The effects of myofascial manual therapy on muscle activity and blood flow in people with low back pain

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I declare that no part of this thesis has been submitted in support of an application for any degree or other qualification of the University of Kent, or any other university or institution of learning.

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Abstract

Over the past ten years structured clinical massage techniques aimed at the myofascial structures of the body have become a common choice of therapy for people with low back pain (LBP) (Ajimsha, Al-Mudahka and Al-Madzhar, 2015), yet the mechanisms behind their effects remain unclear. The overall aim of this study was to determine the benefits of myofascial manual therapy through an evaluation of the possible mechanisms associated with changes to muscle activity and blood flow, in people with low back pain.

The first study aimed to investigate the effects of structured clinical massage techniques (CM) on the flexion relaxation response (FRP) of the paraspinal muscles, range of movement (ROM), pain and disability profiles in subjects with non-specific chronic low back pain compared to a relaxation massage (RM) treatment. Results indicated a main effect of time for trials achieving FRP for the multifidus muscle F (1, 13) = 12.109, p = .004 and a marginal main effect of time for the erector spinae muscle F (1, 13) = 4.495, p = .054. There were significant improvements in VAS F (1, 13) = 6.74, p = .022, and PRI F (1, 13) = 10.254, p = <006, pain scores for the CM intervention compared to RM. There was also a significant improvement in kinesiophobia scores F (1, 13) = 7.77, p = .015 and the ODI disability index F (1, 13) = 11.1, p = .005, for the CM group compared to the RM group.
These findings indicated the need to focus on the specific mechanisms and effects of a specific form of clinical massage, myofascial massage techniques (MT).

The second study aimed to investigate the acute effects of integrated myofascial techniques on the peripheral blood volume at the paraspinal region, compared to a traditional relaxation massage. Results revealed significant increases in oxygenated haemoglobin (O_2Hb), F (2-26.44) = 15.82, p = < .001, deoxygenated haemoglobin (HHb), F (2-41) = 3.59, p = .037 and total haemoglobin (tHb), F (2-26.71) = 15.47, p =

< .001, at the paraspinal region following the MT intervention compared to the RM and control groups. There was no significant difference in blood volume variables between the RM and control groups.

The third study aimed to compare the acute effects of integrated myofascial techniques and kinesiotaping (KT) on blood flow at the lumbar paraspinal region. Results indicated that MT was significantly greater, compared to the KT and the control treatments (P<0.001), for changes in O_2Hb, HHb, and tHb. There were no significant differences for PPT [F (2-41) = 2.69, p = 0.08], between groups.

Study 4 aimed to compare the acute effects of integrated myofascial techniques (IMT) on blood flow and local muscle fatigue, pain pressure threshold and postural sway at the paraspinal muscle region in subjects with non-specific LBP. Results indicated a significant increase in blood volume variables O_2Hb [F (1, 10) = 21.51 p = < .001], and tHb [F (1, 10) = 19.54 p = < .001], following a fatigue task, for the MT group compared to sham TENS. There was a significant improvement in time to fatigue [F (1, 10) = 24.17, p= < .001], for the MT group following a fatigue task, and EMG amplitude was significantly higher at the start of fatigue task 2 for the Sham TENS group trial compared to the MT group trial (U = 20.00, p = .007, f = 0.70).

The purpose of study 5 was to compare the acute effects of integrated myofascial techniques (MT) on blood volume and local muscle fatigue at the paraspinal muscle region in subjects with and without non-specific LBP. Results indicated a significant increase in blood flow variables at F2 compared to F1 for O_2Hb [F (2-40) = 23, P<0.001], tHb [F (2-40) = 15.88, P <0.001] and Tsi [F (2-40) = 19.28, P <0.001]. There were no significant differences for blood volume variables between groups. There was a significant improvement in time to fatigue [F (1-20) = 17.38, p = <0.001], for both groups at F2 compared to F1, but no significant difference between groups. Post hoc

analysis also revealed that PPT was significantly improved in the non-LBP group compared to the LBP group [$F (1, 20) = 8.88$ $p < .007$].

Overall it can be concluded from the results of this thesis that local blood flow is significantly increased at the lumbar paraspinal myofascial region in subjects with and without LBP. MT was also shown significantly improve the fatigability of paraspinal muscles in subjects with and without LBP. Therefore, patients with LBP may benefit from the specific effects of the myofascial massage techniques identified within these studies through improvements in muscle activity and blood flow to the paraspinal muscles.

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List of abbreviations

AA	Activity avoidance
AF	Annulus fibrosis
BMI	Body mass index
CAM	Complementary and alternative medicine
CLBP	Chronic low back pain
CM	Clinical massage
CNS	Central nervous system
COP	Centre of pressure
DOMS	Delayed onset muscle soreness
dPAG	Dorsal peri-aqueductal grey
DV	Dependent variable
ECM	Extra cellular matrix
EMG	Electromyography
ES	Erector Spinae
FRP	Flexion relaxation phenomena
FRR	Flexion relaxation ratio
GAG	Glycosaminoglycan
GM	Gluteus maximus
HA	Hyaluronic acid
Hb	Haemoglobin
HbDiff	O_2Hb and HHb difference
HHb	Deoxygenated haemoglobin
IMT	Integrated myofascial massage techniques
IV	Independent variable
IVD	Intervertebral disc
KT	Kinesiotape
L	Lamina
LBP	Low back pain

LD	Latissimus dorsi
MET	Muscle energy technique
MF	Multifidus
MFR	Myofascial release
MN	Motor neurone
MT	Massage therapy
MT	Myofascial techniques
MTJ	Musculotendinous junction
MVC	Maximum voluntary contraction
NIR	Near Infrared
NIRS	Near Infrared Spectroscopy
NP	Nucleus pulposis
NSCLBP	Non-specific chronic low back pain
NSLBP	Non-specific low back pain
O_2Hb	Oxygenated haemoglobin
ODI	Oswestry Disability Index
PG	Proteoglycans
PLF	Posterior layer of the TLF
PPI	Present pain index
PPT	Pain pressure threshold
PRI	Pain rating index
PRT	position release technique
Ps	Psoas
QL	Quadratus lumborum
QTM	Qualisys Track Manager
RA	Rectus abdominus
RCT	Randomised control trial
RM	Relaxation massage
RMQ	Roland Morris Questionnaire

ROM	Range of movement
SBST	STarT back screening tool
sEMG	Surface electromyography
SF	Somatic focus
SF-MPQ	McGill Short Form Pain Questionnaire
SIJ	Sacroiliac joint
SP	Spinous process
TENS	Transcutaneous electrical nerve stimulation
THb	Total haemoglobin
TLF	Thoracolumbar fascia
TrA	Transversus abdominus
TrP	Trigger point
Tsi	Tissue oxygenation saturation
TSK	Tampa Scale for Kinesiophobia
VAS	Visual analogue scale
VB	Vertebral body

1 Introduction

The use of massage in sport and exercise is widespread and has been used as a form of therapy and medical practice for thousands of years across many ancient cultures (Moyer, Rounds and Hannum, 2004; Weerapong, Hume and Kolt, 2005). Although its popularity diminished during the early 91th century, In modern times interest in massage as a form of manual therapy has continued to grow among the scientific communities, reflecting its popularity and culminating in a relatively extensive body of research within the area (Goats, 1994b) .However, massage is still recognised as a therapeutic modality without scientific foundation. (Moraska, 2005; Weerapong, Hume and Kolt, 2005).

Low back pain (LBP) is a major health problem around the world and one that many patients seek therapy to relieve symptoms and improve function (Hoy *et al.*, 2012; Furlan *et al.*, 2015). Over the past decade a number of complementary and alternative medicines (CAM) have been used to alleviate the symptoms associate LBP (Hughes, Quinn and Baxter, 2011). One of the most prevalent CAM's used by a number of therapists and by sufferers of LBP is massage therapy (Moyer, Rounds and Hannum, 2004; Sherman *et al.*, 2004). However the Effects of massage as a treatment for LBP is reported to be inconsistent across a number of outcome measure when used as a treatment for LBP (Simmons, 2011). These inconsistencies have been largely attributed to methodological limitations, variability in the massage techniques, a lack of a quantitative standardised protocol and variability in obtaining physiological data.

More recently, attempts have been made to quantify and classify massage techniques to improve physiological and clinical outcomes (Sherman *et al.*, 2006; Cherkin *et al.*, 2011). Clinical massage techniques is a classification of massage therapy which include more precisely defined techniques that are typically used to improve clinical outcomes for a number of musculoskeletal conditions including; pain relief, reduced muscle spasm, postural re-education, movement re-education and fascial remodelling

(Sherman *et al.*, 2006). The present thesis aims to determine the effects of these clinical massage techniques on subjects with non-specific low back pain and in particular the effects of myofascial massage techniques on muscle activity and blood flow to the paraspinal muscles. Many of the effects of massage are associated with improvements in blood flow to the muscles (Weerapong, Hume and Kolt, 2005), yet this claim has been questioned due to the equivocal evidence that currently exists (Bervoets *et al.*, 2015). The following section in this chapter will provide an overview of the prevalence and epidemiology of LBP, the various treatment options available to LBP the prevalence of the use of CAMS, the myofascial contributors to LBP and how a structured use of clinical massage techniques could have more favourable outcomes for the LBP population.

1.1 Low back pain

LBP is a common disorder and can be described as pain, muscle tension or stiffness localised below the costal margin and above the inferior gluteal folds, with or without leg pain (Chou, 2010b). Mechanical LBP can be classified into specific, non-specific and radicular syndrome (Van Dillen and van Tulder, 2013). Radicular syndrome can be described as nerve root compression caused by disc herniation or degenerative stenosis of the root or spinal canal (Luijsterburg *et al.*, 2007) . Unlike specific LBP, non-specific low back pain (NSLBP) is defined as LBP for which there is no recognisable pathology, such as tumour, fracture or inflammation (Chou, 2010b) and may be commonly associated with several structures including joints, discs, muscles and connective tissue (Savingley, 2009). Although low back pain is largely self-limiting, about 10-15% of people develop chronic back pain lasting for 12 weeks or more in duration. LBP is, therefore, often sub classified into acute (<6weeks), Sub acute (6-12 weeks) and chronic (>12 weeks) (Balagué *et al.*, 2012).

1.2 Epidemiology of LBP

LBP is a condition that effects many people at some point in their lives, creating a substantial personal, societal and financial burden (Hoy *et al.*, 2012; Meucci, Fassa and Faria, 2015). It is estimated that out of 291 conditions studied throughout the world in 2010, LBP was ranked the greatest contributor to global disability (Hoy *et al.*, 2014). The prevalence data on LBP often varies due to the definitions of LBP and whether LBP or chronic LBP definitions are used (Balagué *et al.*, 2012). However, recently it has been estimated that the mean point prevalence of activity limiting LBP lasting more than one day is 11.9%, with a life time prevalence of 38% (Hoy *et al.*, 2012). However some estimates of lifetime prevalence of LBP have been reported as high as 84% (Balagué *et al.*, 2012). LBP appears to be more prevalent in the 40-70 age group, but across all age groups female prevalence is higher than males (Hoy *et al.*, 2012).

The number of patients that develop chronic low back pain (CLBP) has been said to vary from 10-15% to 40% (Balagué *et al.*, 2012). However, those that do develop chronic symptoms are thought to contribute to high treatment costs, sick leave and is one of the main reasons for seeking health care services (Meucci, Fassa and Faria, 2015). The prevalence of CLBP has been estimated at 23% (Airaksinen *et al.*, 2006). However, prevalence figures appear to be higher for older population groups; with a CLBP prevalence of 4.2% in the 24-39 age group compared to 19.6% in the 20-59 age group; and for people of low socioeconomic status (Meucci, Fassa and Faria, 2015). It is estimated that as the prevalence of LBP and CLBP peaks in older age the economic and disability burden may increase substantially in countries with low and middle income groups and with an ageing population (Hoy *et al.*, 2014).

The cost of LBP have been associated with direct costs to the healthcare system and indirect cost attributable to lost production and days off work (Savingley, 2009). LBP is one of the most costly conditions in the UK, with a direct health care costs of £1,632M,

of which £565M was spent outside of the NHS (Maniadakis and Gray, 2000). Furthermore, it has been estimated that over £500M is spent on hospital care and over £150M is spent on physiotherapy treatments for back pain (Maniadakis and Gray, 2000). Indirect costs also contribute to the economic burden. It has been stated that in the USA, 8% of the working population will be disabled from LBP equating to 40 of all lost working days (Manchikanti, 2000). However, the figures for days of absence per patient per year varies across countries ranging from 9 days in the US, 20 days in Canada and 30 days in Great Britain (Nachemson, 1992). Indeed, in the UK the 2005/2006 Health executive figures estimated that 3.7M working days were lost due to back pain.

Risk factors for LBP include both physical, psychological and social factors such as lifting heavy loads, awkward positioning and physical activity; high levels of psychological distress, lifestyle factors such as being overweight and smoking and work factors such as high job demands, low levels of colleague support and work dissatisfaction (Manchikanti, 2000; Balagué *et al.*, 2012). To date much research into LBP has focussed on structural abnormalities such as disc and facet degeneration (O'Sullivan, 2012). However, the mechanisms surrounding CLBP appear to be multifactorial and are poorly understood. But, as previously mentioned, the majority of LBP cases are classified as non-specific, with no identifiable cause. A number of models have been proposed to help identify the factors associated with the development of CLBP (these will be discussed later in chapter 2) suggesting a more complex interaction of physical, mechanical and psychosocial influences

1.3 Treatment options for low back pain

Treatment choices for NSCLBP can be broadly divided into three categories; conservative, pharmacological and invasive (Airaksinen *et al.*, 2006). While some pharmacological treatments are recommended for pain relief in the acute phase, invasive techniques such intraarticular or trigger point injections appear to be no more effective than placebo, and are not generally recommended (Chou, 2010a) . Therefore, conservative approaches remain the most commonly used treatment options for the non-specific LBP population (Airaksinen *et al.*, 2006). Although not an exhaustive list, conservative treatments include exercise therapy, psychological therapies, educational interventions, physical therapies; such as infrared thermotherapy, TENS, ultrasound and lumbar supports; and manual therapies such as spinal manipulation, mobilisations and massage therapies. Of these conservative approaches education, exercise, manipulations / mobilisations, massage and psychological therapies appear to be recommended for managing NSCLBP (NICE, 2016).

Of the treatment outlined above, exercise therapy, either as an individual treatment or as part of a multidisciplinary program, is the most widely used for the treatment and management of NSCLBP (van Middelkoop *et al.*, 2010). In 2005 a Cochrane Review concluded that there was strong evidence to suggest that exercise is as effective as other conservative treatments in reducing pain and disability for NSCLBP (Hayden *et al.*, 2005). However, more recently evidence appears to reflect that exercise is not necessarily more effective than other treatments, apart from usual care, and it is not clear as to the type of exercise that is most effective (van Middelkoop *et al.*, 2010). Typically, the two approaches to exercise are often compared in the literature are motor control and general (graded) exercise approaches. Motor control exercises programs are typically designed to improve the control and coordination of the spine whereas general (graded) exercise programs are designed to improve all-round conditioning through aerobic, flexibility, and strengthening activities (Macedo *et al.*,

2008). However, The use of exercise therapy for NSCLBP is not within the remit of this thesis and readers may be directed to a recent review for further information (Saragiotto *et al.*, 2016).

1.4 Massage therapy as a form of CAM

CAM's offer an alternative approach to traditional medical care and back pain is one of the most common reasons for patients choosing them (Sherman *et al.*, 2004). The most prevalent CAM therapies used for back pain appear to be spinal manipulations, acupuncture and massage and share commonalities that include prolonged direct patient interaction, hands on therapy and incorporate passive and active elements associated with their modality (Furlan *et al.*, 2010). The number of people in Western society using CAM is reported to be on the increase (Sherman *et al.*, 2004; Hunt *et al.*, 2010; Simmons, 2011). Results of a recent survey of CAM therapies used in England suggest that CAMs are becoming more prevalent with a lifetime and 12-month prevalence of 44% and 26% respectively (Hunt *et al.*, 2010). Furthermore, of the CAM's used for LBP, massage represents one of the most prevalent forms of treatment in western societies (Simmons, 2011).

Massage therapy (MT) has been described as the manipulation of soft tissues of the body to enhance health and well-being (Sherman *et al.*, 2006). Recently, massage therapy has been documented as being one of the most prescribed CAMs most likely to be beneficial and least likely to be harmful (Ezzo, 2007). Recent evidence has pointed out that variable results and general patient dissatisfaction with conventional treatments have led to the increase in the use of CAMs by LBP sufferers (Sherman *et al.*, 2004; Hughes, Quinn and Baxter, 2011). Indeed, one of the most common CAMs used by physiotherapists in the management of low back pain are acupuncture and massage (Hughes, Quinn and Baxter, 2011). With regard to patient treatment choice, Sherman et al., (2004), found the largest sections of patients with CLBP within their study reported massage and chiropractic as being the most commonly used.

Furthermore, among prior users of CAM's, massage was rated most helpful of for LBP (Sherman *et al.*, 2004).

It has been suggested that there are over 100 different types of massage that exist (Eisenberg 1993). Furthermore, different massage styles such as, Swedish (classical) massage, Thai massage (acupressure), sports and remedial massage each use different groups of techniques. Classical (Swedish) massage typically is said to incorporate five main techniques, effleurage, petrissage, friction, tapotement and vibrations (Netchanok *et al.*, 2012). However, Weerapong (2005), identified only the first four as techniques used within this type of massage.

1.5 Structured clinical massage

More recently a variety of other soft tissue techniques have been incorporated into the massage treatments to address clinical and rehabilitative concerns (Cherkin *et al.*, 2009). These include, trigger point therapy, soft tissue release, strain counter strain / positional release, myofascial release and muscle energy techniques. In order to provide some consistency to the terminology when describing treatments Sherman et al. (2006), proposed a taxonomy that could be used to standardise massage treatments according to treatment goals. According to this taxonomy groups of massage techniques can be used for basic relaxation, clinical concerns, movement re-education and energy work (Sherman *et al.*, 2006). Within this classification clinical massage techniques involve focussed manipulations to the myofascial tissues in order to address specific concerns such as pain relief and movement restrictions through their influence on a number of other systems such as the circulatory and nervous systems (Sherman *et al.*, 2006). Typically massage therapists combine a number of treatment techniques however, therapists with more advanced training are able to incorporate clinical massage techniques in a structured format in the treatment of chronic musculoskeletal pain and soft tissue abnormalities such as CLBP (Cherkin *et al.*, 2009, 2011).

The common styles typically used to address chronic musculoskeletal contributors to LBP include friction massage, myofascial release techniques; such as myofascial release and connective tissue work; and neuromuscular techniques, such as myofascial trigger point therapy, muscle energy techniques; and position release / strain counterstain (Sherman *et al.*, 2006; Cherkin *et al.*, 2009). It is proposed that these massage styles and techniques can be used to alleviate the myofascial contributors to low back pain. Friction massage is typically used to address fibrotic tissue and is believed to break up adhesions and improve tissue circulation (Cherkin *et al.*, 2009). Myofascial release techniques are intended to optimise the function of fascia, release identified restrictions in myofascial tissues, address postural abnormalities, decrease pain and improve function (Barnes 1997; Myers 2009; Ajimsha et al. 2014). Neuromuscular techniques are used to address myofascial pain syndromes, lengthening contracted muscles, reducing muscle hypertonicity, relieve musculoskeletal pain and address agonist / antagonist muscle imbalances (Sherman et al. 2006; Cherkin et al. 2011; Ramsook & Malanga 2012; Wong 2012).

1.6 Myofascial contributors to LBP

The multifactorial aetiology and pathogenic mechanisms surrounding NSLBP have led to further investigations into other potential causes. Recently the influence of the myofascial systems and connective tissues have been implicated as potential mechanisms associated with LBP (Langevin and Sherman, 2007; Kao *et al.*, 2008; Myers, 2009a; Langevin *et al.*, 2011; Schilder *et al.*, 2014). Although many structures around the spine have been implicated as a source of LBP (Delitto *et al.*, 2012), there is increasing evidence that the fascial components of the lower back poses a network of nerve fibres including nociceptors sensitive to mechanical and noxious stimulation (Tesarz, Hoheisel, Wiedenhofer, *et al.*, 2011; Schilder *et al.*, 2014). Commonly, in the absence of identified organic lesions, NSLBP has been attributable to myofascial syndrome; which is a disorder that is characterised by the presence of myofascial trigger points within the muscular and fascial networks; leading to pain and disability. It

is largely caused by repetitive strain, muscle overload and poor postural habits (Kao *et al.*, 2008; Ramsook and Malanga, 2012). Furthermore, a number of studies have highlighted the adaptive nature of the fascia of the lower back to injury, overuse and immobility as a potential factor in the development of lower back stiffness, tissue fibrosis, altered movement patterns, and chronic pain (Langevin and Sherman, 2007; Langevin *et al.*, 2011; Klingler *et al.*, 2014).

2 Literature review

2.1 Models for the diagnosis and classification of LBP

The previous description of CLBP as pain, muscle tension or stiffness localised below the costal margin and above the inferior gluteal folds, with or without leg pain, lasting for 12 weeks or more in duration (Chou, 2010b) does not adequately reflect the complexity and multidimensional nature of the condition. Several structures in the spine, as previously outlined, may be involved including muscle, ligaments, zyagapophyseal (facet) joints, intervertebral discs, nerve roots, dura-matter and connective tissues (Delitto *et al.*, 2012). A number of models have been used to attempt to explain the pathology and aetiology of LBP including the pathoanatomical model, motor control models and biopsychosocial models (Hodges and Moseley, 2003; O'Sullivan, 2005; Panjabi, 2006). The following sections will provide an overview of these models.

2.1.1 Pathoanatomical model

Previous approaches to the diagnosis and treatment of LBP has often been focussed on individual dimensions of LBP as opposed to multidimensional approaches (O'Sullivan, 2005). The traditional approach to understanding the causes, diagnosis and treatment of LBP is that LBP is fundamentally a pathoanatomical condition which focuses on biomechanical and motor control deficits that contribute to degenerative influences of the spinal structures (O'Sullivan, 2012). This is based on findings that damage and degeneration to anatomical structures such as IV discs and facet joints are assumed to be the origin of LBP. Indeed, it has been suggested that facet joint pain, IV disc disruption and sacroiliac joint pain account for up to 70% of CLBP (Bogduk, 1995). This claim is supported further through the identification of facet joints pain through anaesthetic injection, and, disk studies that reported a clear statistical correlation between LBP and grade 3 fissures of the annulus fibrosis (McGill, 2007). Furthermore, findings from cross sectional studies also report significant associations between LBP and disc degeneration (Balagué *et al.*, 2012).

Critics of this perspective suggest that such pathoanatomical findings; including disc degeneration, annular tears, and disc bulges; have not been found to predict CLBP, and that only 8-10% of CLBP patients have an identified pathoanatomic diagnosis (O'Sullivan, 2012). Furthermore, there appears to be an issue with determining a pathoanatomic origin as a cause for CLBP as many abnormal findings are also observed in the pain free population (O'Sullivan, 2005). The suggestion that the pathoanatomical model may be limited is highlighted by studies that identify such false-positive findings (Delitto *et al.*, 2012). The authors report evidence of disc herniation present in 20%-76% of subjects without sciatica and 32% of subjects with disc degeneration was asymptomatic for pain. It appears that even with improvements in imaging technology, establishing a direct cause between the patient's condition and a pathoanatomic origin is not always possible (Delitto et al. 2012). Furthermore, despite the number of anatomical structures capable of producing pain, there is often diagnostic uncertainty surrounding NSLBP due to the lack of clinical features or imaging findings, making it difficult to identify any specific pathoanatomical cause (NICE, 2016) . If 8-10% have an identifiable pathoanatomical diagnosis the remaining patients may well be categorised into the NSLBP group (McGill 2007). However, although a lack of a clear pathoanatomical evaluation has often lead to large variations to both the diagnosis and treatment of LBP (Van Dillen and van Tulder, 2013). Therefore, traditional single dimension therapies and commonly prescribed interventions within the traditional biomedical model may not be superior to or show large effects within the NSLBP population (O'Sullivan, 2012). This suggests that there is a need for a more multidimensional approach to the diagnosis and treatment of LBP.

2.1.2 Motor control model

Motor control has been used to describe all aspects of the control of movement, but, alterations in motor control and control of the spine has become a potential factor in the development and persistence of LBP (Hodges, Cholewicki and Van Dieen, 2013). It

has been reported that patients with LBP are associated with compromised deep trunk muscles such as transversus abdominus and lumbar multifidus (Jull and Richardson, 2000), augmented or hyperactive superficial trunk muscles (D'hooge *et al.*, 2013), altered postural strategies following perturbations (Jones *et al.*, 2012), and persistent activation of para-spinal muscles (Colloca and Hinrichs, 2005). However, while evidence suggests that patients with low back pain have been observed to alter motor and postural control (Hodges *et al.*, 2003; Astfalck *et al.*, 2013), there appears to be variability in these findings and it is difficult to establish cause and effect (O'Sullivan 2005). For example, muscle activity of the lumbar erector spine muscle in the LBP population may be increased or decreased compared to healthy controls. (van Dieën, Selen and Cholewicki, 2003). Furthermore, trunk kinematic studies suggest that kinematic outcome measures such as ROM are not consistently affected in LBP subjects while the velocity of lumbar motion is and may be better discriminator of LBP from healthy individuals (van Dieën, Moseley and Hodges, 2013)

The possible mechanisms associated with motor control as a cause of LBP appear to be associated with the effect that motor control has on tissue loading and instability of the spine leading to damaged tissues, pain and inflammation (Yang, Marras and Best, 2011; van Dieën, Moseley and Hodges, 2013). Panjabi (2006), proposed that sub-failure injury and back pain may be caused by instability of the spine. The subsequent increase in mechanical loading secondary to instability may further lead to a decrease in the intrinsic stability of the passive structures, such as osteoligamentous and connective tissue, leading to more pain and disability. As all the structures of both the passive and active systems are extensively enervated by mechanoreceptors, proprioceptors and nociceptors, and the active parts of the stabilising system are under the intimate control of the CNS; at both the spinal cord reflex level and higher brain centres; any abnormal loading of the passive structures which threatens the systems integrity may be experienced as painful and alarming and may in turn further alter motor control (Hodges and Moseley, 2003; Jaap H. van Dieen , Luc P.J. Selen, 2003;

Jones *et al.*, 2012). In this sense motor control changes, could be seen to be both the cause and effect of LBP. In this sense motor control changes, could be seen to be both the cause and effect of LBP.

It has also been identified that specific motor habits such as muscle co-contraction (D'hooge *et al.*, 2013), habitually flexed postures (Solomonow, *et al.*, 2003), and type of mechanical loading (Marras *et al.*, 1995; Yang, Marras and Best, 2011) are factors associated with excessive loading to the spine. Increased muscle co-contraction has been identified in the LBP population and this co-contraction is often associated with high risk postures and during lifting tasks (Granata and Wilson, 2001). However, such contractions may increase spinal compressions during lifting tasks (de Looze *et al.*, 1999), contributing to potential sub failure injuries outlined above. Evidence also exists to suggest that habitually flexed postures such as slouched sitting and sustained trunk bending may lead to creep in viscoelastic tissues and injury. Studies have shown that cyclic loading to the supraspinous ligament can lead to the attenuation of multifidus reflexive activity (Solomonow *et al.*, 1999), potentially exposing the back to instability due to reduced muscular activity. Further evidence suggests that both cyclic and static loading of viscoelastic tissues can lead to micro-damage to the collagen structure, inflammation, reflexive muscular spasms to the multifidus muscle and back extensor hyper-excitability (Solomonow, *et al.*, 2003) . With regard to mechanical loading, occupational, biomechanical factors such as lifting frequency, load moment, trunk twisting velocity and trunk sagittal plane angle have been shown to significantly increase the risk of LPB (Marras *et al.*, 1995). Furthermore, it has been shown that an acute bout of light and heavy weight lifting resulted in an increase in Inflammatory cytokines (Yang, Marras and Best, 2011), and was significantly correlated with spinal tissue loads, suggesting certain occupational tasks may contribute to stresses and the sub-failure injury response outlined above.

Evidence for the effects of LBP on motor control is also associated with the effect of noxious stimulation, pain and cognitive variables on motor control changes in the LBP population. Two theoretical models currently used to explain the motor adaptations to pain have been postulated as potential explanations for the effects of injury and pain on motor control in LBP. These are the pain-spasm-pain model and the pain-adaptation model. Although a review of the literature has revealed that neither of these models consistently predicts the motor response to low back pain (van Dieën, Selen and Cholewicki, 2003; Hodges, 2011) a brief description of these models follows. The pain spasm pain theory (or vicious cycle theory) proposes that there is an intense contraction of muscles surrounding the injured structures or of the muscles that are painful or move the painful region. It is proposed that nociceptors in the damaged tissues of the low back may lead to an increase in muscle spindle output and subsequently a hyper-excitability of the motor neuron pool (Johansson and Sojka, 1991). This increase in muscle activity may then induce ischemia and an accumulation of pain related metabolites and the source of further pain (van Dieën, Selen and Cholewicki, 2003). The pain-adaptation model was developed to explain the lack of predictability of the vicious cycle theory and changes in voluntary movement patterns following pain and injury. It suggests that pain decreases the activation of muscles when acting as agonists and increasing the activation of muscles when acting as antagonists (Lund *et al.*, 1991). This typically would lead to a reduction in movement, movement velocity or force production as a means to minimise pain provocation. For example, low back pain subjects performing trunk flexion exercises have been reported to have reduced agonist abdominal activity and increased antagonist para-spinal muscle activity consistent with the predictions of the pain adaptation theory (D'hooge *et al.*, 2013).

Recent evidence, however, suggests that these theories are relatively simplistic and do not account for the varied redistribution of muscle activity within and between muscles in an individually specific manner (Hodges, 2011). Furthermore, it appears that a

number of RCT's that look at specific exercises addressing motor control impairments are not necessarily superior to conservative approaches for NSCLBP (Hodges, 2011). In the acute phase of injury to the spine, the removal of the noxious stimulus through movement and motor adaptations may be seen as an essential mechanism to support the recovery from injury. However, if the adaptation continues this maintenance of motor adaptations in the chronic phase may not be of benefit to the individual. Failure of the adaptation to be resolved may lead to continued compromise to the quality of movement, increased load on the tissues and an inefficient increase in muscle activity (Hodges, 2011).

2.1.3 Biopsychosocial model

More recently there has been a shift in the clinical and research approach to LBP from a biomedical model to a biopsychosocial model of LBP (Hancock *et al.*, 2011); which places more attention to the psychological and social factors that contribute to the development and maintenance of disability in LBP patient. The biopsychosocial model attempts to make a distinction between pain and disability and highlights the importance of an individual's reaction to pain (Waddell, 1987). It acknowledges the biological functions and limitations of LBP associated with the psychosocial contributors to the pain experience (Koenig *et al.*, 2014). A number of psychological and social factors have been attributed to the development and prevalence of LBP and disability. These include social variables such as job satisfaction, family and cultural issues and psychological variables such as fear avoidance, negative thinking, catastrophising and maladaptive coping strategies (O'Sullivan, 2005). It has been proposed that these psychosocial variables modulate patient attitudes and behaviours leading to greater pain and disability, (Vlaeyen and Linton, 2000), and these variables may be better predictors of LBP than pathoanatomical or traditional biomedical approaches (O'Sullivan, 2012).

Proponents of psychosocial influences on LBP have identified that compared to mechanical factors, psychosocial factors, such as job satisfaction and fear avoidnance,

play a more important role in determining the extent of LBP (Nachemson, 1992). Indeed some early studies that report psychosocial factors as strong predictors of LBP disorders in manual lifting jobs also identify the influence of mechanical factors, identifying peak lumbar shear force and lumbar disc compression biomechanical factors associated with LBP (Norman *et al.*, 1998). This was further highlighted by Hadler (2001), who pointed out that many cross sectional and multivariate studies detect associations between the psychosocial context of working and musculoskeletal disorders. There is further evidence that that poor job satisfaction, poor workplace social environments and reduced social support increase the risk of LBP in jobs that involve manual work and industrial lifting (Marras *et al.*, 1995; Norman *et al.*, 1998). Furthermore, an increased focus on the role of the central nervous system and central sensitisation has revealed that pain may be amplified by these psychosocial determinants of attitudes and behaviours and increase the CNS drive of pain (O'Sullivan, 2005). For example, fear avoidance and catastrophising have been associated with ongoing disability, and having neuromuscular consequences; in the form of modified maladaptive motor planning (Hodges, Cholewicki and Van Dieen, 2013); and neurobiological consequences; such as increased perceptions of pain, disruptions to the opioid analgesic systems and activation of a systemic inflammatory process (Campbell and Edwards, 2009).

Vlaeyen & Linton (2000), point out that fear avoidance is a central mechanism in the development of long-term low back pain leading to deconditioning and a downward spiral of activity avoidance and pain. The authors further point out that catastrophising thoughts may also be a precursor to pain related fear, and has been shown to lead to exaggerated distressful reactions to pain (Vlaeyen and Linton, 2000). It has also been reported that pain related fear associated with LBP may be more disabling in the long term than pain itself (Crombez *et al.*, 1999). Fear appears to promote a strong urge to remove oneself from the threat associated with pain and, in LBP patients, instigates a response that leads to the avoidance of movement and appropriate coping strategies

(Crombez *et al.*, 1999). Therefore, a combination of a catastrophising outlook and fear of injury and re-injury appear to be strongly associated with functional disability in LBP patients. This phenomenon could possibly lead to maladaptive postures; altered biomechanics of the spine that further contribute to the chronicity of LBP. Indeed, there is evidence to suggest that altered stress responses associated with fear avoidance, negative beliefs and catastrophising, may lead to heightened perceptions of pain, a cycle of pain and increased disability that is often associated with CLBP (O'Sullivan, 2012).

While the balance and contribution of the biopsychosocial model for LBP may vary from individual to individual (O'Sullivan, 2012), it has been widely accepted that LBP is a complex condition with multiple contributors to pain and disability, including psychological factors, sociological factors, biophysical factors, and pain procession mechanisms (Hartvigsen *et al.*, 2018). Therefore, the notion that LBP is a pathoanatomical phenomenon and treated from a biomedical point of view should be challenged. This is in part due to the more recent substantial evidence supporting the multifactorial approach to understanding and treating LBP. For example, it has been reported that only 8-15% of patients with LBP have a specific pathoanatomic diagnosis, suggesting that the majority of LBP cases are non-specific (O'Sullivan, 2012). Recently a number of systematic reviews concluded that it was unlikely that a number of mechanical factors; such as manual handling, bending and twisting; were independently causative of LBP (Balagué *et al.*, 2012). Furthermore, with regard to NSLBP, there has been a failure to identify any single biomedical treatment option that is superior to another or for single dimension therapies to exhibit large effects in patients with NSLBP (O'Sullivan, 2012). For example, the benefits of spinal stabilisation exercises continue to be promoted yet a number of RCT's have demonstrated that this approach is not superior to other conservative approaches and that multidisciplinary biopsychosocial approaches to rehabilitation are more effective than usual care and physical treatments (O'Sullivan, 2012; Kamper *et al.*, 2015).

Recommendations now exist in support of the limited use of laboratory and imaging tools for subjects with NSLBP (Foster *et al.*, 2018), as diagnostic labelling and medicalisation associated with a biomedical approach could exacerbate psychological factors and behavioural patterns such as fear avoidance and maladaptive lifestyle strategies. Contrasting this approach, a number or RCT's have also identified that positive outcomes are better predicted by psychosocial factors such as fear avoidance beliefs and pain coping strategies (O'Sullivan, 2012). In a review of the Effects of the treatment and management options for LBP it was recommended that; in view of the associations between behavioural, psychological and social factors; a biopsychosocial model should be used to inform the assessment and management of NSLBP (Foster *et al.*, 2018).

Evidence, therefore, supports the notion that LBP of a non-specific nature is a multifactorial phenomenon and should be treated and managed within a biopsychosocial model. Under the conceptualisation of this model it is therefore recommended that treatment and rehabilitation of NSLBP includes more than one form of treatment. While no firm boundaries appear to exist between the multifactorial contributors and that pain is not merely the result of nociceptive input from one particular anatomical structure, biophysical impairments such as alterations in paraspinal muscle size and function are demonstrable in some people with LBP (Hartvigsen *et al.*, 2018). Furthermore, lifestyle factors such as fear avoidance, catastrophising, maladaptive lifestyle behaviours and psychosocial factors may compound these biophysical features of LBP. Therefore, this thesis will focus on the biopsychosocial model and discuss the inclusion of massage therapy, and in particular integrated myofascial massage techniques, as a form of intervention that focusses on the improvement of biophysical component of the biopsychosocial model of LBP. This will focus on the reduction of pain and functional restoration through the use of a CAM, in the form of myofascial massage, as an intervention that could be utilised as part of a biopsychosocial multidimensional rehabilitation program and will look at the

mechanisms behind some of possible outcomes associated with this particular intervention. Indeed, recent guidelines have indicated that interventions such as self-management physical therapies and some forms of complimentary therapies can be useful as an adjunct or second-line treatment option as part of a multidimensional approach. This will be discussed in more detail in section 2.5 (thesis aims).

2.2 Functional anatomy of the spine

Non-specific LBP is a term commonly used to describe pain in the back between the bottom of the rib cage and the gluteal folds (NICE, 2016) . The description reflects both the complex and multidimensional nature of the condition and an absence of a definitive diagnosis. Although the term suggests that it is unlikely that the condition is caused by a serious pathology a plethora of anatomical structures in the spine are capable of generating pain (Delitto *et al.*, 2012). The following section will provide an overview of the functional anatomy of the lower back and pelvic areas and will be referred to as the lumbo-pelvic region. The pelvic region was included in this review as it includes those structures within the NICE (2016) definition for NSLBP describing pain between the 12th rib and gluteal folds. The review will also focus on the myofascial structures within this region and their contributions to back pain. The term 'myofascia' within this review will refer to the musculature and fascia of the lumbo-pelvic region, where fascia represents the connective tissue sheaths that surround and/or interpenetrate those muscles. A more detailed description of facia will follow in this section.

From a functional point of view the anatomical structures of the lumbo-pelvic region provide mechanical stability to the spine; whilst allowing for movement within a biomechanically determined physiological range; and can be separated into active, passive and neural components (Panjabi, 1992). Passive musculoskeletal structures include the bony structures, intervertebral discs, joint articulations, ligaments and joint capsules. Active musculoskeletal structures include all the muscles and tendons within

the defined region. The neural components refer to the neural and feedback systems contained within the active and passive structures and the neural control centres.

2.2.1 Osseous structures

The bony structures within the lumbo-pelvic region include 5 lumbar vertebrae a sacrum and two innominate bones. Each lumbar vertebra is composed of the vertebral body and a neural arch. The vertebral bodies are cylindrical and increase in diameter from L1-L5 to facilitate loadbearing requirements from L1-L5 (Rawls and Fisher, 2010). The neural arch is composed of the two pedicles that emerge from the posterior lateral surface of the vertebral body, which join with the two laminae that are located further posteriorly. Together the neural arch and vertebral body form a central vertebral foramen which forms part of the verbal canal that allows for protection of the spinal cord. Generally, the neural arch provides attachment for muscles and ligaments and the vertebral bodies provide attachment sites for the anterior and poster longitudinal ligaments. The superior and inferior notches of the pedicles combine to form the intervertebral foramina that allows the peripheral nerves to pass.

2.2.2 Articulations

The two, main articulation in the lumbar spine are the intervertebral (IV) discs and a pair of zygapophyseal or facet joints. The IV disc consists of an annulus fibrosis (AF), nucleus pulposis (NP) and two end plates and are located between each vertebral body. Its principle function is to separate the vertebral bodies and transmit loads between them (Levangie and Norkin, 2011). The vertebral end plates are cartilaginous structures that cover the superior and inferior aspect of the disc. The AF is a collagenous structure composed of concentric sheets of collagen arranged obliquely to the IV disc and whose function resist the expansion of the NP during compression. The AF is slightly thinner posteriorly and has a more vertically arrangement of collagen fibres, which may contribute to the more common posterior lateral disc herniation compared to anterior herniation (Ebraheim *et al.*, 2004). The NP consists of a gelatinous substance that is composed of approximately 70-80% water, proteoglycans

and scattered collagen fibres (Rawls and Fisher, 2010). The ageing process is said to contribute to a reduced disc water content, an increase in collagen and a decrease in the amount of elastin, thus reducing their ability to transfer loads from one disc to another (Levangie and Norkin, 2011).

The facet or zygapathogeal joints are composed of the inferior and superior articular processes of adjacent vertebrae and an articular capsule. The articular surface contains hyaline cartilage and the articular cartilage is composed of an inner synovium and an outer fibrous membrane. The diarthrodial nature of these joints protect the surface of the joints during flexion and extension and resisting shear and torsional forces (Levangie and Norkin, 2011). There regional variations in the structure of the joints in that the superior lumbar facets are concave and face posterio-medially while the inferior facets are convex and face anterio-laterally (Ebraheim *et al.*, 2004). In the lumbar region the facet angles are approximately 120 to 150 degrees to the sagittal plane which facilitates flexion and extension but restricts rotation (Rawls and Fisher, 2010). On their own the passive structures are said to buckle under loading without the integration of the myofascial and ligamentous structures that surround and interpenetrate the lunbo-pelvic region (McGill and ,Sylvain Grenier , Natasa Kavcic, 2003; Willard, 2007). There are many ligamentous structures of the spine and pelvis that are said to represent a dense connective tissue stocking that is critical to the stability of the region (Willard, 2007). The lumbar ligaments include the ligamentum flavum, interspinous ligaments, supraspinous ligaments, intertransverse ligaments, anterior and posterior longitudinal ligaments and the facet joint articular capsule ligaments. Ligaments of the sacrum include the iliolumbar ligament; the articular capsule of the sacroiliac joint; the sacrotuberous ligament and the sacrospinous ligament. The general structure and function of these ligaments are discussed elsewhere (Levangie and Norkin, 2011), However, some of the ligaments have specific fascial connection in the lumbar region, for example, the supraspinous ligament blends with the posterior layer of the TLF (Willard, 2007; Levangie and Norkin, 2011). The

supraspinous ligament is adhered to the lumbar spinal processes and the interspinous ligaments. This osseo-fascial-ligamentous complex is able to translate tension from the TLF to the lumbar vertebrae and, therefore, contribute to stability. Furthermore the ligamentum flavum is said to be continuous with the anterior border of the interspinous ligaments and is associated with reducing neural compression due to buckling (Vleeming, Vert and Stoeckart, 2007). Tensioning of the TLF is said to create an anterior posterior pull on the ligamentum flavum to prevent such buckling and assist in the alignment of the vertebral bodies (Willard, 2007).

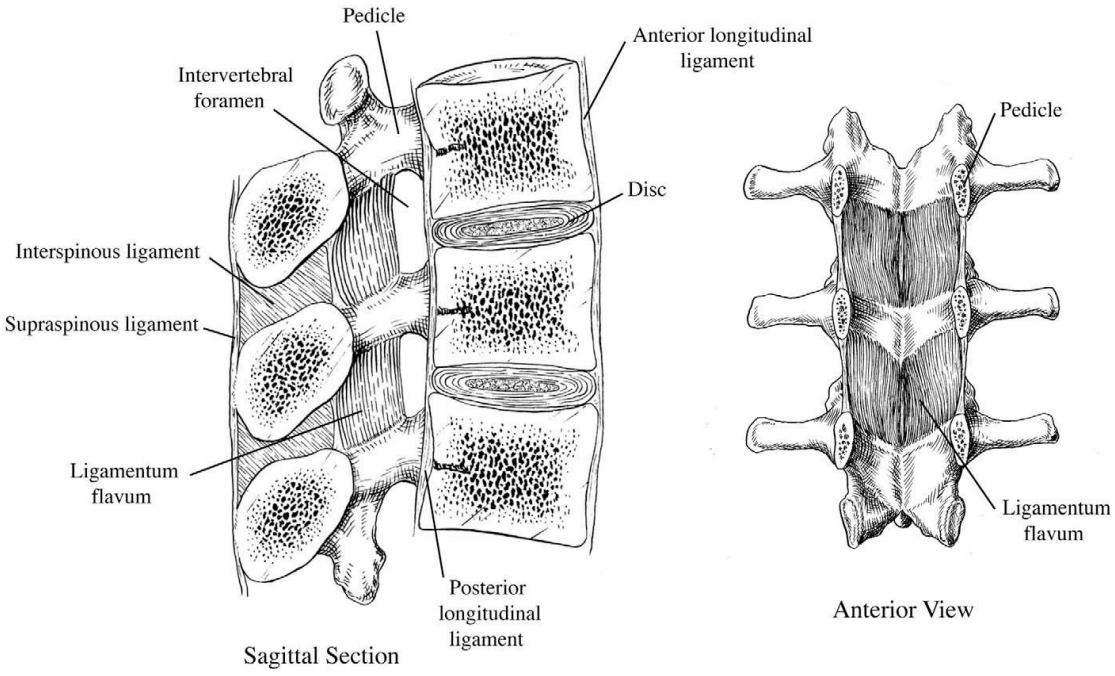

Figure 2. 1: Sagittal section and anterior view of the lumbar ligaments (Ebraheim, et al., 2004 p134)

Further stability is provided by the muscular system that, in coordination with the motor control centres, acts to stiffen the spine. (McGill & ,Sylvain Grenier , Natasa Kavcic 2003). These muscles include lumbar Multifudi (MF), Erector Spinae (ES), Latissimus dorsi (LD), Quadratus lumborum (QL), Psoas (Ps), the Gluteus maximus (GM), as well as the different abdominal muscles such as the Rectus Abdominis (RA), External and Internal obliques and Transversus abdominis (TrA). Previous studies have identified

that spinal segmental stability is achieved through activation of the deeper 'local' muscles whereas the more superficial 'global' muscles act across a much bigger areas to provide stability (Richardson, 1999). However, more recently evidence suggests that muscle activity is more diverse and unpredictable, responding to various challenges of instability through the activation of many muscles in a highly coordinated manner (McGill and ,Sylvain Grenier , Natasa Kavcic, 2003; Hodges, 2011). Through their connection to the passive structures and connective tissues the active muscular component is able to provide stability through a self-bracing mechanism to the lumbo-sacral area (Vleeming et al. 2007) . This myofascial anatomy will be discussed in more detail below.

2.3 Myofascial structures of the lower back

Recently anatomical linkages between the active muscular system and the passive connective tissue structures in the lumbo-pelvic region have suggested a possible role for specialised structures, such as myofascial and aponeurotic tissue, in both the stability of the lumbo-pelvic region and as a potential mechanism associated LBP (Benjamin, 2009; Willard *et al.*, 2012). The thoracolumbar fascia (TLF), or thoracodorsal fascia, is one of those structures and has been described as a myofascial girdle of the lumbo-pelvic region that contributed to posture, load transfer and stability of the lumbar spine (Benjamin 2009; Vleeming et al. 2007; Willard et al. 2012). The following section will review the anatomy and biomechanics of fascia and in particular the TLF and its implications for LBP and lumbo-pelvic stability.

2.3.1 Definition and classification of fascia

Fascia is a form of connective tissue composed of irregularly arranged collagen fibres (Willard *et al.*, 2012). The term fascia is often used to describe connective tissue sheets or bands that surround and separate muscle groups which may be differentiated from the regularly composed collagen fibre connective tissue structures such as ligaments, tendons and aponeurosis. Aponeurotic fascia can be defined as a thin, flat tendon-like expansion of connective tissue important in the attachment of muscles to

bones, other muscles, and in forming sheaths around muscles (Lemoon, 2008). Aponeurotic fascia is often composed of two or three layers of regularly arranged collagen that is separated by a thin layer of lose connective tissue that allows for the layers to glide across each other (Stecco *et al.*, 2013). Functionally, the collagenous arrangement of fascia and aponeurotic fascia are said to differ in that the irregular arrangement is suited to withstanding stresses in multiple directions whereas the regular arrangement is suited to withstand stresses in a particular direction (Willard *et al.*, 2012). Although often described in the literature as 'fascia' the TLF has the structure of aponeurotic fascia (Stecco *et al.*, 2013), and, therefore, can be described as sheets of facia that are aponeurotic in nature. All types of fascia are forms of connective tissues. Fascia is typically classified into three (Stecco, 2004) or four types (Willard *et al.*, 2012), and more regionalised classifications of fascia can also be found (Benjamin, 2009). However, as the focus of the overview is of on the TLF, the following section will focus on the superficial and deep fascia.

Superficial fascia is typically described as fascia that surrounds the body, forms the subcutis layer of the skin and is composed of areolar connective tissue with loosely packed interwoven collagen and an abundance of elastin fibres (Benjamin, 2009; Willard *et al.*, 2012; A. Stecco *et al.*, 2015). The superficial connective tissue has been identified as having independent sublayers that, along with its collagen and fibre arrangement, contribute to its ability to glide across underlying tissue and recoil (Stecco et al., 2015; Willard et al., 2012). The superficial fascia is intimately connected with neurovascular structures, conveying blood vessels and nerves to and from the skin (Benjamin, 2009). Recent evidence has suggested that the superficial fascia is a fibrous layer that separates the adipose tissue into superficial and deep portions (Stecco et al., 2015). Fibrous septa, or retinacula, extend from the superficial fascia to the skin above (sometimes referred to as skin ligaments) and deep fascia below, providing a three-dimensional structural connection between the skin and deep fascia.

The prevalence and thickness of these subcutaneous septa vary according to region, gender, age and levels of physical stress applied to the region (Stecco et al., 2015).

The superficial layer of fascia is formed of loosely packed collagen and many elastic fibres arranged in an irregular fashion. Both the superficial fascia and Retinacula, provide mechanical support for blood vessels and nerve fibres in the subcutaneous region. Furthermore, in separating the skin from the underlying muscle tissue it permits a normal sliding of muscle and skin upon each other (Benjamin, 2009). Within the sub-cutis layer of the skin nerve endings such as Ruffini and Pacinian corpuscles have been shown to be present, with some passing through the superficial fascia and some embedded within the fascia, enabling them to respond to mechanical stimulus such as pressure and stretch (Schleip, 2003b). The elastic component of the superficial retinacula and the superficial fascia act to facilitate the buffering of mechanical stresses from the deep fascia and muscles to the skin and prevent deep fascia nerves from being activated due to normal movement of the skin. Scarring within the sub-cutis region has been shown to create a rigid connection between the skin and deep fascia (Stecco et al., 2015). Therefore, any abnormal fibrosis of the superficial facia may lead to abnormal force transmission to the deep facia.

Deep or investing fascia is deep to the superficial fascia and is generally described as being a more dense fibrous layer of connective tissue and classified as either aponeurotic or epimysial fascia (Benjamin, 2009; Stecco et al., 2015; Willard et al., 2012). It has a more organised and parallel arrangement of collagen fibres (Stecco, 2004; Benjamin, 2009). It is proposed that the deep fascia is composed of a number of layers or sheets, separated by a thin layer of loose connective tissue that enables the sliding of adjacent layers (Pavan *et al.*, 2014). It is also considered to be the fascia that interpenetrates and surrounds the bones, joints, muscles, tendons and ligaments; including myofascial expansions and reinforcing structures such as retinacula (Stecco

et al., 2015; Stecco et al., 2015). However, the following sections will review the deep fascia according to their main classification as aponeurotic or epimysial fascia.

The aponeurotic fascia consists of sheaths of connective tissue that are approximately 1mm thick that cover and keep in place a group of muscles or to act as tendinous insertion for other muscles (Stecco et al., 2015). It is independent of the underlying muscle and can transmit forces over a distance (Willard *et al.*, 2012). The free gliding of muscles under the aponeurosis of the body is permitted due to a layer of loose connective tissue, between the aponeurosis and the underlying epimysium, which is rich in hyaluronan; a hydrophilic glycosaminoglycan (GAG) that contributes to the viscoelastic properties of fascia (see 2.3.2 below). It is mainly composed of type I and III collagen fibres in parallel formation and a reduced elastic component compared to the superficial fascia (Stecco et al., 2015). A number of studies have demonstrated the presence Ruffini and Pacinian fibres in close proximity to blood vessels and closely connected to the collagen fires of the deep fascia (Schleip, 2003a). However, the innervation of the deep fascia cannot be said to be homogenous throughout the body (Stecco et al., 2015) , therefore a more detailed review of the innervation of the thoracolumbar fascia is presented in 2.3.3 below. From a mechanical perspective aponeurotic fascia exhibits stress strain responses similar to other connective tissues (Stecco et al., 2015). The parallel arrangement of collagen fibres within the aponeurotic fascia provides a tensile strength allows it to act as a tendon, transferring muscular forces from one area to another (Willard *et al.*, 2012). However, due to its collagen arrangement this fascia has been shown to have a greater stiffness when stressed in a longitudinal direction compared to the transverse stresses and has been shown to exhibit viscoelastic properties of rate dependent strain, creep, stress relaxation and hysteresis (Stecco et al., 2015).

The epimysial fascia is a thin layer of connective tissue that envelops specific muscles and is continuous with the fascial compartments within the muscle of the perimysium

and endomysium. Its function is to transmit forces between adjacent muscular fibre bundles (Huijing, 1999). All three fascial sheaths contain collagen and elastin fibres and a high quantity of Hyaluronic acid (HA) within the ECM allowing for individual muscle fibres, fascicles and muscle groups to glide within and between each other effectively (Stecco et al., 2015). Both the epimysium and perimysium are considered to be dense regular connective tissue types with parallel collagen formations. They also contain neurovascular tracts that lie between muscle groups and fascicles respectively. Furthermore, both the epimysium and perimysium are connected to the muscle spindle's specialised connective tissue capsule (Stecco et al., 2015). Some studies have suggested that this close relationship may influence muscle contraction and motor control (Stecco, 2004).

2.3.2 Fascia Architecture

The fascial network is primarily composed of cells and an extracellular matrix (ECM). The dominant cell of fascia is the fibroblast which is known to maintain and regulate the ECM (Simmonds, Miller and Gemmell, 2012). Fibroblasts are known to communicate with each other via gap junctions and are capable of responding to mechanical stress and stretch. (Benjamin, 2009; Simmonds, Miller and Gemmell, 2012). The changes in cell shape associated with stretching of connective tissue, are said to be integral to the mechanotrasnduction properties of fascia (Stecco et al. 2015). The principle components of the ECM are the collagen and elastin fibres and a viscous like gel that the cells and fibres reside, called the ground substance (Levangie and Norkin, 2011). The collagen fibres provide the structural integrity and are capable of withstanding high tensile forces while the elastin fibres allow for extensibility and elasticity of fascia (Levangie and Norkin, 2011). As with all connective tissues fascia is a viscoelastic material exhibiting elastic properties as well as viscous resistance to flow along with time and rate dependent deformation characteristics such as creep, stress relaxation, strain rate sensitivity and hysteresis (Levangie and Norkin, 2011; Pavan *et al.*, 2014).

The ground substance of the ECM contains non-collagenous glycoprotein units called proteoglycans (PG) (Levangie and Norkin, 2011). PGs are a heterogeneous group of glycoproteins characterised by one or more linear glycosaminoglycan (GAG) side chain covalently bonded to a core protein (Koźma *et al.*, 2000; Levangie and Norkin, 2011). Many of the he GAGs are highly negatively charged and the high density of negatively charged GAG's attract water into the ECM forming a hydrated gel. This gel contributes to several properties of connective tissues including ECM hydration, maintenance of collagen fibre distance, resistance to compression, diffusion of metabolites, and contributing to the viscoelastic properties of fascia (Levangie & Norkin 2011; Stecco et al. 2013; Stecco et al. 2015). From an intra-muscular perspective the ECM also provides continuations of neurovascular tracts in which nerves and blood vessels are embedded (Voermans *et al.*, 2008). Existing within the ECM is a GAG called hyaluronan (HA). HA does not bond covalently to a core protein but instead PG's attach to HA via link proteins to form giant hydrophilic macromolecules. HA is therefore said to contribute to lubrication of muscles and tendons as they glide over or under aponeurotic fascia (Stecco et al. 2015). Previously the ECM has been considered as an amorphous scaffolding that provides mechanical support (Klingler *et al.*, 2014), however recent insights have revealed that this matrix is a dynamic structure that modifies the mechanical and viscoelastic behaviour of fascia to mechanical loading and unloading; can withstand and transmit contractile forces from muscles; and contribute to the viscoelastic nature of fascia (Kjaer *et al.*, 2006; Voermans *et al.*, 2008; Klingler *et al.*, 2014).

2.3.3 Thoracolumbar fascia

The TLF is the deep aponeurotic component of the posterior aspect of the axial fascia. It surrounds the muscles posterior to the transverse processes and separates the paraspinal muscles from the muscles of the posterior wall. This multi-layered connective tissue structure is attached inferiorly at the iliolumbar ligament, iliac crest and SI joint. Cranially it is continuous with the paraspinal thoracic and cervical fascia and terminates at the cranial base (Willard *et al.*, 2012). In the lumbar region it attaches

medially to the spinous processes and is typically described as having three layers; anterior, middle and posterior layers (Willard, 2007; Loukas *et al.*, 2008; Levangie and Norkin, 2011). The anterior and middle layers surround the QL muscle, the middle and posterior layers surround the paraspinal muscle groups of the ES and MF (Willard *et al.*, 2012) (diagram 2.2).

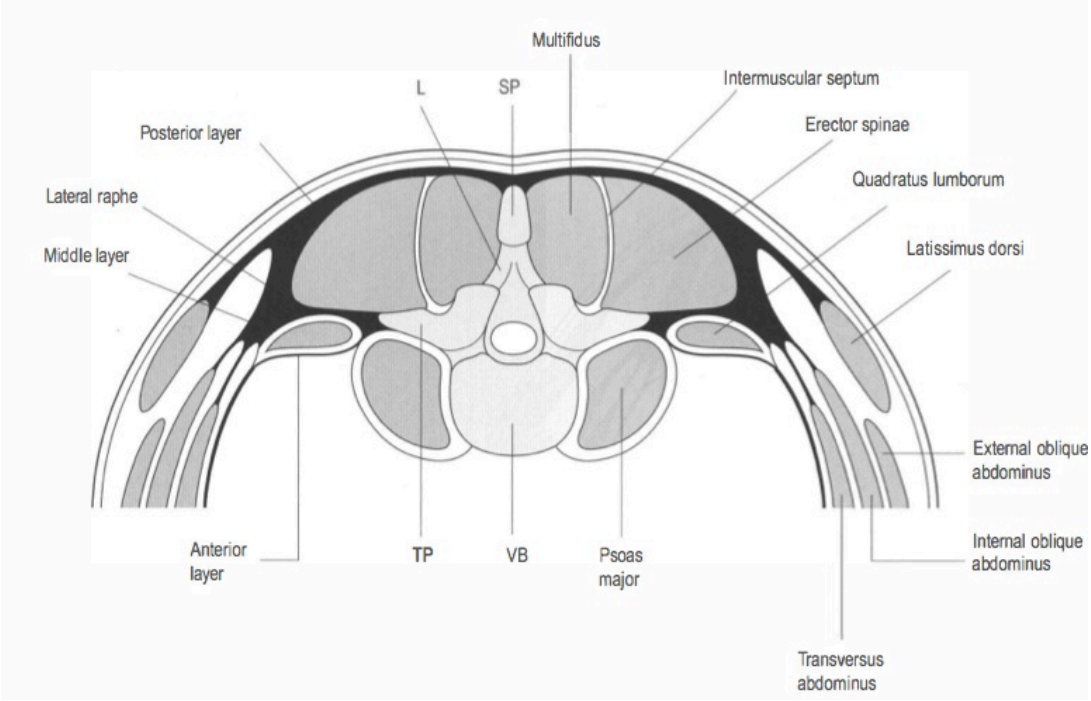

Figure 2. 2 Cross section of the lumbar region containing the anatomical relationships of the TLF. L, lamina; SP, spinous process; TP, transverse process; VB, vertebral body (Vleeming & Stockhart 2007 p 49).

The posterior layer of the TLF (PLF) can be further separated into a deep and superficial layer or lamina (Willard, 2007; Loukas *et al.*, 2008; Willard *et al.*, 2012) . The superficial lamina of the PLF has a mean thickness of 680µm, composed of a parallel arrangement of collagen fibres with few elastic fibres and is reported to be continuous with the aponeurosis of the LD muscle (Willard, 2007) and the GM muscle (Loukas *et al.*, 2008) . The superficial layer of the PLF is also reported to have a number of sub layers composed of loose connective tissue which allows for efficient gliding between the superficial and deep layers of the TLF, and muscular coordination within the area (Willard *et al.*, 2012). For example, at the L3 level of the lumbar spine the aponeurosis of the serratus posterior inferior lies between the superficial and deep layers of the

PLF, while the LD muscle invests and blends in to the superficial layer of the TLF (Willard *et al.*, 2012). Therefore, the contraction and smooth motion of these muscles are facilitated through the loose connective tissue sublayers between these fascia layers. The deep layer fuses with the superficial layer as it projects towards the midline which then attaches to spinous processes and supraspinous ligament. Caudally it fuses with the iliac crest and aponeurosis of the GM muscle. Together the PLF links two of the largest muscles in the body the LD and GM (figure 2.3). The attachment of these two diverse muscle groups into It the TLF has been reported to integrate and co-ordinate muscular activity between the upper and lower extremities (Benjamin, 2009). For example the it is proposed that, through its attachment to the PLF, the GM and LD promote the coordinated, pendulum motion of the arms and legs during walking (Vleeming et al. 2007). EMG evidence has identified contralateral co-contraction of these muscles during gait and torsional forces to the TLF may be stored as elastic energy which contribute to a more energy efficient movement pattern (Vleeming, Vert and Stoeckart, 2007).

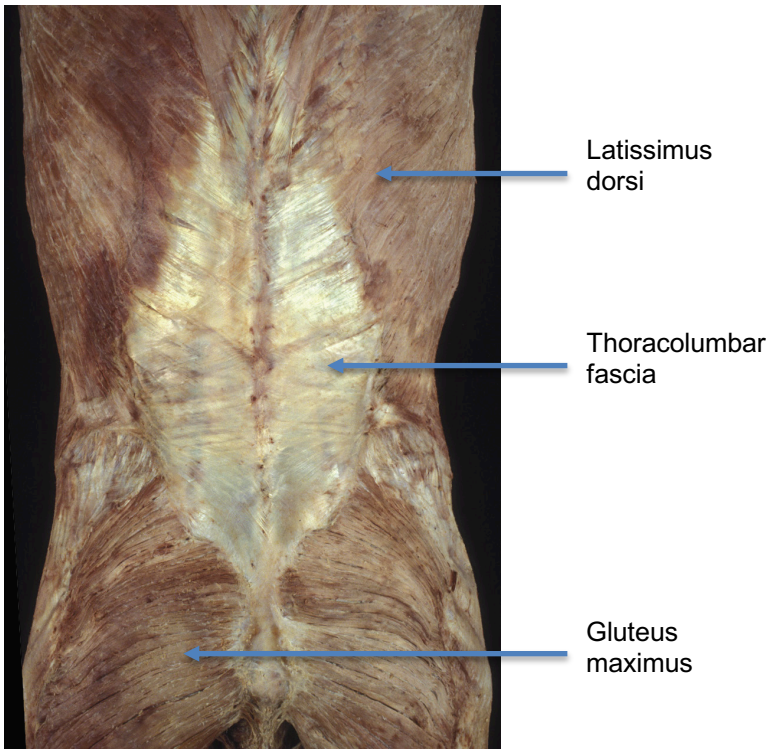

Figure 2. 3 Posterior view of the back, illustrating attachments of the LD and the GM to the thoracolumbar fascia (TLC) (Willard et al. 2012 p 518).

As the deep lamina of the PLF passes from its medial attachment of the spinous processes it then passes anteriorly to the transverse processes where it meets the middle layer of the TLF. The middle layer extends laterally to meet the PLF to form a facial compartment that contains the paraspinal muscle groups the illiocostalis, longissimus and multifidus muscles. On the lateral aspect of this compartment the TLF merges with the aponeurosis of the TrA termed the lateral raphe (Vleeming et al. 2007). The internal abdominal oblique and the transversus abdominal muscles arise from the lateral raphe (fig 2.2). Due to the relative aponeurotic connections with these muscles, contraction of these muscles are said to create tighten effect on the TLF which may contribute to a tensioning and enhanced stability through extension of the spine (Gracovetsky, 2008; Levangie and Norkin, 2011). Furthermore, the Lumbar MF muscles have been identified as having insertional attachments to the interosseous SIJ ligaments and TLF which, when activated, can lead to tensioning of the SIJ ligaments (Willard *et al.*, 2012). Therefore, as well as contributing to movement of the Lumbosacral region, muscles acting on and around the TLF and connective tissue structures of the Lumbosacral region have been proposed to be capable of tensioning the spine to affect spinal segmental stiffness and augment spinal stability. As such the TLF may play a role in the development, prevention and treatment of LBP (Loukas *et al.*, 2008).

2.3.4 Blood supply to the lumbar spine and fascia of the lower back

The blood supply of the lumbar spine is predominantly from the intercostal, iliolumbar and lumbar arteries (fig 2.4), which segment and branch off and supply the spinal cord, vertebrae and cauda equine (Ebraheim *et al.*, 2004). As described above the anterior and middle layers of the TLF surround the QL muscle, the middle and posterior layers surround the paraspinal muscle groups of the ES and MF. In the lumbar region, these muscles are supplied by the dorsal branches of lumbar arteries, the subcostal arteries

and intercostal arteries. The information on the vascularisation of aponeurotic deep fascia more generally remains inadequate (Stecco et al. 2015), however, some histological studies of the TLF have identified a network of blood vessels, particularly at the posterior layer of the TLF, some of which have been reported to accompany sympathetic nerves (Willard *et al.*, 2012). However there is growing evidence that vascular networks have been observe in the regions between the superficial fascia and deep fascia along with well-developed lymphatic vessel (Stecco et al. 2015). Superficially the fascial network contains arteries of small and medium diameter at the level of the sub cutis and superficial fascia that travel in a longitudinal and perpendicular direction and forming two subcutaneous plexuses; the papillary plexus and the deep plexus (Stecco et al. 2015). The superficial fascia contains both reticular and epifascial veins that drain the cutaneous micro circulation along with a well-developed lymphatic plexus in the subdermal level that eventually join the lager lymphatic vessels in the deep fascial layers.

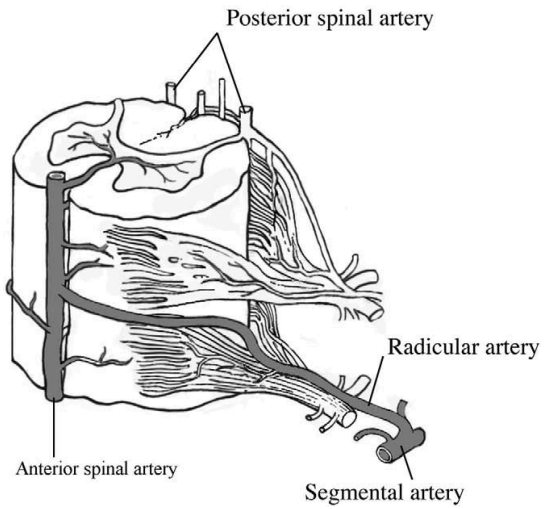

Figure 2. 4 Blood supply to the spinal cord (Ebraheim, et al., 2004 p135)

2.3.5 Innervation of the TLF

The thoracolumbar region of the spine is innervated by the dorsal rami of the spinal nerves (Ebraheim *et al.*, 2004; Willard, 2007; Tesarz, Hoheisel, Wiedenhöfer, *et al.*, 2011) and most structures in the lumbosacral regions receive a generous nerve supply (Willard, 2007). Although there is a lack of studies about the innervation of the TLF compared to other spinal structures, recently a number of reports have identified a range of innervating structures within the TLF (Yahia *et al.*, 1992; Tesarz, Hoheisel, Wiedenhöfer, *et al.*, 2011; Schilder *et al.*, 2014). Of the range of innervating structures identified a high density of sympathetic fibres was reported within the TLF of both rats and humans (Tesarz, Hoheisel, Wiedenhofer, *et al.*, 2011). Furthermore, Some of these nerve ending were in close proximity to blood vessels and because of this proximity have been proposed to have a vasomotor component (Tesarz, Hoheisel, Wiedenhofer, et al. 2011). Schleip (2003), explains that type III and IV interstitial nerve ending identified within fascial tissue may be polymodal as stimulation of these receptors have been shown to affect local blood pressure, and lead to reduction in global muscular tone through an increase in vagal tone. The same author also suggests that sympathetic activity may be reduced through stimulation of ruffini endings within fascia (Schleip, 2003a).

The presence of proprioceptive receptors within the PLF, such as Golgi, Pacini and Ruffini endings have also been reported (Yahia *et al.*, 1992; Schleip, 2003a), suggesting a possible proprioceptive role of the TLF. Yahia et al. (1992) found the TLF to be well innervated with Ruffini and Pacinian corpuscles. The same authors also identified the same receptors within the supraspinous and interspinous ligaments and Goilgi Tendon Organs in the iliolumbar ligament. Cutaneous mechanoreceptors have been identified as important components of the sensorimotor system (Riemann and Lephart, 2002). It has been proposed that tactile stimulation of cutaneous receptors has the capacity to alter muscle activation through the gamma motor neurons (Riemann and Lephart, 2002; Konishi, 2013). Furthermore, stimulation of connective

tissue afferents within the spine have been observed to elicit muscular contractions suggesting a possible mechanoreceptive response to injury to ligaments, discs and facet joints (Holm, Indahl and Solomonow, 2002). With regards to position sense, cutaneous afferents have been speculated to respond mainly at extreme ranges of movement (Riemann and Lephart, 2002; Willard *et al.*, 2012). However, Willard et al. (2012), points out that due to the relatively greater distance from the joint axis, mechanoreceptors in the TLF may experience sufficient stretch to provide sensory feedback on joint positioning. Furthermore, as previously mentioned, the interconnectivity between the TLF, supraspinous and interspinous ligaments may suggest a combined proprioceptive mechanism through a combination of fascial stretch and tensioning through the ligaments during movements that stretch the fascia (Willard *et al.*, 2012). Stecco et al. (2013) suggests that there is a close connection between the capsules of corpuscles and free nerve endings to collagen fibres within the deep fascia. Therefore, any stretching of the deep fascia; either directly or as a result of muscular contractions; could result in stimulation of these nerve endings (Stecco *et al.*, 2013). Additionally, evidence also suggests that the capsules of muscle spindles can correspond to the epimysium, perimysium or to fascial septa (Stecco *et al.*, 2015). Therefore muscle spindle function may be influenced by the structure of this fascia and the surrounding deep facial layers and their myofascial expansions (Stecco *et al.*, 2013)

A potential nociceptive role for fascia has been identified due to the presence of free nerve endings within the posterior layer of the TLF (Yahia *et al.*, 1992; Tesarz, Hoheisel, Wiedenhofer, *et al.*, 2011). Tesarz, Hoheisel, Wiedenhofer, et al. (2011), reported that these nerve fibres were mainly located within the PLF and subcutaneous tissues compared to the deeper layers some of which were classed as being nociceptive in nature. It has been reported that dorsal horn neurons receive input from the TLF (Tesarz, Hoheisel, Wiedenhofer, *et al.*, 2011; Schilder *et al.*, 2014), and the Dorsal Rami of the spinal nerves that innervate the TLF have been shown to contain

myelinated and unmyelinated fibres that are sensitive to mechanical and noxious stimulation (Tesarz, Hoheisel, Wiedenhofer, *et al.*, 2011). Further evidence of a nociceptive function of the TLF has been reported trough a number of studies that attempted to elicit pain through stimulation of the TLF (Willard *et al.*, 2012). Injection of hypertonic saline into the subcutis, muscles and thoracolumbar fascia found that the TLF was more sensitive to pain compared to the subcutis tissue layer and underlying muscles (Schilder *et al.*, 2014). The authors report that the pain response due to hypertonic saline suggests the presence of nociceptive afferents within the TLF (Schilder *et al.*, 2014). Taken together the dense sensory innervation of the TLF may suggest an important link with LBP.

2.4 Massage and low back pain

Although the increased prevalence of massage therapy as a treatment for low back pain has been identified, the Effects of massage and mechanisms by which massage exerts its effects are less well known. Methodological limitations and inconsistencies have been identified within the literature but recent systematic reviews, clinical practice guidelines and evidence reports have shown that massage therapy may have some role to play in the treatment of LBP (Furlan *et al.*, 2009; Chou, 2010b; Simmons, 2011; Brosseau *et al.*, 2012; Netchanok *et al.*, 2012). The Cochrane back review group identified that massage may be beneficial for patients with CLBP especially when combined with exercise and education (Furlan *et al.*, 2009). In a comparison of Swedish massage and traditional Thai massage both were seen to relieve chronic lower back pain. Of the two types of massage techniques compared, traditional Thai massage was seen to have a greater effect on improvements in functional ability (Netchanok *et al.*, 2012). However, the same review revealed that methodological limitations reflected the need for further research in this area.

