The Effects of Lumbar Spine Manipulation on the Flexion-Relaxation Response in Chronic Low-Back Pain Participants.

Nicholas W. Ritter

Thesis submitted in partial fulfilment of the requirements for the degree of Masters of Osteopathy Unitec Institute of Technology, 2014
DECLARATION

Name of candidate: Nicholas Wayne Ritter

This Thesis entitled, “The effects of lumbar spine manipulation on the flexion-relaxation response in chronic low-back pain participants.”, is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

Candidate’s declaration

I confirm that:

- This Thesis represents my own work;

- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: 2011-1221

Candidate Signature: ……………………… Date: …………………

Student number: 1098387
ACKNOWLEDGEMENTS

I would like to thank all participants who volunteered their time towards this study, with special recognition to my supervisors Robert Moran and Jamie Mannion for your valuable help. A special thanks to Mum, Dad, Daniel and friends for all of your support and encouragement. This is for you.
PREFACE

This thesis is submitted in partial fulfilment of the requirements for the Masters of Osteopathy degree at Unitec Institute of Technology.

The following thesis is divided into three sections:

1. The literature review, with the emphasis on:
   - Low back pain prevalence, and its social impacts
   - Chronic low back pain and its classification
   - Investigation of mechanisms associated to the Flexion-Relaxation response, which is a common neuro-muscular phenomenon observed to be predictably absent in individuals with back pain
   - The effects of lumbar spine manipulation on absent flexion-relaxation response in chronic low-back pain participants.

2. A manuscript in the format specified for submission to the *Journal of Electromyography and Kinesiology*, investigating the effects of lumbar spine manipulation on the flexion-relaxation response, range of motion and pain perception in chronic low-back pain participants.

3. Appendices including ethical approval, participant information sheets, consent forms, results information and the guidelines for authors to the *Journal of Electromyography and Kinesiology*. 

THESIS ABSTRACT

High-velocity spinal manipulation continues to gain popularity as treatment for chronic low-back pain (CLBP). The aim of this study was to investigate the effects of high-velocity spinal manipulation on the flexion-relaxation (FR) response, ROM and VAS.

Ten CLBP participants under counter-balanced cross-over design were randomised into manipulation or control groups for either a non-specific bilateral high-velocity manipulation or 30 s side-lying. FR values, finger-floor distance measures and pain perception (100mm VAS) were recorded immediately before and after the intervention.

ROM was controlled during pre-measure FR tasks to individual finger-floor distance values recorded at baseline. Post intervention and control measures removed ROM control and were then compared against pre-measure values to evaluate neuro-muscular changes associated to the intervention.

Repeated-measures two-way ANOVA revealed a significant ($p<0.001$) effect for ROM and VAS for both the control and intervention groups. No changes were observed for FR, despite changes in VAS.

Conclusion: This study shows that there appears to be a temporal asymmetry between restoration of function and pain cessation. Observations in delayed muscle function have been reported in experimental pain research following pain cessation. These findings warrant further investigation to better understand the mechanisms associated to pain, muscle function and spinal manipulation.
# Table of Contents

DEclarAtion ..................................................................................................................... 2

Acknowledgements ......................................................................................................... 3

PREFACE ........................................................................................................................... 4

Thesis Abstract .................................................................................................................. 5

List of Abbreviations ......................................................................................................... 8

Section One: Literature Review ......................................................................................... 9

Introduction ....................................................................................................................... 10

Low Back Pain ................................................................................................................... 10

Pain models ....................................................................................................................... 12

Pain-spasm-pain ............................................................................................................... 12

Lund’s Pain-adaptation ..................................................................................................... 13

Reduced modulation depth .............................................................................................. 13

Flexion relaxation ............................................................................................................ 15

Proposed Mechanism ...................................................................................................... 15

Fear-Avoidance ................................................................................................................ 17

Range of Motion and Flexion-Relaxation ......................................................................... 19

Treatments affecting Flexion-Relaxation ......................................................................... 20

Biofeedback ..................................................................................................................... 20

Spinal Manipulative Therapy ......................................................................................... 21

High-velocity Low-amplitude Manipulation ................................................................... 22

SMT and Neuro-muscular Influences ............................................................................. 24

SMT and ROM and the FRP ............................................................................................ 25

Conclusion ....................................................................................................................... 26

References ....................................................................................................................... 27

Section Two: Manuscript ................................................................................................... 36

Introduction ....................................................................................................................... 38

Methods ............................................................................................................................ 40

Design ............................................................................................................................... 40

Participants ....................................................................................................................... 40

Instrumentation ............................................................................................................... 40

Surface Electromyography ............................................................................................. 41

Skin Preparation and Electrode Placement ..................................................................... 41
Surface EMG Detection and Recording ................................................................. 41
Dependent Variables ......................................................................................... 42
  Visual Analogue Scale .................................................................................. 42
  Finger-floor Distance Measure .................................................................... 42
Independent variables ..................................................................................... 42
  HVLA manipulation ..................................................................................... 42
Control condition ........................................................................................... 43
Procedure ........................................................................................................ 43
  Baseline Measures ....................................................................................... 43
Data Analysis .................................................................................................... 44
Results .............................................................................................................. 44
Discussion ......................................................................................................... 44
  Future Research ............................................................................................ 48
Limitations ......................................................................................................... 49
Conclusion ......................................................................................................... 49
References .......................................................................................................... 50
Section Three: Appendices ......................................................................................
  Appendix A: Images, Diagrams and Tables ....................................................... 60
  Assessed for eligibility .................................................................................... 60
  Appendix B: Information Sheet and Consent Form ............................................. 62
  Appendix C: Ethical Approval ......................................................................... 71
  Appendix D: The Journal of Electromyography and Kinesiology: Guide for Authors ...... 74
**List of Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBP</td>
<td>Low Back Pain</td>
</tr>
<tr>
<td>CLBP</td>
<td>Chronic Low Back Pain</td>
</tr>
<tr>
<td>SMT</td>
<td>Spinal Manipulative Therapy</td>
</tr>
<tr>
<td>ROM</td>
<td>Range Of Motion</td>
</tr>
<tr>
<td>sEMG</td>
<td>Surface Electromyography</td>
</tr>
<tr>
<td>FR</td>
<td>Flexion-Relaxation</td>
</tr>
<tr>
<td>ES</td>
<td>Erector Spinae</td>
</tr>
<tr>
<td>HVLA</td>
<td>High Velocity Low Amplitude</td>
</tr>
<tr>
<td>VAS</td>
<td>Visual Analogue Scale</td>
</tr>
<tr>
<td>kHz</td>
<td>Kilohertz (unit)</td>
</tr>
<tr>
<td>FIR</td>
<td>Finite Infinite Response (filter)</td>
</tr>
<tr>
<td>RMS</td>
<td>Root Mean Square</td>
</tr>
<tr>
<td>MVF</td>
<td>Maximum Voluntary Flexion</td>
</tr>
<tr>
<td>$P$</td>
<td>Statistical Probability</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis Of Variance</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
</tbody>
</table>
Section One: Literature Review
Introduction

LBP is one of the most common presenting complaints seen in clinical practice. This condition incurs a multitude of social and economic costs bridging individual disabilities with diminished work productivity and quality of life. The objective of this review is to discuss current literature in the area of manual therapy, in particular, spinal manipulative therapy and its effect on factors relating to the management of LBP symptoms. These factors include observations on muscle activity and lumbar range of motion and how pain may alter these factors. The context of LBP mechanisms and treatments will be discussed with emphasis on the Flexion Relaxation (FR) response, a phenomenon that is commonly used as an assessment tool for LBP treatment.

Low Back Pain

Low back pain as a condition presents a high incidence rate in society which is multifactorial in its influences. LBP is described as pain that resides between the lower margins of the 12th rib and the gluteal folds. Approximately 85% of the population will experience back pain at some stage in their lifetime with 50% of cases observed to recur within 3 months (Johnson, Adegoke, & Ogunlade, 2010).

LBP is deemed chronic if symptoms persist beyond 12 weeks. Chronic LBP is described not as a clinical entity and diagnosis, but rather a symptom in patients with different stages of impairment, disability and chronicity (Airaksinen et al., 2006). Reports indicate that approximately 10% of chronic LBP cases present with Red Flags which display typical signs and symptoms of specific LBP. Specific LBP is defined by clinical symptoms which originate from specific pathophysiological mechanisms, including herniated nucleus pulposus, infection, osteoporosis, rheumatoid arthritis, fracture, or tumor (Koes, Van Tulder, & Thomas, 2006). The majority of persistent LBP cases presented in clinical practice are regarded as non-specific in nature (Krismer & van Tulder, 2007). Non-specific LBP is regarded as a symptom of unknown cause, which may originate from various innervating structures of the spine (Balagué, Mannion, Pellisé, & Cedraschi, 2012). This condition; however, is not attributed to the same underlying causes observed in specific LBP.

According to the New Zealand Acute Low Back Pain Guide (2004) developed by the Accident Compensation Corporation (ACC), 65% of individuals who have never experienced back pain or sciatica over 50 years of age present with abnormalities on plain x-rays, 33%
will show evidence of disc abnormality on MRI, with 20% under 60 showing evidence of a herniated disk. These percentages illustrate challenges in the diagnosis of discogenic LBP with imaging. Subsequently, development of chronic pain may include psychological dimensions which may provide insights into how an individual may respond to back pain (Balagué et al., 2012). There is strong evidence that the single most consistent predictor of future LBP, and work loss is a previous history of LBP, relative to the frequency and duration of attacks (Waddell & Burton, 2001). As a consequence, this poses an economic burden to society, reflecting in a large number of work days lost and reducing rate of return to usual activity (Krismer & van Tulder, 2007). Chronic LBP is subsequently noted as a diagnosis of convenience for many people, who are actually disabled for socioeconomic, work-related, or psychological reasons (Andersson, 1999).

The associated factors which influence the development and progression of LBP are often multidimensional. Optimal human function may require an inter-related balance between the ‘sum of its parts’ which reflect as the different functioning somatic systems. Current, diagnostic methods exploring the potential mechanisms of LBP are numerous. The exploration of potential influencing LBP factors has done so by developing models which incorporated specific multiple human aspects known to contribute to LBP. This may be illustrated by aspects of the biopsychosocial model which identifies certain dimensions that may contribute to spinal pain (Thomas & France, 2008; Waddell, Newton, Henderson, Somerville, & Main, 1993).

While this model explains associated aspects of disability and forms the basis for management strategies, investigating the experience of pain and subsequent neuro-muscular adaptations remain a crucial component for future pain management research. Maladaptive pain processes are believed to influence the development of chronic pain through altering motor control patterns. This may be observed where increases in muscle activity of stabilizing muscle groups, has adapted to pain in order to ‘splint’ the injured structure (McGorry & Lin, 2012; van Dieën, Selen, & Cholewicki, 2003; Zedka, Prochazka, Knight, Gillard, & Gauthier, 1999). However, these maladaptive mechanisms may result in an increase in abnormal loading forces across pain sensitive structures (Dankaerts & O’Sullivan, 2011) which may be counter-productive and subsequently perpetuate the experienced pain condition.
Pain models

Pain-spasm-pain

The pain-spasm-pain model was first proposed by Travell, Rinzler, and Herman (1942) who illustrated that the presence of somatic pain reflects as an increase in muscle tension and activity resulting in muscle spasm. This model is described as a feedback mechanism where nociception afferents on the gamma motor neuron increase the afferent discharge of the muscle spindles. This in turn activates the alpha motor neuron directly through excitatory interneurons, further increasing muscle activation, subsequently causing spasm (Johansson & Sojka, 1991). Early clinical studies have subsequently shown a substantial amount of LBP patients present with associated muscle spasm along with increased muscle activity on surface electromyography (sEMG) (Ahern, Follick, Council, Laser-Wolston, & Litchman, 1988; Fischer & Chang, 1985; Kravitz, Moore, & Glaros, 1981). These neuro-adaptive changes have been observed during static postures concurrent to a reduction of muscle activity during movement tasks (Roland, 1986). However, this model is debated as experimental and clinical data suggests the pain-spasm-pain model as being too simplistic (Hodges & Moseley, 2003).

