Exploring Low to Moderate Velocity Motor Vehicle Rear Impacts as a Viable Injury Mechanism in the Lumbar Spine

by

Kayla Michelle Fewster

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AUTHOR'S DECLARATION

This thesis consists of material all of which I authored or co-authored: see Statement of Contributions included in the thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I understand that my thesis may be made electronically available to the public.

Statement of Contributions:

I hereby declare that my contribution to each journal publication that was produced from my thesis (currently in press, accepted, and submitted) was from my own work, and for each study that I was responsible for the conception and/or design, preparation, collection and analysis of the data, and the writing and editing of the manuscripts.

I hereby declare that the contribution of author Dr. Jack P. Callaghan on each manuscript was to the conception and/or design, editing of the manuscripts, assisting with instrumentation, and securing external funding for the research.

I hereby declare that the contribution of author Dr. Robert J. Parkinson in each manuscript was to the conception and/or design, editing of the manuscript and providing a database for the research completed in Chapter 3.

I hereby declare that the contribution of author Dr. Chad E. Gooyers was to the conception and design and editing of the manuscript in Chapter 4.

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Abstract

Epidemiological research suggests that up to 50% of individuals involved in low speed rear impact collisions develop acute onset low back pain. Given that little information is known about the low back injury mechanisms as a result of these collisions the overarching goal of this thesis was to explore low to moderate velocity rear-end collisions as a potential low back injury mechanism. Using a combination of data mining, *in vivo* and *in vitro* mechanical testing of porcine functional spinal units, the global purposes of this thesis were to (i) explore the types of low to moderate velocity collisions that frequently result in claims of low back pain and injury (ii) explore the influence of low velocity rear impact collisions on peak *in vivo* joint loading, occupant pain reporting and passive tissue response of the lumbar spine, and (iii) characterize the effects of these mechanical exposures and explore facet joint capsule injury as a potential source of injury and pain generating pathways following low to moderate severity impacts. In-line with these global purposes, four independent studies were conducted, each with their own focused objectives.

Study I - Exploring Low Velocity Collision Characteristics Associated with Claimed Low Back Pain

Background: Up to fifty percent of individuals involved in low to moderate velocity collisions report low back pain. However, our understanding of the specific collision or occupant characteristics that result in such claims of low back pain remains limited.

Objectives: The primary objective of this study was to define the circumstances of low velocity motor vehicle collisions that result in litigation in Ontario with claims of low back injury.

Methods: Data for this investigation were obtained from a forensic engineering firm based in Toronto, Ontario, Canada. The database was searched and only cases with an evaluation of the injuries sustained in passenger vehicle to vehicle collisions, with a collision severity of 24 km/hour or less were included in this analysis. Each identified case was reviewed for collision characteristics, pre-existing medical conditions and injuries claimed. Descriptive statistics (mean, SD and ranges) across low back injury claims were computed for documented variables.

Results: Out the 83 cases reviewed, 77% involved a claim of low back injury. Specific to those who claimed low back injury, examination of the medical history revealed that pre-existing low back pain (LBP) or evidence of lumbar disc degeneration were particularly common with 63% of claimants either having had a history of LBP or evidence of lumbar disc degeneration, or both.

Of all low back injury claims, 97% were accompanied by a whiplash and/or whiplash associated disorder claim. For low back injury claims, a rear-end impact was the most common configuration (70% of all low back injury claims involved a rear-end collision). The majority of all low back injury claimants experienced a change in velocity of 13 km/hour or less (69%), with 42% of all low back injury claims falling between collision severities of 10 – 12 km/hour.

Conclusions: Results indicate that rear-end collision severities of 10 – 12 km/hour appear to be particularly common with respect to low back injury reporting; more severe collisions were not associated with greater low back injury reporting. This result contrasts with previously published neck injury risk data, which demonstrated the risk of neck injury symptom reporting increases with collision severity. Evidence of lumbar disc degeneration was particularly common across claimants with low back injury claims.

Study II - Characterizing Trunk Muscle Activations During Simulated Low Speed Rear Impact Collisions

Background: The internal forces generated by the musculature of the lumbar region create most of the mechanical load placed on the spine. Thus, despite the anticipated low external forces generated between the occupant and the automobile seatback during a low speed rear impact collision, increased muscle tension may influence the resultant peak joint loads experienced in the lumbar spine. Consequently, the risk of low back injury may be altered by muscle activation.

Objective: The purpose of this study was to evaluate the activation profiles of muscles surrounding the lumbar spine during unanticipated and braced simulated rear-end collisions.

Methods: Twenty-two low speed sled tests were performed on eleven human volunteers ($\triangle v = 4$ km/h). Each volunteer was exposed to one unanticipated impact and one braced impact. Accelerometers were mounted on the test sled and participants' low back. Six bilateral channels of surface electromyography (EMG) were collected from the trunk during impact trials. Peak lumbar accelerations, peak muscle activation delay, muscle onset time and peak EMG magnitudes, normalized to maximum voluntary contractions (MVC), were examined across test conditions.

Results: While not statistically significant, bracing for impact tended to reduce peak lumbar acceleration in the initial rearward impact phase of the occupant's motion by approximately 15%. The only trunk muscles with peak activations exceeding 10% MVC during the unanticipated impact were the thoracic erector spinae. *Time of* peak muscle activation was slightly longer for the unanticipated condition (unanticipated = 296 ms; braced = 241 ms).

Conclusions: Results from this investigation demonstrate that during an unanticipated low speed rear-end collision, the peak activation of muscles in the lumbar spine are low in magnitude. As such, muscle activation likely has minimal contribution to the internal joint loads that are experienced in the lumbar intervertebral joints during low speed rear impact collisions.

Study III - Characterizing In Vivo Mechanical Exposures of the Lumbar Spine During Simulated Low Velocity Rear Impact Collisions

Background: Historically, there has been a lack of focus on the lumbar spine during rear impacts because of the perception that the automotive seat back should protect the lumbar spine from injury. As a result, there have been no studies involving human volunteers to address the risk of low back injury in low velocity rear impact collisions.

Objectives: The primary objectives of this study were to explore lumbar kinematics and joint reaction forces in human volunteers during simulated rear impact collisions and to examine the influence of lumbar support on the peak motion and forces experienced in the lumbar spine. A secondary objective was to evaluate lumped passive stiffness changes and low back pain reporting after a simulated rear impact collision

Methods: Twenty-four participants (12 male, 12 female) were recruited. A custom-built crash sled was used to simulate unanticipated rear impact collisions, with a change in velocity of approximately 8 km/h. Randomized collisions were completed with and without lumbar support. Measures of passive stiffness and flexion-relaxation-ratio (FRR) were obtained prior to impact (Pre), immediately post impact (Post) and 24 hours post impact (Post-24). LBP reporting was monitored over the next 24 hours leading up to the final Post-24 measures. For collision simulations inverse dynamics analyses were conducted, and outputs were used to generate estimates of peak L4/L5 joint compression and shear. From the passive trials, lumbar flexion/extension moment-angle curves were generated to quantify time-varying changes in the passive stiffness of the lumbar spine, Post and Post-24 relative to Pre. FRRs were computed as the ratio of thoracic erector spinae and lumbar erector spinae muscle activation in an upright posture to muscle activation in a flexed position

Results: Average [± standard deviation] peak L4/L5 compression and shear reaction forces were not significantly different without lumbar support (Compression = 498.22 N [±178.0]; Shear = 302.2 N [± 98.5]) compared to with lumbar support (Compression = 484.5 N [±151.1]; Shear = 291.3 N [±176.8]). Lumbar flexion angle at the point of peak shear was 36 degrees [±12] without

and 33 degrees [±11] with lumbar support, respectively, with 0 degrees being the lumbar posture in upright standing. No participants developed clinically significant levels of LBP after impact. Time was a significant factor for the length of the low stiffness flexion and extension zone ($p = 0.049$; $p = 0.035$), the length of the low stiffness zone was longer in the Post and Post-24 trial for low stiffness flexion and longer in the Post-24 for low stiffness extension.

Conclusions: Findings demonstrate that during a laboratory-simulation of an unanticipated 8 km/hour rear-impact collision, young healthy adults do not develop LBP. Lumbar support did not significantly influence the estimated L4/L5 joint reaction forces. Changes in the low stiffness portion of the passive flexion/extension curves were observed following impact and persisted for 24 hours. Changes in passive stiffness may lead to changes in the loads and load distributions within the passive structures such as the ligaments and intervertebral discs following impacts.

Study IV - Exploring the Interaction Effects of Impact Severity and Posture on Vertebral Joint Mechanics

Background: To date, no *in vitro* studies have been conducted to explore lumbar soft tissue injury potential and altered mechanical properties from exposure to impact forces. Typically, after a motor vehicle collision, the cause of a reported acute onset of low back pain is difficult to identify with potential soft tissue strain injury sites including the facet joint and highly innervated facet joint capsule ligament (FCL).

Objectives: The purpose of this investigation was to quantify intervertebral translation and facet joint capsule strain under varying postures and impact severities. A secondary objective was to evaluate flexion-extension and shear neutral zone changes pre and post impact.

Methods: A total of 72 porcine cervical FSUs were included in the study. Three levels of impact severity (4g, 8g, 11g), and three postures (Neutral Flexion and Extension) were examined using a full-factorial design. Impacts were applied using a custom-built impact track which simulated impact parameters similar to those experienced in low to moderate speed motor vehicle collisions. Passive flexion-extension and shear neutral zone testing were completed immediately prior to and immediately post impact. Intervertebral translation and the strain tensor of the facet capsule ligament were measured during impacts.

Results: A significant main effect ($p > 0.001$) of collision severity was observed for peak intervertebral translation and peak FCL shear strain ($p = 0.003$). A significant two-way interaction was observed between pre-post and impact severity for flexion-extension neutral zone length ($p =$

0.031) and stiffness (*p>0.001*) and anterior-posterior shear neutral zone length (*p = 0.047*) and stiffness (*p>0.001*). This was a result of increased neutral zone range and decreased neutral zone stiffness pre-post for the 11g severity impact (regardless of posture).

Conclusions: This investigation provides evidence that overall the peak vertebral translations observed across 4g to 11g impacts are below previously published ultimate shear failure displacements. FSU's exposed to the highest severity impact (11g) had significant NZ changes, with increases in joint laxity in flexion-extension and shear testing and decreased stiffness, suggesting that soft tissue injury may have occurred. Despite observed main effects of impact severity, no influence of posture was observed. This lack of influence of posture and small FCL strain magnitudes suggest that the FCL does not appear to undergo injurious or permanent mechanical changes in response to low to moderate MVC impact scenarios.

Study V - Characterizing the Mechanical Properties of the Facet Joint Capsule Ligament

Background: The facet joint capsule ligament (FCL) is a structure in the lumbar spine that constrains motions of the vertebrae. Previous work has demonstrated that under physiological motion the FCL is subjected to significant deformation with FCL strains increasing in magnitude with increasing flexion and extension moments. Thus, it is important to characterize the mechanical response of the FCL for investigations into injury mechanisms. Sub failure loads can produce micro-damage resulting in increased laxity, decreased stiffness and altered viscoelastic responses. Thus, the objective of this investigation was to determine the mechanical and viscoelastic properties of the FCL under various magnitudes of strain from control samples and samples that had been exposed to an impact.

Objectives: The purpose of this investigation was to quantify the mechanical properties and viscoelastic response of control and impacted FCL.

Methods: 200 tissue samples were excised from the right and left FCL of 80 porcine cervical functional spinal units (FSU's). Tissue samples were excised from FSU's obtained from **Study 4**. Twenty FCL tissue samples served as the control group. The remaining 180 FCL tissue samples were randomly obtained from FSU's that had been exposed to one of nine impact conditions (impacted tissue). Each specimen was loaded uniaxially, collinear with the primary fiber orientation. The loading protocol was identical for all specimens: preconditioning with 5 cycles of loading/unloading to 5% strain, followed by a 30 second rest period, 5 cycles of 10% strain and 1

cycle of 10% strain with a hold duration at 10% strain for 240 seconds. The same protocol followed for 30% (cyclic-30% & 30%-hold) and 50% strain (cyclic-50% & 50%-hold). All loading and unloading were performed at a rate of 2%/sec. All impacted FCL properties were compared back to controls. Measures of stiffness, hysteresis and force-relaxation were computed for the 30% and 50% strain conditions.

Results: No significant differences in stiffness were observed for impacted specimens in comparison to control (30% Control = 2.64 N/mm; 4 g = 2.20 N/mm, 8 g = 2.07 N/mm, 16 g = 2.16 N/mm)(50% Control = 5.06 N/mm; 4g = 4.60 N/mm, 8 g = 4.07 N/mm, 16 g =4.64 N/mm). Impacted specimens from the 8g Flexed and 11 g Flexed and Neutral conditions exhibited greater hysteresis during the cyclic-30% and cyclic-50%, in comparison to controls. In addition, specimens from the 8g and 11g Flexed conditions resulted in greater force relaxation for the 50%-hold conditions.

Conclusions: Results from this study demonstrate viscoelastic changes in FCL samples exposed to moderate and highspeed impacts in the flexed posture. However, it is interesting that these viscoelastic changes were not accompanied by changes in stiffness. Findings from this investigation provide novel insight and provide mechanical and viscoelastic properties of the FCL both in control and impacted scenarios.

Global Summary: Findings from this thesis demonstrate that (i) rear-end collision severities of 10 – 12 km/hour appear to be particularly common with respect to low back injury reporting (ii) during a laboratory-simulation of an unanticipated 8 km/hour rear-impact collision, young healthy adults do not develop LBP, however, changes in the low stiffness portion of the passive flexion/extension curves were observed following impact and persisted for 24 hours and (iii) the observed peak displacements in porcine functional spinal units exposed to varying impact severities are below ultimate shear failure displacements and does not support a lumbar spine injury mechanism resulting in acute traumatic bone fractures and/or acute traumatic IVD herniations in previously "healthy" tissues. Overall, the small FCL strain magnitudes during impacts and unchanged FCL mechanical properties post-impact suggest that the FCL does not undergo injurious or permanent mechanical changes in response to low to moderate MVC impact scenarios. Collectively, the findings from this thesis indicate that there are no direct mechanical changes that would indicate the high incidence of low back pain reporting following low to

moderate severity rear-end motor vehicle impacts. However, changes in passive tissue properties were observed, and if persistent over time, may predispose individuals to secondary pain pathways. It is also important to note that this thesis tested healthy conditions and the results do not directly apply to pre-existing LBP cases being exposed to the same impacts.

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Chapter 1: General Introduction

1.1 Scope of the Problem

The underlying mechanisms linking the forces in a low speed rear-end car accident with low back pain are unclear, as there is limited biomechanical data relating low back injury mechanisms to the forces and motions resulting from a low speed impact. Frequently, unlikely claims of low back pain are reported after a low speed collision, even with little vehicle damage, lack of physical evidence upon examination and negative radiographic evidence. The medical community strives to treat and rehabilitate these injuries, however, a major limitation in such cases is the lack of knowledge of injury mechanisms that link to the pain and pathology associated with such collisions. Given the personal and societal costs of such claims, it is important to explore the possible mechanistic factors that may lead to tissue injury during a low speed car accident and the potential link to pain generation.

To date, the majority of epidemiological and laboratory studies on low speed rear impacts have focused on the neck. However, there is increasing concern about the potential for injury risk to the low back. Epidemiological research has demonstrated that up to 50% of individuals involved in a low speed rear impact may develop low back pain (Fast et al., 2002). In contrast, in a review of laboratory studies simulating low speed rear impacts only one volunteer out of 364 human participants reported a low back complaint. A limited number of previous laboratory investigations have examined the motion and joint loads in a cadaver (Fast et al., 2002) and instrumented test dummies (Gates et al., 2010; Gushue et al., 2001) and have demonstrated that the peak exposures in the lumbar spine are well below existing injury reference values and within the range of loads experienced in manual materials handling jobs. While laboratory simulations point in the direction that the exposures during low speed collisions do not cause the potential for acute injury, the continuous reporting of low back pain after low speed collisions demonstrates a clear need to investigate if a link exists between low speed motor vehicle collisions and low back injury.

1.2 Significance

Acute onset of low back pain frequently occurs following a low speed collision. Given that little information is known about the injury mechanisms as a result of these collisions the overarching

goal of this thesis was to explore low to moderate speed rear-end collisions as a potential low back injury mechanism. Using a combination of data mining, *in vivo* and *in vitro* work, the objective of this thesis was to understand the injuries that may occur as a result of a low speed rear-end collision.

1.3 Thesis Overview

The purpose of this thesis was to improve our understanding of underlying low back injury mechanisms that may result from low to moderate speed rear impacts. To achieve this, I completed four studies (Figure 1.1) aimed at eliciting these injury mechanisms through expanding the knowledge of collision characteristics that result in low back injury reporting and characterizing the *in vivo* and *in vitro* mechanical exposures to the lumbar spine during simulated rear impact collisions.

Study one assessed the types of collisions that result in low back injury claims. Data was collected from a Forensic Engineering firm, -30- Forensic Engineering, to characterize and document the dynamics of low velocity motor vehicle collisions that result in low back injury claims. This investigation determined specific collision and occupant characteristics that have a higher likelihood of resulting in low back injury claims.

Study two built on the collision characteristics found in **Study 1** and investigated trunk muscle activations in human volunteers during simulated rear impact collisions. This investigation determined that during an unanticipated low speed rear-end collision, the peak activation of muscles in the lumbar spine are low in magnitude. As such, muscle activation likely has minimal contribution to the internal joint loads that are experienced in the lumbar intervertebral joints during low speed rear impact collisions. The findings from this study justified the use of a simplified joint model in estimating the joint loads in the lumbar spine in **Study 3**.

Study three built on the collision characteristics found in **Study 1** and results from **Study 2** and investigated the lumbar kinematics and kinetics in human volunteers during a simulated rear impact collision. In addition, this study also examined the influence of lumbar seat support on the motion and forces experienced in the lumbar spine during the same magnitude simulated rear impact collision. Lastly, 24 hour pain reporting, as well as lumped passive lumbar spine stiffness was assessed to investigate potential injury mechanisms.

Study four built on the findings from **Study 3** and impacted (forces obtained from **Study 3**) porcine cervical functional spinal units using a custom built impact track. This investigation examined the effects of collision severity and posture on vertebral joint mechanics, as well as passive stiffness testing pre and post impact simulation.

Study five harvested facet capsule ligament tissue samples from **Study 4** to investigate the effect of collision severity and posture on the mechanical properties of the facet capsule ligament under uniaxial strain. Previous work has demonstrated that sub failure loads can produce micro damage to a tissue, which can result in a variety of altered mechanical properties. Samples underwent cyclic, uniaxial tensile loading and force-relaxation testing using a custom commercial apparatus designed to apply tensile loads to biological tissues. Results were compared back to control, non-impacted, samples.

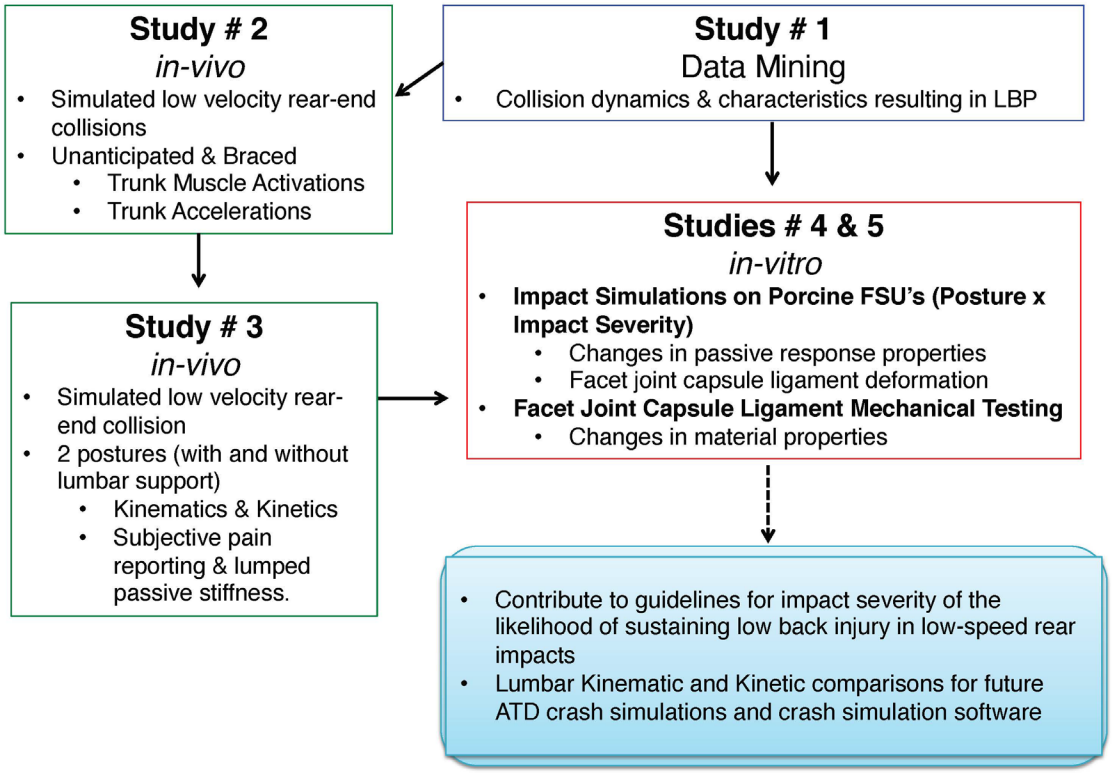

Figure 1.1: Flow chart of the proposed studies for this thesis and anticipated contributions.

1.4 Global Thesis Objectives

The global objective of this thesis was to investigate low velocity rear-end impacts as a potential injury mechanism in the lumbar spine. Collectively, across this thesis, the following injury

assessment reference values (IARVs) were used to explore injury potential in the lumbar spine during exposure to simulated low to moderate severity impacts—3400 N for lumbar spine compression and 1000 N for lumbar spine shear. The compression IARV was established based on the National Institute of Occupational Safety and Health (NIOSH) recommendation of a compressive force threshold of 3400 N during occupational lifting. The shear IARV is based on previous research conducted by McGill et al. (1998) who recommends a maximum limit of 1000 N of lumbar spine shear force during occupational exposures (representing approximately 33% of the ultimate shear failure tolerance observed in cadaveric specimens). This limit was based on work conducted on cadavers which revealed that under anterior shearing forces, the average male cadaver can tolerate approximately 1700 - 2900 N of shear at a loading rate of 50 mm/s (Cripton et al., 1995). McGill and colleagues stated that the limit is further justified from results using a pig model (controlling for age, exercise level, diet and genetic homogeneity), finding very similar tolerance values to human cadavers and that the ultimate anterior shear and posterior shear tolerance values were similar in magnitude (McGill et al., 1998). The chosen occupational IARV's are intended for repeated exposures and are well below any lumbar spine tissue tolerance values. Ultimate tissue tolerances for functional spinal units in the lumbar spine range between 6150 to 13800 for compression (across various postures and age groups) and 1710 to 3538 N for shear (across various postures, loading rates and anterior and posterior shear directions) (Brinckmann et al., 1988; Cripton et al., 1995; Gallagher et al., 2010; Howarth and Callaghan, 2012; Hutton et al., 1979; Yingling and McGill, 1999). In addition, the IARV's utilized represent a very conservative limit given that these values are typically used for assessments involving physiological motion (i.e. repetitive lifting assessments) which results in significantly lower loading rates than an impact event. Rate of loading has been demonstrated to increase a specimens' ultimate load tolerance in both compression and shear (Howarth and Callaghan, 2012; Hutton et al., 1979; Yingling and McGill, 1999)

The following specific objectives guided the experimental approach described in this thesis.

1. To characterize and document the dynamics of low velocity motor vehicle collisions and occupant characteristics that result in low back injury claims (**Study 1**).
2. To establish an understanding of the muscle activations, forces and motions in the low back from low velocity rear impact vehicle collisions and their relationship to low back injury (**Study 2, 3 & 4**).

3. To explore spine mechanical property changes following simulated rear-end collisions (**Study 3 & 4**).
4. To explore whether the facet joint contributes to low back injury after a simulated rear-end collision (**Study 4 & 5**).
5. To investigate the mechanical properties of the facet joint capsule ligament and the effect of exposure to various simulated collision severities on the mechanical properties of the ligament (**Study 5**).

1.5 Global Hypotheses

All hypotheses for each specific study are stated as *null hypotheses* (H_0) such that each hypothesis can be tested on the basis of data/research. An explanation is provided following each hypothesis statement below. Each hypothesis will be revisited in Chapter 7.

Study #1 (Data Mining): This study documented and characterized the types of low velocity collisions that result in low back injury claims. Data was collected and mined from -30- Forensic Engineering. Results from this study guided independent variables for **Study 2 & 3**.

Study 1 Hypotheses:

H_o: There will be no difference in reports of low back injury claims across low velocity collision severities (up to 25 km/hour).

The total number of low back injury claims is expected to increase with increasing collision severity. Claims of whiplash type associated injury have been documented to increase with increasing collision severity (Krafft et al., 2005; Watanabe et al., 2000). Previous epidemiological reports have found a high incidence of low back injury claims accompanying whiplash type injuries (Beattie and Lovell, 2010). Therefore, it is expected that low back injury claims will follow a similar trend to that of whiplash type injuries.

H_o: There will be no difference in reports of low back injury claims across collision types.

It was expected that low back pain reports would be greatest in rear-end collisions. This has been documented previously for whiplash type injuries (Watanabe et al., 2000) and low back injury has been shown to accompany whiplash associated disorders (Beattie and Lovell, 2010).

Study #2 (in vivo): Crash test simulations were conducted on human participants. Each volunteer was exposed to one unanticipated (relaxed) and one braced impact. A total of twelve channels of surface electromyography were used to monitor individual bilateral trunk muscle activity. Peak lumbar accelerations, peak muscle activation following impact, and peak muscle activation delay times were examined. Results from this study helped guide modelling techniques for **Study 3.**

Study 2 Hypotheses:

H_o: There will be no change in peak muscle activation across collision conditions.

It was expected that bracing for impact will result in increased muscle activation prior to impact. Changes in muscle activation patterns between braced and unanticipated collision simulations have been demonstrated in the neck (Siegmund et al., 2003).

H_o: There will be no change peak lumbar accelerations across impact conditions.

It is anticipated that there will be a change in lumbar accelerations across impact conditions. Bracing for impact has been shown to significantly change joint kinematics during low-speed sled tests. This has been demonstrated with lower angular head accelerations in male participants, and smaller head retractions in female participants due to pre-impact bracing of the cervical spine muscles (Siegmund et al., 2003). In low speed frontal collisions, bracing has been shown to reduce the forward excursion of the knees, hips, elbows, shoulders, and head (Beeman et al., 2011). Bracing has also been found to reduce peak shoulder and retractor belt forces in a 50^{th} percentile male population exposed to low speed frontal collisions (5 and 10 km/hour) (Kemper et al., 2014).

Study #3 (*in vivo*): Crash test simulations were conducted on human participants with and without a lumbar support to estimate lumbar spine forces during crash test simulations. Changes in lumbar lumped passive stiffness were quantified pre, post and post-24 hours from the simulated collision. Results from this study helped guide independent variables for *in-vitro* testing for **Study 4.**

Study 3 Hypotheses:

H_o: There will be no change in lumbar spine kinetics across seating conditions.

The use of lumbar support will influence lumbar spine joint loading in a simulated low velocity rear impact. Lumbar supports have been shown to impact vertebral joint rotations, increase lumbar lordosis and decrease low back pain discomfort reporting (De Carvalho and Callaghan, 2015, 2012). Based on the differences in lumbar spine posture, it is

anticipated that the use of lumbar support will also change the kinetics experienced in the lumbar spine during the simulated low velocity collision.

H_o: There will be no change in lumped passive lumbar spine stiffness.

It is anticipated that there will be a change in lumbar spine lumped passive stiffness post collision. Previous investigations (*in vivo*) have demonstrated changes in lumped passive stiffness with prolonged and repetitive flexion postures (Beach et al., 2005; De Carvalho and Callaghan, 2011; Parkinson et al., 2004). In addition, previous *in-vitro* investigations have demonstrated decreased stiffness, increased neutral zone and range of motion with below failure type impacts (Ivancic et al., 2005; Panjabi et al., 2005, 1989). Therefore, it is anticipated that with a sudden impact, changes in lumped passive stiffness will occur.

Study #4 (*in-vitro*): Impact simulations were conducted on cervical porcine functional spinal units using a custom built impact track at peak accelerations of 4, 8 and 11g in three different starting postures (Neutral, Flexion and Extension). Changes in neutral zone range and stiffness, as well as facet strain and vertebral translation during impacts were computed.

Study 4 Hypothesis:

H_o: Peak vertebral translation of the porcine FSU, during each simulated collision, will not be influenced by varying collision severity or posture.

It is anticipated that increases in collision severity will result in increased peak vertebral translation.

H_o: There will be no change in passive flexion-extension and shear translation testing pre and post collision.

It was anticipated that changes in passive flexion-extension and shear neutral zone range will occur. It is anticipated that soft tissue damage will occur with increasing collision severity and increasing flexed postures. Tissue damage will present as increases in neutral zone range, decreased stiffness and increases in joint laxity. This is based on previous cervical whiplash testing on human cadaveric specimens, which has demonstrated statistically significant changes in neutral zone and range of motion testing with increasing collision severity (Ivancic et al., 2005; Panjabi et al., 2005). In addition, flexed postures in a porcine lumbar spine model have been demonstrated to result in a decrease in ultimate compressive (Gunning et al., 2001) and shear tolerance (Howarth and Callaghan, 2012).

Study #5 (*in-vitro*): Uniaxial tensile testing will be conducted on regional sections from the facet capsule ligament. The mechanical and viscoelastic properties of healthy facet capsular ligament will be investigated under uniaxial cyclic loading and during force-relaxation testing. In addition, specimens that have been through a simulated collision loading protocol (Study 4) will also be tested. This study will identify the effects of exposure to a sudden impact on the mechanical and viscoelastic properties of excised facet joint capsule ligament with comparisons to healthy non-impacted tissue.

Study 5 Hypothesis:

H_o: There will be no difference in mechanical properties between healthy control samples and impacted samples.

It is anticipated that specimens that have been through the impacted protocol will exhibit changes in stiffness and force-relaxation responses (Nelson-Wong et al., 2018; Panjabi et al., 1999, 1996)

Chapter 2: Review of Literature

This chapter has been organized into 4 sections. The first section will provide a brief overview of the basic anatomy and function of the osteoligamentous human lumbar spine. The next section includes an overview of epidemiological and laboratory evidence of low back pain reporting and injury in low to moderate velocity rear-end collisions. This section also includes a brief review of the two most commonly used Anthropometric Testing Devices utilized in low to moderate velocity collision testing. The next section provides an overview of potential injury mechanisms to the lumbar spine in a low to moderate velocity rear end collision. The final section includes a review of important methodological consideration pertaining to this proposed work.

2.1 Basic Anatomy of the Osteoligamentous Human Lumbar Spine

The lumbar spine consists of five lumbar vertebrae. Each of the five vertebrae are named L1 to L5 in descending order. The function of the vertebrae is to support the weight of the body, protect the spinal cord and nerve roots and provide attachment for surrounding muscles. A human osteoligamentous, vertebral joint is comprised of two adjacent vertebrae - the intervertebral disc between them and a series of ligaments. An isolated vertebral joint is often called a functional spinal unit (FSU) (Howarth and Callaghan, 2013a; Oxland et al., 1991; Parkinson and Callaghan, 2007). This joint is capable of flexion/extension, lateral bend, axial twist motions and has the capacity to tolerate shear and compressive loading.

Each vertebra making up an FSU can be divided into three main parts: (1) the vertebral body (2) the pedicles and (3) the posterior elements (Figure 2.1).

The **vertebral body** is primarily composed of cancellous bone surrounded by a thin layer of cortical bone. The superior and inferior surfaces of the vertebral body are capped with cartilaginous endplates composed of hyaline cartilage and fibrocartilage (Adams et al., 2002). These endplates are the junctions between adjacent vertebrae and are the attachment points between the vertebral body and the intervertebral disc (IVD) (McGill, 2007).

The **posterior elements** of the vertebrae are composed of a number of defining boney features including, the spinous process, transverse processes, lamina, accessory process, as well as the facets (Figure 2.1). They have a shell of cortical bone but contain a cancellous bone core (McGill, 2007). With the exception of the facets, the primary function of the posterior elements

(spinous process, transverse process, lamina and accessory process) is to provide an area for muscle attachment and protect the spinal cord.

The **pedicles** are the boney connection between the vertebral body and the posterior elements (Figure 2.1). Any forces sustained by the posterior elements of the vertebrae are transmitted to the vertebral body through the pedicles (McGill, 2007).

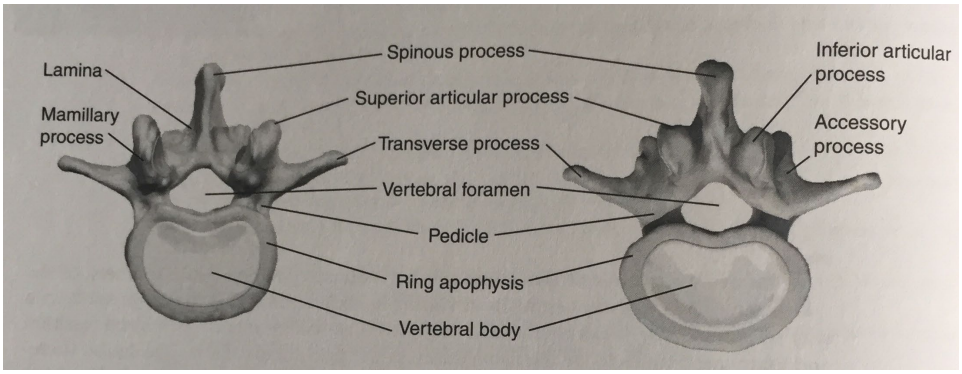

Figure 2.1: The Boney landmarks of a typical lumbar vertebrae. (Image taken from McGill, 2007).

2.1.1 Facets and Facet Joint Capsule

The superior and inferior articular processes (also known as facets) are covered with articular cartilage (Adams et al., 2002). Junctions between the superior facets of the caudal vertebra and the inferior facets of the cephalad vertebra (facet joint) of an FSU are surrounded by an articular capsule creating a synovial joint capsule (Hukins and Meakin, 2000), which forms the facet joint. The articular capsule surrounds the joint and is bounded laterally by the ligamentum flavum and medially by an extension of articular cartilage. To facilitate a gliding movement the facets are lubricated by a film of synovial fluid, which is retained by a synovial membrane that attaches to the articular capsule (Adams et al., 2002). Menisci create a space between the articulating facets of the joint.

The facet joint is one of the structures in the lumbar spine that constrains motions of vertebrae during spine loading and is innervated with mechanically sensitive neurons. They provide a locking mechanism between consecutive vertebrae and are designed to resist axial rotation and forward sliding of the vertebrae.

2.1.2 Intervertebral Disc

Each intervertebral disc is composed of three components; (1) The annulus fibrosus, (2) nucleus pulposus (NP) and (3) the endplates.

The **annulus fibrosus** is arranged into a series of 15-25 lamellar layers arranged in concentric rings surrounding the nucleus pulposus (Tampier, 2006). The inner layers of the annulus are composed primarily of type II collagen and proteoglycans while the outer layers are composed mainly of type I collagen. The collagen fibers of each layer are orientated approximately 45-65 degrees from vertical and the fibers between each layer are orientated approximately 130 degrees relative to each other (Tampier, 2006).

Nucleus pulposus: In healthy young individuals the nucleus pulposus has a gel like consistency. The nucleus pulposus consists mainly of type II collagen fibers and is rich in proteoglycans. It functions as a hydrodynamic system and attracts large amounts of water. The type II collagen is thought to play an important role in resisting high compressive loads, pressurizing the endplates vertically and the annulus laterally (Hayes et al., 2001). Proteoglycan is a molecular complex consisting of a protein core with many side chains of negatively charged glycosaminoglycan molecules. The entire proteoglycan molecule is a negatively charged hydrophilic molecule, thus attracting water into the nucleus and maintaining its hydrostatic pressure.

2.1.3 Ligaments

The complete set of ligaments for an FSU include: the anterior longitudinal ligament, posterior longitudinal ligament, ligamentum flavum, interspinous ligament, supraspinous ligament and intertransverse ligament (Figure 2.2).

The majority of the ligaments in the lumbar spine are composed of collagen fibers (Hukins and Meakin, 2000). The only ligament excluded is the ligamentum flavum because it contains primarily elastin fibers (McGill, 2007). Typically, ligaments surrounding the lumbar spine are able to resist motion only when elongated from their resting length. Ligaments will only resist motion of the bones to which they connect after being elongated outside their toe region (Myklebust et al., 1988). Typically, ligaments are most effective in carrying loads along the direction in which the fibers run.

The ligaments of the lumbar spine have a number of different functions. They must allow

adequate physiological motion between vertebrae and yet constrict motion to protect the spinal cord. Ligaments also share with muscles the role of providing stability to the spine within physiologic ranges of motion (White and Panjabi, 1990). Finally, they must also protect the spinal cord in traumatic situations in which high loads are applied at fast speeds (White and Panjabi, 1990). When the lumbar spine flexes all ligaments except the anterior longitudinal ligament are stretched (Panjabi et al., 1982).

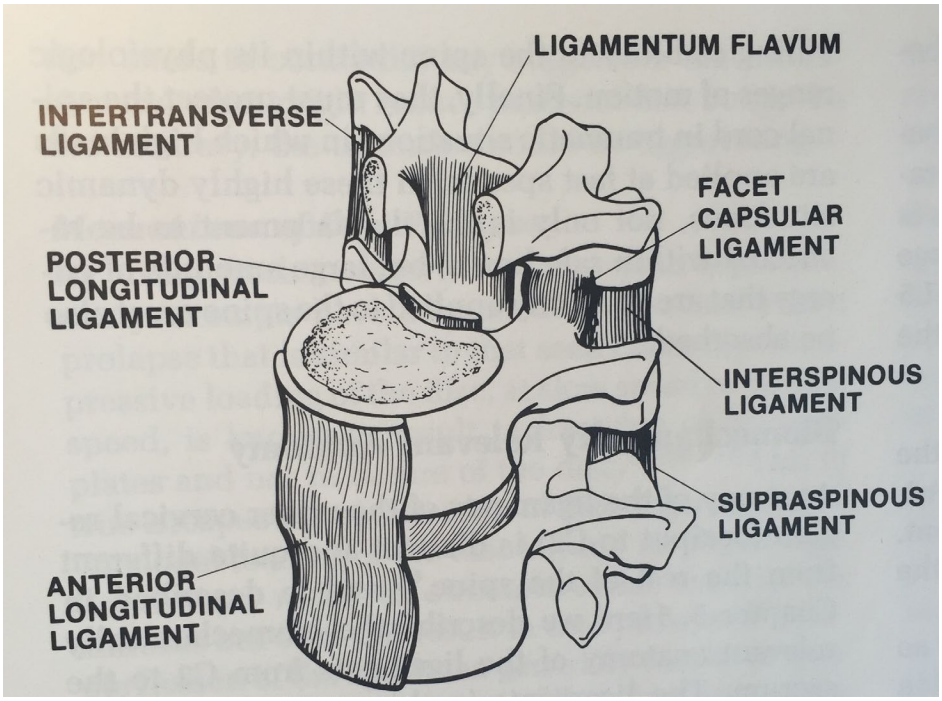

Figure 2.2: The ligaments of the lumbar spine (Image taken from White & Panjabi, 1990). Refer to section 2.6.1.1 for methodological considerations for using a porcine animal model.

2.2 Low Speed Collisions and Evidence of Low Back Pain Reporting

For over fifteen years, researchers have attempted to examine the link between low speed rear impact motor vehicle collisions and the risk of injury through epidemiological and laboratory studies. A large majority of these studies have focused on the link between low speed rear-end collisions and whiplash type injuries in the neck (Castro et al., 1997; Dolinis, 1997; Mayou and Bryant, 1996; Ono and Kanno, 1996). However, there is increasing concern about the potential for injury risk to the low back. Current epidemiological research is conflicting. Some research has demonstrated that up to 50% of individuals involved in a low speed rear impact may develop an acute onset of low back pain, while other studies indicate low back reporting following low speed

rear-end collisions is minimal (Beattie and Lovell, 2010; Magnússon, 1994; Richards et al., 2006; Yang et al., 2013).

2.2.1 Epidemiological Evidence of Low Back Pain Reporting

Epidemiological studies suggest there is an association between developing a whiplash neck injury and reporting low back pain. Several studies have documented the presence of low back pain in conjunction with whiplash injury to the neck, following rear-end collisions, however none of these studies have focused on the low back (Mayou and Bryant, 1996; Radanov et al., 1991; Schrader et al., 1996). Many studies have demonstrated that the largest number of motor vehicle associated injuries occur in rear impacts and with that, a whiplash neck injury is the most frequent type of injury (Watanabe et al., 2000). Over 90 percent of whiplash neck injuries occur at collision severities below 25 km/hour, indicating that whiplash type neck injuries are characteristic of low speed rear impacts (Watanabe et al., 2000). This suggests that low back pain reporting might also be characteristic of lower speed rear impact collisions.

The reported incidence of low back pain with a whiplash injury has been reported to range between 40 to 60% of all whiplash cases (Beattie and Lovell, 2010; Magnússon, 1994). Magnusson (1994), found that 47.4% (18 out of a total of 38 patients) of late whiplash syndrome patients had low back pain and tender points on the low back. In 66.7% (12 out of 18 patients) of these low back pain patients, low back pain was made worse by specific activities. In 27.8% (5 out of 18 patients), there was an associated motion segment failure leading to the diagnosis of chronic mechanical low back pain. In whiplash, follow up studies found chronic low back pain is one of the most common complaints in addition to chronic neck pain (Gay and Abott, 1953). Chapline et al. (2000), completed a comprehensive cross-sectional study investigating neck pain and head restraint position in rear-end collisions. The authors analyzed 585 police reported rear-end crashes with 319 female drivers and 266 male drivers. Self-reported pain to other body areas other than the neck were also reported and it was found that 26% of female drivers and 18% of male drivers reported back pain following rear impacts (Chapline et al., 2000). To date, only one study has looked to investigate low back pain complaints after motor vehicle collisions without an associated whiplash injury and found that out of 800 claimants only 5% of claimed low back pain cases occurred without a subsequent whiplash injury claim, with the majority of these individuals having a pre-existing low back complaint (Beattie and Lovell, 2010). It was also reported that low back pain incidence following a collision was independent of collision severity or the direction of

collision forces (Beattie and Lovell, 2010).

Conversely, some studies do not demonstrate a relationship between low speed rear-end collisions and low back pain reporting. Yang and colleagues (2013), examined the incidence of spine injury in rear impact collisions across all collision severities. Data were taken from the National Automotive Sampling System for rear-end collisions and over 7500 cases were analyzed. The analysis indicated that of all cases analyzed approximately 6.7% of those cases reported low back pain and most of these reports were associated with musculoskeletal strains or sprains (Yang et al., 2013). Richards et al., (2006), performed a review of available frontal crash data and found that there was less than a 2% risk of moderate injury or greater in the lumbar or thoracic spine for belted occupants exposed to a collision severity of less than 20 km/hr (Richards et al., 2006). While this report may not be directly applicable to rear-end collisions, it does demonstrate the reported potential risk to the lumbar spine in low to moderate speed collisions may be lower than reported in other studies.

The association of low back pain with low speed rear impact collisions is unclear with studies reporting a range of very low to high reporting rates. There are a number of limitations associated with the studies completed to date. First, each study assessed a person's low back pain reporting at different stages after the collision, therefore it is unknown if the collision itself resulted in an acute onset of low back pain or if modifications to movement from the collision resulted in low back pain. Second, collision dynamics of the population assessed have not been thoroughly documented. Often the severity of the motor vehicle collision or the direction of collision forces are not reported or the population is classified into one larger group for example, "low speed collisions or low to moderate severity". Finally, low back pain is a common complaint; often studies do not report if prior low back injury, degeneration or low back pain complaints existed prior to the collision.

2.2.2 Laboratory Collision Simulations – Low Velocity Rear Impacts

2.2.2.1 Considerations for Laboratory Testing: Anthropometric Testing Devices

Anthropometric testing devices (ATDs) have been used by the automotive industry to assess vehicle performance since the 1940's. In 1976, General Motors introduced the Hybrid III 50th percentile male ATD for use in both frontal and rear impacts and it is now the current standard for the assessment of automobile performance worldwide. More recently, in addition to the 50th percentile male there is a family of the Hybrid III ATDs that is available for testing, including a

three-year-old child, six-year-old child, ten-year-old child, small female, and large male (Figure 2.3). Injury Assessment Reference Values (IARVs) have been developed for all scaled versions of the Hybrid III ATD. Briefly, IARVs were originally introduced in 1983 when General Motors released the limit values that they impose on the Hybrid III ATD measurements. The values were chosen such that if the IARVs were not exceeded in a prescribed test, then the risks of the associated injuries would be unlikely for that size occupant in the accident condition being simulated (less than a 5% chance). Since the introduction of these values, the IARVs have been continuously updated based on biomechanical studies that have been published and scaled to provide IARVs for the Hybrid III family of ATDs to relate ATD measurements in crash simulations to the likelihood of injury in human occupants. This relies on the Abbreviated Injury Scale (AIS), which is an anatomical-based coding system used to classify injuries from minor (AIS 1) to maximum (AIS 6). The scale is used for specific body regions including the head, face, neck, thorax, abdomen, spine, upper extremity, lower extremity and external or other.

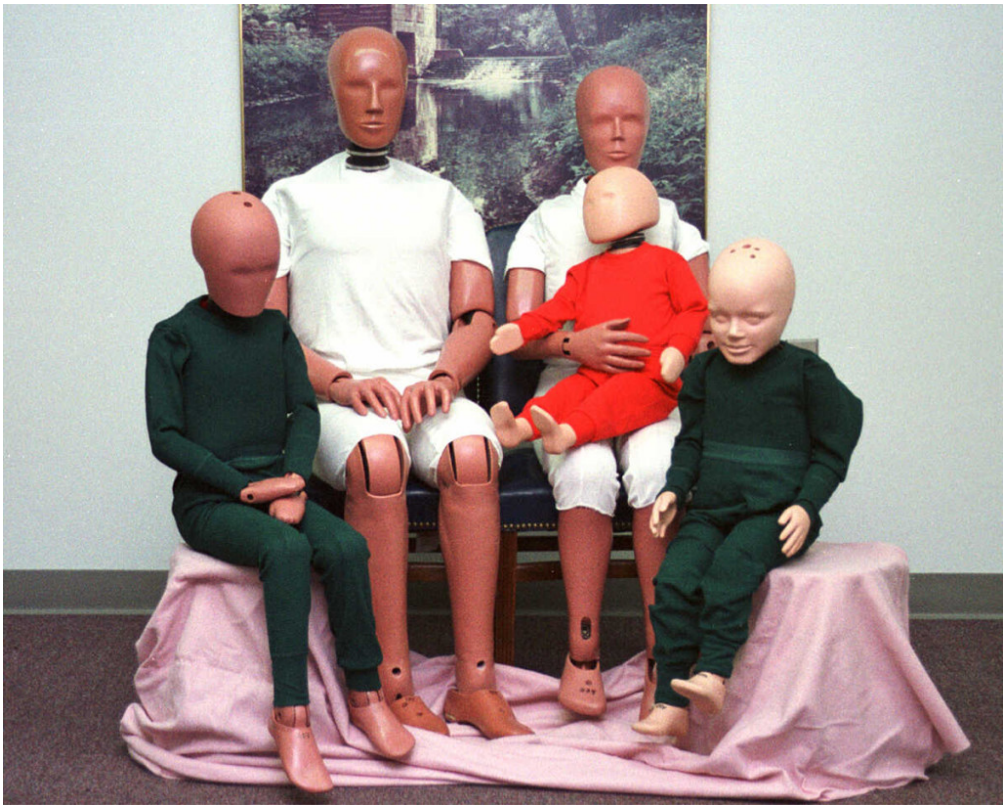

Figure 2.3: The Hybrid III family, including a three-year-old child, six-year-old child, ten-year old child, small female, and large male.

Over the last decade, additional ATDs have been designed to specifically evaluate rear impact performance in automobile collisions. One of these ATDs is known as the Biofidelic Rear Impact Dummy (BioRID). This ATD was introduced in 1999 after being developed at Chalmers University of Technology. The BioRID II (the most current version of the ATD) uses many of the same components found in the Hybrid III, but the BioRID II has a modified Hybrid III pelvis and lumbar spine assembly.

2.2.2.1.1 Hybrid III vs. BioRID II Lumbar Spine Assembly

A major difference between the makeup of the Hybrid III and the BioRID II is the makeup of the lumbar spine. In the BioRID, the spine assembly consists of seven cervical, twelve thoracic and five lumbar "vertebrae". Other than at T1, all the vertebrae are made of plastic and are connected with pins at each joint (Figure 2.4). The pins at each vertebrae keep the vertebrae connected along the modeled spine and allow for rotations about each joint. The pins are made to somewhat represent substitutes for facet joints in the human spine. Rubber pads are attached at the top of each vertebra to simulate compression resistance during flexion and extension (Gates et al., 2010). The BioRID has a 6 channel lumbar spine load cell, which inserts at the location of the L5 vertebrae in the spine model, orientated horizontally on top of a pelvis (Figure 2.4).

In the Hybrid III ATD the lumbar spine is a curved continuous piece of molded material. It is instrumented with two cables that run through the spinal cavity to provide lateral stability. In contrast to the lordotic curvature found in the BioRID II, the Hybrid III lumbar spine is curved forward in a kyphotic curvature. This kyphotic curvature is there to represent "humanlike slouch" to the seated ATD. The Hybrid III lumbar spine attaches to the thoracic spine and pelvis through steel assemblies. A 3-channel lumbar spine load cell inserts between the lumbar and pelvis sections of the Hybrid III and is orientated at a 22 degree angle facing posterior downward (Figure 2.4).

