontal plane moment demands produced by the horizontal transfer task (Table 4.2). These findings are consistent with previous studies reporting that as the external moment (load) for the horizontal transfer task increases, the trunk muscles become more responsive resulting in greater amplitude differences between right and left trunk muscle sites (Butler et al., 2010) and greater responsiveness (PC2 scores) of trunk muscles sites (Quirk & Hubley-Kozey, 2014).

4.4.2 Influence of recovery from LBP

The secondary purpose of this study was to determine whether the effects of strength on muscle activation patterns were modified by the recent experience of low back pain. The rLBI reported, while low, significantly higher levels of pain (Boonstra et al., 2014; Jensen et al., 2003), disability (Turner et al., 2004), along with psychosocial traits consistent with slightly more fear of movement and pain catastrophizing compared to the ASYM group (Table 4.1). Despite mass differences between the rLBI and ASYM group, mass normalized strength (Table 4.1) and the external moments produced by the horizontal transfer task (Table 4.2) were similar between groups. As the primary objective of this study was to compare between WEAK and STRONG participants mass deviation between the rLBI and ASYM group would not modify these differences. However, inclusion of mass as a co-variate in the ANOVA model did not change any group, strength or group by strength interactions captured on PC scores.

The mean strength of participants (Table 4.1) while within the range of supine trunk flexor strength (139.8±35.2 Nm) was above average for prone back extensor strength (179.2±31.2 Nm) when compared to a cohort of similar 20-60 year old males (Hasue et al., 1980). Higher back extensor strength may be explained by the military participants in the current study who have higher levels of fitness (including grip strength) than civilian controls (Deuster et al., 1987), which is associated with higher back extensor strength (Wang, Leger, & Dumas, 2005). A surprising result of this study was that un-normalized back extensor strength was highest in the rLBI group (Table 4.1),

an observation that is different from findings of lower trunk muscle strength in individuals with chronic LBP compared to healthy controls (Hasue et al., 1980; Newton et al., 1993; Steele, Bruce-Low, & Smith, 2014b). In part, these strength differences were explained by the rLBI group having higher mass than the ASYM group. Mass, in particular fat free mass, is partially related to trunk strength (Smith et al., 1985). Once strength was normalized to body mass there was no difference between the rLBI and ASYM groups, a result consistent with others showing that participants with a history of LBP of varying durations, do not have different trunk strength compared to controls (Hultman et al., 1993; McGill, Grenier, Bluhm, Preuss, Brown, & Russell, 2003b).

A key finding of this study was the observation of two interactions between strength and groups on muscle activation patterns. For both the back extensors and abdominals there was an interaction for overall activation amplitudes (PC1). For the abdominals, the WEAK group had relatively greater differences in overall abdominal activation than STRONG within the rLBI group compared to the ASYM group (Table 4.3). To explain this we compared the difference (ratio of WEAK:STRONG) in EMG activation amplitudes to the difference in the strength (ratio of STRONG:WEAK) within each group (rLBI and ASYM). The EMG amplitudes showed that the WEAK group required 117 and 122% higher activation amplitudes than STRONG in the ASYM and rLBI groups respectively. This difference in EMG amplitude was lower, but comparable to the finding that the rLBI had a larger difference in strength (145%) when compared to the ASYM group (120%) for normalized trunk flexor strength (Table 4.1) Thus differences in activation amplitudes are likely explained by the greater flexor strength difference between the respective subgroups.

For the back extensors, it was shown that the WEAK rLBI group had higher activation amplitudes than all other subgroups (Table 4.3). Similar to the abdominals, the rLBI group had a greater difference in EMG amplitudes between WEAK and STRONG when compared to the ASYM subgroups (128 and 137% higher in ASYM and rLBI, Table 4.3), but unlike the abdominals, this difference could not be explained by differences in normalized back extensor strength which was comparable between WEAK and STRONG ASYM and rLBI subgroups (145% and 148% higher respectively, (Table 4.1)).

Previous work suggests that WEAK rLBI are less recovered than STRONG participants based on symptoms (Newton et al., 1993) and that those less recovered report higher pain and disability and would be less likely to produce a true maximum voluntary isometric contraction (Chiou, Shih, Chou, McGregor, & Strutton, 2013) inflating the amplitude of MVIC normalized EMG. The results of the current study do not support this theory as VAS, RMD, PCS, and TSK were not different between the WEAK versus STRONG rLBI subgroups (Table 4.1). To explore whether pain could confound our results we found in the 4 participants with clinically significant VAS scores (ranging from 18-28 /100mm) two were in the STRONG and WEAK group respectively suggesting the current experience of pain was not a confounder.

A more plausible explanation for the higher activation is that the influence of recent pain modified how trunk muscle activation patterns adapt to a stability challenge associated with lower back extensor strength. Experimental work shows, that when an individual is in pain they utilize muscle activation patterns that generally increase the overall activation for all muscle sites to increase joint stiffness and thus stability (Hodges et al., 2013) and according to the motor adaptation to pain theory once pain resolves some individuals retain these adjusted muscle activation patterns (Hodges et al., 2013). This study expands on this model to suggest individuals with lower function (WEAK) of the active spinal system may experience greater adaptations to ensure spinal stability that is beyond a margin of safety whereas those with higher active system function (STRONG) have fewer adjustments. An hypothesis consistent with recent work showing that changes in trunk muscle activation is greatest when the spine has lower stiffness presenting a higher stability demand (Shojaei, Suri, van Dieën, & Bazrgari, 2018). Thus, this study could expand on the motor adaptation to pain theory to suggest those that those with lower function in a spinal system might experience more instability events (painful events) leading to greater adaptations to mitigate the risk of pain and likely have greater retention of these pain developed muscle activation patterns. However, future studies will be necessary to confirm whether pain induced changes are greater in those with lower measures of spinal system function, and the motor adaptation to pain theory still lacks empirical evidence to support motor patterns are retained post injury.

Despite novel findings in the current study there are limitations. First, the current study only reported on one measure of active spinal system function; the maximum voluntary isometric moment of the two main muscle groups with no measure of axial or frontal plane muscle strength. Other work has shown isometric trunk strength is correlated between all fundamental planes (Kocjan & Sarabon, 2014) and hence we chose the sagittal plane moment as a representative of abdominal and back strength given the maximum moment was in this plane. Second, while this study explored whether those with deficits in the active spinal system would have different muscle activation patterns our definition of a deficit was based on participants having below median back extensor strength. While our measure of lower function is a potential risk for experiencing future low back pain, the threshold used in this study does not relate to a clinical threshold of deficient function. A strength of this study is our relatively large sample size with equal numbers between those who have and have not experienced a recent low back injury within the last year.

4.5 CONCLUSION

Individuals classified as having weak back extensors had different muscle activation patterns to complete a controlled dynamic transfer task that those classified with strong back extensors. The results support the hypothesis that participants classified as having weaker back extensor strength adapted muscle activation patterns with higher overall activation amplitudes for both agonist and antagonist sites and had greater responsiveness to changing external moments produced in the frontal plane than stronger individuals. These findings not only show that more motor units were recruited to adapt to impaired maximal force production but the relative increase in muscle activation depends on changes in external moment produced by an external task. Secondly, the findings provide evidence that while both an ASYM and rLBI population adapted their muscle activation in a comparable way, having lower strength resulted in greater adaptations in the rLBI group hence recent LBP can alter the relationships between spinal system deficits and trunk muscle activation pattern adaptations.

CHAPTER 5
TRUNK MUSCLE ACTIVATION PATTERNS DIFFER BETWEEN THOSE WITH LOWER TRANSVERSE PLANE TRUNK STIFFNESS IN UNIQUE WAYS FOR ASYMPTOMATIC AND RECOVERED LOW BACK PAIN PARTICIPANTS

5.1 INTRODUCTION

Early 1990, to understand how the osteoligamentous spinal maintained stability, Panjabi presented a theoretical model to elucidate factors contributing to low back pain (LBP). Panjabi proposed that spinal stability was achieved by having sufficient potential energy to prevent the spine from buckling i.e. a state where it could not return to equilibrium (Crisco et al., 1992; Crisco & Panjabi, 1992). In a clinical definition, buckling represents excessive vertebral body motion resulting in tissue damage thought to be one cause of incapacitating pain (Panjabi, 1992a; 1992b; 2003). Panjabi theorized that stability was achieved through the interaction of all tissues that could modify or contribute to spinal stiffness, acting to resist vertebral body displacement. Spinal tissues were categorized into three functional systems; passive, active and neural (Panjabi, 1992a; 1992b; 2003). To link stability with the risk of LBP, Panjabi suggested that a deficit to any one spinal system can increase the risk of an instability event, but that interaction among spinal systems exist such that each system can modify their function to compensate for a deficit (Panjabi, 2005). While theoretically sound, there is a gap in empirical evidence supporting that deficits in an individual spinal system triggers change in the function of other systems.

This paper focuses on the ability of passive spinal tissues to generate forces and moments to control vertebral body motion. While some authors consider the passive system as osteoligamentous structures (Ibarz et al., 2012; Panjabi, 1992a), others include all structures that exhibit passive elastic mechanical properties including tendons, skin and intramuscular connective tissue (Arjmand & Shirazi-Adl, 2006). Regardless the tissue passive-elements have a physiological function guided by a fundamental mechanical property where the tissue exhibits an elastic behaviour where upon the application of an external force these tissues undergo a deformation (change in length or

strain) resulting in the passive generation of a counter force (stress) dictated by their stiffness (Pintar, Yoganandan, Myers, Elhagediab, & Sances, 1992).

The contribution of passive stiffness becomes more relevant, producing a greater force, as the spine moves into larger ranges of motion. In fact, the contribution of passive stiffness from osteoligamentous and musculotendinous structures can produce a counter moment of nearly sufficient magnitude to support the mass of the trunk in 65 degrees of flexion (Arjmand & Shirazi-Adl, 2006). Confirmed in vivo by minimal back extensor muscle activation amplitudes as measured via electromyography (EMG) when a participant enters full trunk flexion, suggesting forces generated by active-contractile components have a trade off with passive components (Zwambag, De Carvalho, & Brown, 2016). When the spine is in a neutral position, the contribution of passive stiffness has minimal effect on total joint stiffness (Arjmand & Shirazi-Adl, 2006; Cholewicki & McGill, 1996), leading to question whether the structures and function of the passive system have a role in contributing to spinal stability and the prevention of LBP.

Considering only osteoligamentous structures, there is evidence that individuals have changes within the intervertebral disc following low back injuries (LBI). Cross sectional studies have shown, when controlling for factors such as age, those with a history of LBP have greater disc degeneration compared to those with no history (Cheung et al., 2012a; Hassett, Hart, Manek, Doyle, & Spector, 2003; Pye et al., 2004; Wang et al., 2012; de Schepper et al., 2010). Consistent with Panjabi's first theory, a prospective study by Hancook et al, found that radiographic evidence of moderate disc degeneration was associated with a greater risk of experiencing recurrent episodes of LBP in a one-year follow-up (Hancock et al., 2015).

As the vertebral disc degenerates, the ligaments, joint capsule, and fibres of the annulus fibrosis which restrain vertebral body motion (Panjabi, Goel, & Takata, 1982; Panjabi, Hausfeld, & White, 1981) experience a relative increase in toe-length as the vertebral bodies move towards one another (Zander, Krishnakanth, Bergmann, & Rohlmann, 2009). This has an impact on passive stiffness and measurements of vertebral body neutral zone, defined as the range of motion which a functional spinal unit can be moved before it produces a resistive force (Galbusera et al., 2014). In vitro testing of

lumbar spinal segments find that increasing severities of disc degeneration have a significant (Galbusera et al., 2014; Tanaka et al., 2003; Volkheimer et al., 2017) or a trend (Kettler, Rohlmann, Ring, Mack, & Wilke, 2010; Sengupta & Fan, 2014) towards increased axial rotation laxity or neutral zone motion. Whereas, in other planes, disc degeneration has an inconsistent impact on neutral zone motion (Galbusera et al., 2014; Kettler et al., 2010; Sengupta & Fan, 2014; Tanaka et al., 2003; Volkheimer et al., 2017). This increased axial-rotation motion with greater disc degeneration corresponds with decreased angular stiffness, defined as the resistive force produced by a tissue following a set displacement, of functional spinal units (Zirbel et al., 2013).

Much of what is known about the function of the passive system comes from in vitro studies, however, in vivo methods do exist to measure spinal stiffness. The two most common methodologies include rapid loading and unloading (Granata & Rogers, 2006) and quasi-static whole range of motion paradigms (McGill et al., 1994). While both methods are sensitive to measuring changes in passive stiffness as the trunk moves away from a neutral position (Brown & McGill, 2007b; Drake & Callaghan, 2008; Granata & Rogers, 2006; McGill et al., 1994; Scannell & McGill, 2003) the quasi-static range of motion paradigm does not require the participant to generate an active force pre-load controlling for the confounding influence of background muscle activation generating additional active trunk stiffness (Bazrgari, Nussbaum, & Madigan, 2011b; Lee, Granata, & Moorhouse, 2007b; Vera-Garcia, Brown, Gray, & McGill, 2006b). Furthermore the quasi-static whole range of motion paradigm is designed to be performed at slow angular velocities (2-11°/s) (Beach, Parkinson, Stothart, & Callaghan, 2005; Brown & McGill, 2007b; Parkinson, Beach, & Callaghan, 2004; Scannell & McGill, 2003) to reduce the likelihood of evoking a muscle stretch reflex which can contribute to measured stiffness (Moorhouse & Granata, 2006). Despite the ability to minimize confounders, in vivo measures of spinal stiffness still capture a measure of global intrinsic stiffness of the spine generated by passive osteoligamentous, musculotendinous and other sources of stiffness (skin, intra-abdominal pressure etc.) (McGill et al., 1994). Regarding the relevance of in vivo measurements of passive stiffness work around the knee shows that individuals with greater knee laxity, are at an increased risk of experiencing future knee injuries (Myer, Ford, Paterno, Nick, & Hewett, 2008; Uhorchak et al., 2003).

Structural and functional deficits, characterized as higher disc degeneration or lower joint stiffness, of the passive system can capture a risk of LBI and yet it is unknown whether, according to Panjabi's theory, other systems adjust their function to adapt to these deficits. Around the spine cross-sectional studies show that older age (65+ years old) and those with a history of LBP have known changes within the passive system, including disc degeneration (Fujiwara et al., 2001; Galbusera et al., 2014; Sengupta & Fan, 2014), relative to young asymptomatic controls. Independent cross-sectional studies show that these populations have different muscle activation patterns including higher agonist and antagonist activation amplitudes (Hodges & Moseley, 2003; Hubley-Kozey et al., 2013; McGill et al., 1999; Mehta et al., 2010; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014; van Dieën et al., 2003) and sustained muscle activation in response to changing external moments (Butler et al., 2012; D'hooge, Hodges, et al., 2012b; Hubley-Kozey & Vezina, 2002; Hubley-Kozey et al., 2013; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014) when compared to young asymptomatic controls. A direct comparison of older adults and those recovered from a LBI provided evidence that these activation patterns may represent an adaptation for deficit passive stiffness within these populations (Chapter 3 (Quirk & Hubley-Kozey, 2018)). These findings could be confounded as both populations may have deficits in multiple spinal systems (Quirk & Hubley-Kozey, 2018), and pain alone can modify muscle activation patterns independent of tissue deficits (Dubois et al., 2011; Hodges et al., 2003; 2013; Hug et al., 2014; van den Hoorn et al., 2014) where these adaptations may remain post injury (Hodges & Tucker, 2010).

Direct evidence of compensation comes from Shultz et al, who completed a series of studies to show women with greater sagittal and frontal / transverse plane laxity of the knee joint have differences in knee muscle activation patterns including higher antagonist co-activation and agonist amplitudes in preparation to: a standing perturbation or landing from a drop jump (Shultz et al., 2004; Shultz & Schmitz, 2009). While the quantity of these studies is limited, the observed differences are consistent with Panjabi's theory that the neuromuscular system can change muscle activation patterns to compensate for lower passive system stiffness by using activation patterns that increases joint stiffness.

The aim of this study was to probe the spinal system compensation theory by assessing the interaction between passive spinal system function and neuromuscular control of the trunk muscles during a dynamic task. This experiment tested for difference in abdominal and back extensor muscle activation amplitudes and patterns during a controlled dynamic lifting task between those who had higher (STIFF) versus lower (LAX) transverse plane stiffness to assess passive spinal system function. The hypothesis was that LAX individuals would utilize muscle activation patterns that increase the active stiffness of the trunk including higher EMG activation amplitudes of agonist and antagonist muscles, and temporal changes consistent with less responsiveness to changing external moments compared to STIFF individuals. A secondary purpose was to explore whether the recent experience of a low back injury modifies the trunk muscle activation differences identified between those with high and low stiffness.

5.2 METHODS

5.2.1 Participants

Participants for this study were recruited from the Canadian Armed Forces. Asymptomatic participants volunteered by responding to on base posters and base-wide recruitment e-mails. Recovered LBI (rLBI) participants were identified if they reported a back related issue resulting in altered activities of daily living for at least 3 days (Ozguler et al., 2000) to the Canadian Forces Health Services Center and were contacted to see if they were interested in participating in the study. For both groups a self-report questionnaire screened for the following exclusion criteria: previous abdominal or back surgery, cardiovascular, respiratory or neurological conditions that would place them at risk for participating in the study. For the participants in the rLBI group questions determined that their recent LBP occurred 4-12 weeks prior to their data collection, was not chronic lasting longer than 12 weeks, and not recurrent where a previous injury occurred within 12 weeks prior to their most recent episode (Delitto et al., 2012). Asymptomatic participants were screened to determine that they had not experienced an activity limiting LBP (Ozguler et al., 2000) in the last year. All rLBI were deemed recovered, reporting minimal pain (Visual Analog Scale (VAS) <30/100mm (Boonstra et al., 2014; Jensen et al., 2003; Wewers & Lowe, 1990)), minimal disability (Roland

Morris Disability (RMD) <9/24 (Roland & Morris, 1983; Stratford et al., 1998)), and the resumption of normal activities of daily living at the time of testing. Before testing all participants signed an informed consent approved by the Institutes Research Ethics Board.

5.2.2 Protocol

Participants attended two sessions. During session one they were screened by a registered physiotherapist to determine whether they met the minimal pain and disability criteria and confirmed the absence of a neurological condition. During session two participant's pain, demographic and anthropometric data were measured. Self-reports of weekly engagement in physical activity (aerobic sessions >30 minutes, core/ abdominal and strength training), if they frequently lifted objects weighing over 23kg for their work, defined as a heavy job (Seidler et al., 2009) and preferred handedness was recorded. Participants also filled out a Tampa Kinesiophobia (Kori et al., 1990) and Pain Catastrophizing Scale (Sullivan et al., 1995) to characterize their beliefs towards pain.

Participants were prepared for data collection including surface EMG (sections 5.2.3.1) and setup for the quasi-static axial rotation stiffness test. For this test, participants were secured to two rigid bodies. Height of the lower rigid body platform was adjusted so the participant's posterior superior iliac spine was level with a cross bar covered in 1" of dense foam. This design ensured pulley rotation occurred 8cm anterior to the posterior superior iliac spine (cross bar centroid) approximating the L4/L5 transverse plane center of rotation (Dumas et al., 2006; Reed et al., 1999). The lower platform was moved anteriorly or posteriorly relative to the upper rigid body until the participant felt comfortable. Nylon straps secured the participant to the lower and upper rigid bodies to encourage motion occurred primarily between the rotating pelvis (lower rigid body) and rib cage (fixed upper rigid body) (Drake & Callaghan, 2008; McGill et al., 1994) (Figure. 5.1).

Once secured, participants were requested to actively rotate in a clockwise (CW) and counter-clockwise (CCW) position "as far as they felt comfortable", where a hard stop was set. The participant then actively rotated to their most natural "neutral spine" position (Figure 5.1B). A single trial recorded their neutral position as they were

instructed to “stand as relaxed as possible”. EMG recorded from this trial (Section 5.2.3.1) was used in a biofeedback program designed to notify the researcher (via auditory signal) if the participant’s EMG exceeded a resting threshold (mean + 3 standard deviations) during passive rotation trials signaling a need to recollect the trial.

Ten passive rotation trials were performed (five CW and five CCW), alternating the direction between trials. For each trial, participants were encouraged to remain “as relaxed as possible” as they were passively rotated between the two positions (Figure 5.1 A→C). Lower rigid body rotation was produced using a steel cable attached to a pulley system located underneath the lower rigid body. Using an auditory cue emitted from a Labview program a researcher pulled the steel cable approximately 1.2cm every second (tone interval) to simulate quasi-static rotation.

Participants were then prepared for motion capture (see 5.2.3.3) and performed three trials of a right-to-left horizontal transfer task (Butler et al., 2010). Briefly, to a standardized five second count participants were instructed to on “1” lift a 3kg mass orientated 60˚ to the right of the midline of their body with their right hand, on “3” transferring the mass between hands at the midline of their body, and on “5” lower the mass 60˚ to the left of their midline. Mass lift and lower were recorded using a pressure sensor located at the bottom of the mass, and hand transition was measured using an optoelectric switch. A height adjustable table was used to ensure the mass was positioned at elbow height, and the optoeletric switch ensured the mass was lifted no higher than 5cm. During the task, a researcher ensured the participant lifted the mass with the arm in full extension. Participants were asked to minimize trunk motion aided by tactile feedback provided to the upper thoracic spinous process.

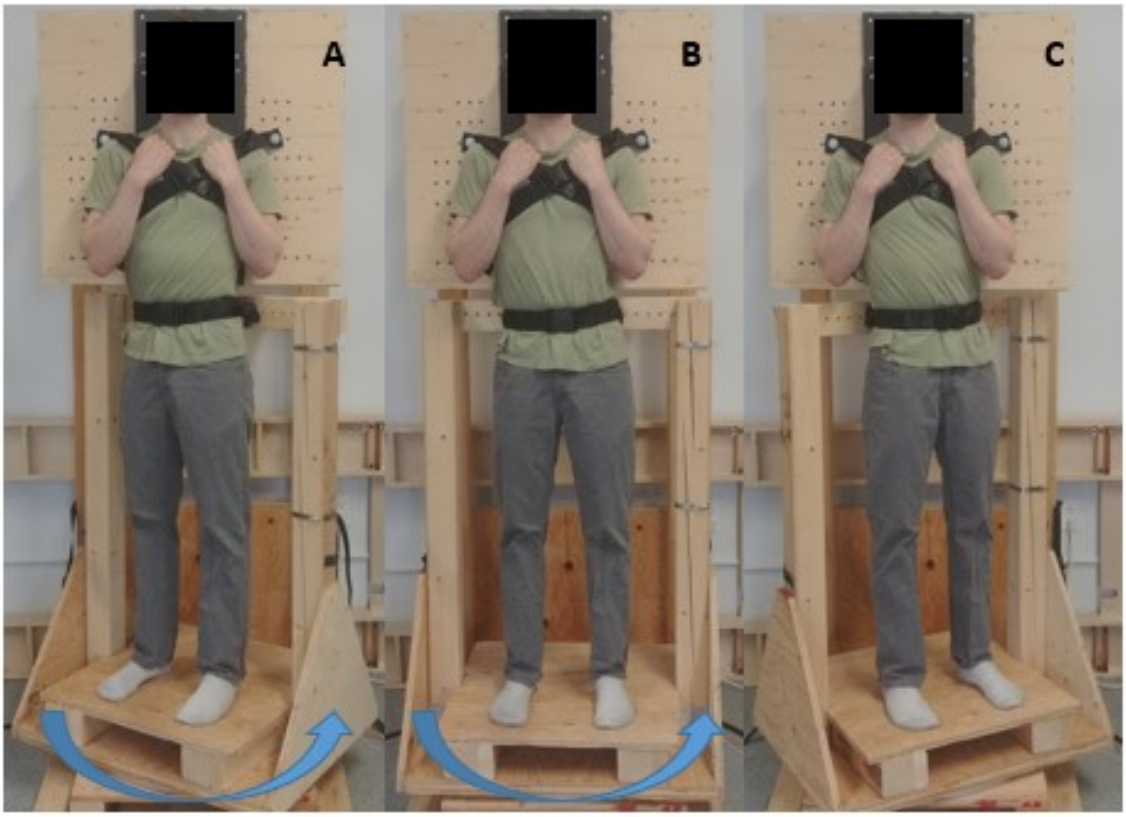

Figure 5.1: Visual representation of the stiffness testing apparatus showing the progression of lower rigid body (secured to pelvis) as it is rotated counter clock wise (A→C) with respect to the upper rigid (attached to torso).

Participants then performed a series of maximum voluntary isometric contractions (MVIC) for EMG amplitude normalization including: i) supine sit-up, ii) side lying lateral flexion (left and right), iii) prone back extension, and iv) prone back extension coupled with rotation (right and left) (Butler et al., 2010). Each exercise was repeated (two trials). For each trial participants were encouraged to maintain maximal effort for 3 seconds against non-elastic straps, with a minute rest between contractions.

Trunk strength (flexor and extensor moment production) was measured during two MVIC exercises. Participants were positioned in a prone or supine crook lying position with a HUMAC Norm Dynamometer (Computer Science Medicine Inc, Strongton, MA, USA) arm positioned anterior and inferior to the clavicle for trunk flexion, or superior and posterior to the spine of the scapula for trunk extension (Hasue et al., 1980). The HUMAC centroid was positioned approximately 5cm anterior to the posterior superior iliac spine, in line with the iliac crest, and non-elastic straps were secured to anchor the pelvis and shank to the HUMAC bed. Following gravity correction,

participants performed two trials with instructions and procedures consistent with the normalization tasks.

5.2.3 Data Collection and Analysis

5.2.3.1 Surface Electromyography

Surface electrodes (Ag/AgCl, 10mm diameter, Red dot, 3M, Maplewood, MN) were placed in a bipolar configuration (30mm interelectrode distance) over 12 bilateral muscle sites using a standard protocol (Butler et al., 2010). Abdominal sites included the upper and lower rectus abdominus (URA & LRA), the internal (IO), and external (EO1-3) oblique sites representing the anterior, lateral and posterior fibres respectively. Back extensor sites included the superficial quadratus lumborum (L48) and multifidus (L52) along with the erector spinae at level of the 1st and 3rd lumbar spinous approximately 3 and 6 cm horizontal to the midline to capture the longissimus (L13 & L33) and iliocostalis (L33 & L36) fibres respectively (Butler et al., 2010).