Debate lies in conflicting evidence surrounding erector spinae (ES) muscle activity observed in LBP populations. Significant variability in sEMG results are reported in LBP populations (Hodges, Moseley, Gabrielsson, & Gandevia, 2003); where participants display increased muscle activity (Arena, Sherman, Bruno, & Young, 1989; Wolf & Basmajian, 1978), increased fatigability or muscle hypoactivity (Demoulin, Crielaard, & Vanderthommen, 2007; Sihvonen, Lindgren, Airaksinen, & Manninen, 1997), or no change in muscle activity (Nouwen & Van Akkerveeken, 1987). Consequently, this model may be challenged where studies have experimentally induced pain in healthy participants and reported evidence of diminished muscle function following cessation of induced pain (Henriksen, Rosager, Aaboe, Graven-Nielsen, & Bliddal, 2011; Hodges et al., 2003). These findings somewhat contradict Travell et al. (1942) model of muscle-tension or pain-spasm-pain mechanisms; however no definitive or consistent outcomes have currently been established.
Lund’s Pain-adaptation

Lund’s pain-adaptation model is a more recent hypothesis challenging the proposed hyperactivity-causality mechanism illustrated in Travell et al. (1942) pain-spasm-pain model. This alternative pain model suggests that pain modifies muscular performance by decreasing the neuronal output of agonist (concentrically contracting) muscles while increasing the level of antagonist (eccentrically contracting) co-contraction (Lund, Donga, Widmer, & Stohler, 1991). This model appears to favour adaptive mechanisms illustrated in recent pain research where muscular changes in strength, range of motion (ROM) and velocity were observed following experimental pain interventions (Arendt-Nielsen, Graven-Nielsen, Svarrer, & Svensson, 1996; Hodges et al., 2003). While support has been found for this model (Farina, Arendt-Nielsen, & Graven-Nielsen, 2005; Stohler, Zhang, & Lund, 1996; van Dieën et al., 2003), it remains incomplete and fails to consider how pain can affect various intensities of contraction, from relaxation through to maximum voluntary contraction.

Reduced modulation depth

Efficacy of the muscular system is suggested to be strongly dependent on the controlling capabilities of the CNS (Harvey & Descarreaux, 2013; Panjabi, 1992). Another current model may be more suitable in explaining certain unaccounted neurological pain anomalies, illustrated as limitations in the pain-spasm-pain and pain adaptation models. Observations in reduction of modulation depth may provide better insight into understanding the mechanisms of pain adaptation. This model considers both the active and passive roles of muscle, where a reduction in modulation depth is observed in muscle activity during pain experience. Modulation response to painful stimuli is an autonomous adaptation, commonly observed in painful spinal muscles. A reduction in muscle activity is observed during specific movement tasks including trunk flexion and re-extension, in contrast to static positions where an increase in muscle activity at end range of maximum voluntary flexion and quite standing is consistently observed (Ahern et al., 1988; Alschuler, Neblett, Wiggert, Haig, & Geisser, 2009; Zedka et al., 1999). The failure of trunk muscle relaxation during full flexion is an observation consistency seen in LBP sufferers (Ahern, Hannon, Goreczny, Follick, & Parziale, 1990; Descarreaux, Lafond, & Cantin, 2010; Floyd & Silver, 1955; Watson, Booker, Main, & Chen, 1997).
Somatic information from peripheral mechanoreceptors and other sensory system afferents are interpreted by the CNS in order to initiate a required reaction such as stability of movement (Hodges & Moseley, 2003). Adaptive modulation is a manifested when peripheral terminals become exposed to noxious stimuli. This produces an increase in excitability of the terminal membranes which are modulated by the CNS, through reducing the amount of depolarisation required to initiate an action potential discharge. (Woolf & Salter, 2000).

Changes in modulation depth may be associated with an increase in muscle activity during baseline measures concurrent to decreased maximal activity (Svensson, Houe, & Arendt-Nielsen, 1997; Zedka et al., 1999). Adaptive processes to pain stimuli reflect as changes in modulation depth in order to provide stabilizing support for failing structures and prevent further damage at the site of pain. It is plausible that the increased activity at rest, and or the effects of reduced activation capacity, lead to aberrant activation and or movement patterns that contribute to the chronicity of pain. Ongoing reduction in muscle activity modulation depth in painful structures may result in pain spreading to associated regions relative to the original epicenter of pain (Woolf & Salter, 2000; Zedka et al., 1999).

Current observations in modulation depth infer that muscle activity is not generally increased or decreased, but that activity is modulated depending on the magnitude of activation. In principle, this adaptive process prevents complete muscle rest and prevents maximal contraction. This may be associated to a biological splint which acts to protect damaged areas (McGorry & Lin, 2012; Sullivan et al., 2001), by increasing localized afferent receptor activity, promoting continual ES activity to limit movement and provide stability (Djupsjobacka, Johansson, Bergenheim, & Wenngren, 1995; Masri, Ro, & Capra, 2005; Svensson et al., 1997).

The development of this model has expanded the current understanding of pain adaptive responses beyond the existing theoretical models described by the pain-spasm-pain and Lund’s pain adaptation models. Furthermore, this model may help to better identify potential underlying mechanisms of non-specific LBP and provide a method of distinguishing individual disability. According to data taken from Geisser et al. (2005) meta-analytic review, there are specific indications citing good accuracy, sensitivity and specificity for observational differences in FR, relative to pain or no pain presentation (Geisser et al., 2005). These observations are consistently supported in studies utilising repeated FR measures.
including both static and dynamic standing postures (Ahern et al., 1988; Ahern et al., 1990; Bicalho, Setti, Macagnan, Cano, & Manffra, 2010; Harvey & Descarreaux, 2013).

**Flexion relaxation**

The term “flexion-relaxation” (FR) was first conceived by Floyd and Silver in 1955 and is described as a sudden onset of myoelectric silence in ES muscles of the back during maximum voluntary flexion in standing. This phenomenon is described by two relaxation mechanisms including a shift of movement to passive structures and the redistribution of muscle recruitment to deeper muscles (Floyd & Silver, 1955). Studies investigating this phenomenon indicate that impaired FR is consistently observed in LBP populations when compared against asymptomatic individuals (sensitivity = 0.89; specificity = 0.81) Geisser et al. (2005) and that FR may be restored following an intervention (Marshall & Murphy, 2008; Neblett, Mayer, Brede, & Gatchel, 2010).

**Proposed Mechanism**

The underlying mechanisms involved in the FR response are proposed by two hypothetical models. These models refer to mechanical and neural alterations as being influential in this phenomenon. The mechanical model suggests that a load sharing effect exists where a decrease in extensor muscle activity incites a reduction in active extensor support, allowing passive structures to provide stability at the end range of dynamic movements (Colloca & Hinrichs, 2005; Solomonow, Hatipkarasulu, Zhou, Baratta, & Aghazadeh, 2003). Elastic forces inducing stretch of passive structures subsequently provide additional support during maximum voluntary flexion (McGill & Kippers, 1994).

The other corresponding mechanism believed to facilitate FR relates to neural alterations, resulting in a reflex response to movement. These propositions infer physiological responses to biomechanical and positional alterations in movement. How much these individual models influence the FR response in ES muscle activity is presently unclear. Neural adaptations associated to the FR response are suggestive of an increase in tension across posterior structures during trunk flexion (Solomonow, Baratta, Banks, Freudenberger, & Zhou, 2003). An inhibitory reflex response during flexion is believed to induce relaxation of ES muscles via stretch receptors situated in posterior spinal structures (Gupta, 2001; Solomonow, Baratta, et al., 2003). Relaxation of ES muscles induces the redistribution of load bearing force, producing a balanced effect between the upper body and gravitational influences, in addition
to viscoelastic properties providing spinal support (Demoulin et al., 2007; Schultz, Haderspeck-Grib, Sinkora, & Warwick, 1985). The aforementioned load bearing viscoelastic forces acting upon the spine are believed to assist passive tissues in extensor support once extensor muscles adopt a neutrally relaxed position (McGill & Kippers, 1994). Active contributions from corresponding muscles such as Quadrates Lumborum and deep ES, further assist load sharing, during trunk flexion (Colloca & Hinrichs, 2005). Corresponding posterior elements of the spine, are suggested to restrict over stretching of intervertebral discs to 80% of full range of flexion (Adams, Green, & Dolan, 1994), while reflex contraction of the back muscles are believed to limit spinal flexion, in order to further protect the underlying spine (Sanchez-Zuriaga, Adams, & Dolan, 2010). The resultant load sharing effect is a reduction in myoelectric activity of lumbar extensor muscles, during lumbar flexion, from an upright standing posture (Hashemirad, Talebian, Hatef, & Kahlae, 2009; Shirado, Ito, Kaneda, & Strax, 1995).

Research investigating FR in LBP populations has revealed a difficulty in developing a unified understanding regarding their underlying mechanisms, and how they relate to the differences in which these variables are measured. Certain studies have placed restrictive boundaries on muscle activity thresholds by applying cut-off points indicating the alleged presence or absence of FR. This is reflected in the view that FR corresponds to a particular angle of trunk flexion, which is proposed to be measurable during later stages of forward flexion. Some studies report that FR characteristically occurs at approximately 40-50 degrees of trunk flexion (Ahern et al., 1988; Solomonow, Baratta, et al., 2003); however, cessation of ES activity has also been observed close too or upon maximum voluntary flexion (Kippers & Parker, 1984; Morris, Benner, & Lucas, 1962; Pauly, 1966).

Discrepancies in FR measures influence the interpretation of outcomes making it difficult to draw comparisons between studies. Setting cut-off point may not be the most reliable method of observing FR. Recent studies have demonstrated value in improved quantitative techniques suggesting that there are no definitive cut-off points identifying FR onset and cessation (Mannion, Taimela, Müntener, & Dvorak, 2001; Marshall & Murphy, 2006; Owens, Gudavalli, & Wilder, 2011).
A contrasting method used when evaluating FR is through normalisation of raw sEMG data to a maximal effort contraction. This is considered a useful form of assessing individual FR where observations have revealed intrinsic variability of sEMG activity between study participants (Lehman & McGill, 1999). Despite its reliability as a measure in asymptomatic and symptomatic populations (Dankaerts, O’Sullivan, Burnett, Straker, & Danneels, 2004) this method has been challenged on the basis that it is subjective and is potentially limited by pain sensation in injured individuals (Marras & Davis, 2001). Currently, the most reliable approach used to quantify the FR response is through ratio based evaluation (Alschuler et al., 2009), corresponding to dynamic and static postures throughout repetitive flexion-extension tasks. Two ratios in particular have demonstrated a high association with the presenting clinical variables of LBP where flexion-extension tasks are measures (Alschuler et al., 2009). These ratios are described as the flexion-relaxation ratio (the FRR), which compares the difference between active flexion and full flexion (FR) and the extension-relaxation ratio (the ERR) which compares differences between active extension and full flexion respectively. When groups represent dichotomous variables, as in symptomatic and asymptomatic presentation, these ratios offer a reliable method of assessing quantitative FR data. Utilising FRR and ERR ratios for quantification removes the need of the normalisation method and hence the use of maximum voluntary isometric contraction which does not ascribe well to symptomatic individuals.