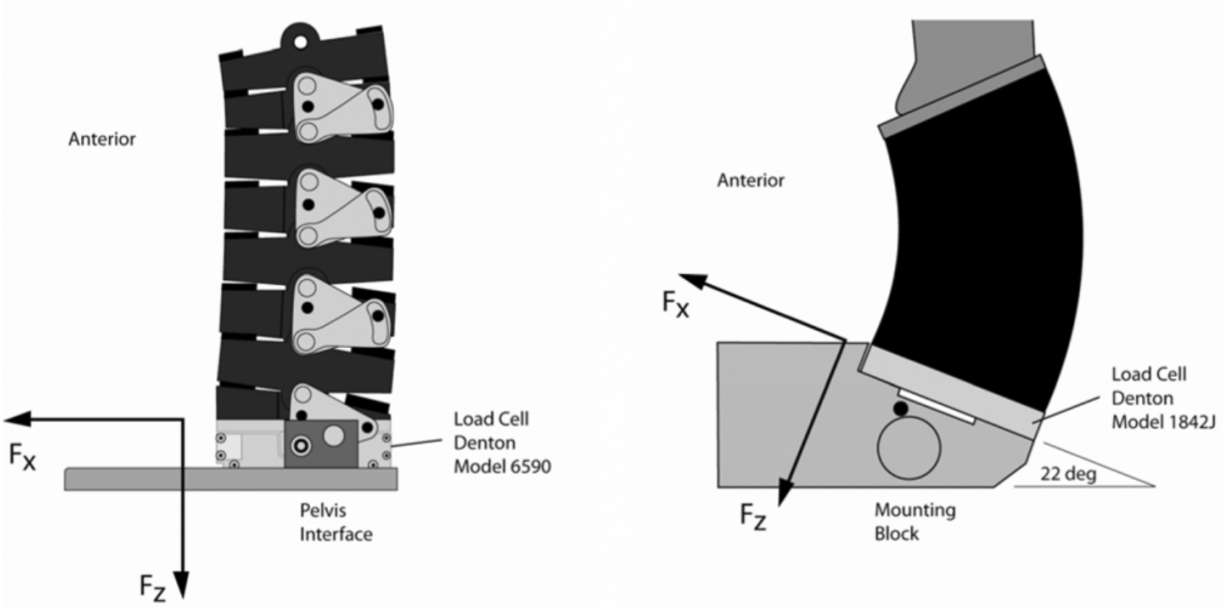

Figure 2.4: The BioRID II (left) and Hybrid III (right) lumbar spines. The images also display the directions of force measurement for shear (Fx) and axial loading (Fz). The pelvis for the BioRID II includes a horizontal plate referred to the pelvis interface. It is through this pelvis interface that the lumbar spine assembly and the load cell attaches to the pelvis. The Hybrid III pelvis includes a posteriorly angled surface called the "mounting block" and it is here the lumbar spine and load cell attach at an angle of 22 degrees. Note that based on the difference in orientation of each modeled lumbar spine, each of the ATDs have a different force measurement orientation (Image taken from Gates et al. 2010).

2.2.2.2 Validation

Validation of the BioRID has mainly included kinematics of the ATD response and how they compare to human subjects under low velocity impact simulations. It is important to note that the majority of these comparisons have focused on the response of the neck. Kinematic comparisons have been made between the BioRID, Hybrid III and human volunteers in low severity rear impacts (Davidsson et al., 2001; Pietsch et al., 2003), and between the BioRID and Hybrid ATD at moderate to high severity rear impacts (Davidsson et al., 2001). All comparisons concluded that the BioRID ATD provided superior kinematic biofidelity over the Hybrid III in low to moderate rear impact simulations. To date, only one collective study has compared the lumbar kinetics of the Hybrid III ATD and BioRID ATD in low to moderate velocity rear impact crashes (Gates et al., 2010; Welch et al., 2010). The ATDs were positioned in paired front row bucket seats and were restrained by a 3-point safety belt and subjected to rear impacts of collision severities of 2.2, 3.6, 5.4 and 6.7 m/s/ (7.92, 12.96, 19.44 and 24.12 km/hour). Head accelerations and rotation rates,

upper and lower neck forces and moments, T1 accelerations, lumbar forces and moments and axial femoral force were measured (Gates et al., 2010; Welch et al., 2010). This was one of the first investigations to pair the lumbar kinematic and kinetic responses of the BioRID II and Hybrid III ATDs across impact severity. In the case of the lumbar spine, the largest discrepancy between the two ATDs was the Hybrid III ATD tended to estimate larger compressive loads in comparison to the BioRID II ATD (Figure 2.5) (Welch et al., 2010). A major limitation to kinetic testing is that to date no human participant data exists for lumbar spine loads of human participants in low velocity rear impact collisions. Therefore, it is difficult to know if the Hybrid III estimates were an over prediction or if the BioRID II estimates were an underestimate of spine loads experienced in the low velocity rear impact simulations.

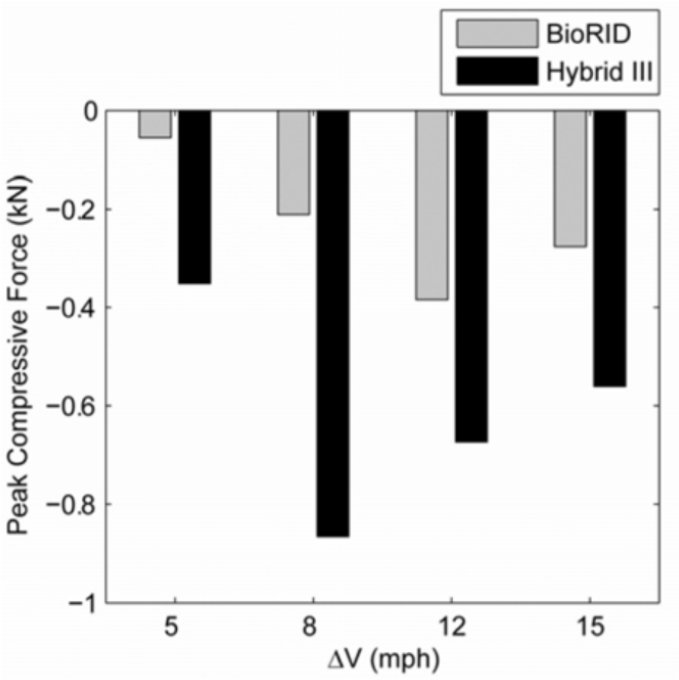

Figure 2.5: Peak compressive load estimates of the lumbar spine at 7.92, 12.96, 19.44 and 24.12 km/hour changes in velocity for the Hybrid III ATD and BioRID II (Image taken from Gates et al. 2010).

2.2.2.3 Laboratory Crash Simulations and the Lumbar Spine

Thousands upon thousands of low to moderate velocity rear-end crash simulations have been completed in the laboratory setting. However, very few of these studies have provided focus examining the forces and motions of the lumbar spine generated during such collisions. The limited number of studies that have been completed have typically demonstrated that the exposures in a

low velocity rear impact collision are below existing injury reference values for the lumbar spine and below manual materials handling limits (Fast et al., 2002; Gates et al., 2010; Gushue et al., 2001). For comparison, the National Institute of Occupational Safety and Health (NIOSH) recommends a compressive force threshold of 3400 N during occupational lifting. Similarly, previous research conducted by McGill et al. (1998) recommends a maximum limit of 1000 N of lumbar spine shear force during occupational exposures (representing approximately 33% of the shear failure tolerance observed in cadaveric specimens). A major limitation to these results is that the estimated lumbar spine forces are extremely variable across different ATDs. Even comparisons across studies with tests run at similar collision severities with the same ATD show extremely variable lumbar kinetic estimates.

Studies examining human occupant kinematics in low velocity rear impacts have demonstrated that the lumbar spine is well supported by the seat back and the mechanisms to cause a sudden or traumatic lumbar injury are un-founded in a properly belted occupant. McConnell and colleagues (1993, 1995) conducted simulated rear-end impact crash testing with human volunteers at collision severities ranging from 3 to 11 km/hour. They demonstrated that during a rear-end collision occupants go through a predictable motion of rearward than forward movement (McConnell et al., 1995a, 1993; Szabo and Welcher, 1996). Initially, human occupants move rearward relative to the vehicle until the seatback is compressed. At this point, the lumbar spine would presumably be in a state of tension as it extends backwards against the seatback and the pelvis and thighs are held in place by the lap belt portion of the seatbelt. The occupant then rebounds forward, away from the seatback. At this point, the lumbar spine would presumably experience compression as it becomes flexed as the torso moves forward and the lower body remains fixed by the lap seat belt. The forward flexion motion of the lumbar spine is restricted within normal physiological range of motion as the cross body seatbelt restricts any further motion (McConnell et al., 1995a).

Insight into the forces and moments experienced in the lumbar spine has been completed using a cadaver (Fast et al., 2002) and instrumented anthropometric test devices (ATDs) (Gates et al., 2010; Gushue et al., 2001). Fast and colleagues (2002), exposed one male cadaver to simulated rear-end collision severities of 13 and 19 km/h. The lumbar spine was instrumented with strain gauges on the lateral and anterior surfaces of T12, L2 and L4. Biaxial accelerometers were mounted on L1, L3 and L5. At both collision severities, radiographic testing revealed that no bony

injuries resulted from either collision. The anterior shear strains resulting from the simulated collisions had a biphasic shape and increased in magnitude with increased collision severity. Principal lateral shear strains were highest at L4 when compared to L2 and T12. This study was able to exclude vertical loading as a mechanism of injury to the low back (Fast et al., 2002). The measured maximum resultant vertical seat forces were approximately 1000 Newtons in the 13 km/hour test, which is well below compression injury tolerance values for the lumbar spine. Maximum resultant horizontal seat forces exceeded 1500 Newtons in the 13 km/hour test, however authors did not report horizontal seat forces for the 19 km/hour collision simulation.

The work by Fast and colleagues (2002), provides an initial assessment of the potential injury mechanisms to the lumbar spine during moderate velocity rear-end collisions. However, there are a number of limitations associated with this work. The first major limitation to this work is that it only involved one cadaver of an elderly person with significant degenerative disc disease and mild scoliosis, which was then exposed to multiple collisions. This data may not be applicable to all drivers and the presence of scoliosis may have modified the response of the cadaver during the simulated collisions. The second limitation is that strain gauge measurements only represent localized deformation at specific attachment points on the three vertebrae. In addition, localized strain measurements make it difficult to make comparisons across other studies. Lastly, the authors made no attempt to use the seat forces to provide an estimate of internal joint loading. In addition, horizontal and vertical seat forces were only presented for the lower collision severity.

More recently, sled testing has been completed on Hybrid III and BioRID ATDs in front bucket seats from a 2001-2003 Ford Taurus. The seats were tested at changes in velocity of 2.2, 3.6, 5.4, and 6.7 m/s (7.92, 12.96, 19.44 and 24.12 km/hour) (Gates et al., 2010). The ATDs were tested side by side in identical bucket seats (Figure 2.6) and lumbar kinetics were compared. The Hybrid III experienced higher bending moments and compressive loads than that of the BioRID II, at all collision severities tested. Out of all testing conditions the Hybrid III exhibited the highest compressive force (870 N) during the 12.96 km/hr impact, which was 2 times that measured in the BioRID II (Gates et al., 2010). This investigation mainly focused on the potential for automobile collisions to result in intervertebral disc injury and therefore the authors concluded that because the measured lumbar axial compression was small, (across both ATDs tested) it is unlikely that low to moderate velocity impacts can cause significant damage to the lumbar discs. This work points out a major discrepancy in lumbar force profile predictions between the BioRID II and

Hybrid III ATDs (Figure 2.7). Across all collision severities the BioRID II predicted the primary axial load to be in tension, with minimal compression force. The Hybrid III, in contrast, predicted substantial compression forces immediately after the impact (Figure 2.7). The results are also conflicting for lumbar shear force profiles. The BioRID II primarily predicted anterior shearing forces (Figure 2.8), while the Hybrid III predicted large posterior shear immediately following impact, prior to anterior shearing (Figure 2.8). In the 12.96 km/hour collisions severity (8 mph), a peak posterior shear load of 1280 N was estimated in the Hybrid III. While in contrast, the BioRID II estimated slightly higher anterior shear loads than the Hybrid III with peak anterior shear loads of 600 N and 400 N in the BioRID II and Hybrid III respectively. Both ATDs sustained comparable tensile loads; the peak tensile load was 1700 N and 1460 N in the BioRID II and Hybrid III, respectively (Gates et al., 2010).

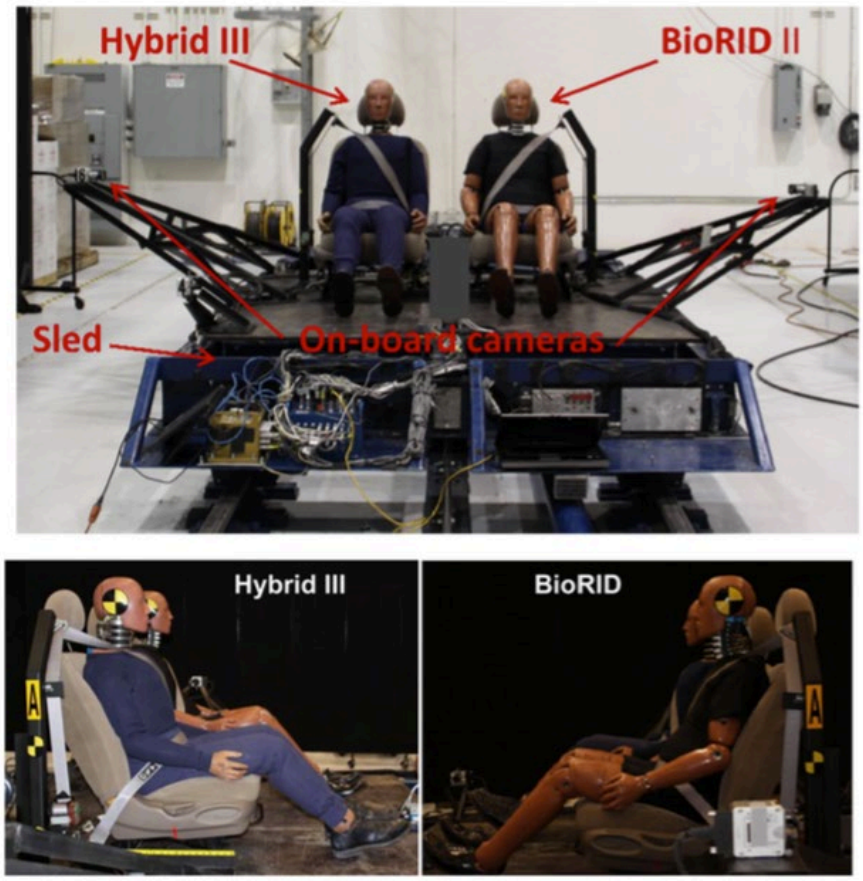

Figure 2.6: A figure displaying the configuration for all crash testing. A 50th percentile Hybrid III ATD and a BioRID II ATD were placed in identical front bucket seats. Taken from Gates et al. 2010.

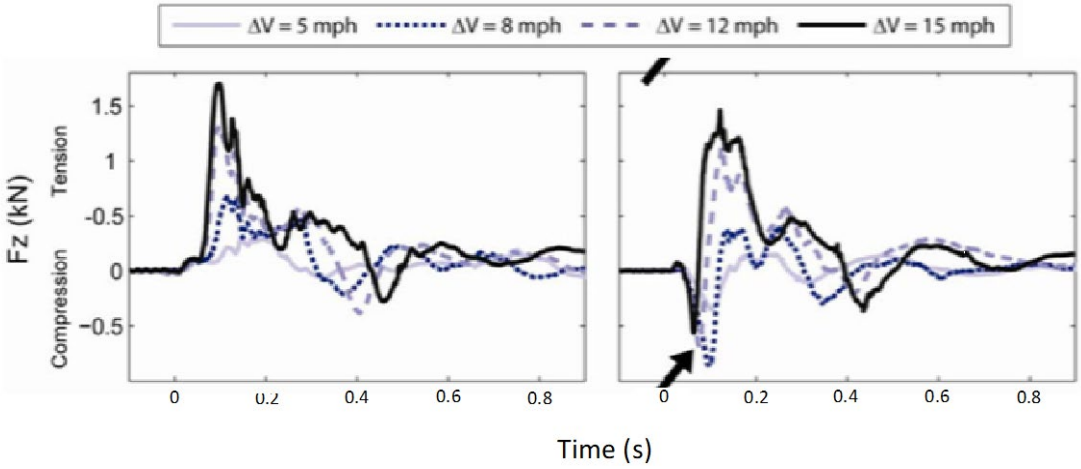

Figure 2.7: A figure displaying the lumbar load cell forces (Fz) throughout the tests for the BioRID II (left) and Hybrid III (right) ATD's. The Hybrid III ATD predicted a compressive force immediately following the impact, while the BioRID II did not. Taken from Gates et al. (2010).

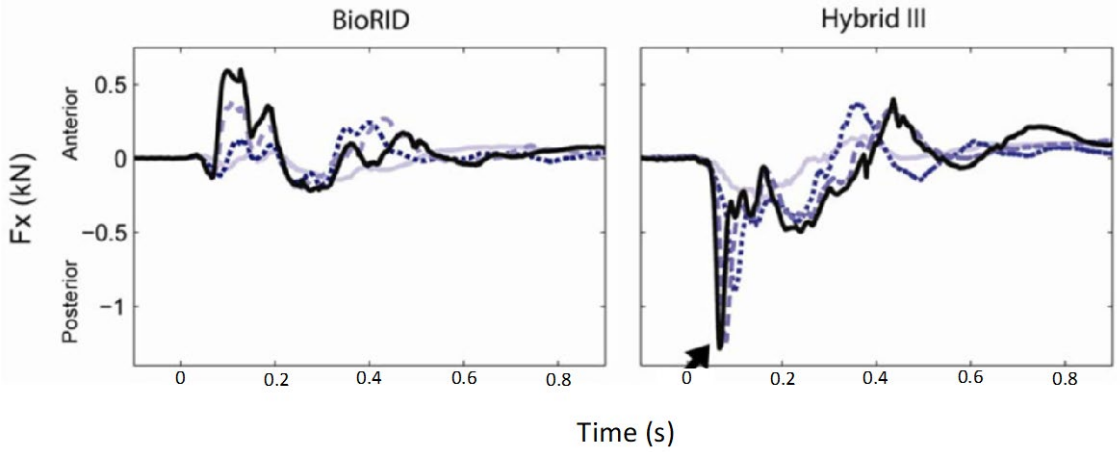

Figure 2.8: A figure displaying the lumbar load cell forces (Fx) throughout the tests for the BioRID II (left) and Hybrid III (right) ATD's. The Hybrid III ATD predicted a large posterior shear force immediately following the impact. Taken from Gates et al. (2010).

The final laboratory study to investigate low to moderate velocity rear-end collision simulations with focus on the lumbar spine was on Hybrid III ATDs (Gushue et al., 2001). Tests were conducted with ATDs seated in a 1999 Buick Park Avenue driver's seat at target changes in velocity of 8 and 12 km/hour (Gushue et al., 2001). The ATDs were positioned in three configurations, termed 'in position,' 6 inches out of position, and 20 inches out of position (Figure 2.9). In all seating postures the motion of the torso of the occupant ATD was arrested and well controlled by the head restraint and seatback (Gushue et al., 2001). Motion of the lumbar spine

was well controlled, even when the ATD was 20 inches out of position (Gushue et al., 2001). Examination of the forces experienced in the lumbar spine found maximum shear forces of 802 N, maximum compressive forces of 539 N and a maximum flexion moment of 43 Nm (Gushue et al., 2001). The authors compared these values to lumbar spine injury thresholds and concluded that these values are well below the injury thresholds for shear, compression, and flexion moment respectively.

Figure 2.9: An image displaying the three sitting postures tested. The ATDs were positioned in three configurations, termed 'in position,' 6 inches out of position, and 20 inches out of position, with 6 and 20 inches referring to the distance between the back of the head and the head rest. Taken from Gushue et al. 2001.

When taking the Hybrid III ATD results from Gushue et al. 2001, and comparing to the results of Gates et al. 2010, the peak compressive force estimates are very different. Gushue and colleagues (2001), completed simulated in position rear impact testing at two collision severities that similarly overlap with the conditions tested by Gates and colleagues (8 km/hour and 12 km/hour), using a Hybrid III ATD. Guschue and colleagues (2001), estimated peak compression forces to be 40.9 and 105 N at the 8 km/hour and 12 km/hour collision severities, respectively. Gates and colleagues (2010), completed in position simulated rear impact testing using a Hybrid III ATD in a 2003 Ford Tarus car seat at collision severities of 7.92 and 12.96 km/hr. They estimated peak compression forces to be 350 N and 870 N at the 8 and 12.8 km/hour collision severities respectively. These values are considerably different considering the similarity in ATD device used and collision severities tested.

To date, laboratory simulations focusing on the lumbar spine have mainly focused on the

potential for low velocity type rear impacts to result in IVD herniation type injuries (Gates et al., 2010; Gushue et al., 2001). IVD herniation injury is typically a fatigue type injury (Adams and Hutton, 1983) or caused by large compressive forces coupled with extension (Adams et al., 1988). Thus, the low compressive force estimates during low velocity rear-end collisions has led authors to conclude that injury to the lumbar spine is unlikely. Previous work has indicated that soft tissue injury resulting from large shearing forces may play an important role in the pathogenesis of lumbar pain after a low velocity collision (Fast et al., 2002). As demonstrated, the joint force estimates in the lumbar spine during a simulated low to moderate velocity collision are extremely variable and largely unknown. Not enough research has been completed to know what the exposures are and if ATD lumbar load estimates in low velocity rear impacts are realistic in comparison to *in vivo* exposures.

2.4 Injury Pathways

The theory for musculoskeletal injury suggests that tissue damage occurs when the physical demands exceed a tissue's capacity (McGill, 1997). There are two primary pathways in which a tissue can be overloaded. The first mechanism is an acute injury, which is known as an over exertion injury. This type of injury often occurs when a single load exposure exceeds a tissue's failure tolerance (McGill, 1997). This would be the typical mechanism of injury in a motor vehicle accident. The second mechanism of injury that can damage a tissue is through repeated (or sustained) application of loads that are of sub-failure in magnitude. This cumulative injury mechanism is often called an overuse injury (McGill, 1997). Here, due to the sustained loading, the tissues capacity is reduced and over time will result in failure when the accumulation of damage to the tissue outpaces the rate of recovery (McGill, 1997). Acute injuries can be easily used to describe the mechanism of failure that occurs during traumatic accidents. In these cases, the mechanical loading applied to the human body exceeds safe exposure levels, which result directly in injury (McGill, 1997).

In the case of a motor vehicle accident, typically the physical evidence from the accident along with reported information is evaluated and the collision dynamics are determined giving an estimate of the collision severity. Based on collision dynamics the mechanical exposure can then be estimated based on occupant motion. From there, the mechanical exposure can be compared to published tissue tolerance values in the literature to determine if a mechanism for injury is present.

2.5 Potential Mechanisms for Lumbar Spine Injury and Pain Reporting After a Rear Impact

2.5.1 Automotive Seating and Flexed Postures

When going from standing to a seated posture you must flex the hips, anteriorly rotate the pelvis, and flex the lumbar spine. Sustained lumbar spine flexion is characteristic of both office (Beach et al., 2005; Dunk and Callaghan, 2005; Gregory et al., 2006) and automobile (Harrison et al., 2000) sitting and is considered an important factor in the hypothesized mechanisms of low back pain development during seated postures. Both office sitting and automotive sitting induce increased lumbar spine flexion. However, the specific postures assumed during automotive driving differ significantly from standard office chair seating. In a study comparing office chairs and automotive seats, Beach et al. (2008), found that males and females exhibited increased trunk reclination, increased pelvic tilt, increased knee extension and increased hip flexion in automotive sitting in comparison to office chair sitting. Males sat at approximately 55% of maximum lumbar flexion range of motion in automotive sitting and females sat at 59% of maximum lumbar flexion range of motion. It was hypothesized that females sat at a greater percentage of maximum lumbar flexion range of motion to compensate for the large automotive seat, which are often not as easily customizable as office seating (Beach et al., 2008). Most automobile seats are designed for the 50th percentile male (Kolich, 2003), decreasing the probability that the average automobile seat will properly fit female drivers. In addition, greater intervertebral flexion has been found at L2/L3, L3/L4 and L4/L5 in automotive sitting when compared to office chair sitting (De Carvalho and Callaghan, 2012).

Changes in lumbar spine posture during simulated driving have been documented radiologically in automotive seating (De Carvalho and Callaghan, 2012). When seated, the pelvis rotates and there is flattening of the lumbar spine (i.e. a loss of lumbar lordosis or a decrease in lumbar lordosis angle). Mean lumbar lordosis angle decreased from 63 degrees (SD 15 degrees) in standing, to 20 degrees (SD 13 degrees) in automotive sitting (De Carvalho and Callaghan, 2012). Authors also investigated individual vertebral joint rotations between automotive sitting and standing and found that with the exception of L5/S1 (which displayed no significant change in angle with respect to standing), all intervertebral joint angles became more flexed in comparison to level ground standing. The lack of flexion at L5/S1 in comparison to all other vertebral joints was considered to be particularly problematic as large strains would be likely to develop given the

amount of rotation in all other intervertebral joints and the pelvis (De Carvalho and Callaghan, 2012).

Relatively few studies have investigated the loads experienced by the lumbar spine during sitting because of the premise that they are below injury thresholds and static. In automotive sitting specifically, minimal movements of the lumbar spine and pelvis are made (Callaghan et al. 2010; De Carvalho & Callaghan, 2011). To date, no study has specifically investigated low back loading in automotive sitting. In a study examining low back loading at the L4/L5 disc level during unsupported sitting, Callaghan and McGill (2001), found average compressive loads to be significantly higher in unsupported sitting (1698 N SD 467) than standing (1076 N SD 243). Average anterior-posterior shear loads were also significantly higher in unsupported sitting (135 SD 200 N) than standing (13 SD 17 N), with positive shear indicating anterior shearing of the trunk on the pelvis (Callaghan and McGill, 2001a). Both the compressive force and anterior-posterior shear force are well below the current shear and compression tolerances. These values are not directly applicable to automotive sitting. However, it provides some estimate of the compressive and shear loads in static sitting.

The L4/L5 compressive and shear joint forces in seated postures are well below injury reference values (Callaghan and McGill, 2001a). However, the static nature and decrease in lumbar lordosis during simulated driving can be problematic. Flexed postures result in elongation of the posterior ligaments (Adams et al. 2004). Increases in strain have been documented with increasing lumbar joint flexion in the supraspinous ligament, posterior longitudinal ligament, ligamentum flavum and capsular ligament (Panjabi et al., 1982). Prolonged flexion can lead to increased strain in the passive tissues (Solomonow et al., 2003). Stress on the passive tissues from prolonged flexion can result in viscoelastic creep of the posterior passive elements of the spine (McGill and Brown, 1992; Solomonow et al., 2003; Twomey and Taylor, 1982). Creep resulting from prolonged flexion has been shown to result in increased laxity, increased reflexive muscle spasm, altered kinesthetic awareness and delayed ligamentomuscular reflexes in the lumbar spine (Sánchez-Zuriaga et al., 2010; Solomonow et al., 2003). Reflective of viscoelastic creep in automotive sitting, a decrease in lumbar spine stiffness has been observed in response to 2 hours of simulated driving while sitting in an automotive seat (De Carvalho and Callaghan, 2011).

Lumbar spine flexion is also associated with elevated disc pressure (Wilke et al., 1999). Intradiscal pressure has been shown to generally increase in sitting from standing (Andersson et

al., 1975). Reflective of an increase in pressure are reports of decreased disc height measured by MRI (Fryer et al., 2010).

Lastly, flexion of a vertebral joint will alter facet interaction. This results in an increase in the gap between the inferior facets of the cephalad vertebrae and the superior facets of the caudal vertebra in the porcine FSU (Drake et al., 2008). The causes increased stretching of the facet joint capsule (Little and Khalsa, 2005a). A flexed posture also directly influences the passive response of an isolated FSU and decreases shear failure tolerance and stiffness (Howarth and Callaghan, 2012). When a human lumbar FSU is placed in flexion, the facets are unable to resist compressive loads (Adams and Hutton, 1980) and therefore are unable to provide an avenue for resisting or transferring compressive load across the joint.

Thus, poor spinal postures associated with automotive seating may put the lumbar spine in a susceptible state for injury. Coupled with sudden unanticipated loading that occurs during a rear-end motor vehicle collision, this may provide a mechanism for the high incidence of low back pain reporting after such collisions (refer to section 2.5.3 for facet joint capsule strain injury mechanisms).

Maintenance of a neutral spine posture has been suggested as an effective intervention to reduce low back discomfort in sitting (O'Sullivan et al., 2010). Lumbar supports in automotive seating have been shown to increase lumbar lordosis (Andersson et al., 1979; De Carvalho and Callaghan, 2015), reduce disc pressure and muscle activity (Andersson et al., 1974; Kingma and van Dieën, 2009) and decrease low back pain reporting (De Carvalho and Callaghan, 2015). A radiographic investigation of lumbar support use confirmed improved lumbar spine posture with increasing lumbar support prominence. Lumbar lordosis angles were found to increase from 20° with 0 cm or no support to 30° with 4 cm of lumbar support prominence (De Carvalho and Callaghan, 2012). Lumbar supports have been shown to be beneficial in driving. However, their effectiveness during a rear impact collision remains unknown. It is possible that the use of a lumbar support may positively influence the kinematics and the resultant joint loads in the lumbar spine during a simulated rear impact collision.

2.5.2 Shear Loading

The mechanisms linking the forces in rear-end collisions with low back pain remain unclear. A potential mechanism for injury includes sub-failure shear forces within the joints resulting from

localized relative motion of the lumbar vertebrae, which could result in altered mechanical joint properties. To date, investigations of *in vitro* shear loading as a mechanism for low back injury have been limited (Howarth et al., 2013; Howarth and Callaghan, 2013b; Yingling and McGill, 1999) and have only encompassed anterior shear force failure exposure.

The ability for the IVD to resist shear loading is largely dependent on posture, compressive load and rate of loading (Howarth and Callaghan, 2012; Yingling and McGill, 1999). Increased compressive load and rate of shear loading has been demonstrated to increase a specimens' ultimate shear load tolerance loading (Howarth and Callaghan, 2012; Yingling and McGill, 1999). While flexed postures have mixed results demonstrating both increased (Yingling and McGill, 1999) and decreased (Howarth and Callaghan, 2012) ultimate shear load tolerances. These results indicate that ultimate shear failure force may be governed by changes in facet articulation.

To date, no study has exclusively investigated changes in joint mechanical properties with sub- failure levels of applied shear force. However, Howarth and colleagues (2013) have used sub-acute failure loads to investigate changes in the shear neutral zone length and average stiffness with specimens in flexed and extended postures. Anterior-posterior shear passive testing was completed at 0.2 mm/s to a target of ±400 N in extended, neutral and flexed postures. For each test shear neutral zone length and average stiffness was quantified. Extended postures produced a 37% increase in shear stiffness within the NZ compared to both flexed and neutral postures (Howarth et al., 2013). Posture did not influence the shear NZ length. The average stiffness increase in extension was likely a result of increased contact area and force of the facet joint in extension. These results demonstrate that postural deviation of the vertebral joint in flexion is likely not a confounding factor when assessing segment stability.

Howarth and Callaghan (2013a), also investigated the effect of sub-acute failure load magnitude on fatigue failure in a repetitive shear loading paradigm. Specimens were repetitively loaded (at a constant loading rate of 1 Hz) to one of four percentages of ultimate shear failure tolerance (20% - 429.2±29.2 N; 40% - 809.4± 27.0 N; 60% - 1226.3±53.0 N or 80% - 1744.4±79.0 N) to failure or 21600 cycles (Howarth and Callaghan, 2013a). Cumulative shear and the number of cycles sustained to failure displayed a strong non-linear decreasing relationship with increasing force magnitude. All specimens assigned to the 60% and 80% groups failed prior to the cycle limit. All specimens exposed to the 20% magnitude and five specimens exposed to the 40% magnitude survived the 21,600 shear loading cycles. Survivors in the 40% group sustained 3.5 MN∗s higher

cumulative shear force than survivors of the 20% magnitude (Howarth and Callaghan, 2013a). Cumulative shear sustained by the failed specimens at 40% was significantly higher than that of the failed specimens in the 60% and 80% groups. This study suggests that tissue micro-damage might begin to non-linearly accumulate with applied shear forces between 30% and 40% of a vertebral joint's acute shear failure tolerance. In addition, throughout the investigation shear force and displacement was continuously monitored. This allows for some evaluation of the time-varying changes in joint properties across sub-acute failure loads. As cycle number continued specimens displayed a general trend of increased shear displacement and decreased average stiffness (Howarth and Callaghan, 2013a).

Previous investigations have provided some indication that sub-failure levels of applied shear force may alter joint mechanics. However, how sub-failure impact type loading links to altered joint mechanics such as, increased joint laxity is largely unknown. Shear loading is an important factor to investigate, as occupational low back pain reporting has been significantly correlated to peak anterior joint reaction shear force (Norman et al., 1998). There is conflicting evidence of the shear force exposure resulting from a low velocity impact. The Hybrid III ATD predicts large posterior and anterior shear forces while the Bio RID II predicts primarily anterior shear loading (Gates et al., 2010). An initial step in exploring low velocity rear-end collisions as a potential injury mechanism to the lumbar spine is to obtain an estimate of the internal lumbar joint loads.

2.5.3 Facet Joint Capsule Strain: Evidence of Facet Joint Capsule Innervation

The lumbar facet joint capsule can be a source for low back pain. In the case of a low velocity rear impact, shear rotational forces within the joints may result in facet joint capsule deformation and compression. Histological analysis of the facet joint capsule has shown that it contains Pacinian corpuscles, Ruffini and free nerve endings which suggests both nociceptive and proprioceptive properties (Cavanaugh et al., 1996). Low back pain has been produced with radiation to the thigh by injecting hypertonic saline into facet joints (Mooney and Robertson, 1976). In addition, facet nerve blocks have a 50-60% success rate in reducing low back pain (Helbig and Lee, 1988). The lumbar facet superior articular process can bottom out on the lamina below when forces replicating the spinal extensor muscles are used to resist flexion loads (Yang and King, 1984). This loading also causes high strains to the facet-joint capsule.

A number of studies have quantified the strains occurring in the lumbar facet joint capsule during physiological motions and have confirmed facet capsule stretching. Cavanaugh and colleagues (1996), tracked the amount of stretch in the facet joint capsule and took note that large strains occurred when many of the specimens were tested in extension. It was noted that the geometry of the superior portion of the facet was a major factor when determining the magnitude of stretch in the capsule (Cavanaugh et al., 1996). Increases in facet capsule strain during static and cyclic flexion have also been noted (Little and Khalsa, 2005a). Ianuzzi et al. (2004), also demonstrated distinct patterns of facet capsule principal strain with physiological movement. Strains increased in magnitude with increased extension and flexion (Ianuzzi et al., 2004). Across all studies, strains were varied across the capsules with no particular pattern of consistent maximum strain location. The variability of strains between capsule locations under stretch may result from individual differences in the capsule insertion locations on the articular processes, inhomogeneity and anisotropy in the facet joint capsule itself.

Fewer studies have attempted to quantify the neurophysical relationship between neural discharge and applied facet capsule stretch. Yamashita and colleagues (1990), demonstrated that 8 out of 30 lumbar facet joint units responded to joint movement (Yamashita et al., 1990). Cavanaugh and colleagues (2006), demonstrated nociceptive neural discharge from facets of anesthetized goats increased with facet capsule stretching. The group was able to demonstrate a quantitative relationship between capsule sensory discharge and applied capsule stretch in cervical facet joints of goats. They stated that facet capsule strains of 47.2% (+/- 9.6%) are most likely noxious and trigger the central nervous system for pain sensation (Cavanaugh et al., 2006). Most of the facet capsule neural receptors sense physiological ranges of capsule stretch and start to fire at strains of 10.2 % (+/- 4.6%) (Cavanaugh et al., 2006). This evidence confirms the hypothesis that facet joint stretching may contribute to lumbar and cervical pain signals resulting from facet joint stretch.

Low back pain can be divided into three time spans based on the potential for recovery (1) acute (up to seven days) (2) subacute (one week to three months) and (3) chronic (over three months) (Mooney, 1989). Based on the typical time course of tissue injury, inflammation and repair, acute traumatic strain in facet joint capsules could lead to acute or sub-acute low back pain. Previous work has already demonstrated facet involvement in patients suffering from chronic neck pain due to acute traumatic injury. In patients involved in motor vehicle collisions suffering from

chronic whiplash symptoms, clinical and pathologic investigations have targeted the facet joints as sources of pain generation. A series of studies have used nerve block and radiofrequency ablation to facet joint afferents and have successfully relieved pain in chronic whiplash patients. (Barnsley et al., 1993; Lord et al., 1996a, 1996b).

Facet joint capsule strain injury may be a possible injury mechanism in the low back resulting from low velocity rear impacts. To date, no work has focused on lumbar facet capsule involvement in low back pain reporting after a low velocity rear-end collision. The flexed lumbar spine posture in automotive seating may place the lumbar spine in a compromised position to result in increased facet joint capsule deformation and capsule strain related injury.

2.5.4 Ligament Injury and Link to Pain Generation

The exact cause of most low back pain remains unidentified. Micro-damage to the ligaments is one potential source for altered joint mechanics and potentially low back pain (Panjabi, 2006). Abnormal mechanics have been hypothesized to lead to low back pain through nociceptive sensors. The path from abnormal mechanics to nociceptive sensation can occur through any number of factors including inflammation (Burke et al., 2002; Cavanaugh et al., 1997), biomechanical and nutritional changes, changes in structure and material of the endplates (Brown et al., 1997) and discs (Osti et al., 1992, 1990). Abnormal mechanics of the spine may be due to degenerative changes (Fujiwara et al., 2000) and or injury of the ligaments (Oxland et al., 1992).

Many studies have demonstrated that the mechanical properties of ligaments can be altered by sub failure injury. To date, ligament injury resulting from mechanical trauma has traditionally been defined by gross measures of mechanical failure or visible rupture. Sub failure loads can produce micro-damage to a tissue, which can result in a variety of altered mechanical properties in ligaments, including increased laxity (Panjabi et al., 1996; Pollock et al., 2000; Provenzano et al., 2002), decreased stiffness (Panjabi et al., 1999; Quinn et al., 2007), increased stiffness (Nelson-Wong et al., 2018; Panjabi et al., 1996) and altered viscoelastic responses (Nelson-Wong et al., 2018; Panjabi et al., 1999). Such responses can be coupled with collagen disorganization, fibroblast necrosis and nociceptor activation.

Panjabi (2006), hypothesized that abnormal mechanics of the spine can be initiated by sub failure damage to the ligaments resulting from some kind of trauma involving the spine. It may be a single trauma due to an accident or continuous micro trauma caused by repetitive motion. Panjabi

(2006), stated that the osteoligamentous spinal column has two functions: structural and transducer. The structural function provides stiffness to the spine. The transducer function uses mechanoreceptors to provide information needed to characterize the spine postures, vertebral motion and loads and transfer that information to the neuromuscular control unit. These mechanoreceptors are present in the ligaments, facet capsules and annulus. If the structural component is compromised through degeneration or injury, then stability from muscular contribution is increased to compensate. However, if the transducer function of the ligaments in the spinal column is compromised as a result of injury, it can lead to altered muscle response patterns influencing coordination and activation patters of muscles surrounding the spine (Panjabi, 2006). Sub failure injury will occur as a result of stretching the ligament beyond physiological limits, but less than the failure point. This can occur from a single trauma, such as a motor vehicle collision or cumulative micro trauma. Altered muscle responses can lead to further sub failure and injury of the spinal ligaments, mechanoreceptors and muscles, as well as overloading of facet joints (Panjabi, 2006). Consequently, over time low back pain may develop.

2.5.5 Muscle and Link to Pain Generation and Injury

As previously established, the exact cause of most low back pain remains unidentified. The muscles that surround the lumbar spine are yet another potential source for low back pain development (Panjabi, 2006).

Muscle as a specific pain-generating source is a somewhat controversial topic in the literature. On one hand it is unlikely that individual muscle fibres contain nociceptors—however—nociceptors are present in blood vessels and in fascia. In addition, muscle spindles are very sensitive to mechanical stimuli (Waddell, 2006). Across the literature, more conclusive evidence supports muscle pain arising from metabolic factors, such as pH decreasing and increased concentration of local metabolites during a sustained muscle contraction (Kumar, 2001; Waddell, 2006)

Another commonly accepted hypothesis for the link between spinal muscles and low back pain generation is lumbar spine stability (Panjabi, 1992a). The muscles surrounding the lumbar spine play an essential role in ensuring proper functioning and maintenance of spine stability (Cholewicki and McGill, 1996; Crisco et al., 1992). For example, the full lumbar spine in the

absence of muscle, will buckle under approximately 90 N of compressive force (Crisco, 1989). This is significantly different from the average range of *in vivo* compressive load estimates ranging between 1500 N to 3500 N, during manual materials handing type tasks (Gooyers et al., 2018; Marras et al., 2001; S. McGill et al., 1998; Toney-Bolger et al., 2019). This difference in ultimate tolerance is exclusively due to the muscles surrounding the lumbar spine, which act as guy wires in stiffening the spine and increasing the critical load and overall stability. Thus, it is important to note that even in neutral low-risk postures, the muscles surrounding the lumbar spine play a critical role in stabilizing the lumbar spine. The amount of muscle activation needed to ensure sufficient stability depends on the task. Generally, for most tasks of daily living, very modest levels of abdominal wall co-contraction is sufficient (Cholewicki et al., 1997; Cholewicki and McGill, 1996). For example, sufficient stability of the lumbar spine is achieved in the neutral posture with modest levels of co-activation from the paraspinal and abdominal wall muscles (Cholewicki et al., 1997; Cholewicki and McGill, 1996). However, if a joint has lost passive stiffness, for example, such as soft tissue injury from a motor vehicle collision or impact type event, the muscles may compensate with increased co-contraction to make up the deficiency (Oxland et al., 1991). This compensation, while helpful from a spine stabilization perspective, could also lead to different movement strategies and if persistent over time, possible secondary pain pathways.

Typically, the muscles surrounding the lumbar spine are thought of in terms of their active force production capabilities, however spine muscles can also passively generate substantial tension when stretched beyond their slack length. Passive muscle tension is particularly important when considering scenarios where muscle activation may be inherently low, yet the postures associated are nearing end range of motion. Recent work by Zwambag and Brown (2020), demonstrated that during a forward flexion-relaxation task, where the muscles surrounding the lumbar spine were virtually in-active, the spine muscles still greatly contribute by passively supporting approximately 47% of the extensor moment demand. Significant passive muscle tension would also be an important consideration in an unanticipated rear-end collision. The flexed postures associated with automotive seating place the lumbar spine in the upper end ranges of maximum lumbar spine flexion (De Carvalho and Callaghan, 2012). In this posture, the muscles surrounding the lumbar spine would be beyond resting slack length and would be capable of passively generating substantial tension in responding to a sudden impact. Changes in passive muscle mechanical properties have been reported across injurious/health events such as

intervertebral disc degeneration (Brown et al., 2011), tendon detachment (Safran et al., 2005; Sato et al., 2014), spine tissue mineralization (Gsell et al., 2017) and cerebral palsy (Fridén and Lieber, 2003). It remains unknown if an acute injurious event can trigger non-recoverable mechanical changes in passive muscle properties, however, it is an important consideration as these mechanical properties have practical relevance for stiffness and spine stability.

2.6 Methodological Considerations

2.6.1 *In Vitro* Techniques

2.6.1.1 Porcine Cervical Spine Model

Human *in vitro* lumbar spine injury research is difficult to conduct. Young healthy spines are preferred, but specimens from older and/or sick donors or donors exposed to traumatic events are the sources most available for research. As a result, porcine cervical FSUs have been used as surrogates for the human lumbar spine (Goertzen et al., 2004; Gunning et al., 2001; Howarth and Callaghan, 2012; Lundin et al., 2000; Panjabi et al., 1989; Parkinson and Callaghan, 2009). This animal model has shown similar mechanical characteristics to a young adult with no disc degeneration or bone injury (Callaghan and McGill, 1995; Yingling et al., 1999, 1997). Both structural and functional similarities between the porcine cervical spine (*i.e.* c34, c56) and the human lumbar spine have been confirmed (Oxland et al., 1991; Yingling et al., 1999). Two slight differences have been identified by Yingling and colleagues (1999) in the porcine cervical FSU, in comparison to the human lumbar spine. This includes the presence of anterior processes, which appear to have no significant mechanical role and the endplates are smaller in the porcine model, with an average area of 500 mm^2 compared to an average of 1000 mm^2 for the human lumbar vertebrae (Yingling et al., 1999). A major benefit of the porcine model is that it allows for control of genetic makeup, age, weight, physical activity levels and diet (Yingling et al., 1999). This would be impossible to collect with human specimens.

2.6.1.2 Influence of Freezing on Mechanical Properties:

Due to cost-effectiveness, transportation issues, and multi-specimen requirements, FSUs are often first frozen and then thawed from a frozen state to be used for *in-vitro* biomechanical testing. Frozen storage allows for large sample experiments on homogenous specimen groups. These specimens can be frozen immediately after harvesting in an attempt to maintain the physical state

at the time of harvest. Conflicting research exists regarding the impact of freezing on the mechanical properties of FSUs, with results varying by methodology, tissue type and animal species (Callaghan and McGill, 1995). With respect to spine tissue specifically, some research has demonstrated that freezing does not affect the tensile properties of human annulus fibrosus (AF) tissue (Hirsch and Galante, 1967). In addition, Smeathers and Joanes (1988) have shown that the compressive stiffness and hysteresis in human lumbar specimens were altered by less than 1% between fresh and thawed testing conditions. Testing conditions were completed under repetitive sub-failure magnitudes (750 ± 250 N) of compressive load (Smeathers and Joanes, 1988). However, Callaghan & McGill (1995) found that frozen storage increased ultimate compressive load by 24% and energy absorbed to failure by 33%, but did not affect stiffness or displacement at failure (in comparison to fresh specimens). Freezing and storage conditions have been shown to have no significant effects on specimen biomechanical properties (displacement due to anterior shear, axial rotation and lateral bending) when comparing fresh specimens to those frozen over a short or long duration (Panjabi et al., 1984). However, fresh specimens showed greater variability in biomechanical properties than previously frozen specimens of any duration (Panjabi et al., 1984). While specimens not loaded to failure seem to show minimal effects of frozen storage, storage medium is an important consideration and must be acknowledged. For this thesis, it is not feasible to acquire and store a sufficient quantity of fresh specimens.

2.6.2 *In Vivo* Techniques

2.6.2.1 EMG Assisted Modeling

The EMG assisted modeling approach has been thoroughly documented in the literature for a variety of tasks including, but not limited to: lifting (Granata and Marras, 1995; Kingma and van Dieën, 2004), pushing and pulling tasks (Knapik and Marras, 2009), standing and unsupported sitting (Callaghan and McGill, 2001a) and walking (Callaghan et al., 1999). In general, contributions to the net L4/L5 joint moments are approximated and partitioned across passive tissues (ligaments IVD etc) and surrounding musculature.

The *in vivo* modelling approach considered for this thesis has been thoroughly described in the literature (J Cholewicki and McGill, 1996; McGill, 1992, 1988; Stuart M. McGill and Norman, 1986). A brief overview of the model will be provided here. First, contributions to the net joint moments from passive tissues are approximated using three-dimensional kinematics of the lumbar

spine (the orientation of the ribcage with respect to the pelvis). It is based on the assumption that each intervertebral joint contributes a specific proportion to the total lumbar spine angle. Based on the angular position of the lumbar spine, the moment contributions from a lumped passive tissue component is calculated based on joint displacement-load relationships. McGill and colleagues (1994) measured passive bending properties of the human torso about the three principle axes of flexion-extension, lateral bending and axial rotation for males and females developing lumped passive parameters for flexion-extension, lateral bend and axial rotation.

Next, the remaining moment (after accounting for passive tissue contribution) is partitioned across surrounding musculature, in combination with a three-dimensional anatomical model from Cholewicki and McGill (1996). The model requires input of surrounding muscle activations of normalized linear enveloped EMG. Three-dimensional kinematics and external kinetics are used in combination with a detailed anatomical model to calculate instantaneous muscle lengths and contraction velocities. Muscle groups that cannot be accessed for surface EMG collection are assigned activation profiles from anatomically and functionally similar muscle groups. For each muscle, in combining an anatomical model with either the distribution-moment (DM) equations (Ma and Zahalak, 1991), or by using a Hill-type muscle model, individual force profiles for each muscle based on normalized linear enveloped EMG can be determined.

The argument in favor of the use of an EMG-driven model is the data source includes a participant's own muscle activations. Individual muscle strategies are accounted for when modeling muscle force estimates. This is especially beneficial in tasks when muscle forces may contribute to a large portion of the net joint moment. In addition, in tasks where large amounts of co-contraction are commonly observed, this results in higher muscle forces to satisfy the required moment and in turn higher spine compressive forces result in comparison to when co-contraction is not present. The use of an EMG assisted model would take this into consideration. However, in certain situations where the impact occurs quickly and the muscles don't have time to react (such as in an unanticipated impact type setting) or tasks where EMG activation is inherently low, this type of modeling approach may not be the most ideal. In this case, the use of simplified joint models in estimating the joint loads in the lumbar spine may be sufficient to obtain an understanding of the resultant joint loads in the lumbar spine.

2.6.2.2 Passive Stiffness and The Neutral Zone – Sensitivity for Injury:

Panjabi (1992), stated that intervertebral motion can be split into two regions – the neutral zone

and elastic zone. The neutral zone is defined as the portion of physiological range of motion in which spinal motion is produced with minimal resistance – a zone with high flexibility and laxity (Panjabi, 1992b). The elastic zone is measured from the end of the neutral zone up to the physiological range of motion. This region has high stiffness and motion within this zone is produced against significant resistance due to stiffening of the ligaments, intervertebral disc or boney contacts. It is the non-linear properties of ligaments that result in a high amount of laxity around the neutral zone and a large amount of stiffening towards the end range of motion (Panjabi, 1992b). These quantities exist in the lumbar spine for each one of the six degrees of freedom (three rotational and three translational).

In vivo, the neutral zone has been defined as the range where the lumbar spine demonstrates the least amount of passive stiffness. Recently, Gallagher (2014), quantified the lumped passive stiffness and location of the neutral zone and related it to self-selected lumbar spine angle when standing. This represented one of the first investigations to quantify the lumbar spine neutral zone *in vivo* and relate it to a physiological task (Gallagher, 2014). Lumped passive stiffness in, flexion (Beach et al., 2005; De Carvalho and Callaghan, 2011; McGill et al., 1994; Parkinson et al., 2004; Scannell and McGill, 2003), extension (McGill et al., 1994; Scannell and McGill, 2003), lateral bend (Gombatto et al., 2008; McGill et al., 1994), axial twist (Drake and Callaghan, 2008; McGill et al., 1994) curves have been quantified *in vivo.* In the case of *in vivo* lumped passive stiffness, the measure of overall lumbar region stiffness includes contributions from muscle, tendon, ligament, cartilage, bone, skin, nerve, adipose tissue and viscera. Changes in passive stiffness have been demonstrated in prolonged office (Beach et al., 2005) and automotive (De Carvalho and Callaghan, 2011) seating, repetitive flexion tasks (Parkinson et al., 2004) prolonged flexion, in older adults in comparison to a younger adult population (Gruevski and Callaghan, 2019) and in clinical LBP patients (Gombatto et al., 2008). Changes in the passive stiffness properties of the lumbar spine can lead to changes in the loads and load distributions within the ligaments, intervertebral discs and muscles. These changes can alter the potential for injury, as well as resulting injury mechanisms.

