THE ROLE OF THE ILIOPSOAS MUSCLE COMPLEX IN CHRONIC SPINAL PAIN AND ASSOCIATED SIGNS AND SYMPTOMS

By

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This thesis is presented as a requirement for the degree of Doctor of Philosophy in the Department of Physiotherapy, Faculty of Medicine, Nursing, and Health Sciences at Flinders University, South Australia.

LIST OF TABLES:

ABBREVIATIONS

ADL: Activities of Daily Living
AUD: Australian dollar
Botox: Botulinum toxin
CLBP: Chronic low back pain
CNS: Central nervous system
CSP: Chronic spinal pain
EMG: Electromyography
FCREC: Flinders Clinical Research Ethics Committee
IMC: Iliopsoas Muscle Complex
IASP: International Association For The Study Of Pain
LBP: Low back pain
L-S joints: Lumbo-sacral joints
MRI: Magnetic Resonance Imaging
n: Number
NSAIDs: Non-steroidal anti-inflammatory drugs
NH&MRC: National Health and Medical Research Council
PM muscle: Psoas Major muscle
PPI: Present Pain Index
RCT: Randomised controlled trial
RCTs: Randomised controlled trials
ROM: Range of motion
SHL: Secondary hyperalgesic locus
TENS: Transcutaneous electrical nerve stimulation
TPPR: Trigger point pressure release
USD: United States dollar
v: versus

vs: versus

VAS: Visual analogue score

ZTTT: Zinc Tally Taste Test

Z joints: Zygapophysial (interchangeable: zygapophyseal, facet, intervertebral).

Definition of chronic low back pain as used in this research

The definition applied in the single blinded study (Chapter Five), and the study undertaken in Chapter Six, was chronic low back pain:

"Defined by its length (more than six months) and its resistance to conventional therapies" (Baszanger, 1990)

(with the included requirement the participant had been medically diagnosed).

Reasons for tense utilisation

The researcher has utilised the use of the past tense in most sections of the thesis to:

"describe the contents, findings, or conclusions of past research. It emphasises the completed nature of a past activity. It is often referred to as the 'reporting' tense, and is traditionally used by scholars to report all past findings, including even very current research in some cases". Monash University © 2015.

SUMMARY OF THIS THESIS

The objectives of these studies were to evaluate the role of the iliopsoas muscle complex (IMC) in chronic low back pain (CLBP) and chronic spinal pain (CSP), and to investigate associated signs and symptoms. Four studies were undertaken, with the content of these detailed in six Chapters.

Chapter Two contained an initial review of identified definitions of pain, CLBP and CSP to better inform further investigations of these perplexing common and confounding conditions. The researcher's clinical observations, experiences, and hypotheses arose from presentations seen in private practice over 39 years, which led to these studies being undertaken.

Six case histories were accessed from the researcher's private practice records of participants who had experienced lumbar and groin pain, identified as arising from myofascial trigger points (MTrPs) in the IMC. Although the presentations and diagnoses of these six participants were markedly different, trigger point pressure treatment (TPPR) returned the six participants to pre-onset functioning, with significant reduction, or abolition, of pain, and improved quality of life. The case studies were of CLBP, and groin pain, sufferers who had active and latent MTrPs in the IMC.

With these reports being so common a review is undertaken attempting to ascertain estimates of incidence, financial, and psychosocial costs, of CLBP, and anatomical structures that may be sources of this condition. Additionally, review is undertaken of many of non-invasive and, invasive treatments undertaken to resolve this frustratingly common disorder, which included a more detailed scrutiny of MTrPs in CLBP. This review detected sufficient evidence of MTrPs, as a potential cause of CLBP, with scrutiny directed to the IMC as being integral to this thesis.

The IMC was scrutinised in Chapter Three, including anatomical positioning, actions, functions, impact on the lumbar intervertebral discs, myofascial trigger point patterns, and sites. On the basis of ascertained evidence, the IMC was ascertained to have the potential to be a primary cause of CLBP, but with a paucity of research supporting this viewpoint. Requirement, then, is the undertaking of a systematic review to examine any previous research conducted on the IMC in CLBP.

Two systematic reviews were conducted in Chapter Four as part of a systematic to detect any previous studies treating the IMC in CLBP and CSP with trigger point pressure release (TTPR) techniques with a notable paucity of studies located. As only one study was identified in these two systematic reviews fitting the criteria, the need to investigate the role of the IMC in CLBP and CSP via a Randomised Controlled Trial (RCT) was ascertained. This RCT was undertaken to assess the effects of treating the

IMC, in CLBP and CSP, with TPPR.

Chapter Five was a RCT undertaken to evaluate the role the IMC in CLBP and CSP, with this muscle complex having received scant attention in the literature as being a potential causation of acute lumbar pain, CLBP and CSP. Subsequent spread of pain was noted to be reported in the lumbosacral (L-S), thoracic, and cervical spines, the medial compartment of the knee, and headaches in various regions. This spread of signs and symptoms led the researcher to hypothesise that these could potentially arise from the presence of MTrPs in the IMC, via its capacity to alter entire body biomechanics (Michele, 1962).

The RCT consisted of 63 CLBP and CSP participants randomised to the intervention or stretching groups to investigate the role of the IMC. Measures utilised were: a personalised questionnaire; the Short-form McGill Pain Questionnaire (Melzack, 1987; the Patient Disability Measure (Stratford et al., 1995; and height and weight measurements. The treatment group (n = 33) received 12 sessions of TPPR to the IMC undertaken twice weekly, and self-managed stretching of the IMC over six weeks, while the stretching group (n = 30) performed the same self-managed stretch twice daily for six weeks. A total of 51 participants completed to follow-up. The evaluation scores utilised in this study focussed on pain impinging on the participants' ability to undertake ADL, as evaluation of range of motion (ROM) had been evidenced as an unreliable

measure (Mellin, 1987). Analysis of outcome measures revealed a significant reduction in pain, and increased ability to perform ADL, in the treatment group when compared with the control group.

Treatment of the IMC using TPPR and self-administered stretching demonstrated an effective, and relatively inexpensive, treatment approach in the reduction of pain, and dysfunction, in CLBP and CSP participants at the cessation of this study.

Chapter Six reported the undertaking and findings of Study Four, being an evaluation of signs and symptoms of zinc deficiency, gastrointestinal and urinary dysfunction, and depression and anxiety, commonly reported by CLBP and CSP participants. Some of these signs and symptoms, frequently reported to the researcher by CLBP and CSP sufferers, were found to have received relatively scant attention in the literature, with others having been reported on more frequently. Sixty six participants, 38 CLBP (as per the definition Bazanger's definition) and CSP sufferers, and 28 non-spinal pain group (that is no participant had ever experienced any spinal pain) entered the study. Each participant underwent an examination of the IMC to detect MTrPs, completing a questionnaire that included known causes of, or factors contributing to, zinc deficiency, gastrointestinal and urinary functioning. The Zinc Tally Taste Test (ZTTT) was also conducted (Bryce-Smith and Hodgkinson, 1986).

Chapter Seven is a discussion of the findings from these studies, and possible implications for future clinical practice into various musculoskeletal disorders. Statistical evidence obtained from these studies indicated a treatment protocol of TPPR, and self-administered stretching of the IMC, effectively reduced pain and improved ADL function in CLBP and CSP participants, at least, in the short-term. CLBP and CSP participants had significantly lower levels of zinc detected by the ZTTT, and a higher incidence of depression, anxiety, gastrointestinal, and urinary dysfunction, when compared to those in the non-spinal pain group.

Statement of authorship

I certify that this thesis does not contain without acknowledgment any material previously submitted for any diploma or degree in any university and, to the best of my knowledge and belief, it does not contain any material previously published or written by another person except where due reference is made.

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Signed: A. Jefferis

Date: 28th November 2015

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Signed: Adrian Schoo

Date: 22 November 2015

Co-supervisor's Name: Prof Paul Worley

Signed:

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Dedication

This thesis is dedicated to two very special people who shaped my thinking in two of my passions; my treatment and my desire to learn more about this. Tragically, neither are here to read this document.

Firstly to the late Emeritus Professor T.G.C. Murrell who died of complications arising from spinal surgery in August of 2002. Thank you Tim for all the support given to my hypotheses and work. You always said I would meet resistance in my work, and this I have. I truly wish you were able to read this thesis, as you wrote the Foreword in my monograph on the iliopsoas muscle complex entitled 'Front to Back'. You were such a source of inspiration to me in bringing this work to fruition. I have continued to explore the role of the iliopsoas muscle complex in spinal pain, and I endeavour to honour you by furthering my work, which you gave so much support to.

The second person I truly honour is the late Professor Alf Nachemson who, as an orthopaedic surgeon, stood strongly in voicing his objection to surgical intervention as the favoured management of low back pain. Shortly before his death in 2006, Professor Nachemson sadly had to decline the invitation to assess my first submission.

To two men of integrity, I owe you my deepest thanks for your wisdom and inspiration.

Acknowledgments

I offer my thanks to the many people who have participated in, and contributed to, the journey that culminated in this thesis. The flame of their belief, and support, has taken me through to completion:

- To all the research participants who gave of their time in both studies
- To the participants who allowed the use of their case histories and X-rays
- John Kaye who previously gave invaluable advice. His constructive feed-back on the first draft of this thesis and advice on its form, structures and format were of inestimable help
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- Trent, my son, who has watched my work evolve over many years and sometimes remembers to ask, "How's it going?"
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who have offered unfaltering support and encouragement in their own wonderful ways.

As submission, for examination, is *de novo*, there are now others to thank for their participation, and assistance:

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- Professor Adrian Schoo
- Peter Jolley
- Leonie Davis
- Matthew Weighton.

CHAPTER ONE: Contextual preface

1.1 Clinical experience

This research has been the culmination of 39 years clinical practice primarily as a musculo-skeletal physiotherapist, with the majority of time having been in the treatment of patients with pain, and in particular, chronic spinal pain (CSP). Eighty percent of patients attending the researcher's practice had seen twelve or more previous practitioners.

The treatment approach used by the researcher has often informed by the principles and work of Janet Travell and David Simons, and more latterly other researchers, in the field of MTrPs. In clinical practice, the researcher observed that the majority of her participants reported low back pain (LBP), CLBP, and then thoracic and cervical pain progressing to headaches. They often remarked to the researcher that other, seemingly unrelated, symptoms in the gastrointestinal and urinary systems, appeared to diminish significantly as their back pain improved with TPPR. Many patients reported their first site of pain was in the lumbar region with subsequent spread to other areas as their LBP became chronic. Patients frequently reported pain, and restricted movements, in the medial compartment of the tibio-femoral joint, and hip joint, with these reports accompanied by radiographic osteo-arthritic changes in these joints. All patients attending the researcher's practice had active or latent MTrPs in the IMC. The researcher noted that there appeared to be a correlation between these reports, being on the ipsilateral side to the more problematic PM muscle or iliacus muscle. Pondering these reports, the researcher noted that the PM and trapezii muscles shared a common attachment on the thoracic 12 vertebræ with these two muscles described as spanning the area from the occiput to the lesser trochanter of the femur attaching to every cervical, thoracic and lumbar vertebræ, the clavicles, scapulæ, pelvis and the lesser trochanter of the femurs. This led the researcher to interrogate the

literature further, with the subsequent recognition that the evidence base for treating her participants was far from complete. This ultimately led to the pursuit of this formal piece of research.

To provide the reader with a snapshot of the clinical context of this research, six brief case studies are provided as illustrative of the many patients whose suffering motivated this research.

1.2 Case Studies

Throughout these Case Studies reference will be made to the use of a coolant gel. This coolant gel was prescribed as a self-management technique, similar to the recommended application of a coolant spray by Simons et al., (1999). Application of a coolant has been demonstrated to reduce nerve conduction velocity and reduce muscle spasm (Kanui, 1987; Oosterveld and Rasker, 1994). MTrPs have been reported to be maintained both locally and centrally (refer p.p.47-48). Prior to self-administered stretching, the coolant gel was instructed to be applied to the lower abdominal area being the skin area associated with the PM muscle thereby facilitating relative relaxation of the PM muscle (refer p.p 55-56).

1.2.1 Case Study One:

History:

A 20 year old female presented with chronic spinal pain (lumbar, thoracic and cervical), accompanied by severe temporal and occipital headaches. She had experienced minor lumbar pain in her mid-teens (plain radiographs were undertaken in the 5 years previously), but had continued to pursue her chosen career in a Bachelor of Dance and had led a normal life.

This patient reported that 18 months prior to her appointment she had been the driver of a motor car that was rear-ended by a truck. Her pain had been present since this accident with the severity of her symptoms forcing her to withdraw from a Bachelor of Dance degree. She then commenced part-time office work. Six months after this accident she again was the driver of a motor vehicle rear-ended by a light truck. After this accident she was forced to give up her office work due to further aggravation of her pain and other symptoms.

Since the two motor vehicle accidents she had consulted multiple practitioners but had experienced no alleviation of her pain with resultant debilitating restrictions on her activities of daily living such as dressing, driving, and hair washing.

Initial observation:

Having failed to respond, or having been aggravated by other assessments, therapies, and multiple pharmacological interventions this patient presented with what appeared to be a degree of distrust. She chose not to sit during history taking, describing this as her most painful position. She described pain in all positions with minor relief being obtained in side lying. The patient also reported gastrointestinal dysfunction, with fluctuations between constipation and loose bowel motions.

Objective assessment:

There was an observable left concave scoliosis in the patient's lumbar spine with left shoulder depression when compared to the right. An antero-inferior rotation of the entire pelvis was evident.

On palpation, active MTrPs were located along the course of the right iliopsoas muscle complex (IMC) and an active MTrP palpable superiorly on the left IMC with latent MTrPs inferiorly.

Evaluation of her zinc status ascertained her to be in Category One on the Zinc Tally Taste Test (ZTTT): (Bryce-Smith and Hodgkinson, 1986), this being reported as a severe deficiency (refer p.132).

Treatment:

Treatment initially focussed on the IMC with trigger point pressure release (TPPR) and self-administered stretching of the IMC, gradually progressing to undertaking a release of the agonists and antagonists of this complex. Treatment was delivered twice-weekly basis for three months initially focussing on the IMC to restore correct pelvic positioning, and correction of biomechanics. Subsequent TPPR was applied to the cervical region, but it was noted that the patient reported reduction of her severe headaches and cervical pain prior to this cervical TPPR commencing.

Outcome:

After 18 months, the patient returned to work 15 hours per week which has been maintained to date.

The chronological radiographic reports below demonstrated that objective improvement had occurred, either because TPPR of the IMC reducing pressures on the lumbar intervertebral discs, or via a natural reabsorption over time ha occurred. As TPPR may have been implicated in improvement these X-rays reports were included.

Dated 10.01.2000: plain radiographs.

"FINDINGS: There is a scoliosis concave to the left which would appear to be into the significant range, centred on T8/9, and is compensated at the L2 level. There does appear to be a slight pelvic tilt, the left hip sitting higher than the right."

- Dated 09.04.2002. MRI SCAN LUMBAR SPINE: "Comment. There are

degenerative changes ins [sic] the L4/5 and L5/S1 discs. There are broad based bulges at both levels. The appearances in the annulus at L5/S1 are suggestive of an annular tear and bulge."

- Dated 15.04.2004: CT CERVICAL AND LUMBAR SPINE:

"L2/3: A minor disc bulge is noted at this level with slight anterior indentation of the thecal sac which is not however significantly compromised. Exit foramina appear clear. No bone or joint abnormality seen."

- "L3/4: A minor disc bulge is noted with minimal anterior indentation of the thecal sac. There is no compromise of the thecal sac or exit foramina. No bone or joint abnormality seen."
- "L4/5: A minor disc bulge is seen at this level also. No disc herniation seen. There is no significant compromise of the thecal sac or of exit foramina. No bone or joint abnormality seen."
- "L5/S1: A broad based diffuse disc bulge is present with slight anterior indentation of the thecal sac which is not significantly compromised. No disc herniation is seen. No bone or joint abnormality seen."

A radiological report dated 20.06.2006 reported (11 months after the commencement of treatment):

- CT CERVICAL AND CT LUMBAR SPINE. "Lumbar spine: At the L2/3, L3/4 and L4/5 levels there are no disc bulges. The spinal canal appears generous at all three levels. The neural foramina appear adequate.

At the L5/S1 level there is a broad based disc bulge which slightly indents the thecal sac. It does not cause stenosis of the thecal sac. There is no foraminal stenosis"

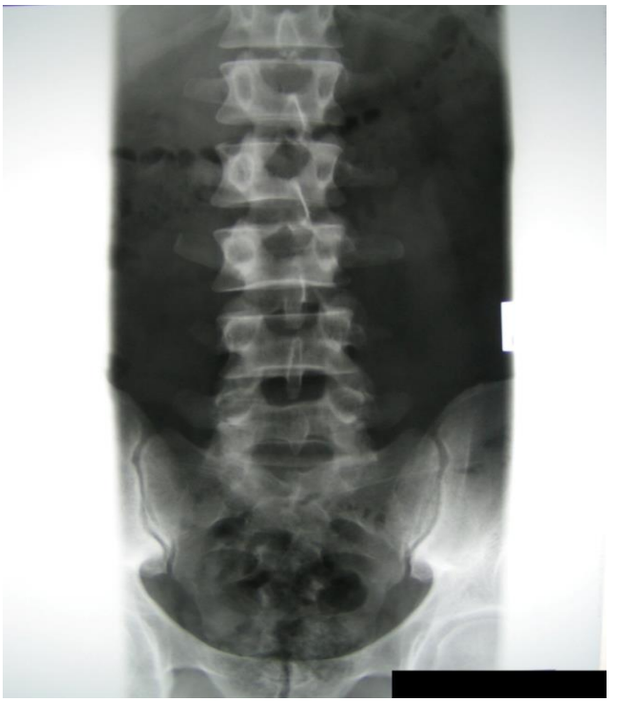
1.2.2 Case Study Two:

History:

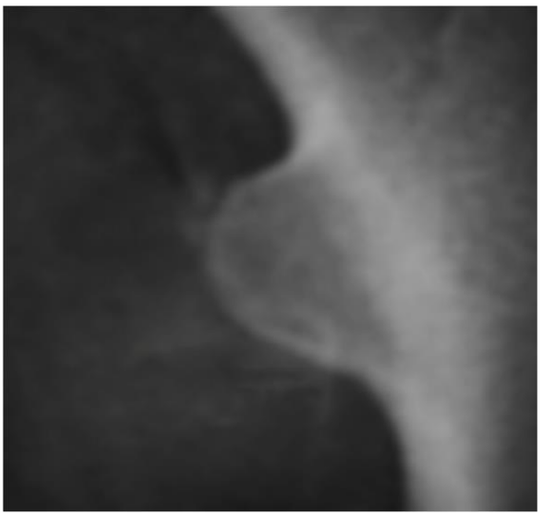
A 41 year old male presented with CSP (lumbar, thoracic, and cervical), right groin pain, and headaches predominantly in the right temporal region.

He had a horse fall on him in 7 years prior to consulting me. An X-ray was taken (X-ray A) at the time of accident. This X-ray showed no evidence of scoliosis but did evidence a slight left rotation of the lumbar 1-4 vertebrae. This injury was diagnosed as bruising to the kidney from which there was no residual pain or functional impairment.

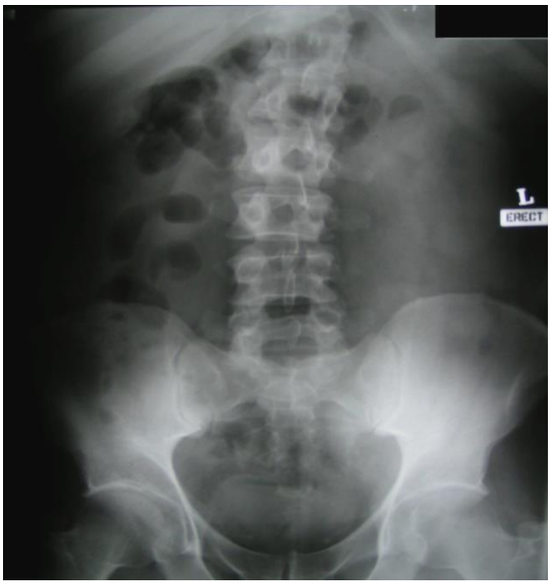
The patient reported no history of spinal pain until trampled by a horse two years before consulting me, at which time he was again X-rayed (X-ray B). X-ray B is a close- up view of the right lesser trochanter of the femur taken one week after the trauma, in which an avulsion fracture of the insertion of the right IMC on the lesser trochanter of the femur is apparent. This fracture was not reported initially, being identified by an orthopaedic surgeon two years later. In the intervening time this patient was repeatedly accused of being a malingerer by medical and other practitioners to whom he had been referred. The patient had also experienced constipation as one of his on-going problems since the second trauma. Faecal loading in the colon was evident in X-ray C with this being taken two and a half years post the second injury, and evidencing a roto-scoliosis concave to the left.



X-ray A



X-ray B



X-ray C

Examination:

Palpation revealed active MTrPs along the length of the right IMC, with a combination of latent and active MTrPs along the left IMC.

Evaluation of his zinc status ascertained he was in Category One on the ZTTT being a severe deficiency.

Treatment:

TPPR was commenced bilaterally on the IMC, its agonists and antagonists, along with concomitant self-management including stretching of the IMC.

Outcome:

This patient returned to part-time work two years after commencing treatment with me gradually being able to resume full time work four years later, which continues to this day. This case study is included as the radiological evidence raises the possibility of the PM muscle exerting an effect on the lumbar spine and possibly playing a role in the causation of scoliosis.

1.2.3 Case Study Three:

History:

A 28 year old male presented describing a 14 year history of progressively worsening chronic spinal pain (lumbar, thoracic, and cervical), and right groin pain. The lumbar region was the first reported site of pain. In the 13 years prior he had undertaken multiple treatment techniques, including chiropractic, physiotherapy joint mobilisation, Bowen therapy and massage, but his pain continued to worsen.

Examination:

The patient attended the researcher's practice with an X-ray (X-ray D) taken that year by a chiropractor, which had been the basis of eight months' chiropractic treatment on a weekly basis. X-ray D evidenced a severe roto-scoliosis of the lumbar spine concave to the right, and elevation of the entire right hemi pelvis. Palpation revealed active MTrPs along the length of the right IMC, with predominantly latent MTrPs along the length of the left IMC.

Treatment:

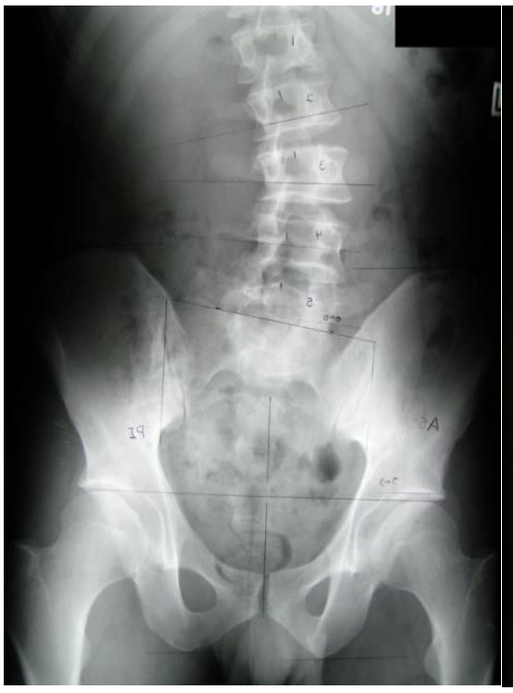
TPPR of the IMC was commenced on a weekly basis for three months and included treatment of the agonists and antagonists. After three months, treatments were progressed to fortnightly for a further period of three months.

Self-management consisted of the use of a coolant gel, concomitant with stretching of the IMC.

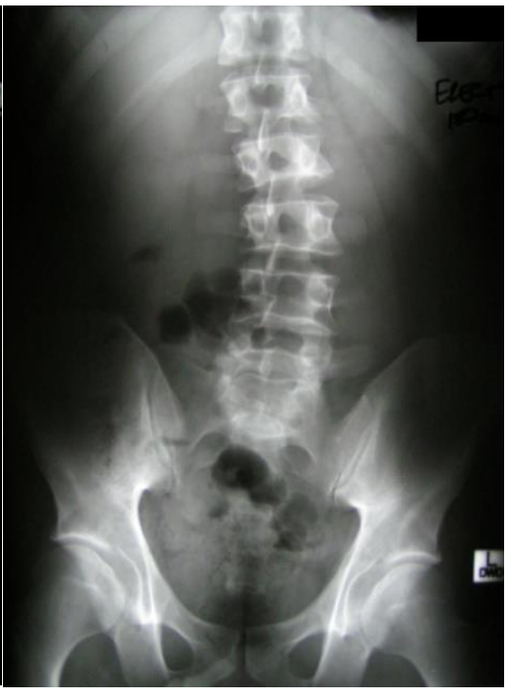
Outcome:

Nine months after commencing treatment the patient was pain-free in the lumbar and right groin regions. Repeat X-rays were performed (X-ray E) demonstrating a significant straightening of the roto-scoliosis. No further treatment was undertaken apart from

continued IMC stretching. He has remained pain free. This case raises the possibility that significant relief of chronic spinal pain and significant radiological improvement in scoliosis could be due to TPPR and a stretching protocol of the IMC.



X-ray D



X-ray E

1.2.4 Case Study Four:

History:

A 47 year old women consulted me with multiple symptoms that began when, as an 11-year old champion dancer, she experienced severe pain in her left groin. This worsened to the point that four days of traction was undertaken prior to the insertion of ‘pins’ into her left hip joint. Following this procedure, she spent three months on axillary crutches, with the ‘pins’ removed two years later.

After seven years from the onset of her initial pain, there had been a marked deterioration in her condition, with pain in the left groin and hip region exacerbating to the point she was reliant on significant quantities of analgesics. She ceased her professional dance career at age 21 due to pain and immobility. Advanced osteoarthritis of the left hip was diagnosed when she was 26 years old by an orthopaedic surgeon who commented that her condition would have most likely been exacerbated by her two pregnancies. Due to the severity of her problems, a left total hip replacement was performed later that year. Her children were, at that time, two years old, and seven months old.

From age 11, until age 47, she consulted five orthopaedic surgeons, multiple physiotherapists, chiropractors, osteopaths, had undertaken a Pain Management Unit course, hydrotherapy, and Pilates training. In the previous 36 years, her groin pain and symptoms persisted despite previous surgical interventions and multiple therapies. She had also developed lumbar pain that was still present, at her first consultation with the researcher, at age 47.

Two months before seeing the researcher, the patient fell forward landing on her knees, further exacerbating her pain and immobility. Due to the severity of her pain and

immobility, just prior to consulting my practice, she had consulted another orthopaedic surgeon who recommended revision surgery to insert a new total hip prosthesis. The surgeon stated he was unable to advise her as to the cause of her continuing symptoms until the surgical revision was undertaken, and informed her that in removing her first total hip replacement a fracture may occur in the shaft of femur. The patient declined the surgery.

The patient presented being unable to walk more than a few metres, and unable to stand to prepare her breakfast. She could not bend forward to put on her under-wear, trousers or foot-wear, and was unable to stand on her left leg. She also reported that she had experienced extreme constipation for the duration of her hip condition.

Observation/Examination:

Visual examination confirmed a six centimetre elevation of the left hemi pelvis compared to the right. The patient was unable to perform a unilateral leg stand on her left lower limb and was unable to lie supine, also demonstrating difficulty in taking the left lower limb into a crook position in an attempt to attain relief.

Palpation revealed absent or latent MTrPs along the right IMC and active MTrPs along the length of the left IMC. Active trigger points were also located in the left adductor muscles.

Evaluation of her zinc status ascertained she was in Category One on the ZTTT, with this being a severe deficiency.

Treatment:

Treatment with TPPR was directed bilaterally to two muscles groups being the IMC and adductors. This was accompanied by self-management techniques for the hip flexors and adductors in conjunction with the use of a coolant gel.

Outcome:

After the first treatment, the patient was able to perform a unilateral leg stand on her left lower limb. After the second treatment, she was able to dress herself, and after the third treatment, she walked for four hours around a Shopping Centre. After three treatments and specific self-management strategies the patient was able to return to the employment of her choice, which she had been unable to undertake for a number of years. She recommenced walking for an hour a day and undertook, and still undertakes, all ADLs independently.

Over the next three years, this patient undertook three further treatment sessions to maintain her health status.

1.2.5 Case Study Five:

History:

A 29 year old professional tennis player presented reporting severe pain in his left lumbar region severely restricting his movements. He had been diagnosed with a significant L5-S1 disc bulge (query rupture) on an MRI examination. As a result of this diagnosis, he had been advised by specialists that he would be unable to play on the professional circuit for a period of up to four months.

Presentation and Examination:

This patient appeared fearful and frustrated on arrival and was reluctant to undertake any further intervention. He walked with a left psoatic gait (or refer p.71 for further

description). His presenting posture revealed a severely scoliosed lumbar spine concave to the left, a three centimetre elevation of the left ilium when compared to the right, and a left hip flexion compensatory deformity of 15 degrees. The patient was unable to lie supine, taking the left leg into the crook position. He was able to forward flex to 15 centimetres above his knees although this movement severely exacerbated his lumbar pain. He was experiencing pain in all positions, somewhat alleviated by side lying.

Palpation revealed predominately latent MTrPs along the right IMC and predominately active MTrPs along the left IMC.

Treatment:

TPPR initially focussed on bilaterally on the IMC. Self-management consisted of advice to use a coolant gel with concomitant stretching of the IMC bilaterally.

Outcome:

This patient returned to the practice court two days after commencement of treatment. His professional career continued for another four years.

1.2.6 Case Study Six:

History:

A 41 year old educator in the equestrian field presented reporting a rapid onset the previous year of numbness of his left hand, with associated motor weakness of the intrinsic muscles. He had found restriction of all cervical movements for over six months and this was progressively worsening. Also, over the last six months, similar symptoms had occurred in his right hand.

The patient reported that pain in the lumbar region had commenced prior to the onset of his upper limb symptoms. This had continued resulting in morphine being prescribed. Subsequently, pain had spread to his cervical region. At the six month point, he consulted a neurosurgeon. The patient reported that no physical examination was conducted, but an MRI of his cervical spine was ordered. The report was:

"**Findings:** Alignment of the cervical spine is anatomical. The posterior fossa structures included within the field of view are unremarkable. Cervical cord signal is normal. No paraspinal masses are identified.

At C2-C3, C3-C4, C4-C5 and at C5-C6, there is [sic] no significant disc bulge. The central canal and neural exit foramina are satisfactory.

At C6-C7, there is a mild right posterolateral broad based disc bulge associated with endplate osteophytes mildly indents [sic] the right anterolateral aspect of the cord. The neural exit foramina remain satisfactory.

A mild disc extrusion at C7-T1 descends 5 mm behind the T1 vertebral body. The central canal and neural exit foramina however remain satisfactory.

Conclusion:

1. The cord is mildly indented at C6-C7 by a right posterolateral disc bulge in [sic] associated with endplate osteophytes. There is however no evidence of cord compression or cervical cord signal abnormality.

2. A small disc extrusion at C7-T1 is noted without neural compromise."

The diagnosis given to the patient was that of cervical seven and eight spinal nerve roots compression. The treatment options offered were that either nothing was undertaken, or that a two level anterior cervical decompression with interbody grafting be performed at C6- C7 and C7-T1 levels with an 80 to 85% chance of relieving the left upper limb symptoms. Risk estimates for surgery were a one per thousand chance of quadraparesis, 20% chance of damage to the recurrent laryngeal nerve damage resulting in hoarseness of

voice, and other known risks of surgery such as wound infection. The patient declined surgery.

Examination:

Palpation revealed predominately latent MTrPs over the length of the right IMC and predominately active MTrPs over the length of the left IMC.