**Fear-Avoidance**

A long established mechanism believed to significantly contribute to the maintenance and progression of LBP relates to the fear-avoidance model. When an individual attempts to negate or lessen the onset of familiar pain, the result may be the development of fear avoidance patterns. Guarded movements during certain tasks may be initiated in order to diminish or prevent potential pain onset. This may lead an individual to experience increased levels of anxiety which subsequently catastrophize pain (Crombez, Vlaeyen, Heuts, & Lysens, 1999). In addition, fear-avoidance patterns may potentially exacerbate pre-existing pain conditions (Linton, Overmeer, Janson, Vlaeyen, & De Jong, 2002; Thomas & France, 2008; Vlaeyen & Linton, 2000). This established model for chronic pain has also been identified as influencing the FR response (Ahern et al., 1988; Floyd & Silver, 1955; Geisser, Haig, Wallbom, & Wiggert, 2004). The FR response was first proposed by Lethem, Slade, Troup, and Bentley (1983) who inferred that fear of pain leads to a cycle of decreased
physical activity and increasingly exaggerated pain perception. As chronic pain develops, the behavioural pattern becomes less a reaction to nociceptive pain signals, and more a fear-avoidance further pain (Trost, France, Sullivan, & Thomas, 2012). Few direct relationships between pain severity and level of disability have previously been identified (Waddell et al., 1993). It has; however, been reported that guarded movement contributes to approximately 27% variability in the FR (Ahern et al., 1990); where relationships between pain-related fear, lumbar flexion and dynamic sEMG muscle activity has been investigated in chronic LBP populations (Geisser et al., 2004). This has directed researchers to conclude that fear of pain maybe more instrumental in maintaining disability than actual perceived pain (Crombez et al., 1999; Waddell et al., 1993). However; Dankaerts and O’Sullivan (2011) report that inherent maladaptive conditioning in movement patterns resulting from consistent peripheral nociceptive input is more relative in the development of chronic pain rather than fear-avoidance itself. This may be supported by Zedka et al. (1999) study where asymptomatic participants performed FR tasks during experimentally induced lumbar pain by way of injected hypertonic saline in lumbar ES muscles. During onset of experimental pain, participants displayed diminished ROM only 60-90% of the baseline range with concurrent reduction in modulation depth. When participants underwent the same FR movements during pain free conditions, elevated muscle activity was still apparent during previous painful positions in spite of overcoming splinting movement (Zedka et al., 1999).
Range of Motion and Flexion-Relaxation

The inquiry of mechanisms believed to influence FR has lead researchers to investigate range of motion as a possible factor, suggesting that a certain percentage of flexion is required before an individual achieves FR. Discrepancies between the particular angle of flexion and FR onset are in question with previous research reporting onset of FR as characteristically occurring at approximately 40-50 degrees of trunk flexion Ahern et al. (1988). These angle values indicate the onset of FR as being much earlier than more recent literature reports where subsequent evidence suggests this phenomenon as occurring typically between 70-90 degrees of trunk flexion (Gupta, 2001; Kippers & Parker, 1984; Olson, Solomonow, & Li, 2006; Shin, Shu, Li, Jiang, & Mirka, 2004). However; predicting or determining definitive and absolute values for the angle of FR onset, in addition to the amount of relaxation that is required to meet the definition of relaxation, provides very vague and often inaccurate values, as individual variability appears to be a significant factor to be considered.

It would appear that ROM is not completely synonymous with the presence or absence of FR as individuals presenting with chronic LBP have illustrated trunk flexion beyond the proposed angles characteristic of FR onset, within asymptomatic populations (Kaigle, Wessberg, & Hansson, 1998; Kippers & Parker, 1984). These findings imply the notion of a stable range FR is displayed; however, this range appears suitable for one person but not another, questioning the relative influences of ROM on FR.

Research exploring neuro-muscular adaptation to pain may rectify certain uncertainties surrounding pain influences on ROM. This was investigated by Zedka et al. (1999) who evaluated the effects of experimentally induced pain on muscle activity in the lumbar spine. Motor output of lumbar ES muscles was examined during trunk flexion-extension tasks, concurrent to the induced pain condition. Controlled muscle pain was induced using a 5% saline infusion into the right lumbar ES muscle group. Results from flexion-extension tasks indicated that muscle pain decreased the modulation depth of ES muscles. Muscle activity observed through sEMG identified an associated decreased range and velocity of motion of the painful vertebral segment, which would normally serve to avoid further injury. An interesting outcome was observed when participants overcome their guarding tendency and made the exact same movements during pain as before pain, where sEMG modulation depth remained reduced. The authors state that these results appeared to reconcile the controversy of previous studies, where both hyper and hypoactivity of back pain had been reported. Their
study concluded that deep back pain modulates the voluntary activation of these muscles (Zedka et al., 1999).

**Treatments affecting Flexion-Relaxation**

Parallel to traditional orthodox medical practice are a number of alternative therapies which focus on integrating the patient into their own recovery, utilising psychological methods including cognitive behavioural therapy, multidisciplinary approaches in functional restoration programmes and the utilisation of biofeedback instrumentation. Other methods include manual therapeutic techniques including SMT which have been identified as a useful and highly scrutinised treatment approach in the treatment of LBP symptoms.

**Biofeedback**

The biofeedback method measures skeletal muscle activation and is utilised in developing treatment programmes by identifying potential muscular influences in LBP. It enables an individual to receive visual and or auditory feedback about the activity of their muscles. This feedback facilitates improvements in volitional motor-control of the monitored region. Studies have identified this method as being a non-invasive and a reliable training tool in the treatment of LBP (Moore, 2013; Neblett, Gatchel, & Mayer, 2003; Neblett et al., 2010).

Studies evaluating FR with biofeedback have indicated significant outcomes (Neblett et al., 2003; Neblett et al., 2010) lending support for its use in understanding the mechanistic relationships between muscle activity and LBP. In 2010, biofeedback methods were incorporated in a study by Neblett et al. (2010), who evaluated the influence of sEMG-assisted stretching protocol within a functional restoration treatment program, on ROM and FR during maximum voluntary flexion. This study specifically investigated changes in FR following an intervention as opposed to producing measurable outcomes. Groups evaluated consisted of chronic LBP participants and asymptomatic control. Results indicated a significant improvement in both FR and ROM (Neblett et al., 2010) supporting previous research where quantified improvements in FR had been observed following an intervention (Neblett et al., 2003; Neblett et al., 2010; R Neblett, 2002; Randy Neblett, 2007). Changes in pain and disability were not assessed in Neblett et al. (2010) study; however, in more recent research by Moore (2013), inclusion of pain and disability measures were utilized while investigating the efficacy of sEMG biofeedback assisted stretching interventions for chronic LBP.
The aim of Moore (2013) study was to explore whether improved FR is associated to improvements in ROM, pain intensity and disability in chronic LBP individuals. Of the nine participants included in this study, three improved pain intensity and FR scores to clinically significant levels. Statistical significance was set at the $p < 0.5$ level and was defined as a change in FR, ROM, pain intensity and disability in individuals with chronic LBP. Results from this study suggest that a sEMG assisted stretching program may benefit individuals with chronic LBP and impaired FR; however, studies of larger population are indicated (Moore, 2013). These recent studies provide good evidence for the use of sEMG assisted biofeedback methods, which have indicated significant improvements in FR ROM and reduction of pain, resulting from an intervention. As improvements were observed in symptomatic LBP individuals, it provides good validation as a method in which other interventions may be applied when investigation other forms of LBP treatment.

**Spinal Manipulative Therapy**

The use of spinal manipulation therapy (SMT) has emerged as a popular form of treatment utilised by osteopaths, chiropractors and physiotherapists for treatment of spinal pain and the associated symptoms of articular joint dysfunction. Research endeavoring to establish a more comprehensive understanding of the underlying mechanisms of SMT are un-unified and conflicted; although, methodological improvements in data acquisition continue to develop and inform a better understanding of the effects of SMT. This ongoing validation process has produced a number of reviews and studies on manipulation, with researchers broadening their search parameters to various physiological mechanisms believed to influence and maintain LBP. Particular areas of interest include the effects SMT on ROM, muscle activity and pain perception which warrant further exploration.

The ever increasing body of literature owing to the efficacy of SMT has shown promising results in the treatment for LBP, but fail to indicate truly significant outcomes due to certain methodological oversights. Although early clinical trials have previously inferred SMT to be effective in the treatment of spinal pain (Paris, 1983), SMT still fails to yield strong effect sizes on LBP. A systematic review and meta-analysis of clinical trials by Licciardone, Brimhall, and King (2005), relating to Osteopathic Manipulative Technique in the treatment of LBP, explored computerised bibliographic search results regarding relevant reports, identifying randomized controlled trials of OMT. Search results revealed 6 trials, conducted between 1973 and 2001, involving eight OMT versus control treatment comparisons. Results
from reviewed trials were taken from 525 participants with LBP, where significant reductions in pain were associated with OMT (effect size, -0.30; 95% CI, -0.47 - -0.13; P = .001). While this review indicates support for the use of SMT in the treatment of LBP, the effect size indicated is mild to moderate at best; therefore research indicating stronger effect sizes are required to improve the validation of SMT as a primary therapy for LBP sufferers.

High-velocity Low-amplitude Manipulation

A specialised technique utilized in the treatment of spinal dysfunction is the high-velocity low-amplitude (HVLA) manipulation. Application of HVLA techniques are regarded as one of the most common forms of SMT (Fritz, Childs, & Flynn, 2005). This technique is commonly applied to a wide range of presenting clinical complaints, including the treatment of chronic LBP (Bicalho et al., 2010; Cecchi et al., 2010; M. Descarreaux, Blouin, Drolet, Papadimitriou, & Teasdale, 2004; Ghroubi, Elleuch, Baklouti, & Elleuch, 2007; Harvey & Descarreaux, 2013; Lalanne, Lafond, & Descarreaux, 2009). HVLA techniques have subsequently been observed to have greater short and long-term clinical outcomes on function and pain relief than other therapies (Cecchi et al., 2010); however, the underlying mechanisms require further investigation to establish a cohesive model of understanding.

Symptoms of non-specific LBP probably originate from a number of different spinal structures which inter-relate in form and function. A multifactorial hypothesis identifying mechanistic effects of HVLA manipulation are theorised in relation to (1) the release of entrapped synovial folds or plica, (2) the relaxation of hypertonic muscles by sudden stretching, (3) the disruption of articular or peri-articular adhesions, and (4) the unbuckling of motion segments that have undergone disproportionate displacement (Evans, 2002). These structures are found throughout the spine and may either individually or collectively contribute to the development of LBP. Dysfunction of these mechanical structures may produce effects on adaptive pain modulation when inflammatory mediators produced during injury excite peripheral terminals upon exposure (Woolf & Salter, 2000). Evidence exploring the underlying mechanisms of SMT suggest there to be a regulatory effect upon afferent discharge, following manipulation (Dishman, Weber, Corbin, & Burke, 2012). Furthermore, HVLA manipulation is postulated to be directed at the articular joints and their corresponding anatomical structures in the human spine (Evans, 2002; Maigne & Vautravers, 2003). The forces applied during HVLA manipulation are believed to ‘distract’ the articular joint (facet) (Cramer et al., 2002; Cramer et al., 2000) and its relative structural components, forcefully
stretching para-spinal muscles, inducing relaxation mechanisms of these structures (Maigne & Vautravers, 2003).