Changes in specific regions of the lumbar spine lumped passive stiffness curve may also provide an indication of specific structures that could be responsible for these changes. Using equations provided by Adams and Dolan (1991), McGill and colleagues (1994) concluded that muscles were the primary flexion-resisting tissues in the moderate ranges of lumbar flexion

(Adams and Dolan, 1991; McGill et al., 1994). Similarly, as previously discussed, Zwambag and Brown (2020), demonstrated that during a full forward flexion-relaxation task, the spine muscles greatly contribute by passively supporting approximately 47% of the extensor moment demand. Thus, suggesting that changes in the moderate to high ranges of lumbar spine passive range of motion may be indicative of passive stiffness changes within the muscles as opposed to the soft tissues surrounding the lumbar spine. Beach and colleagues (2005) observed a trend of increases in stiffness in the moderate ranges of lumbar passive flexion in response to one hour of prolonged office chair sitting (Beach et al., 2005). This work also hypothesized that these changes could be linked to time-varying changes in the passive elastic properties of muscles (Beach et al., 2005). Similarly, De Carvalho & Callaghan (2011) also found initial increases in stiffness in the moderate to high ranges of lumbar flexion following a one hour of prolonged driving simulation. However, after a second hour of prolonged driving there was a gender specific response, with females displaying a trend of decreased stiffness and males displaying a trend of increased stiffness over the moderate and high ranges of lumbar passive flexion (De Carvalho and Callaghan, 2011). Given that during prolonged office seating and driving participants often sit in the mid to upper ranges of maximum lumbar spine flexion, these changes could be in response to the muscles being stretched in a prolonged flexed seated posture. Despite trends across the moderate and high lumbar spine passive flexion ranges, no changes in the low range of passive lumbar flexion were observed in these published studies. Changes to the whole lumbar passive stiffness curve have been observed in flexion following 30 minutes of repetitive lifting and in lateral bend in clinical low back pain patients (Gombatto et al., 2008; Parkinson et al., 2004). It is possible that changes to the lower ranges of lumbar spine passive range of motion may be indicative of changes within the soft tissues such as intervertebral disc, ligaments and fascia. This may also explain the sensitivity for neutral zone changes *in-vitro* (in the absence of muscle) in determining vertebral passive tissue injury during mechanical testing (Oxland and Panjabi, 1992). However, despite speculations, it is currently not possible to validate the specific structures *in-vivo* that are responsible for these changes due to the anatomical and functional complexity of the tissues comprising the lumbar spine passive stiffness properties.

Oxland and Panjabi have previously demonstrated in porcine FSUs, that the neutral zone is a more sensitive measurement than either the elastic zone length or specimen range of motion for determining onset and progression of vertebral passive tissue injury (Oxland and Panjabi,

1992). Larger neutral zones in flexion were indicative of advanced vertebral passive tissue injury and altered passive tissue resistance to applied load. The neutral zone has also been found to be a more sensitive parameter in relating to identification disc degeneration (Panjabi, 1992b). *In vivo*, human patients diagnosed with lumbar spondylolisthesis displayed increased neutral zone length and reduced neutral zone stiffness. In high-speed trauma experiments, both the neutral zone and range of motion have been found to increase with severity of injury (Panjabi et al., 1989). However, in a direct comparison between the neutral zone and range of motion, neutral zone increases were larger than the corresponding increases in range of motion for the same injury (Panjabi et al., 1989). In developing an intervertebral neck injury criterion for the cervical spine in frontal collisions, Ivancic and colleagues (2005), used an incremental trauma model approach with a whole cervical spine. Soft tissue injury at each vertebral level was defined as a statistically significant increase in neutral zone length, flexion neutral zone limit or total range of motion in comparison to physiological limits. At each level, neutral zone length changes either occurred first or accompanied increases in total range of motion. Changes in range of motion did not occur prior to changes in neutral zone range (Ivancic et al., 2005). These findings add evidence that changes in neutral zone may be a sensitive measure in traumatic events when linking to injury or instability and supports the potential utility of neutral zone characteristics for identifying soft tissue injury.

2.7 Summary

In summary, there is evidence to suggest that low back pain reporting after a low speed collision does occur. There is very little evidence in the literature documenting the exposures to the human lumbar spine in a low speed collision. Previous work has demonstrated that traumatic disc injury is unlikely to occur. However, more work needs to be completed in order to explore the potential for soft tissue injury in the lumbar spine and the potential link to pain generation. The flexed posture associated with automotive driving places the lumbar spine in a susceptible posture for injury during sudden impact, with potential soft tissue strain injury sites including the ligaments, intervertebral disc, facet joints and neural components.

Chapter 3: Study I - Exploring Low Velocity Collision Characteristics Associated with Claimed Low Back Pain

Components of this chapter have been published:

Fewster, K.M., Parkinson, R.J., Callaghan, J.P. Low velocity motor vehicle collision characteristics associated with claimed low back pain. *Traffic Injury Prevention* 20(4): 419-423, 2019.

3.1 Overview

Study Design: A data mining experiment was conducted to document the proportion of claimed injuries in low velocity automobile collisions in litigation that result in claims of low back injury. Specific collision and occupant characteristics were documented to determine if specific collision or occupant characteristics result in a higher frequency of low back injury claims.

Background: Up to fifty percent of individuals involved in low to moderate velocity collisions report low back pain. However, our understanding of the specific collision or occupant characteristics that result in such claims of low back pain remains limited.

Objectives: The primary objective of this study was to define the circumstances of low velocity motor vehicle collisions that result in litigation in Ontario with claims of low back injury.

Methods: Data for this investigation were obtained from a forensic engineering firm based in Toronto, Ontario, Canada. The database was searched and only cases with an evaluation of the injuries sustained in passenger vehicle to vehicle collisions, with a collision severity of 24 km/hour or less were included in this analysis. Each identified case was reviewed for collision characteristics, pre-existing medical conditions and injuries claimed. Descriptive statistics (mean, SD and ranges) across low back injury claims were computed for documented variables.

Results: Out the 83 cases reviewed, 77% involved a claim of low back injury. Specific to those who claimed low back injury, examination of the medical history revealed that pre-existing low back pain (LBP) or evidence of lumbar disc degeneration were particularly common with 63% of claimants either having had a history of LBP or evidence of lumbar disc degeneration, or both. Of all low back injury claims, 97% were accompanied by a whiplash and/or whiplash associated disorder claim. For low back injury claims, a rear-end impact was the most common configuration (70% of all low back injury claims involved a rear-end collision). The majority of all low back injury claimants experienced a change in velocity of 13 km/hour or less (69%), with 42% of all low back injury claims falling between collision severities of 10 – 12 km/hour.

Conclusions: Results indicate that rear-end collision severities of 10 – 12 km/hour appear to be particularly common with respect to low back injury reporting; more severe collisions were not associated with greater low back injury reporting. This result contrasts with previously published neck injury risk data, which demonstrated the risk of neck injury symptom reporting increases with collision severity. Evidence of lumbar disc degeneration was particularly common across claimants with low back injury claims.
Keywords: low back pain, low speed collision, motor vehicle accident

3.2 Introduction

Up to fifty percent of individuals involved in low to moderate velocity collisions report low back pain (LBP) (Fast et al., 2002). However, the mechanisms linking the forces of such collisions with LBP remain unclear, as there is limited data available to relate low back injury mechanisms to low velocity collision characteristics. Frequently, claims of lumbar injury and pain are reported after low velocity collisions, even with little vehicle damage, lack of objective injury evidence upon medical examination and negative radiographic evidence. As a result, legal assessments of injury causation remain largely reliant on symptom reporting, despite the psychosocial issues known to be present in such claims. The first step in identifying potential low back injury mechanisms resulting from low velocity automobile collisions is to characterize and identify the types of low velocity collisions that result in LBP claims to establish links between collision circumstances and injury outcomes. Therefore, the primary objective of this investigation was to characterize low velocity motor vehicle collisions that result in claims of LBP in southern Ontario.

There is an abundance of information available in the literature on whiplash and whiplash associated disorders (WAD) and the link to low velocity motor vehicle collisions (Brault et al., 1998; Castro et al., 1997; Howard et al., 1999; Winkelstein et al., 2000). However, the neck isn't the only reported area of injury with claims of LBP after a motor vehicle collision often accompanying WAD (Beattie and Lovell, 2010; Chapline et al., 2000; Gay and Abott, 1953; Magnússon, 1994), with the total percentage of claimed low back injuries accompanying WAD ranging between 27 and 60% (Beattie and Lovell, 2010; Chapline et al., 2000). A limited number of epidemiological investigations have demonstrated links between motor vehicle accidents and LBP reporting. For example, Beattie & Lovell (2010) analyzed over 800 reports for connections between whiplash symptoms and reported LBP. The group found that over 40% of the WAD cases analyzed also included claims of LBP. While no external factors were found to link whiplash symptoms to claimed LBP, it was rare that an individual would claim LBP without some form of neck injury (Beattie and Lovell, 2010). Collisions with low and moderate levels of vehicle damage had higher incidences of LBP claims when compared to collisions with severe damage, suggesting that LBP from automobile collisions may be an outcome of lower severity impacts and associated claims. This is similar to whiplash and WAD, with over 90 percent of whiplash neck injuries from rear-end collisions occurring at collision severities below 25 km/hour (Watanabe et al., 2000). In addition, Nolet and colleagues (2017), found an association between self-reported low back injury

in a motor vehicle collision (in those who had recovered to levels of no or mild LBP) and the development of future LBP. The results demonstrate that the development of future LBP is higher in individuals with a past self-reported low back injury resulting from a motor vehicle collision compared to those who have not (Nolet et al., 2017). Last, a study of insured drivers by Berglund and colleagues found an increased risk of low back pain 7 years post-collision in individuals with some form of injury in a rear-end collision compared to controls (Berglund et al., 2001).

Conversely, some studies have not demonstrated a relationship between low speed collisions and LBP reporting. Yang and colleagues (2013) examined the incidence of spine injury in rear impact collisions across all collision severities with rear-end collision data from the National Automotive Sampling System. Their analysis indicated that of all cases analyzed, approximately 8% of those cases reported some type of lumbar injury and most of these reports were associated with musculoskeletal strains or sprains (Yang et al., 2013). Similarly, Richards et al., (2006), performed a review of available frontal crash data and found that there was less than a 2% risk of moderate or greater injury in the lumbar or thoracic spine for belted occupants exposed to a collision severity of less than 20 km/hour (Richards et al., 2006).

There are a number of limitations associated with the studies completed to date. First, documentation employed in these studies was largely based on occupant reporting, scientific collision reconstruction often did not accompany the reports to estimate specific collision circumstances, such as the severity of the collision (expressed as a change in velocity) and the direction of collision forces. As a result, previous conclusions are limited due to the susceptibility of recall and response bias from the injured parties. Further, the prior studies have not reported if prior low back injury, degeneration or LBP complaints existed prior to the collision. LBP is a common complaint in a review of the epidemiological literature. Hestabek (2003) found estimates for the prevalence of LBP ranging from 7 to 39% for the general population. For those who have had a prior episode of LBP, the risk of developing a recurrent LBP episode doubles, with an increase in prevalence from 14 to 93% (Hestbaek et al., 2003). Therefore, treating all cases of LBP associated with collisions as an isolated occurrence of LBP may lead to erroneous estimates, when prior instances of LBP are not considered. The use of epidemiological data in regard to LBP reporting in the absence of consideration for specific collision circumstances and the presence of prior degeneration and LBP, is insufficient to establish links between collision circumstances and injury outcomes.

To understand causation of low back injuries claimed after collisions, forensic engineering firms are engaged to determine the likelihood that an injury was sustained attributable to the event. As a result, they have unique access to details from the collision to allow for an understanding of the detailed collision characteristics, such as vehicle velocity changes and vehicle orientations. In addition, hospital reports and medical records are reviewed to assess the injuries claimed and to identify any associated pre-existing medical conditions. This unique source of data provides the rare opportunity to match reported collision circumstances with reported LBP outcomes, limiting the need for assessors, researchers, and triers-of-fact to solely rely on occupant recall and reporting in understanding causation. To date, this data is rarely analyzed to understand 'population' trends and relationships. In order to identify potential low back injury mechanisms resulting from low velocity collisions, it is necessary to characterize and identify the physical dynamics of low to moderate velocity collisions that result in LBP claims and establish links between collision circumstances and injury outcomes. Therefore, the primary purpose of this investigation was to define the circumstances of low velocity motor vehicle collisions that result in litigation in Ontario with claims of low back injury.

3.3 Methods

This investigation involved the secondary analysis of a data set obtained from a forensic engineering firm based in Toronto, Ontario, Canada. This approach was reviewed and approved by the University of Waterloo Office of Research Ethics. Each case had a paper file associated with it and was entered into an internal electronic database. The database is searchable by a number of different fields including: start/finish date of the file, incident date, supervising engineer, project description and case number.

The searchable database encompassed 15 years of cases (2000-2014) and was searched using the following specific key words: "low speed", "low velocity", "rear-end", "side swipe", "biomechanical", "WAD", "impact" and/or "lateral impact". All of the cases returned based on the search criteria were documented using the case number to keep personal information confidential. The cases were then evaluated and only cases that included a biomechanical evaluation of the injuries sustained with a determined collision severity of 25 km/hour or less were included in the analysis. Any cases involving air bag deployment or a collision with a larger vehicle (i.e.: transport truck, garbage truck etc.) were excluded from the analysis.

Once a list of useable case numbers was developed, the cases were documented based on the criteria listed in Table 3.1. For each case, information was taken from formal reports completed by the forensic engineering firm. In the case that a formal report was not written, information was collected from available police reports and medical records. If a case included multiple injured persons, each person was entered as a new data entry including specific injury information for each person.

Table 3.1: Reported variables from the data mining

Variable	**Description**
Age	The age of the victim at the time of the loss
Gender	Male or Female
Seating Position	The seating position of the victim prior to the collision (i.e. Driver, Front Passenger etc.)
Severity of Collision	The estimated change in velocity experienced by the claimant
Collision Configuration	The type of collision (e.g. Frontal, Rear-End, Sideswipe)
Restraint Use	Yes or No
Injured Area of Body	The area of the body in which the victim is claiming injury e.g.: Neck, Right Shoulder, Low back etc.
Cost of Vehicle Repair	Obtained from insurance receipts
Event Data Record Present	Yes or No
Tow Away vs. Self-Reported	If the vehicle was towed away at the scene of the accident or if the claimant reported to a collision reporting centre to have the vehicle damage assessed
Frequency of Pre-existing Complaints	List of pre-existing complaints if medical history is present
Vehicle Type	Make, Model and Year of the Vehicle
Number of Occupants	Number of occupants in the vehicle at the time of the collision
Disc Degeneration Present	Yes or No; If present the level where degeneration was present.
Previous Accident	Number of previous accidents and estimated severity of each

3.4 Statistical Analysis

For the total sample, descriptive statistics or percentages of total cases were computed for age, gender, collision severity, collision configuration, tow away vs. self reported accidents and LBP claims.

For those cases that resulted in a LBP claim, descriptive statistics or percentages of total cases were computed for claimant age, seating position, restraint use, history of LBP reporting, evidence of disc degeneration, seating position, collision configuration, collision severity, prior collision and vehicle type. The proportion of claimants with a LBP claim and an accompanying WAD claim was computed.

3.5 Results

3.5.1 Overview of all Cases

In total, eighty-three cases met the inclusion criteria. The automotive collisions assessed occurred between the years of 2002 and 2014. Males and females were roughly equally distributed across all claims; 45.8 percent of all claimants were male and 54.2 percent were female. The mean (Standard Deviation (SD)) age across all claimants was 41.2 (10.6) years of age, with an age range of 15 to 65 years of age. One claimant's age was undisclosed and was therefore left unknown.

The mean collision severity (SD) across all claims was 11.9 km/hour (4.5), with a range of 2 km/hour to 25 km/hour. Rear End collisions dominated the distribution of collision types (70%). The remaining 30 percent of cases involved collisions of Side Swipe (7%), Side Impact (11%), Frontal Collisions (6%) or Other (6%) types of collisions. In total, 7 percent of cases involved vehicles which were towed away from the scene of the accident. The remaining 93 percent of cases were documented through self-reporting at an accident reporting centre. Only one case involved an individual whom was not wearing their seatbelt; this claim did not involve reported LBP.

3.5.2 Low Back Pain Specific Cases

Out of all 83 cases 77% involved a claim of LBP. The mean (SD) age across LBP claimants was 41.5 (10.2) years of age, 78% of claimants were the driver of the vehicle and 100% of claimants were wearing their seat belt.

3.5.2.1 Low Back Pain Cases & Occupant Characteristics

The most common pre-existing medical condition was pre-existing LBP or evidence of lumbar disc degeneration. Of those cases involving a claim of LBP, 63% of claimants either had a history of reporting LBP disclosed in their medical records, displayed evidence of lumbar disc degeneration in their medical records or had both a history of LBP and disc degeneration (Figure 3.1). Of those claimants with evidence of lumbar spine disc degeneration, 55% were between the ages of 41 to 50 years old (Figure 3.2).

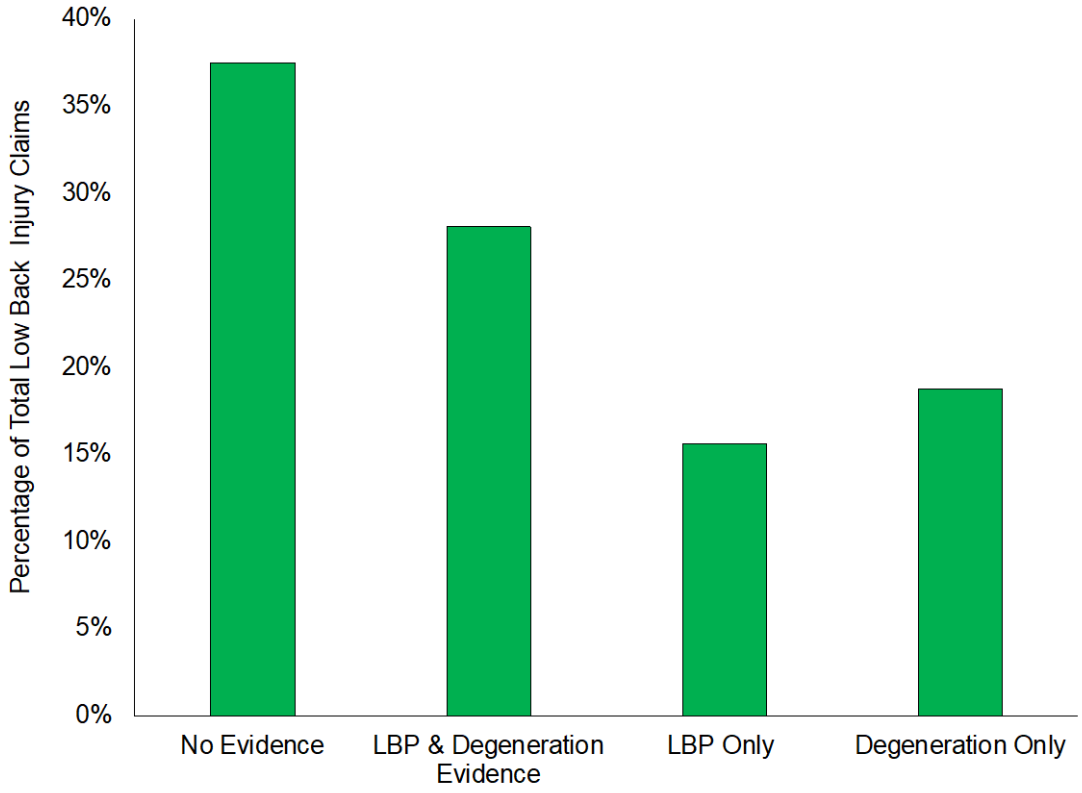

Figure 3.1 Prior LBP reporting and disc degeneration as a percentage of LBP claims (Total = 64).

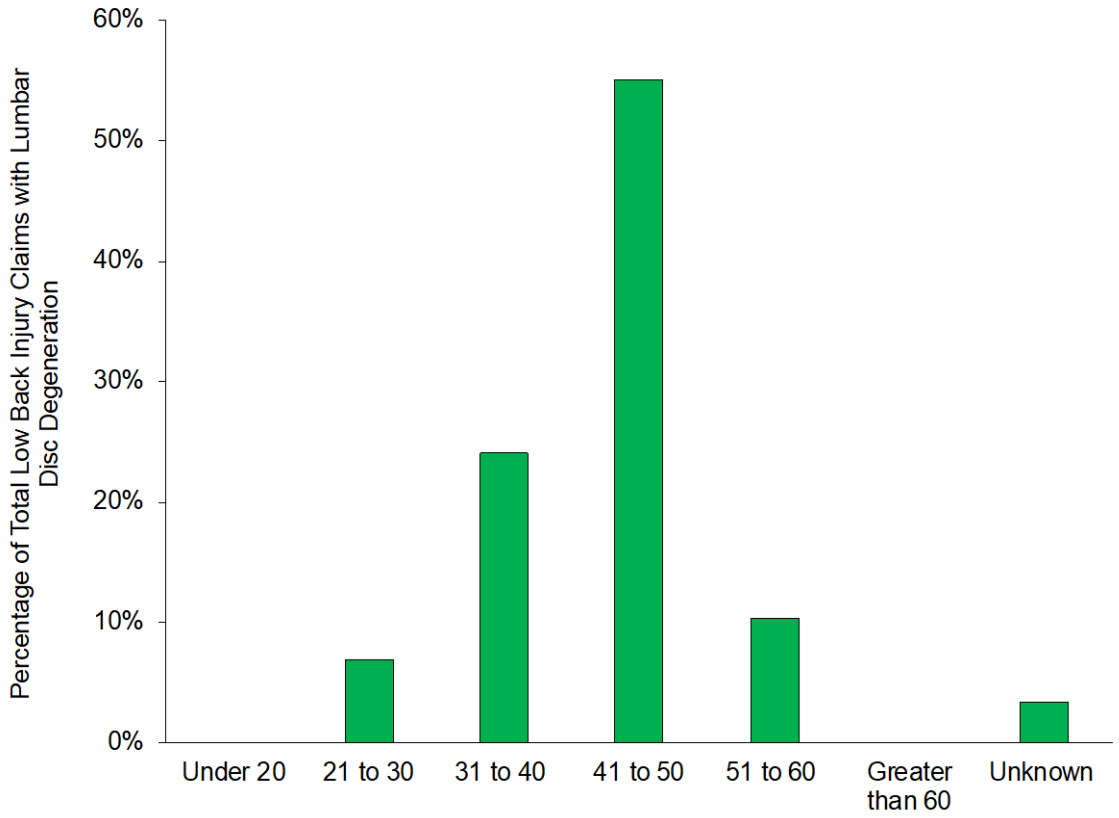

Figure 3.2: Claimants of LBP with evidence of disc degeneration as a function of age (Total = 29 claimants).

Of all low back injury claims, 97% were accompanied by a WAD diagnosis. Thirty-six percent of LBP case claimants had reported that they had been involved in a previous collision. Seventy-eight percent of those in previous collisions also had a history of LBP reporting.

3.2.2 Low Back Pain Cases & Collision Characteristics

A rear-end impact was the most common collision configuration (70% of all LBP claims involved a rear-end collision) (Figure 3.3). The mean collision severity (SD) across all LBP claims was 11.7 km/hour (4.5), with a range of 2 km/hour to 25 km/hour. The majority of all LBP claimants experienced a change in velocity of 13 km/hour or less (69%), with 42% of all LBP claims falling between collision severities of 10 – 12 km/hour (Figure 3.3). Sedans and minivans were the most common types of vehicles, accounting for 72% and 19% of all vehicle types respectively.

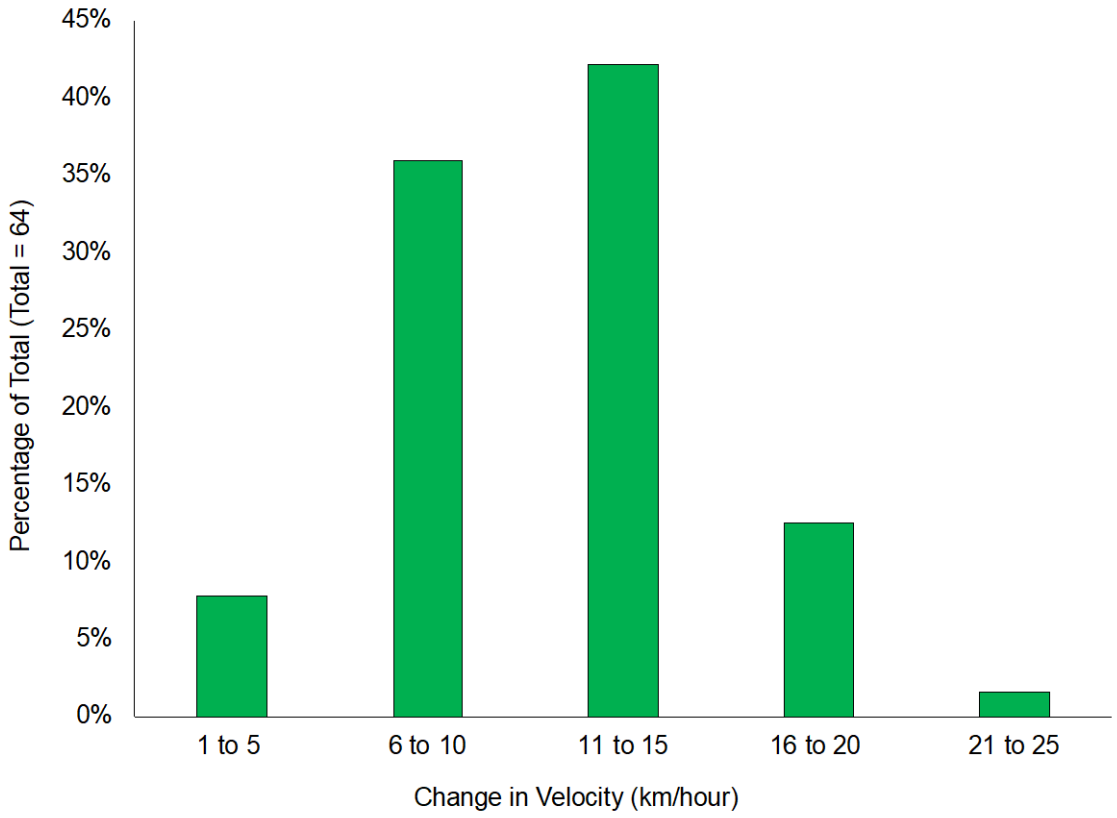

Figure 3.3: Percentage of LBP claims as a function of collision severity.

3.5 Discussion

The current investigation characterized low speed motor vehicle collisions that resulted in claims of LBP in Southern Ontario. In total, 84 forensically investigated claims were reviewed. It was found that low speed rear impact collision configurations at collision severities of 13 km/hour or less were most commonly associated with claims of LBP. This was the first investigation to examine medical history associated with claimed LBP in low speed collisions. It was found that pre-existing LBP and lumbar spine disc degeneration were particularly common in those with LBP complaints. Last, this investigation found that 97 percent of all LBP claims also had an accompanying WAD diagnosis.

This was the first investigation to exclusively investigate low to moderate speed collisions (based on severity determinations obtained from collision reconstruction) and the frequency of LBP reporting following such collisions. The general trends observed in previous work are inconsistent with our study results; previous work has observed a lower percentage of LBP claims. It has been previously reported that 27 to 60% of WAD patients also claim LBP following a low

speed collision (Beattie and Lovell, 2010; Chapline et al., 2000; Gay and Abott, 1953; Goldberg and Neptune, 2007; Hincapié et al., 2010; Magnússon, 1994), which is lower than observed in our study sample (77%). However, one limitation of the current work is that the sample population examined exclusively included individuals advancing an injury claim resulting from a low speed collision that was being investigated and does not account for the thousands of low speed collisions that did not result in any such injury claim or investigation. Thus, it would be expected that this sample population would have a higher percentage of LBP claims. Nevertheless, this work does provide knowledge of the occupant characteristics and the collision characteristics that are resulting in claims of LBP and is the first to focus exclusively on low to moderate speed collisions and LBP reporting, with accompanying collision reconstruction.

This investigation demonstrates that a rear impact collision configuration most commonly resulted in claims of LBP (70% of all LBP claims). However, Yang and colleagues (2013) observed that the risk of obtaining a low back injury from a low speed rear impact collision was extremely low (Yang et al., 2013). Yang and colleagues analyzed cases based on the abbreviated injury scale (AIS) from the National Automotive Sampling System (NASS database) and predominantly focused on determining links between spinal pathologies (such as lumbar disc herniation) and rear end collisions (Yang et al., 2013). The work observed that the majority of documented lumbar spine injuries were classified as lumbar sprains and strains and the incidence of this type of injury was minimal (7.8% across all severities of collisions). However, a major limitation of this work is that the NASS database does not include pre-existing medical records and further medical follow-up. Injury reports in the NASS database are based on immediate medical attention received directly following the collision. Previous clinical work in WAD symptom reporting has demonstrated that nearly all symptoms experienced following a low speed rear-end collision occur within a 24 hour period after the collision and rarely occur immediately after the impact (Brault et al., 1998). In addition, previous whiplash and WAD work has demonstrated that rear-end impact configurations result in a higher frequency of whiplash injuries in comparison to all other crash configurations (Brault et al., 1998; Deans et al., 1987; Jakobsson et al., 2000; Otremski et al., 1989). Following this trend, this work has demonstrated that in comparison to all other collision configurations, rear-end impact configurations result in the highest frequency of LBP reporting.

Collision severities of 13 km/hour or less were most frequently associated with LBP

reporting, with LBP reports most frequently occurring with severities of 11 to 15 km/h. The bell-shaped relationship between collision severity and reporting of LBP contrasts with previous neck injury risk data, which demonstrated the risk of neck injury symptom reporting increasing with collision severity (Krafft et al., 2005). To date, previous work has demonstrated no association of collision severity with LBP reporting across all collision configurations (Beattie and Lovell, 2010) and low back injury reports in rear end collision configurations (Yang et al., 2013). One limitation when analyzing collisions of a lower severity is that the vehicles involved in such collisions sustain little or no visible damage. This reduces the resolution in the determination of collision severity through accident reconstruction. Automotive bumpers are designed to withstand substantial collision forces at low speeds without significant deformation to the bumper. Often, the impact related change in velocity of the struck vehicle cannot be precisely determined, as at low speeds there is no convenient method by which lack of structural damage can be related to a specific change in velocity (Howard et al., 1999). It is also possible that a higher frequency of low speed collisions occurs at collision severities of 13 km/hour or less and as a result it appears as though more injury claims are resulting at these collision severities. Given the low speed nature of the collisions analyzed in this investigation and the limited availability of such databases, it was not possible to compare the results from this study to the total proportion of collisions that occurred across various collision severities.

This study demonstrates that pre-existing LBP and lumbar disc denegation are potential risk factors for reporting LBP following a low to moderate speed collision. LBP is a well-known major contributor to escalating health care costs and disability. It is estimated that 70-85% of all adults experience a significant episode of LBP at some point in their lives (Giesecke et al., 2004). Thus, it is not surprising that a high incidence of LBP claimants in this investigation (44% of all LBP claimants) had a history of LBP reporting prior to the accident. It is estimated that for individuals with a history of LBP, the risk of re-developing LBP doubles, putting these individuals at greater risk of reporting LBP in the future. The results from this investigation agree with these statistics. When looking across all reviewed cases, 31 claimants had a history of prior LBP documented in their medical history and 94 percent of these claimants claimed LBP following the analyzed collision.

This study was limited in a number of respects. First, it is not clear how the sample relates to the general population. In general, cases are brought to litigation if the injuries are severe enough

to affect an individual's quality of life and are associated with significant financial outcomes. Therefore, the current dataset may represent worst case scenarios for injury reporting after low speed automotive collisions. Second, the cases included in this investigation occurred in Ontario, which works on a hybrid based insurance system blending no-fault insurance and tort law. As a result, claimants who are not at fault for the collision in question can access the courts to increase payments for pain and suffering. When financial compensation is determined by pain and suffering, it may promote ongoing illness behavior and disability. Cassidy and colleagues demonstrated a 31% decrease in LBP claims in the province of Saskatchewan in the first 6 months of moving to a no-fault insurance system from tort law (Cassidy et al., 2003). Third, the dataset is small in comparison to the total number of low speed collisions that occur in Southern Ontario. Moving forward, pooling data from other forensic firms or working with insurance companies with larger databases may support greater confidence in the generalizability of findings and possibly allow for the development of predictive models. Fourth, claimants included in this investigation were evaluated exclusively on symptom reporting. Thus, based on this data alone, it is unknown if physical mechanical injury accompanied symptom reporting, as a number of psychosocial issues are known to be present in such claims. Fifth, the average vehicle age within the assessed data base was older (average vehicle age—1999), as such the injury reports here may not be directly comparable to newer vehicles with improved bumper designs. Last, the results from this investigation were descriptive in nature and thus inferences about the association between collision severity and low back injury claims cannot be directly made.

3.6 Conclusions

This study presents a novel analysis of unique dataset and provides a more thorough analysis of low speed rear end collision dynamics and LBP complaints. The results confirm that relationships do exist between LBP reporting, collision dynamics and occupant characteristics. In regard to collision characteristics, it was found that 69% of LBP claims resulted from rear-impact collision configurations of collision severities of 13 km/hour or less. For occupants, it was found that a history of LBP reporting or evidence of lumbar disc degeneration was most commonly associated with LBP reporting following a low to moderate speed collision. The results of this investigation provide knowledge of collision characteristics that can be employed in future studies that may

attempt to identify mechanisms for low back injury in low speed motor vehicle accidents.

Chapter 4: Study II Characterizing Trunk Muscle Activations During Simulated Low Speed Rear Impact Collisions

Components of this chapter have been published:

Fewster, K.M., Viggiani, D., Gooyers, C.E., Parkinson, R.J., Callaghan, J.P., 2019. Characterizing trunk muscle activations during simulated low-speed rear impact collisions. Traffic Inj. Prev.

4.1 Overview

Background: The internal forces generated by the musculature of the lumbar region create most of the mechanical load placed on the spine. Thus, despite the anticipated low external forces generated between the occupant and the automobile seatback during a low speed rear impact collision, increased muscle tension may influence the resultant peak joint loads experienced in the lumbar spine. Consequently, the risk of low back injury may be altered by muscle activation.

Objective: The purpose of this study was to evaluate the activation profiles of muscles surrounding the lumbar spine during unanticipated and braced simulated rear-end collisions.

Methods: Twenty-two low speed sled tests were performed on eleven human volunteers ($\triangle v = 4$ km/h). Each volunteer was exposed to one unanticipated impact and one braced impact. Accelerometers were mounted on the test sled and participants' low back. Six bilateral channels of surface electromyography (EMG) were collected from the trunk during impact trials. Peak lumbar accelerations, peak muscle activation delay, muscle onset time and peak EMG magnitudes, normalized to maximum voluntary contractions (MVC), were examined across test conditions.

Results: While not statistically significant, bracing for impact tended to reduce peak lumbar acceleration in the initial rearward impact phase of the occupant's motion by approximately 15%. The only trunk muscles with peak activations exceeding 10% MVC during the unanticipated impact were the thoracic erector spinae. *Time of* peak muscle activation was slightly longer for the unanticipated condition (unanticipated = 296 ms; braced = 241 ms).

Conclusions: Results from this investigation demonstrate that during an unanticipated low speed rear-end collision, the peak activation of muscles in the lumbar spine are low in magnitude. As such, muscle activation likely has minimal contribution to the internal joint loads that are experienced in the lumbar intervertebral joints during low speed rear impact collisions.

Keywords: Biomechanics, Lumbar Spine, Electromyography, Motor Vehicle Collision, Rear Impact

4.2 Introduction

Few studies have investigated the risk of injury in lumbar spine during rear impacts, which likely reflects the perception that there is minimal risk of injury since this body region is well supported by automotive seat backs. To date, there have been no human volunteer studies conducted to evaluate the risk of low back injury in low velocity rear impact collisions. As a result, the validation of computational models and biofidelic anthropomorphic test devices (ATDs) in the lumbar spine region has been limited, requiring more exposure data to accurately model the mechanical response of the lumbar spine joints during low speed rear impact collisions.

Studies involving post-mortem human surrogates (PMHS) are often used to validate the responses of computational models and ATDs. However, an inherent limitation of PMHS studies is that the responses do not include the effects of muscle activation. An abundance of human volunteer studies have been conducted at various collision severities (ranging from 4 km/h to 15 km/h) to determine the muscle activation responses and thresholds associated with cervical spine symptom reporting (Bailey et al., 1995; Brault et al., 1998; Castro et al., 1997; Mang et al., 2014; McConnell et al., 1995a; Siegmund et al., 2003; Szabo and Welcher, 1996). Previous investigations have demonstrated lower angular head accelerations in male participants, and smaller head retractions in female participants due to pre-impact bracing of the cervical spine muscles (Siegmund et al., 2003). In low speed frontal collisions, bracing has been shown to reduce the forward excursion of the knees, hips, elbows, shoulders, and head (Beeman et al., 2011). Bracing has also been found to reduce peak shoulder and retractor belt forces in a 50^{th} percentile male population exposed to low speed frontal collisions (5 and 10 km/hour) (Kemper et al., 2014). Therefore, the development of muscle tension has been shown to change an occupant's initial posture and joint kinematics during low-speed sled tests (Beeman et al., 2011; Begeman et al., 1980; Kemper et al., 2014) and must be considered if muscle activation levels warrant.

The internal forces generated by the musculature of the lumbar region create most of the mechanical load placed on the spine. A number of electromyography (EMG) driven biomechanical models have been developed to estimate the mechanical loading of the lumbar spine during occupational tasks (S M McGill and Norman, 1986; Reilly and Marras, 1989). These models emphasize the mechanical challenges that exist in the lumbar spine due to the small moment arms of the surrounding trunk muscles. For this reason, when external loads are applied, the compressive

and shear forces acting on the lumbar spine are generated primarily by the surrounding trunk muscles, rather than the reaction forces. Thus, despite the anticipated low external forces generated between the occupant and the automobile seatback during a low speed rear impact collision, increased muscle tension may influence the resultant peak joint loads experienced in the lumbar spine. Consequently, the risk of low back injury may be altered by muscle activation.

Therefore, the purpose of this study was to evaluate muscle activation in muscles surrounding the lumbar spine during unanticipated and braced simulated rear impact collisions. We hypothesized that bracing for impact would result in an increase in muscle activation, which would subsequently influence the resultant lumbar accelerations. Results from this investigation will further provide insight into the likely contribution of trunk muscle tension on the resultant joint loads that are experienced in the lumbar spine during low speed rear impact collisions.

4.3 Methods

Twelve participants (8 Male, 4 Female) between the ages of 18-45 were recruited (8 male, 26.8 ± 3.7 years, 1.81 ± 0.06 m, 85.9 ± 13.4 kg; 4 female, 26.0 ± 3.1 years, 1.62 ± 0.02 m, 60.8 ± 5.9 kg). The inclusion criteria required that all participants were free of any lumbar or cervical injury and had no previous history of prolonged low back or neck pain, previous lumbar surgery, cervical surgery or hip surgery in the past year, or have been involved in a previous automobile collision in the past 24 months. Ethics approval for research involving Human Subjects was obtained from the Office for Research Ethics at the University of Waterloo.

4.3.1 Experimental Setup

A rear impact crash sled, similar to those used in previous investigations (Kaneoka et al. 1999; Ono et al. 1997), was used to simulate rear-end motor vehicle collisions with a severity of 4 km/h (Appendix A, Figure A1, Figure A2, Table A3). A lower collision severity was used (in comparison to Study 3) to facilitate multiple collision simulations within the same collection. Sled acceleration was measured with a triaxial accelerometer (ADXL377, Analog Devices, Norwood, MA, USA), rigidly mounted to the frame directly under the automobile seat. The z-axis of the accelerometer was aligned with the direction of movement and used for all further sled acceleration analysis.

4.3.2 Participant Instrumentation

A triaxial accelerometer (ADXL377, Analog Devices, Norwood, MA, USA), was attached over the approximate location between the 4th and 5th lumbar vertebra using adhesive patches. This location was determined by palpating the spinous processes of the 4th and 5th lumbar vertebra and placing the accelerometer between to two respective spinous processes. Surface electromyography (EMG) was obtained from six muscles bilaterally: latissimus dorsi lateral to the T9 spinous process over the muscle belly (LAT), thoracic erector spinae at the level of the ninth thoracic vertebrae (TES), lumbar erector spinae at the level of the third lumbar vertebrae (LES), rectus abdominus approximately 3 cm lateral to the umbilicus (RA), external abdominal obliques approximately 15 cm lateral to the umbilicus (EO) and internal abdominal obliques midway between the anterior superior iliac spine (ASIS) and symphysis pubis (IO). Disposable, pre-gelled Silver-Silver Chloride electrodes (Blue Sensor, Ambu A/S, Denmark) were applied with a 2 cm center-to-center inter-electrode distance. Raw EMG signals were amplified using an AMT-8 amplifier (Bortec, Calgary, Canada; Bandwidth 10-1000 Hz, CMRR 115 dB @ 60 Hz, input impedance 10 GΩ) and collected at 2500 Hz using a 16-bit AD card with an input range of +/- 10 V. Maximum voluntary contractions (MVC) were collected from each muscle for normalization purposes. For the thoracic and lumbar erector spinae, participants laid prone on a table with their torso hanging off the edge of the table at the level of their ASIS. Participants crossed their arms over their chest, bent their torso towards the ground as a starting position and then extended their trunk to meet resistance applied by the experimenter (Dankaerts et al., 2004). For the rectus abdominus, internal oblique and external oblique participants laid supine on the table in the sit up position and performed right and left rotation against resistance (Dankaerts et al., 2004). Lastly, the latissimus dorsi MVC was conducted by manually resisting participants as they attempted a pull-down maneuver (McDonald et al., 2017). Five second rest trials were collected in the prone and supine positions.

4.3.3 Test Conditions

Two simulated impacts were performed for each participant, one unanticipated and one braced, with a collision severity of 4 km/h. Participants sat in the collision simulation sled with their hips and back centered right to left on the seat pan and seat back. Feet were placed on the foot pedals centered right to left and heel to toe. For all trials the standard 3-point seatbelt was positioned across the participant with the slack removed. The D-ring of the seatbelt was fully adjustable and adjusted such that the shoulder belt was centered across the clavicle and the lap belt was secured

slightly below the right and left ASIS. In addition, the headrest was adjusted in accordance to recommendations from the Insurance Bureau of Canada, with the headrest placed level with the top of the head and 5 to 10 cm back from the occupants' head. During the unanticipated trials, participants wore a blindfold and noise cancelling headphones playing music. Participants were told to remain relaxed, breathe normally, and face forward with their hands placed on their thighs. The only information provided to the participant about the simulated collision was that they would experience a simulated rear-end collision with a speed change of 4 km/hour. They were informed that the impact would occur, at random, sometime within the next 30 minutes. In practice, participants were released within 10 minutes. During the bracing trials, standardized instructions were used and each participant was instructed to push with maximum effort on the poles and foot pedals with their upper and lower extremities. Poles were used to brace against to allow for maximal exertion to provide a best-case bracing effect. A countdown was used to instruct the volunteers when to brace prior to the initiation of the sled release. Each of the two collision simulations were always performed in the same order for each participant; first, unanticipated, and second, braced (Kemper et al., 2014). A standing wait time of approximately 15 minutes was provided between tests. The order of trials and wait time was selected to minimize potential learned behaviour such as bracing during the unanticipated trials, and to minimize time-varying musculoskeletal responses to sitting (De Carvalho and Callaghan, 2011).

4.3.4 Data Acquisition and Processing

All data were recorded at 2500 Hz. Crash sled acceleration and lumbar acceleration data were filtered using SAE Channel Frequency Class (CFC) 60 (Society of Automotive Engineers, 1995). All accelerometers were zeroed based on the average measurement recorded just prior to the release of the sled. The sled accelerometer was used to define the point of impact. Sled accelerometer data were integrated (trapezoidal method) to calculate the resultant change in velocity of the sled assembly. Resultant accelerations recorded from the lumbar spine calculated as the root mean squared (RMS) of the 3 orthogonal directions. The resultant peak lumbar acceleration was quantified, separately, for the rearward and rebound phase of participants' motion. The first peak resultant trunk acceleration following impact was computed as the peak rearward acceleration, while the second peak resultant acceleration following impact was computed as the peak rebound trunk acceleration.

4.3.4.1 Peak Muscle Activation – Time and Magnitude

EMG signals were high-pass filtered using a 30 Hz, second order Butterworth filter, to remove ECG contamination (Drake & Callaghan, 2006). EMG signals were then full-wave rectified and single-pass filtered using a second order digital Butterworth filter with a cutoff frequency of 2.5 Hz (Brereton and McGill, 1998). The resulting linear enveloped signals were then normalized to MVC trials, resulting in normalized EMG signals expressed as a percentage of the maximum voluntary contraction (%MVC). The was done to obtain an estimate of when the muscles turned on (including the electromechanical delay). The peak muscle activation following impact was defined as the first peak in muscle activation following impact, the magnitude and time of peak (Time of Peak Muscle Activation) with respect to the time of impact were then extracted. The time of impact was computed from the accelerometer mounted to the sled assembly. Peak muscle activations were inspected visually and the first peak in muscle activity following the sled impact was chosen as the time in which peak muscle activation occurred. Last, peak magnitudes of normalized EMG throughout the entire collision simulation (regardless of impact time) were computed and averaged across right and left sides.

4.3.4.2 Onset of Muscle Activation

EMG signals were full-wave rectified and dual-pass filtered using a second order digital Butterworth filter with a cutoff frequency of 50 Hz (Hodges and Bui, 1996). To describe the time of onset of muscle activation during the impact trials, the time at which the signal exceeded 2 standard deviations above resting activation for a period of 20 ms or longer was extracted using custom code in MATLAB (vR2017a, Mathworks, Natick, MA, USA) (Hodges and Bui, 1996). Additionally, the onset time was confirmed visually. When a muscle's activation met this criterion, its timing was then referenced to the timing of peak sled acceleration.

4.3.5 Statistical Analysis

Paired t-tests were completed on right and left muscle onset time and peak normalized EMG magnitude. No statistical differences were observed bilaterally, thus right and left muscles were averaged. Paired t-tests comparing unanticipated versus braced collision simulations were completed on peak lumbar acceleration during both phases of occupant motion (rearward and rebound), muscle onset time, peak normalized EMG magnitude.

4.4 Results

Due to malfunction of the sled accelerometer one male participant was removed from the dataset, resulting in eleven participants total.

4.4.1 Peak Lumbar Acceleration

While not statistically significant ($p = 0.092$), a trend emerged that bracing for impact was associated with, on average, a 15% decrease in peak lumbar acceleration in the initial rearward phase of occupant motion (Figure 4.1). Peak lumbar acceleration during the initial rearward phase of occupant motion was 0.58 (0.20) g for the unanticipated condition, and 0.43 (0.11) g for the braced condition. Bracing did not affect the magnitude of peak lumbar acceleration during the forward rebound phase of motion ($p = 0.625$). Peak lumbar acceleration during the rebound phase of occupant motion was 1.56 (0.40) g for the unanticipated condition, and 1.51 (0.17) g for the braced condition. The average timing of peak rearward lumbar acceleration was 49 ms (standard deviation [SD]: 21ms) after the point of impact.

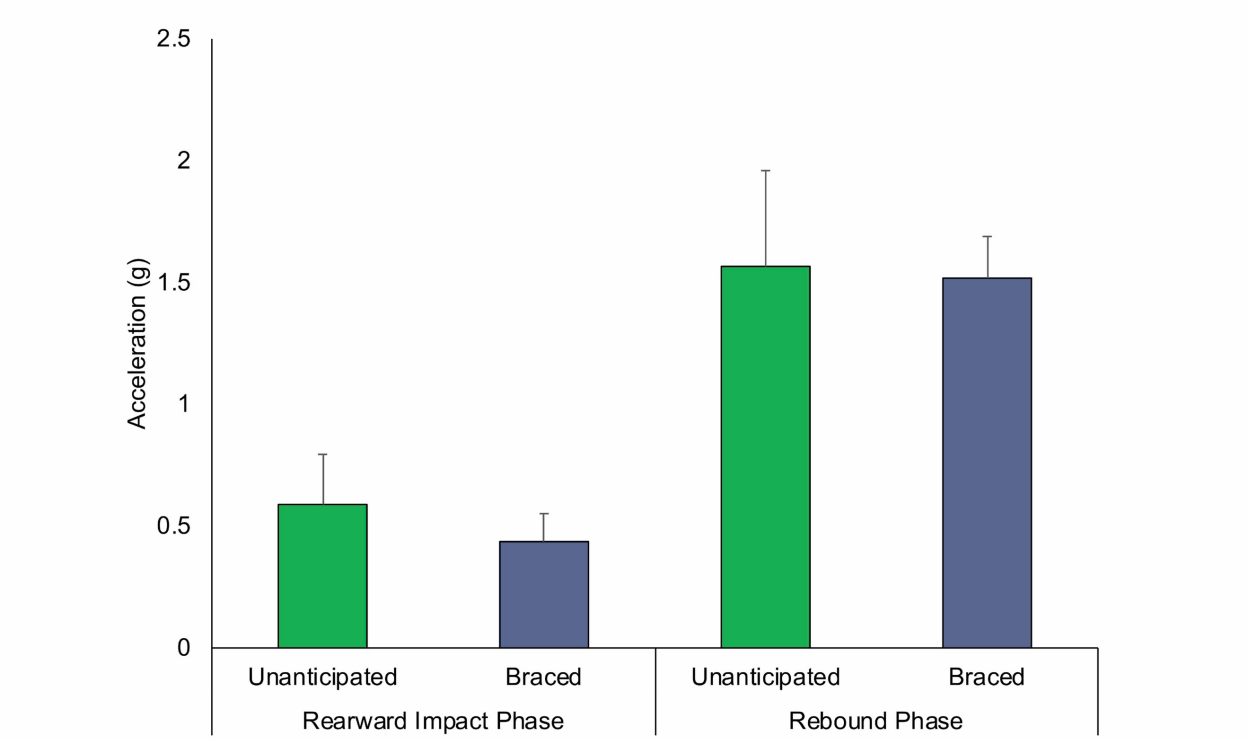

Figure 4.1: Peak resultant lumbar linear accelerations during the rearward impact phase and rebound phase of the collision. Standard deviation bars are displayed.

