An investigation into the effect of experimental knee pain on quadriceps muscle torque during different contraction types

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Declaration

Name of candidate: Lana Torenvlied-Whiting

This thesis titled **'An investigation into the effect of experimental knee pain on quadriceps muscle torque during different contraction types'** 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
- Ethics Approved by the Unitec Ethics Committee (Reference: 2013-1055)

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

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Preface

This 90-credit thesis is submitted in partial fulfillment of the requirement for the Master of Osteopathy degree at Unitec Institute of Technology.

The following thesis is divided into three sections;

1. Literature Review: This literature review commences with a background on pain. Muscle strength and the effect of pain on muscle strength is discussed. Experimental pain modalities are also incorporated. Different contraction types, specifically eccentric are reviewed and a gap in the literature where future research is needed is identified. This literature review provides a basis for the manuscript.

2. Manuscript, titled 'Peak knee extension torque is similar for all muscle contraction types during experimentally induced pain'. This manuscript investigates the effect of experimental pain on eccentric muscle contractions in healthy participants. This manuscript has been prepared in accordance with the format specified for submission to the *Journal of Electromyography and Kinesiology* with the exception of including figures in the body.

3. Appendices: The appendices includes ethics documentation, participant information and consent forms, self-report measures used in the manuscript and the format required for Manuscript submission.

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List of abbreviations

Chronic low back pain	CLBP
Centre meter	cm
Degree	Deg
Degrees per second	deg/s
Electromyography	EMG
Gauge	G
Kilohertz	kHz
Maximal voluntary contraction	MVC
Newton meters	Nm
Numeric Rating Scale	NRS
Pain Catastrophising Scale	PCS
Second	s
Surface Electromyography	SEMG
Δpain	During pain minus baseline
Δpost	Post-pain minus baseline

Section I: Literature review

1. Introduction

There is extensive literature describing the effects of pain on muscle contractions with a unanimous finding of reduced muscle strength (Graven-Nielsen & Arendt-Nielsen, 2008; Hodges, 2011; Lund, Donga, Widmer, & Stohler, 1991). Interestingly, the vast majority of studies have exclusively investigated concentric and isometric contractions (Almosnino, Stevenson, Bardana, Diaconescu, & Dvir, 2012). The few studies that have included eccentric contractions have displayed results that call to question the current generalisation of "muscle weakness" associated with pain (Chaler et al., 2007; Dvir & Keating, 2003; Henriksen, Rosager, Aaboe, Graven-Nielsen, & Bliddal, 2011; Shirado, Ito, Kaneda, & Strax, 1995; Zedka, Prochazka, Knight, Gillard, & Gauthier, 1999). As it is well established that eccentric contractions have a different neural control strategy compared with concentric contractions and are subsequently under less volitional control (Enoka, 1996), it is plausible that eccentric muscle contractions may not be affected by pain in the same way as concentric and isometric contractions.

This following review intends to discuss literature concerning the effect of experimental pain on eccentric muscle contractions. A brief introduction to pain and the physiology of pain will be included, followed by a detailed description of the effect of pain on muscle contractions and motor recruitment during pain. The different types of muscle contractions will be outlined including an in depth discussion regarding eccentric muscle contractions and volitional underperformance.

2. Pain

2.1. Definition

Pain is defined as an "unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (International Association for the Study of Pain, 2014). Pain can be a persistent and debilitating problem that is a prevalent complaint in clinical practice. Pain complaints are commonly associated with dysfunction, injury and disease resulting in reduced quality of life and impaired activities of daily living (Pfizer, 2013). In addition to these physical aspects, social and psychological confounders

also contribute to the pain state (Godfrey, 2005; Moseley, 2003) thus pain is a complex experience (Bogduk, 2002).

2.2. Pain, the economic burden

It is reported that one in eight New Zealanders report experiencing chronic pain (Pfizer, 2013). Chronic pain contributes to lost productivity due to work absentism, compensation claims and health system costs thus being a financial burden on the healthcare system. The expenditure can be divided into direct and indirect costs. Direct costs can include hospital or general practitoner visits, diagnostic investigation including imaging, medication and other therapy. Indirect costs are more difficult to accurately quantify and include expenses such as lost productivity and wage loss due to being absent from work. It is estimated that in Australia, these costs are approximately $10,000 per person who suffers from chronic pain per annum (Access Economics Pty Limited, 2007).

2.3. Physiology of pain

Pain is a multifactorial experience that involves complex underlying processes (Melzack, 2001). There are normally four key neurological processes that occur which result in pain: Transduction, Peripheral Transmission, Central Transmission and Modulation (Holdcroft & Jaggar, 2005). Nociception (the reception of a noxious stimuli), occurs in response to mechanical, chemical or thermal stimulation triggering nociceptors to create an action potential (Garrett & McShane, 1999). This process is known as transduction. For peripheral transmission nociceptive signals are transmitted via afferent fibers into the dorsal root ganglion in the Central Nervous System (CNS) (Moseley, 2003). When the afferent signal has reached the CNS and been conveyed to higher brain centers the individual may become consciously aware of nociception which results in the perception of pain, this is termed ‘central transmission’. Modulation occurs when the nociceptive signal is enhanced or inhibited (Loeser & Melzack, 1999). This occurs multiple times at all stages throughout the pain process. It is important to highlight that pain is only consciously recognized after central transmission has occurred. The perception of pain is an individual experience and depends on individual interpretation of pain, modulation of the electrical signal, pain association and

memory of past experiences (Godfrey, 2005; Melzack, 2001; Moseley, 2003). Subsequently, the pain experience may not accurately represent the damage that has occurred.

3. Motor recruitment during pain

Pain and muscle strength are often evaluated in clinical assessments of muscle function. Clinical intervention of pain often involves addressing motor recruitment patterns alongside manual therapy and techniques to reduce psychological influence (Hodges, 2011). Despite this, many mechanisms of motor recruitment adaptation during pain remains controversial (Hodges, 2011; Tucker et al., 2009). This raises questions regarding the validity of current pain intervention and highlights the necessity of investigating muscle function during pain.

3.1. 'Vicious cycle' theory

The pain adaptation theory and 'vicious cycle' model have been historically used to explain motor recruitment during pain (Peck, Murray, & Gerzina, 2008). Roland (1986), first suggested that there is muscle hyperactivity in painful states. This muscle hyperactivity causes muscle fatigue which results in pain and further hyperactivity, thus a 'vicious cycle' is created. This theory was initially widely accepted however, multiple studies have found no change or decreased muscle activity during pain (Kravitz, Moore, & Glaros, 1981; Roland, 1986; Zedka et al., 1999). Despite this controversy, Peck et al. (2008) state that clinical intervention for pain often involves targeting the 'vicious cycle' as a part of pain management. However, specific interventions were not discussed.

3.2. Pain adaptation model

A more recently proposed theory is Lund's pain adaptation model that explains that there is reduced motor recruitment in the presence of pain and an increase during rest (GravenNielsen, Svensson, & Arendt-Nielsen, 1997; Lund et al., 1991), concurrently the antagonist muscle to the movement is facilitated. It is suggested that this adaptation is protective and may limit further injury. Despite this claim, several studies show that there is variability in motor recruitment during pain (Hodges, 2011; Hodges, Ervilha, & Graven-

Nielsen, 2008; Tucker, Butler, Graven-Nielsen, Riek, & Hodges, 2009). These findings highlight shortcomings in both 'viscous cycle' and the pain adaptation models.