Treatment:

A treatment protocol of TPPR was commenced to the IMC bilaterally with the immediate effect of reducing pain in the left upper limb, cervical and lumbar regions. Three days post-treatment the patient reported full return of sensation bilaterally in the upper limbs. This was then maintained with stretching the IMC. He also reported an absence of lumbar pain, and the cessation of morphine intake.

Five further treatment sessions were subsequently undertaken to address secondary MTrPs in the thoracic and cervical spines.

There has been no re-occurrence of any symptoms with the patient continuing to work and undertake all ADLs.

1.3 Discussion

These six case studies are representative of patients from the researcher's practice who presented with a variety of symptoms in both upper and lower limbs and various regions of the spine. On examination, all had active and latent MTrPs within their IMC, either unilaterally or bilaterally. Despite not responding to multiple previous treatments all of the six in the case studies responded to TPPR and stretching of the IMC, enabling their return

to work, and their ADLs. They also reported a number of symptoms in other systems that appeared to improve with treatment of their MTrPs.

The positive response of these participants to TPPR to their IMC, provoked the curiosity of the researcher to learn more about the potential role of the IMC in CLBP and potential links with symptoms in other body systems.

CHAPTER TWO: Introduction to chronic low back pain

2.1 Background to chronic low back pain

"I've got a bad back". This phrase is so commonly uttered that virtually no-one in the Western World has not heard it! Such is the prevalence of low back pain (LBP) and chronic low back pain (CLBP).

Despite CLBP being so common in lay parlance, a universal definition of this debilitating condition has yet to be agreed on. In addition, the inability of scientists to define the cause, or causes, of CLBP has led to continuation of this condition remaining the source of controversy, debate and confusion.

A number of structures have been identified as potential sources of chronic low back pain, including ligaments (Imai et al., 1995), tendons (Rees et al., 2013), intervertebral discs (Ohnmeiss 1997; Edgar, 2007), thoracolumbar fascia (Hoheisel et al., 2011), and skeletal muscles (Travell, 1976; Simons, 2004).

This thesis focuses on the role of skeletal muscles and associated neural structures in CLBP and, in particular, the role of the IMC comprising the psoas major muscle (PM muscle), the iliacus muscle, the iliocapsularis muscle, and the sometimes present psoas minor muscle.

2.2 Definitions of pain

The International Association for the Study of Pain (IASP, 2015) defines pain as: *"An unpleasant sensory and emotional experience associated with actual or potential tissue damage…The inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain relieving treatment."*

Neuropathic mechanisms have also been implicated in myofascial pain by Backonja, (2003), and Ga et al., (2007). Thus, it may be helpful to consider a definition of neuropathic pain. Woolf and Mannion (1999) suggested that neuropathic pain was considered pathological, not provoked by stimuli, but hypothesised to be dependent on sympathetic nervous system activity. Treede et al., (2008) proposed the definition of neuropathic pain be altered to "*pain initiated or caused by a primary lesion or dysfunction in the nervous system"* with *"pain arising as a direct consequence of a lesion or disease affecting the somatosensory system"* (Treede et al., 2008, p.1631). This later definition is supported by the IASP (IASP 2015 p.p. 4-5).

A further related term used in the literature is "chronic widespread pain" defined by the IASP, (2003) as requiring the following be present: symptoms persisting for three months or longer; pain in the left side of the body, pain in the right side of the body, pain above the waist, pain below the waist or axial skeletal pain (cervical, anterior chest, thoracic, or low back). With so many definitions, it is also important to acknowledge the difficulty of a sufferer to describe their pain (Katz and Melzack, 1999).

2.3 Definitions of low back pain and chronic low back pain

Definitions of low back pain (LBP) differed between those applicable to prevalence studies (Dionne et al., 2008), and clinical studies (Delitto et al., 2012) being impairment/functional subtypes. Establishing a consensus definition of CLBP for the purpose of research has also proven to be difficult. Various definitions have been proposed, including: non-neoplastic pain greater than six months duration without objective physical findings (Rosomoff et al., 1989); pain defined by its length lasting more than six months, and resistance to conventional therapies (Baszanger, 1990); pain prolonged beyond the expected time of recovery usually exceeding three months (Durkin, 1998; Rozenberg, 2008); pain lasting longer than seven to twelve weeks (Andersson, 1999); pain lasing longer than three months (Maher et al., 1999; Bogduk, 2004). To date reference has been made to Rozenberg's definition (2008) in 17 articles, with Andersson's 1999 definition being cited in over 1800 articles.

In an attempt to bring consensus to this field, Dionne and colleagues (2008) brought together a panel of 28 experts to review, through a Delphi process, the common elements of LBP definitions in the literature. The outcome of this produced a consensus on a minimal definition of LBP in the form of two questions (p.98):

1. *In the past four weeks, have you had pain in your low back (in the area shown in the diagram)?*
2. *If yes, was this pain bad enough to limit your usual activities or change your daily routine for more than one day?*

Less consensus emerged in the group with regard to the definition of ‘chronic’. Although it was acknowledged that a cut-off point of “over six months’ had been validated, and found useful, in relation to prognosis (Dunn and Croft 2006), the final consensus was to split the ‘acute’ group into less than three months, and between three and six months.

The research for this thesis commenced in 2002. At this time, it was decided to use the most stringent definition found in literature:
CLBP as “*Defined by its length (more than six months) and its resistance to conventional therapies”* (Baszanger, 1990). Because of the acknowledged difficulty of participants describing their pain (Katz and Melzack, 1999), it was decided to additionally require the study participants to have been medically diagnosed with CLBP. As can be seen, this definition is consistent with the later consensus of Dionne et al., (2008).

In contrast to the extensive debate regarding definitions of LBP, no consensus references were found in the literature with regards to definitions of CSP, thoracic pain, or cervical pain.

2.4 Estimates of the incidence of chronic low back pain

As described previously, there have been many definitions used in relation to chronic low back pain. It is therefore not surprising that estimates of prevalence have varied accordingly. Examples have ranged from 0.1 to 40% (Nachemson, 1985; Papageorgiou et al., 1995; Fraser, 1998). LBP was still considered second only to the common cold as a reason for consulting a medical practitioner (American Academy of Orthopaedic Surgeons, 1988; Fraser, 1998; Binder and Nampiaparampil, 2009). More recently, LBP was identified as the commonest cause of disability worldwide (Buchbinder et al., 2013).

It was considered unlikely that General Practitioner consultations for LBP could be used as an accurate measure of chronicity, as Croft et al (1998) reported that 90% of LBP participants who had consulted their General Practitioner had ceased to do so after three months, with only 25% reporting being pain free, and without any disability, one year after the onset of pain. Burton et al., (2004) found, after one year, 49% of LBP participants reported residual disability, 59% reported mild pain, and 78% reported a relapse of symptoms, with 50% of these seeking some form of intervention. Kent and Keating (2005) reported two out of three cases of LBP had not resolved after 12 months with one out of ten of these cases never resolving.

2.5 Financial and psycho-social costs of chronic low back pain

Despite the difficulties in estimating the prevalence of CLBP, these figures below have been used in an attempt to estimate the financial costs of CLBP.

The American Academy of Orthopaedic Surgeons (2009) stated direct care costs for low back pain were USD193.9 billion, with the indirect costs for lost wages (186.7 million lost work days) being an additional USD22.4 billion in the United States of America in 2004. The incidence of CLBP (defined as being of greater than three months duration and limiting activities of daily living) identified as having risen from 3.9% in 1992 to 10.2% in 2006 in the state of North Carolina (Freburger et al., 2009). The cost of spinal conditions rose by 49% in the United States of America between 1996 and 2004 (Canale, 2009). Back pain was reported to have cost The Netherlands 1.7% of its Gross National Product in 1991 (van Tulder et al., 1995), with CLBP, in some industrialised nations, considered to pose a threat to their welfare systems (Nachemson, 1994; Nachemson, 1997). Ehrlich (2003) similarly identified that LBP, 37% ascribed to occupational factors, was a significant factor worldwide in disability and loss of work place hours and burdened compensation schemes.

Chronic pain, including CLBP, was reported to adversely affect the sufferer's quality of life, with this effect not solely confined to the physical realms (Worden, 1983). Losses in the physical or financial realms shattered the fundamental need for security, causing anguish and unease (Mc Ateer, 1989). Ashburn and Staats (1999) described chronic

pain as having a profoundly negative effect on mood, persona, and capacity to socialise. The effects of chronic pain, negatively impacting on the productivity of the sufferer at home and work, was reported to have a 50% incidence of co-morbid depression (Romano and Turner, 1985; Ashburn and Staats, 1999). This experience was reported to be regulated by many factors, including the attitudes of the attending doctor and family members (Turk and Okifuji, 1999). It was also acknowledged that the psychosocial costs of CLBP could not be confined to the sufferer and their family, as they were known to extend to the larger community (American Academy of Orthopaedic Surgeons, 1988).

Thus, despite the difficulties in agreeing on definitions of CLBP, the costs to individuals, workplaces, and society as a result of people suffering CLBP were ascertained to very significant, with the morbidity embracing both physical and psychosocial elements.

2.6 Anatomical structures of the spine relevant to potential causes of chronic low back pain

There is over fifty years of peer-reviewed literature investigating potential anatomical causes of chronic low back pain. Non-specific CLBP has been described as having many sources including: mechanical in origin (Rozenberg, 2008); from intervertebral discs (Ohnmeiss 1997; Edgar, 2007), ligaments (Imai et al., 1995), tendons (Rees et al., 2013), thoracolumbar fascia (Hoheisel et al., 2011), and skeletal muscles (Travell, 1976;

Simons, 2004). Despite this considerable body of work, consensus has not been achieved, and new studies implicating potential mechanisms have continued to emerge. The focus in these studies in this document was on skeletal muscles, with their inextricable links to the above outlined structures via anatomical, and neurological associations.

Intervertebral discs pressures appear to have first been investigated in 1964 (Nachemson and Morris, 1964), when they reported increased intra-discal pressures in the sitting or standing position. Positional differences were also noted to alter the results of straight leg raising (SLR) evaluation (Maitland, 1977). Tightness in the psoas major (PM) muscle was reported to produce heightened compression on the lumbar discs with this phenomenon being considered damaging (Akuthota and Nadler, 2004). Nachemson (1966) also demonstrated that contraction of the PM muscle increased load on the lumbar intervertebral discs.

Nachemson and Morris's 1964 study on the lumbar 4-5 disc, in one subject, was repeated by Wilke et al., in 1999. Similarities in some intradiscal pressures were found in the second study however significant differences between the two studies were also identified. A three-fold pressure increase from supine lying to side-lying measured in the older study (Nachemson, 1966) was not found by the Wilke study (Wilke et al., 1999). The 1999 researchers questioned whether historical advice given by orthopaedic surgeons, to avoid the side-lying position in cases of LBP, was therefore appropriate. Wilke's group also reported that intradiscal pressure measured in relaxed standing

potentially could have been greater than those pressures measured in relaxed sitting. Findings of a study by Kuo et al., (2010) concluded disc pressures increased more noticeably in flexion when compared to extension or rotation. Spiegl et al., (2014) found that a significant distraction force in an extension position did not render a reduction in intradiscal pressures.

It has been postulated by Porterfield and DeRosa (1991) that extension of the lumbar spine could increase loading on the Z joints. This was confirmed later by Ivicsics et al., (2014). Investigation as to the effects of sustained loading on the lumbar spine in the erect posture, ascertained the lumbar Z joints bore approximately 16% of the weight-bearing load with 84% being carried by the lumbar discs (Adams and Hutton, 1980). One of the noted effects of tightness in the IMC is increased compression on the lumbar Z joints, with this being further increased in the presence of reduced disc height, increased abdominal mass caused by obesity and pregnancy, and true leg length discrepancy (Porterfield and DeRosa, 1991).

Whilst disc pressure studies have used fine wire insertion in vivo, a non-invasive and individualised method to measure alteration intradiscal pressures pre and post treatment was recently proposed by Munoz et al., (2012). This method utilised two pre-treatment, and two post-treatment, specific radiographs one of each being with a four kilogram weight on each of the patient's shoulders to measure *"the remaining level of discal elasticity"* (Munoz et al., 2012, p. 280).

Innervations of the lumbar intervertebral discs have also been the subject of longstanding research (Virgin, 1951; Markolf and Morris, 1974; Bogduk, 1997). Historically, innervation of the intervertebral discs, and adjacent ligaments, was thought to be derived from the sympathetic nervous system (Bogduk, 1997). The lateral, and anterior, aspects of the lumbar discs were reported to derive innervations from branches of the grey rami communicantes, with the posterolateral aspect innervated by the sinuvertebral nerves, and other branches of the ventral rami (Bogduk, 1985; Porterfield and DeRosa, 1991). More recently knowledge has been expanded as to intervertebral disc innervations by Edgar (2007) who suggested they may have a visceral like nerve supply.

Investigating afferent pathways of discogenic LBP, 33 participants underwent injections of local anaesthetic into the L2 nerve root with resultant abolition of or significant decrease in pain (Nakamura et al., 1996). The hypothesis was that this result was achieved because the main afferent pathways of pain were via sympathetic afferents from the sinuvertebral nerves (Nakamura et al., 1996). The sinuvertebral nerves were reported to enter the spinal canal immediately inferior to the pedicles with these nerves being composed of an autonomic source from the grey ramus communicantes and a somatic root from the ventral ramus (Bogduk, 1985). The ventral rami have been identified as penetrating the PM muscle, joining other ventral rami to form the lumbosacral plexus (Porterfield and DeRosa, 1991). Porterfield and DeRosa also noted that the direct innervation of the psoas major, psoas minor, and quadratus lumborum muscles was from a portion of the ventral rami. The finding of nerves containing

neuropeptide Y in histological research of joint capsules provided additional evidence that pain perception may involve a pain source related to autonomic and sensory nerve innervations (Ashton et al., 1992).

Animal studies have also contributed much to knowledge to the function of the human lumbar spine. A study on rats established referred pain to the loin and groin may be related to reflex discharges from lower abdominal nerves, via stimulation of the lumbar discs and zygapophysial (Z) joints (interchangeable: zygapophyseal, facet, intervertebral) of the lumbar spine (Takahashi et al., 2000). Investigation of lumbar intervertebral disc innervations in rats found sensory information from the lumbar intervertebral discs is conveyed via the rami communicantes (Suseki et al., 1998). Suseki suggested that if this pattern of distribution were applied to humans, decompression of intervertebral discs would not result in relief of pain ascribed as being discogenic in origin.

The role of disc degeneration and pain remains contested. (Ohnmeiss et al., (1997) found that 58% of participants with disc disruptions, not causing distortion of the outer annular wall, also had lower limb pain. Sata et al., (1999) established that pressures in degenerated discs were significantly lower than in normal discs. Carragee et al., (1999) suggested, in a study of innervation of disc degeneration, that pain recorded on discography studies may not be as significant as frequently presumed. However, significant levels of substance P immunoreactivity have been found in severely degenerated lumbar discs suggesting nociceptive properties (Coppes et al., 1997).

In a study entitled “The Intrinsic Vasculature of the Lumbosacral Spinal Nerve Root” (Parke and Watanabe, 1985) suggested that mechanical stresses in the lower spinal region, degenerative processes, and pain, all resulted from mechanical stresses on L-S spinal nerve roots. The paradox of a neuroischæmic basis for pain, with many symptoms of LBP arising from this, was commented on by Parke and Watanabe (1985). It was noted that alterations of anatomy, blood supply and biomechanics were generally linked to LBP (Butler, 1991).

Straight leg raising has been a test performed routinely in the assessment of LBP by health care practitioners. Earlier studies (Breig, 1960; Breig and Marions, 1963) demonstrated that the spinal cord lengthened, and the lumbosacral nerve roots became taut, on forward flexion of the spine. In performing a SLR, significant elongation was noted to occur in the lumbar sympathetic trunk (Breig, 1978). Maitland (1977) suggested that the SLR test primarily tested the freedom of movement of the lower lumbar and sacral nerve roots, along with associated sheaths in the vertebral canal and intervertebral foramen.

The sympathetic nervous system has been discussed previously as innervating various structures in, and around, the lumbar spine. Ashton et al., (1992) suggested this provided the potential for pain to arise from other structures innervated by the sympathetic trunk, including skeletal muscles. Certain skeletal muscles, notably the IMC having direct contact with the sympathetic trunk (Grays Anatomy, 2008), acted in

a way that could stimulate both discogenic and neurogenic back pain mechanisms and could also be an additional intrinsic locus of pain.

2.7 Treatment approaches for chronic low back pain

Given the lack of consensus on both definitions of CLBP and many anatomical bases for the causations of this condition, it is not surprising that numerous treatment interventions have been undertaken in those experiencing LBP and CLBP. The results of these have been evidence in the literature as having conflicting outcomes.

2.7.1 Invasive treatments for chronic low back pain

Many invasive treatment techniques have been undertaken in attempts to resolve CLBP. These techniques range from a variety of injections, for example lignocaine and saline, to interbody fusion.

When comparing the results of Z joint injections with lignocaine, and saline injections, Ravel et al., (1998) found greater pain relief in lumbar symptoms was obtained with lignocaine when five of the following conditions were present; age greater than 65 years, pain not exacerbated by coughing, pain arising from hyperextension, forward flexion or the movement of roto-extension, and pain relieved by the recumbency.

A randomised controlled trial of CLBP participants examining pain outcomes

concluded that sclerosant injections may not have been effective in reducing symptoms (Dechow et al., 1999). Commenting on the usefulness of selective nerve blocks to determine surgical candidates two of the three authors argued this procedure was extremely useful however the third author was of the belief that no useful role could be found in the selection process using this technique (Slosar et al., 1998).

Many surgical interventions were, and have continued to be, performed for the condition of CLBP, with these procedures including discectomy, laminectomy, artificial disc replacement, intradiscal electrothermal therapy, spinal cord stimulators, and differing spinal fusion techniques. The reported benefits of these procedures vary.

In regard to CLBP, Nachemson (1994) suggested it was potentially ineffective and financially disadvantageous to undertake spinal surgery in participants without a definitive diagnosis. Much spinal surgery, including spinal fusion, has been performed in First World countries on the unproven assumption that disc degeneration was the origin and cause of LBP (Nachemson, 1997). Without substantive evidence as to the cause of LBP, Nachemson asserted that surgical interventions continue to be used without justification nor positive results. Spinal fusion was reported to have become the foremost procedure in the treatment of LBP worldwide, with this procedure again rendering conflicting results (Høy et al., 2013).

Recent statistics on intervertebral disc procedures could not be found, however the National Hospital Morbidity Database (Australia) reported that there were 93,564

hospital admissions in the period 2010 to 2011 being attributed to back issues. Of these 27.7% of the total were attributed to LBP. A Cochrane review of RCTs (26 involved lumbar disc prolapse, and 14 for surgery for lumbar degenerative spondylosis) provided no evidence supporting either decompressive or spinal fusion surgeries in lumbar spondylosis participants delivering a better outcome than simply the passing of time, sham treatment, or conservative treatments that are non-surgical (Gibson et al., 1999).

At two-year follow- up of a clinical trial in which 29 participants had undergone concurrent posterior lumbar interbody fusion, posterolateral fusion, and pedicle screw instrumentation, reported that nine participants achieved an excellent reduction in pain, and eight had a poor outcome (Leuvfén and Nordwall, 2000). Results demonstrated bony fusion occurred in 93% of participants, with *"excellent"* reduction in pain achieved in 31% of the participants, leading the researchers to propose that solid fusion did not equate to a positive reduction in pain (Leuvfén and Nordwall, 2000). A descriptive case review concluded that those who appeared to have displayed posterolateral solid fusions continued to report postoperative pain. The cause was postulated to be pain originating from a disc within the area of fusion (Barrick et al., 2000).

A ten-year follow up study of 103 participants with anterior lumbar interbody fusions established that 34% were categorised as having achieved *"good or excellent"* scores on the Low-Back Outcome Score (Penta and Fraser, 1997). A further 2 year follow up

study of 135 participants with instrumented posterolateral lumbar spinal fusions established solid bony fusion in 82% of participants, but only 19% categorised symptomatically as *"good"*, utilising the Low Back Outcome Score (Greenough et al., 1998). This study also reported that participants who underwent a second procedure did not have a good outcome, with the recommendation that further surgery be avoided (Greenough et al., 1998). Similar findings were reported by Leuvfén and Nordwall (2000).

A cohort study of 27,111 participants demonstrated that those who had undergone spinal fusions suffered a complication rate 1.9 times greater than those who had had spinal surgery without fusion, with the morbidity rate doubling in the fusion group at six weeks post-surgical follow-up (Deyo et al., 1993). A smaller study by Elias et al., (2000) of 67 participants who had undergone a posterior lumbar interbody fusion via the implantation of a threaded interbody cage, reported one death, ten dural lacerations, and ten participants suffering continued back pain at one-year follow-up. Magnetic resonance imaging (MRI), demonstrated ten of these participants suffered radiculopathy, six incurred epidural fibrosis, one arachnoiditis, and one had recurrent disc herniation. One patient suffered a permanent motor deficit with sexual dysfunction, and additional procedures were required in 14 participants with the total number of participants experiencing post procedure complications being 50%.

In a ten year follow-up study of 143 participants who had undergone midline lumbar fusion, and non-fusion procedures (Frymoyer et al., 1978), researchers found that 30%

of the fusion group, and 37.7% of the non-fusion group, were considered long-term failures. Persistent symptoms, or the need for re-operation, after midline fusion suggested this procedure offered little benefit to those suffering lumbar disc diseases (Frymoyer et al., 1978). Despite this evidence, this procedure was reported as still being undertaken in 2013 (Mukai et al., 2013).

Results from lumbar interbody arthrodesis established nerve and vascular injury as being complications that had arisen from this procedure (Tay and Berven, 2002). Four to six percent of participants, who had undergone lumbar spinal fusion, suffered nerve root damage resultant from pedicle screw malplacement (Hall, 1998). Other complications included reports of: ureter damage (Isiklar et al., 1996); quadriplegia (Langmayr et al., 1996); bilateral cortical blindness (Huber and Grob, 1998); disc herniation (Gertzbein and Hollopeter, 2002); cauda equina syndrome (Chen et al., 2001); bilateral stress fractures of the pedicles (Macdessi et al., 2001; Ha and Kim, 2003).

A retrospective cohort analysis of Medicare claims in the United States (Deyo et al., 2010) identified a 15-fold increase, from 2002 to 2007, in people aged 66 or older undergoing complex fusion procedures (defined as more than two discs levels fused, 360˚ fusion via a single incision, or combinations of anterior fusion accompanied by posterior fusion or transverse fusion) of the lumbar spine. Higher associated morbidity and mortality rates were reported in this group when compared to simple fusions or decompression surgery.

Surgical removal of the disc has been reported to carry significant morbidity. A retrospective follow-up of 35,309 participants who had lumbar discectomies established that 14% had undergone further surgery, and 2.3% had undergone two or more re-operations (Osterman et al., 2003).

The implantation of spinal cord stimulators versus spinal re-operation has constituted a further area of debate. In a five-year follow-up of 50 participants with failed back surgery syndrome (averaging 3.1 operations), 47% of participants achieved a successful outcome in those who received an implantation of a spinal cord stimulator (North et al., 1991). In another RCT, spinal cord stimulation was found to be more effective in alleviating residual post-surgery pain than spinal re-operation (North et al., 1994). Similarly, another trial reported 55% of participants at one-year follow-up had their pain successfully managed by spinal cord stimulation (Burchiel et al., 1996).

Artificial disc replacement is another more recent surgical approach being undertaken in CLBP. An RCT of artificial disc replacement versus 360 degree lumbar spinal fusion reported outcomes at three month suggesting artificial disc replacement was an appealing option to lumbar fusion in participants disabled with lumbar discogenic disease and impairment of the mechanics of the lumbar spine (Zigler et al., 2003). However, a review by de Kleuver et al., (2003) identified a high rate of re-operations associated with artificial disc replacement, with the conclusion being disc replacements were considered experimental and should only be used under stringent controls. A

follow-up of 26 participants who had undergone artificial disc replacement (mean period = 91 months, range 15-157 months) reported complications including anterior subluxation of the lumbar segment and abdominal wall haematoma (van Ooij et al., 2003).

Intradiscal electrothermal therapy (IDET) has been another surgical option performed in CLBP with varying outcomes reported in the literature. Freedman et al., (2003) reviewed a case series of 36 active duty soldiers in which a single trial of IDET had been undertaken. The primary outcome measure was pain reduction, by 50% or greater, with a reported success rate of 16% (five of 31 participants). At two-year follow-up, 19 of the 31 soldiers (61%) were still undertaking active duty. In an analysis of 79 participants treated with IDET, 48% of these participants reported greater than 50% pain relief at six-month follow-up (Cohen et al., 2003). However, a previous study of 20 participants concluded that IDET, at six-month follow up, had not effectively reduced pain levels or improved functional performance (Spruit and Jacobs, 2002).

These data reveal a continuing debate over the efficacy of surgical approaches with, at best, a picture revealing some of the adverse outcomes from surgical intervention being identified. At the least, there was a significant issue about appropriate selection of participants for different surgical treatments. Because of the associated risks involved with invasive procedures for CLBP, and the variable evidence of effectiveness, it is important to consider what non-surgical treatments have been available for CLBP

patients.

2.7.2 Non-invasive treatments for chronic low back pain

Non-invasive interventions for CLBP have included a range of exercise protocols, mobilisation and manipulation, traction, pharmacological therapies, and limited approaches involving MTrPs. The role of MTrPs will be the subject of section 2.8 of the thesis (refer p.42).

Reporting on their extensive review of therapeutic exercise regimes in assisting return to work and activities of daily living, van Tulder et al., (2000) concluded that exercise may be of benefit for CLBP participants but not LBP. A meta-analysis of 23 randomised clinical trials (RCTs) evaluating the role of exercise in the management of CLBP concluded that a better response was achieved from exercise for the entire body than from control interventional treatments (Maher et al., 1999). The other interventions included electrotherapy, manipulative therapy, and massage. Results of an evidenced based review established short–term functional improvement in CLBP participants with interventions such as exercise programmes (unspecified), multidisciplinary approaches, back schools, progressive relaxation, and the use of the COX2 inhibitors, but found no evidence of any long-term benefit was established on evaluation of function and pain levels (van Tulder et al., 2006). In this evidenced based review the reviewers noted a significant number of the reviewed trials had methodological weaknesses. Interestingly, in regard to this thesis, none of the studies reviewed involved the IMC.

For 50 years, physiotherapy teaching and practise for LBP treatment has been dominated by the Maitland approach using the techniques of joint mobilisation and manipulation. This protocol was formulated by Geoffrey Maitland, an Adelaide based physiotherapist, who first presented a three month course on spinal manipulation in 1965. These physical techniques, ascribed as passive, were described as having the potential to render temporary relief to the sufferer, but have lacked data supporting long term clinical improvement (Ashburn and Staats, 1999; Gross et al., 2010). Despite initial resistance, manual therapy had been adopted as a mainstream treatment approach in the United States (Dommerholt, 2004).

The efficacy of using manipulation has been controversial, evidenced by a systemic review of 112 randomised controlled trials (RCTs), 12 met the inclusion criteria utilising manipulation, or a combination of mobilisation and manipulation (Ferreira et al., 2002). Results of the review suggested no significant pain reduction was achieved in CLBP participants who received spinal manipulation versus sham treatment, with no reported improvements in disability. When comparing the treatment techniques of spinal manipulation and massage therapy in CLBP participants, similar outcomes were reported. The efficacy of acupuncture remained unclear in the review.

Assendelft et al., (2003) found no evidence for the efficacy of spinal manipulation in those suffering CLBP. A further RCT of osteopathic and sham manipulation reported no identified benefits between the two techniques in subjects suffering intermittent or

constant non-specific low back pain for three or more months (Licciardone et al., 2003). A meta-analysis of 39 randomised clinical trials compared spinal manipulation with seven conventional treatment approaches, concluding spinal manipulation had no greater benefit than the other treatments in acute LBP and CLBP (Assendelft et al., 2003).

A number of authors questioned why manipulation continued to be performed at such rates with no credible research data to substantiate the undertaking of this treatment approach (Broadhurst, 2002; Avery and Driscoll, 2008). A systematic review conducted by Dagenais et al., (2010) identified comparable or greater benefit in function, and pain, with manipulation when compared with commonly used interventions such as medication and exercise. In summarising their systematic review, Vernon and Schneider (2009, p.20) stated *"... there is moderately strong evidence to support the use of some manual therapies (manipulation, ischemic pressure) in providing immediate relief of pain at MTrPs"* but not so in providing longer term relief.

Lumbar traction has continued to be another treatment undertaken for pain diagnosed arising from lumbar disc disorders, with Malanga and Dunn, (2010) finding no evidence to establish the effectiveness of this technique in reducing pain. An update of a previous Cochrane review (1995) was undertaken to re-evaluate the efficacy of traction in acute, subacute, and chronic non-specific LBP, undertaken with or without other treatments Munoz et al., (2012). Reported was this technique rendered slight or no benefit in reducing pain, function, general improvement, and capacity to return to work (Munoz et

al., (2012). The study also noted the adverse effects of traction ranging from increased pain, to the requirement for surgery, in 21% of the reviewed studies.

Conflicting evidence was located on review of the literature regarding the therapeutic efficacy of various pharmacological interventions in alleviation of CLBP. No pharmacological basis for the use of narcotics was identified by Deyo (1996), with the prescription of antidepressants reported to offer symptomatic relief, described as middle range to positive, in participants experiencing CLBP (Staiger et al., 2003). The reasoning postulated, independent of the participants' depression state, was that specific antidepressants, such as the tricyclics and tetracyclics, have been identified as inhibiting reuptake of norepinephrine. The role of antidepressants in enhancing functional status was reported to be unclear in CLBP (Staiger et al., 2003). The function of nonsteroidal anti-inflammatory medications in CLBP was shown to be more effective than placebo and other medications, such as antidepressants, in reducing pain and with this being the only medication group reported to increase function (Bannwarth et al., 2012). The adverse effects, such as the heightened possibility of cardiovascular disease, with longer term use of nonsteroidal anti-inflammatories in CLBP was discussed by Kuritzky and Samraj (2012).

Similar to the data on surgical interventions, the evidence for non-invasive therapies appeared to suggest that more understanding was required as to which therapies may benefit individuals. An interdisciplinary debate entitled *"Efficacy of manipulation in low back pain treatment: The validity of meta-analysis conclusions"* (Chaitow et al.,

2004) highlighted many factors needing to be considered in treating LBP in a clinical setting, as back pain sufferers were not a standardised group.

It would appear justified to suggest that the treatments described in the preceding sections have rendered limited effectiveness and, for an individual, the treatment approach was to be carefully selected taking into consideration of their signs and symptoms. However, despite this relative paucity of effective treatment outcomes, scant attention had been focussed in manual therapy training on an area that has been emerging with a significant evidence base; that is pain and dysfunction arising directly from muscle, and in particular, from MTrPs. The pathophysiology and clinical presentations of MTrPs, has been previously reported as under-represented in manual therapy training (Dommerholt et al., 2006). The concentrated focus of training has been on mobilisation and manipulation of joints, with teaching on pain and dysfunction arising from muscle comprising around 10% to 15% of course content (Dommerholt et al., 2006). This lack of attention in training, regarding this evidence base, may be an impediment to developing effective treatment approaches. The next section reviews the potential role of MTrPs as a target for therapeutic interventions in CLBP.

2.8 Myofascial trigger points

2.8.1 Introduction to myofascial trigger points

Although MTrPs may have been alluded to in the literature for centuries (Shah and Gilliams, 2008), the pioneers of scientific study of MTrPs are acknowledged to be Doctor Janet Travell (1901 to 1997) later joined by Doctor David Simons (1922 to 2010). Travell and Simons strongly advocated the need for appropriate training in the examination, objective identification, and treatment of MTrPs in skeletal muscle, with this opinion continued by many.

Skeletal muscles were defined as muscles under voluntary control with the capacity to elongate or contract (Marieb, 1994). MTrPs have been considered to be identifiable in skeletal muscles, including a palpable taut band or palpable ropiness within the affected muscle causing excruciating pain on palpation, along with the so described *"jump sign and a local twitch response"* (Simons et al, 1999, p.4).