Compared to other interventions massage was seen to be more effective at improving short and long term pain intensity and function, however, when compared to exercise,

massage was only beneficial in the short term for reducing pain in people with chronic low back pain (Chou, 2010b). In a review of CAMs for low back pain Simmons, (2011), reported that massage was superior to placebo or no treatment in reducing pain and disability immediately post treatment only in acute and sub-acute back pain. However, massage was significantly better than relaxation or physical therapy in reducing back pain intensity for chronic non-specific LBP sufferers but not ROM. The Ottawa Panel evidenced based clinical practice guidelines for therapeutic massage for low back pain (Brosseau *et al.*, 2012) was able to demonstrate that massage was an effective treatment to decrease disability and relieve chronic LBP symptoms at immediate post treatment when compared to acupuncture, self-care, relaxation therapies, conventional physiotherapy and placebo. Cherkin et al., (2011), compared two types of massage with usual care for CLBP and found that both massage interventions improved function and decreased pain compared to usual care after 10 weeks of treatment. Furthermore, these improvements were maintained at 26 weeks' post treatment, suggesting that massage may have beneficial effects over and above the immediate post treatment effects outlined above, however, the study questioned the clinical significance of these effects at 52 weeks.

2.4.1 Massage research limitations

Although recent evidence points towards massage as an effective treatment for low back pain the literature suggests that a number of methodological issues cast some doubt as to the clinical significance of massage as an intervention for low back pain (Ezzo, 2007; Simmons, 2011; van Middelkoop *et al.*, 2011; Patel *et al.*, 2012). In a systematic review of the physical and rehabilitation interventions chronic non-specific LBP Van Middelkoop et al., (2011) found that massage therapy compared to relaxation and acupuncture massage showed no significant difference in pain intensity. The authors also cited that serious limitations contributed to low quality evidence. This was also echoed by the evidence report on CAMs for low back pain (Simmons, 2011), who identified that variations in massage techniques, frequency and duration of treatments,

and types of placebo may all contribute to different results in trial. Therefore, a massage taxonomy with specific definitions and descriptions would significantly assist in researchers selecting and standardising techniques (Patel *et al.*, 2012). Furthermore, where massage has been compared with other modalities the study designs were not such that the relative contribution to massage could be properly interpreted (Ezzo, 2007; Patel *et al.*, 2012). The same studies also point out those optimal parameters for frequency, duration and depth of pressure have not been identified and need to be addressed to further standardise research trials and determine an optimal dose response relationship. Furthermore, the lack of blinding, inherent in massage trials, represents a bias that could exaggerate a treatment effect. Alternative design features, such as inclusion of non-subjective outcomes and a minimum measure of blinding the outcome assessor should be adopted (Ezzo, 2007; Simmons, 2011; Patel *et al.*, 2012).

Despite the popularity of massage many of the reviews on the Effects of massage suggest that many studies are unable to identify whether a massage treatment is truly effective or not. One of the main problems associated with massage research is the lack of consensus over an accepted definition. One of the most prevalent definitions used in massage research is ‘a group of systematic and scientific manipulations of the body tissues best performed with the hands with the purpose of affecting the nervous system, muscular system and general circulation (Lewis 2006). However, many other definitions exist. The American Massage Therapy Association describes it as “a form of manual soft tissue manipulation that includes holding, causing movement and/or applying pressure to the body” (AMTA 2013). Other definitions include “the mechanical manipulation of body tissue with rhythmical pressure and stroking for the purpose of promoting health and well-being” (Caferelli 1992) and, “the management, manipulation and rehabilitation of soft tissues of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being (SMA 2016). In addition to the problem of definition, Morsaka (2007), Identify that variations in the type of massage

technique used, massage therapist skill level, sample size and dosage all contribute to the lack of consensus on the Effects of massage therapy. All of which then contribute to the variability in application of techniques used within the literature. Furthermore, along with the variety of techniques, variability exists within their application. Fritz (2005) identifies that within each technique the depth, duration, direction, drag, frequency, speed and rhythm of application, may vary, yet few studies allow for this variation or are able to control such parameters.

2.4.2 Proposed effects of massage

The possible effects of massage are likely to be achieved through a number of mechanisms, and various propositions have been put forward. These effects and benefits are usually assigned to a variety of types of massage used within a particular study Weerapong et al., (2005), reviewed the effects of what was termed classical (Swedish) massage which was typically used to promote relaxation and is consistent with the techniques within the relaxation massage taxonomy described above. They identified the possible mechanisms and effects on performance, muscle recovery and injury prevention as being biomechanical, physiological, neurological and psychological in nature. However, they concluded that the effects were unclear due to limited evidence.

More recently however, research into the effects of relaxation massage and clinical massage on pain (Sagar, Dryden and Wong, 2007; Frey Law *et al.*, 2008; Adams, White and Beckett, 2010; Simmons, 2011; Brosseau *et al.*, 2012); neuromuscular outcomes and range of movement (ROM) (Maria Hernandez-reif, Tiffany Field, Josh Krasnegor, 2001; Sefton *et al.*, 2011; Netchanok *et al.*, 2012) appears to be more conclusive. When outcome measures of pain and function were examined, Simmons (2011), concluded that massage had more of an impact on pain intensity than on function or ROM. In a comparison of relaxation massage and structured clinical massage with usual care results indicated that both massage groups had improved pain and functional outcomes compared to usual care and with the effects lasting for at

least 6 months (Cherkin *et al.*, 2011). In a double blinded RCT Frey Law et al., (2008) identified that deep tissue massage reduced mechanical hyperalgesia (27.5%) and pain perception (48.4%), however this was following eccentrically induced DOMS to the wrist extensors and not specific to back pain. Adams et al., (2010) sampled 53 hospital patients from a variety of units in an evaluation of the effects of massage on pain in the acute care setting and observed a significant reduction (5.81 pre-treatment to 2.01 post-treatment) in pain recorded on a visual analogue scale. However the massage protocol was not standardised for time or type in this study. Ghanbari, Rahimijaberi, Mohamadi, Abbasi, & Sarvestani, (2012) did specify the treatment protocol and specifically utilised position release technique (PRT) compared to routine medical therapy to manage trigger point tension headache. They observed that both procedures were equally effective in reducing tension type headache frequency and duration but concluded that the reduction in trigger point pain sensitivity was successfully treated by PRT. Trampas, Kitsios, Sykaras, Symeonidis, & Lazarou, (2010) attempted to determine the immediate effects of clinical massage techniques on latent trigger points in muscles innervated by the lumbosacral, sciatic, tibial and common peroneal nerves. They identified significant improvements in ROM and pressure pain threshold following trigger point compression therapy accompanied by a modified PNF stretching technique.

The effects of massage on neurophysiological mechanisms have also been explored. Kostopoulos, Nelson, Ingber, & Larkin, (2008) identified that the use of TrP compression therapy with passive stretching in tandem reduced the spontaneous electrical activity and pain perception of TrP's in the upper trapezius. These techniques can be classified under the clinical massage techniques (Sherman *et al.*, 2006). The authors concluded that the massage techniques utilised may have led to the resolution of the Tr'p and its pathophysiologic ischemic state. Arroyo-Morales et al., (2008) utilised another clinical massage technique, myofascial massage, applied as a passive recovery following intense exercise and recorded reduced EMG amplitude of vastus

medialis muscle during MVC. The authors postulated that the mechanisms of myofascial release may have contributed to the reduced EMG amplitude. Myofascial release is a form of manual medicine that involves the application of low load and long duration stretching to the myofascial complex (Ajimsha, Daniel and Chithra, 2014). The effects of which are proposed to lead to reduced muscle tone through muscle lengthening and changes to the sympathetic / parasympathetic balance (Barnes, 1997; Schleip, 2003a). The technique has also been observed to reduce pain, improve function and range of movement (Castro-Sánchez *et al.*, 2011; Nitsure and Welling, 2014; Namvar and Moghadam, 2016). Although the myofascial massage techniques used were not standardised to the taxonomy described above (Sherman *et al.*, 2006) they were consistent with the myofascial release techniques within the clinical massage category.

Further evidence of a neuromuscular effect was observed following 20 minutes of therapeutic massage to the neck and shoulder region compared to a control and Light touch intervention (Sefton *et al.*, 2011). Results led to a reduction in the alpha MN pool excitability of the flexor carpi radialis muscle; as measured by the Hoffman reflex; and a reduction in EMG signal amplitude of the upper trapezius muscle. As the reductions in the flexor carpi radialis alpha MN pool were observed distal to the area of treatment the authors suggested that there was a centralised effect on the alpha MN pool. The same authors also observed a 12-13% increase in cervical ROM. Furthermore Behm et al., (2013) assessed neural and evoked muscle responses with 2 types of massage techniques; effleurage and circular stroking vs. tapotement massage; combined with static stretching, of plantar flexor muscles and recorded reductions in soleus muscle H/M ratio compared to a control. They concluded that the combination of massage and static stretching may lead to greater inhibitory influences on the muscle, as measured by the H-reflex, than massage alone. It is also worth pointing out that, compared to massage alone, the inclusion of static stretching increased the duration of electromechanical delay suggesting a possible stretch related increase in series elastic

compliance of the muscle. In spite of the above research outcomes our understanding of the mechanisms associated with massage remain limited, however, the evidence cited above does provide some insight into the benefits of combining massage techniques to elicit musculoskeletal improvements. In particular, it appears that the combination of clinical massage techniques such as stretching, TrP release, myofascial release and positional release; or individual effects associated with these techniques; may result in improved outcome measures compared to more traditional forms of massage such as relaxation or Swedish massage techniques.

2.4.3 Effects of massage on blood flow and tissue circulation

One of the major claims of massage techniques and the cause of many of its proposed effects is the improvements in blood flow (Mori *et al.*, 2004; Weerapong, Hume and Kolt, 2005). However a number of studies have questioned this effect with evidence to suggest no influence of massage on blood flow (Shoemaker, Tiidus and Mader, 1997; Hinds *et al.*, 2004). However, such contradictory findings may well be due to a combination of methodological limitations associated with massage research (see 2.4.1 above); a failure to reproduce and or standardise techniques (Wang *et al.*, 2014) and the appropriateness techniques used to measure blood flow changes at muscle tissue (Weerapong, Hume and Kolt, 2005; Munk *et al.*, 2012).

Despite the claimed effect of massage on blood flow, some studies have suggested that massage therapy was not found to have altered blood flow at the quadriceps muscle (Hinds *et al.*, 2004), or the upper limb (Shoemaker, Tiidus and Mader, 1997). Furthermore, Investigations into massage and blood flow have been inconclusive, mainly due to inconsistent study designs and methodological issues with regards to how blood flow is measured (Weerapong, Hume and Kolt, 2005). Studies investigating the effects of massage therapy on blood flow have used Doppler ultrasound, laser Doppler technology (Shoemaker, Tiidus and Mader, 1997; Wiltshire *et al.*, 2010), Xenon washout techniques and venous occlusion plethysmography (Weerapong,

Hume and Kolt, 2005; Munk *et al.*, 2012) Doppler ultrasound blood flow measures are reported to be determined by arterial size and blood velocity in macro regions as opposed to specific muscles (Munk *et al.*, 2012), and may not be sensitive to blood flow in smaller vasculature (Weerapong, Hume and Kolt, 2005), whereas laser Doppler techniques have been criticised for measuring blood flow only at the skin (Munk *et al.*, 2012). Xenon wash out technique has been shown to be invasive, which can interfere with treatment results (Sefton *et al.*, 2010) and possibly overestimate blood flow due to the trauma of the injection tracer (Weerapong, Hume and Kolt, 2005). Venous occlusion plethysmography may underestimate blood flow due to the inflation of the occlusion cuff and may be particularly sensitive to movement artefacts (Weerapong, Hume and Kolt, 2005). Furthermore both Xenon washout technique and venous occlusion plethysmography have methodological issues that make real time measurements of blood flow in relation to massage treatment difficult to record (Munk *et al.*, 2012).

However, in the last decade, the use of Near Infrared Spectroscopy (NIRS) technique has been used to investigate brain oxygenation and muscle oxidative metabolism (Ferrari and Mottola, 2004) and more recently the effects of massage on blood flow (Durkin *et al.*, 2006; Munk *et al.*, 2012). NIRS is a non-invasive optical technique that provides direct and dynamic measures of local oxygenation and haemodynamic changes in muscle tissue (Ferrari and Mottola, 2004). The principles of NIRS technology is based on the relative transparency of tissues for light and the oxygenation dependent absorption of Near Infrared Light (NIR) by haemoglobin and myoglobin. NIR light is able to penetrate the skin and underlying tissues in the range of 700 to 1000nm (Ferrari and Mottola, 2004). This light is either; absorbed by chromophores of variable concentrations, such as haemoglobin (Hb) located within the small blood vessels and myoglobin (Mb) located within the muscle; or scattered within the tissue (Ferrari and Mottola, 2004; Munk *et al.*, 2012). Measurement changes associated with relative absorption rates of light enable measurement changes in

tissue oxygenation to be measured continuously and noninvasively (Fadel *et al.*, 2004; Ferrari and Mottola, 2004). NIRS technique is therefore capable of providing dynamic and continuous measurements of tissue oxygenation saturation (Tsi), oxygenated haemoglobin (O_2Hb), deoxygenated haemoglobin (HHb) total haemoglobin (THb) and the difference between O_2Hb and HHb (HbDiff). A description of the determination of these blood flow variables is reported in the general methods section. NIRS technology has been used to measure blood flow at local tissue directly in the muscle. Its application allows for oxygenation and haemodynamic measurements to be measured during activity and exercise (Olivier *et al.*, 2013), and for blood flow and oxygenation utilisation of muscles between subjects with ad without LBP (Kovacs *et al.*, 2001). Therefore, the use of NIRS technique may be a promising method of measuring blood flow in massage research.

2.5 Thesis aims

2.5.1 Myofascial massage techniques and the biopsychosocial model for LBP

As outlined within section 2.1.3, no firm boundaries appear to exist between multifactorial contributors to LBP and it has been established that they all interact with each other. Although this suggests that pain is not merely a result of nociceptive input, biophysical impairments such as alterations in muscle size, composition and co-ordination are demonstrable in people with LBP (Hartvigsen *et al.*, 2018). Also, lifestyle factors such as physical inactivity and poor general health may compound biophysical factors. If these factors interact with the influence of psycho-social factors such as fear avoidance, catastrophising and maladaptive lifestyles then there is a possibility that the biophysical components can be further negatively influenced to the detriment of subjects with CLBP. However, chronic pain is phasic and may be changeable over time (Campbell and Edwards, 2009), and coping strategies can influence the perception of pain and function (Gatchel *et al.*, 2007). Therefore, addressing trunk impairments may result in decreased LBP and LBP-related disability, indeed there is

evidence to suggest such interventions may result in enhanced physical function, and reduced recurrence rates in younger adults with LBP (Sions *et al.*, 2017).

Fear avoidance and pain catastrophising behaviours suggest that pain is harmful and activity should be avoided. Such avoidance may lead to associated biophysical alterations to the paraspinal myofascial region linked to hypo-activity related changes to the connective tissue structures that surround and connect to the lumbar spine. Therefore, any pain associated with such alterations may exaggerate the negative orientations towards the pain experience. Indeed, it has been shown that interventions that reduce such pain or offset such alterations may counter such perceptions and reduce the catastrophic interpretation of pain, physiologic arousal and reduce behavioural avoidance (Gatchel *et al.*, 2007). Taken within a biopsychosocial view point, this could be seen as the interaction of one component of the model on another in the treatment of NSCLBP. Furthermore, since catastrophising and fear avoidance behaviours have a prominent role in maladaptive responses to pain (Gatchel *et al.*, 2007), treatments that reduce pain and the perception of pain may encourage patients to remain or begin to be active.

Recent systematic reviews have identified that multidisciplinary programs that confer to the biopsychosocial model of chronic pain are more effective than usual, will include more than one single treatment and should be considered in the treatment and management of NSLBP (Kamper *et al.*, 2015; Foster *et al.*, 2018). The relationship between myofascial contributors to low back pain and clinical massage techniques suggests that certain soft tissue massage techniques may have a positive impact on the treatment and management of LBP within a biopsychosocial model. In particular, the use of MT techniques could be used to reduce lumbar pain and improve paraspinal muscle function as a biophysical approach within the wider concept of a multidimensional model. Interventions that focus on the myofascial tissue have been

shown to reduce pain with some evidence suggesting that one possible mechanism behind this is an increase In local blood flow and improved fascial layer sliding (Hotfiel *et al.*, 2017). Blood circulation insufficiency can give rise to hypoxic conditions within tissues, the production and release of algesic substances and tissue fibrosis, thereby leading to pain, muscle spasm and possible joint contracture. Therefore, increasing blood circulation and promoting tissue oxygenation may be clinically important in the management of pain, reduce muscle fatigue, tissue repair and wound healing (Sions *et al.*, 2017; Zügel *et al.*, 2018). However, the mechanisms behind these effects have not been fully addressed. The aims of this thesis are to determine the Effects of clinical massage techniques on subjects with NSCLBP by investigating the mechanisms associated with changes in blood flow muscle activity and pain perception. Clinical massage techniques will specifically refer to myofascial and neuromuscular techniques and will be measured against a number of outcome measures outlined later within the study chapters. A review of the existing literature has been presented in this chapter. The methods employed across the various studies will be described in chapter 3. Chapter 4 aimed to investigate the effects of a structured clinical massage technique on the muscle adaptation pattern associated with LBP referred to as the flexion relaxation phenomena (FRP); along with pain and disability outcomes; compared to a traditional relaxation massage technique (RM). Chapter 5 and 6 attempted to investigate the effects of one particular clinical massage technique, myofascial massage techniques, on peripheral blood volume at the paraspinal region compared to relaxation massage and kinesiotaping method respectively. Chapter 7 investigated the effects of myofascial techniques on peripheral paraspinal blood volume, local paraspinal muscle fatigue and postural sway in the chronic non-specific LBP population. Chapter 8 was built on the previous chapters results and compared the effects of myofascial techniques on peripheral blood volume and local paraspinal muscle fatigue between LBP and non-LBP populations.

3 Methods

This chapter will detail the methods that have been employed during all research studies presented in this thesis. The research studies presented within this thesis were conducted within the University of Kent's School of Sport and Exercise Science laboratory and sports therapy clinic. Participants were recruited by advertisement in the form of participation information sheets sent via email to students at the University of Kent, local colleges and word of mouth. All participants were asked to complete an informed consent form and pre-test questionnaire (appendix 1) prior to any measurement or testing procedure. Participants were also screened to assess inclusion or exclusion criteria. Specific inclusion and exclusion criteria is detailed within each research study. All studies were approved by The University of Kent's School of Sport and Exercise Sciences Research and Ethics Committee.

3.1 Protocol for measuring flexion relaxation phenomenon

3.1.1 Pre-intervention procedures

The study participants were tested prior to the massage intervention during a 1-hour session at the Medway Park laboratory for the Centre for Sport Science at the University of Kent. Participants were asked to complete three self-reported outcome measure questionnaires to assess pre-intervention levels of fear avoidance, pain severity and back function and disability (see section 3.5). Participants were asked in advance to bring appropriate clothing to be able to accurately attach the instrumentation required for EMG and kinematic measurements. The objective outcome measures being tested in the laboratory were: trunk flexion angle, lumbar flexion angle, lateral flexion angle to both sides. Furthermore, EMG and both trunk and lumbar flexion angles were measured to ascertain the flexion relaxation phenomena (FRP) Following the FRP assessment each participant was asked to perform a lateral flexion ROM test to both sides. Following three practice repetitions participants were then given the cue to laterally flex to the left side first as far as possible; back to neutral standing and then laterally flex to the right side before returning to neutral standing at

the end of the test. At the end of the test each participant was assigned to the appropriate massage intervention for six treatments. Following the six treatments, participants were asked to return to the same laboratory to complete the post intervention self-reported outcome questionnaires. This was followed by a re-test of EMG kinematic and self-reported outcome measures as described above.

The protocol for measuring flexion relaxation employed in this study was similar to that used in previous studies (Martin Descarreaux *et al.*, 2008; Descarreaux, Lafond and Cantin, 2010) In order to assess FRP, Participants were required to perform 5 repetitions of trunk flexion, from standing, and back to extension in 20 seconds whilst EMG and kinematic data were simultaneously being recorded. A cycle of flexion to extension was considered to be one trial. Subjects were instructed to perform the trial with 5 seconds of controlled flexion and were instructed to attempt to touch the toes to attain the greatest flexion possible. The criteria for end range of movement were based on the subject's perception of end range and or point of discomfort. For consistency across the study each trial was separated into 5 second intervals of: quiet standing, forward flexion, maintenance of fully flexed position and return to flexion. Once the instrumentation had been set up accordingly (see below) participants were then asked to perform three practice trials of trunk flexion and extension tasks to familiarise themselves with the motion and timing requirements of the test. Participants were then asked to perform 5 repetitions of trunk flexion to extension movements, while the EMG of the para-spinal muscles and kinematic trunk angle data was simultaneously recorded. The tester used verbal cues to inform the participants of each stage of the 20 second trial

3.1.2 Identification of the flexion Relaxation phenomena

The assessment of the flexion relaxation phenomena has been achieved primarily through the measurement of the initiation and cessation of myoelectric activity of the erector spinae muscle group or through the ratio of muscular activity during the forward flexion and fully flexed positions, known as the flexion relaxation (Watson *et al.*, 1997). The following section will outline the justification for the protocols used in study 1, chapter 4, to ascertain the FRP.

To identify the angle at which FRP was achieved we estimated the point at which myoelectric activity for each muscle was deemed to be on or silent and was calculated using 3* SD above the resting mean EMG data, where the resting mean acted as a steady state resting value of the EMG signal. Preliminary testing data had identified that subjects within the back-pain group were not achieving FRP during the flexion phase therefore this method was not deemed to be suitable. Further to this, an evaluation of onset / offset points at 2 and 4 time the standard deviation of the resting mean resulted in either no discernible detection or an over estimation of onset and offset angles. Therefore, the point at which myoelectric activity for each muscle was deemed to be on or silent, was calculated using 3* SD above the resting mean EMG data. Mean resting EMG data was calculated from the data points of a three second quiet standing period moving window, prior to the beginning of the flexion movement. If EMG magnitude exceeded this figure the muscles were deemed to be active. However, if the EMG magnitude fell below this figure the muscle was deemed to be silent. FRP was therefore calculated as the angle at which myoelectric silence occurred during the flexion process. If the myoelectric activity did not fall below 3* SD above the resting mean, the muscle was deemed not to have achieved FRP. The identification of each EMG on-off point was calculated using a script in Matlab.

3.2 Kinematic data collection for FRP measurements

3.2.1 Equipment

Kinematic data was collected using the Qualisys Track Manager 2.2® (QTM). Three Oqus cameras were used to track the motion of the participants. The cameras were firmly fixed on tri-pods and set-up and positioned according to the manufacturers' recommendations. Prior to motion capture the QTM software and camera system was calibrated according to manufacturer's instructions; using a 750mm wand and L-frame that defined the 3D Cartesian origin within the laboratory. The QTM marker capture frequency was set at 250 frames per second and the capture period for 20 seconds. Trunk kinematic measurements of lumbar angle and trunk inclination angle were used in this study to identify local and global measures of flexion respectively (Ning *et al.*, 2011). The Qualisys 3D X-Y-Z global co-ordinates of the sensors were used to measure these angles. Prior to motion capture motion tracking sensors were attached to participants at the levels of S1, T12 and C7 using double side adhesive tape. Lumbar flexion angle was defined as the location of the S1sensor relative to the T12 sensor while the trunk inclination angle was defined as the location of the S1 sensor relative to the C7 sensor, and lateral flexion was defined as the location of S1 relative to C7 in that plane of motion.

3.3 Muscular activity for FRP measurements

Muscle activity patterns were recorded using sEMG. This is comprised of the recording of muscle activity through the use of sensors placed over the skin. The sEMG signal was recorded on a computer and subsequently analysed for the muscle activity level and timing. Signal differential surface EMG sensors (Delsys, Boston, MA USA) were attached over each muscle according to the recommendation of SENIAM (Hermens *et al.*, 2000). Palpation was performed to verify the location of the sensors to be placed over the muscle (Ning et.al 2011). For the lumbar multifidus the electrodes were placed on and aligned with a line from caudal tip posterior superior iliac spine to the interspace between L1 and L2 interspace at the level of L5 spinous process;

approximately 2 - 3 cm from the midline (Hermens *et al.*, 2000). For the erector spinae muscle the electrodes were placed at the L2-L3 interspace 2cm from the midline (Descarreaux, Lafond and Cantin, 2010). The skin was then shaved and cleaned with alcohol prior to placement. Once the sensors were attached tape was applied over these to help maintain their position and the sEMG cables were secured to the subject's garments to prevent movement induced artefacts.

3.3.1 Equipment

sEMG data recordings were performed with a wired EMG system consisting of 2 (study 4) or 4 study 1) EMG sensors (Bagnoli-8, Delsys, Boston, MA USA). Single differential EMG sensors with a fixed inter electrode distance of 1cm were used and connected to a portable device. The portable device was connected to the amplifier unit via a single cable. The EMG signal was amplified by a factor of 1000 and digitised by an A/D board (PCI 6034 National Instruments, Texas, USA) to allow storage of data. The EMG sensors were attached to the skin using Delsys double sided adhesive interfaces for 2-bar sensors (Delsys, Boston, MA USA). Dermatrode® reference electrodes were placed either on the process of C6 or the radial styloid process, depending on location suitability for each individual. Prior to placement the skin was shaven and cleaned with a propyl wipe. To prevent cable movement artefacts the EMG cables were secured to the portable device. Initial trials revealed that sensor adhesiveness was not sufficient to prevent sensor displacement. In these cases, zinc oxide tape was applied over the sensors to secure placement. Muscle activity was recorded for the duration of the tasks within each chapter with a sampling frequency of 1000Hz.

In order to synchronise the timing of EMG and Kinematic data collection for each trial, the electromyography (EMG) data collection system was linked to the Qualysis 3D tracking system via 4 analogue input cables. These cables were attached to the analogue board of the QTM into Chanels 1-4 and represented the EMG data collection for each muscle. However, during the testing session EMG acquisition was monitored through the Bagnoli, Delsys EMG works® 3.1 program screen, while kinematic data

was monitored via the Qualisys monitor screen. This allowed both sets of data to be monitored for the potential of movement artefact that may have compromised the data. All data was visually inspected and any trial with movement artefact was excluded from analysis. EMG Analogue data was exported to TSV file for data analysis.

3.4 Data analysis for FRP calculation

Kinematic data was exported to TSV file for data analysis before converting to excel files. Processing of the raw EMG signals from the four muscles was band passed filtered using a 4th order reverseive Butterworth filter with cut off frequencies of 20-450Hz. Secondly, a linear envelope was created using a low pass reversive Butterworth filter (3Hz cut-off). For study 1, data was then imported into a Matlab ©, (The Mathworks, Natic, MA), programme (see study 1, data analysis) for further processing to compute the smoothed EMG data and kinematic data along with corresponding EMG-off points during each trial. The Matlab programme produced an output file identifying EMG-on /off point and corresponding lumbar and trunk angles. A description of the onset and offset determination is presented in section 3.1.2.

3.5 Outcome Measure Scales for Low Back Pain.

The purpose of this section is to provide an overview of the scales that have been chosen as outcome measures within this study. These scales used will be discussed relative to their appropriateness for measuring pain and function in the low back pain population. Traditional outcome measures have focussed on physiologic outcomes but more recently a biopsychosocial model for chronic pain suggests that physiological, psychological and social factors should be integrated into this assessment (Gentile *et al.*, 2011). Back pain is a complex phenomenon often associated with pain and reduced functional status (Deyo *et al.*, 1998) as well as fear of movement or re-injury and its associated activity avoidance behaviour often described as Kinesiophobia (Swinkels-Meewisse *et al.*, 2003). As the psycho-social aspects as well as the patient's perspective of the treatment becomes more important, Bombardier(2000), has identified a core set of measures that should be included as outcome measures for low

back pain research and treatment, these include: pain, work disability, specific back disability, generic health status and patient satisfaction. The scales that have been used in this study are the Tampa Scale for Kinesiophobia (TSK) (Brockel J. Clark M.E. Kori S.H., 1996) the McGill Short Form Pain Questionnaire (SF-MPQ) (Melzack, 1987), the Oswesrty Disability Index (ODI) and the The Keel STarT back screening tool (SBST) (Fairbank & Pynsent, 2000). The TSK was chosen due to the growing evidence that pain related fear may be more disabling than pain itself in the low back pain population (Crombez *et al.*, 1999). Other scales include the Fear Avoidance Beliefs Questionnaire (FABQ) and the Pain Anxiety Symptoms Scale (PASS). However the TSK is widely used in the literature and have been validated previously (French *et al.*, 2007). Two commonly used measures of back function and disability are the ODI and Roland-Morris Disability Questionnaire (RMDQ) However the ODI has been as one of the most common methods used to assess outcome measures for spinal disorders and has been tested over time (Fairbank & Pynsent, 2000). Finally as pain is considered to be an important and recommended outcome assessment for low back pain (Bombardier, 2000) the SF-MPQ was also chosen for its widespread use in research and ease of use in the clinical setting (Burckhardt and Jones, 2003).Therefore, the use of the scales within this study will allow for the results to be compared to previous studies. The Keel STarT back screening tool (SBT) was chosen for study 4 and 5 only where a tool in which the current bothersome score of the subjects back pain can be used a s a guide to the current level of back bothersomeness.

3.5.1 Tampa Scale for Kinesiophobia

Fear-avoidance refers to the avoidance of movement or activities based on fear and has been discussed as a central theme in the development of long term back pain (Vlaeyen and Linton, 2000). Vlaeyen and Linton's (2000) review article also shows that researchers agree that fear avoidance beliefs may be important in explaining disability and the transition from acute to chronic pain. Kinesiophobia has been described as a vulnerability to painful re-injury leading to a fear of physical movement (Damsgård *et*

al., 2007) and is therefore a phobic fear of movement. Although reliable and valid tools are available to measure pain related fear, the Tampa Scale for Kinesiophobia (TSK) is commonly used to assess such phobic reactions to injury and pain. The TSK was specifically designed to measure fear of movement/re-injury in patients with low back pain (Swinkels-Meewisse *et al.*, 2003). The TSK was originally a one factor, 17-item model with 4 reverse questions designed to identify fear of re-injury due to movement of activity. The 17 statement scores range from 1(strongly disagree) to 4 (strongly agree). Items 2,4,8 and 16 are phrased in reverse key and scored in reverse so that high scores on all items indicate high levels of fear (Bunketorp *et al.*, 2005). The total sum scores range from 17 to 68 and a total score is calculated.

Several studies have found the scale to be valid and reliable psychometric measure (Bunketorp *et al.*, 2005; Damsgård *et al.*, 2007).The TSK demonstrates adequate internal consistency (Cronbach's alphas range from 0.70 to 0.81) and good test-retest reliability (r=0.78) (27-29). The instrument shows acceptable concurrent validity, with TSK scores correlating with other self-report measures of pain-related fear (r values range from 0.54 to 0.60)(Davidson et al., 2008).However, Vlaeyen et al. (1995) conducted a component analysis on the 17-item TSK and identified four non-orthogonal factors, which were labelled harm, fear of re-injury, and importance of exercise and avoidance of activity, which explained 36.2% of total variance. Furthermore, Clark et al. (1996) performed a component analysis and found two factors labelled 'activity avoidance' and 'pathological somatic focus' and concluded that all four reversible items could be excluded due to weak association to the total TSK score leaving only 13 items instead of 17 and a potentially shorter and more reliable scale of kinesiophobia. More recently, the 13-item scale has been developed with the exclusion of the reversed items. It has been proposed that the reversed items had weak association with the total TSK score; therefore omitting these items would improve the factor structure of the TSK (Cook, Brawer and Vowles, 2006).The TSK-13 is a 13-item questionnaire. Each item is provided with a 4-point Likert scale with the scoring

alternatives as discussed above with the 17-item scale. However, the total possible raw score ranges from 13 to 52. The 13-factor model includes two sub-scales of 'Pathological Somatic focus' (SF) (i.e. that pain is a sign of bodily harm or damage) and 'Activity Avoidance' (AA) (i.e. that activities that could be painful should be avoided) and represents the notion that TSK is not one-dimensional but may contain lower order factors.

In an assessment of the TSK French et al., (2007), assessed the construct validity of the TSK to be significantly correlated with other measures of fear avoidance. The same authors also found the internal consistency of the two subscales to be α0.78 and α0.7 for the AA and SF subscales respectively. Finally, the 2-factor model was shown to have a goodness of fit index of 0.93. Wong et al., (2010), demonstrated only marginally acceptable Cronbach's α (0.60) reflecting weak internal consistency. However, this study assessed the TSK-11 item scale and a 4-scale model. The same authors also reported one limitation in that different TSK scales might represent different pain population groups, therefore questioning the homogeneity of the scale. Roelofs et al., (2007) found that the 2-factor model fitted well with patients with work related upper extremity disorders. In examining the links between TSK and disability the TSK-AA was significantly related to disability relating to functioning and sports, whereas the TSK-SF was significantly related to disability related to work. With regard to chronic low back pain the same authors reported internal consistency of the TSK of α0.8 α0.74 α0.68 for the TSK-11, (a further adaptation to the original version) TSK-SF and TSK-AA respectively. In a comparison of 4 TSK models Cook et al., (2006), found the TSK-13 to have the best fit for chronic pain patients and across three adult age groups, however this fit was found to be mediocre suggesting the need for further refinement.

Correlations between the TSK and various pain and depression scores revealed significant correlations with pain, disability, depression and somatic perception (Koho *et al.*, 2001). Examining the factor structure of the TSK in a sample of low back pain

patients and the predictive value of fear of movement / re-injury as measured with the TSK Swinkels-Meewisse et al., (2003) indicated that the 2-factor model was most suitable. Furthermore, the two sub-scales were found to be significant predictors of disability. However, the same authors also point out that the items of the two factors are not always consistent across back pain studies and that there is not always agreement on the factor structure across samples. Damsgård et al., (2007) conducted a Rasch analysis of the 13-item model and concluded that it was robust across age and gender with response patterns similar for patients with low back pain and widespread pain distributions. This result was also echoed by Heuts et al. (2004) who showed that the two factor analysis provided the best fit when examining the pain related fear in Osteoarthritis patients. In a study of activity avoidance in persons with Chronic low back pain Geisser, Haig, & Theisen, (2000), conducted a factor analysis of the TSK subscales and was able to replicate the TSK -13 and showed that the TSK was able to significantly predict pain related fear.

In their review of fear avoidance and its consequences in chronic musculoskeletal pain, Vlaeyen and Linton, (2000), suggest that confrontation and avoidance are two extreme responses to fear, with the former leading to fear reductions over time and the latter possibly leading to a phobic state. Although the phobic avoidance of physical activity in times of pain may be seen as an effective coping strategy, chronic back pain and fear avoidance may contribute to de-conditioning and guarded movement or, altered co-ordination and EMG patterns within the low back pain population (Vlaeyen and Linton, 2000). Therefore the 13-item 2-factor scale has been identified as a valid and reliable tool for measuring fear of movement in both acute and chronic low back pain and highlights the importance of assessing and reducing pain related fear and disability in the hope of fostering increased participation (Geisser, Haig and Theisen, 2000; Swinkels-Meewisse *et al.*, 2003).

3.5.2 The McGill Short Form Pain Questionnaire

The Short Form MPQ (SF-MPQ) was developed to save time as the original was time consuming to administer It is described as a multidimensional measure of perceived pain in adults with chronic pain used to discriminate and evaluate the responsiveness of symptoms to treatment (Hawker *et al.*, 2011). The SF-MPQ contains three components to it: a Pain rating Index (PRI), a Present Pain Index (PPI) and a Visual Analogue Scale (VAS). The PRI contains 15 words from the original MPQ, which can be further divided into two subscales. The 15 Questions within the PRI can be separated into 11 sensory and 4 affective subscales of pain. Each word on the PRI scale is rated on 4-point intensity scale (0-3) with 0= none, 1= mild, 2= moderate and 3= severe. The PRI is scored as the sum total and the sum of sensory and affective components. The PPI is scored on a 0-5 scale and the VAS is scored on a range of 0-10cm. The VAS can be represented in mm or cm measurements with 0-4mm representing no pain, 5-44mm = mild pain, 45-74mm= moderate pain and 75-100mm= severe pain (Hawker *et al.*, 2011).

Initial convergent construct validity of the SF-MPQ demonstrated significant correlations between the SH-MPQ and other measures of pain indicating the two subscales being consistent with the original concept (Burckhardt & Jones, 2003).Overall correlations between the MPQ and SH-MPQ have been good within a range of 0.67-0.87. When used in rheumatoid arthritis (RA) and fibromyalgia patients the Cronbach's alpha was estimated at α = 0.73-0.89, with a test-re-test reliability of 0.45-0.73 for 1 month and 3 month intervals (Hawker *et al.*, 2011). In an Osteoarthritis population, high interclass correlations were also reported for the total, affective and sensory and average pain scores, over a 5-day period, of 0.96, 0.95, 0.88, and 0.89 respectively. Convergent construct validity was demonstrated by significant correlations between the SF-MPQ and other measures of pain (Burckhardt & Jones, 2003). Hawker et al., (2011), report that the content validity of the SF-MPQ was greater in patients with fibromyalgia compared to RA patients. However, for content validity the

SF-MPQ was found to have moderate correlations with the Western and Ontario and McMaster Universities Osteoarthritis Index and the short form 36 Health Survey Bodily Pain Scales with r= 0.36 and -0.36 respectively (P<0.01) (Hawker *et al.*, 2011). The same authors point out that the SF-MPQ has been sensitive to the effects of pain therapies with total scores of >5 on the 0-45 scale demonstrating a clinically important change. Hawker et al., (2011), report that the minimum detectable change for total, sensory, affective and current pain components have been estimated as 5.2, 4.5, 2.8, 1.4 and 1.4cm respectively.

The McGill Pain Questionnaire (MPQ) has been the pre-eminent measure of used in the assessment of multiple types of acute and chronic pain for over 30 years (Dworkin *et al.*, 2009).Cook et al., (2006), measured four hundred and eighty chronic pain patients, the majority of which suffered from low back pain. The authors used the scale as part of a validation of the fear avoidance model, however the population ranged from 15-82, covering a wider age range than the current study and included back pain sufferers with neuropathic symptoms. Trost, France, Sullivan, & Thomas, (2012), used the SF-MPQ to examine pain related fear on kinematic variables and concluded that subjects with back pain adopt avoidant spinal postures that could lead to maladaptive posture patterns, however, the subjects were experimentally induced back pain sufferers. Pain scales were recently used to measure the relationship between muscle activity (using EMG) and psychological factors in patients with low back pain (Lewis *et al.*, 2012). Subjects had similar exclusion criteria to the present study however the authors chose to use the pain catastrophising Scale and PASS to measure feelings of catastrophic pain and anxiety as opposed to the SF-MPQ. Furthermore, a recent and similar study, in terms of subjects and design, identified the Effects of structured massage and relaxation massage on chronic low back pain but used the pain bothersome scale as one of its outcome measures as opposed to the SF-MPQ (Cherkin *et al.*, 2009). Therefore, it does appear that various scales have been utilised in the back-pain population and may reflect individual study design needs. However,

the widely used nature of the SF-MPQ and the need to identify pain related outcomes will allow comparison to other studies.

Overall the SF-MPQ has been identified as an easy to use scale which requires no training to score or interpret (Hawker *et al.*, 2011). However, the same authors also point out that sufficient experience may be needed to complete the questionnaire and new users may require supervision. More recently the SF-MPQ has been revised to the SF-MPQ-2 to include aspects of neuropathic pain that the original version was identified as not being specific enough to detect (Dworkin *et al.*, 2009). This includes seven additional symptoms that relate to neuropathic pain and has shown to be valid and reliable enough to be used in clinical research of treatments for neuropathic conditions as well as non-neuropathic. However, it should be pointed out that the subjects in the current study are of the non-specific low back pain population and were, therefore, excluded if they had any neurological symptoms. Another criticism of the SF-MPQ is that it may not reflect the multidimensionality of pain that it is claimed to do (Hawker *et al.*, 2011). The SF-MPQ identifies sensory and effective descriptors of pain but fails to include socio-cultural aspects of pain (Gentile *et al.*, 2011). The same study also points to evidence suggesting that patients rate pain severity on the VAS scale differently when given the same stimulus and the same patients sometimes provide a different score when presented with the same level of pain at different times.

3.5.3 The Oswestry Disability Index

The assessment of disability and functional changes over time has been shown to be an important outcome measure in people with low back pain (LBP) (Deyo *et al.*, 1998). Of the many measures and scales that have emerged the Oswestry Disability Index (ODI) has been as one of the most common methods used to assess outcome measures for spinal disorders and has been tested over time (Fairbank and Pynsent, 2000b).First published in 1980 and subsequently adapted, the purpose of the ODI is to assess pain related disability in persons with low back pain. The ODI has been used to

assess this disability by questionnaire in acute, sub-acute and chronic back pain groups but was seen to reflect an American rather than British usage (Fairbank and Pynsent, 2000b). The Medical Research Council Group has since developed the ODI version 2 in the United Kingdom.

The ODI-2 has 10 sections each with 5 statements. The first statement is scored as 0 and the last 5. The ODI is scored by the sum of the statements divided by the total possible score (50) x100 to gain a percentage disability. For example, if the subject scored 14 the patient would score 14/50x100 = 28% disability. If one of the sections has been missed out, then the total scored is divided by the remaining total e.g. 45 instead of 55. The test is administered as a self-test questionnaire, which can be completed in approximately 5 minutes and scored in less than a minute. Each category in the ODI is ordinal in nature but the data gathered is in categorical format. Summing the scores gives the data a quantitative dimension to it. Fairbanks advises against viewing this as a linear correlation with disability e.g. that a 40% score means twice as much disability as a 20% score. Rather, it is suggested to aggregate the index into several categories (Fairbank and Pynsent, 2000b). The disability categories are described below:

0% to 20%: minimal disability: The patient can cope with most living activities. Usually no treatment is indicated apart from advice on lifting sitting and exercise.

21%-40%: moderate disability: The patient experiences more pain and difficulty with sitting lifting and standing. Travel and social life are more difficult and they may be disabled from work. Personal care sexual activity and sleeping are not grossly affected and the patient can usually be managed by conservative means.

41%-60%: severe disability: Pain remains the main problem in this group but activities of daily living are affected. These patients require a detailed investigation.

61%-80%: crippled: Back pain impinges on all aspects of the patient's life. Positive intervention is required.

81%-100%: These patients are either bed-bound or exaggerating their symptoms.

In terms of its reliability a test of internal consistency has produced a Cronbach's alpha score of between 0.71to 0.87 (Fairbank and Pynsent, 2000b). The test-retest correlation is also good but the same authors point out that the retest interval is a factor that needs to be considered. The test-retest score ranged from r-0.99 (n=22) when retested at 24-hour interval, to 0.91 (n=22) and 0.83 (n=22) if the re-test is extended to 4 days and a week respectively. It is possible that symptom fluctuation and memory may contribute to these findings (Fairbank and Pynsent, 2000b). Construct cross-sectional tests of validity showed convergent correlation results of 0.8 with the Quebeck Back pain scale, 0.82 with the Roland Morris Questionnaire (RMQ), and 0.62 with the Jan Van Breeman pain and function questionnaire (JVB) (Fitch *et al.*, 2002).With regard to sensitivity to change, convergent correlations with global rating of change range from 0.31 to 0.57. Correlations with the RMQ change score was 0.79 and 0.61 and 0.64 with JVB pain and function respectively (Fitch *et al.*, 2002).

The ODI has been used extensively to measure self-reported outcomes in the LBP population. In a sample of subjects with mechanical low back pain, The ODI showed greater ability to measure change over time than a Jan Van Breeman Institute function questionnaire. Subjects were measured at 4 and 6 weeks after the initial questionnaire was completed. However, one surprising feature of this study was the number of blank and multiple responses associated with ODI. Blank responses may be due to questions being not applicable; which may apply to the sex life question; and phrases being perceived as confusing. It was also noted that multiple responses might be due to the options being perceived as not being mutually exclusive and not representing a hierarchy (Stratford *et al.*, 1994).In an examination of 5 questionnaires that assess disability in people with low back pain, interclass correlation coefficients to measure reliability found the ODI to be over 0.8. Measurements obtained with the modified ODI SF36 physical functioning scale and Quebec back pain disability scale were the most reliable and was sufficient to detect improvement or worsening in most subjects (Davidson and Keating, 2002).Furthermore, in an examination of the measurement

properties of the ODI and Quebec Back Pain Disability Scale, the ODI was preferable with higher test re-test reliability over 4 weeks with an ICC of 0.9 and 0.55 respectively. Responsiveness was also better for the ODI, interpreted as the probability of correctly identifying the improved patient from randomly selected pairs of improved and unimproved patients, and ranges between 0.5 (no diagnostic accuracy beyond chance) to 1.0 (perfect diagnostic accuracy); responsiveness was found to be 0.94 0.87 for the ODI and QUE respectively.

Fairbank & Pynsent, (2000) suggest that the ODI remains a valid and vigorous measure for spinal disorders. The authors believe that the management of disability is an important component of the managements of low back pain patients and the ODI is a better measure of this than some objective measures. The authors also recommend the use of version 2 used in the current study. However, there does seem to be an issue with the wording of the questions leading to subject misinterpretation or leaving the questions blank (Stratford *et al.*, 1994), suggesting the need to for subject specific explanations and clarification of terminology. Furthermore, this would also mean that the use of this questionnaire through means other than a one to one setting may not be appropriate. Examples of this include clinical trials that require outcome measures that are identified over the phone or through other similar means. For this purpose, it is proposed that the RMDQ may be a more appropriate measure which has recently been used in two similar massage trials.(Cherkin *et al.*, 2009).

3.5.4 Keel STarT Back Screening Tool

The Keel STarT back screening tool (SBST) is a validated screening tool that allows for a stratified care approach that matches patients to treatment based on prognosis or risk of poor clinical outcome (Main *et al.*, 2012a). It has been described a simple prognostic questionnaire that helps clinicians identify modifiable; biomedical, psychological and social risk factors; for back pain disability. There is currently a 9-item tool with 9 questions relating to predictors for persistent back pain. All 9-items use a

response format of ‘agree' or ‘disagree', with exception to the bothersomeness item, which uses a Likert scale. The tool produces an overall score and a distress (psychological) subscale. The overall score and subscales are then used to stratify patients into low, medium and high subgroups. Scores of 3 or below are considered low risk. Scores of 4 or more are then subject to the subscale questions 5-9. A score of 3 or less from these questions is regarded as a medium risk, while 4 or more is considered to be high risk. Beyond this tool there is a 9-item clinical measuring tool that is designed to objectively measure the severity, of the domains screened by the 9-item tool described above, and used as a repeated measure tool over time. Cut of scores have been provided to indicate subgrouping as above. The clinical measurement tool has scores for; leg pain, shoulder and neck pain, dressing, walking, fear, worry, catastrophizing, mood and a current bothersome score.

The validity of the SBST tool as a subgrouping tool has previously been established (Hay *et al.*, 2008). In comparison to the Orebro Musculoskeletal Pain Screening Questionnaire (OMPSQ) showed a high correlation (rs = 0.8). The results indicated that the SBT tended to stratify fewer patients into the high risk group, and while the OMPSQ was better at discriminating pain intensity the SBT was better for discriminating the bothersomness and referred leg (Hill *et al.*, 2010). The questionnaire’s shortness and ease of use has made it effective in translation to other languages, however some patients with non-specific LBP found it more difficult to understand (Gusi *et al.*, 2011). In a comparison of the tool’s predictive ability with subjective clinical decision making, there was observed agreement with the BST in 47% of cases, indicating a fair agreement (k = 0.22) (Hill *et al.*, 2010). More recently, test-retest reliability of the BST total score provided an interclass coefficient of 0.9, and a good Chronbach alpha of 0.73 for internal consistency for the psychological subscales (bruyere at.al 2014). Furthermore in comparison with the Roland Morris Disability Questionnaire and the OMPSQ scale, the BST correlated highly with correlation coefficients of 0.74 for both (Bruyère *et al.*, 2014).

3.6 Blood volume and tissue oxygenation

Blood volume and tissue oxygenation measurements were recorded using NIRS technique. NIRS is a non-invasive optical technique that measures the concentration changes in oxyhaemoglobin, deoxyhaemoglobin and total haemoglobin. This is achieved through the means of spectrophotometers placed on the participant's skin. NIRS relies mainly on the relative transparency of tissues for light in the NIR range and the oxygenation dependent absorbance of haemoglobin. NIR light is transported to the tissues by means of an optical fibre referred to as an optode. The NIR light is received by a detector placed parallel to the optode and placed over the tissue of interest. Light detected from the optode is said to describe a banana shape travelling through the tissue and represents the mean path of multiple detected photons (Munk *et al.*, 2012). The maximum penetration depth is said to be approximately half the distance between the optode and the detector (interoptode distance). As a result adipose tissue confounds the measurement as greater adipose tissue thickness reduces penetration depth into muscle tissue (van Beekvelt *et al.*, 2001). Therefore, Adipose tissue measurements and or BMI will be evaluated at the site of NIRS recordings throughout this thesis where NIRS is utilised. The optical signals from NIRS acquisition are converted to haemoglobin changes using the modified Beer Lambert Law (Ferrari and Quaresima, 2012), which enables calculation of the optical the density in non-homogenous biological tissues. The optical density changes are converted into oxygenated (O_2Hb) haemoglobin and deoxygenated (HHb) haemoglobin. The measurement of changes in tissue oxygenation can, therefore, be followed noninvasively.

The sum of O_2Hb and HHb is a measure of total blood volume (tHb). NIRS is unable to distinguish between haemoglobin and myoglobin because the absorbency signals overlap in the NIR range (Ferrari and Mottola, 2004). For this reason, both haemoglobin and myoglobin are referred to as haemoglobin within this thesis.

Oxygenated haemoglobin (O_2Hb) was determined by the relative concentration changes in oxygenated haemoglobin and myoglobin. De-oxygenated haemoglobin (HHb) was determined by the relative concentration changes in deoxygenated haemoglobin and myoglobin. Total haemoglobin (tHb) was determined by the sum of O_2Hb and HHb and was used to provide the total change in blood volume. Tissue oxygen saturation (TSi); an absolute measure of oxygen saturation within the tissues; was also measured at the lumbar paraspinal region. TSi is the estimation of percentage tissue oxygen saturation and is the concentration of O2Hb in relation to the total amount of haemoglobin (O_2Hb/ (HHb+O_2Hb). Haemoglobin difference (HbDiff) is the difference between the relative concentrations of O_2Hb and HHb. The data obtained from this process represents relative changes in concentrations chromophores and at a depth of approximately 15mm deep that represents the capillary level. Within this thesis the sums of O_2Hb and HHb will be used as a measure of total blood volume (tHb), and, the concentration of O2Hb in relation to the total amount of haemoglobin (O_2Hb/ (HHb+O_2Hb) will be referred to as changes in tissue oxygen saturation (TSi). Relative changes in the concentration's haemoglobin will also be measured. Any reference to blood 'flow' will only be presented where previous studies have used such terminology to describe blood and or circulation changes within those studies.

3.7 Protocol for measuring blood volume and tissue oxygenation at the lumbar paraspinal region

In order to assess blood volume and oxygenation measurements Near Infrared Spectroscopy (NIRS) technique was used. The NIRS data was collected Bi-laterally at the lumbar para-spinal region. Sampling locations for the spectrophotometers were bi-laterally at the level of L3, 3 cm from the spinous process (Ning *et al.*, 2011). All testing was conducted in the same room in which the intervention was applied, minimising the effect of movement on the test results. Before and after each testing session, the NIRS instrumentation was calibrated according to manufacturer's specifications. Prior to any

testing subjects were placed on a couch in a prone position where NIRS data collection reference points were identified. Subjects were prepared for the measurements (see below) at which point NIRS spectrophotometers were then applied to the skin. Samples were recorded for two minutes before and after each treatment at 20 samples / second. Spectrophotometers were removed prior to any further measurements or intervention applied to the lumbar paraspinal region. Following any re-measurement, intervention or treatment the sensor locations were re-cleaned with a sterile wipe and the spectrophotometers were returned to the same pre-treatment sensor locations, at which point NIRS blood volume measurements were recorded for a further 2 minutes. NIRS data was extrapolated for further analysis (see below).

3.7.1 NIRS Equipment

The NIRS data was collected Bi-laterally at the lumbar para-spinal region using the Oxysoft Mk III Near Infrared Spectroscopy System, (Artinis Medical Systems ®Arnhem). The Oxysoft Mk III is a continuous wave NIRS system with two wavelengths of emitting light. The cabled NIRS system consisted of two spectrophotometers attached to the multi-channel Mk III system. The spectrophotometers consisted of three light transmitters and one receiver in parallel to each other. The distance between the transmitter and receiver was set at 25 mm. Therefore, as the depth in which NIRS measure is approximately half the distance between the transmitter and receiver the depth of measurement for the purpose of this thesis will be approximately 12.5 mm. The area was prepared by removing any hair and cleaned with a sterile wipe. Palpation was performed to verify proper location of for each muscle to be measured. The skin was shaved and cleaned with alcohol wipes prior to placement of spectrophotometers onto the skin. The spectrophotometers were then applied to the skin using double sided tape at the receiver end. Further adhesive tape was then applied over both ends of the spectrophotometers to ensure maximum connection to the skin. Cables were secured to the participants and supported to prevent any movement induced artefacts. A black cloth was placed loosely over the area, completely covering the NIRS sensors

to block any ambient light from reaching the sensors. The NIRS was calibrated and set to record tissue saturation index (Tsi), O2Hb, HHb, tHb and Hb Diff. Figure 3.1. below depicts the NIRS set apparatus positioning within this thesis.

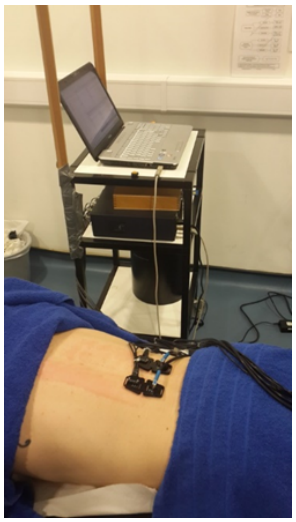

Figure 3. 1 NIRS spectrophotometer placement at the paraspinal region

3.7.2 NIRS Data analysis

The NIRS protocol recorded Tsi, O2Hb, HHb and tHb. NIRS raw data for the 2-minute period before and after interventions were exported to excel for further analysis. Tsi, O2Hb, HHb and tHb measurements were averaged over the 2-minute period. Left and right-side data was then averaged prior to further statistical analysis. A description of the statistical analysis is presented in each of the relevant chapters.

3.8 Massage interventions

The following sections outline the massage interventions used within this study. For the purpose of this thesis the massage techniques used within these studies have been described previously by Sherman et.al. (2006) as; relaxation massage and clinical massage techniques and have been described in section 1.5. However, in chapters 5-8 the massage intervention utilised an integration of techniques used to manipulate the connective tissue or fascial structures of the paraspinal region. These were; myofascial release (Barnes, 1997), fascial release techniques (Myers, 2009b; Earls

and Myers, 2010), connective tissue skin rolling (Ali *et al.*, 2012), soft tissue release (Sanderson, 1998; Lowe, 2000) and collectively referred to in this thesis as integrated myofascial techniques (MT).

3.8.1 Relaxation massage techniques (RM)

Due to the nature of the intention only certain techniques were allowed to be used to facilitate general relaxation and comply with the industry recognised techniques that pertain to a Swedish body massage routine. While variations do exist between therapists these techniques were as follows:

- Effleurage (gliding, superficial and deep)
- Petrissage (kneading, wringing, rolling)
- Petrissage Frictions (circular only)
- Tapotement (hacking, cupping, beating)

Specific timings and treatment descriptions can be seen in the methods section of study 4 and 5.

3.8.2 Clinical massage techniques (CM)

Clinical massage techniques used within this thesis were as follows:

Myofacsial techniques.

- Myofascial release techniques (MFR) including – cross hands MFR, skin rolling, pin and stretch, longitudinal fascial planes release, active and passive techniques
- Soft Tissue Release including – active, passive and resistive techniques.
- Longitudinal and transverse muscle stripping including – deep muscle stripping, J-stroking, cross fibre and longitudinal friction massage.

Neuromuscular techniques.

- Trigger point compression therapy
- Muscle Energy Techniques (MET) including:

    - post isometric relaxation
    - reciprocal inhibition
- Positional release techniques
    - Strain counter strain

Specific application of these techniques are described in section 4.7.2

3.8.3 Integrated myofascial techniques (MT)

The MT intervention was designed to be standardised across all subjects so that treatments did not fluctuate and were repeatable within a standardised time frame. Therefore, the therapist was instructed to conduct the treatment within a time frame of 30 minutes to be in line with practice time slots. Prior to the study beginning the therapist was taken through a familiarisation process for conducting the treatments. As the therapist was familiar with the techniques the familiarisation process was designed to ensure that the treatments were kept within the treatment time frame. The MT techniques utilised in chapters 5-8 are detailed in table 3.1 below.

Table 3. 1 Integrated myofascial massage techniques (MT) treatment descriptions.

Client position	*Technique Description*	*Repetitions/ duration*
Prone position:	Compressions and vibrations applied through a towel. Myofascial release (MFR) cross hands technique, applied bi-laterally, to the spine at the lumbar region.	90 seconds second each side.
	Fascial release, longitudinal myofascial stripping applied bi-laterally with the knuckles, to the erectus spinae from approximately C7 to S1/2.	Ten repetitions.
	Connective tissue manipulation, skin rolling over the thoraco-lumbar fascia region from the sacrum, in a cephalad direction, through to T12.	Six repetitions each side
	Fascial release, transverse myofascial stripping applied bi-laterally, with the fingers from a medial to lateral direction (from L5 to the mid thoracic region). Transverse myofascial stripping across the gluteal muscles in a medial to lateral direction from approximately L5 to S2.	Three repetitions each side. Three repetitions each side.
Side lying	MFR cross hands to Quadratus Lumborum (QL) region between the iliac crest and T12.	90 seconds each side.
	Fascial release, myofascial stripping and active lengthening to the latissimus dorsi and QL, in a caudal direction, from T7 to the iliac crest using knuckles and or forearms.	Six repetitions each side
	Myofascial stripping and active lengthening to the lumbar erector spinae, in a caudad direction, from T12 to S1 using knuckles and or forearms.	Four repetition each side.
	Soft Tissue Release to the QL muscle.	Three repetitions each side
Seated	Fascial release, myofascial stripping and active lengthening from C7 to S1/2 region, using knuckles and or elbows with active forward flexion performed by the participant.	8 to 10 repetitions each side.

3.9 Biering-Sorensen test (time to fatigue measurements)

The Biering-Sorensen test is designed to test the resistance of the lumbar extensor muscles to fatigue (Biering-Sørensen, 1984). The test is considered a specific tool for evaluating back muscle endurance and is by far the most widely used test for assessing trunk extensor muscle groups (Demoulin *et al.*, 2006). Although the test has been described in detail previously (Demoulin, 2012), a brief description will follow. The test requires participants to produce and sustain an isometric contraction of the paraspinal extensors. This involves participants having to maintain a horizontal position with the trunk and upper body unsupported for as long as possible. As the muscle fatigues and muscle contraction is no longer sufficient the participant drops the trunk below the horizontal line, at which pint the test is stopped.

Prior to the fatigue task the test protocol was fully explained to each participant and participants were informed that if the test was too uncomfortable or painful that the test would be terminated. Participants were placed on a hydraulic couch in a prone position with the upper edge of the iliac crest aligned with the edge of a plinth. A chair was then placed in front of the plinth so the clients were able to support themselves in the correct position before initiation of the test. The plinth was placed at a height above the chair that allowed for a 10 degree drop from the horizontal position while the participant was using it for support. The client lay with their arms in lateral rotation at 90 degrees of shoulder abduction, elbows flexed and forearms in a prone position. This was assumed to be the resting starting position. The participant was then secured in place using three straps that were placed around the participant and secured to the plinth. One placed at the level of the greater trochanter, the second one just proximal to the popliteal fossa and the third strap at the level of the Achilles tendon. In order to negate irritability of the straps on the client's limbs a towel was placed between the strap and the client. Each strap was then tightened so that the client felt secure. On initiation of the test the subjects were requested to remove themselves from the support of the

chair, take themselves into a horizontal position and place their arms across their chest while maintaining the unsupported position. This was referred to as the neutral test position. One tester visually observed the participants position for changes in the horizontal position and timed the task using a stop watch. Another tester provided continuous motivation to maintain a neutral spine to ensure the test was a true measure of resistance to fatigue. The test ended when a participant touched the chair; when the participant flexed, or extended their torso away from the neutral position and was unable to return to that position or if they repeatedly dropped into flexion 3 times or more. At this point the stop watch was stopped and the time to fatigue recorded.

3.10 Muscular activity for fatigue task evaluation

EMG signals were recorded during the entire sustained Biering-Sorensen fatigue task using a Bagnoli, Delsys EMGworks ® 3.1(Bagnoli-8, Delsys, Boston, MA USA). Two bipolar surface sensors were used in total to measure the left and right paraspinal muscles. These were placed bilaterally over the lumbar illiocostalis and longissimus muscle groups as described by the recommendation of SENIAM (Hermens, Freriks 2000). The sampling locations were 2-4 cm lateral to the L2 spinous process. The exact placement was determined following client extension of the spine and palpation of the paraspinal muscle bulk. Once the sensors were placed at the appropriate locations each client was instructed to lie still in a prone position for 10 seconds to determine a baseline, resting EMG trace. Subjects were then instructed to carry out the first Biering-Sorensen fatigue task (task1). Following the intervention (MT or sham tens) the EMG sensors locations were re-prepared and the sensors re-attached. Precise replacement of the sensors was achieved through the use of a permanent marker to identify sensor location sites. EMG signal recordings were exported to excel files for further analysis. Raw EMG data was visually checked to identify start and end time frames. EMG data was then smoothed across the recorded time frame using root mean squared method.

EMG signal recordings were exported to excel files for further analysis. Raw EMG data was visually checked to identify start and end time frames. EMG data was then smoothed across the recorded time frame using root mean squared method. Data for the left and right side paraspinal muscles were side averaged. Each EMG recording at fatigue task 1 and 2 was divided into 2 second segments of raw EMG data. To eliminate the start transition the first 2 seconds were excluded (Tanaka *et al.*, 2002). The root mean (RMS) squared amplitude was calculated in each 2 second segment. RMS of the first 5 and last 5 segments of the fatigue task were used for later analysis. The first and last 10 second time segments were used to differentiate the start and end RMS amplitudes.

3.11 Sham transcutaneous electrical nerve stimulation

The TENS treatment was conducted by a therapist trained in the use of electrotherapy. The therapist had 2 years' experience of using electrotherapy in practice. Participants were required to lay prone on a treatment plinth. The area for treatment was cleaned and shaved (if required). Two sterile electrodes were placed bi-laterally at the level of L3, three centimetres from the spinous process (figure 3.2 below). The Intellect ® Mobile Combo Electrotherapy and ultrasound machine (Chattanooga Medical Inc, US) was programmed for a 30-minute treatment. Following electrode placement, the participant lay prone while no electrotherapy was performed. In order to blind participants to the SHAM condition, the machine was placed out of sight so that

participants could not see the display (Conn *et al.*, 1986). The treatment lasted for 30 minutes before the electrodes were removed for retesting.

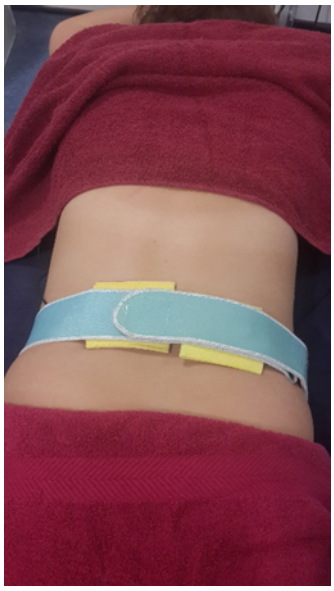

Figure 3. 2 Sham TENS electrode placement at the paraspinal region

3.12 Sham ultrasound measurements

The therapist had 2 years' experience of using electrotherapy in practice. Participants were required to lay prone on a treatment plinth. The area for treatment was cleaned and shaved (if required). The Intellect ® Mobile Combo Electrotherapy and ultrasound machine (Chattanooga Medical Inc, US) was programmed for a 15-minute treatment to reflect typical ultrasound treatments to the musculature of the lower back at the lumbar paraspinal region, to coincide with the area that the blood flow measurements were recorded. The Ultrasound time was calculated based on the treatment principle of 1 minute per treatment head area, multiplied by the pulse factor. As the intervention was a pilot study for the treatment of chronic low back pain the sham ultrasound was set for 10 minutes, 5 minutes for each of the paraspinal muscle groups in the L1-5 region. Subjects were placed in a prone position. Ultrasound gel was applied to the area while the therapist applied the ultrasound head in a continuous circular motion over the paraspinal muscles. In order to blind participants to the SHAM condition, the machine

was placed out of sight so that participants could not see the display (Jaffer, Daly, Yadav, Marshall & Graeme, 1986).

3.13 Pain pressure threshold measurements (PPT)

Perceptions of pain pressure were taken before and after the treatment on both the left and right sides three centimetres from the L3 vertebra, using a Baseline ® Algometer, (Wagner Instruments, Greenwich, US). Pain pressure threshold (PPT) within this thesis can be defined as the minimum pressure or force required to cause pain. Subjects were positioned in a prone position with the lumbar area exposed for testing. The subjects were informed of the procedure prior to the testing and they were informed that they were to provide a verbal indication of the onset of pain by saying the word 'yes'. The specific area for measurement was identified by measuring 3cm latera from the L3 spinous process and corresponded with the area of paraspinal myofascial tissue NIRS placements (see 3.7 above), and marked with a pen. The PPT applicator was placed over the mark perpendicular to the skin. Force was applied by the researcher perpendicular to the skin gradually increasing the pressure at 1kg per second until the patient said 'yes'. At that point the ergometer was removed and the measurement was recorded. Following the measurement, the area was cleaned with a sterile wipe.

3.14 Skinfold measurements

In order to ascertain the possible confounding effects of adiposity on the NIRS measurements skinfold measurements were taken on the right side three centimetres from the L3 vertebra using Harpenden Callipers ®, (Batty International, Sussex, and UK). The subjects were placed in a standing position with the area exposed to the tester. The procedure was explained to the subject. The area for measurements was determined by measuring 3cm lateral to the L3 spinous process and marked with a pen. The tester then pinched a double layer of skin and the underlying adipose tissue 1cm above the marked site, but not the muscle tissue, producing a vertical skinfold. The callipers were then applied perpendicular to the pinched skin and lest to settle for 1 second. The measurement was then recorded. This process was repeated twice more and the mean

reading recorded for analysis. The area was cleaned with a sterile wipe following the test.

3.15 Postural balance

Postural balance or sway was assessed by an RSscan® (RSscan Ipswich) balance system 7.22 at 32Hz. All subjects performed the balance tasks bare foot to eliminate the effect of shoe type. Subjects were asked to sand on a balance pad placed on top of the RSscan® balance pressure mat. This was a foam balance pad of medium density ($45cm^2$ x 13cm thick, density = 60kg thick, load deflection 80-90). Subjects were asked to stand in a double leg support position with feet parallel and hands on hip. At the first session Participants were asked to one perform one 10 second practice trial followed by two baseline trials of 30 seconds with eyes open and eyes closed respectively. Balance was defined as the displacement of entre of pressure (COP). The centre of pressure area was recorded which represented the maximum anterior, posterior, medial and lateral sway (in mm^2) during the trial. The balance test was then repeated with eyes closed immediately after fatigue task 1 (F1) and fatigue task 2 (F2).

3.16 Discussion of methods used and their limitations

3.16.1 Study 4

FRP calculation

FRP was used to determine the myoelectric activity of the paraspinal muscles during a forward flexion motion in subjects with CLBP. This outcome measure and method was chosen as previous studies comparing massage techniques had not used an objective measure of muscle activity specific to pathogenesis of LBP. Activities using full flexion are a common in activities of daily living, occupation and sport therefore knowledge of the trunk biomechanics and the transfer of load to paraspinal muscles in flexion can provide some indication of the pathogenesis of LBP, furthermore, there is evidence to suggest that EMG differences exist between patients with back pain and healthy

subjects during dynamic flexion tasks performed at peak flexion (Colloca and Hinrichs, 2005).

Use of the Qualisys Track Manager 2.2® (QTM), provided an objective measure of trunk flexion ROM and the ability to identify segmental changes in ROM required for accurate FRP calculation. In study 4 trunk flexion and lumbar flexion determined to provide an overall measure of trunk flexion and lumbar flexion required to determine the angles at which FRP was achieved. Furthermore, the Delsys EMG works® 3.1 system was able to be synchronised to the QTM system, this allowed both sets of data to be monitored for the potential of movement artefact that may have compromised the data.

Although the synchronisation of EMG and kinematic motion was achievable there are some limitations to the methods surrounding the extraction of data to ascertain FRP. Identification of the onset and offset of the paraspinal muscle was calculated through the method of using 3* SD above the resting mean EMG data, where the resting mean acted as a steady state resting value of the EMG signal. However there appears to be no common agreement to define EMG on and off FRP angles (Jin, Ning and Mirka, 2012). Onset and offset methods have previously been determined as a percentage MVC, however MVC was not measured in this study. Other studies have utilised similar steady state reference points and developed cut off points based on a number of standard deviations above a mean time period during the fully flexed position (Ning *et al.*, 2011). However, these have been shown to produce artificially high cut off points if full flexion relaxation is not achieved (Jin, Ning and Mirka, 2012). Further limitations to the use of FRP include the suggestion that FRP calculations are difficult to compare between subjects and further studies may utilise the use of a flexion relaxation ratio (FRR). FFR is calculated as the maximum EMG during forward flexion divided by the minimum resting (fully flexed) EMG. This method has been suggested to provide a normalising EMG factor to the EMG data, making it possible to compare factors across

time and individuals (Owens, Gudavalli and Wilder, 2011). Furthermore, FRR may be a more sensitive evaluation of back muscle activity where subjects exhibit lower disability scores (Watson, Booker, Main and Chen, 1997),

Within this study it should also be identified that pre and post placements of track manager and EMG sensors may have differed between visits leading to confounding results. Subject activities and lifestyle patterns were not standardised and may have influenced the subject's response to forward flexion and the associated myoelectrical activity. Finally, movement artefact cannot be ruled out within this study. While every attempt was made to reduce the movement of QTM and EMG sensors the flexion motion may have led to sliding of soft tissue layers at the paraspinal region affecting the reliability of results within this study.

Outcome Measure Scales for Low Back Pain

Three self-reported questionnaires were administered prior to and following the massage interventions, these were the 13-item Tampa Scale for Kinesiophobia, the McGill SF-MPQ pain scale questionnaire and the Oswestry Disability Index. A detailed description of these self-reported outcome measures and their limitations can be found in section 3.5.

3.16.2 Study's 5-8

Blood volume measurements

Paraspinal blood flow/volume was measured using near infrared spectroscopy (NIRS) at the L3 vertebral level before and after a 30 minute treatment. NIRS light penetrates superficial tissues and is absorbed by chromophores (Haemoglobin and myoglobin). It can measure changes in haemoglobin volume (total Blood volume) and tissue saturation (Tsi). Tsi represents a balance between o2 supply and o2 consumption in the capillary vessels. The use NIRS allows for the measurement of blood volume variables to be measured non-invasively and continuously at the capillary level. Furthermore it has been identified that other methods of measuring blood flow / volume

have been limited in their ability to provide reliable results. Investigations into massage and blood flow have been inconclusive, mainly due to inconsistent study designs and methodological issues with regards to how blood flow is measured (Weerapong, Hume and Kolt, 2005). Studies investigating the effects of massage therapy on blood flow have used Doppler ultrasound, laser Doppler technology (Shoemaker, Tiidus and Mader, 1997; Wiltshire *et al.*, 2010), Xenon washout techniques and venous occlusion plethysmography (Weerapong, Hume and Kolt, 2005; Munk *et al.*, 2012) Doppler ultrasound blood flow measures are reported to be determined by arterial size and blood velocity in macro regions as opposed to specific muscles (Munk *et al.*, 2012), and may not be sensitive to blood flow in smaller vasculature (Weerapong, Hume and Kolt, 2005), whereas laser Doppler techniques have been criticised for measuring blood flow only at the skin (Munk *et al.*, 2012). Xenon wash out technique has been shown to be invasive, which can interfere with treatment results (Sefton *et al.*, 2010) and possibly overestimate blood flow due to the trauma of the injection tracer (Weerapong, Hume and Kolt, 2005). Venous occlusion plethysmography may underestimate blood flow due to the inflation of the occlusion cuff and may be particularly sensitive to movement artefacts (Weerapong, Hume and Kolt, 2005). Furthermore both Xenon washout technique and venous occlusion plethysmography have methodological issues that make real time measurements of blood flow in relation to massage treatment difficult to record (Munk *et al.*, 2012). Therefore NIRS presents a reliable as well as a convenient way to measure musculoskeletal blood flow, especially in manual therapy setting (Sampath et al 2017).