Although this relaxation phenomenon is not fully understood, part of the mechanism at least, is inferred to activate Ib fibres of the flexor muscles, inducing pre-synaptic inhibition of afferent Ia fibres of the agonists. This in turn contributes to a reduction in activity of extensor muscle alpha motor neurons (Maigne & Vautravers, 2003). Observations in FR research have illustrated altered function of extensor muscles by sEMG, depicting a state of impaired FR being synonymous with continual ES muscle activation during maximum voluntary flexion (Ahern et al., 1988; Ahern et al., 1990; Watson et al., 1997). SMT studies subsequently indicate significant decreases in pain when investigating its effect on symptomatic populations (Bicalho et al., 2010; Cecchi et al., 2010; M. Descarreaux et al., 2004; Licciardone et al., 2005). Furthermore, changes in motor control to pain are reported in numerous acute and chronic LBP studies (Hodges & Richardson, 1996; Radebold, Cholewicki, Panjabi, & Patel, 2000; Sihvonen et al., 1997) which are often associated with significantly decreased lumbar ROM (Neblett et al., 2010; Shum, Tsung, & Lee, 2013). Altered ROM is reported to subsequently correspond to an absence of FR (Colloca & Hinrichs, 2005; Hashemirad et al., 2009). The restoration of these three variable to baseline muscle activity, flexibility and pain perception contribute to vertebral stabilization and the redistribution of forces across passive connective tissue structures (Colloca & Hinrichs, 2005; Farfan, 1975; Kippers & Parker, 1984; Lalanne et al., 2009). As mentioned, SMT acts on the various components of the vertebral motion segment by distracting the facet joints (Maigne & Vautravers, 2003); thus, improving segmental ROM through the ‘unbuckling’ of motion segments that have undergone disproportionate displacement (Evans, 2002). The restoration of both FR and ROM is reported as contributing to improved vertebral stabilization, redistributing forces to passive connective tissue structures (Colloca & Hinrichs, 2005; Farfan, 1975; Kippers & Parker, 1984; Lalanne et al., 2009). These findings provide good support for investigating the efficacy of SMT in LBP populations utilising FR measures compared with ROM evaluation methods.
**SMT and Neuro-muscular Influences**

There are a number of theorised mechanisms in which SMT is believed to influence the CNS. A theoretical model by (Pickar, 2002) pertains to certain relationships between SMT, segmental biomechanics, and the CNS. Biomechanical changes during SMT are reported to decrease the inflow of sensory information to the CNS (Pickar, 2002). The CNS is known to employ adaptive mechanisms in response to physiological pain (modulation); however, at times these adaptations can hinder function as opposed to help. Such hindrances may be observed through effects attributed to neural plasticity. Plasticity is an observed phenomenon of the CNS in which neurons display the capacity to change their function, chemical profile or structure (Woolf & Salter, 2000); and are considered to play a crucial role in the development of neurogenic pain (Coderre, Katz, Vaccarino, & Melzack, 1993; Woolf, 1983).

Adaptive modulation is triggered within the CNS when peripheral terminal nociceptors are exposed to noxious input. The response is a reduction in the amount of depolarisation required to initiate an action potential discharge (Wall & Woolf, 1984; Woolf & Salter, 2000). Ongoing exposure to pain without intervention may lead to a subsequent state of central sensitisation. This state reflects an enhanced response to normal inputs and is believed to interfere with adaptive modifications that are initiated to promote recovery following injury. The subsequent heightened synaptic transmission results in a reduction in pain threshold parallel to amplification of pain responses, which may support the spread of pain sensitivity to adjacent regions (Ji, Kohno, Moore, & Woolf, 2003). This persistently increasing response of the CNS to noxious stimuli is described as sharing a strong relationship to memory, in a way that information is processed, stored and made accessible for retrieval (Ferguson et al., 2012; Grau & Hook, 2006; Ji et al., 2003; Woolf, 2007).

Sensitisation is currently believed to undermine adaptive modulation, the application of SMT may encourage functional adaptive plasticity; through response-outcome dependent mechanisms, promoting functional learning patterns within the CNS. This may in turn, lessen the adverse consequences of uncontrolled stimulation (Crown & Grau, 2001; Ferguson et al., 2012; Grau & Hook, 2006).

Biomechanical changes in articular joint function which are postulated to result from SMT (Evans, 2002), may induce regulatory effects upon afferent discharge (Dishman et al., 2012); furthermore, initiating a ‘break’ in nociceptive feedback loops responsible for reinforcing the maladaptive state of sensitisation. Furthermore, induced effects of SMT may be sufficient to
inhibit maladaptive progression within the spinal cord, allowing adaptive modulatory mechanisms to be restored. Restoring modulation may be observed where improvements in FR have been reported as a result of SMT (DeVocht, Pickar, & Wilder, 2005; Harvey & Descarreaux, 2013; Keller & Colloca, 2000; Lalanne et al., 2009; Lehman & McGill, 2001).

When developing potential SMT treatment methods for chronic LBP, restoration of adaptive modulation may consider a repetitious approach. This could potentially be achieved utilising response-outcome measures previously observed within the instrumental learning model. This model infers that instrumental learning typically requires immediate feedback of information to induce neurological change. When feedback is in some way delayed, learning ability of the CNS deteriorates (Grau & Hook, 2006). Improving sensory afferent feedback through SMT may induce a pattern of ‘retraining’ within the CNS by inhibiting maladaptive feedback resulting from sensitisation and allow an environment for adaptive modulation to be restored. Such restorations in modulation have been identified through FR assessment in conjunction with SMT in LBP individuals in a recent study by Harvey and Descarreaux (2013).

Including ROM assessment pre and post interventions may provide further objective means of identifying potential neuro-muscular changes associated to SMT and inform on postulated relationships between pain and ROM which are currently in debate. Improvements in ROM have been reported in SMT studies with LBP participants (Bicalho et al., 2010; Shum et al., 2013), suggesting a relationship between lumbar flexibility and FR (Hashemirad et al., 2009); however, the methodological oversight of controlling for ROM during baseline assessment, questioning the validity of pervious claims of increased ROM attributed to SMT.

**SMT and ROM and the FRP**

Studies researching associations between manipulation and LBP have identified an inter-relative triad between FR, ROM and pain perception. Initial acute intervention studies report benefits of improved FR and ROM in symptomatic populations, matched against asymptomatic controls (Bicalho et al., 2010; Descarreaux et al., 2010; Martin Descarreaux, Lafond, Jeffrey-Gauthier, Centomo, & Cantin, 2008). Furthermore, improvements in objective ROM methods are observed with the incorporation of finger-floor distance measures (Bicalho et al., 2010). However; in spite of improved ROM methodology, limitations are identified where post intervention ROM values are observed. Bicalho et al.
subsequently reported a mobility increase in the control group despite participants not receiving manipulation. The authors theorised that initial flexion-extension cycles during base-line evaluation could be responsible for acutely increasing ROM, allowing smaller finger-floor distance values the during secondary evaluation following baseline measures. This challenges the hypothesis that the reduction in finger-floor distance in intervention group was a direct result of manipulation. ROM was measured but not controlled for in this study, identifying a significant limitation, as it is unclear if changes in FR were attributable to the increase in ROM or an effect of neuro-muscular alteration. Therefore, it is important to control for ROM as spinal pain has previously been observed to reduce ROM, which concurrently affects EMG and FR (Zedka et al., 1999). Therefore, ROM objectification requires re-evaluation to truly understand its association between FR, pain perception and adaptation to SMT.

**Conclusion**

It appears that SMT may be a suitable treatment method applied to chronic LBP where associated absence of FR and diminished ROM are present; however, understanding the significance of these individual components is still a point of query. The recent study by Bicalho et al. (2010) highlights the possibility of SMT to improve ROM, but with the methodological oversight of lack of ROM controls at baseline, speculation on whether improvements of ROM are an effect of the neuro-muscular changes associate to SMT, or some other unknown mechanism. Significant observations in FR research where ROM is concurrently evaluated warrant further investigation to better understand the potential neuro-muscular influences of SMT where chronic LBP populations exist.
References


30


Controlled Clinical Trial


Section Two: Manuscript
The effects of lumbar spine manipulation on the flexion-relaxation response in chronic low-back pain participants.
**Introduction**

Low back pain (LBP) is a highly prevalent complaint that is routinely encountered by practitioners with an interest in diagnosis and therapy of musculoskeletal conditions (Hoy, Brooks, Blyth, & Buchbinder, 2010; Walker, 2000). Approximately 85% of the population will experience LBP at some stage in their life with 50% of cases recurring within 3 months (Johnson et al., 2010). Of these percentages, approximately 80% of acute LBP presentation resolves spontaneously, regardless of treatment (Andersson, 1999).

Despite a majority of acute LBP conditions reported as resolving within 4 weeks; recurrent episodes are a commonly incurred (Thomas et al., 1999). LBP symptoms lasting longer than 4 weeks but less than 3 months infer a classification of sub-acute conditioning (Karjalainen et al., 2003). Anywhere beyond a 3 month time frame of persistence, indicates a shift towards a chronic status (Andersson, 1999; Koes et al., 2006). Chronic LBP (CLBP) is not regarded as a clinical entity or diagnosis, but rather a symptom in people with different stages of impairment, disability and chronicity. Percentage wise, approximately 85% of chronic LBP cases are classified as being ‘non-specific’ owing to a lack of identifiable pathology through radiographic imaging (Dillingham, 1995; Krismer & van Tulder, 2007). Furthermore, chronic LBP is considered to be multidimensional in presentation representing pathoanatomical, neurophysiological and psychological components (Main & Waddell, 2004).

The pathoanatomical model represents a diagnostic method for chronic LBP where tissue damage of anatomical structures are attributed to onset (Nachemson, 1999). However; this model has previously been unsuccessful in explaining pain and disability in LBP with abnormal findings being commonly observed in pain free populations (Nachemson, 1999; Senbursa, Baltaci, & Atay, 2007).

Neurophysiological modelling of chronic LBP is suggestive of nervous and biochemical interrelationships involved in the regulation of pain conditions. This is observed where neuro-modulatory reflexes are activated at peripheral spinal and cortical levels in adaptation to pain (Flor, Braun, Elbert, & Birbaumer, 1997; Hodges & Moseley, 2003; Hodges et al., 2003). Constituents of this model describe peripheral structures as potential pain generators which progressively mediate chronicity through the development of centrally facilitated sensitisation (Ji et al., 2003; O’Sullivan, 2005; Woolf, 2007). Modulation and progression of sensitisation is discussed in-depth in later sections.
In addition to structural contributors identified in pain development and progression, psychosocial barriers to recovery indicate a strong correlation to chronic LBP (Kendall, 1999; Main & Williams, 2002); supporting a substantial cause of absence from work and associated healthcare costs and compensation (Goossens & Evers, 1997; Linton et al., 2002). A common progression is a reduced rate of individuals returning to usual activity, subsequently incurring substantial loss of income. The identification of effective LBP interventions has largely been unsuccessful (Van Tulder, Koes, & Bouter, 1997); however, SMT in acute intervention studies indicate strong evidence for short-term benefit for LBP (Baker, 2014; Swenson & Haldeman, 2003). Although associations to short-term benefits of SMT are identified, recent reviews on efficacy in chronic LBP treatment, subsequently report little evidence (Assendelft, Morton, Yu, Suttorp, & Shekelle, 2013). Assendelft et al. (2013) systematic review and meta-analysis concluded SMT as being no more effective than other standard forms of treatment including sham manipulation, conventional general practitioner care, analgesics and physical therapy. Despite the less than convincing outcomes for systematic reviews, it would be helpful to better understand the underlying mechanisms of SMT; however, a lack of clarity exists regarding neuro-muscular effects of SMT in people with pain and these effects require further exploration.