MTrPs have been classified as either being active (the source of pain or other signs and symptoms) (Simons et al., 1999, p.1), or latent (where pain or tenderness is provoked on palpation) (Simons et al., 1999, p.4). MTrPs have been described as being pathological with diverse dysfunctions manifesting in the motor, sensory, and autonomic systems (Dommerholt et al., 2006; Bron and Dommerholt, 2012). Pain and other symptoms, such as tinnitus and restricted range of movement (ROM) in specific muscles, have also

been suggested to arise from MTrPs (Rocha and Sanchez, 2012). The capacity of MTrPs to alter bio-mechanics, and cause muscle stiffness, and weakness has also been suggested by Dommerholt et al., (2006). Progression from localised MTrP activity to involving extensive areas of the body were described as a myofascial pain syndrome (MPS) by Gerwin (2005), the symptoms of which have been detailed as *"sensory, motor and autonomic"* (Lavelle et al., 2007, p.841). Hong (2006) also asserted that MPS was a clinical condition commonly arising from MTrPs.

2.8.2 Hypotheses of mechanisms of causation and aetiology of myofascial trigger points

Various mechanisms have been hypothesised regarding the origin of MTrPs. Histo-pathological findings at, or near, active myofascial trigger point sites have been reported (Shah et al., 2008; Shah and Gilliams, 2008). After further histopathological review, local tissues, the peripheral and autonomic nervous systems, and the central nervous system (CNS) were all suggested to be implicated in myofascial trigger point development and perpetuation (Dommerholt et al., 2006; Shah and Gilliams, 2008).

An earlier hypothesis regarding the causation of MTrPs was that acute loading in areas of skeletal muscle within the contractile region led to chronic stress within the affected muscle (Travell and Simons, 1983). Dommerholt et al., (2006, p.207) further enhanced this hypothesis by outlining *"low-level muscle contractions, uneven intramuscular pressure distribution, direct trauma, unaccustomed eccentric contractions in*

unconditioned muscle, and maximal or submaximal concentric contractions" as feasible mechanisms leading to the development of MTrPs.

Other hypotheses regarding the development and perpetuation of MTrPs have included: the *energy crisis theory* (Simons and Travell, 1981) incorporated into the *integrated trigger point hypothesis*, and the *motor endplate hypothesis* (Simons, 2002; Hong, 2002; Simons and Mense, 2003). A more likely explanation proposed was that abnormal depolarisation occurred at the post junctional membrane (Simons et al., 1999) resulting in the so-described *"energy crisis"*. This was hypothesised to be caused by continuous muscle contraction in proximity to an irregular endplate resulting in disproportionate release of acetylcholine causing sarcomere rigidity, greater metabolic stresses, and impairment of the capillary capacities (Shah and Gilliams, 2008). On this basis, the *energy crisis hypothesis* was incorporated into *the integrated trigger point hypothesis.* Further research has continued to expand and explain *the integrated trigger point hypothesis* (Huguenin, 2004; Gerwin et al., 2004; Ge et al., 2011). Dommerholt and Huijbregts (2011, p.35) stated *"there is no other evidence-based hypothesis that explains the phenomena of MTrPs in as much detail and clarity as the expanded integrated trigger point hypothesis"*.

The *motor endplate* hypothesis was based on recorded spontaneous electrical activity in MTrPs having a contiguous relationship with motor endplates (Hong and Simons, 1998). It was hypothesised that disproportionate amounts of acetylcholine were continuously released into the synaptic cleft of a dysfunctional motor nerve terminal

(Simons et al., 1999). Simons (1998) suggested that the positive results achieved by injection of Phentolamine (a sympathetic nervous system blockade agent) into MTrPs (Hong et al., 1997), offered additional support to the hypothesised involvement of the autonomic nervous system in MTrP pathophysiology. Further support for this hypothesis was provided by the evidence that sympathetic hyperactivity was reported to diminish pain pressure thresholds at MTrP sites accompanied by concomitant increase in the intensities of local and referred pain by Ge at al. (2006). The effective use of botulinum toxin injections as one of the release techniques in MTrPs supported the motor endplate hypothesis (Royal, 2002; Smith et al., 2002; Kuan et al., 2002; De Andres et al., 2003; Anderson, 2004). Despite this, other researchers have reported no benefit from botulinum toxin injections into MTrPs (Gerwin, 2012; Climent et al., 2013). The origin of 'spontaneous electrical activity' was hypothesised to arise from a *"contraction knot"* resulting from endplate dysfunction (Simons et al., 1999, p.111). Subsequently, it has been proposed that 'spontaneous electrical activity' is actually endplate noise (Simons et al., 2002). In endplate areas, close to the location of MTrPs, frequencies of electrical discharges had been recorded 10 to 1000 times higher than in normal endplate areas (Simons et al., 2002). Endplate potentials were reported as more common in MTrPs located in the midfibre regions of skeletal muscles (Simons et al., 2002). Alteration in the activities of acetylcholine and acetylcholinesterase receptors was hypothesised to cause alteration of endplate behaviour (Simons and Mense, 2003).

More recent hypotheses have also been published. The conclusion of a neurophysiological review by Partanen et al., (2009) was that muscle spindles played a

role in myofascial pain, with correlation noted between "*painful muscle spindles"* (p.19), and taut bands associated with active MTrPs. The *central modulation* hypothesis by Hocking (2010) proposed MTrPs do not result from defects at the motor endplate, but were the result of *"centrally maintained α-motor neurone plateau depolarization"* (p.187) with this depolarisation being responsible for the continuation of the localised muscle shortening associated with MTrPs. Hocking's hypothesis (2013,) was based on MTrP formation being the result of *"nociception- induced CNS plasticity"* (). Included in this hypothesis was categorisation of MTrPs into *"antecedent"* (p.187), commonly, but not always, found in the flexor muscle groups, and *"consequent"* (p.187) again commonly, but not always found, in the extensor muscle groups. Hocking further expanded the central modulation hypothesis to include mechanisms that were primordial. The *neurogenic* hypothesis by Srbely et al., (2010) implicated, at the least, partial segmental spinal mechanisms as having a primary role in MTrP formation with these being a secondary, peripheral event. This hypothesis was tested in a study utilising dry needling on the so described *"secondary hyperalgesic locus* [SHL]*"* (p.463), with the report that induced decreased short-term pain perception occurred in other SHL that were segmentally correlated. This study also suggested segmental effects may be the result of central sensitisation, not a peripheral phenomenon. Vulfson et al., (2012) also reported MTrPs as being a secondary peripheral phenomena arising from central sensitisation.

The *Cinderella hypothesis* (Hägg, 1988) suggested that the so called repetitive strain

injury were the result of initial disproportionate recruitment, and de-recruitment, of Type 1 muscle fibres resulting in metabolic stresses, tissue degradation, and pain (as reported by Dommerholt et al., 2006). While not a hypothesis as to a possible causation of MTrPs, the *Cinderella hypothesis* might support a correlation between low level muscle contractions and MTrP formation. A study, utilising electromyography as one of the assessment tool on the upper trapezius muscles of 16 females undertaking high speed typing on a computer, was conducted by Treasters et al., (2006). This study describes the development of MTrPs in the upper trapezius muscle within 30 minutes of typing despite these MTrPs, along with any identified MTrPs in the agonists and antagonists, having been released prior to commencement of typing. These findings offered collaboration to the theory that injury can be sustained by muscles at efforts of low force levels, and validated an association between *"visual and postural work demands"* (Treasters et al., 2006, p.122).

Histopathological studies of MTrPs have demonstrated hypertrophy, degeneration of Type 1 fibres with concomitant Type II fibre atrophy, and pathological changes in the mitochondria (Reitinger et al., 1996). Biopsies of the upper trapezius muscle obtained from 27 participants (nine with fibromyalgia, nine with myofascial pain, and nine controls) ascertained participants with the highest measurements of substance P were from the myofascial pain group when compared with fibromyalgia and normal subjects (DeStefano et al., 2000). Shah and Gilliams (2008) found elevated levels of various biochemical substances involved in nociception and tissue damage in tissue obtained from active MTrPs when compared to tissue from latent and normal sites. The presence

of MTrPs, and their role in the causation of pain in many musculo-skeletal conditions, has been described (Hong and Simons, 1998; De Andrés et al., 2003; Simons, 2004; Chen et al., 2008). Advances in radiological techniques such as sonoelastography and magnetic resonance elastography have enhanced knowledge as to local effects, and identification, of MTrPs (Chen et al., 2009; Sikdar et al., 2009; Ballyns et al., 2011; Dong-wook et al., 2011; Shanker and Reddy, 2012; Ballyns et al., 2012; Thomas and Shankar, 2013). In the study by Chen et al., (2013), a positive correlation was made between taut bands in muscles, previously identified by palpation, and identification of specific abnormalities on magnetic resonance elastography. Utilising ultrasound, substantial tissue irregularities, and morphological alterations, at MTrP sites previously identified by physical examination were reported by Sikdar et al., (2009), with reported correlation between constriction of blood flow and MTrPs pathophysiology.

Moraska et al., (2013) analysed interstitial fluid samples obtained from active MTRP sites in the upper trapezius muscles of two subjects to assess vascular flow and cellular metabolism before, during, and after TPPR. The findings demonstrated enhanced vascular flow, with increased levels of the tested carbohydrate metabolites, throughout the three post-intervention sample testings timed at 20 minute intervals.

The central nervous system has also been implicated in MTrP formation and MPS, with the myofascial trigger point mechanism reported to have an immediate association with spinal cord integration (Wong and Wong, 2012). A study to detect anomalous brain responses to painful stimuli in MPS participants identified heightened activity in the

somatosensory and limbic regions, and pronounced diminishment of activity in the hippocampus (Niddam et al., 2007). Increased activity in the somatosensory cortices, cerebellum, and inferior parietal lobe regions was identified in response to painful stimulation of a neutral site (the thumb) in fibromyalgia and CLBP sufferers, but not healthy controls (Giesecke et al., 2004). Changes in various areas of the central nervous system were noted to be a precursor to the onset of widespread muscle pain (DeSantana and Sluka, 2008). The findings of a study by Hsieh et al., (2007) established that dry needling of an active MTrP substantially increased pressure pain thresholds in MTrPs located within the zone of pain referral, improved ROM, and suggested diminishment of central sensitisation.

Thus there remains a number of alternative hypotheses, if not to the cause, but the mechanisms of causation and aetiology of MTrPs, with continuation of work in these area seemingly so important.

2.8.3 Clinical relevance of myofascial trigger points

The existence, prevalence, and effects of MTrPs within skeletal muscle continues to be strongly debated. Frustratingly, from within Australia, alternate rationalisations have been proposed for development of the signs and symptoms ascribed to MTrPs with the *theory* [emphasis by the researcher as is it their respectful opinion that *hypothesis* would have been considered a more appropriate term] of myofascial pain and MTrPs being dismissed (Quinter and Cohen 1994; Quinter et al., 2014).

In a review entitled "A critical evaluation of the trigger point phenomenon" Quinter et al., (2014) stated *"the theory* [of myofascial pain syndrome being caused by MTrPs] *is flawed both in reasoning and in science"* (p.1). The reviewers rejected the model of MPS, proposing *"sufficient research has been performed to allow TrP theories to be discarded"* (p.5). This so described 'critical evaluation' contained many unsubstantiated statements, and to a large extent was selective in referencing earlier to current evidence into the aetiology, pathogenesis, identification, and treatments of MTrPs. Two alternate hypotheses (the *neuritis model*, and *allodynia*) causing development of MTrPs, and MPS, were proposed by Quinter et al., (2014, p.5) with neither underpinned by *"local pathophysiology"*. Neither of the hypotheses fully explained the clinical presentation and findings in a sufferer of MPS. These reviewers were acknowledged to hold positions of significance in areas potentially important in the dissemination of information to others. Conversely, Hong (2006, p.345) stated *"… the existence and nature of MTrPs have now been widely accepted".*

Various studies have been identified that include the utilisation of TPPR in MTrPs treatment including Hsieh et al., 2004, Hsieh et al., 2006, and Bron et al., 2011. The two studies by Hsieh et al., were conducted on the low back region, while the third study by Bron et al., conducted in chronic shoulder pain groups. Each study reported significant improvements for the groups receiving TPPR.

It should be noted that muscle injuries, including sprains, have long been recognised as a source of pain and disability resulting from extreme stretch or stretch during muscle

activation (Garrett, 1996). However this universal acceptance has not extended to MTrPs. Muscle strain was reported to occur commonly during *"high-intensity eccentric loading"* (Mair et al., 1996, p.137), with eccentric loading having also been suggested in the causation of MTrPs (Dommerholt et al., 2006). Correlation between healed muscle injuries, including strains, and subsequent development of MTrPs, was reported by Melzack et al., (1977).

Previously, MTrPs have been suggested to be the most frequently over-looked, and under diagnosed, source of regional pain by Fricton and Steenks, (1996). This was re-iterated in a publication edited by Dommerholt and Huijbregts (2011, p.25), in which it is stated that MPS had been a frequently overlooked clinical finding. Accurate identification of active MTrPs, then the release of these using appropriate treatment methods, was asserted as being obligatory for amelioration of symptoms by Baldry (2002).

The clinical relevance of MTrPs remains contested, despite the coherent theories for their causation and the evidence from radiological identification. The debate about systems to classify MTrPs and their clinical manifestations is the subject of the next section.

2.8.4 Classifications and clinical manifestations of myofascial trigger points

As of 2011 there had yet to be an internationally accepted standardised definition of MTrPs (Dommerholt and Huijbregts, 2011, p.85). As previously noted, this has been also the case for CLBP. Without a standardised definition there has been variance in the identification of MTrPs. It has been suggested that the ability to diagnose MTrPs was dependent upon considerable clinical practice, informed teachings, and gifted palpatory skills (Simons and Mense, 2003).

Despite this, there has been evidence of emerging consensus in the literature regarding both active and latent MTrPs. This body of evidence has also been enhanced by research as to other signs and symptoms possibly arising from MTrPs.

An active MTrP was described as being a source of pain (Simons et al., 1999; Bron and Dommerholt, 2012). With the presence of MTrPs within skeletal muscle or muscles, findings on examination have been described as a palpable taut band contained within the endplate zone, a hyperirritable spot of tenderness within these palpable taut bands, restricted ROM with heightened consciousness of stretching, and stiffness on arising, or after overuse and immobility (Hong and Simons, 1998; Simons and Mense, 2003). Other described findings included weakness not accompanied by discernible atrophy, localised autonomic dysfunction, pain when the affected muscle was contracted against resistance, and a local twitch response more easily elicited from some muscles than others (Travell and Simons, 1983). Vasoconstriction has been noted to occur over a

myofascial trigger point site, often accompanied by coldness of the overlying cutaneous area (Travell and Simons, 1992), while in contrast the normal response of skeletal muscle contraction was reported to be an elevation of arterial blood pressure (Shepherd and Shepherd, 1989).

Latent MTrPs were defined *"as a focus of hyperirritability in a muscle taut band that is clinically associated with local twitch and tenderness and/or referred pain on manual examination"* (Ge and Arendt-Nielsen, 2011, p.386). Latent MTrPs were reported as being more common with aging (Hong and Simons, 1998; Simons et al., 1999), with latent MTrPs having the potential to become an active myofascial trigger point (Simons et al., 1999; Dommerholt, 2011).

Central sensitisation resultant from mechanical stimulation of latent trigger points was reported by Xu et al (2010), while results from a previous study by Li et al., (2009) confirmed the presence of both nociceptive and non-nociceptive hypersensitivity at latent MTrP sites, when compared with non-MTrP sites, post-injection. A study utilising intramuscular electromyography in latent MTrPs offered evidence of the association between amplified synergistic muscle movements that might, in turn, have induced disjointed synergistic muscle activation (Ge et al., 2014). Correlation between latent MTrPs and the hastened onset of muscle fatigue was evidenced in a study by Ge et al., (2012), while the presence of latent MTrPs had been reported to adversely affect muscle recruitment patterns in scapula muscles (Lucas et al., 2004; Lucas et al., 2010). Enhanced intramuscular electromyography activity of latent MTrPs in antagonist

muscles during agonist muscle shortening was reported by Ibarra et al., (2011), who suggested the effect of impaired reciprocal inhibition may have contributed to significantly altered motor functioning in musculoskeletal pain disorders.

Pain described as arising from MTrPs was that of being *"poorly localized, regional,* [and] *aching"* with varying degrees of disablement (Simons et al., 1999, p.19). There have been a few notable exceptions to this such as the description being a *"superficial sharp or tingling pain"* from MTrPs in the sartorius muscle described by the same authors (Travell and Simons, 1993, p. 226).The pain patterns originating from MTrPs have been reported to not strictly correspond to dermatomes, myotomes, or spinal segmental levels (Travell and Simons, 1983). MTrPs have been demonstrated to have the capacity to cause nerve entrapment, in some instances mimicking other syndromes, with an example being the piriformis muscle impinging on the sciatic nerve, causing sciatica (Travell and Simons, 1992). Therefore, the capability of MTrPs to mimic other musculo-skeletal conditions had led to erroneous diagnosis, with failure to diagnose and treat the causative muscle or muscles (Gerwin, 1991; Wysoki et al., 1997). MTrPs have been suggested to sometimes occur secondary to other medical conditions. Examples of this being kidney stones causing secondary MTrPs in the lumbar area with pain persisting after the stone has been excreted, but abolished by treatment of the MTrPs (Giamberardino et al., 2011). The development of MTrPs in association with dental procedures (Rosted and Jorgensen, 2002) has also been reported.

2.8.5 Treatments of myofascial trigger points

A variety of treatment techniques to facilitate the release of MTrPs have been detailed in the literature. These techniques have included: spray and stretch (Simons et al., 1999); cold application (Simons et al., 1999); stretching (Simons et al., 1999); injections of local anaesthetic (Simons et al., 1999; Ashburn and Staats, 1999); sterile water injections (Byrn et al., 1993); botulinum toxin injections (Royal, 2002; Smith et al., 2002; Kuan et al., 2002; De Andres et al., 2003; Anderson, 2004); dry needling (Hong, 1994; Hong, 2002; Baldry, 2002; Dommerholt and Fernández-de-las-Peñas, 2013); electrotherapy devices (Hong, 2002); acupuncture (Strauss, 1987; Hong, 2002; Chu, 2002; Goddard et al., 2002); massage (Hong, 2002); and trigger point pressure release (TPPR), previously known as ischemic compression or acupressure (Simons et al., 1999).

In facilitating MTrP release, the spray and stretch technique required the affected muscle to be placed on stretch, followed by longitudinal application of a coolant spray from origin to insertion (Simons et al., 1999). Subsequently, the muscle is to be worked isometrically, warmed and active ROM undertaken (Simons et al., 1999). With autonomic nervous system involvement in MTrPs noted, it is hypothesised that the effectiveness of this technique is achieved via an acute drop in skin temperature (Simons et al., 1999). Concomitant short-term anaesthesia was stated to create a blockade of the spinal stretch reflex allowing the affected muscle to be stretched with the application of a coolant (Simons et al., 1999), with an additional demonstrated effect

of cooling being the slowing of nerve conduction velocity (Kimura, 1984).

Deemed paramount was the requirement to stretch any skeletal muscle following myofascial trigger point treatment, including injection (Simons et al. 1999), with the need for a self-managed home stretching programme also considered obligatory (Travell and Simons, 1983). Following deactivation of MTrPs it was suggested the patient be taught suitable stretching exercises, accompanied by rectification of any postural abnormalities that may cause reactivation of MTrPs (Baldry, 2002). Stretches of the IMC and trapezii muscles complexes were noted to stretch nervous system structures (Butler, 1991). It was recommended stretching of muscles, including the PM muscle, was to be achieved by stair climbing, step or low impact aerobics, walking and swimming (Bachrach, 1997). The iliacus and PM muscles, while somewhat stretched in these activities, were also reported to be loaded and shortened (Porterfield and DeRosa, 1991)

Injections into MTrPs (local anaesthetics or saline), were described to be of benefit when the affected muscle could not be stretched, or the muscle is resistant to the spray and stretch technique (Simons et al., 1999). Immediately post- injection or the application of coolant spray, the requirement for moist heat to be applied followed by the treated muscle being put through a full range of movement (Simons et al., 1999). The efficacy of lignocaine injections, when compared with dry needling, in MTrPs was reported by Kamanli et al., (2005), with intramuscular stimulation reported to be more effective than lignocaine injections (Ga et al., 2007). Botulinum toxin injections have

been undertaken, to facilitate release of MTrPs (Royal, 2002). The effects of botulinum toxin in tension-type headache and migraine were established, co-incidentally, when injected into head and cervical muscles for cosmetic purposes (Blumenfield et al., 2002). Effects of botulinum toxin have been reported to be due to a neuromuscular blockade in the target skeletal muscle, achieved by inhibition of acetylcholine release, with resultant flaccid paralysis of the injected muscle (Royal, 2002; Smith et al., 2002; Kuan et al., 2002; De Andres et al., 2003; Anderson, 2004). Additionally, there have been other reported benefits of the use of botulinum toxin (Mense, 2004; Zhou and Wang, 2014). Mense reported pain relief immediately after injection of botulinum into MTrPs, not attributable to the *"hyperactivity of the muscle",* thereby implicating other mechanisms. Such mechanisms were postulated to be the prevention of sympathetically maintained pain being diminished,as the sympathetic system had previously been implicated in MTrPs activity (Ge at al. (2006). Further outlined by Mense (2004) was pain relief may be achieved, almost immediately post-injection of botulinum toxin, via the prevention of neuropeptides being released from nociceptive nerve endings.

While the mechanisms of botulinum toxin injections in releasing MTrPs have been elucidated, the mechanisms involved in dry needling of MTrPs have yet to be established (Dommerholt and Huijbregts, 2011, p.174). A systematic review by Cummings and White (2001) concluded the type of substance injected into MTrPs did not alter outcomes, and suggested that the utilisation of needling techniques rendered significant improvements in MTrPs. The findings of Cummings and White were confirmed by Vulfson et al., (2012), who outlined MTrPs as a secondary peripheral

phenomena arising from central sensitisation. The findings of a study by Hsieh et al., (2007) established that dry needling of an active MTrP substantially increased pressure pain thresholds in MTrPs located within the zone of pain referral.

The technique of ischaemic compression has become commonly referred to as trigger point pressure release (TPPR), despite differences in the application of these techniques. Ischaemic compression was described as pressure applied with sufficient strength to cause blanching of the skin (Travell and Simons, 1983). This technique was later modified to become known as TPPR being described as *'the application of slowly increasing, nonpainful pressure until a barrier of resistance is encountered. Contact is then maintained until the tissue barrier releases, and pressure is increased to reach a new barrierto eliminate the trigger point tension and tenderness"* Simons et al., 1999, p.8). Studies specifying "ischaemic compression", as the treatment technique, have been identified as recently as 2015 (Kaur et al., 2014; Cagnie et al., 2015; Martin-Pintado-Zugasti et al., 2015).

In conclusion, there have been many treatment techniques described to facilitate the release of MTrPs, and no consistent evidence to suggest superiority of one method over another. However, recent studies utilising TPPR appear to have promising results.

2.8.6 Perpetuating factors of myofascial trigger points

If competent provision of the above listed techniques has failed to provide more than

temporary relief, Travell and Simons (1983) suggested considering the possibility that perpetuating factors may be present. Potential perpetuating factors highlighted have included: overload or repeated trauma; nutritional (vitamin or mineral) deficiencies; mechanical stress: structural, postural, sustained pressure and/or constriction of muscles; metabolic and endocrine inadequacies; bacterial, viral, or parasitic infection; prolonged cooling or chilling; strengthening exercise; excessive caffeine intake; excessive alcohol intake; specific pharmacological agents; psychological factors including anxiety or stress; and other undiagnosed medical conditions (Travell and Simons, 1983). As stated by Gerwin (1993, p.87) the most prevalent *"systemic factors that we encounter among persons with MPS are hypothyroidism, folic acid inadequacy and iron insufficiency."* Some of these perpetuating factors, for example nutritional and metabolic deficiencies, have yet to be extensively researched however.

2.9 Conclusion

This chapter has attempted to review the varied efficacy of standard surgical and non-invasive treatments, treating muscular causes for this condition, particularly those associated with MTrPs, which may warrant further investigation. One muscle identified as a potential target for such therapy is the IMC. The next chapter explores the IMC in more depth.

CHAPTER THREE: The iliopsoas muscle complex

3.1 Introduction to the iliopsoas muscle complex

The IMC has been described by Michele (1962; Michele, 1971) as one of the most significant muscles units in the human body, being the muscle complex responsible for the majority of presentations arising from muscle imbalances. However, further review of the composition, and actions, of the muscles that comprise the IMC revealed a lack of consensus (King et al., 1993; Hanson et al., 1999). This chapter will review the anatomical and clinical evidence regarding the questions that have arisen from the previous chapter regarding the IMC.

In this regard the IMC, considered to be a mammalian muscle, was functionally required to lengthen at the hip in land based quadrupeds from approximately 100 degrees hip flexion to 180 degrees for humans in the upright posture, with this then being the attributed cause of its increased susceptibility to overload, and dysfunction, in humans (Michele, 1962; Michele, 1971). The process of evolutionary adaptation to the upright stance may be important in understanding the finding of numerous MTrPs in the posterior extensor muscles of the human trunk as outlined by Hocking (2010).

3.2 Anatomical composition and considerations of the iliopsoas muscle complex

The IMC in humans was historically described as being comprised of three muscles; the

psoas major (PM), psoas minor, and iliacus muscles (Grays Anatomy, 2008). A further muscle in this complex consistently present in humans, variously named iliocapsularis, iliotrochantericus, or iliacus minor, has more recently been reported by Ward et al., (2000). Despite this report by Ward and colleagues, no reference of this muscle was located in Gray's Anatomy (2008).

The PM muscle, described as the most anteriorly positioned of the low back muscles, was reported to composed of an anterior and a posterior mass (Gray's Anatomy, 2008). The anterior mass as outlined (Gray's Anatomy, 2008) were those of slip attachments to the bodies and discs of the twelfth thoracic to fifth lumbar vertebræ, with tendinous arches located between the slip attachments across the lumbar intervertebral bodies. The posterior mass attachments were reported as being to the first to fifth lumbar transverse processes on the anterior surfaces and lower margins (Gray's Anatomy, 2008). Innervation of the PM muscle was described as being derivation from the first, second, and sometimes third lumbar spinal nerve roots (Gray's Anatomy, 2008).

The iliacus muscle was reported to originate from the upper two-thirds of the inner rim of the iliac fossa spanning the region from the anterior superior iliac spine to the anterior inferior iliac spine with minimal fibres from the hip joint capsule, the inner lip of the iliac crest, the anterior aspects of the sacroiliac and iliolumbar ligaments, and the upper, lateral aspects of the sacrum (Gray's Anatomy, 2008). Innervations for the iliacus muscle were stated to be derived from the second and third lumbar spinal nerves, and the femoral nerve (Gray's Anatomy, 2008). Conjointly, the PM and iliacus muscles

were noted to insert on the lesser trochanter of the femur as the IMC, with iliacus also having attachments to inferior and anterior regions adjacent to the lesser trochanter of the femur (Gray's Anatomy, 2008). The PM muscle was noted to be the only muscle connecting the lumbar spine to the lower limb (Grays Anatomy, 1991; Bogduk, 1997; Kimura, 2002).

Psoas minor, although not always present, was reported as originating from the vertebral bodies of the twelfth thoracic and first lumbar vertebræ, and the intervening disc, inserting onto the iliopubic ramus, the pectineal line, and the iliac fascia. Innervation being by the first lumbar spinal nerve (Gray's Anatomy, 2008). From 144 ultrasound scans performed on the PM muscles, the psoas minor muscle could not be identified as a separate structure to the PM muscle by King et al., (1993), while in some (number not specified) dissections performed by Anatómico et al., (2012) the psoas minor could not be macroscopically differentiated from the PM muscle. Absence of psoas minor was identified in 74%, of 60 human foetuses examined by King et al., (1993), while the psoas minor was reported to be present in 59% of 22 foetuses examined by Anatómico et al., (2012). Routine autopsies of 44 males reported the absence of psoas minor in 13% of white subjects, and 91% of black subjects (Hanson et al., 1999). To date there has been no hypothesis found to explain the disparity of these findings.

The presence of iliocapsularis muscle was first proposed by Ward et al., in 2000, at which time it was stated as being universally present (Ward et al., 2000). This muscle

was identified as having a small origin from the inferior margin of the anterior inferior iliac spine with a larger site of origin arising from the anteromedial aspect of the hip joint capsule, inserting onto an area distal to the lesser trochanter of the femur (Ward et al., 2000).

On MRI evaluation, the PM muscle attained its maximum circumference at the level of fourth and fifth lumbar vertebrae, with the right PM muscle noted to be generally larger than the left PM muscle (Dangaria and Naesh, 1998). No explanation was given in regard to this asymmetrical finding (Cronin et al., 2008). Females were noted to have a smaller cross-sectional area of the PM muscle than males with no rationale established as to the significance of this finding (Gatton et al., 1999). In males, the maximal cross-sectional surface measurement of the PM muscle was noted to be attained by 30 years of age, quickly deteriorating by the age of 40 years to about two thirds of its size at 30 years of age, with then being half of this again by the age of 60 (Imamura et al., 1983). Females had a mild size-related decline with diminishment of overall size noted from age 20 to 80 years as reported by Imamura et al., (1983).

The PM muscle was identified as being anterior to the axis of rotation of movement of the Z joints from the twelfth thoracic to fifth lumbar vertebrae, the lumbo-sacral joint, and the sacroiliac and hip joints (Porterfield and DeRosa, 1991). Contraction of the iliacus muscle, in the closed kinetic chain, created anterior torsioning of the ipsilateral ilium and lumbar Z joints, and lumbosacral joint extension, resulting in anterior rotation of the pelvis (Porterfield and DeRosa, 1991). Contraction or shortening, of the PM

muscle caused compression of the lumbar Z joints, further aggravated by increased abdominal mass (pregnancy or obesity), or any other factor that extended the lumbar spine, for example the wearing of high heeled shoes (Porterfield and DeRosa, 1991). This finding was contrary to the report that wearing high heeled shoes reduces lumbar lordosis (Franklin et al., 1995). Elevation of the heel has been suggested to create increase lumbar lordosis (Porterfield and DeRosa, 1991). It was suggested that the PM muscle anterior torsioning the contralateral ilium via the contralateral sacro-iliac joint (Travell and Simons, 1992).

The hip joint capsules, capsular ligament of the hip, iliopectineal bursa, and sub-tendinous iliac bursa, were in direct contact with the IMC, with other skeletal muscles such as the quadratus lumborum sometimes having slip attachments to the iliacus muscle as reported by Gray's Anatomy (2008). The diaphragm was described as having a common attachment at the twelfth thoracic vertebra forming a fascial arch overlying and blending with the PM muscle and fascia (Gray's Anatomy, 2008). The PM muscle was noted to also share a common attachment with the inferior portion of the trapezius muscle on the twelfth thoracic vertebra (Gray's Anatomy, 2008).

Organs stated to be in direct contact with the PM included the kidneys, ascending and descending colons and the urogenital system (Gray's Anatomy, 2008). Vascular structures in contact, or close proximity, included the lumbar arteries, vena cava, aorta, and femoral arteries (Gray's Anatomy, 2008). The vascular supply to the PM muscle was reported as being complex, including contributions from the "*lumbar, iliolumbar,*

obturator, external iliac and common femoral arteries" (Pillet et al., 1989, p.33).