Limitations of the use of NIRS within this study exist and are identified below. Within the current thesis NIRS sensors were disconnected during treatment, which raises three important questions. Firstly, a key advantage of using NIRS is its ability to measure blood flow continuously (Ferrari et al 2004). However, the instrument is also extremely sensitive to minimal movements and muscle contractions (Ferrari et al 2004). NIRS sensors were reconnected to the exact location as they were before

intervention. However, the use of a marking method that was not removed by the massage intervention could be used to achieve this reliably every time with all the participants. Further, the main findings were blood volume changes immediately (2 minutes) after intervention. However, their methodology demands reconnecting the sensors (bilaterally) after intervention suggesting a time delay (the average minutes the procedure took is not reported), which invariably means that the results may not reflect immediate changes following the intervention. More generally, NIRS measurements are also subject to the confounding influence of adiposity; which was measured and factored in from study 6 onwards. NIRS technique cannot be used to determine the flow of blood through the capillaries only the relative change in haemoglobin concentration at the site of measurement. Baseline measures of haemoglobin are arbitrary figures which make comparisons between individuals difficult to evaluate. Due to the nature of the position in which the blood volume measurements were recorded, venous occlusion arterial occlusion method, could not be used to calculate oxygen consumption and re-oxygenation using these methods.

3.16.3 Study's 6-8

PPT measurements

Musculoskeletal symptoms such as CLBP have a variety of causes and vary widely among individuals and is characterised by pain at particular sites, in the case of no-specific CLBP is defined as being between the areas of the gluteal folds and T12. Within this region it is important to measure muscle pain and tenderness of the paraspinal muscles as a potential contributing factor of LBP (Sung, Lammers and Danial, 2009). Furthermore, we were interested in the association between paraspinal blood flow and changes in the perception of pain following structured MT massage interventions either directly in response to a treatment or due to the effects of MT treatment n pain perception following a fatigue task. Pressure algometers are designed to measure deep pressure pain thresholds or tenderness resistance and are advantageous for quantifying the pressure pain thresholds of muscles. Also, the

reliability of PPT measurements are reported to be high with intra-class coefficients ranging from 0.9 to 0.95, implying very high reliability (Park *et al.*, 2011). Furthermore, PPT is reported to be responsive to a physical therapeutic intervention such as that used within this thesis (Mutlu and Ozdincler, 2015). However, a number of limitations exist with the use of PPT within this thesis should be identified. It has been reported that there are differences in the perception of pain between males and females with females exhibiting lower thresholds than males. (Park *et al.*, 2011). PPT tends to decrease with age and age may also lead to greater sensitivity to pain independent of the disease process (Mutlu and Ozdincler, 2015). Within the current thesis PPT was consistently measured at the lumbar paraspinal region, however, the reliability of PPT measurements may alter depending on the muscle group measured, for example it has been suggested that the reliability of measuring the paraspinal muscle group may be less that other muscle groups as the lumbar area is subject to greater change in PPT (Mutlu and Ozdincler, 2015). Finally, although the protocol of applying pressure at a rate of 1kg/s was utilised this was not monitored and the studies would have benefitted form an inter-tester reliability study.

3.16.4 Study 7 and 8

Biering-Sorensen test (time to fatigue measurements)

The Biering-Sorensen test was used to assess the back muscle endurance of the lumbar paraspinal muscles within this study and is designed to test the resistance of the lumbar extensor muscles to fatigue (Biering-Sørensen, 1984). Within this thesis the test was chosen to assess back muscle fatigue in relation to the effects of MT on blood flow and any potential influence this would have on the fatigability of the paraspinal region. LBP subjects have been identified as having poor muscular endurance, increased fatigability and reduced oxygenation to the paraspinal muscle groups (Kramer *et al.*, 2005; Kell and Bhambhani, 2006).

The test is considered a specific tool for evaluating back muscle endurance and is by far the most widely used test for assessing trunk extensor muscle groups (Demoulin *et al.*, 2006). The test has been validated with an interrater and interrater reliability coefficient of >0.95 (Simmonds et.al 1998) and good reproducibility co-efficient of 0.82 and 0.68 (Simmonds *et al.*, 1998; Demoulin *et al.*, 2006) respectively. Although a number of patients have experience back pain during the test, no persistent adverse effects have been reported for this test.

Within the studies that employed the use of this test, specific protocols used to determine the pint at which time to fatigue occurred. However, one limitation of this test is thought to be the reason for termination of the test. Some subjects report discomfort in the hip muscles or upper back as contributing factors as opposed to pain or discomfort in the lumbar region. The lack of specificity associated with testing the lumbar paraspinal muscles only is a limitation of this method within these studies. Furthermore, subjects with current or previous nonspecific LBP may be more likely to end testing due to fatigue, and asymptomatic subjects have been reported to end the test due to pain in the low back area suggesting that factors limiting holding time is unclear (Ropponen *et al.*, 2005). Therefore, within these studies it was not possible to fully ascertain if the time to fatigue was due to fatigue, nor was it possible to ascertain if the time to fatigue was centrally driven in individuals.

In order to facilitate an evaluation of muscle fatigue due to the Biering-Sorensen test, RMS EMS fatigue measurements within this study were used to identify changes in amplitude and has been identified as a reliable measure reflective of the force of muscle contraction. However, it may have been more appropriate to measure fatigue in the frequency domain either through mean frequency (MNF) or median frequency (MDF). Measurements in the frequency domain have been described as a more accurate indicator of muscle fatigue (Criswell, 2011). Changes in the power spectrum shift to lower frequencies are said to be as a result of changes in the waveform of the

motor unit and reduced conduction velocities associated with lactate and H^+ accumulation (Tanaka *et al.*, 2002; Kramer *et al.*, 2005). Therefore, if MT application improved blood volume and time to fatigue it may be more prudent to include EMG measures in the frequency domain and supplementary measures of lactate and H^+.

3.16.5 Study 7

Postural balance

Postural balance or sway was assessed by an RSscan® (RSscan Ipswich) balance system 7.22. The rational for including this measure as part of this study's methodology was due to the premise that muscle fatigue and reduced blood flow have also been observed to influence lumbo-sacral position sense (Brumagne *et al.*, 2013). Johanson et al. (2011) observed that acute back muscle fatigue lead to increased postural sway and a shift in proprioceptive dependency from the lumbo-sacral region to the ankles, while standing on an unstable surface. Furthermore, people with low back pain showed greater postural sway and were more dependent on ankle signals compared to healthy subjects. Increases in postural sway and reduced proprioception may, therefore, further contribute to spinal instability, and an increased risk of further injury. Furthermore, the lack of experimental data regarding the effects of fatigue on postural sway in subjects with LBP provided the scope to also evaluate the influence of MT on blood flow and its effect on low back fatigue and possible interactions with postural sway.

Although the above test is a valid measure of postural sway, it is limited in its ability to differentiate between whole body and ankle steered postural strategies. Furthermore, the protocol used in this study is not a standardised form of balance assessment for LBP subjects. Future studies could utilise muscle vibration, which is used as a strong stimulation for muscle spindles and can be used to appraise the role of proprioception in postural control. Displacement of the centre of pressure (CoP) specifies how the subject makes use of proprioceptive signals from the vibrated muscle to control posture

(Johanson *et al.*, 2011). In addition to this, the mechanism by which the fatigue task in this study alters balance is not known and may differ between individuals and groups.

4 The Effects of structured myofascial massage techniques on the flexion relaxation phenomena of the lumbar paraspinal musculature, range of movement, pain and disability in subjects with non-specific chronic low back pain.

4.1 Abstract

Background

Few studies have evaluated the Effects of a structured clinical massage (CM) approach for low back pain as compared to a traditional relaxation massage (RM) on pain, function and motor adaptations to LBP.

Objectives

To investigate the effects of CM techniques on the flexion relaxation response (FRP) of the lumbar spine musculature, range of movement (ROM), pain and disability profiles in subjects with non-specific chronic low back pain compared to a RM treatment.

Methods

Sixteen subjects (8 male and 8 female) with a history of chronic, non-specific LBP volunteered for the study. Subjects were randomly assigned to either a CM or RM massage intervention group and received 6 treatments over a six-week period. Subjects were measured for improvements in FRP at the multifidus (MF) and erector spinae (ES) muscle groups, ROM, pain and disability scales before and after the six-week intervention.

Results

A Two-way ANOVA indicated a main effect of time for trials achieving FRP for the MF muscle F (1, 13) = 12.109, p = .004 and a marginal main effect of time for the ES muscle F (1, 13) = 4.495, p = .054. There were significant improvements in VAS F (1, 13) = 6.74, p = .022, and PRI F (1, 13) = 10.254, p = <006, pain scores for the CM intervention compared to RM. There was also a significant improvement in

kinesiophobia scores F (1, 13) = 7.77, p = .015 and the ODI disability index F (1, 13) = 11.1, p = .005, for the CM group compared to the RM group.

Conclusions

This study did not find any difference in the FRP outcome measures between the CM and RM interventions for LBP. Analysis of the pain and disability scales revealed a significant interaction effect in favour of the clinical massage group, indicating that clinical massage was more effective in reducing patient perceptions of pain, fear avoidance and disability.

4.2 Introduction

Non-specific low back pain (NSLBP) is defined as LBP for which there is no recognisable pathology (Chou, 2010b). It has been reported that once low back pain has been present for more than a year, few people with long term pain return to normal activities (Savingley, 2009), and patients with a history of LBP are often associated with inadequate or incorrect motor control strategies (Panjabi, 2003; Hodges, 2011). Panjabi (2006), proposed that sub-failure injuries, due to instability and mechanical loading, may lead to a decrease in the intrinsic stability of the passive structures, such as ligaments and connective tissue, leading to an increase in the neutral zone of the spine and further overloading of spinal structures. As all the structures of both the passive and active systems are extensively enervated by mechanoreceptors, proprioceptors and nociceptors, any abnormal loading of the passive structures, which threatens the systems integrity, is experienced as painful and alarming and may in turn alter motor control (Hodges and Moseley, 2003; Jones et al., 2012; van Dieën et al., 2003)

In the acute phase of injury to the spine, the removal of the noxious stimulus through movement and motor adaptations may be seen as an essential mechanism to support the recovery from injury. However, if the adaptation continues this maintenance of motor adaptations in the chronic phase may not be of benefit to the individual. If this adaptation is not resolved, this may lead to continued compromise to the quality of movement, increased load on the tissues and an inefficient increase in muscle activity (Hodges, 2011). The adaptations associated with CLBP include; altered repositioning sense (Astfalck *et al.*, 2013). Altered trunk muscle co-ordination (Lamoth *et al.*, 2006; D'hooge *et al.*, 2013), and persistent activation of para-spinal muscles leading to an absence of the Flexion Relaxation Phenomena (FRP) (Colloca and Hinrichs, 2005).

Active and passive structures of the spine play an important role in load sharing of the spine in flexion and extension. During the flexion movement it has been identified that there is a transfer from the role of the active extensors to the passive structures of the spine, including posterior spinal ligaments and the thoraco-lumbar fascia, as the active extensors are relieved of their moment supporting role. To this extent a number of researchers have examined the electromyographic (EMG) activity of the trunk musculature and have identified this transfer from active to passive support as a period of myoelectric silence. This reduction in EMG activity typically occurred between mid and full flexion and was termed the Flexion relaxation Phenomena (Floyd and Silver, 1955).

The FRP appears to be modulated by a number of factors including muscular fatigue (Martin Descarreaux *et al.*, 2008), asymmetry of movement (Ning *et al.*, 2011), creep of the posterior passive structures (Solomonow, *et al.*, 2003), gravitational loading (Olson, Solomonow and Li, 2006) and clinical status (Colloca and Hinrichs, 2005). There is evidence to suggest that the FRP, is reduced or absent in patients with back pain and healthy subjects (Colloca and Hinrichs, 2005). The electrical signal reduction or silence that occurs in healthy subjects was initially hypothesised to represent a neural reflexive response from stretch receptors in the passive structures sending efferent impulses to cause a reflex inhibition of the trunk musculature. However, more recently Olsen et al (2006) have suggested that a complex interplay of kinematics, gravitational orientation and neurological responses from the posterior lumbar connective tissue structures exists in the development of FRP.

Compared to healthy subjects, low back pain sufferers experience either a smaller reduction in EMG activity of the lumbar spine musculature or a complete absence of the FRP, indicating persistent activation of musculature of the back. Likely explanations for the absence of FRP reside in the LBP population's clinical status. It

has been reported that the continued EMG activity may represent co-contraction stiffening of the spine reflecting an adaptation to pain exposure (Lalanne, Lafond and Descarreaux, 2009) , muscle spasm, guarding and exaggerated stretch reflex activity of the para-spinal musculature (Watson, Booker, Main and a C. N. Chen, 1997). Solomonow et al. (2003), have also observed a reflexive hyper-excitability in the para-spinal muscles and have attributed this result as a response to creep and micro-damage in the ligamentous fibres.

Massage therapy (MT) has been described as the manipulation of soft tissues of the body to enhance health and well-being (Sherman *et al.*, 2006) and represents one of the most prevalent forms of complementary and alternative medicine (CAM) treatments for LBP (Simmons, 2011). However, recent reviews have identified massage as being limited in its Effects as a treatment for LBP (Furlan *et al.*, 2015). One of the most common types of massage practiced is Swedish or relaxation massage (Cherkin *et al.*, 2009). However more recently structured massage, using techniques that are designed to facilitate outcomes within a more clinical setting, have been used for the treatment of various musculoskeletal conditions (Sherman *et al.*, 2006; Cherkin *et al.*, 2011). Previous studies have compared the Effects clinical massage techniques with a focus on LBP (Preyde, 2000; Trampas *et al.*, 2010), however comparisons between relaxation massage and other massage techniques are scarce (Cherkin *et al.*, 2011; Netchanok *et al.*, 2012). The study by Cherkin et al. (2011), compared RM with a structured CM massage technique for outcome measure of self-perceived pain and, disability. However, to date, there has been no study that has compared relaxation massage and a structured clinical massage on motor adaptations to low back pain. Within this background the present study was designed to investigate the effects of clinical massage (CM) techniques on improving outcome measures in subjects with CLBP. It was hypothesized that CM techniques would lead to an improved flexion relaxation response (FRP) of the lumbar spine musculature, range of movement

(ROM), pain and disability profiles in subjects with non-specific chronic low back pain compared to a RM treatment.

H_0: CM treatment does not lead to improved FRP responses, ROM, pain and disability score in subjects with CNSLBP compared to a RM treatment.

H_1: CM treatment does lead to improved FRP responses, ROM, pain and disability score in subjects with CNSLBP compared to a RM treatment.

4.3 Methods

4.3.1 Participants

Participants were sixteen students (8 male and 8 female) whose ages ranged from 18-47 and with a mean age of 30 (SD = 10) and had a history of chronic non-specific low back pain (CLBP). Participants were recruited by advertisement in the form of participation information sheets sent via email to students at the University of Kent and K College Folkestone. Ethical permission was obtained through the School of Sports and Exercise Sciences ethics committee. All participants were asked to complete an informed consent form and pre-test questionnaire (appendix 1) prior to any measurement or testing procedure. Participants were also screened at this point to assess inclusion or exclusion criteria. The inclusion criteria for this study were that participants had to have a history of chronic, non-specific, low back pain and be between18 and 65 years of age. This meant that subjects had to have a history of recurrent low back pain, occurring in multiple episodes over a 12-month period; or chronic low back pain defined as being present and or persistent within a 3-12-month period. Participants were excluded from this study if they reported the following: low back pain which has lasted less than 3 months, self-reported incidences of severe back or lower limb injury, surgery to the spine, major structural spinal deformity, ankylosing spondylitis, rheumatoid arthritis, spinal fracture, cancer, tumour or infection, nerve root compression, neurological conditions, psychiatric conditions, bleeding disorders,

corticosteroid medication via inhaler, pregnancy, acute systemic infection, severe fibromyalgia.

4.3.2 Study design

The study was a parallel designed, non-cross over randomised control trial pilot study designed to compare the effects of clinical massage with relaxation massage on FRP, ROM at the lumbar spine and pain and disability scales. Participants were randomly assigned to one of the two massage groups using a Microsoft excel program randomisation function. The two groups were: structured CM or relaxation massage (RM). Participants in the two massage groups received six treatments over a six-week period (figure 4.1 summarises the study design). To minimise bias the participants were not told which group they were assigned to, only that they were to receive massage for their low back pain. Table 4.1 below summarises the participant demographics.

Table 4. 1: Participant demographics, (mean [SD]), for CM and RM groups.

Variable	**CM**	**RM**	**p-Value**
N	8	8	-
Male: Female	4:4	4:4	-
Age (years)	27 (11)	32 (10)	.443
Body weight (KG)	74.89 (12.07)	78.73 (16.26)	.485
Height (M)	1.71 (0.08)	1.72 (0.11)	.778
BMI (kg/m^2)	25.72 (3.53)	26.68 (6.16)	.540

4.3.3 Procedures

Pre-intervention procedures

A description of the pre-intervention procedures can be found in section 3.1

Self-reported outcome measure questionnaires.

Three self-reported questionnaires were administered prior to and following the massage interventions, these were the 13-item Tampa Scale for Kinesiophobia, the McGill SF-MPQ pain scale questionnaire and the Oswestry Disability Index. A detailed description of these self-reported outcome measures can be found in section 3.5.

Surface electromyography

A description of the surface EMG procedures can be found in section 3.3

4.3.5 Data Analysis

A description of the data analysis can be found in section 3.4

4.3.6 Statistical Analysis

The normality assumption was assessed using Shapiro-Wilk*W* tests. Data were analysed using a 2X2 mixed analysis of variance (ANOVA). The independent variables (IVs) were time (2 levels: pre, post; within-participants) and group (2 levels: Relaxation Massage, Clinical Massage; between-participant). Dependent variables (DVs) were: percentage of trials achieving FRP at two muscles (ES, MF); relative angles at which FRP was achieved at two muscles (ES, MF) and two locations (trunk and lumbar); ratings of pain using two scales (VAS, PRI); fear avoidance ratings using the TSK-13 scale and back disability ratings using the ODI-V2. Separate ANOVA's were carried out for each DV. All effects were reported as significant at $p < .05$ and with marginal significance at $p < .01$.

4.3.7 Intervention

A description of the massage interventions used within this study can be found in section 3.8.

Relaxation massage group

In this treatment group the massage therapist provided 6 full body massages intended to provide a generalised relaxation approach to the treatment of low back pain.
In order to standardise the treatment each session was allocated a 60-minute time slot with specific areas of the body included and in particular time slots. All participants received the same treatment and, in the order, described below.

- Back: 15-20 minutes
- Back of legs: 10-15 minutes
- Front of legs: 10 minutes
- Arm and hands: 10 minutes
- Neck and shoulders: 10 minutes

Structured Clinical Massage

A description of the massage techniques used within this study can be found in section 3.8.2. In this treatment group the therapist provided 6 treatments designed specifically to identify and address musculoskeletal contributors to low back pain. Prior to the first treatment the participant was taken through a musculoskeletal assessment to identify possible contributors to low back pain. This included postural, palpatory, functional assessment and muscle testing. The therapist was able to use a wide range of CM techniques at his/her discretion, and outlined in section 3.8.2 specifically designed to affect the musculoskeletal system and facial tissue, following a similar protocol (Cherkin *et al.*, 2011). Therefore, there was no specific order to the application of techniques as the aim of this intervention strategy was to integrate assessment and treatment techniques to develop individualised treatment approaches for low back pain. Effleurage stroking and kneading petrissage was also permitted to prepare the area for the more advanced clinical massage techniques, to warm the area and to be used as transition strokes and are described above.

4.4 Results

Figure 4.1 below outlines participant flow including enrolment, participant allocation, randomisation and analysis.

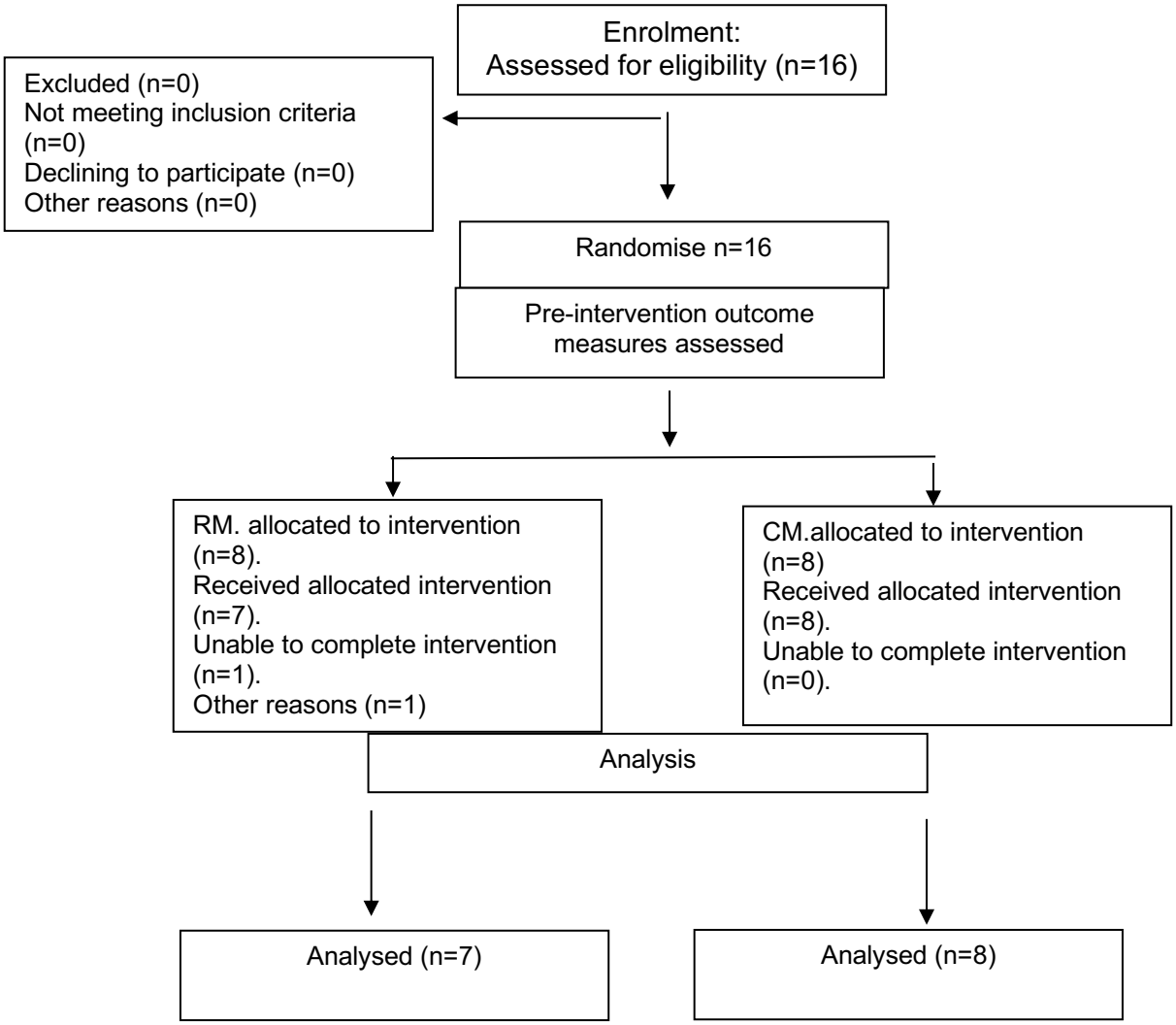

Figure 4. 1 Flow diagram depicting flow and allocation of participants from allocation to analysis.

4.4.1 Percentage of Trials Achieving FRP

The percentage of trials achieving FRP did not meet the normality assumption. In order to side average, the percentage of FRP across trials, a Man-Whitney U test was used to identify any significant differences between left and right-side results. A Man-Whitney U tests revealed no difference between sides. The side average was therefore determined to allow each muscle's FRP count to be compared pre, and post and between groups. For both groups the total number of trials achieving FRP increased

following the interventions. Figure 4.2 below shows the percentage of trials across both muscles that achieved FRP for both RM and CM groups. A 2X2 mixed ANOVA was used to compare within (time) and between (group) factors for the MF and ES muscles and are described below.

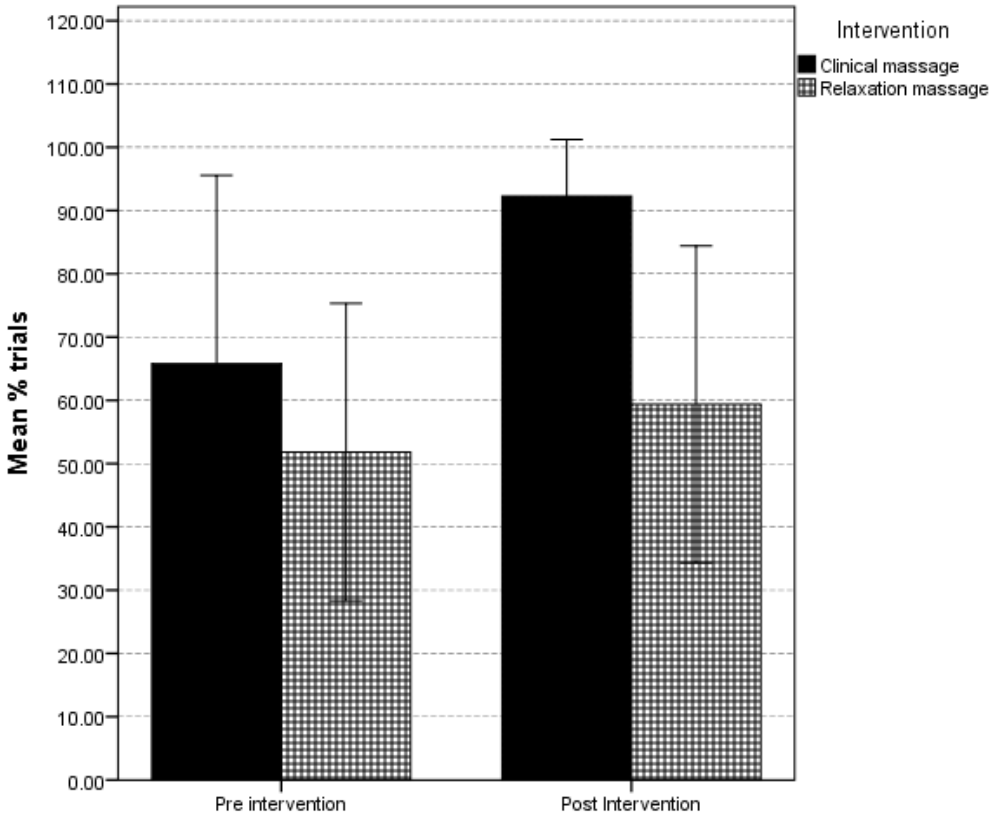

Figure 4. 2 Percentage of total number of trials where FRP was achieved for both the ES and MF muscle groups at time pre, and post.

Percentage of FRP achieved (MF)

There was a significant main effect of time, F (1, 13) = 12.109, p = .004, η_p^2= .48. Percentage of FRP achieved increased significantly from time pre (M = 45°, SEM = 8°) to time post (M = 76°, SEM = 7°). There was no interaction effect F (1, 13) = 1.437, p = .252, η_p^2= .100, or group effect F (1, 13) = 1.644, p = .222, η_p^2= .112. The profile plot below represents the change in percentage of FRP achieved following interventions for both groups for the MF muscle.

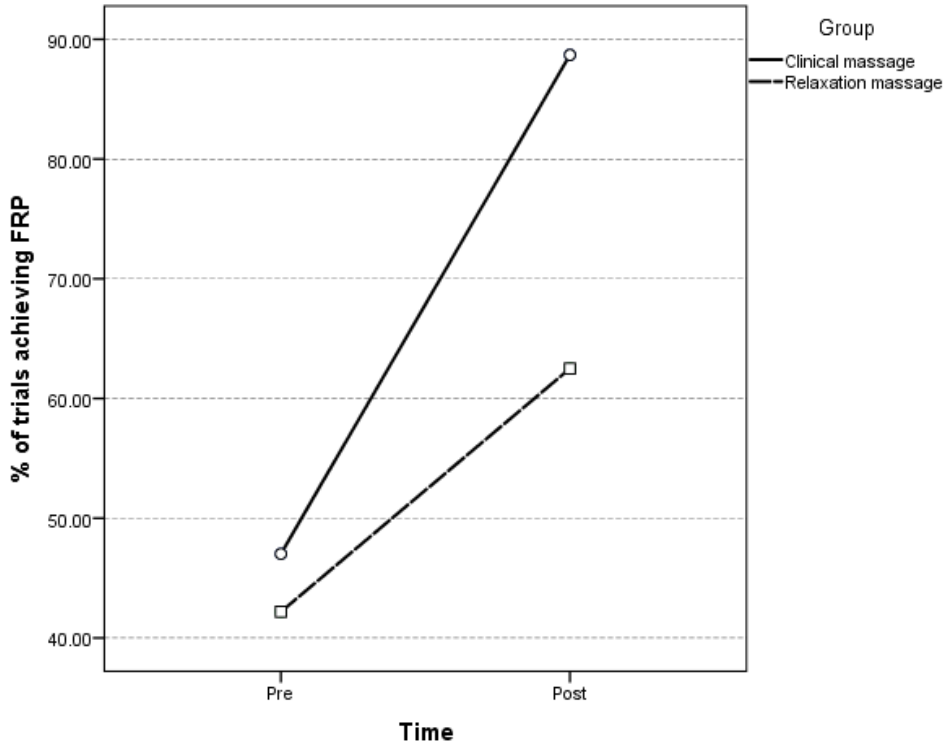

Figure 4. 3 Within and between group comparisons of means for the percentage of FRP achieved for multifidus muscle (MF) at time pre, and post intervention for the clinical massage (CM) and relaxation massage (RM) groups.

Percentage of FRP achieved (ES)

There was a marginal main effect of time, F (1, 13) = 4.495, p = .054, η_p^2= .257.

Percentage of FRP achieved increased with marginal significance from time pre (M = 60°, SEM = 10°) to time post (M = 81°, SEM = 7°). There was no interaction effect F (1, 13) = .750, p = .402, η_p^2= .055, or group effect F (1, 13) = .1.305, p = .274, η_p^2= .091.

The profile plot below represents the change in percentage of FRP achieved following interventions for both groups for the ES muscle.

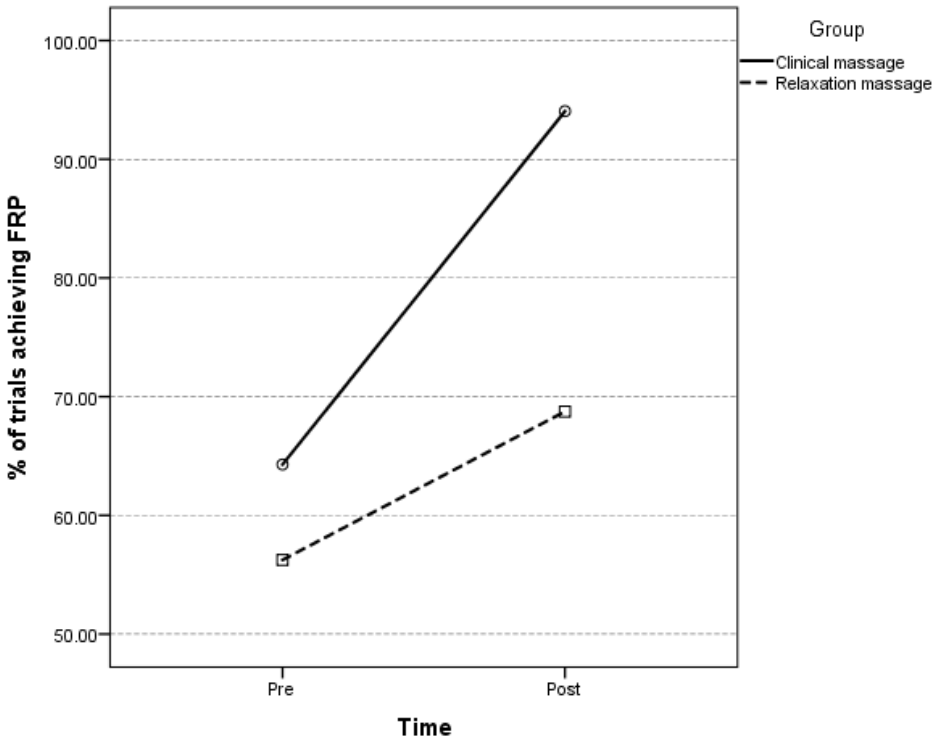

Figure 4. 4 Within and between group comparisons of means for the percentage of FRP achieved for erector spinae muscle (ES) at time pre, and post intervention for the clinical massage (CM) and relaxation massage (RM) groups.

4.4.2 Angles at which FRP was achieved.

Side averaged angles, relative to total flexion, angles measured at the MF lumbar and trunk sites and ES lumbar and trunk sites met the normality assumption. For data averaged over left and right, we want to know if there is a significant effect of Time (pre, post, within), or Group (RM, CM, between) or an interaction between the two. To check this, we conduct a 2 x 2 mixed analysis of variance (ANOVA). Results of the 2 x 2 mixed ANOVA showed only a significant main effect of time for ES lumbar, F (1, 7) = 13.662, p = .008, η_p^2= .661, and trunk F (1, 7) = 15.078, p = .006, η_p^2= .68. Relative angles increased significantly from Time pre (M = 62°, SEM = 6.1°) to Time post (M = 78°, SEM = 4.2°) for ES Lumbar and from Time pre (M = 68°, SEM = 5°) to Time post

(*M* = 82°, *SEM* = 3°) for ES Trunk. Means and standard deviations are reported in Table 4.2 below.

Table 4. 2 Means and standard deviations data for the relative angles at which the FRP was achieved for the erector spinae (ES) trunk and Lumbar measures for the clinical massage (CM) and relaxation massage (RM) groups.

Group	ES Trunk (Mean/SD)		ES Lumbar (Mean/SD)	
	Pre	Post	Pre	Post
CM	68.84 (12.44)	79.65 (10.69)	60.06 (12.75)	74.72 (14.49)
RM	66.32 (17.53)	83.4 (8.98)	60.85 (21.37)	81.12 (10.56

4.4.3 Pain and disability scales

The means and standard deviation for the pain scales and the relevant sub-scales measured within this study can be viewed below (see table 4.3).

Table 4. 3 Mean and standard deviation scores for the pain and disability scales

Pain and disability scale	Clinical massage		Relaxation massage	
	Pre-intervention (mean/SD)	Post intervention (mean/SD)	Pre-intervention (mean/SD)	Post intervention (mean/SD)
VAS pain scale	4.69 (1.72)	1.84 (0.97)	4.31 (1.72)	3.84 (1.32)
TSK-13 Total	25.00 (6.30)	19.86 (6.79)	24.25 (3.15)	23.28 (3.93)
PRI-total	13.89 (3.14)	5.25 (2.25)	12.00 (7.62)	9.88 (10.10)
ODI	9.57 (2.70)	5.00 (2.83)	7.63 (4.57)	6.63 (5.26)
TSK-13 AA	16.29 (3.95)	12.86 (4.74)	15.88 (1.13)	15.38 (1.99)
TSK-13 SF	8.71 (2.69)	7.00 (2.24)	8.38 (2.45)	7.88 (2.23)
PRI Sensory	13.00 (3.61)	4.86 (2.12)	10.00 (4.81)	8.25 (6.80)
PRI Affective	1.14 (1.46)	0.29 (0.76)	2.00 (3.38)	1.63 (3.46)

Visual Analogue Scale (VAS)

A 2X2 mixed ANOVA revealed a significant main effect of time for pain scales as measured by the VAS, F (1, 13) = 13.09, p = .003, η_p^2= .502. Pain scores reduced significantly from time pre (M = 4.4°, SEM = 0.5°) to post (M = 2.7°, SEM = 0.3°). There was also a significant interaction effect between group and time F (1, 13) = 6.74, p = .022, η_p^2= .34, with a significant reduction in VAS score for the CM group (M = 5°, SEM = 0.7° at time pre, and M = 1.6°, SEM = 0.4° at time post) compared to the RM group (M = 4.3°, SEM = 0.6° at time pre, and M = 4 °, SEM = 0.4° at time post). The profile plot below represents the change in VAS score between and within groups prior to and after the intervention.

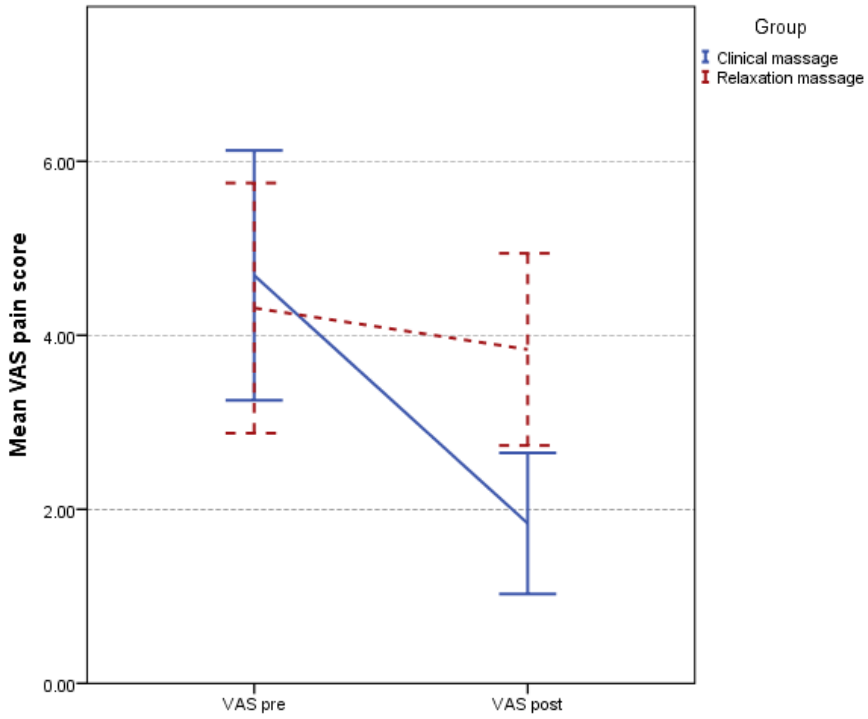

Figure 4. 5 VAS values at time pre, and post intervention for the clinical massage (CM) and relaxation massage (RM) groups.

Kinesiophobia.

TSK-13 total score: A 2X2 mixed ANOVA revealed a significant main effect of time, F (1, 13) = 15.45, p = .002, η_p^2= .543, with a reported reduction in kinesiophobia, from time pre (M = 25°, SEM = 1°) to time post (M = 22°, SEM = 1°). There was also a significant interaction between time and group, F (1, 13) = 7.77, p = .015, η_p^2= .423,

indicating that the change in kinesiophobia scores over time were different between CM and RM groups. The table below presents the mean TSK scores

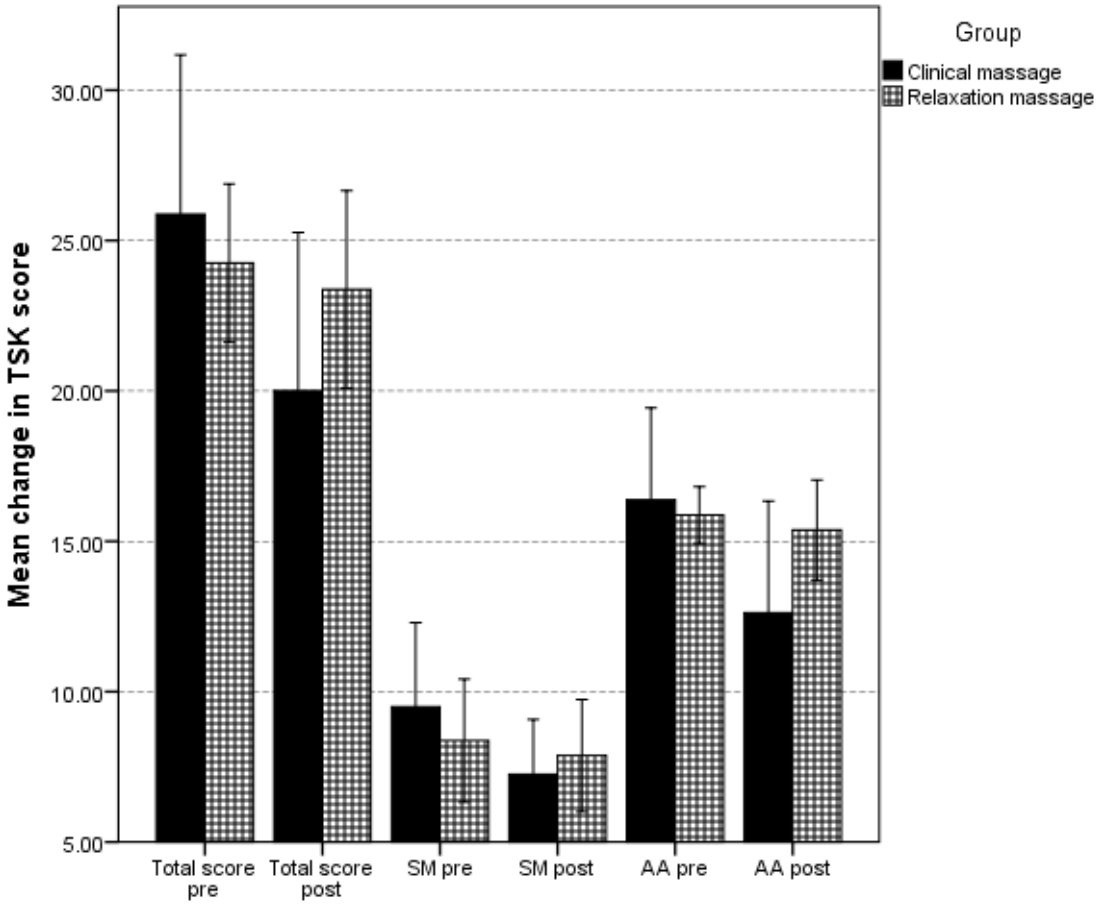

Figure 4. 6 Mean values for the TSK-13 total score, SM and AA scores at time pre, and post intervention.

TSK-13 AA score: A 2X2 mixed ANOVA revealed a significant main effect of time *F* (1, 13) = 17.14, *p* = .001, η_p^2= .569, where mean TSK-13 AA score was reduced over time (*M* = 16°, *SEM* = 0.7° at time pre, and *M* = 14°, *SEM* = 0.9° at time post). There was also a significant interaction effect between time and group: *F* (1, 13) = 9.52, *p* = .009, η_p^2= .423, where mean TSK-13 AA scores were lower at time post for the CM group (*M* = 13°, *SEM* = 1°), compared to the RM group (*M* = 15°, *SEM* = 1°). The profile plot below depicts the mean scores over time and between groups.

TSK-13 SF scores: A 2X2 mixed ANOVA revealed only a marginal main effect of time F (1, 13) = 4.627, p = .051, η_p^2= .262, where mean TSK-13 SF score was reduced over time (M = 9°, SEM = 0.7° at time pre, and M = 7°, SEM = 0.6° at time post).

Pain Rating Index (PRI)

PRI-Total score: Analysis of Pain Rating Index revealed there was a significant main effect of time, F (1, 13) = 28.05, p = <.001, η_p^2= .667. Mean PRI scores reduced from time pre (M = 13°, SEM = 1°), to time post (M = 8°, SEM = 2°). There was also a significant interaction effect between time and group F (1, 13) = 10.254, p = <006, η_p^2= .423. After 6 weeks of intervention the PRI rating was significantly reduced in the CM group (M = 5°, SEM = 3°) compared to the RM group (M = 10°, SEM = 3)

PRI-Sensory Score: Results for the PRI sensory component revealed a significant main effect of time F (1, 13) = 22.16, p = <.001, η_p^2= .630. Estimated marginal means showed a significant reduction in score from pre, to post intervention, M = 11°, SEM = 1° and M = 7°, SEM = 1° respectively. There was also a significant interaction effect of time and group, F (1, 13) = 9.25, p = <.009, η_p^2= .416, with mean scores significantly reduced at time post for the CM group (M = 5°, SEM = 2°) compared to the RM group (M = 8°, SEM = 2°).

PRI-Affective Score: Results for the PRI affective component revealed only a significant main effect of time. F (1, 13) = 5.79, p = <.032, η_p^2= .308. Mean scores reduced from time pre (M = 1.6°, SEM = 0.7°) to time post (M = 0.96°, SEM = 0.7°). The table below represent the mean scores for PRI, sensory and affective subscales.

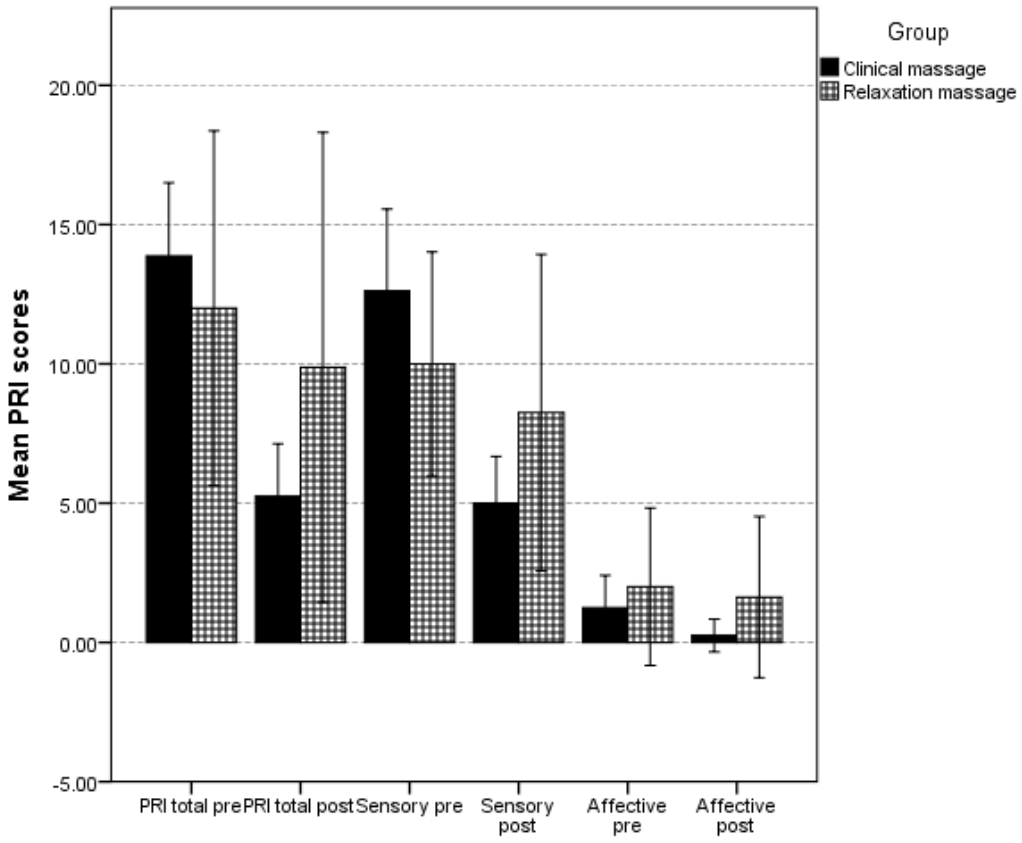

Figure 4. 7 Mean Pain Rating Index (PRI) along with sensory and affective subscale scores pre, and post intervention for the Clinical massage (CM) and relaxation massage (RM) groups

Oswestry Disability Index

A 2X2 mixed ANOVA revealed a significant main effect of time F (1, 13) = 27.04, p = <.001, η_p^2= .675. Mean scores reduced over time from pre (M = 9°, SEM = 0.9°) to post (M = 6°, SEM = 1.1°). There was also a significant interaction effect between group and time F (1, 13) = 11.1, p = .005, η_p^2= .461. The ODI scores significantly reduced from time pre (M = 9.6°, SEM = 1.4°) to post (M = 5°, SEM = 1.6°) for the CM group, compared to the RM group at pre (M = 7.6°, SEM = 1.4°) to post (M = 6.6°, SEM = 1.5°). The profile plots below show the mean scores over time and between groups.

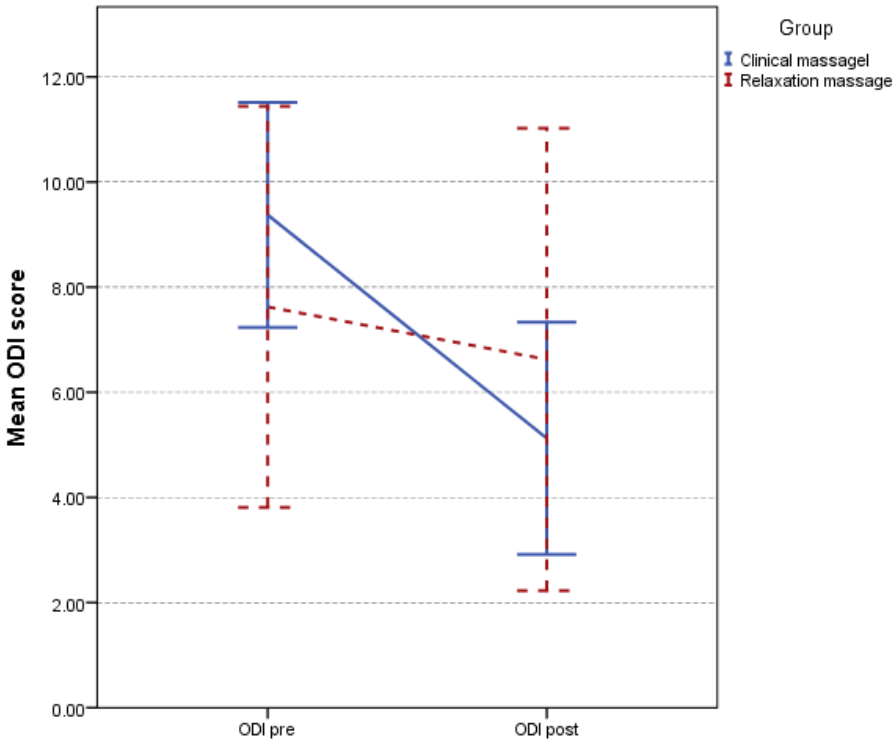

Figure 4. 8 Oswestry Disability Index (ODI) values at time pre, and post intervention for the clinical massage (CM) and relaxation massage (RM) groups.

Dependent variable	Research Question	Test	Sig.	Effect size	ANOVA G-Power Sensitivity: Effect size, actual power, sample size
%FRP Achieved Multifidus	Change in % FRP achieved following interventions? Multifidus	Mixed ANOVA Time	.004	$\eta_p^2 = 0.48$	η^2 = 0.456 / 46% Cohen's f = 0.9158 Actual power = 0.9188 required sample @ 80% power = 13
	Was there an interaction effect between the groups? Multifidus	Mixed ANOVA Interaction	.252	n/a	η^2 = 0.0542/ 5.4% Cohen's f = 0.2394 Actual/observed power = 0.09 required sample @ 80% power = 195
%FRP Achieved Erector Spinae	Was there a change in % FRP achieved following interventions? Erector spinae	Mixed ANOVA Time	.054	η_p^2= .257	η^2 = 0.246/ 24.6% Cohen's f = 0.572 Actual/observed power = 0.56 required sample @ 80% power = 27
	Was there an interaction effect between the groups? Erector spinae	Mixed ANOVA Interaction	.402	n/a	η^2 = 0.0410 / 4.1% Cohen's f = 0.21 Actual/observed power = 0.08 required sample @ 80% power = 252
Kinesiophobia	Was there a change in Kinesiophobia score following interventions?	Mixed ANOVA Time	.002	η_p^2 = .543	η^2 = 0.4266 / 43% Cohen's f = 0.863 Actual/observed power = 0.89 required sample @ 80% power = 14
	Was there an interaction effect between the groups?	Mixed ANOVA Interaction	.015	η_p^2 = .423	η^2 =0.2146 / 22% Cohen's f = 0.523 Actual/observed power = 0.29 required sample @ 80% power = 44
VAS Pain Scale	Was there a change in VAS score following interventions?	Mixed ANOVA Time	.003	η_p^2 = .502	η^2 = 0.39882 / 39.9% Cohen's f = 0.812 Actual/observed power = 0.85 required sample @ 80% power = 15
	Was there an interaction effect between the groups?	Mixed ANOVA Interaction	.022	η_p^2 = .341	η^2 = 0.20521 / 20.5% Cohen's f = 0.51 Actual/observed power = 0.3 required sample @ 80% power = 47

Dependent variable	Research Question	Test	Sig.	Effect size	ANOVA G-Power Sensitivity: Effect size, actual power, sample size
PRI Pain Scale	Was there a change in PRI score following interventions?	Mixed ANOVA Time	<.001	η_p^2 = .667	η^2 = 0.54/ 54% Cohen's f = 0.1.08 Actual/observed power = 0.98 required sample @ 80% power = 10
	Was there an interaction effect between the groups?	Mixed ANOVA Interaction	.006	η_p^2 = .423	η^2 = 0.2 / 20% Cohen's f = 0.50 Actual/observed power = 0.3 required sample @ 80% power = 48
ODI back function scale	Was there a change in ODI score following interventions?	Mixed ANOVA Time	<.001	η_p^2 = .675	η^2 = 0.53/ 53% Cohen's f = 0.1.06 Actual/observed power = 0.98 required sample @ 80% power = 11
	Was there an interaction effect between the groups?	Mixed ANOVA Interaction	.005	η_p^2 = .461	η^2 = 0.22 / 22% Cohen's f = 0.53 Actual/observed power = 0.3 required sample @ 80% power = 43

Table 4. 4 Mixed ANOVA effect sizes converted to Cohen's f, actual observed statistical power and required sample sizes for the main dependent variables

4.5 Discussion

4.5.1 Flexion Relaxation Phenomena (FRP)

The results of this study indicated that both interventions resulted in significant improvements in the percentage of FRP trials for the multifidus muscle group. Although the percentage of trials achieving FRP for the erector spinae muscle group did improve after the six-week intervention, this was not significant at the 0.5 level set within this study. FRP has been widely investigated within the literature however there are few studies that have explored the effects of massage on FRP in patients with NSCLBP or the effects of manipulative therapy in general using FRP. However, the results of this study are in accordance with Lalanne et al., (2009) who observed a reduction of EMG activity at the L2 ES level following spinal manipulation therapy. The manipulation therapy utilised in that study was a single chiropractic manipulative thrust delivered at the L3 segment as opposed to soft tissue treatments. Ritvanen et al., (2007) found no improvements in EMG parameters following traditional bone setting or physical therapy. In Ritvanen's study the physical therapy intervention included therapeutic stretching, trunk stabilisation and massage, however, the massage techniques were not specified and a fitness centre specialist carried out these treatments without reference to the massage practitioner's skills or experience.

The absence of FRP in the low back pain population is said to be an indication of persistent activation of the para-spinal muscles to increase spinal stabilisation in response to pain or altered muscle activation patterns (Colloca and Hinrichs, 2005); however, it is just one myoelectric measure that can be used to assess neuromuscular adaptations to an intervention. Previous studies have identified other myoelectric changes following massage therapy implicating both central and reflexively mediated changes in muscle activation (Arroyo-Morales et al., 2011; Behm et al., 2013; Sefton et al., 2011). For example, Sefton *et al.*, (2011), observed reduced EMG activity following therapeutic massage at the upper trapezius. The same authors also observed a reduction in activity of

the motor neurone (MN) pool of the flexor carpi radialis muscle. As the reductions in the FCR alpha MN pool were observed distal to the area of treatment the authors suggested that this might be a centrally mediated response to massage. The therapeutic massage intervention applied in this study was developed as a clinical intervention for neck and shoulder pain and included a combination of effleurage, petrissage, frictions, passive stretching and trigger point compressions.

Behm *et al.*, (2013) measured spinal reflex excitability of the triceps surae muscle following two types of massage. They observed reductions in the Hoffman reflex (H-reflex), following massage combined with static stretching. They concluded that the combination of massage and static stretching could have led to greater inhibitory influences on the muscle, than massage alone. The massage techniques employed were either vigorous tapotement techniques or effleurage and circular stroking to the musculotendinous junction (MTJ). Improvements following MTJ massage were attributed to autogenic inhibition following stimulation of Golgi tendon organs, whereas the tapotement technique may have stimulated vibratory and pressure sensitive mechanoreceptors facilitating pre-synaptic inhibition of 1a afferents. The addition of static stretching may have further attenuated muscle activation through reflexive presynaptic inhibition of 1a afferents. The effects of static stretching on H-reflex amplitude have been documented (Cuissard, Duchateau and Hainaut, 1988; Avela, Kyrolainen and Komi, 1999) and may be attributed to a reduction in muscle spindle afferent discharge due to increased compliance of the muscle following stretching, reducing the muscle spindles sensitivity to stretch (Avela, Kyrolainen and Komi, 1999).

The results of the present study indicated that the CM intervention may be more effective in altering FRP than RM. The evidence outlined above suggests that muscle activity may be influenced by many factors, and at multiple levels, leading to the muscle relaxation. The CM group experienced a variety of soft tissue treatment techniques that might have

contributed to the improved FRP patterns. CM techniques have been identified as focussed manipulations to muscle and facia, capable of addressing the nervous system and used to reduce muscle spasm (Sherman *et al.*, 2006). Many of the individual CM techniques employed within this study have been shown to influence muscle activity through local or central neural mechanisms and / or pain perception. (Schleip, 2003a; Lucas, Polus and Rich, 2004; Kostopoulos *et al.*, 2008; Simmonds, Miller and Gemmell, 2012; Fryer and Pearce, 2013). Fryer & Pearce, (2013), demonstrated that MET's to the L5/SI spinal segment resulted in immediate inhibition of MN excitability in the motor cortex and spinal cord. Even though the study measured muscle responses at the gastrocnemius muscle and on asymptomatic participants, it suggests that MET's used within the present study may have the ability to decrease motor excitability, facilitating muscle relaxation, decreasing tension and pain. Kostopoulos *et al.*, (2008) identified that ischemic compression therapy combined with passive stretching significantly reduced the spontaneous electrical activity (SEA) and perceived pain in TrP's in the upper trapezius muscle. The combination of treatments was superior in effect to the same treatments in isolation. It is proposed that TrP therapy and stretching may increase blood circulation resulting in a normalisation of the metabolic state at the area of the TrP. Subsequent reductions in ACh levels in the myofascial active loci may deactivate the contracted sarcomeres. The same authors also propose that the passive myofascial stretching inhibits gamma muscle spindle response through slow lengthening of contracted sarcomeres. Furthermore, Lucas *et al.*, (2004) established that latent TrP's altered the muscle activation patterns in the scapular rotator muscles indicating a coping strategy associated with movement inefficiency. However, once the latent TrP's had been removed muscle activation patterns were normalised. Therefore, alterations in FRP observed in the CM group within the present study might reflect an improvement in motor recruitment and joint positioning following the inactivation of TrP's within and around the lumbo-pelvic musculature.

Myofascial massage techniques have also been associated with improvements in pain and function (Ajimsha, Daniel and Chithra, 2014). Schleip (2003) proposed that slow deep pressure, often a feature of myofascial release and connective tissue massage, leads to stimulation of mechanoreceptors in the densely innervated fascia, triggering an autonomic response that lowers sympathetic activity in favour of a more parasympathetic state. This response is proposed to alter global muscle tone as well as intrafascial smooth muscle cells. Simmonds *et al*., (2012) also propose that the benefits of myofascial therapies are due in part to neurophysiological effects through stimulation of pacini and Ruffini corpuscles; which respond to vibratory techniques and deep slow myofascial techniques respectively. Stimulation of these mechanoreceptors has been shown to produce various autonomic responses often associated with a therapeutic effect. MFR techniques were employed considerably as part of the intervention for the CM group but the myofascial network may also be manipulated through some of the techniques received by the RM group. As the results of the present study showed a significant and marginally significant effect of time for improved FRP counts in the MF and ES muscles respectively, it is possible that some these effects may be due to the reflexive autonomic mechanisms identified above. Therefore, further investigation of this particular technique utilised in comparison with others may prove useful in discriminating such effects.

4.5.2 Pain and Disability Scales

The results of the present study revealed that the CM intervention was significantly better at reducing perceptions of pain, disability and kinesiophobia than the RM group, however these results were in slight contrast to Cherkin *et al*., (2011), who found that both RM and structural massage were effective at reducing pain and improving function compared to usual care in chronic low back pain patients. Both massage treatments within their study were very similar to the present study however the disability and pain scales used were the RDQ and a 'bothersomeness' pain scale of between 1 and 10. Furthermore, subjects in their study received 10 instead of 6 treatments within the present study. The authors

suggest that the time spent in a relaxing environment may, in part, contribute to non-specific effects of both massage treatments. The RM group in the present study spent less time in this environment than their study which may have contributed to the different results observed. Furthermore, in their study participants were randomly assigned to 27 different licensed therapists instead of a single therapist conducting both treatments. Although these therapists had a minimum of 5 years' experience, variability between treatments could not be discounted. However, in agreement with some of the results of the present study, Little *et al*. (2008), found massage, compared to controls, to be effective in the short term in improving disability and a pain troublesome scale in back pain subjects but did not show any difference for fear avoidance of physical activity. Lower back pain and sleep disturbance was also shown to be improved following 5 weeks of massage (Field, Hernandez-Reif, Diego, & Fraser, 2007). Furthermore, in a trial that used similar techniques to that administered to the CM group within the present study Preyde, (2000) also observed significant improvements in RDQ scores and pain (McGill's PPI and PRI) scales for chronic low back pain.

When outcome measures of pain and function are examined Simmons, (2011), proposed that massage may have a more consistent impact on pain than function or ROM. However, the mechanisms that are involved in pain relief following massage are not fully understood. It is possible that manipulation of muscle and fascia may contribute to local and centrally mediated alterations in pain perception. For example, massage has been shown to modulate pain through local blood flow changes and the removal of metabolic waste (Mori *et al*., 2004), while acupuncture and light touch have been observed to modulate the limbic system and subcortical grey structures via the stimulation of peripheral sensory receptors (Hui *et al*., 2000). Sagar *et al*., (2007), therefore, postulates that massage may directly affect muscle physiology which is then communicated through dorsal horn afferents to the central nervous system leading to subcortical gaiting and modulation of the limbic system. The results of the present study, indicating that CM

massage was significantly better at reducing pain over RM, may be due to the specific role of CM techniques in manipulating tissues for pain relief. Furthermore, it has been suggested that the dorsal peri-aqueductal grey (dPAG) may also be implicated in pain relief through descending inhibitory pain pathways (Frey Law *et al.*, 2008; Simmonds, Miller and Gemmell, 2012). Frey Law *et al*., (2008) suggest that deep tissue massage may produce an anti-nociceptive effect via oxytocin-mediated influences on the dPAG. Previous studies have identified that intra-dPAG injection of oxytocin increases pain threshold, therefore oxytocin release following massage may reduce pain through descending pathways and its interaction with the opioid system (Lund *et al.*, 2002). Finally, local effects of clinical massage may have contributed to reductions in pain associated with myofascial trigger points. The combination of TrP therapy, MET's and myofascial stretching, as applied to the CM participants of this study, may be responsible for pain reduction directly at the site of the TrP and general improvements in perceived pain through increased stretch tolerance of the whole muscle.

Improved self-reported measures of kinesiophobia found within the present study have been reported elsewhere (Little *et al.*, 2008). However, another study, (Lara-Palomo *et al.*, 2013), found that a form of inferential 'electro' massage was effective in reducing VAS scores, ODI scores but not kinesiophobia scores. The paucity of studies measuring the effects of massage on kinesiophobia makes it difficult to draw firm conclusions. However, the link between fear avoidance behaviours and biomechanical changes have been well documented (D'hooge et al., 2012; Hodges, 2011; Trost et al., 2012; Williams, Haq, & Lee, 2010). Within the present study, when the TSK scale was analysed for its component subscales it was activity avoidance beliefs that showed the greatest improvements, suggesting that subject's fear of activity attenuated following treatment. Trost *et al*., (2012) reported that individuals with high pain related fear adopted avoidant spinal strategies. Although this study's results were based on experimentally induced back pain, early avoidant strategies may lead to chronic adaptations. Furthermore, when

back pain was induced on subjects it was shown to reduce walking and forward bending velocities as well as an elevation of superficial muscle activity (Williams, Haq and Lee, 2010). Therefore, the reductions in pain perception and fear avoidance scores following CM identified within the present study may have contributed to improved FRP counts also observed. As altered muscle activation patterns probably reflect changes at multiple levels of the nervous system the removal of the pain stimulus only would not necessarily bring about the required muscle activation pattern changes in CLBP sufferers (Hodges, 2011). However, CM techniques employed within this study include techniques to reduce pain as well as myofascial manipulation and stretching strategies that may contribute to movement re-education. Therefore, CM may be well placed to reduce pain and fear avoidance and help to facilitate movement re-education.

One of the main claims touted by massage therapists is that massage has the ability to improve blood flow (Moraska, 2005). Furthermore, one of the possible mechanisms for pain reduction and functional changes within spinal muscles may be attributed to improvements in blood flow following massage (Kovacs *et al.*, 2001; Mori *et al.*, 2004). As outlined in the literature review, the massage research has a number of methodological limitations affecting the reliability of the results. This study attempted to compare the effects of two classifications of massage on parameters associated with a LBP population, however, within these classifications are a number of different techniques that could have individually accounted for the results found within this study. Therefore, the following chapters will attempt to address some of these issues through the utilisation of one of the techniques used within the CM classification. Previous studies into the effects and utilisation of neuromuscular techniques have been well documented (Penas *et al.*, 2005; Kao *et al.*, 2008; Trampas *et al.*, 2010; Ramsook and Malanga, 2012; Wong, 2012; Fryer and Pearce, 2013). However, very few studies have attempted to identify the effects of Myofascial techniques LBP and the mechanisms associated with this technique. The use of and popularity of this as a manual therapy technique has grown in recent years in

parallel with the interest and research into fascia as a form of connective tissue (Schleip, 2003b). Therefore, the following chapters will attempt to identify the mechanisms associated with myofascial techniques, specifically the effects of this intervention on blood flow and its efficacy in the treatment of non-specific LBP.

4.5.3 Limitations and areas for future research

The present study was a pilot study to determine the effects of clinical massage techniques compared to a more traditional relaxation massage technique on subjects with non-specific LBP. Sources of bias within this study are discussed more generally in section 9.1. The sample size in this study was therefore relatively small resulting in some of the results being statistically underpowered. Table 4.4 represents the main dependent variable effect sizes and observed power. Appendix 2 provides effect sizes and observed power for all remaining dependent variables and research questions including post hoc tests. It is clear from this analysis that the interaction effect for pain and disability scores achieved low to moderate effect sizes and observed statistical power of below the 0.8 level. This was also evident with the FRP achieved analysis for both muscle groups, although only a main effect of time was achieved. The analysis also clearly identifies the need for greater sample sizes across all variables. The protocol within this study identified the number of trials experiencing flexion relaxation (see 3.1.2). However further studies may utilise the use of a flexion relaxation ratio (FRR). FFR is calculated as the maximum EMG during forward flexion divided by the minimum resting (fully flexed) EMG. This method has been suggested to provide a normalising EMG factor to the EMG data, making it possible to compare factors across time and individuals (Owens, Gudavalli and Wilder, 2011). Furthermore, FRR may be a more sensitive evaluation of back muscle activity where subjects exhibit lower disability scores (Watson, Booker, Main and A. C. N. Chen, 1997), as a complete cessation of FRP in these subjects is not always measured. Therefore, FRR may be a more effective criterion in the identification of EMG alterations at the lumbar spine during the flexion motion.

4.5.4 Conclusion

The results of the present study indicate that both forms of massage treatments may be effective as an intervention to modify the FRP in subjects with chronic low back pain. Analysis of the pain and disability scales revealed a significant interaction effect in favour of the clinical massage group, indicating that clinical massage was more effective in reducing patient perceptions of pain, fear avoidance and disability. The findings may suggest a relative role for clinical massage techniques for subjects with non-specific LBP

5 The acute effects of integrated myofascial massage techniques on Peripheral blood volume at the lumbar paraspinal region compared to relaxation massage

5.1 Abstract

Background

One of the most touted effects of massage therapy is the ability to improve or increase blood flow to the tissues being treated. However, the evidence for this effect remains equivocal. Myofascial massage techniques (MT) are a popular form of clinical massage used to reduce pain and improve function as well as blood flow and circulation. However, to date, very few studies have compared the effects of MT on peripheral blood volume changes at the paraspinal region to relaxation massage (RM) using Near Infrared Spectroscopy (NIRS).

Objectives

To investigate the acute effects of myofascial techniques on the peripheral blood volume to the paraspinal myofascial region, compared to a traditional relaxation massage.

Methods

A non-crossover, parallel RCT design. Forty-four healthy participants (19 male, 15 female) volunteered for the study and randomly assigned to either MT, RM and sham ultrasound interventions. Paraspinal blood flow was measured at the L4 vertebral level using NIRS, before and after each of the interventions. Blood volume variables were compared from the pre-intervention to post intervention measures.

Results

A one-way ANOVA revealed significant increases in oxygenated haemoglobin (O_2Hb), F (2-26.44) = 15.82, p = < .001, deoxygenated haemoglobin (HHb), F (2-41) = 3.59, p = .037 and total haemoglobin (tHb), F (2-26.71) = 15.47, p = < .001, at the paraspinal region following the MT intervention compared to the RM and control groups. There was no significant difference in blood volume variables between the RM and control groups.

Conclusions

Myofascial massage techniques significantly improved peripheral volume to the paraspinal region compared to traditional relaxation massage and a sham ultrasound control treatment. The findings of this study suggest that myofascial techniques may be a treatment of choice for those practitioners wishing to facilitate improvements in blood volume to the paraspinal region.

5.2 Introduction

The number of people utilising therapeutic massage to treat low back pain is increasing and massage therapy is becoming a mainstream treatment for multiple condition (Hunt *et al.*, 2010; Simmons, 2011). Despite this prevalence, study limitations and inconsistencies in the literature have made it difficult to make firm conclusions as to the physiological effects of massage. One of the most commonly referred to effects of massage is its ability to increase blood flow and many of its therapeutic effects have been attributed to changes in local blood flow, (Sefton *et al.*, 2010; Munk *et al.*, 2012). The benefits of local changes in circulation include improved circulation to damaged or painful tissues and greater removal of metabolic waste products (Sefton *et al.*, 2010). Massage is also thought to increase local tissue circulation, thereby reducing muscle tension and fatigue (Goats, 1994a). Mori *et al*., (2004), have suggested that massage application to the lumbar region has been shown to increase muscle blood volume and reduce muscle fatigue. Similarly, Durkin, Harvey, Hughson, & Callaghan, (2006), reported that lumbar massage has the potential to improve muscle blood flow and oxygenation. Massage therapy has also been shown to improve blood flow and skin temperature at the neck and shoulder region (Sefton *et al.*, 2010).

In a review of the effects of massage on blood flow (Weerapong, Hume and Kolt, 2005), nine controlled clinical trials used either classic whole body massage or techniques that can be classified as relaxation massage techniques (Sherman *et al.*, 2006). One study has utilised a combination of relaxation with clinical massage techniques such as frictions, muscle stretching and trigger point compressions (Sefton *et al.*, 2010), but to date no study has used myofascial massage techniques as an intervention. Myofascial massage technique is a manual therapy technique that is used to help reduce restrictive barriers or fibrous adhesions between the fascial layers (Barnes, 1997; Ercole *et al.*, 2010). Various methods and systems have been proposed including myofascial release (MFR) (Barnes,

1997), connective tissue massage (Holey, 2000), fascial manipulation (Picelli *et al.*, 2011) and fascial release (Earls and Myers, 2010). Generally speaking, these techniques employ low load and or long duration (90s-300s) stretching to the connective tissue or fascial structures (Barnes, 1997; Earls and Myers, 2010; Shah and Bhalara, 2012). MFR techniques have been described as either direct or indirect (Ajimsha, Daniel and Chithra, 2014) . Direct MFR techniques work directly into the fascial restrictions with the application of a few grams of force, with either knuckles fingers elbows, or other tools, to contact and stretch the restricted fascia. Indirect MFR involves the application of the same amount of force to the fascial restriction, but held for longer, where the traction is sustained but the stretch is not taken through the restriction barrier; the tissue is said to experience an unwinding (Shah and Bhalara, 2012; Ajimsha, Daniel and Chithra, 2014). Other techniques involve deeper friction manipulation (Ercole *et al.*, 2010), short and long finger stripping, pulling and lifting techniques that are designed to mobilise and or separate connective or fascial tissue (Goats and Keir, 1991; Earls and Myers, 2010).