The continued utilisation of surface electromyography (sEMG) has provided consistent objective means of evaluating changes in neuro-muscular activity. This method has extended its evaluation to better understanding specific neuro-muscular phenomenon. This includes the flexion relaxation (FR) response which was first reported by Floyd and Silver (1955) and has been used in to investigate the neuro-muscular mechanisms associated to LBP. Recent studies continue to demonstrate that SMT can have a positive effect on pain and FR (Bicalho et al., 2010; Harvey & Descarreaux, 2013). Bicalho et al. (2010) explored the effects of SMT on lumbar ES muscles in chronic LBP participants indicating significant outcomes in pain perception and lumbar ROM. However, there is an inherent limitation in FR research that is often overlooked in intervention studies, and that is the relationship between lumbar ROM and FR (Zedka et al., 1999). Subsequently, improvements in FR that are accompanied by improvements in ROM may be attributable to neuro-muscular changes, or simply, to improved ROM. Without controlling for ROM, the mechanisms of change in FR cannot be confidently inferred. Thus, the mechanism by which an intervention has an effect is unknown.
The present study aims to determine if lumbar HVLA manipulation can induce neuro-muscular changes in FR ROM and pain perception as previously observed in research by Bicalho et al. (2010). In addition, ROM control measures are incorporated in order to better evaluate potential neuro-muscular changes resulting from the intervention.

**Methods**

**Design**

A pseudo-randomized, controlled, counterbalanced cross-over design (See Figure 1). The intervention was a bilateral non-specific high velocity low amplitude (HVLA) thrust manipulation applied to participant’s lumbar spinal region (See Figure 2).

**Participants**

Participants were recruited from members of the general public and from the tertiary education campus at which the study was conducted. Recruitment was achieved using notices posted to an online study recruitment service (http://www.researchstudies.co.nz) and the campus. Participants underwent pre-screening to determine eligibility. Inclusion criteria were: i) male or females aged 18-50 years; ii) presence of LBP reported as ≥30mm on a visual analogue pain scale; reported presence of LBP on more than half the days over previous 3 months or longer. Participants were excluded from this study if they presented with: i) history of back or hip surgery; ii) presence of medical condition that may cause back pain; iii) presence of a contraindication to SMT (Koes et al., 2006); iv) were currently undergoing any other treatment for LBP. All participants gave written informed consent prior to enrollment and the study was approved by the Unitec Research Ethics Committee (UREC No.: 2011-1221).

**Instrumentation**

Control for ROM was applied through the use of a rig designed to act as a physical barrier to trunk flexion during pre-measure FR tasks. The rig was composed of a horizontal plastic strut which was secured to a vertical rule which measured individual finger-floor distance values. The rig ensured that pre intervention flexion measures were matched to ROM measures at baseline and was adjustable to accommodate individual participant ROM (See Figure 3).
Surface Electromyography

Skin Preparation and Electrode Placement

To reduce skin impedance to below 5 kΩ, the skin surface was gently abraded with fine-grade sandpaper (Red Dot, Trace Prep, 3M Corp., MN) and wiped with an alcohol swab. Inter-electrode impedance below this level was considered to be acceptable. To ensure electrodes were placed in the correct region of the lumbar spine, palpation of bony landmarks indicating the L4 and L5 vertebral levels were identified. Disposable pre-gelled Ag-AgCl surface electrodes (Micropore, 3M Corp., MN), with circular conductive pads 1cm in diameter were attached overlaying bi-lateral lumbar ES (5cm lateral to the L4 and L5 spinous processes, approximate to the posterior superior iliac spine and the L1-2 interspace). The electrodes were attached parallel to lumbar ES fiber orientation with an inter-electrode space of 2cm. Application of electrodes required participants to adopt a semi-flexed trunk position to ensure sufficient skin adhesion during flexion-extension tasks. An additional reference electrode was placed over left olecranon process. Following application of electrodes, hypoallergenic medical tape (Micropore, 3M Corp., MN) was applied to ensure secure skin contact, once snap lead were attached to electrodes.

Surface EMG Detection and Recording

Surface EMG signals were detected utilising an Octal Bio Amp (ML138; ADInstruments Pty Ltd., NSW.) differential method, with an input impedance of 200 M Ohms. Recorded data were sampled and processed at a 2 kHz (16 bit) level using the PowerLab® (ML785) data acquisition unit and LabChart 7® software (ADInstruments Pty Ltd., NSW.) with a gain range of 2 mV parallel with a common mode rejection ratio >85db typically (at 60HZ) with a signal to noise ratio of 66.11db. Band-pass filtering of data was between 30Hz (high-pass Finite Infinite Response (FIR) filter with a half-amplitude frequency of 30Hz and transition width of 23Hz) and 500Hz (low-pass FIR filter with a half-amplitude frequency of 500Hz and transition width of 100Hz). A low-pass filter of 30Hz was applied in order to reduce potential electromyographic artifact at the site of lumbar muscle attachment (Drake & Callaghan, 2006). Visual spectrum analysis (1-sec epoch Fast Fourier Transformation) revealed consistency in spikes at 50Hz indicative of uncharacteristic muscle activity, thereupon a 50Hz second-order notch filter with 32 dB attenuation was incorporated to filter noise artefact.
Dependent Variables

Visual Analogue Scale

The VAS was a 100mm horizontal line labelled with ‘no pain’ on the left anchor and ‘worst pain possible’ on the right anchor. Participants were instructed to mark a point on the line which represented their pain intensity. The VAS is an instrument with acceptable validity, reliability, moderate distribution-based responsiveness and good anchor-based responsiveness compared to multi-item questionnaires (De Boer et al., 2004). Due to the subjective nature of pain, self-reports from participants are considered to provide the most valid measure of the experience (Katz & Melzack, 1999).

Finger-floor Distance Measure

Finger-floor distance is a measure for assessing changes in posterior chain ROM when recorded from the standing position during maximum voluntary flexion (MVF). This test is regarded as having excellent validity, reliability and responsiveness in clinical practice and therapeutic trials (Perret et al., 2001) and is identified as providing a good relationship to self-reported disability when assessing LBP populations (Ekedahl, Jönsson, & Frobell, 2012).

Independent variables

HVLA manipulation

Application of bilateral rotational primary leverage HVLA manipulations to participant’s lumbar spinal region were applied as the intervention. Manipulation was applied in the side-lying position. The practitioner, a registered osteopath with 29 years clinical experience in HVLA manipulation applied the intervention.

Practitioner hand contact was placed upon the posterior-lateral aspect of both superior and inferior articular facets of participants T12/L1 segment. Body position faced towards the anterior aspect of the side-lying participant with feet approximately one-and-a-half shoulder-lengths apart with a stance directed slightly cephalad (See Figure 2).
The participant’s lumbar spine was then rotated to a position where the superior articular facets moved posterior to inferior articular facets below. Using the pelvis as a lever, lumbar spinal segments below T12 were rotated towards the practitioner’s position inducing para vertebral tissue tension traversing lumbar facet joints between L1 and L5 spinal segments. Once appropriate pre-manipulation tissue tension was achieved, a primary lever rotational HVLA thrust was applied to the lumbar spine with no intension to achieve cavitation at any particular segment.

**Control condition**

Control group participants were placed in the side-lying position for 30s each side. The interval between sides, and the duration of side-lying positioning matched the average time participants were side-lying during the intervention phase.

**Procedure**

**Baseline Measures**

Participants were instructed to perform an initial round of 5 repetitive FR tasks reflecting as baseline measurements, with recordings taken during a 3s trunk flexion movement, during a 3s relaxation period and a 3s re-extension phase. Participants were guided by an audible metronome indicating the pace to change trunk position in relation to the 3s sEMG recording intervals. Finger-floor distance and FR were recorded at baseline, and following the intervention at both ROM–controlled and ROM-uncontrolled conditions. Pain intensity was recorded using the VAS, before and immediately following the intervention. Average and maximum values for FR and finger-floor distance measures, respectively, were taken from all five FR tasks for evaluation.

Following baseline sEMG and ROM recording, participants were assigned to their respective intervention or control groups by way of block-randomisation counter-balancing to ensure equal numbers in each group. Following the first session, participants underwent a 14-day wash-out period before crossing-over to the alternate group. The same procedures were undertaken at both sessions (See Figure 1).
Data Analysis

Raw data from exported from the data acquisition software into Excel, and was tabulated before importing into statistical software for analysis. A two-way repeated-measures ANOVA with statistical significance set at the p<0.05 level to test for an interaction between time (pre and post intervention) and group (control and intervention) for all outcome variables. Unless stated otherwise, all values are reported as mean (SD). Data analysis was conducted using SPSS (v21) statistical software (SPSS, IBM Corp.).

Results

Twenty respondents were assessed for eligibility at the beginning of the trial (See Figure 1). Due to varying circumstances, 10 applicants were excluded from the trial. Once assessment for eligibility was complete, 10 participants were enrolled into the study. All 10 volunteers completed the study (7 females; 3 males). The mean age of participant’s was 31.3 (8.8) years. A significant interaction of time*group was observed for VAS (p=0.001), revealing a difference between treatment and control interventions, however, there was no significant interaction for time*group for measures of FR or ROM. A significant (p<0.001) time effect was observed for ROM and VAS for both the control and intervention groups (Table 1).

Discussion

The aim of this study was to determine the effect of lumbar HVLA manipulation on neuro-muscular changes in FR, ROM and pain perception in participants presenting with chronic LBP.

The main finding is that HVLA manipulation did not decrease myoelectric activity during maximum trunk flexion. Increases in forward bending ROM were observed between pre and post measure finger-floor distance values, however, this increased range occurred under both intervention and control conditions indicating that ROM changes were most likely an effect of repetition. The present study found that the HVLA intervention had a significant effect on perceived pain. Several previous studies have observed improvements in FR within chronic LBP participants following an acute HVLA intervention (DeVocht et al., 2005; J. M. Fritz et al., 2011; Harvey & Descarreaux, 2013; Keller & Colloca, 2000).
The design of the present study was similar to that of Bicalho et al. (2010) who reported a significant increase in FR after the manipulation intervention but not for the control group. In the present study, there were no significant differences between treatment and control conditions pre and post intervention in FR.

It is unlikely that the differences in FR outcomes between the study by Bicalho et al. (2010) and the present study were owing to the intervention, as both interventions were effective at reducing pain. It is possible that the difference in findings for FR between Bicalho et al. (2010) and present study are owing to differences in how ROM was measured and controlled. This understanding was based on previous research that shows that FR is related to flexion angle and that pain changes both ROM and FR (Zedka et al., 1999). Bicalho et al. (2010) observed improvements in both FR and ROM during repetitive FR tasks but due to the absence of ROM controls, changes in FR could simply have been from changes in ROM. Significant reductions in pain perception were concurrently observed following the intervention in Bicalho et al. (2010) study. Therefore a change in pain could affect a change in ROM which would therefore influence FR. This methodological oversight challenges several previously reported outcomes, as ROM is typically reported to increase (Ahern, Follick, Council, Laser-Wolston, & Litchman, 1988; Bicalho et al., 2010; Harvey & Descarreaux, 2013) or is not measured at all (DeVocht et al., 2005; Dwornik, Kujawa, Bialoszewski, Slupik, & Kiebzak, 2009; Granata, Rogers, & Moorhouse, 2005; Lalanne et al., 2009).