To provide further insight into this theory, Tucker et al. (2009), investigated whether motor recruitment is altered during pain. The authors found that during pain, low threshold motor recruitment was reduced and new motor units were recruited to maintain force. Hodges, (2011) recently published a re-conceptualised theory of muscle adaptation to pain. This theory proposes that rather than being inhibited, motor recruitment during pain is reorganized within and between muscle groups. This reorganization of muscle activity appears to be task dependent. Hodges, (2011), suggest that pain changes mechanical behavior (e.g., modified movement and stiffness) which may be owing to protective mechanisms used to prevent further damage (Hodges, 2011; Tucker et al., 2009). This adaptation may be beneficial in the short term, however, potentially has long term negative consequences. For instance, behavioral (i.e., fear-avoidance beliefs) and mechanical (modified movement and stiffness) adaptations as a result of pain can cause increased mechanical load on other structures that can result in more pain and injury reoccurrence (Hodges, 2011; Hodges & Tucker, 2011).

Although attractive, Hodges (2011) re-conceptualisation does not provide an explanation for the unique activation found in trunk muscles that suggests that no single theory can explain pain adaptation (Hodges, 2011). Alternatively it has been proposed that pain is a unique individual experience that is task dependent (Falla, Farina, Dahl, & Graven-Nielsen, 2007) and based on previous pain experience and anthropometrics (Hodges, 2011). In the shorter term, the pain response is likely to be a protective mechanism that involves the CNS and complex higher brain processes. Thus rather than one model to explain motor adaptation during pain, a combination of the 'vicious cycle' and pain adaptation theories may exist (Hodges, 2011; Tucker et al., 2009).

4. Muscle strength during pain

Previous studies have shown that pain typically decreases maximal voluntary contraction strength (Henriksen et al., 2011; Palmieri-Smith, Villwock, Downie, Hecht, & Zernicke, 2013; Wassinger, Sole, & Osborne, 2012). This adaptation is probably owing to both neural (Hodges, 2011; Tucker et al., 2009) and behavioral (Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995) factors. A common behavioral observation is reduced trunk flexion in people with low back pain. Zedka et al., (1999) used an experimental pain model and showed decreased trunk flexion with experimental pain. When range of movement was controlled, altered muscle activation during movement phases persisted. This indicates an involuntary component that is likely to be a protective mechanism, reducing the likelihood of joint damage (Farina, Arendt-Nielsen, & Graven-Nielsen, 2005). Furthermore, when the amount of muscle force is controlled in both pain and pain-free conditions, alternate motor strategies are employed, indicating a mechanism that influences individual motor unit recruitment regardless of volitional effort (Hodges, 2011). The combination of behavioral and neural adaptations to pain presents a challenge to the clinician when attempting to assess and treat causes of activity restriction disability, and can confound outcomes of comparison studies between symptomatic and asymptomatic groups.

5. Arthrogenic Muscle Inhibition

Arthrogenic muscle inhibition (AMI) is the reduced ability to activate muscles due to sustained neural inhibition after pain has dissipated (Rice & McNair, 2010). This means that after an injury occurs, there may be a limited ability to contract muscles, despite there being no pain experienced. This is a commonly observed phenomenon in exercise rehabilitation and can be a significant obstacle to recovery progress (Hopkins & Ingersoll, 2010). The exact mechanism of inhibition is unknown, however, many pathways and central adaptations have been associated with AMI.

Firstly, it is suggested that during pain, central adaptations can occur in ascending pathways where modulation occurs without nociception being perceived (Henriksen, Alkjær, Simonsen, & Bliddal, 2009). Additionally, AMI could be a result of a short delay in the neural inhibition

networks (inhibitory interneurons) disconnecting, hence the delayed effect. Secondly, when nociception occurs, the flexion reflex occurs where extension is inhibited and a flexion response occurs (Sterling, Jull, & Wright, 2001). Wide dynamic range neurons aid in mediation of the flexion reflex (You, Colpaert, & Arendt-Nielsen, 2008). When joint inflammation occurs, there is an increase in afferent signals being transmitted to the central nervous system. This results in hyper-excitability of wide dynamic range neurons which increases the flexion reflex subsequently reducing motor neuron pool excitability and inhibiting extension (Rice & McNair, 2010). Thirdly, the gamma loop reflex pathway has been found to be dysfunctional when tissue damage occurs (Konishi, Fukubayashi, & Takeshita, 2002). Residual muscle inhibition, after pain has dissipated may be an inappropriate adaptation and may contribute to development of chronic pain conditions (Henriksen et al., 2011). Experimental pain can be used to simulate some aspects of the pain experience and may provide more comprehensive explanations into the adaptations occurring during AMI.

6. Experimental pain

Due to the diverse nature of pain, identifying the causal mechanisms of observed strength and functional deficits in pain populations can be problematic; thus experimentally induced pain is often used to simulate the pain experience (Capra & Ro, 2004). This is essential in being able to explore pain conditions and mechanisms in a controlled environment (Reddy, Naidu, Rani, & Rao, 2012). Moreover, experimental pain allows participants to be their own controls, thereby eliminating between-subject variability and controlling for complex psychosocial factors associated with pathological pain conditions. There are multiple different experimental pain methods including mechanical, chemical, ischemic, thermal, electrical and exercise induced. The most viable modalities, including their strengths and weaknesses, are reviewed in the next sections.

6.1. Strengths and limitations of different pain modalities

Mechanical pain, using pressure stimuli and ischemic stimulation, are non-invasive methods, however, both evoke a non-specific pain response (Staahl & Drewes, 2004). Additionally, ischemic stimulation has a long duration (up to 2 hours) of pain response and has an inherent

risk of tissue necrosis. Electrical stimulation is a non-invasive, reliable source of muscle and referred pain, however, is not nociceptive specific and causes concurrent muscle fibre activation (Olesen, Andresen, Staahl, & Drewes, 2012). Thermal stimulation (including hot, cold and ice water) is also non-invasive. Its limitations are that it can result in tissue damage, cutaneous nociceptor activation and has a cardiovascular risk associated with vaso-vagal events (Staahl & Drewes, 2004). Exercise induced pain is non-invasive, although can result in delayed onset muscle soreness 24-48 hours later, muscle damage and has an unpredictable pain response. Chemical stimulation, specifically hypertonic saline reliably mimics muscle pain, lasts for a short duration and has no reported adverse effects (Capra & Ro, 2004). Its limitations are that it has poor inter-individual reliability and the injection has a risk of infection, although minimal risk. A more detailed review can be found below.

6.2. Hypertonic Saline review

Of the modalities considered, hypertonic saline (HS) is the most accepted method of inducing experimental pain that is commonly and successfully used in pain studies (Henriksen et al., 2011;Korotkov et al., 2002). It is considered to be safe with minimal adverse effects reported (Graven‐Nielsen, 2006). Pain induced by HS is said to reliably mimic musculoskeletal pain and chronic pain (Capra & Ro, 2004). Hypertonic saline also possesses satisfactory intra-individual reliability in pain intensity and distribution (Graven‐Nielsen, 2006). This reliability is especially important for studies comparing individual results. In addition to these factors, HS is accessible and does not produce an inflammatory response, thus is considered an appropriate method for inducing experimental pain.