4.4.2 Peak Muscle Activation – Time and Magnitude

4.4.2.1 Time of Peak Muscle Activation Following Impact

While not statistically significant (IO: *p=0.553*, RA: *p= 0.492*, EO: *p=0.3146*, LES: *p=0.480*, TES: *p=0.348* LAT: *p=0.221*), with exception of TES, the time of peak muscle activation was later in all trunk muscles during the unanticipated impact (Figure 4.2). The TES had the reverse trend in which the time to peak muscle activation was longer in the braced impact. Across all muscles studied (except the TES), the average delay in peak muscle activation during the unanticipated impact was 297 ms (SD: 361 ms), while for the braced impact it was 211 ms (SD: 127 ms). For the TES the average delay in peak muscle activation during the unanticipated impact was 288 ms (SD: 194 ms), while for the braced impact it was 390 ms (SD: 348 ms).

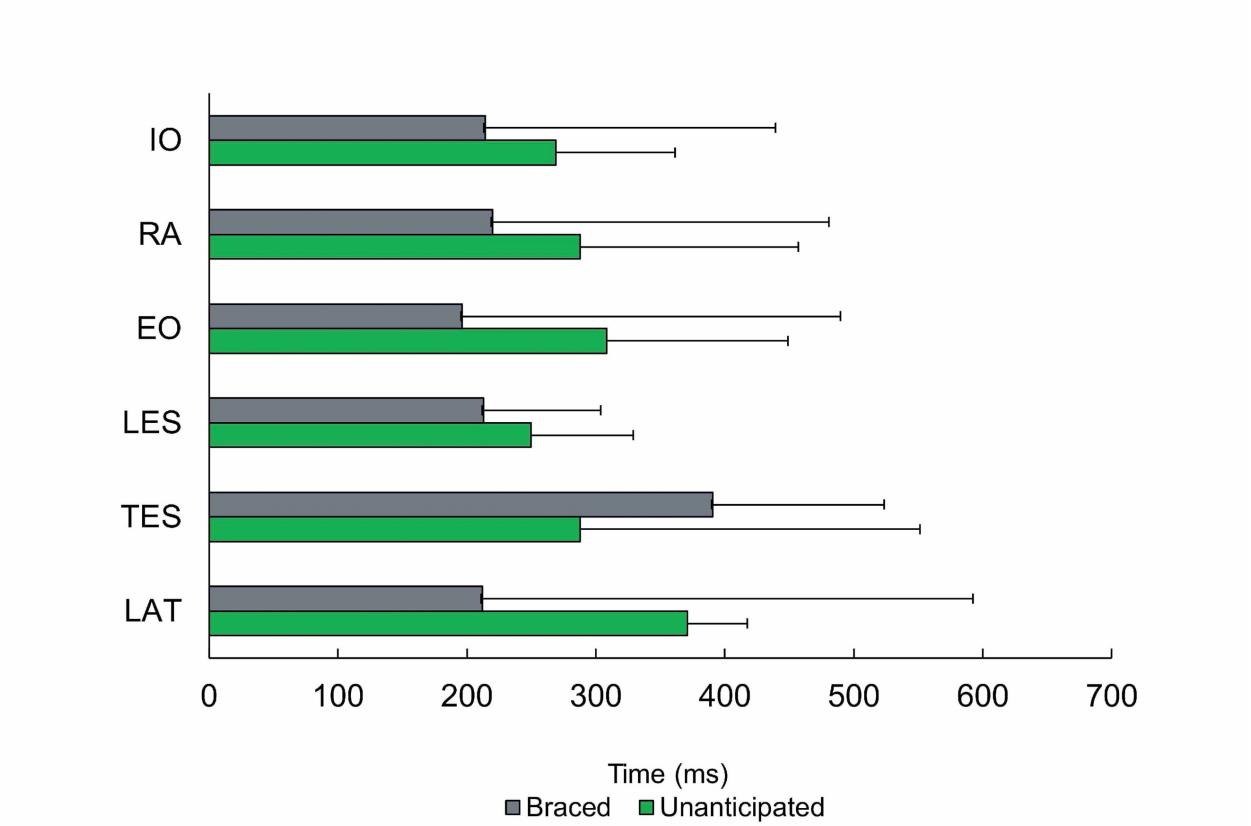

Figure 4.2: Peak muscle activation delay times following the simulated rear end collision. Delay times are averaged across the right and left muscles for Braced and Unanticipated impacts. Time zero is the point of impact of the sled. Standard deviation bars are displayed.

4.4.2.2 Peak EMG Magnitude Following Impact

LAT exhibited significantly higher peak muscle activation ($p = 0.042$) during the braced impact in comparison to the unanticipated impact (Figure 4.3). While none of the other muscles exhibited statistically significant differences in peak muscle activation (IO: $p=0.104$, RA: $p= 0.329$, EO: $p=0.312$, LES: $p=0.051$, TES: $p=0.172$), between unanticipated and braced impacts, there was a trend observed that peak muscle activation was generally higher during the braced impact. During the unanticipated impact the only muscle with peak activation magnitudes exceeding 10% MVC was the TES (Figure 4.3). During the braced impact the only muscle with peak activation magnitudes exceeding 20% MVC was the TES (Figure 4.3). During the braced impact the IO, LES, TES and LAT exceeded 10% MVC.

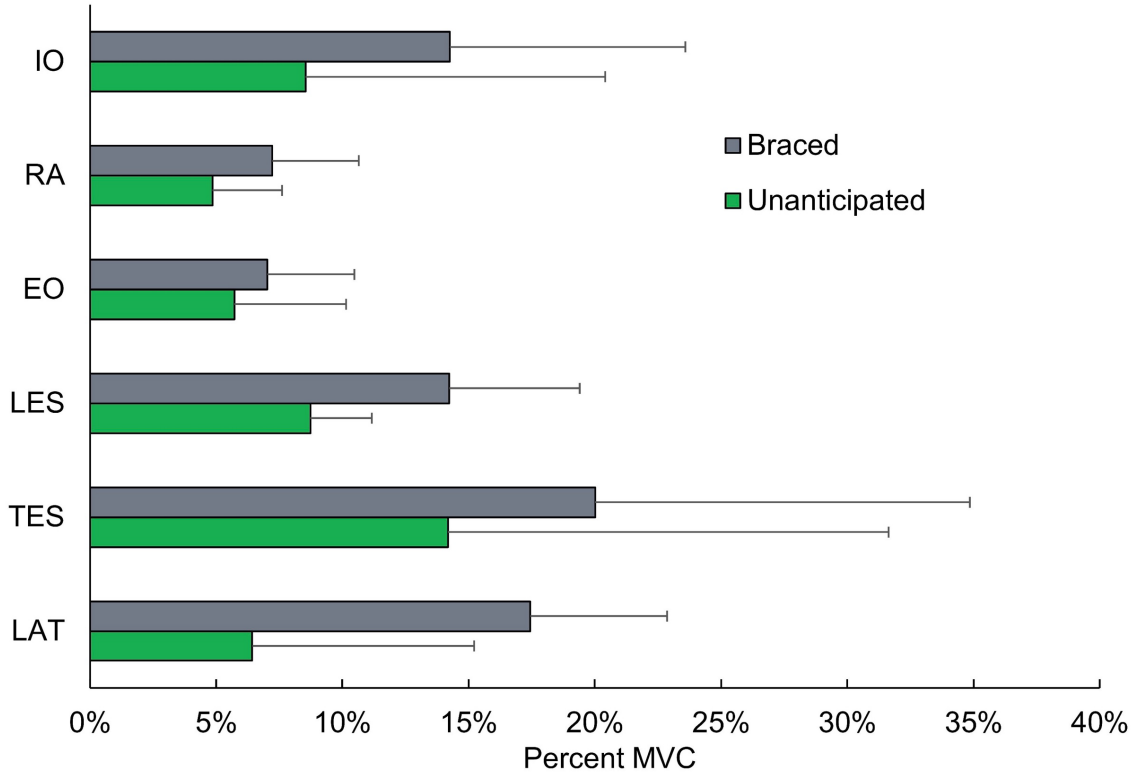

Figure 4.3: Average peak muscle activation as a percent of maximum voluntary contraction (MVC) for each of the six bilateral trunk muscles examined during the unanticipated and braced impacts.

4.4.3 Muscle Activation Onset Time

During the unanticipated trials, only a few instances occurred in which trunk muscles had activation onsets prior to impact (20 instances out of a total of 132 possible instances (6 muscles (bilaterally) x 11 participants)) (Figure 4.4). In contrast, for the braced trials, muscles more frequently met the activation criterion prior to impact (84 instances out of a total of 132 possible instances) (Figure 4.5).

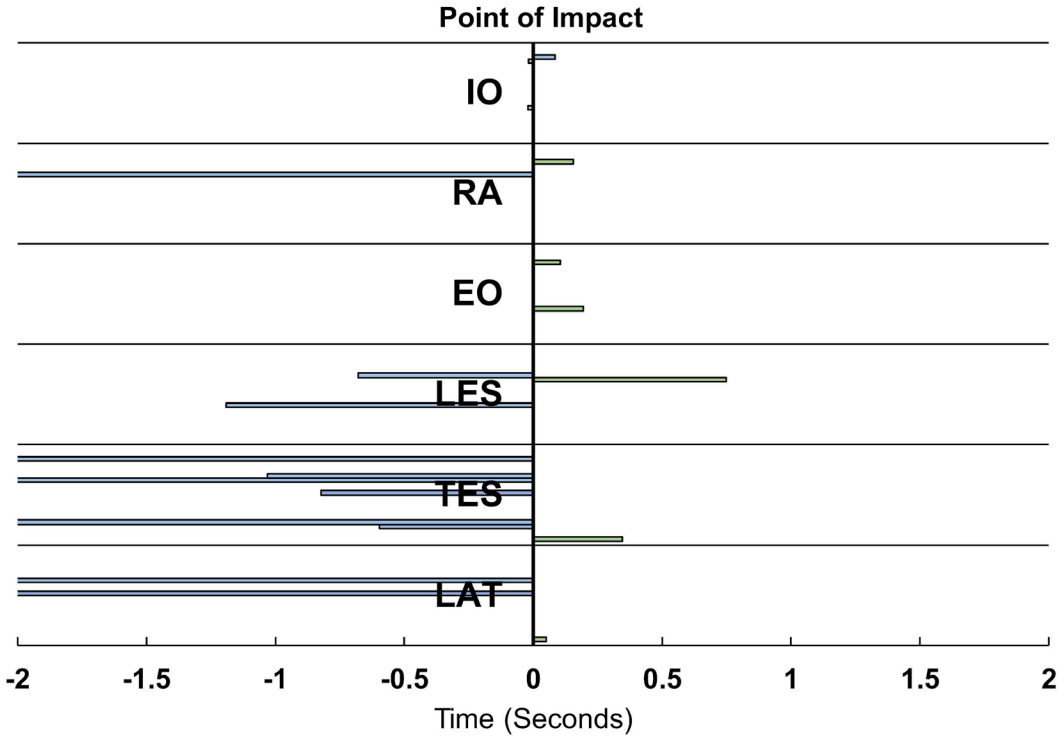

Figure 4.4: The number of instances (across 11 participants examined; unanticipated impact) and the timing relative to impact in which a muscle reached the activation criterion (2 standard deviations above resting activation for a period of 20 ms). Each bar represents a separate participant. Time is normalized to the point of impact (at time point zero) in which a negative value indicates pre-impact muscle activation onset, where a positive value indicates post-impact muscle onset.

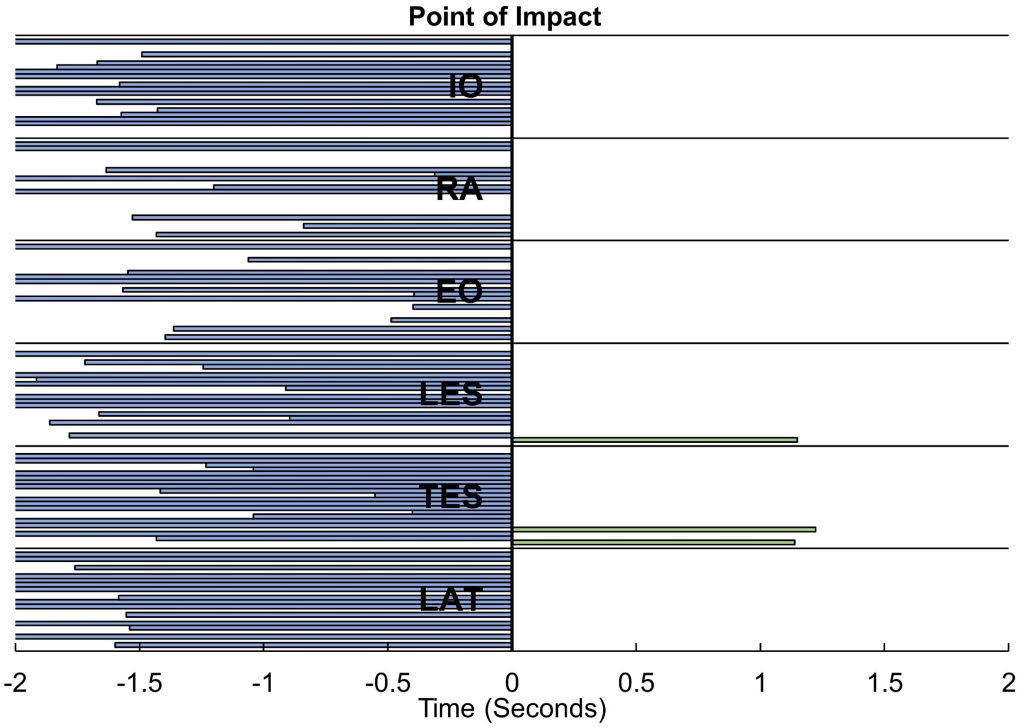

Figure 4.5: The number of instances (across 11 participants examined; braced impact) and the timing relative to impact in which a muscle reached the activation criterion (2 standard deviations above resting activation for a period of 20 ms). Each bar represents a separate participant. Time is normalized to the point of impact (at time point zero) in which a negative value indicates pre-impact muscle activation onset, where a positive value indicates post impact muscle activation onset.

4.5 Discussion

The current investigation exposed participants to two simulated rear impact collisions, one unanticipated and one braced, to quantify differences in trunk muscle activation and lumbar accelerations. Consistent with our hypothesis, greater peak muscle activations were observed in LAT during the braced impact in comparison to the unanticipated impact. This result may explain the slight decrease observed in the peak resultant lumbar accelerations in the initial rearward motion with the seatback. However, contrary to our hypothesis, no other trunk muscles exhibited statistically significant differences in peak activation magnitude between the braced and unanticipated impact. However, a trend was observed across all trunk muscles that greater peak activation occurred during the braced impact. In general, all muscles investigated had relatively low magnitudes of activation with muscles rarely exceeding 10% MVC in the unanticipated impact and no muscles exceeding 25% MVC in the braced impact.

The peak muscle activity observed during the unanticipated rear-end impacts was very low in magnitude (less than 15% MVC). This finding, combined with the activation timing, supports the hypothesis that muscle forces likely do not contribute a significant amount to the internal joint loads during unanticipated low speed rear impact collisions and measured reaction forces likely provide a good estimate of the internal joint loads. The average peak muscle activation onset times were delayed relative to peak impact accelerations by 296 ms in the unanticipated impact, and 241 ms in the braced impact. This misalignment of peak muscle activity and peak acceleration indicates that muscular responses of the trunk and lumbar spine are too slow to resist any initial impact forces, unless those muscles are already contracted (as in the braced collision scenario). In the braced impact, the LAT, TES and LES were the muscle groups most frequently activated prior to impact, in comparison to the abdominal muscles (EO, IO, RA). Models of lumbar spine stability indicate that an increase in compressive forces and stiffness caused by higher muscle activation levels will decrease shearing and horizontal translation of lumbar vertebrae (Cholewicki et al., 1997; Gardner-Morse and Stokes, 1998). Thus, it is likely that the activation of the trunk muscles prior to the impact may have stiffened the lumbar spine, which in turn may have slightly decreased rearward linear lumbar accelerations. Since the magnitude of compressive force and stiffness is proportional to muscle activation (Gardner-Morse and Stokes, 1998), larger activations (up to 30% of MVC) may decrease this rearward acceleration even further.

The lack of statistically significant differences in peak muscle activations when braced was surprising. The only muscle with statistically significant peak muscle activation differences between unanticipated and braced impact was LAT. One explanation for the lack of statistically significant differences across braced and unanticipated impacts could be the low collision severity used in this investigation. Previous work by Kemper and colleagues (2014) has shown greater differences in linear and angular head accelerations as well as C7, sternum and sacrum acceleration between unanticipated and braced trials in their higher severity frontal impacts (10 km/h) than in their lower severity impacts (5 km/h). It is possible that this finding may also hold true for rear impact collision, where a higher collision severity might show greater differences in peak muscle activation magnitude. It is also noted that the unanticipated impact was always performed prior to the braced impact in the current study. Therefore, it is possible that once participants experienced the unanticipated low severity impact this could have influenced their behavior in the braced impact because they knew what the impact experience was like. Nonetheless, both impacts were

triggered at random within a 10-minute window to combat the potential influence of any learning effects.

The findings from this study should be interpreted in the context of some limitations. First, the sample size was small and reflected a healthy university-aged population. Therefore, the data from this study may not be applicable to all occupants across different ages. A second limitation was that whole-body kinematics were not monitored and therefore comparisons cannot be made in how bracing for impact influenced occupant joint motions and body segment excursions. Lastly, while we did review each participants' muscle activation traces manually, it is possible that due to the impact nature of the experiment, peak muscle activations may have been confounded by soft-tissue artifacts and/or the interaction of the EMG electrodes with the seatback.

4.6 Conclusions

In summary, results from this work demonstrate that during unanticipated rear impact collisions, the peak activation of trunk muscles are of low magnitudes. Based on our observations, it can be concluded that muscle activation likely has minimal contribution to the joint forces that are experienced in the intervertebral joints of the lumbar spine during low speed rear impact collisions. Furthermore, the delay observed between peak muscle activity and peak acceleration of the lumbar spine indicates that muscular contractions are too slow to resist any initial impact forces, unless they are recruited (braced) before impact. These findings justify the use of simplified models in estimating the resultant joint loads in the lumbar spine during low speed rear impact collisions and provide support the application of cadaveric testing to characterize the mechanical response in the lumbar spine.

Chapter 5: Study III - Characterizing *In Vivo* Mechanical Exposures of the Lumbar Spine During Simulated Low Velocity Rear Impact Collisions

5.1 Overview

Study Design: An experiment was conducted to explore *in vivo* lumbar kinematics, joint reaction forces and lumped passive tissue changes in response to laboratory-simulated 8 km/hour rear-end collisions with and without lumbar support.

Background: Historically, there has been a lack of focus on the lumbar spine during rear impacts because of the perception that the automotive seat back should protect the lumbar spine from injury. As a result, there have been no studies involving human volunteers to address the risk of low back injury in low velocity rear impact collisions.

Objectives: The primary objectives of this study were to explore lumbar kinematics and joint reaction forces in human volunteers during simulated rear impact collisions and to examine the influence of lumbar support on the peak motion and forces experienced in the lumbar spine. A secondary objective was to evaluate lumped passive stiffness changes and low back pain reporting after a simulated rear impact collision

Methods: Twenty-four participants (12 male, 12 female) were recruited. A custom-built crash sled was used to simulate unanticipated rear impact collisions, with a change in velocity of approximately 8 km/h. Randomized collisions were completed with and without lumbar support. Measures of passive stiffness and flexion-relaxation-ratio (FRR) were obtained prior to impact (Pre), immediately post impact (Post) and 24 hours post impact (Post-24). LBP reporting was monitored over the next 24 hours leading up to the final Post-24 measures. For collision simulations inverse dynamics analyses were conducted, and outputs were used to generate estimates of peak L4/L5 joint compression and shear. From the passive trials, lumbar flexion/extension moment-angle curves were generated to quantify time-varying changes in the passive stiffness of the lumbar spine, Post and Post-24 relative to Pre. FRRs were computed as the ratio of thoracic erector spinae and lumbar erector spinae muscle activation in an upright posture to muscle activation in a flexed position

Results: Average [± standard deviation] peak L4/L5 compression and shear reaction forces were not significantly different without lumbar support (Compression = 498.22 N [±178.0]; Shear = 302.2 N [± 98.5]) compared to with lumbar support (Compression = 484.5 N [±151.1]; Shear =

291.3 N [±176.8]). Lumbar flexion angle at the point of peak shear was 36 degrees [±12] without and 33 degrees [±11] with lumbar support, respectively, with 0 degrees being the lumbar posture in upright standing. No participants developed clinically significant levels of LBP after impact. Time was a significant factor for the length of the low stiffness flexion and extension zone ($p = 0.049$; $p = 0.035$), the length of the low stiffness zone was longer in the Post and Post-24 trial for low stiffness flexion and longer in the Post-24 for low stiffness extension.

Conclusions: Findings demonstrate that during a laboratory-simulation of an unanticipated 8 km/hour rear-impact collision, young healthy adults do not develop LBP. Lumbar support did not significantly influence the estimated L4/L5 joint reaction forces. Changes in the low stiffness portion of the passive flexion/extension curves were observed following impact and persisted for 24 hours. Changes in passive stiffness may lead to changes in the loads and load distributions within the passive structures such as the ligaments and intervertebral discs following impacts.

Keywords: lumped passive stiffness, lumbar spine, low speed rear impact

5.2 Introduction

Historically, there has been a lack of focus on the lumbar spine during low speed rear impact collisions because of the perception that the automotive seat back should protect the lumbar spine from injury. As a result, there have been no studies involving human volunteers to address the risk of low back injury in low velocity rear impact collisions. Given the limited information provided (Fast et al. 2002) and the high incidence of reported low back pain resulting from such collisions, this study seeks to establish an understanding of the lumbar spine forces and kinematics in simulated low velocity motor vehicle collisions.

Previous investigations, that have examined the motion and joint loads in Anthropometric Testing Devices (ATD's) (Gates et al. 2010; Gushue et al. 2001) and cadavers (Fast et al. 2002), have demonstrated that the peak exposures in the lumbar spine during a simulated rear impact collision are below existing injury reference values and within the range of loads experienced in manual materials handling. Gates and colleagues (2010) compared estimates of lumbar loads in an Hybrid III and a Biofidelic Rear Impact Dummy (BioRID) ATD's, at rear impact collision severities ranging from 8 to 24 km/h. The measured peak compressive force, across all collision severities, was well below the NIOSH action limit (3400 N) for occupational exposures. This finding is further supported by cadaveric research where a single male cadaver was exposed to collision severities of 13 and 19 km/h (Fast et al. 2002) where no boney injuries were observed and the lumbar compression forces were insufficient to result in acute injury. A major limitation of these results is that these physical models (ATDs and cadavers) have not been fully validated against human subject responses during low velocity rear-end collisions. This is because no such study exists, knowledge of lumbar kinematics and joint loads in human participants would add valuable insight into the validity of ATDs and cadaver responses in low velocity rear impact collisions.

Large differences in posture exist between standing with neutral lumbar lordosis and when seated in an automotive seat. When seated, the lumbar spine flattens completely, becoming more flexed, the pelvis rotates posteriorly and the intervertebral joints become flexed throughout the spine (De Carvalho and Callaghan, 2012). This decrease in lumbar lordosis in automotive seating can lead to increased strain in the posterior passive tissues (Solomonow et al., 2003), greater muscle activity (Andersson and Ortengren, 1974) and elevated disc pressure (Wilke et al., 1999). Thus, poor posture coupled with the effects of sudden loading resulting from a rear impact may be

a plausible mechanism for the high incidence of reported low back pain after a low velocity collision. Maintenance of a neutral spine posture has been suggested as an effective intervention to reduce low back discomfort in sitting (De Carvalho and Callaghan 2015). Lumbar supports in automotive seating have been shown to increase lumbar lordosis, or reduce the flexion induced by a seated posture, and decrease low back pain reporting (De Carvalho and Callaghan 2015; De Carvalho and Callaghan 2012); however, their effectiveness during a rear impact collision remains unknown.

Most patients involved in low velocity rear-end collisions presenting with low back pain display no significant findings upon radiographic examination and present without discernable tissue damage. Subjective pain reporting makes it difficult to associate low velocity collision exposure to physical mechanical injury or pathology. Passive stiffness and neutral zone characteristics have been demonstrated to be a sensitive measure for spinal instability and injury (Ivancic et al., 2005; Panjabi et al., 2005, 1989). Passive intervertebral joint range of motion can be split into a neutral zone and elastic zone. The neutral zone is the portion of physiological range of motion where spinal motion is produced with minimal resistance (Panjabi, 1992). Aberrant neutral zone characteristics have been associated with injury (Panjabi, 1992b). By characterizing lumped lumbar spine stiffness *in vivo*, changes in passive stiffness characteristics can be tracked pre and post collision. Such changes could indicate a mechanical insult to the passive tissues, which is a mechanism that has previously been shown to trigger transient low back pain (Winkelstein and DeLeo, 2004). In addition, changes in passive stiffness properties of the lumbar spine can lead to changes in the loads and load distributions within the ligaments, intervertebral discs, and muscles.

The exposures to the lumbar spine resulting from low velocity rear impacts remain unknown. Further, it is unknown how lumbar supports may alter these exposures. Therefore, the purpose of this study was to access peak lumbar spine loads and kinematics during simulated low velocity rear-end collisions with and without lumbar spine support. A secondary purpose of this study was to determine if passive lumbar spine stiffness properties change in response to a simulated low velocity rear impact collision and assess this relation with low back pain symptom reporting post collision. In line with these purposes, it was hypothesized that the mechanical exposures during a low velocity rear-end collision will change with lumbar support and this change will result in a different lumbar spine passive response.

5.3 Methods

5.3.1 Study Design

An *in vivo* biomechanics study to estimate L4/L5 joint loading during a simulated rear-end impact at a change in velocity of 8 km/hour (Figure 5.1). Participants were exposed to two rear-impact collision simulations; one without lumbar support and one with lumbar support, in a randomized order, separated by at least 2 weeks. Pre, Post and Post 24 hour testing was completed which included lumped lumbar passive stiffness quantification using a near frictionless jig and forward repeated flexion trials (Figure 5.1).

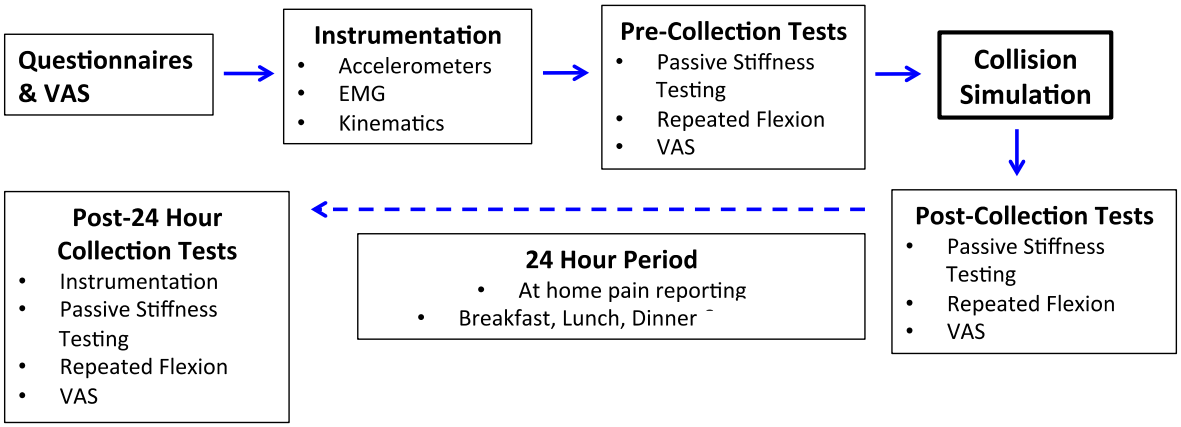

Figure 5.1: Experimental protocol for Study #3 which occurred for each simulated collision (with lumbar support and without lumbar support).

5.3.2 Participants

Twenty-four participants (12 male, 12 female) between the ages of 18-35 were recruited. All participants fell within the 10th to 90th percentile heights for gender and 10th to 90th percentile weights for their height (Table 5.1). The inclusion criteria to participate in this study was that all participants had to be free of any lumbar or cervical injury and have no previous history of low back or neck pain, previous lumbar surgery, cervical surgery or hip surgery, or have been involved in a previous automobile collision in the past 24 months.

5.3.3 Pre-Collection Recordings

5.3.3.1 Psychosocial Questionnaires

Upon entering the lab, participants filled out questionnaires to assess their pain attitudes and fear avoidance beliefs (Appendix B). Because these tests were administered on an asymptomatic population, rather than a clinical population, a modified questionnaire (Gallagher, 2014; Nelson-

Wong, 2009) with questions from the Cognitive Risk Profile for Pain, Survey of Pain Attitudes – Brief and Fear Avoidance Belief Questionnaire, were administered to the participants. This modified questionnaire has been used previously to assess psychosocial differences between people who develop low back pain and people who do not during prolonged standing (Gallagher, 2014; Nelson-Wong, 2009). It has been demonstrated that beliefs about activity, disease and work can contribute to the level of pain and disability experienced by an individual (Waddell, 2004).

5.3.3.2 Visual Analog Scale and Quality of Pain

To assess subjective low back pain following the low velocity rear-end collision simulation a digital 100 mm visual analog scale (VAS) was administered when participants first entered the laboratory, right before the simulated collision, right after the simulated collision and as they left the laboratory (approximately 30 minutes after the impact). The 100 mm horizontal line was anchored on either end with "No Pain" and "Worst Pain Imaginable" and this pain scale has been previously used to identify asymptomatic pain developers during prolonged standing (Gallagher and Callaghan 2015; Marshall, Patel, and Callaghan 2011; Nelson-Wong and Callaghan 2010).

Because participants could not be observed continuously after the test collisions, the assessment of the participants' pain experience was followed over a 24 hour period using at home pain reporting cards (Figure 5.2). This pain reporting card included a 100 mm VAS along with descriptions of pain sensations to be detected from the short-form McGill Pain Questionnaire (Gallagher, 2014; Melzack, 1975). Participants were required to check a box next to the words to describe their current status of their pain (Figure 5.2). The sensory words included: "throbbing", "shooting", "stabbing", "sharp", "cramping", "gnawing", "hot-burning", "aching", "heavy", "tender" and the affective will be "tiring-exhausting", "sickening", "fearful", and "cruel-punishing" (Melzack, 1975).

When each participant left the laboratory they were given 3 cards identical to Figure 5.2 in which they recorded their level of pain and pain sensation type at three different times throughout the day (e.g. dinner, bedtime, breakfast) between the time of their first post-impact reporting and the 24-hour follow-up testing. This provided a method of documentation away from the test site and allowed for continuous tracking of symptoms (Brault et al., 1998). One last digital pain measure was taken when the participant first arrived for their post-24 hour assessment.

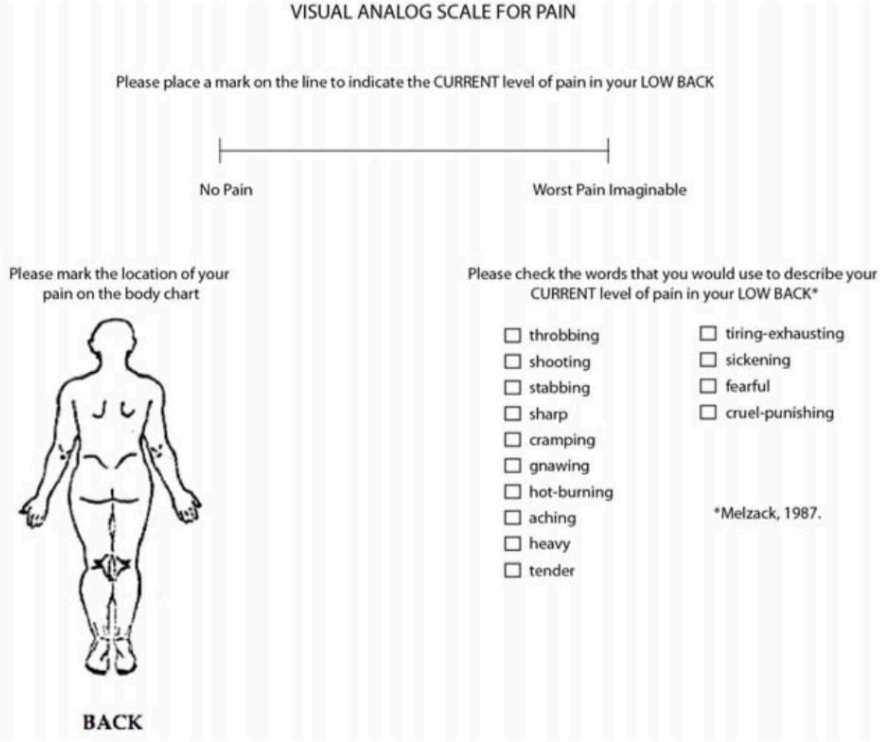

Figure 5.2: Visual Analog Scale for measurement of subjective pain ratings of low back pain

5.3.4 Instrumentation

5.3.4.1 Electromyography

Electromyography was used to track muscle activity during pre and post testing of forward flexion trials and passive testing. Pairs of surface electrodes were placed over the upper erector spinae (approximately 5 cm lateral to the T9 spinous process); lower erector spinae (approximately 3 cm lateral to the L3 spinous process); rectus abdominus (approximately 3 cm lateral to the umbilicus); external abdominal oblique (approximately 15 cm lateral to the umbilicus). A reference electrode was placed over a rib. Disposable, pre-gelled electrodes (Product #272, Noraxon, USA Inc., Arizona, USA) were applied with a 2 cm center-to-center inter-electrode distance. Raw EMG signals were amplified using an AMT-8 amplifier (Bortec, Calgary, Canada; Bandwidth 10-1000 Hz, CMRR 115 dB @ 60 Hz, input impedance 10 GΩ) and collected at 2500 Hz using a 16-bit AD card with an input range of +/- 10 V. Maximum voluntary contractions were collected from each muscle for normalization purposes. For the thoracic and lumbar erector spinae, participants laid prone on a table with their torso hanging off the edge of the table at the level of their anterior superior iliac spine (ASIS). Participants crossed their arms over their chest, bent their torso towards

the ground as a starting position and then extended their trunk to meet resistance applied by the experimenter (Dankaerts et al., 2004). For the rectus abdominus and external oblique participants laid supine on the table in the sit up position and performed right and left rotation against resistance (Dankaerts et al., 2004). Ten second rest trials were taken in both the prone and supine positions.

5.3.4.2 Accelerometers:

Sled accelerations were measured using one tri-axial accelerometer (ADXL377, Analog Devices, Norwood, MA, USA) rigidly attached to the sled frame. The z-axis of the accelerometer was orientated along the axis of motion. The accelerometer was sampled at 5000 Hz using a 16-bit card with a range of +/- 10 V.

5.3.4.3 Motion Capture

Motion capture was used to monitor the location of the torso and pelvis. Two rigid bodies were used to track the location of the trunk and pelvis during the pre, post and post-24 and during the simulated collisions. Because of the difference in setup of the pre/post measures and collision simulations, different anatomical landmarks were used for kinematics for each portion of the respective experimental procedure. Motion capture data were continuously sampled at 100 Hz.

5.3.4.3.1 Collision Simulation Motion Capture:

Two rigid bodies were firmly attached to the participant – one on the sternum, and one on the superior surface of the left thigh. In order to define anatomical landmarks, digitized points were created relative to the rigid bodies on each segment. While each participant stood in anatomical posture, a digitizing probe containing four markers and a known location of a point at the end of the probe was placed on the anatomical landmark of interest (Table 5.1) to capture the location with respect to the rigid body of interest. This created a fixed relationship with the rigid body of interest and the digitized anatomical landmark. Because the participant was seated in an automotive seat, it was not possible to place a rigid body directly on the sacrum to track digitized points on the pelvis. To combat this issue, the anatomical landmarks defining the pelvis (Table 5.1) were initially found and marked with permanent marker while the participant stood in anatomical posture. Once the initial digitization process was completed with the participant standing in anatomical posture they were then seated and belted into the automotive seat. After this was completed, the pelvis points were then re-digitized to reflect the movement of the rigid body placed on the left thigh with respect to the pelvis. After the anatomical pelvis points were re-

digitized in the seated-belted position, it was assumed the thigh did not move with respect to the pelvis, resulting in fixed relationship between the rigid body placed on the thigh and the pelvis.

Table 5.1: Location of digitized anatomical landmarks for each respective rigid body.

Rigid Body	Anatomical landmark for coordinate systems
Trunk	Left and Right Acromion Left and Right Iliac Crest
Pelvis	Left and Right Iliac Crest Left and Right Greater Trochanter

5.3.4.3.2 Pre-Post Collision Testing Motion Capture:

Two rigid bodies were attached to the participant and placed over the estimated locations of the 1st lumbar vertebra and sacrum. Each rigid body had a slot cut out of the back which fit directly over each of the two accelerometers adhered to the participant. The accelerometers remained adhered to the participant over the duration of the experimental protocol, however after the pre-passive test the rigid bodies were removed and re-attached after the collision simulation. Placing the rigid body over the accelerometers ensured the rigid bodies were attached at the same location. In the case of the 24 hour testing session, the rigid bodies were traced over with permanent marker to ensure a similar placement the second day of return. Similar to above, anatomical landmarks were defined within the rigid bodies on each segment. While the participant stood in anatomical posture, a digitizing probe containing four markers and a known location of a point at the end of the probe was placed on the anatomical landmark of interest (Table 5.1) to capture the location with respect to the rigid body of interest. Each time the rigid bodies were re-attached to the participant (Pre, Post and Post-24) the anatomical landmarks were re-digitized. Anatomical landmarks were marked with permanent marker to ensure repeatability over Pre, Post and Post-24 testing.

5.3.4.4 Pressure Data

Seat back forces were estimated using a ferroresisitive pressure measurement system (Version 3150, Tekscan Inc., Boston, MA, USA). The pressure mat had sensing dimensions of 36.8 cm by 43.5 cm with a total of 2288 sensing elements and a spatial resolution of 1.4 sensels per cm^2. Prior to data collection, the mat was conditioned to 103.4 kPa five times in 5 second cycles, equilibrated for 30 seconds at three points (13.8 kPa, 27.6 kPa, and 48.3 kPa) then calibrated following the

manufacturer's nonlinear (power) procedure. The pressure mat was rigidly fixed to the automotive seat back with the inferior edge of the sensing area positioned at the interface between the seat back and the seat pan. Pressure measurements were recorded using F-Scan Research 7.01 software (Tekscan Inc., Boston, MA, USA) at a sample rate of 500 Hz and synchronized with kinematic and analog data through a custom external trigger connected to the Optotrak Data Acquisition Unit (Northern Digital Inc., Waterloo, ON). One rigid body with X optical markers was rigidly attached to the sled platform which allowed tracking of the boundaries of the pressure mat at the upper and lower corners of the seat back. These points were digitized in the absence of the participant (Figure 5.3) and were used to define the position and orientation of pressure mat with the assumption that the pressure mat did not move relative to the optical tracking cluster.

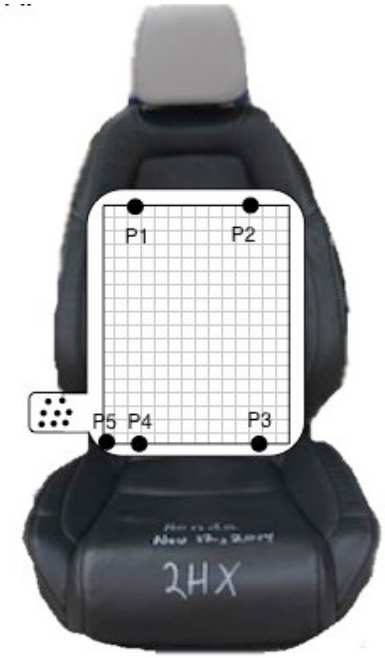

Figure 5.3: Location of the pressure mat with respect to the automotive seat and the 5 digitized points used to track the location of the pressure mat with respect to the seat and occupant.

5.3.4.5 Frictionless Jig

A custom built near frictionless jig (Figure 5.4) was used to measure lumbar spine passive stiffness. This jig allowed for the moment-angle relationship of the lumbar spine to be computed. The legs and pelvis remained in a fixed position and the upper body was free to move through flexion and extension.

The jig was comprised of three main components:

1. Nylon ball bearing (1.2 cm diameter) evenly distributed over a Plexiglas surface (1.22 m x 1.83 m x 2.54 cm).
2. Upper body wooden cradle lined with Plexiglas on the inferior surface, which glides over the ball bearings and a lower body support that restricts motion at the hip and is vertically adjustable.
3. A force transducer placed in series with a cable to pull the participants into flexion and extension, a metal rod fixed to the point of application of the applied force, and a parallel cable to ensure that applied forces are perpendicular to the thoracic harness.

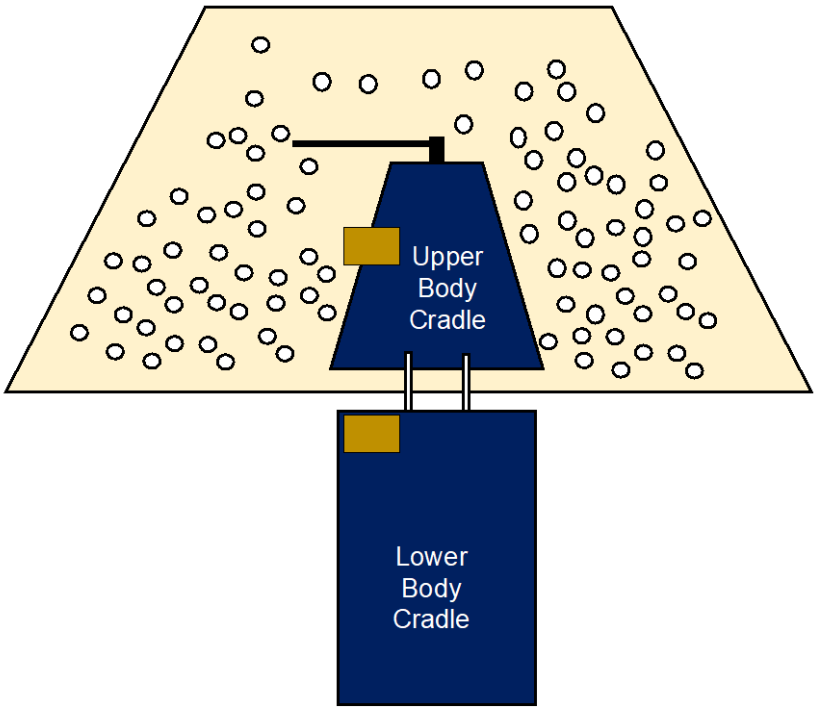

Figure 5.4: A schematic of the frictionless jig used to assess passive stiffness of the lumbar spine. Initially the upper and lower body cradle components were secured together using wooden dowels on either side of the cradle. The upper body cradle was adjusted horizontally for each participant such that the lumbar spine was isolated between the upper and lower body cradle.

5.3.4.6 Collision Simulation Sled

All collision simulation tests were conducted on a custom-built sled-track unit (Figure 5.5). The moveable platform contained a Honda CRV automobile seat reclined to an angle of 27° (Siegmund et al., 1997) (2017 Model: 10 cm bilateral trim; 50 cm seat pan; 30 cm seat back; adjustable head restraint), an adjustable foot platform to facilitate a knee angle of 110°, and the belted occupant.

The platform accelerated down 3.86 m plane that had an inclination angle of 5.8° until it simultaneously struck four custom designed springs (Omnicoil, Ayr, ON) with stiffness and damping parameters of 31991 N/m and 593.78 Ns/m, respectively. The total mass of the platform was standardized to 113.4 kg (250 lbs) by fastening additional mass (i.e., the difference between the seat with participant body mass and 113.4 kg) to the platform base. This ensured a collision-induced velocity change (ΔV) of approximately 8 km/h for participants of varying body mass when released from the same track location. Further details pertaining to the collision simulation sled, accelerometer trace and validation can be found in Appendix C.

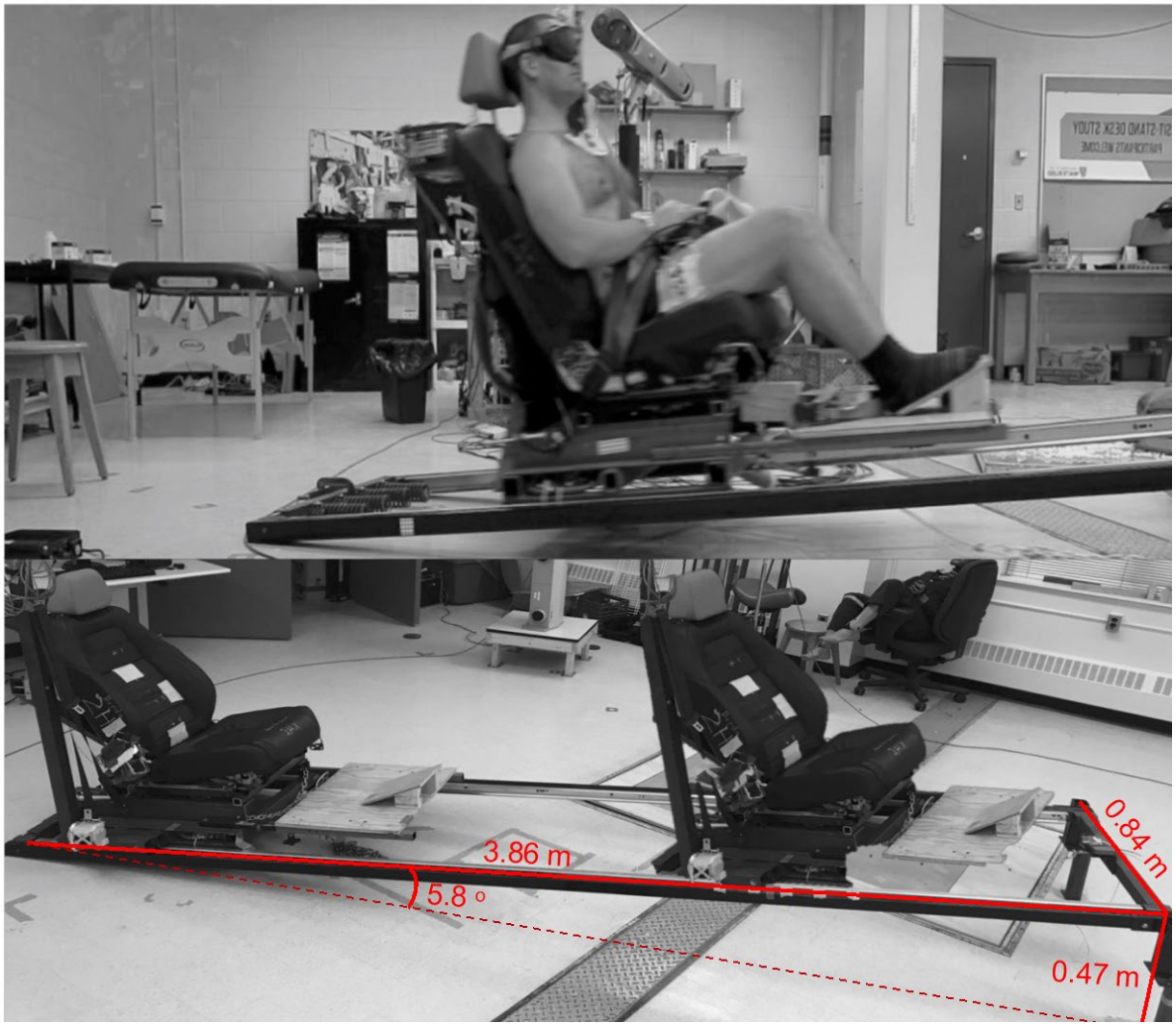

Figure 5.5: The custom-built collision simulation sled. Participants were seated and belted in the automotive seat. The seat assembly was then released from the top and rolled back to hit 4 springs.

5.3.5 Experimental Protocol

The experimental protocol consisted of two separate unanticipated rear-end collision simulations. One collision simulation involved the participant sitting in a normal automobile seat and the second collision simulation involved the participant sitting in the same automobile seat with fully engaged lumbar support. In randomized order, the collisions were conducted with the application of mechanical lumbar support at the third lumbar spinal level and without. The lumbar support depth was mechanically adjusted to a 4 cm horizontal shell deflection, which was consistent across all of the participants.

The following protocol outlined below occurred for each collision condition (without lumbar support and with lumbar support). Each collision was separated by a minimum of 2 weeks to ensure there were no residual effects from the prior collision and sufficient time had passed such that the participant did not recall the previous collision (Brault et al., 1998).

5.3.5.1 Pre Collision Measures

Prior to the simulated rear-end collision baseline measures of Pain, Forward-Flexion, and Passive Flexion-extension were measured.

5.3.5.1.1 Frictionless Jig Protocol

The participant laid with their anterior superior iliac spine aligned with a vertical column on the lower body support and shoulders aligned with the vertical column on the upper body support. Straps secured the ankles, thighs and pelvis to the lower body platform. The torso was strapped to the upper body cradle such that when the platform was moved motion only occurred about the lumbar spine (Figure 5.6).

The passive trial began with the upper body cradle moved into a lumbar spine posture away from the intended movement direction (i.e. for the extension trial the participant began in mild flexion and for the flexion trial the participant began in mild extension). The participant was then pulled by the experimenter into flexion or extension and the trial was stopped when the experimenter felt the participant was at their maximum range of motion requiring an increase in applied force or if the participant indicated they could not move any further. A trial was confirmed to be passive if muscle activity of the lumbar erector spinae muscles remained below 5% MVC.