It has been postulated that the fascial system can develop adhesions, restriction to movement and become inflamed due to injury and trauma (Barnes, 1997). Fascial restrictions have been said to create undue stress and tension on any structures that are enveloped or surrounded by the fascia (Ajimsha, Daniel and Chithra, 2014). Fascial restrictions and adhesions are said to be due to an increase in the viscosity of ground substance and or the development of abnormal cross links between collagen fibres (Shah and Bhalara, 2012). As described in the literature review, these structures include a network of neural and vascular tissue. Therefore, such fascial stresses may cause pain or ischemic conditions (Barnes, 1997). The proposed effects of MT techniques are to restore optimal length to the connective tissues, release restrictions within the fascial layers, release pressure on neuro-vascular structures, increase blood flow and reduce pain (Goats and Keir, 1991; Barnes, 1997; Ajimsha, Daniel and Chithra, 2014). Therefore, the aim of this study was to compare the acute effects of myofascial techniques on the

peripheral blood volume to the paraspinal myofascial region, compared to a traditional relaxation massage.

H_0: An acute bout of MT does not lead to improved blood volume at the paraspinal region compared to a RM treatment in healthy subjects

H_1: An acute bout of MT leads to improved blood volume at the paraspinal region compared to a RM treatment in healthy subjects.

5.2 Methods

5.2.1 Participants

Forty-four healthy participants (age: 22 ± 4 years, height: 176 ± 8 cm, mass: 73.5 ± 10.1, BMI: 23.5 ± 2.9) were drawn from the student population at the University of Kent and the local area (table 5.1). Participants were recruited using electronic mail, and posters located at the university campus. Ethical permission was obtained through the School of Sports and Exercise Sciences ethics committee. Subjects were excluded from this study if they were suffering from low back pain, diagnosed with serious infection in the preceding two weeks, previous severe back or leg injury, surgery on the back, spinal deformity, ankylosing spondylitis, rheumatoid arthritis; in any part of the body; any history of spinal fracture, a tumour in the back, an infection around the spine, any root compression or spinal disc damage, cancer, or any bleeding disorder. Other exclusion criteria included currently taking warfarin or similar blood thinning medication, taking corticosteroid medication, e.g. Prednisolone, or high doses of inhaled steroids.

Table 5. 1 Participant mean and standard deviation (SD) data for the massage technique (MT), relaxation massage (RM) and control groups.

Group	N (male/female)	Age (yrs)	Height (cm)	Weight (kg)	BMI
MT	17(M=8, F=9)	23 (5.01)	175.7 (8.15)	75.3 (14.82)	24.4 (3.38)
RM	17(m=11, F=6)	21 (1.16)	177.4 (8.02)	73.6 (10.08)	23.4 (2.77)
Control	10(m=5, F=5)	22 (2.88)	177.2 (9.7)	70.4 (10.5)	22.4 (2.04)

5.2.2 Study design

This was a pilot study using asymptomatic subjects. The study was a parallel designed, non-cross over randomised control trial pilot study designed to compare the effects of two types of massage (RM and MT) on blood flow to the lumbar paraspinal region. Recruitment of subjects was cumulative therefore randomisation was achieved through a simple random allocation. On arrival, subjects were asked to choose one of three envelopes, which concealed the allocation group. Subjects were allocated by drawing the next consecutive envelope. A member of the team; not involved in administering the interventions; conducted randomisation and assignment of subjects. Subjects were then allocated to that intervention. The independent variable consisted of the treatment condition (MT), RM and a sham ultrasound control condition). The dependent variables were the relative changes in haemoglobin and total blood volume at flow at the lumbar paraspinal region.

5.2.3 Measurements

Blood flow measurements

Blood volume and relative changes in haemoglobin was measured using Near Infrared Spectroscopy Technique (NIRS). A description of the blood volume and oxygenation measurements can be found in section 3.7.

5.2.4 Treatment Protocols

Integrated Myofascial techniques (MT)

A trained massage therapist with 3 years of practical experience conducted the MT treatments. A description of the MT treatment protocol can be found in section 3.8.3

Relaxation massage (RM)

In order to standardise the treatment each session was allocated a 30-minute time slot with specific areas of the body included and in particular time slots. All participants within the RM group received the same treatment and, in the order, described below.

Table 5. 2 Relaxation Massage technique (RM) treatment description

Client position	***Treatment Description***	***Repetitions / duration***
Supine: neck and shoulders	Effleurage (gliding) Petrissage (kneading, wringing, rolling) Petrissage Frictions (circular only) Tapotement (hacking, cupping, beating)	5 minutes
Prone position: Whole of back	Effleurage (gliding) Petrissage (kneading, wringing, rolling) Petrissage Frictions (circular only) Tapotement (hacking, cupping, beating)	15 minutes
Side lying position torso region	Effleurage (gliding) Petrissage (kneading, wringing, rolling) Petrissage Frictions (circular only) Tapotement (hacking, cupping, beating)	10 minutes each side

Sham Ultrasound

The Ultrasound treatment was conducted by a therapist trained in the use of electrotherapy. A description of the ultrasound measurements can be found in section 3.12.

5.2.5 Statistical analysis

Statistical analyses were performed in SPSS (IBM SPSS for Windows, version 21.0. Armonk, NY: IBM Corp). The normality assumption was assessed using Shapiro-Wilk tests. The dependent variables of Tsi O2Hb, HHb, tHb anfd HbDiff, were analysed using a one-way ANOVA comparing the change in values from pre to post measurements between each group. The independent variables were the treatment groups (MT, RM and sham TENS). Statistical significance level was set at alpha <0.05. Post hoc analysis was used to identify pairwise differences in blood flow and PPT between each group. Estimation of effect size from the pairwise comparisons was calculated using the pre-test, post-test-control design according to (Morris, 2008).

$$d = \frac{(\mathrm{M\ post, T} - \mathrm{M\ pre, T}) - (\mathrm{M\ post, c} - \mathrm{M\ pre, c})}{\mathrm{Pooled\ SDpre}}$$

Where M pre, and post T represent the pre-test and post-test means for the treatment group; M pre c and post c represent the pre-test and post-test means for the control group and SDpre represents the pooled standard deviation from the treatment and control groups. Effect sizes were considered as small (0.2), moderate (0.5) and large (0.8). As this was a pilot study statistical power was analysed using a G*Power programme (Faul *et al.*, 2007) to determine if the experimental results were statistically powerful at the 0.8 level and to facilitate sample size calculations for future studies within the thesis.

5.3 Results

Figure 5.1 below outlines participant flow including enrolment, participant allocation, randomisation and analysis.

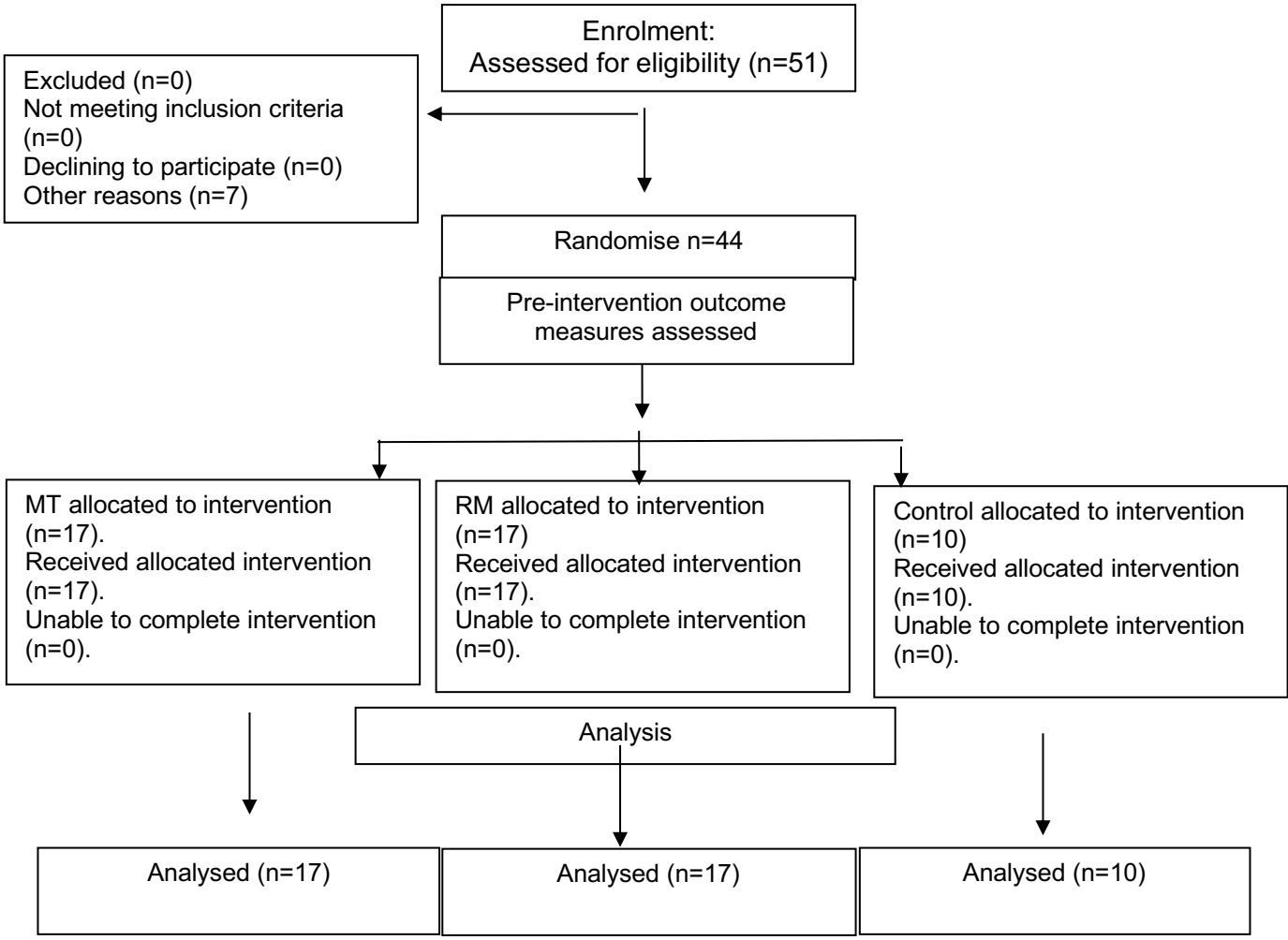

Figure 5. 1 Flow diagram depicting flow and allocation of participants from allocation to analysis.

Table 5.1 above reflects the mean and standard deviation figures for age, height, weight and BMI for each group. A one-way ANOVA revealed no significant difference in the above variables between each group. Table 5.4 below represents the mean and SD's for the change in blood flow from pre, to post treatment for each group. The results and

statistical analysis of this study had to be altered due to technical issues with the Oxysoft NIRS equipment which meant that the Tsi data was not able to be used.

There was a statistically significant difference between groups for O2Hb, however the assumption of homogeneity was violated; therefore, the Welch F-ratio is reported, F (2-26.44) = 15.82, p = < .001. There was a statistically significant difference between groups for tHb, however the assumption of homogeneity was violated; therefore, the Welch F-ratio is reported, F (2-26.71) = 15.47, p = < .001. There was also a significant difference between groups for HHb; F (2-41) = 3.59, p = .037 and Hb Diff; F (2-41) = 5.08, P = .011.

In order to compare the between groups effects for the MT and RM groups a post hoc analysis revealed that there was a significantly greater increase in O2Hb for the MT group compared to the RM group and control (p <0.001). Post hoc tests also revealed significantly greater increases in HHb (p = .032) and tHb (p = <.001) for the MT group compared to the RM group. There was no statistically significant change in HbDiff between the MT and RM group (p = .072)

In order to compare the between groups effects for the MT and control groups a post hoc analysis revealed significantly greater increases in tHb (p = <0.001) and O2Hb (p = <0.001) for the MT group compared to the control group. However, there was no significant increase in HbDiff (p = .014) of HHb (p = .451) between groups.

In order to compare the between groups effects for the RM and control groups a post hoc analysis revealed no significant difference in the change in O2Hb (p = 0.897), HHb (p = 0.707), tHb (p = 0.100) or HbDiff (P = .706) between the RM and control groups.

Table 5. 3 Mean and SD values for the change in blood flow variables for each group, results of the comparison between groups for Oxygenated haemoglobin (O_2Hb), De-oxygenated haemoglobin (HHb), Total haemoglobin (tHb), Hb difference (HbDiff), effect sizes and post hoc observed power calculations.

Group	MT	RM	Control	MT vs RM				MT vs control				RM vs Control			
				Difference (95%CI)	**P-value**	**Effect size**	**Observed Power**	**Difference (95%CI)**	**P-value**	**Effect size**	**Observed Power**	**Difference (95%CI)**	**P-value**	**Effect size**	**Observed Power**
Change in O2Hb (Mean ±SD)	19.57 (12.15)	3.95 (9.37)	1.55 (3.64)	15.62 (7.32 to 23.95)	< .001	1.41	0.97	18.02 (8.46 to 27.58)	< .001	1.12	0.80	-2.40 (-11.96 to 7.16)	.897	0.13	0.06
Change in HHb (Mean ±SD)	3.01 (3.49)	-2,65 (9.22)	-0.29 (0.99)	5.66 (0.391 to 10.92)	.032	0.51	0.25	3.30 (-2.77 to 9.36)	.451	0.20	0.34	2.36 (-3.78 to 8.42)	.707	0.13	0.06
Change in tHb (Mean ±SD)	22.58 (15.02)	1.31 (7.00)	1.26 (4.02)	21.27 (12.30 to 30.24)	< .001	1.92	0.99	21.32 (10.99 to 31.65)	< .001	1.32	0.91	-0.46 (-10.38 to 10.29)	1.00	0.002	0.005
Change in HbDiff (Mean ±SD)	16.56 (9.70)	6.60 (17.22)	1.84 (3.51)	9.96 (-6.57 to 20.58)	.072	0.89	0.71	14.72 (2.49 to 26.95)	.014	0.91	0.62	4.76 (-16.99 to 7.47)	.706	0.26	0.09

RM = relaxation massage

MT = Integrated Myofascial techniques

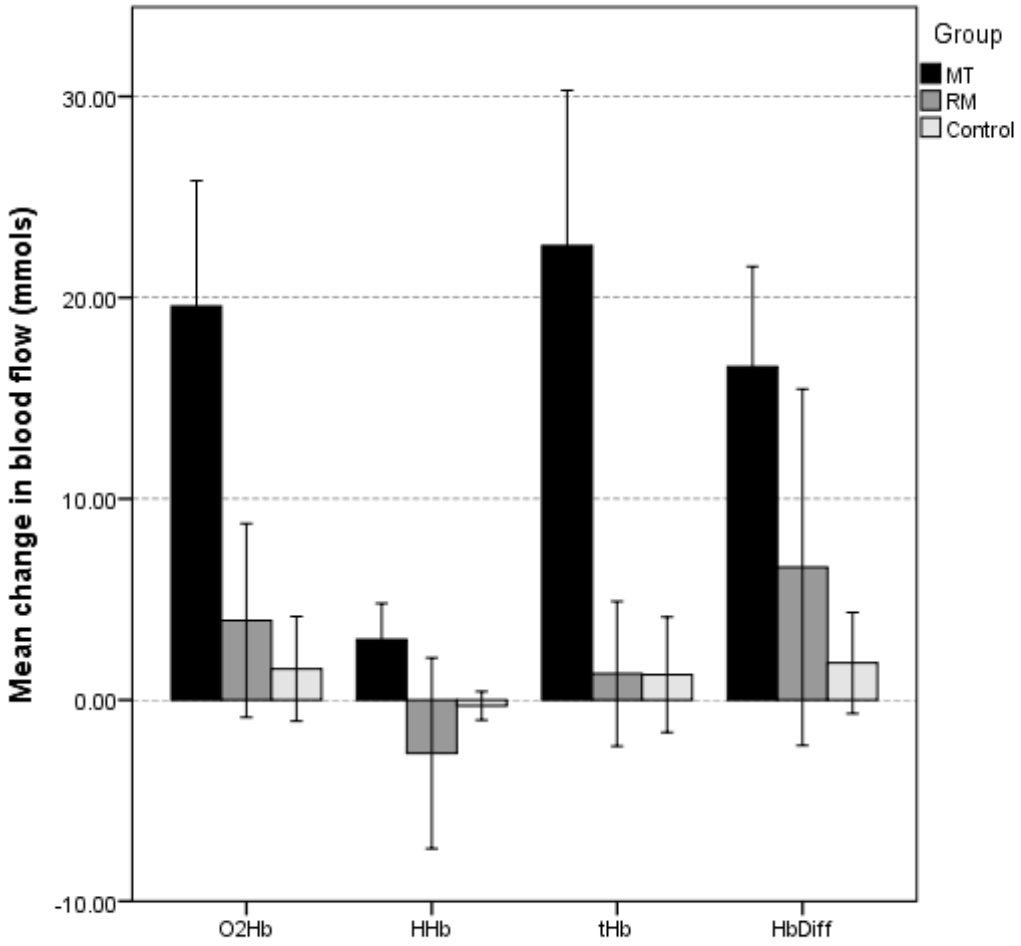

Figure 5. 2 Bar chart representing mean change in blood haemoglobin variables oxygenated haemoglobin (O_2Hb), De-oxygenated haemoglobin (HHb), total haemoglobin (tHb) and Hb difference (HbDiff) for each group (MT = Integrated myofascial techniques, RM = relaxation massage, control = sham ultrasound)

5.4 Discussion

The main findings from this study are that the MT techniques led to significantly greater improvements in tHb, O2Hb and HHb to the paraspinal region compared to relaxation massage and the control method, sham ultrasound. Although the blood volume changes for relaxation massage and the control intervention showed some increases there was no statistical difference between these groups for any of the blood volume variables, suggesting that relaxation massage, in this study, does not alter local blood volume at the paraspinal region. This finding is in agreement with previous authors who have suggested that massage does not alter blood flow (Shoemaker, Tiidus and Mader, 1997; Martin *et al.*, 1998; Hinds *et al.*, 2004; Wiltshire *et al.*, 2010), but not in agreement with some studies that have recorded improvements in blood flow following massage (Mori *et al.*, 2004; Cambron, Dexheimer and Coe, 2006; Sefton *et al.*, 2010; Taspinar *et al.*, 2013).

Proponents of the theory that massage increases blood flow suggest that this is achieved through either a reflex hyperaemia (Plakornkul *et al.*, 2016), an increase in tissue temperature (Portillo-Soto *et al.*, 2014) and a vasodilatory action mediated through reduced sympathetic tone (Wiltshire *et al.*, 2010). However, the inconsistencies within the literature make it difficult to be conclusive about these effects. One of these inconsistencies is the general lack of homogeneity in the massage techniques employed and the time frames of the treatments. Studies addressing blood flow have often used combinations of massage styles such as relaxation and clinical massage strokes (Cambron, Dexheimer and Coe, 2006; Sefton *et al.*, 2010), and or timeframes that may be unrealistic to the use of massage in a clinical setting (Shoemaker, Tiidus and Mader, 1997; Hinds *et al.*, 2004; Wiltshire *et al.*, 2010). Furthermore, as outlined in the thesis introduction, the range of blood flow assessment techniques within the methodologies also contributes to the equivocal findings. Many techniques involve measurements of arterial blood flow using Doppler ultrasound (Shoemaker, Tiidus and Mader, 1997; Hinds

et al., 2004; Wiltshire *et al.*, 2010), which may not be sensitive enough to measure small changes or peripheral blood flow alterations that massage would most likely achieve (Sefton *et al.*, 2011; Munk *et al.*, 2012). However; in the case of those studies that have reported improvements in blood flow; it is possible that these methods have contributed to an overestimation of blood flow through the muscle (Martin *et al.*, 1998). Furthermore, some studies reporting improvements in blood flow have used indirect methods such skin temperature (Portillo-Soto *et al.*, 2014) and infrared thermography (Sefton *et al.*, 2010), which require an interpretation of blood flow as opposed to a direct measurement.

The results of the effects of blood volume following RM in this study are in stark contrast to the effects on blood volume following MT techniques that target the connective tissue structures. The significant improvement in tHb following the MT intervention represents a 178% difference between that and the RM value. The tHb values reflects the change in the sum of O_2HB and HHb, both of which significantly improved following the MT intervention. To our knowledge this is the first study to compare blood volume changes at the paraspinal region using MT techniques and NIRS technique. Previous studies have identified improvements in blood volume at the paraspinal region following exercise (Olivier *et al.*, 2013), traditional RM type massage therapy (Mori *et al.*, 2004); or using MT techniques at different musculature measured through venous pressure changes (Ramos-González *et al.*, 2012). Interestingly, one relaxation massage study found that improvements in blood volume were not as great as those from a comparative vibrational technique (Taspinar *et al.*, 2013). The authors postulate that the reason for this may have something to do with this technique being similar to connective tissue massage. It is therefore possible that techniques directed at the connective tissues and, in this case, fascia of the lower back are capable of eliciting improvements in blood volume due to the structure and function of this tissue and the nature of its response to mechanical stimulation (Barnes, 1997). The current study found no significant difference for the HbDiff value between the massage RM and MT groups. The Hbdiff is derived from subtracting

HHB form O_2Hb and is sometimes referred to as a surrogate measure of muscle tissue oxygenation (Janssens *et al.*, 2013). As the study protocol did not require back muscle activity it is assumed that changes in tissue oxygenation would not be observed.

The effects of MT on blood volume appear to be related to alterations in the ground substance of fascia or the connective tissues (Barnes, 1997; A. Stecco *et al.*, 2015), and or a neurophysiological response that facilitates the above but also contributes to localised vasodilation (Goats and Keir, 1991; Schleip, 2003a; Ajimsha, Daniel and Chithra, 2014). The low load, long duration type stretch associated with myofascial release techniques is said to contribute to the viscoelastic deformation of the connective tissue and therefore elongation of shortened or restricted fascia (Barnes, 1997). Furthermore, mechanical pressure applied to the tissue is said to change the ground substance form a viscous to fluid state through thixotropic or piezoelectric events (Barnes, 1997; Schleip, 2003b). There is some evidence to suggest that heat and pressure to the connective tissue is effective in affecting fascial fibroses through thixotropic mechanisms (Ercole *et al.*, 2010). While others report that local effects of MT include the release of histamine and a subsequent vasodilatory response (Goats and Keir, 1991) . However, more recently the effects of MT on blood flow have been attributed to the stimulation of fascial mechanoreceptors that influence autonomic function. Schleip (2003a), proposed that stimulation of Interstitial and Ruffini receptors alter the local pressure in local arterioles and capillaries, through lowering of sympathetic activity, affecting either local blood supply and or the viscosity of the extracellular matrix. Previous evidence suggest that increases in sympathetic tone can lead to vasoconstriction (Thomas and Segal, 2004), while increases in parasympathetic activity have a vasodilatory effect (Goats and Keir, 1991). Furthermore to this it has been shown that the application of MT techniques is capable of resulting in an increase in parasympathetic activity (Simmonds, Miller and Gemmell, 2012). The lack of significant difference between RM and the control group within the current study suggest that the effect of touch or heat transfer from the hands of the

therapist during the RM intervention cannot be attributed to the change in blood volume. Therefore, in light of this, and the above discussion, it is possible to suggest that the specific nature of MT that directly focus on fascia and connective tissues are able to stimulate mechanisms associated with increased blood volume compared to relaxation massage techniques that do not; suggesting a technique specific response.

This study utilised NIRS technique to measure haemodynamic changes within the paraspinal myofascial region. Previous challenges with regard to blood volume measurements in massage therapy research is that of using a method that does not interfere with the treatment, that can measure local changes in muscle blood volume, and that directly measures blood volume (Munk *et al.*, 2012) . NIRS provides a non-invasive, continuous and direct method to determine blood volume changes within the muscle including absolute changes in O_2HB HHb and the sum of which represents the total amount of haemoglobin in the tissue (tHb) (Ferrari and Mottola, 2004). NIRS, therefore, is capable of detecting small changes in muscle blood volume and relative changes in haemoglobin in real time. Furthermore, the effects of manual therapies on blood volume that are directed at skeletal muscle are more likely to affect peripheral blood volume as opposed to venous or arterial measurements (Sefton *et al.*, 2010) . Therefore, peripheral blood volume measurement techniques such as NIRS may be a more appropriate method of measuring blood volume in manual therapy research. Measuring changes in blood volume at skeletal muscles may have implications for measuring recovery from exercise (Wiltshire *et al.*, 2010), healing of soft tissue injuries (Portillo-Soto *et al.*, 2014) and discriminating between normal and pathological states (Sakai *et al.*, 2012), and further investigations using NIRS could be used to measure a variety of clinical conditions (Munk *et al.*, 2012). Therefore, the following chapters will use NIRS technology to assess blood volume in the a-symptomatic and LBP populations and comparing different interventions.

5.4.1 Study Limitations

Sources of bias within this study are discussed more generally in section 9.1. The penetration depth of near-infrared light is roughly half the distance between the light source and the detector, adipose tissue thickness (ATT) can be a confounding factor in the measurement of blood volume using NIRS (van Beekvelt *et al.*, 2001). The current study did not consider the ATT of subjects within each group. BMI assessment revealed no significant difference between groups but the adiposity of the region at the L3 should be taken into consideration for future studies. The number of subjects in the control group (n=10) was lower than those in the RM and MT groups (n = 17) due to recruitment issues. However, a post hoc analysis of observed power for the change in total blood volume (tHb) was 0.99. Based on the average post hoc effect sizes between the MT and MR group's results for all blood volume variables, a post hoc power analysis revealed a total sample of 36 would be required for statistical power at the recommended 0.8 level (Faul *et al.*, 2007). The current study had a total sample size of 44 across three groups. Due to technical issues with the Oxysoft Mk III Near Infrared Spectroscopy System, (Artinis Medical Systems ®Arnhem), the tissue oxygen saturation profiles of the subjects could not be measured. Although a useful measure to have recorded, the aim of the study was to evaluate the effects of MT on blood volume to the desired muscle as opposed to muscle tissue oxygenation. Finally, the sham ultrasound group may have experienced an initial reduction in temperature at the paraspinal region due to the application of the ultrasound gel, which may have some effect on the relative change in blood volume and blood volume. For the purpose of future studies within this thesis, sham TENS was used instead of sham ultrasound in order to offset the possible confounding effects of temperature change due to the ultrasound gel application.

5.4.2 Conclusions

Myofascial massage techniques significantly improved peripheral volume to the paraspinal region compared to traditional relaxation massage and a sham ultrasound control treatment. The effect of massage on blood volume is equivocal and the mechanisms are poorly understood. The findings of this study suggest that myofascial techniques have a greater influence on peripheral blood volume than relaxation massage and may be a treatment of choice for those practitioners wishing to facilitate improvements in blood volume to the paraspinal region.

6 The acute effects of integrated myofascial techniques on lumbar paraspinal blood volume compared with kinesiotaping: A pilot study

The research presented in this chapter has been published in Journal of Bodywork and Movement Therapies, Volume 21, Issue 2, April 2017, Pages 459-467.

6.1 Abstract

Background

Integrated myofascial massage techniques (MT) and Kinesio Taping (KT) are therapeutic interventions used to treat low back pain. However, limited research has been conducted into the underlying physiological effects of these types of treatments.

Objectives

The purpose of this study was to compare the acute effects MT and KT on blood flow at the lumbar paraspinal musculature.

Methods

Forty-four healthy participants (18 male and 26 female) (age, 26 ± SD 7) volunteered for this study and were randomly assigned to one of three interventions, MT, KT or a control group (Sham TENS). Paraspinal blood flow was measured at the L3 vertebral level, using Near Infrared Spectroscopy (NIRS), before and after a 30-minute treatment. Pain Pressure Threshold (PPT) was also measured before and after treatments.

Results

A one-way ANOVA indicated a significant difference between groups for O_2Hb [F (2-41) = 41.6, P<0.001], HHb [F (2-41) = 14.6, P<0.001] and tHb [F (2-41) = 42.2, P <0.001]. Post hoc tests indicated that MT was significantly greater, from the KT and the control treatments (P<0.001), for changes in O_2Hb, HHb, and tHb. There were no significant differences for PPT [F (2-41) = 2.69, p = 0.08], between groups.

Conclusions

This study demonstrated that MT increases peripheral blood flow at the paraspinal muscles in healthy participants compared to KT and sham TENS. The change in blood flow had no impact on pain perception in the asymptomatic population group.

6.2 Introduction

Impaired blood flow and greater fatigability of the paraspinal muscles have been identified as possible mechanisms associated with LBP (Mori *et al.*, 2004). Previous studies have suggested that LBP subjects exhibit higher muscular loads, increased intramuscular hypoxia and a limited capacity for the paraspinal muscles to consume oxygen (Kovacs *et al.*, 2001; Sakai *et al.*, 2012). Decreases in blood flow to the lumbar paraspinal region have also been associated with detrimental adaptations to proprioception (Thomas and Segal, 2004) and lumbosacral position sense (Brumagne *et al.*, 2013).

Two interventions that have been proposed to improve blood flow are massage therapy and kinesio taping (KT) (Mori *et al.*, 2004; Hagen, Jeffrey J Hebert, *et al.*, 2015). The effects of massage on blood flow are equivocal and may be due to inconsistencies in the research such as small sample sizes, lack of control groups (Weerapong, Hume and Kolt, 2005) and measurement limitations that make real time measurements of blood flow in massage problematic (Munk *et al.*, 2012). However, these studies refer to more traditional forms of massage and did not use NIRS technology, which provides a non-invasive, dynamic measurement of blood flow to the muscle tissues (Munk *et al.*, 2012)

Myofascial techniques are a form of manual therapy that involves focal soft tissue work to fascia and connective tissues and is widely employed to reduce pain and improve physiological functions (Ajimsha, Daniel and Chithra, 2014). For clinical purposes a variety of fascial techniques can be integrated to manipulate and stretch the myofascial or connective tissue layers to achieve various focussed therapeutic goals (Sherman *et al.*, 2006). These include restoring optimal tissue length, reduce pain, improve tissue circulation and improve function (Barnes, 1997; Myers, 2009b; Ajimsha, Al-Mudahka and Al-Madzhar, 2015; Celenay, Kaya and Akbayrak, 2016). It has been postulated that,

following injury or a lack of movement, fascia can form adhesions and abnormal cross-links rendering the fascia less pliable and resistant to movement (Bouffard *et al.*, 2007). Repeated bouts of overuse or extended periods inactivity may therefore contribute to the pathophysiology of back pain (Langevin and Sherman, 2007). It has also been proposed that myofascial techniques can influence the ground substance of the connective tissues and mechanoreceptors within fascia, contributing to changes in local fluid dynamics, reducing excessive muscle tension, capillary constriction, and improve local blood flow (Schleip, 2003a). Although these studies were not specifically related to LBP it suggests that myofascial techniques may have a role to play in improving blood flow in LBP patients.

Kinesio taping is a popular intervention choice in the treatment of low back pain (Álvarez-Álvarez *et al.*, 2014). It is proposed that the application of Kinesio taping to a stretched muscle creates convolutions to the skin (Kase K, Wallis J, 2003). These convolutions are believed to lift the skin and underlying fascia, creating room for increased blood and lymphatic flow (Kase K, Wallis J, 2003), reducing pressure on subcutaneous nociceptors, and subsequently reducing pain (Parreira *et al.*, 2014). Studies on the effects of KT on blood flow are also limited and show conflicting results (Stedge, Kroskie and Docherty, 2012; Williams *et al.*, 2012), however, the ability to affect muscle endurance and improve fatigue has been identified and may be effective in the management of LBP (Hagen, Jeffrey J Hebert, *et al.*, 2015).

Currently, it is not known whether KT or myofascial techniques can improve blood flow, nor have these techniques been compared directly. Therefore, the aim of the present study is to determine whether KT or integrated myofascial techniques (MT) have the potential to increase blood volume and relative changes in haemoglobin at the paraspinal

region. The acute effects of KT and MT were determined in a healthy population to determine the efficacy of both treatments compared with a sham treatment.

H_0: An acute bout of MT does not lead to improved blood volume and pain pressure threshold scores at the paraspinal region compared to a KT treatment in healthy subjects

H_1: An acute bout of MT leads to improved blood volume and pain pressure threshold scores at the paraspinal region compared to a KT treatment in healthy subjects

6.3 Methods

6.3.1 Participants

Participants were drawn from the student population at the University of Kent and the local area (table 6.1). An a-priory analysis for sample size N was calculated using G* Power 3.1 (Faul et.al., 2007) as a function of the required power level set at 80% and a pre-specified significance level of 0.05. An overall sample size of 42 was calculated. Participants were recruited using electronic mail, and posters located at the university campus. Ethical permission was obtained through the School of Sports and Exercise Sciences ethics committee. Subjects were excluded from this study if they were suffering from low back pain, diagnosed with serious infection in the preceding two weeks, previous severe back or leg injury, surgery on the back, spinal deformity, ankylosing spondylitis, rheumatoid arthritis; in any part of the body; any history of spinal fracture, a tumour in the back, an infection around the spine, any root compression or spinal disc damage, cancer, or any bleeding disorder. Other exclusion criteria included currently taking warfarin or similar blood thinning medication, taking corticosteroid medication, e.g. Prednisolone, or high doses of inhaled steroids. None of the participants were excluded under the exclusion criteria above. One subject was unable to attend due to personal reasons (see figure 3). All testing and data collection was conducted at the University of Kent's Sports Ready Clinic.

Table 6. 1 Participants characteristics by group (mean ±SD)

Group	N (male/female)	Age (yrs)	Height (cms)	Weight (kg)	BMI
MT	15(M=8, F=7)	28.3 (7.6)	172 (11.5)	70.6 (10.3)	24.0 (3.7)
KT	15(m=6, F=9)	25.4 (8.8)	170 (7.6)	70.2 (13.3)	24.3 (3.6)
Control	14(m=4, F=10)	23.5 (6.6)	170 (7.6)	67.1 (10.4)	23.9 (3.2)

6.3.2 Study design

This was a pilot study using asymptomatic subjects. The study was a parallel designed, non-cross over randomised control trial pilot study designed to compare MT with Kinesio Tape as possible interventions for patients with LBP. Participants were randomly assigned into a 30-minute treatment of either KT, MT or sham Transcutaneous Electrical Nerve Stimulation (TENS) control group. Randomisation was achieved through simple allocation using a random allocation. Recruitment of subjects was cumulative. On arrival, subjects were asked to choose one of three envelopes, which concealed the allocation group. Subjects were allocated by drawing the next consecutive envelope. A member of the team; not involved in administering the interventions; conducted randomisation and assignment of subjects. Subjects were then allocated to that intervention. The independent variable consisted of the treatment condition (MT, KT and a sham TENS control condition). The dependent variables were the change in peripheral blood flow, relative changes in haemoglobin and pain pressure threshold values at the lumbar paraspinal region.

6.3.3 Measurements

Blood volume measurements

Blood volume was measured using Near Infrared Spectroscopy Technique (NIRS). A description of the blood volume and oxygenation measurements can be found in section 3.7.

Skinfold measurements

A skinfold measurement of adipose tissue was obtained on all participants. A description of the skinfold measurement protocol can be found in section 3.14.

Pain pressure threshold measurements (PPT)

A description of the measurements to identify the PPT of participants within this study can be found in section 3.13.

6.3.4 Treatment Protocols

Integrated myofascial techniques (MT)

A trained massage therapist with 3 years of practical experience conducted the MT treatments. A description of the MT treatment protocol can be found in section 3.8.3

Kinesio taping condition

A therapist trained in the application of this taping technique conducted the KT treatment. The therapist had 2 years' experience of using Kinesio Tape in practice. The KT method was a standardised from a technique by Kase et al., (2003). Two "I"- Shaped Kinesio Rock Tape® elastic bandage was attached directly to the patients' skin over the erector spinae parallel to the spinous processes of the lumbar vertebrae (figure 6.2). A standardised reference point from the posterior superior iliac spine (PSIS) to the thoracic eight vertebrae (T8) was implemented according to the Kenzo Kase's Kinesio Taping Method Manual, (1996). The skin was cleaned and shaved (if required) in preparation for the tape. The tape was anchored (approximately five centimetres) at the base of the KT strip to the posterior superior iliac spine with no tension. The therapist removed the paper backing from the base of the 'I' strip leaving the remainder of the paper backing on the "I" strip. Care was taken not to handle the adhesive side of the tape. The clients were asked to flex the lumbar spine by leaning forward from the hip therefore placing the erector spinae muscles in a lengthened position. Two vertical 'I' strips were equally applied

upwards on either side of the spine over the skin area with a light tension of ten to fifteen percent stretch. The application was completed when the proximal base of the KT was placed approximately five centimetres above the vertebra T8 with no tension. The therapist activated the adhesive glue by rubbing the tape onto the skin using the paper backing from the tape. The KT treatment lasted for 30 minutes before the tape was removed for retesting.

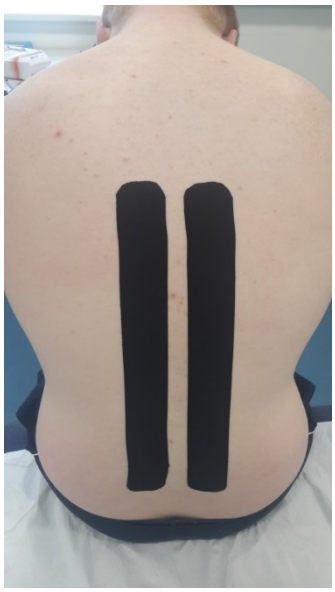

Figure 6. 1: Kinesiotape method tape placement

Sham TENS

A description of the sham TENS treatment protocol can be found in section 3.11.

6.3.5 Statistical Analysis

Statistical analyses were performed in SPSS (IBM SPSS for Windows, version 21.0. Armonk, NY: IBM Corp). The normality assumption was assessed using Shapiro-Wilk tests. Skinfold thickness and BMI values were measured to determine any differences in body composition between groups. The dependent variables of Tsi, O2Hb, HHb, tHb were obtained by averaging the values over the two-minute measurement period. The dependent variables of Tsi, O2Hb, HHb, tHb, and ratings of pain pressure threshold were

analysed using a one-way ANOVA comparing the change in values from pre to post measurements between each group. The independent variables were the treatment groups (MT, KT and sham TENS). Statistical significance level was set at alpha<0.05. Post hoc analysis was used to identify pairwise differences in blood volume and PPT between each group. Estimation of effect size from the pre-test, post-test-control design was calculated according to (Morris, 2008).

$$d = \frac{(\mathrm{M\ post, T} - \mathrm{M\ pre, T}) - (\mathrm{M\ post, c} - \mathrm{M\ pre, c})}{\mathrm{Pooled\ SDpre}}$$

Where M pre and post T represent the pre-test and post-test means for the treatment group; M pre and post c represent the pre-test and post-test means for the control group and SDpre represents the pooled standard deviation from the treatment and control groups. Effect sizes were considered as small (0.2), moderate (0.5) and large (0.8).

6.4 Results

Figure 6.4 below outlines the participant flow including, enrolment, participant allocation, randomisation and analysis. One participant was unable to take part in the study due to personal reasons.

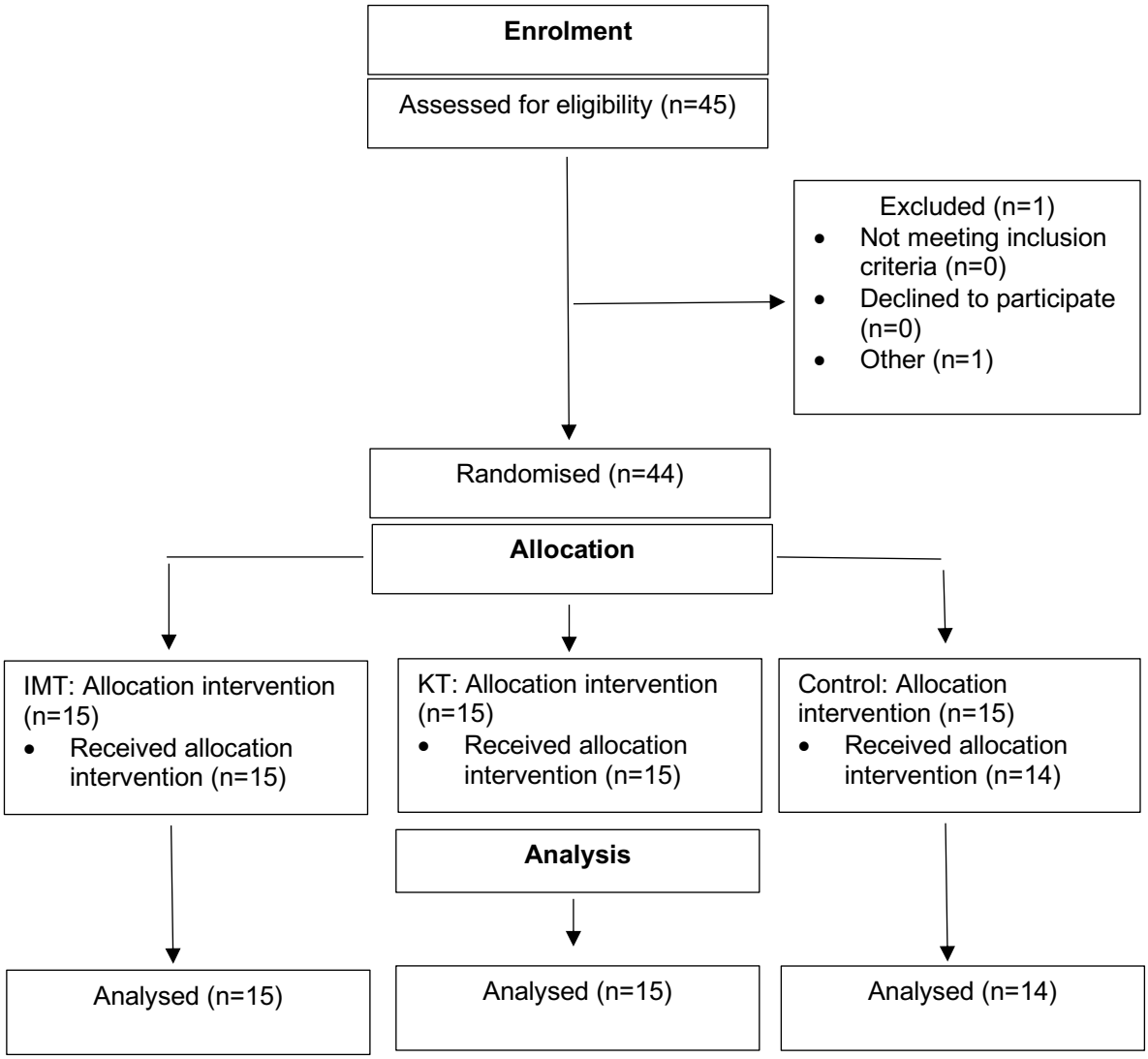

Figure 6. 2 Flow diagram depicting flow and numbers of participants from allocation to analysis

6.4.1 Tissue Oxygenation and blood volume

Table 6.3 represents the mean and SD values for the change in tissue oxygenation and blood volume between groups. Results of a one-way ANOVA revealed statistically significant differences between groups for the change in O2Hb [$F_{(2\text{-}41)}$ =41.6, P= <0.001], HHb [$F_{(2\text{-}41)}$ = 14.6, P= <0.001] and tHb [$F_{(2\text{-}41)}$ = 42.2, P= <0.001]. Post hoc analysis revealed that significantly greater changes in O2Hb for the MT group compared to the KT group and control (p <0.001). There was no significant difference in the change in O2Hb between the KT group and control (p = 0.789). Post hoc tests also revealed significantly

greater changes in HHb and tHb for the MT group compared to the KT group and the control group ($p = <0.001$). There was no significant difference between changes in HHb ($p = 0.881$) and tHb ($p = 0.769$) between the KT and control group (figure 6.3 and 6.4).

Table 6. 2 Mean and SD values for the change in blood volume variables for each group, results of the comparison between groups for Oxygenated haemoglobin (O_2Hb), De-oxygenated haemoglobin (HHb), Total haemoglobin (tHb), Tissue oxygen saturation (TSi) and effect sizes.

Group	MT	KT	Control	MT vs KT			MT vs control			KT vs Control		
				Difference (95%CI)	**P-value**	**Effect size**	**Difference (95%CI)**	**P-value**	**Effect size**	**Difference (95%CI)**	**P-value**	**Effect size**
Change in O2Hb (Mean ±SD)	22.34 (9.52)	1.87 (3.66)	1.16 (7.10)	20.46 (15.17 to 25.76)	< .001	1.73	21.18 (15.79 to 26.57)	< .001	0.85	0.717 (-4.67 to 6.11)	.799	0.03
Change in HHb (Mean ±SD)	3.42 (3.30)	-1.02 (1.62)	-1.25 (2.75)	4.44 (2.48 to 6.39)	< .001	0.39	4.67 (6.66 to 2.68)	< .001	0.19	0.24 (-1.75 to 2.22)	.811	0.003
Change in tHB (Mean ±SD)	25.76 (10.88)	0.86 (4.13)	-0.09 (9.60)	24.9 (18.49 to 31.31)	< .001	2.14	25.85 (19.33 to 32.37)	< .001	1.05	0.954 (-5.57 to 7.47)	.769	0.03
Change in Tsi (Mean ±SD)	2.81 (7.01)	0.31 (8.61)	1.32 (9.23)	2.50 (-3.6 to 8.65)	.416	0.28	1.50 (-4.76 to 7.75)	.632	0.07	-1.01 (-7.26 to 5.25)	.747	-0.07

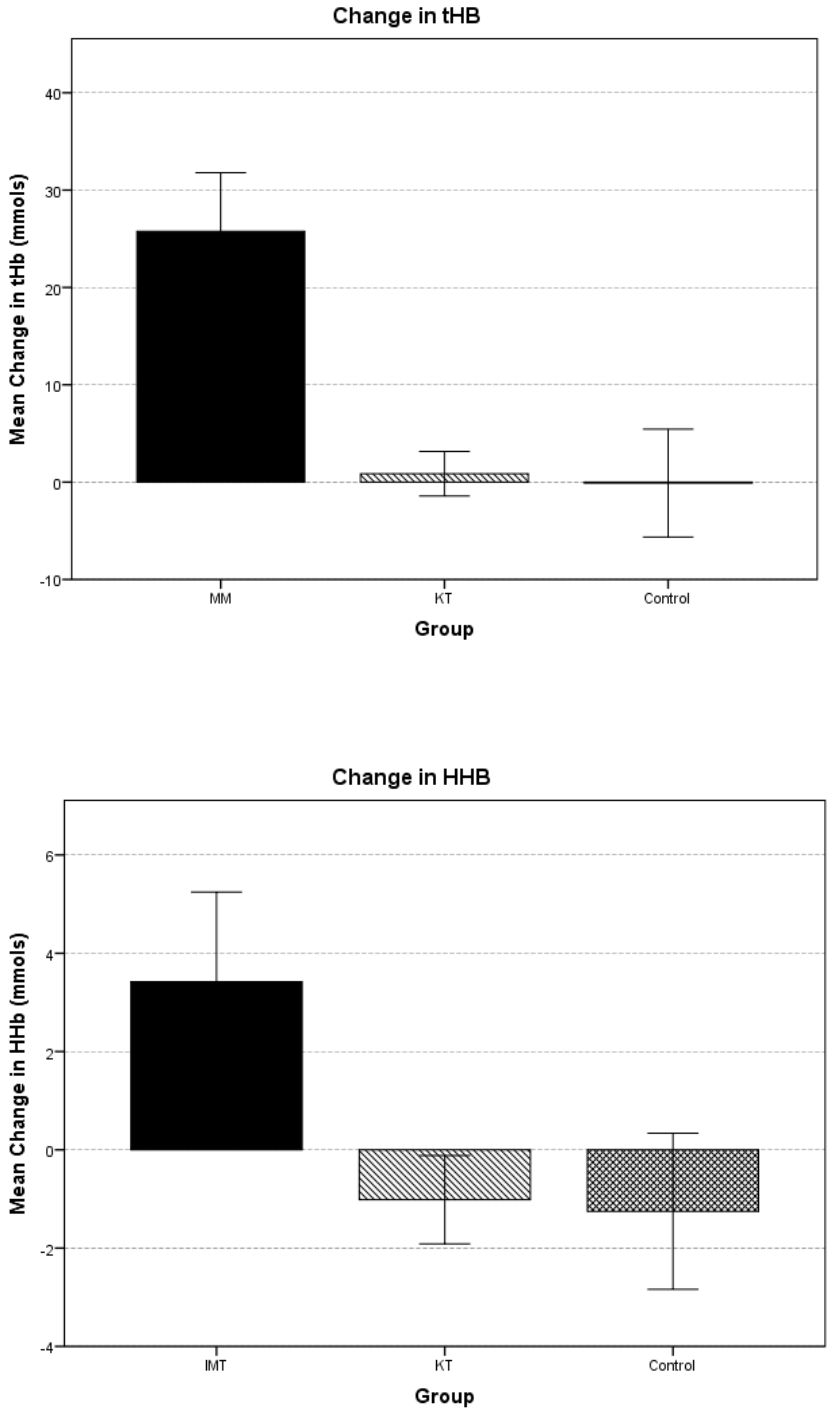

Figure 6. 3 Comparison of the change in blood volume variables between each group for total haemoglobin (tHb) and deoxyhaemoglobin (HHb). (MM = Myofascial Massage KT = Kinesio Tape)

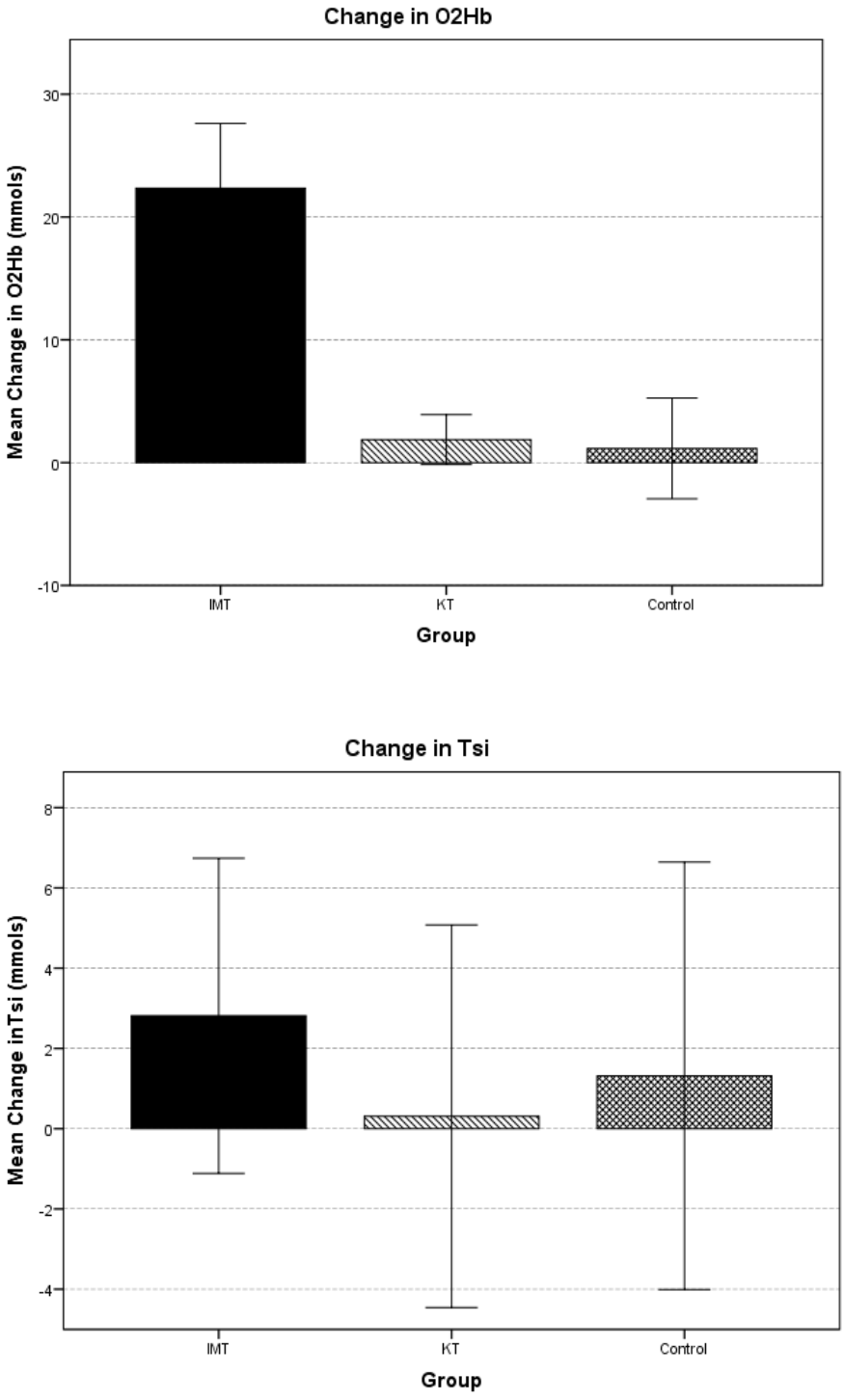

Figure 6. 4 Comparison of the change in blood volume variables between each group for oxyhaemoglobin (O2Hb) and tissue saturation index (Tsi). (MM = Myofascial Massage KT = Kinesio Tape)

6.4.2 Body Fat and Body Mass Index

Descriptive data analysis revealed that skinfold thicknesses were not normally distributed, however BMI was. A one-way ANOVA revealed no significant difference for BMI [$F_{(2-41)}$ =.049 P = 0.953] between groups. A Kruskal-Wallis Test revealed no difference in skinfold measurement between groups, [$H_{(2)}$ = 4.3, p = 0.59].

6.4.3 Pain Pressure Threshold

A one-way ANOVA revealed no significant difference between groups for PPT changes, [$F_{(2-41)}$ = 2.69, p = 0.80].

6.5 Discussion

The results of this study indicate that, in healthy participants, a 30-minute treatment using MT techniques produced significantly greater increases in peripheral blood volume at the lumbar paraspinal region compared to KT and a control group. However, changes in PPT and Tsi did not differ between groups.

The present study is the first to compare changes in peripheral blood volume following manual MT therapy and KT using NIRS at the paraspinal region. Previous studies into massage and blood flow have been largely inconclusive due to the inconsistencies surrounding Xenon wash-out technique and the inability of Pulsed Doppler ultrasound to measure microcirculation within the muscle (Weerapong, Hume and Kolt, 2005; Munk *et al.*, 2012) . Previous studies have assessed the effects of massage on changes in blood flow using Doppler (Shoemaker, Tiidus and Mader, 1997; Hinds *et al.*, 2004; Taspinar *et al.*, 2013), Dynamic Infrared Thermography (Sefton *et al.*, 2010) and NIRS techniques (Mori *et al.*, 2004; Durkin *et al.*, 2006). Previous studies have both supported (Mori *et al.*, 2004; Sefton *et al.*, 2010); and not supported (Shoemaker, Tiidus and Mader, 1997) the premise that massage has the potential to improve blood volume and oxygenation. The current study utilised NIRS technique to measure blood volume. NIRS is a non-invasive

technique that provides dynamic measures of blood volume and tissue oxygenation and has been widely used to monitor these parameters within muscle (Ferrari and Mottola, 2004; Durkin *et al.*, 2006; Munk *et al.*, 2012; Sakai *et al.*, 2012).

The major effects of MT found in this study were markedly higher levels of O2Hb, HHb and tHb. As NIRS provides continuous monitoring of both oxygenated and deoxygenated haemoglobin and myoglobin and the sum of O2Hb and HHb are considered to represent the change in blood volume (Olivier *et al.*, 2013), It is reasonable to assume that the results of this study indicate a change in total blood volume at the lumbar paraspinal region following MT. One possible explanation for the increase in blood volume may be due to the restoration of normal tissue structure and function following MT. It has been argued that fascial restrictions within the connective tissue network may result in stress on muscle tissues and pain sensitive structures that are enveloped, supported or divided by these connective tissue (Ajimsha, Daniel and Chithra, 2014). It has been suggested that these restrictions may result from alterations in ground substance (Hanten and Chandler, 1994) and the presence of adhesions due to injury, stress and repetitive use (LeBauer, Brtalik and Stowe, 2008). It is proposed that MT techniques are able to alter the ground substance (Ercole *et al.*, 2010), and muscle architecture through thixotropy and tensegrity (Arroyo-Morales, Olea, Martínez, *et al.*, 2008), restoring extensibility between fascial layers, relieving pressure on structures such as nerves and blood vessels and, therefore, restoring normal neural and vascular dynamics to the region.

Another possible theory is that MT techniques may lead to an increase in intramuscular blood volume through stimulation of the autonomic nervous system. It is reported that the lumbar paraspinal muscles in a state of contracture of around 20-30% of MVC result in a significant decrease in oxygenation (Jensen *et al.*, 1999). MT techniques may have

stimulated mechanoreceptors within the fascial network resulting in an autonomic nervous system mediated vasodilatation and a reduction in muscle tension and tonus (Schleip, 2003a). Reducing excessive muscular tension may, therefore, reduce capillary constriction and improve blood volume. It is possible that the sustained pressure and traction associated with MT is able to stimulate type III and IV mechanoreceptors, triggering a predominantly parasympathetic response (Arroyo-Morales, Olea, Martínez, *et al.*, 2008). In contrast, the findings of this study did not support the use of KT to improve blood volume at the paraspinal region. Although it is theorised that KT creates convolutions in the skin that could increase circulation and lymphatic volume, our data did not support this theory and is in agreement with previous studies (Stedge, Kroskie and Docherty, 2012). One possible reason for this may have been that the length of time that the KT tape was applied within this study may not have been optimal for this type of intervention. It has been proposed that KT tape should be applied for 3-5 days (Kase K, Wallis J, 2003). Tissue saturation was not significantly altered following any of the treatments. The Tsi value can be seen as a measure of dynamic balance between oxygen delivery and use (Janssens *et al.*, 2013). This result may be explained by the relatively sedentary nature of the interventions, as the paraspinal muscles were not active enough to alter the balance between oxygen demand and oxygen use.

6.5.1 Limitations

Sources of bias within this study are discussed more generally in section 9.1. The results of this study indicated PPT did not alter after MT and there was no difference between groups. This result was at odds with a number of other studies that demonstrated reductions in pain following the use of MT (LeBauer, Brtalik and Stowe, 2008; Castro-Sánchez *et al.*, 2011; Ajimsha, Daniel and Chithra, 2014). Indeed, the study by Castro-Sánchez et.al. (2011), identified a significant fall in the number of painful spots associated with Fibromyalgia including lower cervical, left trapezius muscle, the second ribs and gluteal muscles. While one of the proposed benefits of MT is to reduce pain, the

population group used for this study was a healthy a-symptomatic one. Previous investigations into the effects of MT on healthy populations have also found that pain was not altered following treatment (Arroyo-Morales, Olea, Martínez, *et al.*, 2008).

This was a pilot study with several limitations and it is, therefore, important to point out that the conclusions of this study are limited. The myofascial intervention within this study utilised an integrative approach that, although may be applied in a clinical setting, does not allow us to attribute the results of this study with any single myofascial technique. Indeed, the acute changes in blood volume identified in this study could be attributed to any one or all of the techniques outlined. Furthermore, this study investigated the effects of MT on asymptomatic subjects; therefore, any effects identified in this study could not be used to imply clinical significance to the LBP population. Future studies should attempt to make a comparison between different myofascial techniques and or with different types of massage methods using symptomatic subject groups. Future studies should also look to control participant's activities before testing were not controlled. The depth, pressure and speed of MT techniques could not be fully standardised, however, in this study; in order to standardise these variables as much as possible; the same therapist was used to conduct all the MT treatments. The current study only identified immediate changes in blood volume. To assess the effects of MT in the long term, future studies should explore the blood volume changes associated with repeated treatments and or the duration of the treatment effects following myofascial techniques.

6.5.2 Conclusions

In conclusion, this study demonstrated that the application of 30 minutes of integrated myofascial techniques increases peripheral blood volume at the paraspinal region, in healthy participants, compared to kinesiology tape and sham transcutaneous electrical nerve stimulation. The changes in peripheral blood volume had no impact on pain

perception in this asymptomatic population. Therefore, integrated myofascial techniques may have a possible role in the management of LBP, through improved O2 delivery and blood volume increases to the paraspinal muscles.

7 The acute effects of integrated myofascial techniques on lumbar paraspinal peripheral blood volume, local muscle fatigue and postural sway in subjects with non-specific low back pain

7.1 Abstract

Background

Low back pain (LBP) has been associated with poor back muscle endurance, reduced resistance to fatigue and altered postural control compared to those without LBP. Myofascial massage techniques (MT) are a form of manual therapy that have been postulated to reduce pain and increase blood flow to the paraspinal region and, therefore, may influence some of the mechanisms associated with back muscle fatigue. However, research to support this association is lacking.

Objectives

The purpose of this study was to compare the acute effects of integrated myofascial techniques (IMT) on blood flow and local muscle fatigue, pain pressure threshold and postural sway at the paraspinal muscle region.

Methods

This was a repeated measure, crossover design study design. 11 subjects with non-specific chronic LBP (5 male and 6 female) volunteered for this study. Participants were required to visit the clinic / laboratory on two separate occasions after being randomised to one of two treatments (MT or sham ultrasound). Paraspinal blood flow was measured at the L3 vertebral level, using Near Infrared Spectroscopy (NIRS), at rest and during two Biering-Sorensen fatigue tests; at baseline, before a 30-minute intervention treatment (F1) and once after a 30-minute intervention treatment (F2). Time to fatigue, sEMG, pain Pressure Threshold (PPT) and postural sway was measured before and after the treatments were administered.

Results

A Two-way ANOVA indicated a significant increase in volume variables at F2 compared to F1 for O_2Hb [F (1, 10) = 21.51 p = < .001], and tHb [F (1, 10) = 19.54 p = < .001] for the MT group compared to sham TENS. There was a significant improvement in time to fatigue [F (1, 10) = 24.17, p= < .001], for the MT group at F2 compared to F1, and EMG amplitude was significantly higher at the start of fatigue task 2 for the Sham TENS group trial compared to the MT group trial (U = 20.00, p = .007, f = 0.70). There was a main effect of time for PPT [F (1, 10) = 9.55 p < .011] but no change in postural sway measures.

Conclusion

the application of 30 minutes of integrated myofascial techniques increases peripheral blood volume at the paraspinal region and improved time to fatigue of the erector spinae muscle group in subjects with non-specific chronic LBP

7.2 Introduction

It has been shown that suboptimal lumbopelvic control and resistance to fatigue of the lumbar extensor muscles are some of the factors responsible for the development of CLBP (D'hooge *et al.*, 2013; Álvarez-Álvarez *et al.*, 2014), and previous studies have identified patterns of back muscle fatigue in the LBP population compared to controls (Roy *et al.*, 1990; Candotti *et al.*, 2008). Previous studies have also suggested muscle blood flow may be compromised in the trunk muscles of LBP patients (Yoshihito *et al.*, 2005). Furthermore, it has been reported that the LBP population experience muscle fatigue due to greater levels of intramuscular pressure in the spinal muscles and tissue hypoxia (Sakai *et al.*, 2012). This may be as a result of an increased muscular component to some LBP sufferers who are inclined to increase muscular tension to stabilise the spine and reduce pain provocation postures. This protective solution has been identified in subjects with recurring low back pain (D'hooge *et al.*, 2013), and that this abnormal strategy that increases co-contraction and paraspinal muscle activity may further augment the loads placed on the spine leading to long term mal-adaptive postures (Hodges and Moseley, 2003). These findings suggest that paravertebral muscles in a state of fatigue may compromise spinal stability (Descarreaux *et al.*, 2008; D'hooge *et al.*, 2013) and, therefore, affect the interaction between the active, passive and control subsystems for spinal stabilisation. Given the proposition that spinal stability relies on the interaction of these subsystems, disorders of the myofascial system may therefore contribute to instability of the spine often associated with low back pain (Panjabi, 2003). There is now ample evidence to suggest that back pain leads to altered motor and postural control and reductions in proprioceptive acuity which are associated with back muscle fatigue (Johanson *et al.*, 2011), reduced back muscle oxygenation (Janssens *et al.*, 2013) and alterations in proprioception (Thomas and Segal, 2004). Muscle fatigue and reduced blood flow have also been observed to influence lumbo-sacral position sense (Brumagne *et al.*, 2013). Johanson et al. (2011) observed that acute back muscle fatigue lead to

increased postural sway and a shift in proprioceptive dependency from the lumbo-sacral region to the ankles, while standing on an unstable surface. Furthermore, people with low back pain showed greater postural sway and were more dependent on ankle signals compared to healthy subjects. Increases in postural sway and reduced proprioception may, therefore, further contribute to spinal instability, and an increased risk of further injury.

Patients with low back pain have been observed to alter motor and postural control (Hodges and Moseley, 2003; Astfalck *et al.*, 2013). Several mechanisms have been described that could explain these alterations, these included pain and pain adaptation (Lund *et al.*, 1991), fear and fear-avoidance beliefs (Hodges and Moseley, 2003), muscle fatigue and decreased blood flow (Brumagne *et al.*, 2013), and are discussed in detail elsewhere (Hodges, 2011). Whatever the adaptation it is generally recognised that short term alterations in muscle activation patterns may be useful as a protective mechanism to limit pain provocation and re-injury but maintenance of this motor adaptations may be maladaptive in the long term (Hodges, 2011). Therefore, any intervention that can positively affect blood flow and reduce the effects of fatigue may be a useful tool in the prevention and management of low back pain as outlined above. The aim of this study was to compare the acute effects of myofascial techniques on local paraspinal muscle fatigue, blood volume; local muscle fatigue of the paraspinal muscles; pain pressure threshold and postural sway in a non-specific, chronic low back pain population group (NSLBP).

H_0: An acute bout of MT does not lead to improved blood volume, time to fatigue, sEMG profiles, pain pressure threshold at the paraspinal region, and postural control compared to a control treatment in subjects with NSCLBP.

H_1: An acute bout of MT leads to improved blood volume, time to fatigue, sEMG profiles, pain pressure threshold at the paraspinal region, and postural control compared to a control treatment in subjects with NSCLBP.

7.3 Methods

7.3.1 Participants

Participants were 11 subjects (5 male and 6 female) drawn from the student population at the University of Kent and the local area. Ethical permission was obtained through the School of Sports and Exercise Sciences ethics committee. Table 7.1 provides general participant information data. All participants were asked to complete an informed consent form and pre-test questionnaire (appendix 1) prior to any measurement or testing procedure. Participants were also screened at this point to assess inclusion or exclusion criteria. The inclusion criteria for this study were that participants had to have a history of chronic, non-specific, low back pain and be between18 and 65 years of age. This meant that subjects had to have a history of recurrent low back pain, occurring in multiple episodes over a 12-month period; or chronic low back pain defined as being present and or persistent within a 3-12-month period. Participants were excluded from this study if they reported the following: low back pain which has lasted less than 3 months, self-reported incidences of severe back or lower limb injury, surgery to the spine, major structural spinal deformity, ankylosing spondylitis, rheumatoid arthritis, spinal fracture, cancer, tumour or infection, nerve root compression, neurological conditions, psychiatric conditions, bleeding disorders, corticosteroid medication via inhaler, pregnancy, acute systemic infection, severe fibromyalgia.

Table 7. 1 Participant information data for the subjects within this study

Subjects n (male/female)	Age (yrs)	Height (cm)	Weight (kg)	Back Bothersome Score
11(M= 5, F= 6)	24 (6)	170.6 (9.3)	71.1 (14)	1.6 (0.7)

7.3.2 Study design

This was a repeated measure, crossover design study designed to compare the acute effects of myofascial techniques on local paraspinal muscle fatigue, blood volume, relative changes in haemoglobin, pain pressure threshold and postural sway in a non-specific, chronic LBP population group (NSLBP). Participants were required to visit the clinic / laboratory on two separate occasions after being randomised to one of two treatments (MT or sham ultrasound). Randomisation was achieved through simple allocation using a random allocation method. Recruitment of subjects was cumulative, therefore on arrival, subjects were asked to choose one of two envelopes, which concealed the treatment allocation. On their first visit subjects were familiarised with the protocol prior to any testing. The subjects were first assessed for postural sway with eyes open and eyes closed (see balance testing below). Subjects were then taken into the clinic environment where baseline resting EMG of the paraspinal muscles, paraspinal blood volume and PPT were measured. Subjects were then asked to complete a timed Biering-Sorensen fatigue task (F1) for as long as possible while blood volume and EMG were recorded at the paraspinal region. Immediately after the F1 task EMG, and NIRS sensors were removed and the subjects were retested for PPT at the paraspinal region and postural balance. Subjects were then given their allocated treatment of MT or sham ultrasound for 30 minutes. Following the treatment, the subjects were re assessed for paraspinal blood volume as a post treatment measure. EMG sensors were re-attached at the same site as the pre-intervention measures. The subjects were then asked to complete a second timed Biering-Sorensen fatigue task (F2) while EMG and blood volume were recorded at the

paraspinal region. Following the F2 task subjects were retested for PPT at the paraspinal region and postural balance. The second visit was organised one week apart from the first. This visit involved the same protocol as described above except the subjects would have received the second treatment allocation (MT or sham tens). Figure 7.1 below summarises the experimental procedure. The Biering-Sorensen test was used to measure the time to fatigue and was recorded at both F1 and F1 at both sessions (see below).

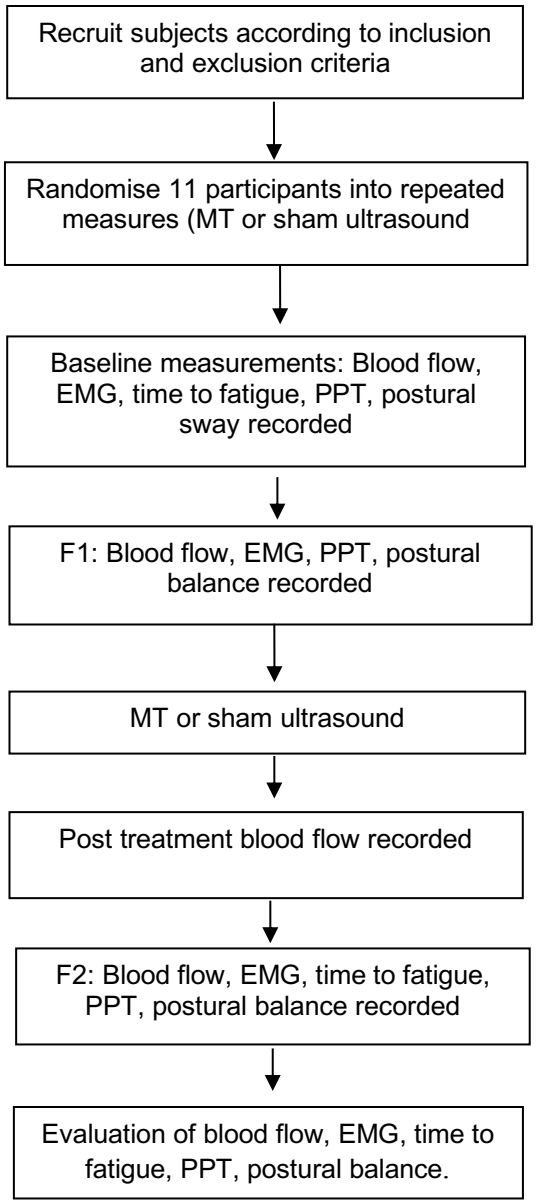

Figure 7. 1 Flow chart summarising the experimental procedure

7.3.3 Measurements

Blood volume measurements

Blood volume was measured using Near Infrared Spectroscopy Technique (NIRS). A description of the blood volume and oxygenation measurements can be found in 3.7.

Muscle activity

Details of the equipment and procedures for muscle activity patterns and EMG measurements are described in section 3.10

Postural balance

Postural sway was assessed by an RSscan® (RSscan Ipswich) balance system 7.22 A detailed description of the protocol can be found in section 3.15.

Biering-Sorensen test (time to fatigue measurements)

A description of the time to fatigue task measurements can be found in section 3.9.

Skinfold measurements

A skinfold measurement of adipose tissue was obtained on all participants. A description of the skinfold measurement protocol can be found in section 3.14.

Pain pressure threshold measurements (PPT)

A description of the measurements to identify the PPT of participants within this study can be found in section 3.13. Back pain bothersome scores were identified using the Keel STarT back screening tool (SBST). This was used to identify the current back bothersomness of the subject group. Details of the SBST, its administration and scoring are described in the general methods section 3.5.4

7.3.4 Treatment protocols

Integrated myofascial techniques (MT)

A trained massage therapist with 3 years of practical experience conducted the MT treatments. A description of the MT treatment protocol can be found in section 3.8.3.