7. Different types of muscle contractions

There are three main types of muscle contractions: concentric, eccentric and isometric. As described by Enoka (1996), concentric contraction is when the muscle fibers shorten under tension (e.g., lifting a weight). An eccentric contraction is when the muscle fibers lengthen under tension (e.g., lowering a heavy object). During an isometric contraction the muscle fiber length stays the same (e.g., carrying a book in a static position). A naive presumption may be to assume that the only difference between contraction types is the amount of opposing force and the subsequent direction of change in muscle length. However, several

decades of developments in neuromuscular research has revealed that this is an over-simplistic and inaccurate differentiation.

7.1. Eccentric muscle contractions

Eccentric contractions are different from concentric as they are capable of generating up to 30-40% greater force (Lindstedt, LaStayo, & Reich, 2001; Tesch, Dudley, Duvoisin, Hather, & Harris, 1990). It is reported that they also have greater resistance to fatigue (Enoka, 1996) however, the quality of these reports are variable. One study that investigated fatigue used a constant force, intensity and duration to achieve fatigue for concentric and eccentric contractions (Gray & Chandler, 1989). As eccentric contractions are capable of generating more force, this force is not relative to eccentric MVCs in which case this is not a fair comparison of fatigue. Another study used concentric and eccentric MVCs to achieve fatigue which is a more appropriate protocol (Grabiner & Owings, 1999). Eccentric contractions are also innervated by a different neural activation strategy as indicated by lower EMG activity for a given amount of force production, and activation of different motor units, some of which are exclusively activated during eccentric contractions (Enoka, 1996). Due to the recruitment of eccentric specific motor units, neural activation in eccentric contractions is considered to be unique (Enoka, 1996).

Eccentric contractions are essential in coordinating postural control and preventing joint damage yet the performance of concentric and isometric contractions are the mainstay for muscle function tests (Wilson & Murphy, 1996). Very seldom have eccentric contractions been investigated, particularly in conditions of disease and/or pain. The studies that have investigated eccentric muscle contractions provide evidence that suggest that eccentric contractions are not affected by pain the same way that concentric contractions are, if they are effected at all (Marshall, Mannion, & Murphy, 2010; Shirado et al., 1995; Zedka et al., 1999). A review of these studies can be found in Section 8.

8. Review of studies investigating eccentric muscle contractions

Divr & Keating, (2003) investigated trunk extension during maximal contractions in participants with low back pain. Marshall et al., (2010) similarly explored the eccentric-concentric strength relationship using maximal contractions in chronic low back pain. Both authors found more pronounced concentric strength loss compared to eccentric strength in patients with low back pain. Divr & Keating, (2003) observed a particularly significant finding where the reduction in strength during an eccentric contraction was 7% compared to 32% in concentric. Other studies have struggled to report differences that exceed between-subject variation, which reflects the inherent challenge of studying organic pain (Boling, Padua, & Alexander Creighton, 2009).

Zedka et al. (1999) investigated experimental pain during functional tasks (lumbar flexion) in human back muscles and established that electromyogram (EMG) activity changed during standardized isometric and concentric contraction movement, but not during the eccentric component (i.e., forward flexion). The authors found that the EMG activity of the flexion phase (eccentric component) was unchanged between pain and no pain conditions, even though the relaxation (isometric) and extension (concentric) phases were significantly affected. This is important to know as the change in EMG activity highlights that eccentric contractions are being effected differently compared to other contraction types. Zedka et al. (1999) suggested that this phenomenon could be due to the larger gravitational force acting on eccentric movement compared to concentric, however this is a physically flawed explanation. One downfall to this study was the small sample size (n=5), however, this is a minor failing, and the clear findings are enough to provide confidence in their results.

9. Volitional underperformance

Given the different neural control systems and pathways employed for eccentric contractions, and the role of eccentric contractions as postural stabilisers (Enoka, 1996) one may hypothesise that different contraction types are influenced by different levels of volitional control. For example, the involuntary stretch-reflex would occur during a fast eccentric contraction but not during concentric or isometric contraction. Reduced volitional control of eccentric contractions has been evidenced by the inability to modulate eccentric force (Dvir

& Keating, 2001). Divr & Keating, (2001) explored this by using a test-retest comparison of MVCs and feigned lower back extension in pain free participants. In the protocol, participants performed two tasks, the first was a round of eccentric and concentric MVCs. For the second task, participants were then asked to repeat the contractions imagining "that they were suing their insurer for a previously sustained a low back injury (now completely recovered) whilst trying to convince the clinician that the claim is sincere". The authors concluded that eccentric-concentric strength ratios were effective in identifying feigned weakness. As pain exhibits both a behavioural and involuntary response, it is possible that the reduced volitional control of eccentric contractions may result in decreased behavioral effects of pain on eccentric contractions. Reduced behavioral effect, combined with difference in neural strategy, may mean that pain affects eccentric contractions differently. If so, eccentric contractions may provide a valuable tool for clinical assessment of muscle strength. This highlights limitations of existing research that cannot control for behavioral factors of strength loss. Multiple authors have found muscle weakness in pain conditions, however, it cannot be concluded that this is actually a neuromuscular weakness or simply behavioral weakness (Hamlin & Quigley, 2001; Hodges & Richardson, 1996; Palmieri-Smith et al., 2013).

10. Conclusion

Current evidence suggests that eccentric contractions may not be effected by pain in the same way as concentric or isometric. However, the limited number of studies and controversial results highlight that further research is required to identify the exact effect of pain on eccentric contractions. Benefits may exist for future research in identifying the role of experimental pain on eccentric muscle contractions. Identification of these results may bridge a gap in current literature and act as facilitation for future research. This knowledge will also give a better understanding of potential functional changes that occur with pain. This could be clinically useful in identifying behavioral factors in clinical strength loss and in aiding the identification of appropriate assessment and treatment plans that are based on the individuals' pain experience. To address the gap in the literature, the manuscript reported in Section II of this thesis aimed to investigate the effect of experimental pain on eccentric muscle contractions.

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Section II: Manuscript

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Peak knee extension torque is similar for all muscle contraction types during experimentally induced pain

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Abstract

Peak knee extension torque is similar for all muscle contraction types during experimentally induced pain

Reduced isometric and concentric muscle strength during pain is a common finding, however, the few investigations into the effect of eccentric muscle strength during pain report inconsistent findings. Eccentric contractions are innervated by different neural control strategies than concentric and isometric contractions, therefore may not be affected by pain in the same way. The aim of this study was to investigate the effect of experimentally induced knee pain on maximal voluntary eccentric inhibition in comparison to other contraction modes using a repeated measures experimental design. Twenty asymptomatic volunteers completed the study in a physiology laboratory setting. Maximal strength of the quadriceps was assessed using an isokinetic dynamometer. Experimental pain was induced by injecting 1.0ml of 5.8% hypertonic saline into the infrapatellar fat pad. Eccentric strength was reduced immediately with pain for most participants, however the difference was not statistically significant ($p = 0.083$, $z = -1.736$, $r = 0.39$ '*small*'). Eccentric strength was reduced from the baseline to post-pain condition ($p = 0.017$, $z = -2.389$, $r = 0.53$ '*medium*'). In conclusion, eccentric contractions are affected by experimental pain similarly to other contraction types. However, differences exist in the variability of the effect of pain across contraction modes.