EMG signals were pre-amplified (500x) and further amplified using three AMT-8 amplifiers (band-pass 10-1000Hz, CMRR=115 dB, input impedance 10GΩ; Bortec Inc., Calgary, AB). EMG signals, and event markers were digitized at 2000Hz using a 16-bit (±5V) analog-to-digital board (PCI-6033E, National Instruments, Austin, TX) and Labview^{TM} (Version 2017, National Instruments). Custom Matlab (Math Works, Natick, MA) code corrected EMG signals for participant bias, electrocardiogram artifact (high pass zero-lag 30Hz filtered) (Butler et al., 2007) and noise from electromagnetic sources (inverse fast-Fourier filtered removing frequency spikes with a power 3 times greater than their nearest frequency (+/-15Hz) neighbours). Corrected filtered data were rectified and low pass filtered at 6Hz using a second order zero-lag Butterworth filter, to produce a linear envelope. Data were time-normalized to specific events. For the stiffness test average EMG activation amplitudes were measured from 0-3 degrees of rotation for all muscles from each trial in both CW and CCW rotation. For the horizontal transfer task, EMG signals were time normalized from 0-100% of the total task time (from the event markers) using a quadratic interpolation algorithm. For both tasks, EMG signals were amplitude normalized, to the maximum average 500ms moving window linear envelope

regardless of what task evoked the MVIC (Vera-Garcia et al., 2009; Vezina & Hubley-Kozey, 2000).

For the horizontal transfer task, ensemble average waveforms were calculated from the three trials. To characterize overall neural drive the average (% MVIC) activation was calculated for the waveform for the entire task. To characterize spatial temporal characteristics of the multiple muscles sites collected, ensemble average waveforms for each participant (N) and muscle site (12) created two data matrices ((N*12) X 101) for the abdominals and back extensors separately. Each data matrix was entered into a principal component (PC) analysis model (Hubley-Kozey & Vezina, 2002). Briefly, each data matrix was transformed into a covariance matrix and underwent an eigenvector decomposition to identify eigenvectors (PCs) which explained patterns of maximum variation within the waveforms. For each EMG waveform, a PC score was calculated to fit the PC to the original waveform. The number of PCs examined was determined such that the total explained variance for the combinations of PCs reached 90%, and that each PC explained at least 1% variance (Hubley-Kozey & Vezina, 2002).

5.2.3.2 Stiffness Testing

During the stiffness test, an angular potentiometer (3547S, Bourns Technology, Riverside CA) was instrumented to measure the angular displacement of the lower rigid body pully. To measure cable tension applied to the pulley, a load cell (FTD-IU-200, Schaevitz Sensors, Hampton VA) was attached to the steel cable used to produce motion. For each trial, load cell and potentiometer data were digitized at 2000Hz using the same 16-bit (±5V) analog-to-digital board that sampled EMG data (section 5.2.3.1). Custom Matlab code filtered potentiometer and load cell data using a 2Hz second order zero-lag low pass Butterworth filter. Potentiometer and load cell voltage data were converted to angle (Θ) and force (newtons (N)) using calibration equations (Equation 5.1 and 5.2 respectively). Force data were converted to moments by calculating the product of the time-varying force (N) and pully radius (0.1m) to express data in newton-meters (Nm). Angle data were expressed relative to the standing neutral position by subtracting the bias position.

$$Angle(\theta(t)) = 1.00\theta + (Voltage(v(t)) * -156.05(\theta/v))$$

Equation 5.1: Measured angle (θ(t)) via time varying change in potentiometer voltage (v(t))

$$Force(N(t)) = [51.76lbs + (Voltage(v(t)) * -38.04(lbs/v))] * 4.45N/lbs$$

Equation 5.2: Measured force (N(t)) via time varying change in load cell voltage (v(t))

For each trial, the average moment measured at -0.5 to 0.5˚ relative to the participant's neutral position (0˚) was subtracted to remove bias (Drake & Callaghan, 2008; McGill et al., 1994). The time-varying angle was measured from 0-±15˚ in the direction desired for the trial, moment data was determined for the same time points. For each participant, the moment-angle profile for all trials developed a data matrix. Using the Polyfit function in Matlab a 4th degree polynomial modeled the external moment ($\overline{Nm}$) for each angle from -15 to 15˚ (Equation 5.3) (Figure 5.2) (Scannell & McGill, 2003).

$$\overline{Nm} = c + a\theta + a_2\theta^2 + a_3\theta^3 + a_4\theta^4$$

Equation 5.3: Forth degree polynomial equation used to calculate external moment (Nm) from known angle (θ) using predicted constants (c) and coefficients (a_n)

Stiffness (Nm/˚) was calculated from the modeled moment-angle curve as the quotient between the derivative of moment ($\dot{Nm}$) and angle ($\dot{\theta}$) (Equation 5.4). Using this stiffness profile two outcome measures were calculated. The primary outcome measure was average neutral zone stiffness. Calculated from 0-3˚ (within the 75th percentile of the measured neutral zone width ($\Delta\Theta$, to be discussed)) in both the CW or CCW direction relative to (0˚) the minimum of the absolute value stiffness. Because the stiffness of a functional spinal unit is known to increase with compressive forces (Noguchi et al., 2015; Zirbel et al., 2013), average stiffness was normalized to the participant's mass as Nm/kg˚. The second, outcome measure was in vivo neutral zone calculated in two steps. First, the angle at which the stiffness in the CW (Θ^1) or CCW (Θ^2) direction exceeded the threshold of 0.1Nm/˚ was calculated (Scannell & McGill, 2003), then the neutral zone width ($\Delta\Theta$) was calculated as the difference between the CCW and CW angle (Figure 5.2).

$$Stiffness\left({}^{Nm}/_{\circ}\right) = {}^{\dot{Nm}}/_{\dot{\theta}}$$

Equation 5.4: Calculated stiffness ($^{Nm}/_{\circ}$) measured as the quotient of the derivative of the moment ($\dot{Nm}$) and angle ($\dot{\theta}$)

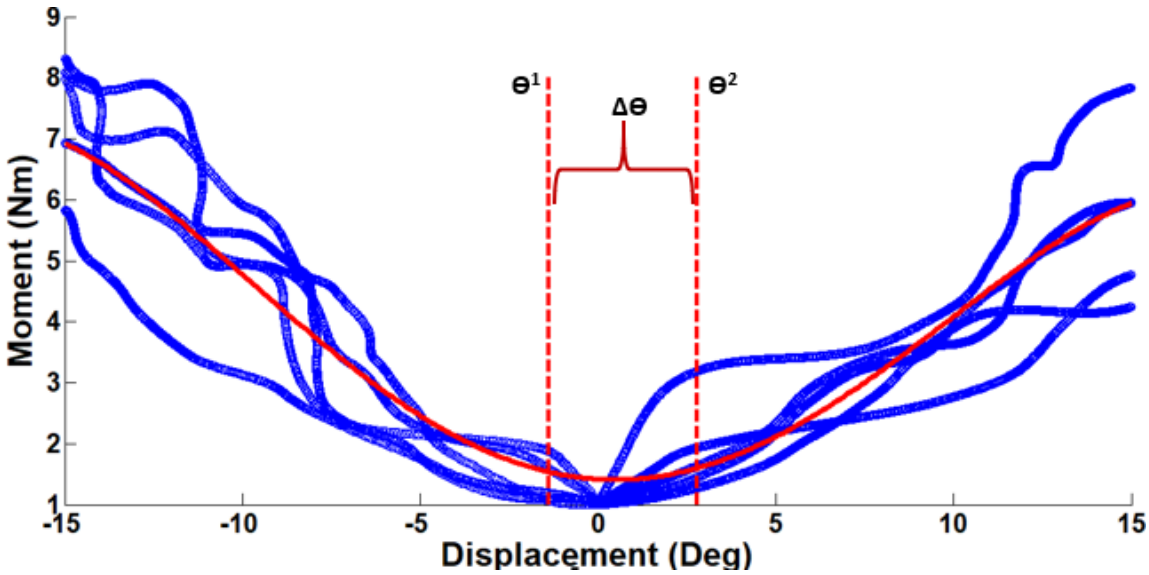

Figure 5.2: Representative moment-displacement curve for one participant from five trials in both the clockwise (CW) and counter CW direction (blue). These data were modeled to a fourth-degree polynomial (red). Stiffness was calculated as the derivative of the modeled moment-displacement curve. In both the CW and CCW direction the angle at which absolute value stiffness exceeded 0.1Nm was calculated as Θ^1 and Θ^2. Neutral zone range ($\Delta\Theta$) was calculated as the difference between Θ^2 and Θ^1.

5.2.3.3 Kinematic and Kinetic Analysis

Passive reflective markers were positioned on the suprasternal notch and 7th cervical spinous process along with 16 bilateral landmarks including the: 5th and 2nd metacarpal, radial styloid, ulnar styloid, medial and lateral humeral epicondyle, the mid acromion clavicular joint, and the anterior superior iliac spine. Four marker rigid body clusters were affixed to the thorax and pelvis, both forearms and upperarms. Following marker setup, a single standing calibration trial captured the marker position relative to the rigid bodies using six infrared emitting cameras (ProReflex 240, Qualisys^{TM}, Goteborg, Sweden) sampled at 100Hz using Qualisys Track Manager Software (Version 2.10, Qualisys^{TM}, Goteborg, Sweden). For synchronization, the Labview program collecting analog-to-digital data triggered the motion capture system.

Marker data were processed in Qualisys Track Manager, to label marker's coordinates. Coordinate data were entered into a custom Matlab^{TM} script for quadratic interpolation of missing data points, and low pass filtered at 4Hz using a fourth order zero-lag Butterworth filter (Wicks, 2017). Kinematic data were processed in accordance with the International Society of Biomechanics recommendations (Wu et al., 2002; 2005). Within each joint, an anatomical coordinate system was defined using bony landmarks, and joint centers calculated using regression equations (Dumas et al., 2006). Segment angles were calculated as Euler angles in a Z-Y-X (lateral flexion, axial rotation, flexion-extension) rotation sequence. For the horizontal transfer task segment angles were filtered using a 1Hz second order, zero-lag low pass Butterworth filter (Butler et al., 2010; Wicks, 2017). For each trial, the total displacement (maximum-minimum) of the torso and pelvis was calculated in lateral flexion, flexion extension, and axial rotation and averaged over the three trials for each participant.

Kinematic data were used to calculate the moments of force around the trunk using a top-down static inverse dynamics approach. Known external mass, along with joint mass and position (center of mass), estimated using anthropometrically derived regression equations (Dumas et al., 2006), were inputs to an open kinetic model that, by using a series of Newton-Euler equations, calculated forces and moments occurring at proximal segment, which was then propagated to the distal end of any linked segment (Winter, 2009). This sequence was continued to predict the forces and moments applied to the trunk (L4/L5). This model assumed the mass of the lifted object applied a weight vector at the middle of the 2nd and 5th metacarpal of the right hand for the beginning of the lift, both hands during hand transition and the left hand following hand transition. Following this calculation, the peak lateral flexion and flexion trunk moment was calculated for each trial and then averaged across trials for each participant.

5.2.4 Strength Analysis

For strength testing, gravity corrected external moments sampled from the HUMAC, were digitized at 2000Hz using a 16-bit analog to digital converter (Section 5.2.3.1). Custom Matlab code filtered these data using a 6Hz second order zero-lag low pass Butterworth filter, and converted to a moment (Nm) according to equations provided

by HUMAC. The maximum isometric moment for flexors and extensors were calculated as the highest average moment over 500ms window and normalized to body mass to compensate for anthropometric differences between participants (Smith et al., 1985).

5.2.5 Statistical Analysis

To test whether there were differences in muscle activation amplitudes and patterns between those with higher (STIFF) or lower stiffness (LAX), a median split approach was used on averaged mass normalized CCW and CW stiffness values, within both the rLBI and asymptomatic group. Two-way analysis of variance models (ANOVA) (group and stiffness) tested for differences in demographics, anthropometrics, strength (trunk flexor and extensor moments), and horizontal transfer task performance including trunk motion, peak moments and timing data. From the stiffness task average stiffness in both the CW and CCW direction motion (condition), neutral zone motion, rotation velocity were compared using a three-factor ANOVA (group, stiffness, and condition). Categorical data were compared using a Chi-square test for independence.

EMG data were analyzed in different ways according to the task. Normality of EMG data (amplitude or PC scores) was assessed using a Kolmogorov-Smirnov test, with non-normal data transformed using a Johnson transformation. For the stiffness test average EMG amplitudes (%MVIC) were compared using a four-factor (group, stiffness, condition (CW or CWW) and muscle) mixed model ANOVA. For the horizontal transfer task PC scores were analyzed using three-factor (group, stiffness and muscle) mixed model ANOVAs for the abdominals and back extensors separately. Tukey simultaneous tests compared pairwise differences when significant. Statistical analyses were performed in Minitab (version 17, State Collage, PA). Alpha was set at 0.05 and Bonferroni corrected.

5.3 RESULTS

5.3.1 Anthropometrics, Demographics, Strength and Task Performance

Sixty-nine participants volunteered for the study and of those nine were women. Given sex modifies muscle activation patterns (Hubley-Kozey et al., 2012) and only two

women were in the rLBI group, the analyses were performed on the 60 men (30 rLBI). For normalized stiffness participants were separated into the STIFF and LAX categories, the median average (CW and CWW) mass normalized stiffness threshold of 0.0013 and 0.0011 Nm/˚kg for the ASYM and rLBI respectively. General characteristics of the separate participant groups are shown in Table 5.1. No stiffness main effects or interactions were identified. Significant group main effects show the rLBI group had higher mass ($p=0.001$), and despite being recovered from LBI (9±2 weeks from the day of testing) pain (VAS ($p=0.001$)), disability (RMD ($p=0.003$)), pain catastrophizing (PCS ($p=0.004$)) and kinesiophobia (TSK ($p<0.001$)) scores were higher than the ASYM. Non-normalized extensor strength was greater in the rLBI group ($p<0.001$) but this difference was not significant when strength was normalized to body mass (Table 5.1).

5.3.2 Stiffness Testing Performance Outcomes

Results of the stiffness test are shown in Table 5.2. As stiffness was not different between conditions (CW and CCW rotation) ($p>0.05$) only the combined stiffness was shown. Stiffness main effects captured the LAX group was rotated at a higher velocity ($p<0.001$), had greater neutral zone displacement ($p<0.001$) and had lower unnormalized ($p<0.001$) and normalized ($p<0.001$) stiffness than STIFF. EMG activation amplitudes were not different (main or interaction) between conditions and were averaged for the CW and CCW direction. A group by stiffness interaction for the abdominals sites ($p<0.001$) found the LAX ASYM subgroup had lower activation amplitudes than both rLBI subgroups, and the LAX rLBI subgroup had higher activation amplitudes than the STIFF rLBI subgroup (Table 5.2). For the back extensors, a stiffness by group interaction ($p<0.001$) captured the LAX rLBI subgroup had higher activation amplitudes than all other subgroups (Table 5.2).

Table 5.1: Demographics, anthropometrics, pain characteristics, self reported physical activity, occupational loading and trunk strength

Stiffness (n)	STIFF (30)		LAX (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Age (years)	33.9 (6.4)	39.5 (10.5)	35.9 (11.7)	38.2 (9.6)
Mass (kg)	80.3 (10.8)*	87.3 (13.6)	80.1 (13.0)*	91.0 (15.6)
Height (m)	173.4 (6.2)	180.6 (7.3)	177.1 (9.4)	179.2 (6.7)
BMI (kg/m^2)	26.7 (3.1)	26.8 (3.8)	25.6 (3.7)	28.3 (4.5)
Heavy Job (n (%))	6 (40%)	3 (20%)	2 (13%)	7 (47%)
L. Hand (n (%))	3 (20%)	2 (13%)	3 (20 %)	1 (7%)
VAS (mm)	0.8 (1.8)*	4.9 (8.5)	0.3 (1.3)*	7.3 (8.3)
PCS	10.6 (8.1)*	14.0 (8.7)	3.4 (4.2)*	13.8 (11.6)
TSK	31.4 (7.5)*	35.3 (6.1)	28.4 (6.2)*	37.5 (6.1)
Aerobic Training (/ week)	4.1 (1.6)	2.6 (2.1)	4.7 (3.2)	4.0 (3.7)
Strength Training (/ week)	2.6 (1.9)	2.3 (2.3)	2.2 (2.1)	2.7 (3.0)
Core Training (/ week)	2.0 (1.6)	2.7 (2.0)	2.3 (2.2)	1.6 (2.2)
RMD	0.2 (0.8)*	2.1 (2.4)	0.1 (0.3)*	2.5 (2.7)
Flexor Mo (Nm)	129.6 (30.6)	137.0 (39.8)	123.2 (29.7)	145.9 (36.5)
Extensor Mo (Nm)	193.6 (48.2)*	219.1 (57.7)	198.0 (41.8)*	238.4 (45.4)
Norm Flexor Mo (Nm/kg)	1.6 (0.4)	1.6 (0.4)	1.6 (0.4)	1.6 (0.4)
Norm Ext. Mo (Nm/kg)	2.4 (0.5)	2.5 (0.7)	2.5 (0.5)	2.6 (0.6)

Significant differences ($p<0.05$) represented by * to indicate difference between the ASYM and rLBI group. Abbreviations: asymptomatic (ASYM), recovered low back injury (rLBI), body mass index (BMI), left handed (L. Hand), visual analog scale (VAS), pain catastrophizing scale (PCS), tampa scale of kinesiophobia (TSK), Roland Morris Disability score (RMD), moment (Mo), normalized (Norm) and extensor (Ext.).

Table 5.2: Stiffness test outcomes including resting electromyography activation amplitudes

Stiffness (n)	STIFF (30)		LAX (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Rotation Velocity	6.4 (0.8)	6.4 (1.2)	7.0 (1.4)†	7.9 (1.5)†
NZ (°)	2.8 (0.5)	2.8 (0.7)	5.2 (2.0)†	4.9 (1.7)†
Stiffness (Nm/°)	0.12 (0.03)	0.13 (0.05)	0.07 (0.03)†	0.07 (0.02)†
Norm. Stiffness (Nm/°kg)	0.0015 (0.0002)	0.0015 (0.0003)	0.0009 (0.0003) †	0.0008 (0.0002)†
Abs Avg. EMG (% MVIC)	3.6 (3.0)	3.7 (2.7) ¶	3.2 (2.6)	4.2 (3.2) ¶§
Back Avg. EMG (% MVIC)	3.6 (2.6) ‖	3.1 (2.7) ‖	3.4 (2.3) ‖	4.7 (3.3)

Significant differences ($p<0.05$) represented by † to represent a difference between the STIFF and LAX group. Significant group by stiffness interactions are indicated by a specific symbol to show differences relative to the: LAX ASYM subgroup (¶), STIFF ASYM subgroup (§), and LAX rLBI subgroup (‖). Abbreviations: normalized (Norm.), abdominals (Abs), back extensors (Back), average (Avg.), and maximum voluntary isometric contraction (% MVIC)

Table 5.3: Horizontal transfer task performance data, timing, motion, and peak external moments

	STIFF (30)		LAX (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Time (s)	4.3 (0.3)	4.2 (0.3)	4.2 (0.3)	4.3 (0.3)
Torso Flex/Ext (°)	3.3 (1.8)	5.3 (2.7)	4.0 (1.6)	3.6 (1.4)
Torso Lat. Flex (°)	2.9 (1.2)	2.4 (1.2)	3.0 (1.2)	2.5 (1.4)
Torso Ax Rot (°)	5.5 (2.5)	5.4 (2.9)	4.6 (1.6)	4.3 (1.7)
Pelvis Flex/Ext (°)	1.8 (1.1)	1.4 (1.1)	1.4 (0.9)	1.6 (0.8)
Pelvis Lat. Flex (°)	1.5 (0.9)	1.4 (0.9)	1.1 (0.7)	1.4 (0.9)
Pelvis Ax Rot (°)	2.7 (1.5)	2.4 (1.2)	2.2 (1.1)	2.3 (0.8)
Peak Flexion (Nm)	27.2 (3.0)	28.4 (3.8)	27.1 (3.9)	29.4 (3.5)
Peak Lateral Flexion (Nm)	11.7 (2.8)	12.4 (2.9)	11.7 (2.1)	11.3 (1.7)
Norm Peak Flex (Nm/kg)	0.34 (0.02)	0.33 (0.03)	0.34 (0.03)	0.33 (0.02)
Norm Peak Lat. Flex (Nm/kg)	0.15 (0.04)	0.14 (0.04)	0.15 (0.03)	0.13 (0.03)

No significant group or stiffness main effects or interactions ($p>0.05$). Abbreviations: flexion (Flex), extension (Ext), lateral (Lat.), axial (Ax), rotation (Rot), normalized (Norm), asymptomatic (ASYM), recovered low back injured (rLBI)

5.3.3 Horizontal Transfer Task Timing, Motion and External Moment

Time to complete the task, trunk and pelvis motion, and peak external moments for the horizontal transfer task are shown in Table 5.3. There were no group or stiffness main effects or interactions ($p> 0.05$) supporting that the controlled task parameters of timing (approximately 4 seconds), minimizing trunk and pelvis motion and task demand of the external moment was consistent among groups.

5.3.4 Trunk Muscle Activation Patterns

As the primary objective was to explore differences between STIFF and LAX participants, any stiffness main effects and interactions are first presented along with any group or muscle main effects to fully describe the data. Visual representation of the ensemble average calculated across select oblique muscles (EO1-3 and IO) is provided in Figure 5.3A. For the back extensors' ensemble average muscle activation patterns for the medial left and right combined back extensor sites, the left and right lateral back extensor sites are shown in Figure 5.3B-D.

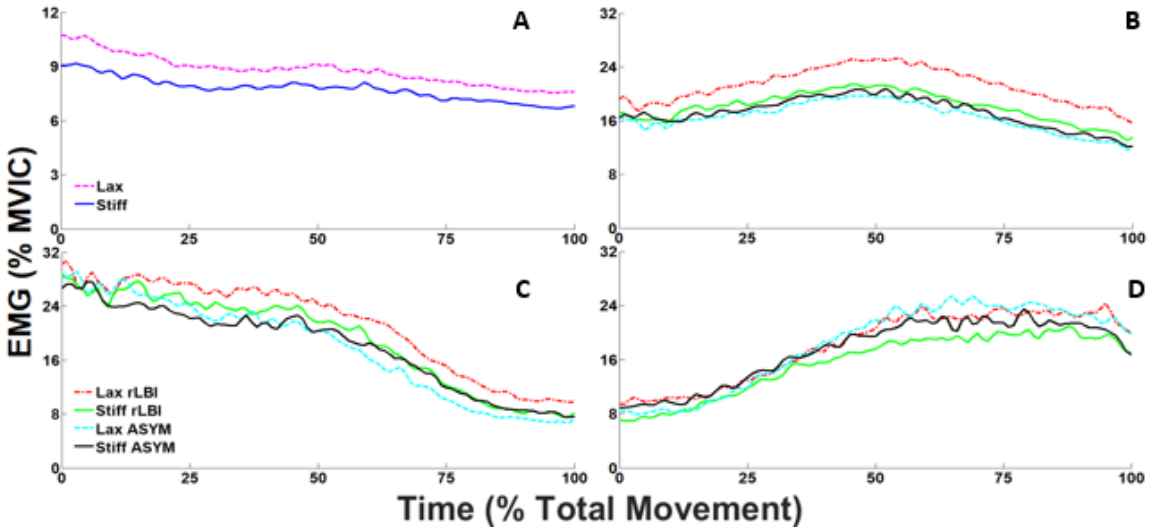

Figure 5.3: Normalized to maximum voluntary isometric contraction (%MVIC) ensemble average trunk electromyograms for recovered low back injury (rLBI) and asymptomatic (ASYM) STIFF and LAX subgroups. For the abdominals stiffness main effects are depicted by the contrast between A) the ensemble average of right anterior external (REO1) and internal obliques (RIO) and the left middle (LEO2) and lateral external oblique sites (LEO3). For the back extensors group by stiffness interactions are depicted by B) the ensemble average of both the right and left medial back extensor sites (L13, L33, & L52) and the C) left and D) right lateral back extensor sites (L16 & L36).

5.3.4.1 Abdominal Muscle Activation Patterns

Two PC's explained 96.5% of the total abdominal muscle activation waveform variance. For both the abdominals and back extensors PC1 captured the overall shape and amplitude of abdominal muscles (Figure 5.4 A&E) where high scores captured higher activation amplitudes (Figure 5.4 B&F). PC1 scores were highly correlated (r=1) with the

average (%MVIC) amplitude of muscle sites across the entire task so the average activation amplitudes in %MVIC are also provided to give context to PCs unitless score. Overall the average amplitudes were less than 8% MVIC for the abdominals (Table 5.4), with the IO sites having the highest (12% MVIC) activation (Table 5.5). There was a significant stiffness main effect ($p<0.001$) where LAX had higher activation amplitudes than the STIFF group (Table 5.4 & Figure 5.3A). In addition, a muscle main effect ($p<0.001$) captured differences in the activation amplitudes between muscle sites showing IO sites had the highest activation, followed by EO sites and finally the RA sites (Table 5.5).

For both the abdominals and back extensors, PC2 captured the muscular activation response to the lateral flexion moment (Figure 5.4 C&G). High scores corresponded with muscle sites having higher initial activation and lower terminal activation, whereas low (negative) scores corresponded to the opposite with lower initial activation (Figure 5.4 D&H). There were no significant stiffness main effects or interactions. A group by muscle interaction ($p=0.005$) found that the ASYM had higher responsiveness of the right EO1 compared to the rLBI group (Table 5.5). This interaction also captured differences in the synergistic relationship among muscle sites where both groups had asymmetries in the same three sites EO1, EO3 and IO. Finally, the ASYM had more differences among ipsilateral muscle sites than rLBI which only had differences between IO and EO3 (Table 5.5).

Given PC2 captured both the directionality (positive or negative Table 5.5) and magnitude (coefficient) of the responsiveness to the lateral flexion moment generated by this task, data were transformed to absolute values to explore whether the absolute magnitude of responsiveness regardless of direction could capture differences between stiffness groups (Quirk & Hubley-Kozey, 2018). Following this transformation, a stiffness main effect ($p=0.011$) found LAX was more responsive to the lateral flexion moment (higher scores) than STIFF (Table 5.4).