The thoracolumbar sympathetic nerve trunks, and the rami communicantes, were noted to be in direct contact with the PM muscle (Nathan, 1987; Bogduk, 1997; Banagan et al., 2011). The innervations of the lumbar intervertebral discs (Bogduk, 1997), and the lumbar Z joint capsules (Ashton et al., 1992), were derived from the sympathetic component of the autonomic nervous system. Through the development of new techniques more recent research has greatly enhanced knowledge of lumbar disc and joint innervations as reported by Edgar, 2007. As previously noted this newer information renders possible support to a 'visceral pain hypothesis' that is the lumbar intervertebral discs may have a visceral like nerve supply (Edgar, 2007). Adaption, by the autonomic nervous system, to accommodate correct functioning of the [human] body with movements into spinal flexion probably elongate the thoracic and lumbar chains of the sympathetic nervous system (Butler, 1991). Lumbar spinal nerve roots were identified in the PM muscle by Banagan et al., (2011), with the PM muscle also being traversed, or penetrated, by a number of peripheral nerves, including the femoral, obturator, genito-femoral, iliohypogastric, ilioinguinal, and lateral cutaneous nerve of the thigh (Gray's Anatomy, 2008).

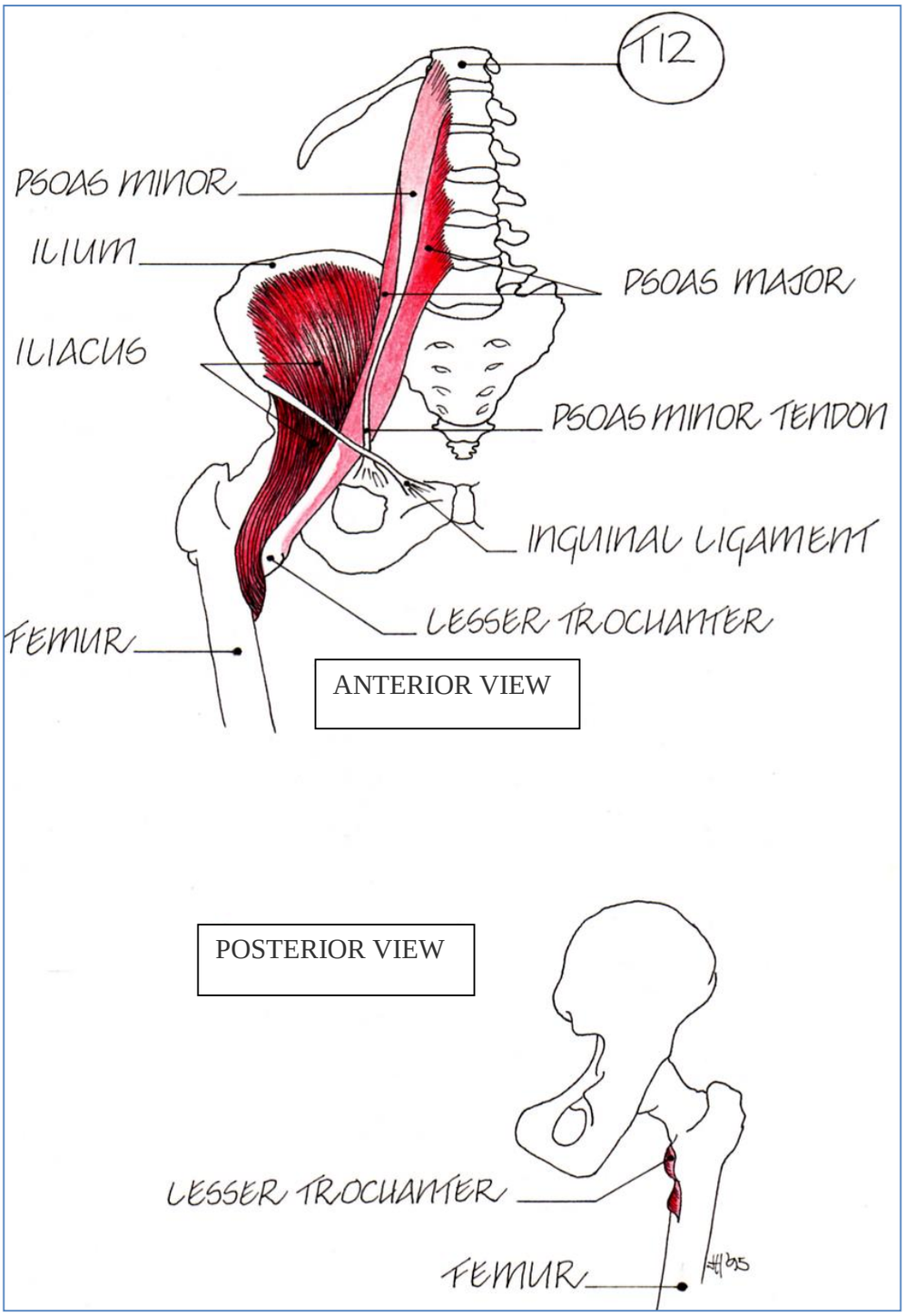


Diagram 3.1 of the iliopsoas muscle complex: anterior and posterior views

(Diagrams sourced from 'Front to Back': Jefferis, 2011, p.p.17-18).

3.3 Actions and functions of the iliopsoas muscle complex

Despite the IMC being in proximity to this vast array of structures, authors have stated that little research has been undertaken into its actions (Takahashi et al., (2006), with some dispute having been raised with regard to its functions (Bogduk, 1997; Penning, 2000). The reason for this disparity may lie in the recognition that the IMC functions vary with differing angles of flexion at the hip joint (Yoshio et al., 2002).

The IMC has been universally accepted as the primary hip flexor in the open kinetic chain (Dykyj, 1988; Grays Anatomy, 1991; Skyrme et al., 1999). More specifically, Yoshio et al., (2002) identified the PM muscle as an effective hip flexor between 45 degrees to 60 degrees hip flexion. Beyond this primary hip flexion role, however, the views of researchers have been more diverse. Most of the research has focused on the PM component of the IMC.

Takahashi et al., (2006) proposed three primary roles of the PM muscle. These were elevation of the upper leg and extension of stride in ambulation; stabilising the pelvis and drawing the lumbar spine laterally to allow ambulation; and to maintain head position. The PM muscle could continuously, and significantly, participate in support of the erect stance in the standing or sitting positions (Yoshio et al., (2002). Significant participation of the PM muscle in maintenance of the up-right posture were also described (Nachemson, 1966; Porterfield and DeRosa, 1991; Hu et al., 2011). Anatomical studies of the PM muscle (Penning, 2000) supported previous findings (Nachensom, 1966; Nachemson, 1968) that it could potentially stabilise the lumbar

spine in the upright posture. This assertion was reinforced by Porterfield and DeRosa, (1991). The PM muscle was suggested to be active in sitting with a straight back (Andersson et al., 1977), while the iliacus muscle stabilised the pelvis during contralateral hip extension when performed in standing (Andersson et al., 1995). The ipsilateral iliacus, rectus femoris, adductor longus, and bilateral PM muscles were noted to create frontal stabilisation of the lumbar spine, with bilateral PM muscle contraction creating forward flexion of the trunk and pelvis (Hu et al., 2011).

Researchers using MRI scans of living subjects and cadaveric specimens concluded that the PM muscle could potentially stabilise the lumbar spine, participate in lateral flexion of the lumbar spine, and create large anterior shear forces at the level of L5-S1 (Santaguida and McGill, 1995). Dependent on the position of the spine, the PM muscle could flex, extend, or stabilise the lumbar spine, and was continuously active in stabilising the lumbar spine in lifting as reported by Sullivan, (1989). More recent research by Park et al., (2014) proposed the central nervous system was involved in differential activation of the PM muscle and the quadratus lumborum muscles in spinal stabilisation, in view of recorded electrographic responses to vigorous upper limb movements. The PM muscle has been reported to act as an erector of the lumbar spine by Yoshio et al., (2002), while Basmajian and Deluca (1985) as quoted by Travell and Simons (1992), noted its participation in spinal extension (when there is a normal lordotic curve present) and participation in spinal flexion (when moving into forward flexion). Using biomechanical analysis, (Bogduk, 1997) described the PM muscle as only having a weak action on lumbar spine flexion and extension. However it was described as a significant lumbar spine flexor by Penning (2000). Participation in lateral

flexion of the lumbar spine renders the PM muscle an agonist to the quadratus lumborum muscle, acknowledged to be the prime lumbar lateral flexor and was deemed to be primarily responsible for 'Failed low back syndrome' (Travell and Simons, 1992), who noted the quadratus lumborum muscle sometimes having slip attachments to the iliacus muscle. Bogbuk and Hadfield (1992, p.119) concluded that the PM muscle was responsible for *"extreme compression loads and shear loads … on the lumbar spine".*

With the insertion of the IMC being noted by some researchers to be on the posteromedial surface of the femur, one of the ascribed actions of PM muscle was participation in lower limb lateral rotation, thereby rendering it an agonist to the piriformis muscle (Grays Anatomy, 1991; Porterfield and DeRosa, 1991). Hu et al., (2011) considered the piriformis muscle to be a lateral rotator of the hip. In investigating the role of PM muscle and iliacus muscles in various positions and activities, both muscles were reported to be co-activated under hip flexor torque (Andersson et al., 1977). Skyrme et al., (1999), reporting on a cadaveric study of traction applied to the PM muscle along its long axis, suggested that there was differing participation of the PM muscle in rotation, with this being hip angle dependent. No rotation occurred in neutral and adduction positions of the hip, however when positioned in abduction, lateral rotation did occur. The ipsilateral iliacus, rectus femoris, adductor longus, and PM muscles were reported to be active in straight leg raising (Hu et al., 2011). Porterfield and DeRosa (1991) noted when the IMC forces were placed inferiorly and posteriorly on the lateral area of the superior ramus, anterior movement of the lesser trochanters results when the lower limb placement is under the trunk. Yoshio et al., (2002) suggested participation of the PM muscle in stabilisation of the femoral

head in the acetabulum, occurred at neutral to 15 degrees of hip flexion. Dysfunction of the PM muscle may manifest as a psoatic gait resulting from the lower limb being held in the relatively shortened position of hip flexion, external rotation, and adduction (Michele, 1960; Magee, 2008). The psoatic gait is evident when the one PM muscle is relatively tighter than the other. The effect of this unilateral tightening becomes apparent on ambulation when the lower limb, on the tighter side, has the capacity to move in to full hip flexion. On moving the contralateral lower limb into hip flexion the tighter side is unable to, or has reduction in its capacity move into hip extension (more moderately the movements of adduction and internal rotation are also restricted). On the side of the shortened psoas, therefore, a normal forward stride can be achieved but when the contralateral lower limb moves into flexion the step has to shortened.

Andersson et al., (1977) suggested that selective activation of the iliacus muscle stabilised the pelvis in the movement of contralateral hip extension during standing. The closed kinetic chain contraction of the iliacus muscle created antero-inferior movement of the ipsilateral ilium, extension of the lumbosacral joints, and superior movement of the first sacral superior articular process (Porterfield and DeRosa, 1991). The authors noted that these movements of the iliacus could compromise of one or both of the sacroiliac joints. Correlation between sacroiliac movement, positioning of the hip joints, and the lumbosacral junction was reported from a study on cadavers by Smidt et al., (1997). The *"iliacus test"* was designed to evaluate the capacity of the *"iliacus complex"* (that is the iliacus muscle, the iliofemoral ligament, and associated fascia) to allow hip joint extension (Eland et al., 2002). This test required stabilisation of the ipsilateral innominate bone over the region of the anterior superior iliac spine in

addition to the protocol of the standard clinical test for hip joint extension, the Thomas test (Eland et al., 2002). Statistical differences were reported between the iliacus and Thomas tests with the iliacus test consistently rendering smaller hip extension angles.

Anterior tilt of the pelvis increased lumbar lordosis, potentially leading to tightness in the IMC (Jorgensson, 1993) and shortening of the PM muscle (Michele, 1960; Michele, 1962; Norris, 1993; Norris, 1995). Prolonged standing increased the likely accentuation of the lumbar lordosis with the resultant requirement of the lumbar Z joints to sustain an increased proportion of body weight (Twomey and Taylor, 1994). Accentuation of the lumbar lordosis has been directly attributed to increased Z joint pressures created by the PM muscle, increased further by axial compression load (Shirazi-Adi and Parnianpour, 1999). The Z joints of the lumbar spine were hypothesised to be a mechanically based source of low back pain (Yang and King, 1984). As previously noted, the PM muscle is a flexor of the lumbar spine (Penning, 2000; Hu et al., 2011), with this action reported to flatten the lordosis, with then the greatest portion of load being borne by the anteromedial area of the Z joints (Norris, 1995).

The PM muscle has also been implicated in the causation of scoliosis (Michele, 1960; Michele, 1962; Michele, 1971; Cohen et al., 1985; Travell and Simons, 1992; Gerwin, 2005; Advić, 2010), with anterior pelvic torsion due to the PM muscle having been reported in the presence of leg length discrepancy (Young et al., 2000). A leg length discrepancy of one centimetre or greater was associated with alterations in the structure of articular cartilage and subchondral bone of the L-S joints in scoliosis (Giles and Taylor, 1984). Pelvic tilt mimicking a leg length discrepancy and causing scoliosis has

been reported to occur in the presence of MTrPs in the PM muscle or quadratus lumborum muscles (Gerwin, 2005).

Travell and Simons (1992) suggested the psoas minor muscle assists the PM muscle in increasing normal lordotic angulation, resulting in ipsilateral elevation of the anterior aspect of the pelvis. More recently however, the psoas minor muscle has been implicated in contributing to posterior pelvic tilting, possibly the cause of flexion of the lower lumbar spine relative to the sacrum (Neumann and Garceau, 2014). It has also been suggested the psoas minor muscle may be involved in regulating both the mechanical stability and location of the PM muscle over the hip joint, with possibility that these actions may have an association with both IMC tendon and anterior hip pathologies (Neumann and Garceau, 2014).

Various hypotheses have been proposed regarding the function of the iliocapsularis muscle. Ward et al., (2000), who first described this muscle, hypothesised its action may be to tighten the anterior aspect of the hip joint capsule. Based on MRI findings, Babst et al., (2011) hypothesised that it may act as a stabiliser of dysplastic hip joints. Domb et al., (2011) implicated this muscle in labral tears due to tendon hypertrophy.

Given the wide variety of observed actions of the IMC over a number of joints, it is unsurprising that Lee and Wong (2002) recommended that any evaluation of the lumbar spine also required obligatory kinematic evaluation of the hip joints. This brief review has demonstrated the impact that the IMC has on the biomechanics of the hip joint, the pelvis and the lumbar spine, including posteriorly situated structures such as the Z

joints. This supported the hypothesis of Porterfield and DeRosa (1991) that pain arising from these posterior lumbar structures may be directly related to the IMC altering the local biomechanics.

3.4 The potential participation of the iliopsoas muscle complex on intradiscal pressures and disc pathology

Contraction of the PM muscle has been demonstrated experimentally to increase loading on the lumbar intervertebral discs (Nachemson and Morris, 1964; Nachemson, 1966). Bogduk (1977) agreed that contraction of the PM muscle may cause excessive pressures on the low lumbar discs, while the impact of the PM on intradiscal pressures and strains of discal fibres was confirmed by Shirazi-Adi and Parnianpour (1999).

Intradiscal, and intra-abdominal, pressures were reported as higher in loaded trunk rotation than in lateral flexion (Andersson et al., 1977). Intradiscal pressures and spine positioning were suggested as a direct result of PM muscle contraction by Nachemson (1968) and Michele (1971). The PM muscle has been identified as a cause of lumbar disc herniations, and consequently a cause of sciatica by Michele (1971), and Dangaria and Naesh (1998). In commenting on discogenic back pain, Ingber (1989) noted that the positions that rendered the greatest comfort to a patient with LBP was side-lying in the foetal position and crook lying with each placing a relatively lesser strain on a tight IMC.

3.5 The potential role of the iliopsoas muscle complex in chronic low back pain

Despite its anatomical positioning and reported impact on known sources of chronic low back pain, the IMC has remained largely ignored as a treatable cause of acute LBP, CLBP, and other musculoskeletal conditions (Michele, 1971; Inger, 1989; Travell and

Simons, 1992). Two case series suggested further research in this area may be beneficial.

Ingber (1989) investigated the effect of dry needling of the PM muscle in six *'failed back syndrome'* participants who then followed the dry needling with self-administered post isometric exercise of the IMC. Of the six participants, five also had lower limb pain, with all six having previously undergone radiological investigations and conservative treatments or surgical interventions without reduction in pain or improvement in function. Ingber notes a correlation between the presence of palpable tenderness and weakness of the IMC with both diminished hip extension and LBP in all six participants. Although deficits in spinal extension had previously been considered a noteworthy diagnostic finding in LBP participants, Ingber reported that loss of hip extension had not been previously identified on examination in this patient group. Dry needling of the PM in the six participants in this study significantly increased spinal and hip extension ranges, reduced pain levels, and facilitated resumption of activities of daily living (ADL).

Marrè-Brunenghi et al., (2008) studied four participants (age range 6 – 21 years) with cerebral palsy who all suffered severe LBP resistant to all previous treatment interventions. This manoeuvre was undertaken to ascertain the role of the PM muscle in potential causation of LBP, each patient required to perform an isometric contraction of the hip while positioned at 90 degrees that resulted in LBP, hypothesised support the PM muscle as the source of the pain. In the first of the four participants botulinum toxin was injected into the PM muscle with abolition of LBP. In the second patient LBP was

obliterated by bilateral surgical release of the PM muscle at the level of the pelvic rim. Following this, the PM muscles of the two further participants were injected with botulinum toxin via an ultrasound guided transabdominal approach, with the intestines having been previously moved prior to injection. In both cases there was initial abolition of pain and reduction in hip flexion deformity. When both participants experienced a re-occurrence of pain post-injection, surgical release of the PM muscles was performed with reported abolition of pain up to their 12-month follow-up.

3.6 Potential myofascial trigger point sites and pain patterns in the iliopsoas muscle complex

Travell and Simons (1992) suggested the location of three principle MTrPs in relation to the IMC in patients positioned in supine lying. The reported locations of these MTrPs, and their associated pain referral patterns were:

- Abdominal: indirect palpation of the PM muscle with a posterior directed palpation on the lateral border of the rectus abdominis, then directing palpation slightly medially, usually at the level of the umbilicus. Pain referral is primarily to the low back in a vertical pattern however this can extend as high as the inferior border of the scapula
- Pelvic: palpating the iliacus muscle inside the iliac crest. Pain referral is primarily to the low back in a vertical pattern with spill-over to the sacro-iliac joint.
- Proximal medial thigh: palpation near the insertion of the IMC on the lesser trochanter of the femur. Pain referral is primarily to the groin and the anteromedial region of the thigh with spill-over pain that can extend as far as the knee.

It should be noted that some colleagues have described difficulty in palpating the iliacus muscle as described by Travell and Simons above. In addition, and more recently, it has been recognised that MTrP sites have the potential to be located with considerable variation within the endplate zones of the IMC (Barbero et al., 2013). Supporting this, the author of this thesis has determined two further common MTrPs related to the IMC in her clinical practice. The region of the IMC motor endplate, as identified by Van Campenhout et al., (2010) correlated closely with the two abdominal sites.
These were:

- A further abdominal MTrP located seven centimetres inferiorly and two centimetres medially below the above described abdominal site. Pain referral is primarily localised to the abdomen in an area of approximately 10 centimetres, and/or rarely referring to the low back in a vertical pattern
- At the musculotendinous junction of the IMC located by pressing against the lateral wall of the femoral triangle. Pain referral being primarily to the low back in a vertical pattern, and/or the antero-medial region of the thigh.

3.7 Stretching protocols for the iliopsoas muscle complex

The use of post-isometric relaxation in treatment, or self-management, regimes for MTrPs was considered to be significant in the reduction of pain (Lewit and Simons, 1984). Various references have been identified in the critical literature review outlining the reasons as to why the hip flexors and lumbar spine should be stretched into extension (McKenzie, 1981; Travell and Simons, 1992; Broadhurst, 1998). One stretching technique recommends the patient hang their lower limb off the end of the

bed with the other limb pulled into flexion, in order to maintain the lumbar spine on the bed (Travell and Simons, 1992). This maintains a neutral position of the spine while also stretching other hip flexors (Travell and Simons, 1992). To stretch the IMC in kneeling, the patient is required to extend their lumbar spine, inhale and then forward flex to stretch the hip on the contralateral side (Broadhurst, 1998). Previously identified is that a tight PM muscle or IMC moved the lumbar spine into extension with the instruction not to arch the back as this movement increased pressure on the lumbar discs and Z joints (Porterfield and DeRosa, 1991). In a randomised controlled trial of chronic musculoskeletal pain participants utilising self-administered stretching, reported no improvement gained in muscle length, however stretch tolerance was improved (Law, 2009). As reported by Knudson (2006) range of movement was increased as an acute effect of stretching but was accompanied by a substantial diminishment of muscle functioning, while dynamic stretching is reported to decrease both concentric and eccentric strength (Costa et al., 2013). As has been evidenced with other treatment interventions, differing types of stretching appear to render benefit to specific target populations (Costa et al., 2013, p, 2012).

3.8 Summary

This chapter has described the IMC as a muscle complex that links the lower thoracic vertebrae and the lumbar spine with the pelvis and leg. Its actions have been demonstrated to impact on many of the structures recognised as sites of CLBP. A small number of case study series have reported clinical benefit from specific interventions

directed at the IMC, and a number of reproducible MTrPs have been identified in relation to the IMC with predictable patterns of referred pain.

At the time that this research was undertaken, this represented the evidence base available to the author to help understand why participants with CLBP in her practice were responding to treatment directed to the release of MTrPs. It was not a strong evidence base. In particular, there were no studies with the level of evidence of a randomised controlled trial.

To address this apparent evidence deficit, the author proceeded to design and undertake a randomised controlled trial of MTrP release in participants with CLBP. This study is presented in Chapter Five. Due to the author undertaking this research part time with a number of unforeseen interruptions, the author also decided to undertake a further systematic review of the literature after the RCT was completed to ensure that the evidence resulting from this study remained contemporaneous. This systematic review is presented in the next chapter.

CHAPTER FOUR: Systematic review of the literature on treatment of myofascial trigger points in people with chronic low back pain.

4.1 Search method

After reviewing two relevant, but unindexed, journals (the Journals of Musculoskeletal Medicine, and Musculoskeletal Pain), a computerised search using the Ovid MEDLINE database was undertaken. After only two relevant articles were found (refer p.83), a further computerised search of both MEDLINE and CINAHL was undertaken, a year later, with expanded search terms. No further articles were found to meet the search criteria.

4.2 Inclusion criteria

The inclusion criteria used in the first review were: articles published between 1946 and May 2012; myofascial trigger point; acupressure; TPPR; spray and stretch; experiencing pain; and activities of daily living function. The exclusion criteria were studies conducted on animals and studies published in a language other than English. This search initially yielded 14 articles (see Table 4.1).

The second search was expanded to include the additional criteria: experiencing back pain; LBP, or CLBP; psoas muscles or iliopsoas muscles; myofascial release; myofascial trigger point injections; pelvic asymmetry; scoliosis; spinal curvature; lumbar deviation; mobility; mobility limitation; and muscle length. The exclusion

criteria were again studies conducted on animals, and studies published in a language other than English. This search resulted in an initial yield of 45 articles (Table 4.2).

Table 4.1 Search strategy

Database: Ovid MEDLINE(R) <1946 to May Week 1 2012>

1	chronic low back pain.mp.	2857
2	myofascial trigger point?.mp.	286
3	1 or 2	3141
4	trigger point therapy.mp.	32
5	exp Acupressure/	391
6	TPPRmp.	2
7	trigger point injection.mp.	58
8	spray and stretch.mp. [mp=title, abstract, original title, name of substance word, subject heading word, protocol supplementary concept, rare disease supplementary concept, unique identifier]	34
9	4 or 5 or 6 or 7 or 8	511
10	exp Pain/	275041
11	function.mp.	1269452
12	exp "Activities of Daily Living"/	45156
13	10 or 11 or 12	1562326
14	3 and 9 and 13	20
15	limit 14 to (abstracts and English language and humans)	17

Search for: limit 14 to (abstracts and English language and humans)

Results: 10 (excluding doubles)

4.3 Results of systematic search one

The first computerised search yielded a total of ten papers however, after scrutiny, only one study matched all inclusion criteria (Hsieh et al., 2006). A second paper was included in the final tally, as 67 percent of participants had a history of CLBP (Hsieh et al., 2004). The remaining eight articles were excluded. Two were reviews, one on the use of onabotulinum toxin A, but was not specific as to the sites injected (Ho and Tan, 2007), and the other was a generalised overview of MTrPs (Majlesi and Unalan, 2010). From the remaining six experimental studies, one used ultrasound-guided trigger point injections in the cervicothoracic musculature and not manual treatment (Botwin et al. 2008), one did not pertain directly to MTrP treatment (Miyakoshi et al. 2007), one was yet to be undertaken and was to include sub-acute LBP patients (Buselli et al. 2011), one was a case study regarding an intervention to an unidentified site in the trapezius muscle (Montanez-Aguilera et al. 2010), and one was an RCT on MTrPs located in the upper trapezii muscles (Unalan et al. 2011). There was one duplication (Hsieh et al., 2006).

Table 4.2 Search Strategy: MEDLINE (Ovid) and CINAHL (EBSCO).

Step	Keywords	Medline	CINAHL
1	exp Back Pain/ or exp Low Back Pain/ or chronic low back pain.mp.	28,259	14,710
2	chronic spinal pain.mp.	90	135
3	Psoas Muscles/ or iliopsoas.mp.	1,887	133
4	1 or 2 or 3	30,134	7,948
5	TPPRmp.	2	5
6	Trigger point therapy.mp.	38	56
7	Myofascial release.mp.	66	87
8	Myofascial trigger point treatment.mp.	0	12
9	Trigger point injection.mp.	60	19
10	Spray and stretch.mp.	36	14
11	exp Acupressure/	446	247
12	5 or 6 or 7 or 9 or 10	194	424
13	exp Pain/	297,143	60,930
14	Pelvic asymmetry.mp.	34	23
15	exp Scoliosis/ or exp Spinal Curvatures/ or pelvic distortion.mp.	17,829	1,633
16	lumbar deviation.mp.	0	6
17	muscle length.mp.	1,734	330
18	mobility.mp. or Mobility Limitation/	10,2854	8,325
19	function.mp.	1,409,478	67,451
20	13 or 14 or 15 or 16 or 17 or 18 or 19	1,792,289	126,850
21	4 and 12 and 20	22	68
22	21 (limit to abstracts and english language)	18	31
	Total (excluding doubles)	**45**	

4.4 Results of systematic search two

The two papers included in the final review were reports of RCTs of acupressure (also known as TPPR) versus physical therapies in CLBP and LBP conducted by Hsieh et al., (2004) and Hsieh et al., (2006). The duration of each trial was four weeks with follow-up at six months. The 2004 study involved 146 participants with an age range of 16 to 84 years of age. Of these, 67 percent presented with CLBP. There was no rigid adherence to randomisation of the patients in the study, as four patients received both acupressure and physical therapies. There was transfer of participants between groups with no explanation given by the authors. One patient was transferred from the acupressure to physical therapy group, and four patients were transferred from physical therapy to the acupressure group. Five different physical therapies were undertaken but these therapies were not detailed. A total of 129 CLBP patients participated in the 2006 study that aimed to evaluate acupressure versus unspecified physical therapy. Patients in this study ranged in age between 18 and 81 years. After randomisation, two patients in the acupressure group and two patients from the physical therapy group refused to participate in the randomised treatment, and were switched to the other group. A total of 20 participants (15.5 percent) were lost to follow up during the study but their data was included in the final analysis. Both studies concluded that acupressure (also known as TPPR) produced statistically significant improvements according to the Chinese Short Form Pain Questionnaire (2004 study), and the Roland and Morris Disability Questionnaire (2006 study).

Table 4.3

Studies investigating myofascial trigger points in chronic low back pain and low back pain

First author + **Design**	**Treatment/ Intervention**	**Sample**	**Sample size** + **Nos randomised**	**Baseline** + **Follow up measures**	**Re-assessments**	**Results**
Hsieh, 2004 RCT	Acupressure or physical therapies	CLBP (67%) LBP (33%)	N = 146 Randomised to: N = 69 Acupressure N = 77 Physical therapy	Chinese SFPQ	4 weeks + 6 months	Significantly lower post treatment pain scores in the acupressure group 2.28; the physical therapies group 5.05 (p = 0.0002). At 6 month follow up a significant difference was again detected: acupressure 1.08; physical therapies 3.15 (p = 0.0004).
Hsieh, 2006 RCT	Acupressure or physical therapies	CLBP (100%)	N = 129 Randomised to: N = 64 Acupressure N = 65 Physical therapy	Roland and Morris disability questionnaire	4 weeks + 6 months	Mean differences after treatment = 5.4 in the acupressure group and 9.2 in the physical group (*p* = 0.000); at 6 month follow up = 2.2 and 6.7 respectively (*p* = 0.000)

Table 4.4

Criteria for methodological assessment of myofascial trigger point studies

Criteria	Description	Points deducted for non-inclusion
BAS 1[1]	Eligibility criteria specified	1
BAS 2	Random allocation to groups	1
BAS 3	Allocation concealment	1
BAS 4	Similarity of groups at baseline regarding most important prognostic indicators	1
VAL 1[2]	Blinding of participants	1
VAL 2	Blinding of treating therapists	1
VAL 3	Blinding of assessors	1
STAT 1[3]	Measure of at least one key outcome was greater than 85 percent	1
STAT 2	Outcome measures that both treatment and control groups were treated as allocated	1
STAT 3	Statistical comparisons between-groups reported one key outcome	1
STAT 4	Point measures and measures of variability for one key outcome	1

BAS[1]
VAL[2]
STAT[3]

Table 4.5

Methodologic assessment details of the studies examined using trigger point pressure release in chronic low back pain and low back pain

First author Information	Insufficient information	Potential bias	Additional comments	Insufficient (n)	Potential bias (n)	Quality rating (n)
Hsieh 2004	the specific muscles treated were not identified	VAL[2] 3, STAT[3] 2	5 different physical therapies were undertaken	1	2	5
			Physical therapies were not randomised			
			The consistency of practitioner /patient was not identified		1	
			4 participants received both acupressure and physical therapies, 1 switched from acupressure to physical therapies, 4 participants switched from physical therapies to acupressure		1	
Hsieh 2006	the specific muscles treated were not identified	VAS[2] 3, STAT[3] 2	6 different physical therapies were undertaken, but were not randomised	1	2	7

.

4.5 Summary

The first search identified one article utilising acupressure [TPPR] as one of the interventions in a study of CLBP (Hsieh et al., 2006). A second article has been included in which participants suffered both LBP and CLBP (Hsieh et al., 2004). The second search identified 45 articles, all of which were excluded as not fulfilling the search criteria.

While both studies identified reported acupressure [TPPR] as having rendered statistically significant improvements in CLBP and LBP on outcome measures, there were significant methodological flaws in the studies, including it was not specified which muscle, or muscles, were treated by TPPR. Thus, these reviews had failed to find any further contemporary robust evidence to elucidate the potential role of treating MTrPs in the IMC for participants with CLBP.

The following chapter details original research, conducted by the author from 2003, to address the identified and persisting evidence deficit.

CHAPTER FIVE: The participation of the iliopsoas muscle complex in chronic low back pain

5.1 Introduction

As this thesis has demonstrated, there has been support in the literature for a potential role for trigger point pressure release (TPPR) of MTrPs in the IMC as a treatment for chronic low back pain (CLBP). A relative paucity of high quality studies in this field was evidenced, however.

The primary objective of the study reported in this chapter was to explore the hypothesis that TPPR treatment to the IMC may provide effective relief of symptoms for participants with CLBP in a single-blind randomised controlled study.

5.2 Study methods

5.2.1 Ethical requirements and undertakings

Ethics approval was obtained (Appendix One, p.217 and Appendix Two, p.227) from the Flinders Medical Centre Clinical Research Ethics Committee prior to the undertaking of this study.