Keywords: knee pain, hypertonic saline, muscle strength, physical function

1. Introduction

Chronic pain is a persistent and debilitating problem [Harstall and Ospina, 2003] that has become one of the most common causes of disability worldwide [Dreisinger, 2014; Pfizer, 2013]. It is well established that pain alters muscle function, including muscle activation and strength [Hodges et al., 2013]. Moreover, pain is strongly associated with impaired physical activity [Henriksen et al., 2011] and strongly correlates with reduced quality of life, thus it is not surprising that pain is amongst the most common presenting complaints in clinical settings [Dvir and Keating, 2001; Khan et al., 2011].

Rehabilitation of muscle activity, including strengthening and neuromuscular retraining is commonly utilized for management of pain conditions [Hodges, 2011]. There is extensive literature describing the effects of pain on muscle contractions with a unanimous finding of reduced muscle strength [Graven-Nielsen and Arendt-Nielsen, 2008; Hodges, 2011; Lund et al., 1991]. Interestingly, the majority of studies have investigated concentric and isometric contractions. The few studies that have included eccentric contractions have reported somewhat inconsistent findings that challenge the current generalisation of 'muscle weakness' associated with pain [Dvir and Keating, 2003; Henriksen, Rosager, 2011; Zedka et al., 1999].

It is plausible that the effect of pain on eccentric contractions may be dissimilar to what has been reported for concentric and isometric contractions. Eccentric contractions are served by different neural control strategies [Enoka, 1996; McHugh et al., 2002], have greater resistance to fatigue [Enoka, 1996], and are capable of generating more force [Lindstedt et al., 2001]; eccentric contractions are required in coordinating postural control and preventing joint damage [Hodges, 2011] and often evoke an involuntary stretchreflex contraction [Fukutani et al., 2015]. In light of the different characteristics of eccentric contractions, it has been argued that eccentric muscle contractions are under less volitional control than concentric contractions [Dvir and Keating, 2003]. Given the behavioural effects (decreased performace with pain expectation) of pain on strength tasks [Geisser et al., 2000; Vlaeyen et al., 1995] a strength measure that is less affected by volitional apprehension could be advantageous in clinical assessments.

Eccentric contraction characteristics have received little research attention, particularly in the context of pain. In the few studies where this has been investigated, the results have not closely mirrored those reported for isometric and concentric contractions. Marshall et al., [2010] and Shirado et al., [1995] both found more pronounced concentric strength loss compared to eccentric strength in patients with low back pain. Zedka et al., [1999] observed no change in eccentric muscle activity of trunk muscles during experimental pain. Other studies in the clinical setting have struggled to report differences that exceed between-subject variation [Boling et al., 2009]. At least in people with low back pain, it appears that eccentric contractions may be less affected than concentric or isometric contractions. However, these observations made in people with naturally occurring back pain have yet to be investigated under well-controlled laboratory conditions. Therefore, the present study employed an experimental pain model to assess the extent to which eccentric contractions are affected by pain. This unexplored area is important as further insight may aid clinicians in differentiating between neural and behavioural mechanisms of muscle weakness as a result of pain. Therefore, the aim of this study was to investigate the effects of experimental muscle pain on eccentric muscle inhibition in comparison to other contraction modes.

2. Methods

2.1. Participants

Healthy participants (12 female, 9 male) were recruited as consistent with Henriksen et al. (2011), from the local community using convenience sampling. Participants were excluded if they reported a history of significant trauma to the lower limbs and/or trunk with any current musculoskeletal or neural symptoms; any diagnosed neurological or spinal disorders; use of pain medications on the day or day prior to testing; or pain (score >2/10 on numerical rating scale) anywhere in the body on the day of testing. Further exclusion was made if participants scored >30 on the Pain Catastrophising Scale [Osman et al., 2000; Sullivan et al., 2009]. Five individuals were unable to be recruited in the study due to being able to overcome the maximum torque capacity of the dynamometer. All participants gave written informed consent and the study received ethics approval from the Unitec Research Ethics Committee (UREC 2013- 1055).

2.2. Isokinetic Dynamometer

Strength of the knee extensors was measured using isokinetic dynamometry (Biodex system 3, Biodex Medical Systems, New York). Participants were positioned in the dynamometer with adjustable straps placed around their shoulders, waist, and right thigh to restrain movement. The right center of the knee joint was aligned with the axis of rotation of the dynamometer and the shin was then strapped into a resistance cuff. Velocity settings for concentric and eccentric maximal voluntary contractions (MVCs) was 30deg/s and the position for isometric MVCs was set to 70deg knee flexion as consistent with Henriksen et al., (2011). Raw voltage output from the dynamometer for torque and position was sampled at 2 kHz using LabChart v7.2.1 (AD Instruments Pty Ltd, NSW).

2.3. Surface electromyography detection and recording

In parallel to investigating the aim, muscle activation amplitude and frequency of vastus lateralis and vastus medialis were measured using surface electromyography (SEMG) together with a complementary force-matching task. These data were beyond the scope of the aim of the current project and will be reported in a separate report.

2.4. Procedure

Clinical observations indicate that most people are unable to achieve a maximum voluntary eccentric contraction without practice. Participants were therefore required to attend a predata collection session that involved familiarising participants with the equipment and tasks (torque testing using MVCs and force-matching). At least 1 week later participants attended a second data collection session where participants underwent dynamometry. Measures were recorded for three conditions: baseline (MVCs then force-matching), pain (force-matching then MVCs), and post-pain (force-matching then MVC) conditions. Peak torque values for the force-matching tasks were taken during the baseline measures. A 5-minute rest for recovery was given after completion of the baseline measures (see figure 1 for time course of procedure).

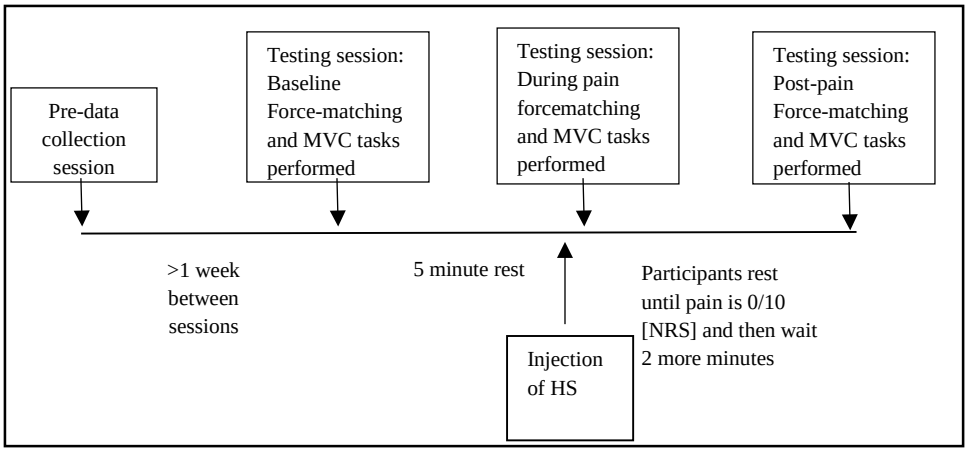

Figure 1: Timeline of procedure. The procedure involved one pre-data collection session and one testing session (involving three sets of measurements being collected). Note: HS = hypertonic saline, MVC = maximum voluntary contraction, NRS = numerical rating scale.