Table 5.4: Stiffness main effects and group by stiffness interactions for abdominals and back extensor PC scores

Stiffness (n)	STIFF (30)		LAX (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Abs AVG	6.3		7.3	
(% MVIC)	±5.0		±5.7	
Abs. PC1	63.3†		73.0	
(Unitless)	±50.4		±57.2	
Abs |PC2|	4.8†		6.3	
(Unitless)	±8.1		±10.4	
Back AVG	17.7	17.6	17.1	20.9
(% MVIC)	±8.3	±9.3	±7.7	±10.0
Back PC1	178.9‖	178.3‖	173.3‖	211.6
(Unitless)	±84.9	±94.0	±78.1	±100.9
Back |PC2|	38.6¶	42.8	47.0	42.2
(unitless)	±27.4	±29.1	±31.2	±28.0

If no main effect or interaction was identified the combined population mean was presented in the center of the table. Comparisons for PC2 are performed on the absolute value |PC2| scores. Significant differences ($p<0.05$) for stiffness main effects are indicated by a †. Significant group by stiffness interactions are indicated by a specific symbol to show difference relative to the: LAX rLBI (‖), and LAX ASYM subgroup (¶). Abbreviations: asymptomatic (ASYM), recovered low back injury (rLBI), Maximum voluntary isometric contraction (% MVIC), principal component (PC), abdominals (Abs), average activation amplitudes (AVG), and back extensors (Back).

.

Table 5.5: Muscle main effects and interactions for abdominal sites

	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO
AVG	3.7	3.7	3.8	3.3	6.9	7.8	6.8	6.9	7.5	8.2	11.1	11.6
(%MVIC)	±2.6	±2.4	±3.2	±2.7	±4.7	±6.3	±4.4	±5.5	±4.8	±4.9	±6.0	±6.4
PC1	36.8^{cdef}	37.1^{cdef}	38.5^{cdef}	33.5^{cdef}	69.6^{f}	78.0^{f}	67.9^{f}	69.8^{f}	75.7^{f}	82.2^{f}	111.4	117.1
	±25.9	±24.3	±31.7	±26.9	±47.5	±63.8	±44.1	±55.2	±48.6	±48.8	±60.1	±65.1
PC2	0.1^{c}	0.0^{ce}	0.4^{c}	-1.0^{ce}	**10.3***	**-13.4**	-2.1^{c}	4.9^{cef}	**-5.7^{cf}**	**14.7^{cf}**	**3.1**	**-4.0**
ASYM	±0.6	±0.7	±1.3	±1.4	**±15.5**	**±16.4**	±4.9	±12.1	**±11.8**	**±18.1**	**±7.7**	**±7.0**
PC2	-0.1	-0.5^{e}	-0.1	-0.7^{e}	**1.6***	**-6.5**	-2.8	0.7^{ef}	**-6.4^{f}**	**10.9^{cf}**	**3.5**	**-6.7**
LBI	±1.1	±1.4	±2.0	±1.8	**±6.2**	**±17.0**	±4.7	±6.4	**±9.0**	**±11.4**	**±8.5**	**±11.2**

All values are mean ± standard deviation and unitless unless otherwise indicated. If only a muscle main effect exists the combined average all groups is presented, otherwise for interactions the respective group (LBI vs ASYM) is indicated by the row title. Significant differences (<0.05) between muscle sites are indicated by **bold** lettering to show an asymmetry between left and right sites. Differences amongst ipsilateral muscle sites are depicted by superscript letters indicating a difference between the muscle site and: a) LRA, b) URA, c) EO1, d) EO2, e) EO3, and f) IO. Significant group by muscle interactions ($p<0.05$) are shown by * to indicate differences between the LBI and ASYM group within a specific muscle site. Abbreviations: average activation amplitudes (AVG), maximum voluntary isometric contractions (% MVIC), principal component (PC), asymptomatic (ASYM), recovered low back injured (rLBI).

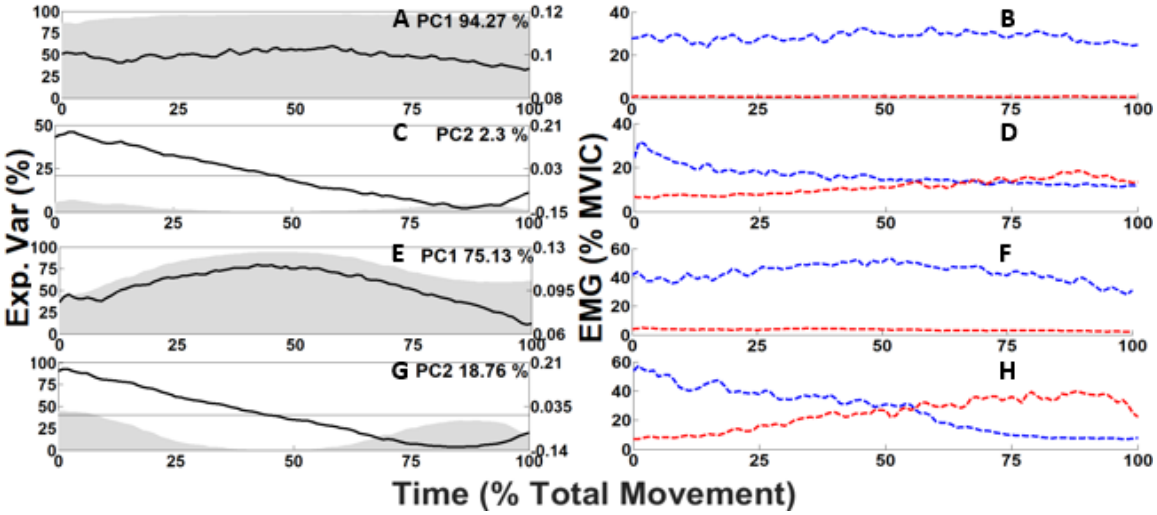

Figure 5.4: Visual representation of abdominal (A,C) and back extensor (E,G) principal component (PC) waveforms (black line) and ensemble average representations of high (blue) and low (red) electromyogram waveforms corresponding to each PC waveform (right) (B,D,F,H). For PC waveform plots grey shading depicts the time-varying explained variance with the collective total displayed on the top right of the sub-plot.

5.3.4.2 Back Extensor Muscle Activation Patterns

Two PC's explained 93.9% of the total waveform variance for the back-extensor muscle activation patterns. For PC1 there was a group by stiffness interaction ($p<0.001$) where the LAX rLBI subgroup had higher activation amplitudes than all other subgroups (Table 5.4 & Figure 5.3 B-D). In the ASYM there were no differences between LAX and STIFF (Table 5.4). Differences in muscle activation amplitudes were captured by a group by muscle interaction ($p=0.013$) showing while no specific muscle site had higher activation amplitudes between the rLBI and ASYM group (Table 5.6), there were differences in muscle synergies. The rLBI group had higher activation amplitudes of the left L52 site compared to other muscle sites whereas the ASYM group had no differences between muscle sites (Table 5.6).

For PC2, a muscle main effect ($p<0.001$) captured differences in the synergistic relationship amongst back extensor muscle sites (Table 5.6) showing that all back extensors were responsive to the changing lateral flexion moment resulting in asymmetries between right and left sites. In terms of the magnitude of responsiveness superior and lateral back extensors sites were the most responsive (highest absolute scores) to the changing lateral flexion moment (Table 5.6).

Table 5.6: Muscle main effects and interactions for back extensor sites.

	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52
AVG ASYM (%MVIC)	17.6 ±7.0	19.2 ±8.2	17.9 ±6.9	19.2 ±8.1	16.4 ±7.5	16.7 ±7.7	14.0 ±6.9	15.6 ±8.2	16.6 ±9.7	17.0 ±8.6	19.9 ±8.2	18.9 ±8.2
AVG rLBI (%MVIC)	17.3 ±8.5	21.7 ±10.1	16.4 ±7.1	20.9 ±9.3	18.2 ±8.9	19.8 ±10.2	15.6 ±10.3	18.3 ±9.9	18.4 ±10.3	18.6 ±11.1	22.0 ±8.9	24.4 ±10.0
PC1 ASYM	176.9 ±70.6	196.8 ±84.3	178.2 ±69.3	195.5 ±83.6	164.8[f] ±75.7	171.1 ±79.0	139.2[f] ±68.8	158.9 ±83.4	166.5[f] ±97.6	171.4 ±86.7	201.6 ±83.1	191.6 ±8.2
PC1 rLBI	172.7[f] ±85.0	222.2 ±102.8	**162.8[f] ±70.5**	**214.0 ±94.7**	181.8 ±88.8	203.0 ±103.1	155.4[f] ±103.0	186.8[f] ±100.5	183.9 ±103.5	187.9[f] ±112.3	221.4 ±88.9	247.7 ±100.8
PC2	**-57.3 ±29.1**	**52.1 ±32.7**	**-56.8 ±29.6**	**63.6 ±33.4**	**-47.5 ±27.7**	**37.2[a] ±24.6**	**-41.3[a] ±23.8**	**42.8[a] ±29.5**	**-31.0[ab] ±19.3**	**26.2[abd] ±19.2**	**-34.7[abc] ±22.7**	**13.1[abcd] ±25.8**

All values are mean ± standard deviation and unitless unless otherwise indicated. If only a muscle main effect exist the combined average across subgroups is shown. For all interactions, the respective group (LBI vs ASYM) is indicated by the row title. Significant differences ($p<0.05$) between muscle sites are indicated by **bold** lettering to show an asymmetry between left and right sites, and between muscle site differences amongst ipsilateral sites are depicted by superscript letters indicating differences between the muscle site and: a) L13 b) L16, c) L33, d) L36, e) L48, and f) L52. Interaction effects also include the symbols * to indicate significant ($p<0.05$) differences between the LBI and ASYM group within a specific muscle site. Abbreviations: average activation amplitudes (AVG), maximum voluntary isometric contractions (% MVIC), principal component (PC), asymptomatic (ASYM), recovered low back injured (rLBI).

Transforming PC2 scores to capture general responsiveness found a group by stiffness interaction (p=0.003). In the ASYM group, LAX participants were more responsive (higher scores) to the changing lateral flexion moment than STIFF participants (Table 5.4). Whereas, in the rLBI group LAX and STIFF participants were not different (Table 5.4).

5.4 Discussion

This study explored the spinal system compensation theory by examining whether those with lower passive spinal system function (lower stiffness) would have adaptations to trunk muscle activation patterns during a controlled dynamic task. Secondly, it examined whether the presence of a recent LBI modified these relationships. Fundamental was that all demographics, strength and task performance variables were not different between the STIFF and LAX groups. Despite no differences in task performance, there were differences in the muscle activation amplitudes and patterns for abdominal and back extensors between individuals with relatively low (LAX) or high (STIFF) mass normalized stiffness, resulting in stiffness main effects or interactions for all muscle activation patterns analyzed. Consistent with the hypothesis of the study, LAX had higher antagonist activation amplitudes than STIFF. Contrary to the hypothesis, LAX had greater responsiveness, of the abdominal muscles in both groups and in the back muscles of the ASYM group only, to the changing lateral flexion moment than STIFF.

To understand whether the experience of recent LBI modified how muscle activations adapted to passive laxity, the presence of group by stiffness interactions suggests each group had unique strategies. Participants who recovered from a LBI adapted to laxity using a different recruitment strategy, increasing overall activation amplitudes, whereas the ASYM, increasing the time-varying responsiveness of back extensor muscles.

5.4.1 Influence of stiffness on muscle activation patterns

All PCs examined had significant stiffness main effects or group by stiffness interactions. Consistent with our hypothesis, and studies around the knee (Shultz et al., 2004; Shultz & Schmitz, 2009), LAX had higher activation amplitudes of abdominal

muscle sites than STIFF (Table 5.4). The abdominals act primarily as an antagonist to the flexion moment produced during the horizontal transfer task. Higher antagonist activation amplitudes were thought to offset suspected deficits within the passive system that occur with advanced age or LBI (Quirk & Hubley-Kozey, 2018). Both experimental (Hodges et al., 2013; Vera-Garcia, Brown, & McGill, 2006a; Wong et al., 2016) and computational (Granata & Marras, 2000; Kavcic et al., 2004) work support that higher abdominal (antagonist) activation would increase active spinal stiffness. Thus, higher activation could compensate for lower passive stiffness as found in this study.

Novel was the higher responsiveness of the abdominals to the changing lateral flexion moment produced during the horizontal transfer task represented by higher absolute value PC2 scores (Table 5.4). Considering the line of action of the oblique can generate lateral flexor moments (Brown & Potvin, 2007), these muscles could participate as an agonist to the lateral flexion moments generated at the beginning and end of the transfer task. Our initial hypothesis was that LAX participants would utilize muscle activation patterns consistent with reduced responsiveness capturing a bracing strategy of higher antagonist co-activation in the frontal plane as observed previously in populations with suspected deficits of passive stiffness (Quirk & Hubley-Kozey, 2018; Trudel, 2014). While computational modeling supports that increased antagonist co-activation acts as a generalized mechanism to increase spinal stiffness and thus stability, McGill et al. have shown the time-varying recruitment of agonist is potentially more important for controlling spinal stability as a single period of inadequate agonist activation can result in instability (Kavcic et al., 2004). Thus, the observed increased frontal plane agonist responsiveness supports a change in active stiffness to compensate for lower stiffness contributed by the passive system.

Greater responsiveness to the lateral flexion moment has not been previously observed in populations with suspected passive system deficits (Quirk & Hubley-Kozey, 2018) and could be explained by confounding factors. In cross-sectional studies comparing older adults or those with chronic LBP to young healthy controls more responsive muscle activation patterns may be less suitable as impaired trunk proprioception (Brumagne et al., 2004; Claeys et al., 2010; Goldberg et al., 2005; Laird et al., 2014; Tong et al., 2015), and delayed reflexes (Hwang et al., 2007; Klass et al., 2011;

Ramprasad et al., 2010; Reeves et al., 2004; Shenoy et al., 2013) were also found in these populations. Adjusting agonist activation requires an accurate and rapidly changing feedback to modify muscle activation patterns potentially making increased frontal plane co-activation (reduced responsiveness) a more generalized and simplistic compensatory pattern (Reeves et al., 2011) consistent with computational models of how muscles would act with impaired feedback control (Stroeve, 1996).

5.4.2 Influence of recovery from LBP

The second purpose of this study was to determine whether the effects of passive stiffness on muscle activation patterns were modified by the recent experience of LBI. From the above discussion, the abdominal responses were influenced by laxity assignment, whereas for the back extensors there were group by stiffness interactions for both overall activation amplitudes (PC1) and responsiveness to the lateral flexion moment (PC2). Each group had a unique way to adapt muscle activation patterns to reduced passive stiffness.

The rLBI LAX subgroup had higher overall back extensor activation amplitudes compared to the STIFF subgroup (Table 5.4) but back extensor strength values were similar between rLBI subgroups (Table 5.1). Thus higher activation amplitude should result in higher moments produced by the back extensors in the sagittal plane to counter the flexion moment produced by the transfer task and would capture higher antagonist co-activation between left and right back extensor sites to balance moments produced in the frontal plane (Arjmand & Shirazi-Adl, 2006; Dumas, Poulin, Roy, Gagnon, & Jovanovic, 1991; Jorgensen, Marras, Granata, & Wiand, 2001). In the rLBI group, those in the LAX subgroup adapted to lower passive stiffness with a higher generalized active stiffness strategy in both the frontal and sagittal planes. This finding is consistent with another study showing greater generalized stiffness, sustained activation of the back extensor muscles in the frontal plane, for participants recovered from an LBI with suspected deficits in the passive spinal system based on clinical tests (Trudel, 2014).

A novel finding of this study was how this interaction modified muscle activation pattern adaptations to reduced passive stiffness in the ASYM compared to the rLBI group. In the ASYM group, the LAX subgroup had no difference in overall activation

amplitudes of their back extensors (PC1 Table 5.4) but the responsiveness in the frontal plane (PC2) shows the back extensors were more responsive to the changing lateral flexion moment than the STIFF subgroup (Table 5.4 & Figure 3C&D). As previously discussed, this greater responsiveness reflects an adaptation pattern that could lead to increase spinal stiffness through a counter moment produced by modulating activation of the agonist in the frontal plane. However, one must be cautious with this interpretation as MVIC normalized EMG amplitudes alone would require further knowledge of muscle size, moment arm length, line of action, and muscle specific force and stiffness in order to truly characterize the impact of this activation pattern on trunk stiffness (Bergmark, 1989). What is most interesting is that this difference was not found between the LAX and STIFF rLBI subgroups (Table 5.4). This supports the above speculation (Section 5.4.1) that potential deficits in the other spinal systems associated with current or recent LBP may limit the ability to utilize complex mechanism of neural control to adapt to lower passive system stiffness similar to the asymptomatic controls.

A secondary reason the rLBI may have adapted to reduced transverse plane stiffness in a unique way could be explained by their recent experience of activity limiting LBP independently modifying their muscle activation patterns. This study supports the use of a generalized stiffness pattern in participants recovered from LBP who are LAX, consistent with the motor adaption to pain theory by Hodges where individuals experiencing LBP modified their muscle activation patterns to generally increase spinal stiffness and stability (Hodges et al., 2013). Unique to this study is the finding that muscle activation pattern adaptations are modified by the presence of a recent LBP and the level of passive spinal system function. While LAX participants in both the rLBI and ASYM subgroups needed to adapt to passive laxity, the generalized (rLBI) vs more complex neural control (ASYM) mechanism used may be driven by the cost (potentially pain) associated with instability events. This tradeoff supports a recent theoretical framework proposed by Van Dieen and colleagues suggesting that optimization criteria to adapt muscle activation patterns may change when an individual is in a pain state (van Dieën, Flor, & Hodges, 2017).

5.4.3 Limitations

Although a number of novel findings were noted there are a few limitations that need to be addressed when interpreting the results. Testing time relative to participants waking time was not measured and could have an effect on the stiffness of the spine through diurnal changes, as intervertebral disc water loss results in marginal increases of neutral zone transverse plane laxity (Zander et al., 2009). Our participants attended either a morning (40/60) or afternoon (20/60 participants) data collection session. In vitro work suggests that most of this height loss of the disc occurs within the first hour of loading (Adams, Dolan, Hutton, & Porter, 1990). Thus, most of our morning participants likely experienced their normal loss of disc height by the time testing took place as they would have traveled to the lab and participant preparation took approximately half an hour. Consistent with our assumption Drake and Callaghan showed that in vivo measures of axial rotation were not different for test retest measurements taken 30 minutes after the participant awoke to those measured in the afternoon (Drake & Callaghan, 2008).

Secondly, in vivo measurements of spinal stiffness could be influenced by the compressive loads experienced by the spine. In vitro work shows that angular stiffness of spinal segments increases with compressive forces (Noguchi et al., 2015; Zirbel et al., 2013), reducing motion segment neutral zone (Noguchi et al., 2015). Indeed, our data found participants with greater mass had higher axial rotation stiffness (Pearson correlation coefficient of r=0.43). Normalizing stiffness to mass addressed the confounding influence of mass differences between STIFF and LAX subgroups (Table 5.1). The inclusion of the participants natural mass in our spinal stiffness measures reflect the in vivo passive spinal stiffness measures for this study represents a summative intrinsic passive stiffness including stiffness provided by spinal compression, skin and musculotendinous structures rather than a measure of pure osteoligamentous stiffness (McGill et al., 1994). We feel this is a pragmatic measure representing the extent this natural intrinsic stiffness is involved in controlling spinal stability during activities of daily living performed in standing.

To what extent our measurement of passive stiffness captured stiffness from osteoligamentous tissues alone, we found that the results of our average angular stiffness from 0-3 degrees (0.10±0.04 Nm/° or 0.0013 Nm/°kg) were similar yet slightly lower to

the measure of 0.13 Nm/° or 0.0018 Nm/°kg reported by McGill in a group of 22 young (21±1 years old) men (McGill et al., 1994). McGill et al., also questioned the validity of their findings showing their results were similar yet somewhat higher compared to in vitro specimens for testing in flexion-extension. Compared to in vitro work our measure of neutral zone range of motion (3.9±1.8°) falls within the range of an entire lumbar segment (0.5-8°) when considering that the neutral zone of any singular functional spinal unit with varying degrees of disc degeneration is approximately 0.1-0.8° of axial rotation in the CW or CCW direction alone (Volkheimer et al., 2017). Our data in part captured stiffness from the osteoligamentous system but is limited in that it represents a measure of functional stiffness from other sources. Another method to characterize the passive system could include radiographic evidence of lumbar segment disc degeneration which has been associated with in vitro measures lower of spinal segment stiffness in the transverse plane (Galbusera et al., 2014; Tanaka et al., 2003; Volkheimer et al., 2017). Secondly, the stiffness measure in the current study focused on a range of motion that captured the participant's neutral zone. The decision to analyze this region of stiffness was made as disc degeneration is known to consistently lower stiffness within this neutral region for axial rotation (Galbusera et al., 2014; Tanaka et al., 2003; Volkheimer et al., 2017). No consistent relationship exists in other planes or across larger (full range) of motion as stiffness has a trend to decrease with moderate degeneration and increase when moving to severe degeneration (Galbusera et al., 2014; Tanaka et al., 2003; Volkheimer et al., 2017). This relationship is explained by earlier bone on bone contact leading to increased stiffness when disc degeneration becomes severe (Fujiwara et al., 2001; Galbusera et al., 2014; Kong et al., 2009; Tanaka et al., 2003). Thus, findings must be interpreted for this plane only.

Although there were statistical differences between subgroups (Table 5.3) in angular velocity during our in vivo measurements of passive stiffness, the actual impact on our findings is minimal. The LAX subgroups were rotated at a 1˚/s higher angular velocity than STIFF. While in vitro work supports that step-wise quasi-static loading of functional spinal units have larger neutral zone motion and lower stiffness compared to dynamic (1˚/s) loading no difference was identified in neutral zone or stiffness when comparing dynamic loading velocities ranging from 0.25-14˚/s (Stolworthy, Zirbel,

Howell, Samuels, & Bowden, 2013). Thus the 1˚/s difference between groups would have negligible effects on measured stiffness and if anything would increase the stiffness measured in the LAX subgroup.

The LAX rLBI subgroup had significantly higher but small (0.5-1.2% MVIC) activation amplitudes of abdominal and back extensor muscles during the testing paradigm (Table 5.3). Given trunk muscles activation impacts spinal stiffness (Cholewicki et al., 1997), our measures of spinal stiffness in the LAX rLBI subgroup may be higher than if their muscles were relaxed to the same background activation amplitudes as the other subgroups. Work by Brown and McGill showed that increasing levels of muscle activation can increase measures of stiffness in flexion-extension and lateral bending. However, this effect is not significant when participants were encouraged to completely relax (2±2%MVIC across all trunk muscles) to when they were encouraged to perform a light contraction of obliques (4±3% MVIC across all trunk muscles) (Brown & McGill, 2007b), suggesting the higher background muscle activity in this study likely had minimal effect on passive stiffness measures and stiffness group classification. Attempts were made to reduce the effects of these confounders including EMG biofeedback. The small differences reflect the success of these controls and likely the statistically significant differences are due to the small variance in the data. Similar to the velocity confounder, if background activation had an effect it would place the participant in the higher stiffness group, and thus likely had no effect on group allocation.

A final limitation of this study was while this study explored whether those with deficits in the passive spinal system would have different muscle activation patterns our definition of a deficit was based on participants having below median transverse plane stiffness. Thus, while our measure of lower function is a potential risk for experiencing future low back pain the threshold used in this study does not relate to a clinical threshold of deficient function. Despite a less distinct difference between subgroups, measures of spinal stiffness between groups did capture muscle activation pattern differences with lower spinal system function, supporting Panjabi's compensation theory.

5.5 CONCLUSION

In conclusions, the interactions between passive trunk stiffness and trunk muscle activation patterns during a controlled transfer task in part support Panjabi's compensation theory. Participants classified as lower (LAX) passive stiffness had different muscle activation patterns to complete a controlled transfer task compared to those with higher (STIFF) stiffness. Not only did muscle activation patterns differ between LAX and STIFF groups, with LAX participants having higher antagonist co-activation and greater responsiveness of the abdominals to changing lateral flexion moments, but the presence of a recent LBI modified these responses. Group by stiffness interactions captured that those recovered from a recent LBI had higher overall activation amplitudes of the back extensors to generally increase active stiffness, whereas asymptomatic participants adapted to laxity by making agonist muscles more responsive to changing lateral flexion moments produced by this task. Consistent with our hypothesis, the trunk muscle activation patterns observed adapt to lower passive system stiffness in a way to increase the active contribution of stiffness to the spine. Furthermore, there is preliminary evidence that pain or simultaneous deficits in the other spinal systems may modify how muscle activation patterns adapt to lower passive system stiffness.

CHAPTER 6
TRUNK MUSCLE ACTIVATION PATTERNS DIFFER BETWEEN THOSE WITH DELAYED TRUNK MUSCLE REFLEXES IN ASYMPTOMATIC AND RECOVERED LOW BACK PAIN PARTICIPANTS

6.1 INTRODUCTION

The lumbar spine is an inherently unstable structure. Computational and experimental modeling establish that the osteoligamentous spine alone would become unstable, unable to return to a state of equilibrium, with the application of low compressive forces (80-90N) below those produced by the mass of the head (Crisco et al., 1992; Crisco & Panjabi, 1992). However, in vivo, the human spine can safely perform tasks at considerably higher (3400N) compressive forces (Waters & Putz, 1994; Waters, Putz-Anderson, Garg, & Fine, 1993). The explanation of this is articulated in a theoretical model proposed by Panjabi, stating the ability of the spine to remain stable is not explained by any singular tissue system, but rather is achieved by all tissues that can contribute to or modify forces generated around the spine (Panjabi, 1992a; 1992b). In Panjabi's model tissues are categorized into three fundamental systems, passive, active and neural, which function to predict, monitor and respond to changes in the forces or motion generated around the spine (Panjabi, 2005). While stiffness generated by the active system is a determinant of spinal stability (Bergmark, 1989; Cholewicki et al., 1997), a highly organized neural controller is needed to ensure the active system can generate forces in such a way that it would not lead to spinal instability (Cholewicki & VanVliet, 2002; Kavcic et al., 2004; McGill, Grenier, Kavcic, & Cholewicki, 2003a). Not only does the neural system play a role in maintaining the distribution of activation to multiple, often redundant, muscles to maintain stability, it is theorized the neural system also attempts to find an optimal solution to minimize the cost of stress to any singular muscle (Brown & Potvin, 2005; Rashedi et al., 2009; van Dieën et al., 2017).