In accordance with the ethics contained in The Physiotherapists Act (1991), and the NH&MRC National Statement on Ethical Conduct in Research Involving Humans, all appropriate and professional procedures were followed with clinical responsibilities overriding research responsibilities. In keeping with sound

scientific principles, the researcher undertook that all information acquired during this study would be: kept strictly confidential; used only in accordance with the stated objectives; stored on a secure data base; and, that hard copy would be secured in a locked filing cabinet in the Rehabilitation and Ageing Studies Unit, Flinders University for a total of fifteen years. With a change of supervisor for the study, the agreed storage site became the Department of Surgery, Flinders Medical Centre, South Australia. Participants were advised of their right to withdraw, at any time, without prejudice to their ongoing care and, or, treatment. Because of the inclusion criteria, it had been anticipated that the participants would continue any medically prescribed medication, and, or, treatment during the study.

All assessment, treatment, and re-assessment techniques performed were non-invasive. Palpation of the abdominal area was acknowledged as a potential source of discomfort during assessment, treatment or reassessment.

5.2.2 Sample Size

The sample size of 106 participants required in this study was calculated according to the criteria of Gridley and van den Dolder (2001) for two independent samples being compared. Sample size calculations were based on an estimated mean difference of 1.80 and a standard deviation of 3.25 with the test of equality of means to be carried out at the significance level of 0.05. A sample size of 53 in each group then rendered a beta of 0.194. This means a greater than 80% probability that a true difference between the intervention and stretching groups would be detected in this study.

A total of 63 participants with CLBP (34 female and 29 males) were recruited with 51 (29 females and 22 males) completing to follow up. Recruitment of participants was terminated at 63 participants as the analysis of the data, at that point, demonstrated inferior outcomes for participants in the stretching group (see below p.p. 84-110). At the cessation of the study the final number of participants who completed the program was 51; 27 in the intervention group and 24 in the stretching group.

5.2.3 Criteria

Inclusion criteria were:

- chronic low back pain, medically diagnosed;
- pain being of six months, or greater, duration;
- failure to respond to conventional therapy or therapies; and
- a participant age range of 18 to 65 years (Appendix Three p.228).

Note that some participants with CLBP also had symptoms in the thoracic and cervical regions. No discrimination occurred for such participants.

Exclusion criteria were:

- osteoporosis or those taking corticosteroids (known to affect bone density);
- a medical condition in which bone integrity may be compromised by pressure (including spinal fusion);
- diagnosis of aortic or vena cava conditions; and
- pregnancy (Appendix Three, p.228).

Participants could withdraw from the study at any stage without prejudice to their ongoing care and treatment (Appendix Three, p.228).

5.2.4 Recruitment

Participants were recruited through an advertisement and editorial in a local newspaper, a mailed letter of request to private physiotherapists (Appendix Four, p.229), and by word of mouth between participants.

All but three participants resided in the Adelaide metropolitan area. Too great a distance to travel was cited in two instances of refusal to participate (Consort flow diagram 5.1, p.87). A total of 95 inquiries were received in regard to the study, with various reasons given as to inability to participate (Consort flow diagram 5.1, p.87). Of 95 inquiries, the most common question asked by 31 people was the likelihood of potential aggravation, or re-aggravation, of their spinal problems by their undertaking the required examination, and intervention, protocols.

After eligibility had been ascertained by phone contact, each participant was mailed a Participation Information sheet (Appendix Five, p.230) outlining the requirements of the study, the inclusion, exclusion and withdrawal criteria (Appendix Three, p.228), and the Participants Information form (Appendix Five, p.230). Eligible participants were then invited to attend an appointment for baseline assessment.

The protocols for the two groups were as follows.

5.2.5 Initial baseline assessment and randomisation into intervention and stretching groups

Any further questions were answered at the baseline assessment appointment by the researcher prior to the consent form (Appendix Six, p.232) being signed by the participant and witnessed by an independent person over the age of 18. This was then checked, signed, and dated by the researcher. Reasons for non-participation were ascertained wherever possible.

When signed consent had been obtained, the participant was asked to complete a Patient History Questionnaire (Appendix Seven, p.233) that included date of birth, gender, duration of pain, whether the onset of pain was sudden or gradual, had there been any spread of pain and if so where, details of pain, hand and leg dominance, and previous and current treatments undertaken. The Short-Form McGill Pain Questionnaire (Melzack, 1987) comprising three parts was then completed being the 15 pain descriptors, the Visual Analogue Scale, and the Present Pain Index (Appendix Eight, p.234). The Patient Specific Disability Measure (Stratford et al., 1995) was also utilised. This required the participant to identify five to seven activities they had difficulty in performing, or were unable to perform, due to their CLBP at the time of initial assessment (Appendix Nine), or refer p. 235). The identified activity required the participant to grade their level of disability related to this activity on an 11-point scale (0 being unable to perform the activity to 10 being able to perform at the level prior to the onset of their problems).

Physical examination included height and weight measurements, and palpation examination for five potential IMC trigger point sites. To ensure consistency, and

reproducibility, the study protocol restricted the assessment to five sites for the location of MTrPs on each side. These were:

1. Abdominal: MTrP located by indirect palpation of the PM muscle with a posterior directed palpation on the lateral border of the rectus abdominis, then directing palpation slightly medially, usually at the level of the umbilicus.
2. Abdominal: MTrP located seven centimetres inferiorly and two centimetres medially below the above described abdominal site.
3. Pelvic: MTrP located by palpating the iliacus muscle inside the iliac crest.
4. Groin: MTrP at the musculotendinous junction of the IMC located by pressing against the lateral wall of the femoral triangle.
5. Proximal medial thigh: palpation near the insertion of the IMC on the lesser trochanter of the femur.

The five MTrPs were palpated on the right and left side, numbered 1 to 5 (superior to inferior), and graded as absent, or latent according to the criteria of Ge and Arendt-Nielsen, 2011, or active according to the criteria of Simons et al., 1999.

A standing stretch of the IMC was then demonstrated by the researcher, with this stretch then performed by the participant with the following instructions being given:

"Looking straight ahead, not down, one leg 20 centimetres behind the other as in a normal standing position, tightening the bottom muscles and sucking in the tummy, stretching the same arm to the ceiling, on the same side as the leg is back, hold for five seconds, relax. Do ten repetitions on each side."

A diary, which included a photograph demonstrating the position for the stretch to be undertaken and instructions, was supplied to record compliance (Appendix Ten) (or refer p.236).

Attendance was also required for outcome assessment, and participants were informed that this would be conducted by an independent 'blind' assessor (Appendix Eleven, p.237).

Randomisation was performed by way of telephone contact with the researcher's student co-ordinator, who was located within the Department of Rehabilitation and Ageing Studies Unit, Repatriation General Hospital, South Australia. There was no input from the researcher in the randomisation process, performed via a computer-generated process, with the outcomes sealed in opaque envelopes. Subsequently, these envelopes were handed to the researcher's student co-ordinator for opening and allocation of participants to the intervention or stretching group.

Of the 63 participants, 33 were randomised to the intervention group and 30 to the stretching group.

5.2.6 Procedure: intervention group

The intervention group received a total of 12 sessions of TPPR to the five designated MTrPs in the IMC, twice weekly for a total of six weeks. Ethics approval had been gained for the use of coolant spray as per the technique described by Simons et al., 1999, however, in order to limit variables, this technique was not used. Weight measurement was undertaken prior to each

intervention session, with height measurements recorded before and after each session. A copy of the Patient Specific Disability Measure form (Stratford et al., 1995) was supplied by the treating practitioner, to be completed after every second session, with instructions given verbally and written on the sheet (Appendix Nine) (or refer p.235). Participants were also required to undertake daily stretches as instructed in the initial assessment interview.
A stretching compliance diary was supplied to be filled in after stretches were performed (Appendix Ten) (or refer p.236).

5.2.7 Procedure: Stretching group

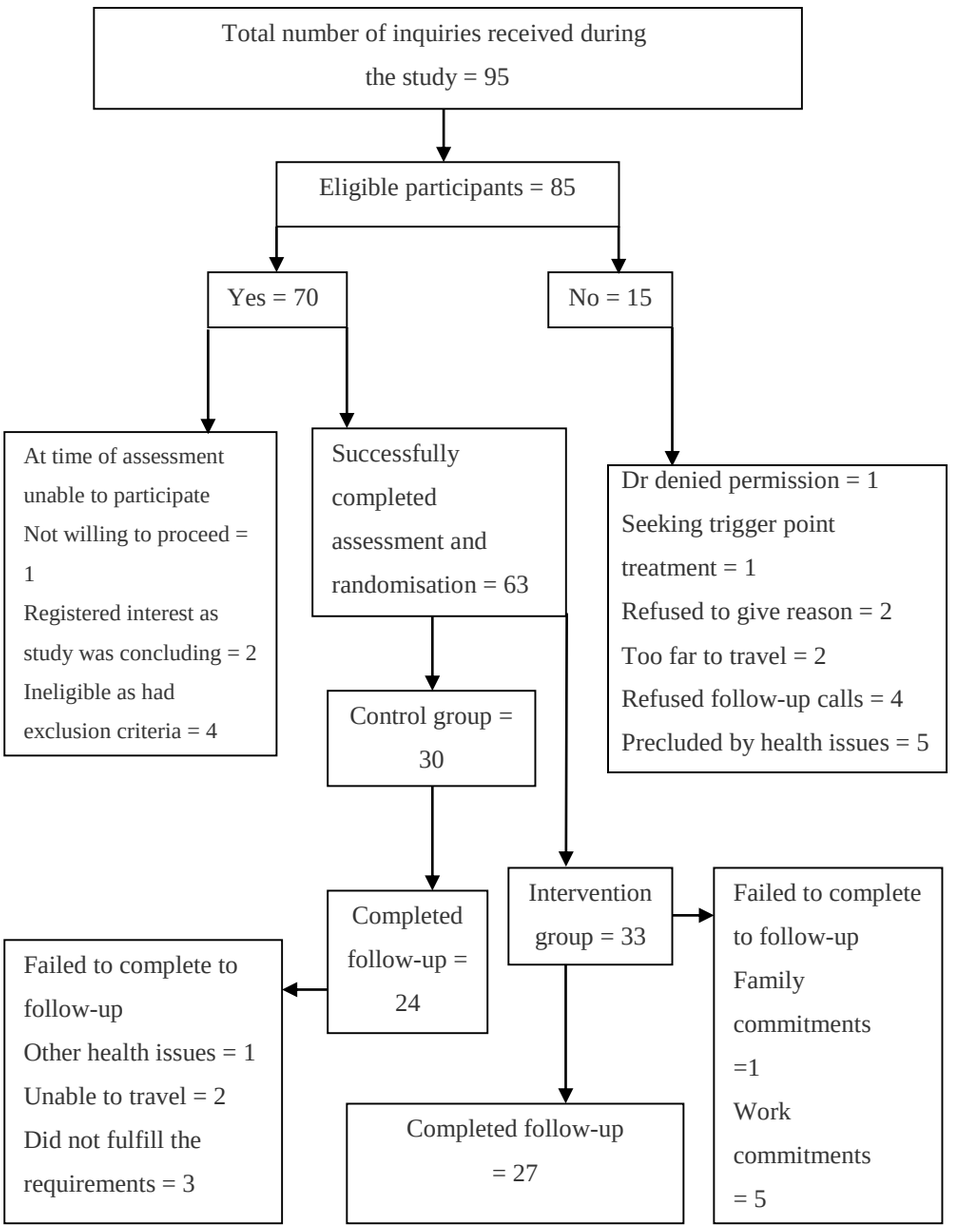
The procedures for the stretching group were

- weight and height measurements undertaken at baseline
- the requirement to complete the supplied Patient Specific Disability Measure form (Stratford et al., 1995) weekly for six weeks with instructions given verbally and being written on the form (Appendix Nine) (or refer p.235)
- daily self-administered stretching of the IMC as demonstrated prior to randomisation for six weeks (Appendix Ten) (or refer p.236).

No regular follow-up could be undertaken during the course of the study due to financial and clinical constraints. Despite being 'blinded'to the nature of techniques utilised in the two groups contact was received by the researcher from a number of participants (n = 17) in the stretching group, asking if they could participate in the intervention group. Knowledge of the two different approaches being utilised may have been ascertained by participants via the newspaper editorial or by "word of mouth", but was not disclosed by the researcher. These

requests were refused, but this did provide opportunities to re-inforce the stretching protocol and the need for data collection on the supplied forms, as outlined above.

Figure 5.1. Consort Flow Diagram: Recruitment to Completion



5.2.8 Outcome Assessment

The outcome assessor had no knowledge of which group each participant had been randomised to. The ‘blind’ assessor received appropriate training in palpation to examine for the presence of MTrPs, having had five years’ experience post training. The researcher was not present during the outcome assessment.

The outcome assessment was conducted six weeks after the commencement of either the intervention or the stretching program and included:

- weight and height measurements;
- bilateral palpation of five IMC MTrP sites;
- the Short-Form McGill Pain Questionnaire (Melzack, 1987); and
- calculation of scores based on entries recorded in the Patient Specific Disability Measure (Stratford et al., 1995).

The ' blind' assessor recorded clinical findings in the Outcome Assessment form (Appendix 11) (refer p.237), signed and dated the form. These data, and the second completed Short-Form McGill Pain Questionnaire and the Patient Specific Disability Measure form, were then sealed in an envelope and forwarded to the Student co-ordinator, Rehabilitation and Ageing Studies Unit, Repatriation General Hospital, Daw Park, South Australia. These were retained by the co-ordinator for ‘blinded’ entry into the SPSS participant data file.

There was no involvement by the researcher in the data entry process, which was performed by a person without knowledge or involvement in the study. The researcher’s supervisors recommended ‘blinded’ interim analysis after 63

participants had entered into the study. The statistician at the Repatriation General Hospital South Australia conducted this 'blind' interim analyses. Termination of the research component of the study was based on the interim analyses of these 63 participants demonstrating significant differences between the intervention and stretching groups. At the cessation of the study the final number of participants who completed the program was 51; 27 in the intervention group, and 24 in the stretching group.

5.3 Data analyses

Data collected were entered and analysed on SPSS version 10. The significance level was set at the 5 % level ($p = 0.05$).

For normally distributed continuous data, mean and standard deviation were calculated. Differences across the groups were tested at baseline and also at completion using independent sample t-tests. For non-normally distributed data, median and interquartile range were calculated and differences across the groups were tested using the Mann-Whitney *U* test. For categorical data, the chi square test of association was used.

5.4 Results

Of the 95 inquiries, 63 eligible people with CLBP or CSP participated, 51 of whom completed the study to full outcome assessment. Of the 63 participants who entered this study, there were no significant differences between those who completed, and those who failed to complete, the study in relation to age, gender,

the first site of pain, the duration of pain, the visual analogue score and the present pain index (Table 5.1).

Table 5.1: Baseline data of the 63 consented participants

Total n = 63	Participants who completed n = 51	Participants who failed to complete n = 12	Significance (*p*)
Age, years Mean (SD)	46.6 (10.13)	46.7 (10.15)	t(61) = 0.03* (*p* = 0.974)
Gender no (%) Male Female	 22 (43.1%) 29 (56.9%)	 7 (58.3%) 5 (41.7%)	 X^2 (1) = .90^ (*p* = 0.342)
First site of pain no (%) Lumbar Thoracic Cervical	 46 (96.8%) 0 (0%) 2 (4.2%)	 8 (80%) 1 (10%) 1 (10%)	 X^2 (2) = 5.56^ (*p* = 0.134)
Duration of pain (years) Mean (SD)	19.35 (11.00)	15.25 (10.60)	t(61) = 1.17* (*p* = 0.247)
Visual Analogue Scale Median (IQR)	5.43 (1.56)	5.36 (1.65)	Z = 294.5˚
Present Pain Index Mean (SD)	2.00 (1.01)	2.58 (1.30%)	t(60) = 1.69* (*p* = 0.096)

3=Completed, 2=Failed to complete) subjects recorded multiple first sites and were excluded from this analysis.
**Fisher Exact test*
^Pearsons X^2 square
˚Mann-Whitney U test

As would be expected with randomisation, no statistical differences were found between the intervention and stretching groups, baseline assessment of age, gender, height, weight, hand and leg dominance (Tables 5.1 and 5.2). Participants had a mean age of 46.6 years, the majority (56.9%) were female, and had a BMI in the overweight, but not obese, range. Over 90% were right hand and right leg dominant. The majority of participants in both groups described the onset of pain

as being in the lumbar region, being of sudden onset, and present for a mean of over 19 years.

Table 5.2: Baseline data of the 51 participants who completed to follow up

	Baseline		
	Intervention n = 27	Stretching n = 24	Significance (*p*)
Age Mean (SD)	46.0 (10.57)	47.4 (9.79)	t(.48) = 49^ (p = 0.631)
Gender n (%) Female Male	 18 (69.2%) 9 (33.3%)	 11 (44%) 13 (54%)	 $X^2(1) = 2.24$* (*p* = 0.134)
First site of pain Lumbar Cervical	 24 (96%) 1 (4.0%)	 22 (95.7%) 1 (4.3%)	 *p* = 0.952●
Height Mean (SD)	169.93 (7.55)	171.96 (8.21)	t(0.91 = 49^ (*p* = 0.363)
Weight Mean (SD)	82.70 (19.08)	80.94 (12.26)	t(.701) = 49^ (*p* = 0.701)
Onset (years) Sudden Gradual	 18 (66.7%) 9 (33.3%)	 14 (58.3%) 10 (41.7%)	 $X^2(1) = .37$* (*p* = 0.539)
Duration of pain (years) Mean (SD)	19.88 (11.65)	18.75 (10.43)	t(.36) = 49^ (*p* = 0.716)
Dominant hand n (%) Right Left	 26 (96.3%) 1 (3.8%)	 22 (91.7%) 2 (8.3%)	 *p* = 0.483●
Dominant leg n (%) Right Left	 26 (96.3%) 1 (3.7%)	 22 (91.7%) 2 (8.3%)	 *p* = 0.483●

∗2 treatment and 1 stretching reported multiple pain sites and were excluded
●Fishers Exact Test
****Pearsons X^2 Test***
^ Independent Samples Test

There were no differences between the groups in the attributed cause of the pain (Table 5.3). The three most common attributed activities were trauma, lifting, and an occupational incident. Six participants were unable to identify any specific event or cause to explain the onset of their pain.

Table 5.3: Attributed cause of spinal pain in the 51 participants who completed to follow-up

Activity classification	Intervention n = 27 (no%)	Stretching n = 24 (no%)
Trauma	6 (22.2%)	4 (16.7%)
Lifting	5 (18.5%)	5 (20.8%)
Occupational incident	5 (18.5%)	5 (20.8%)
Recreational	3 (11.1%)	2 (8.3%)
Unknown	2 (7.4%)	4 (16.7%)
Pregnancy	2 (7.4%)	1 (4.2%)
Sitting	2 (7.4%)	0 (0%)
Carrying	1 (3.7%)	1 (4.2%)
Growth spurt	1 (3.7%)	2 (8.3%)

The majority of participants in both groups reported that the pain had spread from its initial site (Table 5.4). When asked where the pain had spread to 43.3% (n = 13) of the intervention group and 32.3% (n = 10) of the stretching group reported the thoracic region, and 23.3% (n = 7) of the intervention group, and 22.6% (n = 7) in the stretching group reported the lower limb. Other less commonly reported sites were the gluteal region, cervical region, and the genitals.

Table 5.4: Spread of pain from the original site

			Has the pain spread n (% rounded to whole number)		**Total**	Pearson X^2 Significance (*p*)
			Yes	No		
Group	Intervention	Count	31	2	33	X^2 (1) (*p* = 0.157)
		% within group	94%	6%	100.0%	
	Stretching	Count	30	0	30	
		% within group	100.0%	0%	100.0%	
Total		Count	61	2	63	
		% within group	97%	3%	100.0%	

Participants had used a wide range of previous treatments for in their attempts to address their CLBP (Table 5.5). The majority, perhaps reflecting the recruitment base, had used physiotherapy and chiropractic treatments.

Table 5.5: Past treatments undertaken by the 51 participants who completed to follow up

Treatment received	Past (n%) Intervention n = 27	Past (n%) Stretching n = 24	Current (n%) Intervention n = 27	Current (n%) Stretching n = 24
Physiotherapy	22 (81.5%)	19 (79.2%)	3 (11.1%)	4 (16.7%)
Chiropractic	16 (59.3%)	15 (62.5%)	3 (11.1%)	2 (8.3%)
Massage	6 (22%)	4 (16.7%)	4 (14.8%)	2 (8.3%)
Prescribed exercise	3 (11.1%)	4 (16.7%)	0 (0%)	2 (8.3%)
Hydrotherapy	2 (7.4%)	2 (8.3%)	2 (7.4%)	0 (0%)
Acupuncture	2 (7.4%)	4 (16.7%)	1 (3.7%)	0 (0%)
Orthopaedic	1 (3.7%)	7 (29.2%)	1 (3.7%)	0 (0%)
Medication	4 (14.8%)	6 (25%)	4 (14.8%)	4 (16.7%)
Other	5 (18.5%)	3 (12.5%)	0 (0%)	1 (4.2%)

MTrPs were examined with palpation in the 51 participants who completed, and the findings were categorised as absent, or latent by the criteria of Ge and Arendt-Nielsen (2011), or active as per the criteria of Simon et al., (1999), as contained in Tables 5.6a.and 5.6b

A greater number of participants in the intervention group were found to have an active MTrP at site 2 on the side of their dominant leg (70% compared with 38%) and dominant hand (63% compared with 29%). Having two out of twenty variables show a difference at the level of $p = 0.05$ is within normal statistical expectations.

Table 5.6a: Baseline trigger point data of the 51 participants who completed to follow-up

Baseline		Intervention n = 27	Stretching n = 24	Significance (*p*)
Trigger Point 1 no (%) within group				
Dominant Hand	Absent	4(14.8%)	0(0%)	
	Latent	9(33.3%)	9(37.5%)	*p* = 0.190*
	Active	14(51.9%)	15(62.5%)	
Dominant Leg	Absent	2(7.4%)	0(0%)	
	Latent	9(33.3%)	8(33.3%)	*p* = 0.679*
	Active	16(59.3%)	16(66.7%)	
Non-Dominant Hand	Absent	3(11.1%)	4(16.7%)	
	Latent	13(48.1%)	8(33.3%)	*p* = 0.552*
	Active	11(40.7%)	12(50%)	
Non-Dominant Leg	Absent	5(18.5%)	4(16.7%)	
	Latent	13(48.1%)	9(37.5%)	*p* = 0.663*
	Active	9(33.3%)	11(45.8%)	
Trigger Point 2 no (%)				
Dominant Hand	Absent	5(18.5%)	2(8.3%)	
	Latent	5(18.5%)	15(62.5%)	*p* = 0.006*
	Active	17(63%)	7(29.2%)	
Dominant Leg	Absent	3(11.1%)	2(8.3%)	
	Latent	5(18.5%)	13(54.2%)	*p* = 0.026*
	Active	19(70.4%)	9(37.5%)	
Non-Dominant Hand	Absent	3(11. 1%)	9(37.5%)	*p* = 0.097
	Latent	12(44.4%)	8(33.3%)	
	Active	12(44.4%)	7(29.2%)	
Non-Dominant Leg	Absent	5(18.5%)	9(37.5%)	*p* = 0.254
	Latent	12 (44.4%)	10(41.7%)	
	Active	10(37%)	5(20.8%)	
Trigger Point 3 no (%)				
Dominant Hand	Absent	6 (22.2%)	4(16.7%)	
	Latent	10(37%)	12(50%)	*p* = 0.711*
	Active	11(40.7%)	8(33%)	
Dominant Leg	Absent	5(18.5%)	4(16.7%)	
	Latent	10 (37%)	12(50%)	*p* = 0.617*
	Active	12(44.4%)	8(33. %)	
Non-Dominant Hand	Absent	5(18.5%)	9(37.5%)	
	Latent	14(51.9%)	10(41.7%)	*p* = 0.311
	Active	8(29.6%)	5(20.8%)	
Non-Dominant Leg	Absent	6(22.2%)	9(37.5%)	*p* = 0.513
	Latent	14(51.9%)	10(41.7%)	
	Active	7(25.9%)	5(20.8%)	

Table 5.6b Baseline trigger point data of the 51 participants who completed to follow-up

Trigger Point 4 no (%)				
Dominant Hand	Absent	7(25.9%)	6(25%)	$p = 0.156$
	Latent	6(22.2%)	11(45.8%)	
	Active	14(51.9%)	7(29.2%)	
Dominant Leg	Absent	6(22.2%)	4(16.7%)	$p = 0.113$
	Latent	6(22.2%)	12(50%)	
	Active	15(55.6%)	8(33.3%)	
Non-Dominant Hand	Absent	7(25.9%)	9(37.5%)	$p = 0.732$
	Latent	10(37%)	8(33.3%)	
	Active	10(37%)	7(29.2%)	
Non-Dominant Leg	Absent	8(29.6%)	11(45.8%)	$p = 0.601$
	Latent	10(37%)	7(29.2%)	
	Active	9(33.3%)	6(25%)	
Trigger Point 5 no (%)				
Dominant Hand	Absent	5(18.5%)	3(12.5%)	
	Latent	9(33.3%)	9(37.5%)	$p = 0.865$*
	Active	13(48.1%)	12(50%)	
Dominant Leg	Absent	3(11.1%)	2(8.3%)	
	Latent	9(33.3%)	10(41.7%)	$p = 0.919$*
	Active	15(55.6%)	12(50%)	
Non-Dominant Hand	Absent	5(18.5%)	5(20.8%)	$p = 1.00$
	Latent	11(40.7%)	9(37.5%)	
	Active	11(40.7%)	10(41.7%)	
Non-Dominant Leg	Absent	7(25.9%)	6(25%)	$p = 0.936$
	Latent	11(40.7%)	8(33.3%)	
	Active	9(33.3%)	10(41.7%)	

** Fishers Exact Test*

The Short-Form McGill Pain Questionnaire contains 15 affective descriptors, a Visual Analogue Scale (VAS), and the Present Pain Index (PPI). Participants were able to choose more than one affective descriptor for their pain. There were no significant differences found on any of these elements between the intervention and stretching groups at baseline (Table 5.7)

Table 5.7: Baseline data per item of the Short Form McGill pain questionnaire for the 51 participants who completed to follow-up

Descriptor	Intervention n = 27* Median (IQR)	Stretching n = 24* Median (IQR)	Mann-Whitney *U* test (*p*)
Throbbing	0 (2)	0 (2)	Z = .42 (*p* = 0.673)
Shooting	2 (3)	2 (3)	Z = .79 (*p* = 0.428)
Stabbing	2 (3)	1 (3)	Z = .33 (*p* = 0.736)
Sharp	2 (3)	3 (3)	Z = .41 (*p* = 0.677)
Cramping	0 (3)	1 (2)	Z = .66 (*p* = 0.504)
Gnawing	1 (3)	2 (3)	Z = .05 (*p* = 0.960)
Hot/burning	0 (2)	1 (2)	Z = .05 (*p* = 0.959)
Aching	3 (1)	3 (1)	Z = .66 (*p* = 0.504)
Heavy	1 (2)	5 (2)	Z = .00 (*p* = 1.000)
Tender	2 (2)	2 (2)	Z = .22 (*p* =0 .819)
Splitting	0 (3)	0 (2)	Z = 1.25 (*p* = 0.210)
Tiring/exhausting	2 (2)	2 (2)	Z = .07 (*p* = 0.943)
Sickening	0 (2)	0 (2)	Z = .29 (*p* = 0.770)
Fearful	1 (3)	1 (3)	Z = .072 (*p* = 0.944)
Punishing/cruel	0 (1)	5 (2)	Z = 1.45 (*p* = 0.146)
Visual analogue scale	5.3(2.3)	6.5 (1.88)	Z=-1.88 (*p* = 0.059)
Present pain index No pain Mild Discomforting Distressing Horrible	Number (%) 2(7.4%) 4(14.8%) 13(48.1%) 7(25.9%) 1(3.7%)	Number (%) 1(4.2%) 8(33.3%) 8(33.0%) 4(16.7%) 3(12.5%)	$X^2 = 4.514$ (p = 0.341)

** unless otherwise stated Median and interquartile range is shown.*

Tables 5.8 (a-d), and 5.9(a-b)contain the trigger point data of the intervention and stretching groups who completed to follow-up, while Figures 5.1 to 5.10 contain baseline versus completion data of the intervention and stretching groups. Figures 5.11 and 5.12 contain average trigger point prevalence in the intervention and stretching groups, pre and cessation of the study.

Table 5.8a: Trigger point data baseline vs completion

		Dominant hand MTrP 1 at follow up				
		Absent	Latent	Active	**Total**	*p*
Dominant hand MTrP1 at baseline Total	Absent	1	3	0	4	0.000
	Latent	6	12	0	18	
	Active	12	6	11	29	
		19	21	11	51	
		Dominant hand MTrP 2 at follow up				
Dominant hand MTrP 2 at baseline Total	Absent	4	2	1	7	0.000
	Latent	11	6	3	20	
	Active	14	5	5	24	
		29	13	9	51	
		Dominant hand MTrP 3 at follow up5				
Dominant hand MTrP 3 at baseline Total	Absent	4	4	2	10	0.246
	Latent	8	10	4	22	
	Active	6	7	6	19	
		18	21	12	51	
		Dominant hand MTrP 4 at follow up				
Dominant hand MTrP 4 at baseline Total	Absent	8	3	2	13	0.090
	Latent	5	10	2	17	
	Active	10	4	7	21	
		23	17	11	51	
		Dominant hand MTrP 5 at follow up				
Dominant hand MTrP 5 at baseline Total	Absent	2	3	3	4	0.203
	Latent	5	8	5	18	
	Active	10	7	8	29	
		17	18	16	51	

Table 5.8b Trigger point data baseline v. completion

		Dominant leg MTrP 1 at follow up				
		Absent	Latent	Active	**Total**	*p*
Dominant leg MTrP1 at baseline	Absent	0	2	0	4	0.000
	Latent	6	11	0	18	
	Active	14	8	10	29	
Total		20	21	10	51	
		Dominant leg MTrP 2 at follow up				
Dominant leg MTrP 2 at baseline	Absent	2	2	0	5	0.000
	Latent	10	4	4	18	
	Active	15	7	6	28	
Total		28	13	10	51	
		Dominant leg MTrP 3 at follow up				
Dominant leg MTrP 3 at baseline	Absent	4	2	3	9	0.076
	Latent	8	11	3	22	
	Active	6	8	6	20	
Total		18	21	12	51	
		Dominant leg MTrP 4 at follow up				
Dominant leg MTrP 4 at baseline	Absent	7	2	1	10	0.014
	Latent	6	10	2	18	
	Active	10	5	8	23	
Total		23	17	11	51	
		Dominant leg MTrP 5 at follow up				
Dominant leg MTrP 5 at baseline	Absent	1	2	2	5	0.036
	Latent	6	7	6	19	
	Active	11	8	8	27	
Total		18	17	16	51	

Table 5.8c: Trigger point data baseline v. completion

		Absent	Latent	Active	**Total**	*p*
Non dominant hand MTrP1 at baseline Total	Absent	5	1	1	7	0.014
	Latent	6	10	5	21	
	Active	8	10	5	23	
		19	21	11	51	
		Non dominant hand MTrP 2 at follow up				
Non dominant hand MTrP 2 at baseline Total	Absent	5	5	2	12	0.015
	Latent	11	7	2	20	
	Active	11	6	2	19	
		27	18	6	51	
		Non dominant hand MTrP 3 at follow up				
Non dominant hand MTrP 3 at baseline Total	Absent	9	3	2	14	0.273
	Latent	8	9	7	24	
	Active	5	5	3	13	
		22	17	12	51	
		Non dominant hand MTrP4 at follow up				
Non dominant hand MTrP 4 at baseline Total	Absent	12	2	2	16	0.041
	Latent	9	7	2	18	
	Active	8	3	6	17	
		29	12	10	51	
		Non dominant hand MTrP 5 at follow up				
Non dominant hand MTrP 5 at baseline Total	Absent	3	5	2	10	0.069
	Latent	11	5	4	20	
	Active	9	6	6	21	
		23	16	12	51	

Table 5.8d Trigger point data baseline v. completion

		Non dominant leg MTrP 1 at follow up				
		Absent	Latent	Active	**Total**	*p*
Non dominant leg MTrP1 at baseline	Absent	6	2	1	9	0.066
	Latent	6	11	1	22	
	Active	7	8	5	20	
Total		19	21	11	51	
		Non dominant leg MTrP 2 at follow up				
Non dominant leg MTrP 2 at baseline	Absent	6	5	3	14	0.038
	Latent	12	9	1	22	
	Active	10	4	1	15	
Total		28	18	5	51	
		Non dominant leg MTrP 3 at follow up				
Non dominant leg MTrP 3 at baseline	Absent	9	5	1	15	0.196
	Latent	8	8	8	24	
	Active	5	4	3	12	
Total		22	17	12	51	
		Non dominant leg MTrP 4 at follow up				
Non dominant leg MTrP 4 at baseline	Absent	13	3	3	19	0.208
	Latent	8	7	2	17	
	Active	8	2	5	15	
Total		29	12	10	51	
		Non dominant leg MTrP 5 at follow up				
Non dominant leg MTrP 5 at baseline	Absent	4	6	3	13	0.287
	Latent	10	6	3	13	
	Active	8	5	6	19	
Total		22	17	12	51	

As shown in Table 5.9 significant improvements were achieved in 17 of the 20 trigger point sites of the intervention and stretching groups. Reductions of active MTrPs in the intervention group, baseline to completion, by 53.72%, and in the stretching group the decrease in active MTrPs was 13.33% as can be seen Figure 5.11. In the findings of absent MTrPs, this was demonstrated to increase from an average of 5 pre-intervention to 13.85 in the intervention group, evidencing a change of 177%, with an increase from 5.1 to 6.95 in the stretching group (refer Figure 5.12).