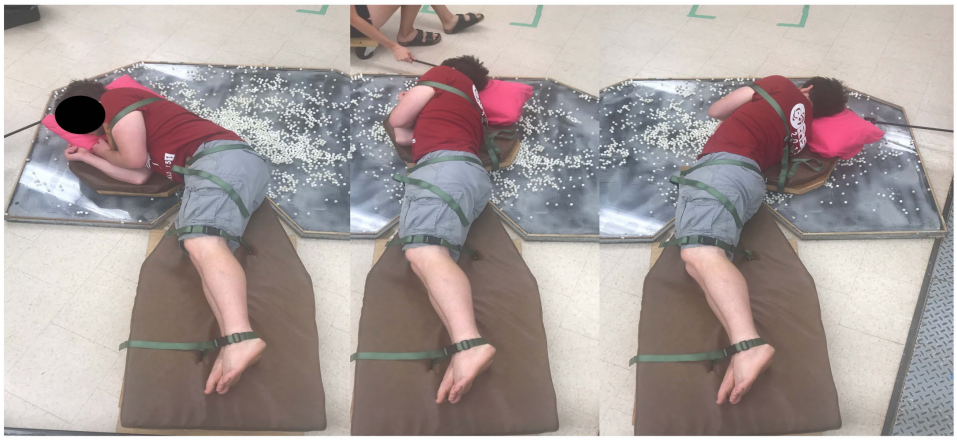

Figure 5.6: The frictionless jig positions. The figure displays the neutral position (center), which was used as the posture to secure the participant into the jig, extension trial (right) and flexion trial (left).

5.3.5.1.2 Forward Flexion

Three repetitions of standing forward flexion were then performed with the collection of continuous EMG. Participants were asked to stand quietly for several seconds to obtain baseline EMG values in upright standing, and then bend forward from the hips into their maximum range of lumbar flexion while maintaining extended knees. They were then asked to hold this position for several seconds, and then return into upright standing.

5.3.5.2 Simulated Low Velocity Rear-End Collision

Following the pre-test measures and instrumentation, participants were exposed to their first collision. Participants were seated in the collision simulation sled and instructed to sit with their backs against the seat back, face forward, feet on the floor, hands in the lap, and to otherwise assume a normal seated position. The D-ring of the seat belt was fully adjustable and was adjusted such that the shoulder belt was centered across the clavicle and the lap belt was secured slightly below the right and left ASIS. In addition, the head- rest was adjusted in accordance to recommendations from the Insurance Bureau of Canada (2002), with the headrest placed level with the top of the head and 5 to 10cm back from the occupant's head. Because of the potential effect of pre-impact muscle contraction on kinematics, special attention was made to replicate an unanticipated impact by eliminating visual and auditory cues of the impending impact. Participants wore ear buds playing music and were blindfolded such that they were not able to see the experimenter releasing the sled. A standard 3-point seat belt was worn.

5.3.5.3 Post-Test Examination

Immediately following the simulated collision participants went through a post-test series of tests. The post-test was identical to all of the measures taken in the pre-test (Pain Reporting, Passive Flexion-extension and Forward Flexion).

5.3.5.4 24-Hour Post Test Examination

Participant's then returned for a third series of measures approximately 24 hours post the initial impact. Participants were re-instrumented with electromyography and kinematics identical to that of testing the previous day. In addition, all MVC trials were repeated. Both the Passive Flexion-extension and Forward Flexion tests were repeated.

5.3.6 Data Analysis

5.3.6.1 Pain Development

In total, 7 VAS scores were reported for each participant, for each collision. To assess the relative increase in VAS score attributed to each collision, VAS scores taken right before the simulated collision were subtracted from all VAS scores that followed. As a result, participants started with a relative VAS score of 0 mm and any increases in pain were attributed to the experimental protocol.

5.3.6.2 EMG

Processing of EMG signals began with removing the DC bias from the EMG channels. The signals were then band pass filtered from 10 – 500 Hz and band pass filtered from 30 – 500 Hz (Drake and Callaghan, 2006) to remove any ECG contamination. Signals were then full wave rectified and then low pass filtered (Butterworth, 2^{nd} order, single pass) with an effective cut off of 2.5 Hz (Brereton and McGill, 1998). The resulting linear enveloped signals were then normalized to MVC trials, resulting in normalized EMG signals expressed as a percentage of the maximum voluntary contraction (%MVC).

5.3.6.3 Motion Capture & Force

All kinematic and kinetic signals were padded with one second of data (Howarth, Beach, Pearson & Callaghan, 2009) using an end-point reflection method (Smith, 1989) and smoothed using a second-order, lowpass (dual-pass) digital Butterworth filter with an effective cutoff frequency of 6 Hz. Coordinate systems were constructed using the landmarks for each segment with +y pointing proximally, +x pointing anteriorly, and +z pointing laterally to the right of the participant. Lumbar angular displacements were represented as the orientation of the distal segment (the pelvis) with respect to the adjacent segment (the thorax). The rotation matrix describing the 3D orientation

between the two segments was calculated using the joint coordinate system with a flexion/extension-lateral bend-axial twist rotation sequence.

5.3.6.4 Accelerometers

Crash sled acceleration data were filtered using SAE Channel Frequency Class (CFC) 60 (Society of Automotive Engineers, 1995). Sled accelerometer data was zeroed based on the average measurement recorded just prior to the release of the sled. The sled accelerometer was used to define the point of impact (peak acceleration) and was integrated (trapezoidal method) to calculate the resultant change in velocity (ΔV) of the sled assembly.

5.3.6.5 Pressure Mat

Raw position and pressure data were imported into a custom Matlab® program (Math Works, Natick, MA, USA) for further processing. The total pressure was computed by summing the calibrated output from each sensel (Equation 4.1). The total pressure quantity was converted from kPa to Pa and then multiplied by the sensel area ($7.02 \times 10^{-5}\,m^2$) to determine the seat back reaction force recorded from the entire sensing surface. The peak of the total force-time history was then used to identify when the moment of peak seatback contact (Figure 5.7). At the identified frame, the region of force concentration was manually identified using a masking technique, to determine the area and centre-of-force (CoF) (Kingston and Acker, 2018). This mask was then applied to all frames collected (Figure 5.9). The employed masking approach was used to eliminate noise due to the mat crinkling and/or bending around the seat bolsters and permitted an analysis of regions where seat reaction forces were concentrated (Kingston and Acker, 2018). The total force and the CoF of the masked polygon were determined with respect to the labelled corner 4 (C4) of the pressure mat sensing area (Figure 5.8).

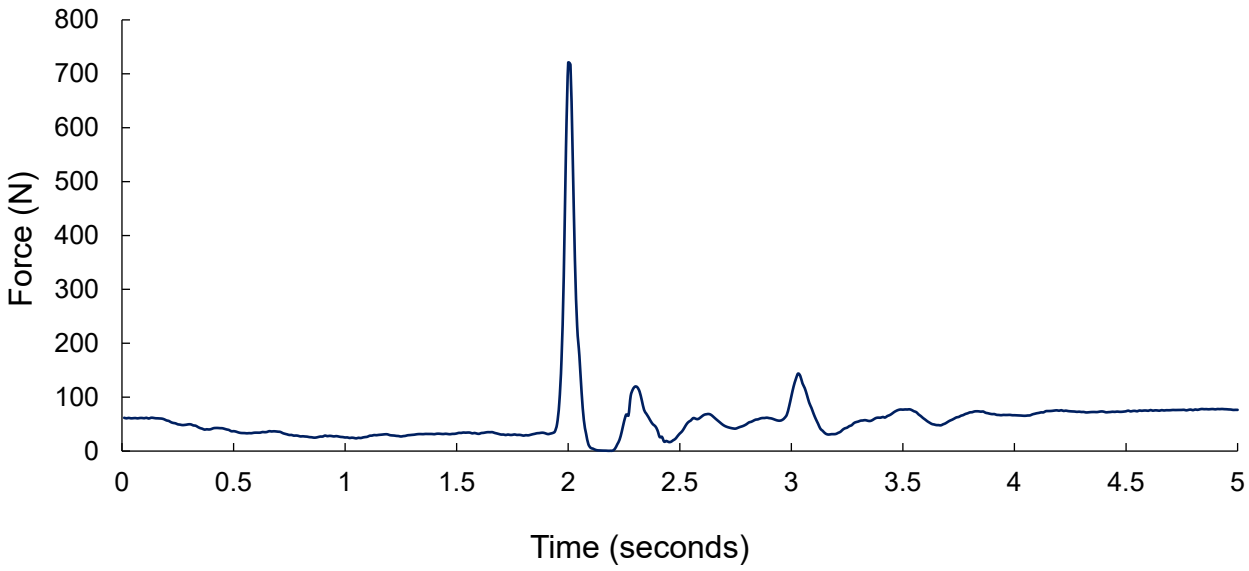

Figure 5.7: Sample calibrated force trace from the pressure mat.

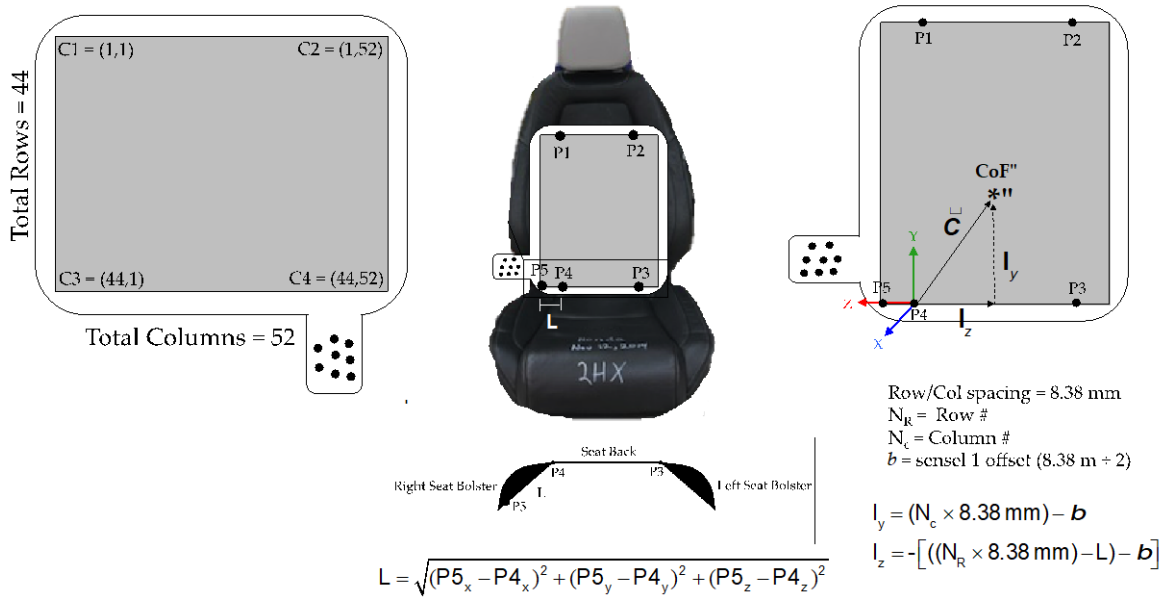

Figure 5.8: A schematic of the pressure mat setup. Left - the sensel number (X, Y) within the mat coordinate system are with respect to corner 1 (C1). Centre – the position of the pressure mat on the seatback and quantification of the row offset (L) and digitized points. Right - quantification of center-of-force coordinates with respect to the pressure mat local coordinate system.

A mat local coordinate system (LCS) was constructed using the digitized seat endpoints and was originated at digitized point 4 (P4). The CoF location with respect to the mat LCS origin

was determined by subtracting the distance between P4 and P5 (Figure 5.8) from the horizontal component and then transformed to the global (laboratory) coordinate system by multiplying the mat direction cosine matrix by the CoF coordinates expressed in the LCS. The global coordinates of the LCS origin were accounted for by addition of the derived CoF coordinates to represent the vector in the global coordinate system.

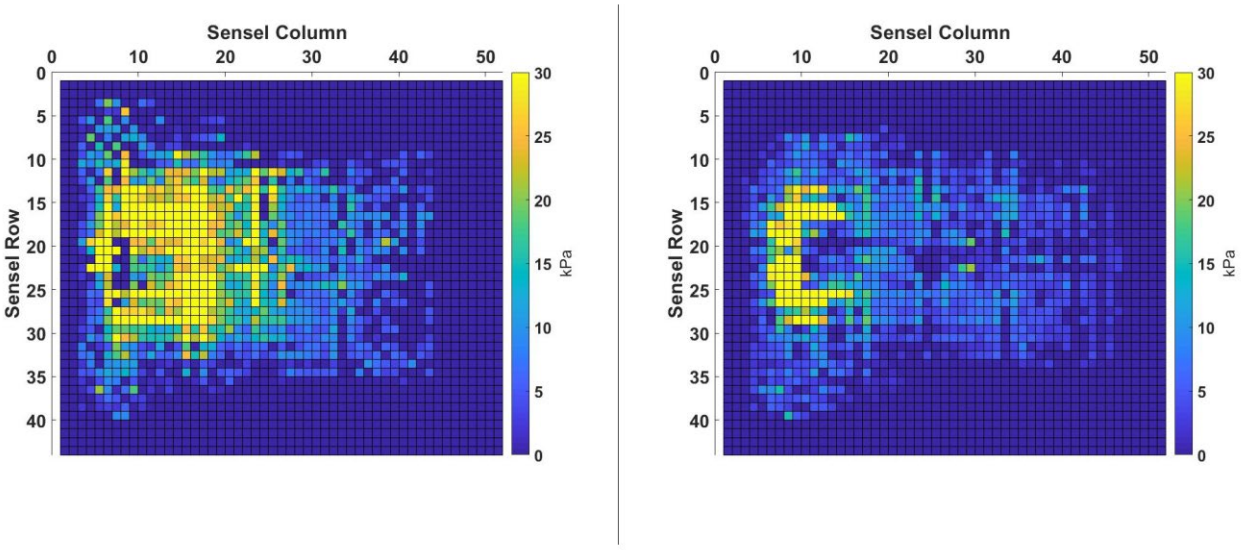

Figure 5.9: Processed pressure data at the point of peak pressure for one participant during Supported (left) and Unsupported (right) impacts.

5.4 Outcome Measures

5.4.1 Simulated Rear-End Collision

As previously described, all kinematic and kinetic signals were padded with one second of data (Howarth and Callaghan, 2009) using an end-point reflection method (Smith, 1989) and smoothed using a second-order, low-pass (dual-pass) digital Butterworth filter with an effective-cutoff frequency of 6 Hz (Bisseling and Hof, 2006). This cutoff frequency was chosen based on pilot work, which compared peak accelerations of the trunk computed by numerical differentiation of position data and accelerometer data. Due to noise in the kinematic data and the need to compute segmental accelerations by numerical differentiation of position data a cutoff filter was chosen such that the peak accelerations measured by the accelerometer closely matched that of the differentiated of position data. Next, for each simulated collision (with lumbar support and without lumbar support), the kinematic data and kinetic data (seatback forces obtained from the pressure

data) were combined in a 3D biomechanical model to estimate instantaneous reaction forces (about the origin of the pelvis coordinate system) (Beach et al., 2014; Gooyers et al., 2018). This was calculated based on a top down inverse dynamics approach using Visual3D^{TM} software (Version 5.0, C-Motion, Inc., Germantown MD, USA). The origin of the pelvis-segment coordinate system was calculated as the midpoint between iliac crests and a least-squares plane fit to the locations of the iliac crests and greater trochanter represented the frontal plane of the pelvis segment (Beach et al., 2014; Gooyers et al., 2018). The component of the reaction force acting normal to the plane was assumed to be equivalent to the anterior/posterior shear force acting at L4/L5 (Beach et al., 2014; Gooyers et al., 2018). Default parameters within the Visual3D^{TM} software for segmental mass and inertial properties (scaled for individual participants based using height and mass) were used in the inverse dynamics calculations. The decision to negate muscle activation was based on **Study 2**, which showed that trunk muscle activation levels are extremely low in an unanticipated 4 km/hour collision.

5.4.1.1 Simulated Rear-End Collision - Data Reduction

Five dependent measures were computed from the simulated rear-end collision data, including: (i) seat back peak total force at impact (ii) peak L4/L5 compression; (iii) peak L4/L5 shear; (iv) lumbar angle at the time of peak shear loading (i.e. seat back contact); (v) peak lumbar spine flexion angle. In addition to the above outcome measures, peak sled acceleration and ΔV were computed across all collision simulations to ensure the simulated collision exposures were the same across and within participants.

5.4.2 Pre, Post & Post-24 Hour Testing

5.4.2.1 Passive Trials

For all passive measures the first passive trial deemed acceptable in flexion and extension was used to characterize passive stiffness. Muscle activation was tracked in real-time, a trial was deemed acceptable when muscle activation was below 5% MVC for the duration of the trial. A moment-angle relationship was created by multiplying the force from the force transducer by the moment arm measured by placing a marker at the point of force application and the L4/L5 joint. Changes in passive stiffness were then quantified using a newly developed Breakpoint Method (Barrett et al., 2020), which employed a tri-linear fit separating the moment-angle curve in to three linear regions. The moment-angle relationship in flexion and extension was obtained by plotting the moment against the measured lumbar angle. Similar to previous literature (Beach et al. 2005;

De Carvalho and Callaghan 2011), each moment-angle curve was partitioned into three zones (low, transition and high), by locating the points at which the greatest percentage of change in stiffness were evident (Figure 5.10). Thus, for both Flexion and Extension curves two breakpoints of lumbar spine angle were determined separating the curve into the three zones (low, transition and high stiffness). For across participant comparison, all lumbar spine angles were normalized to initial pre-collision maximum lumbar spine flexion and extension for the passive flexion and extension curves respectively. From this, the slopes in each zone were used as a measure of passive stiffness (low, transition and high). The angle of low and high moment-angle breakpoint (%maximum Flexion and Extension) were also documented along with the range of flexion angles between these breakpoints (the transition zone range) (Figure 5.10). In addition, the maximum lumbar flexion and extension angle obtained for each trial was also determined. A list of all outcome measures extracted from the passive testing is listed in Table 5.2.

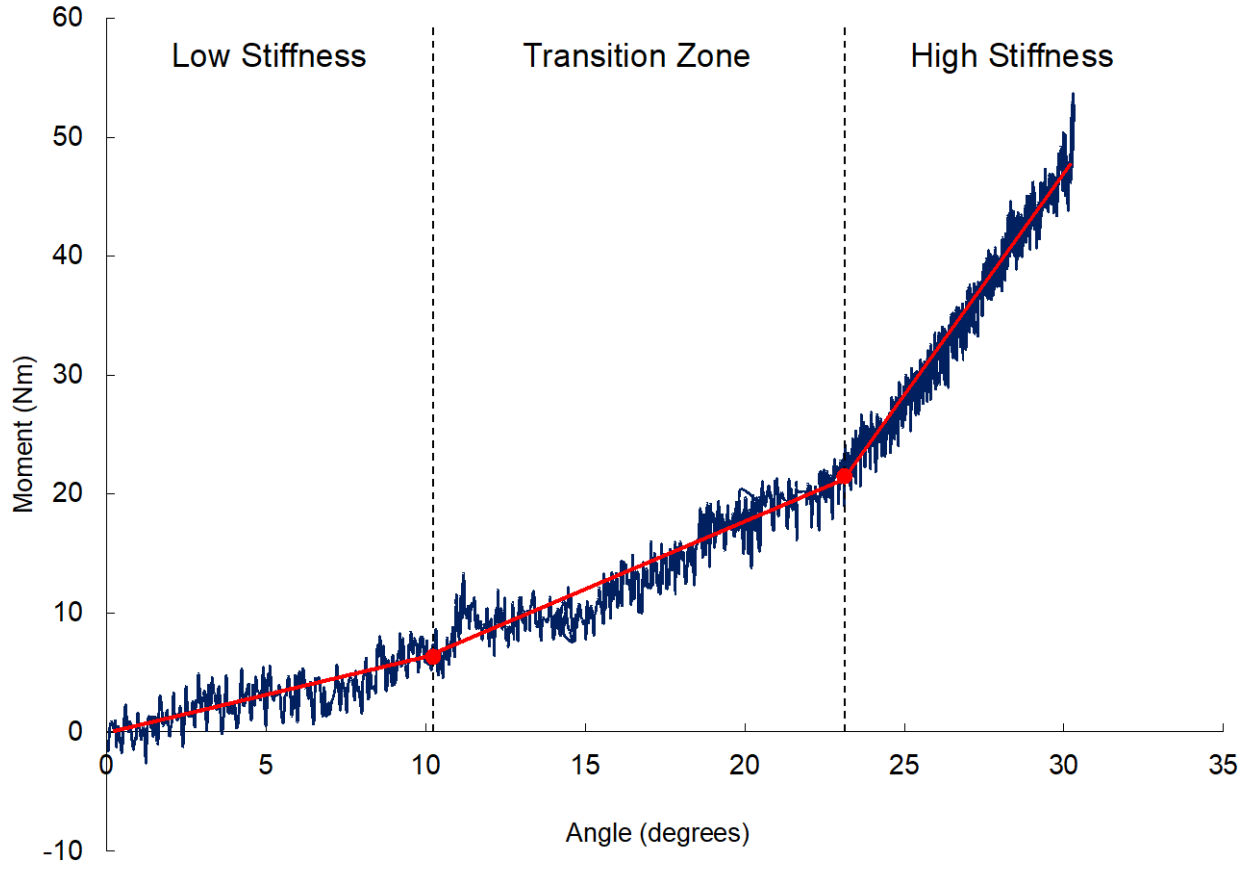

Figure 5.10: A sample figure displaying one of the collected passive flexion curves and the use of the tri-linear fit. The method finds the best fitting piecewise linear fit to the curve and separated

the curve into 3 regions. Here the 3 zones are displayed: the low, transition, and high stiffness zones. The solid lines represent slopes for each region superimposed over the raw data.

Table 5.2: A list of the outcome measures obtained from the passive jig for both flexion and extension curves.

Outcome Measure	Definition
Slope	The slopes of linear trend-lines that were independently fit to the original moment-angle data in each of the low, transition and high stiffness zones to provide an estimate of stiffness in each of the three zones.
Moment Angle Curve Breakpoint	The points at which the greatest percentage of change in the slope was evident (low and high breakpoints)
Maximum Lumbar Flexion and Extension	The maximum voluntary lumbar flexion/extension angle to which participants were pulled on the frictionless jig.
Transition Zone Range	The range in angle from the low breakpoint to the high breakpoint, covering the transition zone stiffness range.

5.4.2.2 Repeated Flexion Trials

For each period of testing (Pre, Post, Post-24), normalized linear enveloped EMG from the three forward bending flexion trials for the lumbar erector spinae muscle was used to determine a flexion-relaxation ratio using Equation 4 (Dankaerts et al., 2004; Nelson-Wong, 2009).

$$FRR = \frac{average\left(EMG_{upright}\right)}{average\left(EMG_{flexed}\right)} \qquad \textbf{Equation 5.1}$$

This ratio was computed for both the right and left TES and LES and averaged across each of the 3 repeated flexion trials. Right and left muscles were then averaged for a total of 6 FRR measures for each participant (Not Support/Support; Pre, Post, Post-24). Similar to the passive measures, the Pre measures were subtracted from the Post and Post-24 as a baseline measure. Or to yield a relative change attributable to the impact events.

5.4.3 Statistical Analysis

All statistical analyses were computed using SPSS statistical software (SPSS v20, IBM Corporation, Somers, NY, USA) with an alpha criterion of 0.05. A paired t-test was completed to

compare peak acceleration and ΔV across collision simulations. A 2-way mixed general linear model was performed to determine the effects of the use of lumbar support and gender. In addition, a three-way mixed general linear model was employed to determine the effects of Time (pre, post and post-24), Lumbar Support (Support and No Support) and Gender on the pre-post testing outcome measures. When significant effects of Gender were not observed data was then collapsed across Gender. Tukey's *post hoc* test was used to further explore all significant main and interaction effects.

5.5 Results

Of the 24 participants, three (1 male and 2 female) were excluded from the collision statistical analysis due to marker occlusion and/or failure of the pressure mat to trigger during collision testing. Additional trials could not be performed on these participants due to concern for wellbeing and to remain within the approved ethics protocol that only permitted two separate impacts per participant.

5.5.1 Simulated Rear-End Collision

5.5.1.1 Collision Characteristics

Mean differences in ΔV (p = 0.060) and peak acceleration (p = 0.826) were not statistically significant across test days or individuals. The average ΔV and peak acceleration were 7.66 km/h (± 0.30) and 4.75 g (± 0.29), respectively. This confirms that impact parameters were similar for all simulated rear impact collisions.

5.5.1.2 Total Seatback Force

There were no significant main effects (p = 0.197) or interaction effects (p = 0.445) found for total seatback force magnitude. The mean seat back force was 820.5 N and 818.7 N during collisions with and without lumbar support, respectively.

5.5.1.3 Peak Reaction Compression Force

There were no significant main effects (p = 0.08) or interaction effects (p = 0.726) found for peak compressive reaction force. Peak compression reaction force was slightly greater without lumbar support (498.22 N (±178.0) in comparison to with lumbar support (484.5 N (±151.1)).

5.5.1.4 Peak Reaction Shear Force

There were no significant main effects ($p = 0.292$) or interaction effects ($p = 0.326$) found for peak shear reaction force. While not statistically significant, peak shear force was slightly greater without lumbar support (302.2 N (±98.5) in comparison to with lumbar support (291.3 N (±76.8).

5.5.1.5 Lumbar Flexion at the Time of Peak Shear Loading

There were no significant main effects ($p = 0.365$) or interaction effects ($p = 0.337$) found for lumbar flexion angle at the point of peak shear reaction force (Figure 5.11). Lumbar flexion angle at the point of peak shear was 36.3 degrees (±11.8) and 33.4 (±11.1) without and with lumbar support, respectively.

5.5.1.6 Peak Lumbar Flexion Angle

There were no significant main effects ($p = 0.365$) or interaction effects ($p = 0.337$) found for peak lumbar flexion angle (Figure 5.11). Peak lumbar flexion was 45.2 degrees (±13.1) and 44.8 (±11.7) without and with lumbar support, respectively.

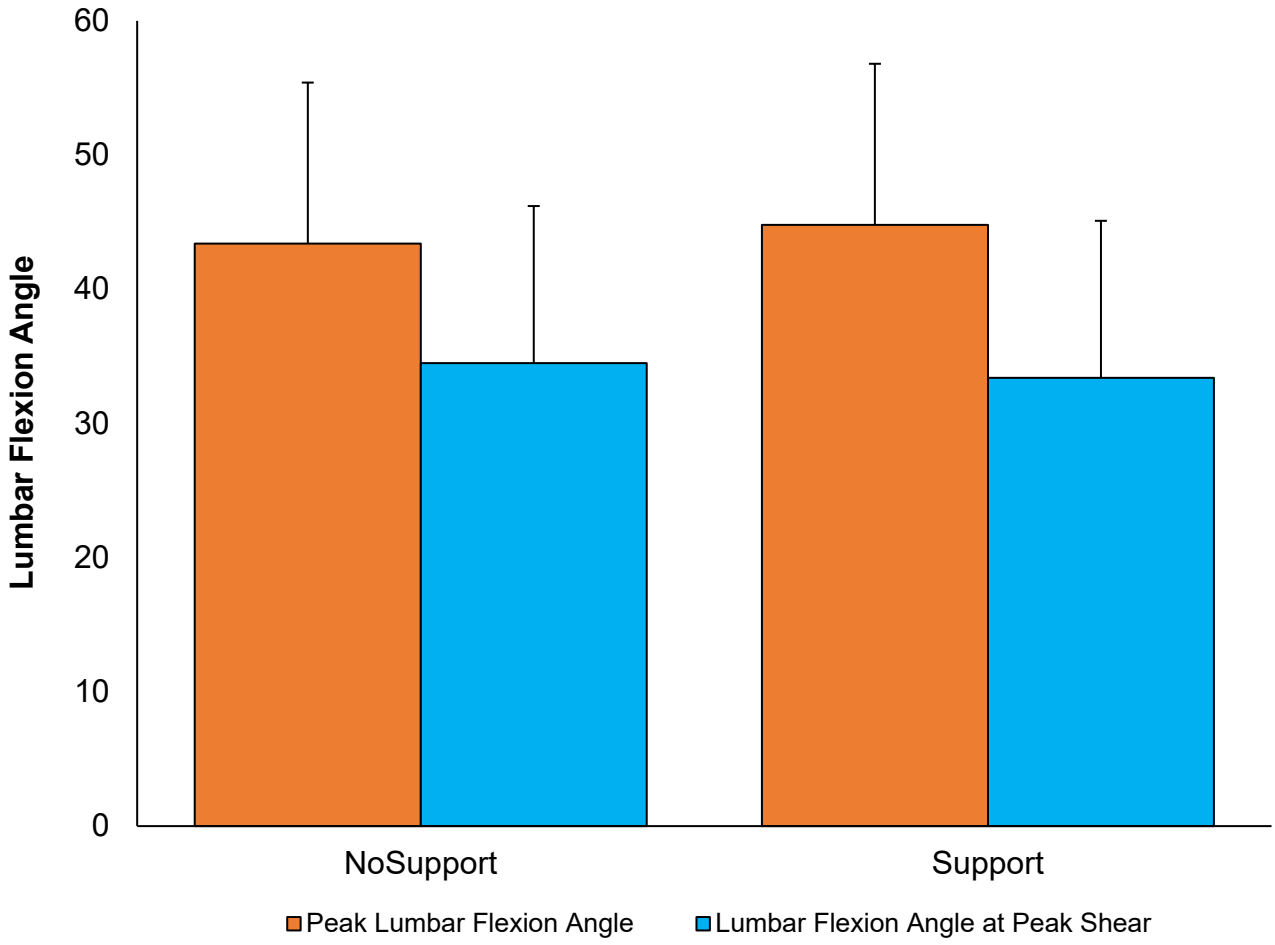

Figure 5.11: Summary of peak lumbar flexion angle for both No Support and Support collision simulations as well as lumbar spine flexion angle at the point of peak shear.

5.5.2 Pre, Post & Post-24 Hour Testing

5.5.2.1 Pain Scores

Overall, reported discomfort levels were extremely low. No differences were observed in discomfort reporting across gender or collision conditions (No Support vs. Support) (Figure 5.12). While extremely low, the highest average peak reported discomfort occurred during the home reporting portion of the protocol and returned to near baseline by the post collision 24 hour point.

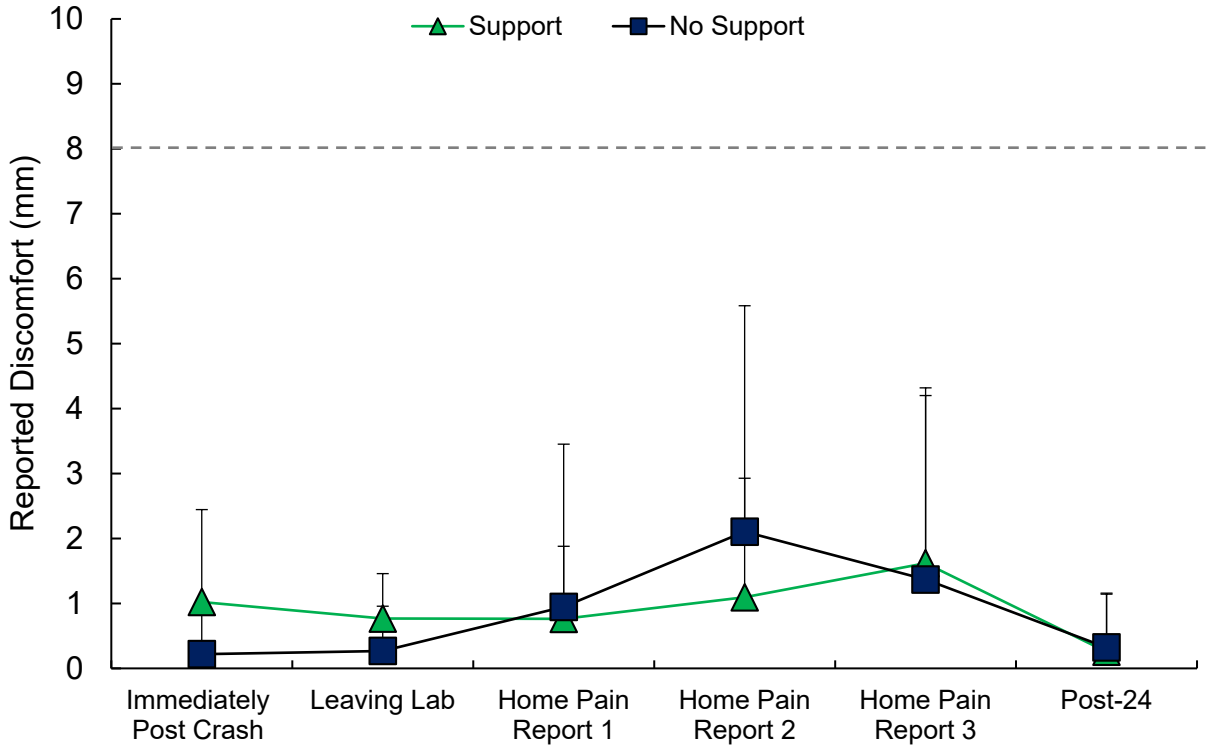

Figure 5.12: Peak reported lumbar spine discomfort throughout the experimental protocol (baseline removed) for both No Support and Support collision conditions. The dashed line represents the clinical important difference for patients to feel their LBP symptoms worsening (Hägg et al., 2003).

5.5.2.2 Passive Trials: Flexion

5.5.2.2.1 Low, High and Transition Slopes

There was a Gender x Support interaction effect (p = 0.016) for the slope of the low stiffness zone. This was as a result of female participants having a significantly greater change in low stiffness slope Post collision for the No Support collision, in comparison to all other measures. Specifically, females had a significant decrease in stiffness in comparison to males. No Significant differences in slope for the transition (Condition p = 0.210; Time p = 0.341 and high stiffness zones were observed (Condition p = 0.773; Time; p = 0.210) (Figure 5.13, Table 5.3).

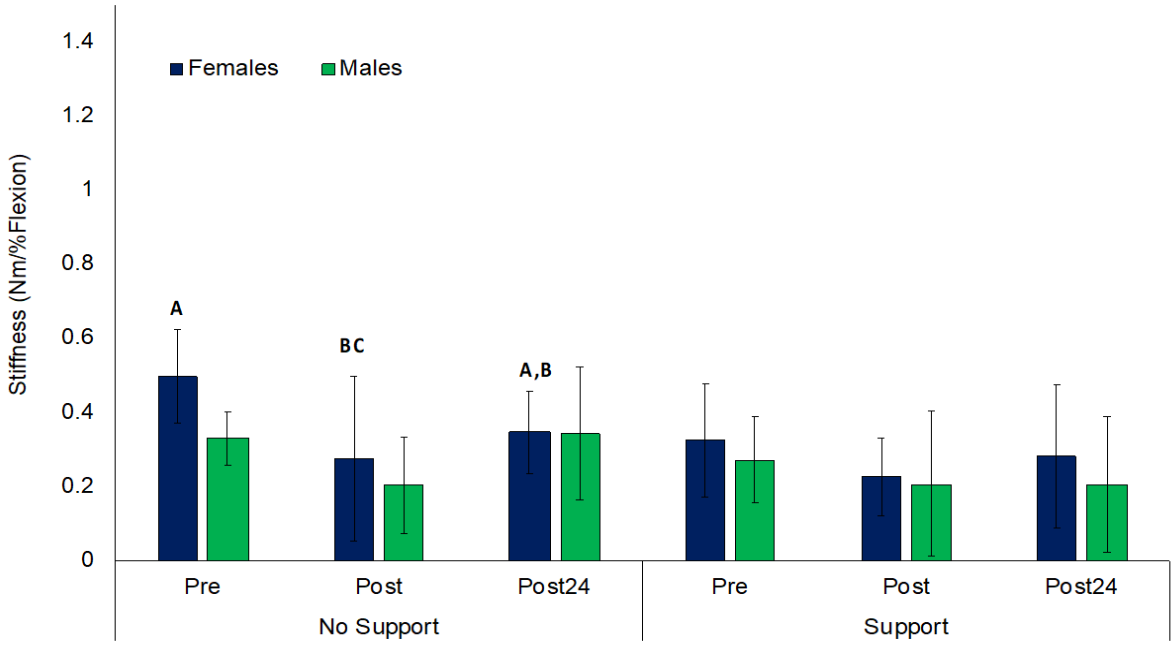

Figure 5.13: Changes in low stiffness zone slope across Supported and Un-Supported simulated collisions. Standard deviation bars are displayed. Statistically significant differences are displayed.

Table 5.3: Comparisons of mean (SD) low, transition and high slope zones for male and female subjects.

		No Support			Support		
		Pre	Post	Post24	Pre	Post	Post24
Low Stiffness Nm/^{o} (SD)	Females	0.50 (0.13)	0.28 (0.22)	0.35 (0.11)	0.32 (0.15)	0.23 (0.10)	0.28 (0.19)
	Males	0.33 (0.10)	0.20 (0.18)	0.34 (0.18)	0.27 (0.12)	0.21 (0.20)	0.21 (0.18)
Transition Zone Nm/^{o} (SD)	Females	0.38 (0.31)	0.51 (0.26)	0.38 (0.21)	0.42 (0.26)	0.33 (0.22)	0.45 (0.54)
	Males	0.44 (0.23)	0.45 (0.28)	0.42 (0.23)	0.41 (0.24)	0.35 (0.34)	0.51 (0.31)
High Stiffness Nm/^{o} (SD)	Females	1.56 (0.33)	1.42 (0.39)	1.65 (0.29)	1.04 (0.13)	0.91 (0.26)	1.32 (0.20)
	Males	1.42 (0.17)	1.03 (0.27)	0.89 (0.19)	1.09 (0.11)	1.00 (0.27)	1.23 (0.21)

5.5.2.2.2 Moment Angle Curve Breakpoints

A significant effect of Time was observed for the low stiffness breakpoint (p = 0.049)(Figure 5.14). This was a result of significantly different Post and Post24 breakpoint differences in both the No Support and Support conditions. Specifically, the low stiffness breakpoint occurred at a greater angle in the Post trial and the Post24 trial, in comparison to Pre. No significant differences were observed for the high stiffness breakpoint across Condition (p = 0.697) or Time (p = 0.162).

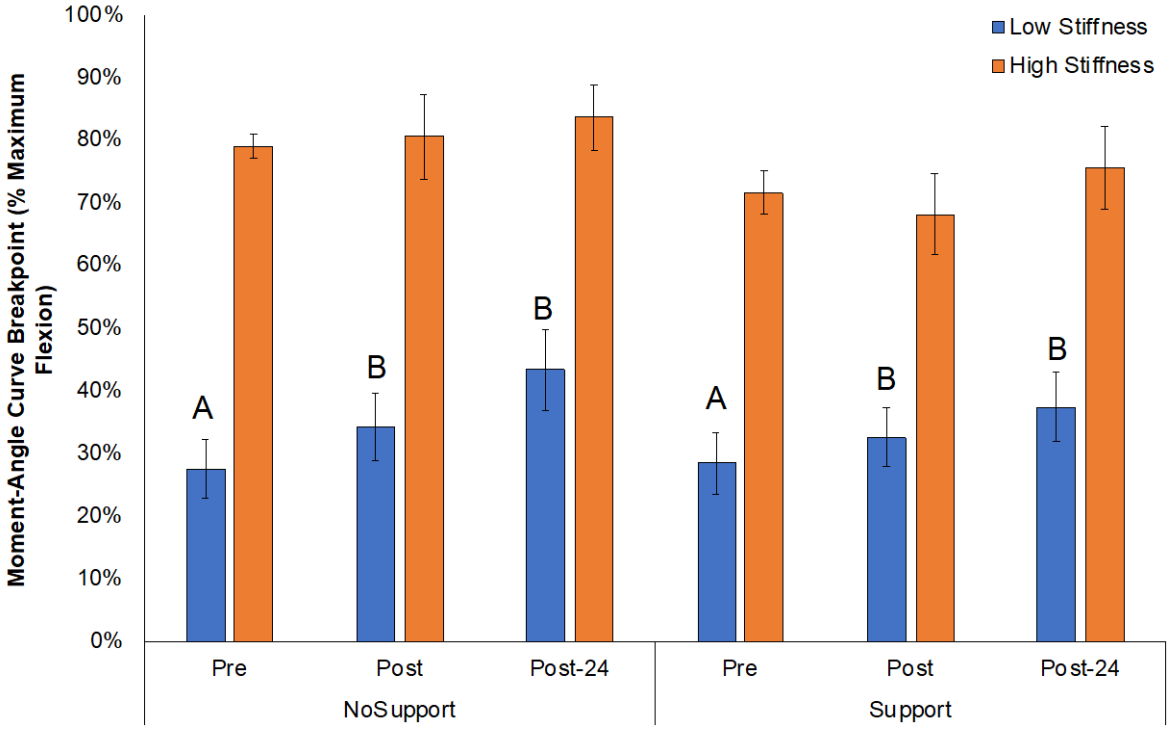

Figure 5.14: Average low and high breakpoints over time across support types. Standard Deviation bars are displayed. No significant differences were found for the high stiffness breakpoint. Statistically significant differences are displayed for the low stiffness breakpoints.

5.5.2.2.3 Maximum Passive Lumbar Flexion

No significant differences were observed for Support Type ($p = 0.315$) or Time ($p = 0.068$) for Maximum Lumbar Flexion angle.

5.5.2.2.4 Transition Zone Range

No significant differences were observed for transition zone range across Condition ($p = 0.768$) or Time ($p = 0.498$).

5.5.2.3 Passive Trials: Extension

5.5.2.3.1 Low, High and Transition Slopes

No Significant differences in stiffness were observed. Slopes for the low stiffness (Condition $p = 0.245$; Time; $p = 0.143$), transition zone (Condition $p = 0.333$; Time; $p = 0.387$) and high stiffness zone were not statistically different (Condition $p = 0.888$; Time; $p = 0.457$) (Figure 5.15).

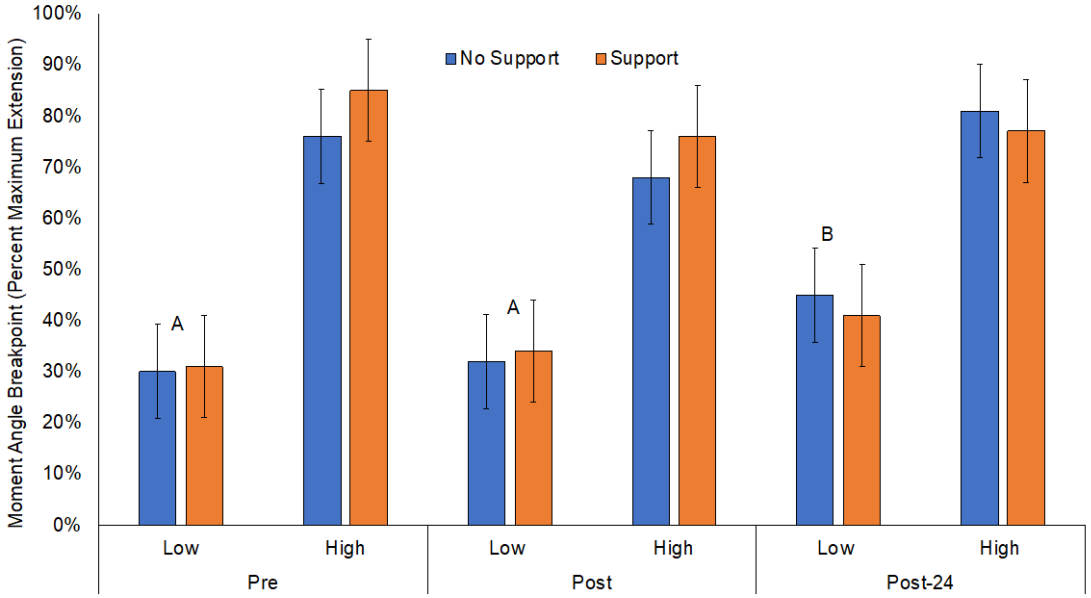

Figure 5.15: Extension low, transition and high stiffness zone slope across Supported and Un-Supported simulated collisions. Standard deviation bars are displayed. No significant differences were observed.

5.5.2.3.2 Moment Angle Curve Breakpoints

A significant effect of Time was observed for the low stiffness breakpoint (p = 0.035). This was a result of significantly different Post24 breakpoint differences in both the No Support and Support conditions (Figure 5.16). Specifically, the low stiffness breakpoint occurred at a greater angle in the Post24 trial, in comparison to Pre and Post. No significant differences were observed for the high stiffness breakpoint across Condition (p = 0.728) or Time (p = 0.144).

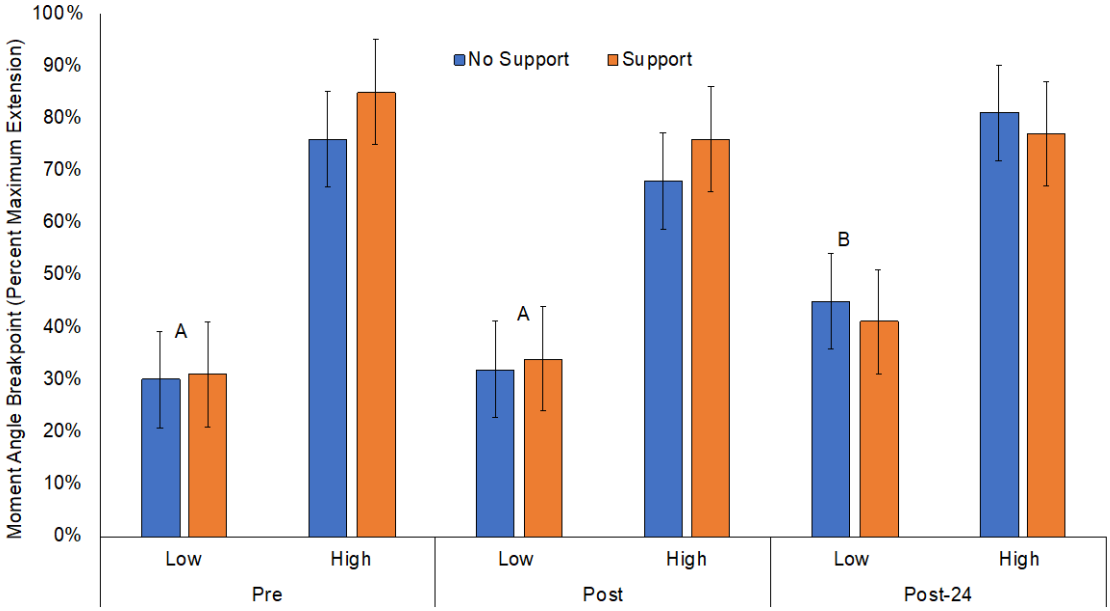

Figure 5.16: Average low and high breakpoints over time across support types. Standard Deviation bars are displayed. No significant differences were found for the high stiffness breakpoint. Statistically significant differences are displayed for the low stiffness breakpoints.

5.5.2.3 Repeated Flexion Trials

No significant differences were observed for FRR for the TES (Support Type $p = 0.587$; Time = $p = 0.101$) or LES (Support Type $p = 0.685$; Time = $p = 0.232$). Average values for FRR for the TES and LES, Pre, Post and Post-24 are displayed in Figure 5.17.

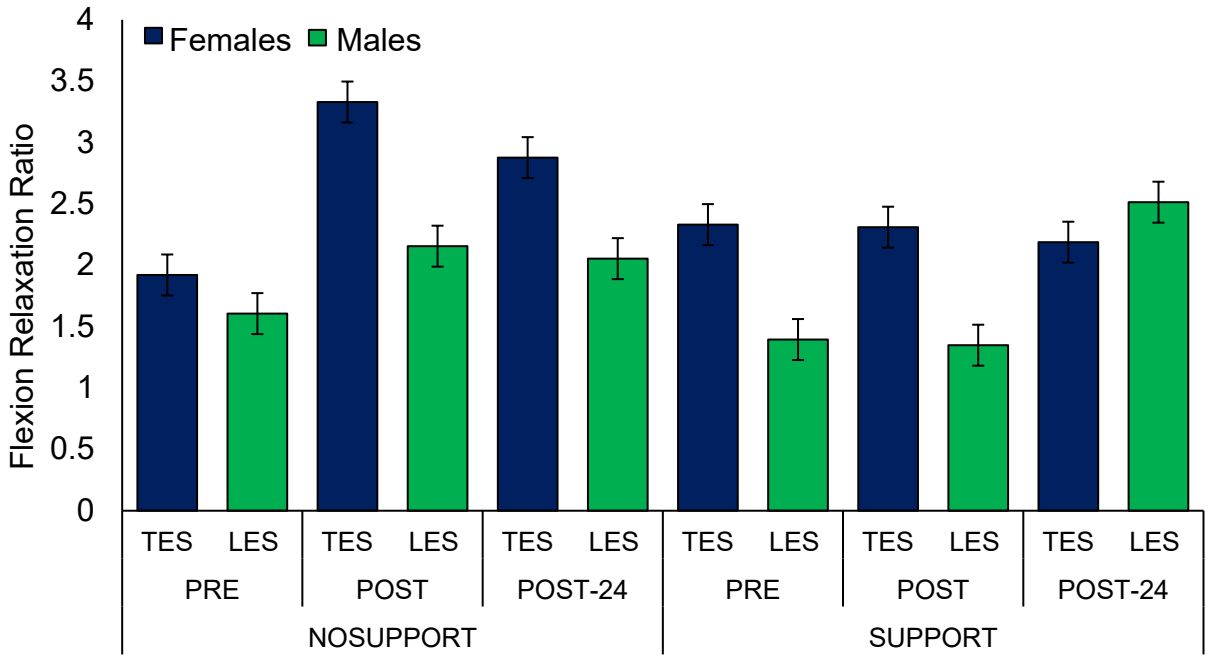

Figure 5.17: Average FRR values for the TES and LES for Pre, Post and Post-24 hour testing for No Support and Support crashes.

5.6 Discussion

The purpose of this study was to access peak lumbar spine loads and kinematics during simulated low velocity rear-end collisions with and without lumbar spine support. Contrary to our hypothesis, lumbar spine loads and kinematics were not significantly different across collision support types. Significant changes in the low stiffness zone of lumped passive lumbar spine stiffness were observed in both flexion and extension post impact. Specifically, we observed a decrease in stiffness in the low stiffness zone, in female participants, immediately following the un-supported collision simulation. Across all impacts and gender, participants exhibited an increase in low stiffness zone range, in flexion, immediately following and 24 hours post impact. In addition, all participants exhibited a significant increase in low stiffness zone range in extension 24 hours post impact. Changes in low stiffness zone range were not accompanied by low back pain symptom reporting post collision or changes in muscle activation strategies.

To date, laboratory simulations focusing on the lumbar spine have mainly focused on the potential for low velocity type rear impacts to result in IVD herniation type injuries (Gates et al., 2010; Gushue et al., 2001). An IVD herniation injury is typically a fatigue type injury (Adams and Hutton, 1983) or caused by large compressive forces coupled with extension (Adams et al., 1988).