To control for order effects, the order of muscle contraction types was randomised using a counterbalanced design ('latin square') [Winer, 1962]. Each participants order was repeated throughout both tasks (MVC and force-matching). For the torque testing, five full-range concentric and eccentric MVCs at 30deg/s and five isometric MVCs at 70deg flexion were performed. Each contraction was sustained for 3s, and a 3-5s rest was given between each contraction (isometric, eccentric and concentric). After each contraction type was complete, a 30s rest was given. Participants were instructed and verbally encouraged throughout the MVCs to perform all contractions as "hard and fast as possible".

2.5. Experimental pain

With the participant positioned in the dynamometer, an injection of 1.0ml of 5.8% hypertonic saline (Waitemata District Health Board hospital pharmacy, Auckland, New Zealand) using a 27G needle was administered at a 45 degree angle into the anterior medial aspect of the right infrapatella fat pad with a needle insertion depth of 1cm. The onset of pain took approximately 30s to begin after needle withdrawal. A numerical rating scale (NRS) [Hartrick et al., 2003] was used to rate pain intensity at post needle withdrawal and every 90s thereafter until completion of the tasks. When the NRS reached 0/10, participants waited 2-

min before repeating the force-matching and MVC tasks to attain the post-pain condition measure.

2.6. Data analysis

All statistical analyses were performed using SPSS (v22, IBM Corp., Armonk, NY). Mean values for each pain condition (baseline, during pain and post-pain) and contraction type (isometric, concentric and eccentric) were obtained by taking the average of the three greatest peak torque values from the five contractions recorded. The greatest three contractions were chosen as they more closely represent the physiological maximum force. Calculated variables were created to quantify the change in torque from baseline to pain condition (Δpain: during pain minus baseline) and change from baseline to post-pain conditions (Δpost: post-pain minus baseline). Calculated variables were also created to quantify the contrast between contraction types (eccentric v isometric, isometric v concentric and concentric v eccentric). Data distribution was visually identified as not normally distributed. This observation was affirmed statistically with the Kolmogorov-Smirnov and Shapiro-Wilk tests of normality with all but baseline eccentric and Δpost isometric, concentric and eccentric conditions being significantly different from normal. Pairwise analyses were therefore conducted using the Wilcoxon Signed-Rank test to compare changes in torque between baseline and during pain, and between baseline and post-pain conditions for all contractions. Effect sizes were calculated using the equation $r=Z/\sqrt{N}$ as described by Field [2005] and interpreted using Cohen's criteria where $d = 0.2$ is 'small', 0.5 is 'medium', and > 0.8 is 'large' [Cohen, 1977]. Statistical significance was set at $p < 0.05$.

Data is reported as median (*Mdn*) unless otherwise stated.

3. Results

3.1. Experimental pain duration

One participant did not complete data collection due to an adverse reaction to injection of hypertonic saline. Between the during-pain and post-pain condition it took an average of 18 ± 4.5 min for the NRS (pain intensity) to reach 0/10. The average peak pain intensity rating was 5.3 ± 2.1.

3.2. Contrast within conditions (isometric, eccentric, concentric)

There was a significant reduction of muscle torque during the Δpain (during pain minus baseline) condition for concentric contractions (baseline *Mdn* = 144 Nm, during pain *Mdn* = 135 Nm, $p = 0.005$, $z = -2.8$, $r = 0.63$ '*medium'*). For the Δpost (post-pain minus baseline) condition significance was not achieved for concentric contractions (baseline *Mdn* = 144 Nm, post-pain *Mdn* = 140 Nm, $p = 0.151$, $z = -1.437$, $r = 0.32$ '*small'*).

For isometric contractions there was a significant reduction of muscle torque during the Δpain condition for isometric contractions (baseline *Mdn* = 188 Nm, during pain *Mdn* = 180 Nm, $p = 0.011$, $z = -2.539$, $r = 0.57$ '*medium'*). During the Δpost condition significance was not achieved (baseline *Mdn* = 188 Nm, post-pain *Mdn* = 185 Nm, $p = 0.079$, $z = -1.755$, $r = 0.39$ '*small*').

Statistical significance during the Δpain condition for eccentric contractions was not achieved (baseline *Mdn* = 236 Nm, during pain *Mdn* = 234 Nm, $p = 0.083$, $z = -1.736$, $r = 0.39$ '*small*'). For the Δpost condition significance was achieved (baseline *Mdn* = 236 Nm, postpain *Mdn* = 216, $p = 0.017$, $z = -2.389$, $r = 0.53$ '*medium*') for eccentric contractions.

In summary, considering Δpain *within* concentric and isometric contractions, there was a significant reduction of muscle torque. However, there was no significant reduction of torque for eccentric contractions (figure 2). During the Δpost condition (figure 3), there was no

prolonged effect of pain on torque for concentric and isometric contractions. In comparison, eccentric contractions exhibited a delayed inhibitory response after pain had dissipated.

3.3. Contrast between contraction types (eccentric v isometric, isometric v concentric and concentric v eccentric)

For the Δpain condition a 'small' non-significant effect was observed between isometric-concentric (p = 0.376, z = -0.885, r = 0.20 '*small'*). For eccentric-concentric contractions a negligible effect (p = 0.010, z = -2.576, r = 0.058 *'small'*) was observed. For eccentric-isometric a 'medium' effect was observed in favor of eccentric contraction (p = 0.017, z = -2.389, r = 0.53 *'medium'*).

For the Δpost condition a negligible effect size was found between isometric-concentric (p = 0.601, z = -0.523, r = 0.12 *'small'*). For isometric-eccentric, a negligible effect (p = 0.478, z = -0.709, r = 0.16 *'small'*) was observed. Between eccentric-concentric a 'medium' effect size, (p = 0.173, z =-1.363, r = 0.3 *'small'*) was observed, eccentric having a higher torque.

3.4. Variability in eccentric contractions

There appears to be variability in responses to experimental pain stimuli during eccentric contractions (see Figure 2). Indeed, approximately 25% of participants displayed an increase in eccentric torque measures, while at least 50% of participants displayed the greatest torque loss during eccentric contractions.

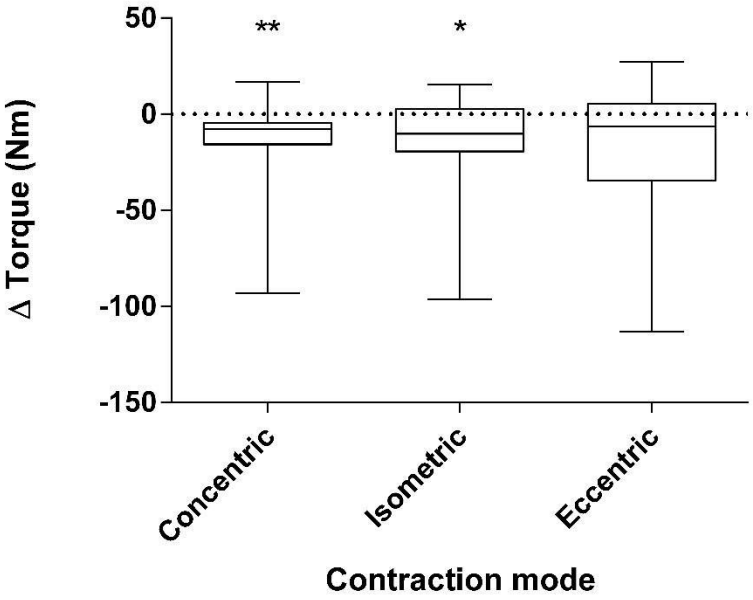

Figure 2: Boxplot to show change in torque (Nm) from baseline to pain condition during each contraction mode (concentric, isometric and eccentric). The whiskers represent the interquartile range and the dotted line shows zero change in force. Note: * Indicates significance $p < 0.05$, ** indicates significance $p < 0.01$.