Table 5.9a Trigger point data at completion

Completion (n = 51)		**Intervention (n = 27)**	**Stretching (n = 24)**	*P*
Trigger Point 1 n (%) within group				
Dominant Hand	Absent	4(14.8%)	5(20.8%)	
	Latent	9(33.3%)	12(50%)	t(5.06)=50
	Active	14(51.9%)	7(29.2%)	*p* = 0.000
Dominant Leg	Absent	15(55.6%)	5(20.8%)	
	Latent	9(29.6%)	13(54.2%)	t(5.97) = 50
	Active	4(14.8%)	6(25%)	*p* = 0.000
Non-Dominant Hand	Absent	14(51.9%)	6(25%	
	Latent	9(33.3%)	12(50%)	t(3.68) = 50
	Active	4(14.8%)	6(25%)	*p* = 0.001
Non-Dominant Leg	Absent	13(48.1%)	6(25%)	
	Latent	10(37%)	11(45.8%)	t(2.77) = 50
	Active	4(14.8%)	7(29.2%)	*p* = 0.008
Trigger Point 2 n (%) within the group				
Dominant Hand	Absent	21(77.8%)	8(33.3%)	
	Latent	4(14.8%)	9(37.5%)	t(4.97) = 50
	Active	2(7.4%)	7(29.2%)	*p* = 0.000
Dominant Leg	Absent	21(77.8%)	7(29.2%)	
	Latent	4(14.5%)	9(37.5%)	t(3.68) = 50
	Active	2(7.4%)	8(33.3%)	*p* = 0.000
Non-Dominant Hand	Absent	17(63%)	10(41.7%)	
	Latent	10(37%)	8(33.3%)	t(3.56) = 50
	Active	0(0%)	6(25%)	*p* = 0.001
Non-Dominant Leg	Absent	17(63%)	11(45.8%)	
	Latent	10(37%)	8(33.3%)	t(3.00) = 50
	Active	0(0%)	5(20.8%)	*p* = 0.004

Table 5.9b Trigger point data at completion

Trigger Point 3 n (%) within the group				
Dominant Hand	Absent	11(40.7%)	7(29.2%)	
	Latent	13(48.1%)	8(33.3%)	t(2.08) = 50
	Active	3(11.1%)	9(37.5%)	*p* = 0.042
Dominant Leg	Absent	5(18.5%)	6(25%)	
	Latent	10(37%)	8(33.3%)	t(2.34) = 50
	Active	12(44.4%)	10(41.6%)	*p* = 0.023
Non-Dominant Hand	Absent	18(66.7%)	8 (33.3%)	
	Latent	6(22.2%)	9(37.5%)	t(1.26) = 50
	Active	3(11.1%)	7(29.2%)	*p* = 0.211
Non-Dominant Leg	Absent	14(51.9%)	8(33.3%)	
	Latent	8(29.6%)	9(37.5%)	t(1.00) = 50
	Active	5(18.5%)	7 (29.2%)	*p* = 0.322
Trigger Point 4 n (%) within the group				
Dominant Hand	Absent	17(63%)	6(25%)	
	Latent	7(25.9%)	10(41.7%)	t(2.69) = 50
	Active	3(11.1%)	8(33.3%)	*p* = 0.010
Dominant Leg	Absent	6(22.2%)	4(16.7%)	
	Latent	6(22.2%)	12(50%)	t(3.62) = 50
	Active	15(55.6%)	8(33.3%)	*p* = 0.001
Non-Dominant Hand	Absent	18(66.7%)	11(45.8%)	
	Latent	6(22.2%)	6(25%)	t(2.85) = 50
	Active	3(11.1%)	7(29.2%)	*p* = 0.006
Non-Dominant Leg	Absent	18(66.7%)	11(45.8%)	
	Latent	6(22.2%)	6(25%)	t(2.01) = 50
	Active	3(1%)	7(29.2%)	*p* = 0.050
Trigger Point 5 n (%) within the group				
Dominant Hand	Absent	13(48.1%)	5(20.8%)	
	Latent	9(33.3%)	8(33.3%)	t(2.20) = 50
	Active	5(18.5%)	11(45.8%)	*p* = 0.032
Dominant Leg	Absent	3(11.1%)	2(8.3%)	
	Latent	9(33.3%)	10(41.7%)	t(3.00) = 50
	Active	15(55.6%)	12(50%)	*p* = 0.004
Non-Dominant Hand	Absent	16(59.3%)	7(29.2%)	
	Latent	9(33.3%)	7(29.2%)	t(2.80) = 50
	Active	2(7.4%)	10(41.7%)	*p* = 0.007
Non-Dominant Leg	Absent	16(59.3%)	6(25%)	
	Latent	8(29.6%)	9(37.5%)	t(1.99) = 50
	Active	3(11.1%)	9(37.5%)	*p* = 0.051

Figure 5.1 Baseline vs Completion – Trigger Point 1 (Intervention group)

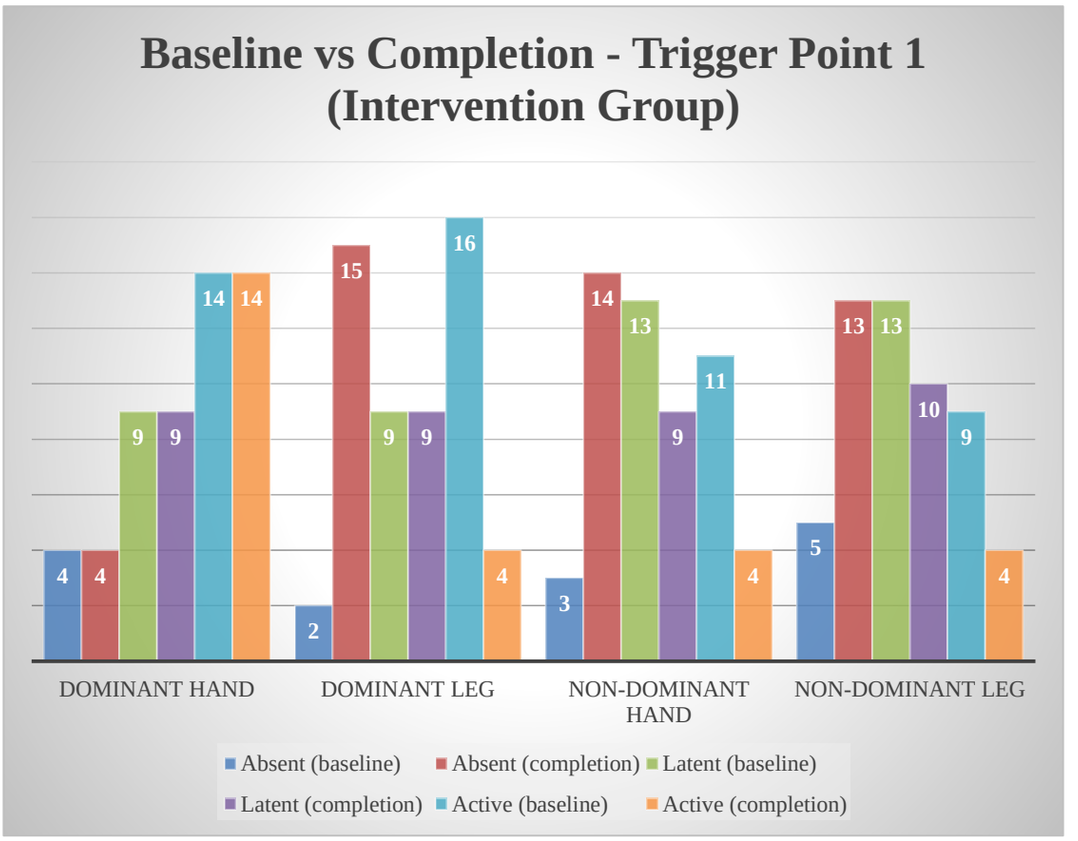


Figure 5.2 Baseline vs Completion – Trigger Point 1 (Stretching group)

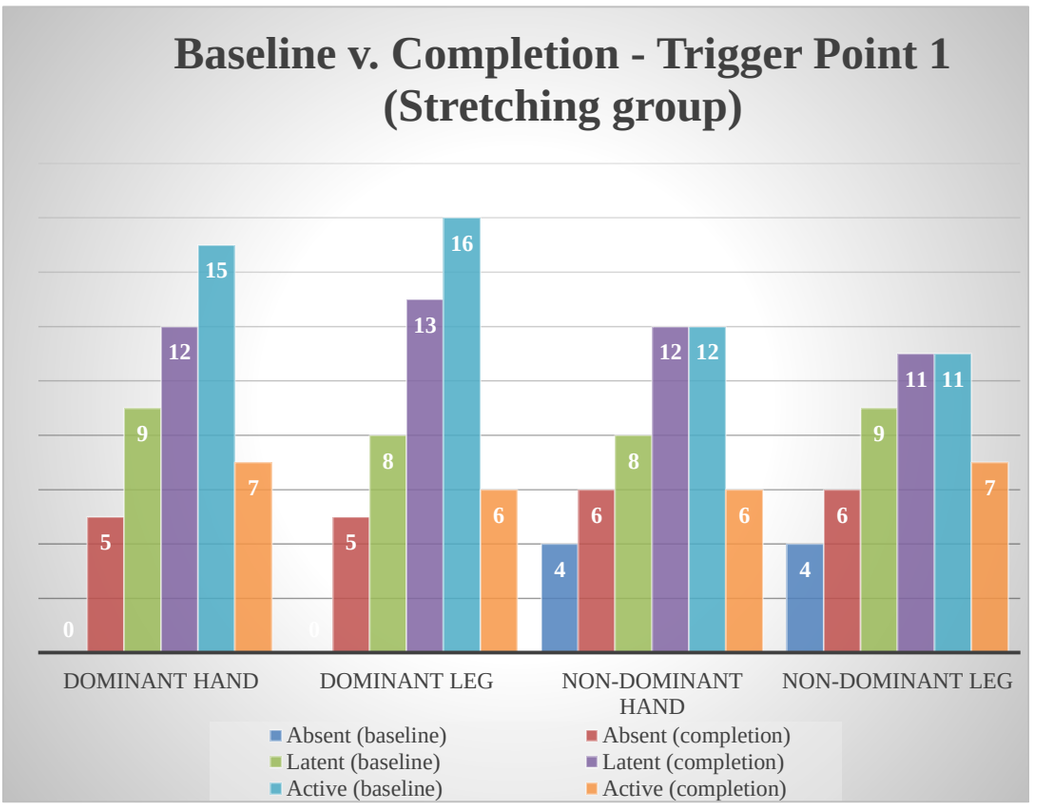


Figure 5.3 Baseline vs Completion – Trigger Point 2 (Intervention group)

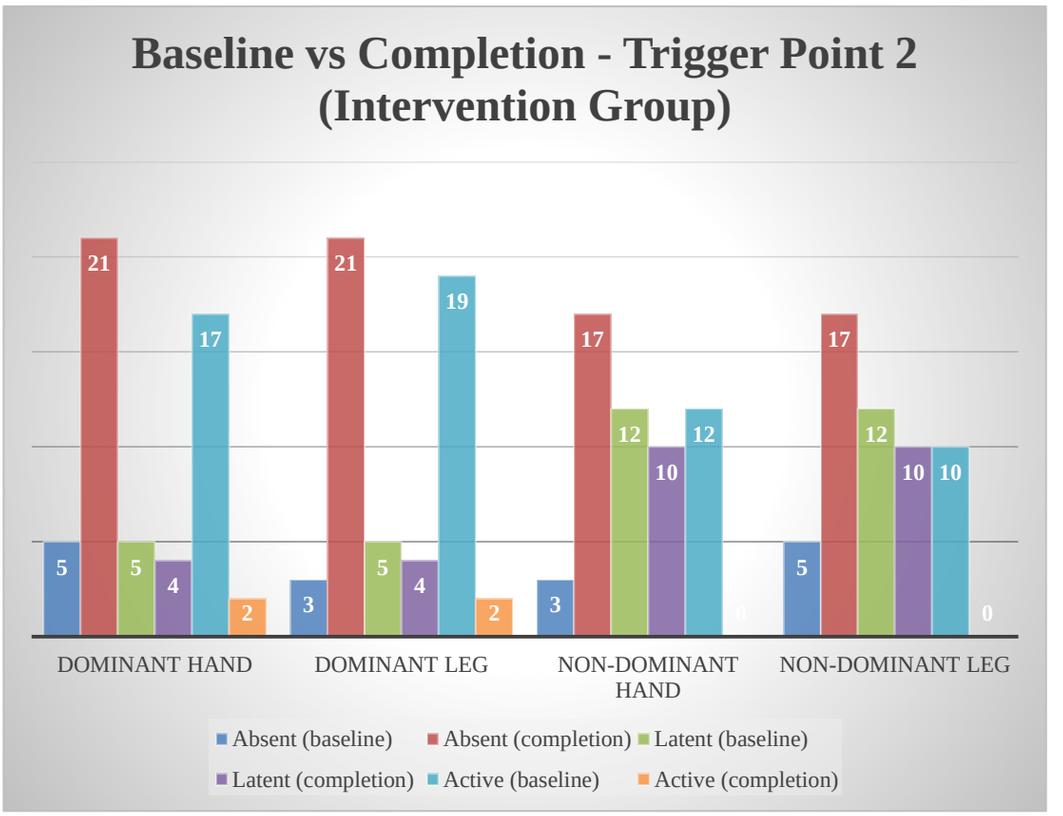


Figure 5.4 Baseline vs Completion – Trigger Point 2 (Stretching group)

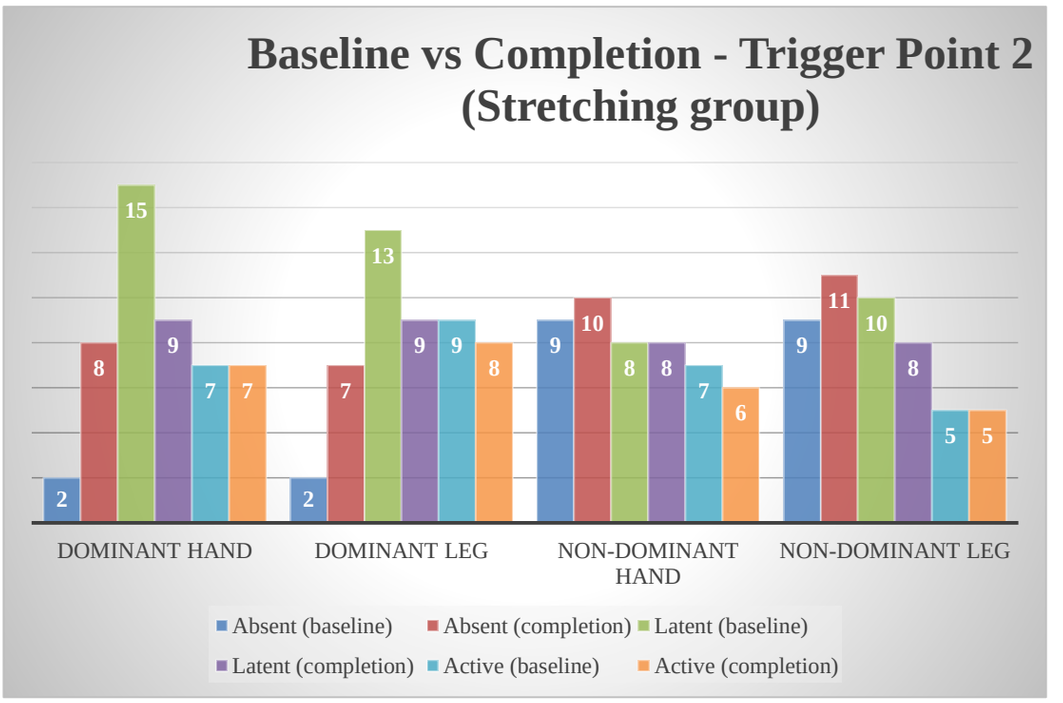


Figure 5.5 Baseline vs Completion – Trigger Point 3 (Intervention group)

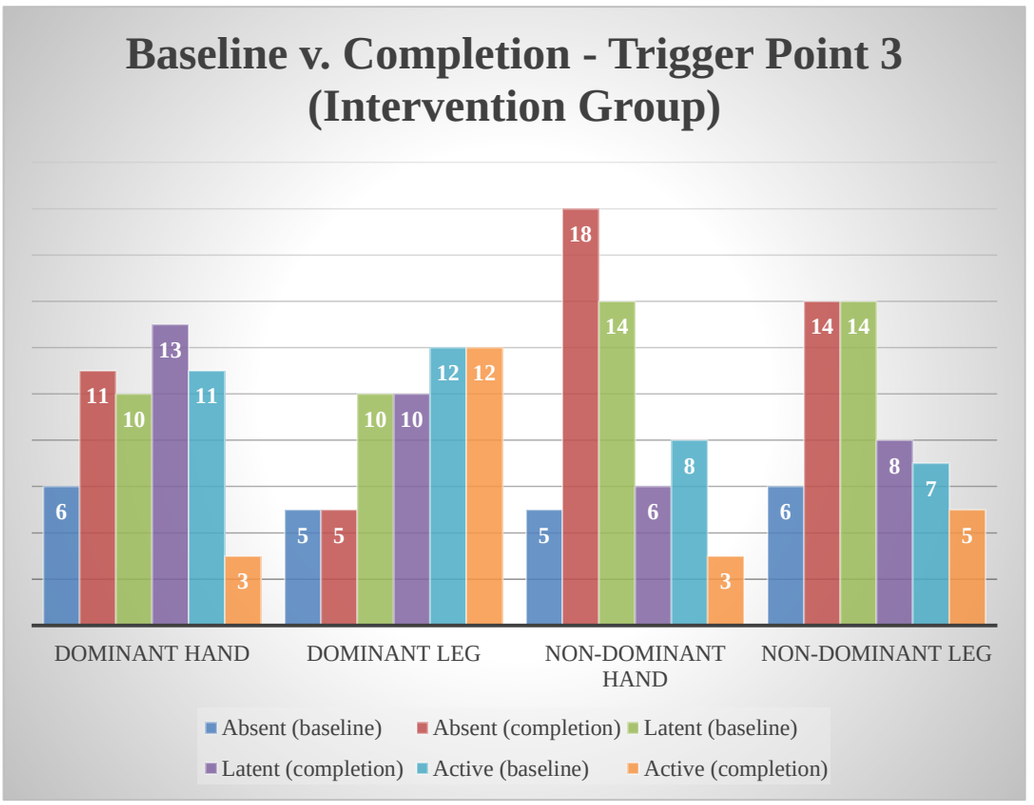


Figure 5.6 Baseline vs Completion – Trigger Point 3 (Stretching group)

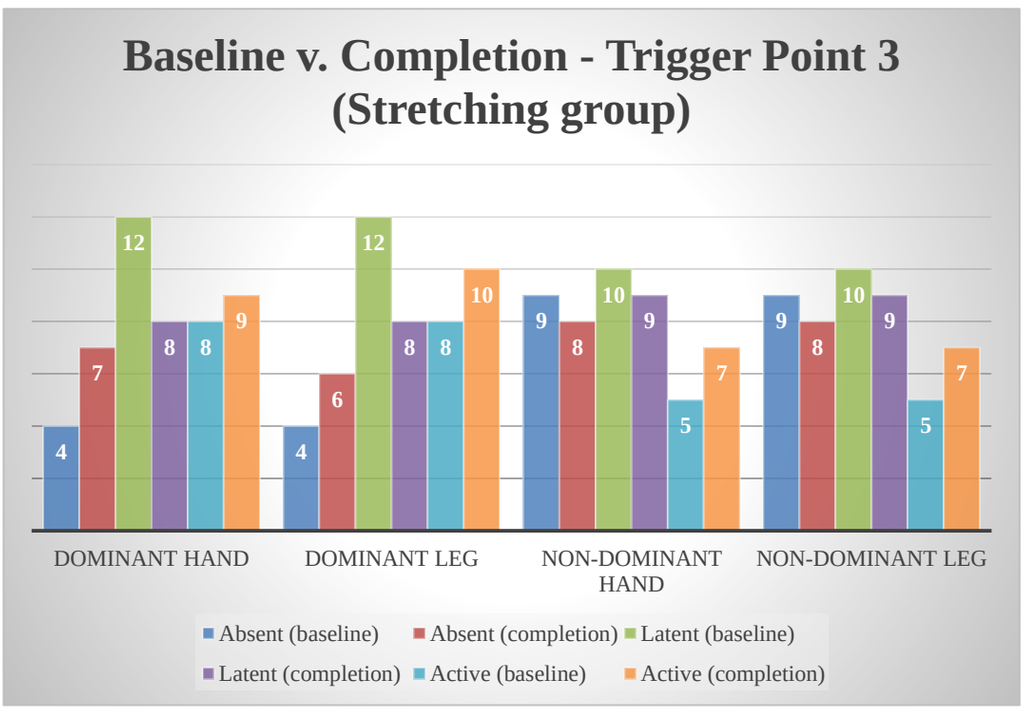


Figure 5.7 Baseline vs Completion – Trigger Point 4 (Intervention group)

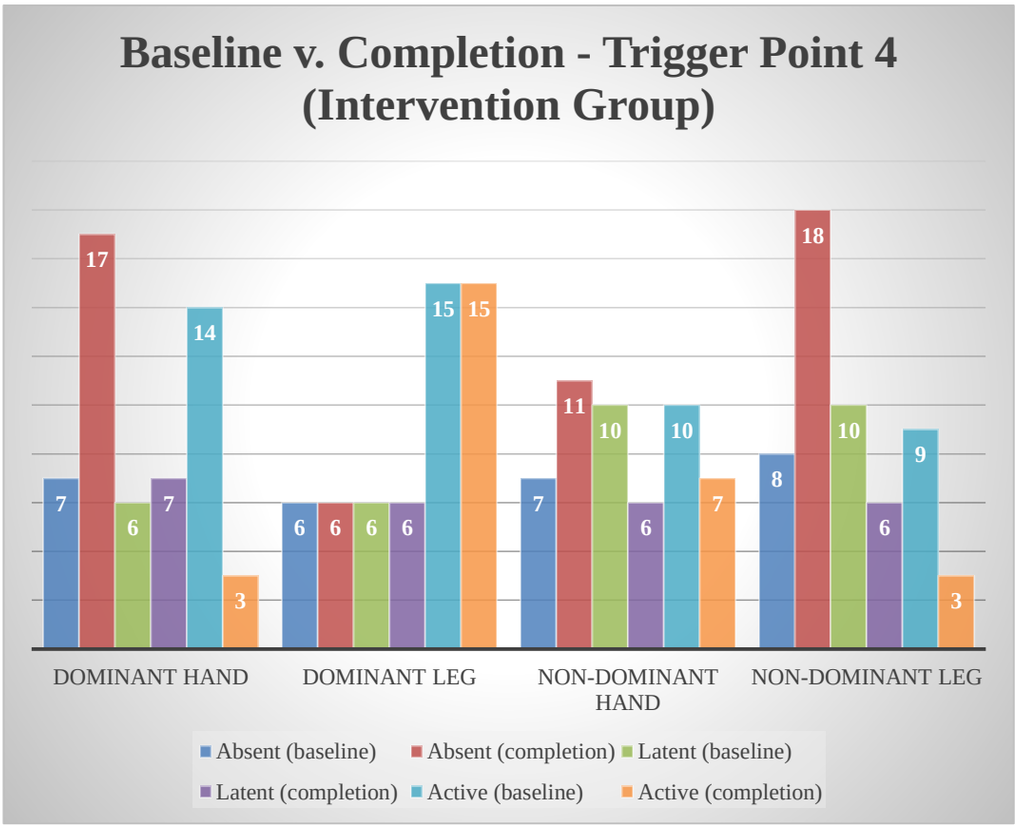


Figure 5.8 Baseline vs Completion – Trigger Point 4 (Stretching group)

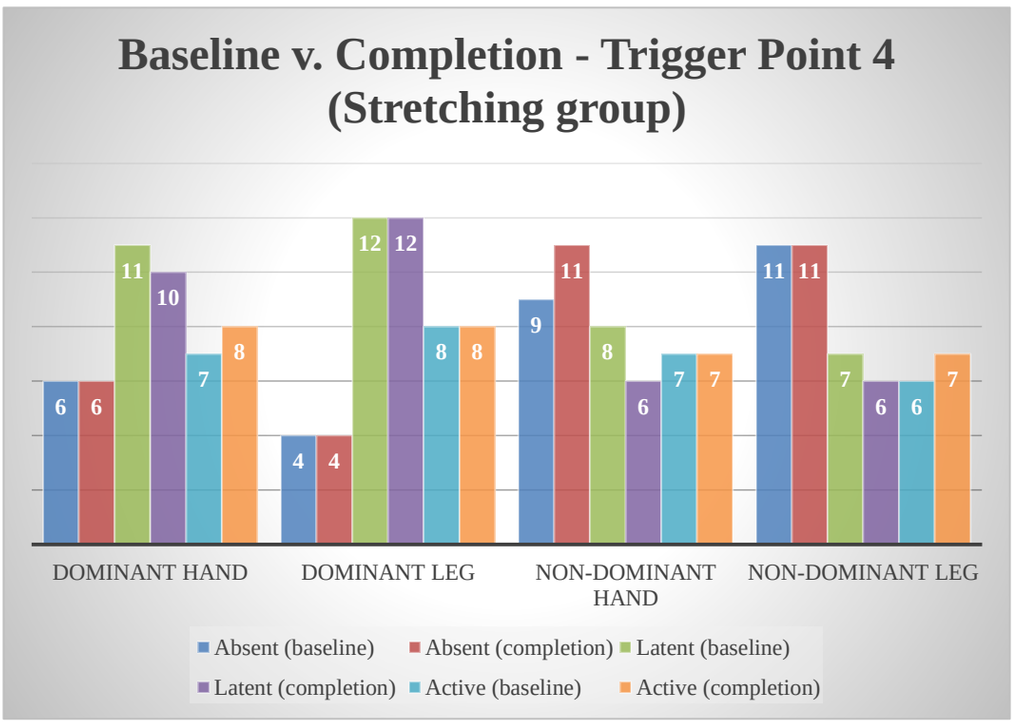


Figure 5.9 Baseline vs Completion – Trigger Point 5 (Intervention group)

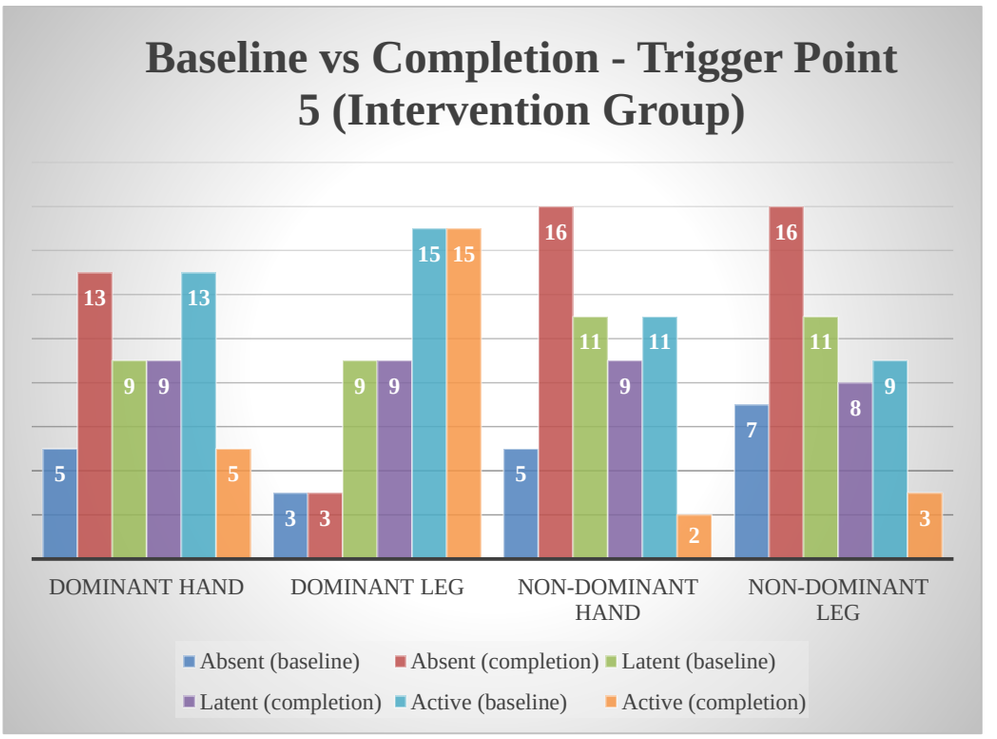


Figure 5.10 Baseline vs Completion – Trigger Point 5 (Stretching group)

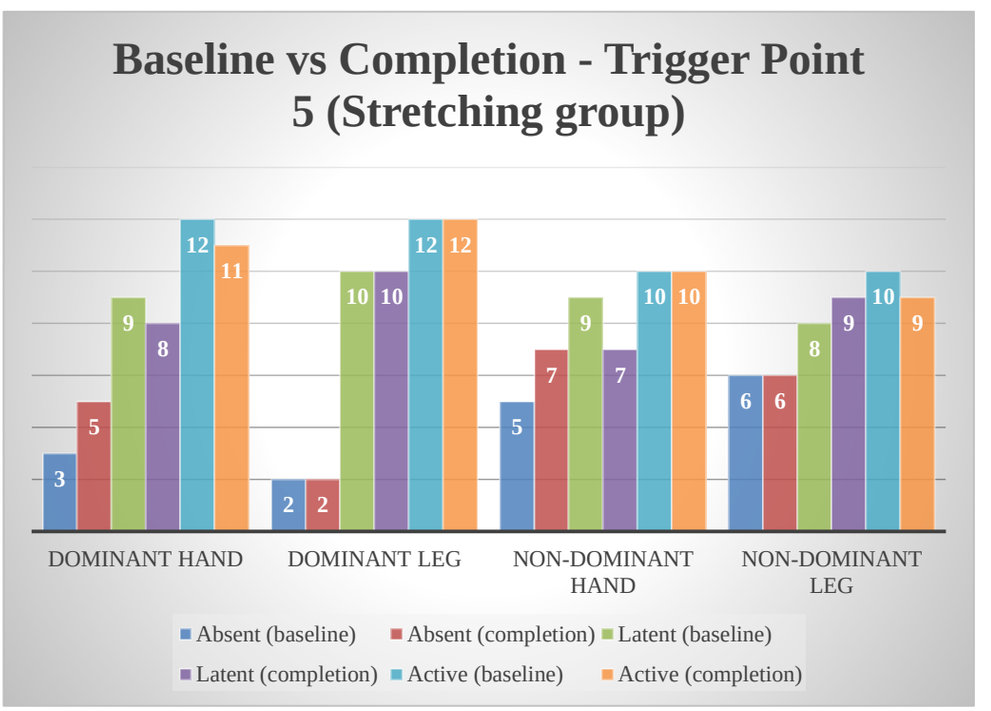


Table 5.10a Trigger point data at baseline vs completion in the intervention group