In terms of the compression force estimates observed in the current study, results from this investigation agree with previous cadaver and ATD testing results. Fast and colleagues (2002) recorded vertical seat forces of 500 N, for a 13 km/hour severity collision, which was similar to the lumbar compression forces measured in ATDs exposed to rear-end impacts. Gates et al. (2010) examined the response of instrumented BioRID and Hybrid III ATDs exposed to rear-end collision severities at 8 km/hour, while Gushue et al. (2001) examined peak compression forces of the Hybrid III ATD exposed to rear-end impact severities at 8 km/h, with the ATD seated both "in position," as well as up to "20 inches out-of-position" (i.e., centered in the seat but leaning forward). Across all investigations, peak lumbar compression force estimates ranged between 56 N (BioRID) and 350 N (Hybrid III) for "in position" impacts. The estimates in the current investigation align with previous investigations, with estimates of 498 N and 484 N for unsupported and supported impacts. All lumbar spine compression estimates to date have been well below the National Institute of Occupational Safety and Health (NIOSH) recommendations, which recommends a compressive force threshold of 3400 N during occupational lifting.

Previous investigations on the tolerance of healthy lumbar vertebrae to acute anterior shear exposures have revealed an ultimate shear limit of approximately 1000 N during occupational exposures (representing approximately 33% of the shear failure tolerance observed in cadaveric specimens) (Adams et al., 1994; Gallagher and Marras, 2012; Norman et al., 1998). Based on this threshold, our results indicate a margin of safety of 698 N and 709 N for the lumbar spine, for shear loading, during the simulated low-velocity rear impact collisions with and without lumbar support, respectively. While the current investigation employed a simplified joint model to estimate bone-on-bone shear loading, shear loading is not expected to be markedly different even with a more superior biofidelic model. Typically, the internal forces generated by the musculature of the lumbar region create most of the mechanical load placed on the spine. However, unlike standardized occupational tasks, muscle activation does not significantly increase during unanticipated low-velocity collision testing with a collision-induced velocity change of 4 km/h (**Study 2**). Thus, the measured reaction forces in this investigation likely provide a reasonable estimate of the internal joint loads and support the high frequency of negative radiographic findings for bone failure following low-velocity rear impact collisions, leaving potential pain generation sources to soft tissues of the lumbar spine (e.g., ligaments, joint capsules, intervertebral disc).

Time-varying changes in the lumbar spine low stiffness zone were observed following simulated rear-end collisions. Decreasing spinal stiffness has been previously reported due to repetitive lifting, repetitive bending and prolonged driving (De Carvalho and Callaghan, 2011; Dolan and Adams, 1998; Parkinson et al., 2004). The observed changes in the low stiffness zone are likely due to creep in the passive soft tissues of the lumbar spine, such as the intervertebral discs and ligaments (Dolan and Adams, 1998). Results indicate that after a sudden impact, changes in passive stiffness within the lumbar spine may result in increases in intervertebral joint laxity. This increased laxity could subject ligaments and intervertebral discs to exceed their initial range of motion, altering joint mechanics and loading patterns potentially leading to an increased risk of developing low back pain. Interestingly, this investigation did not find any significant differences in transition and high stiffness zones. Increased stiffness in the moderate to high ranges of lumbar flexion, which would be indicated by increased transition or high stiffness slopes, could be due to time-varying changes in the passive elastic properties of muscles. Using equations provided by Adams and Dolan (1991), McGill and colleagues (1994) concluded that muscles were the primary flexion-resisting tissues in the moderate ranges of lumbar flexion (Adams and Dolan, 1991; McGill et al., 1994). Thus, the lack of change in transition and high stiffness zones following collision simulations indicate that changes to the passive properties of muscle are unlikely and suggest such changes may be attributable to stiffness changes in the passive soft tissues.

In a standard automotive seat, without lumbar support, females have been shown to sit with the same pelvic posture but with greater lumbar flexion in comparison to males. De Carvalho and Callaghan (2011) observed a trend of decreasing passive flexion stiffness and a slight right shift in breakpoints, in females, throughout the course of a prolonged driving protocol. De Carvalho and Callaghan (2011) hypothesized that postural differences prompt the suggestion that there may be a gender specific response to passive tissue strain caused by the greater degree of lumbar flexion present in automotive seating for female participants (De Carvalho and Callaghan, 2011). The significant interaction effect observed across gender and seat support for low stiffness zone stiffness also suggests that postural differences may influence passive tissue strain during simulated motor vehicle rear-end impacts. While no significant gender effects were observed across unsupported and supported impacts for lumbar spine kinematics, it is interesting to note that on average, for unsupported impacts, female participants experienced an approximate 6 degree average increase in peak lumbar spine flexion in comparison to males (48 degrees vs. 42 degrees).

In supported impacts, only a 1 degree difference in peak flexion was observed (45 degrees vs. 44 degrees). Considering the normal ranges of motion of the lumbar spine are between 40° to 60° in flexion (Magee, 2006), it does appear that the peak magnitudes of lumbar flexion achieved during the simulated rear-end impacts are approaching end-range flexion for the lumbar spine.

An important finding to note from this investigation is that changes in passive stiffness were not accompanied by clinically significant LBP reporting (Hägg et al., 2003). Previous assessments of low speed rear impact collisions that relied entirely on symptom reporting (e.g., pain, discomfort and self-reported stiffness), with minimal consideration of the physical circumstances of the collision have made it challenging to confirm or infer lumbar spine injury causation in low speed rear impacts (Beattie and Lovell, 2010; Chapline et al., 2000; Magnússon, 1994; Richards et al., 2006; Yang et al., 2013). Pain and duration of symptom reporting are not solely biomechanical issues and are known to be subject to psychosocial factors. To the best of our knowledge, no laboratory study, has been able to replicate prolonged LBP reporting in human volunteers following a simulated low speed rear-end collision with severities of up to 11 km/h (Bailey et al., 1995; Brault et al., 1998; Braun et al., 2010; Castro et al., 1997; Keifer et al., 2010; McConnell et al., 1995b; Szabo and Welcher, 1996). Results from this investigation agree with previous observations, as there was no sign of any prolonged LBP symptom reporting, even with repeated exposures separated over a 2-week period. This lack of pain reporting is supported by the peak exposures measured in this investigation which were low, below existing injury reference values and within the range of loads considered safe for manual materials handling tasks.

Before interpreting and implementing the findings from this study, a number of limitations should be considered. For the collision simulations, pressure-sensing units are limited to measuring forces acting normal to the seatback. As such, any frictional forces that may exist between the occupant-seat interface during the simulated impacts could not have been measured. Second, rear-end collision responses were characterized on one automotive seat model (i.e., 2017 Honda CRV model). Differences in seat design can alter how an individual positions themselves in the seat and responds to a simulated rear-end motor vehicle collision. Third, a single simulated crash severity was examined in this study (i.e., ΔV = 7.66 km/h), while this severity is at the higher-end of simulated rear-impact collision testing performed with human volunteers, it is unknown how changes in collision severity would influence responses. Fourth, trunk muscle activations were not included in the estimation of L4/L5 compression and shear forces. Previous work within our group

has observed significant differences between peak muscle activity and peak acceleration of the lumbar spine and indicates that muscular contractions are too slow to resist any initial impact forces, unless they are recruited (braced) prior to impact. Although the inclusion criteria included a large anthropometric range, participants from this study had a stature that was within the 25th to 75th percentiles and an average BMI that represents a healthy population. As such, occupant interactions driven by body dimensions may be exacerbated for individuals beyond the examined height and BMI ranges. Lastly, all participants included in this investigation were healthy young adults. The current work cannot comment on how the simulated impacts would/could cause or exacerbate LBP in individuals who have had a medical history of prior LBP reporting.

For passive testing, the passive range of motion measurement approach used in this investigation determined end range of motion subjectively by the experimenter and feedback from the participant. To minimize the effect of this limitation all passive range of motion measurements were conducted by the same experimenter who was trained in this technique. A second limitation is that slight changes in participant positioning within the passive jig across trials may have occurred across stiffness collections. A change in position will have some influence on stiffness measurements and it is possible that slight changes in the orientation of the participant within the passive jig may have changed passive measurements, every effort was made to ensure consistent positioning and that the tests isolated the lumbar spine. A final limitation of this study stems from the young age group tested. The benefit of using a younger population is that confounding degenerative changes and other co-morbidities typically present in older persons can be controlled for. The downside is that the results of this study are at best generalized to a healthy young population. Tissues tend to lose hydration with degenerative changes and become stiffer, therefore, one could speculate the passive response following a low speed impact might be more dramatic in older individuals. Future research should incorporate a larger study population with an age range representative to the entire population.

5.7 Conclusions

Findings demonstrate that during a laboratory-simulation of an unanticipated 8 km/hour rear-impact collision, young healthy adults do not report LBP. A lumbar support did not significantly influence the estimated L4/L5 joint reaction forces. Changes in the low stiffness portion of the passive flexion/extension curves were observed following impact and persisted for 24 hours. Changes in passive stiffness may lead to changes in the loads and load distributions within the

passive structures such as the ligaments and intervertebral discs following impacts. This investigation represents the first experiment to characterize the *in vivo* mechanical exposures to the lumbar spine during simulated low-speed rear impacts.

Chapter 6: Exploring the Interaction Effects of Impact Severity and Posture on *In-vitro* Vertebral Joint Mechanics

6.1 Overview

Background: To date, no *in vitro* studies have been conducted to explore lumbar soft tissue injury potential and altered mechanical properties from exposure to impact forces. Typically, after a motor vehicle collision, the cause of a reported acute onset of low back pain is difficult to identify with potential soft tissue strain injury sites including the facet joint and highly innervated facet joint capsule ligament.

Objectives: The purpose of this investigation was to quantify intervertebral translation and facet joint capsule strain under varying postures and impact severities. A secondary objective was to evaluate flexion-extension and shear neutral zone changes pre and post impact.

Methods: A total of 72 porcine cervical FSUs were included in the study. Three levels of impact severity (4g, 8g, 11g), and three postures (Neutral Flexion and Extension) were examined using a full-factorial design. Impacts were applied using a custom-built impact track which simulated impact parameters similar to those experienced in low to moderate speed motor vehicle collisions. Passive flexion-extension and shear neutral zone testing were completed immediately prior to and immediately post impact. Intervertebral translation and the strain tensor of the facet capsule ligament were measured during impacts. For passive testing independent variables of impact severity, posture and pre/post were assessed. For dependent variables obtained during impacts, independent variables of impact severity and posture were assessed.

Results: A significant main effect ($p > 0.001$) of collision severity was observed for peak intervertebral translation and peak FCL shear strain ($p = 0.003$). A significant two-way interaction was observed between pre-post and impact severity for flexion-extension neutral zone length ($p = 0.031$) and stiffness ($p>0.001$) and anterior-posterior shear neutral zone length ($p = 0.047$) and stiffness ($p>0.001$). This was a result of increased neutral zone range and decreased neutral zone stiffness pre-post for the 11g severity impact (regardless of posture).

Conclusions: This investigation provides evidence that overall the peak vertebral translations observed across 4g to 11g impacts are below previously published ultimate shear failure displacements. FSU's exposed to the highest severity impact (11g) had significant NZ changes, with increases in joint laxity in flexion-extension and shear testing and decreased stiffness, suggesting that soft tissue injury may have occurred. Despite observed main effects of impact

severity, no influence of posture was observed. This lack of influence of posture and small FCL strain magnitudes suggest that the FCL does not appear to undergo injurious or permanent mechanical changes in response to low to moderate MVC impact scenarios.
Keywords: impact, facet joint capsule, strain, neutral zone, shear

6.2 Introduction

Previous epidemiological studies have identified a link between low speed rear-end collisions and acute onset of low back pain (Beattie and Lovell, 2010). A limited number of studies have attempted to simulate rear impacts to measure the mechanical exposures of the lumbar spine (Fast et al. 2002; Gushue et al. 2001; Gates et al. 2010). All investigations determined that traumatic injury (such as acute disc herniation or avulsion fractures) in the lumbar spine is unlikely given the estimated kinematics and forces (Gates et al., 2010; Gushue et al., 2001). To date, no *in vitro* studies have been conducted to explore lumbar soft tissue injury potential and altered mechanical properties from exposure to low velocity rear impact forces. Typically, after a motor vehicle collision, the cause of a reported acute onset of low back pain is difficult to associate with potential soft tissue strain injury sites including the ligaments, intervertebral disc, facet joints muscle and neural components. Therefore, the purpose of this investigation was to characterize intervertebral joint mechanics of porcine soft tissues in response to variations of posture and impact severity.

Prior investigations have demonstrated that when exposed to a simulated low velocity rear-end collision the peak lumbar spine loads are relatively low in comparison to published tissue failure tolerances in the lumbar spine (Fast et al. 2002; Gushue et al. 2001; Gates et al. 2010). A cadaver study replicated low velocity rear-end collisions at changes in velocity of 13 and 19 km/hour and they found no boney injuries and that the mechanisms to result in acute traumatic intervertebral disc (IVD) injury were not present (Fast et al., 2002). Most often disc herniations are described primarily as a fatigue injury (Adams and Hutton, 1983). As a result, it has been considered that irritation or injury to the lumbar soft tissues surrounding the IVD from shear loading may play a more important role in the pathogenesis of low back pain after a low velocity collision (Fast et al. 2002).

When seated in an automotive seat the intervertebral joints rotate into flexion, which results in a change in orientation of the passive tissues. *In vitro* tests have demonstrated that flexed postures alter a intervertebral joint's shear injury potential (Howarth and Callaghan, 2012), and result in increased strain of the posterior lumbar ligaments (Panjabi et al., 1982). Flexed postures also increase the gap between articulating facets (Drake et al., 2008), and result in facet joint capsule deformation (Little and Khalsa, 2005a). The facet joint capsule (FJC) has been the focus of several research studies investigating whiplash injury mechanisms (Pearson et al. 2004; Siegmund et al. 2001; Lu et al. 2005). Increasing capsule deformation has been statistically

correlated to neural responses, indicating a potential pain link to FJC deformation (Lu et al., 2005b). Exposure to a sudden impact such as a rear-end collision, coupled with a flexed posture, may result in sub-failure micro-damage to the soft tissues surrounding the IVD. The mechanical properties of soft tissues can be altered by sub failure injury (Quinn et al., 2007; Quinn and Winkelstein, 2008). This could, in part, be responsible for low back pain generation after low velocity automobile collisions.

How sub-failure impact loading may be linked to altered joint mechanical properties such as increased joint laxity or decreased stiffness is largely unknown. Therefore, the primary objective of this study was to explore the combined effects of posture and impact severity on intervertebral joint mechanics. A secondary purpose was to quantify the amount of intervertebral translation and FJC strain during impacts. Low to moderate severity impacts were simulated on isolated functional spinal units. Horizontal intervertebral translation and FJC capsule strain were assessed during impacts. Flexion-extension and shear neutral zone (NZ) testing was completed prior to and post impact. In line with these objectives, it was hypothesized that as impact severity increased there would be a subsequent change in intervertebral joint mechanics following impact evidenced by significant changes in neutral zone length. A secondary hypothesis was that horizontal intervertebral translation and FJC strain would increase with increasing impact severities.

6.3 Methods

6.3.1 Study Design

In vitro testing of FSUs to characterize potential soft tissues disturbances. Three independent variables were used for passive testing measures; impact severity, posture and pre-post impact. Two independent variables were used for impact testing measures; impact severity and posture.

6.3.2 Specimen Preparation

The cervical spines of 36 porcine specimens were obtained following death and stored at -20ºC. For testing, each spine was separated into two FSUs; one at the C34 level and one at the C56 (72 FSUs total). Porcine cervical FSUs were used as surrogates for the human lumbar spine due to the anatomical and functional similarities (Oxland et al., 1991; Yingling et al., 1999). The porcine model provides greater control over potential confounding factors such as age, disc degeneration, nutrition and physical activity that can impact the mechanical integrity of the tissues surrounding the IVD.

Prior to testing, specimens were thawed at room temperature for 12 hours. Dissection of the cervical spine included isolating the two FSUs (C34 & C56) and removal of the surrounding musculature. Next, tissues superficial to the outer facet joint capsule surface were carefully removed until the glossy joint capsule was completely visible (this was necessary for facet joint capsule tracking – see section 5.3.3.3). Following dissection, the quality of the exposed superior and inferior IVD was assessed using the grading scale outlined by Galante (1967). Only specimens with disc quality of Grade 1 were used for this investigation (Galante, 1967). Next, measurements of superior and inferior endplate width and depth were recorded using digital calipers. These measurements were used to estimate an average intervertebral joint endplate surface area using the equation of an ellipse. Estimates of endplate surface area were then used as inputs to a regression equation to approximate each FSU's ultimate compressive tolerance (UCT) without destructive testing (Parkinson et al., 2005). This allowed for normalization of compressive loading across specimens during impacts. The FSU was then fixed in custom aluminum cups using a combination of screws, steel wire and dental plaster. To prevent specimen dehydration throughout preparation all FSUs were misted with a saline solution approximately every 15 minutes.

6.3.3 Instrumentation

6.3.3.1 Impacts

Similar to Study 3, a custom-built impact track was developed to apply impact parameters of rear-end collisions corresponding to approximate collision severities of 8, 16 and 24 km/hour representing impact severities of 4, 8 and 11g (Figure 6.1). The impact device consisted of a 206 cm plexiglass track, in which a 6.5 kg sled was launched down the track and struck a single custom designed spring (inner diameter = 9.32 cm, outer diameter = 11 cm, active number of coils = 12.37, free length =34.26 cm) (Omnicoil, Ayr, ON), with damping and stiffness parameters of 3816.1 N/m and 0.0999 Ns/m respectively. The spring was rigidly attached, in-line, to the FSU loaded in the mechanical systems testing device under compressive load. Flat stainless-steel plates were attached to both the impact truck and spring to ensure the truck directly struck the spring. The centre of the track was elevated to constrain the rolling truck and ensure the truck remained centred while rolling down the track and hit the spring centred (Figure 6.1). During all testing, the FSU was orientated such that the anterior surface of the specimen was facing away from the impact track, with the inferior vertebra being impacted resulting in anterior shear created in the joint (Figure 6.1).

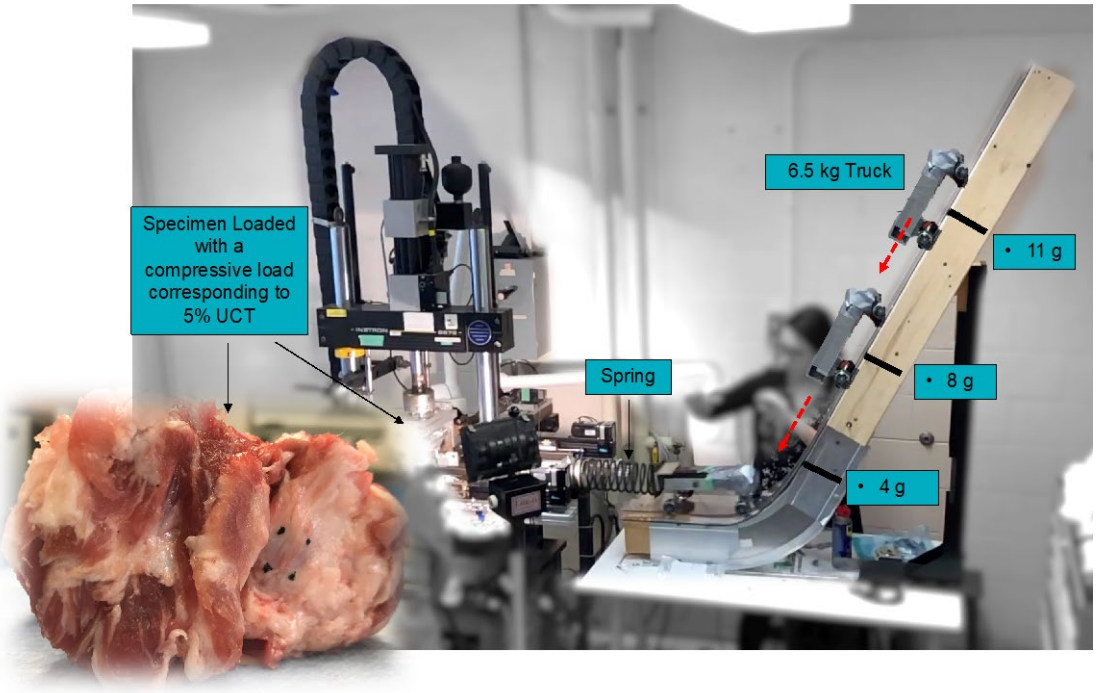

Figure 6.1: Orientation of the specimen in the materials testing device and the impact track setup displaying the 3 drop heights for each of the 3 respective impact severities. The single spring was rigidly in series with the mounted specimen in the materials testing device, attached to the cup containing the specimens' inferior vertebra. For each impact the truck was released such that the trucks' centre of mass was aligned with the drop height, centered on the track.

For design of the impact device, reaction forces obtained from **Study 3**, coupled with currently published ATD lumbar spine reaction force estimates for an 8 km/hour rear-end collision severity were used (Gates et al., 2010; Gushue et al., 2001). The impact device was designed that the impact parameters for the simulated 8 km/hour collision severity consisted of the following: a peak acceleration of 4 g, an approximate 130 ms impact duration, and an applied shear load of approximately 250 N (**Study 3**)(Gates et al., 2010; Gushue et al., 2001). For simulated impact severities of 16 and 24 km/hour, the peak applied acceleration was scaled up based on previously published ATD testing, corresponding to peak accelerations measured during 16 km/hour and 24 km/hour sled testing (Fast et al., 2002; Gates et al., 2010; Gushue et al., 2001). For such simulations, impact parameters remained fixed, however the height at which the truck was released on the designed track was changed to increase the peak acceleration of the truck and as a result, the peak applied force (Table 6.1). This impact track was validated using the identical test setup however during validation trials the linear spring apparatus was rigidly attached to a cement cinderblock. Validation results can be found in Table 6.1.

Table 6.1: Estimated impact parameters and measured impact parameters during the validation phase of the impact track. Peak accelerations were measured using one tri-axial accelerometer (ADXL377, Analog Devices, Norwood, MA, USA) rigidly attached to the truck.

	Estimated Impact Parameters			Measured	
Simulated Collison Severity (km/hour)	Launch Height (m)	Peak Acceleration (g)	Applied Peak Force	Peak Acceleration (g)	Applied Peak Force (N)
8	0.138	3.9	250	4.12	264.1
16	0.538	7.9	506.5	8.11	520.2
24	1.02	11	705.2	10.91	698.2

6.3.3.2 Intervertebral Translation

FSU intervertebral translation was measured using a linear potentiometer with a 50 mm stroke (TS50, Novotechnik U.S. Inc., Southborough, MA, USA) that was rigidly mounted in parallel with the caudal vertebrae during impacts. Following pilot testing, this method was chosen over 3D kinematics for obtainable sampling rates. Voltages from the linear potentiometer were digitally sampled at a rate of 5000 Hz using a 16-bit analog to digital conversion board for all impacts (PCI 6034E, National Instruments Inc., Austin, TX, USA).

6.3.3.3 Facet Joint Capsule Deformation

Deformation of the facet joint capsule ligament was tracked during all impacts. Immediately following dissection of the FSU, four markers were drawn in a rectangular configuration on the left exposed superficial facet capsule layer using an India ink marking pen (Figure 6.2). The India ink markers were configured to capture strain of the superior facet joint capsule fibers together with the inferior fibers. Two-dimensional video of the facet capsule markers was recorded using a high-speed camera (Chronos 1.4, Kron Technologies Inc., Burnaby, BC, Canada) equipped with a Canon EF-S 18-55mm lens. The camera was oriented perpendicular to the plane of the facet joint and sampled at a rate of 1502 frames per second (resolution: 1280 × 720 pixels).

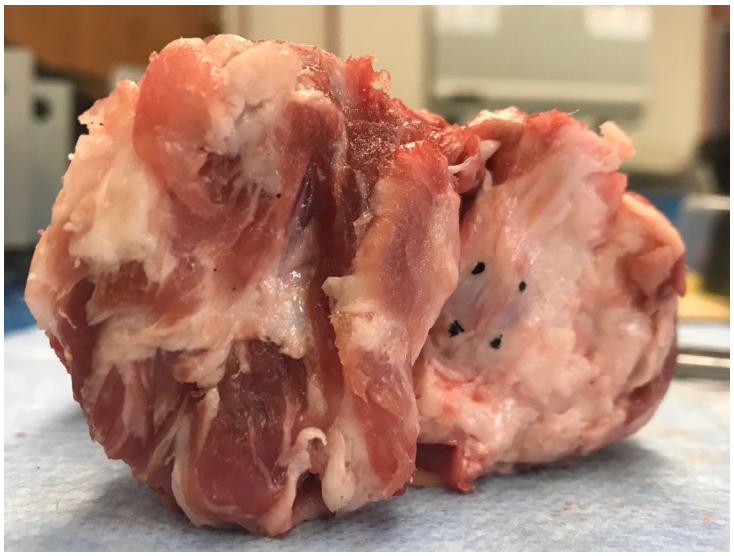

Figure 6.2: The left lateral view of a specimen with four India ink markers.

6.3.4 Procedure

The potted specimens were mounted into a custom servo-materials testing system that has been modified to apply flexion/extension rotations to FSUs under compressive load. FSUs were first preloaded with a 300 N compressive preload to counter any post mortem swelling that occurred within the IVD (Callaghan and McGill, 2001b). During this preload test, a brushless servomotor connected in series with a torque cell was used to establish the angular position of minimal stiffness (Nm/degree) about the flexion/extension axis. This position defined each specimen's neutral position. Following the preload test, three cycles of controlled passive Flexion-extension (PFE) testing was completed at a rate of 0.5 degrees/second, under a constant load of 300N. Using custom software, the applied moment (Nm) and angular displacement (degrees) were sampled at 25 Hz. The cup containing the FSU's caudal vertebrae was free to translate on metallic ball bearings. The flexion extension limits of the NZ were identified using methods defined by Thompson and colleagues (Thompson et al., 2003). The first derivative of a fourth-order polynomial fit to the moment-angle data from the last two loading cycles were used to detect when the angular curve deviates from linear (Noguchi et al., 2015). These endpoints were used to objectively define the maximum NZ flexion and extension limits as well as the neutral posture for passive shear testing.

Next, three cycles of controlled passive shear testing were completed at a rate of 0.2 mm/second, under a constant compressive load of 300N, in the neutral posture. To apply shear loading, the cup containing the specimen's caudal vertebrae (either C4 or C6) was attached to two linear actuators (RSA24, Tolomatic Inc., Hamel, MN, USA) via a metal extension rigidly attached

for use during the passive shear protocol, driven by a pair of brushless servomotors (AKM22E, Danaher Motion Inc., Radford, VA, USA) (Figure 6.3). The linear actuators were controlled in parallel to ensure equal movement and were equal distance from the specimen's mid-sagittal plane to prevent the application of an axial twisting moment. A uniaxial load cell (MLP-500, Transducer Technologies, Temecula, CA, USA) was mounted in series to each linear actuator and was used to measure applied shear force during the preconditioning testing. Cycles of displacement during anterior-posterior shear passive testing were applied using a continuous motion control algorithm to shear force targets of +/- 400 N (Howarth and Callaghan, 2012). This is approximately 14% of previously measured ultimate anterior shear failure force (Gallagher et al., 2010). Shear force and displacement from each combination of load cell and linear actuator were continuously sampled at a rate of 7 Hz.

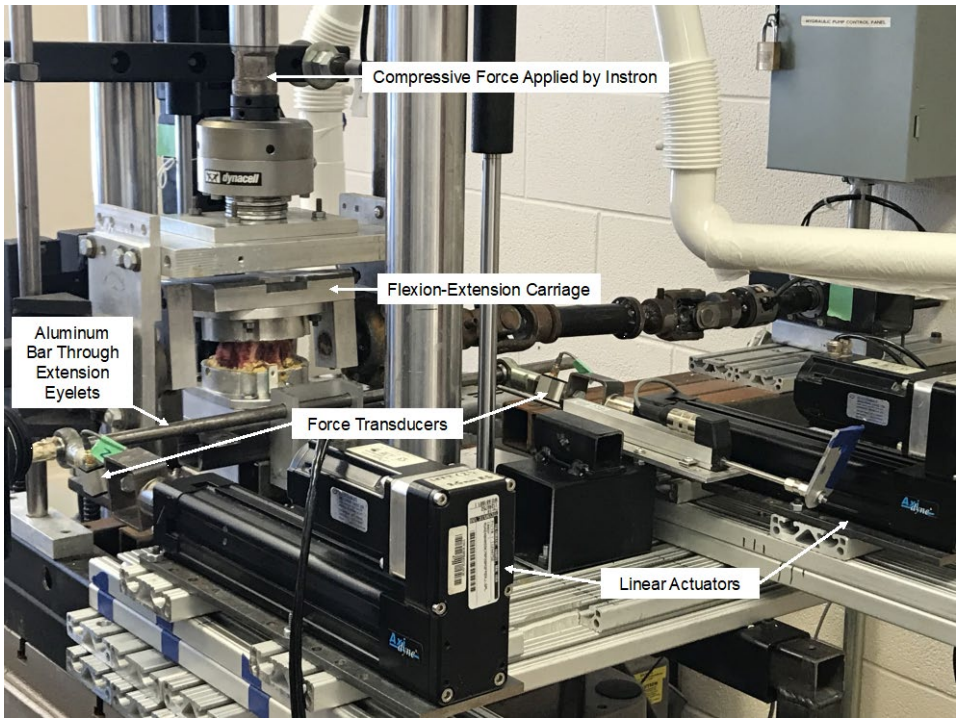

Figure 6.3: The material testing system setup for shear passive testing. Shear displacement was applied by moving the linear actuators forward (anterior shear) and backwards (posterior shear) while force was measured using two force transducers. The Instron applied constant compressive force (in load control) while the flexion/extension carriage maintained the specimen's posture in the neutral position.

After completion of the shear passive tests, the aluminum bar was removed, and the specimen was rigidly attached in series to the linear spring using nuts and bolts. Specimens were randomized into one of eight conditions (an equal number of C34 and C56 were used in each

condition). The conditions were made up of variations of simulated impact severity and posture. Three impact severities were simulated and were designed to apply corresponding impact parameters of rear-end collisions representing similar collision severities of 8, 16 and 24 km/hour (4g, 8g and 11g). Impact profiles were applied at three different starting postures. The three levels of posture consisted of Neutral (defined as the center of the NZ), 300% of the Flexion NZ limit and 300% of the Extension NZ limit (Neutral, Flexion and Extension). Flexion and Extension postures were designed to bring the isolated FSU to the end of the natural physiological range (i.e., where the passive rotational stiffness begins to deviate from linear). Impacts were applied using the above-mentioned impact device.

Each specimen was only exposed to a single impact. Prior to impact, the FSU was first compressively loaded (in load control) corresponding to 5% of the specimens' estimated UCT. This was completed to simulate the approximate compressive load of the upper body (approximately 500 N), while scaling the compressive load to each specific FSU. Next, the specimen was moved to the desired posture and the static specimen was then impacted. Immediately following the impact, the specimen was returned to the neutral posture, the spring was removed and three cycles of controlled passive Flexion-extension testing and three cycles of anterior-posterior passive shear testing were re-completed (under 300 N of compressive load) to monitor changes in the passive Flexion-extension and shear NZ.

6.3.5 Post Impact Analysis

Specimens were removed after completion of the impact protocol. Specimens were visually inspected for any possible locations of damage, and the right and left facet joint capsules were removed for testing in **Study 5**. Following facet capsule removal, specimens were then further dissected and the investigator assessed visually if any damage had occurred.

6.3.6 Data Processing and Analysis

6.3.6.1 Translation

Potentiometer signals were first filtered using a third order, dual pass, Butterworth low-pass filter with a cutoff frequency of 30 Hz. This frequency was originally obtained from previous *in vitro* work, using similar peak accelerations (Ivancic et al., 2005; Panjabi et al., 2005) and was tested on pilot data with the criterion of minimizing the vibration signals from the materials testing device without losing the measured signal. Next, sampled voltages from the linear potentiometer were calibrated to displacement. Peak horizontal intervertebral displacement during impact (with zero

being the original starting position prior to impact) were extracted. Sample displacements are presented in Figure 6.4.

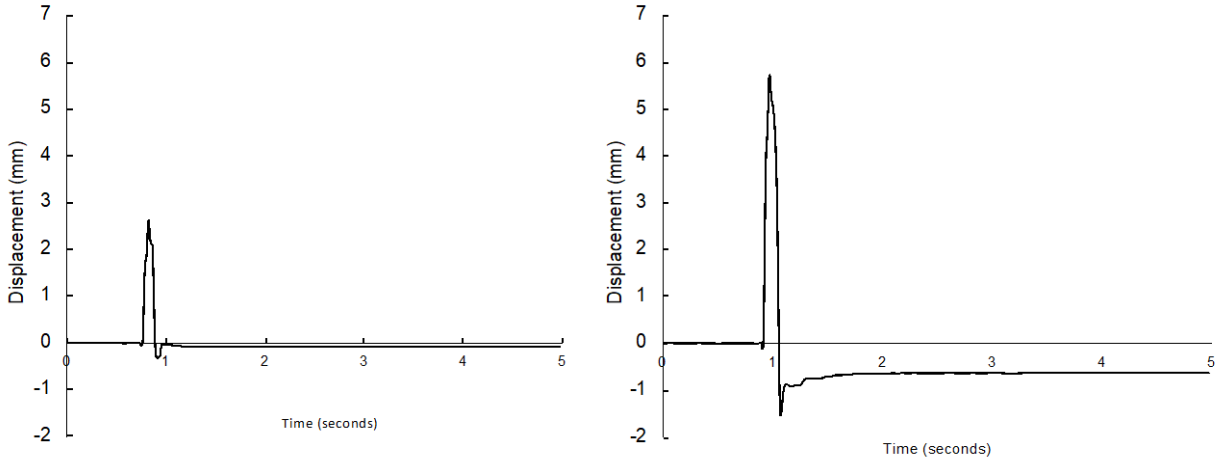

Figure 6.4: Sample calibrated potentiometer displacements for 4g (left) and 8g (right) impacts. A positive value indicates anterior shearing of the caudal vertebra relative to the cranial vertebra while a negative value indicates posterior shearing of the caudal vertebra.

6.3.6.2 Facet Capsule Tracking

Raw highspeed video were input into ProAnalyst® software (Xcitex Inc., Woburn, MA, USA). A two-point calibration was performed (Figure 6.5) and the regions of India ink markers were manually defined (Figure 6.5). Following identification, the two-dimensional coordinates of the marker centroids were tracked during the impact (Figure 6.5) and exported.

Coordinate data were then input into a custom Matlab (version, etc.) program where the Green strain tensor (***E***) was obtained by first quantifying the homogeneous transformation that mapped each marker's coordinates from the initial starting posture (compressively loaded under 5% of estimated UCT and in the desired posture) to the deformed configuration on a frame-by-frame basis (Equation 6.1).

$$\vec{x}' = \boldsymbol{F}\vec{x} + \vec{b} \tag{6.1}$$

Where $\boldsymbol{F}$ is the deformation gradient; $\vec{x}$ is the position of a marker in the neutral posture; $\vec{x}'$ is that marker's position on a given frame; and $\vec{b}$ absorbs any rigid body translation that may occur. The deformation gradient, and rigid translation, were quantified with a least-squares solution. Finally, the Green strain tensor was obtained from the deformation gradient (Equation 6.2):

$$\boldsymbol{E} = \frac{1}{2}(\boldsymbol{F}^T\boldsymbol{F} - \boldsymbol{I}) = \begin{bmatrix} E_{xx} & E_{xy} \\ E_{yx} & E_{yy} \end{bmatrix} \tag{6.2}$$

Where $\boldsymbol{I}$ is the 2×2 identity matrix; and $\boldsymbol{E}$ is the Green strain tensor, with components representing the anterior-posterior (E_{xx}), superior-inferior (E_{yy}), and shear strain (E_{xy}). These strains were quantified in favor of principal strains since the camera was situated to ensure an anatomically relevant coordinate system.

Peak anterior-posterior (E_{xx}), superior-inferior (E_{yy}), and shear strain (E_{xy}) were extracted for each impact.

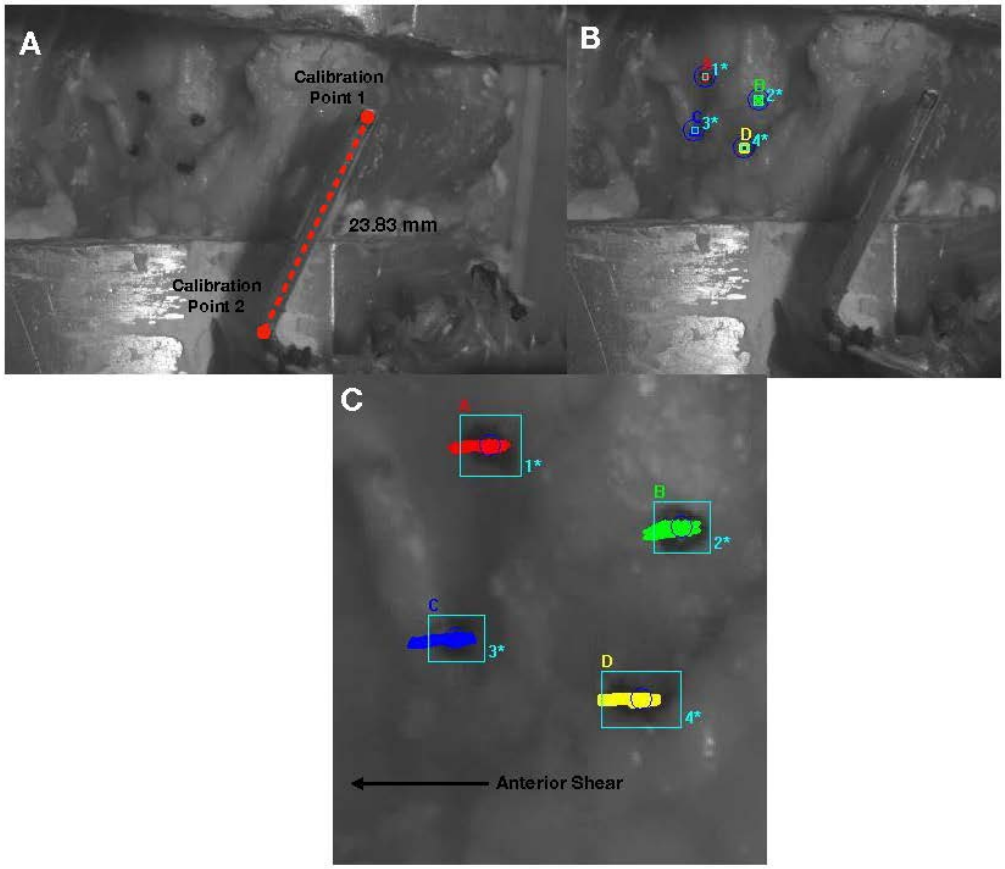

Figure 6.5: Labelling of India ink points and two-point calibration (A), definition and of the contrasted marker regions (B), tracking of the defined points during the impact (C).

6.3.6.3 Passive Testing

To establish the Flexion-extension NZ range, the first derivative was taken from a fourth-order polynomial fit to the moment angle data sampled during each test, and the range between ± 0.05 Nm/degree from the minimum point was used to indicate limits (Thompson et al., 2003). The

passive rotational stiffness within the NZ was described using the slope of a linear fit to the moment angle data within the boundaries that defined the NZ (Figure 6.6). The last two cycles of each test were considered for analysis, as previous research has shown reduced variability between the second and third cycles (Wilke et al., 1998).

For shear NZ analysis, the force and displacement data from each combination of linear actuator and load cell were tracked for the final two cycles of the shear tests (Howarth et al., 2013). Total shear force was calculated as the sum of the forces measured by the two load cells and displacement was calculated as the average displacement of the two linear actuators. Force and displacement were divided into individual posterior shear to anterior shear (Phase 1) and anterior to posterior shear (Phase 2) segments for separate analysis (Figure 6.7)(Howarth et al., 2013). Endpoints of the shear NZ were computed using a previously defined technique to quantify the shear NZ (Howarth et al., 2013). First to combat noise in the system the raw force and displacement curves were fit to a third-order polynomial (Howarth et al., 2013). Next, a double-sigmoid mathematical representation expressed shear displacements as a function of the measured shear force values. The NZ endpoints were selected from the maximum and minimum values of the second derivative of the fitted double-sigmoid function (Howarth et al., 2013). The NZ length was determined as the distance between the endpoints and the stiffness within the NZ was then determined as the reciprocal of the slope of a linear fit to the data endpoints (Howarth et al., 2013) (Figure 6.6).

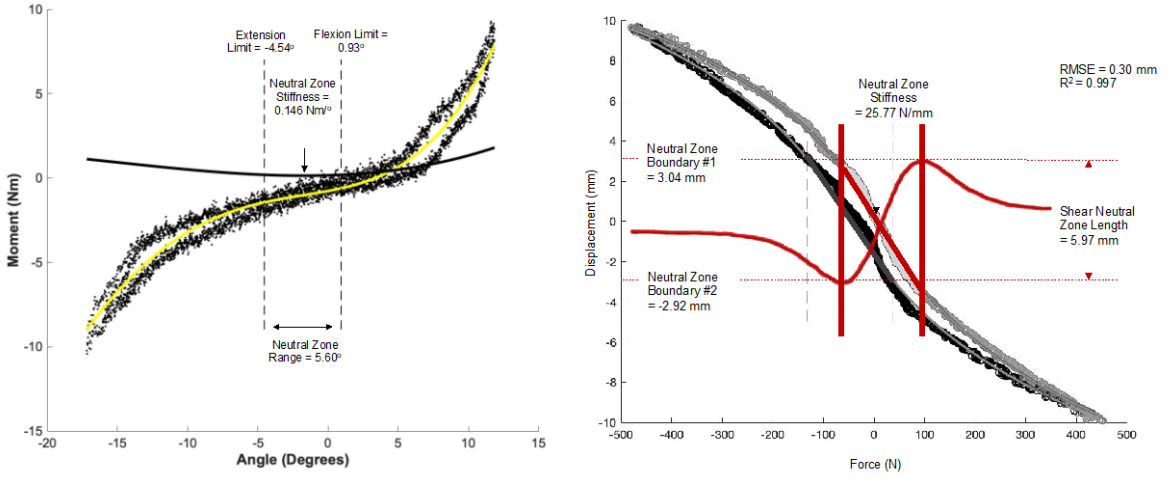

Figure 6.6: Left – sample raw data from one passive Flexion-extension test pre-impact, showing the fourth order polynomial fit, computed NZ limits and range, and the passive rotational stiffness within the NZ using the slope of the fit to the moment angle data. Right – sample raw data from

one complete shear passive test pre-impact, for the posterior-anterior shear cycle, the double-sigmoid fit, computed NZ limits and range, and stiffness are displayed.

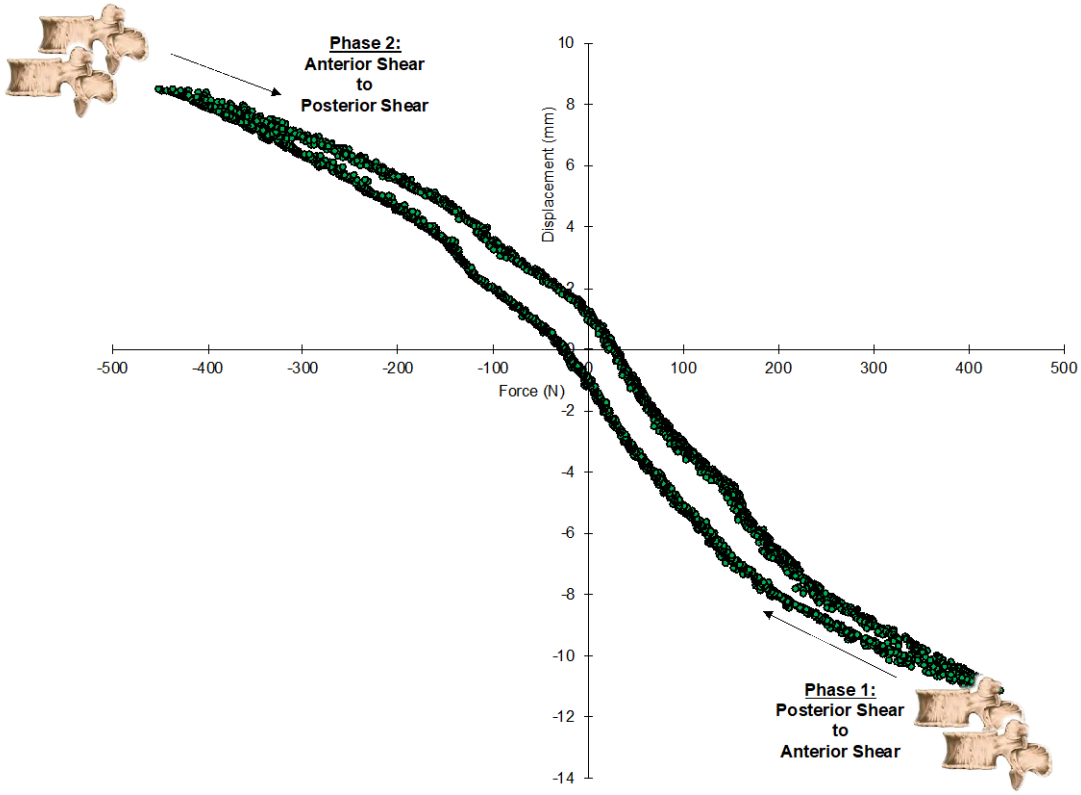

Figure 6.7: Sample raw data from one passive shear NZ test demonstrating how the shear NZ test was separated. Phase 1 consists of the shear NZ test in which the specimen moved from maximum posterior shear to maximum anterior shear. Phase 2 consists of the portion of shear NZ test in which the specimen moved from maximum anterior shear to maximum posterior shear.

In total, 6 dependent variables were analyzed: Flexion-extension NZ Length and Stiffness, Shear NZ Length and Stiffness during Phase 1 and Shear NZ Length and Stiffness during Phase 2.

6.3.7 Statistics:

Significant differences for impact specific variables were assessed using a three-way (Level, Impact, and Posture) ANOVA for measurements of peak intervertebral translation, anterior-posterior strain, superior-inferior strain, and shear strain). Significant differences for passive variables were assessed using a four way, (Level, Pre-Post, Impact, and Posture) general linear model. Where significant differences of Level were not observed specimens were collapsed across level. Tukey's test with Bonferroni adjustments were used for *post hoc* testing when appropriate.

6.4 Results

6.4.1 Impact Results

6.4.1.1 Peak Horizontal Displacement

A main effect of impact severity was observed for peak intervertebral translation ($p > 0.001$). Not surprisingly, all 3 impact exposures resulted in significantly different peak intervertebral translation (Figure 6.8). This was a result of significantly greater intervertebral translation from the 4g impact (2.8 $\pm$ 0.53 mm) to the 8g impact (6.4 $\pm$ 2.9 mm), to the 11g impact (8.3 $\pm$ 0.45 mm). No main effects or interaction effects of posture were observed.

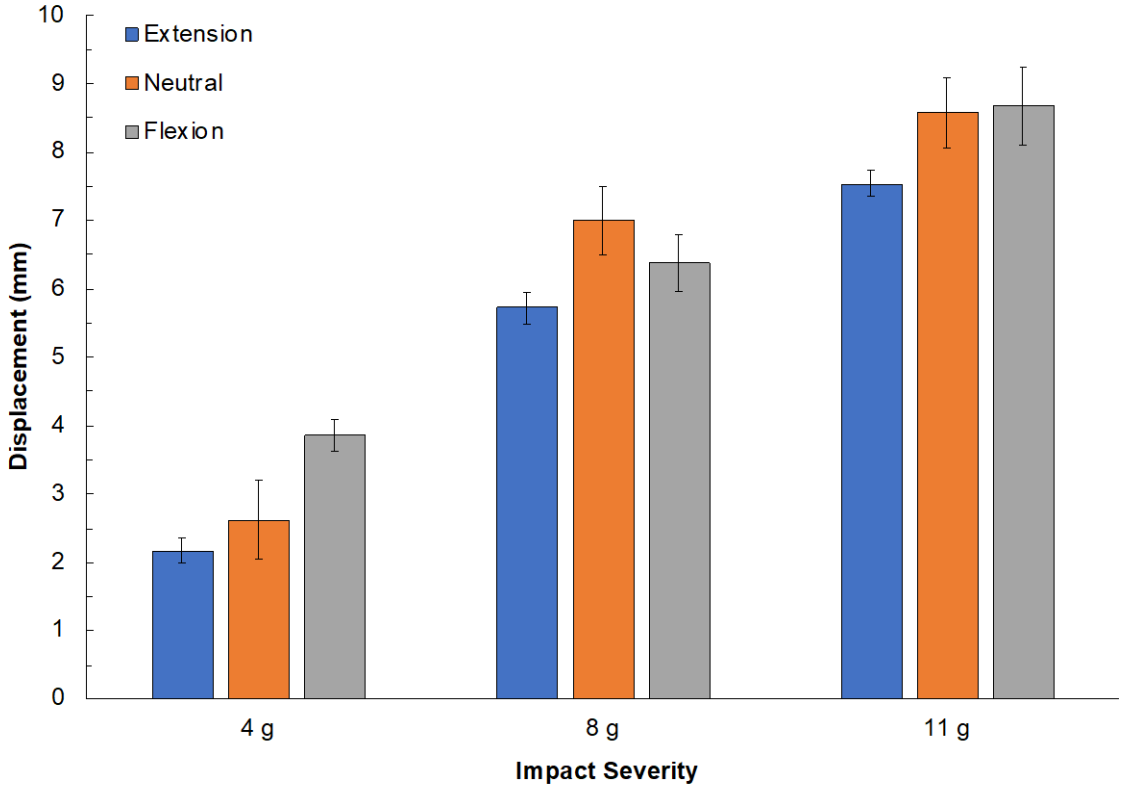

Figure 6.8: Caudal vertebrae translation separated across posture for each of the impact severities tested. A main effect of impact severity was observed with average translation for each impact severity being statistically different. Standard Deviation bars are displayed.

6.4.1.2 Peak FCL Strain

No significant differences for peak anterior-posterior ($p = 0.228$) and peak superior-inferior strain ($p = 0.200$) were observed across impact severity. A significant main effect of impact severity was observed for peak shear strain ($p = 0.003$)(Table 6.2).