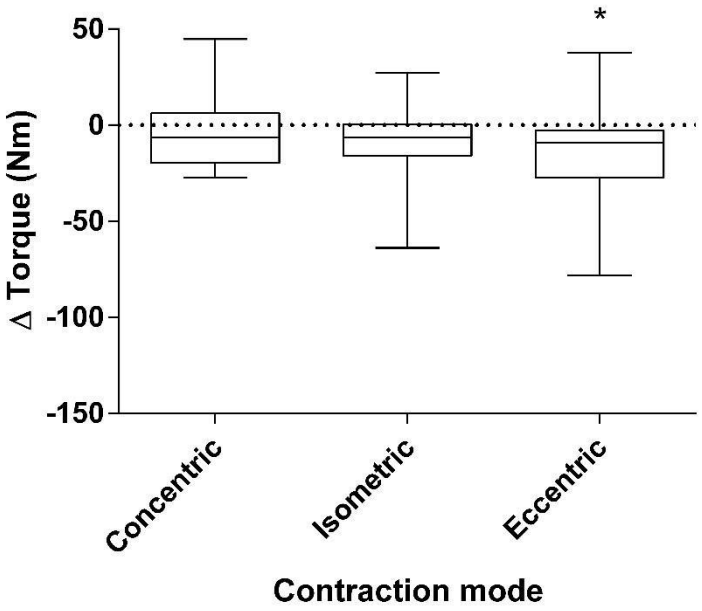

Figure 3: Boxplot to show change in torque (Nm) from baseline to post-pain condition during each contraction mode (concentric, isometric and eccentric). The whiskers represent the interquartile range and the dotted line shows zero change in force. Note: * indicates significance $p < 0.05$.

4. Discussion

The aim of this study was to investigate the effect of experimental pain on eccentric muscle torque. The main finding of the study is that, for most individuals, eccentric contractions were similarly affected by experimentally induced pain as concentric and isometric. This observation contrasts with previous reports of eccentric strength being less influenced by non-experimental (i.e. naturally occurring low back pain) pain compared to concentric and isometric contractions [Dvir and Keating, 2003; Shirado et al. , 1992; Zedka et al.,1999].

There are variable findings reported in relation to the effect of eccentric muscle strength on pain. Shirado et al., [1992] were one of the first to investigate eccentric strength. They investigated eccentric and concentric strength in healthy subjects compared with patients

reporting chronic low back pain (CLBP). The authors noted that concentric and eccentric strength was lower in patients with CLBP compared to healthy subjects. Although this finding is similar to the findings of the current study, non-experimental or naturally occurring pain (i.e. CLBP), cannot be compared to experimental pain because in chronic pain the variability in pain experience between individuals is likely to be high. A further explanation for Shirado et al's finding is that people with CLBP may not have experienced sufficient pain intensity to evoke enough change during eccentric contractions. Shirado et al., [1992] results do not include adequate detail to calculate the effect size to compare to the current study.

Divr and Keating, [2003] investigated the strength of trunk muscles in participants with CLBP. The results show that during concentric contractions, the strength reduction relative to healthy subjects for woman was 32% compared to a 7% reduction during eccentric. Men showed a similar effect, concentric strength being reduced by 28% compared to a 6% reduction during eccentric. The authors conclude that in patients with CLBP the strength deficit during eccentric contractions is much less affected compared to concentric. However, the results of this study do not account for between-subject variability. Experimental pain allows participants to be their own controls, thereby eliminating between-subject variability and controlling for complex psychosocial factors associated with pain [Turk and Okifuji, 2002]. Other studies have failed to include eccentric strength [Khan et al., 2011; Palmieri-Smith et al., 2013]. Of the studies that have included eccentric contractions [Dvir and Keating, 2003; Henriksen et al., 2011; Shirado et al., 1992; Zedka et al., 1999], variance in the effect of pain during different contraction types has been observed. In contrast, the current study found that all contraction types were affected similarly in relation to reduced torque, although the magnitude of the effect was greater for concentric and isometric during pain ('medium') than eccentric ('small'). Post pain, the magnitude of the effect was greater for eccentric ('medium') than concentric and isometric ('small').

The findings highlight that although not statistically significant, eccentric contractions appear to be more variable in response to pain than concentric and isometric. These observations may explain some of the differences in how pain affects muscle function in previous reports [Dvir and Keating, 2003; Zedka et al., 1999]. The variable effect of pain on eccentric

contraction may confound clinical observations about strength changes. The observed variability may be due to lack of familiarisation of performing eccentric contractions. More extensive familiarisation may reduce variability, which may improve statistical detection of smaller effects.

During concentric contractions, pain had a significant immediate effect (decreasing muscle torque) on concentric contraction torque. This decrease appeared to reduce over time and was no longer apparent after pain had dissipated. Pain also had a significant and immediate effect on isometric contractions. While there were indications of the effect of pain (decreased torque) present after pain had dissipated, the lasting effect was not statistically significant. In contrast, pain did not produce a statistically significant immediate effect for eccentric contractions, however, eccentric force production was found to be strongly inhibited after pain had dissipated.

Residual muscle inhibition that exceeds pain perception observed in the current study is in accordance with previous findings [Ervilha et al., 2004; Henriksen et al., 2007; Shakespeare et al., 1985]. The exact mechanism of this inhibition is unknown [Henriksen et al., 2011], however, some authors suggest that sustained muscle inhibition is due to central adaptations [Henriksen et al., 2009; Slater et al., 2003]. Central adaptations can occur in ascending pathways where modulation occurs without nociception being perceived or could be a result of a small delay in the neural inhibition networks (inhibitory interneurons) disconnecting [Henriksen et al., 2009], hence the delayed effect. It has been suggested that sustained muscle inhibition, after pain has dissipated is an inappropriate adaptation and may contribute to development of chronic pain conditions [Henriksen et al., 2011]. This adaptation is multifactorial and can be influenced by a large number of factors including memory of past experiences, modulation of the electrical signal, and pain association [Bogduk, 2002; Godfrey, 2005; Peck et al., 2008]. An interesting approach that may provide further insight could be to examine longer exposure to pain to enable a more comprehensive neurological adaptive response that more closely resembles clinically observed persistent pain.

A possible explanation for eccentric contractions being significantly effected post-pain compared to isometric and concentric contractions may be due to the different neural control strategies found in eccentric contractions [Enoka, 1996]. Eccentric contractions have a unique activation of motor units by altering recruitment patterns and recruiting eccentric specific motor units [Enoka, 1996]. Eccentric contractions also require less activation during MVCs compared to other contraction types. Perhaps, due to the different neural pathways, the difference between contraction types is more apparent once pain has been present for a longer duration.