The present study controlled ROM in an effort to isolate neuro-muscular effects of HVLA manipulation. Although ROM-controlled FR studies have established that long-term interventions can restore FR (Neblett et al., 2010), and experimentally it has been observed that FR is immediately affected by the onset of pain (Zedka et al., 1999), no study has explored the immediate effects of an intervention on FR while controlling for ROM (Bicalho et al., 2010; Descarreaux et al., 2010; DeVocht et al., 2005; Harvey & Descarreaux, 2013; Hashemirad et al., 2009; Herzog, Scheele, & Conway, 1999; Lehman & McGill, 2001). Subsequently, there is little convincing evidence that FR can be affected following an acute intervention.
The majority of cases of LBP are mechanical in nature and involve musculoskeletal structures (O’Sullivan, 2005) including muscles, ligaments, dura mater, facet and sacroiliac joints (M. A. Adams, Burton, Bogduk, & Dolan, 2006). The physiological mechanisms of SMT are proposed to affect structures such as theses (Evans, 2002); therefore, if the source of back pain is from a structure that is directly affected by the manipulation we may expect the pain stimulus to be affected. However, nociception from structures contributing to pain experience not under direct mechanical influence may develop from non-peripheral processes such as central sensitisation process (Ferguson et al., 2012; Ji et al., 2003; Woolf, 1983, 2007).

In the present study, it is possible that the intervention was too brief to evoke changes in the pain stimulus; however the perception of the pain may have changed owing to changes in ‘Kinesiophobia’ or fear of movement. This term was first introduced by Kori, Miller, and Todd (1990), describing excessive, irrational and debilitating fear of physical movement which is an effect of feeling vulnerable to pain and the potential of re-injury. This subsequently affects mobility, coordination and strength resulting in significantly diminished levels of activity (Crombez et al., 1999; Vlaeyen & Linton, 2000). Linton et al. (2002) states, patients do not conceive ‘kinesiophobia’ as a psychological form of fear, but instead as a medical problem. Therefore, graded activity exposure from repetitive FR tasks may have helped develop psychological confidence in forward bending movements, resulting in decreased pain perception.

Subsequently, it is unlikely there were peripheral neuro-muscular changes in nociception from the intervention, despite improved pain perception. This would imply that the FR response is contingent upon nociception more so than perception of pain. Although previous studies have demonstrated changes in pain perception following psychological or behavioral interventions (Crombez et al., 1999; Linton et al., 2002), no study has explored the effects of such interventions on pain-related neuro-muscular changes. Therefore, it may be of value to explore this area of research to investigate neuro-muscular changes in LBP following a psychological intervention.
It is also possible that the process of neuro-muscular restoration exhibits a temporal delay. Research investigating the adaptive changes to pain stimuli illustrates a temporal asymmetry between onset and cessation of dysfunction where neurological adaptations to pain are quickly adopted but display greater restoration latency upon pain cessation (Henriksen et al., 2011; Hodges et al., 2003).

A recent study investigating the effects of experimental pain and delayed restoration by Henriksen et al. (2011) induced pain using injection of a hypertonic solution into the infrapatella pad of the knee joint. The authors found that the sustained muscle inhibition post-pain indicates that the immediate adaptive response to knee joint pain in muscle strength is not restored when the pain experience has gone, but in some cases is maintained for at least 20 minutes post-pain (Henriksen et al., 2011). This phenomenon had previously been observed by Hodges et al. (2003) where the effects of experimentally induces LBP indicated that trunk muscle recruitment strategies did not resolve spontaneously in participants with response of the transverse abdominus (TrA) muscle to single arm movements. This delay was observed across the whole group during the follow-up period, with one participant not recovering within 1 hour of pain cessation. In addition, these observed latencies in functional restoration of muscle have also been observed within surgical populations, where functional delays have been noted anywhere between ten and fifteen days post-surgery (Shakespeare, Stokes, Sherman, & Young, 1985). Overall, it appears that the restoration of normal function is delayed for at least 20min (Henriksen et al., 2007). This delay in neuro-muscular restoration may be explained through mechanisms associated to central sensitisation. Central sensitization is a phenomenon associated with changes in the function of the CNS displaying states of increased neuro-muscular excitability with diminished inhibitory response produced within the spinal cord by peripheral noxious inputs. The introduction of noxious mediators produce a local inflammatory response which can induce a lasting increase in neuro-muscular excitability, mediated by cellular mechanisms linked to memory (Ji et al., 2003). This mechanism reflects an increased response to normal stimuli as in the case of allodynia. This central synaptic modification is considered a form of short-term pain memory where nerves innervating muscles and joints produce lasting changes in function which may persist after the pain stimulus is gone (Wall & Woolf, 1984).
The single session of manipulation applied as the intervention in the present study may not have been sufficient stimulus to invoke a learning pattern within the CNS. In order to disrupt the existing maladaptive process and influence FR, longer intervention duration may be required to induce positive plastic changes within the CNS and restore normal function. Subsequently, it may be argued that more time may have been required to observe results owing to this neuro-muscular delay. This argument may be supported through studies that have failed to demonstrate an immediate effect on FR restoration following an acute intervention, despite reported improvements in pain (Bicalho et al., 2010; Lalanne et al., 2009; Ritvanen, Zaproudina, Nissen, Leinonen, & Hanninen, 2007). Further support for this argument is observed where long-term interventions utilising ROM-controlled measures have reported restoration in the FR response (Neblett et al., 2010).

In summary, the intervention applied was effective in reducing pain scores between pre and post manipulation despite no significant change in FR. A strong interaction was observed within and between groups for VAS scores post intervention. As previously illustrated, FR, ROM and pain are inter-connected. Observing significant effects in VAS and ROM supports the effectiveness of the intervention; however, post intervention measures should be evaluated at least a 20 minute period post intervention to compensate for any potential neuro-muscular delay mechanisms. Significant reductions in pain observed in the present study is an encouraging observation in itself and is consistent with findings from Bicalho et al. (2010) who’s study was of similar repeated measure design. Ongoing research should include a study design that implements continued treatments may produce a greater effect.

**Future Research**

Owing to the observed repetition effect on ROM, future studies should conduct longer familiarisation sessions so that ROM plateaus prior to intervention delivery. Future research should also include delayed measures. Findings in previously illustrated research suggests that a time longer than 20 minutes is required (Henriksen et al., 2011) or greater than an hour (Hodges et al., 2003); however, timing would need to consider the duration of the treatment effect. All future research in the area of LBP and FR must include methods to control for ROM to discriminate between restoration of behavioral and neuro-muscular factors. It would be interesting to explore the immediate effect of local analgesics, or investigate the effects of a psychological intervention on changes in ROM and FR.
Limitations

Using immediate measures of FR as an outcome measure may not be appropriate for acute interventions, regardless of the effectiveness of the intervention on other measures. Although improvements were noted in pain perception, there were no observed changes in ROM. As illustrated by Zedka et al. (1999) pain and ROM are inter-related, so it is possible that the magnitude of the observed effect was insufficient to facilitate neuro-muscular changes. This may be owing to the sample recruited or to the intervention. Participants in Bicalho et al. (2010) study reported a much higher VAS value; therefore, a study of similar design needs to be replicated with a more severely affected sample.

Conclusion

Findings from this study demonstrate that an acute HVLA manipulation intervention to the lumbar spine of chronic LBP participants can significantly improve pain perception scores in participants between baseline and post intervention measures. Significant increases in participant ROM was observed in both intervention and control groups; however, magnitude of effect appears to favor changes attributed to the intervention. Although significant improvements in ROM within and between groups were observed, it is suggested that these changes be attributed to repetition of movement during FR tasks as opposed to the intervention itself. No significant changes were observed in FR resulting from the intervention; however, as changes were observed in ROM and pain perception, which are reported as being related to the FR response, it is possible that post intervention measure were taken to soon following manipulation, not allowing enough time to observe neuro-muscular change in FR. This study shows that there appears to be a temporal asymmetry between restoration of function and pain cessation. Observations in delayed muscle function have been reported in experimental pain research following pain cessation. These findings warrant further investigation to better understand the mechanisms associated to pain, muscle function and spinal manipulation.
References


Pickar. (2002). Neurophysiological effects of spinal manipulation. [Comparative Study Research Support, U.S. Gov't, P.H.S.]


Controlled Clinical Trial


Figure 2. The participant’s lumbar spine is rotated to a position where the superior articular facet moved posterior to the inferior articular below. Using the pelvis as a lever, lumbar spinal segments below T12 were rotated towards the practitioner. This induced erector spinae pre-manipulation tension across lumbar facet joints between L1 and L5 spinal segments.

Figure 3. Rig measuring finger-floor distance and controlling participant’s range of motion during flexion-relaxation tasks. The Rig was composed of a horizontal strut which was secured to a vertical ruler. The strut ensured that pre intervention flexion measures were matched to ROM measures at baseline. The Rig was adjustable to accommodate individual participant ROM.
CONSORT diagram (Figure 1)

Assessed for eligibility

Pre-screening:
Excluded $n = 10$

Randomized

Group 1 $n = 10$

Group 2 $n = 10$

Measures: FRR, ROM: Finger-Floor measurements, VAS

Experiment: Bilateral non-specific HVLA manipulation of Lumbar Spine.

Sham intervention: HVLA side-lying position with practitioner contact maintaining sham positioning.

Post-measures (FRR, ROM, VAS)

2-week washout period, prior to cross-over of groups

Pre-measures (FRR, ROM, VAS)

Control: 30 sec side lying each side.

Experiment: Bilateral non-specific HVLA manipulation Lumbar Spine.

Post-measures (FRR, ROM, VAS)
<table>
<thead>
<tr>
<th></th>
<th>VAS</th>
<th>ROM</th>
<th>FR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Post</td>
<td>Baseline</td>
</tr>
<tr>
<td>Control</td>
<td>5.3 (1.89)</td>
<td>4.7 (2.06)***</td>
<td>5.95 (5.98)</td>
</tr>
<tr>
<td>Intervention</td>
<td>5.2 (1.69)</td>
<td>2.5 (1.58)***</td>
<td>6.00 (6.04)</td>
</tr>
<tr>
<td>Interaction</td>
<td>P&lt;0.001</td>
<td>p=0.33</td>
<td>p=0.79</td>
</tr>
</tbody>
</table>

Note: *** denoted significance at the p<.001 level. Interactions are reported for time*group for each measure.

ER= Extension-Relaxation Re-extension, ROM= Range Of Motion, VAS= Visual Analogue Scale.
Appendix B: Information Sheet and Consent Form
Name: ___________________________ Date Of Birth: _______ / _______ / _______

At present are you working?  ☐ Yes, full time
☐ Yes, part time
☐ Not working

If not working, is it because of the pain?  ☐ Yes  ☐ No

Main occupation over the last 12 months: ______________________________

Females Only:

Are you currently pregnant, planning on becoming pregnant in the next 2 months, given birth within the last 6 months or are you currently breastfeeding?  ☐ Yes  ☐ No

Have you been diagnosed with any medical condition?  ☐ Yes  ☐ No

If yes, what?  _______________________________________________________

Do you take any medications?  ☐ Yes  ☐ No

If yes, what/amounts:  ____________________________________________
Do you currently have Low Back Pain?  □ Yes    □ No

Site of pain:  ________________  VRS:  ____/10____

Quality of pain:  ________________  Progression:  ________

Aggravating factors:  ______________________________________________________

Relieving factors:  ______________________________________________________

Daily Pattern:  ______________________________________________________

Mode of onset?  □ Gradual  □ Traumatic  Details:  ____________________________

How long (approximately, in months) have you had Low Back Pain?  _____________

Has your Low Back Pain changed significantly over the last month?  □ Yes    □ No

Associated symptoms:  □ Saddle anaesthesia  □ Overt loss of balance  □ Incontinence

How often did you experience Low Back Pain in the last month?  □ More than half the time
                                                      □ Less than half the time

Do you have pain into your legs?  □ Yes    □ No

If yes, is it sharp, shooting or lancinating pain?  □ Yes    □ No
Does your back pain stop you from doing any regular daily activities? (Examples of activities: running, playing squash, getting out of bed, vacuuming, sitting for longer than 1 hour, playing soccer with your children, gardening, bending down to tie your shoe laces).