Intervention Group		Baseline	Completion
Trigger Point 1 n (%) within group			
Dominant Hand	Absent	4(14.8%)	4(14.8%)
	Latent	9(33.3%)	9(33.3%)
	Active	14(51.9%)	14(51.9%)
Dominant Leg	Absent	2(7.4%)	15(55.6%)
	Latent	9(33.3%)	9(29.6%)
	Active	16(59.3%)	4(14.8%)
Non-Dominant Hand	Absent	3(11.1%)	14(51.9%)
	Latent	13(48.1%)	9(33.3%)
	Active	11(40.7%)	4(14.8%)
Non-Dominant Leg	Absent	5(18.5%)	13(48.1%)
	Latent	13(48.1%)	10(37%)
	Active	9(33.3%)	4(14.8%)
Trigger Point 2 n (%) within the group			
Dominant Hand	Absent	5(18.5%)	21(77.8%)
	Latent	5(18.5%)	4(14.8%)
	Active	17(63%)	2(7.4%)
Dominant Leg	Absent	3(11.1%)	21(77.8%)
	Latent	5(18.5%)	4(14.5%)
	Active	19(70.4%)	2(7.4%)
Non-Dominant Hand	Absent	3(11. 1%)	17(63%)
	Latent	12(44.4%)	10(37%)
	Active	12(44.4%)	0(0%)
Non-Dominant Leg	Absent	5(18.5%)	17(63%)
	Latent	12(44.4%)	10(37%)
	Active	10(37%)	0(0%)
Trigger Point 3 n (%) within the group			
Dominant Hand	Absent	6 (22.2%)	11(40.7%)
	Latent	10(37%)	13(48.1%)
	Active	11(40.7%)	3(11.1%)
Dominant Leg	Absent	5(18.5%)	5(18.5%)
	Latent	10 (37%)	10(37%)
	Active	12(44.4%)	12(44.4%)
Non-Dominant Hand	Absent	5(18.5%)	18(66.7%)
	Latent	14(51.9%)	6(22.2%)
	Active	8(29.6%)	3(11.1%)
Non-Dominant Leg	Absent	6(22.2%)	14(51.9%)
	Latent	14(51.9%)	8(29.6%)
	Active	7(25.9%)	5(18.5%)

Table 5.10b Trigger point data at baseline vs completion in the intervention group

Intervention Group		**Baseline**	**Completion**
Trigger Point 4 n (%) within the group			
Dominant Hand	Absent	7(25.9%)	17(63%)
	Latent	6(22.2%)	7(25.9%)
	Active	14(51.9%)	3(11.1%)
Dominant Leg	Absent	6(22.2%)	6(22.2%)
	Latent	6(22.2%)	6(22.2%)
	Active	15(55.6%)	15(55.6%)
Non-Dominant Hand	Absent	7(25.9%)	18(66.7%)
	Latent	10(37%)	6(22.2%)
	Active	10(37%)	3(11.1%)
Non-Dominant Leg	Absent	8(29.6%)	18(66.7%)
	Latent	10(37%)	6(22.2%)
	Active	9(33.3%)	3(1%)
Trigger Point 5 n (%) within the group			
Dominant Hand	Absent	5(18.5%)	13(48.1%)
	Latent	9(33.3%)	9(33.3%)
	Active	13(48.1%)	5(18.5%)
Dominant Leg	Absent	3(11.1%)	3(11.1%)
	Latent	9(33.3%)	9(33.3%)
	Active	15(55.6%)	15(55.6%)
Non-Dominant Hand	Absent	5(18.5%)	16(59.3%)
	Latent	11(40.7%)	9(33.3%)
	Active	11(40.7%)	2(7.4%)
Non-Dominant Leg	Absent	7(25.9%)	16(59.3%)
	Latent	11(40.7%)	8(29.6%)
	Active	9(33.3%)	3(11.1%)

Table 5.11 Average number of trigger points in the intervention group

	Absent	Latent	Active
Baseline	5	9.9	12.1
Completion	13.85	8.1	5.6
Percentage Change	177%	-18.18%	-53.72%

Figure 5.11 Average number of trigger points in the intervention group

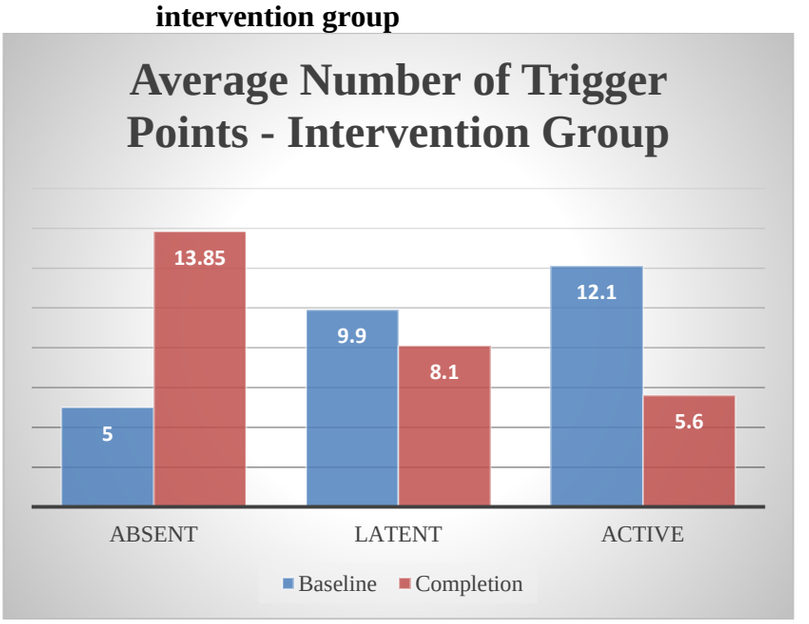


Table 5.12a Trigger point data baseline vs completion in the stretching group

Stretching Group		**Baseline**	**Completion**
Trigger Point 1 n (%) within group			
Dominant Hand	Absent	0(0%)	5(20.8%)
	Latent	9(37.5%)	12(50%)
	Active	15(62.5%)	7(29.2%)
Dominant Leg	Absent	0(0%)	5(20.8%)
	Latent	8(33.3%)	13(54.2%)
	Active	16(66.7%)	6(25%)
Non-Dominant Hand	Absent	4(16.7%)	6(25%
	Latent	8(33.3%)	12(50%)
	Active	12(50%)	6(25%)
Non-Dominant Leg	Absent	4(16.7%)	6(25%)
	Latent	9(37.5%)	11(45.8%)
	Active	11(45.8%)	7(29.2%)
Trigger Point 2 n (%) within the group			
Dominant Hand	Absent	2(8.3%)	8(33.3%)
	Latent	15(62.5%)	9(37.5%)
	Active	7(29.2%)	7(29.2%)
Dominant Leg	Absent	2(8.3%)	7(29.2%)
	Latent	13(54.2%)	9(37.5%)
	Active	9(37.5%)	8(33.3%)
Non-Dominant Hand	Absent	9(37.5%)	10(41.7%)
	Latent	8(33.3%)	8(33.3%)
	Active	7(29.2%)	6(25%)
Non-Dominant Leg	Absent	9(37.5%)	11(45.8%)
	Latent	10(41.7%)	8(33.3%)
	Active	5(20.8%)	5(20.8%)
Trigger Point 3 n (%) within the group			
Dominant Hand	Absent	4(16.7%)	7(29.2%)
	Latent	12(50%)	8(33.3%)
	Active	8(33%)	9(37.5%)
Dominant Leg	Absent	4(16.7%)	6(25%)
	Latent	12(50%)	8(33.3%)
	Active	8(33. %)	10(41.6%)
Non-Dominant Hand	Absent	9(37.5%)	8 (33.3%)
	Latent	10(41.7%)	9(37.5%)
	Active	5(20.8%)	7(29.2%)
Non-Dominant Leg	Absent	9(37.5%)	8(33.3%)
	Latent	10(41.7%)	9(37.5%)
	Active	5(20.8%)	7 (29.2%)

Table 5.12b Trigger point data baseline vs completion in the stretching group

Trigger Point 4 n (%) within the group			
Dominant Hand	Absent	6(25%)	6(25%)
	Latent	11(45.8%)	10(41.7%)
	Active	7(29.2%)	8(33.3%)
Dominant Leg	Absent	4(16.7%)	4(16.7%)
	Latent	12(50%)	12(50%)
	Active	8(33.3%)	8(33.3%)
Non-Dominant Hand	Absent	9(37.5%)	11(45.8%)
	Latent	8(33.3%)	6(25%)
	Active	7(29.2%)	7(29.2%)
Non-Dominant Leg	Absent	11(45.8%)	11(45.8%)
	Latent	7(29.2%)	6(25%)
	Active	6(25%)	7(29.2%)
Trigger Point 5 n (%) within the group			
Dominant Hand	Absent	3(12.5%)	5(20.8%)
	Latent	9(37.5%)	8(33.3%)
	Active	12(50%)	11(45.8%)
Dominant Leg	Absent	2(8.3%)	2(8.3%)
	Latent	10(41.7%)	10(41.7%)
	Active	12(50%)	12(50%)
Non-Dominant Hand	Absent	5(20.8%)	7(29.2%)
	Latent	9(37.5%)	7(29.2%)
	Active	10(41.7%)	10(41.7%)
Non-Dominant Leg	Absent	6(25%)	6(25%)
	Latent	8(33.3%)	9(37.5%)
	Active	10(41.7%)	9(37.5%)

Table 5.13 Average number of trigger points in the stretching group

	Absent	Latent	Active
Baseline	5.1	9.9	9
Completion	6.95	9.25	7.8
Percentage Change	36%	-6.57%	-13.33%

Figure 5.12 Average number of trigger points in the stretching group

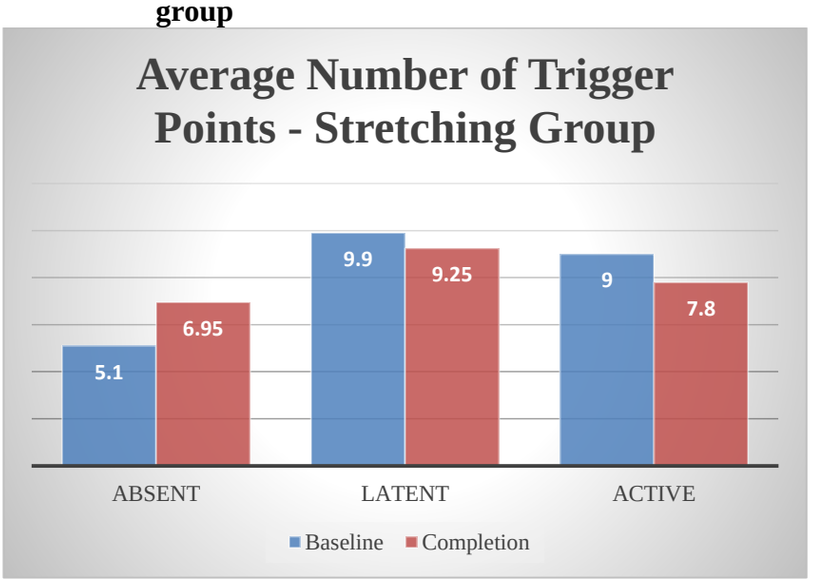


Table 5.14 Outcome analyses at completion

Analysis description Mean (SD)	**Intervention**	**Stretching**	*P*
Short-form McGill pain questionnaire: Affective change	3.44(2.80)	1.58(2.46)	t(2.50) = 49 *p* = 0.016
Short-form McGill pain questionnaire: Sensory change	11.22(5.87)	4.16(6.01)	t(4.23) = 49 *p* = 0.000
Short-form McGill pain questionnaire: Total change: affective and sensory scores	14.66(7.65)	5.75(7.75)	t(4.12) = 49 (*p* = 0.000)
Short-form McGill pain questionnaire: Total change Visual analogue scale	3.47(1.81)	1.92(2.04)	t(2.87) = 49 (*p* = 0.006)
Short-form McGill pain questionnaire: Total change Present Pain Index	1.14(.90)	.33(1.12)	t(2.85) = 49 (*p* = 0.006)
Height	.03(.85)	.30(.52)	t(1.66) = 48 (*p* = 0.103)
Weight	.82(2.56)	.53(18.9)	t(2.08) = 48 (*p* = 0.043)
Patient Specific Disability Outcome Measure	4.45(2.52)	1.36(1.74)	t(4.95) = 48 (*p* = 0.000)

5.5 Summary

At the cessation of the study, the results demonstrated that the participants who received 12 sessions of TPPR, and had undertaken self-administered stretching gained significant improvements in the sensory, combined sensory and affective categories, and present pain index of the Short-Form McGill Pain Questionnaire, the Patient Specific Disability Measure, with reduction in MTrP sites when compared to those who undertook self-administered stretching.

The incidence, and costs, of LBP, CLBP, and CSP were stated to threaten the Welfare systems in industrialised countries by Nachemson (2004). Again, in 2013, LBP was identified as the foremost reason of worldwide disability (Buchbinder et al., 2013). Perhaps, this treatment protocol may potentially offer a relatively inexpensive treatment approach to some sufferers of these conditions that have been variously resistant to other interventions.

Possible limitations of this experiment, and further discussion, are considered in Chapter Seven.

Having completed this study the researcher turned attention back to the Case Studies (Chapter One), and the anedoctal reports by so many patients, of associated signs and symptoms in CLBP and CSP patients indicating zinc deficiency, gastro-intestinal and urinary dysfunction amongst other problems. With all 63 CLBP/CSP participants in the above study having MTrPs in the IMC, as had those in the case studies, the researcher designed a study to investigate these reportedly problematic associated signs and symptoms.

This research is contained in Chapter Six.

CHAPTER SIX: The role of the iliopsoas muscle complex in associated signs and symptoms in chronic low back pain and chronic spinal pain

6.1 Abstract

Empirical signs and symptoms including zinc deficiency, gastrointestinal, and urinary symptoms have received limited attention in the literature with regard to CLBP. Previous research has been undertaken on anxiety, depression and cognitive function in CLBP.

6.1.1 Objectives

To examine zinc levels, levels of depression and anxiety, gastrointestinal and urinary symptoms, and the prevalence of MTrPs in the IMC of CLBP or CSP sufferers compared to those who had never experienced spinal pain.

6.1.2 Method

Sixty-six participants (38 in CLBP and CSP group, 28 controls) underwent examination of the ten potential sites for MTrPs in the IMC. All participants were administered the Zinc Tally Taste Test (ZTTT) and completed a questionnaire regarding any reports of depression, anxiety, gastrointestinal, and urinary symptoms.

6.1.3 Results

No MTrPs were located in the IMC of the participants who had no history of spinal pain with significant incidences of MTrPs of the IMC in the CLBP and CSP sufferers. CLBP and CSP sufferers were found have significantly lower levels of zinc as assessed by the ZTTT and had a higher incidence of self-reported depression and anxiety, cognitive dysfunction, gastrointestinal, and urinary dysfunction.

6.1.4 Conclusions

Zinc deficiency, depression and anxiety, cognitive dysfunction, gastrointestinal and urinary dysfunction were more prevalent in CLBP or CSP participants. These signs and symptoms may require closer attention by clinicians.

6.2 Introduction

The previous chapter demonstrated that, in a randomised controlled trial of participants with CLBP, there was a clear association between 6 weeks of TPPR treatment to MTrPs of the IMC and an improvement in pain and disability. These findings supported the hypothesis that had emerged from the author's practice and further literature review that the IMC was a potential site for intervention in participants with CLBP.

The clinical cases from the author's practice also raised other unanswered questions, including the possibility that IMC dysfunction may potentially be associated with changes to body function outside the musculoskeletal system. In

particular, features of gastro-intestinal and urinary function appeared more prominent, and the possibility that these clinical cases may have an associated zinc deficiency was also noted. Following the encouraging findings in the first study, the author undertook designing a study that would shed more light on these clinical questions.

6.3 The aim of the study

The aim of this second study was to explore the hypothesis that, in participants with CLBP and CSP, IMC dysfunction may be associated with symptoms and signs of participants' zinc deficiency, depression and anxiety, cognitive dysfunction, gastrointestinal and urinary dysfunction.

The first component of the study was a comprehensive search and analyses of the literature to elucidate further understanding of these possible associations.

6.4 Zinc

In its pure form, zinc was identified as a metal being Number 30 on the Atomic Scale with an atomic weight of 65.39 (National Institute of Standards and Technology, reference E95). Zinc was reported as not utilised by the body in this form (Bryce-Smith and Hodgkinson, 1986).

6.4.1 Zinc Measurement

Obtaining a clinically relevant measurement of zinc has been acknowledged to be difficult. Chasapis et al., 2012 reported 85% of the body's zinc was found in muscles and bones. Whilst Sarukura et al., (2012) asserted that zinc nutrition could be assessed via zinc serum concentration, Sian et al., (1996) reported no association between plasma zinc levels and the assimilation or ingestion of zinc, while Yanagisawa (2004) demonstrated normal serum zinc levels in participants with known zinc deficiency. Hambidge (2003, p.951) stated that *"There is a compelling demand for improved zinc biomarkers"*.

At the time this study was conducted, (2004 to 2006), one of the most accurate and inexpensive methods of testing zinc status in the human body was the Zinc Tally Taste Test (ZTTT), devised by Bryce-Smith (Bryce-Smith and Hodgkinson, 1986). The ZTTT was approved by The British Pharmacopoeia in 1988 as being a quick and reliable test of zinc status.

The ZTTT was based on the essential role zinc plays in the taste system (Tamayo et al., 1978). The synthesis of gustin (a metalloprotein enzyme) by the parotid gland was regarded as zinc-dependent, and when secreted into the saliva, gustin was reported to regulate taste perception (Henkin et al., 1999). Eaton et al., (1990) referred to the ZTTT as as a diagnostic tool, widely applicable in medical practice. In comparing the ZTTT and sweat mineral analysis, Eaton et al., (1990) established a highly significant correlation between the two tests. Whilst Gruner and Arthur (2012) questioned the validity of the ZTTT, particularly for use with pregnant women, Saling et al., (2013) found a significant correlation between the ZTTT and a unique visual analogue scale, suggesting the results of the ZTTT may be enhanced with this additional measure.

The ZTTT consists of a test solution of one gram of zinc sulphate heptahydrate ($ZnSO_4$ 7-H_2O) dissolved in one litre of distilled water (Bryce-Smith and Hodgkinson, 1986). This test was not to be administered within one hour after eating, drinking, or smoking. The procedure requires 10 mls of the zinc-hydrated solution to be held in the mouth for ten seconds, after which any taste response was to be reported by the patient. The reported response is that which occurs while the solution is held in the mouth, not on swallowing (Bryce-Smith and Hodgkinson, 1986).

Four categories are to be used to interpret the taste response (Bryce-Smith and Hodgkinson, 1986):

- Category one (severe deficiency): no specific taste or other sensation is noticed, even after the solution has been kept in the mouth for about ten seconds;
- Category Two (moderately severe deficiency): no immediate taste noted, but after a few seconds a slight taste variously described as ‘dry’, ‘mineral’, ‘furry’ or (more rarely) ‘sweet’ develops;
- Category Three (mild deficiency): a definite though not unpleasant taste is noted almost immediately, tending to intensify with time;
- Category Four (normal levels): a strong and unpleasant taste is noted immediately.

If zinc status falls within a Category 1 or 2, Bryce-Smith and Hodgkinson (1986) recommended that the patient then requires liquid zinc supplementation.

6.4.2 Signs and symptoms of zinc deficiency

Zinc deficiency has long been known to exist in humans (Prasad et al., 1963; Prasad, 2013), with the Food and Nutrition Board of the United States (1974) declaring zinc an essential nutrient (Prasad, 2003). Zinc deficiency has been implicated in over 30 disease processes affecting the human body (Bryce-Smith and Hodgkinson, 1986), with one author suggesting zinc deficiency was as prevalent as iron deficiency in the Western world (Sandstead, 1995). The recommended daily allowance for zinc intake in Australia was 14 milligrams for non-vegetarian adult males, eight milligrams for non-vegetarian females increasing to 10 milligrams for pregnant or lactating females (Australian Government: Department of Health and Ageing: National Health and Medical Research Council, Nutritional Reference Values for Australia and New Zealand, 2005).

Reported signs of zinc deficiency included: dry skin, dry hair or excess hair loss, vertical ridges, white spots (Figure 6.1), or brittle nails, photosensitivity or inability to adjust from light to dark or vice versa, night-blindness, stretch marks on the skin, afternoon fatigue, sugar and chocolate cravings (Bryce-Smith and Hodgkinson, 1986). Hanstead (2000) noted an association between zinc deficiency and diminished neuropsychological performance.

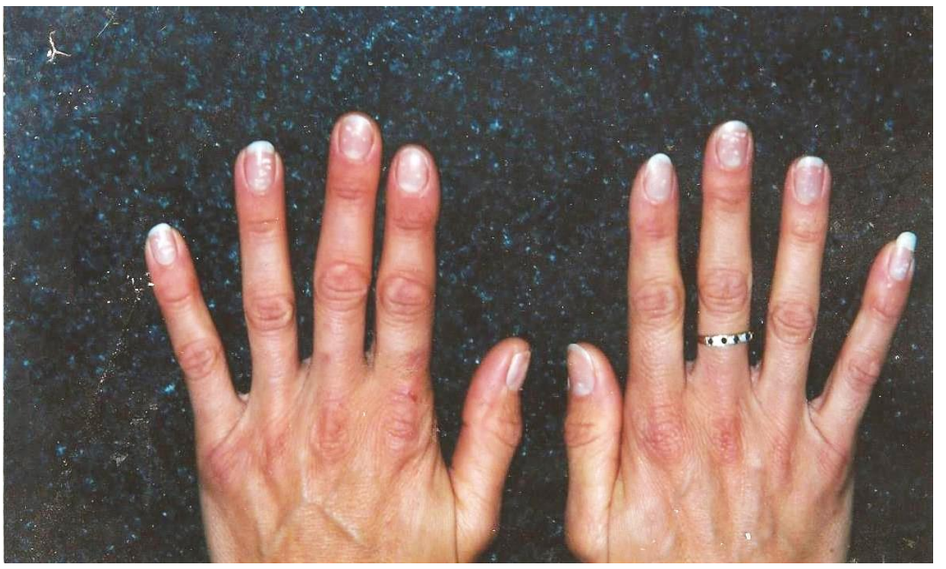


Figure 6.1. Photograph of finger nails exhibiting evidence of zinc deficiency

Zinc has previously been identified in over 300 catalytically active metalloproteins, and in excess of 2000 dependent transcription factors, reflecting key roles in many vital functions of the human body (Bryce-Smith and Hodgkinson, 1986). Zinc was deemed to be an essential trace element, important in many bodily functions including metabolism, cellular growth, and collagen synthesis (Tengrup et al., 1981; Bryce-Smith and Hodgkinson, 1986; Cabot and Jasinska, 2006; Chasapis et al., 2012). Specific metabolic functions included the metabolism of proteins, carbohydrates, and fats; reproductive and immune function; thyroid function; combating toxins; development and functioning of the CNS; essential for taste, vision, smell, and appetite; and formation of DNA and RNA (Bryce-Smith and Hodgkinson, 1986; Cabot and Jasinski, 2006; Chasapis et al., 2012).The antioxidative and anti-inflammatory properties of zinc were outlined by Prasad (2013).

Hotz and Brown (2004) suggested there were four significant causes of zinc deficiency: dietary inadequacies, impaired absorption, enhanced excretion, and compromised utilisation. Weston (2000) reported that there were limited sources of zinc available in the food chain, with depletion occurring through the use of

herbicides, pesticides, and certain fertilisers. The primary site of zinc absorption has been hypothesised to be in the proximal region of the small intestine (Lee et al., 1989; Bahl et al., 1998; Krebs, 2000; Yanagisawa, 2004). Zinc depletion has been recognised in malabsorption syndrome (Prasad, 2013), with other factors such as phytate ingestion also recognised to inhibit zinc absorption (Sandstead, 2000).

Zinc has been reported to be important in the function of the musculoskeletal system. Second only in concentration to iron in the human body, 60 % of the body's zinc was reported as being stored in muscle, and 30 % in bone (Saunders et al., 2012), being similar to the report by Chasapis et al., 2012. With zinc being required for protein synthesis, zinc deficiency was postulated to directly impair collagen formation (Bryce-Smith and Hodgkinson, 1986). Decreased bone collagen turnover was identified in the presence of zinc deficiency (Starcher et al., 1980), with Tengrup et al., (1981) demonstrating adequate zinc levels were required for collagen synthesis.

Dastych and Vlach (1990) analysed the zinc levels in muscle, hair, leucocytes and serum of 50 scoliosis participants when compared to analyses from 20 participants with spinal column complaints other than scoliosis. The findings of this analysis were that lower zinc levels were found in the spinal muscles of the scoliosis participants, but the researchers did not consider this to be a primary causation of spinal deformation. Abnormalities in collagen obtained from the musculature of scoliosis participants were reported by other researchers including Francis et al., (1976), Uden et al., (1980), and Worthington and Shambaugh (1991). Interestingly, in relation to this thesis, the IMC has been suggested as a causative

factor in scoliosis by a number of authors (Michele, 1960; Michele, 1962; Cohen et al., 1985; Advić, 2010).

6.5 Depression and Anxiety

Many researchers have made correlations between chronic pain, CLBP in particular, and psychological distress. Depression has been reported in 50 % of chronic pain sufferers (Romano and Turner, 1985; Ashburn and Staats, 1999). Similar findings of elevated levels of depression and anxiety in participants with CLBP were found in a prospective cross-sectional study by Sagheer et al., (2013). In a further study of CLBP participants, Michalski and Hinz (2006) found increased pain levels directly correlated with higher levels of depression and anxiety, while Seminowicz et al., (2011) also identified an association between CLBP and cognitive dysfunction.

In looking for possible mechanisms for these associations, several authors have suggested that fear and stress could have been causative in, or contributory, to disease processes. As far back as 1942, Williams (1942), as quoted by Sampson, (2003) stated that fear was the causative factor in most disease processes with the first obligation of any doctor being to alleviate this. Chapman and Gavrin (1999) suggested that the physiological effects of chronic pain created, and perpetuated, anxiety and disablement. Krantz et al., (2004) reported an association between stress and musculo-skeletal disorders. Esteves et al., (2013) identified dysfunction in the processing of emotions was statistically higher in participants with CLBP than those with no history of CLBP. Michalski and Hinz (2006: p.38), in a study of participants with CLBP, noted that *"fear-and depression-related behaviour"* was frequently identified in these participants.

Waddell et al., (1993) suggested that the very nature, and progression, of pain in CLBP provoked participants to fear their future capacity regarding work and physical activities. A potential causal link between pain intensity, depression and disability was suggested by Karoly et al., (2007, p.428), who further commented that *"severe pain gives rise to fear at multiple levels"*. Interestingly, the PM muscle was labelled the 'muscle of fear' in yoga philosophy and stretching of the IMC considered paramount to overall health and a general sense of wellbeing (personal communications, Smith, 2006; Koch, 2012).

There have also been suggestions in the literature of a link between zinc and psychological function, with Bryce-Smith and Hodgkinson (1986) suggesting an association between stress and zinc depletion. Cope and Levenson (2010) described an association between zinc deficiency, depression and stress, with the recommendation of zinc administration as a treatment in these disorders. Swardfager et al., (2013) reported a correlation between lower zinc levels and increased symptoms of depression in their met analysis. Siwek et al., (2013, p.1513) proposed that zinc concentrations be included in the investigation of participants suffering depression, and suggest that its action was *"anti-depressant-like in both clinical and preclinical studies".*

Thus, without there being definitive evidence, there are numerous observations in the literature of associations between CLBP, psychological distress and zinc levels.

6.6 Gastrointestinal Dysfunction

A search of the literature located only limited references linking gastrointestinal motility and LBP (Cover et al., 1983; Zeiss et al., 1987; Heaton et al., 1989; Mendoza-Lattes, 2005). Mendoza-Lattes (2005) hypothesised that gastrointestinal motility dysfunction could be the result of altered sensory information from the lumbar spine in LBP. Mendoza-Lattes (2005) reported a 2.2 times greater incidence of inhibition of the motility of the gastrointestinal tract in LBP patients when compared with shoulder pain patients, and suggested that this effect could be attributed to inhibition of overflow from the thoracolumbar plexus.

Cover et al., (1983) reported a case of compression of the ascending colon diagnosed with ultrasound imaging attributing this to enlargement of the PM muscle. Similarly, Zeiss et al., (1987) reported a case study of marked hypertrophy of the PM muscle impinging on the medial caecum and adjacent small intestine. Travell and Simons (1992) also reported a barium study of the colon demonstrating hypertrophy of the PM muscle causing compression of the adjacent large intestine.

Pinto et al., (1997) reported anatomical variations in the positioning of the ascending colon between the PM muscle and kidney in six of 428 of participants on Computerised Axial Tomography, suggesting that the capacity to identify these variations in anatomical positioning could avert erroneous diagnoses and post-interventional difficulties. Travell and Simons (1992) suggested that MTrPs in the PM muscle had the potential to be activated by the passage of a faecal bolus.

6.7 Urinary dysfunction

Urinary dysfunction was reported by 58 % (n = 22) of the participants with CLBP (Table 6.11). There were no reports of urinary dysfunction in the non-spinal pain group.

6.7 Summary of the literature findings

The literature, despite the absence of rigorous clinical studies, provided theoretical mechanisms to link CLBP due to IMC dysfunction with zinc levels, depression and anxiety, gastrointestinal symptoms and urinary symptoms. Therefore, the author determined to undertake a rigorous clinical case-control study to further investigate these potential associations.

6.8 Method

The study null hypothesis was that there was no association between CLBP, low zinc levels, depression and anxiety, cognitive dysfunction, gastrointestinal dysfunction, and urinary dysfunction.

6.8.1 Ethical requirements and undertakings

Ethics approval was gained (Appendix 12, p.238: Appendix 13, p.243), from the Flinders Medical Centre Clinical Research Ethics Committee prior to the undertaking of this study.

All appropriate and professional procedures were followed in accordance with the ethical requirements in The Physiotherapists Act (1991), and the NH&MRC

National Statement on Ethical Conduct in Research Involving Humans, with clinical responsibilities overriding research responsibilities. The researcher undertook that all information acquired during this study would be: kept strictly confidential; used only in accordance with the stated objectives; stored on a secure database; and, that hard copy would be secured in a locked filing cabinet in the Rehabilitation and Ageing Studies Unit, Flinders University for a total of fifteen years. With subsequent change in supervision, the storage site became the Department of Surgery, Flinders Medical Centre, South Australia. Participants were advised of their right to withdraw, at any time, without prejudice to their ongoing care and/or treatment. Because of the inclusion criteria used, it was anticipated that the participants would continue any medically prescribed medication and, or, treatment during the study. All assessment, treatment, and re-assessment, techniques performed were non-invasive. Palpation of the abdominal area was acknowledged as a potential source of discomfort during assessment.

6.8.2 Sample Size

The study involved two groups, one being people with CLBP, and a second group comprising people who had never suffered spinal pain. Baszanger's definition of CLBP (1990) was again applied as in the previous study (Chapter Five).