In his theoretical model Panjabi proposed that if one spinal system experienced a deficit, an individual would be at an increased risk of low back pain (LBP) via an instability event (Panjabi, 1992a; 1992b). Considering the neural system, the function of

these tissues is to detect joint motion and forces using sensory organelles embedded within osteoligamentous (Dimitroulias et al., 2010; Goble et al., 2008; McLain & Pickar, 1998; Sjölander, Johansson, & Djupsjöbacka, 2002), and musculotendinous tissues (Amonoo-Kuofi, 1983; Boyd-Clark, Briggs, & Galea, 2002; Ge & Pickar, 2012) which are transmitted to sensory afferents. Afferent signals are transmitted to a variety of structures in the central nervous system (spinal, sub-cortical, or cortical) which must resolve whether these sensory afferents are consistent or divergent with one's movement goals (Jacobs & Horak, 2007). The speed at which the nervous system can resolve these conflicts and adjust muscle activation is important for spinal stability. Studies show individuals with chronic LBP have delayed reflexes compared to pain free controls (Liebetrau, Puta, Anders, de Lussanet, & Wagner, 2013; Radebold et al., 2000; Radebold, Cholewicki, Polzhofer, & Greene, 2001; Reeves et al., 2004), as summarized in two reviews (Knox, Chipchase, Schabrun, Romero, & Marshall, 2018; Prins et al., 2017). Furthermore, a prospective study in approximately 300 college athletes, found there was a 3% increase in the chance of experiencing a low back injury (LBI) in a 2-3 year follow up period for each millisecond trunk reflexes (muscle shut off times) were delayed (Cholewicki et al., 2005). This suggests, consistent with Panjabi's hypothesis that deficits in the neural system, characterized as delayed trunk muscle reflexes, represents a predisposing risk to LBP arising from an instability event.

Despite evidence suggesting individuals with delayed reflexes, have compromised spinal stability, a secondary theory by Panjabi proposed deficits within individual spinal systems could be compensated by an adaptation in the function of another spinal system (Panjabi, 2003). To our knowledge, no study has attempted to understand how the spinal systems adapt to delays in muscle stretch reflexes to maintain joint stability during functional tasks. Computational modeling and experimental work suggests stiffness produced by modifying muscle activation through a reflexive mechanism is paramount to maintain stability in the event of sudden loading (Brown & McGill, 2008; Franklin & Granata, 2006; Liebetrau et al., 2013; Moorhouse & Granata, 2006; van Drunen, Maaswinkel, van der Helm, van Dieën, & Happee, 2013). Control of the neural system to maintain joint stability is a delicate balance. Computational modeling highlights if a joint had no baseline level of agonist and antagonist co-activation, to increase intrinsic

stiffness, it would become unstable despite any effort to manipulate reflexive activation in both timing and amplitude (DeMers, Hicks, & Delp, 2016). However, despite experimental work highlighting that increased intrinsic spinal stiffness is successful in limiting the velocity and displacement of spinal motion following sudden loading (Lee, Rogers, & Granata, 2005; Shahvarpour, Shirazi-Adl, Mecheri, & Larivière, 2014; Vera-Garcia et al., 2006b), intrinsic stiffness alone is insufficient to return a joint to a state of equilibrium without adjusting muscle activation (Brown & McGill, 2008; Moorhouse & Granata, 2006).

When the nervous system is delayed a joint can experience more motion before reflexive adjustments occur increasing the likelihood of joint instability. Consistent with Cholewicki, computational modeling confirms spinal stability is compromised with each millisecond of reflex delay (Franklin & Granata, 2006; Liebetrau et al., 2013). In addition, experimental studies show delayed reflexes can impact postural stability. Studies by Cholewicki's group, have shown delays in spinal reflexes are associated with participants having a greater deviation in center of pressure motion (Radebold et al., 2001) and a greater likelihood to fail (lose balance) (Reeves, Cholewicki, & Narendra, 2009) during seated balance tasks. While no study has attempted to understand how the spinal systems adapt to delayed reflexes; most suspect individuals with an deficient neural system would settle on higher intrinsic stiffness to minimize the motion that occurred following unexpected loading reducing the need of the neural system to generate a quick and accurate restorative force (Davarani, Shirazi-Adl, Hemami, Mousavi, & Parnianpour, 2007; Panjabi, 2003; 2005; Reeves et al., 2011). Indirectly there may be some support for this proposed compensation. A study by Fu et al, have shown deficits in the neural system, characterized as reduced ankle position sense, was associated with increased ankle co-activation for a drop jump landing task (Fu & Hui-Chan, 2007).

Further indirect evidence regarding how muscle activation patterns adjust to neural deficits come from cross-sectional studies. Older adults and those with LBP have delayed trunk muscle reflexes compared to younger pain free controls (Reeves et al., 2004; Shojaei, Nussbaum, & Bazrgari, 2016), and have different trunk muscle activation patterns to complete a variety of tasks. These muscle activation patterns include higher

agonist and antagonist activation amplitudes (Hodges & Moseley, 2003; Hubley-Kozey et al., 2013; McGill et al., 1999; Mehta et al., 2010; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014; van Dieën et al., 2003), and sustained muscle activation in response to changing external moments (Butler et al., 2012; D'hooge, Hodges, et al., 2012b; Hubley-Kozey & Vezina, 2002; Hubley-Kozey et al., 2013; Moreside et al., 2013; Quirk & Hubley-Kozey, 2014) compared to younger asymptomatic cohorts. These changes in muscle activation patterns are consistent with methods of increasing the intrinsic stiffness of the spine (Brown et al., 2006; Granata & Marras, 2000; Kavcic et al., 2004; Lee et al., 2005; Shahvarpour et al., 2014) and thus support a shift towards higher intrinsic stiffness in those with reflex delays. However, there is a need for caution when interpreting these results in LBP populations as pain alone can modify trunk muscle activation patterns changing both overall activation amplitudes and temporal recruitment (Dubois et al., 2011; Hodges et al., 2003; 2013; Hug et al., 2014; van den Hoorn et al., 2014), shifting participants towards muscle activation patterns that would exaggerate intrinsic spinal stiffness (ie. co-activation) without any changes to the individual spinal systems (Hodges et al., 2013). Recent theoretical models suggest even when LBP resolves, individuals may retain their painful muscle activation patterns (Hodges et al., 2013; van Dieën et al., 2017) increasing spinal stiffness above what is necessary to compensate for impairments in individual spinal systems (Quirk & Hubley-Kozey, 2018).

The aim of this study was to probe the spinal system compensation theory by assessing the interaction between neural spinal system function and neuromuscular control of the trunk muscles during a dynamic task. This experiment tested for differences in abdominal and back extensor muscle activation amplitudes and patterns during a controlled dynamic lifting task between those who had slow (SRL) versus fast reflex latency (FRL) times in response to a sagittal plane velocity controlled reflex protocol to assess a component of the neural spinal system. The hypothesis was that slow individuals would utilize muscle activation patterns that increase the active stiffness of the trunk including higher EMG activation amplitudes of agonist and antagonist muscles compared to fast individuals, and temporal changes consistent with less responsiveness (sustained activation) to changing external moments. A secondary purpose was to explore

whether the recent experience of a low back injury modifies the trunk muscle activation differences between those with slow versus fast reflex latencies.

6.2 METHODS

6.2.1 Participants

Participants for this study were recruited from the Canadian Armed Forces. Asymptomatic participants volunteered by responding to on base posters and base-wide recruitment e-mails. Recovered LBI (rLBI) participants were identified if they reported a back related issue resulting in altered activities of daily living for at least 3 days (Ozguler et al., 2000) to the Canadian Forces Health Services Center and were contacted to see if they were interested in participating in the study. For both groups a self-report questionnaire screened for the following exclusion criteria: previous abdominal or back surgery, cardiovascular, respiratory or neurological conditions that would place them at risk for participating in the study. For the participants in the rLBI group questions determined that their recent LBP occurred 4-12 weeks prior to testing, was not chronic lasting longer than 12 weeks, and not recurrent where a previous injury occurred within 12 weeks prior to their most recent episode (Delitto et al., 2012). Asymptomatic participants were screened to determine that they had not experienced an activity limiting LBP (Ozguler et al., 2000) in the last year. All rLBI were deemed recovered at the time of testing reporting minimal pain (Visual Analog Scale (VAS) <30/100mm (Boonstra et al., 2014; Jensen et al., 2003; Wewers & Lowe, 1990)), minimal disability (Roland Morris Disability (RMD) <9/24 (Roland & Morris, 1983; Stratford et al., 1998)), and the resumption of normal activities of daily living at the time of testing. Before testing all participants signed an informed consent approved by the Institutes Research Ethics Board.

6.2.2 Protocol

Participants attended two sessions. During session one, participants' were screened by a registered physiotherapist to determine whether they met the minimal pain and disability criteria and confirmed the absence of neurological conditions. During session two, participant's pain, demographic and anthropometric data were measured.

Self-reports of weekly engagement in physical activity (aerobic sessions >30 minutes, core/ abdominal and strength training), if they frequently lifted objects weighing over 23kg for their work, defined as a heavy job (Seidler et al., 2009) and handedness were recorded. Participants also completed a Tampa Kinesiophobia (Kori et al., 1990) and Pain Catastrophizing Scale (Sullivan et al., 1995) to characterize their beliefs towards pain.

Participants were prepared for data collection including EMG setup (Section 6.2.3.2) followed by reflex testing. For this test, the participant was secured to two rigid bodies designed so the torso could articulate to a fixed pelvis (Figure 6.1). Height of the lower rigid body platform was adjusted such that the axis of the HUMAC Centroid was located 8 cm anterior to the participant's posterior superior iliac spine to approximate the L4/L5 center of rotation (Dumas et al., 2006; Reed et al., 1999). A steel arm attached to a HUMAC Norm Dynamometer (Computer Science Medicine Inc, Strongton, MA, USA) was positioned behind the participants scapular spine. Non-elastic straps were positioned over the participants shoulders and pelvis securing them to the steel arm and lower rigid body (Figure 6.1).

For reflex testing participants were requested to stand in their most comfortable position, defined as their neutral standing (0˚). Electronic stops were set at 3˚ extension and 12˚ flexion (Figure 6.1A and C respectively) using the HUMAC software. Participants were instructed "for this test they would be flexed forwards (Figure 6.1 A→C) or extended backwards seven times in each direction with an unexpected time between these motions". To limit the confounding influence of visual reflexes (Goodworth & Peterka, 2009) or background muscle activation (Larivière et al., 2010) (A. S. Lee et al., 2007a), participants were instructed to "keep their eyes closed and relax their trunk muscles during the entire test". However, participants were also requested to "keep tension in their neck muscles to prevent unwanted head motion". Delivery of the perturbations were controlled by the HUMAC software to produce a 120˚/s perturbation every 7 seconds (Sánchez-Zuriaga et al., 2010).

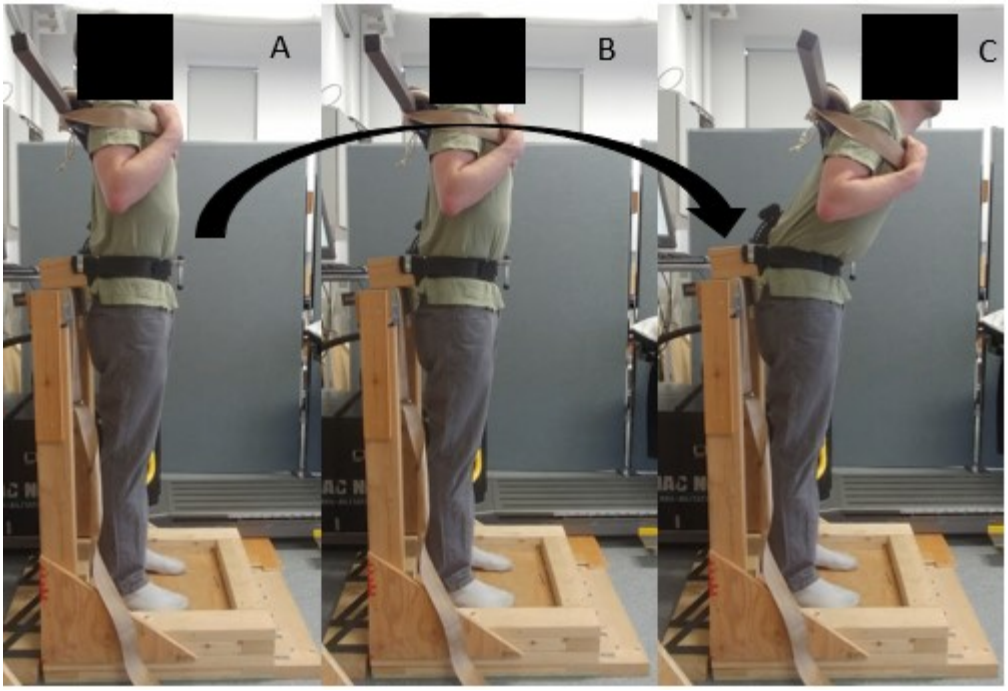

Figure 6.1: Visual representation of reflex testing apparatus showing the progression of electronically controlled motion as a participant was rotated from 3˚ extension (A) to 12˚flexion (C). All photos depict lower rigid body secured to the participant's pelvis, and the upper rigid body attached to the HUMAC dynamometer.

Following the reflex test, participants were prepared for kinematic analysis (Section 6.2.3.3) and then performed three trials of a highly controlled horizontal transfer task (Butler et al., 2010). Briefly, to a standardized five second count, participants were instructed to on "1" lift a 3kg mass orientated 60˚ to the right of the midline of their body with their right hand, on "3" transferring the mass to their left hand at the midline of their body, and then on "5" lower the mass 60˚ to the left of their midline. Mass lift and lower were recorded using a pressure sensor located at the bottom of the mass, and hand transition was measured using an optoelectric switch. A high adjustable table was used to ensure the mass was lifted just below elbow height, and the optoeletric switch ensured the mass was lifted no higher than 5cm. During the task, a researcher ensured the participant lifted the mass with the arm in full extension. Participants were asked to minimize trunk motion aided by tactile feedback provided to the upper thoracic spinous process.

Participants then performed a series of maximum voluntary isometric contractions (MVIC) for EMG amplitude normalization including: i) supine sit-up, ii) side lying lateral flexion (left and right), iii) prone back extension, and iv) prone back extension

coupled with back extension (right and left) (Butler et al., 2010) with each exercise repeated (two trials). Participants were encouraged to maintain maximal effort for 3 seconds against non-elastic straps, with a minute rest between contractions.

Trunk strength (flexor and extensor moment) was measured during two MVIC exercises. Participants were positioned in a prone or supine crook lying position with the HUMAC Norm Dynamometer arm positioned anterior and inferior to the clavicle for trunk flexion, or superior and posterior to the spine of the scapula for trunk extension (Hasue et al., 1980). The HUMAC centroid was positioned approximately 5cm anterior to the posterior superior iliac spine, in line with the iliac crest, and non-elastic straps were secured to anchor the pelvis and shank. Following gravity correction, participants performed two trials with instructions and procedures consistent with the normalization tasks.

6.2.3 Data Collection and Analysis

6.2.3.1 Reflex Testing

During the reflex test, analog signals (angle and moment) from the HUMAC and surface electromyograms (Section 6.2.3.2) from 24 muscles were simultaneously digitized at 2000Hz using a 16 bit (±5V) analog-to-digital board (PCI-6033E, National Instruments, Austin, TX) and Labview™ (Version 2017, National Instruments). Custom Matlab code filtered the HUMAC data using a 10Hz second order zero-lag low pass Butterworth filter, and converted it to angle (˚) according to equations provided by HUMAC. The first derivative of the position data were calculated to determine perturbation velocity. The velocity signal was monitored for event detection, as analog data was collected across the entire reflex experiment. Perturbation onset was calculated as the sample at which the velocity exceeded a threshold of -110˚/s or 110˚/s for flexion and extension perturbations respectively.

6.2.3.2 Surface Electromyography

Surface electrodes (Ag/AgCl, 10mm diameter, Red dot, 3M, Maplewood, MN) were placed in a bipolar configuration (30mm interelectrode distance) over 12 bilateral muscle sites using a standard protocol (Butler et al., 2010). Abdominal sites included the

upper and lower rectus abdominus (URA & LRA), the internal (IO), and external (EO1-3) oblique sites representing the anterior, lateral and posterior fibres respectively. Back extensor sites included the superficial quadratus lumborum (L48) and multifidus (L52) along with the erector spinae at level of the 1st and 3rd lumbar spinous approximately 3 and 6 cm horizontal to the midline to capture the longissimus (L13 & L33) and iliocostalis (L33 & L36) fibres respectively (Butler et al., 2010).

EMG signals were pre-amplified (500x) and further amplified using three AMT-8 amplifiers (band-pass 10-1000Hz, CMRR=115 dB, input impedance 10GΩ; Bortec Inc., Calgary, AB). Custom Matlab (Math Works, Natick, MA) code corrected EMG signals for participant bias, electrocardiogram artifact (high pass zero-lag 30Hz filtered) (Butler et al., 2007) and noise from electromagnetic sources (inverse fast-Fourier filtered removing frequency spikes with a power 3 times greater than their nearest frequency (+/-15Hz) neighbours). Data were then processed separately for the reflex and horizontal transfer task.

For the reflex test, EMG data were rectified and low pass filtered at 50Hz using a second order zero-lag Butterworth filter, to produce a linear envelope to determine reflex onset (Hodges & Bui, 1996). Using the event signals from the HUMAC (see section 6.2.3.1) EMG data were cut into epochs -500ms before and 1000ms following the perturbation event (Figure 6.2). Primary outcome measures were calculated only for the stretched (lengthened) muscle sites, back extensor or abdominal sites, depending on whether the perturbation was in the flexion or extension direction respectively. Three outcome measures were calculated i) muscle onset, defined as the period at which the EMG signal exceeded 3x the mean and standard deviation of EMG muscle activation measured -500 to -400ms prior to the perturbation onset (time 0) for at least 25ms (Hodges & Bui, 1996) in a period of 25-150ms following the perturbation to suggest the source was from involuntary reflex (Radebold et al., 2000; Stokes et al., 2000; Taube et al., 2006). Each muscle onset was confirmed using visual detection (Figure 6.2). If no reflex was detected in this period the reflex was defined as ii) (responsive or) not-responsive. Finally iii) the short latency reflex amplitude, quantified as the average linear-envelope amplitude from the time of muscle onset and the 30ms following reflex onset, was calculated (Klass et al., 2011; Obata et al., 2009; 2011). Reflex amplitude was

normalized to the maximum average 500ms moving window linear envelope regardless of what task evoked the MVIC (Vera-Garcia et al., 2009; Vezina & Hubley-Kozey, 2000).

For the horizontal transfer and MVIC tasks, EMG data were rectified and low pass filtered at 6Hz using a second order zero-lag Butterworth filter, to produce a linear envelope. These EMG signals were amplitude normalized and time normalized from 0-100% of the total task time (from the event markers) using a quadratic interpolation algorithm. Ensemble average waveforms were calculated from the three trials. To characterize overall neural drive the average (% MVIC) activation was calculated for the waveform for the entire task. To characterize spatial temporal characteristics of various muscles collected, ensemble average waveforms for each participant (N) and muscle site (12) created two data matrices [(N*12) x 101] for the abdominals and back extensors separately. Each data matrix was entered into a principal component (PC) analysis model (Hubley-Kozey & Vezina, 2002). Briefly, each data matrix was transformed into a covariance matrix and underwent an eigenvector decomposition to identify eigenvectors or PCs which explained patterns of maximum variation within the waveforms. For each EMG waveform, a coefficient or PC score was calculated to fit the PC to the original waveform. The number of PCs extracted was determined such that the total explained variance for the combinations of PCs reached 90%, and that each PC explained at least 1% of the waveform variance (Hubley-Kozey & Vezina, 2002).

6.2.3.3 Kinematic and Kinetic Analysis

Following reflex testing, passive reflective markers were positioned on the suprasternal notch and 7th cervical spinous process along with 16 bilateral landmarks on the limbs: 5th and 2nd metacarpal, radial styloid, ulnar styloid, medial and lateral humeral epicondyle, the mid acromion clavicular joint, and the anterior superior iliac spine. Four marker rigid body clusters were affixed to the thorax and pelvis, along with bilateral triads on the forearm and upperarm to capture motion. Following marker setup, a single standing calibration trial captured the marker position relative to the rigid bodies using six infrared emitting cameras (ProReflex 240, Qualisys^{TM}, Goteborg, Sweden) sampled at 100Hz using Qualisys Track Manager Software (Version 2.10, Qualisys^{TM}, Goteborg,

Sweden). For synchronization, the Labview program collecting analog-to-digital data triggered the motion capture system.

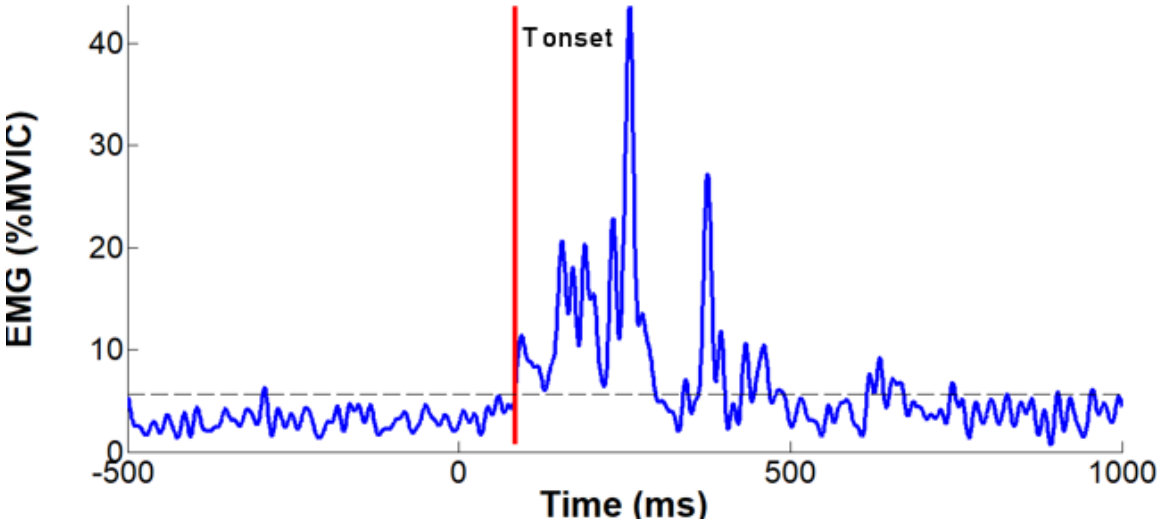

Figure 6.2: Representative trial of an individual muscle sites reflex response. For each perturbation (EMG) data (blue line) were cut -500ms prior to and 1000ms following perturbation (time 0ms). If a muscle were responsive reflex onset (red line) was measured at the time (T onset) EMG activation exceeded a mean + 3 standard deviation threshold (black dashed line).

Marker data were processed in Qualisys Track Manager, to label marker's coordinates. Marker coordinate data were entered into a custom Matlab™ script for quadratic interpolation of missing data points, and low pass filtered at 4Hz using a fourth order, zero-lag Butterworth filter (Wicks, 2017). Kinematic data were processed in accordance with the International Society of Biomechanics recommendations (Wu et al., 2002; 2005). Within each joint, an anatomical coordinate system was defined using bony landmarks, and joint centers as calculated using regression equations (Dumas et al., 2006). Segment angles were calculated using Euler angles in a Z-Y-X (lateral flexion, axial rotation, flexion-extension) rotation sequence. To quantify the motion during the horizontal transfer task segment angles were low pass filtered using a 1Hz second order, zero-lag Butterworth filter (Butler et al., 2010; Wicks, 2017). For each trial, the total displacement (maximum – minimum) of the torso and pelvis was calculated in lateral flexion, flexion extension, and axial rotation and averaged over the three trials for each participant.

Kinematic data were also used to calculate the moments of force around the trunk using a top-down static inverse dynamic model. Known external mass, along with segment mass magnitude and location estimated using anthropometrically derived regression equations (Dumas et al., 2006) were inputs for the force and moment calculation using a system of Newton-Euler equations (Winter, 2009). An open kinetic model including joint forces and moments from distal joints were used to calculate forces and moments occurring at the proximal joint. The mass of the lifted object was assumed to apply a weight vector at the middle of the 2nd and 5th metacarpal of the right hand for the beginning of the lift, both hands during hand transition, and the left-hand following hand transition. Following this calculation, the peak lateral flexion and flexion moment around the trunk was calculated for each trial and then averaged across trials for each participant.

6.2.4 Strength Analysis

For strength testing, gravity corrected external moments sampled from the HUMAC, were entered in a custom Matlab code, filtered using a 6Hz second order zero-lag low pass Butterworth filter, and converted to a moment (Nm) according to equations provided by HUMAC. The maximum isometric moment for flexors and extensors were calculated as the highest average moment over a 500ms window and normalized to body mass to compensate for anthropometric differences between participants (Smith et al., 1985).

6.2.5 Statistical Analysis

To test whether there were differences in muscle activation amplitudes and patterns between those with slow or fast reflexes, a median split approach was used on averaged muscle onset, calculated as the average of all agonist muscles in both the flexion and extension direction, within both the rLBI and asymptomatic group. The choice to split participants into groups based on average (trunk flexor and extensor) onset was used as it is consistent with previous studies (Cholewicki et al., 2005; Radebold et al., 2001; Reeves et al., 2004), and has been shown signal averaging across multiple muscle sites improves the reliability of reflex latency measures (Santos et al., 2011).

Two-way analysis of variance models (ANOVA) (group and onset) tested for differences in demographics, anthropometrics, strength (maximum flexor and extensor moment), lifting motion, lifting moment, and lifting timing data. Categorical data were compared using a Chi-square test for independence. These data determine if there were confounders to be included as co-variates or adjust populations testing for muscle activation (PC) differences.

EMG data were analyzed in different ways according to the task observed. Normality of EMG data (amplitude and PC scores) was assessed using a Kolmogorov-Smirnov test, with non-normal data transformed using a Johnson transformation. For the reflex test, reflex onset, average EMG amplitude (% MVIC), and percent detected for both flexion and extension perturbations were compared using a three-factor (group, onset and muscle) mixed model ANOVA for abdominals and back extensors separately. For the horizontal transfer task, PC scores were analyzed using three-factor (group, onset and muscle) mixed model ANOVAs for the abdominals and back extensors separately. Tukey simultaneous tests compared pairwise differences when significant. Statistical analyses were performed in Minitab (version 17, State Collage, PA). Alpha was set at 0.05 and Bonferroni corrected.