☐ No ☐ Yes

If yes, what?

________________________________________________________________________

How do you currently manage your low back pain? (rest/meds/exercise):

________________________________________________________________________

Have you ever had any spinal fractures, infections, tumours or surgery?

☐ Yes ☐ No

If yes, what/when?

________________________________________________________________________

Have you ever been diagnosed with osteoporosis, spinal stenosis or spinal disc injuries?

☐ Yes ☐ No

If yes, what/when?

________________________________________________________________________

Have you had any major injuries or surgeries to the legs?

☐ Yes ☐ No

If yes, what/when?

________________________________________________________________________
Do you have unrelenting pain, which wakes you from your sleep?  ☐ Yes  ☐ No

Do you have difficulty controlling your bowel or bladder?  ☐ Yes  ☐ No
Medical History Questionnaire:

Have back pain (perceived as 3/10 or greater) on more than half the days for the past 3 months or greater

Yes ☐ No ☐ Do not recall ☐

Structural or surgical history of the back or hip region

Yes ☐ No ☐ Do not recall ☐

Other medical and or physical conditions that may affect back pain or treatment efficacy

Yes ☐ No ☐ Do not recall ☐

Currently undergoing any physical treatments

Yes ☐ No ☐ Do not recall ☐

Serious low back injuries

Yes ☐ No ☐ Do not recall ☐

Underlying spinal pathologies:

Lumbar spondylosis

67
<table>
<thead>
<tr>
<th>Condition</th>
<th>Yes</th>
<th>No</th>
<th>Do not recall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disc herniation</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Spinal canal stenosis</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Cauda equine</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Lumbar spine vertebrae fracture</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Pregnancy or given birth in the last 6 months</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>
The Effects of Lumbar Spine Manipulation on the Flexion-Relaxation Response in Chronic Low-Back Pain Participants

This research project aims to evaluate if lumbar spinal joint manipulation effects surface electromyography (sEMG) reading in lumbar erector spinae muscles of the low-back and its effectiveness at correcting dysfunctional muscle activity and reducing pain.

Name of Participant: _______________________________  D.O.B : _____ / ____ / ____

I have been given and have understood an explanation of this research project.

I confirm that I have read and understand the information sheet.

I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

I understand that I may withdraw myself (or any information I have provided) from this project (before data collection and analysis is complete) without having to give reasons or without penalty of any sort.

I understand that any information I provide will be kept confidential to the researcher and the supervisor.

I understand the published results will not use my name, and that no opinions will be attributed to me in any way that will identify me.

I understand that the data I provide will not be used for any other purpose or released to others without my written consent.
I understand that I can see the finished research document.

I have had time to consider everything and I give my consent to be a part of this study.

I know whom to contact if I have any questions or concerns about this study.

I agree to take part in the above research study.

Participant Signature: ___________________________    Date: _____ / _____ / _____

Study explained by: _____________________________

Signature: _____________________________    Date: _____ / _____ / _____
Appendix C: Ethical Approval
Nicolas Ritter  
34 Kiernan Place  
Kelston  
Auckland  
21.11.2011

Dear Nicolas,

Your file number for this application: 2011-1221  
Title: An Investigation into the effects of lumbar spinal joint manipulation on muscle activity, range of motion and pain perception in subjects presenting with non-specific chronic low-back pain.

Your application for ethics approval has been reviewed by the Unitec Research Ethics Committee (UREC) and has been approved for the following period:

Start date: 16.11.11  
Finish date: 16.11.12

Please note that:

1. The above dates must be referred to on the information AND consent forms given to all participants.

2. You must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.

Yours sincerely,

Scott Wilson  
Deputy Chair, UREC

Cc: Jaime Mannion  
Cynthia Almeida
Nicolas Ritter
34 Kiernan Place
Kelston
Auckland

22.3.12

Dear Nicolas,

Re: Request for amendments

Your file number for this application: 2011-1221
Title: An Investigation into the effects of lumbar spinal joint manipulation on muscle activity, range of motion and pain perception in subjects presenting with non-specific chronic low-back pain.

Your request for amendments to your research project has been reviewed by the Unitec Research Ethics Committee (UREC) and has been approved for the following period:

Start date: 22.3.12
Finish date: 16.11.12

Please note that:

1. The above dates must be referred to on the information AND consent forms given to all participants.

2. You must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.

Yours sincerely,

Scott Wilson
Deputy Chair, UREC

Cc: Jaime Mannion
Cynthia Almeida
Appendix D: The Journal of Electromyography and Kinesiology: Guide for Authors
Journal of Electromyography and Kinesiology

The *Journal of Electromyography and Kinesiology* aims to provide a single, authoritative forum for the publication of original research and clinical studies on muscle contraction and human motion through combined or separate mechanical and electrical detection techniques. Some of the key topics covered include: control of movement; muscle and nerve properties; electrical stimulation; sports and exercise; rehabilitation; muscle fatigue; joint biomechanics; motion analysis; measures of human performance; neuro-muscular diseases; physiological modelling; posture and movement. The Journal welcomes the submission of original papers, reviews and letters to the Editors. The Journal will also publish book reviews and a calendar of forthcoming events. Please note that, at the discretion of the Editor in Chief, some papers may be accepted for online publication only.

**Open Access**

This journal offers authors two choices to publish their research:

1. **Open Access**
   - Articles are freely available to both subscribers and the wider public with permitted reuse
   - An Open Access publication fee is payable by authors or their research funder

2. **Subscription**
   - Articles are made available to subscribers as well as developing countries and patient groups through our access programs ([http://www.elsevier.com/access](http://www.elsevier.com/access))
   - No Open Access publication fee

All articles published Open Access will be immediately and permanently free for everyone to read and download. Permitted reuse is defined by your choice of one of the following Creative Commons user licenses:

*Creative Commons Attribution-Non Commercial-ShareAlike (CC BY-NC-SA):* for non-commercial purposes, lets others distribute and copy the article, to create extracts, abstracts and other revised versions, adaptations or derivative works of or from an article (such as a translation), to include in a collective work (such as an anthology), to text and data mine the article, as long as they credit the author(s), do not represent the author as endorsing their adaptation of the article, do not modify the article in such a way as to damage the author's honor or reputation, and license their new adaptations or creations under identical terms (CC BY NC SA).
**Creative Commons Attribution-NonCommercial-NoDerivs (CC-BY-NC-ND):** for non-commercial purposes, lets others distribute and copy the article, and to include in a collective work (such as an anthology), as long as they credit the author(s) and provided they do not alter or modify the article.

**Creative Commons Attribution (CC-BY):** available only for authors funded by organizations with which Elsevier has established an agreement. For a full list please see [http://www.elsevier.com/fundingbodies](http://www.elsevier.com/fundingbodies)

Elsevier has established agreements with funding bodies. This ensures authors can comply with funding body Open Access requirements, including specific user licenses, such as CC-BY. Some authors may also be reimbursed for associated publication fees. [http://www.elsevier.com/fundingbodies](http://www.elsevier.com/fundingbodies)

To provide Open Access, this journal has a publication fee which needs to be met by the authors or their research funders for each article published Open Access. Your publication choice will have no effect on the peer review process or acceptance of submitted articles. The Open Access publication fee for this journal is **$3000 USD**, excluding taxes.

Learn more about Elsevier's pricing policy [http://www.elsevier.com/openaccesspricing](http://www.elsevier.com/openaccesspricing)

**PUBLICATION CONDITION**

A manuscript submitted to this journal can only be published if it (or a similar version) has not been published and will not be simultaneously submitted or published elsewhere. A violation of this condition is considered as fraud, and will be answered by appropriate sanctions against all authors. Two manuscripts are considered similar if their subjects concern the same hypothesis, question or goal, addressed with the same scientific methodology.

**REFEREEING**

All contributions are read by two or more referees to ensure both accuracy and relevance, and amendments to the script may thus be required before final acceptance. On acceptance, contributions are subject to editorial amendment to suit house style.
AUTHORSHIP

All authors should have made substantial contributions to all of the following: (1) the conception and design of the study, or acquisition of data, or analysis and interpretation of data, (2) drafting the article or revising it critically for important intellectual content, (3) final approval of the version to be submitted.

CHANGES TO AUTHORSHIP

This policy concerns the addition, deletion, or rearrangement of author names in the authorship of accepted manuscripts:

Before the accepted manuscript is published in an online issue: Requests to add or remove an author, or to rearrange the author names, must be sent to the Journal Manager from the corresponding author of the accepted manuscript and must include: (a) the reason the name should be added or removed, or the author names rearranged and (b) written confirmation (e-mail, fax, letter) from all authors that they agree with the addition, removal or rearrangement. In the case of addition or removal of authors, this includes confirmation from the author being added or removed. Requests that are not sent by the corresponding author will be forwarded by the Journal Manager to the corresponding author, who must follow the procedure as described above. Note that: (1) Journal Managers will inform the Journal Editors of any such requests and (2) publication of the accepted manuscript in an online issue is suspended until authorship has been agreed.

After the accepted manuscript is published in an online issue: Any requests to add, delete, or rearrange author names in an article published in an online issue will follow the same policies as noted above and result in a corrigendum.

ACKNOWLEDGEMENT OF OTHER CONTRIBUTORS

All contributors who do not meet the criteria for authorship as defined above should be listed in an acknowledgements section. Examples of those who might be acknowledged include a person who provided purely technical help, writing assistance, or a department chair who provided only general support. Authors should disclose whether they had any writing assistance and identify the entity that paid for this assistance.
CONFLICT OF INTEREST

"Conflict of interest statement" all authors must disclose any financial and personal relationships with other people or organisations that could inappropriately influence (bias) their work. Examples of potential conflicts of interest include employment, consultancies, stock ownership, honoraria, paid expert testimony, patent applications/registrations, and grants or other funding. If there are no conflicts of interest, the authors should state there are none.

ROLE OF THE FUNDING SOURCE

All sources of funding should be declared as an acknowledgement at the end of the text. Authors should declare the role of study sponsors, if any, in the study design, in the collection, analysis and interpretation of data; in the writing of the manuscript; and in the decision to submit the manuscript for publication. If the study sponsors had no such involvement, the authors should so state.

PREPARATION OF SCRIPTS

All publications will be in English. Authors whose 'first' language is not English should arrange for their manuscripts to be written in idiomatic English before submission. Please also ensure that your manuscript has been thoroughly checked for errors prior to submission.

Language Editing: International Science Editing and Asia Science Editing can provide English language and copyediting services to authors who want to publish in scientific, technical and medical journals and need assistance before they submit their article or, it is accepted for publication. Authors can contact these services directly: International Science Editing http://www.internationalscienceediting.com and Asia Science Editing http://www.asiascienceediting.com or, for more information about language editing services, please contact authorsupport@elsevier.com who will be happy to deal with any questions.