Sham TENS

A description of the sham TENS treatment protocol can be found in section 3.11

7.3.5 Statistical analysis

Statistical analyses were performed in SPSS (IBM SPSS for Windows, version 23.0. Armonk, NY: IBM Corp). The normality assumption was assessed using Shapiro-Wilk tests. The independent variables were the treatment conditions (Myofascial Techniques and sham TENS) and time (fatigue 1 and fatigue 2) dependent variables were time to fatigue, sEMG of the paraspinal muscles, pain pressure threshold (PPT), centre of pressure displacement and tissue oxygenation and blood volume variables. Statistical significance level was set at alpha<0.05.

A 2x3 repeated measures, crossover, design ANOVA was conducted to analyse the change in blood volume variables. Change in blood volume was calculated at three time points; during fatigue 1 (F1), post intervention (post) and during fatigue 2 (F2). Change in blood volume was calculated by subtracting the blood volume measures at each time point from the baseline measurements taken at the beginning of the session. The ANOVA assessed for differences based on factors of: (a) treatment condition (MT and sham TENS), and NIRS blood volume variables (Tsi, O2Hb, HHb, tHb and HbDiff). A 2x2 repeated measures, crossover, design ANOVA was conducted to analyse the time to fatigue at F1 and F2. The ANOVA assessed for differences based on factors of: (a) treatment condition (MT and sham TENS) and (b) time (F1 and F2). A 2x4 repeated measures, crossover design, ANOVA was conducted to analyse the EMG data at the left

and right side paraspinal muscles at F1 and F2. The ANOVA assessed for differences based on factors of (a) treatment condition (MT and sham TENS) and the mean percentage change in RMS amplitude relative to F1. The change in RMS EMG was therefore normalised relative to the start of F1. The change in amplitude was measured at the start and end of F1 and F2 providing 4 time points for analysis (F1 start, F1 end, F2 start, F2 end). The first and last 10 second time segments were used to differentiate the start and end RMS amplitudes.

A 2x2 repeated measures, crossover, design ANOVA was conducted to analyse the change in pain pressure threshold scores at F1 and F2, from baseline measurements, for the two treatment conditions MT and sham TENS. The ANOVA assessed for differences based on factors of: (a) time (F1 and F2) and treatment condition (MT and sham TENS). A 2x2 repeated measures, crossover, design ANOVA was conducted to analyse the change in COP displacement at F1 and F2 for the two treatment conditions MT and sham TENS. The ANOVA assessed for differences based on factors of: (a) time (F1 and F2) and treatment condition (MT and sham TENS).

A Pearson's moment correlation was run to assess the relationship between the change in tHb, PPT and time to fatigue measurements.

Effect sizes for main effects were calculated by calculating f from η^2 where:

$$f = \text{Sqrt}\ \frac{\eta 2}{1 - \eta 2}$$

Effect size interpretations for f were described as 0.10 = small, 0.25 = medium and 0.40 = large. Effect sizes were also calculated for focused comparisons of contrasts. Where 1 degree of freedom exists F statistic values were converted to r to compare any main effects or contrasts using the formula below (Field, Miles and Field, 2013)

$$r = \text{Sqrt}\ \frac{\text{F}(1, \text{dfR})}{\text{F}(1, \text{dfR}) + dfR}$$

Where F = F score, 1,dfr = 1 degree of freedom, dfr = error degrees of freedom.

Effect size interpretations for f were described as 0.10 = small, 0.30 = medium and 0.50 = large.

7.4 Results

Figure 7.2 below outlines participant flow including enrolment, participant allocation, randomisation and analysis.

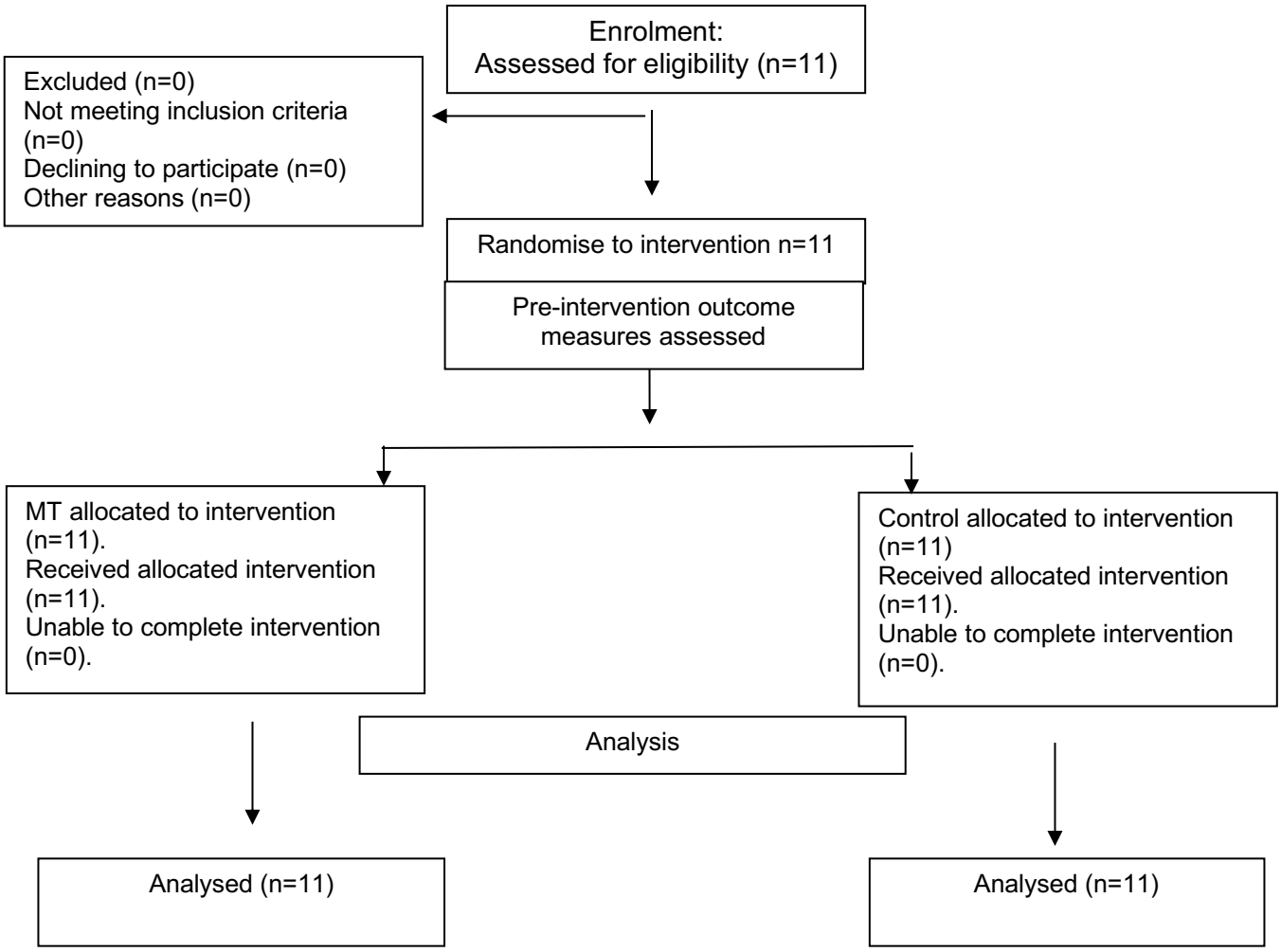

Figure 7. 2 Flow diagram depicting flow and allocation of participants from allocation to analysis.

7.4.1 Tissue oxygenation and blood volume

Blood volume variables at baseline (pre-fatigue) and during fatigue task 1 (F1) are shown in figure 7.3 and 7.4 below. Results indicated no difference at baseline or during F1 for both treatment conditions. The results indicate consistency and a standardised protocol for the fatigue task across both sessions. A comparison of blood volume measurements at rest and during fatigue load 1 indicated a reduction in tissue saturation and O2Hb with an increase in HHb, possibly as a result of the muscular activity during the fatigue task.

Table 7.2 below shows the means and SD's for the change tissue oxygenation and blood volume variables from baseline to F1, post treatment and F2. For the change in O2Hb, Mauchly's Test of Sphericity was not violated. There was Main effect of treatment $F(1, 10) = 12.9$ $p = < .005$; a Main effect of time $F(2, 20) = 12.5$ $p = < .001$; and an Interaction effect of treatment and time $F(2, 20) = 13.4$ $p = < .001$. The results indicated that the type of treatment interacted in some way with time for the change in O2Hb. The interaction contrasts compared MT and Sham tens treatments at time points F1, post treatment and F2. Contrast revealed that the change in O2Hb at F2 was significantly greater than at F1 for the MT treatment compared to Sham tens $F(1, 10) = 21.51$ $p = < .001$.

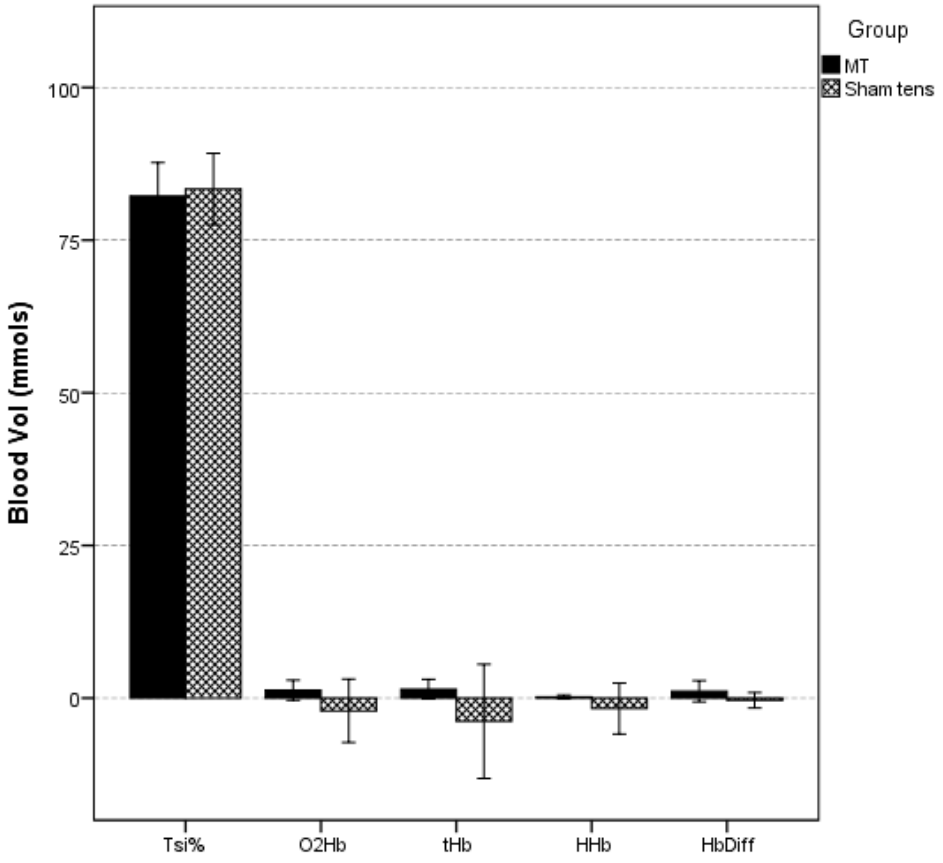

Figure 7. 3 Comparison of the mean blood volume variables between the myofascial techniques (MT), solid line; and sham TENS, dashed line; treatment conditions for tissue saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (O2Hb), deoxyhaemoglobin (HHb), and haemoglobin difference (HbDiff) at baseline.

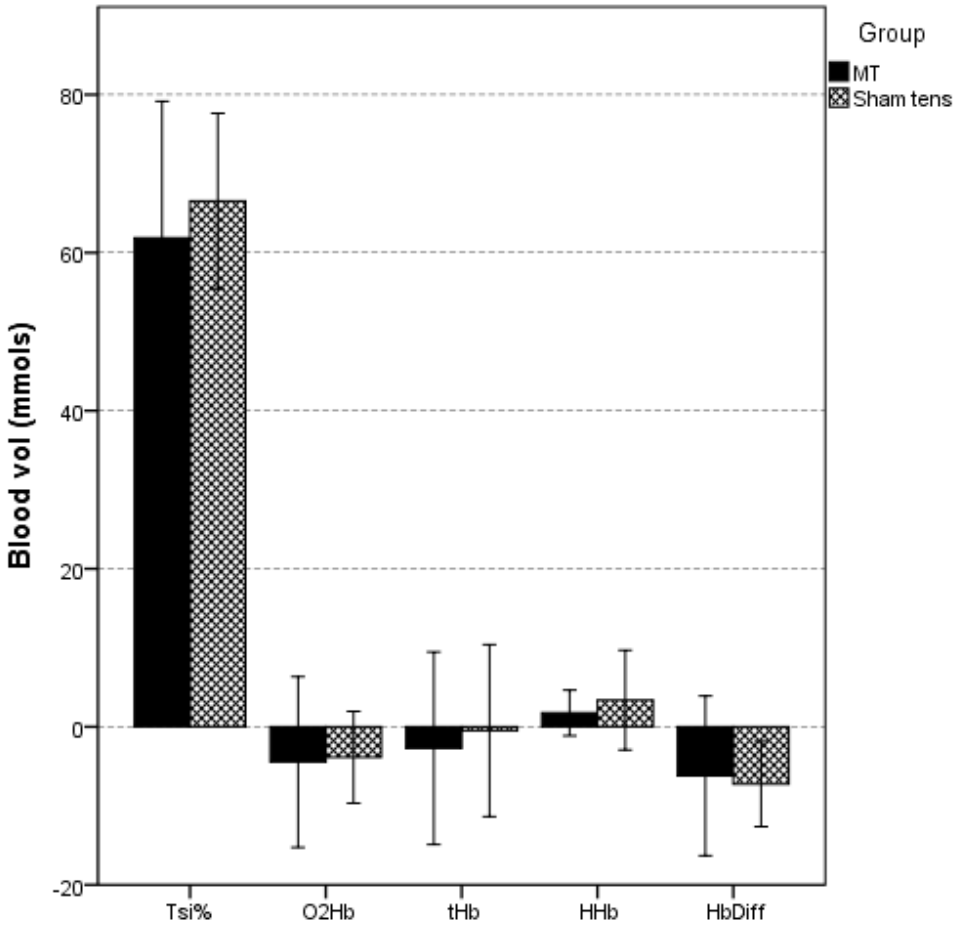

Figure 7. 4 Comparison of the mean blood volume variables between the myofascial techniques (MT), solid line; and sham TENS, dashed line; treatment conditions for tissue saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (O2Hb), deoxyhaemoglobin (HHb), and haemoglobin difference (HbDiff) at F1.

Table 7. 2 Change in blood volume from baseline at fatigue task 1 (F1) and fatigue task 2 (F2) for tissue saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (O2Hb), deoxyhaemoglobin (HHb), and haemoglobin difference (HbDiff).

Blood volume variable	Change in blood volume (MT)				Change in blood volume (Sham tens)			
	F1 (Mean ±SD)	**F2 (Mean ±SD)**	**95% confidence intervals**	**p-value**	**F1 (Mean ±SD)**	**F2 (Mean ±SD)**	**95% confidence intervals**	**p-value**
Change in Tsi	-20.33 (18.91)	-11.8 (17.96)	-7.87 to 24.93	.291	-16.83 (14.71)	-10.63 (17.78)	-8.31 to 20.71	.384
Change in tHb	-4.16 (17.54)	14.89 (20.63)	2.02 to 36.08	.030	3.32 (13.80)	1.92 (9.80)	-12.05 to 9.25	.787
Change in O2Hb	-5.75 (15.02)	13.70(16.32)	5.50 to 33.39	.009	-1.78 (9.52)	0.46 (10.34)	-6.60 to 11.08	.603
Change in HHb	1.59 (4.42)	1.20 (5.50)	-4.89 to 4.04	.864	5.10 (6.50)	1.72 (2.52)	-7.76 to 0.12	.123
Change in HbDiff	-7.33 (13.52)	12.50 (12.94)	0.73 to 24.27	.034	-6.88 (8.67)	-6.35 (11.53)	-8.58to 9.64	.905

For the change in tHb, Mauchly's Test of Sphericity was not violated. There was a main effect of treatment F (1, 10) = 5.99 p = < .034; a main effect of time F (2, 20) = 6.17 p = < .008; and an interaction effect F (2, 20) = 12.33 p = < .001. The results indicated that the type of treatment interacted in some way with time for the change in tHb. The interaction contrasts revealed that the change in tHb at fatigue F2 was significantly greater than at F1 for the MT treatment, F(1, 10) = 19.54 p = < .001 compared to Sham tens.

For the change in HbDiff, Mauchly's Test of Sphericity was assumed for treatment and time. There was a main effect treatment F (1, 10) = 22.19 p = < .001; a main effect of time F (2, 20) = 21.20 p = < .001; and an interaction effect, Greenhouse-Geisser adjusted, F (2, 20) = 9.36 p = < .001. The results indicated that the type of treatment interacted in some way with time for the change in HbDiff. The interaction contrast was significant f (1, 10) = 19.50 p = < .001 indicating that the change in Hbdiff at fatigue F2 was significantly greater than at F 1 for the MT treatment.

For the change in Tsi, Mauchly's Test of Sphericity was violated. There was a main effect of time F (2, 20) = 8.39, p = 0.11. There was no effect of treatment or interaction for Tsi. Figures 3-6 below represent the change in blood volume at the lumbar paraspinal region for O2Hb, tHb, HbDiff and Tsi. Contrasts revealed that blood volume variables O2, tHb and HbDiff, at the lumbar paraspinal region, were significantly higher at F2 than F1, following the MT treatment when compared to sham TENS. Table 7.5 and 7.6 Below identifies the effect sizes (f) for the time and treatment interactions and the relevant effect sizes for the contrasts (r). There were no significant differences for the HHb results across time or between groups.

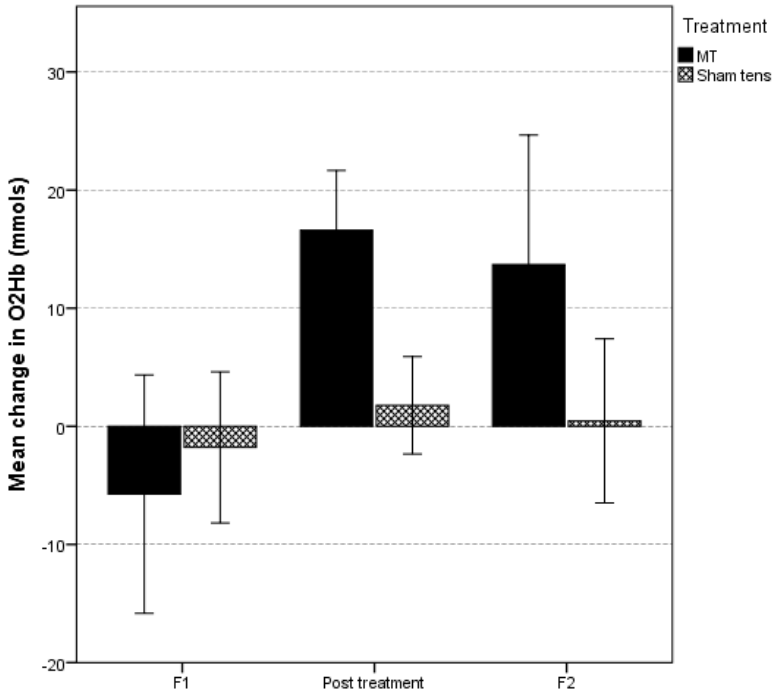

Figure 7. 5 Comparison of the change in O2Hb (oxyhaemoglobin) between the myofascial techniques (MT), dark bars and sham TENS checked bars; treatment conditions; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

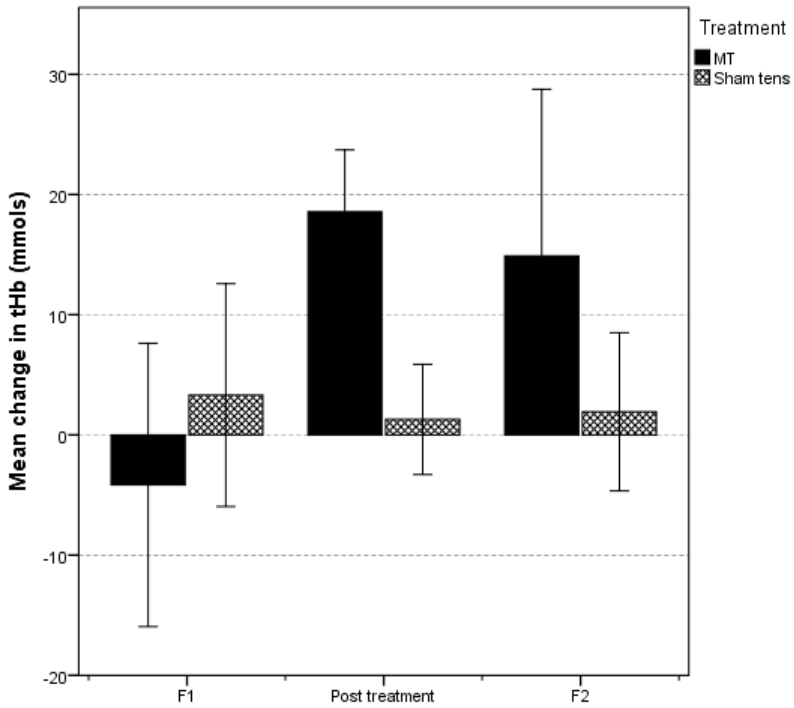

Figure 7. 6 Comparison of the change in tHb (total haemoglobin) between the myofascial techniques (MT), dark bars and sham TENS checked bars; treatment conditions; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

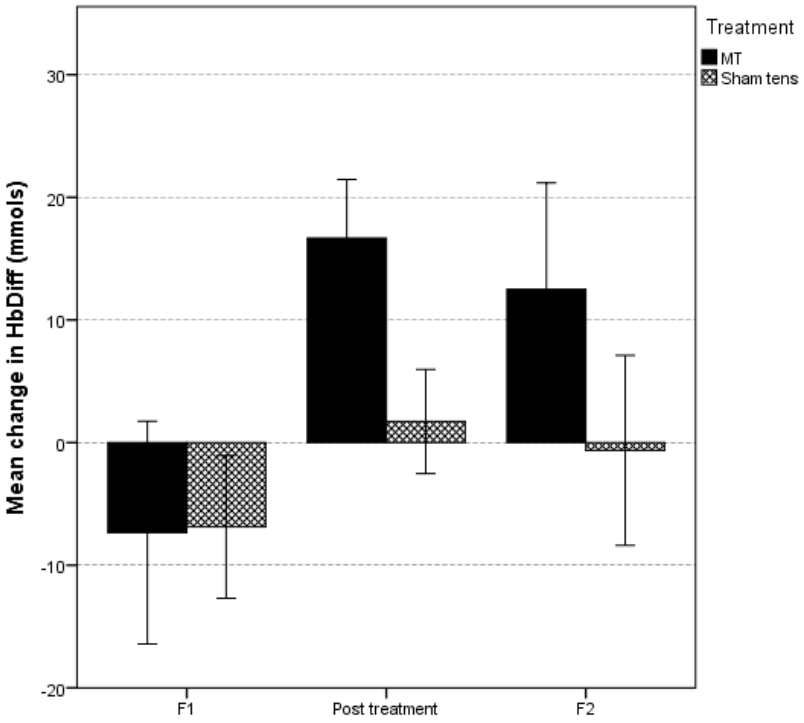

Figure 7. 7 Comparison of the change in HbDiff between the myofascial techniques (MT), dark bars and sham TENS checked bars; treatment conditions; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

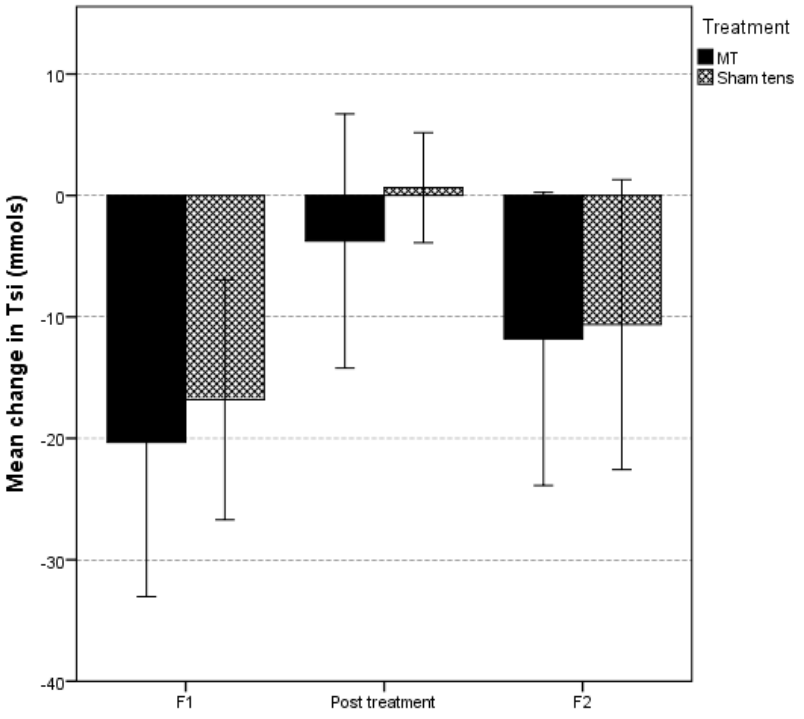

Figure 7. 8 Comparison of the change in Tsi (tissue saturation index) between the myofascial techniques (MT), dark bars and sham TENS checked bars; treatment conditions; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

Table 7. 3 Effect sizes (f) for main effects of treatment, time and interaction effects for tissue saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (O2Hb), deoxyhaemoglobin (HHb), and haemoglobin difference (HbDiff).

Blood variable	**Effect sizes for main effects f**		
	Treatment	**Time**	**Interaction**
Change in O2Hb	1.14	0.86	0.56
Change in tHb	0.77	0.57	0.73
Change in Hb Diff	1.49	0.45	0.2
Change in Tsi	0.23	0.9	0.07

Table 7. 4 P-values and effect sizes (r) for time and interaction contrasts; for tissue saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (2Hb), deoxyhaemoglobin (HHb), and haemoglobin difference (HbDiff); between myofascial techniques (MT) and Sham TENS interventions at F1 and F2 (fatigue task 1 and 2)

Contrasts	**Time (F1-F2)**			**Interaction of treatment and time (F1-F2)**		
	F-score	**P-value**	**Effect size (r)**	**F-score**	**P-value**	**Effect size (r)**
Change in Tsi	17.84	.002	0.80	-	-	-
Change in O2Hb	42.2	< .001	0.90	21.51	.001	0.83
Change in tHb	17.77	.002	0.80	19.5	.001	0.81
Change in HbDiff	43.45	<.001	0.90	9.62	0.11	0.70

- No significant interaction

7.4.2 Time to fatigue

Table 7.5 below shows the mean and SD figures for time to fatigue for F1 and F2 and both treatment conditions. The table 5 and Figure 7 below shows that the time to fatigue increased following the MT treatment at F2 and reduced following the sham TENS treatment at F2. Mauchly's Test of Sphericity was not violated and results revealed that there was an interaction effect of treatment and time $F(1, 10) = 24.17$, $p = < .001$; with effect size $f = 0.9$.

7.4.3 Pain Pressure Threshold

Table 7.5 below represents the mean and standard deviation data for PPT scores. Results indicated an improvement in PPT scores following both conditions. Results revealed a main effect of time $F(1, 10) = 9.55$ $p < .011$ with effect size $f = 0.9$ However, there was no effect for treatment or interaction for PPT.

7.4.4 Centre of Pressure displacement

Centre of pressure displacement means and standard deviations can be viewed in table 7.5 and depicted in figure 7.8 below. Results indicated a trend towards a reduction in COP travelled following the MT treatment and an increase in COP for the sham TENS at F2 compared to F1. However, there was no main effect for time or an interaction effect between groups at F1 and F2.

*Table 7. 5 Time to fatigue, pain pressure threshold (PPT) scores and centre of pressure (COP) measurements at fatigue task 1 (F1) and fatigue task 2 (F2) for the integrated myofascial techniques (MT) and sham TENS conditions. P-values for main effects. * = significant at 0.05 level.*

Fatigue Task	**Time to fatigue (s)**		**PPT score (kg/cm^2)**		**COP (mm^2)**	
	MT	**Sham TENS**	**MT**	**Sham TENS**	**MT**	**Sham TENS**
F1 (Mean ±SD)	101.27 (46.94)	108.55 (47.93)	6.64 (2.42)	6.56 (2.25)	337.18 (121.38)	370.91 (169.80)
F2 (Mean ±SD)	127.73 (34.46)	89.27 (33.85)	7.57 (2.35)	6.95 (2.75)	308.36 (98.39)	391 (210.26)
	Time to fatigue (s)		**PPT score (kg/cm^2)**		**COP (mm^2)**	
Main effects	**Time**	**Interaction**	**Time**	**Interaction**	**Time**	**Interaction**
P-value	-	<. 001	< .011	-	<.001	-

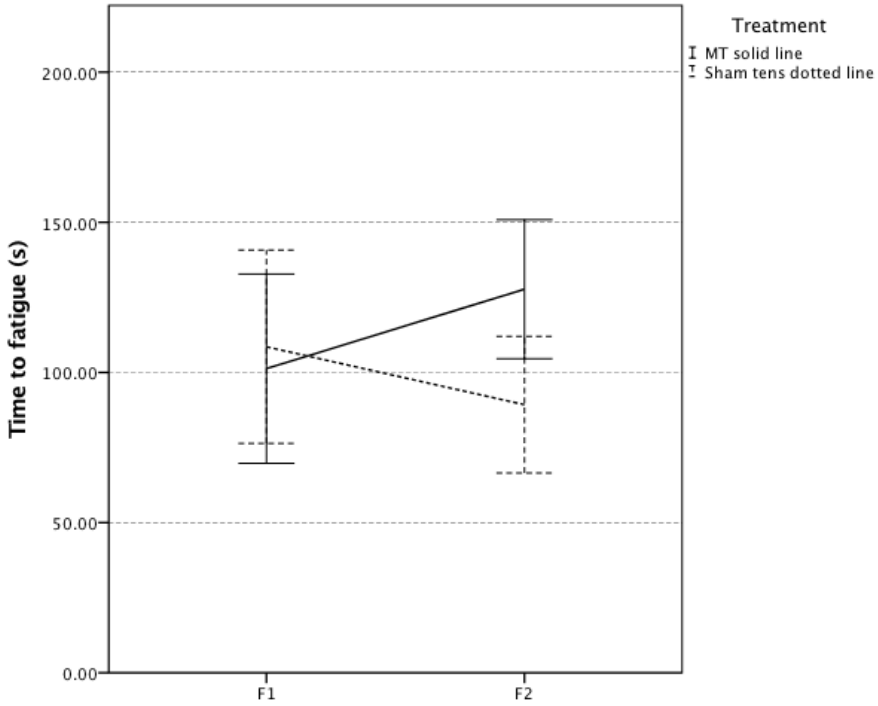

Figure 7. 9 Time to fatigue scores for the myofascial techniques (MT,) solid line, and sham TENS conditions, dotted line; during fatigue task 1 (F1) fatigue task 2 (F2).

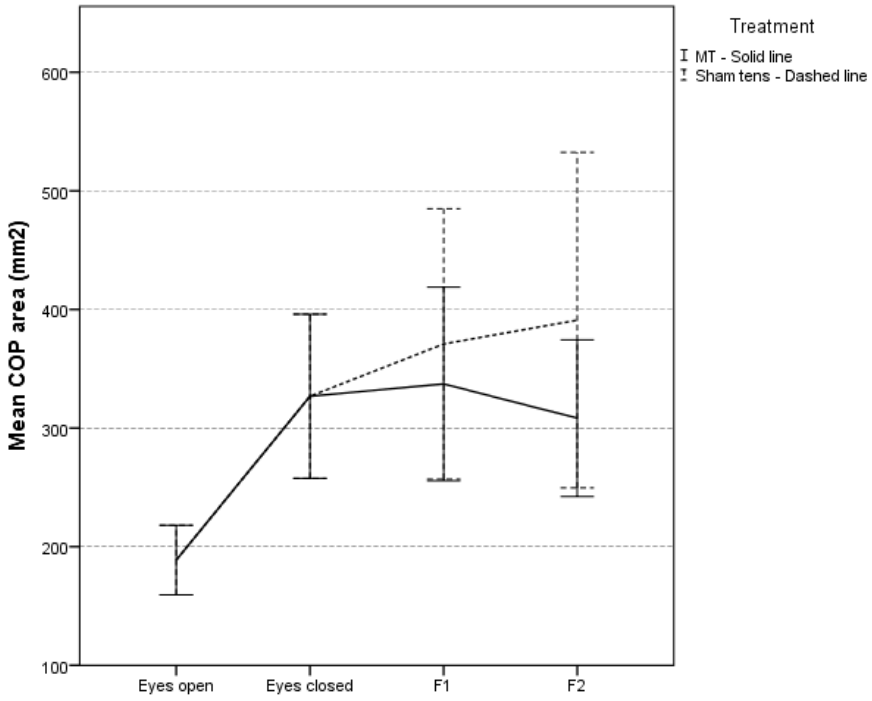

Figure 7. 10 Centre of pressure (COP) measurements for the myofascial techniques (MT,) solid line, and sham TENS conditions, dotted line; at baseline (eyes open and eyes closed) and at fatigue task 1 (F1) fatigue task 2 (F2).

7.4.5 EMG analysis

The RMS distribution was not normally distributed therefore the logarithm of the RMS EMG data was used for analysis. A Kolomov-Smirnov test of normality, indicated that the transferred data was also not normally distributed. Therefore, the percentage change log RMS EMG was analysed using non-parametric methods. Table 7.6 and figure 7.8 below shows the side averaged log RMS EMG values at the start and then the percentage change for points 2, 3 and 4.

A Friedman test reported a statistical difference in the percentage change in log RMS EMG, depending on the fatigue task time point, χ^2 (7) = 34.61, p = <.001. To examine the difference between the changes in EMG values within each group across time points a Wilcoxon signed-rank test was conducted. As the comparisons compared three time points a Bonferroni correction was applied resulting in a new significance level to 0.017 (0.05/3). Post hoc analysis with Wilcoxon signed-rank test revealed a significant increase in the change in EMG amplitude between the end of fatigue task 1 (time point 2) and the beginning of fatigue task 2 (time point 3) for the sham TENS group trial only (Z = -2.40, p = 0.007, f = 1.15). No other within group differences wire significant. In order to examine the difference in the change in EMG amplitude between group trials at time points 2, 3 and 4 a Mann-Whitney U test was conducted with a Bonferroni correction significance level of p = 0.017. From this data analysis, the Mann-Whitney U test revealed theta the change in EMG amplitude was significantly higher at the start of fatigue task 2 (time point 3) for the Sham TENS group trial compared to the MT group trial (U = 20.00, p = .007, f = 0.70).

Table 7. 6 Side averaged log RMS EMG percentage change values from F1 start to F1 end, F2 start and F2 end for the myofascial techniques (MT,) and sham TENS conditions.

	Fatigue task 1		Fatigue task 2	
Treatment	**Start (1)**	**End (2)**	**Start (3)**	**End (4)**
	Mean (SD) Log RMS Start (MV)	Mean (SD) % change log RMS	Mean (SD) % change log RMS	Mean (SD) % change log RMS
MT	2.19 (0.31)	-3.15 (8.19)	3.10 (9.74)	-3.93 (11.52)
Sham tens	2.06 (0.18)	7.67 (19.7)	15.82 (26.7)	4.00 (12.79)

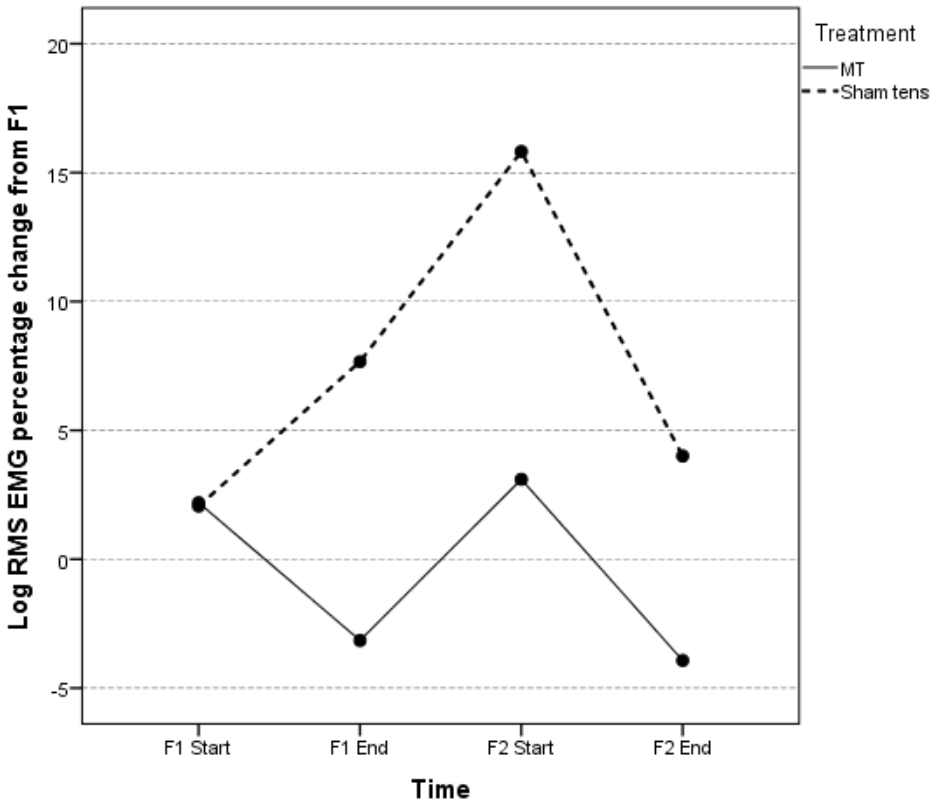

Figure 7. 11 Log RMS EMG measurements for the myofascial techniques (MT,) solid line, and sham TENS conditions, dotted line; at F1 start and then percentage change values at F1 end, F2 start and F2 end.

7.4.6 Correlation results

A Pearson's moment correlation was run to assess the relationship between the change in tHb, PPT and time to fatigue measurements. Preliminary analysis showed the relationship to be linear with both variables normally distributed.

There was a non-significant weak negative correlation between the change in tHb from f1 to F2 and the change in PPT from F1 to f2 r(9) = -.251, p = .457

There was a non-significant weak correlation between the change in tHb from baseline to F2 and the change in PPT from baseline to f2 r(9) = .08, p = .981

There was a non-significant weak negative correlation between the change in tHb from F1 to F2 and the change in TTF from f1 to f2 r(9) = -.001, p = .994

7.5 Discussion

The results of this study demonstrate that following local muscular fatigue, MT techniques significantly improve peripheral blood volume variables of tHb, O2Hb and HBdiff, during a subsequent fatigue task, at the paraspinal myofascial region. Although Tsi was improved from F1 to F2 this was not significantly so between groups. Time to fatigue was also significantly improved following the MT intervention and compared to sham TENS, suggesting MT may have the capacity delay the onset of fatigue in the pasrapsinal muscles. However, this was also corroborated with the results of EMG parameters, which indicated a significant difference in log RMS EMG amplitude between groups following the intervention. Improvements in PPT and COP for the MT group at F2 were not significantly different from the sham TENS intervention.

7.5.1 Blood volume and tissue oxygenation

The alterations in blood volume variable results in this study are in agreement with previous studies investigating the effects of massage on local muscle fatigue (Mori *et al.*, 2004; Durkin *et al.*, 2006). However, to date there ae no studies that identify the effects of

MT on blood volume, postural sway and fatigue. Previous studies have identified an improved blood flow following massage (Sefton *et al.*, 2010; Portillo-Soto *et al.*, 2014; Plakornkul *et al.*, 2016; Iwamoto and Mizukami, 2017) but none of these involved MT and as identified earlier these claims have been inconclusive and the effects of massage on blood flow remain questionable (Weerapong, Hume and Kolt, 2005; Bervoets *et al.*, 2015). There is a paucity of information on the effects of myofascial techniques on blood flow, volume and circulation (Ramos-González *et al.*, 2012; T Hotfiel *et al.*, 2017), with only one study directly measuring integrated MT techniques on blood volume using NIRS at the paraspinal myofascial region (Shah *et al.*, 2017).

Alterations in blood volume identified following MT have may be attributed to the normalisation of vascular dynamics at the paraspinal region through alterations in the fascial structure and autonomic nervous system mediated changes in muscle tone in the LBP population, and have been previously discussed in study 3 (Shah *et al.*, 2017). The Changes in blood volume following MT could be also explained by changes in sympathetic and parasympathetic activation as a result of the treatment. It has been reported that massage and myofascial techniques have the potential to shift the activity of the ANS from SNS to PSNS through stimulation of vagal tone (Schleip, 2003a; Moyer, Rounds and Hannum, 2004). These alteration in PSNS and SNS activity have been associated with reductions in stress hormones and levels of anxiety and arousal following massage (Moyer, Rounds and Hannum, 2004; Netchanok *et al.*, 2012). As previously identified fascia is densely innervated with mechanosensitive nerve endings that are capable lowering sympathetic tone and altering local blood volume and fluid dynamics (Schleip, 2003b). Furthermore, the TLF appears to be innervated with sympathetic efferent fibres (Tesarz, Hoheisel, Wiedenhöfer, *et al.*, 2011); a significant proportion of which are accompanied by blood vessels, implying a potential vasomotor component (Willard *et al.*, 2012). In a review of the theoretical framework behind the potential effects of MT Simmonds *et al*, (2012) have identified a number of studies that show a reduction in

SNS following MT. Therefore, it is plausible to suggest that MT to the paraspinal myofascial region may stimulate mechanosensitive structures and sympathetic afferents within the TLF resulting in an autonomic response that lead to a favourable increased blood volume. Another potential factor in the increase in blood volume is the potential for MT techniques to stimulate the release of vasoactive substances such as nitric oxide (NO). Previous evidence supports the principle that mechanical stimulation of the fascia is enough to distort the endothelium of blood vessels and stimulate NO release (T Hotfiel *et al.*, 2017), however, it should be noted that this evidence pertains to foam rolling myofascial techniques which may be more aggressive in terms of pressure applied to the fascia compared to the MT techniques within this study.

7.5.2 Postural sway and COP measurements

The results of this study also identified no significant change in the postural sway profiles following MT intervention. These results are in agreement with one other study investigating the effects of MT on postural stability (Castro-Sánchez *et al.*, 2011). Both back muscle fatigue and reduced back muscle oxygenation have been shown to adversely affect postural control and postural strategies in the LBP population (Johanson *et al.*, 2011; Janssens *et al.*, 2013). There are different hypothesis regarding the underlying causes of change in position sense in the presence of fatigue. Gandevia's (2001) review identified that muscle fatigue is accompanied by changes in all groups of muscle receptors which, in turn, would affect their contribution to proprioception. Therefore, fatigue related declines in muscle afferent feedback activity might lead to erroneous feedback to the CNS resulting compensation. Pedersen et al. (1999) identified that position sense may be affected by localised responses to muscle fatigue and point out that local muscle fatigue leads to an increase in intramuscular concentrations of metabolites, and inflammatory substances, that are known to alter alpha – gamma co-activation and have a direct impact on the discharge pattern of muscle spindles. Therefore, as muscle spindles have been known play a major role in proprioception and

position sense of the lumbo-sacral region (Brumagne *et al.*, 2000), local muscle fatigue may confound muscle spindle sensibility and induce errors in joint position sense (Ribeiro, Mota and Oliveira, 2007). It has been suggested that chemo sensitive muscle afferents in the paraspinal musculature increase the fusimotor drive to muscle spindles. Such afferent stimulation is also associated with ischemic muscle conditions, prolonged static muscular contractions and pain or the fear of pain (Brumagne *et al.*, 2000).

The influence of MT on blood volume and the autonomic nervous system may have also contributed to the results seen in this study. There is evidence to suggest that stimulation of the SNS can lead to a reduced muscle spindle afferent output (Hellström *et al.*, 2005), and over stimulation of the SNS has been shown to contribute to vasoconstriction to the vascular supply to skeletal muscle (Thomas and Segal, 2004). Previous evidence have shown that LBP populations exhibit reduced blood volume and oxygenation to the paraspinal region (Kovacs *et al.*, 2001; Yoshihito *et al.*, 2005) , increased levels of pain and fear avoidance patterns (Wong *et al.*, 2010). Furthermore, pain and noxious stimuli in the spine has been associated with increases in SNS activity (Simmonds, Miller and Gemmell, 2012). Therefore, suboptimal motor control strategies may develop in situations and pain syndromes that are characterised by increase in SNS activity, such as LBP (Roatta *et al.*, 2002). The results of this study showed some improvement in COP at F2 for the MT group compared to the control group but this was not significantly different. However, although the Biering-Sorrensen fatigue task at F1 resulted in a reduced blood volume to the paraspinal region, the PPT values were not significantly changed at this point. It is possible that the fatigue task and level of blood volume reduction during the fatigue task was not enough to cause pain and or the accumulation of noxious stimuli and, therefore significant changes in postural sway. Similarly, the significant improvements in blood volume seen at F2 for the MT group may have been enough to explain the slight improvements in COP but not enough to significantly influence postural sway.

7.5.3 Time to fatigue and RMS EMG

The results of this study indicated that the MT at the parsapinal region lead to an increase in the time to fatigue during the Biering-Sorensen back endurance task at F2 compared to F1. RMS EMG analysis and post hoc analysis revealed a significant increase in EMG amplitude at the beginning of fatigue task 2 compared to the end of fatigue task 1 for the sham TENS group only. Furthermore, the increase in EMG amplitude at this time point was significantly higher than the same time point for the MT group. Previous studies are in agreement with the effects of massage and fatigue (Tanaka *et al.*, 2002; Mori *et al.*, 2004; Durkin *et al.*, 2006) and only one study that identified the effects of MT on fatigue (Arroyo-Morales, Olea, Martínez, *et al.*, 2008). These authors did identify reductions in EMG amplitude during MVC following myofascial massage. However, the study differed to the current one in that it measured sEMG of the quadriceps muscle following Wingate tests. Considering the increase in blood volume at the parapaspinal region following the MT intervention, as outlined above, it would be reasonable to assume that the fatigue results may be attributed to metabolic and neuromuscular alterations. It has been suggested that continued isometric contractions of the paraspinal musculature lead to increases in intramuscular pressure sufficient enough to reduce blood volume and oxygenation to the muscle (Kramer *et al.*, 2005; Kell and Bhambhani, 2006), and reductions in blood volume have been associated with both peripheral and central fatigue (Broxterman *et al.*, 2015). Indeed, within this study the mean time to fatigue was below 120 seconds and the Biering-sorensen contractions in this study are typically associated with an MVC of 40-55%MVC (Kell and Bhambhani, 2006). Previous studies have suggested that the energy metabolism for this type of activity depends primarily on the anaerobic glycolysis (Kramer *et al.*, 2005).

The combination of reduced blood volume and the fatigue task may have led to metabolic alterations in the active muscle including H^+ and inorganic phosphate (Pi) accumulation

(Murthy *et al.*, 2001; Allen, Lamb and Westerblad, 2008). Although the causes of fatigue are multifactorial, H^+ and Pi accumulation has been associated with inhibition of Ca^{2+} release from the sarcoplasmic reticulum and cross bridge cycling and, therefore, fatigue development (Allen, Lamb and Westerblad, 2008). Therefore, the significant improvement in time to fatigue at F2 following the MT intervention may have been attributed to the changes in blood volume seen within this study and identified above. The Improvements in tissue oxygenation and total blood volume at the paraspinal region following the MT intervention may have offset blood volume occlusion associated with increases in IMP, and, may have contributed to metabolite clearance (Murthy *et al.*, 2001).

The significant increase in EMG amplitude at the start of fatigue task 2 for the sham TENS group compared to the MT group may suggest that the MT treatment induced something within the muscle to require less muscle activation to maintain the same position. It has been postulated that sustained isometric contractions, as used in the current study, may stimulate group III and IV afferents (Taylor and Butler, 2000), speculated to be as a result of metabolite accumulation and a rise in pain levels in muscles that are ischemic (Kennedy *et al.*, 2014) . Furthermore, muscles in an ischemic states have been shown to have reductions in motor unit firing rates (Kennedy *et al.*, 2014), and, type III and IV afferent stimulation during sustained muscle contraction have been shown to contribute to motor unit inhibition (Gandevia, 2001). Thus, the fatiguing effect at the end of fatigue task 1 may have led to an increase in intramuscular metabolites that contributed to fatigue development by affecting the firing frequency of type III and IV muscle afferents. Furthermore, during the continuous isometric contraction associated with the Biering-Sorrensen task, blood volume may have been reduced to further contribute to the fatigue development as outlined above. Although muscle afferent activity was not measured in this study, it is possible that the increase in blood volume at fatigue the start of fatigue task 2 following the MT intervention may have contributed to either a reduction or

prevention of an ischemic state and a reduced stimulation of type III and IV muscle afferents leading to the change in EMG amplitude and time to fatigue results found within this study.

7.5.4 Pain pressure threshold

The improvements in pain across time are in agreement with other studies into the effects of MT on LBP (Hsieh *et al.*, 2002; Tozzi, Bongiorno and Vitturini, 2011; Ajimsha, Daniel and Chithra, 2014). However, the results of this study did not identify significant differences in PPT between interventions, indicating that MT was not better than sham TENS in reducing pain perception in the LBP group. One of the reasons behind this discrepancy may be the lack of homogeneity in the MT techniques and the methodologies associated with this and the previous studies mentioned. Furthermore, as with many non-pharmacological therapies, it is often difficult to completely blind subjects to the intervention which may have some effect on the outcome measures associated with a placebo effect (Chou *et al.*, 2017). It is possible that subject's expectations and perceptions of both interventions may have contributed to the results in this study. However, patient expectations for pain perception improvements in the LBP are seen to be modest and dependent on a number of factors such as age, sociodemographic factors and previous experiences (Hsu *et al.*, 2014). The subjects in this study were largely from a university setting and with a mean age of under 25 and a back bothersome score of 1.55. These figures suggest that the subjects were not within the age bracket of sufferers with the highest frequency of symptoms (Manchikanti *et al.*, 2014). This may also be reflected in the low to moderate bassline back bothersome score of the subject group (Main *et al.*, 2012b).

7.5.5 Limitations

The RMS EMS fatigue measurements within this study were used to identify changes in amplitude and has been identified as a reliable measure reflective of the force of muscle contraction. However, it may have been more appropriate to measure fatigue in the

frequency domain either through mean frequency (MNF) or median frequency (MDF). Measurements in the frequency domain have been described as a more accurate indicator of muscle fatigue (Criswell, 2011). Changes in the power spectrum shift to lower frequencies are said to be as a result of changes in the waveform of the motor unit and reduced conduction velocities associated with lactate and H^+ accumulation (Tanaka *et al.*, 2002; Kramer *et al.*, 2005). Therefore, if MT application improved blood volume and time to fatigue it may be more prudent to include EMG measures in the frequency domain and supplementary measures of lactate and H^+. As outlined previously in chapter 6, the MT intervention used an integrative approach that makes it difficult to attribute effects to any single myofascial technique and the depth and pressure of the techniques could not be quantified. Future studies should also look at the effects of MT over repeated treatments and the duration of the treatment effects over time. Finally, limited statistical power because the modest sample size of n = 11, may have contributed to limiting the significance of some of the statistical comparisons. Furthermore, based on the mean between groups comparison for changes in postural sway, with an effect size of f = 0.24, a sample size of 36 would be required to achieve statistical power at the 0.8 level. Finally, based on the mean between groups comparison for PPT, with an effect size of f = 0.39, sample size of 16 would be required to achieve statistical power at the 0.8 level.

7.5.6 Conclusion

In conclusion, this study demonstrated that the application of 30 minutes of integrated myofascial techniques increases peripheral blood volume at the paraspinal region and improved time to fatigue of the erector spinae muscle group in subjects with non-specific chronic LBP. Therefore, integrated myofascial techniques may have a possible role in the management of LBO thorough improving O2 and blood volume delivery and reduce local muscular fatigue of the parsapinal musculature.

8 The acute effects of integrated myofascial techniques on lumbar paraspinal blood volume and local paraspinal muscle fatigue in back pain and non- back pain subjects

8.1 Abstract

Background

Low back pain (LBP) has been associated with poor back muscle endurance and reduced resistance to fatigue compared to those without LBP. Myofascial massage techniques (MT) are a form of manual therapy that have been postulated to reduce pain and increase blood flow to the paraspinal region and, therefore, may influence some of the mechanisms associated with back muscle fatigue. However, research to support this association is lacking.

Objectives

The purpose of this study was to compare the acute effects of integrated myofascial techniques (MT) on blood volume and local muscle fatigue at the paraspinal muscle region in subjects with and without LBP.

Methods

22 subjects (10 male and 12 female) (age, 23 ± SD 8) volunteered for this study, 11 with LBP and 11 without LBP. Paraspinal blood volume was measured at the L3 vertebral level, using Near Infrared Spectroscopy (NIRS), at rest and during two Biering-Sorenses fatigue tests; once before a 30-minute MT treatment (F1) and once after a 30-minute MT treatment (F2). Time to fatigue and pain Pressure Threshold (PPT) was also measured before and after the treatments were administered.

Results

A Two-way ANOVA indicated a significant increase in blood flow variables at F2 compared to F1 for O_2Hb [F (2-40) = 23, P<0.001], tHb [F (2-40) = 15.88, P <0.001] and Tsi [F (2-40) = 19.28, P <0.001]. There were no significant differences for blood volume variables between groups. There was a significant improvement in time to fatigue [F (1-

20) = 17.38, p = <0.001], for both groups at F2 compared to F1, but no significant difference between groups. There was also a significant increase in PPT [F (2-40) = 28.2, p = <0.001] for both groups at F2 compared to F1. Post hoc analysis also revealed that PPT was significantly improved in the non-LBP group compared to the LBP group [F (1, 20) = 8.88 p < .007].

Conclusions

This study demonstrated that Integrated MT increases peripheral blood volume and time to fatigue at the paraspinal muscles in LBP and non-LBP group. MT also improved PPT scores for both groups but was significantly greater in the non-LBP group.

8.2 Introduction

Poor back muscle fitness (Kell and Bhambhani, 2006) and lumbar paraspinal muscle dysfunction (Hagen, Jeffrey J. Hebert, *et al.*, 2015) have been associated with LBP. Furthermore, It has been shown that poor back muscle endurance (Kell and Bhambhani, 2006; da Silva *et al.*, 2015) and resistance to fatigue (Sung, Lammers and Danial, 2009) are factors responsible for the development of LBP. Candotti *et al*. (2008) observed significantly reduced force capacity of the back muscles in subjects with low back pain compared to those without. The same authors also identified the fatigue indexes and force value results from the study enabled 89.5% of subjects to be accurately classified with pathology. Da Silva *et al*. (2015) also identified that subjects with CLBP displayed more back fatigue than people without CLBP, while Sung *et al*. (2009), have suggested that subjects with LBP demonstrate higher fatigability of the lumbar and thoracic erector spinae muscles. Furthermore it appears that trunk muscle fatigue may be associated with reduced spinal stability, delayed muscle reaction times and reduced lumbar position sense (Richmond, 2012; Boucher *et al.*, 2015; da Silva *et al.*, 2015).

Paraspinal blood volume and tissue oxygenation impairments have also been identified in subjects with LBP compared to those without LBP (Kovacs *et al.*, 2001; Yoshihito *et al.*, 2005). Paraspinal muscles have been shown to exhibit reduced muscle oxygen utilisation during dynamic movements (Kovacs *et al.*, 2001) and during sustained static contractions of the paraspinal muscles in the LBP population (Kell and Bhambhani, 2006; Dehner *et al.*, 2009). Dehner et al. (2009) reported that subjects with LBP exhibited reduced tissue oxygenation during an isometric extension task which was also associated with local muscular fatigue. These results suggest that the paraspinal muscles of the LBP population present an impaired capacity to deliver and use oxygen (Olivier *et al.*, 2013). Fatigue is also said to be accelerated during isometric contractions due to an increase in intramuscular pressure, capillary constriction and restricted blood volume to the working

muscles (Kramer *et al.*, 2005; Kell and Bhambhani, 2006; Álvarez-Álvarez *et al.*, 2014). Previous evidence has suggested that the decreasing oxygen content of tissue can lead to fatigue (Murthy *et al.*, 2001). There are a number of fatiguing protocols used to assess fatigue of the paraspinal muscles (da Silva *et al.*, 2015), however, the Biering-Sorensen test is a popular test designed to measure the isometric endurance of the trunk muscles and has been used to measure the resistance of the trunk muscles to fatigue (Demoulin *et al.*, 2006). It has been identified that shorter muscular endurance times during the test is associated with an increased risk of developing LBP (Kell and Bhambhani, 2006), and is considered to be sensitive enough to differentiate between healthy subjects and those with LBP (Álvarez-Álvarez *et al.*, 2014).

The technique of near-infrared spectroscopy (NIRs) is a non-invasive and continuous method of monitoring changes in blood volume and tissue oxygenation and has been widely used to monitor these parameters within muscle (Ferrari and Mottola, 2004; Durkin *et al.*, 2006; Munk *et al.*, 2012; Sakai *et al.*, 2012). Blood flow and oxygenation to the paraspinal muscles in the LBP population have been shown to be improved through the use of exercise based interventions (Sakai *et al.*, 2012; Olivier *et al.*, 2013). However, to date the is a paucity of research identifying the effects of manual massage therapies on blood flow or volume at the paraspinal region (Shah *et al.*, 2017). Furthermore, to our knowledge no study to date has compared the effects of myofascial massage techniques (MT) on local muscular fatigue and blood volume at the paraspinal region in subjects with and without LBP.

Myofascial massage techniques (MT) are an increasingly popular manual therapy treatment for musculoskeletal conditions (Ajimsha, Daniel and Chithra, 2014; Ajimsha, Al-Mudahka and Al-Madzhar, 2015). MT has been described as a form of manual medicine

that involves the application of low load and long duration stretching to the fascial layers of the connective tissue system (Barnes, 1997; Nitsure and Welling, 2014; Ajimsha, Al-Mudahka and Al-Madzhar, 2015). It has been suggested that injury or dysfunction to the fascial network can result in adhesions and restrictions within the fascial layers and may place stress on vascular and neural structures that are structurally associated with the fascia (Schleip, 2003a; Pavan *et al.*, 2014; Ajimsha, Al-Mudahka and Al-Madzhar, 2015), contributing to pain, reduced ROM, and decreased strength and motor coordination (Stecco *et al.*, 2013; A. Stecco *et al.*, 2015; Namvar and Moghadam, 2016). Proponents of MT suggest that a combination of mechanical and neuro-reflexive mechanisms may restore the integrity of the fascia, relieving pressure on pain sensitive structures such as nerves and blood vessels (Schleip, 2003a; A. Stecco *et al.*, 2015; Celenay, Kaya and Akbayrak, 2016), contributing to changes in local fluid dynamics, reducing excessive muscle tension, capillary constriction, and improve local blood volume (Schleip, 2003a). Although these studies were not specifically related to LBP it suggests that myofascial techniques may have a role to play in improving blood volume in LBP patients. Recently, evidence has emerged to suggest that myofascial techniques can increase blood volume at the paraspinal region (Shah *et al.*, 2017). Therefore, the purpose of the study was to examine the acute effects of myofascial techniques on paraspinal blood volume, local muscle fatigue of the paraspinal muscles and pain pressure threshold in subjects with and without LBP.

H_0: An acute bout of MT does not lead to improved blood volume, time to fatigue, and pain pressure threshold at the paraspinal region, compared to a control treatment in subjects with NSCLBP compared to subjects without NSCLBP.

H_1: An acute bout of MT leads to improved blood volume, time to fatigue, and pain pressure threshold at the paraspinal region, compared to a control treatment in subjects with NSCLBP compared to subjects without NSCLBP.

8.3 Methods

8.3.1 Participants

Participants were 22 subjects (10 male and 12 female) drawn from the student population at the University of Kent and the local area. Ethical permission was obtained through the School of Sports and Exercise Sciences ethics committee. Table 8.1 provides general participant information data. All participants were asked to complete an informed consent form and pre-test questionnaire (appendix 1) prior to any measurement or testing procedure. Participants were also screened at this point to assess inclusion or exclusion criteria. The inclusion criteria for the non-specific LBP group was a history of chronic, non-specific, low back pain and be between18 and 65 years of age. This meant that subjects had to have a history of recurrent low back pain, occurring in multiple episodes over a 12-month period; or chronic low back pain defined as being present or persistent within a 3-12 month period. Inclusion criteria for the non-back pain group was an absence of a history of LBP or the lower extremity in the past 12 months. Participants were excluded from this study if they reported the following: low back pain which has lasted less than 3 months, self-reported incidences of severe back or lower limb injury, surgery to the spine, major structural spinal deformity, ankylosing spondylitis, rheumatoid arthritis, spinal fracture, cancer, tumour or infection, nerve root compression, neurological conditions, psychiatric conditions, bleeding disorders, corticosteroid medication via inhaler, pregnancy, acute systemic infection, severe fibromyalgia.

Table 8. 1 Participants characteristics by group (mean ±SD). For all values P = > 0.05

Group	N (male/female)	Age (yrs)	Height (cm)	Weight (kg)	BMI
LBP	11(M=5, F=6)	24 (5.6)	170 (9.3)	71.1 (13.9)	24.4 (3.9)
Non-back pain	11(M=5, F=6)	22 (5.8)	170 (7.5)	75.3 (13.9)	25.8 (5.1)

8.3.2 Study design

This was a parallel designed non-crossover study designed to compare the acute effects of myofascial techniques on local paraspinal muscle fatigue, blood volume, pain pressure threshold between subjects with non-specific, chronic low back pain (NSLBP) and subjects without NSLBP. The study was a composite of results from the LBP group's results from study 4 and the recruitment of 11 healthy subjects subsequent to this in order to make a comparison between population groups. Participants were therefore required to visit the clinic / laboratory on one occasion. Subjects were taken into the clinic environment where baseline paraspinal blood volume and PPT were measured. Subjects were then asked to complete a timed Biering-Sorensen fatigue task (F1) for as long as possible while blood volume was recorded at the paraspinal region. Immediately after the F1 task NIRS sensors were removed and the subjects were retested for PPT at the paraspinal region. Subjects were then provided with a MT treatment for 30 minutes. Following the treatment, the subjects were re assessed for paraspinal blood volume as a post treatment measure. The subjects were then asked to complete a second timed Biering-Sorensen fatigue task (F2) while blood volume was recorded at the paraspinal region. Following the F2 task subjects were retested for PPT at the paraspinal region. The

Biering-Sorensen test was used to measure the time to fatigue and was recorded at both F1 and F2 at both sessions (see below).

8.3.3 Measurements

Blood volume and tissue oxygenation

Blood volume was measured using Near Infrared Spectroscopy Technique (NIRS A description of the blood volume and oxygenation measurements can be found in 3.7.

Biering-Sorensen test (time to fatigue measurements)

A description of the time to fatigue task measurements can be found in section 3.9.

Skinfold and pain pressure threshold

A skinfold measurement of adipose tissue was obtained on all participants on the right side three centimetres from the L3 vertebra. A description of the skin fold and PPT measurements can be found in section 3.13-3.14.

8.3.4. Treatment protocol

Integrated myofascial techniques (MT)

A trained massage therapist with 3 years of practical experience conducted the MT treatments. A description of the MT treatment protocol can be found in section 3.8.3.

Sham TENS

A description of the sham TENS treatment protocol can be found in section 3.11

8.3.5 Statistical analysis

Statistical analyses were performed in SPSS (IBM SPSS for Windows, version 23.0. Armonk, NY: IBM Corp). The normality assumption was assessed using Shapiro-Wilk tests. A Two-way ANOVA with repeated measures was used to compare the effects of Myofascial techniques (MT) on paraspinal blood volume PPT and time to fatigue between LBP and non-LBP groups. The dependent variables were paraspinal blood volume PPT

and time to fatigue. The within subject's factor was time and the between subject's factor was group (LBP and non-LBP)

A 2x3 ANOVA was conducted to analyse the change in in blood volume variables. Change in blood volume was calculated at three time points; during fatigue 1 (F1), post intervention (post) and during fatigue 2 (F2). Change in blood volume was calculated by subtracting the blood volume measures at each time point from the baseline measurements taken at the beginning of the session. The ANOVA assessed for differences based on factors of: (a) group (LBP and non-LBP), and NIRS blood volume variables (Tsi, O2Hb, HHb, tHb and HbDiff) over time (F1, post and F2). A 2x2 ANOVA was conducted to analyse the time to fatigue at F1 and F2. The ANOVA assessed for differences based on factors of: (a) group (LBP and non-LBP) and (b) time (F1 and F2). A 2x3 ANOVA was conducted to analyse the PPT scores between groups (LBP and non-LBP) at baseline, F1 and F2. Effect sizes for main effects were calculated by calculating f from η^2 where:

$$f = \text{Sqrt}\ \frac{\eta 2}{1 - \eta 2}$$

Effect size interpretations for f were described as 0.10 = small, 0.25 = medium and 0.40 = large. Effect sizes were also calculated for focused comparisons of contrasts. Where 1 degree of freedom exists F statistic values were converted to r to compare any main effects or contrasts using the formula below (Field, Miles and Field, 2013)

$$r = \text{Sqrt}\ \frac{\text{F}(1, \text{dfR})}{\text{F}(1, \text{dfR}) + dfR}$$

Where F = F score, 1, dfr = 1 degree of freedom, dfr = error degrees of freedom.

Effect size interpretations for f were described as 0.10 = small, 0.30 = medium and 0.50 = large.

8.4 Results

Figure 8.1 below outlines participant flow including enrolment, participant allocation, randomisation and analysis.

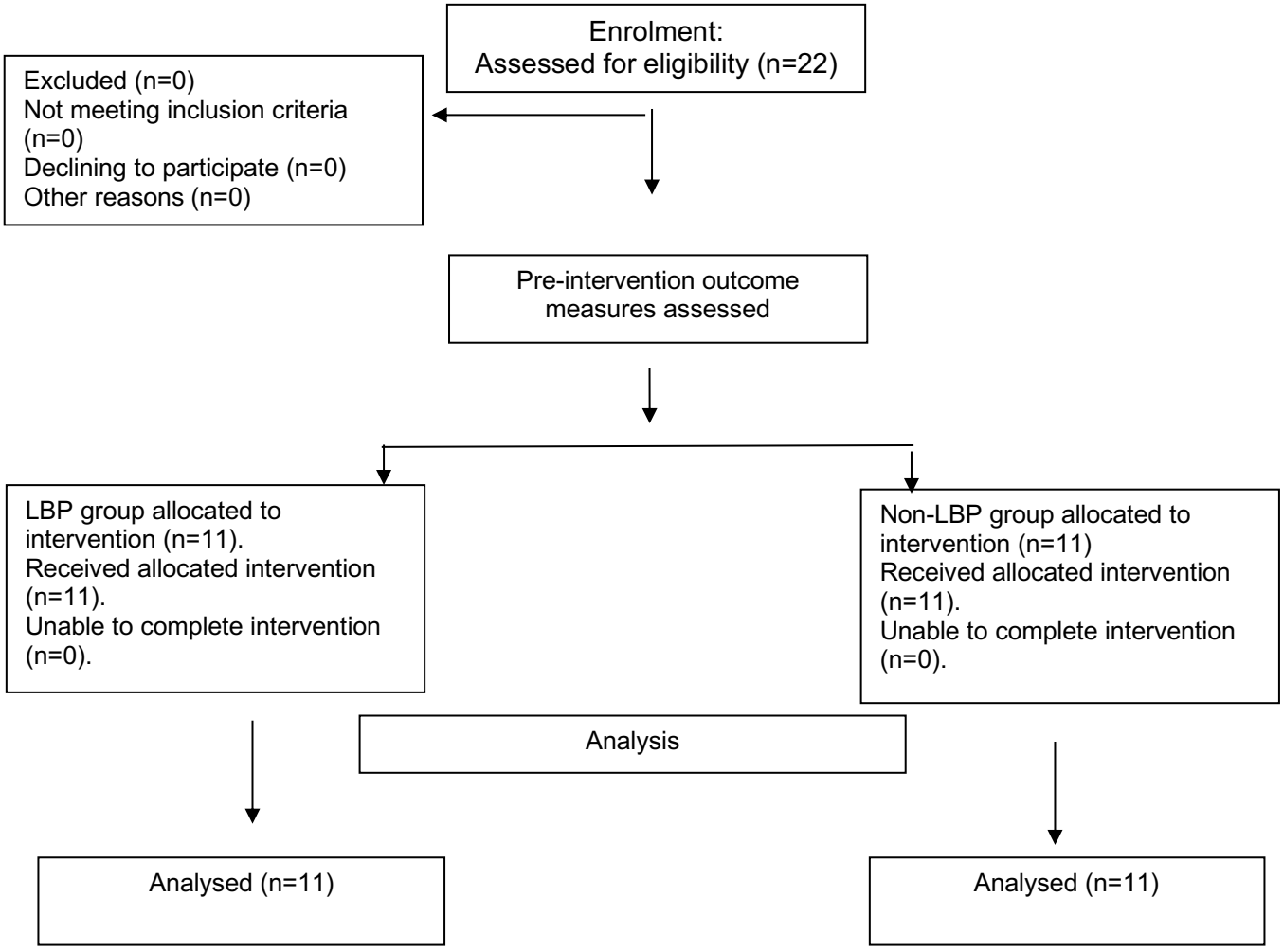

Figure 8. 1 Flow diagram depicting flow and allocation of participants from allocation to analysis.

8.4.1 Pain pressure threshold

For the change in PPT Mauchly's Test of Sphericity was assumed for PPT across time. Results for the pain pressure threshold tests indicated a main effect of time F (2, 40) = 28.2, p = < .001; f = 1.18. There was also a marginal main interaction effect of time and group F (2, 40) = 3.262, p = .049 f = 0.4. Contrasts revealed that the PPT score for the

non-back pain group were significantly higher than LBP group following fatigue task 2 (PPT3) compared to fatigue task 1 (PPT2) F (1, 20) = 8.88 p < .007; f = 0.75. Table 8.5 represents the effect sizes for the time to fatigue results.

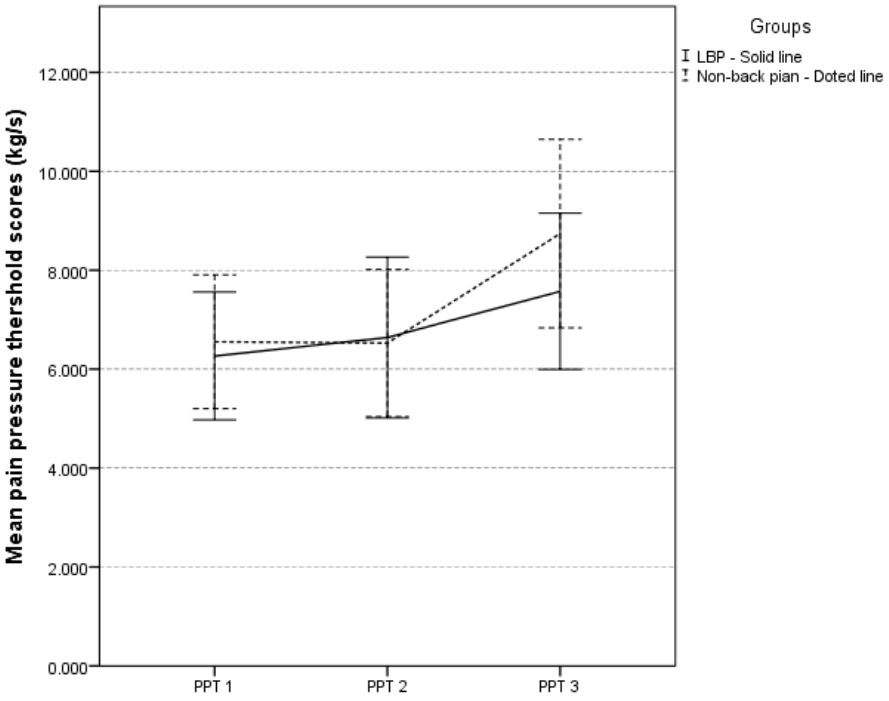

Figure 8. 2 Pain pressure threshold scores (PPT) for the low back pain group (LBP) solid line, and the non-back pain group, dotted line; at baseline (PPT1); following fatigue task 1 (PPT2) and following fatigue task 2 (PPT3).

8.4.2 Time to fatigue

Results for the time to fatigue at F1 and F2 indicate that there was a main effect of time F (1, 20) = 17.38 p = <.001; f = 0.93; with both groups increasing their time to fatigue following the MT intervention. However, there was no significant difference between groups at time points F1 and F2. Table 8.2 and 8.3 below shows the mean and standard deviation values for the time to fatigue and PPT measurements for both groups and each time point measured. Table 8.5 represents the effect sizes for the time to fatigue results.