6.3 RESULTS

6.3.1 Anthropometrics, Demographics and Strength

Sixty-nine participants volunteered for the study and of those nine were women. Only two women were in the rLBI group. Thus, the analyses were performed on the 60 men (30 rLBI). Participants recovered from a LBI were tested 9±2 weeks from their most recent episode of LBP. For the fast (FRL) and slow reflex latency (SRL) groups, participants were identified if their average muscle onset for the combined abdominals and back was below (FRL) or above (SRL) 62 and 63.7ms for the ASYM and rLBI respectively. General characteristics of the separate participant groups are shown in Table 6.1. No significant onset main effects or interactions were identified ($p>0.05$). Significant group main effects found that the rLBI group had higher mass, ($F(1,56)=11.80$, $p=0.001$) pain (VAS ($F(1,56)=13.45$, $p=0.001$)) and disability (RMD ($F(1,56)=9.04$, $p=0.003$)) scores and higher perceived barriers with pain catastrophe (PCS ($F(1,56)=8.62$, $p=0.004$)

and kinesiophobia (TSK ($F(1,56)=14.82, p<0.001$)). Non-normalized extensor strength was greater in the rLBI group ($F(1,56)=6.82$, $p<0.001$) but this difference was not significant when strength was normalized to body mass (Table 6.1).

Table 6.1: Demographics, anthropometrics, pain characteristics, self reported physical activity, occupational loading and trunk strength

Reflex Group (n)	FRL (30)		SRL (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Age (years)	35.3 (8.6)	39.6 (9.3)	34.5 (10.8)	38.1 (10.8)
Mass (kg)	79.9 (12.0)*	86.0 (10.0)	80.6(11.9)*	92.3 (17.8)
Height (m)	174.9 (8.3)	181.0 (6.9)	175.6 (8.0)	178.8 (7.1)
BMI (kg/m^2)	26.1 (3.8)	26.4 (3.8)	26.1 (3.2)	28.7 (4.3)
Heavy Job (n (%))	4 (27%)	5 (33%)	4 (27%)	5 (33%)
L. Hand (n (%))	3 (20%)	2 (13%)	3 (20 %)	1 (7%)
VAS (mm)	0.8 (1.8)*	3.7 (6.0)	0.3 (1.3)*	8.5 (9.8)
PCS	6.9 (8.1)*	13.5 (8.0)	7.3 (6.6)*	14.3 (12.1)
TSK	30.7 (7.6)*	37.9 (5.4)	29.1 (6.3)*	34.9 (6.6)
Aerobic Training (/ week)	3.6 (1.5)	3.4 (2.5)	5.2 (3.1)	3.2 (3.5)
Strength Training (/ week)	2.6 (2.0)	1.7 (2.0)	2.2 (2.0)	3.3 (3.0)
Core Training (/ week)	2.1 (2.0)	1.9 (1.6)	2.1 (1.8)	2.5 (2.5)
RMD	0.3 (0.8)*	2.2 (2.1)	0.0 (0.0)*	2.4 (3.0)
Flexor Mo (Nm)	130.0 (30.2)	132.8 (40.9)	123.0 (41.9)	150.2 (33.5)
Extensor Mo (Nm)	199.7 (47.9)*	219.8 (47.0)	192.0 (41.9)*	237.7 (56.6)
Norm Flexor Mo (Nm/kg)	1.7 (0.5)	1.6 (0.5)	1.5 (0.3)	1.6 (0.3)
Norm Ext. Mo (Nm/kg)	2.5 (0.5)	2.6 (0.6)	2.4 (0.5)	2.6 (0.6)

Significant differences ($p<0.05$) represented by * to indicate the difference between the ASYM and rLBI group. Abbreviations: Fast (FRL) and slow reflex latency (SRL), asymptomatic (ASYM), recovered low back injury (rLBI), body mass index (BMI), left handed (L. Hand) visual analog scale (VAS), pain catastrophizing scale (PCS), tampa scale of kinesiophobia (TSK), roland morris disability score (RMD), moment (Mo), normalized (Norm) and extensor (Ext.).

6.3.2 Reflex Testing Performance Outcomes

Results of the reflex testing showing onset main effects or onset by group interactions are in Table 6.2, with a description of the performance of individual muscle sites shown in Table 6.3-4. Considering muscle onset for both the abdominals and the back extensors, a group by onset interaction ($F(1,616)>7.55$, $p<0.006$) captured the SRL

rLBI subgroup had the longest reflex latency compared to all other subgroups, and the SRL ASYM subgroup had longer reflex latencies than both FRL subgroups (Table 6.2). For both the trunk flexors (Table 6.3) and back extensors (Table 6.4), muscle main effects captured differences in the relative onset time between trunk muscle sites. For the abdominals, the internal obliques and lateral fibers of the external obliques were slower than other abdominal sites (Table 6.3). For the back extensors, the superficial quadratus lumborum (L48) were slower than other back extensor sites (Table 6.4).

Considering the amplitude of reflexes, a group by onset interaction ($F(1,616)=51.95$, $p<0.001$) in the abdominals captured in the rLBI the SRL subgroup had lower reflex amplitudes than FRL subgroup, whereas an opposite effect (FRL>SRL) was found in the ASYM group (Table 6.2). In the back extensors, a group by onset interaction ($F(1,616)=4.57$, $p=0.033$) captured in both groups (ASYM and rLBI) the SRL group had lower reflex amplitudes than FRL group (Table 6.2). Muscle main effects captured differences in the relative activation amplitude of reflex responses. In the abdominals the main effect ($F(11,616)=3.75$, $p<0.001$) captured REO2 had higher activation than REO1 (Table 6.3). In the back extensors the muscle effect ($F(11,616)=5.10$, $p<0.001$) captured lower reflex amplitudes in the inferior iliocostalis sites (L36) compared to the multifidus (L52) sites (Table 6.4).

For the number of trials with a detectable reflex response an onset main effect in both the abdominals and back extensors ($F(1,616)>13.88$,$p<0.001$) found there was an increased likelihood that no reflex would be detected 150ms following the perturbation in the SRL group compared to the FRL group (Table 6.2). Muscle main effects captured differences in responsiveness between muscle sites. In the abdominals, the muscle main effect ($F(11,616)=12.85$, $p<0.001$) captured IO were consistently less likely to have a reflex response to the perturbation compared to other abdominal sites (Table 6.3). In the back extensors the muscle main effect ($F(11,616)=7.27$, $p<0.001$) captured LL48 were less responsive than LL33 (Table 6.4).

Table 6.2: Onset main effects and group by onset interactions for reflex measures

Onset Group (n)	FRL (30)		SRL (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Abs Onset (ms)	52.7‖¶	52.7‖¶	80.5‖	94.7
	±25.0	±30.4	±26.7	±34.5
Back Onset	47.1‖¶	51.8‖¶	77.9‖	89.9
(ms)	±24.7	±19.0	±31.1	±40.6
Abs Amplitude	11.7‖¶	16.1‖¶	16.0	12.3
(%MVIC)	±7.8	±10.2	±11.0	±7.8
Back Amplitude	17.8‖¶	17.0‖¶	13.1	14.4
(%MVIC)	±12.9	±10.9	±10.5	±10.5
Abs Responsive	91.0		85.5†	
(% Detectable)	±20.8		±26.8	
Back Response	94.1		86.4†	
(% Detectable)	±18.1		±26.2	

If no main effect or interaction is identified the combined population mean is indicated in the center of the table. Significant differences ($p<0.05$) for reflex main effects are indicated by a † to show differences between the fast (FRL) and slow reflex latency (SRL) groups. Significant group by reflex interactions ($p<0.05$) are indicated by a specific symbol to show differences relative to the: SRL rLBI (‖), and SLR ASYM (¶) subgroup. Abbreviations: Maximum voluntary isometric contraction (%MVIC), abdominals (Abs) and back extensors (Back).

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Table 6.3: Muscle main effects and interactions for reflex outcome measures for abdominal sites

	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO
Abs Onset (ms)	68.5[f] ±38.2	67.1[f] ±34.1	63.6[ef] ±32.6	61.5[ef] ±33.1	66.5[f] ±31.4	73.0 ±34.7	63.8[ef] ±28.9	66.2[f] ±34.8	77.3 ±35.6	75.3 ±30.0	82.0 ±36.2	76.7 ±39.7
Abs Amp (%MVIC)	14.5 ±10.1	14.8 ±9.6	13.8 ±8.3	12.7 ±8.3	10.9 ±8.4	13.9 ±10.1	16.3[c] ±9.7	14.3 ±9.5	12.5 ±7.5	12.5 ±8.9	15.8 ±10.4	16.9 ±11.2
Abs Response (%Detect)	89.8[f] ±19.7	91.2[f] ±21.2	96.8[f] ±10.3	94.6[f] ±14.7	94.4[f] ±13.8	88.6[f] ±21.9	96.8[f] ±14.7	93.4[f] ±18.3	88.8[f] ±25.9	85.3[f] ±26.0	72.3 ±32.7	67.9 ±37.6

All values are mean ± standard deviation. If only a muscle main effect exists, the combined average for each site is shown. Significant differences ($p<0.05$) amongst ipsilateral muscle sites are indicated by superscript letters to show differences between noted muscle and: a) LRA, b) URA, c) EO1, d) EO2, e) EO3 and f) IO. Abbreviations: reflex amplitudes (Amp), normalized to maximum voluntary isometric contraction (%MVIC) and abdominals (Abs).

Table 6.4: Muscle main effects and interactions for back extensor

	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52
Back Onset (ms)	60.8^{bde} ±33.8	54.3^{def} ±27.9	**76.0 ±35.8**	**58.1^{f} ±31.1**	58.1^{bde} ±31.1	64.4 ±33.2	72.2 ±42.0	69.3 ±34.8	76.8 ±40.7	74.4 ±35.3	63.6^{b} ±30.7	65.4 ±28.9
Back Amp (%MVIC)	15.6 ±11.8	17.9 ±12.3	16.5 ±11.0	16.7 ±12.7	15.1 ±9.7	14.3 ±10.6	12.8^{f} ±10.4	11.9^{acf} ±8.9	15.6 ±13.4	14.0^{f} ±10.4	18.3 ±11.7	18.5 ±12.2
Back Response (%Detect)	92.0 ±22.8	92.7 ±23.0	92.2 ±24.1	93.7 ±19.7	93.3 ±13.6	97.3 ±10.6	90.0 ±22.3	94.0 ±15.3	87.3 ±25.5	85.4^{d} ±27.9	92.0 ±21.9	93.0 ±20.1

All values are mean ± standard deviation. If only a muscle main effect the grand mean for each muscle site is shown. Significant differences ($p<0.05$) between muscle sites are indicated by **bold** lettering to show an asymmetry between left and right sites, and muscle site differences amongst ipsilateral sites are indicated by superscript letters showing a difference relative to: a) L13 b) L16, c) L33, d) L36, e) L48 and f) L52. Abbreviations: reflex amplitudes (Amp), normalized to maximum voluntary isometric contraction (%MVIC), and back extensors (Back)

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6.3.3 Horizontal Transfer Task, Timing, Motion and External Moments

Time to complete the task, trunk and pelvis motion, and peak external moments for the horizontal transfer task are shown in Table 6.5. There were no group or reflex main effects or interactions ($p>0.05$) supporting that the controlled task parameters of timing (approximately 4 seconds), minimizing trunk and pelvis motion and task demand of the external moment was consistent among groups.

Table 6.5: Horizontal transfer task performance data, timing, motion, and peak external moments

Onset Group (n)	FRL (30)		SRL (30)		Combined (60)
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)	
Time (s)	4.1 (0.3)	4.3 (0.3)	4.3 (0.3)	4.2 (0.3)	4.3 (0.3)
Torso Flex/Ext (°)	3.0 (1.4)	4.5 (2.6)	4.3 (1.8)	4.5 (2.1)	4.1 (2.0)
Torso Lat. Flex (°)	2.9 (0.9)	2.2 (1.3)	3.0 (1.4)	2.6 (1.3)	2.7 (1.2)
Torso Ax Rot (°)	4.1 (2.0)	5.4 (2.7)	5.9 (1.8)	4.3 (2.0)	4.9 (2.2)
Pelvis Flex/Ext (°)	1.6 (1.2)	1.1 (0.9)	1.6 (0.9)	1.8 (0.9)	1.5 (1.0)
Pelvis Lat. Flex (°)	1.1 (0.8)	1.3 (1.0)	1.5 (0.9)	1.5 (0.8)	1.4 (0.9)
Pelvis Ax Rot (°)	2.7 (1.7)	2.3 (1.0)	2.2 (0.8)	2.4 (1.0)	2.4 (1.2)
Peak Flexion (Nm)	26.6 (3.2)	28.6 (2.7)	27.7 (3.6)	29.2 (4.4)	28.0 (3.6)
Peak Lateral Flexion (Nm)	11.8 (2.6)	12.1 (2.0)	11.7 (2.3)	11.7 (2.9)	11.8 (2.4)
Norm Peak Flex (Nm/kg)	0.34 (0.03)	0.33 (0.03)	0.35 (0.03)	0.32 (0.02)	0.33 (0.03)
Norm Peak Lat. Flex (Nm/kg)	0.15 (0.04)	0.14 (0.03)	0.15 (0.03)	0.13 (0.03)	0.14 (0.04)

No significant group or onset main effects or interactions ($p>0.05$). Abbreviations: Fast (FRL) and slow reflex latency (SRL), flexion (Flex), extension (Ext), lateral (Lat.), axial (Ax), rotation (Rot), normalized (Norm), asymptomatic (ASYM), recovered low back injured (rLBI).

6.3.4 Trunk Muscle Activation Patterns

As the primary objective of this study was to explore differences between the FRL and SRL groups any onset main effects and interactions are first presented along with any group or muscle main effects to fully describe the data. Visual representation of the ensemble average calculated across select oblique muscles are provided in Figure 6.3A. For the back extensors, the ensemble average muscle activation patterns for the medial left and right combined back extensor sites, along with the left and right back extensor sites are shown in Figure 6.3B-D respectively. Data were presented to display group by onset interactions.

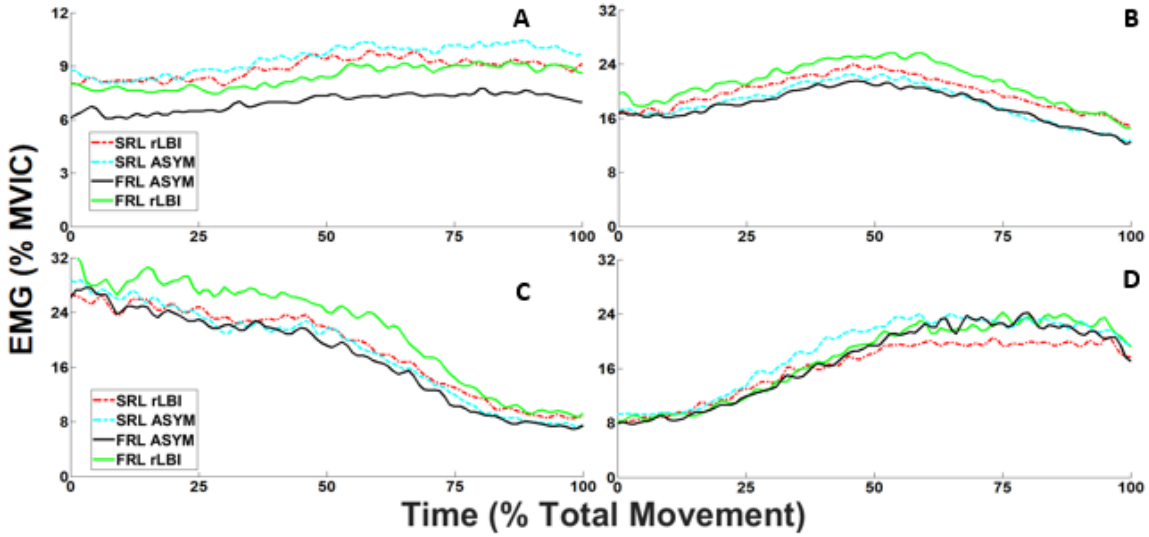

Figure 6.3: Normalized to maximum voluntary isometric contraction (%MVIC) ensemble average trunk electromyograms for recovered low back injury (rLBI) and asymptomatic (ASYM) slow (SRL) and fast reflex latency (FRL) subgroups. For the abdominals group by onset interactions are depicted by a) the ensemble average of left anterior external (LEO1) and internal obliques (LIO) and the right middle (REO2) and lateral external oblique sites (REO3). For the back extensors group by onset interactions are depicted by b) the ensemble average of both the right and left medial back extensor sites (L13, L33, & L52) and the c) left and d) right lateral back extensor sites (L16 & L36).

6.3.4.1 Abdominal Muscle Activation Patterns

Two PC's explained 96.5% of the total abdominal muscle activation waveform variance. For the abdominals and back extensors, PC1 captured the overall shape and

amplitude of abdominal muscles (Figure 6.4A) with high scores capturing higher activation amplitudes (Figure 6.4B). PC1 scores were highly correlated (r=1) with the average amplitude of muscle sites across the entire task so the average activation amplitudes in %MVIC are also provided to give context to the data whereas PCs are a unitless score (Table 6.6 and 6.7). There was a significant group by onset interaction ($F(1,616)=11.43$, $p<0.001$) capturing the SRL ASYM subgroup had higher activity than both FRL subgroups (Table 6.6). Both the SRL and FRL rLBI subgroups had higher activation amplitudes than the FRL ASYM subgroup (Table 6.6 & Figure 6.3A). In addition, a muscle main effect ($F(11,616)=42.74$, $p<0.001$) captured differences in the activation amplitudes between muscle sites showing IO sites had the highest activation, followed by EO sites and finally the RA sites (Table 6.7).

For the abdominals and back extensors, PC2 captured changes in muscle activation in response to the lateral flexion moment (Figure 6.4C). High scores corresponded with muscle sites having higher initial and lower terminal activation, whereas low (negative) scores corresponded to the opposite with lower initial activation (Figure 6.4D). For this feature, a group by muscle interaction ($F(11,616)=2.45$,$p=0.005$) found that the ASYM group had higher responsiveness of the right EO1 compared to the rLBI group (Table 6.6) with higher initial activation and a large drop off. This interaction also captured differences in the synergistic relationship between muscle sites where both groups had asymmetries in the same three sites EO1, EO3 and IO. Finally, the ASYM group had more differences among ipsilateral muscle sites than the rLBI which only had differences between IO and EO3 (Table 6.6).

Given PC2 captured both the directionality (positive or negative Table 6.6) and magnitude (coefficient) of the responsiveness to the lateral flexion moment generated by this task, data were transformed to absolute values to explore whether the absolute magnitude of responsiveness regardless of direction could capture differences between stiffness groups (Quirk & Hubley-Kozey, 2018). Following this transformation, there was no onset main effect or interaction ($p>0.05$) (Table 6.6 & Figure 6.3A).

Table 6.6: Onset main effects and group by onset interactions for trunk muscle activation patterns

Onset Group (n)	FRL (30)		SRL (30)	
Group (n)	ASYM (15)	rLBI (15)	ASYM (15)	rLBI (15)
Abs AVG	5.8	6.5	7.7	7.1
(% MVIC)	±5.0	±5.2	±5.7	±5.5
Abs PC1	58.5‖¶*	65.8¶	77.5	70.8
(Unitless)	±50.0	±51.8	±57.3	±55.7
Abs |PC2|		5.5		
(Unitless)		±9.3		
Back AVG	17.2	20.0	17.7	18.6
(% MVIC)	±8.4	±8.7	±7.6	±10.9
Back PC1	173.3*	202.4	178.9*	187.5
(Unitless)	±85.6	±85.6	±77.4	±110.6
Back |PC2|	43.5*‖	47.6	42.0‖*	37.4*
(unitless)	±30.3	±27.0	±29.0	±29.2

If no main effect or interaction is identified the combined population mean is indicated in the center of the table. Comparisons for PC2 are performed on the absolute value |PC2| scores. Significant group by reflex interactions ($p<0.05$) are indicated by specific symbol to show a difference relative to the: (‖) SLR rLBI, (*) FRL rLBI and (¶) SLR ASYM subgroup. Abbreviations: Fast (FRL) and slow reflex latency (SRL), asymptomatic (ASYM), recovered low back injury (rLBI), maximum voluntary isometric contraction (% MVIC), average activation amplitudes (AVG), principal component (PC), abdominals (Abs), and back extensors (Back).

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Table 6.7: Muscle main effects and interactions for abdominal sites

	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO
AVG	3.7	3.7	3.8	3.3	6.9	7.8	6.8	6.9	7.5	8.2	11.1	11.6
(%MVIC)	±2.6	±2.4	±3.2	±2.7	±4.7	±6.3	±4.4	±5.5	±4.8	±4.9	±6.0	±6.4
PC1	36.8^{cdef}	37.1^{cdef}	38.5^{cdef}	33.5^{cdef}	69.6^{f}	78.0^{f}	67.9^{f}	69.8^{f}	75.7^{f}	82.2^{f}	111.4	117.1
	±25.9	±24.3	±31.7	±26.9	±47.5	±63.8	±44.1	±55.2	±48.6	±48.8	±60.1	±65.1
PC2	0.1^{c}	0.0^{ce}	0.4^{c}	-1.0^{ce}	**10.3***	**-13.4**	-2.1^{c}	4.9^{cef}	**-5.7^{cf}**	**14.7^{cf}**	**3.1**	**-4.0**
ASYM	±0.6	±0.7	±1.3	±1.4	**±15.5**	**±16.4**	±4.9	±12.1	**±11.8**	**±18.1**	**±7.7**	**±7.0**
PC2	-0.1	-0.5^{e}	-0.1	-0.7^{e}	**1.6***	**-6.5**	-2.8	0.7^{ef}	**-6.4^{f}**	**10.9^{cf}**	**3.5**	**-6.7**
rLBI	±1.1	±1.4	±2.0	±1.8	**±6.2**	**±17.0**	±4.7	±6.4	**±9.0**	**±11.4**	**±8.5**	**±11.2**

All values are mean± standard deviation and unitless unless otherwise indicated. If only a muscle main effect exist the grand mean for each muscle site is shown. For all interactions the respective group (rLBI vs ASYM) is indicated by the row title. Significant differences (<0.05) between muscle sites are indicated by **bold** lettering to show a asymmetry between left and right sites. Differences amongst ipsilateral muscle sites are indicated by superscript letters indicating a difference between the indicated muscle site and: a) LRA, b) URA, c) EO1, d) EO2, e) EO3, and f) IO. Significant group by muscle interactions ($p<0.05$) are shown by * to indicate differences between the LBI and ASYM group within a specific muscle site. Abbreviations: average activation amplitudes (AVG), maximum voluntary isometric contractions (% MVIC), principal component (PC), asymptomatic (ASYM), recovered low back injured (rLBI).

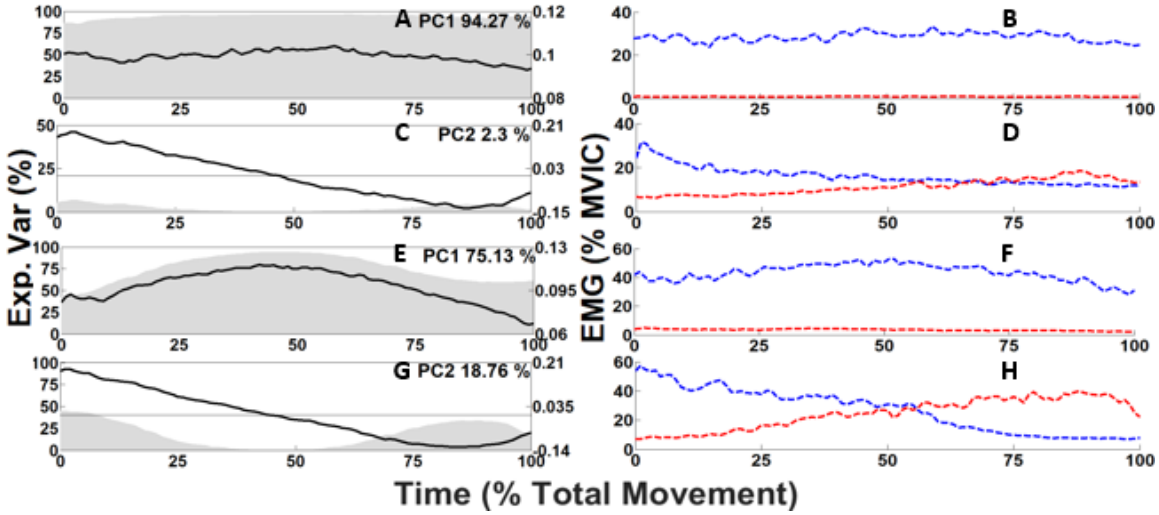

Figure 6.4: Visual representation of abdominal (A,C) and back extensor (E,G) principal component (PC) waveforms (black line) and ensemble average representations of high (blue) and low (red) electromyogram waveforms corresponding to each PC waveform (right) (B,D,F,H). For PC waveform plots grey shading depicts the time-varying explained variance with the collective total displayed on the top right of the sub-plot.

6.3.4.2 Back Extensor Muscle Activation Patterns

Two PC's explained 93.9% of the total waveform variance for the back extensor muscle activation patterns. For PC1 there was a group by onset interaction ($F(1,616)=28.89$, $p<0.001$) where the FRL rLBI subgroup had higher activation amplitudes than both ASYM subgroups, but not the SRL rLBI subgroup (Table 6.6 & Figure 6.3B&C). Additional differences in muscle activation amplitudes were captured by a muscle main effect ($F(11,616)=13.89$, $p=0.013$) showing the superficial multifidus (L52) had higher activation amplitudes than other back sites.

For PC2 a muscle main effect ($F(11,616)=193.2$, $p<0.001$) captured differences in the synergistic relationship amongst back extensor muscle sites (Table 6.8) showing while all back extensors were responsive to the changing lateral flexion moment resulting in asymmetries between left (Figure 6.3C) and right sites (Figure 6.3D), in terms of the magnitude of responsiveness superior and lateral back extensors sites were the most responsive to lateral flexion moment (Table 6.8). Transforming PC2 scores to absolute values to capture general responsiveness found a group by onset interaction ($F(1,616)=17.4$, $p<0.001$) that captured the FRL rLBI subgroup were more responsive (higher scores) to the changing lateral flexor moment than the SRL rLBI subgroup (Table

6.6 & Figure 6.3C). Whereas, in the ASYM the FRL and SRL were not different to one another.