Please note Elsevier neither endorses nor takes responsibility for any products, goods or services offered by outside vendors through our services or in any advertising. For more information please refer to our terms & conditions http://authors.elsevier.com/terms_and_conditions.html.
You should have your contribution typed in double-line spacing, on one side only of A4 paper. Do not underline anything and leave wide margins. Please also add line numbers to your submitted manuscript (e.g. 5, 10, 15 etc.) and number every page.

EMG data should be collected and presented according to the 'Standards for Reporting EMG Data' printed at the back of each issue of this journal.

All authors should sign a cover note to acknowledge that they have read, and approve of, the content of the manuscript as submitted.

SUBMISSIONS

Authors are requested to submit their original manuscript and figures online via http://ees.elsevier.com/jek. This is the Elsevier web-based submission and review system. You will find full instructions located on this site. Please follow these guidelines to prepare and upload your article. Once the uploading is done, the system automatically creates an electronic pdf proof, which is then used for reviewing. All correspondence, including notification of the Editor's decision and requests for revisions, will be managed via this system. Paper copies and email submissions are also currently accepted. Please submit to:

For the Americas, Europe, Africa and the Middle East:

Professor M. Solomonow, Professor & Director, Bioengineering Division & Musculoskeletal Disorders Research Laboratory, University of Colorado Health Sciences Center, Mailstop 8343, PO Box 6511, Aurora, CO., 80045, USA; Tel.: (303) 724-0383, Fax: (303) 724-0394

For the Far East and Australia:

Professor T. Moritani, Laboratory of Applied Physiology, The Graduate School of Environmental Studies, Kyoto University, Sakyoku, Kyoto 606, Japan; Tel: 81 75 753 6888, Fax: 81 75 753 6734

No page charges are made to authors for material published.

Arrangement of papers

JEK now accepts original articles within a word limit of 5,000 words (including title page, abstract, text, references & figure legends). Reviews and special articles (keynote lectures or a Special issue articles) are exempted from this limit. You should arrange your contribution in the following order:
1. Title page including the article title, author(s), affiliation(s), keywords and one author identified for correspondence

2. A 200 word abstract outlining the purpose, scope and conclusions of the paper

3. The text, suitably divided under headings

4. Acknowledgements (if any)

5. References

6. Tables (each on separate sheet)

7. Captions to illustrations (grouped on a separate sheet or sheets)

8. Illustrations, each on a separate sheet containing no text.

All submissions should be accompanied by a declaration signed by each author that the paper has not been previously published or submitted for consideration elsewhere.

TEXT

Subdivide your paper in the simplest way possible, consistent with clarity using the standard format of introduction, methods, results and discussion.

TABLES

Number tables consecutively throughout the paper (with Arabic numerals) referring to them in the text as Table 1, Table 2 etc. with a caption at the top of each table. Avoid the use of vertical rules. Tables should not duplicate results presented in graphs.

ILLUSTRATIONS

All illustrations should be identified with the author's name and figure number marked in pencil.

Line illustrations

Articles may be published more quickly if illustrations are supplied to the required standards, authors should not be deterred if they are unable to meet these standards as illustrations can be redrawn in-house. The originals must be supplied on separate sheets, with two photocopies. Illustrations will be reduced in size photographically, typically to fit one or two columns of the journal and this should be borne in mind to ensure that lines and lettering
remain clear when reduced. If you label the original illustrations do so in black ink using a suitable stencil. Lower case letters should be used throughout, with an initial capital letter for the first word only. If suitable stencils are unavailable label a photocopy, not the original illustrations, and our studio will complete the work to the correct standard. If your illustrations are computer-generated follow the lettering standards as above and supply the blackest possible laser printout.

For full instructions on the electronic submission of artwork, please visit: http://ees.elsevier.com/jek.

Graphs

The minimum amount of descriptive text should be used on graphs and drawings (label curves, points, etc, with single-letter symbols). Descriptive matter should be placed in the figure caption. Scale grids should not be used in graphs, unless required for actual measurements. Graph axes should be labelled with variables written out in full, along the length of the axes, with the unit in parentheses (for example, Time(s)). A table is usually more satisfactory for recording data.

Photographs

Supply glossy, black and white, unmounted prints or 35 mm transparencies, plus two photocopies. A scale, where appropriate, should be marked on the photographs or included in the caption.

Colour Illustrations

If, together with your accepted article, you submit usable colour figures then Elsevier will ensure, at no additional charge, that these figures will appear in colour on the web (e.g., Science Direct and other sites) regardless of whether or not these illustrations are reproduced in colour in the printed version. For colour reproduction in print, you will receive information regarding the costs from Elsevier after receipt of your accepted article. For further information on the preparation of electronic artwork, please see http://ees.elsevier.com/jek. Please note: Because of the technical complications which can arise by converting colour figures to 'grey scale' (for the printed version should not opt for colour in print) please submit in addition usable black and white prints corresponding to all the colour illustrations. Submit colour illustrations as original photographs high-quality computer prints or transparencies,
close to the size expected in publication, or as 35 mm slides. Polaroid colour prints are not suitable.

REFERENCES

The reference list should be constructed alphabetically. Where more than one reference has the same first author, use the next named author to construct the list alphabetically. For identical author groups, list the references by date. References should be cited in the text using the first author name plus the year of the paper, eg Solomonow et al, 2004, in square brackets. References should be in the following form:

*Journal article*


*Book*


*Article or chapter in edited book*


Please ensure that references are complete, in that they include where relevant, author's name, article or book title, volume and issue number, publisher, year and page reference. Journal titles should appear in full.

UNITS AND ABBREVIATIONS

SI units and their accepted abbreviations should be used.

RANDOMISED CONTROLLED TRIALS

All randomised controlled trials submitted for publication in the journal should include a completed Consolidated Standards of Reporting Trials (CONSORT) flow chart. Please refer to the CONSORT statement website at [http://www.consort-statement.org](http://www.consort-statement.org) for more information. The Journal of Electromyography and Kinesiology has adopted the proposal from the International Committee of Medical Journal Editors (ICMJE) which require, as a
condition of consideration for publication of clinical trials, registration in a public trials registry. Trials must register at or before the onset of patient enrolment. The clinical trial registration number should be included at the end of the abstract of the article. For this purpose, a clinical trial is defined as any research project that prospectively assigns human subjects to intervention or comparison groups to study the cause-and-effect relationship between a medical intervention and a health outcome. Studies designed for other purposes, such as to study pharmacokinetics or major toxicity (e.g. phase I trials) would be exempt. Further information can be found at www.icmje.org.

ETHICS

Work on human beings that is submitted to the Journal should comply with the principles laid down in the Declaration of Helsinki; Recommendations guiding physicians in biomedical research involving human subjects. Adopted by the 18th World Medical Assembly, Helsinki, Finland, June 1964, amended by the 29th World Medical Assembly, Tokyo, Japan, October 1975, the 35th World Medical Assembly, Venice, Italy, October 1983, and the 41st World Medical Assembly, Hong Kong, September 1989. The manuscript should contain a statement that the work has been approved by the appropriate ethical committees related to the institution(s) in which it was performed and that subjects gave informed consent to the work. Studies involving experiments with animals must state that their care was in accordance with institution guidelines. Patients' and volunteers' names, initials, and hospital numbers should not be used.

CHECKLIST

Have you told readers, at the outset, what they might gain by reading your paper?

Have you made the aim of your work clear?

Have you explained the significance of your combination?

Have you set your work in the appropriate context by giving sufficient background (including a complete set of relevant references) to your work?

Have you addressed the question of practicality and usefulness?

Have you identified future developments that may result from your work?

Have you structured your paper in a clear and logical fashion?
COPYRIGHT

Upon acceptance of an article, authors will be asked to sign a "Journal Publishing Agreement" (for more information on this and copyright see http://ees.elsevier.com/jek. Acceptance of the agreement will ensure the widest possible dissemination of information. An e-mail (or letter) will be sent to the corresponding author confirming receipt of the manuscript together with a "Journal Publishing Agreement" form. If excerpts from other copyrighted works are included, the author(s) must obtain written permission from the copyright owners and credit the source(s) in the article. Elsevier has preprinted forms for use by authors in these cases: contact Elsevier's Rights Department, Philadelphia, PA, USA: Tel. (+1) 215 238 7869; Fax (+1) 215 238 2239; e-mail healthpermissions@elsevier.com. Requests may also be completed online via the Elsevier homepage (http://www.elsevier.com/locate/permissions).

PROOFS

One set of page proofs in PDF format will be sent by e-mail to the corresponding author (if we do not have an e-mail address then paper proofs will be sent by post). Elsevier now sends PDF proofs which can be annotated; for this you will need to download Adobe Reader version 7 available free from http://www.adobe.com/productsacrobat/readstep2.html. Instructions on how to annotate PDF files will accompany the proofs.

The exact system requirements are given at the Adobe site: http://www.adobe.com/productsacrobat/acrreqsystemreqs.html#70win. If you do not wish to use the PDF annotations function, you may list the corrections (including replies to the Query Form) and return to Elsevier in an e-mail. Please list your corrections quoting line number. If, for any reason, this is not possible, then mark the corrections and any other comments (including replies to the Query Form) on a printout of your proof and return by fax, or scan the pages and e-mail, or by post. Please use this proof only for checking the typesetting, editing, completeness and correctness of the text, tables and figures. Significant changes to the article as accepted for publication will only be considered at this stage with permission from the Editor. We will do everything possible to get your article published quickly and accurately. Therefore, it is important to ensure that all of your corrections are sent back to us in one communication: please check carefully before replying, as inclusion of any subsequent
corrections cannot be guaranteed. Proofreading is solely your responsibility. Note that Elsevier may proceed with the publication of your article if no response is received.

**OFFPRINTS**

The corresponding author, at no cost, will be provided with a PDF file of the article via e-mail or, alternatively, 25 free paper offprints. The PDF file is a watermarked version of the published article and includes a cover sheet with the journal cover image and a disclaimer outlining the terms and conditions of use. Additional paper offprints can be ordered by the authors. An order form with prices will be sent to the corresponding author.

**PREPARATION OF SUPPLEMENTARY DATA**

Elsevier now accepts electronic supplementary material to support and enhance your scientific research. Supplementary files offer the author additional possibilities to publish supporting applications, movies, animation sequences, high-resolution images, background datasets, sound clips and more. Supplementary files supplied will be published online alongside the electronic version of your article in Elsevier web products, including Science Direct: [http://www.sciencedirect.com](http://www.sciencedirect.com). In order to ensure that your submitted material is directly usable, please ensure that data is provided in one of our recommended file formats. Authors should submit the material in electronic format together with the article and supply a concise and descriptive caption for each file. For more detailed instructions please visit: [http://ees.elsevier.com/jek](http://ees.elsevier.com/jek).

**AUTHOR ENQUIRIES**

For enquiries relating to the submission of articles (including electronic submission where available) please visit: [http://ees.elsevier.com/jek](http://ees.elsevier.com/jek).

Contact details for questions arising after acceptance of an article, especially those relating to proofs, are provided after registration of an article for publication.

*Audio Slides*

The journal encourages authors to create an AudioSlides presentation with their published article. AudioSlides are brief, webinar-style presentations that are shown next to the online article on ScienceDirect. This gives authors the opportunity to summarize their research in their own words and to help readers understand what the paper is about. More information and examples are available at [http://www.elsevier.com/audioslides](http://www.elsevier.com/audioslides). Authors of this journal
will automatically receive an invitation e-mail to create an Audio Slides presentation after acceptance of their paper.