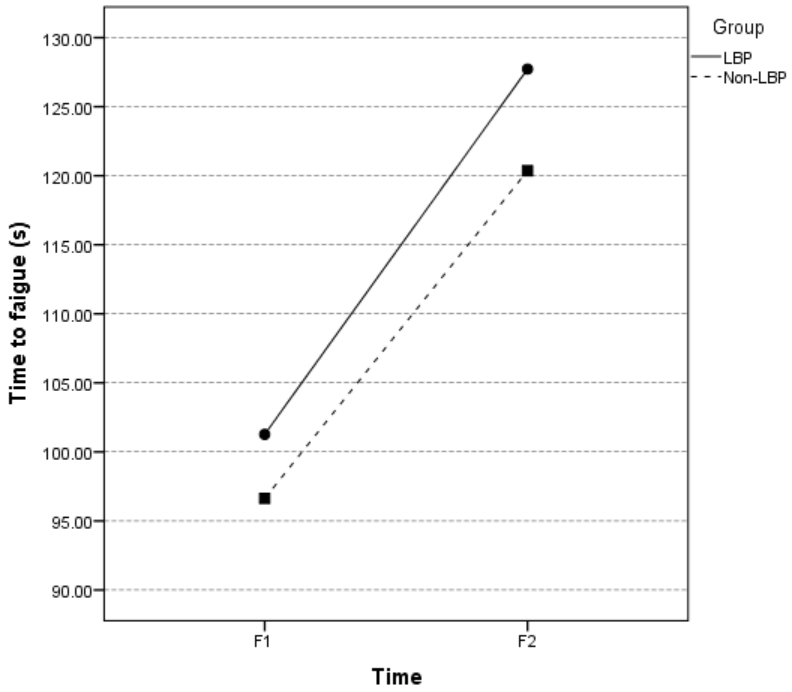

Figure 8. 3 Time to fatigue measurements for the low back pain group (LBP), and the non-back pain group; for fatigue task 1 (F1) fatigue task 2 (F2).

Table 8. 2 Pain pressure threshold measurements (PPT) for the low back pain group (LBP), and the non-back pain group; at fatigue task 1 (F1) fatigue task 2 (F2).

Group	**F1 PPT2 (kg/s)**	**F2 PPT3 (kg/s)**	**P-value**
LBP (mean/SD)	6.64 (2.42)	7.57 (2.35)	< .001
Non-LBP (Mean/SD)	6.53 (2.21)	8.74 (2.84)	< .001

Table 8. 3 Time to fatigue (seconds) measurements for the low back pain group (LBP), and the non-back pain group at fatigue task 1 (F1) fatigue task 2 (F2).

Group	**F1 (s)**	**F2 (s)**	**P-value**
LBP (mean/SD)	101.27 (46.94)	127.73 (46.94)	< .001
Non-LBP (Mean/SD)	96.64 (49.54)	120.36 (45.95)	< .001

8.4.3 Blood volume measurements

Table 8.4 below shows the means and standard deviations for the change in blood volume variables for tHb, Tsi, O2Hb and HHb for the LBP and non-LBP groups. The change in blood volume results indicated a main effect of time for the change in total blood volume, as measured by tHb at the lumbar paraspinal region F (2, 40) = 15.88, p = <.001; f = 0.89. There was no between groups effect. Pairwise comparisons revealed a significant increase in total blood volume post treatment (p = .002) and at F2 (p = .001) compared with the total blood volume at F1. There was no significant difference between the post treatment and F2 tHb result. There was also a main effect of time for Tsi, F (2, 40) = 19.28, p = < .001; f = 0.98. However, there was no between groups effects. Pairwise comparisons indicate a significantly increased tissue oxygenation levels at time post treatment (p =<.001) and F2 (p = .001) compared to F1, and between post treatment and F2 (p = 0.12). Results also indicated a main effect of time for O2Hb, F (2, 40) = 23.69, p = < .001; f = 1.08. There was no between groups effect. Pairwise comparisons indicated a significant increase in levels of O2Hb at time post treatment (p = < .001) and F2 (p = < .001) compared to F1. There was also a main effect of time for the change in HbDiff, F (2, 40) = 43.1, p = < .001; f = 1.47. There was no between groups effect. Levels of HHb showed no main effects of time or between group effects. Table 8.5 represents the effect sizes for the time to fatigue results

8.4.4 Correlation results

A Pearson's moment correlation was run to assess the relationship between the change in tHb, PPT and time to fatigue measurements. Preliminary analysis showed the relationship to be linear with both variables normally distributed.

There was a non-significant weak negative correlation between the change in tHb from f1 to F2 and the change in PPT from F1 to f2 r(9) = -.251, p = .457 for the LBP group

There was a non-significant weak correlation between the change in tHb from baseline to F2 and the change in PPT from baseline to f2 r(9) = .08, p = .981 for the LBP group

There was a non-significant weak negative correlation between the change in tHb from F1 to F2 and the change in TTF from f1 to f2 r(9) = -.001, p = .994 for the LBP group

There was a non-significant moderate correlation between the change in tHb from baseline to F2 and the change in TTF from baseline to f2 r(9) = .504, p = .114, for the NBP group

There was a non-significant weak negative correlation between the change in tHb from F1 to F2 and the change in PPT from pre to f2 r(9) = -.189, p = .578, for the NBP group

Table 8. 4 Change in blood volume from baseline at fatigue task 1 (F1) and fatigue task 2 (F2) for tissue saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (O2Hb), deoxyhaemoglobin (HHb), and haemoglobin difference (HbDiff), for the LBP and non-LBP groups.

Blood volume variable	LBP group				Non-LBP group			
	F1 (Mean ±SD)	**F2 (Mean ±SD)**	**95% confidence interval**	**P-Value**	**F1 (Mean ±SD)**	**F2 (Mean ±SD)**	**95% confidence interval**	**P-value**
Change in Tsi	-20.33 (18.91)	-11.8 (17.96)	-7.88 to 24.94	.291	-20.88 (19.68)	-12.18 (17.01)	-5.80 to 23.20	.225
Change in tHb	-4.16 (17.54)	14.89 (20.63)	2.02 to 36.08	.030	-4.81 (10.44)	30.30 (33.86)	12.82 to 57.39	.004
Change in O2Hb	-5.75 (15.02)	13.70(16.32)	5.50 to 33.40	.009	-10.66 (11.89)	17.68 (25.71)	10.52 to 46.16	.003
Change in HHb	1.59 (4.42)	1.20 (5.50)	-1.65 to 7.23	.205	5.86 (5.60)	6.91 (10.62)	-6.50 to 8.60	.775
Change in HbDiff	-7.33 (13.52)	12.50 (12.94)	8.60to 31.60	.002	-16.52 (15.37)	5.06 (21.10)	5.16 to 37.99	.013

Table 8. 5 Effect sizes (f) for main effects of time and between groups interaction effects interaction for pain pressure threshold (PPT), time to fatigue, tissue oxygen saturation (Tsi), total haemoglobin (tHb), oxyhaemoglobin (O2Hb), and haemoglobin difference (HbDiff).

Dependent variable	**Effect sizes (f) for main effects and observed power (1 – β err prob)**	
	Time	**Between groups Interaction**
PPT	f = 1.18 (1.0)	f = 0.40 (0.77)
Time to fatigue	f = 0.93 (0.99)	-
Change in O2Hb	f = 1.08 (1.0)	-
Change in tHb	f = 0.89 (0.99)	-
Change in Hb Diff	f = 1.47 (1.0)	-
Change in Tsi	f = 0.98 (0.99)	-

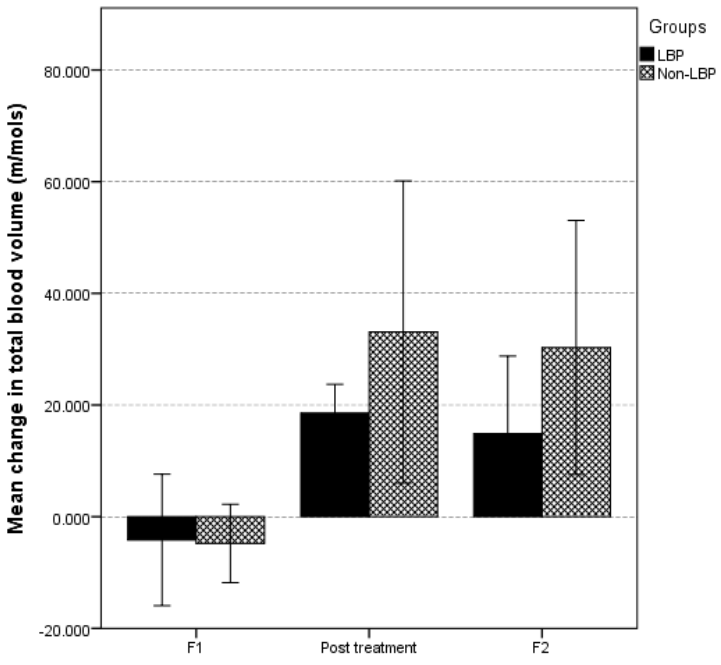

Figure 8. 4 Comparison of the change in tHb (total haemoglobin) between the LBP (dark bars) and non-LBP (checked bars) groups; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

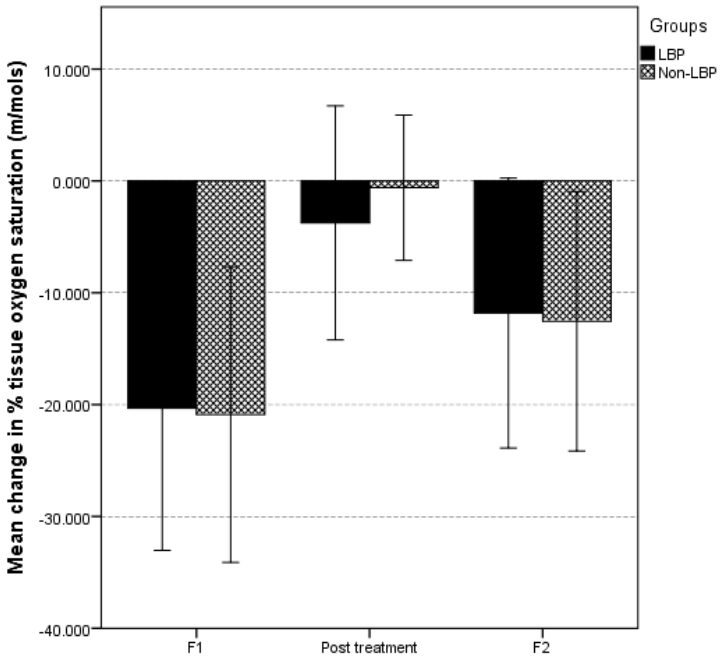

Figure 8. 5 Comparison of the change in Tsi (tissue oxygen saturation) between the LBP (dark bars) and non-LBP (checked bars) groups; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

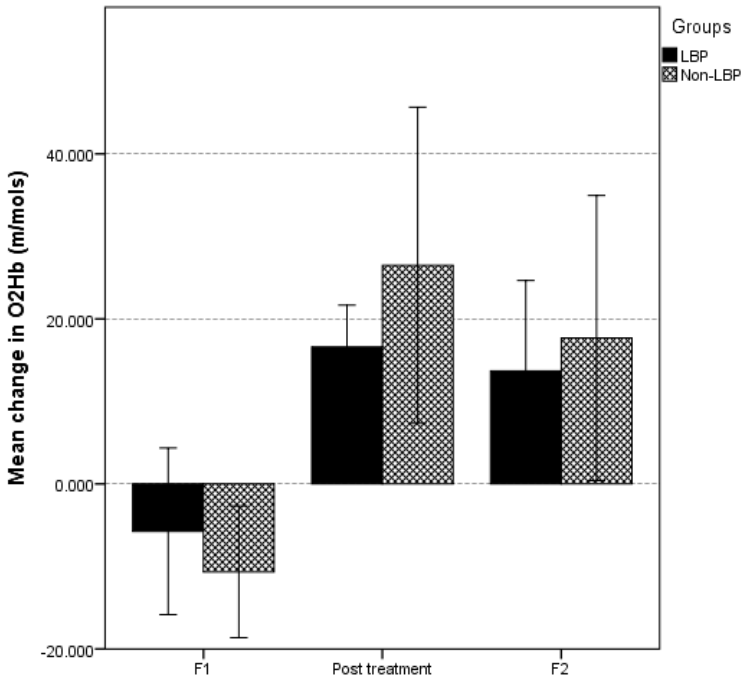

Figure 8. 6 Comparison of the change in O2hb (oxyhaemoglobin) between the LBP (dark bars) and non-LBP (checked bars) groups; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

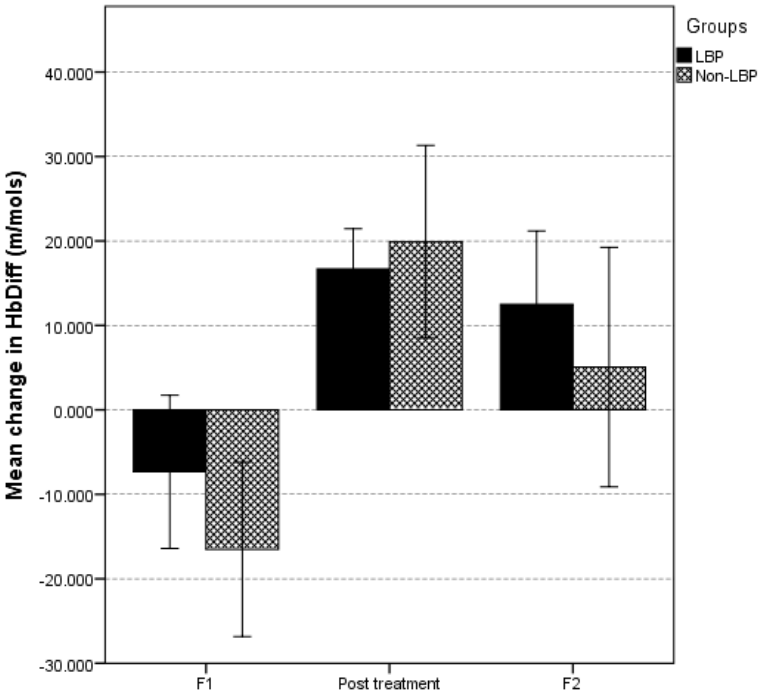

Figure 8. 7 Comparison of the change in Hbdiff (haemoglobin difference) between the LBP (dark bars) and non-LBP (checked bars) groups; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

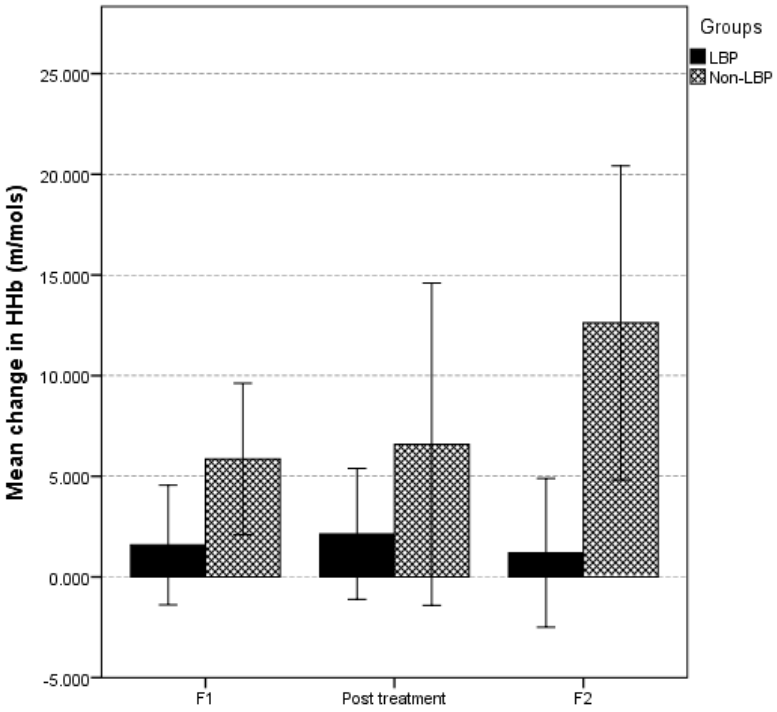

Figure 8. 8 Comparison of the change in HHb (deoxyhaemoglobin) between the LBP (dark bars) and non-LBP (checked bars) groups; at time points F1 (fatigue task 1), post treatment and F2 (fatigue task 2).

8.5 Discussion

The main results from the study indicate that the MT intervention resulted in significant improvements in levels of tHb, Tsi and O2H at the paraspinal musculature following a sustained isometric contraction to fatigue in subjects with and without non-specific LBP. Previous studies have supported the premise that massage therapy can improve blood flow and oxygenation to muscles (Mori *et al.*, 2004; Durkin *et al.*, 2006; Sefton *et al.*, 2010), however very few studies have found improvements utilising MT (Ramos-Gonzalez *et al.*, 2012; Shah *et al.*, 2017). Furthermore, where MT techniques have been identified as a potential post exercise passive recovery strategy (Arroyo-Morales, Olea, Martínez, *et al.*, 2008), to our knowledge this is the first study to identify the effects of MT on paraspinal blood volume and local muscle fatigue in subjects with and without LBP.

Total blood volume (tHb) was significantly increased at F2 compared to F1 for both groups. The change in tHb between scores at post treatment and F2 were not significantly different indicating that blood volume was maintained during the second fatigue task at a significantly higher level following the MT intervention. Although not significant, the change in tHb was greater for the non-LBP group at time post treatment and F2 with a percentage increase of 56% and 68% respectively. The change in Tsi revealed significantly improved tissue oxygen saturation rates at post treatment and F2 for both groups following the MT treatment. The reduction in Tsi during the first fatigue task is similar to that experienced in previous studies (Kell and Bhambhani, 2006; Olivier *et al.*, 2013), and may be attributed to an increase in the metabolism of active motor units (Kell and Bhambhani, 2006) and an increase in intramuscular pressure as the fatigue task developed (Jensen *et al.*, 1999). Although the Tsi reduced again at F2, the level of tissue oxygen saturation was still greater following the MT treatment compared to F1 indicating that tissue O2 saturation was also maintained following the treatment. In a similar trend to both tHb and Tsi, O2hb was also significantly improved at time post treatment and at F2

compared to F1. While levels of O2Hb dropped during F1 this appeared to be reversed following the treatment and levels were maintained significantly higher at F2. There was a slight drop in O2hb at F2 compared to the post treatment and may be attributed to metabolic demands of the fatigue task (Kell and Bhambhani, 2006; Olivier *et al.*, 2013). Although not significant, the level of O2Hb for the non-back pain group was greater than that of the LBP group. The percentage difference between groups O2Hb levels was 26% and 25% higher for the non-back pain group at time post treatment and F2 respectively. The non-significant differences in blood volume between groups in this study are in agreement with previous studies (Kell and Bhambhani, 2006; Dehner *et al.*, 2009).

Results for the time to fatigue tests revealed a significant increase in time to fatigue of 26% and 24% respectively from F1 to F2 for the LBP and non-LBP groups respectively following an acute bout of MT. Interestingly the LBP group's time to fatigue was greater than that of the non-LBP group but this may have been due to the relative times recorded at F1. The percentage difference revealed very little difference between the two groups. The results of this study are in agreement with previous studies identifying improvements in local muscle fatigue of the paraspinal musculature following massage therapy (Tanaka *et al.*, 2002; Mori *et al.*, 2004). However, studies supporting improvements in fatigue following myofascial techniques have either related to passive recovery of leg musculature (Arroyo-Morales, Olea, Martínez, *et al.*, 2008) or to interventions that influence the fascial network as opposed to those identified in this study (Álvarez-Álvarez *et al.*, 2014; Healey *et al.*, 2014). The effects of back fatigue on subjects with and without LBP have been previously been identified (Kell and Bhambhani, 2006; Candotti *et al.*, 2008; Dehner *et al.*, 2009; da Silva *et al.*, 2017). The results of the time to fatigue scores in this study are in agreement with Kell & Bhambhani (2006), who found no significant difference in time to fatigue scores following a Biering-Sorensen test, and Dehner et al. (2009), who found no significant differences in back muscle fatigue during an isometric extension exercise between rowers with LBP and healthy controls. However, other studies are not in

agreement suggesting that subjects with LBP show significantly greater fatigability of the paraspinal musculature compared to healthy controls (Candotti *et al.*, 2008; Olivier *et al.*, 2013; Parreira *et al.*, 2014; da Silva *et al.*, 2015).

The similarity in time to fatigue responses, and blood volume variables, may be due in part to the relative homogeneity of subjects in the LBP and non-LBP groups. The subjects for this study were taken from a student environment mainly studying sports courses and displayed similar physical characteristics (table 8.1). Factors such as age, physical fitness levels and muscle recruitment patterns have been known to influence the blood volume responses and fatigability of the paraspinal muscles (Kell and Bhambhani, 2006; da Silva *et al.*, 2015). Therefore, although specific fitness tests were not conducted health screening forms identified that both groups were active 2-3 time per week. Tissue oxygenation, blood volume and fitness of the paraspinal muscles are known to improve following exercise (Olivier *et al.*, 2013) . It is, therefore, possible that both groups were similarly conditioned.

Pain pressure threshold readings showed a significant improvement in pain scores following the intervention for both groups at F2 compared to F1, suggesting that MT in this study had a positive effect on LBP during a back-fatigue task. However, the scores for the non- LBP group's PPT responses were significantly greater than those of the LBP group at F2. The results of the study are in agreement with other studies on the effects of MT on pain (Castro-Sánchez *et al.*, 2011; Ramos-González *et al.*, 2012; Nitsure and Welling, 2014) , and those specifically relating to LBP (Cherkin *et al.*, 2011; Ajimsha, Daniel and Chithra, 2014). Pain reductions associated with MT have been attributed to a number of factors including improving the integrity of the fascial network and relieving pressure on pain sensitive structures (Ajimsha, Al-Mudahka and Al-Madzhar, 2015). Pain relief may also have been improved through reductions in muscular tension (Lewis *et al.*, 2012) and or intramuscular pressure of the paraspinal muscles (Kramer *et al.*, 2005) and improved

circulation (see 5.4.1 below). Furthermore pain reduction and increases in pain thresholds following massage have been associated with segmental pain modulation (Ajimsha, Daniel and Chithra, 2014) and descending pain inhibitory systems (Frey Law *et al.*, 2008) . As previously mentioned, the non-LBP group had significantly improved PPT scores compared to the LBP group. The PPT scores at baseline were not significantly different therefore, the changes observed at F2 may have been associated with population specific response to the intervention. LBP populations have been shown to exhibit greater levels of muscular tension (Lewis *et al.*, 2012), intramuscular pressure (Kramer *et al.*, 2005), thickened fascia (Langevin and Sherman, 2007) and enhanced pain sensitisation (O'Sullivan, 2012). Therefore, although pain was significantly improved following MT in both groups, the greater pain relief identified in the non-LBP population maybe attributable to individual factors associated with the LBP population.

8.5.2 Study Limitations

Table 8.5 highlights the effect sizes for the main effects of time and any between group interactions. A post hoc power analysis revealed the main effects of time achieved statistical power at the 0.8 level, (Faul *et al.*, 2007). The only between group interaction obtained within this study was the very small significant difference in PPT between groups. However, the sample size required to achieve power at 0.8 would have needed to have been 12. Furthermore, as in study 4 the assessment of local muscle fatigue could have included EMG measurements measure changes in amplitude or alterations in the power spectrum in the frequency domain. While time to fatigue provides us with an indication of muscle endurance capability EMG analysis would have provided the additional information with regard to neuromuscular or metabolic influences of MT on local muscle fatigue.

8.5.3 Conclusion

Integrated myofascial techniques applied to the parapsinal myofascial region significantly improve local peripheral blood volume, pain pressure threshold and significantly delays

the onset of paraspinal muscle fatigue in both LBP and non-LBP subjects. The results of this study may support the use of integrated myofascial techniques in the management of back pain and improve the fatigability of the paraspinal musculature.

9 Combination of results across studies

9.1 Subgrouping analysis and discussions

In order to further identify relationships and themes within this thesis a number of sub-analysis were conducted to pull subgroups from different studies for analysis. The following subgroup analysis were conducted:

1. Effects of Blood volume changes between the combined MT intervention of studies 2 and 3 compared with the combined control group interventions for studies 2 and 3.
2. Effects of blood volume changes between LBP and non-LBP subjects following an MT intervention conducted at study 5 compared to a control sham tens group from study 3

9.1.1 Effects of Blood volume changes between the combined MT intervention of studies 2 and 3 compared with the combined control group interventions for studies 2 and 3.

A One-way MANOVA identified that there was a significant difference between the combined dependent variables [F (3,51) = 25.32, P=<.001; Wilks = .402 η_p^2 = .6].

Tests of between subjects' effects revealed that there were significant differences between MT and control groups for changes in:

- O_2Hb F (1,53) = 73.94, P=<.001; η_p^2 = .6.
- tHb F (1,53) = 71.53, P=<.001; η_p^2 = .6.
- HHb F (1,53) = 23.60, P=<.001; η_p^2 = .35

Pairwise comparisons revealed that O_2Hb was significantly higher for the combined MT group compared to the combined control group [(95% CI, 18.65-30.24), P=<.001)]. tHb was significantly higher for the combined MT group compared to the combined control group [(95%CI,15.63-24.97); P=<.001]. HHb was significantly higher for the combined MT group compared to the combined control group [(95%CI, 2.56-5.7); P=<.001], The results were in keeping with the theme of the previous studies which indicated that an acute bout of MT significantly improved blood volume variables at the paraspinal region compared to control groups.

9.1.2 Effects of blood volume changes between LBP and non-LBP subjects, following an MT intervention conducted at study 5 compared to a control sham tens group from study 3

A one-way MANOVA revealed that there was a statistically significant difference between the groups for the combined dependent variables [F (8,54) = 13.14, P=<.001; Wilks = .115 η_p^2 = .66].

Tests of between subjects' effects revealed that there were the following significant differences between groups:

- There was a significant difference in the change in O_2Hb [F (2,30) = 5.83, P=<.001; η_p^2 = .28].
- There was a significant difference in the change in tHb [F (2,30) = 5.12, P=<.001; η_p^2 = .26].
- There was a significant difference in the change in tsi [F (2,30) = 71.2, P=<.001; η_p^2 = .82].

Tukay post hoc analysis revealed that there was a significant increase in O_2Hb for the non-LBP group compared to the control group (P=.006), and a significant increase in tHb for the non-LBP group compared to controls (P=.009). There were no significant

differences between the LBP and non-LBP groups or between the LBP group and controls. The above results further reflected the significant improvements in blood volume variables following an acute MT intervention to the paraspinal region. The non-significant difference between the LBP and back pain groups also reflected the results obtained in study 5. However, this sub-group analysis did reveal no significant difference in the blood volume variables between LBP and controls. While the results may have been affected by chronology bias it should be noted that there may be a possible reduced effect of MT on the LBP group not previously identified from the comparison of LBP and non-LBP. It is possible that LBP subjects do exhibit some reduction in BV changes due to population specific muscle function and tissue oxygenation (Kell and Bhambhani, 2006). Furthermore subjects with CLBP, as within this thesis, have been shown to exhibit increased intramuscular pressure which may contribute to blood flow occlusion and reduced tissue oxygenation (Kramer *et al.*, 2005; Dehner *et al.*, 2009). A more detailed discussion on the relationship between blood flow and muscle function in subjects with LBP follows below, however, this result could be subject to further investigation in future studies.

10 General discussion

The aims of this thesis were to investigate the Effects of MT techniques on muscle activity and blood volume changes at the paraspinal region in people with NSCLBP. The first study (chapter 4) within this thesis identified improvements in pain and dysfunction in subjects with non-specific LBP following a six-week intervention of clinical massage techniques. Further to this, improvements in the flexion relaxation ratio, as indicated by myoelectric silence of the paraspinal muscles, were also observed following both clinical massage and relaxation massage interventions. Although not significant, greater improvements in this muscle relaxation phenomenon were seen in the clinical massage group. It has been established that a number of techniques within the clinical massage classification may have contributed to these results. The variability of massage techniques within massage studies makes it difficult to attribute specific effects of massage on the LBP population. Therefore, the following studies attempted to investigate the relative contribution of specific treatment effects of one particular clinical massage technique within that classification model.

The effects of myofascial techniques (MT) on blood volume was determined through chapters 5 to 8. It was found that MT consistently improved blood volume, following an acute treatment, when compared to a traditional relaxation massage technique and kinesiotaping. This improvement in blood volume was evident in both the asymptomatic and low back pain populations. Chapters 7 and 8 attempted to identify the effects of MT on local muscle fatigue. In chapter 7, MT techniques significantly increased blood volume following paraspinal muscle fatigue in subjects with non-specific, CLBP. The application of MT to the parspinal region lead to a reduced fatigability of the muscle group as evidenced by an increase in time to fatigue and a lower RMS EMG amplitude during a fatigue task following the MT intervention. The improvements in time to fatigue might have been explained by the metabolic changes associated with an increase in blood volume during

the fatigue task (see chapter 7). Chapter 7 revealed no significant change in postural sway for LBP subjects following either the MT or control treatments. However, there was a trend towards an improvement following the MT treatment (see chapter 7). The same influence of MT on improved blood volume and time to fatigue were also seen in chapter 8, where blood volume and time to fatigue were consistently improved in both the back pain and non-back pain subject groups. Chapter 7 revealed that MT improved pain pressure threshold scores across time but were not different compared to controls. However, in chapter 8, it was identified that both the non-back pain and low back pain groups' pain pressure threshold scores improved during a fatigue task but the non-back pain's improvements were significantly greater than the LBP group. Table 9.1 below provides a summary of the effects of myofascial techniques (MT) for the dependent variables per study.

Table 9. 1 Summary of the effects of myofascial techniques (MT) for the dependent variables per study. Interpretation is provided in text.

Dependent variables	Study 2 A comparison between massage MT and RM techniques	Study 3 A comparison between MT and Kinesiotaping	Study 4 The effects on the LBP population compared to control	Study 5 A comparison between LBP and non-LBP groups
Blood volume **tHb**	Increased for MT group	Increased for MT group	Increased post MT intervention and following local muscle fatigue	Increased post treatment for both groups and following local muscle fatigue
O_2Hb	Increased for MT group	Increased for MT group	Increased post MT intervention and following local muscle fatigue	Increased post treatment for both groups and following local muscle fatigue
HHb	Increased for MT group	Increased for MT group	No Change	No change
HbDiff	n/a	No change	Increased post MT intervention and following local muscle fatigue	No change
Tsi	n/a	No change	Increased across time but not significantly between groups	Increased post treatment for both groups and following local muscle fatigue

Table 9. 2 Summary of the effects of myofascial techniques (MT) for the dependent variables per study. Interpretation is provided in text.

	Study 2	**Study 3**	**Study 4**	**Study 5**
Dependent variables	**A comparison between massage MT and RM techniques**	**A comparison between MT and Kinesiotaping**	**The effects on the LBP population compared to control**	**A comparison between LBP and non-LBP groups**
Pain pressure threshold	n/a	No interaction effect on asymptomatic subjects	Improved but no interactions effect	Improved for both groups
Time to fatigue	n/a	n/a	Improved following MT	Improved following MT for both groups
Postural sway	n/a	n/a	No change	n/a

The improvements in blood volume seen in chapters 5 to 8 suggest there may be a technique specific adaptation when using MT techniques as a form of manual therapy. As outlined in the literature review, these effects may have a basis in the neuroanatomy and neurophysiology of fascia. Chapter 5 and 6 highlight the mechanical and neurobiological effects of MT on blood volume. In this sense the changes in blood volume can be explained by the mechanical influence of MT techniques on the viscoelastic properties of fascia leading to greater compliance and mobility between layers. Such a change is said to correspond with a reduction in pressure on neurovascular structures alleviating restrictions to local blood volume (Barnes, 1997; Ajimsha, Al-Mudahka and Al-Madzhar, 2015). Similarly, the heat developed through such techniques may have a thixotropic influence on the ground substance of the extracellular matrix reducing the viscosity and densification of fascia (Ercole *et al.*, 2010; Pavan *et al.*, 2014). However, it should be pointed out that the heat from the physical act of touch alone; as with the application of relaxation massage in chapter 4; was not responsible for blood volume changes. From a neurobiological perspective, chapters 5 and 6 explained that the techniques employed within the intervention could have stimulated mechanoreceptors within the fascia capable of producing a parasympathetic mediated vasodilatory response to local blood vessels (Schleip, 2003a; Simmonds, Miller and Gemmell, 2012).

The improvements in blood volume and time to fatigue in chapters 7 and 8 further developed the potential implications and the utility of MT techniques for the lower back pain population. The mechanisms associated with this change have been addressed above. However, in the context of chronic LBP the potential for structural and mechanical changes to the fascia of the low back in the LBP population is thought to be the linked to plasticity responses of the fascia associated with immobility, overuse and or injury, resulting in either fibrosis or densification of the fascia (Langevin and Sherman, 2007; Pavan *et al.*, 2014). Previous studies have identified the effects of injury and inflammation on the development of fibrosis in fascia (Bouffard *et al.*, 2007; Stratton and Shiwen, 2010).

Furthermore, scaring of fascia has been shown to result in an increase in the proportion of type III collagen creating a possible localised diffusion barrier to oxygen and nutrients (Koźma *et al.*, 2000). Other studies have reported increased intramuscular fibrosis and collagen deposition as a result of immobilisation (Uebelhart *et al.*, 2000; Järvinen *et al.*, 2002) and patients with LBP exhibit reduced levels of activity due to pain related fear and fear avoidance behaviours (Cook, Brawer and Vowles, 2006; Damsgård *et al.*, 2007). While these levels of inactivity may be useful in the early stages of repair long term deconditioning, and altered movement patterns may lead to connective tissue fibrosis.

Pavan et al., (2014) further suggest that the mechanical properties of facial may be altered due to a densification of the ground substance. One of the potential mechanisms behind these changes may be the influence of mechanical stress on the GAGs of the extra cellular matrix and in particular Hyaluronic acid (HA) (Koźma *et al.*, 2000; Stecco *et al.*, 2013). As described in the literature review these GAGs are hydrophilic and contribute to the hydration and space filling properties of the fascia, as well as contributing to the efficient gliding of facial layers. Damage to the fascia has been reported to increase the content of GAG's (Koźma *et al.*, 2000) and an increase in the amount and viscosity of HA (Pavan *et al.*, 2014). Furthermore, alterations in HA production and an increase in loose connective tissue viscosity is associated with rises in tissue pH levels and inflammatory mediators (Kwong and Findley, 2014). Increased paraspinal fatigueability and ischemia have been shown to be present in the low back population (Kell and Bhambhani, 2006). Taken together it is possible to suggest the following.

1. Injury, overuse and immobilisation may influence the structure of the facia and have an effect on the fascial PG and GAG content.
2. Excessive or under production of HA can lead to densification of the fascia, leading to fascial restrictions and abnormal pressure on microvasculature and neural structures.
3. Fatigue mediated increases in pH levels may further exacerbate these changes.

The results of this study show improvements in blood volume and reduced fatigability of the paraspinal muscles. It is therefore possible that manual therapies such as MT may be used to facilitate blood volume and pH homeostasis and maintain the integrity of ground substance. In addition to the above, blood volume may have been enhanced through a reduction of paraspinal muscle tone and intramuscular pressure, following the MT treatment, facilitating re-oxygenation to previously ischemic muscles (Murthy *et al.*, 2001; Candotti *et al.*, 2008). The improvements in PPT for both groups may also be explained through changes to ischemic muscles and associated metabolite removal (Blangsted *et al.*, 2005; Kell and Bhambhani, 2006). The corresponding improvements in time to fatigue suggests a metabolic influence within the muscle that may help to maintain muscle function in the LBP population and offset the effects of fatigue and associated implications to stability of the spine. The improvements in time to fatigue have been explained by the increased delivery of oxygen and removal of metabolites following and during a fatigue task (Mori *et al.*, 2004; Kell and Bhambhani, 2006). Furthermore, the effects of MT techniques on time to fatigue were reflected in chapter 8, however this response did not differ between back pain and non-back pain populations. The explanation for this may be linked to the type and severity of LBP in the subject groups within chapter 8.

10.1 Relationship between blood volume, time to fatigue, PPT and CLBP

The significant improvements in time to fatigue and pain following the MT intervention may, in part, be due to the improvements in blood volume and oxygenation to the paraspinal musculature and the associated metabolic changes that come with this. Improving circulation and recovery from muscle fatigue have been identified as contributing to pain relief (Mori *et al.*, 2004; Kachanathu *et al.*, 2014). The improvements in blood volume variables within study 7 and 8 support previous suggestions associated with improvements in circulation and may therefore have contributed pain relief through a reduction in ischemia associated with sustained muscle contraction. Indeed, the removal

of metabolites of static or ischemic contractions; such as lactic acid, potassium, bradykinin and serotonin; may also be associated with reductions in muscular tone and pain relief via the gamma feedback loop (Knutson, 2000). It has been proposed that increased intramuscular concentration of these metabolites stimulate gamma motor neurones and muscle spindle afferents, thereby increasing muscle stiffness (Knutson, 2000). The fatigue task in study 7 and 8 employed the Biering-Sorensen test is proposed to equate to 40-50% of MVC of the paraspinal muscles (Kell and Bhambhani, 2006). Such contractions are said to contribute to an increase in intramuscular pressure, chronic compartment syndrome, compromised blood volume and an associated build-up of metabolites (Blangsted *et al.*, 2005; Kell and Bhambhani, 2006; Candotti *et al.*, 2008). Therefore, the application of MT and the corresponding increase in blood volume may have facilitated re-oxygenation of the paraspinal region and reduced the effects of ischemia, metabolite build up and pain. This also has implications for the fatigue results in this study. Intramuscular pressure, Ischemia and associated alterations in muscle pH have been shown to facilitate muscle fatigue (Murthy *et al.*, 2001; Candotti *et al.*, 2008; Broxterman *et al.*, 2015). Therefore, it is plausible to suggest that the improvements in blood volume following the MT intervention may have contributed to metabolic changes that could offset the development in fatigue and explain the significant improvements in time to fatigue found in this study.

Throughout this thesis MT was consistently shown to improve blood volume variables at the paraspinal region. However, pain pressure threshold improvements could not be attributed to MT interventions only. Correlation analysis revealed no significant correlation between changes in blood flow PPT and time to fatigue. Therefore, the clinical meaningfulness of MT may lie in the biopsychosocial model of LBP and away from the tissue pathology. Previous discussion in sections 2.1.3 and 2.5 have identified that psychological factors and emotional distress are associated with the transition from acute and subacute LBP to CLBP. A key feature of CLBP within the biopsychosocial model is

pain related fear of movement with a consequent decrease in physical activity. The fascia of the back has been shown to be susceptible to micro injury and inflammation due to the abundance of nociceptive nerve endings within the TLF and subjects with CLBP have been shown to have generalised augmented pain sensitivity linked to abnormal central pain processing (Langevin and Sherman, 2007). Both pain and the fear of pain has been shown to alter trunk muscle activation and subjects with LBP have been shown to have reduced back muscle endurance, muscle spasm or co-contraction as outlined above. Furthermore, it has been identified that hypermobility and hypomobility can cause abnormal changes in the connective tissue. Overloading of the connective tissue is associated microinjury and inflammation, whereas hypomobility can lead to connective tissue fibrosis, adhesions and contractures, with the presence of facial fibrosis linked to the concurrent presence of inflammation and hypo-oxygenation. Furthermore, densification and connective tissue fibrosis has been shown to increase pressure on neurovascular structures with blood and lymphatic flow chronically compromised (Ajimsha, Al-Mudahka and Al-Madzhar, 2015) and vulnerable to unusual muscle activity (Langevin and Sherman, 2007).

Therefore, subjects with CLBP may exhibit connective tissue adaptations linked to involving altered movement patterns, connective tissue remodelling, connective tissue inflammation, peripheral and central sensitisation. The Effects of MT techniques for LBP subjects has been attributed to three potential mechanisms: (1) the mechanical effects of MT altering the mechanical properties of the lumbar connective tissues, (2) heat mediated thixotropic changes to the ground substance of the connective tissue and, (3) neurobiological mechanisms associated with the stimulation of mechanoreceptors responsible for vasodilation and reductions in muscle tone. Taken together the Effects of MT can be utilised to promote an alteration in the abnormal plastic responses of connective tissue to LBP. This includes a restructuring of connective tissue to improve peripheral blood volume at the paraspinal muscles; an alleviation of pressure on

neurovascular structures and pain associated with this phenomenon; a reduction in any abnormal muscle tone that may contribute to pain and or muscle fatigue. This may have clinical meaningfulness as a possible treatment to facilitate factors associated with pain, fear of pain and altered movement patterns associated with the biopsychosocial model for LBP as part of a behaviour modification and movement re-education treatment protocol.

10.2 Sources of Bias within experimental chapters 7 and 8

Bias can be described as any tendency which prevents unprejudicial consideration of a question (Pannucci and Wilkins, 2011). The following section will outline the sources of bias within the experimental chapters 7 and 8 and focus on pre-trial bias, data collection bias and confounding associated with these chapters. Both chapters 7 and 8 may have been subject to selection bias as part of the study design process. Within both studies, the study population was defined in terms of the selection criteria for LBP and non-LBP populations, however, the criteria used to select and enrol subjects did not meet the criteria used to define the LBP population. Recruitment of subjects were mainly from a university sports science course background. This excluded the wider population and therefore not wholly representative of the LBP population as defined by the selection criteria. Selection and Inclusion bias therefore exists as the population group within these two studies may have different physical characteristics, and preconceived thoughts regarding the interventions provided within these studies and therefore leading to confounding effects of association. Furthermore, the allocation of participants into intervention groups were not based on a prospective design which affected the randomisation process affecting selection bias in terms of the similarity between groups. Foer future research participation selection should be more accurately planned in accordance with the aims and criteria associated with the study population.

The choice of participants within these chapters may also have led to channelling bias as potential prognostic factors or degree of subjects reported conditions may differ from

those of the wider population suffering from chronic LBP who may be perceived as being higher risk. Therefore, a selection bias may exist for the subject participants within the studies as the therapist involved in applying the MT treatments may have altered the depth and pressure of MT treatments based on the relative perception of low risk subjects within those chapters. As depth and pressure are significant components of the application of MT techniques (Barnes, 1997), the effects of MT on blood volume changes may have been influenced by such bias.

Standardised protocols were implemented to standardise the MT and control interventions and the data collection process to reduce inter-observer variability. One trained therapist was used to provide the interventions within this study and one person was assigned to the data collection and analysis process. However, blinding of study personnel to the subject's exposure and outcome status was not possible within these studies. Future designs would include an independent examiner blinded to the type of intervention performed.

Data collection recall bias may have also existed within these studies. Subjects with LBP had to recall information with regard to pain perception over a period of time relating to their chronicity of their condition and thus affecting the reliability of information provided. Furthermore, subject perception of the treatment / intervention type may have influenced the effect of the intervention in terms of a placebo effect. Where subjects have knowledge of an intervention and its effect this may influence the reporting of PPT within these studies due to their perception of the potential outcome of the intervention. Another source of bias linked with both study design and data collection was the use of historic LBP subjects used as a comparison with non-LBP subjects. This chronology bias may have acted as a source of inequality between groups in terms of NIRS sensor placements

and PPT protocols. NIRS measures are sensitive to minimal movement of sensors and muscle contractions (Ferrari and Mottola, 2004).

During the delivery of the treatment interventions, performance bias may have existed within the two clinical chapters. Technical variability in terms of strokes and depth of pressure applied during the MT treatments may have varied between subjects. These parameters were not measured therefore differences in performance of the therapist may have confounded the results of the blood volume measurements within these studies and between groups and time. Furthermore, differences inherent in the intervention procedures may have contributed to the different blood volume changes between MT and control results. The MT intervention involved subjects moving from prone to side lying and supine positions; whereas control groups maintained a prone position. The movement of the subjects within the MT intervention may have led to postural effects which have been associated with changes in blood volume variables (Dupeyron *et al.*, 2009).

Further confounding features within these studies should also be addressed. Subjects exposure to different levels of activity may be independently associated with the outcome of interest. For example muscular characteristics of paraspinal muscles may influence baseline levels of blood volume variables and responses to intervention effects (Yoshihito *et al.*, 2005). Furthermore, intrinsic factors such as physiological and biochemical characteristics of a muscle may have confounded the results of the EMG measurements within chapter 7. Differences such as number of active motor units at any particular time of contraction, fibre type composition and blood flow to a muscle are difficult to control but may have influenced the sEMG results (Luca, 1997).

10.3 Practical implications

Given the increasing popularity of massage as a form of complementary and alternative therapy MT may be the technique of choice in order to improve blood volume or blood volume in order to facilitate a treatment aim. The overall implication from the results of this thesis is that practitioners can integrate these techniques into any manual therapy in the treatment of musculoskeletal conditions. The translation of these results into practice is further improved by the use of an integrated approach within these chapters. These provide a more realistic approach to the design of many manual therapy treatments. One of the criticisms associated with massage research is the confounding effects of variability in technique and dosage. However, in practice many manual therapists include a number of techniques across various massage classifications and based on a number of treatment aims and goals (Sherman *et al.*, 2006). Therefore, the effects on blood volume found with MT techniques can be incorporated into the clinical reasoning process should improvements in blood volume be required. The comparison between MT and RM in chapter 5 and MT and kinesiotape in chapter 6 provides the most obvious practical implication to achieve this effect.

The results of this thesis also have some important implications for the treatment of LBP. It has been clearly identified that blood volume to the paraspinal lower back musculature can be improved through the use of MT techniques. Thus, the implications of this may lie in the use of MT techniques as a standalone therapy to facilitate healing of injured tissue particularly in the subacute phase of healing. Furthermore, reductions in pain perception may contribute to improvements in psychosocial profiles and chronicity risk factors (Trost *et al.*, 2012). Improvements in perceptions of pain and mobility of lumbar fascia may help to facilitate movement. Such adaptations could be incorporated into an integrated approach to reduce the negative effects of fear voidance and kinesiophobia. However, it is the combination of improved blood volume and the effect on local muscle fatigue that may

be of greatest practical importance. Subjects with LBP have been shown to exhibit reduced blood volume and oxygenation and earlier manifestations of fatigue to these muscles (Kovacs *et al.*, 2001; Candotti *et al.*, 2008). Therefore, MT techniques may be utilised to offset the effects of muscle fatigue and its effects of spinal stability and pain. Furthermore, there is a potential for its use within a multidisciplinary treatment program particularly where exercise is used in combination with other modalities. Subjects experiencing fatigue and pain may cease to adhere to exercise therapy, yet It has been well documented that exercise may help to condition lower back muscles and improve their aerobic capacity (Olivier *et al.*, 2013). Therefore, the combination of fatigue and pain reducing manual therapies may contribute to exercise adherence and exercise progression.

One of the possibilities associated with the improved pain perception in the NBP group compared to the LBP group may be linked with a generalised embodiment of pain and interoceptive sensitivity. Described as the sense of physiologic condition of the body or an ability to concisely perceive signals from the body, subjects with high levels of interoceptive sensitivity have been shown to exhibit reduced pain tolerance level and increases in autonomic reactivity in the form of heightened sympathetic activity (Pollatos, Fustos and Critchley, 2012). LBP subjects within this study may therefore have had reduced pain pressure threshold scores due to heightened pain perception associated with their condition. Evidence of interoceptive differences between the LBP population and healthy subjects have been identified (Mehling *et al.*, 2013). If interoceptive sensitivity can guide an individual's response to noxious stimuli (Pollatos, Fustos and Critchley, 2012), then it would be interesting to identify the effects of interoceptive sensitivity on pain reliving treatments.

10.4 Future directions and implications for research

Future studies should look to identify the relative contribution of the various myofascial techniques to improvements in blood volume. The current thesis took an integrated approach. Identification of similar effects with individual techniques may have time and cost saving implications. Similarly, a dose response study would enhance the techniques utility. Future research into the effects of MT on blood volume should also look to identify the mechanisms proposed to contribute to these changes. These include structural changes to the connective tissues, autonomic nervous system changes, biochemical analysis of the ground substance and chemical markers associated with muscle fatigue and or ischemia. Within this thesis the subjects with LBP were chosen from a largely student population. However, evidence exists to suggest that LBP sub classification strategies should be incorporated into LBP outcome research (Fersum *et al.*, 2010). This may be particularly appropriate for those subjects with pain adaptation profiles reduced flexion relaxation profiles or those with chronic functional compartment syndrome (Martin Descarreaux *et al.*, 2008; Dehner *et al.*, 2009; D'hooge *et al.*, 2013). It is possible that better sub classification could further target the use of the technique more accurately. Furthermore, the use of these techniques could be investigated as part of a multidisciplinary intervention as outlined above. Multi-disciplinary approaches to back pain have been shown to be more effective than massage on its own (Finn, 2013; Kamper *et al.*, 2015), Therefore incorporating MT techniques into this approach would be important to identify how these techniques work synergistically with LBP treatment and rehabilitation.

10.5 Conclusions

The use of clinical massage techniques in the form of myofascial manual therapy has been shown to improve blood volume variables and time to fatigue in subjects with and without LBP. Local blood volume is significantly increased to the lumbar paraspinal myofascial region in response to integrated myofascial techniques when compared to

relaxation massage and kinesiology taping techniques. The effects of these interventions on pain and the relationship between these outcomes requires further investigation. However, the results suggest a possible utility for population groups where reduced paraspinal blood flow, muscular co-contraction and increased fatigability of the paraspinal muscles are concerned. Therefore, patients with LBP receiving practitioner-based therapies such as massage may benefit from the specific effects of integrated myofascial techniques identified within these studies. These findings may be of use for those looking to add second line treatments to facilitate a multidimensional approach to treating LBP within a biopsychosocial paradigm.

References

Adams, R., White, B. and Beckett, C. (2010) 'The effects of massage therapy on pain management in the acute care setting.', *International journal of therapeutic massage & bodywork*, 3(1), pp. 4–11.

Airaksinen, O., Brox, J. I., Cedraschi, C., Hildebrandt, J., Klaber-Moffett, J., Kovacs, F., Mannion, A. F., Reis, S., Staal, J. B., Ursin, H. and Zanoli, G. (2006) 'Chapter 4. European guidelines for the management of chronic nonspecific low back pain.', *European spine journal :* 15 Suppl 2, pp. S192-300.

Ajimsha, M. S., Al-Mudahka, N. R. and Al-Madzhar, J. a. (2015) 'Effectiveness of myofascial release: systematic review of randomized controlled trials', *Journal of Bodywork and Movement Therapies*. Elsevier Ltd, 19(1), pp. 102–112.

Ajimsha, M. S., Daniel, B. and Chithra, S. (2014) 'Effectiveness of Myofascial release in the management of chronic low back pain in nursing professionals', *Journal of Bodywork and Movement Therapies*. Elsevier Ltd, 18(2), pp. 273–281.

Ali, A., Kahn, J., Rosenberger, L. and Perlman, A. I. (2012) 'Development of a manualized protocol of massage therapy for clinical trials in osteoarthritis.', *Trials*. Trials, 13(1), p. 185.

Allen, D. G., Lamb, G. D. and Westerblad, H. (2008) 'Skeletal muscle fatigue: cellular mechanisms', *Physiological Reviews*. Am Physiological Soc, 88(1), pp. 287–332.

Álvarez-Álvarez, S., José, F. G.-M. S., Rodríguez-Fernández, a L., Güeita-Rodríguez, J. and Waller, B. J. (2014) 'Effects of Kinesio® Tape in low back muscle fatigue: randomized, controlled, doubled-blinded clinical trial on healthy subjects.', *Journal of back and musculoskeletal rehabilitation*, 27(2), pp. 203–12.

Arroyo-Morales, M., Fernandez-Lao, C., Ariza-Garcia, A., Toro-Velasco, C., Winters, M., Diaz-Rodriguez, L., Cantarero-Villanueva, I., Huijbregts, P. And Cesar., F.-D.-L.-P. (2011) 'Psychophysiological Effects Of Preperformance Massage Before Isokinetic Exercise', *Journal of Strength and Conditioning Research*, 25(2), pp. 481–488.

Arroyo-Morales, M., Olea, N., Martínez, M. M., Hidalgo-Lozano, A., Ruiz-Rodríguez, C. and Díaz-Rodríguez, L. (2008) 'Psychophysiological Effects of Massage-Myofascial Release After Exercise: A Randomized Sham-Control Study', *The Journal of Alternative and Complementary Medicine*. Mary Ann Liebert, Inc. 140 Huguenot Street, 3rd Floor New Rochelle, NY 10801-5215 USA, 14(10), pp. 1223–1229.

Arroyo-Morales, M., Olea, N., Martinez, M., Moreno-Lorenzo, C., Daz-Rodrguez, L. and Hidalgo-Lozano, A. (2008) 'Effects of myofascial release after high-intensity exercise: a randomized clinical trial', *Journal of manipulative and physiological therapeutics*. Mosby, Inc., 31(3), pp. 217–223.

Astfalck, R. G., O'Sullivan, P. B., Smith, A. J., Straker, L. M. and Burnett, A. F. (2013) 'Lumbar spine repositioning sense in adolescents with and without non-specific chronic low back pain - An analysis based on sub-classification and spinal regions.', *Manual therapy*, 18(5), pp. 410–417.

Avela, J., Kyrolainen, H. and Komi, P. V (1999) 'Altered reflex sensitivity after repeated and prolonged passive muscle stretching', *Journal of Applied Physiology*, 86(4), pp. 1283–1291.

Balagué, F., Mannion, A. F., Pellisé, F. and Cedraschi, C. (2012) 'Non-specific low back pain.', *Lancet*, 379(9814), pp. 482–91.

Barnes, M. F. (1997) 'The basic science of myofascial release: morphologic change in connective tissue', *Journal of Bodywork & Movement Therapies*. Elsevier, 1(4), pp. 231–238.

Van Beekvelt, M. C., Borghuis, M. S., van Engelen, B. G., Wevers, R. a and Colier, W. N. (2001) 'Adipose tissue thickness affects in vivo quantitative near-IR spectroscopy in human skeletal muscle.', *Clinical science (London, England : 1979)*, 101(1), pp. 21–28.

Behm, D. G., Peach, A., Maddigan, M., Aboodarda, S. J., DiSanto, M. C., Button, D. C. and Maffiuletti, N. a. (2013) 'Massage and stretching reduce spinal reflex excitability without affecting twitch contractile properties', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 23(5), pp. 1215–1221.

Benjamin, M. (2009) 'The fascia of the limbs and back-a review', *Journal of anatomy*, 214(1), pp. 1–18.

Bervoets, D. C., Luijsterburg, P. A., Alessie, J. J., Buijs, M. J. and Verhagen, A. P. (2015) 'Massage therapy has short-term benefits for people with common musculoskeletal disorders compared to no treatment: a systematic review.', *Journal of physiotherapy*. Korea Institute of Oriental Medicine, 61(3), pp. 106–16.

Biering-Sørensen, F. (1984) 'Physical measurements as risk indicators for low-back trouble over a one-year period.', *Spine*, 9(2), pp. 106–19.

Blangsted, A. K., Vedsted, P., Sjøgaard, G. and Søgaard, K. (2005) 'Intramuscular pressure and tissue oxygenation during low-force static contraction do not underlie muscle fatigue', *Acta Physiologica Scandinavica*, 183(4), pp. 379–388.

Bogduk, N. (1995) 'The anatomical basis for spinal pain syndromes.', *Journal of manipulative and physiological therapeutics*, 18(9), pp. 603–5.

Bombardier, C. (2000) 'Outcome Assessments in the Evaluation of Treatment of Spinal dissorders: Summary and General Recommendations', *Spine (Phila Pa 1976)*, 25(24), pp. 3100–3103.

Boucher, J.-A., Abboud, J., Nougarou, F., Normand, M. C. and Descarreaux, M. (2015) 'The Effects of Vibration and Muscle Fatigue on Trunk Sensorimotor Control in Low Back Pain Patients.', *PloS one*, 10(8), p. e0135838.

Bouffard, N. A., Cutroneo, K. R., Badger, G. J., White, S. L., Buttolph, T. R., Ehrlich, H. P., Stevens-tuttle, D. and Langevin, H. M. (2007) 'Tissue Stretch Decreases Soluble TGF-b1 and Type-1 Procollagen in Mouse Subcutaneous Connective Tissue : Evidence From Ex Vivo and In Vivo Models', *Journal of Cellular Physiology*, (May), pp. 389–395.

Brockel J. Clark M.E. Kori S.H. (1996) 'Kinesiophobia and chronic pain: psychometric characteristics and factor analysis of the tampa scale.', *American Pain society Abstracts.*, 15, p. 77.

Brosseau, L., Wells, G. a., Poitras, S., Tugwell, P., Casimiro, L., Novikov, M., Loew, L., Sredic, D., Clément, S., Gravelle, A., Kresic, D., Hua, K., Lakic, A., Ménard, G., Sabourin, S., Bolduc, M.-A., Ratté, I., McEwan, J., Furlan, A. D., Gross, A., Dagenais, S., Dryden, T., Muckenheim, R., Côté, R., Paré, V., Rouhani, A., Léonard, G., Finestone, H. M., Laferrière, L., Haines-Wangda, A., Russell-Doreleyers, M., De Angelis, G. and Cohoon, C. (2012) 'Ottawa Panel evidence-based clinical practice guidelines on therapeutic massage for low back pain', *Journal of Bodywork and Movement Therapies*, 16(4), pp. 424–455.

Broxterman, R. M., Craig, J. C., Smith, J. R., Wilcox, S. L., Jia, C., Warren, S. and Barstow, T. J. (2015) 'Influence of blood flow occlusion on the development of peripheral and central fatigue during small muscle mass handgrip exercise', *J Physiol*, 593. 17(593), pp. 4043–4054.

Brumagne, S., Cordo, P., Lysens, R., Verscheuren, S. and Swinnen, S. (2000) 'The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain.', *Spine*, 25(8), pp. 989–994.

Brumagne, S., Janssens, L., Claeys, K. and Pijnenburg, M. (2013) 'Altered variability in proprioceptive postural strategy in people with recurrent low back pain', in Hodges, P., Cholewicki, J., and Van Dieen, J. (eds) *Spinal Control: The Rehabilitation of Back Pain*. Curchill Livingstone Elsevier, pp. 135–144.

Bruyère, O., Demoulin, M., Beaudart, C., Hill, J. C., Maquet, D., Genevay, S., Mahieu, G., Reginster, J.-Y., Crielaard, J.-M. and Demoulin, C. (2014) 'Validity and Reliability of the French Version of the STarT Back Screening Tool for Patients With Low Back Pain', *Spine*, 39(2), pp. E123–E128.

Bunketorp, L., Carlsson, J., Kowalski, J. and Stener-victorin, E. (2005) 'Evaluating the reliability of multi-item scales: A non-parametric approach to the ordered categorical structure of data collected with the Swedish version of the Tampa Scale for Kinesiophobia and the Self-Efficacy Scale', *Journal of Rehabilitation Medicine*, 37(5), pp. 330–334.

Burckhardt, C. S. and Jones, K. D. (2003) 'Adult measures of pain: The McGill Pain Questionnaire (MPQ), Rheumatoid Arthritis Pain Scale (RAPS), Short-Form McGill Pain Questionnaire (SF-MPQ), Verbal Descriptive Scale (VDS), Visual Analog Scale (VAS), and West Haven-Yale Multidisciplinary Pain Inven', *Arthritis & Rheumatism*, 49(S5), pp. S96–S104.

Cambron, J. A., Dexheimer, J. and Coe, P. (2006) 'Changes in Blood Pressure After Various Forms of Therapeutic Massage: A Preliminary Study', *The Journal of Alternative and Complementary Medicine*, 12(1), pp. 65–70.

Campbell, C. M. and Edwards, R. R. (2009) 'Mind-body interactions in pain: the neurophysiology of anxious and catastrophic pain-related thoughts.', *Translational research : the journal of laboratory and clinical medicine*, 153(3), pp. 97–101.

Candotti, C. T., Loss, J. F., Pressi, A. M. S., Castro, F. A. D. S., La Torre, M., Melo, M. D. O., Araújo, L. D. and Pasini, M. (2008) 'Electromyography for assessment of pain in low back muscles.', *Physical therapy*, 88(9), pp. 1061–1067.

Castro-Sánchez, A. M., Matarán-Peñarrocha, G. a, Arroyo-Morales, M., Saavedra-Hernández, M., Fernández-Sola, C. and Moreno-Lorenzo, C. (2011) 'Effects of myofascial release techniques on pain, physical function, and postural stability in patients with fibromyalgia: a randomized controlled trial.', *Clinical rehabilitation*, 25(9), pp. 800–813.

Celenay, S. T., Kaya, D. O. and Akbayrak, T. (2016) 'Cervical and scapulothoracic stabilization exercises with and without connective tissue massage for chronic mechanical neck pain: A prospective, randomised controlled trial', *Manual Therapy*. Elsevier Ltd, 21, pp. 144–150.

Cherkin, D. C., Sherman, K. J., Kahn, J., Erro, J. H., Deyo, R. a, Haneuse, S. J. and Cook, A. J. (2009) 'Effectiveness of focused structural massage and relaxation massage for chronic low back pain: protocol for a randomized controlled trial.', *Trials*, 10, p. 96.

Cherkin, D. C., Sherman, K. J., Kahn, J., Erro, J. H., Deyo, R. a, Haneuse, S. J. and Cook, A. J. (2011) 'A Comparison of the Effects of 2 Types of Massage and Usual Care on Chronic Low Back Pain', *Annals of Internal Medicine*, 155, pp. 1–9.

Chou, R. (2010a) 'Low back pain (chronic).', *BMJ clinical evidence*, 10(1116), pp. 1–41.

Chou, R. (2010b) 'Musculoskeletal disorders Low back pain (chronic)', *Clinical Evidence Online*, 10:1116(October 8), p. P1116.

Chou, R., Deyo, R., Friedly, J., Skelly, A., Hashimoto, R., Weimer, M., Fu, R., Dana, T., Kraegel, P., Griffin, J., Grusing, S. and Brodt, E. D. (2017) 'Nonpharmacologic therapies for low back pain: A systematic review for an American College of physicians clinical practice guideline', *Annals of Internal Medicine*, 166(7), pp. 493–505.

Colloca, C. J. and Hinrichs, R. N. (2005) 'The biomechanical and clinical significance of the lumbar erector spinae flexion-relaxation phenomenon: a review of literature.', *Journal of manipulative and physiological therapeutics*, 28(8), pp. 623–31.

Conn, I., Yadav, S., Daly, J., Jaffer, M. and Marshall, A. H. (1986) 'Transcutaneous electrical nerve stimulation following appendicectomy: the placebo effect.', *Annals of the Royal College of Surgeons of England*, p. 191.

Cook, A. J., Brawer, P. a and Vowles, K. E. (2006) 'The fear-avoidance model of chronic pain: validation and age analysis using structural equation modeling.', *Pain*, 121(3), pp. 195–206.

Criswell, E. (2011) *Cram's Introduction to Surface Electromyography*, *Training*.

Crombez, G., Vlaeyen, J. W., Heuts, P. H. and Lysens, R. (1999) 'Pain-related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability.', *Pain*, 80(1–2), pp. 329–39.

Cuissard, N., Duchateau, J. and Hainaut, K. (1988) 'Muscle stretching and motoneuron excitability', *European Journal of Applied Physiology and Occupational Physiology*. Springer-Verlag, 58(1–2), pp. 47–52.

D'hooge, R., Hodges, P., Tsao, H., Hall, L., Macdonald, D. and Danneels, L. (2013) 'Altered trunk muscle coordination during rapid trunk flexion in people in remission of recurrent low back pain.', *Journal of Klectromyography and kinesiology*, 23(1), pp. 173–81.

Damsgård, E., Fors, T., Anke, A. and Røe, C. (2007) 'The Tampa Scale of Kinesiophobia: A Rasch analysis of its properties in subjects with low back and more widespread pain.', *Journal of rehabilitation medicine : official journal of the UEMS European Board of Physical and Rehabilitation Medicine*, 39(9), pp. 672–8.

Davidson, M. a, Tripp, D. a, Fabrigar, L. R. and Davidson, P. R. (2008) 'Chronic pain assessment: a seven-factor model.', *Pain research & management : the journal of the Canadian Pain Society = journal de la société canadienne pour le traitement de la douleur*, 13(4), pp. 299–308.

Davidson, M. and Keating, J. L. (2002) 'Research Report A Comparison of Five Low Back Disability Questionnaires : Reliability', *Physical Therapy*, 82, pp. 8–24.

Dehner, C., Schmelz, A., Völker, H.-U., Pressmar, J., Elbel, M. and Kramer, M. (2009) 'Intramuscular pressure, tissue oxygenation, and muscle fatigue of the multifidus during isometric extension in elite rowers with low back pain.', *Journal of sport rehabilitation*, 18(4), pp. 572–81.

Delitto, A., George, S., Whitman, J., Sowa, G., Shekelle, P., Denniger, T. and Godges, J. (2012) 'Low Back Pain Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopaedic Section of the American Physical Therapy Association', *Journal of Orthopaedic and Sports Physical Therapy*, 42(4), pp. A1–A157.

Demoulin, C. (2012) 'Muscular Performance Assessment of Trunk Extensors: A Critical Appraisal of the Literature.', *Low Back Pain*, pp. 141–166.

Demoulin, C., Vanderthommen, M., Duysens, C. and Crielaard, J. M. (2006) 'Spinal muscle evaluation using the Sorensen test: A critical appraisal of the literature', *Joint Bone Spine*, 73(1), pp. 43–50.

Descarreaux, M., Lafond, D. and Cantin, V. (2010) 'Changes in the flexion-relaxation response induced by hip extensor and erector spinae muscle fatigue', *BMC Musculoskeletal Disorders*, 11(112).

Descarreaux, M., Lafond, D., Jeffrey-Gauthier, R., Centomo, H. and Cantin, V. (2008) 'Changes in the flexion relaxation response induced by lumbar muscle fatigue.', *BMC musculoskeletal disorders*, 9, p. 10.

Descarreaux, M., Lafond, D., Jeffrey-Gauthier, R., Centomo, H. and Cantin, V. (2008) 'Changes in the flexion relaxation response induced by lumbar muscle fatigue', *BMC Musculoskeletal Disorders*. England, 9(10).

Deyo, R. a, Battie, M., Beurskens, a J., Bombardier, C., Croft, P., Koes, B., Malmivaara, a, Roland, M., Von Korff, M. and Waddell, G. (1998) 'Outcome measures for low back pain research. A proposal for standardized use.', *Spine*, 23(18), pp. 2003–13.

Van Dieën, J. H., Moseley, G. L. and Hodges, P. W. (2013) 'Motor control changes and low back pain: cause or effect', in Hodges, P. W., Cholewicki, J., and Van Dieen, J. (eds) *Spinal Control: The Rehabilitation of Back Pain*. 1st edn. Curchill Livingstone Elsevier, pp. 207–217.

Van Dieën, J., Selen, L. and Cholewicki, J. (2003) 'Trunk muscle activation in low-back pain patients, an analysis of the literature', *Journal of Electromyography and Kinesiology*, 13(4), pp. 333–351.

Van Dillen, L. and van Tulder, M. W. (2013) 'Targeting interventions to patients: development and evaluation.', in *Spinal Control: The Rehabilitation of Back Pain*, pp. 195–206.

Dupeyron, A., Lecocq, J., Vautravers, P., Pélissier, J. and Perrey, S. (2009) 'Muscle oxygenation and intramuscular pressure related to posture and load in back muscles', *Spine Journal*.

Durkin, J., Harvey, A., Hughson, R. and Callaghan, J. (2006) 'The effects of lumbar massage on muscle fatigue, muscle oxygenation, low back discomfort, and driver performance during prolonged driving.', *Ergonomics*, 49(1), pp. 28–44.

Dworkin, R. H., Turk, D. C., Revicki, D. a, Harding, G., Coyne, K. S., Peirce-Sandner, S., Bhagwat, D., Everton, D., Burke, L. B., Cowan, P., Farrar, J. T., Hertz, S., Max, M. B., Rappaport, B. a and Melzack, R. (2009) 'Development and initial validation of an expanded and revised version of the Short-form McGill Pain Questionnaire (SF-MPQ-2).', *Pain*. International Association for the Study of Pain, 144(1–2), pp. 35–42.

Earls, J. and Myers, T. W. (2010) *Fascial Release for Structural Balance*. North Atlantic Books.

Ebraheim, N. A., Hassan, A., Lee, M. and Xu, R. (2004) 'Functional anatomy of the lumbar spine', *Seminars in Pain Medicine*, 2(3), pp. 131–137.

Ercole, B., Antonio, S., Julie Ann, D. and Stecco, C. (2010) 'How much time is required to modify a fascial fibrosis?', *Journal of bodywork and movement therapies*, 14(4), pp. 318–25.

Ezzo, J. (2007) 'What can be learned from Cochrane systematic reviews of massage that can guide future research?', *Journal of alternative and complementary medicine (New York, N.Y.)*, 13(2), pp. 291–5. d

Fadel, P. J., Keller, D. M., Watanabe, H., Raven, P. B. and Thomas, G. D. (2004) 'Noninvasive assessment of sympathetic vasoconstriction in human and rodent skeletal muscle using near-infrared spectroscopy and Doppler ultrasound.', *Journal of applied physiology (Bethesda, Md. : 1985)*, 96(4), pp. 1323–30.

Fairbank, J. C. and Pynsent, P. B. (2000a) 'The Oswestry Disability Index.', *Spine*, 25(22), p. 2940–52; discussion 2952.

Fairbank, J. C. and Pynsent, P. B. (2000b) 'The Oswestry Disability Index.', *Spine*, 25(22), p. 2940–52; discussion 2952.

Faul, F., Erdfelder, E., Lang, A. G. and Buchner, A. (2007) 'G*Power3: A Flexible Statistical Power Analysis Program for the Social, Behavioral, and Biomedical Sciences', *Behavioral Research Methods*, 39(2), pp. 175–191.

Ferrari, M. and Mottola, L. (2004) 'Principles, techniques, of near-infrared spectroscopy.', *Canadian Journal of Applied Physiology*, 29(4), pp. 463–87.

Ferrari, M. and Quaresima, V. (2012) 'NeuroImage A brief review on the history of human functional near-infrared spectroscopy (fNIRS) development and fi elds of application', *NeuroImage*. Elsevier Inc., 63, pp. 921–935.

Fersum, K. V, Dankaerts, W., O'Sullivan, P. B., Maes, J., Skouen, J. S., Bjordal, J. M. and Kvåle, a (2010) 'Integration of subclassification strategies in randomised controlled clinical trials evaluating manual therapy treatment and exercise therapy for non-specific chronic low back pain: a systematic review.', *British journal of sports medicine*, 44(14), pp. 1054–62.

Field, A., Miles, J. and Field, Z. (2013) *Discovering Statistics Using SPSS*, *Sage*.

Field, T., Hernandez-Reif, M., Diego, M. and Fraser, M. (2007) 'Lower back pain and sleep disturbance are reduced following massage therapy', *Journal of Bodywork and Movement Therapies*, 11(2), pp. 141–145.

Finn, C. (2013) 'Rehabilitation of Low Back Pain in Golfers: From Diagnosis to Return to Sport', *Sports Health: A Multidisciplinary Approach*. doi: 10.1177/1941738113479893.

Fitch, E., Brooks, D., Stratford, P. and Mayo, N. (2002) *Physical Rehabilitation Outcome Measures*. 2nd Editio. Hamilton, ON: BC Decker Inc.

Floyd, W. F. and Silver, P. H. S. (1955) 'The function of the erectores spinae muscles in certain movements and postures in man', *The Journal of Physiology*, 129, pp. 184–203.

Foster, N., Anema, J., Cherkin, D. and al., et (2018) 'Prevention and treatment of low back pain: evidence, challenges, and promising directions', *Lancet*, 6736(18), pp. 1–16.

French, D. J., France, C. R., Vigneau, F., French, J. a and Evans, R. T. (2007) 'Fear of movement/(re)injury in chronic pain: a psychometric assessment of the original English version of the Tampa scale for kinesiophobia (TSK).', *Pain*, 127(1–2), pp. 42–51.

Frey Law, L. a, Evans, S., Knudtson, J., Nus, S., Scholl, K. and Sluka, K. a (2008) 'Massage reduces pain perception and hyperalgesia in experimental muscle pain: a randomized, controlled trial.', *The journal of pain : official journal of the American Pain Society*, 9(8), pp. 714–21.

Fryer, G. and Pearce, A. J. (2013) 'The effect of muscle energy technique on corticospinal and spinal reflex excitability in asymptomatic participants', *Journal of Bodywork and Movement Therapies*, 17(4), pp. 440–447.

Furlan, A. D., Giraldo, M., Baskwill, A., Irvin, E. and Imamura, M. (2015) 'Massage for low-back pain.', *The Cochrane database of systematic reviews*, 9, p. CD001929.

Furlan, A. D., Imamura, M., Dryden, T. and Irvin, E. (2009) 'Massage for low back pain: An updated systematic review within the framework of the Cochrane Back Review Group', *Spine*, 34(16), p. 1669.