6.4 Discussion

This study explored the spinal system compensation theory to determine whether lower neural spinal system function, as measured by reflex delays, would be compensated by change in trunk muscles activations during a dynamic task. Secondly, we examined whether the presence of a recent low back injury modified that relationship. Fundamental to this comparison was that all demographic, strength and task performance variables were not different between the SRL and FRL groups. Furthermore, while demographics did differ between the rLBI and ASYM groups all reflex and muscle activation patterns were analyzed with both mass and trunk flexor or extensor strength as a co-variate to find no significant main effects or interactions were modified when controlling for these differences.

Despite no differences in task performance, there were differences in reflex testing outcomes (onset time, reflex amplitude, and reflex responsiveness), and muscle activation patterns between the SRL and FRL groups. For muscle activation patterns, this study found onset main effects or interactions for 3/4 muscle activation patterns analyzed. However, these differences were dependent on whether the participant was in the ASYM or rLBI group, suggesting these subgroups had unique adaptation strategies. This discussion will focus on these muscle activation patterns but will also elaborate on some novel features that were revealed through our comprehensive analysis of reflex outcome measures presented in section 6.4.3.

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Table 6.8: Muscle main effects and interactions for back extensor

	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52
AVG	17.5	20.4	17.2	20.0	17.3	18.3	14.8	17.0	17.5	17.8	21.0	21.6
(%MVIC)	±7.7	±9.2	±7.0	±8.7	±8.2	±9.1	±8.7	±9.1	±10.0	±9.9	±8.5	±9.5
PC1	174.8[f]	209.5	170.5 [f]	204.8	173.3[f]	187.1	147.3[f]	172.9	175.2[f]	179.6	211.6	219.8
	±77.5	±94.1	±69.7	±89.1	±82.3	±92.5	±87.3	±92.6	±100.2	±99.8	±85.9	±96.1
PC2	**-57.3**	**52.1**	**-56.8**	**63.6**	**-47.5**	**37.2[a]**	**-41.3[a]**	**42.8[a]**	**-31.0[ab]**	**26.2[abd]**	**-34.7[abc]**	**13.1[abcd]**
	±29.1	**±32.7**	**±29.6**	**±33.4**	**±27.7**	**±24.6**	**±23.8**	**±29.5**	**±19.3**	**±19.2**	**±22.7**	**±25.8**

All values are mean ± standard deviation and unitless unless otherwise indicated. If only a muscle main effect exist, the grand mean for each muscle site is shown. Significant differences ($p<0.05$) between muscle sites are indicated by **bold** lettering to show an asymmetry between left and right sites, and between muscle site differences amongst ipsilateral sites are depicted by superscript letters indicating a difference between the muscle site and: a) L13 b) L16, c) L33, d) L36, e) L48, and f) L52. Abbreviations: average activation amplitudes (AVG), maximum voluntary isometric contractions (% MVIC), principal component (PC), asymptomatic (ASYM), recovered low back injured (rLBI).

6.4.1 Influence of onset on dynamic muscle activation patterns

Fundamental to the studies objective, our results show that separating participants into two groups (SRL and FRL) based on muscle onsets, did capture differences in muscle activation patterns during the performance of a highly controlled transfer task. Consistent with our hypothesis was the SRL had higher activation amplitudes of abdominal muscle sites and evidence of reduced responsiveness in the back extensor muscles to the changing lateral flexion moment compared to the FRL (Table 6.6). However, these findings were captured within group by onset interactions and will be discussed below.

6.4.2 Influence of recovery from LBP

The second purpose of this study was to determine whether the effects of onset latency on muscle activation patterns were modified by the recent experience of LBP. In the back extensors, two group by onset interactions were found for the overall activation amplitudes and the responsiveness to the lateral flexion moment, and in the abdominals, a group by onset interaction was captured for overall activation amplitudes. These interactions captured each group had a unique way to adapt muscle activation patterns to delayed muscle onsets.

Results from this study found rLBI in the SRL subgroup adapted back extensor muscle activation patterns by reducing lateral flexor responsiveness (PC2) compared to the fast group (Table 6.6). This muscle activation pattern suggests greater antagonist co-activation between left and right back extensor sites as they balance moments in the frontal plane (Arjmand & Shirazi-Adl, 2006; Dumas et al., 1991; Jorgensen et al., 2001). These data are consistent with our initial hypothesis that the muscle activation of the back extensors would change towards reduced responsiveness. This activation pattern has been identified in populations suspected to have deficits within the neural system of the lower back and has been interpreted to capture bracing or increased co-activation in the frontal plane to increase active spinal stiffness (Quirk & Hubley-Kozey, 2018; Trudel, 2014), to be discussed.

Of interest was the finding that participants in the ASYM group did not adapt to delayed onset in a similar way as the rLBI group. In the ASYM, SRL subgroup had higher abdominal activation amplitudes than the FRL (Table 6.6). Similar to the finding in the rLBI group, this difference suggests those in the SRL move towards a co-activation pattern, however in the ASYM group this was balanced in the sagittal plane. While our data does capture higher agonist activation amplitudes and can be interpreted as capturing higher co-activation, true co-activation represents the relative ratio of antagonist to agonist force (Thelen, Schultz, & Ashton-Miller, 1995), a feature which is not captured when comparing MVIC normalized activation amplitudes between muscle sites as they likely do not have comparable muscle cross sectional area, gain (activation-force conversion factor) and effective moment arm length which would have to be corrected for to understand the relative ratio of antagonist to agonist forces (Le et al., 2017).

Higher co-activation in either the sagittal or frontal plane could be interpreted as an adaptation for delays in muscle reflexes. Increased baseline activation of muscles surrounding a joint, increases the intrinsic or baseline stiffness of that joint (Lee et al., 2005). While intrinsic stiffness alone is unlikely to prevent joint motion in the event of a large magnitude sudden load (Brown & McGill, 2008; Moorhouse & Granata, 2006; van Drunen et al., 2013), increased stiffness through antagonist co-activation has been shown effective in minimizing displacement following a perturbation of a set external force (Shahvarpour et al., 2014; Vera-Garcia et al., 2006b) and is shown both computationally (Stroeve, 1996) and experimentally (Fu & Hui-Chan, 2007) to be the most likely mechanism to adapt to impaired feedback control. An interesting aspect of this study was these adaptation patterns were observed in this highly controlled lifting task where changes in external forces remained constant and predictable throughout the task. This suggests that despite the low likelihood that the participants would be exposed to sudden loading for this task, participants in the SRL settled on muscle activation patterns that would provide higher active spinal stiffness as a "margin of safety" by mitigating spinal displacement to unexpected sudden loading, despite the cost associated with increased muscle co-activation (Granata & Marras, 2000; van Dieën et al., 2017; Cholewicki & McGill, 1996).

Despite these findings being novel, a limitation of the current study is that only a single measure was used to characterize neural function. The rationale for choosing a spinal mediated stretch reflex to measure neural control was based on Panjabi's model which suggested that deficits in the neural system represent "corrupted feedback control" (Panjabi, 1992a; 1992b; 2003). Much of his focus was on sub-cortical structures, sensory organelles, reflex speed etc., where a variety of methods are used to measure deficits in the neural system including appraisal of proprioception via vibration (Brumagne, Cordo, & Verschueren, 2004; Brumagne, Janssens, Knapen, Claeys, & Suuden-Johanson, 2008; Claeys, Brumagne, Dankaerts, Kiers, & Janssens, 2010; Claeys et al., 2014), joint position repositioning sense (Georgy, 2011; Osthoff et al., 2015; Tong, Mousavi, Kiers, & Ferreira, 2015), and motion detection threshold (Silfies, Cholewicki, Reeves, & Greene, 2007). The latter two were considered less reliable than the stretch reflex utilized by the current study as they required either: i) a heavy cognitive component to remember the initial position to which one must reposition (Han, Waddington, Adams, & Anson, 2015), or ii) an indirect measure of sub-cortical neural control as the stimulus would have to be detected, consciously recognized and then acted upon by executing a button press (Verschueren, Brumagne, Swinnen, & Cordo, 2002). The muscle stretch reflex utilized in this study circumvents these confounders as the measure would assess the speed at which the spinal cord can excite motor units at the joint of interest. Of course, the neural system is highly complex with the capacity to modify muscle activation with a variety of structures. Evidence suggests that with low back pain, cortical structures also change altering motor (Pelletier, Higgins, & Bourbonnais, 2015; Tsao, Galea, & Hodges, 2010) and sensory cortex organization (Flor, Braun, Elbert, & Birbaumer, 1997; Hotz-Boendermaker, Marcar, Meier, Boendermaker, & Humphreys, 2016; Schabrun, Burns, & Hodges, 2015). These changes in the central nervous system could characterize deficits within the neural system leading to low back injuries. Excluding this central component suggest there is still future areas for study to confirm whether similar adaptations would be observed in trunk muscle activation patterns with measured differences within different neural structures.

A final limitation of this study is that our definition of a spinal deficit in the neural system was based on participants having below median trunk muscle onset times. While

lower function as measured by slower reflex is a potential risk for experiencing future low back pain, the threshold used in this study does not relate to a clinical threshold of deficient function. However, important to note is that despite lower function not being related to a deficit, slower trunk reflex latency between groups did capture muscle activation pattern differences, providing novel information to support Panjabi's compensation theory.

6.4.3 Difference in onset groups on measures of reflexes

To our knowledge, this is the first study to compare the reflex response between participants recently recovered from an LBI to asymptomatic controls. The results of this study show interactions between participant groups and those classified in the SRL and FRL subgroup for both muscle onset and reflex amplitude.

The onset of agonist muscles reported in this study were consistent with the average onset latency of abdominals (40-90ms) (Cholewicki et al., 2005; Lee et al., 2007a; Radebold et al., 2000; 2001; Vera-Garcia et al., 2006b) and back extensors (40-100ms) (Cholewicki et al., 2005; Larivière et al., 2010; Lee et al., 2007a; Miller, Slota, Agnew, & Madigan, 2010; Radebold et al., 2000; 2001; Santos et al., 2011; Shahvarpour et al., 2014; Shojaei et al., 2016; Sánchez-Zuriaga et al., 2010). Considerable range in onset times likely reflects differences in the protocols used. This is particularly true for studies that use rapid loading or unloading paradigms where participants can increase levels of pre-perturbation intrinsic stiffness leading to reductions in post perturbation velocity, motion (Vera-Garcia et al., 2006b) and delay reflex onsets (Larivière et al., 2010; Miller et al., 2010). As this study was velocity controlled, this feature may have had less of effect on reflex outcome measures. Considering our results to those of Dolan and Colleagues who used a similar a perturbation design (100°/s from 20 to 40° of flexion), show our muscle onset times (Table 6.4) were comparable to visually detected onsets from the L36 muscle site (62±13ms) (Sánchez-Zuriaga et al., 2010).

The findings of this study did not show group differences in muscle onset for the abdominals or back extensors as these main effects were superseded by the group interactions. However, for flexion and extension perturbations our results did capture a significant group main effect ($p<0.001$) showing the rLBI group had longer muscle onset

times compared to the ASYM group (Table 6.2). This finding is consistent with prospective (Cholewicki et al., 2005) and cross-sectional (Liebetrau et al., 2013; Radebold et al., 2000; 2001; Reeves et al., 2004), for review see (Knox et al., 2018; Prins et al., 2017) data suggesting those who have or will experience LBP have slower reflexes compared to asymptomatic controls. Unique to this study was the finding that these group differences were attributed to participants in the SRL group (Table 6.2). Our group by onset interaction for both the abdominals and back extensors found the SRL rLBI subgroup had the slowest reflexes, whereas no group differences were identified between the ASYM and rLBI in the FRL group (Table 6.2).

Reflex amplitudes for the back extensors, were higher (Table 6.4) than those reported by Santos et al. of 6±2% MVIC as was the velocity of the perturbation delivered by this study (120˚/s) versus that of Santos et al, (35±6°/s) (Santos et al., 2011). Thus high amplitudes in this study would be expected given a positive correlation between muscle reflex amplitude and stretch velocity (Bedingham & Tatton, 1984; Nakazawa, Yamamoto, Ohtsuki, Yano, & Fukunaga, 2001; Obata et al., 2009; 2011). While the angular velocity of other studies were not reported, our amplitudes (Table 6.4) were within the amplitude reported at the L52 (17-25% MVIC) (Granata, Slota, & Bennett, 2004), and the peak reflex activity measured at a variety of back extensors (18-30% MVIC) measured 0-200ms following a perturbation (Shahvarpour et al., 2014). This study found for the back extensors the SRL had lower reflex amplitudes than the FRL group. These results are consistent with a combination of computational modeling (Franklin & Granata, 2006), and experimental studies (Liebetrau et al., 2013; Mühlbeier, Puta, Boström, & Wagner, 2017) that show as reflexes become delayed the relative amplitude of that reflex decreases as a greater response could result in a spinal instability event (Franklin & Granata, 2006; Liebetrau et al., 2013).

Few studies report typical reflex amplitudes for the abdominals. Vera-Garcia and colleagues using a posterior rapid loading paradigm captured peak reflex amplitudes 0-250ms following a perturbation of 25-40% MVIC (Vera-Garcia et al., 2006b) a result higher than the amplitudes reported by the current study (Table 6.3). However, such differences could be explained by how reflex amplitude was measured. In the current study, reflex amplitudes were averaged over 30ms rather than using a peak (Vera-Garcia

et al., 2006b). Second, reflex amplitudes occurring 0-30ms following muscle onset (short latency reflex) are often smaller than longer latency reflex components (Figure 6.2) that would have been captured in Vera-Garcia's longer window (Obata et al., 2009; 2011). Comparing between our SRL and FRL groups violated the theory that slower reflexes should have lower reflex amplitudes. While this finding was consistent in our rLBI subgroups the opposite relationship was found in our ASYM group (Table 6.2). Deviation in the ASYM group may be explained that the strength of this relationship in other experimental studies has been reported to be weak (R^2=0.088) (Mühlbeier et al., 2017) with some muscle sites having a non-significant relationship (Liebetrau et al., 2013).

Considering responsiveness, the likelihood of measuring a detectable reflex was consistent with previously reported ranges of 80-100%. Additionally, our results were similar to trends reported by Vera-Garcia et al. showing that the internal obliques and more lateral back extensor sites were less likely to have a measurable reflex response (Table 6.3 and 6.4) (Vera-Garcia et al., 2006b). Unique to the current study was that muscles are less likely to have a "responsive" reflex if participants are in the SRL than the FRL (Table 6.2). This is not surprising as when reflexes become progressively slower it is more likely that they could be defined as being non-responsive occurring beyond the 150ms window used by the study to ensure the captured reflex response was mediated by sub-cortical structures (Radebold et al., 2000; Stokes et al., 2000). No group differences were identified which is not consistent with previous studies that found a reduced likelihood of measuring a reflex response in participants with chronic LBP (Radebold et al., 2000; Stokes et al., 2005). However, differences with the previous work on participants with chronic LBP may have been confounded by the use of a rapid loading or unloading paradigm which as previously mentioned can be confounded by increased muscle activation (intrinsic stiffness) prior to the perturbation (Larivière et al., 2014; Lee et al., 2007a; Stokes et al., 2000).

6.5 CONCLUSION

Individuals classified as having slower reflexes had different muscle activation patterns to complete a controlled dynamic transfer task, supporting the spinal system

compensation theory. Not only did muscle activation patterns differ between FRL and SRL, but these features had an interaction based on whether the participant recently recovered from a LBI or not. In ASYM group, those with SRL had higher abdominal antagonist activation amplitudes, whereas, in the rLBI those in the SRL had reduced responsiveness, capturing increased co-activation of back extensor muscles in response to changing flexion moments in the frontal plane. Overall both features potentially lead to higher baseline intrinsic stiffness of the trunk suggesting this may act as an adaptation to a delay in rapidly adjusting muscle activation to restore spinal stability in the event of unexpected loading. This thorough investigation of reflex response also found that the rLBI group had slower reflexes than the ASYM group, but did not differ for other properties such as reflex amplitude or percent of responsive muscles. Investigating between SRL and FRL revealed not only did SRL have longer reflex latencies but also reduced the likelihood of having a responsive reflex. Regarding reflex amplitudes, this study revealed for most subgroup comparisons reflex amplitudes were smaller in those placed in the SRL, with one exception in the ASYM group which warrants further investigation regarding the robustness of this relationship.

CHAPTER 7 CONCLUSION

7.1 CONCLUSION AND SUMMARY

The overarching purpose of this dissertation was to examine the interaction between deficits within the three spinal systems and trunk muscle activation patterns during a dynamic task to probe Panjabi's spinal system compensation theory. Prior to this dissertation, Panjabi's theory was supported with limited empirical evidence. Around the spine, most of what was known regarding muscle activation pattern adaptations came from cross-sectional studies comparing populations with suspected spinal system deficits to a reference control group. These cross-sectional comparisons have confounders that could further explain the differences observed in muscle activation patterns. To build on these cross-sectional studies, this dissertation simultaneously compared populations with suspected deficits to a reference control. Though different in magnitude, common muscle activation pattern adaptations could be observed in the deficit groups compared to controls with fewer suspected deficits. The conclusions that could be inferred from this study while novel, were limited in that they could not determine whether these changes were specific to a deficit within one spinal system. To further strengthen our understanding of muscle activation pattern adaptations, empirical evidence should be made relative to measurable spinal system deficits to determine whether specific muscle activation pattern adaptations exist for each deficit. However, there was a gap in the literature making such specific comparisons and what was published examined joint systems other than the spine and did not examine temporal differences in muscle activation patterns (Chapter 1). The four studies that make up this dissertation support that muscle activation patterns adapted to suspected spinal system deficits i.e. older adults and those with recovered low back injury (rLBI) (Chapter 3) and to measures of lower (below median) spinal system function (Chapters 4-6). Furthermore, by exploring individual spinal system function of the active, passive, and neural systems, the findings support that adaptations are unique to specific spinal system measured. This summary chapter discusses the collective findings contained in this dissertation and their impact on

our understanding of Panjabi's compensation theory while identifying factors that modify the relationships and could be considered potential confounders

Objective 1 (Chapter 3) directly tested whether trunk muscle activation patterns differed between older adults and participants recovered from an LBI, both suspected of having deficits within all three spinal systems, to a young asymptomatic control population. Novel findings were related to determining whether both groups had similar muscle activation patterns and whether both differed in a similar way when compared to a young asymptomatic control population. There was evidence that older adults and the rLBI had similar activation adaptations, but the magnitude of the change was greater in the older adult group for all features except antagonist co-activation amplitudes. This finding was consistent with Panjabi's compensation theory that if a deficit existed in one spinal system function that the other systems would adapt to maintain spinal stability. Patterns observed characterized higher agonist activation amplitudes, higher antagonistic activation amplitudes, and reduced responsiveness of trunk muscles to changing external moments. Higher activation amplitudes compensate for reduced trunk strength acknowledged in both older adults and those with chronic low back pain (LBP) when compared to asymptomatic controls (Keller et al., 1999; Newton et al., 1993; Sinaki et al., 2001; Singh et al., 2013). Reduced responsiveness and higher antagonist co-activation amplitudes were consistent with other studies and thought to be mechanisms to increase the general stiffness of the spine (Cholewicki & VanVliet, 2002; Granata & Marras, 2000; Kavcic et al., 2004). Increased stiffness could act as an adaptation to reduced neutral zone passive stiffness (Fujiwara et al. 2001; Galbusera et al. 2014; Sengupta and Fan 2014) or changes in feed-back control, characterized by delayed reflexes (Hwang et al., 2007; Klass et al., 2011; Ramprasad et al., 2010; Reeves et al., 2004; Shenoy et al., 2013; Shojaei et al., 2016), also acknowledged within each population. The activation patterns for the rLBI and older adult groups were similar in direction however, they differed in magnitude. Older adults likely had deficits within spinal systems that were of higher magnitude requiring more muscle activation pattern adaptations than the rLBI group. However, there was one notable exception of higher antagonistic activation amplitudes in the rLBI group. This suggests that the unique experience of recent low back pain modified how individuals adapted to spinal system deficits and thus was

treated separately in Chapters 4-6 to further understand this influence of current or recent pain.

The findings in Chapter 3 provided the framework to show how muscle activation patterns adapted to suspected deficits within spinal systems. Like much of the literature, this study could not elucidate whether these two populations had deficits in a single or all three spinal systems. It was also not possible to determine whether these muscle activation pattern adaptations were unique to a deficit in a specific spinal system or whether they captured a generalized adaptation to deficits within multiple spinal systems.

To explore whether muscle activation patterns adapted in a specific way to lower function within a single spinal system, objective two directly measured individual spinal system function (active, passive, and neural) and trunk muscle activation patterns during a highly controlled transfer task. Data were collected from 69 participants, including rLBI, and those who did not experience low back pain within one year prior to testing. This rLBI group was included to address the secondary purpose related to whether specific muscle activation adaptations differed between those who have or have not recently recovered from a low back injury.

In each of the three separate papers (Chapters 4-6), direct measures were used to characterize the function of each spinal system (active, passive, and neural), where a median split approach divided groups for each measure to better understand how trunk muscle activation patterns adapted to lower function within a specific spinal system. In the end, the analysis was conducted on 60 men as only 9 of the 69 participants were women and only two women were in the rLBI group it was not possible to perform the median split to probe the influence of recent pain. However, if the analysis was performed only on the direct measure of each spinal system without considering the interaction with recent LBI women could be included without impacting the key findings between the high and low functioning groups, for example in the passive system (Quirk & Kozey, 2018b). Through the series of experiments, unique muscle activation patterns were identified showing an adaptation for each specific spinal system (Table 7.1).

Table 7.1: Summary of findings of the various cross-sectional comparisons in this dissertation, comparing and contrasting how populations adapted to suspected or measured deficits in spinal system function for the horizontal transfer task

Deficit Group	Older vs ASYM	rLBI vs ASYM
Suspected (Active/ Passive/ Neural) (Chapter 3)	↑↑ Agonist Activation ↑ Antagonist Activation ↓ Responsive (LF) BE ↓ Responsive (F) BE	↑ Agonist Activation ↑↑ Antagonist Activation ↓ Responsive (LF) BE ↓ Responsive (F) BE
Measured Function	Low Functioning ASYM	Low Functioning rLBI
Active (Back extensor strength) (Chapter 4)	↑ Agonist Activation ** ↑ Antagonist Activation ↑ Responsive (LF) BE	↑↑ Agonist Activation ↑↑ Antagonist Activation ↑ Responsive (LF) BE
Passive (Transverse plane mass normalized stiffness) (Chapter 5)	↑ Antagonist Activation ↑ Responsive (LF) Abs ** ↑ Responsive (LF) BE	↑ Antagonist Activation ↑Responsive (LF) Abs ** = Responsive (LF) BE ↑ Agonist Activation
Neural (Reflex latency) (Chapter 6)	↑ Antagonist Activation	= Antagonist Activation ↓ Responsive (LF) BE **

All comparisons were made to contrast those with deficit (lower functional) measures compared to those with above median measures for each respective subgroup (asymptomatic (ASYM) or rLBI) except for the first row where comparisons were made to the young healthy controls. The symbols are used to display a non-significant (=) or a significantly higher (↑) or lower (↓) feature was characterized. Double arrows (↑↑) reflect interactions where the magnitude of difference within the specific population was greater than the difference in the comparable comparison. Double asterix (**) reflect differences unique to lower function in a specific spinal system. Abbreviations: Lateral flexion (LF), flexion moment (F), back extensor (BE), and abdominals (Abs).

In the active system those with lower back extensor strength (weak) had higher muscle activation amplitudes of agonist muscle sites during the transfer task, as hypothesized. These differences captured the recruitment of additional motor units to compensate for reduced maximum moment production (muscle strength), and to overcome the moment produced by higher antagonist activation amplitudes. An interesting finding was higher antagonist (abdominal) co-activation in the weak compared to strong group suggested there was an additional external moment demand that must be met by antagonist muscles, requiring participants with lower strength to increase motor unit recruitment. Furthermore, participants with lower strength had greater

responsiveness of trunk muscles to changing external moments during the transfer task, a finding that has not been reported around the trunk or other joints. Previous work has shown individuals with weaker muscles have higher agonist activation amplitudes around the knee (Fujita, Kanehisa, Yoshitake, Fukunaga, & Nishizono, 2011; Mizelle et al., 2003; Takai et al., 2008) and ankle thank stronger individuals (Lucas-Cuevas, Baltich, Enders, Nigg, & Nigg, 2015; Takai et al., 2008), but to our knowledge no study compared temporal responses. Previous studies have shown increasing task load also increases the spatial and temporal responsiveness of trunk muscles to dynamic tasks (Butler et al., 2010; Quirk & Hubley-Kozey, 2014). Chapter 4 found, consistent with increasing task load, participants with lower strength had higher temporal responsiveness of trunk muscles.

Previous knowledge regarding how muscle activation patterns adapt to lower measured function of the passive system were found for the knee joint (Shultz et al., 2004; Shultz & Schmitz, 2009). The present results (Chapter 5) were consistent with this work finding higher antagonist, abdominal, co-activation amplitudes in participants with lower mass normalized transverse plane stiffness (laxity or lax). The higher antagonist activation amplitudes could act as a generalized strategy to increase spinal stiffness via active mechanisms to compensate for measured reductions in passive stiffness. Novel to this dissertation was the characterization of trunk muscles response to changing forces in the frontal plane, capturing participants with lower passive stiffness had greater temporal responsiveness. This mechanism was contrary to the hypothesis of reduced responsive trunk muscle activation in the frontal plane, as observed in populations suspected to have passive stiffness deficits (Quirk & Hubley-Kozey, 2018; Trudel, 2014). However, greater responsiveness in the frontal plane captured that agonist sites increased their activation, a mechanism that is also acknowledged to prevent instability events by increasing active spinal stiffness (Kavcic et al., 2004). Why greater responsiveness was not observed in our populations with suspected deficits in the passive system was thought to be partially explained by co-varying deficits in the neural system, previously reported in older adults and LBP populations when compared to young healthy controls (Brumagne et al., 2004; Claeys et al., 2010; Goldberg et al., 2005; Laird et al., 2014; Tong et al., 2015). Neural system deficits could impair the speed and accuracy of feedback control needed to adjust

the time-varying recruitment of trunk muscles. Congruent with this interpretation was that greater back extensor responsiveness identified in the lax asymptomatic group, was not found for the lax rLBI group. One might suspect simultaneous deficits in the neural system of participants in the rLBI group could impair the ability to increase back extensor responsiveness, explaining the interaction in this study. However, this interpretation must be made with caution for, despite participants being recovered (minimal pain and disability) on the day of testing, the previously experienced pain by our rLBI group could shift participants towards a higher co-activation strategy (Hodges et al., 2013) which is theorized to be retained when pain resolves (Hodges & Tucker, 2010).