Isometric and concentric strength is commonly utilized in clinical examination of muscle function tests [Almosnino et al., 2012; Shirado et al., 1995]. As there is insufficient evidence that eccentric strength is affected differently to concentric and isometric strength, including eccentric testing in muscle function tests in practitioners' routine investigations of pain, may not be necessary. Additionally, eccentric muscle contractions are often associated with unfamiliarity, pain, and possible delayed onset muscle soreness [Hamlin and Quigley, 2001; MacIntyre et al., 1995]. In light of the findings of this study, the utility of eccentric muscle testing could be investigated further.

4.1. Strengths and limitations

Experimental pain is a well-established method used to investigate muscle function during pain conditions [Arendt-Nielsen and Graven-Nielsen, 2008; Bennell et al., 2004] and allows participants to be their own controls, thereby eliminating between-subject variability and controlling for complex psychosocial factors associated with pain [Capra and Ro, 2004; Palmieri-Smith et al., 2013]. Of the different experimental pain modalities, hypertonic saline is commonly utilized in assessments of muscle function [Graven-Nielsen, 2006; Tsao et al., 2010]. Hypertonic saline is accessible, reliably mimics musculoskeletal and chronic pain [Capra and Ro, 2004] and has minimal risk of adverse reaction [Korotkov et al., 2002].

There were several limitations to the current study. Firstly, maintaining the desired pain intensity for a period long enough to complete the tasks involved is difficult to control with a

single injection of hypertonic saline. Three participants reached a pain rating of 0/10 before they had completed all of the pain condition measures. As decreasing pain intensity was experienced during the 'pain' measure this could have influenced the isometric, concentric and eccentric pain outcomes. Use of alternative methods such as an infusion pump may be more appropriate for achieving and maintaining desired pain intensity [Rice et al., 2009]. However, because of in-dwelling needle placement, additional care would need to be taken to avoid injury during dynamic movements. Recruiting participants for pain studies is already challenging, enduring pain for a longer period may also considerably reduce willing participants. Secondly, this study may have suffered from selection bias owing to technical limitations of the Biodex system 3. During piloting testing approximately five out of nine people were able to overcome the maximum torque limit of 420 Nm during eccentric contractions and therefore subsequent recruitment was limited to those whose peak torque was less than 420 Nm. Due to this technical limitation, the sample studied was not representative of a full spectrum of the population. These findings have not been demonstrated in those who can generate more than 420 Nm and therefore should be cautiously generalized. Thirdly, volitional underperformance during assessment of strength can be a limitation. In the current study two sessions were used where participants underwent MVCs measures. If the maximum torque was similar across both sessions, it was interpreted as a representative measure of peak torque because achieving similar torque over different days without feedback would be highly challenging. Additionally, when performing the five MVCs per contraction type, contractions were visually assessed by the investigator for consistency in torque. In order to promote consistent contraction torque, participants were encouraged to perform as "hard and fast as possible".

4.2. Recommendations

As the results of this study are inconsistent with previous findings [Dvir and Keating, 2003; Zedka et al., 1999] more research in this area is needed to corroborate the findings. The use of more extensive pre-experimental familiarisation may reduce variability in pain responses during eccentric contractions. Alternatively, a sample of well-trained individuals, who are familiar with heavy eccentric movements, may provide a convenient cohort in this regard, however, their superior strength may be challenging for practical and clinical use of eccentric strength testing. Alternatively a dynamometer with higher capacity than 420 Nm could be

used. Finally, a pain stimulus of longer duration may be beneficial to allow slower inhibition networks to establish.

5. Conclusion

The effect of experimental pain on eccentric muscle contractions was investigated. This study found that, in general, eccentric muscle force was similarly affected as concentric and isometric by pain, however, eccentric contractions produced more variability in torque. Future studies need to appreciate the different neural networks involved in eccentric contractions, and the possibilities that pain has a different temporal effect and the unique experience of pain.

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Section III: Appendices

Appendix A: Ethics approval

Lana Torenvlied-Whiting
465 Ararimu Valley Rd
RD2, Waimauku
Auckland

22.8.13

Dear Lana,

Your file number for this application: **2013-1055**
Title: **To what extent are eccentric muscle contractions affected by experimental 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: 15.8.13
Finish date: 15.8.14

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.

3. Organisational consent/s must be cited and approved by your primary reader prior to any organisations or corporations participating in your research. You may only conduct research with organisations for which you have consent.

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

Yours sincerely,

pp.

Deputy Chair, UREC

cc: Jamie Mannion
Cynthia Almeida

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Henderson
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New Zealand

Appendix B: Information Sheet for Participants

RESEARCH INFORMATION FOR PARTICIPANTS

You are invited to participate in our research investigation. Please read carefully through this information sheet before you make a decision about volunteering.

Research question

To what extent are eccentric muscle contractions affected by experimental pain?

Researcher

My name is Lana Torenvlied-Whiting and I am a Master of Osteopathy student at Unitec New Zealand. As part of this programme I am conducting a research project.

Purpose of the study

The purpose of this study is to identify the effect of experimentally induced muscle pain on eccentric contractions. The results of this study will assist in understanding more functional and dynamic pain-related movement alterations, and will test the validity of Eccentric:Concentric ratio methods for detecting feigned weakness.

What the study involves

Taking part in this study will require you to attend up to 2 sessions at AUT. One session will last approximately 1 hour. The following procedure will occur on the day of testing:

- Informed consent process with questions and answers.
- Participant positioned in dynamometer.
- EMG preparation (This involves abrading the area of skin where the electrodes will be placed and then cleansing it with a sterile wipe. The electrodes will be placed at the same reference point on each participant. This reference point will be determined during piloting).
- Four repetitions at 25%, 50%, 75% and maximal voluntary concentric and eccentric strength will initially be used to familiarize participants with the movement required. Additional familiarization sessions may be required (see piloting section below).
- 5 minute break.
- Three baseline maximal concentric and then eccentric contractions will be made at angular velocity of 60 degrees per second.

- 5 minute break.

- Hypertonic Saline (HS) injection. Then wait for 30 seconds for pain to peak and get participants to report Verbal analogue scale.

- Three maximal concentric and then eccentric contractions will be made at angular velocity of 60 degrees per second.

- 10 minute break.

- Three maximal concentric and then eccentric contractions will be made at angular velocity of 60 degrees per second.

About the experimental pain

Experimental pain can be expected to produce an intensity of approximately 5 or 6/10 and last for about 5 minutes. The perceptual characteristics are most commonly described as 'aching', 'cramping', and 'dull'. It's often likened to sore muscles after vigorous exercise.

"Adverse reactions are extremely rare and may consist of infection (as associated with any needle injection; risk will be managed by following World Health Organization (WHO) infection control practices), bruising or an undesirable level of pain. Excessive pain may be alleviated quickly by stretching and contracting the painful area. If you would like us to contact your GP prior to your participation, please provide their contact details below:

_____

For immediate and after-hour concerns, you may contact an A&E clinic. The most local clinics that are open 24 hours are Ascot White Cross, contact number 520 9555 and Henderson White Cross, contact number 836 3336. The injections for inducing experimental pain will be performed by Vivienne Axon from the Department of Nursing, Unitec New Zealand. Participants will be observed for 15 to 20 minutes following the injection, or until pain has completely subsided.