Furlan, A. D., Yazdi, F., Tsertsvadze, A., Gross, A., Van Tulder, M., Santaguida, L., Cherkin, D., Gagnier, J., Ammendolia, C., Ansari, M. T., Ostermann, T., Dryden, T., Doucette, S., Skidmore, B., Daniel, R., Tsouros, S., Weeks, L. and Galipeau, J. (2010) *Complementary and alternative therapies for back pain II.*, *Evidence report/technology assessment*.

Gandevia, S. C. (2001) 'Spinal and supraspinal factors in human muscle fatigue.', *Physiological reviews*, 81(4), pp. 1725–89.

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N. and Turk, D. C. (2007) 'The Biopsychosocial Approach to Chronic Pain: Scientific Advances and Future Directions', *Psychological Bulletin*, 133(4), pp. 581–624.

Geisser, M. E., Haig, A. J. and Theisen, M. E. (2000) 'Activity Avoidance and Function in Persons with Chronic Back Pain', *Journal of Occupational Rehabilitation,* 10(3), pp. 215–227.

Gentile, D. a, Woodhouse, J., Lynch, P., Maier, J. and McJunkin, T. (2011) 'Reliability and validity of the Global Pain Scale with chronic pain sufferers.', *Pain physician*, 14(1), pp. 61–70.

Ghanbari, A., Rahimijaberi, A., Mohamadi, M., Abbasi, L. and Sarvestani, F. K. (2012) 'The effect of trigger point management by positional release therapy on tension type headache.', *NeuroRehabilitation*, 30(4), pp. 333–9.

Goats, G. C. (1994a) 'Massage--the scientific basis of an ancient art: Part 2. Physiological and therapeutic effects.', *British journal of sports medicine*, 28(3), pp. 153–6.

Goats, G. C. (1994b) 'Massage--the scientific basis of an ancient art: Part 2. Physiological and therapeutic effects', *British Journal of Sports Medicine*, 28(3), pp. 153–156.

Goats, G. C. and Keir, K. a (1991) 'Connective tissue massage. [Review]', *British journal of sports medicine*, 25(3), pp. 131–133.

Gracovetsky, S. (2008) 'Is the lumbodorsal fascia necessary?', *Journal of bodywork and movement therapies*, 12(3), pp. 194–7.

Granata, K. P. and Wilson, S. E. (2001) 'Trunk posture and spinal stability.', *Clinical biomechanics (Bristol, Avon)*, 16(8), pp. 650–9.

Gusi, N., del Pozo-Cruz, B., Olivares, P. R., Hernández-Mocholi, M. and Hill, J. C. (2011) 'The Spanish version of the "STarT Back Screening Tool" (SBST) in different subgroups', *Atención Primaria*, 43(7), pp. 356–361.
Hadler, N. (2001) 'The bane of the ageing worker', *Spine*, 26(12), pp. 1309–10.

Hagen, L., Hebert, J. J., Dekanich, J. and Koppenhaver, S. (2015) 'The effect of elastic therapeutic taping on back extensor muscle endurance in patients with low back pain: a randomized, controlled, crossover trial.', *Journal of Orthopaedic & Sports Physical Therapy*, 45(3), pp. 215–219.

Hagen, L., Hebert, J. J., Dekanich, J. and Koppenhaver, S. (2015) 'The Effect of Elastic Therapeutic Taping on Back Extensor Muscle Endurance in Patients With Low Back Pain: A Randomized, Controlled, Crossover Trial', *Journal of Orthopaedic & Sports Physical Therapy*, 45(3), pp. 215–219.

Hancock, M. J., Maher, C. G., Laslett, M., Hay, E. and Koes, B. (2011) 'Discussion paper: What happened to the bio in the bio-psycho-social model of low back pain?', *European Spine Journal*, 20(12), pp. 2105–2110.

Hanten, W. P. and Chandler, S. D. (1994) *Effects of myofascial release leg pull and sagittal plane isometric contract-relax techniques on passive straight-leg raise angle.*, *The Journal of orthopaedic and sports physical therapy*. 20(3), pp.138-144.

Hartvigsen, J., Hancock, M. J., Kongsted, A., Louw, Q., Ferreira, M. L., Genevay, S., Hoy, D., Karppinen, J., Pransky, G., Sieper, J., Smeets, R. J., Underwood, M., Buchbinder, R., Hartvigsen, J., Cherkin, D., Foster, N. E., Maher, C. G., Underwood, M., Tulder, M. van, Anema, J. R., Chou, R., Cohen, S. P., Costa, L. M., Croft, P., Ferreira, M., Ferreira, P. H., Fritz, J. M., Genevay, S., Gross, D. P., Hancock, M. J., Hoy, D., Karppinen, J., Koes, B. W., Kongsted, A., Louw, Q., Öberg, B., Peul, W. C., Pransky, G., Schoene, M., Sieper, J., Smeets, R. J., Turner, J. A. and Woolf, A. (2018) 'What low back pain is and why we need to pay attention', *The Lancet*, 0(0).

Hawker, G. a, Mian, S., Kendzerska, T. and French, M. (2011) 'Measures of adult pain: Visual Analog Scale for Pain (VAS Pain), Numeric Rating Scale for Pain (NRS Pain), McGill Pain Questionnaire (MPQ), Short-Form McGill Pain Questionnaire (SF-MPQ), Chronic Pain Grade Scale (CPGS), Short Form-36 Bodily Pain Scale (SF', *Arthritis care & research*, 63 Suppl 1(November), pp. S240-52.

Hay, E. M., Dunn, K. M., Hill, J. C., Lewis, M., Mason, E. E., Konstantinou, K., Sowden, G., Somerville, S., Vohora, K., Whitehurst, D. and Main, C. J. (2008) 'A randomised clinical trial of subgrouping and targeted treatment for low back pain compared with best current care. The STarT Back Trial Study Protocol', *BMC Musculoskeletal Disorders*, 9(1), p. 58.

Hayden, J. A., van Tulder, M. W., Malmivaara, A. and Koes, B. W. (2005) *Exercise therapy for treatment of non-specific low back pain.*, *The Cochrane database of systematic reviews*.

Healey, K. C., Hatfield, D. L., Blanpied, P., Dorfman, L. R. and Riebe, D. (2014) 'The effects of myofascial release with foam rolling on performance', *Journal of Strength and Conditioning Research*, 28(1), pp. 61–68.

Hellström, F., Roatta, S., Thunberg, J., Passatore, M. and Djupsjöbacka, M. (2005) 'Responses of muscle spindles in feline dorsal neck muscles to electrical stimulation of the cervical sympathetic nerve.', *Experimental brain research*, 165(3), pp. 328–42.

Hermens, H. J., Freriks, B., Disselhorst-Klug, C. and Rau, G. (2000) 'Development of recommendations for SEMG sensors and sensor placement procedures.', *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*, 10(5), pp. 361–74.

Heuts, P. H. T. G., Vlaeyen, J. W. S., Roelofs, J., de Bie, R. a, Aretz, K., van Weel, C. and van Schayck, O. C. P. (2004) 'Pain-related fear and daily functioning in patients with osteoarthritis.', *Pain*, 110(1–2), pp. 228–35.

Hill, J. C., Dunn, K. M., Main, C. J. and Hay, E. M. (2010) 'Subgrouping low back pain: A comparison of the STarT Back Tool with the Örebro Musculoskeletal Pain Screening Questionnaire', *European Journal of Pain*. Blackwell Publishing Ltd, 14(1), pp. 83–89.

Hill, J. C., Vohora, K., Dunn, K. M., Main, C. J. and Hay, E. M. (2010) 'Comparing the STarT Back Screening Tool's Subgroup Allocation of Individual Patients With That of Independent Clinical Experts', *The Clinical Journal of Pain*, 26(9), pp. 783–787.

Hinds, T., McEwan, I., Perkes, J., Dawson, E., Ball, D. and George, K. (2004) 'Effects of massage on limb and skin blood flow after quadriceps exercise', *Medicine and Science in Sports and Exercise*, 36(8), pp. 1308–1313.

Hodges, P. (2011) 'Pain and motor control: From the laboratory to rehabilitation.', *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*, 21(2), pp. 220–8. d

Hodges, P., Cholewicki, J. and Van Dieen, J. (2013) *Spinal Control: The Rehabilitation of Back Pain*. Edited by P. Hodges, J. Cholewicki, and J. Van Dieen. Curchill Livingstone Elsevier.

Hodges, P. W. and Moseley, G. L. (2003) 'Pain and motor control of the lumbopelvic region: effect and possible mechanisms.', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 13(4), pp. 361–370.

Hodges, P. W., Moseley, G. L., Gabrielsson, A. and Gandevia, S. C. (2003) 'Experimental muscle pain changes feedforward postural responses of the trunk muscles.', *Experimental brain research. Experimentelle Hirnforschung. Expérimentation cérébrale*, 151(2), pp. 262–71.

Holey, E. A. (2000) 'Connective tissue massage : a bridge between complementary and orthodox approaches', *Journal of Bodywork and Movement Therapies*, 4(March 1999).
Holm, S., Indahl, A. and Solomonow, M. (2002) 'Sensorimotor control of the spine.', *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*, 12(3), pp. 219–34.

Hotfiel, T., Swoboda, B., Krinner, S., GRIM, C., Engelhardt, M., UDER, M. and HEISS, A. R. U. (2017) 'Acute effects of lateral thigh foam rolling on arterial tissue perfusion determined by spectral doppler and power doppler ultrasound.', *Journal ofStrength and Conditioning Research*, 31(4), pp. 893–900.

Hotfiel, T., Swoboda, B., Krinner, S., Grim, C., Engelhardt, M., Uder, M. and Heiss, R. (2017) 'Acute Effects of Lateral Thigh Foam Rolling on Arterial Tissue Perfusion Determined by Spectral Doppler and Power Doppler Ultrasound.', *Journal of Strength and Conditioning Research*, 31(4), pp. 893–900.

Hoy, D., Bain, C., Williams, G., March, L., Brooks, P., Blyth, F., Woolf, A., Vos, T. and Buchbinder, R. (2012) 'A systematic review of the global prevalence of low back pain', *Arthritis and Rheumatism*, 64(6), pp. 2028–2037.

Hoy, D., March, L., Brooks, P., Blyth, F., Woolf, A., Bain, C., Williams, G., Smith, E., Vos, T., Barendregt, J., Murray, C., Burstein, R. and Buchbinder, R. (2014) 'The global burden of low back pain: estimates from the Global Burden of Disease 2010 study.', *Annals of the rheumatic diseases*, 73(6), pp. 968–74.

Hsieh, C. Y., Adams, A. H., Tobis, J., Hong, C. Z., Danielson, C., Platt, K., Hoehler, F., Reinsch, S. and Rubel, A. (2002) 'Effectiveness of four conservative treatments for subacute low back pain: a randomized clinical trial', *Spine*, 27(11), pp. 1142–1148.

Hsu, C., Sherman, K. J., Eaves, E. R., Turner, J. A., Cherkin, D. C., Cromp, D., Schafer, L. and Ritenbaugh, C. (2014) 'New perspectives on patient expectations of treatment outcomes: results from qualitative interviews with patients seeking complementary and alternative medicine treatments for chronic low back pain.', *BMC Complementary & Alternative Medicine*, 14(1), pp. 276–285.

Hughes, C. M., Quinn, F. and Baxter, G. D. (2011) 'Complementary and alternative medicine: perception and use by physiotherapists in the management of low back pain.', *Complementary therapies in medicine*, 19(3), pp. 149–54.

Hui, K. K., Liu, J., Makris, N., Gollub, R. L., Chen, a J., Moore, C. I., Kennedy, D. N., Rosen, B. R. and Kwong, K. K. (2000) 'Acupuncture modulates the limbic system and subcortical gray structures of the human brain: evidence from fMRI studies in normal subjects.', *Human brain mapping*, 9(1), pp. 13–25.

Huijing, P. A. (1999) 'Muscular force transmission: a unified, dual or multiple system? A review and some explorative experimental results', *Archives of Physiology and Biochemistry*. Informa UK Ltd UK, 107(4), pp. 292–311.

Hunt, K. J., Coelho, H. F., Wider, B., Perry, R., Hung, S. K., Terry, R. and Ernst, E. (2010) 'Complementary and alternative medicine use in England: results from a national survey.', *International journal of clinical practice*, 64(11), pp. 1496–502.

Iwamoto, K. and Mizukami, M. (2017) 'Effects of friction massage of the popliteal fossa on blood flow velocity of the popliteal vein', *The Journal of Physical Therapy Science*, 29, pp. 511–514.

J.Pannucci, C. and G.Wilkins, E. (2011) 'Identifiyed and Avoiding Bias in Research', *Plastic and Reconstructive Surgery*, 126(2), pp. 619–625.

Jaap H. van Dieen , Luc P.J. Selen, J. C. (2003) 'Trunk muscle activation in low-back pain patients, an analysis of the literature.', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 13(4), pp. 333–351.

Janssens, L., Pijnenburg, M., Claeys, K., McConnell, A. K., Troosters, T. and Brumagne, S. (2013) 'Postural strategy and back muscle oxygenation during inspiratory muscle loading.', *Medicine and science in sports and exercise*, 45(7), pp. 1355–62.

Järvinen, T. a H., Józsa, L., Kannus, P., Järvinen, T. L. N. and Järvinen, M. (2002) 'Organization and distribution of intramuscular connective tissue in normal and immobilized skeletal muscles. An immunohistochemical, polarization and scanning electron microscopic study.', *Journal of muscle research and cell motility*, 23(3), pp. 245–54.

Jensen, B., Kurt, J., Alan R., H., Pernille K., N. and Nicolaisen (1999) 'Physiological Response to Submaximal Isometric Contractions of the Paravertebral Muscles', *Spine*, 24(22), p. 2332.

Jin, S., Ning, X. and Mirka, G. A. (2012) 'An algorithm for defining the onset and cessation of the flexion-relaxation phenomenon in the low back musculature', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 22(3), pp. 376–382.

Johanson, E., Brumagne, S., Janssens, L., Pijnenburg, M., Claeys, K. and Pääsuke, M. (2011) 'The effect of acute back muscle fatigue on postural control strategy in people with and without recurrent low back pain.', *European spine journal :*, 20(12), pp. 2152–9.

Johansson, H. and Sojka, P. (1991) 'Pathophysiological mechanisms involved in genesis and spread of muscular tension in occupational muscle pain and in chronic musculoskeletal pain syndromes: a hypothesis.', *Medical Hypothesis*, Johansson(35), pp. 196–203.

Jones, S., Henry, S., Raasch, C., Hitt, J. and Bunn, J. (2012) 'Individuals with non-specific low back pain use a trunk stiffening strategy to maintain upright posture.', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 22(1), pp. 13–20.
Jones, S. L., Hitt, J. R., Desarno, M. J. and Henry, S. M. (2012) 'Individuals with non-specific low back pain in an active episode demonstrate temporally altered torque responses and direction-specific enhanced muscle activity following unexpected balance perturbations.', *Experimental brain research. Experimentelle Hirnforschung. Experimentation cerebrale*, pp. 413–426.

Jull, G. A. and Richardson, C. A. (2000) 'Motor Control Problems in Patients With Spinal Pain : A New Direction for Therapeutic Exercise Spinal Segmental Stabilization Training', *Journal of Manipulative and Physical Therapies*, 23(2), pp. 1999–2001.

Kachanathu, S. J., Alenazi, A. M., Seif, H. E., Hafez, A. R. and Abdulmohsen, M. A. (2014) 'Comparison between Kinesio Taping and a Traditional Physical Therapy Program in Treatment of Nonspecific Low Back Pain.', *Journal of Physical Therapy Science*, 26(8), pp. 1185–1188.

Kamper, S. J., Apeldoorn, A. T., Chiarotto, A., Smeets, R., Ostelo, R., Guzman, J. and van Tulder, M. W. (2015) 'Multidisciplinary biopsychosocial rehabilitation for chronic low back pain: Cochrane systematic review and meta-analysis.', *BMJ (Clinical research ed.)*, 350(February), p. h444.

Kao, M.-J., Kuan, T.-S., Hsiegh, Y.-L., Yang, J.-F. and Hong, C.-Z. (2008) 'Myofascial low back pain.', *Journal of Physical Medicine and Rehabilitation*, 36(1), pp. 1–14.

Kase K, Wallis J, and K. T. (2003) 'Clinical therapeutic applications of the kinesio taping method', *Tokyo, Japan: Ken I kai Co Ltd*, pp. 12, 32.

Kell, R. T. and Bhambhani, Y. (2006) 'Relationship between erector spinae static endurance and muscle oxygenation-blood volume changes in healthy and low back pain subjects', *European Journal of Applied Physiology*, 96(3), pp. 241–248.

Kennedy, D. S., Fitzpatrick, S. C., Gandevia, S. C. and Taylor, J. L. (2014) 'Fatigue-Related Firing of Muscle Nociceptors Reduces Voluntary Activation of Ipsilateral But Not Contralateral Lower Limb Muscles.', *Journal of applied physiology (Bethesda, Md. : 1985)*, 118(18), pp. 408–418.

Kjaer, M., Magnusson, P., Krogsgaard, M., Boysen Møller, J., Olesen, J., Heinemeier, K., Hansen, M., Haraldsson, B., Koskinen, S., Esmarck, B. and Langberg, H. (2006) 'Extracellular matrix adaptation of tendon and skeletal muscle to exercise.', *Journal of anatomy*, 208(4), pp. 445–50. d

Klingler, W., Velders, M., Hoppe, K., Pedro, M. and Schleip, R. (2014) 'Clinical relevance of fascial tissue and dysfunctions', *Current Pain and Headache Reports*, 18(8).

Knutson, G. A. (2000) 'The role of the γ-motor system in increasing muscle tone and muscle pain syndromes: A review of the Johansson/Sojka hypothesis', *Journal of Manipulative and Physiological Therapeutics*, 23(8), pp. 564–572.

Koenig, A., Kupper, A., Skidmore, J. and Murphy, K. (2014) 'Biopsychosocial functioning and pain self-efficacy in chronic low back pain patients', *Journal of Rehabilitation Research & Development (JRRD)*, 51(8), pp. 1277–1286.

Koho, P., Aho, S., Watson, P. and Hurri, H. (2001) 'Assessment of chronic pain behaviour: reliability of the method and its relationship with perceived disability, physical impairment and function.', *Journal of rehabilitation medicine : official journal of the UEMS European Board of Physical and Rehabilitation Medicine*, 33(3), pp. 128–32.

Konishi, Y. (2013) 'Tactile stimulation with kinesiology tape alleviates muscle weakness attributable to attenuation of Ia afferents.', *Journal of science and medicine in sport / Sports Medicine Australia*. Sports Medicine Australia, 16(1), pp. 45–8.

Kostopoulos, D., Nelson, A. J., Ingber, R. S. and Larkin, R. W. (2008) 'Reduction of Spontaneous Electrical Activity and Pain Perception of Trigger Points in the Upper Trapezius Muscle through Trigger Point Compression and Passive Stretching', *Journal of Musculoskeletal Pain*, 16(4), pp. 266–278.

Kovacs, K. M., Marras, W. S., Litsky, a S., Gupta, P. and Ferguson, S. a (2001) 'Localized oxygen use of healthy and low back pain individuals during controlled trunk movements.', *Journal of spinal disorders*, 14(2), pp. 150–8.

Koźma, E. M., Olczyk, K., Głowacki, A. and Bobiński, R. (2000) 'An accumulation of proteoglycans in scarred fascia.', *Molecular and cellular biochemistry*, 203(1–2), pp. 103–12.

Kramer, M., Dehner, C., Hartwig, E., Völker, H. U., Sterk, J., Elbel, M., Weikert, E., Gerngroß, H., Kinzl, L. and Willy, C. (2005) 'Intramuscular pressure, tissue oxygenation and EMG fatigue measured during isometric fatigue-inducing contraction of the multifidus muscle', *European Spine Journal*, 14(6), pp. 578–585. d

Kwong, E. H. and Findley, T. W. (2014) 'Fascia— Current knowledge and future directions in physiatry: Narrative review', *Journal of Rehabilitation Research & Development*, 51(6), pp. 875–884.

Lalanne, K., Lafond, D. and Descarreaux, M. (2009) 'Modulation of the flexion-relaxation response by spinal manipulative therapy: a control group study.', *Journal of manipulative and physiological therapeutics*, 32(3), pp. 203–9.

Lamoth, C. J. C., Meijer, O. G., Daffertshofer, A., Wuisman, P. I. J. M. and Beek, P. J. (2006) 'Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control.', *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*, 15(1), pp. 23–40.

Langevin, H. M., Fox, J. R., Koptiuch, C., Badger, G. J., Greenan-Naumann, A. C., Bouffard, N. a, Konofagou, E. E., Lee, W.-N., Triano, J. J. and Henry, S. M. (2011) 'Reduced thoracolumbar fascia shear strain in human chronic low back pain.', *BMC musculoskeletal disorders*, 12(203), pp. 1–29.

Langevin, H. M. and Sherman, K. J. (2007) 'Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms', *Medical hypotheses*. Elsevier, 68(1), pp. 74–80.

Lara-Palomo, I. C., Aguilar-Ferrándiz, M. E., Matarán-Peñarrocha, G. a, Saavedra-Hernández, M., Granero-Molina, J., Fernández-Sola, C. and Castro-Sánchez, A. M. (2013) 'Short-term effects of interferential current electro-massage in adults with chronic non-specific low back pain: a randomized controlled trial.', *Clinical rehabilitation*, 27(5), pp. 439–49.

LeBauer, A., Brtalik, R. and Stowe, K. (2008) 'The effect of myofascial release (MFR) on an adult with idiopathic scoliosis', *Journal of Bodywork and Movement Therapies*. Elsevier, 12(4), pp. 356–363.

Lemoon, K. (2008) 'Terminology Used in Fascia Research', *Journal of Bodywork and Movement Therapies*, 12(3), pp. 204–212.

Levangie, P. K. and Norkin, C. C. (2011) *Joint structure and function : a comprehensive analysis*. 5th edn. Edited by P. K. Levangie and C. C. Norkin. Philidelphia: FA Davis Company.

Lewis, S., Holmes, P., Woby, S., Hindle, J. and Fowler, N. (2012) 'The relationships between measures of stature recovery, muscle activity and psychological factors in patients with chronic low back pain.', *Manual therapy*, 17(1), pp. 27–33.

Little, P., Lewith, G., Webley, F., Evans, M., Beattie, A., Middleton, K., Barnett, J., Ballard, K., Oxford, F., Smith, P., Yardley, L., Hollinghurst, S. and Sharp, D. (2008) 'Randomised controlled trial of Alexander technique lessons ', *BMJ*, 337:a884, pp. 13–15.

de Looze, M. ., Groen, H., Horemans, H., Kingma, I. and van Dieën, J. . (1999) 'Abdominal muscles contribute in a minor way to peak spinal compression in lifting', *Journal of Biomechanics*. Elsevier, 32(7), pp. 655–662.

Loukas, M., Shoja, M. M., Thurston, T., Jones, V. L., Linganna, S. and Tubbs, R. S. (2008) 'Anatomy and biomechanics of the vertebral aponeurosis part of the posterior layer of the thoracolumbar fascia', *Surg Radiol Anat*. 2007/12/19, 30(2), pp. 125–129.

Lowe (2000) 'Active engagement strokes'.

Luca, C. J. De (1997) 'The Use of Surface Electromyography in Biomechanics', *Journal of applied biomechanics*, 13(2), pp. 135–163.

Lucas, K. R., Polus, B. I. and Rich, P. A. (2004) 'Latent myofascial trigger points : their effects on muscle activation and movement efficiency', *Journal of Bodywork & Movement Therapies*, 8, pp. 160–166.

Luijsterburg, P. A. J., Verhagen, A. P., Ostelo, R. W. J. G., Van Os, T. A. G., Peul, W. C. and Koes, B. W. (2007) 'Effectiveness of conservative treatments for the lumbosacral radicular syndrome: A systematic review', *European Spine Journal*, 16(7), pp. 881–899.

Lund, I., Yu, L.-C., Uvnas-Moberg, K., Wang, J., Yu, C., Kurosawa, M., Agren, G., Rosen, A., Lekman, M. and Lundeberg, T. (2002) 'Repeated massage-like stimulation induces long-term effects on nociception: contribution of oxytocinergic mechanisms', *European Journal of Neuroscience*, 16(2), pp. 330–338.

.

Lund, J. P., Donga, R., Widmer, C. G. and Stohler, C. S. (1991) 'The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity', *Canadian Journal of Physiology and Pharmacology*. NRC Research Press, 69(5), pp. 683–694.

Macedo, L. G., Latimer, J., Maher, C. G., Hodges, P. W., Nicholas, M., Tonkin, L., McAuley, J. H. and Stafford, R. (2008) 'Motor control or graded activity exercises for chronic low back pain? A randomised controlled trial', *BMC Musculoskeletal Disorders*. England, 9(65).

Main, C. J., Sowden, G., Hill, J. C., Watson, P. J. and Hay, E. M. (2012a) 'Integrating physical and psychological approaches to treatment in low back pain: the development and content of the STarT Back trial's "high-risk" intervention (StarT Back; ISRCTN 37113406)', *Physiotherapy*, 98(2), pp. 110–116.

Main, C. J., Sowden, G., Hill, J. C., Watson, P. J. and Hay, E. M. (2012b) 'Integrating physical and psychological approaches to treatment in low back pain: the development and content of the STarT Back trial's "high-risk" intervention (StarT Back; ISRCTN 37113406)', *Physiotherapy*, 98(2), pp. 110–116. d

.

Manchikanti, L. (2000) 'Epidemiology of Low Back Pain', *Pain Physician*, 3(2), pp. 167–192.

Manchikanti, L., Singh, V., Falco, F. J. E., Benyamin, R. M. and Hirsch, J. A. (2014) 'Epidemiology of low back pain in Adults', *Neuromodulation*, 17(S2), pp. 3–10.

Maniadakis, N. and Gray, a (2000) 'The economic burden of back pain in the UK.', *Pain*, 84(1), pp. 95–103.

.

Maria Hernandez-reif, Tiffany Field, Josh Krasnegor, and H. T. (2001) 'Lower Back Pain is Reduced and Range of Motion Increased After Massage Therapy', *International Journal of Neuroscience*, 106(3–4), pp. 131–145.

Marras, W. S., Lavender, S. A., Leurgans, S. E., Fathallah, F. A., Ferguson, S. A., Allread, W. G. and Rajulu, S. L. (1995) 'Biomechanical risk factors for occupationally related low back disorders.', *Ergonomics*. Taylor & Francis Group, 38(2), pp. 377–410.

Martin, N. A., Zoeller, R. F., Robertson, R. J. and Lephart, S. M. (1998) 'The comparative effects of sports massage, active recovery, and rest in promoting blood lactate clearance after supramaximal leg exercise', *Journal of Athletic Training*, 33(1), pp. 30–35.

McGill, S. (2007) *Low back Pain Dissoeders: Evidence based Prevention and Rehabilitation*. 2nd edn. Human kinetics.

McGill, S. M. and ,Sylvain Grenier , Natasa Kavcic, J. C. (2003) 'Coordination of muscle activity to assure stability of the lumbar spine.', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 13(4), pp. 353–359.

Mehling, W. E., Daubenmier, J. J., Price, C. J., Acree, M., Bartmess, E. and Stewart, A. L. (2013) 'Self-reported interoeptive awareness in primary care patients with past or current low back pain', *Journal of Pain Research*, 6, pp. 403–418.

Melzack, R. (1987) 'The short-form McGill pain questionnaire', *Pain*, 30(2), pp. 191–197.
Meucci, R. D., Fassa, A. G. and Faria, N. M. X. (2015) 'Prevalence of chronic low back pain: systematic review.', *Revista de saúde pública*, 49(73), pp. 1–10.

Van Middelkoop, M., Rubinstein, S. M., Kuijpers, T., Verhagen, A. P., Ostelo, R., Koes, B. W. and van Tulder, M. W. (2011) 'A systematic review on the effectiveness of physical and rehabilitation interventions for chronic non-specific low back pain.', *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*, 20(1), pp. 19–39.

Van Middelkoop, M., Rubinstein, S. M., Verhagen, A. P., Ostelo, R. W., Koes, B. W. and Van Tulder, M. W. (2010) 'Exercise therapy for chronic nonspecific low-back pain', *Best Pract Res Clin Rheumatol*, 24(2), pp. 193–204.

Moraska, A. (2005) 'Sports massage: A comprehensive review', *Journal of Sports Medicine and Physical Fitness*, pp. 370–380.

Mori, H., Ohsawa, H., Tanaka, T., Taniwaki, E., Leisman, G. and Nishijo, K. (2004) 'Effect of massage on blood flow and muscle fatigue following isometric lumbar exercise.', *Medical science monitor : international medical journal of experimental and clinical research*, 10(5), pp. CR173-8.

Morris, S. B. (2008) 'Estimating Effect Sizes From Pretest-Posttest-Control Group Designs', *Organizational Research Methods*, 11(2 April 2003), pp. 364–386.

Moyer, C. a, Rounds, J. and Hannum, J. W. (2004) 'A meta-analysis of massage therapy research.', *Psychological bulletin*, 130(1), pp. 3–18. d

Munk, N., Symons, B., Shang, Y., Cheng, R. and Yu, G. (2012) 'Noninvasively measuring the hemodynamic effects of massage on skeletal muscle: a novel hybrid near-infrared diffuse optical instrument.', *Journal of bodywork and movement therapies*. Elsevier Ltd, 16(1), pp. 22–8.

Murthy, G., Hargens, A. R., Lehman, S. and Rempel, D. M. (2001) 'Ischemia causes muscle fatigue', *Journal of Orthopaedic Research*, 19(3), pp. 436–440.

Mutlu, E. K. and Ozdincler, A. R. (2015) 'Reliability and responsiveness of algometry for measuring pressure pain threshold in patients with knee osteoarthritis', *Journal of Physical Therapy Science*, 27(6), pp. 1961–1965.

Myers, T. W. (2009a) *Anatomy Trains, 2nd edition*, *The Foot*.

Myers, T. W. (2009b) *Anatomy Trains: Myofascial Meridians for Manual and Movement Therapists*. Elsevier Health Sciences.

Nachemson, G. (1992) 'Newest knowledge of low back pain: A critical look.', *Cinical Orthopedics and Related Research*, 279, pp. 8–20.

Namvar, H. and Moghadam, B. A. (2016) 'Effect of myofascial release technique on pain , disability , maximum isometric contraction of the extensor muscles , and pressure pain threshold in patients with chronic nonspecific neck pain : Double blinded randomized clinical trial', *International Journal of Medical Research & Health Sciences*, 5(7S), pp. 500–506.

Netchanok, S., Wendy, M., Marie, C. and Siobhan, O. (2012) 'The effectiveness of Swedish massage and traditional Thai massage in treating chronic low back pain: a review of the literature.', *Complementary therapies in clinical practice*. Elsevier Ltd, 18(4), pp. 227–34.

NICE (2016) *National Clinical Guideline Centre Low back pain and sciatica: management of non-specific low back pain and sciatica Assessment and non-invasive treatments*.
Ning, X., Haddad, O., Jin, S. and Mirka, G. a (2011) 'Influence of asymmetry on the flexion relaxation response of the low back musculature.', *Clinical biomechanics (Bristol, Avon)*. Elsevier Ltd, 26(1), pp. 35–9.

Nitsure, P. and Welling, A. (2014) 'Effect of Gross Myofascial Release of U pper Limb and Neck on Pain and Function in Subjects with Mechanical Neck Pain with Upperlimb Radiculapathy- A Clinical Trial', *Int J Dent Med Res*, 1(3), pp. 8–16.

Norman, R., Wells, R., Neumann, P., Frank, J., Shannon, H. and Kerr, M. (1998) 'A comparison of peak vs cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry', *Clinical Biomechanics*, 13(8), pp. 561–573.

O'Sullivan, P. (2005) 'Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism.', *Manual therapy*, 10(4), pp. 242–55.

O'Sullivan, P. (2012) 'It's time for change with the management of non-specific chronic low back pain', *British Journal of Sports Medicine*, 46(4), pp. 224–227.

Olivier, N., Thevenon, A., Berthoin, S. and Prieur, F. (2013) 'An exercise therapy program can increase oxygenation and blood volume of the erector spinae muscle during exercise in chronic low back pain patients', *Archives of Physical Medicine and Rehabilitation*, 94(3), pp. 536–542.

Olson, M., Solomonow, M. and Li, L. (2006) 'Flexion–relaxation response to gravity', *Journal of biomechanics*. Elsevier Science, pp. 2545–2554. A

Owens, E. F., Gudavalli, M. R. and Wilder, D. G. (2011) 'Paraspinal muscle function assessed with the flexion-relaxation ratio at baseline in a population of patients with back-related leg pain.', *Journal of manip and physiological therapeutics*, 34(9), pp. 594–601.

Panjabi, M. M. (1992) 'The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement.', *Journal of spinal disorders*, 5, p. 383–389; discussion 397.

Panjabi, M. M. (2003) 'Clinical spinal instability and low back pain', *Journal of Electromyography and Kinesiology*, 13(4), pp. 371–379.

Panjabi, M. M. (2006) 'A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction.', *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society*, 15(5), pp. 668–76.

Park, G., Jang, S. H., Kim, C. W., Kim, M. J. and Park, S. B. (2011) 'Reliability and Usefulness of the Pressure Pain Threshold Measurement in Patients with Myofascial Pain', *Annals of Rehabilitation Medicine*, 35(3), p. 412.

Parreira, P. D. C. S., Costa, L. D. C. M., Takahashi, R., Junior, L. C. H., Junior, M. A. D. L., Silva, T. M. Da and Costa, L. O. P. (2014) 'Kinesio Taping to generate skin convolutions is not better than sham taping for people with chronic non-specific low back pain: A randomised trial', *Journal of Physiotherapy*, 60(2), pp. 90–96.

Patel, K., Gross, A., Graham, N., Ch, G., Ezzo, J., Morien, A. and Pmj, P. (2012) 'Massage for mechanical neck disorders (Review)', *The Cochrane Collaboration.*, (9).
Pavan, P. G., Stecco, A., Stern, R. and Stecco, C. (2014) 'Painful connections: Densification versus fibrosis of fascia', *Current Pain and Headache Reports*, 18(8).

Pedersen, J., Lonn, J., Hellstome, F., Djupsjobacka, M. and Johansson, H. (1999) 'Localized muscle fatigue decreases the acuity of the movement sense in the human shoulder', *Medicine & Science in Sports & Exercise*, 31(7), pp. 1047–1052.

Penas, C., Campo, M. S., Carnero, J. F. and Page, J. C. M. (2005) 'Manual therapies in myofascial trigger point treatment: a systematic review', *Journal of Bodywork & Movement Therapies*, 9(1), pp. 27–34.

Picelli, A., Ledro, G., Turrina, A., Stecco, C., Santilli, V. and Smania, N. (2011) 'Effects of myofascial technique in patients with subacute whiplash associated disorders: A pilot study', *European Journal of Physical and Rehabilitation Medicine*, 47(4), pp. 561–568.

Plakornkul, V., Vannabhum, M., Viravud, Y., Roongruangchai, J., Mutirangura, P., Akarasereenont, P. and Laohapand, T. (2016) 'The effects of the court-type Thai traditional massage on anatomical relations, blood flow, and skin temperature of the neck, shoulder, and arm.', *BMC complementary and alternative medicine*. BMC Complementary and Alternative Medicine, 16, p. 363.

Pollatos, O., Fustos, J. and Critchley, H. (2012) 'On the generalised embodiment of pain: How interoceptive sensitivity modulates cutaneous pain perception', *PAIN®*. No longer published by Elsevier, 153(8), pp. 1680–1686.

Portillo-Soto, A., Eberman, L. E., Demchak, T. J. and Peebles, C. (2014) 'Comparison of blood flow changes with soft tissue mobilization and massage therapy.', *Journal of alternative and complementary medicine*, 20(12), pp. 932–6.

Preyde, M. (2000) 'Effectiveness of massage therapy for subacute low-back pain: a randomized controlled trial.', *CMAJ : Canadian Medical Association journal = journal de l'Association medicale canadienne*, 162(13), pp. 1815–20.

Ramos-Gonzalez, E., Moreno-Lorenzo, C., Matar??n-Pe??arrocha, G. A., Guisado-Barrilao, R., Aguilar-Ferr??ndiz, M. E. and Castro-S??nchez, A. M. (2012) 'Comparative study on the effectiveness of myofascial release manual therapy and physical therapy for venous insufficiency in postmenopausal women', *Complementary Therapies in Medicine*, 20(5), pp. 291–298.

Ramos-González, E., Moreno-Lorenzo, C., Matarán-Peñarrocha, G. a., Guisado-Barrilao, R., Aguilar-Ferrándiz, M. E. and Castro-Sánchez, A. M. (2012) 'Comparative study on the effectiveness of myofascial release manual therapy and physical therapy for venous insufficiency in postmenopausal women', *Complementary Therapies in Medicine*, 20(5), pp. 291–298.

Ramsook, R. R. and Malanga, G. A. (2012) 'Myofascial low back pain', *Current Pain and Headache Reports*, 16(5), pp. 423–432. doi: 10.1007/s11916-012-0290-y.

Rawls, A. and Fisher, R. E. (2010) 'Development and Functional Anatomy of the Spine', in Kusumi, K. and Dunwoodie, S. L. (eds) *The Genetics and Development of Scoliosis*. 1st edn. New York: Springer, pp. 21–46.

Ribeiro, F., Mota, J. and Oliveira, J. (2007) 'Effect of exercise-induced fatigue on position sense of the knee in the elderly.', *European journal of applied physiology*, 99(4), pp. 379–85.

Richardson, C. (1999) *Therapeutic exercise for spinal segmental stabilization in low back pain : scientific basis and clinical approach*. Churchill Livingstone.

Richmond, J. (2012) 'Multi-factorial causative model for back pain management; relating causative factors and mechanisms to injury presentations and designing time- and cost effective treatment thereof', *Medical Hypotheses*. Elsevier Ltd, 79(2), pp. 232–240.

Riemann, B. L. and Lephart, S. M. (2002) 'The sensorimotor system, part I: The physiologic basis of functional joint stability', *Journal of Athletic Training*, 37(1), pp. 71–79.

Ritvanen, T., Zaproudina, N., Nissen, M., Leinonen, V. and Hänninen, O. (2007) 'Dynamic surface electromyographic responses in chronic low back pain treated by traditional bone setting and conventional physical therapy.', *Journal of manipulative and physiological therapeutics*, 30(1), pp. 31–7.

Roatta, S., Windhorst, U., Ljubisavljevic, M., Johansson, H. and Passatore, M. (2002) 'Sympathetic modulation of muscle spindle afferent sensitivity to stretch in rabbit jaw closing muscles.', *The Journal of physiology*, 540(Pt 1), pp. 237–248.

Roelofs, J., Sluiter, J. K., Frings-Dresen, M. H. W., Goossens, M., Thibault, P., Boersma, K. and Vlaeyen, J. W. S. (2007) 'Fear of movement and (re)injury in chronic musculoskeletal pain: Evidence for an invariant two-factor model of the Tampa Scale for Kinesiophobia across pain diagnoses and Dutch, Swedish, and Canadian samples.', *Pain*, 131(1–2), pp. 181–90.

Ropponen, A., Gibbons, L. E., Videman, T. and Michele C. Battié (2005) 'Isometric Back Extension Endurance Testing ':, *Journal of Orthopaedic & Sports Physical Therapy*, 35(7), pp. 437–442.

Roy, S., De Luca, C., Snyder-Mackler, M., Crenshaw, R. L. and Lyons, J. (1990) 'Fatigue, recovery, and low back pain in varsity rowers', *Medicine & Science in Sports & Exercise*, 22, pp. 463–469.

Sagar, S. M., Dryden, T. and Wong, R. K. (2007) 'Massage therapy for cancer patients: a reciprocal relationship between body and mind.', *Current oncology (Toronto, Ont.)*, 14(2), pp. 45–56.

Sakai, Y., Imagama, S., Ito, Z., Wakao, N. and Matsuyama, Y. (2012) 'Outcome of Back Exercise for Flexion-and Extension-Provoked Low Back Pain', *Orthopedic & Muscular System*, 1(5), pp. 1–6.

Sanderson, M. (1998) *Soft tissue release : a practical handbook for physical therapists*. Corpus.

Saragiotto, B. T., Maher, C. G., Yamato, T. P., Costa, L. O., Menezes Costa, L. C., Ostelo, R. W. and Macedo, L. G. (2016) *Motor control exercise for chronic non-specific low-back pain*, *Cochrane Database of Systematic Reviews*. Edited by B. T. Saragiotto. Chichester, UK: John Wiley & Sons, Ltd.

Savingley, P. (2009) *Low back pain : early management of persistent non-specific low back pain Full guideline May 2009 National Collaborating Centre for Primary Care*. Schilder, A., Hoheisel, U., Magerl, W., Benrath, J., Klein, T. and Treede, R.-D. (2014)

'Sensory findings after stimulation of the thoracolumbar fascia with hypertonic saline suggest its contribution to low back pain.', *Pain*. International Association for the Study of Pain, 155(2), pp. 222–31.

Schleip, R. (2003a) 'Fascial plasticity–a new neurobiological explanation Part 2', *Journal of Bodywork and Movement Therapies*. CHURCHILL LIVINGSTONE, 7(1), pp. 11–19.
Schleip, R. (2003b) 'Fascial plasticity – a new neurobiological explanation: Part 1', *Journal of Bodywork and Movement Therapies*, 7(1), pp. 11–19.

Sefton, J. M., Yarar, C., Berry, J. W. and Pascoe, D. D. (2010) 'Therapeutic massage of the neck and shoulders produces changes in peripheral blood flow when assessed with dynamic infrared thermography.', *Journal of alternative and complementary medicine (New York, N.Y.)*, 16(7), pp. 723–32.

Sefton, J. M., Yarar, C., Carpenter, D. M. and Berry, J. W. (2011) 'Physiological and clinical changes after therapeutic massage of the neck and shoulders.', *Manual therapy*, 16(5), pp. 487–94.

Shah, S. and Bhalara, A. (2012) 'Myofascial Release', *International Journal of Health Sciences and Research*, 2(2), pp. 417–423.

Shah, Y., Arkesteijn, M., Thomas, D., Therapy, S. and Whyman, J. (2017) 'The acute effects of integrated myofascial techniques on lumbar paraspinal blood flow compared with kinesio-taping : A pilot study', *Journal of Bodywork & Movement Therapies*. Elsevier Ltd, 21, pp. 459–467.

Sherman, K. J., Cherkin, D. C., Connelly, M. T., Erro, J., Savetsky, J. B., Davis, R. B. and Eisenberg, D. M. (2004) 'Complementary and alternative medical therapies for chronic low back pain: What treatments are patients willing to try?', *BMC complementary and alternative medicine*, 4(9).

Sherman, K. J., Dixon, M. W., Thompson, D. and Cherkin, D. C. (2006) 'Development of a taxonomy to describe massage treatments for musculoskeletal pain.', *BMC complementary and alternative medicine*, 6(24).

Shoemaker, J. K., Tiidus, P. M. and Mader, R. (1997) 'Failure of manual massage to alter limb blood flow: measures by Doppler ultrasound.', *Medicine and science in sports and exercise*, 29(5), pp. 610–614.

da Silva, R. A., Vieira, E. R., Cabrera, M., Altimari, L. R., Aguiar, A. F., Nowotny, A. H., Carvalho, A. F. and Oliveira, M. R. (2015) 'Back muscle fatigue of younger and older adults with and without chronic low back pain using two protocols: A case-control study', *Journal of Electromyography and Kinesiology*. Elsevier Ltd, 25(6), pp. 928–936.

da Silva, T., Mills, K., Brown, B. T., Herbert, R. D., Maher, C. G. and Hancock, M. J. (2017) 'Risk of Recurrence of Low Back Pain: A Systematic Review', *Journal of Orthopaedic & Sports Physical Therapy*, 47(5), pp. 305–313.

Simmonds, M. J., Olson, S. L., Jones, S., Hussein, T., Lee, C. E., Novy, D. and Radwan, H. (1998) 'Psychometric characteristics and clinical usefulness of physical performance tests in patients with low back pain.', *Spine*, 23(22), pp. 2412–21.

Simmonds, N., Miller, P. and Gemmell, H. (2012) 'A theoretical framework for the role of fascia in manual therapy.', *Journal of bodywork and movement therapies*. Elsevier Ltd, 16(1), pp. 83–93.

Simmons, S. (2011) 'Complementary and alternative therapies for low-back pain.', *Nursing*, 41(5), pp. 54–5.

Sions, J. M., Elliott, J. M., Pohlig, R. T. and Hicks, G. E. (2017) 'Trunk Muscle Characteristics of the Multifidi, Erector Spinae, Psoas, and Quadratus Lumborum in Older Adults With and Without Chronic Low Back Pain', *Journal of Orthopaedic & Sports Physical Therapy*, 47(3), pp. 173–179. d

Solomonow, M., Baratta, R. V., Zhou, B. H., Burger, E., Zieske, A. and Gedalia, A. (2003) 'Muscular dysfunction elicited by creep of lumbar viscoelastic tissue', *Journal of Electromyography and Kinesiology*. Elsevier, 13(4), pp. 381–396.

Solomonow, M., Baratta, R., Zhou, B., Burger, E., Zieske, A. and Gedalia, A. (2003) 'Muscular dysfunction elicited by creep of lumbar viscoelastic tissue', *Journal of Electromyography and Kinesiology*, 13(4), pp. 381–396.

Solomonow, M., Zhou, B.-H., Baratta, R. V., Lu, Y. and Harris, M. (1999) '1999 Volvo Award Winner in Biomechanical Studies', *Spine*, 24(23), p. 2426.

Stecco, A., Gesi, M., Stecco, C. and Stern, R. (2013) 'Fascial components of the myofascial pain syndrome.', *Current pain and headache reports*, 17(8).

Stecco, A., Stern, R., Fantoni, I., De Caro, R. and Stecco, C. (2015) 'Fascial Disorders: Implications for Treatment', *Pm&R*. Elsevier Ltd, 2015, pp. 1–8. d

Stecco, C., Hammer, W., Vleeming, A. and De Caro, R. (2015) *Functional Atlas of the Human Fascial System*, *Functional Atlas of the Human Fascial System*. Churchill Livingstone.

Stecco, L. (2004) *FASCIAL MANIPULATION for Musculoskeletal Pain*. Padova: Piccini.

Stedge, H. L., Kroskie, R. M. and Docherty, C. L. (2012) 'Kinesio taping and the circulation and endurance ratio of the gastrocnemius muscle', *Journal of Athletic Training*, 47(6), pp. 635–642.

Stratford, P. W., Binkley, J., Solomon, P., Gill, C. and Finch, E. (1994) 'Assessing change over time in patients with low back pain.', *Physical therapy*, 74(6), pp. 528–33.

Stratton, R. and Shiwen, X. (2010) 'Role of prostaglandins in fibroblast activation and fibrosis.', *Journal of cell communication and signaling*, 4(2), pp. 75–7. d

Sung, P. S., Lammers, A. R. and Danial, P. (2009) 'Different parts of erector spinae muscle fatigability in subjects with and without low back pain', *Spine Journal*. Elsevier Inc., 9(2), pp. 115–120.

Swinkels-Meewisse, I. E. ., Roelofs, J., Verbeek, A. L. ., Oostendorp, R. a. . and Vlaeyen, J. W. . (2003) 'Fear of movement/(re)injury, disability and participation in acute low back pain', *Pain*, 105(1–2), pp. 371–379.

Tanaka, T. H., Leisman, G., Mori, H. and Nishijo, K. (2002) 'BMC Complementary and Alternative Medicine The effect of massage on localized lumbar muscle fatigue', *BMC complementary and alternative medicine*, 2(9), pp. 1–8.

Taspinar, F., Aslan, U. B., Sabir, N. and Cavlak, U. (2013) 'Implementation of matrix rhythm therapy and conventional massage in young females and comparison of their acute effects on circulation.', *Journal of alternative and complementary medicine (New York, N.Y.)*, 19(10), pp. 826–32.

Taylor, J. L. and Butler, J. E. (2000) 'Changes in muscle afferents, motoneurons and motor drive during muscle fatigue', *European Journal of Applied Physiology*, 83, pp. 106–115.

Tesarz, J., Hoheisel, U., Wiedenhofer, B. and Mense, S. (2011) 'Sensory innervation of the thoracolumbar fascia in rats and humans', *Neuroscience*. 2011/09/29, 194, pp. 302–308.

Tesarz, J., Hoheisel, U., Wiedenhöfer, B. and Mense, S. (2011) 'Sensory innervation of the thoracolumbar fascia in rats and humans.', *Neuroscience*. Elsevier Inc., 194, pp. 302–8.

Thomas, G. D. and Segal, S. S. (2004) 'Neural control of muscle blood flow during exercise.', *Journal of applied physiology (Bethesda, Md. : 1985)*, 97(2), pp. 731–8.

Tozzi, P., Bongiorno, D. and Vitturini, C. (2011) 'Fascial release effects on patients with non-specific cervical or lumbar pain', *Journal of Bodywork and Movement Therapies*, 15(4), pp. 405–416.

Trampas, A., Kitsios, A., Sykaras, E., Symeonidis, S. and Lazarou, L. (2010) 'Clinical massage and modified Proprioceptive Neuromuscular Facilitation stretching in males with latent myofascial trigger points.', *Physical therapy in sport : official journal of the Association of Chartered Physiotherapists in Sports Medicine*. Elsevier Ltd, 11(3), pp. 91–8.

Trost, Z., France, C. R., Sullivan, M. J. and Thomas, J. S. (2012) 'Pain-related fear predicts reduced spinal motion following experimental back injury.', *Pain*, 153(5), pp. 1015–21.

Uebelhart, D., Bernard, J., Hartmann, D. J., Moro, L., Roth, M., Uebelhart, B., Rehailia, M., Mauco, G., Schmitt, D. A. and Alexandre, C. (2000) 'Modifications of bone and connective tissue after orthostatic bedrest', *Osteoporosis International.* Springer, 11(1), pp. 59–67.

Vlaeyen, J.W.S, Kole Snijders A.M.J, Rotteveel A.M, Rueskin R, Heutts, P. (1995) 'The role of fear/(re)injury in pain disability.', *Journal of Occupational Rehabilitation*, 5, pp. 235–252.

Vlaeyen, J. W. and Linton, S. J. (2000) 'Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art.', *Pain*, 85(3), pp. 317–32.

Vleeming, A., Mooney, V. and Stoeckart, R. (2007) *Movement, Stability & Lumbopelvic Pain: Integration of Research and Therapy*. 2, illustr edn. Edited by R. S. Andry Vleeming, Vert Mooney. Churchill Livingstone Elsevier.

Vleeming, A., Vert, M. and Stoeckart, R. (2007) *Movement Stability and Lumbopelvic pain*. Edited by V. Andre, V. Mooney, and R. Stoeckart. CHURCHILL LIVINGSTONE.
Voermans, N. C., Bönnemann, C. G., Huijing, P. A., Hamel, B. C., van Kuppevelt, T. H., de Haan, A., Schalkwijk, J., van Engelen, B. G. and Jenniskens, G. J. (2008) 'Clinical and molecular overlap between myopathies and inherited connective tissue diseases', *Neuromuscular Disorders*, 18(11), pp. 843–856.

Waddell, G. (1987) '1987 Volvo award in clinical sciences. a new clinical model for the treatment of the low back pain', *Spine*, 12, pp. 632–644.

Wang, Q., Zeng, H., Best, T. M., Haas, C., Heffner, N. T., Agarwal, S. and Zhao, Y. (2014) 'A mechatronic system for quantitative application and assessment of massage-like actions in small animals', *Annals of Biomedical Engineering*, 42(1), pp. 36–49.

Watson, P. J., Booker, C. K., Main, C. J. and Chen, a C. N. (1997) 'Surface electromyography in the identification of chronic low back pain patients: the development of the flexion relaxation ratio.', *Clinical biomechanics (Bristol, Avon)*, 12(3), pp. 165–171.

Watson, P. J., Booker, C. K., Main, C. J. and Chen, A. C. (1997) 'Surface electromyography in the identification of chronic low back pain patients: the development of the flexion relaxation ratio', *Clinical Biomechanics*. Elsevier, 12(3), pp. 165–171.

Weerapong, P., Hume, P. a and Kolt, G. S. (2005) 'The mechanisms of massage and effects on performance, muscle recovery and injury prevention.', *Sports medicine (Auckland, N.Z.)*, 35(3), pp. 235–56.

Willard, F. (2007) 'Biomechanical,clinical-anatomical and evolutiinary aspects of lumbopelvic pain: Clinical anatomical aspects', in Andry Vleeming, Vert Mooney, R. S. (ed.) *Movement, Stability & Lumbopelvic Pain: Integration of Research and Therapy*. 2, Illustr edn. Churchill Livingstone Elsevier, 2007, pp. 5–113.

Willard, F. H., Vleeming, a, Schuenke, M. D., Danneels, L. and Schleip, R. (2012) 'The thoracolumbar fascia: anatomy, function and clinical considerations.', *Journal of anatomy*, 221(6), pp. 507–36.

Williams, J. M., Haq, I. and Lee, R. Y. (2010) 'Is pain the cause of altered biomechanical functions in back pain sufferers?', *Human Movement Science*, 29, pp. 311–325.

Williams, S., Whatman, C., Hume, P. a. and Sheerin, K. (2012) 'Kinesio taping in treatment and prevention of sports injuries: A meta-analysis of the evidence for its effectiveness', *Sports Medicine*, 42(2), pp. 153–164.

Wiltshire, E. V., Poitras, V., Pak, M., Hong, T., Rayner, J. and Tschakovsky, M. E. (2010) 'Massage Impairs Postexercise Muscle Blood Flow and "Lactic Acid" Removal', *Med. Sci. Sport.*, 42, pp. 1062–1071.

Wong, C. K. (2012) 'Strain counterstrain: current concepts and clinical evidence.', *Manual therapy*. Elsevier Ltd, 17(1), pp. 2–8.

Wong, W. S., Kwok, H. Y., Luk, K. D. K., Chow, Y. F., Mak, K. H., Tam, B. K. H., Wong, E. T. and Fielding, R. (2010) 'Fear of movement/(re)injury in Chinese patients with chronic pain: Factorial validity of the Chinese version of the Tampa Scale for Kinesiophobia.', *Journal of rehabilitation medicine : official journal of the UEMS European Board of Physical and Rehabilitation Medicine*, 42(7), pp. 620–9.

Yahia, H., Rhalmi, S., Newman, N., Isler, M. and Isle, M. (1992) 'Sensory innervation of human thoracolumbar fascia Sensory innervation of human thoracolumbar fascia. An immunohistochemical study', *Acta Orthopaedica Scandinavica Acra Orthop S c a d*, 63(632), pp. 195–197.

Yang, G., Marras, W. S. and Best, T. M. (2011) 'The biochemical response to biomechanical tissue loading on the low back during physical work exposure.', *Clinical biomechanics (Bristol, Avon)*. Elsevier Ltd, 26(5), pp. 431–7.

Yoshihito, S., Yukihiro, Matsuyama and Naoki, I. (2005) 'Intramuscular oxygenation of exercising trunk muscle in elderly persons', *Journal of Lumbar Spine Dissorders*, 11(1), pp. 148–156.

Zügel, M., Maganaris, C. N., Wilke, J., Jurkat-Rott, K., Klingler, W., Wearing, S. C., Findley, T., Barbe, M. F., Steinacker, J. M., Vleeming, A., Bloch, W., Schleip, R. and Hodges, P. W. (2018) 'Fascial tissue research in sports medicine: from molecules to tissue adaptation, injury and diagnostics', *British Journal of Sports Medicine*, August(0), pp. 1–9.

Appendix 1

HEALTH QUESTIONNAIRE

Name……

Date of Birth…….. Age……

Please answer these questions truthfully and completely. The sole purpose of this questionnaire is to ensure that you are in a fit and healthy state to complete the exercise test.

ANY INFORMATION CONTAINED HEREIN WILL BE TREATED AS CONFIDENTIAL.

SECTION 1: GENERAL HEALTH QUESTIONS

Please read the 8 questions below carefully and answer each one honestly: check YES or NO.

	YES	NO
1. Has your doctor ever said that you have a heart condition or high blood pressure?	□	□
2. Do you feel pain in your chest at rest, during your daily activities of living, or when you do physical activity?	□	□
3. Do you lose balance because of dizziness or have you lost consciousness in the last 12 months? (Please answer NO if your dizziness was associated with over-breathing including vigorous exercise).	□	□
4. Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)?	□	□
If yes, please list condition(s) here:		
5. Are you currently taking prescribed medications for a chronic medical condition?	□	□
If yes, please list condition(s) and medications here:		
6. Do you currently have (or have you had within the past 12 months) a bone, joint or soft tissue (muscle, ligament, or tendon) problem that could be made worse by becoming more physically active? Please answer NO if you had a problem in the past but it *does not limit your ability* to be physically active.	□	□
If yes, please list condition(s) here:		
7. Has your doctor ever said that you should only do medically supervised physical activity?	□	□
8. Are you, or is there any chance you could be, pregnant?	□	□

If you answered NO to all of the questions above, you are cleared to take part in the exercise test

Go to SECTION 3 to sign the form. You do not need to complete section 2.

If you answered YES to one or more of the questions in Section 1 - PLEASE GO TO SECTION 2.

SECTION 2: CHRONIC MEDICAL CONDITIONS

Please read the questions below carefully and answer each one honestly: check YES or NO.

		YES	NO
1.	**Do you have arthritis, osteoporosis, or back problems?** If YES answer questions 1a-1c. If NO go to Question 2.	□	□
1a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking any medications or other treatments).	□	□
1b.	Do you have joint problems causing pain, a recent fracture or fracture caused by osteoporosis or cancer, displaced vertebrae (e.g. spondylolisthesis), and/or spondyloysis/pars defect (a crack in the bony ring on the back of the spinal column)?	□	□
1c.	Have you had steroid injections or taken steroid tablets regularly for more than 3 months?	□	□
2.	**Do you have cancer of any kind?** If YES answer questions 2a-2b. If NO, go to Question 3.	□	□
2a.	Does your cancer diagnosis include any of the following types: lung/bronchogenic, multiple myeloma (cancer of plasma cells), head and neck?	□	□
2b.	Are you currently receiving cancer therapy (such as chemotherapy or radiotherapy)?	□	□
3.	**Do you have heart disease or cardiovascular disease? This includes coronary artery disease, high blood pressure, heart failure, diagnosed abnormality or heart rhythm.** If YES answer questions 3a-3e. If NO go to Question 4.	□	□
3a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking any medications or other treatments).	□	□
3b.	Do you have an irregular heartbeat that requires medical management?	□	□

	(e.g. atrial fibrillation, premature ventricular contraction)		
3c.	Do you have chronic heart failure?	□	□
3d.	Do you have a resting blood pressure equal to or greater than 160/90mmHg with or without medication? Answer YES if you do not know your resting blood pressure.	□	□
3e.	Do you have diagnosed coronary artery (cardiovascular) disease and have not participated in regular physical activity in the last 2 months?	□	□

		YES	NO
4.	**Do you have any metabolic conditions? This includes Type 1 Diabetes, Type 2 Diabetes and Pre-Diabetes.** If YES answer questions 4a-4c. If NO, go to Question 5.	□	□
4a.	Is your blood sugar often above 13mmol/L? (Answer YES if you are not sure).	□	□
4b.	Do you have any signs or symptoms of diabetes complications such as heart or vascular disease and/or complications affecting your eyes, kidneys, OR the sensation in your toes and feet?	□	□
4c.	Do you have other metabolic conditions (such as thyroid disorders, current pregnancy related diabetes, chronic kidney disease, or liver problems)?	□	□
5.	**Do you have any mental health problems or learning difficulties?** This includes Alzheimer's, dementia, depression, anxiety disorder, eating disorder, psychotic disorder, intellectual disability and down syndrome. If YES answer questions 5a-5b. If NO go to Question 6.	□	□
5a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking any medications or other treatments).	□	□
5b.	Do you also have back problems affecting nerves or muscles?	□	□
6.	**Do you have a respiratory disease?** This includes chronic obstructive pulmonary disease, asthma, pulmonary high blood pressure. If YES answer questions 6a-6d. If NO, go to Question 7.	□	□
6a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking any medications or other treatments).	□	□
6b.	Has your doctor ever said you blood oxygen level is low at rest or during exercise and/or that you require supplemental oxygen therapy?	□	□
6c.	If asthmatic, do you currently have symptoms of chest tightness, wheezing, laboured breathing, consistent cough (more than 2 days/week), or have you used your rescue medication more than twice in the last week?	□	□
6d.	Has your doctor ever said you have high blood pressure in the blood vessels of your lungs?	□	□
7.	**Do you have a spinal cord injury?** This includes tetraplegia and paraplegia. If YES answer questions 7a-7c. If NO, go to Question 8.	□	□
7a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking any medications or other treatments).	□	□

7b.	Do you commonly exhibit low resting blood pressure significant enough to cause dizziness, light-headedness, and/or fainting?	□	□
7c.	Has your physician indicated that you exhibit sudden bouts of high blood pressure (known as autonomic dysreflexia)?	□	□

		YES	NO
8.	**Have you had a stroke?** This includes transient ischemic attack (TIA) or cerebrovascular event. If YES answer questions 8a-8c. If NO go to Question 9.	□	□
8a.	Do you have difficulty controlling your condition with medications or other physician-prescribed therapies? (Answer NO if you are not currently taking any medications or other treatments).	□	□
8b.	Do you have any impairment in walking or mobility?	□	□
8c.	Have you experienced a stroke or impairment in nerves or muscles in the past 6 months?	□	□
9.	**Do you have any other medical condition which is not listed above or do you have two or more medical conditions?** If you have other medical conditions, answer questions 9a-9c. If NO go to Question 10.	□	□
9a.	Have you experienced a blackout, fainted, or lost consciousness as a result of a head injury within the last 12 months OR have you had a diagnosed concussion within the last 12 months?	□	□
9b.	Do you have a medical condition that is not listed (such as epilepsy, neurological conditions, and kidney problems)?	□	□
9c.	Do you currently live with two or more medical conditions?	□	□
	Please list your medical condition(s) and any related medications here:		
10.	**Have you had a viral infection in the last 2 weeks (cough, cold, sore throat, etc.)?** If YES please provide details below:	□	□
11.	**Is there any other reason why you cannot take part in this exercise test?** If YES please provide details below:	□	□
12.	**Please provide brief details of your current weekly levels of physical activity (sport, physical fitness or conditioning activities), using the following classification for exertion level:** **L = light (slightly breathless)** **M = moderate (breathless)** **V = vigorous (very breathless)** **Activity** **Duration (mins.)** **Level (L/M/V)** **Monday** **Tuesday** **Wednesday**		

	Thursday **Friday** **Saturday** **Sunday**

Please see below for recommendations for your current medical condition and sign this document:

If you answered NO to all of the follow-up questions about your medical condition, you are cleared to take part in the exercise test.

If you answered YES to one or more of the follow-up questions about your medical condition it is strongly advised that you should seek further advice from a medical professional before taking part in the exercise test.

Back pain specific Questions

Would you please complete the following Questionnaire? If any of the conditions below apply to you please tick. The researcher will discuss with you whether or not it is appropriate to take part in the study. If none of the questions below apply to you, please proceed to question 1

ANY INFORMATION CONTAINED WITHIN HEREIN WILL BE TREATED AS CONFIDENTIAL

☐ If you have a serious infection you will have to wait 2 weeks before taking part.

☐ If you have had a previous severe back or leg injury, or surgery on your back.

☐ If you have any spinal deformity, ankylosing spondylitis, or rheumatoid arthritis in any part of the body.

☐ If you ever had a spinal fracture, a tumour in your back, or an infection around your spine.

☐ If you have ever had root compression or spinal disc damage.

☐ If you have cancer

☐ If you have a bleeding disorder, for example haemophilia. Or if you take warfarin or similar blood thinning medication.

☐ If you take corticosteroid medication, e.g. Prednisolone. Or high doses of inhaled steroids. Or if you have injections in your lower back.

☐ If you are pregnant or are planning pregnancy.

☐ If your back pain has lasted less than 3 months.

Further information:

1. Have you had lower back pain in the last 12 months?

☐ No

☐ Yes

Please state how many times in the last 12 months:

SECTION 3: DECLARATION

Please read and sign the declaration below:

I, the undersigned, have read, understood and completed this questionnaire to the best of my knowledge.

NAME: ……

SIGNATURE: ……DATE: ……..

SIGNATURE OF PARENT/GUARDIAN: ……

This health questionnaire is based around the PAR-Q+, which was developed by the Canadian Society for Exercise Physiology www.csep.ca

The School of Sport and Exercise Sciences
Participant Code:

Research Project: The acute effects of clinical massage on peripheral blood flow at the lumbar para-spinal myofascia.

Consent Form

	Please sign below
I have been informed of and understand the purpose of the study.	
I have been given the opportunity to ask questions.	
I understand I can withdraw at any time from this project without giving a reason.	
I agree to participate in the study as outlined to me.	
In the event of abnormality being discovered as a result of an ultrasound scan, I agree that I should be informed of the abnormality, that I may be referred, if necessary to the appropriate health care provider.	
Any information, which may potentially identify me, will not be used in published material.	

I would like to receive a summary of the results of this study. Please send to:	(postal or email address)

Name of participant:
Phone number: day: eve:
Email:
Participant signature: Date:

Appendix 2

Power and effect size calculation (Chapter 4)

Kinesiophobia - AA	Was there a change in Kinesiophobia AA score following interventions?	6	Mixed ANOVA Time	<001	η_p^2 = .569	η^2 = 0.43208 / 43.2% required sample @ 80% power = 45 Actual power = 0.35
	Was there an interaction effect between the groups?	6	Mixed ANOVA Interaction	.009	η_p^2 = .423	η^2 = 0.240 / 24% required sample @ 80% power = 194 Actual power = 0.09
	Was there a difference Kinesiophobia AA score between groups time pre?	6	Ind. T test	.782	n/a	
	Was there a difference in Kinesiophobia AA score between groups time post?	6	Ind. T test	.192	n/a	
	Was there a difference between Kinesiophobia AA score at time post compared to pre in the experimental group?	6	Paired T test	.004	Dz = 1.73 Statistical power = 0.96 given sample size. Required sample = 5 in each group	
	Was there a difference between Kinesiophobia-AA, score at time post compared to pre in the control group?	6	Paired T test	.198	n/a	
	Was there a change in Kinesiophobia SF score following interventions?	7	Mixed ANOVA Time	.051	η_p^2 = .262	η^2 = 0.2433 / 24.3% required sample @ 80% power = 139 Actual power = 0.14
	Was there an interaction effect between the groups?	7	Mixed ANOVA Interaction	.259	η_p^2 = .097	η^2 =0.07316 / 7.3%

Kinesiophobia - SF						required sample @ 80% power = 2229 Actual power = 0.05
	Was there a difference Kinesiophobia SF score between groups time pre?	7	Ind. T test	.802	n/a	
	Was there a difference in Kinesiophobia SF score between groups time post?	7	Ind. T test	.463	n/a	
	Was there a difference between Kinesiophobia SF score at time post compared to pre in the experimental group?	7	Paired T test	.086	d = 0.77 Statistical power = 0.41 given sample size. Required sample = 16 in each group	
	Was there a difference between Kinesiophobia SF score at time post compared to pre in the control group?	7	Paired T test	.451	n/a	
PRI Sensory	Was there a change in PRI sensory score following interventions?	8	Mixed ANOVA Time	<.001	η_p^2 = .630	η^2 = 0.4989 / 49.9% required sample @ 80% power = 34 Actual power = 0.45
	Was there an interaction effect between the groups?	8	Mixed ANOVA Interaction	.009	η_p^2 = .416	η^2 = 0.20835 / 20.8% required sample @ 80% power = 252 Actual power = 0.08
	Was there a difference PRI sensory score between groups time pre?	8	Ind. T test	.200	n/a	
	Was there a difference in PRI sensory score between groups time post?	8	Ind. T test	.229	n/a	
	Was there a difference between PRI sensory score at time post	8	Paired T test	.002	d z= 2.04 Statistical power= 0.99 given sample size.	

	compared to pre in the experimental group?				Required sample = 5 in each group	
	Was there a difference between PRI sensory score at time post compared to pre in the control group?	8	Paired T test	.270	n/a	
	Was there an interaction effect between the groups?	9	Mixed ANOVA Interaction	.364	η_p^2 =	η^2 = 0.045 / 4.5% required sample @ 80% power = 5388 Actual power = 0.05

Appendix 3

Reliability results for EMG and kinematic data trials (chapter 4)

*Table 10.1: mean coefficient of variation calculations for EMG onset and offset corresponding angles for the lumbar multifidus muscle and erector spinae muscle. Calculated as CV (%) = SD /mean *100%*

Muscle	**Lumbar multifidus (pre)**				**Erector spinae (pre)**			
	EMG onset angle lumbar	EMG onset angle trunk	EMG offset angle lumbar	EMG offset angle trunk	EMG onset angle lumbar	EMG onset angle trunk	EMG offset angle lumbar	EMG offset angle trunk
Left	0.526253	0.606816	0.159330173	0.114168561	0.631467	0.709726	0.105769035	0.105769
Right	0.56243	0.480603	0.199494585	0.13579	0.114719	0.538843	0.221608347	0.152409

Reliability results for EMG and data trials (chapter 7)

*Table 10.2: Coefficient of variation for RMS EMG trials per subject at first and last 10 second time intervals during fatigue tasks F1 and F2. Calculated as CV (%) = SD /mean *100%*

subject	**RMS Sham TENS**				**RMS MT**			
	F1 first 10 seconds	F1 last 10 seconds	F2 first 10 seconds	F2 last 10 seconds	F1 first 10 seconds	F1 last 10 seconds	F2 first 10 seconds	F2 last 10 seconds
1	0.367815235	0.055931628	0.015277387	0.040621731	0.025920189	0.092722245	7.67E-03	0.01442005
2	0.061268906	0.007961479	0.026464716	0.01844488	0.002488417	0.004280599	7.60E-03	0.00128413
3	0.092038465	0.011788095	0.003482956	0.006750248	0.00621849	0.069240919	1.32E-03	0.00679147
4	0.133894635	0.056118051	0.00684094	0.002946112	0.002324521	0.019852112	1.18E-03	0.01475953
5	0.051922458	0.03259407	0.01178497	0.009739449	0.013072808	0.004438198	6.42E-03	0.02667698
6	0.034234944	0.003622856	0.006038407	0.001125401	0.007331616	0.004852515	2.04E-03	0.00818593
7	0.041602701	0.049855547	0.005465129	1.045816689	0.030751183	0.014030925	5.92E-03	0.03048053
8	0.050161401	0.004887444	0.117919815	0.002621485	0.00214769	0.023821885	5.70E-03	0.02496136
9	0.229869161	0.07562217	0.11218172	0.000501622	0.014473634	0.026055844	2.70E-02	0.02952891
10	0.059221362	0.015201015	0.039022623	0.078758147	0.024115819	0.011131528	2.71E-02	0.19489184
11	0.043875371	0.004360628	0.014310867	0.032771285	0.006740069	0.03871369	4.11E-03	0.05875404

Appendix 4

Participant information sheet (generic)

Participation Information Sheet

Centre for Sports Studies
Medway Building
Chatham Maritime
Kent ME4 4AG

Research Project:

The effectiveness of structured, myofascial, massage for non-specific chronic low back pain.

Aims of the project:

The aim of this project is to look at the effects of structure myofascial massage techniques on changes in the lower back of people with and without lower back pain.

Background information

Most low back pain remains a diagnostic enigma and is appropriately classified as nonspecific low back pain (NSLBP). There are diverse views regarding the underlying causes however Injury and lifestyle may contribute to detrimental changes to various tissues of the lower back. This could contribute to pain, altered movement patterns and increased muscular activity in the lower back. The aim of this project is to investigate the effects of massage techniques on these symptoms of lower back pain.

What does the project involve?

The project involves looking at people with back pain. We will measure how your back muscles are working during a forward bending movement. This will be carried out using electromyography (EMG). Essentially when muscles are active they generate small electric currents. EMG sensors will be placed on the skin over the muscles to record the electric current generated during the

forward bending movement. It should be noted that these sensors are incapable of producing electricity themselves they can only detect electric currents.

During this forward bending pattern we will also measure how far forward the participant moves using a 3 dimensional motion tracker. Markers will be placed on the skin, which will be monitored with 3 cameras that will track the movement throughout.

What do I have to do?

The study requires participants to make 6-8 visits. Initially you will be invited a 1 hour session at a time convenient to you at either the University of Kent Sports Clinic at Medway Park, Mill Road, Gillingham, Kent or another suitable venue for an ultrasound scan; a measure of muscle activity at the lower back and motion analysis as outlined above.