To our knowledge, no study has examined whether individuals with delayed stretch reflexes around any joint have different muscle activations (amplitudes or patterns) to perform functional tasks. Work characterizing lower function of the neural system, reduced proprioception or greater repositioning error, around the ankle show muscle sites have higher antagonist activation amplitudes in preparation to landing from a drop jump (Fu & Hui-Chan, 2007). In this dissertation comparing those with lower function (slower) reflexes to those with faster reflexes (Chapter 6), similar to Fu et al., participants with delayed muscle onsets had higher antagonist abdominal activation amplitudes. Higher antagonist activation could increase the general stiffness of the spine (Granata & Marras, 2000; Kavcic et al., 2004). Furthermore, there was evidence of reduced responsiveness to the frontal plane moment for back extensor sites. This suggests that those with delayed reflexes settle upon a pattern consistent with a strategy of generalized stiffness in multiple planes to provide the trunk intrinsic stiffness to act as a margin of safety in the event of any unexpected adjustments in external loads. Collectively the findings of these three studies comparing those with high or low function across three different spinal systems (Chapters 4-6) provide support that muscle activation patterns adapt measurable changes in spinal system function and that these muscle changes are unique for each spinal system (Table 7.1, shown by **). In particular, this dissertation found lower function of: the active system resulted in greater responsiveness to the lateral flexion moment for the back extensors, the passive system had greater responsiveness for the abdominals, and in the neural system an interaction captured only rLBI had reduced responsiveness of back extensors to the changing lateral

flexion moment. This interaction supports the sub-objective that the recent experience of low back injury modified how participants adapted to lower function of the spinal system.

The general conclusions observed in the three spinal system papers (Chapter 4-6), comparing equal numbers of men representing two different populations (rLBI and asymptomatic) helps understand how the recent experience of pain might independently influence muscle activation pattern adaptations to lower function of spinal systems despite the recent recovery of painful symptoms. Collectively across the three spinal systems interactions suggest participants recently recovered from a LBI with lower spinal system function often had greater differences in activation amplitudes (active), no evidence of greater responsiveness of back extensor sites (passive), and evidence of reduced responsiveness of back extensor sites to changing lateral flexion moments (neural) (Table 7.1). These interactions support that rLBI adapt muscle activation patterns in a way that would seek to increase the generalized stiffness of the spine and potentially the magnitude of this compensation is magnified in those with lower function within the spinal systems. To explain this feature, we focused our interpretation on the primary difference between the asymptomatic and rLBI group. Despite symptomatic resolution (minimal pain and disability) on the day of testing, the recent experience of low back pain, may have residual effects on how participants adapted muscle activation patterns to lower spinal system function, consistent with the motor adaptation to pain theory (Hodges & Tucker, 2010).

Theoretical, computational modeling, and experimental work suggest that pain may represent a factor to which participants adapt muscle activation patterns to modify spinal stiffness and thus stability (van Dieën et al., 2017). Despite lacking experimental evidence it is theorized that in some these patterns are retained when pain resolves, which could be a risk for future back pain, in what is known as the motor adaptation to pain theory (Hodges & Tucker, 2010). Novel data from this study supports a new hypothesis, that the extent muscle activation patterns adapt to pain may relate to both the level of function within an individual spinal system and the experience of pain. In this hypothesis, those with lower spinal system have augmented adjustments in muscle activation patterns than those who do not. The rational to explain this heightened adaptation in the presence of recent pain may be related to when the participant was in their painful state. In this

painful state lower spinal system function could increase the risk of experiencing small instability events, provoking pain. This spinal system function dependent pain could act as a cost to incentivize the neural system to explore new motor patterns to prevent these events. In participants with higher spinal system function fewer small instability events occur, reducing the need (cost) to adapt motor patterns. While this theory fits within recent computational modeling suggesting that pain can act as a cost criteria for muscle pattern optimization (van Dieën et al., 2017), the integration of spinal system function would require additional computational modeling to see how a system might adapt to an increased likelihood of experiencing "small instability events" (spinal system function level) gained by the experience of pain.

Despite summary data suggesting adaptations to lower spinal system function were often different for specific spinal systems, the most apparent question is whether the same participants populated the various lower function groups. From a physiological perspective, the literature suggests that lower function within individual spinal systems may lower the function of other systems. Evidence from our studies supports Panjabi's theory of compensation where individuals adapt muscle activation patterns to these deficits. The interrelation of lower function in the three spinal systems could be explained by the dependence of some tissue systems on one another. For example, mechanoreceptors of the neural system are embedded within osteoligamentous (Jiang et al., 1995; Yahia, Newman, & Rivard, 1988) or musculotendinous tissue (Amonoo-Kuofi, 1983; Boyd-Clark et al., 2002).

Considering the interrelationship between spinal systems one might expect with increased osteoligamentous laxity tissues would undergo greater strain until they experience enough stress to cause deformation to the imbedded mechanoreceptors. Thus, impairing information provided by sensory afferents. This relationship has been shown in previous studies for example in those with knee osteoarthritis, participants with greater anterior-posterior neutral zone stiffness had a weak association (r=0.09-0.4) with greater motion detection error (reduced proprioception) (Cammarata & Dhaher, 2012). In the spine, much of the work linking the passive and neural system has been explored by inducing transient changes to passive tissues following tissue creep protocols. Exposing an in vitro vertebral joint to a static or repetitive load results in tissue creep, increasing

range of motion (Adams & Dolan, 1996; Busscher et al., 2011; Zhao, Pollintine, Hole, Dolan, & Adams, 2005) and increasing strain to a specific stress (Adams & Dolan, 1996). It has been shown, in vivo, that moving the trunk into full (deep) flexion increases loading on passive tissues (Arjmand & Shirazi-Adl, 2006). In vivo sustained (Abboud, Nougarou, & Descarreaux, 2016; Bazrgari et al., 2011a; Muslim et al., 2013; Shin, D'Souza, & Liu, 2009) and repetitive deep trunk flexion (Olson, Li, & Solomonow, 2004; Shin & Mirka, 2007; Toosizadeh et al., 2013) produces ligamentous creep, increasing trunk flexion range of motion (Abboud et al., 2016; Milosevic et al., 2015; Olson et al., 2004; Shin et al., 2009; Shin & Mirka, 2007) and decreasing trunk stiffness in response to rapid perturbations (Bazrgari et al., 2011a; Muslim et al., 2013; Toosizadeh et al., 2013) relative to pre-creep measures. Following such paradigms, it has been shown that post creep there are delays in muscle reflex onset (Sánchez-Zuriaga et al., 2010), supporting that mechanoreceptor deformation may occur later as a consequence of changes to the mechanoreceptors host osteoligamentous tissues. However, a recent literature review has concluded that there is no consensus of decreased reflex latency, or reflex amplitude following spinal creep (Abboud, Lardon, Boivin, Dugas, & Descarreaux, 2017), suggesting the strength of this relationship is weak.

Similar changes occur within mechanoreceptors embedded within the active system. Ito et al., have found that reduced erector spinae cross-sectional area was associated with reduced trunk motion following vibration to the paraspinal muscles (impaired proprioception) (Ito et al., 2015). However, the strength of this relationship was weak (r=-0.26) and only revealed in superior (L1/2 level) erector spinae muscles and no other (3/4) muscle sites.

Further rationale for mutual deficits in spinal systems might also be explained by the proximity of tissue systems. Work from Hodges and others show the inflammatory environment created following an osteoligamentous (disc) injury is associated with changes in the size and quality (presence of intramuscular fat and fibrosis) of nearby musculotendinous tissues (Brown et al., 2011; Hodges et al., 2015; Hodges & James, 2018; Hodges, Holm, Hansson, & Holm, 2006; James, Klyne, Millecamps, Stone, & Hodges, 2019). Thus, multiple tissues systems, located close to one another, could

experience a reduction in function following a period of inflammation induced in an unrelated tissue system.

Collectively, the above studies suggest that lower function in the passive, active and neural systems do interact with one another where decrements in one system coincide with decrements in another. This is contrary to Panjabi's theoretical model, where all tissue systems are treated in silo adapting for a deficit in one another (Figure 7.1A). Considering mutual deficits between spinal systems one participant could have lower function in 2 or 3 of the spinal systems simultaneously yet they would still retain the capacity to impact one another as shown in Figure 7.1B. To explore whether lower function were identified in multiple spinal systems for the same participants the data set was separated between those with low (deficit) or high function for the three fundamental spinal system tests used in this dissertation (back extensor moment, neutral zone transverse plane spinal stiffness, and trunk muscle onset latency). Agreement on whether participants were placed in a low or high (above median function) group for the three tests are displayed in Table 7.2 and the Venn diagram for lower function populations in Figure 7.2. What is evident is whether participants were considered high or low function for a specific spinal system test was random. The probability of participants having high or low function between two spinal system tests was 47-53% close to random probability (50%). When considering the function of three spinal systems participants who scored as having high or low function across all test had a probability of 20-23% which was slightly, but not significantly, lower than the expected random probability (25%). Thus, there was minimal interrelation between the measures of spinal system function (Figure 7.2) and evidence was not strong that lower function co-vary between systems. Secondly, while 20-23% of participants were tested within the same group across all three studies these data suggest that this potential confounder acts only as the random chance of conducting all three studies on the same population.

A secondary limitation of the above series of studies was the differences between populations classified as having low spinal system function were small yet significantly different when compared to those with high function. This suggests that while the data supports the function of an individual spinal system acts as a criterion to detect significant differences in muscle activation patterns, there still exist other factors

accounting for variability in muscle activation patterns. Part of this variability could be explained by the use of the median split methodology in this dissertation resulting in some participants specific function measures around the median being separated in the low or high function group lowering the signal separating these two groups. In addition, unaccounted variability could be explained by considerable redundancy that is present within the trunk muscles allowing for multiple adaptations. Indeed, experimental pain work around the lower back suggests while no singular muscle or muscle group consistently increases or decreases activation in the presence of low back pain (Hodges et al., 2013; van den Hoorn et al., 2014) collectively the subtle changes within each muscle could act to achieve a common goal such as increasing trunk stiffness (Hodges, van den Hoorn, Dawson, & Cholewicki, 2008). Future work dedicated to understanding whether adaptations to spinal systems function act as a potential risk in developing future LBI requires an understanding of these sources of additional variability to predict what is an "optimum" adaptation and whether over or under adaptation to unique spinal system function measure modifies back pain risk.

The measurements of spinal system function in this dissertation, were often calculated such that lower function would represent a risk of a future low back injury and thus were characterized being deficient, but there are limitations with using this terminology. First, the level of low function used in this dissertation was produced on a median split within our sample population and thus do not reflect a level of spinal system function that could be used as a clinical threshold to predict who will have future low back injuries. Second, while the introduction to each study stated lower function has the potential to represent a risk to low back injury it is unknown if extremely high function (high back extensor strength, transverse plane spinal stiffness, or fast reflexes) could also represent a risk to LBI. Despite this limitation, the results in this dissertation did find that spinal system functional measures magnitude did impact muscle activation patterns consistent with the theme of Panjabi's compensation theory.

Finally despite this dissertation being comprehensive by including a functional measure of all three spinal systems proposed within Panjabi's original framework, it was limited in that only a single measure was used for each spinal system. Mentioned within each paper were alternative methods to characterize structural or functional deficits

within the spinal systems. However, for each study considerable a prior decision making determined the functional metric that could represent a risk of low back pain and could be implemented in a way to limit potential confounders. Despite this, the conclusions reached in this dissertation may reflect the single outcome measure that was used. A more rigorous method would have included multiple metrics within each spinal system to determine the best singular metric or combination of metrics to account for the variability in muscle activation patterns. However, such analysis would require an even larger sample size to account for errors related to performing multiple comparisons on the same dataset.

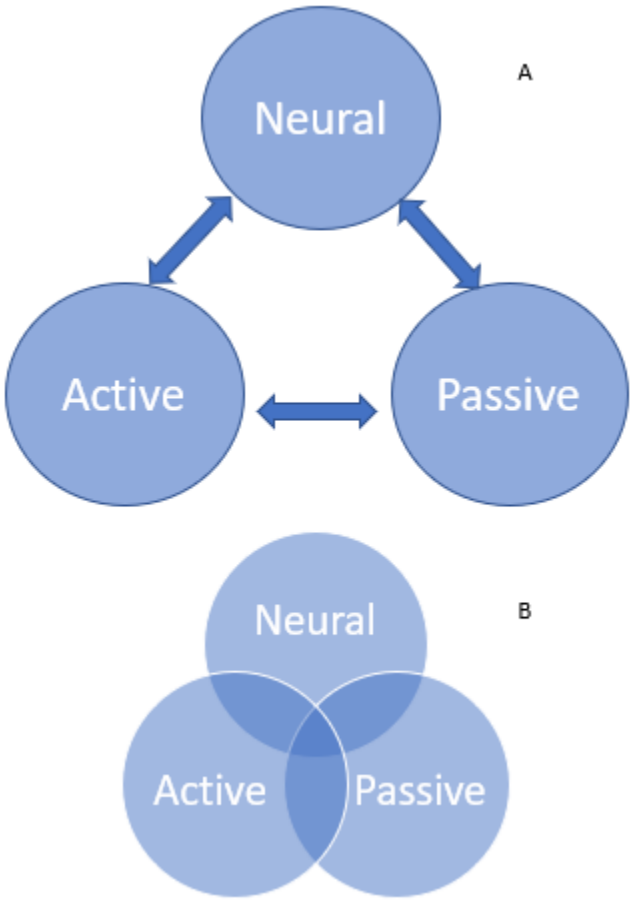

Figure 7.1: Comparison of Panjabi's original spinal system model (A) and a theoretical model of covarying deficits amongst multiple spinal systems (B).

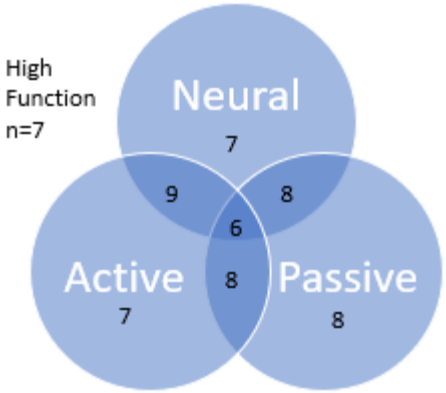

Figure 7.2: Venn diagram of participants classified as having low function within each individual or combination of various spinal systems. Outside depicts the number of participants with high function for all test, then the number of participants with low function in only one system (single circle), two systems (double overlap), or all three systems (center) are conveyed.

Table 7.2: Agreement of participants placed in the low function (below median) or high function (high score) category for the three test used in this dissertation. The first 3 rows represents participants placed in the same category (high or low function) for the two compared functional measures. The final row representing participants who were consistently placed in the same group for all three tests

Comparison	Subgroup	Total (n=60)	ASYM (n=30)	rLBI (n=30)
Moment & Stiffness	Total Agreement	28 (47%)	14 (47%)	14 (47%)
	Low Score	14 (47%)	7 (47%)	7 (47%)
	High Score	14 (47%)	7 (47%)	7 (47%)
Moment & Onset	Total Agreement	30 (50%)	16 (53%)	14 (47%)
	Low Score	15 (50%)	8 (53%)	7 (47%)
	High Score	15 (50%)	8 (53%)	7 (47%)
Onset & Stiffness	Total Agreement	28 (47%)	14 (47%)	14 (47%)
	Low Score	14 (47%)	7 (47%)	7 (47%)
	High Score	14 (47%)	7 (47%)	7 (47%)
Moment, Onset & Stiffness	Total Agreement	13 (22%)	7 (23%)	6 (20%)
	Low Score	6 (20%)	3 (20%)	3 (20%)
	High Score	7 (23%)	4 (27%)	3 (20%)

In conclusion, this dissertation provided empirical evidence that the abdominal and back extensor muscle activation patterns adapted to the function of individual spinal systems. Through a comprehensive analysis, these adaptations were unique to measures of each spinal system. Additionally, the evidence showed that recent recovery from low back pain independently influenced how trunk muscle activation patterns adapted to lower function within each spinal system. The data presented in this dissertation adds to our general body of knowledge, providing empirical evidence that deficits or lower function within individual spinal systems could represent a factor that the neural controller must adapt to prevent the likelihood of instability events. Overall findings of this dissertation fill some gaps in the current literature, advancing our knowledge on how muscle activation patterns interact with measures of joint function to maintain joint stability of the lower back spinal segments. These findings have implications towards understanding how musculoskeletal injuries develop suggesting muscle activations must adapt to the function of individual spinal systems. Furthermore, our data may provide information on how low back injuries can be prevented and/or managed (in the workplace or recreational activities) to ensure joint health can be maintained while individuals age allowing them to remain physically active.

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APPENDIX COMPARISION OF HYPOTHETICAL AND STATIC TOP-DOWN INVERSE DYNAMIC KINETIC MODEL OF THE HORIZONTAL TRANSFER TASK

8.1 INTRODUCTION

Comparing electromyograms (EMG) between individuals is challenging. The time-varying interference signal is the spatial and temporal summation of the number and firing frequency of motor unit action potentials captured within the volume of surface electrodes (Contessa, De Luca, & Kline, 2016; De Luca & Hostage, 2010; Fuglevand, Winter, Patla, & Stashuk, 1992). As the force of a task increases muscle activation increases linearly (Brown & McGill, 2007a) or curvilinearly (Larivière et al., 2002) as illustrated by muscle force-activation relationships. When comparing EMG signals between participants many authors normalize EMG amplitudes to a physiological reference (Burden, 2010). One established method is normalization to a maximum voluntary isometric contraction, believed to represent the physiological maximum activation of the sampled muscle. Despite normalization accounting for discrepancies due to skin and sub-cutaneous tissue attenuation (Burden, 2010; Larivière, Gagnon, & Loisel, 2000; Lehman & McGill, 1999), comparing EMG amplitudes between groups or individuals can be confounded if the external moment of the task or the absolute strength of individuals compared are systematically different (Quirk & Hubley-Kozey, 2018). To account for this, it is often important to control for or characterize external moments of the experimental task. Discrepancies can exist in interpreting EMG amplitude as a surrogate for muscular force as the direct relationship between the activation of a muscle site and the moment produced around a joint depends on: the size, angle of pennation, length, and the velocity of the muscle along with the effective moment arm length between the muscle's line of action and the joints center of rotation (Le et al., 2017; Marras, Ferguson, Burr, Davis, & Gupta, 2004; McGill & Norman, 1986).

Inverse-dynamics is one method where the forces and moment acting on a body segment can be calculated through measurements and estimates of external forces applied to the body and propagating these along a linked segment kinetic chain using basic Newtonian equations (Robertson, Caldwell, Hamill, Kamen, & Whittlesey, 2013; Winter,

2009). However, the external moments calculated through inverse-dynamics assumes no co-activation around the joint. Around the trunk, which is often the center of the kinetic chain, a top-down or bottom-up approach can be used to estimate forces and moments acting on the spine (Faber, Chang, Kingma, Dennerlein, & van Dieën, 2015; Iino & Kojima, 2011; Kingma, Toussaint, & de Looze, 1996; Larivière & Gagnon, 1999b). While differences exist between these approaches, primarily explained by top-down inverse dynamics being more sensitive to estimations of body segment inertial parameters (BSIP) (Larivière & Gagnon, 1999a; Riemer, Hsiao-Wecksler, & Zhang, 2007), both methods produce similar estimates of the time-varying changes in forces and moments acting on a joint of interest (Faber et al., 2015; Iino & Kojima, 2011; Kingma et al., 1996; Larivière & Gagnon, 1999b). Thus, either method applied systemically across a population should be considered internally valid to understand whether an experimental task is comparable between individuals or participant groups.

A top-down approach can often be completed with just measurements of body kinematics and regression equations to estimate BSIP. It is a feasible alternative if direct measurements of external forces cannot be made via a force plate, needed for the bottom-up approach. Calculating forces and moments can be performed using a static, quasi-dynamic/static, or dynamic analysis. Dynamic analysis uses full Newtonian equations requiring accurate measurements of the position of body segments and masses relative to a joint of interest, direct measurements of external forces applied to the body, and accounting for forces and moments needed to overcome linear and angular inertial properties. A quasi-dynamic approach excludes estimates of inertial factors at body segments but retains all other information necessary for dynamic analysis (including direct measures of external force). Static analysis assumes external forces applied to the body are purely represented as weight vectors and there are no direct measures of external forces nor estimation of linear or angular inertial factors (Callaghan et al., 2005; McGill & Norman, 1985). Using these definitions suggests that static analysis is possible through measurements of total body kinematics without any direct measures of external forces making it suitable for fieldwork (Callaghan et al., 2005). Comparing between methods show consistent temporal changes in the forces and moments measured around a joint of interest, despite differences in peak magnitude. Dynamic methods often capture

higher peak forces and moments than static methods. This difference is accounted for by ignoring the relative contribution of forces needed to produce acceleration to initiate, terminate or change the direction of a movement (McGill & Norman, 1985). However, errors between these methods become marginal, and often not significantly different, when comparing static versus dynamic methods for slow motion tasks (Bernard et al., 1999; De Looze et al., 1994).

For nearly a decade the neuromuscular function lab has used a highly controlled horizontal dynamic lifting task to understand how muscle activation patterns differ between populations or among tasks (Butler et al., 2010; Hubley-Kozey et al., 2012; 2013; Quirk & Hubley-Kozey, 2014). By controlling the timing of the motion and the displacement of an external mass and minimizing trunk motion, this task produced a hypothetical change in the flexion and lateral flexion moments around the lumbar spine as illustrated in Figure A.1. These moments at time points have been calculated using a simple static model based on many assumptions but have not been calculated using actual segment masses and displacements for the trunk and arms. The purpose of this paper is to describe the kinetic model used by a series of papers contained within this dissertation and to understand how the time-varying external moment profile measured by static kinetic analysis is qualitatively comparable to our hypothetical model.

8.2 METHODS

8.2.1 PARTICIPANTS

Data analyzed in this appendix were from 60 male participants who volunteered for this study from the Canadian Armed Forces to understand how muscle activation patterns adjust for a variety of spinal system deficits (Chapters 4-6). Before testing all participants signed an informed consent approved by the Institutes Research Ethics Board.

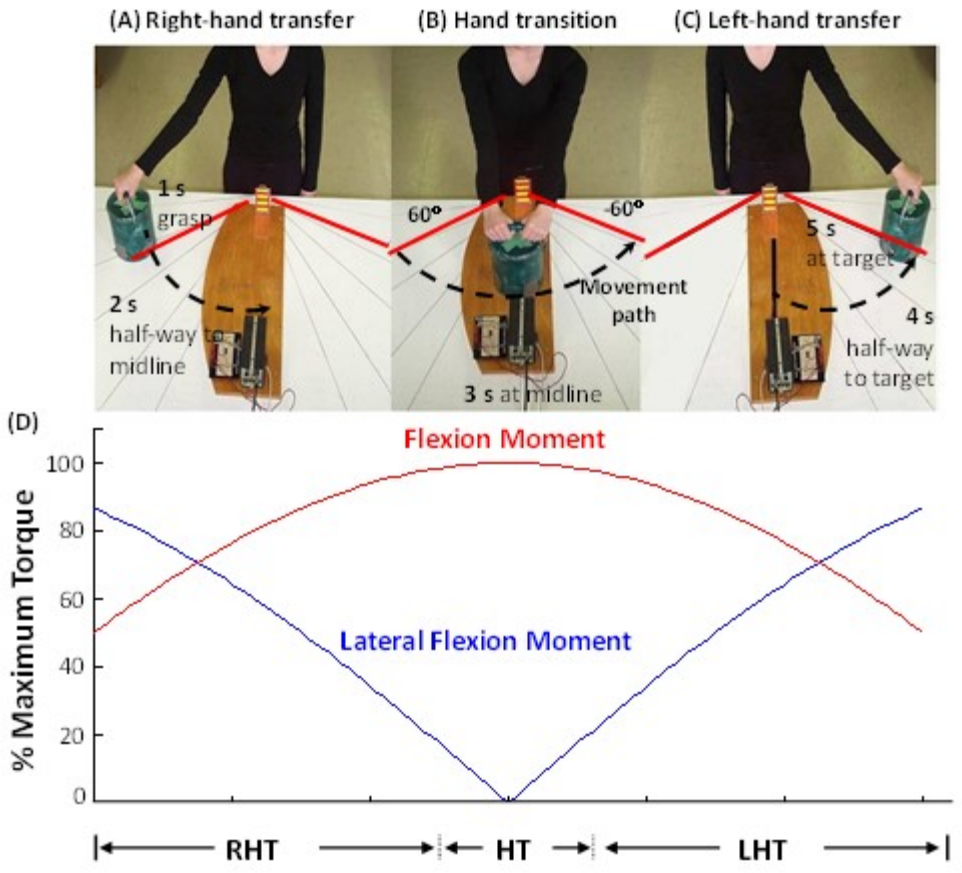

Figure A.1: Example of the right to left horizontal transfer task. Below is the hypothetical time-varying change in the external moments produced from this task as the mass moves from 60˚ to right-side of the midline of the body to 60˚ to the left.

Participants were prepared for electromyographic analysis (Section 2.3.1) and data were collected for the passive stiffness (Section 2.3.2.1) and reflex (Section 2.3.2.2) test. Participants were then prepared for kinematic analysis (Section 2.3.3 & 8.2.2) where they performed the highly controlled horizontal transfer task (Section 2.3.4) to collect their neuromuscular patterns. Briefly, to a standardized five second count participants were instructed to on "1" lift a 3kg mass orientated 60˚ to the right of the midline of their body with their right hand, on "3" transferring the mass to their left hand at the midline of their body, and then on "5" lower the mass 60˚ to the left of their midline (Figure A.1). Mass lift and lower were recorded using a pressure sensor located at the bottom of the mass, and hand transition was measured using an optoelectric switch. A height adjustable table was used to ensure the mass was lifted just below elbow height, and the optoeletric switch ensured the mass was lifted no higher than 5cm. During the task a researcher

ensured the participant lifted the mass with the arm in full extension. Participants were asked to minimize trunk motion aided by tactile feedback provided to the upper thoracic spinous process. This task was designed to be controlled so that a predictable hypothetical moment profile would be applied to the trunk as the arms transitioned the mass from a mixed lateral flexion/flexion to a pure flexion moment (Figure A.1).