If required, counselling services are available at Unitec, Mt. Albert campus for all Maori and non-Maori Unitec students and staff. Contact number: 815 4321, extension 7248. Maori consultation services are also available (Hare Paniora, Phone 815 2934)

Your voluntary participation

Your participation in this study is entirely voluntary and you may withdraw at any time during the practical procedures. Data collected from your involvement in the study may be withdrawn up until 1 week following data collection.

Who may participate?

You are eligible to participate if you:

- Are aged between 18-60
- Speak and write English
- Have normal upper limb function

- Are a New Zealand citizen or permanent resident Are male or female

You are not eligible to participate if you:

- Have had previous significant trauma to upper/lower limbs and/or trunk with current symptoms
- Are diagnosed with any neurological disorders
- Use pain medications on the day or day prior to testing
- Have pain (2/10 or greater) anywhere in body on the day of testing
- Have Elevated Pain catastrophising as determined by the Pain Catastrophising Scale (PCS)
- Have any diagnosed spinal disorders

Please inform the researcher if any of the above pertains to you.

What we do with the data and results, and how we protect your privacy.

Personal information is collected and stored under the guidelines provided by the Privacy Act 1993 and the Health Information Privacy Code 1994. For information collection your identity will remain anonymous and you will simply have an identification number. If the information you provide is reported or published, this will be done in a way that does not identify you as its source. All the data recorded will be stored in a password-locked computer and archived in a locked file room and will be stored for a minimum of 10 years. Access to this data will be limited to the principle researcher, the research supervisor, and yourself. This research project is not sponsored by any commercial company. This research project is part of Master of Osteopathy Programme.

Compensation for Adverse Reactions

Compensation may be available in the unlikely event of injury of negligence. As this procedure can be defined as a treatment, you may be eligible for compensation for treatment injury as described under Accident Compensation Act, 2001. Should you incur a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act 2002. You may or may not be entitled to ACC compensation, depending on several factors such as whether or not you are an earner. ACC will usually cover a proportion of income lost due to a physical injury, this does not cover mental injury unless as a direct result from a physical injury. ACC cover may affect your right to sue. Please contact your nearest ACC office for further information (0800 735 566) or visit their website: www.acc.co.nz

A summary of the final report will be available to you if you are interested.

Please contact me if you require further information about the study.

Principal investigator	Supervisor
Lana Torenvlied-Whiting	Jamie Mannion
Phone: 021 1538404	Phone: 021 0629007
Email:lana.whiting@hotmail.com	Email: jmannion@unitec.ac.nz

This study has been approved by the Unitec Research Ethics Committee from (15/08/2013) to (15/08/2014). If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC Secretary (Ph: 09 815 4321 ext.7254). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.

Appendix C: Participant Consent form

Participant Consent Form

To what extent are eccentric muscle contractions affected by experimental pain?

I have had the research project explained to me and I have read and understand the information sheet given to me.

I understand that I don't have to be part of this if I don't want to and I may withdraw at any time up to two weeks after receipt of the transcript.

I understand that I will be injected with hypertonic saline to produce experimental pain. I understand the possibility of an adverse reaction to the injection.

I understand that everything is confidential and none of the information I give will identify me and that the only persons who will know what I have said will be the researcher and their supervisors. I also understand that all the information that I give will be held in a secure location if printed or password controlled if electronic for a period of 5 years.

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 project.

Participant Name: …….. Date: ……

Participant Signature: …….. Date: ……

Project Researcher: …….. Date: ……

UREC REGISTRATION NUMBER: (insert number here)
This study has been approved by the UNITEC Research Ethics Committee from (date) to (date). If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC

Secretary (ph: 09 815-4321 ext 6162). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.

Participant Consent Form

To what extent are eccentric muscle contractions affected by experimental pain?

I have had the research project explained to me and I have read and understand the information sheet given to me.

I understand that I don't have to be part of this if I don't want to and I may withdraw at any time up to two weeks after receipt of the transcript.

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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 project.

Participant Name: …….. Date: ……

Participant Signature: …….. Date: ……

Project Researcher: …….. Date: ……

UREC REGISTRATION NUMBER: (insert number here)
This study has been approved by the UNITEC Research Ethics Committee from (date) to (date). If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC Secretary (ph: 09 815-4321 ext 6162). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.

Appendix D: Pain Catastrophising scale

PCS-EN

Client No.: _____ Age: _____ Sex: M(__) F(__) Date: _____

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

0 – not at all **1** – to a slight degree **2** – to a moderate degree **3** – to a great degree **4** – all the time

When I'm in pain …

1 ☐ I worry all the time about whether the pain will end.

2 ☐ I feel I can't go on.

3 ☐ It's terrible and I think it's never going to get any better.

4 ☐ It's awful and I feel that it overwhelms me.

5 ☐ I feel I can't stand it anymore.

6 ☐ I become afraid that the pain will get worse.

7 ☐ I keep thinking of other painful events.

8 ☐ I anxiously want the pain to go away.

9 ☐ I can't seem to keep it out of my mind.

10 ☐ I keep thinking about how much it hurts.

11 ☐ I keep thinking about how badly I want the pain to stop.

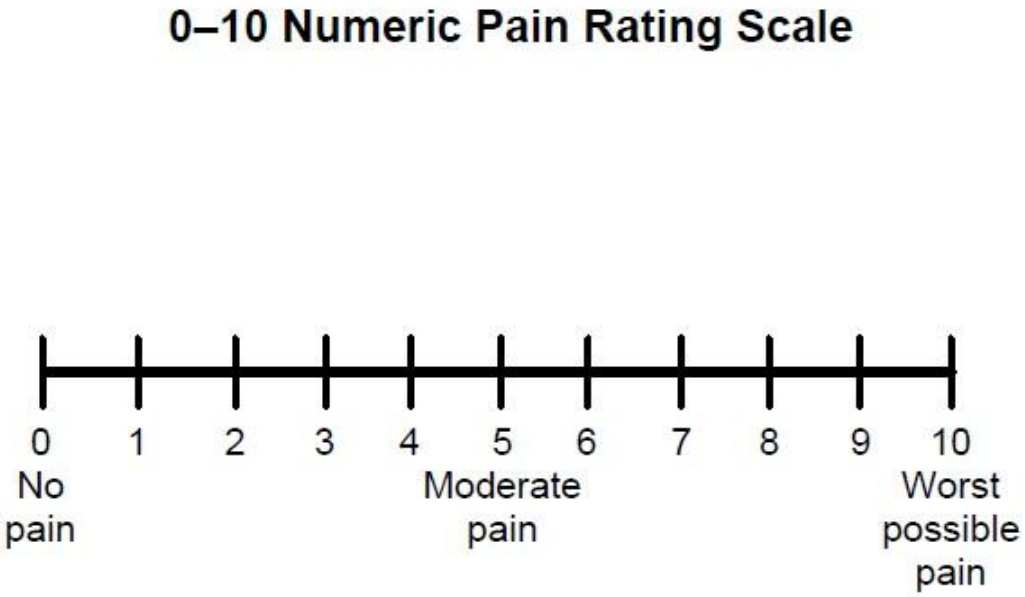
12 ☐ There's nothing I can do to reduce the intensity of the pain.

13 ☐ I wonder whether something serious may happen.

…Total

Updated 11/11

Appendix E: Numerical rating scale



Appendix F: Instructions for Authors manuscript submission to the Journal of Electromyography and Kinesiology

Guide for Authors

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; neuromuscular 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.

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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.

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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,

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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:

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