The magnitude of peak anterior-posterior strain did not significantly differ across posture or impact severity. Anterior-posterior strain slightly increased across impact severity (average 1.6% increase from 4g to 11g) (Table 6.2). Across impact severities there was a slight increase (average 0.2% increase) in superior-inferior strain between 4g and 8g severity impacts and a slight decrease (average 0.4% decrease) in superior-inferior strain between 8g and 11g severity impacts (Table 6.2).

The magnitude of peak shear strain increased across impact severity with an average increase of 0.6% from the 4g to 8g impact severity and an average increase of 2.1% from the 4g to 11g impact severity (Table 6.2). Post-hoc testing revealed that peak shear strain observed during the 11g impact was significantly greater than that observed during the 4g impact ($p = 0.004$) and 8 g impact ($p = 0.041$).

Table 6.2: Computed mean peak strains across impact severity. Standard deviations depicted in brackets

Impact (g)	Anterior-Posterior *Exx* (%)	Superior-Inferior *Eyy* (%)	Shear *Exy* (%)
4	2.10 (2.5)	-0.66 (1.69)	-0.95 (1.51)
8	2.95 (3.3)	-0.85 (2.2)	-1.55 (2.05)
11	3.73 (4.4)	-0.45 (4.0)	-3.32 (3.59)

6.4.2 Passive Testing Results

6.4.2.1 Passive Shear Testing: Phase 1

A significant Pre-Post x Impact Severity interaction effect was observed for shear passive stiffness ($p>0.001$) and NZ range length ($p = 0.047$) for Phase 1. For shear passive stiffness, this was a result of a significant difference in Pre-Post stiffness for the 8g and 11g impact severities (Figure 6.9). Pre-Post passive shear stiffness was not significantly different for the 4g impact. For shear NZ length, a significant difference in Pre-Post NZ length was observed for the 11 g impact severity only (Figure 6.10). For both the 4g and 8g impact severities, no differences in NZ length were observed Pre-Post impact.

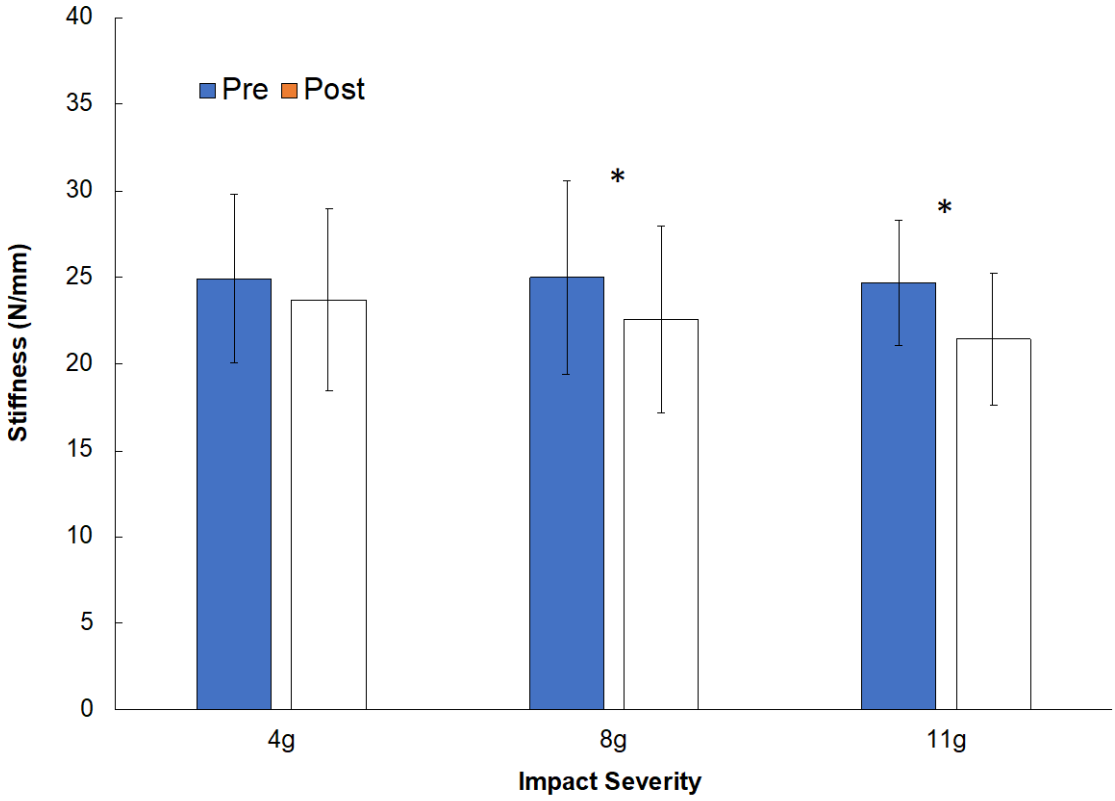

Figure 6.9: Phase 1 shear NZ stiffness pre impact and post impact across the three impact severities tested. Anterior-posterior shear NZ stiffness significantly decreased post impact for the 8g and 11g impact severities. No significant differences were observed for the 4 g impact severity. Average root mean squared error (RMSE) and explained variance (r^2) of the double-sigmoid fit were 0.29 mm and 0.997 respectively. Standard deviation bars are displayed.

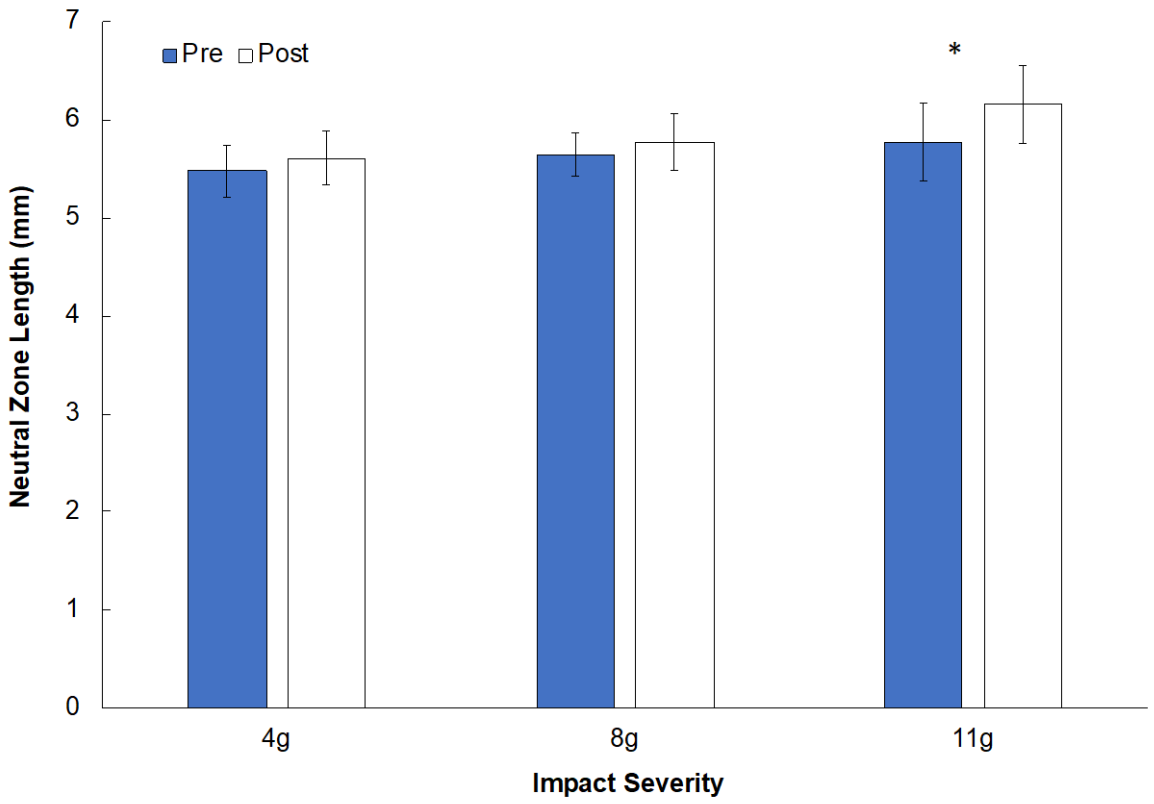

Figure 6.10: Phase 1 shear NZ length pre impact and post impact across the three impact severities tested. Anterior-posterior shear NZ length significantly increased post impact for the 11g impact severity. No significant differences were observed for the 4 g impact severity. Standard deviation bars are displayed.

6.4.2.2 Passive Shear Testing: Phase 2

Similar to Phase 1, a significant Pre-Post x Impact Severity interaction effect was observed for shear passive stiffness ($p=0.021$) and NZ range length ($p = 0.004$) for Phase 2. For shear passive stiffness, this was a result of a significant difference in Pre-Post stiffness for the 8g and 11g impact severities (Table 6.3) and a significant difference in Phase 2 shear NZ length for the 11g impact severity (Table 6.3).

Table 6.3: Extracted NZ length and stiffness for pre-impact and post-impact Phase 2 shear NZ tests.

	Pre Impact			**Post Impact**		
	4g	**8g**	**11g**	**4g**	**8g**	**11g**
Stiffness (N/mm)	22.99	23.25	21.65	21.54	21.16	19.24
SD Stiffness	4.54	5.62	3.06	4.87	5.62	3.66
NZ Length (mm)	5.28	5.53	5.20	5.42	5.66	5.73
SD NZ Length	0.53	0.39	1.22	0.53	0.43	0.76

6.4.2.3 Passive Flexion-Extension Testing

A significant main effect (*p>0.001*) of Pre-Post was observed for NZ passive stiffness. Across all conditions, there was a significant decrease in NZ stiffness post impact (Pre-Impact = 0.117 (± 0.01) Nm/degree; Post-Impact = 0.0690 (± 0.01) Nm/degree). A significant Pre-Post x Impact interaction (*p=0.031*) was observed for Flexion-extension NZ range. This was a result of there being a significant increase in NZ range for the 11 g impact severity only (Figure 6.11).

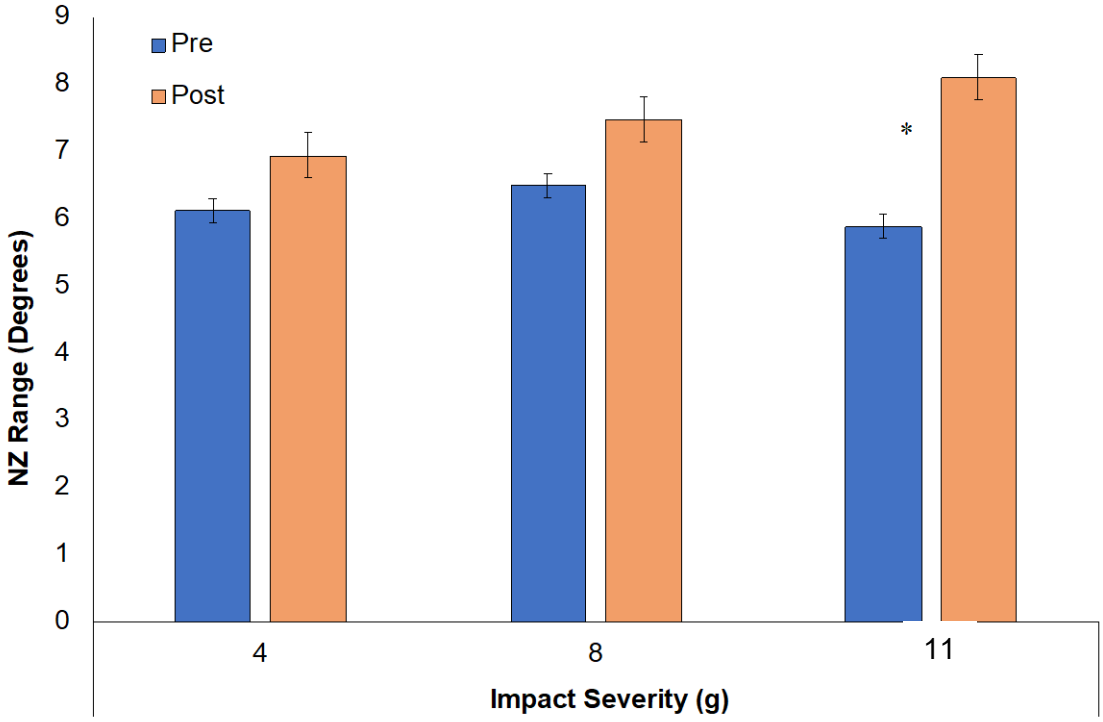

Figure 6.11: NZ length pre impact and post impact across the three impact severities tested. NZ length significantly increased post impact for the 11g impact severity. No significant differences were observed for the 4 g or 8 g impact severity.

6.5 Discussion

In line with our hypotheses, FSU's exposed to the highest severity impact (11g) had significant passive NZ changes, with increases in joint laxity in flexion-extension and shear testing and decreased stiffness. This investigation observed a significant effect of collision severity on peak intervertebral translation, with increasing horizontal displacement as impact severity increased and a subsequent increase in peak FCL shear strain as impact severity increased. Despite the observed main effects of impact severity, no influence of posture was observed. In addition, no significant

differences were observed for anterior-posterior FCL strain or superior-inferior FCL strain across impact severity or posture.

This investigation found that FSU's exposed to the highest severity impact (11g) had significant passive NZ changes in both flexion-extension and shear, with increases in NZ length and decreased NZ stiffness. Changes in NZ have been reported as a sensitive measure for determining onset and progression of intervertebral passive tissue injury (Oxland and Panjabi, 1992), with larger NZs in flexion associated with advanced intervertebral passive tissue injury and altered passive tissue resistance to applied load. In high-speed trauma experiments the flexion-extension NZ range has been found to increase with severity of injury (Panjabi et al., 1989). Results from this investigation align with previous *in-vitro* cervical spine whiplash testing on whole human cadaveric specimens, which demonstrated statistically significant changes in NZ with collision severities exceeding 8g for frontal collision simulations and 6.5g for rear-end collision simulations (Ivancic et al., 2005; Panjabi et al., 2005). Increases in NZ length can have negative consequences on the joint in question. As the ligaments and the muscles surrounding the joint work together to provide stability to the joint, Panjabi and colleagues (1999) hypothesized that increased joint laxity and NZ characteristics could be compensated by increased muscular activity (Panjabi et al., 1999). If injury is severe enough such that muscles are not able to fully compensate for the loss of the passive stability, the joint may develop chronic laxity. This can result in changes in the articulations to the joint, leading to possible aberrant movement patterns, nociception and altered mechanical joint properties. Based on the documented changes in NZ length for the highest severity 11g impact, results suggest that damage to the lumbar soft tissues surrounding the IVD did occur, which was reflective by the significant increase in NZ range post impact. Future work should attempt to document micro-injuries using a histological approach.

This investigation consistently observed changes in NZ stiffness occurring prior to changes in physical NZ length. For shear Phase 1 NZ testing, significant changes in post shear NZ stiffness occurred in specimens exposed to 8g and 11g severity impacts, while actual significant changes in shear NZ length were only observed for the 11g severity impact (average of 0.43 mm increase). Similar results were found for flexion-extension testing, with post changes in NZ stiffness occurring for all impact severities, but only the 11g severity impacts resulted in significant NZ range changes (average 2.2 degree increase). While not directly comparable to human investigations, this result does align with results from **Study 3**, which observed significant changes

in flexion low stiffness zones in female participants immediately following impact. It is possible that changes in NZ stiffness may be a recoverable precursor to soft tissue sub failure type injuries. Recoverable changes in passive zone stiffness have also been observed in human volunteers exposed to repetitive lifting, prolonged office seating and prolonged driving (Beach et al., 2005; De Carvalho and Callaghan, 2011; Parkinson et al., 2004).

To the best of my knowledge, this is the first investigation to report peak displacements across varying impact severities and posture. The horizontal displacements observed ranged between 2.2 and 8.7 mm and are within the range of the shear displacements observed during shear NZ testing. Peak anterior displacements across impact severities were approximately 50%, 120% and 150% of the average shear NZ range measured prior to impact, for 4g, 8g and 11g severity impacts respectively. Gallagher and colleagues (2010) reported ultimate acute shear failure displacements, for porcine cervical FSU's, ranging between 16 and 22 mm across anterior shear displacement rates ranging between 1 mm/s and 16 mm/s. In contrast, Yingling and McGill (1999) and Cripton and colleagues (1995) have reported much smaller ultimate shear failure displacements for porcine cervical FSUs and human cadaveric specimens, ranging between 10 to 13 mm. Such differences may be accounted for in terms of failure criteria and the amount of compressive load applied, increased compressive load has been demonstrated to increase a specimens' ultimate shear load tolerance loading (Howarth and Callaghan, 2012; Yingling and McGill, 1999). The absolute highest anterior displacement measured in this investigation was 8.6 mm during the Flexed-11g impact, which is approximately half of the ultimate shear displacements estimated by Gallagher et al. (2010), and also below ultimate shear displacements measured by other groups. In addition, across all previous shear failure testing the displacement rates used were significantly lower than a typical impact scenario and the rates used in this study. The rate of shear loading has been demonstrated to increase a specimens' ultimate shear load tolerance, ultimate shear displacement and average stiffness to failure (Howarth and Callaghan, 2012; Yingling and McGill, 1999). Thus, it is likely that the hypothetical ultimate shear tolerance would be greater for specimens in the current investigation, based on the significantly higher loading rate. Overall, the measured peak horizontal displacements observed in this investigation were below previously documented ultimate anterior shear failure displacements. In alignment with this finding, this investigation did not document any boney injuries and does not support an injury mechanism that results in acute traumatic fractures, such as pars interarticularis fractures and/or endplate avulsion

injuries, commonly observed in repetitive anterior shear failure mechanical testing (Cripton et al., 1995; Gallagher et al., 2010; Yingling and McGill, 1999). This result is also in agreement with previous cadaver work completed by Fast and colleagues (2002), who also found no boney injuries and that the mechanisms to result in acute traumatic IVD injury were not present (Fast et al., 2002).

Similar to the human cervical spine (Siegmund et al., 2001b; Winkelstein et al., 2000), the results of the current study demonstrate that a combination of strains concurrently occur in the FCL when subjected to low to moderate impacts. To the best of my knowledge, this is the first investigation to report facet capsule strain while spinal joints were exposed to impacts. Until recently, previous work tracking lumbar FCL strains in human cadaveric specimens were limited to low range flexion-extension tests and did not investigate the influence of shear (Ianuzzi et al., 2004). The range of horizontal intervertebral displacements observed during impact exposures are of similar magnitude to recently published work within our group examining anterior-posterior, superior-inferior and shear FCL strains during physiological translation ROM tests (Zehr et al., 2019). Zehr and colleagues (2019) conducted ROM testing on porcine FSU's to horizontal displacements reaching 200% of the estimated shear NZ range (Zehr et al., 2019). During anterior displacement of the caudal vertebrae, Zehr and colleagues (2019) also observed a similar trend for strains occurring in the FCL. During anterior shear, when exceeding 50% of the shear NZ range, anterior-posterior FCL strains and superior-inferior FCL strains remained unchanged, while the magnitude of FCL shear strain systematically increased from 50% of the shear NZ range out to 200% of the shear NZ range. The current work directly aligns with these findings, with no effect of impact severity observed for anterior-posterior FCL strain and superior-inferior FCL strain and a main effect of impact severity observed for shear FCL strain. Similar to Zehr and colleagues (2019), the magnitude of anterior-posterior FCL strains and superior-inferior FCL strains quantified were very low (below 4% and 1% respectively) and considerably less than the average horizontal (≈8%) and vertical (≈8-14%) principal strains quantified by Iannuzzi and colleagues (2004) under modest amounts of flexion-extension. While shear strain systematically increased across impact severity, the observed strains were still quite small in magnitude (below 5%). The small facet capsule strain quantities observed suggest that the facet joint capsule does not appear to undergo injurious or permanent mechanical changes during the tested impact scenarios.

The current investigation is the first to demonstrate that altering flexion/extension posture has a negligible effect on peak shear displacement and peak FCL strain of the porcine cervical

spine exposed to low to moderate severity impacts. Howarth and colleagues (2013) also found a negligible effect of flexion/extension posture on shear NZ length when exposed to anterior/posterior shear. Howarth et al. (2013), hypothesized that the absence of a significant difference in NZ length across posture suggests that the facets may not be the primary structure resisting low magnitude shear forces. In addition, the recent work reported by Zehr et al. (2019), also reported that despite translational joint stiffness increasing with increasing horizontal displacement a similar relationship was not evident for FCL strain during range of motion shear testing (Zehr et al., 2019). Aligning with previous work, the forces used within this experiment were also low in magnitude, ranging between approximately 10–30% of shear failure force for specimens tested in a flexed posture (Howarth and Callaghan, 2012). Lu et al. (2005) found that transection of the intervertebral disc resulted in a 23% decrease in shear stiffness while removal of facets generated a 78% decrease in shear stiffness. Conversely, Yingling and McGill (1999) reported that the intervertebral disc can resist up to 77% of shear force. It is possible that the intervertebral disc may resist the majority of shear load at low magnitudes and may be the primary load bearing structure during low to moderate severity impacts. This would explain the minimal effect of posture deviation on peak intervertebral shear displacement and the low magnitude of FCL strain observed in the current investigation.

It is important to interpret the findings from the investigation in the context of the limitations. The application of findings from this study to the human lumbar spine is limited by the use of porcine cervical FSUs. Secondly, the current investigation did not take into account any physiological repair of the tissues and/or the long term effects of soft tissue injuries. It is possible that the changes in NZ observed at the higher severity impacts may have been recoverable. Third, during facet capsule tracking, despite the prescribed motion and loading being highly controlled, it is possible that not all tissue deformation was planar in nature. Although, out-of-plant motion is believed to have been minimal under the prescribed impacts, this method is insensitive to any out-of-plane deformation that may have occurred. Fourth, the present study reported responses from single functional spinal units exposed to sagittal impact loading only, it did not simulate coupled intervertebral motions or rotational movements within a functional spinal unit that most likely occur during real life rear-end motor vehicle accidents. The current investigation was not meant to directly replicate a rear-end motor vehicle collision scenario and was designed as an initial step to investigate the interacting effects of impact severity and posture on possible injury mechanisms to

the lumbar spine. Fifth, during the current impact simulations, the superior vertebra remained fixed and the shear load was applied directly to the inferior vertebrae. It is possible and likely, that during a real rear-end motor vehicle collision, the actual loading scenario may be different.

6.6 Conclusions

This investigation provides evidence that overall the peak intervertebral translations observed across 4g to 11g impacts are below previously published ultimate shear failure displacements and do not support a lumbar spine injury mechanism resulting in acute traumatic bone fractures and/or acute traumatic IVD herniations. FSU's exposed to the highest severity impact (11g) had significant NZ changes, with increases in joint laxity in flexion-extension and shear testing and decreased stiffness, suggesting that soft tissue injury may have occurred. Despite observed main effects of impact severity, no influence of posture was observed. The lack of influence of posture and small facet capsule strain quantities observed across impact severities tested suggest that the facet joint capsule does not appear to undergo injurious or permanent mechanical changes during the tested impact scenarios.

Chapter 7: Characterizing the *In-Vitro* Mechanical Properties of the Facet Joint Capsule Ligament

7.1 Overview

Study Design: An *in vitro* biomechanics study exposing the facet joint capsule ligament (FCL) tissue to sub-maximal, cyclic uniaxial tensile loading.

Background: The FCL is a structure in the lumbar spine that constrains motions of the vertebrae. Previous work has demonstrated that under physiological motion the FCL is subjected to significant deformation with FCL strains increasing in magnitude with increasing flexion and extension moments. Thus, it is important to characterize the mechanical response of the FCL for investigations into injury mechanisms. Damaged tissue behaves differently from healthy tissue. Sub failure loads can produce micro-damage resulting in increased laxity, decreased stiffness and altered viscoelastic responses. Thus, the objective of this investigation was to determine the mechanical and viscoelastic properties of the FCL under various magnitudes of strain from control samples and samples that had been through an impact protocol.

Objectives: The purpose of this investigation was to quantify the mechanical properties and viscoelastic response of control and impacted FCL.

Methods: 200 tissue samples were excised from the right and left FCL of 80 porcine cervical functional spinal units (FSU's). Tissue samples were excised from FSU's obtained from **Study 4**. Twenty FCL tissue samples served as the control group. The remaining 180 FCL tissue samples were randomly obtained from FSU's that had been exposed to one of nine conditions (impacted tissue). These conditions included three different severity impacts (4g, 8g and 11g) and three levels of posture during the impact (Flexion, Extension and Neutral) (**Study 4**). Each specimen was loaded uniaxially, collinear with the primary fiber orientation. The loading protocol was identical for all specimens: preconditioning with 5 cycles of loading/unloading to 5% strain, followed by a 30 second rest period, 5 cycles of 10% strain and 1 cycle of 10% strain with a hold duration at 10% strain for 240 seconds (4 minutes). The same protocol followed for 30% (cyclic-30% & 30%-hold) and 50% strain (cyclic-50% & 50%-hold). All loading and unloading were performed at a rate of 2%/sec. All impacted FCL properties were compared back to controls. Measures of stiffness, hysteresis and force-relaxation were computed for the 30% and 50% strain conditions.

Results: No significant differences in stiffness were observed for impacted specimens in comparison to control (30% Control = 2.64 N/mm; 4 g = 2.20 N/mm, 8 g = 2.07 N/mm, 11 g =

2.16 N/mm) (50% Control = 5.06 N/mm; 4g = 4.60 N/mm, 8 g = 4.07 N/mm, 11 g =4.64 N/mm). Impacted specimens from the 8g Flexed and 16 g Flexed and Neutral conditions exhibited greater hysteresis during the cyclic-30% and cyclic-50%, in comparison to controls. In addition, specimens from the 8g and 16g Flexed conditions resulted in greater stress decay for the 50%-hold conditions.

Conclusions: Results from this study demonstrate viscoelastic changes in FCL samples exposed to moderate and highspeed impacts in the flexed posture. However, it is interesting that these viscoelastic changes were not accompanied by changes in stiffness. Findings from this investigation provide novel insight and provide mechanical and viscoelastic properties of the FCL both in control and impacted scenarios. Results suggest that the FCL is not exposed to sub failure loading during low to moderate severity impacts.

Keywords: facet joint capsule ligament, uniaxial tension, force-relaxation,

7.2 Introduction

The facet joint capsule ligament (FCL) is a structure in the lumbar spine that constrains motions of the intervertebral joint. It is innervated by mechanically sensitive neurons, sensitive to capsular ligament stretch, and is a recognized source of low back pain (Cavanaugh et al., 2006). Previous work has demonstrated that under physiological motion the FCL is subjected to deformation (Panjabi et al., 1982) with multidirectional facet capsular strains increasing in magnitude with increasing flexion and extension moments (Ianuzzi et al., 2004; Zehr et al., 2019). Thus, it is important to accurately characterize the mechanical response of the FCL for investigations into injury mechanisms. Damaged tissue may behave differently from healthy tissue, with sub failure loads potentially producing micro-damage. The first step in quantifying microdamage and potential injury in the FCL is to establish the mechanical properties for healthy tissue. Thus, the objective of this investigation was to determine the mechanical properties of the FCL in healthy tissue and investigate the viscoelastic response of the FCL under various magnitudes of strain. A secondary objective was to compare potentially injured FCL tissue, which has been exposed to an impact protocol, to healthy tissue properties in attempt to quantify possible FCL microdamage.

In comparison to the mechanical properties of healthy tissue, tissue that has been exposed to mechanical trauma may behave quite differently. Many studies have demonstrated that the mechanical properties of ligaments can be altered by sub failure injury. To date, tissue injury resulting from mechanical trauma has traditionally been defined by gross measures of mechanical failure or visible rupture. Sub failure loads can produce micro-damage to a tissue, which can result in a variety of altered mechanical properties in ligaments. These properties include, increased laxity (Panjabi et al., 1996; Pollock et al., 2000; Provenzano et al., 2002) altered stiffness (Panjabi et al., 1999; Provenzano et al., 2002; Quinn et al., 2007; Quinn and Winkelstein, 2007) and altered viscoelastic responses (Nelson-Wong et al., 2018; Panjabi et al., 1999). Such responses can be coupled with collagen disorganization, fibroblast necrosis and nociceptor activation. For example, exposure to vibration has been shown to influence the mechanical properties of annulus fibrosus tissue long before the initiation of disc herniation (Gregory and Callaghan, 2010). Vibrated annulus tissue displayed a larger toe region, hypothesized to be due to damaged elastin fibers. In porcine thoracolumbar fascia tissue, fascia from injured pigs were found to have greater stiffness, less energy dissipation, and less stress decay compared to tissues from healthy control pigs (Nelson-Wong et al., 2018). In rat facet joint capsular ligament, the structural response and collagen fiber

organization have been demonstrated to be altered at sub-failure loading conditions (Quinn et al., 2007). Capsular ligaments displayed significant mechanical changes in laxity and stiffness under sub-failure tensile loading (Quinn et al., 2007). These findings demonstrate a link to sub failure loading conditions and altered joint mechanics.

Therefore, the primary objective of this study was to measure the mechanical properties and observe the viscoelastic response of excised FCL from porcine functional spinal units (FSU's) under physiological tensile loading. A secondary objective was to identify the effect of exposure to a sudden impact on mechanical properties and viscoelastic responses of excised FCL and compare changes relative to un-impacted tissue. In line with these objectives, it was hypothesized that changes in mechanical and viscoelastic response would be observed in specimens exposed to a sudden impact. A secondary hypothesis was that these changes in response would be greater in those specimens exposed to a higher impact severity.

7.3 Methods

7.3.1 Study Design

An *in vitro* study exposing excised FCL to sub-maximal, cyclic uniaxial tensile loading comparing the mechanical properties of control FCL tissue samples to "impacted" FCL tissue samples.

7.3.2 Tissue Preparation

Two-hundred (200) FCL samples were excised from the left and right facet joint capsules of 80 porcine cervical FSU's (40 C34, 40 C56). These were obtained immediately following collection of **Study 4.** Porcine cervical FSUs were used as surrogates for the human lumbar spine due to the anatomical and functional similarities (Oxland et al., 1991; Yingling et al., 1999). The porcine model provides greater control over potential confounding factors such as age, nutrition and physical activity that can impact the mechanical integrity of the tissues surrounding the IVD. The quality of the IVD was assessed using the grading scale outlined by Galante (1967). Only specimens with disc quality of Grade 1 will be used for this investigation.

Briefly, from **Study 4**, the cervical spines of 80 porcine FSU specimens were obtained following death and stored at -20^{o}C. Specimens were thawed at room temperature for a minimum of 12 hours. Dissection of the cervical spine included isolating the two FSUs (C34 & C56) and removing the surrounding musculature leaving the osteoligamentous structure intact. All specimens were exposed to a 15 minute, 300 Newton, preload to reduce postmortem swelling and passive Flexion-extension testing and Anterior-Posterior shear testing (**Study 4**). Eight FSUs (4

C34, 4 C56) served as a control group, and only underwent the 15-minute preload as well as passive flexion extension and shear testing. The remaining 72 were randomized into one of nine conditions (impacted tissue). These conditions included three different severity impacts (4g, 8g and 11g) and three levels of posture during the impact (Flexion, Extension and Neutral) (**Study 4**). To prevent specimen dehydration throughout testing all FSUs were misted with a saline solution approximately every 15 minutes.

After testing was completed, tissue samples were harvested from the left and right facet joint capsule (Figure 7.1). Each condition (10 conditions, including control), consisted of 20 facet joint capsule specimens (10 C34 and 10 C56 and 5 right and 5 left within each level). Prior to testing, the extracted ligaments were reduced to 5 x 2 mm sections. The cross-sectional area of each tissue sample was measured using a 2D laser displacement sensor (LJ-V7080, Keyence Corporation, Osaka, Japan).

7.3.3 Mechanical Testing

Each FCL sample was mounted in a BioTester loading system (Cellscale, Waterloo, ON), a commercial apparatus designed to apply tensile loads to biological tissues (Figure 7.1). The apparatus secured the biological tissue using stainless steel clamps which were used to apply uniaxial tension to the tissue samples (Figure 7.1).

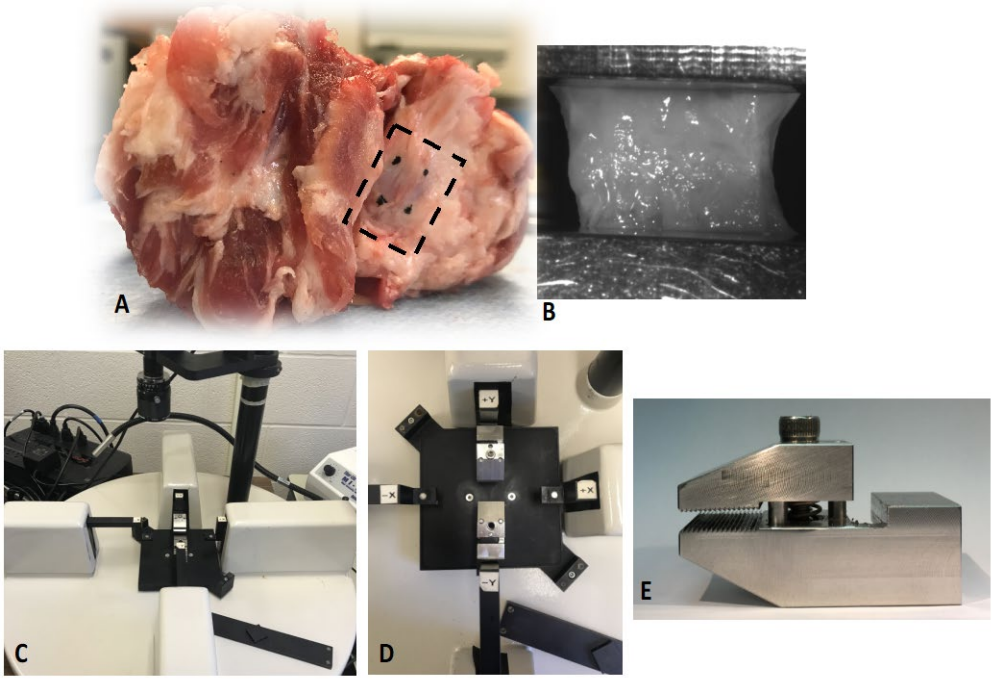

Figure 7.1: (A) Sample image of the area harvested from the left and right facet joint capsule. (B) Sample image of 5 x 2 mm FJC section mounted in stainless steel clamps. (C) BioTester loading system used in this experiment. (D) Stainless steel clamps interfaced and aligned with y-axis of the BioTester loading system. (E) Closeup of stainless steel clamp.

Tensile load was applied to the FCL tissue using linear actuators connected in series with a 10 N load cell. Force and actuator displacement were continuously collected at 10 Hz throughout all testing.

Once the tissue was mounted and secured within the testing device, the sample was preloaded to 10 mN (Little and Khalsa, 2005b). Each sample was mounted such that the sample was positioned with the predominate orientation of the fiber direction along the axis of pull. Next, each sample was pre-conditioned with five cycles to a maximum of loading/unloading to a 5% strain ratio at a rate of 2%/s (Gregory and Callaghan, 2012; Little and Khalsa, 2005b; Nelson-Wong et al., 2018). Following preconditioning, samples were returned to the zero position for a 60 sec rest period (Nelson-Wong et al., 2018).

The testing protocol consisted of cyclic loading/unloading and stress relaxation testing at increasing strain increments: 10%, 30% and 50%. This was based on previous work that observed approximately 10% strain at 3 degrees of flexion for the L4/L5 joint (Panjabi et al., 1982). L4/L5 intervertebral flexion ranges between 13.4 to 17.7 degrees in seated postures (De Carvalho and Callaghan, 2012). For each test the sample was strained at 2%/s, with one minute of rest in between

each condition. Force and displacement were measured at 30 Hz. The testing began with 5 cycles of 10% strain, followed by 1 cycle of 10% strain with a hold duration at 10% strain for 240 seconds. The same followed for 30% and 50% strain, with repeated cycles and one strain cycle being held for 240 seconds (Figure 7.2) (Little and Khalsa, 2005b).

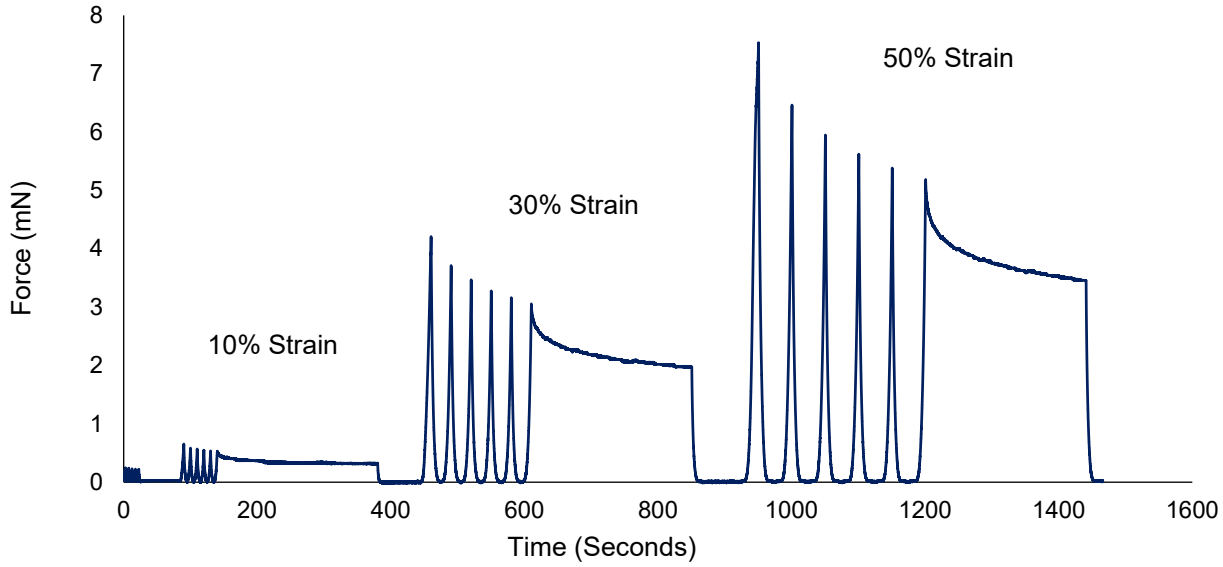

Figure 7.2: Representative raw force data of a control sample depicting all components of testing (preloading and protocol).

7.3.4 Data Analysis

Force and displacements were synchronized during collection and time-varying force and actuator displacement data were processed in Matlab (version R2015a, Mathworks, Natick, MA). Signal noise was attenuated using a fourth-order, zero-lag, low-pass Butterworth filter with a cutoff frequency of 3 Hz (Nelson-Wong et al., 2018). Due to noise issues with the force transducer only the 30% and 50% cycles were analyzed. All results were plotted and analyzed as force-displacement curves. A sample force-displacement curve depicting the cycles used for analysis is available in Figure 7.3.

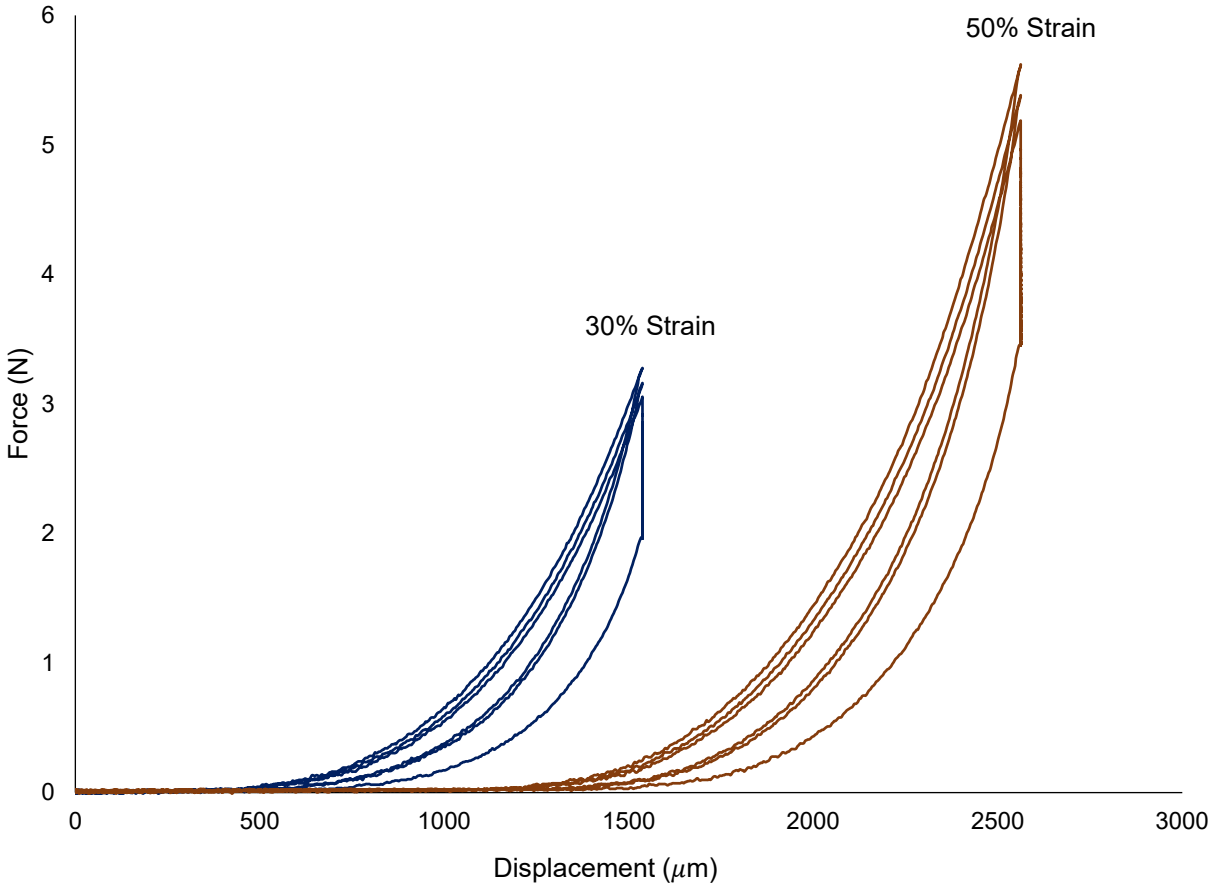

Figure 7.3: Representative raw force data of a control sample depicting all components of testing (preloading and protocol).

7.3.4.1 Measure of Stiffness

Stiffness of the linear loading region was computed as the slope of the linear region of the force-displacement curve, consistent with previous work (Dumas et al., 1987; Mattucci et al., 2012; Yoganandan et al., 1989). Stiffness values were averaged from the fourth and fifth cycles as loading responses were most stable during these cycles. The linear region was defined starting at the second transition point of a trilinear curve fit to the loading region separating the curve into toe and linear regions (Figure 7.4). Previous work has used a similar approach (bilinear curve) for repeatably in defining the linear region (Chandrashekar et al., 2008; Elliott and Setton, 2001; Lynch et al., 2003; Mattucci et al., 2012). In the previously mentioned work, all specimens were taken to failure, thus obtaining a significantly greater portion of the linear loading region. Because the current work did not fail specimens, the specimen was brought through a smaller portion of the linear loading region and as a result a bilinear curve did not provide a sufficient fit. Pilot work

demonstrated that a trilinear fit effectively separated the curve into toe and linear regions for estimates of stiffness to be obtained.

7.3.4.2 Hysteresis During Cyclic Loading

Hysteresis was represented as the energy dissipated in the load-unload cycle and was calculated as the difference between loading and unloading energy, expressed as a percentage of loading energy (Nelson-Wong et al., 2018)(Figure 7.4).

7.3.4.3 Force Relaxation Response

Relaxation Response was calculated as the ratio of the force at the end of force-relaxation test to the peak force measured at the beginning of the test. This value was expressed as the percentage of peak force for both the 30% (30%-hold) and 50% (50%-hold) (Nelson-Wong et al., 2018)(Figure 7.4).

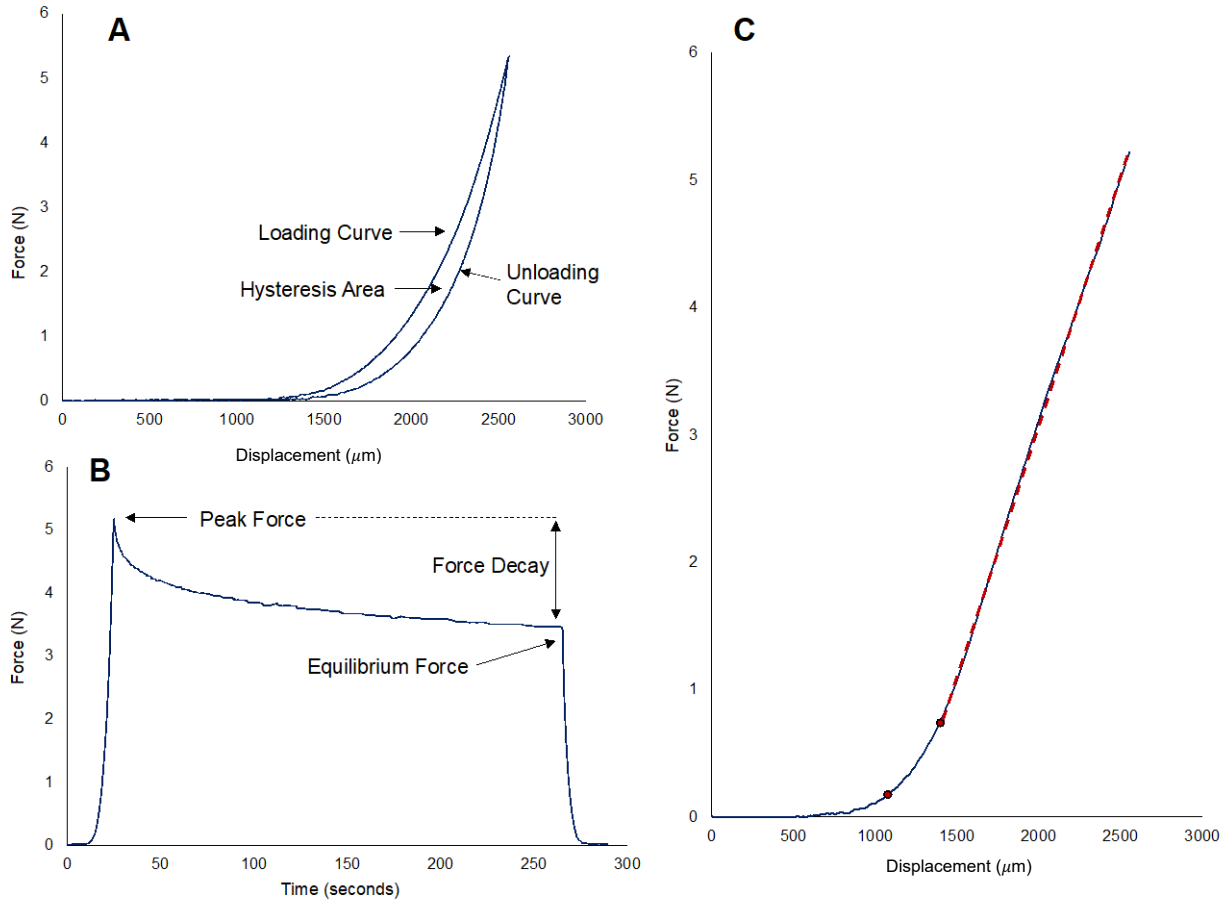

Figure 7.4: Representative data for one control sample for the 50% strain condition. A - Loading and unloading curve for one 50% strain cycle. B - Force-relaxation curve for one 50%-hold test.

C – One 50% strain cycle depicting the 2 breakpoints chosen for the trilinear fit as well as the linear loading region used to compute stiffness for this particular cycle.

7.3.5 Statistical Analysis

To determine if the mechanical properties of excised FCL from impacted specimens were significantly different from control specimens Dunnett's test was completed for all computed mechanical properties. Mechanical properties of stiffness (30% and 50%), hysteresis (30% and 50%) and stress-relaxation ratio (30% and 50%) of impacted specimens were compared back to the control group. In total 9 separate groups (Impact x Posture) were compared to the control group. Lastly, a 2-way general linear model was completed (with factors of impact and posture) on impacted specimens only, to determine if significant differences in mechanical properties existed across Impact or Posture groups. For all statistical tests an alpha level of .05 (SPSS Inc., Chicago, IL) was used, Bonferroni adjustments were completed when appropriate.

7.4 Results

No significant differences were observed across impact or posture groups, thus for the remainder of the document all results will pertain to comparisons of mechanical properties within an impact-posture group back to the control group.

7.4.1 Stiffness

No significant differences in stiffness were observed for the 30% or 50% strain conditions in comparison to control groups (Table 7.1). In general, there was a trend for impacted specimens to have a slight decrease in estimates stiffness in comparison to control (30% Control = 2.64 N/mm; 4 g = 2.20 N/mm, 8 g = 2.07 N/mm, 16 g = 2.16 N/mm)(50% Control = 5.06 N/mm; 4g = 4.60 N/mm, 8 g = 4.07 N/mm, 16 g =4.64 N/mm).

Table 7.1: Average stiffness for the final 2 cycles of the 30% and 50% strain tests

	Control	Impact Level (g)	Extension	Flexion	Neutral
30% Stiffness (N/mm)	2.64	4	2.71	1.59	2.33
		8	1.50	2.61	2.17
		11	2.19	2.31	2.07
30% Stiffness Standard Deviation	1.44	4	1.17	0.95	1.18
		8	1.01	1.31	1.21
		11	0.99	1.75	1.10
50% Stiffness (N/mm)	5.06	4	5.75	2.95	4.80
		8	3.13	5.44	3.89

		11	4.52	4.45	4.83
50% Stiffness Standard Deviation	2.78	4	3.72	1.45	2.47
		8	2.07	2.43	1.79
		11	1.47	3.33	2.97

7.4.2 Hysteresis During Cyclic Loading

Significantly greater hysteresis was observed in impacted specimens, during the cyclic-30% cycles, in comparison to the control group ($p = 0.026$). Specifically, 8g-Flexion ($p = 0.014$), 16g-Flexion ($p = 0.012$) and 11 g-Neutral ($p = 0.021$) conditions were greater in comparison to control (Figure 7.5).