8.2.2 KINEMATIC ANALYSIS

The kinematic analysis for these studies has been described briefly in Section 2.3.3. Passive reflective markers were positioned on the suprasternal notch (SSN) and 7th cervical spinous process (C7) along with 16 bilateral landmarks on the limbs: 5th (5MC) and 2nd (2MC) metacarpal, radial styloid (RS), ulnar styloid (US), medial (MHE) and lateral humeral epicondyle (LHE), the mid acromion clavicular (Ac) joint, and the anterior superior iliac spine (ASIS). Rigid body clusters (4 markers) were affixed to the thorax and pelvis, along with bilateral clusters on the forearm and upperarm (Figure A.2). Following marker setup, a single standing calibration trial captured the marker position relative to the rigid bodies using six infrared emitting cameras (ProReflex 240, Qualisys^{TM}, Goteborg, Sweden) sampled at 100Hz using Qualisys Track Manager Software (Version 2.10, Qualisys^{TM}, Goteborg, Sweden). For synchronization, the Labview program collecting analog-to-digital data triggered the motion capture system.

Marker data were processed in Qualisys Track Manager, to label marker's coordinates and entered into a custom Matlab^{TM} script for quadratic interpolation of missing data points, and low pass filtered at 4Hz using a fourth order, zero-lag Butterworth filter (Wicks, 2017).

For the standing calibration trial the bony landmark marker set was used in a variety of regression equations to determine joint (wrist, elbow, shoulder, trunk) center of rotation (Figure A.3) (Campbell et al., 2009; Dumas et al., 2006; Reed et al., 1999), and the location of each body segments (hand, forearm, upper arm, and torso's) center of mass (CoM) (Dumas et al., 2006). In addition, these markers were used to determine the anatomical coordinate system (ACS) around each segment's center of mass (Figure A.4) using the recommendations set forth by the International Society of Biomechanics (Wu et al., 2002; 2005).

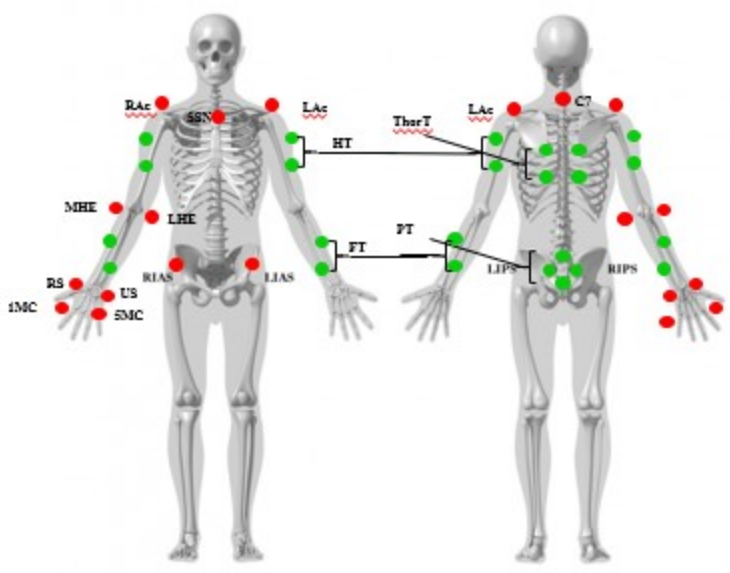

Figure A.2: Representation of kinematic marker setup. Including anatomical landmarks (red) and markers to represent the 4 marker rigid bodies (green).

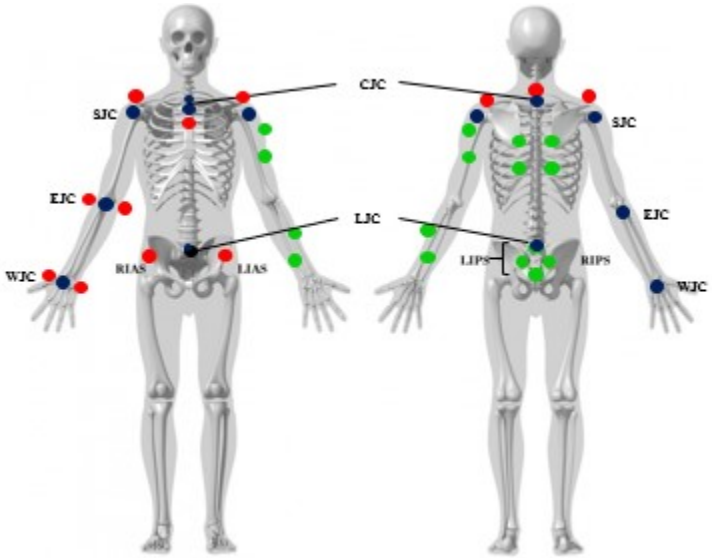

Figure A.3: Representation of calculated joint centers. Wrist (WJC), elbow (EJC), shoulder (SCJ), cervical (CJC) and lumbar (LJC) depicted in blue, along with single markers (red) needed as input to calculate these joint centers.

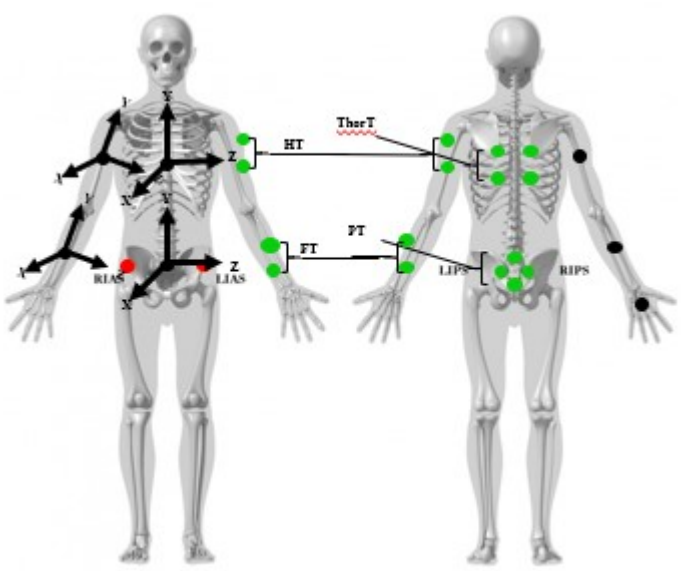

Figure A.4: Representation of the calculated segment center of mass and anatomical coordinate system. Each center of mass is depicted by a black dot, along with the vectors for each anatomical coordinate system.

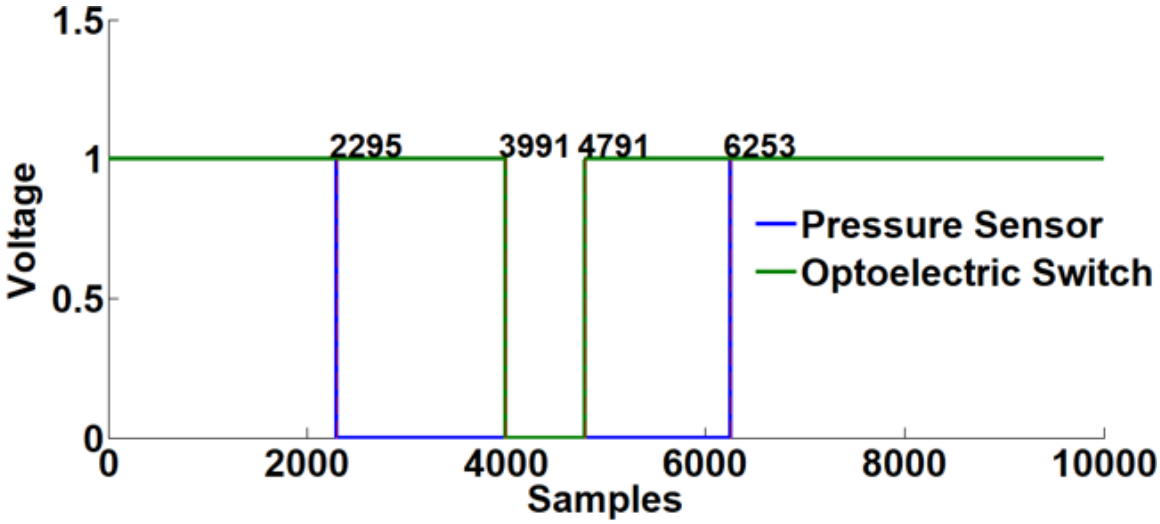

Figure A.5: Example of voltage data from the optoelectric switch (green) and pressure sensor (blue) from a single trial.

Following this calibration trial, all bony landmark markers were removed leaving only the rigid body clusters that were tracked for the horizontal transfer task. Assuming each segment acted as a rigid body the global coordinate system (GCS), the position of any individual marker, joint center of rotation, or CoM could all be defined as a virtual marker (VM) with a rigid vector relationship (VM_{LCS}) to a technical coordinate system (TCS) of the rigid body such that it could be translated and rotated with respect to a local to global (LtoG) TCS transpose matrix to define VM's position in the GCS using

Equation A.1. By the same definition a rigid relationship would exist where the ACS could be defined with respect to the TCS via similar matrix multiplication.

$$\overrightarrow{VM_{GCS}} = [T_{LtoG}]\overrightarrow{VM_{LCS}}$$

Equation A.1: Calculation of points in the global coordinate system using a technical coordinate system. Abbreviations include how to calculate a virtual marker (VM) point in the global coordinate system (GCS) using a transpose matrix of the technical coordinate system (T) to produce a local to global (LtoG) to translate and rotate the VM vector measured in the local coordinate system (LCS) for the static trial.

Kinematic data were used to calculate the moments of force around the trunk using a static top-down inverse dynamics approach (Callaghan et al., 2005; Iino & Kojima, 2011). For this approach, the relative magnitude of each segments mass was estimated as a proportion of total body mass using anthropometrically derived regression equations (Dumas et al., 2006). For this model, necessary inputs were the magnitude and position (CoM) of all segment masses and estimates of joint center of rotation. The largest assumption from this kinetic model was the estimate of the magnitude and position of the external load lifted by participants. As only the mass of the external object was known (3kg) and its direct position was not measured it was assumed the center of mass of the lifted object was a weight vector that acted at the middle of the 2nd and 5th metacarpal of the right hand for the beginning of the lift (3kg right hand), both hands during hand transition (1.5 kg each hand), and the left hand following hand transition (3kg left hand), as determined by event markers for the pressure sensor and movement through the optoelectric switch (Figure A.5).

Starting at the most distal and open-ended hand segment forces and moments were calculated in the global coordinate system and propagated from one segment to the next using a system of Newton equations where all linear and angular acceleration terms were assumed to be zero. Assuming the human body acted as a link of frictionless rigid bodies an open kinetic model approach was used where the predicted forces and moments calculated at the proximal (p) end of a body segment were propagated, assumed to be equal and opposite, to the subsequent body segments distal (d) end with respect to Newton's third law (Winter, 2009). Fundamental equations were modified from a generic equation designed for static analysis (Equation A.2 and A.3) with acceleration (a) and

angular momentum (Hdot) set to 0 (Callaghan et al., 2005; McGill & Norman, 1985; Robertson et al., 2013). For all equations, essential inputs were the mass (m) of a segment or external load and acceleration due to gravity (g) to produce a weight vector acting in the vertical direction of the global coordinate system. At the torso segment (Equation A.10 and A.11) forces and moments were included from the global coordinate system of both the right and left shoulders, the mass and center of mass location of the head was assumed to be included in the mass of the torso (Dumas et al., 2006). The distal moment of the torso as calculated at the lumbar joint center (L4/L5) (Equation A.11) was defined with respect to the torso coordinate system to provide anatomical context for these moments with respect to the International Society of Biomechanics recommendations defining moments around lateral-flexion (x) as the floating axis calculated as the cross product between the (y and z axis), the axial rotation axis (y) defined as the long axis between the cervical and lumbar joint centers, and the flexion-extension (z) as the floating axis defined by the x-y cross product but initially assumed to be the axis defined between the suprasternal notch and the seventh cervical spinous process (Wu et al., 2002; 2005).

$$\sum F = ma = 0$$

Equation A.2: Generic linear static equation. Abbreviations include forces (F), mass (m) and acceleration (a).

$$\sum M = Hdot = 0$$

Equation A.3: Generic angular static equation. Abbreviations include moment (M), and the angular momentum of a segment (Hdot) derived knowing the mass moment of inertia of a segment and a combination of angular velocity and acceleration.

$$F_{Wp} = -m_o g - m_h g$$

Equation A.4: Calculation of proximal wrist force. Abbreviations include mass (m) of the object (o) and hand (h), acceleration due to gravity (g), and the force (F) calculated at the proximal (p) wrist (W).

$$M_{Wp} = -m_o g(CoM_o - CoM_h) - F_{Wp}(WJC - CoM_h)$$

Equation A.5: Calculation of proximal wrist moment. Abbreviations include those in Equation 4 in addition to the center of mass (CoM) position of the object (o) and hand (h), along with the position of the wrist joint center (WJC) to calculate the moment (M) at the proximal (p) end of the wrist (W).

$$F_{Ep} = -m_f g - F_{Ed}$$

Equation A.6: Calculation of proximal elbow joint force. Abbreviations include mass (m) of the forearm (f), acceleration due to gravity (g), the force (F) at the distal (d) elbow (E) joint which was equal and opposite to the proximal wrist joint Equation 4 to calculate the force at the proximal (p) elbow.

$$M_{Ep} = -M_{Ed} - F_{Ed}(WJC - CoM_f) - F_{Ep}(EJC - CoM_f)$$

Equation A.7: Calculation of proximal elbow moment. Abbreviations include those in Equation 6 in addition to the center of mass (CoM) position of the forearm (f), along with the position of the wrist (WJC) and elbow joint center (EJC) the moment (M) at the distal (d) elbow (E) was equal and opposite to the proximal wrist joint Equation 5 to calculate the moment at the proximal (p) elbow.

$$F_{Sp} = -m_u g - F_{Sd}$$

Equation A.8: Calculation of proximal shoulder force. Abbreviations include mass (m) of the upper arm (u), acceleration due to gravity (g), the force (F) at the distal (d) shoulder (S) joint which was equal and opposite to the proximal elbow joint Equation 6 to calculate the force at the proximal (p) shoulder.

$$M_{Sp} = -M_{Sd} - F_{Sd}(EJC - CoM_u) - F_{Sp}(SJC - CoM_u)$$

Equation A.9: Calculation of proximal shoulder moment. Abbreviations include those in Equation 8 in addition to the center of mass (CoM) position of the upper arm (u), along with the position of the elbow (EJC) and shoulder joint center (SJC) the moment (M) at the distal (d) shoulder (S) was equal and opposite to the proximal elbow joint Equation 7 to calculate the moment at the proximal (p) shoulder.

$$F_{Tp} = -m_t g - RF_{TSd} - LF_{TSd}$$

Equation A.10: Calculation of proximal trunk force. Abbreviations include mass (m) of the torso (t), acceleration due to gravity (g), the force (F) at the distal (d) aspect of the trunk (T) joint which was equal and opposite to the proximal right (R) and left (L) shoulder (TS) joint Equation 8 to calculate the force at the proximal (p) trunk.

$$M_{Tp} = -RM_{TSd} - RF_{TSd}(RSJC - CoM_t) - LM_{TSd} - LF_{TSd}(LSJC - CoM_t) - F_{Tp}(LJC - CoM_u)$$

Equation A.11: Calculation of proximal trunk moment. Abbreviations include those in Equation 10 in addition to the center of mass (CoM) position of the torso (t), along with the position of the right (R) and left (L) shoulder (SJC) and lumbar joint center (LJC) the moment (M) at the distal (d) trunk (T) was equal and opposite to the proximal shoulder joint Equation 9 to calculate the moment at the proximal (p) trunk.

Following these calculations, the time varying trunk moments in the flexion extension, and lateral flexion planes were time normalized as a percentage of total movement from mass lift (0%) to mass lower (100%) using a quadratic spline function. For each participant peak absolute value lateral flexion and flexion moments were determined to understand the peak loading of this task. Ensemble average waveforms were generated to provide the time-varying profile of moments directed in both the lateral flexion and flexion direction. Finally, to compare our data with the hypothetical moments generated by this lateral flexion task the flexion and absolute value lateral flexion moment were amplitude normalized to the peak flexion moment measured for each participant (Figure A.1).

8.3 Results/ Discussion

The ensemble average external moment profile as a mean and standard deviation are displayed in Figure A.6 and A.7 for the lateral flexion and flexion moment respectively. Qualitatively while there is variance in the absolute magnitude for each moment the calculated waveforms show for the lateral flexion moment a peak right flexion moment occurred at the initiation of the lift, and peak left lateral flexion moment occurred near the end of the lift. The relative magnitude of the left lateral flexion moment (10.3±3.5Nm) was lower than the right moment (11.7±2.7Nm). Potentially explained as while mass lift could be set up to be lined prior to initiating the task, mass lower required the participant to approximate the location of where the mass was lowered while maintaining the external count of the task. In the sagittal plane, a flexion moment was produced for the entire lifting task with a minimum moment of 15.7±2.9 Nm and a peak moment of 27.8±3.5Nm. Reviewing the waveform, it would appear on average the peak

flexion moment occurred around 54% of the total movement during hand transition (Figure A.7).

When data were transformed to determine how they compared to the hypothetical moment profile generated by this task (Figure A.1), the static model performed similarly, predicting approximately 60% of the total flexor moment would be produced for the entire task, and the absolute magnitude of the lateral flexion moment would transition to nearly zero by the middle of the task when the mass was transitioned from one hand to the other (Figure A.8). Compared to the hypothetical moment profile, the general transition between moments, particularly in lateral flexion, was not as smooth around the phase of hand transition (40-65%). The measured moment curve had a noticeable inflection around 40 and 65% with a less abrupt transition around the middle of the task 50-55% when compared to the hypothetical moment profile (Figure A.8). This divergence was likely explained by the production of a counter moment as the contralateral limb moved into position to reach the mass in preparation to hand transition occurring around 40-60% of the total movement time (Figure A.8). A feature that was not considered in the hypothetical model that only considered how the transition of the lateral flexion moment would vary as mass transitioned from a 60 to 0˚ orientation relative to the midline of the body with no correction for potential motion in the contralateral arm (Figure A.8).

The results of this appendix confirm the hypothetical controlled external moment profile for this highly controlled lifting task was similar to direct measurements from a static kinetic analysis (Figure A.8). Some discrepancies did exist by not considering the influence of the contralateral limb reducing the relative magnitude of the lateral flexion moment produced by this task during the hand transition phase.

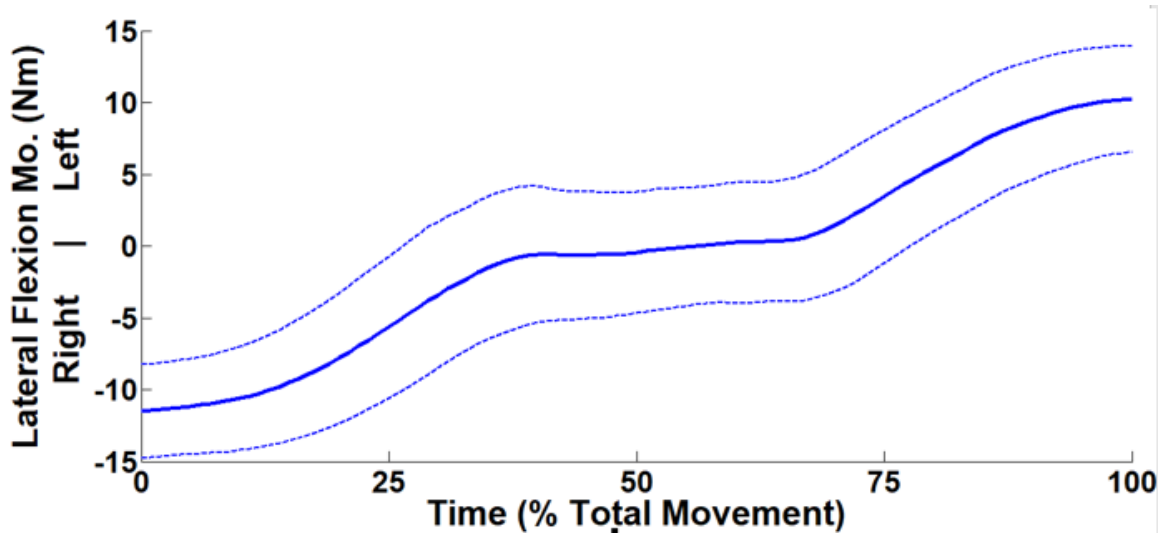

Figure A.6: Calculated ensemble average lateral flexion moment from the horizontal transfer task. Data displays participant grand mean (solid) and standard deviation (dashed).

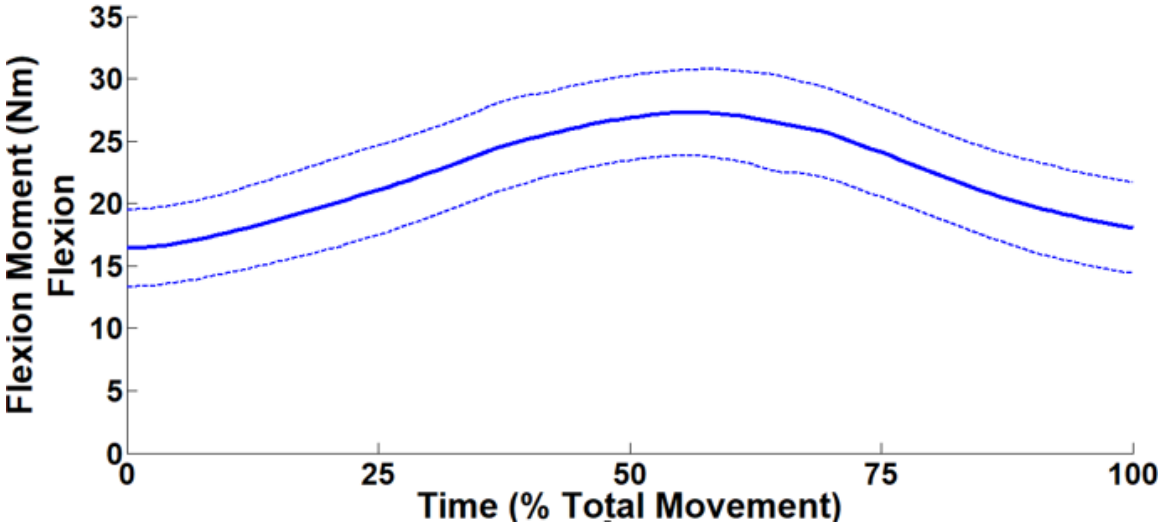

Figure A.7: Calculated ensemble average flexion moment from the horizontal transfer task. Data displays participant grand mean (solid) and standard deviation (dashed).

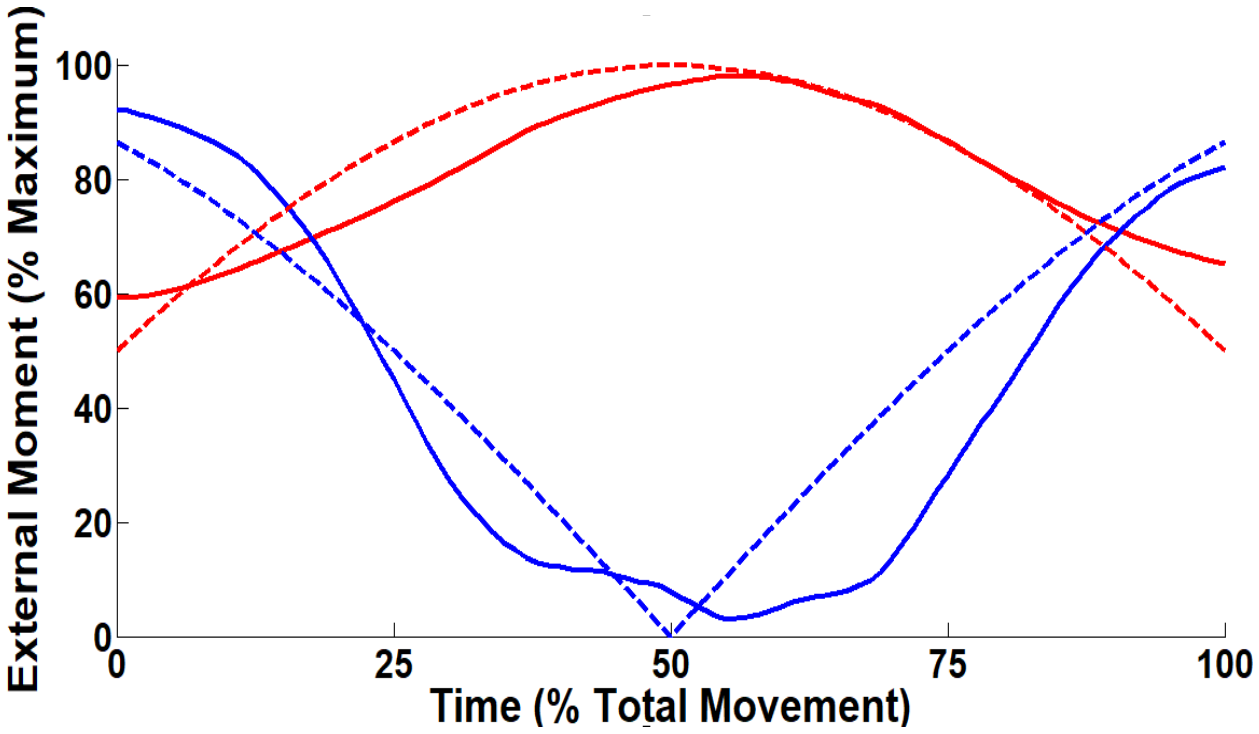

Figure A.8: Comparison of ensemble average and hypothetical external moments. This model shows the calculated ensemble average (solid) and hypothetical (dashed) lateral flexion (blue) and flexion (red) moments produced around the lumbar spine for the horizontal transfer task.

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27 March 2019

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***Journal of Motor Behavior*, 51 (2): 222-238.**
DOI: 10.1080/00222895.2018.1458280

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