- At the onset of the session you will be asked to complete a questionnaire about your health, back pain levels and activity levels.
- A member of the research team will measure your height and weight.
- For the EMG measurements the skin has to be shaved (if applicable) and cleaned to get a clear signal. Sensors will be placed on the skin at locations identified by a member of the research team.
- For the motion analysis a member of the research team will place 3 markers on the skin at three areas of the spine.
- You will then be asked to bend forward in a controlled manner while the motion tracker records the movement of the markers and the EMG sensors record the muscular activity at the lower back.

Once this preliminary information has been taken, you will then be placed into one of two massage groups. The next 6 visits will involve 1-hour sessions of massage therapy. These should be within 1 week of each other or twice a week for 3 weeks. If visiting twice a week the sessions should be on non-consecutive days. On the last visit you will be asked to perform the ultrasound scan, EMG and motion analysis once again.

It is important to note that no diagnosis will be made. However, if you have concerns about your lower back, we are able to refer you to suitable health practitioners. We also provide sources of further information about lower back pain at the end of the leaflet.

Who can be included in this project?

- If you are over 18
- If you currently have, or have had, lower back pain in the last 12 months

Who cannot take part?

- If you have a serious infection you will have to wait 2 weeks before taking part.
- If you have had a previous severe back or leg injury, or surgery on your back.
- If you have any spinal deformity, ankylosing spondylitis, or rheumatoid arthritis in any part of the body.
- If you ever had a spinal fracture, a tumour in your back, or an infection around your spine.
- If you have ever had root compression or spinal disc damage.
- If you have cancer
- If you have a bleeding disorder, for example haemophilia. Or if you take warfarin or similar blood thinning medication.
- If you take corticosteroid medication, e.g. Prednisolone. Or high doses of inhaled steroids. Or if you have injections in your lower back.
- If you are pregnant or are planning pregnancy.
- If your back pain has lasted less than 3 months.
- If your back pain has a score of less than 6/100 on the Oswestry Disability Questionnaire.

Are there any risks?

- EMG recordings are non-invasive and only detect electric current in the muscle.
- EMG electrodes by themselves do not produce any electricity.

Can I choose to withdraw from the project?

- You can change your mind and withdraw from the project at anytime and without giving any reason

Will taking part in the project affect my health or back care?

- Taking part in this project will not affect your usual care. You can maintain your usual medications and visit you doctor, or any other health care professional, if required.

How can I find out the results of the study?

- We can send you a summary of the results when the study is completed. You can indicate if you want to receive this on the consent form

Will the information be confidential?

- All data will be kept in accordance with the data protection act 2003. All data will be coded and no names will appear on the results sheets or ultrasound images. All data will be stored in a locked filing cabinet or on password protected files by the main researcher. No data will be

passed on to a third party. If the paper is published no individual references will be made.

How can I take part?

- Email the researcher ys70@kent.ac.uk
- Contact me on 07813090773
- A member of the research team will contact you to arrange an appointment

Researcher: Yusuf Shah

Yusuf Is a PhD Student at the university of Kent and a lecturer at K-College. Yusuf has worked as a sports and remedial massage therapist for over 14 years and is particularly interested in the effects of massage on lower back pain. This project is part of his PhD research.

PhD Supervisor: Professor Louis Passfield

Professor Passfield is the director of the Centre for Sports Studies at the University of Kent.

The Centre has approved this study for Sports Studies Ethics Committee (to be included only when approved)

Address for appointments and baseline measurements.

University of Kent Sports Lab
Medway Park
Mill Road
Gillingham
Kent
ME7 1HF

http://www.medwaypark.org.uk/medway_park/map_directions/

Sources of information about back pain

http://www.nhs.uk/conditions/back-pain/pages/introduction.aspx

If you have any further questions please feel free to contact Yusuf Shah, the main researcher:

Email: ys70@kent.ac.uk

Participation Information Sheet

The School of Sport and Exercise Sciences
Medway Building
Chatham Maritime
Kent ME4 4AG

Research Project:
The acute effects of massage on blood flow, localised muscle fatigue and postural balance

Aims of the project:
The aim of this project is to look at the effects of massage on blood flow and lower back muscle fatigue and balance.

Background information

Our understanding of back pain has improved in recent years. However the exact causes are still not entirely clear. Therapeutic massage is becoming one of the most prescribed treatments for many conditions. The possible effects of massage are likely to be achieved through a number of mechanisms due, in part, to changes in local blood flow. These improvements in local blood flow may have important implications for recovery, fatigue, improved balance and co-ordination.

What does the project involve?

The project involves measuring local blood flow and muscle activity of the back and postural balance after a fatiguing task to the muscles in the lower back region. This will be followed by two sessions of soft tissue therapy to the lower back after which the measurements are repeated. All measurements are non-invasive.

What do I have to do?

The study requires participants to make 3 visits. Initially you will be invited a 1-hour session at a time convenient to you at the University of Kent Sports Clinic at Medway Park, Mill Road, Gillingham, Kent for an initial measurement of blood flow, muscle activity and postural balance.

At the onset of the session you will be asked to complete a questionnaire about your health, back pain levels and activity levels.
A member of the research team will measure your height and weight.

For the muscle activity measurements the skin will be prepared (shaved if applicable) and cleaned to get a clear signal. Sensors will be placed on the skin at locations identified by a member of the research team.
For the blood flow measurements you will be asked to lie on a couch while the blood flow sensors are attached for recording. Two sensors will be placed over the skin at the lower back region, each one either side of your spine. This will be recorded for two minutes.
For the postural balance measurements you will be asked to stand on a foam balance pad for 30 seconds

Once this preliminary information has been taken, you will then be placed into one of two treatment groups, which will last approximately 30-40 minutes. On a separate day (with at least 1 week gap) you will then receive the second treatment, which will last the same time.

It is important to note that no diagnosis will be made. However, if you have concerns about your lower back, we are able to refer you to suitable health practitioners. We also provide sources of further information about lower back pain at the end of the leaflet.

Who can be included in this project?

If you are over 18
If you have lower back pain
If you currently have, or have had, lower back pain in the last 12 months

Who cannot take part?

If you have a serious infection you will have to wait 2 weeks before taking part.
If you have had a previous severe back or leg injury, or surgery on your back.
If you have any spinal deformity, ankylosing spondylitis, or rheumatoid arthritis in any part of the body.
If you ever had a spinal fracture, a tumour in your back, or an infection around your spine.
If you have ever had root compression or spinal disc damage.
If you have cancer
If you have a bleeding disorder, for example haemophilia. Or if you take warfarin or similar blood thinning medication.
If you take corticosteroid medication, e.g. Prednisolone. Or high doses of inhaled steroids. Or if you have injections in your lower back.
If you are pregnant or are planning pregnancy.
If your back pain has lasted less than 3 months.
Neurological or psychiatric conditions
Acute systemic infections

Are there any risks?

Muscle activity recordings are non-invasive and only detect electric current from the surface of the skin.

Muscle activity electrodes by themselves do not produce any electricity.
Near Infrared Spectroscopy is a non-invasive method of obtaining information about blood flow and oxygenation of tissues.

Can I choose to withdraw from the project?

You can change your mind and withdraw from the project at any time and without giving any reason

Will taking part in the project affect my health or back care?

Taking part in this project will not affect your usual care. You can maintain your usual medications and visit you doctor, or any other health care professional, if required.

How can I find out the results of the study?

We can send you a summary of the results when the study is completed. You can indicate if you want to receive this on the consent form

Will the information be confidential?

All data will be kept in accordance with the data protection act 1998. All data will be coded and no names will appear on the results sheet. All data will be stored in a locked filing cabinet or on password protected files by the main researcher. No data will be passed on to a third party. If the paper is published no individual references will be made.

How can I take part?

Email the researcher at ys70@kent.ac.uk
A member of the research team will contact you to arrange an appointment

Researcher: Yusuf Shah

Yusuf Is a PhD Student at the university of Kent and a Sessional Lecturer. Yusuf has worked as a sports and remedial massage therapist for over 15 years and is particularly interested in the effects of massage on lower back pain. This project is part of his PhD research.

PhD Supervisor: Professor Louis Passfield

Professor Passfield is the director of the School of Sport and Exercise Sciences at the University of Kent.

This study has been approved by the School of Sports and Exercise Sciences
Address for appointments and baseline measurements.

University of Kent Sports Clinic
Medway Park

Mill Road
Gillingham
Kent
ME7 1HF

http://www.medwaypark.org.uk/medway_park/map_directions/

Sources of information about back pain

http://www.nhs.uk/conditions/back-pain/pages/introduction.aspx

Appendix 5

Ethics approval forms

FULL ETHICS APPLICATION FOR RESEARCH WITH HUMAN PARTICIPANTS – FACULTY OF SCIENCES

University of Kent

- If any significant changes are made to the design of the research I will notify the Faculty of Sciences Research Ethics and Advisory Group (REAG) and understand that further review may be required before I can proceed to implement the change(s)
- I agree that I will notify the Faculty of Sciences Research Ethics Advisory Group of any unexpected adverse events that may occur during my research
- I agree to notify the Faculty of Sciences Research Ethics Advisory Group of any complaints I receive in connection with this research project

Signed: Name: Yusuf Shah	Date: 24/9/13

What to do next

Send your completed form, along with all supporting documentation, to the Faculties Support Office, at fso@kent.ac.uk.

Checklist	
Please ensure you have included the following with your application (where relevant): • Full research proposal (current project) • Participant information sheet • Consent form • Covering letter (if relevant) • Any questionnaires/interview schedules/topic guides to be used • Any approved instruments/measures to be used • Any advertising material to be used to recruit participants • Confirmation that project is covered by UoK insurance policies (if necessary)	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

REJ/ARC/12.02.13
S:\Committees\Research Ethics\Forms\Sciences\sciences-reag-full-app-form-feb-2013.docx

9-10-13

10.10.2013.

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If any of the questions in Section IV B is answered 'yes', a full ethics application must be made to the REAG. This also applies for studies not defined as 'research' in the narrow sense, i.e. evaluations/audits, etc. Complete this form and send it to the Faculties Support Office along with supporting documentation: a copy of the full research proposal; any participant information sheets and consent forms; any surveys, interview schedules; any advertising material or proposed website wording.

Overview
Name of Applicant(s)
Yusuf Shah
Contact Details
ys70@kent.ac.uk, ys99@kent.ac.uk
Title of Project::
The effectiveness of myofascial massage on localised lumbar muscle fatigue and postural control strategy in people with low back pain
Lay Summary (Please provide a brief summary of the study):
This project is a follow up study to determine the efficacy of structured myofascial massage techniques as an intervention for subjects with chronic non-specific lower back pain (CLBP). CLBP is considered to have a complex aetiology and many sufferers of the condition seek different types of therapies to relieve their symptoms, including soft tissue therapies such as massage. However, the variability in soft tissue massage techniques has made it difficult to determine its effectiveness in improving outcome measures.
Name of Supervisor(s) (If applicable):
Louis Passfield, Marco Arkesteijn
Application Reference Number (For office use only)

Risks and ethical issues
Please list the principal inclusion and exclusion criteria
Inclusion criteria for CLBP will be a history of recurrent or chronic lower back pain for at least 12 months. Recurrent lower back pain is defined as being present on occurring in multiple episodes over a 12-month period. Chronic lower back pain is defined as being present in a single episode within a 3-12 month period. Exclusion criteria are self-reported incidences of previous severe back or lower limb injury or surgery, major structural spinal deformity, ankylosing spondylitis or rheumatoid arthritis, spinal fracture, cancer, tumor or infection, nerve root compression, neurological or psychiatric conditions, bleeding disorders, corticosteroid medication via inhaler, or steroid injections at L2-3 in the lower back, pregnancy or attempting to conceive and acute systemic infection.
How long will each research participant be in the study in total, from when they give informed consent until their last contact with the research team?
3 hours. Following informed consent the participants will take part in a pre-intervention measurement session lasting approximately one hour. The participants with then take part in 2 intervention sessions which will last approximately 1 hour each.
What are the potential risks and burdens for research participants and how will you minimise them? (Describe any risks and burdens that could occur as a result of participation in the research, such as pain, discomfort,

distress, intrusion, inconvenience or changes to lifestyle. Describe what steps would be taken to minimise risks and burdens as far as possible)
There are minimal risks for the clients. All subjects will provide informed consent. Both interventions are non-invasive and utilise renowned soft tissue techniques that are taught as part of the sports Therapy programme. The myofascial massage techniques may involve more focussed pressure to 'tender points' or 'trigger points' but these techniques are relieving and will not exacerbate the area of intervention or have any other detrimental outcome for the participant. Only qualified massage therapists will be able to deliver the massage interventions. All the intervention treatments will be carried out at Medway park clinic to ensure client privacy and to minimise intrusion. The outcome measures in this study are non-invasive and the safety guidelines for, EMG analysis, balance analysis and NIRS data collection will be adhered to.
Please describe what measures you have in place in the event of any unexpected outcomes or adverse effects to participants arising from involvement in the project
Equipment malfunction A maintenance agreement has been agreed with the suppliers of ultrasound, EMG and balance analysis and NIRS equipment **Subject drop-out** Every effort will be made to provide a thorough understanding of the subject requirements. Extra subjects will be recruited in the event of drop out. Participants will be asked about any adverse experiences at each intervention visit as defined by Cherkin et.al. (2009).
Will interviews/questionnaires or group discussions include topics that might be sensitive, embarrassing or upsetting, or is it possible that criminal or other disclosures requiring action could occur during the study?
NO
If yes, please describe the procedures in place to deal with these issues
What is the potential benefit to research participants?
An initial pilot study revealed that back pain sufferers may show improved muscle activation patterns, improved blood flow to the paraspinal musculature following the use of massage as an intervention. Subjects will receive 2 free treatments which will be offered free of charge to all participants. Participants will also receive a summary of their results following their participation.
What are the potential risks to the researchers themselves?
Cross infection – Hygiene protocols will be maintained throughout. Subjects will sign a pre participation questionnaire informing them of the inclusion exclusion criteria
Will there be any risks to the University? (Consider issues such as reputational risk; research that may give rise to contentious or controversial findings; could the funder be considered controversial or have the potential to cause reputational risk to the University?)
No
Will any intervention or procedure, which would normally be considered a part of routine care, be withheld from the research participants? (If yes, give details and justification). For example, the disturbance of a school child's day or access to their normal educational entitlement and curriculum).
No

Recruitment and informed consent
How and by whom will potential participants, records or samples be identified?

Participants will initially be identified from the University of Kent's Sport an exercise Science student population. Subjects will be informed of the study thought the school's procedures. Information in the form of a participant information sheet (see attached) will be emailed to the school's administration department to disseminate to the students. The researcher may also take advantage of opportunistic sampling to increase the sample size and to meet the needs of potential back pain sufferers outside of the University environment. The participant information sheet and the informed consent will be applied as above for these potential subjects.

Will this involve reviewing or screening identifiable personal information of potential participants or any other person? (If 'yes', give details)

No

Has prior consent been obtained or will it be obtained for access to identifiable personal information?

Yes

Will you obtain informed consent from or on behalf of research participants? (If 'yes' please give details. If you are not planning to gain consent, please explain why not).

All subjects will provide informed consent prior to all procedures and interventions carried out within the study. Students will also complete an initial pre-test screening form to identify inclusion and exclusion criteria.

Will you record informed consent in writing? (If 'no', how will it be recorded?)

Yes

How long will you allow potential participants to decide whether or not to take part?

Participants will have as long as they wish to decide on whether to take part or not. Potential participants will have the opportunity to refrain from inclusion or to withdraw their participation at any time.

What arrangements have been made for persons who might not adequately understand verbal explanations or written information given in English, or have special communication needs? (eg, translation, use of interpreters?)

None

If no arrangements will be made, explain the reasons (eg, resource constraints)

Participants will initially be recruited from the student pool at the university of Kent. The researcher anticipates that verbal and written information in English will not be a barrier for the participants in any way. If any participant has any special communication needs the researcher will seek advice from the student support services at the university and either facilitate the subjects individual needs or review the subject's suitability to participate. However, as this is not a funded research project there are resource concerns with regards to the use of translators or interpreters.

Confidentiality

In this section personal data means any data relating to a participant who could potentially be identified. It includes pseudonymised data capable of being linked to a participant through a unique code number.

If you will be undertaking any of the following activities at any stage (including in the identification of potential participants) please give details and explain the safeguarding measures you will employ

-

- Email communication of the study to potential participants will be disseminated via the school's administrative department.

- All data will be kept in accordance with the data protection act (1998). All data will be coded and no names will appear on the results sheets or ultrasound images. All data will be stored in a locked filing cabinet or on password protected files by the main researcher. No data will be passed on to a third party. If the paper is published no individual references will be made

- RS Scan balance system will only record centre of pressure changes. No images of subjects will be recorded in any way. Centre of pressure data will be anonymous and dealt with as above.

How will you ensure the confidentiality of personal data? (eg, anonymisation or pseudonymisation of data)

All data will be kept in accordance with the data protection act (1998). All data will be coded and no names will appear on the results sheets or ultrasound images. All data will be stored in a locked filing cabinet or on password protected files by the main researcher. No data will be passed on to a third party. If the paper is published no individual references will be made.

Who will have access to participants' personal data during the study?

The lead researcher will be the only person with access to personal data. The PhD supervisors may wish to have access to certain data in his capacity to advise and supervise where necessary. However this data will not include any identifiable data. Subjects will be allocated a subject code to anonymise them during any data review process that may include external parties such as supervisors.

How long will personal data be stored or accessed after the study has ended? (If longer than 12 months, please justify)

The data will form part of a PhD thesis which will progress for approximately 3 more years. Data relevant to the study may be kept for the duration to review if required. However personal data not required will not be kept longer than is necessary.

Please note: as best practice, and as a requirement of many funders, where practical, researchers must develop a data management and sharing plan to enable the data to be made available for re-use, eg, for secondary research, and so sufficient metadata must be conserved to enable this while maintaining confidentiality commitments and the security of data.

Incentives and payments

Will research participants receive any payments, reimbursement of expenses or any other benefits or incentives for taking part in this research? (If 'yes', please give details)

No

Will individual researchers receive any personal payment over and above normal salary, or any other benefits or incentives, for taking part in this research? (If 'yes', please give details)

No

Does the Chief Investigator or any other investigator/collaborator have any direct personal involvement (e.g. financial, shareholding, personal relationship, etc) in the organisations sponsoring or funding the research that may give rise to a possible conflict of interest? (If 'yes', please give details)

NO

Publication and dissemination

How do you intend to report and disseminate the results of the study? If you do not plan to report or disseminate the results please give your justification

The results of the study will be disseminated initially to the PhD supervisor and possibly for publication or presentation as part of the PhD thesis requirements.

Will you inform participants of the results? (Please give details of how you will inform participants or justify if not doing so)

Participants will be given the opportunity to review the results as a matter of personal interest. Participants will provide an email address that can be used to forward a summary of the study's findings.

Management of the research		
Other key investigators/collaborators. (Please include all grant co-applicants, protocol authors and other key members of the Chief Investigator's team, including non-doctoral student researchers)		
NA		
Has this or a similar application been previously rejected by a research Ethics Committee in the UK or another country? (If yes, please give details of rejected application and explain in the summary of main issues how the reasons for the unfavourable opinion have been addressed in this application)		
NO		
How long do you expect the study to last?		
• Planned start date: Jan 2016	• Planned end date: March 2016	• Total duration: 3 months
Where will the research take place?		
University Of Kent		

Insurance/indemnity
Does UoK's insurer need to be notified about your project before insurance cover can be provided? *The majority of research carried out at UoK is covered automatically by existing policies, however, if your project entails more than usual risk or involves an overseas country in the developing world or where there is or has recently been conflict, please check with the Insurance Office that cover can be provided. Please give details below.*
NO

Children
Do you plan to include any participants who are children under 16? (If no, go to next section)
NO
Please specify the potential age range of children under 16 who will be included and give reasons for carrying out the research with this age group
NA
Please describe the arrangements for seeking informed consent from a person with parental responsibility and/or from children able to give consent for themselves
NA
If you intend to provide children under 16 with information about the research and seek their consent or agreement, please outline how this process will vary according to their age and level of understanding
NA

Participants unable to consent for themselves
Do you plan to include any participants who are adults unable to consent for themselves through physical or mental incapacity? (If yes, the research must be reviewed by an NHS REC or SCREC)
NO
Is the research related to the 'impairing condition' that causes the lack of capacity, or to the treatment of those with that condition?

☐ Yes	If 'yes' proceed to next question
☒ No	If 'no' the study should proceed without involving those who do not have the capacity to consent to participation
Could the research be undertaken as effectively with people who do have the capacity to consent to participate?	
☐ Yes	If 'yes' then the study should exclude those without the capacity to consent to participation
☐ No	If 'no' then the inclusion of people without capacity in the study can be justified
Is it possible that the capacity of participants could fluctuate during the research? (If yes, the research must be reviewed by an NHS REC or SCREC)	
NA	
Who inside or outside the research team will decide whether or not the participants have the capacity to give consent? What training/experience will they have to enable them to reach this decision?	
The subject sample will be taken, primarily, from the university population who will have the capacity to give consent. All participants will be subject to the participant information and consent documentation prior to inclusion. The researcher will be able to make an informed judgment to exclude subjects that are unable to give consent.	
What will be the criteria for withdrawal of participants?	
Participants will be screened for inclusion and exclusion prior to participation. Those included in the study will have the right to withdraw at any stage.	

Declaration

To be signed by the Chief Investigator

- I agree to comply, and will ensure that all researchers involved with the study comply with all relevant legislation, accepted ethical practice, University of Kent policies and appropriate professional ethical guidelines during the conduct of this research project
- If any significant changes are made to the design of the research I will notify the Faculty of Sciences Research Ethics and Advisory Group (REAG) and understand that further review may be required before I can proceed to implement the change(s)
- I agree that I will notify the Faculty of Sciences Research Ethics Advisory Group of any unexpected adverse events that may occur during my research
- I agree to notify the Faculty of Sciences Research Ethics Advisory Group of any complaints I receive in connection with this research project

Signed: Name: Yusuf Shah	Date: 5/11/15

What to do next

Send your completed form, along with all supporting documentation, to the Faculties Support Office, at fso@kent.ac.uk.

Checklist	
Please ensure you have included the following with your application (where relevant): • Full research proposal (current project) • Participant information sheet • Consent form • Covering letter (if relevant) • Any questionnaires/interview schedules/topic guides to be used • Any approved instruments/measures to be used • Any advertising material to be used to recruit participants • Confirmation that project is covered by UoK insurance policies (if necessary)	☒ ☒ ☒ ☒ ☒ ☒ ☒ ☒

ETHICS REVIEW CHECKLIST FOR RESEARCH WITH HUMAN PARTICIPANTS – FACULTY OF SCIENCES

University of Kent

A checklist should be completed for every research project in order to identify whether a full application for ethics approval needs to be submitted.

The principal investigator or, where the principal investigator is a student, the supervisor, is responsible for exercising appropriate professional judgement in this review.

This checklist must be completed before potential participants are approached to take part in any research.

All forms must be signed by the School's Research Ethics Advisory Group representative.

Section I: Project details			
Project title:	The effectiveness of myofascial massage on localised lumbar muscle fatigue and postural control strategy in people with low back pain		
Planned start date: Jan 2016		Planned end date: July 2016	
Funder:			

Section II: Applicant details			
Applicant name:	Yusuf Shah		
Department:	Sport and Exercise Sciences		
Email: ys70@kent.ac.uk		Telephone number: 07813090773	
Contact address:	17 Beaconsfield Road, Bexley. DA52AE		
Applicant signature:	Yusuf Shah	**Date**	24/9/13

Section III: Students only				
Supervisor:				
Undergrad. ☐	Postgrad ☐	Masters ☐	Doctorate ☒	Other (please specify)
Supervisor signature:			**Date**	4/12/15
School REAG rep signature:	S.N. Hamilton.		**Date**	12-1-16

If all questions in the checklist are answered as 'no', **send the completed and signed form to the Faculties Support Office (at fso@kent.ac.uk), with any further required documents, for their records.**

If any question in Section IV(A) are answered 'yes', **you will need to send the completed form to the Faculties Support Office (at fso@kent.ac.uk) for reference and submit your research for ethics approval to the appropriate body. For advice and assistance with this process please contact the Research Ethics & Governance Officer.** Once ethical approval is granted, a copy should be sent to the Faculties Support Office for their records.

If any questions in Section IV(B) are answered 'yes', **you will need to complete the full application form and send it to the Faculties Support Office (at fso@kent.ac.uk) for review by the Sciences Research Ethics Advisory Group (REAG) along with a copy of the project protocol and any supporting documentation such as patient information sheets and consent forms.**

Please note that it is your responsibility to follow, and to ensure that all researchers involved with your project follow, accepted ethical practice and appropriate professional ethical guidelines in the conduct of your study. You must take all reasonable steps to protect the dignity, rights, safety and well-being of participants. This includes providing participants with appropriate information sheets, ensuring informed consent and ensuring confidentiality in the storage and use of data.

Any significant change in the question, design or conduct over the course of the research should be notified to the Faculties Support Office (at fso@kent.ac.uk) and may require a new application for ethics approval.

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ETHICS REVIEW CHECKLIST FOR RESEARCH WITH HUMAN PARTICIPANTS – FACULTY OF SCIENCES

University of Kent

A checklist should be completed for every research project in order to identify whether a full application for ethics approval needs to be submitted. The principal investigator or, where the principal investigator is a student, the supervisor, is responsible for exercising appropriate professional judgement in this review.

This checklist must be completed before potential participants are approached to take part in any research. All forms must be signed by the School's Research Ethics Advisory Group representative.

Section I: Project details		
Project title:	The effectiveness of myofascial massage on localised lumbar muscle fatigue and postural control strategy in people with low back pain	
Planned start date: Jan 2015		Planned end date: July2015
Funder:	NA	

Section II: Applicant details		
Applicant name:	Yusuf Shah	
Department:	School of Sport and Exercise Sciences	
Email: ys99@kent.ac.uk		Telephone number: 01322315442
Contact address:	17 Beaconsfield Road Bexley DA52AE	

Applicant signature	Yusuf Shah	**Date**	20/12/15

Section III: Students only			
Supervisor:			
Undergraduate ☐	Masters ☐	Doctorate ☒	Other (please specify)

Supervisor name	Professor Louis Passfield	**Date**	
Supervisor signature			

School REAG rep signature (required for both staff and students)	S.M. Mackenzie	**Date**	12.01.2016

Prop 34 - 2015 -16

If any question in Section IV(A) are answered 'yes':
1. **Contact Nicole Palmer** (University Research Ethics & Governance Officer) for advice
2. Send a copy of ethical approval to the Faculties Support Office, once received

If any questions in Section IV(B) are answered 'yes':
1. **Complete full application form**
2. **Send to the Faculties Support Office** for review by the Research Ethics Advisory Group (REAG)

If all questions in Section IV(A) and IV(B) are answered as 'no', **send the completed and signed form to the Faculties Support Office.**

Declaration: Please note that it is your responsibility to follow, and to ensure that, all researchers involved with your project follow accepted ethical practice and appropriate professional ethical guidelines in the conduct of your study. You must take all reasonable steps to protect the dignity, rights, safety and well-being of participants. This includes providing participants with appropriate information sheets, ensuring informed consent and ensuring confidentiality in the storage and use of data.

Appendix 6

Chapter 8, study 5 raw data

Participant data, PPT and time to fatigue data

group	Code	Gender	Age	Height (cm)	Weight (KG)	Skinfold	PPT 1	PPT 2	PPT 3
1	1	M	20.00	188.00	97.80	18.60	9.31	8.435	9.08
1	2	M	20.00	179.00	75.80	15.20	7.42	9.2	10.55
1	3	F	19.00	165.00	91.40	24.00	7.22	6.04	7.27
1	4	F	20.00	164.00	61.80	21.00	4.00	5.41	5.845
1	5	F	19.00	164.00	61.00	15.40	7.74	7.2	6
1	6	F	29.00	163.00	59.80	19.80	7.50	9.2	10.5
1	7	F	27.00	168.00	74.80	25.00	2.98	2.11	3.595
1	8	M	32.00	166.00	71.20	11.20	5.14	5.795	7.775
1	9	F	29.00	163.00	59.80	19.80	7.50	9.2	10.55
1	10	M	32.00	186.00	76.00	6.50	5.34	7.26	6.66
1	11	M	19.00	170.00	53.00	9.50	4.79	3.18	5.47
2	1	M	18	162	49	5.6	3.43	3.25	5.35
2	2	M	20	177	81	8	7.4	8.15	11.5
2	3	M	22	181	63.2	8.2	7.85	8.35	11.5

2	4	F	35	161	90.6	17	6.75	6.4	9
2	5	F	19	163.3	64.4	9.5	4.7	4.5	6.1
2	6	F	18	174	69	19	4.7	4.2	5.2
2	7	M	18	180	78	11	8.25	9.45	12.6
2	8	M	18	177	79	13.5	7.4	6.5	9.9
2	9	F	20	163	68.6	19	8	8.3	9.4
2	10	F	22	170	95	27.5	4	4	5
2	11	F	31	172	90	15.5	9.6	8.7	10.6

change_PPT_pre_L2	change_PPT_L1_L2	F1 time	F2 time	NBP_change_TTF_F1_F2	BMI
-0.23	0.65	93.00	90.00	-3.00	27.7
3.13	1.35	173.00	195.00	22.00	23.7
0.05	1.23	69.00	116.00	47.00	33.6
1.85	0.44	70.00	103.00	33.00	23.0
-1.74	-1.20	32.00	78.00	46.00	22.7
3.00	1.30	163.00	140.00	-23.00	22.5
0.62	1.49	90.00	142.00	52.00	26.5
2.64	1.98	54.00	108.00	54.00	25.8
3.05	1.35	163.00	140.00	-23.00	22.5
1.32	-0.60	101.00	170.00	69.00	22.0
0.68	2.29	106.00	123.00	17.00	18.3
1.92	2.10	205	270	65.00	18.7
4.10	3.35	94	115	21.00	25.9

3.65	**3.15**	**132**	**135**	**3.00**	**19.3**
2.25	**2.60**	**138**	**140**	**2.00**	**35.0**
1.40	**1.60**	**84**	**118**	**34.00**	**24.2**
0.50	**1.00**	**105**	**80**	**-25.00**	**22.8**
4.35	**3.15**	**52**	**85**	**33.00**	**24.1**
2.50	**3.40**	**76**	**91**	**15.00**	**25.2**
1.40	**1.10**	**85**	**118**	**33.00**	**25.8**
1.00	**1.00**	**15**	**47**	**32.00**	**32.9**
1.00	**1.90**	**77**	**125**	**48.00**	**30.4**

NIRS data

group	code	Change_Tsi%_1_MT	Change_O2Hb_1_MT	Change_HHb_1_MT	Change_tHb_1_MT	Change_HbDiff_1_MT	Change_Tsi%_2_MT	Change_O2Hb_2_MT	Change_HHb_2_MT	Change_tHb_2_MT	Change_HbDiff_2_MT	Change_Tsi%_3_MT	Change_O2Hb_3	Change_HHb_3_MT	Change_tHb_3_MT	Change_HbDiff_3_MT
1	1	-12.45456274	-12.36638586	2.239832404	-10.12655347	-14.60621823	14.83532255	21.97435349	3.030052026	25.00440551	18.94430147	14.38724858	29.8717892	8.449921707	38.32171081	21.42186748
1	2	-20.83901163	-16.14791502	7.582527715	-8.565387241	-23.73044255	-4.968882213	24.51320984	0.229679212	24.74288905	24.28353064	-13.22800703	18.3904566	4.103978767	22.49443535	14.28647782
1	3	-10.88385904	-0.392978432	-0.958475704	-1.351454124	0.565497263	-12.60473666	21.49055575	4.423893705	25.91444949	17.06671205	-15.2093818	18.0986707	-3.042004398	15.05666637	21.14067515
1	4	-20.89301371	-1.992030645	-0.540170304	-2.532200955	-1.451860341	3.82606748	7.797775188	14.65573614	20.52599355	17.60249362	-14.83442764	10.4226551	-0.041694756	10.3809603	10.46434983
1	5	-7.334249453	-7.884560761	-2.304984215	-10.18954498	-5.57957654	-6.064478337	15.97251477	1.702381476	17.67489625	14.27013326	1.756222035	10.918383	0.50172094	11.42010401	10.41666213
1	6	-3.795860466	6.689097929	5.781099049	12.47019697	0.907998878	6.574647137	9.831343683	0.67827802	10.50962171	9.153065664	4.968798541	21.5664695	4.737052152	26.30352168	16.82941738
1	7	-7.779277426	15.28508107	0.062056888	15.34713795	15.22302418	1.447495562	27.89228448	-2.721746034	25.17053843	30.61403051	-12.02841165	39.7595028	1.823535362	41.58303817	37.93596735
1	8	-17.85695513	-1.497524008	6.094375065	4.596851078	-7.591899086	0.076393234	18.20396619	3.390778433	21.59474464	14.81318774	-17.07187021	13.4490313	8.012699249	21.46173056	5.436332077
1	9	-10.39933956	2.47272553	2.849257461	5.321982997	-0.376531926	-3.448448574	8.616230449	2.324438252	10.94066871	6.291792201	4.041527229	10.1236406	3.170070938	13.29371153	6.953569649
1	10	-66.12315774	-42.79626688	-7.578547482	-50.37481423	-35.21771884	4.340287822	20.55691692	-0.531914147	20.02500276	21.08883105	-45.24804137	-24.558799	-10.00587344	-34.56467242	-14.55292551
1	11	-45.28307785	-4.596008688	4.24946128	-0.346547413	-8.845469967	-45.28307785	5.7343386	-3.666569936	2.06816866	9.401108512	-37.41586739	2.59649948	-4.516859641	-1.920060158	7.113409111
2	1	-24.44128063	-21.52114565	4.01408264	-17.50746302	-25.53532827	-3.059838199	110.8661343	42.25257001	153.1185546	68.61336438	-1.205571894	83.654833	42.7865665	126.4412495	40.86806646
2	2	-39.60368547	-12.67127285	14.23614867	1.564875878	-26.9074216	-20.06053212	14.99506142	2.359504556	17.35456593	12.63555683	-28.90400247	-0.6411363	13.26710417	12.62596789	-13.90824049
2	3	-60.16845329	-34.06577659	14.44804795	-19.61772864	-48.5138245	-15.53789971	16.95830727	6.466372696	23.42467979	10.49193457	-42.31108819	-20.414183	19.97200691	-0.442175809	-40.38618954
2	4	13.17177481	6.560656128	3.47256295	10.03321907	3.088093176	9.312514396	17.60563107	2.178237117	19.78386817	15.42739392	10.76114573	26.329023	4.876918869	31.2059419	21.45210418
2	5	-25.610298	-7.51134513	5.798703536	-1.712641582	-13.31004867	-0.001119877	14.62377913	1.723944357	16.34772345	12.89983478	-23.17256076	17.1247114	10.48521424	27.60992566	6.63949716
2	6	-5.499458721	4.589245728	5.787392251	10.37663797	-1.198146529	1.993119791	19.93400136	1.048356773	20.98235814	18.88564458	-2.900209265	22.8184602	5.72578559	28.54424578	17.09267464
2	7	-28.73864058	-22.09999642	6.933979115	-15.16601732	-29.03397562	1.672234283	29.22928067	3.941397263	33.17067781	25.28788341	-21.67489373	4.59254862	13.12980578	17.72235442	-8.537257182
2	8	-28.82024175	-11.60669257	10.43017171	-1.176520841	-22.03686431	-0.049253091	22.99081917	3.819759587	26.81057868	19.17105955	-30.18362365	17.9245071	16.2231549	34.14766202	1.701352233
2	9	-14.81950406	-5.483420729	2.599803089	-2.883617642	-8.083223819	1.996682313	21.9402618	4.053386293	25.99364812	17.88687552	-4.386782375	18.6932561	7.647747937	26.34100403	11.04550814
2	10	-3.449650286	-9.416879485	-4.378979775	-13.79585923	-5.037899676	3.995271456	7.892557598	2.404048507	10.2966061	5.488509091	0.800084091	3.9483436	-0.923824812	3.0245188	4.872168408
2	11	-11.73973823	-4.041877817	1.064576427	-2.977301388	-5.106454247	13.08247027	14.43479364	2.266958334	16.70175197	12.16783531	5.161431088	20.4853567	5.618375646	26.10373234	14.86698109

Chapter 7 study 4 data

Participant, PPT, time to fatigue data

Code	**gender**	Ht	Wt	age	skinfold	Bothersome score	fatigue 1 time mr	fatigue 2 time mr	fatigue 1 time tens	fatigue 2 time tens
1	M	188.00	97.80	20.00	18.60	2.00	1.33	1.30	1.06	1.02
2	M	179.00	75.80	20.00	15.20	2.00	2.53	3.15	2.27	1.54
3	F	165.00	91.40	19.00	24.00	1.00	1.09	1.56	2.13	2.29
4	F	164.00	61.80	20.00	21.00	2.00	1.10	1.43	0.58	1.09
5	F	164.00	61.00	19.00	15.40	1.00	0.32	1.18	0.45	0.50
6	F	163.00	59.80	29.00	19.80	0.00	2.43	2.20	2.45	2.00
7	F	168.00	74.80	27.00	25.00	1.00	1.30	2.22	1.07	1.47
8	M	166.00	71.20	32.00	11.20	2.00	0.54	1.48	1.03	0.53
9	F	163.00	59.80	29.00	19.80	2.00	2.43	2.20	2.45	2.00
10	M	186.00	76.00	32.00	6.50	2.00	1.41	1.50	2.20	1.50
11	M	170.00	53.00	19.00	9.50	2.00	1.46	2.03	2.25	1.80

Code	**gender**	PPT baseline	PPT baseline	PPT L_Load1	PPT L_load 2	PPT R_load1 MR	PPT R_load2 MR	PPT L_load1 tens	PPT L_load2 tens	PPT R_load1 tens	PPT R_load2 tens
1	M	9.40	9.21	8.93	9.08	7.94	9.08	9.00	10.10	9.4	11
2	M	6.82	8.02	9.00	10.10	9.40	11.00	7.50	6.82	7.8	8.02
3	F	7.25	7.18	5.90	7.03	6.18	7.51	7.25	7.52	7.19	7.23
4	F	4.00	4.00	5.49	5.73	5.33	5.96	4.00	6.00	3.6	5.1
5	F	6.81	8.66	7.70	6.00	6.70	6.00	5.75	5.05	6.07	6.1
6	F	7.26	7.74	9.00	10.00	9.40	11.00	9.03	10.36	9.06	10.48
7	F	3.53	2.43	2.16	3.64	2.06	3.55	4.23	4.52	5.19	6.23
8	M	5.50	4.78	5.25	7.72	6.34	7.83	4.42	3.50	3.5	1.86
9	F	7.26	7.74	9.00	10.10	9.40	11.00	9.03	10.36	9.05	10.48
10	M	5.19	5.49	6.79	6.65	7.73	6.67	8.66	7.96	7.55	7.51
11	M	4.82	4.76	3.69	4.83	2.67	6.11	2.76	3.24	4.28	3.48

NIRS data

code	Change_Tsi%_1_MT	Change_TSiQCF_1_MT	Change_O2Hb_1_MT	Change_HHb_1_MT	Change_tHb_1_MT	Change_HbDiff_1_MT	Change_Tsi%_1_tens	Change_TSiQCF_1_tens	Change_O2Hb_1_tens	Change_HHb_1_tens	Change_tHb_1_tens	Change_HbDiff_1_tens
1	-12.45456274	-40.1217169	-12.36638586	2.239832404	-10.12655347	-14.60621823	-10.49698199	-0.13090154	3.40707228	0.78737146	4.193843736	2.61970081
2	-20.83901163	-23.79165409	-16.14791502	7.582527715	-8.565387241	-23.73044255	-11.84678056	0.944547984	-6.289461442	2.550933905	-3.738527536	-8.84039534
3	-10.88385904	2.272808832	-0.392978432	-0.958475704	-1.351454124	0.565497263	-20.11644211	-0.191779627	-1.232547381	2.503564222	1.271016838	-3.736111608
4	-20.89301371	-1.129904376	-1.992030645	-0.540170304	-2.532200955	-1.451860341	-20.28992861	-2.131437995	-13.0716327	5.175808048	-7.895824644	-18.24744074
5	-7.334249453	-8.265940144	-7.884560761	-2.304984215	-10.18954498	-5.57957654	-15.31075663	0.964983711	-8.063334151	-1.15284646	-9.216180605	-6.910487684
6	-3.795860466	-10.17718522	6.689097929	5.781099049	12.47019697	0.907998878	-3.182808089	-0.08647771	3.920999727	4.810561207	8.73156093	-0.889561475
7	-7.779277426	0.028257889	15.28508107	0.062056888	15.34713795	15.22302418	-24.76976239	-43.32700502	14.92446649	23.37742691	38.30189342	-8.452960414
8	-17.85695513	0.861735888	-1.497524008	6.094375065	4.596851078	-7.591899086	-20.58637024	-0.164793277	-8.352622341	5.135524158	-3.217098185	-13.48814651
9	-10.39933956	1.829656646	2.47272553	2.849257461	5.321982997	-0.376531926	-4.823974474	-0.118885255	-0.509462107	1.661249141	1.15178704	-2.170711248
10	-66.12315774	-25.9024009	-42.79626688	-7.578547482	-50.37481423	-35.21771884	-53.89375263	-0.137428852	-15.09988347	7.030158872	-8.06962459	-22.12999231
11	-45.28307785	-0.435386659	-4.596008688	4.24946128	-0.346547413	-8.845469967	0.243181873	0.046895346	10.78592826	4.180024536	14.96595273	6.605903749

Change_Tsi%_2_MT	Change_TSiQCF_	Change_O2Hb_2	Change_HHb_2_	Change_tHb_2_M	Change_HbDiff_	Change_Tsi%_2_	Change_TSiQCF_	Change_O2Hb_2	Change_HHb_2_	Change_tHb_2_t	Change_HbDiff_
14.83532255	-15.230647	21.9743535	3.03005203	25.0044055	18.9443015	5.46984927	-0.0938599	8.95542052	2.87854165	11.8334622	6.07687887
-4.968882213	-25.63109	24.5132098	0.22967921	24.7428891	24.2835306	-6.661392	0.90873329	-2.6377346	0.98417828	-1.6535563	-3.6219129
-12.60473666	45.3187873	21.4905558	4.4238937	25.9144495	17.0667121	5.54104849	0.05764329	-4.0034209	0.08620704	-3.9172138	-4.0896279
3.82606748	5.04938737	7.79777519	14.6557361	20.5259936	17.6024936	-7.2809712	-0.0973124	-1.8381913	0.83994997	-0.9982413	-2.6781412
-6.064478337	14.0226982	15.9725148	1.70238148	17.6748962	14.2701333	-1.0370345	-0.166133	-0.6782216	-1.564302	-2.2425236	0.88608033
6.574647137	-1.6818764	9.83134368	0.67827802	10.5096217	9.15306566	9.78568399	-0.276986	5.30037727	-1.7467307	3.55364659	7.04710794
1.447495562	-0.0233649	27.8922845	-2.721746	25.1705384	30.6140305	-2.8384015	0.02310527	15.4459411	-0.5756286	14.8703124	16.0215697
0.076393234	0.67916955	18.2039662	3.39077843	21.5947446	14.8131877	-10.552442	-0.2299751	-4.8527261	1.08319398	-3.7695321	-5.9359201
-3.448448574	0.94040924	8.61623045	2.32443825	10.9406687	6.2917922	6.97864534	0.04858981	3.27445033	0.92128146	4.19573179	2.35316887
4.340287822	-0.0919661	20.5569169	-0.5319141	20.0250028	21.088831	5.68443059	0.0880006	-0.8050141	-3.3848163	-7.2948424	-0.3152226
-45.28307785	-0.4353867	5.7343386	-3.6665699	2.06816866	9.40110851	1.95648153	0.02739659	1.40783358	-1.8202828	-0.4124493	3.22811643

Change_Tsi%_3_	Change_TSiQCF_	Change_O2Hb_3	Change_HHb_3_	Change_tHb_3_MT	Change_HbDiff_3_MT	Change_Tsi%_3_	Change_TSiQCF_	Change_O2Hb_3	Change_HHb_3_	Change_tHb_3_t	Change_HbDiff_3_tens
14.3872486	-28.289151	29.8717892	8.44992171	38.32171081	21.42186748	3.95670602	-0.0518124	3.95172539	0.57245903	4.52368443	3.379266352
-13.228007	-18.361742	18.3904566	4.10397877	22.49443535	14.28647782	-12.448727	0.96489655	-8.8892517	0.53299922	-8.3562525	-9.422250915
-15.209382	18.0672506	18.0986707	-3.0420044	15.05666637	21.14067515	2.15061252	-0.0098485	1.80765342	-1.5266869	0.28096648	3.33434036
-14.834428	-12.284592	10.4226551	-0.0416948	10.3809603	10.46434983	-11.525802	-0.0415003	-3.7259286	1.44650363	-2.279425	-5.172432239
1.75622203	14.9131477	10.918383	0.50172094	11.42010401	10.41666213	-18.636715	0.91987381	-7.7320034	-0.6745555	-8.4065589	-7.057447899
4.96879854	-1.5103642	21.5664695	4.73705215	26.30352168	16.82941738	9.86409911	-0.1493399	7.5438565	6.22599063	11.0473973	8.214910255
-12.028412	-15.026464	39.7595028	1.82353536	41.58303817	37.93596735	-12.114811	-0.0210315	23.248072	-1.0308319	22.2172401	24.27890389
-17.07187	0.96182685	13.4490313	8.01269925	21.46173056	5.436332077	-13.337989	0.02635269	-7.5894315	2.91390784	-4.6755237	-10.50333938
4.04152723	1.6543026	10.1236406	3.17007094	13.29371153	6.953569649	1.8727337	0.04943802	5.8740493	3.35311151	9.22716082	2.520937785
-45.248041	-0.0819534	-24.558799	-10.005873	-34.56467242	-14.55292551	-56.774058	-0.0664262	-13.857	5.27100099	-8.5858989	-19.12795085
-37.415867	0.3142733	2.59649948	-4.5168596	-1.920060158	7.113409111	-9.9840968	0.11514855	4.37801846	1.80320081	6.18121928	2.574817697

EMG data (RMS log percentage change from F1 start)

group	F1_Start	f1_end%change	f2_start%change	F2_end%change
1	1.9635	2.223919871	10.37433155	4.089635854
1	2.045	1.662591687	2.454767726	3.02200489
1	2.1235	2.197629699	4.092300447	3.301153756
1	2.018	55.17013545	92.79484638	5.609514371
1	2.100833333	-2.268940896	2.768742562	-0.315747719
1	2.173166667	-0.490835187	6.637012041	-0.891172636
1	2.0095	-2.944347682	2.811644688	3.329186365
1	2.2935	-13.49465882	7.852626989	-18.20361892
1	2.164166667	9.272237197	8.263380824	6.757027339
1	1.585166667	34.49689833	30.34591526	36.81842078
1	2.152	-1.510223048	5.62267658	0.520446097
2	2.083	-2.880460874	2.726836294	-0.931349016
2	1.8596	0.053775005	16.82082168	4.205205421
2	1.991	-2.50125565	-1.67754897	-5.524861878
2	2.2494	0.248955277	-3.387570019	-3.343113719
2	2.0742	-3.683347797	1.870600714	-1.330633497
2	2.2528	1.846590909	-2.237215909	-8.016690341
2	2.1282	-4.943144441	0.131566582	-4.990132506
2	2.8938	-25.606469	-1.803856521	-28.10145829
2	2.5228	4.915173617	-6.984303155	-4.891390518
2	1.7584	3.594176524	26.14877161	20.41628753
2	2.3278	-5.730732881	2.474439385	-10.72257067

EMG RMS raw data

Clientcode	Sham_L1start	Sham_L1end	sham_L2start	Sham_L2end	MT_L1start	MT_L1end	MT_L2start	MT_L2end
1	0.00008234	0.00008772	0.000108342	0.000110871	0.00008214	0.00007906	0.000122665	0.00012222
2	9.86634E-05	0.000099287	0.000109197	0.000105916	0.000115793	0.00011425	0.000122342	0.000119587
3	0.000121683	0.000121378	0.000117218	0.000116117	0.00009742	0.000108134	0.000105344	0.000105273
4	0.000138829	0.000135367	0.000137332	0.000136445	0.000100599	9.84942E-05	0.000101227	0.0000983
5	0.000116106	0.000110774	0.000117968	0.000119486	0.000134657	0.000134336	0.000124975	0.00012277
7	0.000126923	0.000120288	0.000130716	0.00012318	0.000118777	0.000108423	0.000125372	0.000116922
8	0.000118928	0.000114741	0.000124147	0.000224553	0.000127671	0.000128763	0.000119872	0.000115871
9	0.000108496	0.000105949	0.000118165	0.000115985	0.000120581	0.000114165	0.000121036	0.000114609
10	0.000105504	0.00007858	0.000134197	0.000100263	0.000178479	0.000120019	0.000186739	0.000121767
11	0.000118289	0.000127347	0.000131844	0.000134191	0.000131123	0.00013729	0.000156098	0.000142322
6	0.000124775	0.000117477	0.000130653	0.000127569	0.00012819	0.000123328	0.000134009	0.000116451

Footscan, COP data

group	subject	Eyes open	Eyes closed	f1	f2
1	1	173	324	365	363
1	2	175	401	253	175
1	3	253	135	210	168
1	4	265	449	420	364
1	5	151	284	220	275
1	6	165	263	626	270
1	7	130	223	235	245
1	8	188	444	388	434
1	9	174	458	392	484
1	10	164	293	316	306
1	11	238	321	284	308
2	1	173	324	467	366
2	2	175	401	267	175
2	3	253	135	197	258
2	4	265	449	500	375
2	5	151	284	308	337
2	6	165	263	234	236
2	7	130	223	212	753
2	8	188	444	781	829
2	9	174	458	392	422
2	10	164	293	313	275
2	11	238	321	409	275

Chapter 6 study 3 data

Participant data

Group	Name	PPT left pre	PPT right p	PPT left pos	PPT right p	PPT left cha	PPT right C	skinfold	wt	Ht	age
1	EM_1	16	13.2	18.8	14.2	2.8	1	15	53.6	1.58	21
1	OS_2	11.5	23	17	19.5	5.5	-3.5	12.2	78	1.79	21
1	JW_1					0	0	11	57	1.65	30
1	CP_1	12.5	17.8	16	15.8	3.5	-2	15	52	1.6	52
1	DT_1					0	0	24	91	1.82	34
1	JB_1	8	6.1	8	7	0	0.9	5	64.2	1.72	20
1	KG_1	31	33.5	27.2	34	-3.8	0.5	9	73.4	1.73	23
1	AC_2	25.5	26.2	28.4	30.2	2.9	4	22	96	1.79	18
1	LP_1	12	10	15.5	11.2	3.5	1.2	25	65	1.78	19
1	HS_1	15.2	15.6		18.8	19	3.2	22	77	1.7	28
1	VD_1					0	0		70	1.69	21
1	G3_1	15.8	15.2	14.8	17	-1	1.8	10	69	1.68	30
1	DT_2	10.5	9	13.2	13.2	2.7	4.2	10	60	1.61	23
1	LP_4	29	31.5	27	23	-2	-8.5	25	87	1.63	21
1	CO_1	12.2	18	13.1	15.2	0.9	-2.8	13	61	1.64	20
2	KL_1	13.5	15.5	12	15	-1.5	-0.5	9.2	65	1.75	21
2	AC_1					0	0	20	72	1.68	29
2	MR_1a	23	21.4	20	19.2	-3	-2.2	12.5	62	1.64	23
2	JH_1	21	24.5	24.4	25	3.4	0.5	8.5			
2	RC_2	10	11	14	15	4	4	13	85	1.88	34
2	AS_1	18.5	12	26.5	17.2	8	5.2	6	73	1.86	30
2	PI_1	14.8	19.25	19	21	4.2	1.75	6.4	69	1.65	34
2	AL_1	21.8	21.2	24	25.5	2.2	4.3	9	61	1.48	49
2	KP_2	12.2	13	9.2	8	-3	-5	12.5	72	1.65	34
2	IS_1	19	19	19.4	19.2	0.4	0.2	9.25	85.5	1.81	30
2	AK_1	12.2	13.2	16	9.5	3.8	-3.7	10.2	57.2	1.72	21
2	JV_1	13.2	6	12	9	-1.2	3	23	79	1.55	29
2	KH_1	24	31	29	33	5	2	15.6	86.4	1.81	21
2	AG_1	9	9	11	13.5	2	4.5	23	52	1.66	20
2	RE_2	36	30	36	36	0	6	16.5	68.4	1.78	24
3	KS_1	5.5	5.5	7.8	7.5	2.3	2	11	58.4	1.74	19
3	NH_2	10.8	14.5	13.4	10	2.6	-4.5	13.5	63	1.65	34
3	SA_1	11	11.2	12.4	11.8	1.4	0.6	12	71	1.72	20
3	MB_1	14.8	16.4	12	13.5	-2.8	-2.9	17	58	1.65	18
3	DB_1	17.8	19.8	14	14	-3.8	-5.8	13	68	1.63	18
3	KC_1	9	10.4	11.2	11.4	2.2	1	29	73	1.6	18
3	SS_1	14	13.5	10.2	11	-3.8	-2.5	21	84	1.74	21
3	RF_1	25	27.8	17	28	-8	0.2	12	76	1.76	23
3	AJ_1	28.4	32.6	26.4	30.2	-2	-2.4	28	77	1.62	24
3	AK_1	12.2	16	13.2	9.5	1	-6.5	10.2	57.2	1.72	23
3	G3_2	17.5	15	15.5	18.2	-2	3.2	30	69.8	1.68	39
3	KF_1	12.5	11	12	11.2	-0.5	0.2	15	58	1.75	32
3	NW_3	15	16	10	6.2	-5	-9.8	11.5	50	1.52	21
3	CR_5	5.2	4.5	10.2	8	5	3.5	23	54	1.6	21

NIRS Data

Group	Gender	Name	Tsi% pre	Tsi QCF pre	O2Hb pre	HHb pre	tHb pre	HbDiff pre	Tsi% post	Tsi QCF post	O2Hb post	HHb post	tHb post	HbDiff post
1	F	EM_1	78.6942266	99.85088906	1.24649929	-0.2083238	1.0383255	1.45512308	78.44394	99.8434643	0.16253678	-1.1028497	-0.940163	1.26568653
1	M	OS_2	66.6651261	99.8744159	-1.3253051	0.18918263	-1.1365225	-1.5147377	69.7655339	99.5703671	2.65434421	-0.5703335	2.08361066	3.22442777
1	F	JW_1	73.5963981	99.75483614	-0.1126194	-0.4879323	-0.6005517	0.37526283	62.2079256	99.6342538	2.76197899	0.13914359	2.90112258	2.62278541
1	F	CP_1	51.9025699	99.89082615	-4.3239437	-0.501004	-4.8252977	-3.8228898	73.8222683	99.7580665	5.24629109	-0.4122994	4.83364165	5.65864052
1	M	DT_1	61.43237	92.89636195	0.08698102	0.19744045	0.28482147	-0.1102594	55.9040453	91.6349321	0.6057734	-0.7100837	-0.1039103	1.31605715
1	M	JB_1	62.1423342	99.68545924	0.72441191	-0.5046266	0.21963527	1.22908855	63.3698984	99.6453625	3.33465873	-3.1265999	0.20790879	6.46130867
1	M	KG_1	54.9890265	99.72906498	1.98187308	0.31243019	2.29400328	1.66934289	45.5042724	99.728309	6.53671997	-0.2828155	6.25360448	6.81943545
1	M	AC_2	73.4243003	99.77445626	-1.9504275	-0.1936476	-2.1439751	-1.7567299	79.660183	99.8418671	-0.335091	-5.3495424	-5.6845333	5.01450141
1	F	LP_1	76.0794905	99.83055739	-3.1350401	-0.9967041	-4.1315942	-2.138236	73.3382187	99.7694016	-0.3191725	-3.1310243	-3.4500467	2.81195175
1	F	HS_1	71.3285328	99.82629455	1.32695251	-1.7682211	-0.4411186	3.09527362	74.1942738	99.8161709	2.04451622	-4.5714628	-2.5267965	6.61607899
1	F	VD_1	76.9912762	99.7240969	-18.805616	-30.582653	-49.387619	11.7772366	74.1056782	99.7662196	-19.1545	-30.880969	-50.034818	11.7266689
1	F	G3_1	78.3803479	99.8305792	48.0395209	12.5399766	60.5794974	35.4994943	72.5942077	99.8660612	43.9801732	11.1286395	55.1088127	32.8514836
1	M	DT_2	66.0234701	98.95480269	20.8702222	17.0685951	37.9383172	3.8014271	58.0015657	99.4506938	16.2682727	17.8668176	34.1345902	-1.5987449
1	F	LP_4	76.0216898	99.78955855	-3.2049471	-1.8957711	-5.1012183	-1.309376	80.6838862	99.7240936	3.11829386	-1.9440407	1.17375314	5.06213456
1	F	CO_1	56.6202669	99.76003115	0.37077439	0.52140063	0.89267502	-0.1502762	67.3388138	99.7306133	3.00453853	1.41225012	4.41728864	1.59263841
2	F	KL_1	77.2250828	76.59125102	19.518918	5.56649222	25.0859102	13.9521258	77.2732353	76.9804769	42.9903975	5.83827518	48.8291726	37.1518223
2	F	AC_1	81.2045762	78.57185295	1.21057462	0.23648399	1.44680861	0.97384063	81.8251649	78.4954431	21.6232433	4.02076109	25.6437544	17.6022322
2	M	MR_1a	61.4238783	76.94834574	1.60912235	0.56570625	2.1749286	1.0434661	61.4970316	78.5325593	30.5669453	6.6853168	37.252362	23.8816784
2	M	JH_1	54.440424	99.59087696	-0.7886039	-0.9348159	-1.7235698	0.14611208	62.9302777	99.7210005	24.9296954	4.3722845	29.3018299	20.5573109
2	M	RC_2	65.7962729	99.76279714	-0.2907503	-0.178613	-0.4694133	-0.1122373	69.5348622	99.8566429	26.7148011	4.38874	31.1034911	22.3259611
2	M	AS_1	56.2674214	99.64613445	0.33603899	0.14266304	0.47870203	0.19307595	59.9991578	99.7203254	6.21309308	-1.7333921	4.47970096	7.94618521
2	M	PI_1	62.5391724	99.68040551	-1.1686754	0.7162358	-0.4524896	-1.8848112	54.4470643	99.8439539	10.0684859	6.54018144	16.6086174	3.52840449
2	M	AL_1	56.9280535	99.74153916	3.44093437	0.54134452	3.98242889	2.89963985	54.9910495	99.7067639	22.8420429	7.77608877	30.6182817	15.0660041
2	F	KP_2	74.4023751	99.64797994	1.92069931	-0.0411787	1.87932058	1.96177804	72.1425008	99.1242153	21.4713665	4.35215938	25.8233259	17.1191071
2	M	IS_1	50.2905492	86.55392239	-6.1653503	3.45261691	-2.7120834	-9.6179172	45.9768297	79.4963545	30.786232	2.01254981	32.7994318	28.7737322
2	F	AK_1	43.7333902	99.53360149	-17.274245	-4.1820975	-21.456492	-13.092147	62.6781042	99.4900634	22.149984	5.78379023	27.9336242	16.3661937
2	F	JV_1	78.7598261	99.70160171	0.88618813	-0.3399409	0.54609727	1.22587898	80.8465281	99.8041184	11.1013324	0.98679812	12.0879805	10.1142843
2	M	KH_1	32.1947712	98.52798389	-1.2672841	-2.9996417	-4.2670758	1.73220754	47.9005713	95.0190584	30.0936335	-2.5504567	27.5430268	32.6439401
2	M	AG_1	74.6941035	99.8326231	-0.1304771	-0.2054589	-0.3354359	0.07533179	78.9047033	99.8709689	18.5032066	2.50766366	21.0113702	15.9958929
2	M	RE_2	64.3012497	99.82315141	1.63116203	-0.2163375	1.41517456	1.84779951	65.4463757	99.7863313	18.4977078	2.4470657	20.9451235	16.0509421
3	F	KS_1	33.3675063	99.08502876	0.53233442	-0.9333965	-0.4011621	1.4656809	61.963794	99.4522924	2.81087947	-4.9489298	-2.1381504	7.7597593
3	F	NH_2	47.9616098	99.49507791	95.073973	41.6284139	136.702537	53.4455641	50.8104259	99.4553161	94.7636559	40.9301416	135.693947	53.8335192
3	F	SA_1	72.4696255	99.87289083	0.86369882	0.47317544	1.33712426	0.39062338	73.8130815	99.8112857	0.46251189	0.11910862	0.58132051	0.34340328
3	F	MB_1	73.2992991	99.79149542	-3.504675	-2.2325923	-5.7371674	-1.2720327	73.0435878	99.8364585	-11.376609	-5.6892837	-17.065792	-5.6872751
3	F	DB_1	78.7082441	99.86281691	-10.760377	-4.9337945	-15.694072	-5.8265325	79.4172784	99.7269474	-1.5605293	-4.3364762	-5.8969055	2.77599688
3	F	KC_1	73.7956808	99.77443443	-1.2279096	-0.0785095	-1.3063191	-1.14935	72.696132	99.4588739	-15.068286	-6.8472704	-21.915456	-8.2209654
3	M	SS_1	80.4537991	99.85842698	-1.3061838	-0.7282991	-2.0348329	-0.5778347	83.8993819	99.8227025	-0.3017535	-2.8656956	-3.1677991	2.56399206
3	M	RF_1	63.1820978	99.8045567	14.1735108	6.78953745	20.9631982	7.38407334	58.4301307	99.5439928	22.3475942	7.5701945	29.9179387	14.7774997
3	F	AJ_1	75.1846922	99.72861601	-10.544874	-11.420032	-21.964755	0.87525767	77.2490496	99.6108124	0.26524508	-10.062715	-9.7973204	10.3280606
3	M	AK_1	68.2999213	99.64551518	-1.8264932	-0.0026163	-1.8284595	-1.8236769	52.5285136	98.9223565	9.35881818	4.45341858	13.8128868	4.90559959
3	F	G3_2	75.2883708	99.77246301	81.558709	22.6266213	104.18533	58.9320377	78.2129009	99.863344	78.8836931	19.2767032	98.1603963	59.6069399
3	F	KF_1	71.7882965	99.80084855	-0.1231948	-0.2015932	-0.325288	0.07819844	70.6572271	99.8230332	-1.9735673	-1.3048294	-3.2788967	-0.6689379
3	F	NW_3	69.138962	99.82907168	13.6813322	15.0377241	28.7185563	-1.3565918	70.3093227	99.7325594	11.6830267	12.601998	24.2845247	-0.9191714
3	F	CR_5	74.7272006	99.76038536	-23.77104	9.81288107	-13.958659	-33.584121	73.0697671	99.8721825	-21.269397	9.42111978	-11.848777	-30.690717

Chapter 5 study 2 data

Participant data

Group	gender	age	ht cm	wt	BMI
1	f	22	190	88	24.4
1	f	21	180	69	21.3
1	m	20	180	68	21
1	f	18	170	54	18.7
1	f	21	180	67	20.7
1	f	21	170	69	23.9
1	f	21	170	66	22.8
1	f	22	180	62	19.1
1	m	22	180	85	26.3
1	m	20	170	78	27
1	m	22	160	73	28.5
1	m	22	180	82	25.3
1	m	21	189	89	25.6
1	f	22	169	65	22.8
1	f	22	180	70	21.6
1	m	23	1.82	84	25.4
1	m	22	186	82	23.7
2	f	33	165	72	26.4
2	m	22	190	81.5	22.6
2	m	21	180	96	29.6
2	m	34	183	91	27.2
2	f	20	172	64.2	21.7
2	f	30	1.65	57	21
2	f	19	170	65	22.5
2	m	19	170	65	22.5
2	m	22	176	62	20
2	f	18	165	58	21.3
2	m	21	170	70	24.2
2	m	20	179	78	27
2	m	22	190	95	26.3
2	f	27	170	54	18.7
2	m	25	180	83	25.6
2	m	18	179	96	29.9
2	m	24	183	92	27.7
3	m	22	190	80	22.2
3	m	22	190	80	22.2
3	m	20	190	79	21.9
3	m	21	170	73	25.3
3	f	20	170	60	20.8
3	f	21	170	54	18.7
3	m	30	181	85	25.9
3	f	22	172	65	22
3	f	22	174	67	22.1
3	f	21	165	61	22.4

NIRS data

Group	Name	O2Hb pre	HHb pre	tHb pre	HbDiff pre	O2Hb post	HHb post	tHb post
1	CL_1	-0.881677031	0.5803436	-0.301133429	-1.461920631	3.496438981	1.269246293	4.765885273
1	HC_1	0.728924027	-0.27542854	0.453745489	1.004602565	1.021729086	0.532495121	1.554474204
1	M_1	1.036380034	0.027533546	1.06406358	1.008996488	9.811145393	4.213986721	14.02528211
1	RB_1	-0.131204345	-0.249825426	-0.381079771	0.118921084	-3.55778384	-4.402877633	-7.96071147
1	RC_1	1.634680908	0.722881453	2.357562361	0.911849453	2.681743525	1.258778308	3.940521832
1	SN_1	-35.21157733	36.15361699	0.94198967	-71.36524428	-2.654490112	0.180498722	-2.47404139
1	K_1	0.845169952	0.147685535	0.99305549	0.697784418	4.307024623	1.927849532	6.235074148
1	AR_1	-0.196264495	0.519373229	0.323108737	-0.715387721	3.940086439	-1.168534932	2.771551508
1	EU_1	-3.393775917	-1.026490251	-4.420166171	-2.367035672	-4.383925972	-3.296692959	-7.680518926
1	MO_1	-9.535361945	-2.553079813	-12.08844176	-6.982632129	-22.92930284	-0.620682437	-23.54998526
1	RL_1	1.863638818	0.006109476	1.869798294	1.857179343	-1.892704097	0.355915532	-1.536738563
1	SCH_1	-0.611499507	-0.067915278	-0.679764786	-0.543684229	8.791724723	1.675719212	10.46709394
1	SL_1	-0.215713488	-0.101361216	-0.317224703	-0.114302271	4.803644133	0.831460843	5.634954978
1	AP_1	14.23796204	12.36491567	26.60272769	1.873096383	22.16578604	10.46010142	32.62573751
1	LH_1	0.521394947	0.037299923	0.558444869	0.484245025	1.724695054	1.660306282	3.38475134
1	TV_1	26.57776501	23.85619116	50.43415605	2.722073824	27.15818964	13.07529465	40.23368427
1	SSC_1	-21.6453681	-8.114545719	-29.75971382	-13.53032239	-11.66907357	-10.92740227	-22.59627585
2	KP_1	1.603417391	-0.086109029	1.517708363	1.689726418	18.15607073	4.544282538	22.70075325
2	AS_1	-1.120579966	-0.265245782	-1.386275747	-0.855584183	5.177005719	-1.22629696	3.950258758
2	DM_1	-0.571400267	0.504655596	-0.066894669	-1.076005862	44.7460273	8.44727582	53.19315298
2	DT_1	-0.51762167	0.419584968	-0.098236701	-0.937256636	14.75476402	1.771008511	16.52557256
2	JB_1	-0.808342794	0.291966107	-0.516426688	-1.100208903	9.171194508	0.195786259	9.366930748
2	JW_2	0.54602937	0.093542484	0.627942076	0.468202353	12.92668203	1.761764743	14.68839678
2	LF_1	-0.277384908	-0.087849778	-0.365084688	-0.189685131	20.15355201	9.634094376	29.78779639
2	SR_1	9.655211513	12.22219127	21.87755277	-2.567129756	8.677721395	11.0110848	19.68895621
2	MA_1	-1.72570634	0.255534894	-1.470021444	-1.981341227	39.87950426	7.30387237	47.18352666
2	MBK_1	0.911755679	0.4540282	1.365333876	0.457427481	17.27513487	3.068876637	20.34356148
2	MH_1	0.245141768	0.128735442	0.373577213	0.116356326	28.29469486	7.80396047	36.09835534
2	OS_1	-2.079016563	1.609959409	-0.469657155	-3.689225974	21.48871626	4.875031827	26.36314808
2	TF_1	0.822607245	-0.787046346	0.035660898	1.61000359	25.55817482	0.996522082	26.55479694
2	SC_1	-0.334336611	1.100524642	0.765838031	-1.435011254	15.07030291	2.304928163	17.37488108
2	JZ_1	-0.113287131	-0.386142525	-0.499279656	0.272655393	33.26261902	4.735805239	37.9985742
2	ACC	0.431010013	0.547429932	0.978539945	-0.11626992	13.34664092	1.900985662	15.24772659
2	RBC_1	1.789902806	-0.558957303	1.231245505	2.349160108	13.20699497	-2.530156568	10.67713842
3	CSU_1	0.33982493	-1.783177047	-1.443852115	2.122651984	-3.076178914	-3.310080994	-6.386759915
3	CED_1	-21.23750683	-24.26769195	-45.50569876	3.029835117	-18.77554913	-25.13709007	-43.9131392
3	CTN_1	-13.0130439	-37.82325088	-50.83679474	24.80985697	-7.168681448	-35.89823082	-43.06741238
3	CJV_1	-7.450851575	-37.53097735	-44.98232892	30.07977578	-4.019598975	-37.81926776	-41.83936675
3	CBS_1	-33.14624521	-51.68395692	-84.83070216	18.53736176	-31.86891362	-52.88240096	-84.75181454
3	CGS_1	-21.82715758	-42.4820019	-64.30965947	20.65449433	-19.48964337	-42.75138764	-62.24153107
3	CIS_1	0.431010013	0.547429932	0.978539945	-0.11626992	2.672807974	-0.130006153	2.54290182
3	CIK_1	54.16227898	21.05780351	75.21988255	33.10422553	47.95716259	21.57007138	69.52703401
3	CSB_1	1.014832562	-0.831369561	0.183663	1.846702123	5.387295963	-1.55587279	3.831623169
3	CAM_1	7.933898852	-7.245564758	0.688534095	15.17996362	11.08223679	-7.030024024	4.052412761

Chapter 4 study 1 data

Participant disability, pain and ROM data

subject	Gender	Group	Age	Height (m)	Weight (kg)	Tampa score pre	Tampa score post	ODI score pre	ODI score post	PRI pre	PRI post	VAS score pre	VAS score post	Lumbar flexion (deg) pre	Lumbar flexion (deg) post	Trunk Flexion (deg) pre	Trunk Flexion (deg) post	Left lateral flexion (deg) pre	Left lateral flexion (deg) post	Right lateral flexion (deg) pre	Right lateral flexion (deg) post
1	male	A	47	1.71	89.9	25	23	9	3	12	3	4	1	96.1	94.6	118.1	116.7	33	35.5	30	32.1
2	female	A	40	1.62	62.5	28	18	10	4	18	3	5	2.2	92.6	84.9	117.4	113.3	44.9	45.7	40.8	38.4
3	female	A	19	1.66	59.4	24	15	9	4	19	6	4	2	83.4	78.6	105.6	104.6	34	35.2	32	34
4	female	A	19	1.66	69.4	21	17	15	9	11	8	3.5	3.2	55.7	52.5	86.5	92.5	43.2	40.9	41.9	38.8
5	male	A	21	1.88	81.5	37	34	9	9	11	2	7.7	0.8	43.7	72.4	76.2	104	32.2	37.6	35.5	42
6	male	A	19	1.68	65.1	17	15	6	2	15	7	2	1.1	87.6	84	108	108.1	38.6	51.9	32.7	56.2
7	female	A	21	1.68	85.8	32	21	8	6	12	6	5.8	3.2	89.7		101		31.5		29.5	
8	male	A	33	1.75	85.5	23	17	9	4	13	7	5.5	1.2	52	68	82	100.3	25.8	32.3	23.3	28.5
9	male	B	39	1.76	89.4	25	25	6	6	6	7	4	2.3	80.1	84.2	107.1	107.3	33.9	38.2	34.7	38.2
10	female	B	31	1.69	61	23	23	7	7	6	6	5	5.1	88.4	78.8	109	106.4	37	36	33	34.7
11	female	B	27	1.63	53	21	24	8	7	10	11	5.5	3	85.7	97.5	106.9	115.8	34	36	31	38.6
12	male	B	18	1.88	67	23	21	3	3	11	5	6.5	5.2	61.4	73.1	76.2	93.5	31.9	30.7	33.2	33.5
13	male	B	24	1.78	89.5	28	23	7	4	10	3	3	2.6	86.5	81.2	113.3	115.4	35.3	45.5	43.4	42.6
14	male	B	47	1.85	98.4	25	23	4	4	12	9	2.4	3.8	66.6	68.9	97.4	97.3	29.5	48.5	30.6	34.8
15	female	B	45	1.65	82.5	20	17	8	3	11	4	2	3	65.9	70.2	92.2	97.6	27.2	33.9	23	28.7
16	female	B	31	1.55	89	29	31	18	19	30	34	6.1	5.7	105.9	110.8	127.03	124	36.1	34.7	27.9	32.5