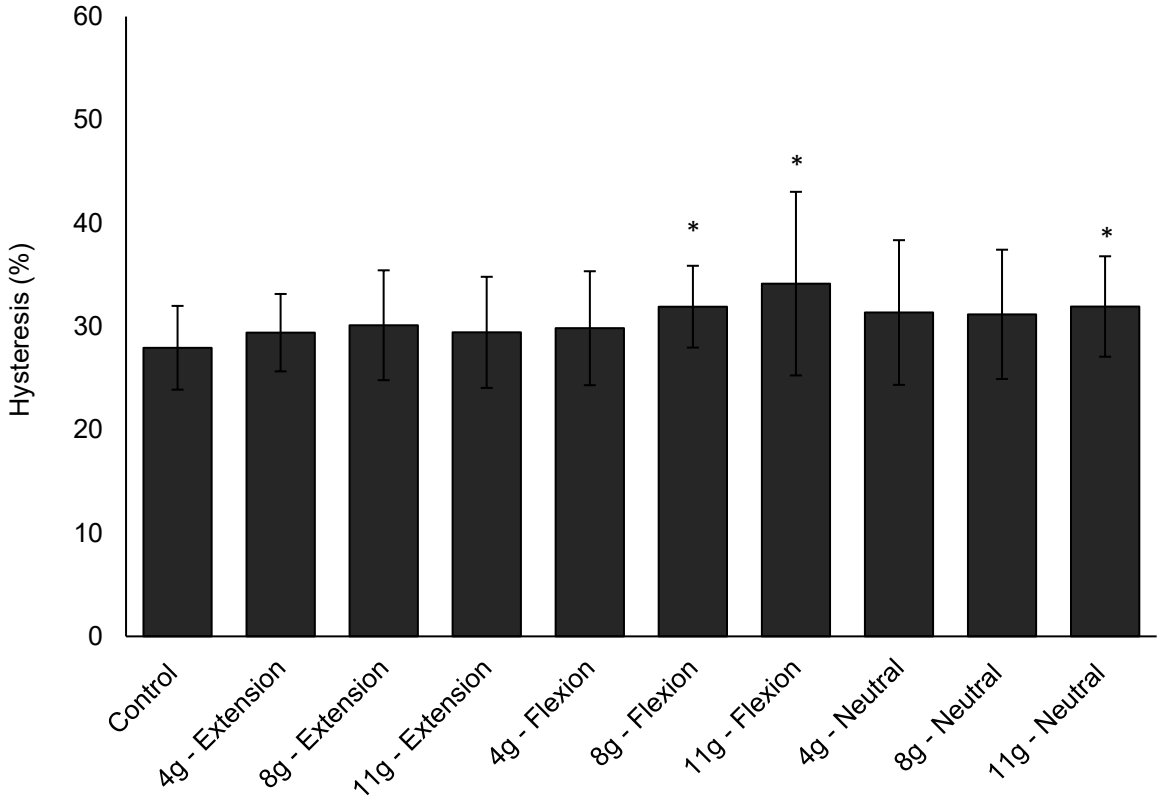

Figure 7.5: Average Hysteresis for cyclic 30% testing. Standard Deviations are displayed along with statistically different means.

Greater hysteresis was observed in impacted specimens, during the cyclic-50% cycles, in comparison to the control group ($p > 0.001$). Specifically, 8g-Flexion ($p = 0.044$), 16g-Flexion (p

= *0.026*) 8g-Neutral (*p =0.021*) and 16g-Neutral (*p = 0.020*) conditions were statistically greater in comparison to the control (Figure 7.6).

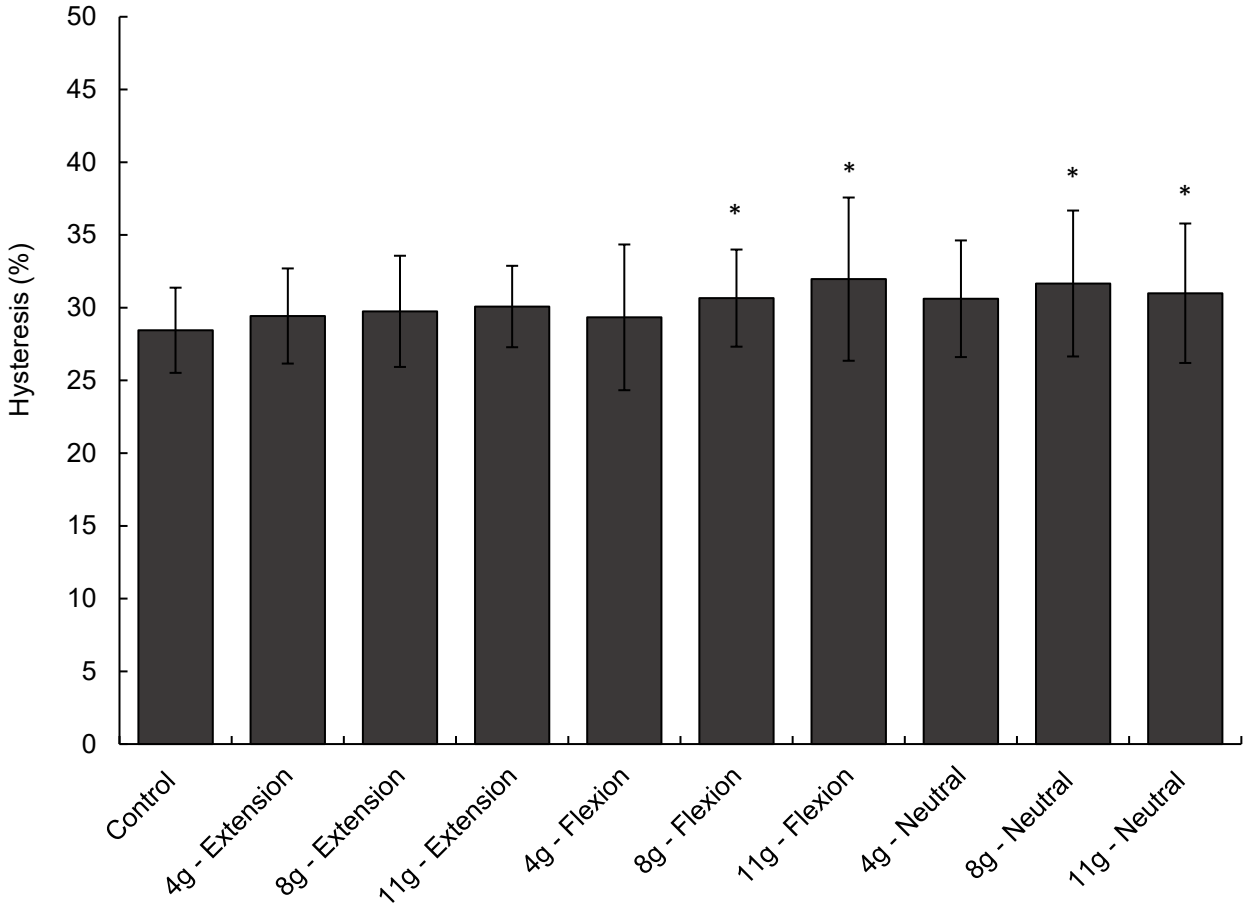

Figure 7.6: Average Hysteresis for cyclic 50% testing. Standard Deviations are displayed along with statistically different means.

7.4.3 Force Relaxation Response

No significant differences were observed for the 30%-hold condition (*p = 0.643*) (Figure 7.7). For the 50%-hold condition significant differences were observed in comparison to control (*p = 0.026*). Specifically, 8g-Flexion (*p = 0.014*), 16g-Flexion (*p = 0.005*) conditions resulted in statistically greater force decay in comparison to control (Figure 7.8).

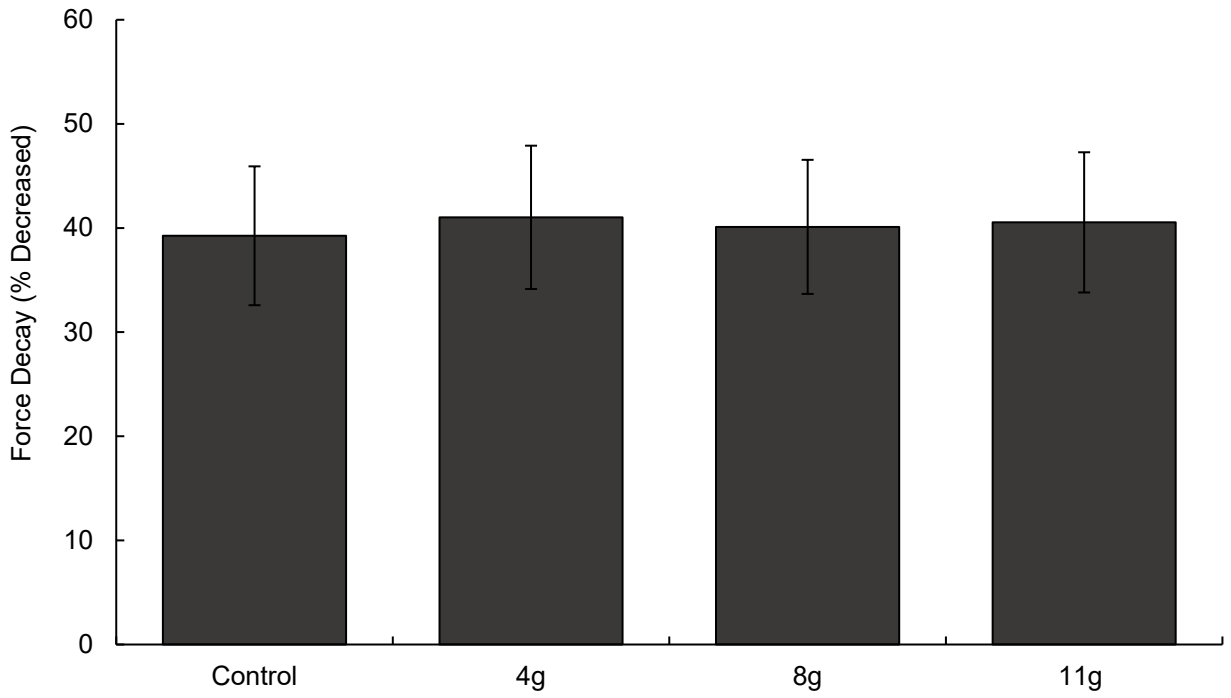

Figure 7.7: Average force decay for the 30%-hold condition for control and impacted specimens (collapsed across posture). Standard deviation bars are displayed.

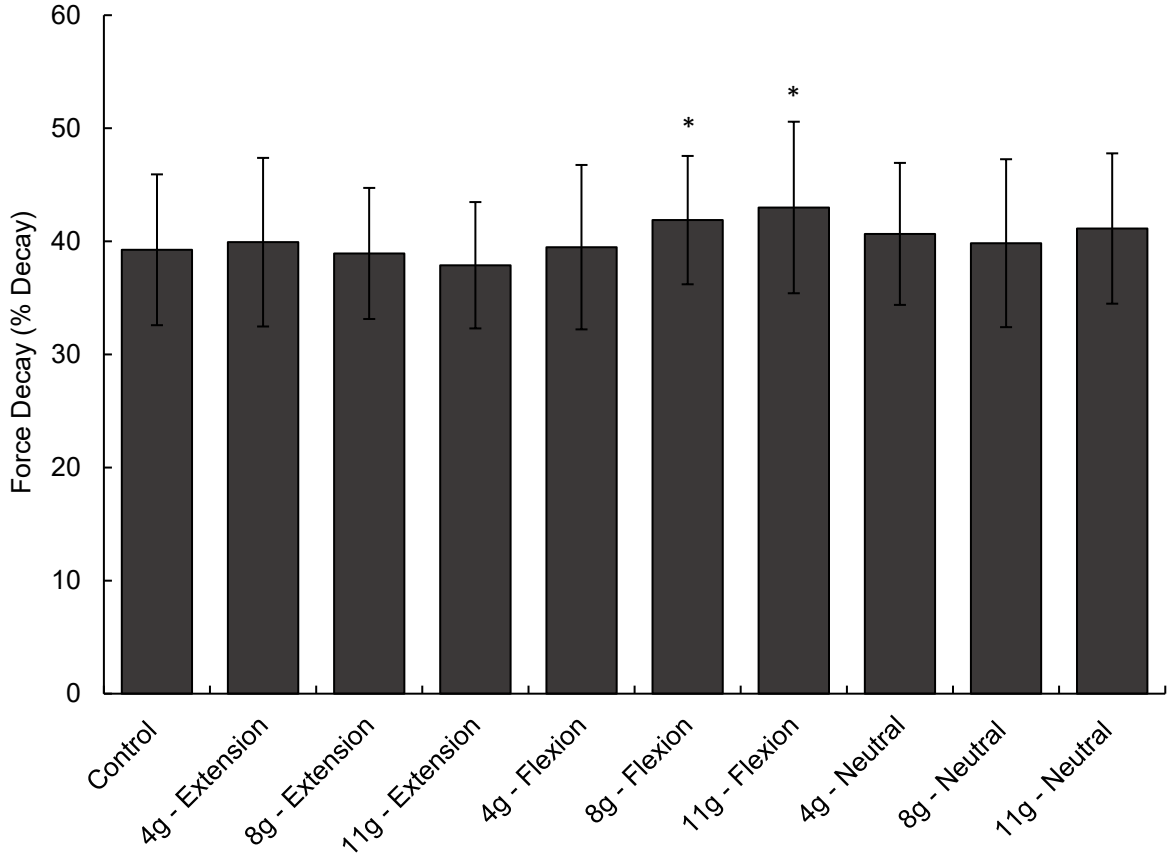

Figure 7.8: Average force decay for the 50%-hold conditions for control and impacted specimens. Standard deviation bars are displayed and statistically different means.

7.5 Discussion

In line with our hypothesis, this study observed significant differences in viscoelastic response in FCL's from impacted specimens in comparison to control. Specifically, a trend was observed displaying increases in hysteresis and force-relaxation across the higher impact conditions in Flexion. However, contrary to our hypothesis, despite a trend in altered viscoelastic response, no significant differences in stiffness were observed across impact groups in comparison to controls.

FCL samples taken from impacted specimens were not found to have significantly different estimates of stiffness in comparison to control. This result indicates that the primary fibers within the FCL were likely not significantly damaged or exposed to sub failure loading across the impact conditions tested. Changes in ligament stiffness have frequently been used as a parameter to identify potential micro-damage within a tissue, suggesting potential micro-damage to the collagen

fibres or fibre cross-links and/or realignment of the collagen fibers within the ligament (Panjabi et al., 1999; Provenzano et al., 2002; Quinn et al., 2007; Quinn and Winkelstein, 2007). When looking at investigations specific to the FCL, previous work by Quinn and colleagues (2007) assessed the effect of sub failure loading on the structural response of the FCL. Specimens were exposed to a tensile loading protocol that lengthened FCL's to corresponding physiological and sub failure magnitudes, with stiffness and joint laxity measures following each distraction. Results demonstrated significantly greater laxity and a significantly greater decrease in stiffness for sub failure distractions in comparison to physiological (Quinn et al., 2007). Polarized light microscopy also revealed an increase in collagen fiber angular deviation (indicating increased collagen fiber disorganization) following sub failure loading in comparison to physiological (Quinn et al., 2007). Quinn and colleagues (2007) concluded that sub failure loading conditions are associated with altered joint mechanics and collagen fiber disorganization which implies ligament damage. Panjabi and colleagues (1996), also documented significant changes in rabbit anterior cruciate ligament load deformation curves following an 80% sub failure stretch injury. Significant increases in deformation were observed post injury in the lower portion of the load-deformation curve. No significant differences in failure parameters (load, deformation or total energy) were observed (Panjabi et al., 1996). Lastly, Nelson-Wong and colleagues (2018) observed differences in thoracolumbar fascia stiffness in a living porcine model. Thoracolumbar fascia extracted from pigs that were exposed to a sub failure injury exhibited greater thoracolumbar fascia stiffness in comparison to controls (Nelson-Wong et al., 2018). This increase in stiffness was likely due to the fact that a live animal model was used and biological changes such as chronic inflammation and/or biochemical changes may have influenced stiffness properties. Because stiffness was not significantly influenced across impact conditions and taken with the small strains observed during the impacts in **Study 4**, it is unlikely that the impacts damaged the primary collagen fibres in the FCL. While this current investigation did not use high resolution microscopy to track FCL fibre organization or damage, previously mentioned work shows a consistent trend in altered mechanical properties following known sub failure injury exposures.

This investigation found significant changes in hysteresis (energy dissipation) in impacted FCL in comparison to controls. Specimens in the 8g-Flexion and 11g-Flexion groups exhibited greater hysteresis (i.e. greater energy dissipation between loading and unloading curves) in both the 30% and 50% strain conditions. Specimens in the 8g and 11g, Flexion and Neutral groups,

exhibited greater hysteresis at the 50% strain condition. Contrary to typical tissue mechanical properties such as stiffness and laxity, changes in hysteresis in a tissue exposed to a prior sub failure injury have been significantly less studied. To the authors' knowledge only one study has specifically looked at hysteresis properties following a sub failure type injury. In addition to stiffness, Nelson-Wong and colleagues (2018) also found that hysteresis changes were a sensitive measure for thoracolumbar fascia injury. Injured thoracolumbar fascia resulted in significantly less energy dissipation in comparison to controls (Nelson-Wong et al., 2018). This result was attributed to biological changes (such as changes in morphological and biochemical factors) with higher collagen content and larger fibril diameter being observed during aging and wound healing (Shadwick, 1990). Because this current study did not use a live animal model, such changes in FCL structure would be impossible. Panjabi and colleagues (1996) hypothesized that increased energy loss during the load/unload cycle within a ligament may, in part, be due to damage to the crosslinking fibers. It is possible that the higher impact conditions resulted in altered and/or damaged crosslink fibers, with no damage to the predominate collagen fibres. Gregory and Callaghan (2012) observed changes in toe-region length in excised single annular lamellae exposed to vibration, with no changes in elastic modulus, initial failure force, ultimate tensile strength and ultimate stretch ratio (Gregory and Callaghan, 2012). It was hypothesized that vibration resulted in damage to the secondary structures including the dense fibrous connections that can exist between adjacent collagen fibres. Thus, it is possible that the higher severity and flexed impact conditions may have altered and/or damaged secondary structures within the FCL resulting in preliminary damage to such structures and altered energy dissipation responses during cyclic tensile testing.

In addition to greater energy dissipation, this study also observed greater force-decay during the 50% force-relaxation test for the 8g and 11g flexion groups in comparison to control. Similar to hysteresis measures, very limited work has investigated changes in stress-relaxation response of a tissue in response to a sub failure injury. Of the limited investigations available for comparison, Panjabi and colleagues (2000, 2001) examined the influence of a sub failure injury and highspeed sub failure injury on the viscoelastic response of rabbit anterior cruciate ligament during a force-relaxation test (held for 180 seconds). It was found that an 80% sub failure stretch injury (80% of the failure deformation of control) for both highspeed and non-highspeed, resulted in at least a 50% decrease in relaxation force at all time points along the relaxation curve. However,

based on an approximated ratio of total percent force decay, the percentage of total force decay throughout the relaxation protocol remained less changed. Total force decay was approximately 22% for the un-injured ligaments and then dropped to 17% and 14% following an 80% standard stretch and highspeed stretch respectively. It is unknown why greater force-decay was observed during the 50% force-relaxation test for the 8g and 11g flexion groups. Similar to changes in hysteresis, it is possible that creep related changes and/or damage to secondary structures occurred in the FCL during impact testing, particularly in the flexed posture, and such changes altered the force-relaxation response of the FCL during the 50% force-relaxation test.

To the best of the author's knowledge, this investigation represents one of the first studies to characterize the mechanical properties and viscoelastic response of the FCL, both in control and impacted scenarios. However, there are a number of potential limitations that should be addressed. First, a porcine animal was used and may not be directly comparable to humans. The porcine animal model allows for control of factors such as diet, age and activity level that would otherwise not be possible to control in a human population and may confound comparisons across groups. Further investigations have demonstrated that the cervical porcine spine is a good anatomical and functional match to human lumbar spines (Oxland et al., 1991; Yingling et al., 1999). Therefore, it is reasonable to expect that mechanical similarities would exist between the two models. Second, this study only completed tensile testing in the primary fiber direction, thus it is unknown if the tested properties would provide a more sensitive measure for injury perpendicular to the primary fiber orientation. Results from this investigation suggest that the primary fibres within the FCL were not damaged and thus future work could investigate damage to possible crosslinking fibers as a potential avenue for microdamage within the FCL. Lastly, the tissue examined in the current study was obtained post-mortem and therefore does not consider biological responses with which a living body may respond. Thus, it is unknown if the changes observed in the FCL were viscoelastic (i.e. creep) in nature and may have returned to baseline if recovery time was permitted.

7.6 Conclusions

Results from this investigation demonstrate significant changes in the FCL viscoelastic response following a moderate to high velocity impacts in the flexed posture. Conversely, no significant changes in FCL stiffness were observed in comparison to controls, indicating that the FCL did not to undergo injurious or permanent mechanical changes during the simulated impacts. These findings provide novel insight into FCL responses following a sudden impact. Future research

should examine damage to possible crosslinking fibers within the FCL as a possible source of altered viscoelastic response.

Chapter 8: Synthesis of Contributions

8.1 Thesis Summary

The global objective of this thesis was to investigate low velocity rear-end impacts as a potential injury mechanism in the lumbar spine. In particular, the four studies included in this thesis were conducted to:

1. To characterize and document the dynamics of low velocity motor vehicle collisions that result in low back injury claims (**Study 1**).
2. To establish an understanding of the forces and motions in the low back from low velocity motor vehicle rear impact vehicle collisions and their relationship to low back injury potential (**Study 2, 3 & 4**).
3. To explore passive spine stiffness changes following simulated motor vehicle rear-end collisions (**Study 3 & 4**).
4. To explore the contribution of potential facet joint injury and damage to low back injury after a sudden impact (**Study 4 & 5**).
5. To investigate the mechanical properties of the FCL and the interacting effects impact severity and posture have on the mechanical properties of the ligament (**Study 5**)

Study one provided insight into the types of low velocity collisions that frequently result in claims of low back pain (LBP). Using database from a forensic engineering firm based in Toronto, Ontario, Canada, data was obtained to evaluate the injuries sustained in passenger vehicle to vehicle collisions, with a collision severity of 24 km/hour or less. Each identified case was reviewed for collision characteristics, pre-existing medical conditions and injuries claimed. Descriptive statistics across low back injury claims were computed. Results from study one indicated that relationships do exist between LBP reporting, collision dynamics and occupant characteristics. With regards to collision characteristics, it was found that a high proportion of LBP claims resulted from rear-impact collision configurations with collision severities of 13 km/hour or less. For occupants, it was found that a history of LBP reporting or evidence of lumbar disc degeneration was most commonly associated with LBP reporting following a low to moderate speed collision. The results from this investigation provided knowledge of collision characteristics

associated with LBP reporting that were employed in the remaining thesis studies in attempt to explore possible mechanisms for low back injury in low speed motor vehicle accidents.

Study two explored differences in trunk muscle activation magnitudes and timing in human volunteers during simulated rear impacts during braced and unanticipated impacts. Findings demonstrated that during a laboratory-simulation of an unanticipated 4 km/hour rear-impact collision, the peak activation of muscles in the lumbar spine are low in magnitude. In addition, a significant delay was observed between peak muscle activity and peak acceleration of the lumbar spine, which indicates that muscular contractions are too slow to resist any initial impact forces, unless they are recruited (braced) before impact. As such, muscle activation likely has minimal contribution to the internal joint loads that are experienced in the lumbar intervertebral joints during low speed rear impact collisions. Findings from this study encourage the use of simplified joint models in estimating the joint loads in the lumbar spine during low speed rear impact collisions and support the application of cadaveric and anthropomorphic test device (ATD) testing in understanding the resultant joint loads in the lumbar spine associated with rear-end collisions.

Study three explored peak lumbar kinematics and joint reaction forces in human volunteers during simulated rear impact collisions with and without the use of a lumbar support. In addition, this investigation evaluated lumped lumbar spine passive stiffness changes and low back pain reporting following such collisions. Findings demonstrated that during a laboratory-simulation of an unanticipated 8 km/hour rear-impact collision, young healthy adults did not develop LBP over a 24 hour follow up period. Significant changes in lumped passive lumbar spine stiffness, specifically a decrease in stiffness, were observed in the low stiffness zone for flexion and extension. Lumbar support did not significantly influence peak lumbar kinematics and joint reaction forces. This investigation represents the first experiment to characterize the *in vivo* mechanical exposures to the lumbar spine during simulated low-speed rear impacts.

Study four explored the combined effects of impact severity and posture on porcine cervical FSUs by characterizing the mechanical exposures and potential soft tissues disturbances in response to sub failure impacts. Impacts were applied using a custom-built impact track, which simulated impact parameters similar to those experienced in low to moderate speed motor vehicle collisions (4g, 8g and 11g). All FSUs were impacted, passive Flexion-extension and shear range of motion testing were completed immediately prior to and immediately post impact. Vertebral horizontal translation and facet joint capsule (FCL) strain were measured during impacts. FSUs

exposed to the highest severity impact (11g) had significant neutral zone changes, with increases in joint laxity in flexion-extension and shear testing and decreased stiffness. This investigation observed a significant effect of collision severity on peak vertebral translation, with increasing horizontal displacement as impact severity increased and a subsequent increase in peak FCL shear strain as impact severity increased. Despite the main effects of impact severity all horizontal translations were well below previously measured ultimate shear failure displacements and all observed FCL strains were very low in magnitude (below 6% strain). Across impact severity, no influence of posture was observed. The lack of influence of posture and small facet capsule strain quantities observed across impact severities tested, suggest that the facet joint capsule does not appear to undergo injurious or permanent mechanical changes. Based on the significant neutral zone changes post impact for the 11g impacts, results suggest that soft tissue injury may have occurred during the highest severity impact, however, results do not suggest injury to the facet capsule ligament.

Using the same cohort of porcine FSUs, study five characterized the mechanical properties and viscoelastic response of excised facet capsule ligament (FCL) from porcine FSUs that had been through the impact protocol in Study 4. Specifically, 180 FCL tissue samples were randomly obtained from FSUs that had been exposed to one of nine impact conditions in Study 4. Each specimen was loaded uniaxially, collinear with the primary fiber orientation. The loading protocol was identical for all specimens: preconditioning with 5 cycles of loading/unloading to 5% strain, followed by a 30 second rest period, 5 cycles of 10% strain and 1 cycle of 10% strain with a hold duration at 10% strain for 240 seconds (4 minutes). The same protocol followed for 30% (cyclic-30% & 30%-hold) and 50% strain (cyclic-50% & 50%-hold). All loading and unloading were performed at a rate of 2%/sec. All impacted FCL properties were compared back to controls. Overall, no significant changes in FCL stiffness were observed in comparison to controls. However, a trend was observed displaying increases in hysteresis and force-relaxation across the higher impact conditions in Flexion. Results from this investigation demonstrated significant changes in the FCL viscoelastic response following a moderate to high speed impact in the flexed posture. However, no significant changes in FCL stiffness were observed in comparison to controls, indicating that the FCL was not exposed injurious or permanent mechanical changes during the simulated impacts. These findings provide novel insight into FCL responses following

a sudden impact and together with the results from Study 4 suggest that the FCL is likely not responsible for low back pain generation after low velocity automobile collisions.

8.2 Hypotheses Revisited

As outlined in section 1.5, the general purpose of this thesis was to improve our understanding of underlying low back injury mechanisms that may result from low to moderate speed rear impacts given the high rate of LBP reporting in these accidents. Four studies aimed at eliciting these injury mechanisms, focussed on expanding the knowledge of collision characteristics that result in low back injury reporting and characterizing the *in vivo* and *in vitro* mechanical exposures to the lumbar spine during simulated rear impact collisions, were completed. Collectively, the findings from this thesis do not support that the facet joint capsule is responsible for low back pain generation after low velocity automobile collisions and that the forces experienced by the lumbar spine are below magnitudes associated with acute failure. High severity impacts did alter the mechanical response of functional spinal units post impact, resulting in increased neutral zone range and stiffness. This suggests that LBP reporting following such impacts may be related to irritation and/or injury to the IVD.

i. There will be no difference in reports of low back injury claims across low velocity collision severities (up to 25 km/hour) (**Study 1**).
   DECISION: The null hypothesis is rejected.
   Significantly greater low back injury reports were documented for collision severities under 13 km/hour

ii. There will be no difference in reports of low back injury claims across collision types (**Study 1**).
   DECISION: The null hypothesis is rejected.
   Significantly greater low back injury reports were documented for the rear-end collision configuration.

iii. There will be no change in peak muscle activation across collision conditions (**Study 2**).
   DECISION: The null hypothesis is rejected

Greater peak muscle activations were observed in the latissimus dorsi muscle group during the braced impact in comparison to the unanticipated impact.

iv. There will be no change peak lumbar accelerations across impact conditions (**Study 2**).
DECISION: The null hypothesis is accepted.
No significant differences in peak lumbar accelerations were observed between unanticipated and braced impact conditions.

v. There will be no change in lumbar spine kinetics across seating conditions (**Study 3**).
DECISION: The null hypothesis is accepted
No significant differences in lumbar spine kinetics were observed between unsupported and supported seating conditions.

vi. There will be no change in lumped passive spine stiffness (**Study 3**).
DECISION: The null hypothesis is rejected
Significant changes in the low stiffness zone range were observed immediately post and post-24 hours in flexion. Significant changes in low stiffness the low stiffness zone range were observed post-24 hours in extension. Females exhibited a significant decrease in low stiffness zone stiffness immediately following the unsupported collision simulation.

vii. Peak vertebral translation of the porcine FSU, during each simulated collision, will not be influenced by varying collision severity or posture (**Study 4**).
DECISION: The null hypothesis is rejected.
Significant differences in peak vertebral translation were observed across impact severities, with peak vertebral translation increasing as impact severity increased. However, no effects of posture were observed across impact severities.

viii. There will be no change in passive flexion-extension and shear neutral zone testing pre and post impact (**Study 4**).
DECISION: The null hypothesis is rejected.

A significant decrease in passive flexion-extension neutral zone stiffness was observed across all specimens post impact. In addition to a decrease in neutral zone stiffness, an increase in flexion-extension neutral zone range was observed in specimens exposed to the highest severity impact. Similarly, for Phase 1 of shear neutral zone testing, a significant decrease in shear neutral zone stiffness was observed post impact in specimens exposed to the moderate and high severity impacts. An increase in shear neutral zone range was also observed in specimens exposed to the highest severity impact. However, no significant differences in Phase 2 shear neutral zone testing were observed.

ix. There will be no difference in mechanical properties between healthy control samples and impacted samples (**Study 5**).

DECISION: The null hypothesis is accepted.

No significant changes in FCL stiffness were observed across impact groups in comparison to control.

8.3 Summary of Contributions

Study I:

i. Results from this study provide evidence that low to moderate speed rear-end collision configurations frequently result in claims of low back injury reporting.

ii. A significant contribution from this work is the examination of medical history associated with claimed LBP in low speed collisions. It was found that pre-existing LBP and lumbar spine disc degeneration were particularly common in those with LBP complaints. This investigation also found that 97 percent of all LBP claims also had an accompanying whiplash associated disorder diagnosis.

Study II:

i. This study represents one of the first efforts to characterize peak trunk muscle activations and lumbar accelerations in human volunteers exposed to 4 km/hour simulated rear-end collisions.

ii. Results from this study provide evidence that during an unanticipated low speed rear-end collision, the peak activation of muscles in the lumbar spine are low in magnitude. As such,

muscle activation likely has minimal contribution to the internal joint loads that are experienced in the lumbar intervertebral joints during a low speed rear impact collision.

iii. Results from this investigation support the use of simplified joint models in estimating the joint loads in the lumbar spine during low speed rear impact collisions and support the application of cadaveric and anthropomorphic test device (ATD) testing in understanding the resultant joint loads in the lumbar spine associated with rear-end collisions.

Study III:

i. This study represents one of the first efforts to measure peak lumbar spine kinematics and joint reaction forces in human volunteers exposed to 8 km/hour simulated rear-end collisions.

ii. Results from this study provide evidence that young healthy adults do not report clinically significant levels of low back pain follow a laboratory simulated low velocity rear end collision.

iii. Results from this investigation also demonstrate that similar to tasks such as repetitive lifting, prolonged driving and prolonged office seating, changes in lumbar spine lumped passive stiffness do occur following a simulated rear-end collision and persist for 24 hours.

Study IV:

iv. This study represents one of the first efforts to measure pre/post changes in flexion-extension and shear neutral zone properties across varying impact severities and posture.

v. This study also represents the first effort to quantify peak vertebral translations and facet joint capsule strain across impact severity and posture.

vi. Results from this investigation suggest that the FCL does not appear to undergo injurious or permanent mechanical changes in response to low to moderate MVC impact scenarios.

vii. Results from this investigation also demonstrate that posture does not interact with impact severity in the mechanical exposures of functional spinal units to sudden impacts.

Study V:

i. This investigation represents one of the first efforts to characterize the mechanical and viscoelastic properties of FCL tissue with tensile loading in control specimens and specimens which have been exposed to varying impact severities.

ii. Results from this study provide novel insight into the viscoelastic response that occurs in FCL tissue.

iii. Results from this investigation provide strong evidence that the FCL is not exposed to sub failure loading during impact severities up to 11g.

8.4 Global Summary

Epidemiological research suggests that up to 50% of individuals involved in a low speed rear-end car accident will develop an acute onset of low back pain (Fast et al., 2002). To date, laboratory simulations have pointed in the direction that the exposures during low speed collisions do not cause the potential for injury. However, the continuous reporting of low back pain after low speed collisions demonstrated a clear need to investigate if a link exists between low speed motor vehicle collisions and low back injury. This thesis demonstrates that moderate severity rear-end collision configurations frequently result in claims of low back injury and that pre-existing low back pain and lumbar disc denegation are potential risk factors for reporting low back pain following a low to moderate speed collision. This thesis provides evidence that the exposures to the lumbar spine, in properly position healthy young human volunteers, during simulated 8 km/hour low severity rear-end impacts (both supported and unsupported) are low and well below existing lumbar spine injury reference values. Results do not support an acute traumatic injury mechanism in the lumbar spine. However, results from both *in vivo* and *in vitro* testing demonstrate altered passive stiffness responses in the lumbar spine following low to moderate severity impacts. These passive stiffness changes do not appear to be linked to pain reporting in human volunteers or sub failure loading/injury to the facet joint capsule (*in vitro*) but may lead to changes in the loads and load distributions within the ligaments, intervertebral discs, and muscles immediately following impacts. Such mechanical changes may have future implications for spine range of motion, increased laxity and possible future LBP development.

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Appendix A: Study II Collision Simulation Sled Construction

The collision simulation sled consisted of a stainless-steel frame with two longitudinal rails spaced 68 cm apart and reinforced with two horizontal rails at each end. The entire frame sat at an angle of 3 degrees above the horizontal. The seat assembly consisted of a square metal base, an automotive seat (Crown Victoria Model #EN114 2007, Lear Seating Corporation, Southfield, MI), a platform for the feet with two foot pedals, and two poles in line with the left and right sides of the automotive seat. The seat assembly was bolted onto the metal base, which contained wheels running along either track. A deflated tire (tire pressure = 101.3 kPa (atmospheric pressure), tire diameter = 0.39 m (15.5 inches), tire width = 0.099 m (3.9 inches), rim size = 0.20m (8 inches)) was used to stop the moving seat assembly during the simulated collision. Resultant acceleration traces using the above stopping mechanism were compared to previously published 4 km/hour simulated collisions and showed similar peak accelerations and impact durations (Siegmund et al., 2001a). Comparisons of peak accelerations, collision severity and duration between the current investigation and Siegmund and colleagues (2001) was completed (Table A1). A sample acceleration profile trace is available in Figure A2. A standard 3-point passenger side seat belt was used to restrain participants.

Table A1: A comparison of the impact parameters from the current investigation to Siegmund and colleagues whom completed 4 km/hour vehicle to vehicle collision simulations. Duration of impact was estimated from the sample human corridor trace provided in the manuscript while peak acceleration and delta-v were taken directly from the reported values in the manuscript.

	Current Investigation	**Siegmund et al. 2001**
Peak Accel (g)	1.90 (0.25)	2.10 (0.07)
Delta-V (km/hour)	4.18 (0.31)	3.92 (0.11)
Duration (ms)	119 (6.58)	114

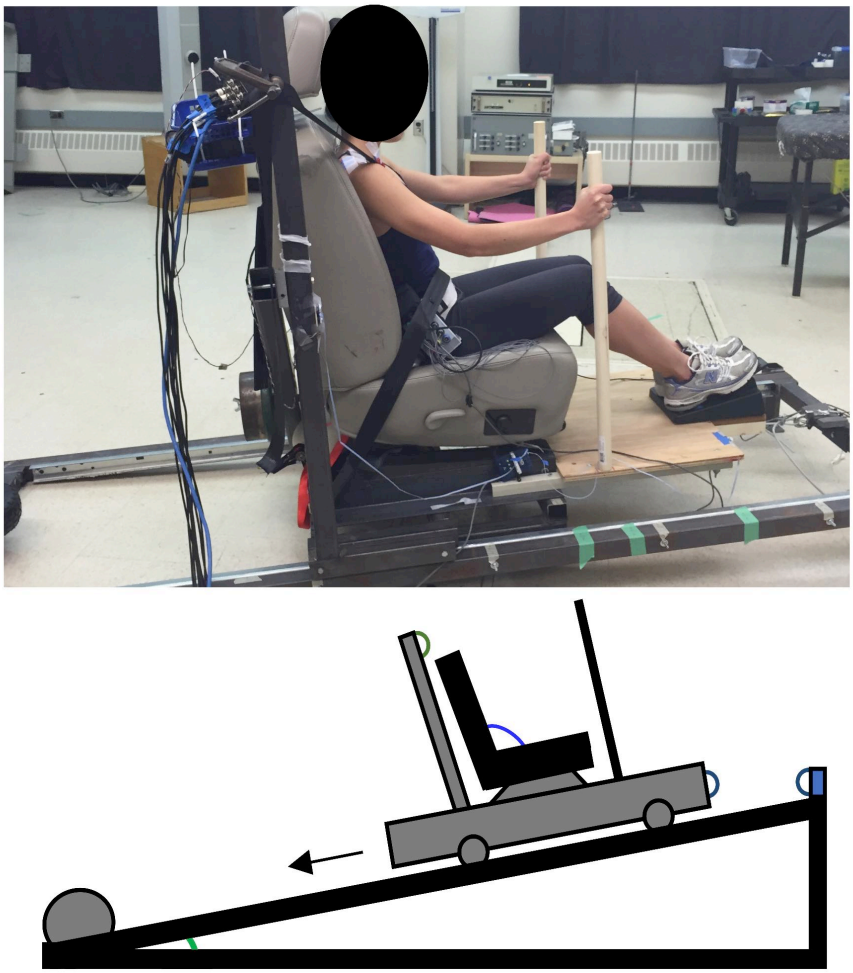

Figure A1: Subject sitting on rear impact crash sled (top), Collision simulation sled schematic (bottom), which illustrates the stainless-steel frame, seat assembly and stopping mechanism.

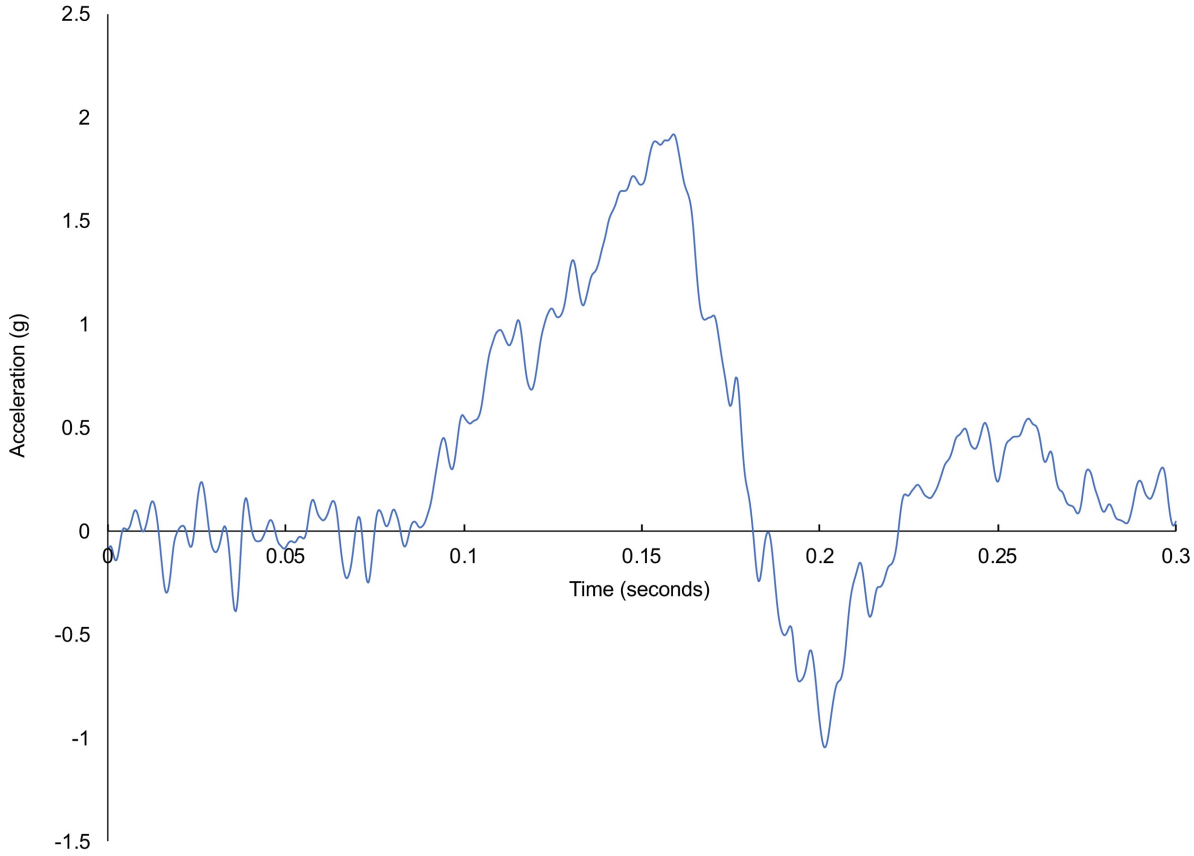

Figure A2: Sample acceleration trace of one of the simulated rear-end collisions

Appendix B: Questions Taken for Psychosocial Surveys

Table B1: Questions take from the Cognitive Risk Profile for Pain (Cook and Degood, 2006)

Strongly Agree	Moderately Agree	Slightly Agree	Slightly Disagree	Moderately Disagree	Strongly Disagree	Please Rate your level of agreement with the following statements
1	2	3	4	5	6	Feeling angry can increase my pain
1	2	3	4	5	6	Pain can put me in a bad mood
1	2	3	4	5	6	Exercise can help me manage my pain
1	2	3	4	5	6	My life should be pain free
1	2	3	4	5	6	Worry can increase the pain I feel
1	2	3	4	5	6	My attitude and the way I think are an important part of how to manage my pain
1	2	3	4	5	6	Stress in my life can make my pain feel worse
1	2	3	4	5	6	Pain can make me feel depressed

Table B2: Questions from the Survey of Pain Attitudes – Brief Version (Tait and Chibnall, 1997)

Please rate your level of agreement with the following statements	Very Untrue	Somewhat Untrue	Neither True nor Untrue/ or Does Not Apply	Somewhat True	Very True
There are many times when I can influence the amount of pain I feel	0	1	2	3	4
When I hurt, I want my family to treat me better	0	1	2	3	4
Anxiety increases the pain I feel	0	1	2	3	4

When I am hurting, people should treat me with care and concern	0	1	2	3	4
It is the responsibility of my loved ones to help me when I feel pain	0	1	2	3	4
Exercise and movement are good for a pain problem	0	1	2	3	4
Just by concentrating or relaxing, I can ‘take the edge’ off my pain	0	1	2	3	4
Medicine is one of the best treatments for chronic pain	0	1	2	3	4
Depression increases the pain I feel	0	1	2	3	4
If I exercise, I could make my pain problem much worse	0	1	2	3	4
I believe that I can control how much pain I feel by changing my thoughts	0	1	2	3	4
Often I need more tender loving care than I am now getting when I am in pain	0	1	2	3	4
There is a strong connection between my emotions and my pain level	0	1	2	3	4

Table B.3: Questions from the Fear Avoidance Beliefs Questionnaire (Waddell et al., 1993)

Please rate your level of agreement with the following statements	**Completely Disagree**	**Moderately Disagree**	**Slightly Disagree**	**Unsure**	**Slightly Agree**	**Moderately Agree**	**Completely Agree**
Physical activity might harm my back	0	1	2	3	4	5	6
I should not do physical activities that (might) make my pain worse	0	1	2	3	4	5	6
My work is too heavy for me	0	1	2	3	4	5	6
My work might harm my back	0	1	2	3	4	5	6

Appendix C: Study III Collision Simulation Sled Construction

To ensure the responses of human volunteers collected in this investigation were representative of real-world collisions, it was imperative that the collision simulation device used repeatably mimicked the impact parameters of a low velocity impact. Therefore, a repeatable collision simulation device was developed to simulate rear-end impacts, on human volunteers, using inputted impact parameters obtained from real vehicle to vehicle rear-end collisions.

Based on previous work by Siegmund and colleagues the following impact parameters were desired (Siegmund et al., 2001a):

Variable	Desired Value
Duration	135 ms
Coefficient of Restitution	0.60
Maximum Acceleration	3.5 g
Delta-V	7 km/hr

The collision simulation sled was initially modelled as a rear-facing cart mounted on an inclined plane. This collision simulation sled was developed based off of previous work (Kaneoka et al., 1999; Ono et al., 1997). The cart accelerated under gravity until it simultaneously collided with the springs and dashpots at the base. Altering the mechanical parameters of the device—the mass of the sled, spring stiffness, damper viscosity, length and angle of the ramp—allowed for tuning of the desired impact parameters. The optimal mechanical parameters were determined by minimizing the squared difference between impact parameters obtained from the model (Equation C1) and the desired impact parameters (Table C1).

$$m\ddot{s} + b\dot{s} + ks = -mg \sin\theta \;,\; s(0) = 0,\; \dot{s}(0) = -\sqrt{2Lg}(\sin\theta - \mu\cos\theta) \quad \text{(Equation C1)}$$

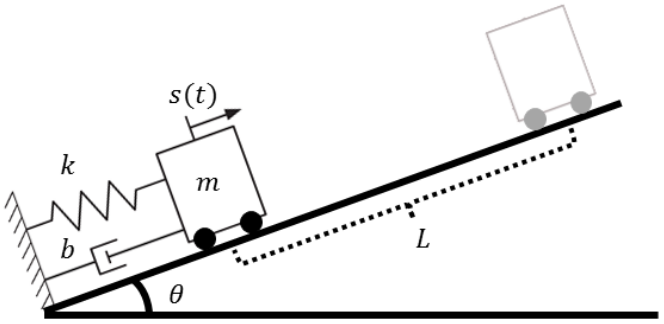

Figure C1: Initial 1-D Sled Model. The sled starts from the greyed position at zero velocity, and accelerates under gravity. It strikes the springs and dashpots, whose mechanical parameters are selected to yield the desired impact parameters.

Where $s(t)$ is the deflection of the springs, m is the mass of the sled (350 lbs), k is the stiffness of the spring, b is the viscosity of the damper, L and θ are the length and angle of the ramp, respectively, and μ is the coefficient of friction for the sled (measured to 0.02). Optimization was done in the Python (version 3.5.1) programming language, using the minimize function in the

'Scipy' package. Springs were custom ordered (Omnicoil, Ayr, Ontario, Canada) such that four springs in parallel would supply the estimated required stiffness (stiffness and damping parameters of 31991 N/m and 593.78 Ns/m, respectively).

Initial pilot impact parameters for physical trials were recorded from a 3 degree-of-freedom accelerometer, which was processed in accordance with SAE standards (Society of Automotive Engineers, 1995). During all pilot testing a Hybrid II ATD was belted in the automotive seat and additional mass was added to the seat assembly such that the total added mass of the seat assembly was 250 lbs. A comparison of desired values for impact parameters against those estimated by the model and those measured from the physical sled are provided in Table C1 and Figure C2.

Table C1: Comparison of the desired values for impact parameters against those estimated by the model and those measured from the collision simulation sled

Variable	Desired Value	Model Estimated Value	Measured Value
Duration	135 ms	123. 5 ms	107 ms
Coefficient of Restitution	0.60	0.599	0.511
Maximum Acceleration	3.5 g	3.3 g	3.99 g
Delta-V	7 km/h	7.99 km/h	7.26 km/h

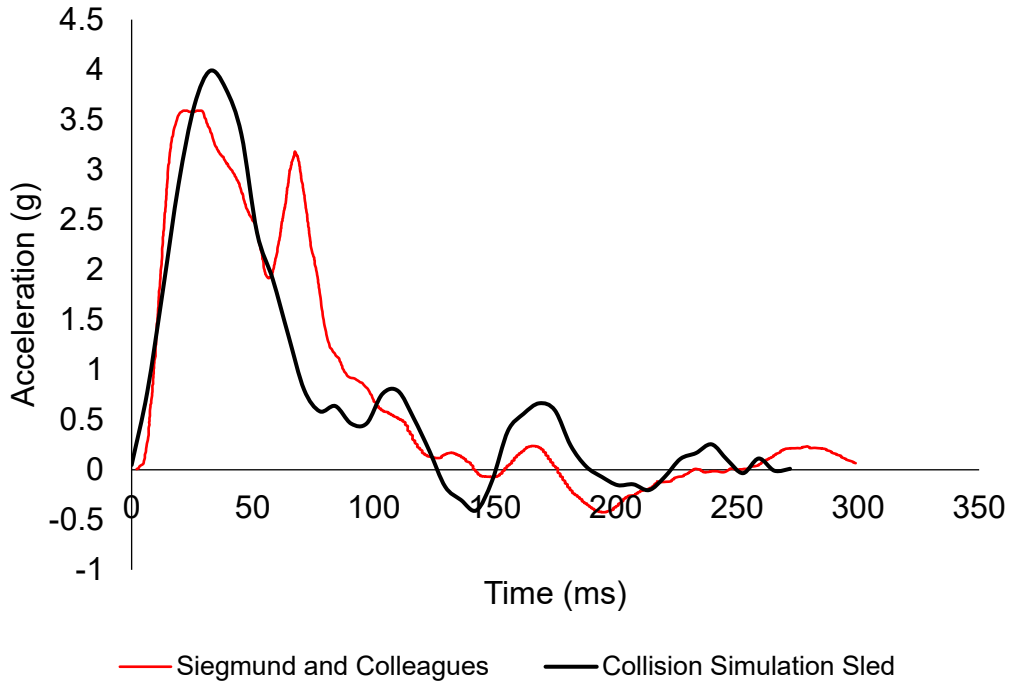

Figure C2: Sample comparison of acceleration trace from the current investigation versus a digitized acceleration trace from Siegmund and colleagues (2001).