An assumption was made that there may be a 25% incidence of zinc deficiency in the control group and an incidence of 50 % in the CLBP group. On this basis, n = 58 people were required in each group (116 in total) to have a power of alpha = 0.80 to detect a statistically significant difference at p = 0.05.

Blinded interim analyses were undertaken when data on 66 participants had been collected. Based on the results of these analyses, conducted by an independent statistician at Flinders University, South Australia, the research was terminated at this point as the null hypothesis was already disproven.

6.8.3 Criteria

Inclusion criteria (Appendix Fourteen, p.244) included:

For the CLBP group:

- Age from 18 to 65 years
- The participant had CLBP for longer than six months, and
- CLBP had been medically diagnosed
- The participant may or may not have other chronic, but not acute, spinal pain in the thoracic or cervical regions

For the non-spinal pain group:

- Age from 18 to 65 years
- No history of spinal pain

Exclusion criteria for both groups (Appendix 14, p.244) for this study included:

- Being outside the age range
- Having acute, but not chronic low back pain
- Suspicion or diagnosis of osteoporosis
- A history of problems involving the aorta or vena cava (for example aneurysm)
- Taking of corticosteroids or any medication known to affect bone density
- Pregnancy

- Taking supplements containing calcium and, or, zinc.

Participants were able to withdraw from the study at any stage without prejudice to their ongoing care and, or, treatment.

6.8.4 Recruitment

Participants were recruited from within two private physiotherapy practices and one Massage/Naturopathic clinic in and around the Adelaide metropolitan area. (Appendix 15, p.245). Further participants were recruited by word of mouth. It took in excess of two years to recruit sufficient participants with no history of any spinal pain (n = 28). A total of 76 people responded to information regarding this study, each respondent receiving a Participants Information Sheet (Appendix 16, p.246) outlining the aims of, and requirements for, participation in the study. Also included in the information package was a form outlining inclusion, exclusion, and withdrawal criteria (Appendix 14, p.244). Of a total of 76 respondents, 66 entered and completed the study as outlined in Consort Flow Diagram 6.1 (refer p.140).

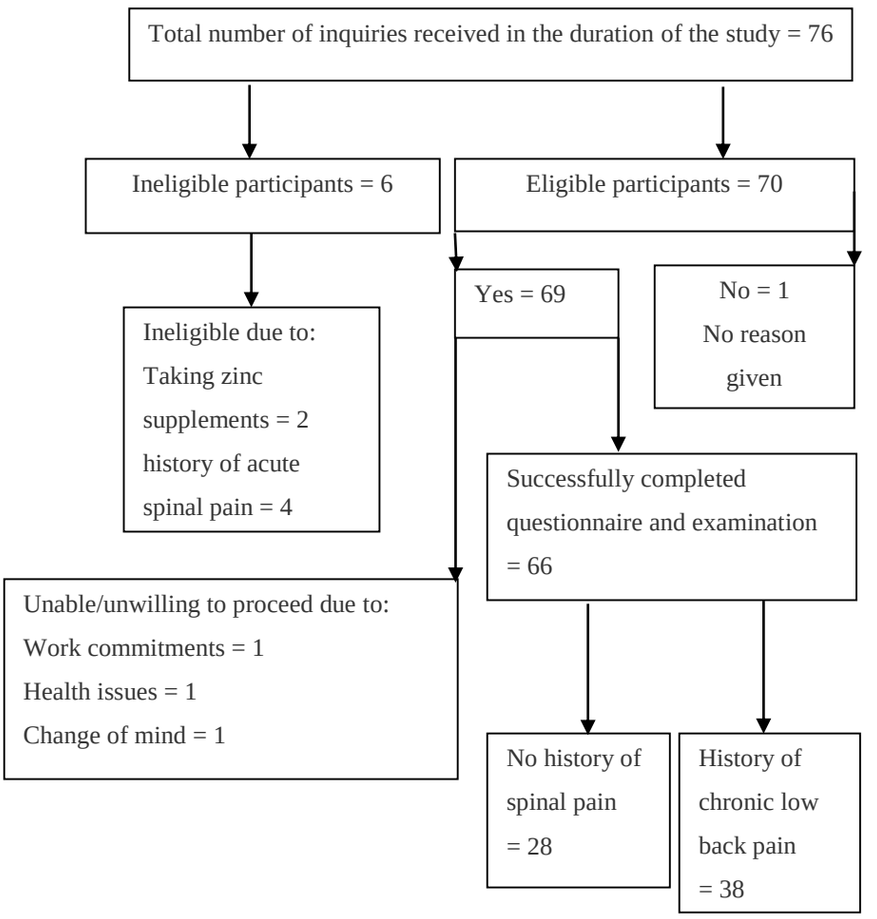
After eligibility was determined, the participant completed a Consent form witnessed by a person over the age of 18 years (Appendix 17, p.248). Of the 66 participants, 38 participants suffered CLBP, and 28 participants had no history of spinal pain.

6.8.5 Procedure

All participants were asked to complete the Patient History Questionnaire (Appendix 18, p. 250). All participants then underwent an examination of the 10

standardised MTrP sites (as used in the previous study, Chapter Five), and completed a ZTTT (Zinc Tally Taste Test) (or refer p.132).

6.1 Consort Flow Diagram: Recruitment to Completion



6.8.5 Data analyses

All analyses between the two independent groups utilised Fishers Exact test with the exception of the ZTTT in which the Chi-square test was used.

6.9 Results

6.9.1 Zinc findings

The results of the ZTTT are presented in Table 6.1 As can be seen, there was a strong association between CLBP and more severe Zn deficiency as assessed by the ZTTT ($p<0.001$). With a $\phi = 0.635$, a larger effect size was observed than was predicted in the pre-study calculations.

Table 6.1: Zinc Tally Taste Test Individual Categories 1, 2, 3, 4.

n (% rounded to whole number)		Zinc Taste Tally Test					
		CAT 1 Severe	CAT 2 Moderate severe	CAT 3 Mild	CAT 4 Normal	Total	*p**
CLBP	Group 1	22	14	2	0	38	p < 0.01
		58%	37%	5%	0%	100%	
No spinal pain	Group 2	1	9	16	2	28	
		4%	32%	57%	7%	100%	
Total		23	23	18	2	66	
		35%	35%	27 %	3.0%	100%	

* Chi-square test

As evidenced, 94.7 % (n = 36) of participants with CLBP had a zinc status in the classification of moderate or severe deficiency, while only 36 % (n = 10) in the non-spinal pain group had zinc classifications of this severity. Notably, while 58 % (n = 22) of the participants with CLBP were assessed as having severe zinc deficiency, only 4% (n = 1) of the participants without spinal pain were assessed as being in this category.

There were also significant differences between the frequency of reported signs and symptoms, identified in the literature, as being associated with zinc deficiency in the two groups of participants (Table 6.2). All questions asked showed significant associations, with the exception of stretch marks and impaired smell.

Table 6.2: Signs and symptoms that may be associated with zinc deficiency in the spinal and non-spinal pain groups

Total (n = 66)	CLBP Group 1 (n = 38)		Non spinal pain Group 2 (n = 28)		Fishers Exact test (*p*)
	Yes	No	Yes	No	
Impaired taste	7 (18%)	31 (82%)	0 (0%)	28 (100%)	0.01
Impaired smell	9 (24%)	29 (76%)	2 (7%)	26 (93%)	0.07
Stretch marks	13 (34%)	25 (66%)	7 (25%)	21 (75%)	0.29
Skin problems	12 (32%)	26 (68%)	3 (11%)	25 (89%)	0.04
Dry skin	27 (71%)	11 (29%)	2 (7%)	26 (93%)	0.00
Dry hair/excess loss	23 (61%)	15 (39%)	0 (0%)	28 (100%)	0.00
Brittle nails	21 (55%)	17 (45%)	3 (10%)	25 (90%)	0.00
White spots in nails	28 (78%)	8 (22%)	7 (26%)	20 (74%)	0.00
Vertical ridges in nails	36 (95%)	2 (5%)	8 (29%)	20 (71%)	0.00
Photosensitivity/ night blindness	33 (87%)	5 (13%)	1 (4%)	27 (96%)	0.00
Afternoon fatigue	32 (84%)	6 (16%)	5 (18%)	23 (82%)	0.00
Sugar or chocolate cravings	27 (71%)	11 (29%)	8 (29%)	20 (71%)	0.00

Participants were questioned about factors known to impact adversely on zinc status with regard to uptake and depletion. No significant differences were identified in the consumption of cigarettes and alcohol between the two groups (Table 6.3).

Table 6.3 Smoking and daily alcohol consumption

	CLBP Group 1 n (% rounded to whole number)		Non-spinal pain Group 2 n (% rounded to whole number)		Fishers Exact test (*p*)
	Yes	No	Yes	No	
Smoking	9 (24%)	29 (76%)	6 (21 %)	22 (79%)	n = 56 *p* = 0.53
Daily alcohol consumption	7 (18%)	31 (82%)	5 (18%)	23 (82%)	n = 56 *p* = 0.63

Participants were asked if they were taking any of a number of medications known to have an adverse effect on zinc status. These medications included anticonvulsants, laxatives, oral contraceptives, hormone replacement therapy, antacids, steroids, and diuretics. There were no statistical differences between the two groups in the ingestion of medications known to adversely impact on zinc status using Fishers Exact test (Table 6.4).

Table 6.4: Medications adversely affecting zinc status

	CLBP n (% rounded to whole number)		Non-spinal pain n (% rounded to whole number)		Fisher Exact test (*p*)
	Yes	No	Yes	No	
Taking medications adversely affecting zinc status	27 (71%)	11 (29%)	24 (86%)	4 (14%)	0.13

6.9.2 Myofascial Trigger Point findings

As an assessment of the presence or absence of IMC dysfunction, all participants in both groups were examined to assess the status of 10 standardised MTrPs. All 66 participants were right hand and leg dominant, with MTrPs being examined, as outlined in the previous study (Chapter Five) sequentially from superior to inferior (one to five). The data presented in Table 6.5 revealed no active, or latent, MTrPs were detected in the participants in the group without spinal pain. All participants in the CLBP group had at least three active or more latent trigger points.

Table 6.5: Trigger point prevalence data in spinal pain and non-spinal pain participants

Total (n = 66)		CLBP n (% rounded to whole number)	Non-spinal pain n (% rounded to whole number)
Right trigger point 1	Absent	1 (3%)	28 (100%)
	Latent	12 (31%)	0 (0%)
	Active	25 (66%)	0 (0%)
Right trigger point 2	Absent	1 (3%)	28 (100%)
	Latent	12 (31%)	0 (0%)
	Active	25 (66%)	0 (0%)
Right trigger point 3	Absent	6 (16%)	28 (100%)
	Latent	21 (55%)	0 (0%)
	Active	11 (29%)	0 (0%)
Right trigger point 4	Absent	5 (13%)	28 (100%)
	Latent	19 (50%)	0 (0%)
	Active	14 (37%)	0 (0%)
Right trigger point 5	Absent	1 (3%)	28 (100%)
	Latent	10 (26%)	0 (0%)
	Active	27 (71%)	0 (0%)
Left trigger point 1	Absent	0 (0%)	28 (100%)
	Latent	12 (32%)	0 (0%)
	Active	26 (69%)	0 (0%)
Left trigger point 2	Absent	0 (0%)	28 (100%)
	Latent	17 (45%)	0 (0%)
	Active	21 (55%)	0 (0%)
Left trigger point 3	Absent	3 (8%)	28 (100%)
	Latent	23 (61%)	0 (0%)
	Active	12 (31%)	0 (0%)
Left trigger point 4	Absent	3 (8%)	28 (100%)
	Latent	22 (58%)	0 (0%)
	Active	13 (34%)	0 (0%)
Left trigger point 5	Absent	0 (0%)	28 (100%)
	Latent	13 (34%)	0 (0%)
	Active	25 (66%)	0 (0%)

Figure 6.2 Trigger point prevalence in spinal pain and non-spinal pain participants

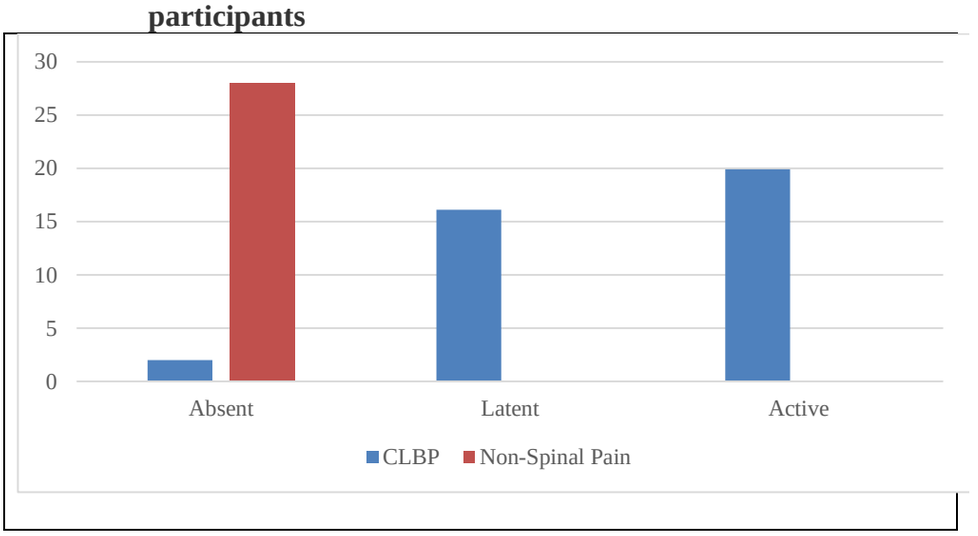


Table 6.6 Trigger point prevalence averages

Chronic spinal pain				No spinal pain			
Trigger point	Absent	Latent	Active	Trigger point	Absent	Latent	Active
1	1	12	25	1	28	0	0
2	1	12	25	2	28	0	0
3	6	21	11	3	28	0	0
4	5	19	14	4	28	0	0
5	1	10	27	5	28	0	0
6	0	12	26	6	28	0	0
7	0	17	21	7	28	0	0
8	3	23	12	8	28	0	0
9	3	22	13	9	28	0	0
10	0	13	25	10	28	0	0
Total	20	161	199	Total	280	0	0
Average	2	16.1	19.9	Average	28	0	0

	Absent	Latent	Active
CLBP	2	16.1	19.9
Non-Spinal Pain	28	0	0

6.9.3 Depression, anxiety/agitation, mood swings, impaired concentration, word finding difficulties and word transposition

Participants were asked to self-report the presence or absence of depression, agitation/anxiety (without apparent cause), mood swings, impaired concentration, and word finding difficulties/word transposition (Table 6.6). There was a significant association between participants reporting each of these symptoms and the presence of CLBP in this study group (Fisher's Exact Test $p < 0.001$). There was a strong effect size with a phi = 0.599.

Table 6.7 Depression, anxiety/agitation, mood swings, impaired concentration, word finding difficulties/word transposition

Total n = 66	Chronic spinal pain n (% rounded to whole number)		No spinal pain n (% rounded to whole number)		Fishers Exact test (*p*)
	Yes	No	Yes	No	
Depression	18 (47%)	20 (53%)	2 (7%)	26 (93%)	0.000
Anxiety/ agitation	23 (61%)	15 (9%)	3 (11%)	25 (89%)	0.000
Mood swings	20 (53%)	18 (47%)	3 (11%)	25 (89%)	0.000
Impaired concentration	22 (58%)	16 (42%)	2 (7%)	26 (93%)	0.000
Word finding difficulties/ word transposition	23 (61%)	15 (39%)	4 (14%)	24 (86%)	0.000

6.9.4 Gastrointestinal findings

Participants were asked to report the presence or absence of any problems in relation to their current gastrointestinal function (Appendix 18, refer p.250).

Table 6.8 shows that 66 % (n = 25) of the participants in the CLBP group reported gastrointestinal dysfunction, with eight participants having sought medical advice for the problem. No participant in the non-spinal pain group reported any gastrointestinal dysfunction.

Table 6.7: Gastrointestinal function

			Changes in gastrointestinal function			
			Yes n (% rounded to whole number)	No n (% rounded to whole number)	Total	Fishers Exact test (*p*)
CLBP	Group 1	n = 38	25	13	38	<0.0001
			66%	34%	100.0%	
No spinal pain	Group 2	n = 28	0	28	28	
			0%	100.0%	100.0%	
Total		n = 66	25	41	66	
		%	38%	62%	100.0%	

6.9.5 Urinary findings

Urinary dysfunction was reported by 58 % (n = 22) of the participants with CLBP (Appendix 18, p.250) (Table 6.9). There were no reports of urinary dysfunction in the non-spinal pain group.

Table 6.9 Urinary function

			Changes in urinary function			Fishers Exact test
			Yes n (% rounded to whole number)	No n (% rounded to whole number)	Total	(*p*)
CLBP	Group 1	n = 38	22	16	38	<0.0001
			58%	42%	100.0%	
No spinal pain	Group 2	n = 28	0	28	28	
			0%	100.0%	100.0%	
Total		n = 66	22	44	66	
		%	33%	67%	100.0%	

6.10 Summary

This case-control study identified significant differences between the group of participants who had medically diagnosed as having CLBP and CSP, and the group of participants who had never experienced spinal pain. Participants suffering CLBP and CSP reported lower reactions to the ZTTT suggesting depleted zinc levels, recorded higher self-reported levels of depression, anxiety, mood swings, impaired concentration, word finding difficulties/word transposition, and reported higher levels of gastrointestinal and urinary

dysfunction than the group of participants who had no spinal pain. All participants with CLBP and CSP (n = 38) had active or latent MTrPs identified in the IMC, with none of the participants without any history of spinal pain had no MTrPs detected.

CHAPTER SEVEN: Summation

7.1 Discussion

Six case series from the researchers' patient population, identified as being representative, were reported in Chapter One where initial complaints were low back pain or pain in the inguinal region. Commonalities in findings were MTrPs in the IMC, signs and symptoms of zinc deficiency, reported cognitive dysfunction, and gastrointestinal and urinary dysfunction. Observations obtained in the researchers' clinical practice led to this thesis to find out whether there was solid evidence on the possible link between MTrPs in the IMC and back pain, and whether myofascial treatment could alleviate symptoms.

Two systematic searches of the literature were undertaken, one year apart, to identify previous studies examining the role of the PM muscle/IMC in CLBP. Results rendered only one study utilising TPPR in CLBP, although a second study reviewed has been entered as participants suffered both LBP and CLBP. The muscles treated in both studies were not identified, and it is not able to be ascertained whether the IMC was included and treated, therefore.

A single-blinded randomised controlled trial, identified improvements in the outcome on pain scores and function at six weeks in people suffering CLBP or CSP when a protocol of TPPR and stretching is used, compared to the group who undertook stretching (Chapter Five). As no other studies were identified investigating the role of TPPR on MTrPs in the IMC, this is considered original research with findings contributing to the body of knowledge in this field. The measures used pre and post interventions were the Short-Form Pain Questionnaire

to assess pain levels (Melzack, 1987), and the Patient Specific Disability Measure (Stratford et al., 1995) to evaluate the effects of interventions on ADL. These evaluation measures were chosen to enable the participants' to identify their self-perception of their pain and function, with these measures identified as being objective and valid. Also, any outcome measure is required to be sensitive enough to reflect and record relevant changes in reported symptoms (Gridley and van den Dolder, 2001).

When comparing the two groups, significant improvements were found in the group who received TPPR with self-administered stretching of the IMC. There was a reduction in the number of active MTrPs together with an increase in the absence of MTrPs in this group. This group also recorded lower pain scores on the Short-Form Pain Questionnaire (Melzack, 1987) and achieved improved ADL, and function, on the Patient Specific Disability Measure (Stratford et al., 1995) at cessation. Smaller improvements were also gained with a protocol of stretching. These results are in line with a previous randomised controlled clinical trial of plantar heel pain (Renan-Ordine et al., 2011). The group which received myofascial trigger point therapy had significant reduction in pain, and improved function, with a protocol of TPPR and with a self-administered stretching, over stretching alone, at the completion of a four week study of (Renan-Ordine and colleagues, 2011). These results may indicate that a protocol of TPPR to MTrPs in the IMC, in combination with stretching of the IMC, alleviated or resolved CLBP or CSP for some participants by the cessation of the study.

Additionally, this study may potentially enhance the investigation of CLBP via the examination and identification of MTrPs in the IMC in people with CLBP and

CSP.

The findings of a second primary study in this thesis presented in Chapter Six may advance understanding of associated signs and symptoms reported by people experiencining CLBP and CSP. This study investigated the role of the IMC in development, or maintenance, of the associated signs and symptoms identified by CLBP and CSP participants. The focus is on zinc deficiency, depression, anxiety/agitation, mood swings, impaired concentration, word finding difficulties/word transposition, and gastrointestinal and urinary dysfunction. It is hypothesised that these signs and symptoms may be associated with MTrPs in the PM muscle. This hypothesis is based on one of the noted effects of an MTrP being the shortening of an affected muscle (Travell and Simons, 1983). This shortening in the PM muscle could potentially provoke stimulation of the thoracolumbar sympathetic plexus, with the participation of the autonomic system in MTrPs having previously been identified (Dommerholt et al., 2006).

The lower levels of zinc and higher levels of depression identified in this study, correlated with previous findings of zinc deficiency linked to depression and stress (Cope and Levenson, 2010). The self-reported incidence of depression and anxiety, in the CLBP and CSP group correlated closely with previous findings (Romano and Turner, 1985; Ashburn and Staats, 1999; Michalski and Hinz, 2006; Sagheer et al., 2013). The attributable cause as to the lower levels of zinc found in people with CLBP and CSP is unable to be ascertained as any stressor is identified as instigating zinc deficiency (Bryce-Smith and Hodgkinson, 1986).

Significant differences in zinc levels were identified between the CLBP and CSP group, and non-spinal pain group, on administration of the ZTTT. In the CLBP

and CSP group zinc deficiency is identified as being at a statistically higher incidence, as are reports of depression and anxiety. Although no participant in the non-spinal pain group identified gastrointestinal or urinary dysfunction, a significant number of people in the CLBP and CSP group reported these symptoms.

7.2 Limitations of this research

With the study described in Chapter Five being a single-blinded clinical trial some of the participants were cognisant of which group they had been allocated to as previously detailed (refer p.97). An acknowledged weakness, also, is the stretching group did not receive the same level of care as the intervention group, potentially biasing results toward the intervention group. It appears from the results, however, that the protocol of TPPR and self-administered stretching rendered encouraging outcomes at cessation of the study. Follow up was only conducted immediately after the cessation of the research but with future studies to include longer term follow-up to assess the ongoing effects of the intervention.

The strength of the results suggest that further studies to overcome these limitations maybe of value in contributing to the body of knowledge regarding CLBP. Larger studies would enable analysis of the effect of this intervention on different sub-groups of participants with CLBP. Such studies could also be double-blinded and undertake longer follow up periods to provide further evidence in this area.

Study Two, Chapter Six, reflects participants recruited from three private practices in the Adelaide region. The ZTTT, as described previously, has been

supported by a number of researchers as an appropriate test of zinc status. However, this study cannot determine for how long the current zinc status has been present, and as such, cannot make any inferences about causality. Neither can the study differentiate the relative potential contributions to zinc status of diet, absorption, pain, and other neurological factors.

The study used a group of ten standardised sites to detect MTrPs. Whilst these results do not exclude the possibility that participants without spinal pain may have had other active or latent MTrPs related to the IMC present at other sites, the significant differences between the two groups indicate a strong association between the presence of CLBP and latent or active MTrPs in sites known to be associated with IMC dysfunction.

The study used self-reporting of symptoms by the participants, reflecting patients' perceptions and relative understanding of what are sometimes technical medical terms. Given the large effect size found in this study, further research using more formalised diagnostic tools for the psychological, gastrointestinal, urinary and zinc deficiency symptoms would appear worthwhile.

7.3 Implications for policy and practice

The costs of CLBP are both financially and personally high, with the impact spreading to the Community in terms of the burden placed on government (American Academy of Orthopaedic Surgeons, 1988; American Academy of Orthopaedic Surgeons, 2004; Canale, 2009). The apparent short-term effectiveness and relative simplicity of the treatment used in this study raises some relevant economic issues. A total of 12 TPPR treatments were delivered to

active MTrPs within the IMC in treatment group in the first RCT study. The cost of these twelve sessions and teaching of a self-management programme, as performed in this study, would have been AUD 600 in a typical private practice at the time this study was conducted. The prevalence estimates of CLBP varied from 0.01 to 10% of the Western world populations (Nachemson, 1994; Fraser, 1998). Although not all back pain is likely to be related to the IMC, even if the highest prevalence rate is taken and every person in the USA received this treatment, the cost would be in the realm of 18 billion dollars. This compares to the current estimated for LBP being USD100 to 200 billion per annum in the US in regard to direct and indirect costs (Carey and Freburger, 2014).

The findings in this thesis have potential implications for clinical practice. These findings may contribute to the evidence base for practitioners dealing with CLBP sufferers. It could benefit clinicians to have these findings translated into clinical guidelines for safe and effective practice, as the findings of this thesis offer evidence that may inform diagnosis and treatment of CLBP and CSP that have continued to be perplexing disorders, and that are frustratingly resistant to various treatments.

In addition to larger and stronger studies, and studies focused on investigating whether the associations demonstrated in this study are indeed causal, this research suggests many areas for further investigation in relation to the role of the PM muscle and IMC in musculo-skeletal conditions. These conditions include participation in acute and subacute LBP, and other chronic pain conditions that have remained elusive to resolution. The capacity for the IMC to spread pain from the lumbar spine to other areas of the spine and periphery, as suggested by Michele, 1962; Michele, 1971; Travell and Simons, 1992 warrants future studies.

Although not flowing directly from theses studies, future studies may investigate the proposed role that MTrPs in the IMC may potentially contribute to osteoarthritis of the hip, and medial compartment syndrome of the knee, as the result of altered bio-mechanics. The hypothesised effects on lower limb joints are based on the effect of antero-inferior torsion of the hemi pelvis, in the presence of MTrPs in IMC, thereby creating compression of the head of the femur into the acetabular cup, and excessive force on the medial compartment of the tibio-femoral joint. Early treatment of the PM muscle and IMC, prior to the alteration in pelvic positioning and altered biomechanics, could prevent the spread of pain to sites well beyond the lumbar spine. Hamstrings, knee and low back problems are common in sports medicine and it is therefore urged that recognition be focussed on the potential role of the PM muscle and IMC in these conditions.

In the field of Paediatric Medicine, infantile, juvenile and adolescent idiopathic scoliosis is a further area of research that may be enhanced on the basis of findings from this study. The prevalence of adolescent idiopathic scoliosis is estimated to be between two and four percent of children aged ten to sixteen years (Reamy and Slakey, 2001). While genetic factors have been implicated (Reamy and Slakey, 2001), other causes have been identified such as spasticity associated with cerebral palsy and true leg length discrepancy. However, it is a generally held consensus that the cause is unknown (the Scoliosis Association of Australia, 2013). Previously recommended interventions have been those of spinal bracing and surgery. As previously been outlined tightness of the PM muscle has been outlined as a cause of scoliosis (Michele, 1960; Michele, 1962; Michele, 1971; Cohen et al., 1985; Travell and Simons, 1992; Gerwin, 2005; Advić, 2010). Of the six case studies presented in Chapter One, three had radiographic evidence of scoliois (refer case studies one, two, and three). In case study three radiographic

evidence of improvement in roto-scoliosis, after TPPR, is demonstrated radiographically. Specifically, it has been stated that MTrPs in the *'psoas'* can cause scoliosis (Gerwin, 2005, p.126). In scoliosis sufferers MTrPs are commonly identified in the IMC with a protocol of TPPR and self-administered stretching previously demonstrated improvement in this condition in the researcher's practice. Future research into idiopathic scoliosis may add to this body of knowledge.

One area that could be included in such guidelines could be more appropriate ordering of X-rays to assist diagnosis. The most common request for X-rays is that they be taken in the supine position thereby negating the effect of gravity on the postural muscles (personal communications, McKay, 1999). A series of X-rays in supine and erect postures were undertaken with the results demonstrating X-rays performed in the supine evidenced correct anatomical alignment, and X-rays of the same patient taken in the erect position demonstrating scolioses. This study, and the experience of the researcher, suggests that radiological assessment of the spine in both supine and erect postures could be helpful in reporting rotations, elevations and other deviations from all planes and axes. The greater value of weight-bearing, as opposed to horizontal, X-rays of the spine in identifying postural and structural abnormalities is reported by Inklebarger and Clarke, (2015).

A further area to re-address, in clinical guidelines, is the recommended seating posture by Occupational Health and Safety of the so called 90-90-90 position of the hips, knees, and ankles. This posture places the PM and iliacus muscles in a relatively shortened position as previously noted (Travell and Simons, 1992). Pain is provoked by alteration of tension in a muscle containing an MTrP, with the

affected muscle being *functionally shortened* (Simons et al., 1999, p.113). This may be an aggravating factor in patients with LBP, CLBP, and CSP, particularly when moving from the seated to the erect position since the IMC is required to rapidly elongate. Prescribing a footstool may have a compounding effect since it tends to shorten the PM muscle even more and, therefore, it is recommended that this practice be considered for review.

7.4 Conclusion

This thesis has presented a comprehensive overview of possible relationships between MTrPs in the IMC and symptoms of CLBP and CSP.

The prospective studies have provided new evidence that treatment of TPPR applied to MTrPs in the IMC may offer a cost-effective treatment to reduce pain, at least in the short-term, and significantly improve ADL and function, for those suffering CLBP and CSP. Appropriate identification and treatment of active or latent MTrPs in the IMC may improve pain and quality of life, reducing financial and psychosocial costs currently threatening to overwhelm individuals, governments, and compensation systems.

This evidence may have significant implications for policy and practice. The IMC is referred to as ‘Hidden Prankster’ (Travell and Simons, 1992), although the research suggests it may be more appropriate to refer to it as the ‘Hidden Culprit’. The extent of its role in CLBP and CSP, dysfunction in the adjacent lumbar region, and pelvic organs, and wider impact on the wider musculoskeletal system can now be identified through further research. Given the high prevalence of these disorders, and associated costs, the appropriate translation of this evidence into

clinical guidelines that are readily available to all may assist in alleviating the suffering of many who experience CLBP.

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APPENDICES:

Appendix One

FLINDERS MEDICAL CENTRE/FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

FLINDERS CLINICAL RESEARCH ETHICS COMMITTEE

1. Project Title	"The connection between the iliopsoas muscle complex and chronic spinal pain"
2. Investigator Details: Neil Piller – Professor Course Co-ordinator MSc (PHC) Dept of Public Health School of Medicine G5 Flats, Flinders medical Centre Bedford Park 5042 T/Leader Lymphoedema Assessment Clinic Flinders Medical Oncology Unit Flinders medical Centre Bedford park S.A. 5042	Ms Aileen Jefferis Student in Masters of Clinical Rehabilitation (Research based) Flinders University Telephone (08) 83760748 <u>Qualifications</u> Diploma of Physiotherapy (NZ), Otago Polytechnic, 1976. Graduate Diploma in Social Science (Rehabilitation) University of South Australia, 2000.
3. List of places research is being undertaken:	This project is a study being undertaken by a student from Flinders University (Masters of Clinical Rehabilitation – Topic Co-ordinator- Prof Maria Crotty) and supervised by Prof Neil Piller from the Department of Public Health at Flinders University. Recruitment of study participants will take place via advertisement/editorial in the Messenger Newspaper and Private Practitioners (allied health). Ethics approval is sought from Flinders Clinical Research Committee.
4. Project details:	The iliopsoas muscle complex is herein referred to as the IMC. Pain, as defined by the International Association for the Study of Pain is *"An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. The inability to communicate in no way negates the possibility that an individual is experiencing pain and is need of appropriate pain relieving treatment"* [1] While chronic back pain is widely reported in the medical and epidemiological literature with specific reference to the low

back, the definition of chronic back pain remains contentious with no generally agreed criteria being accepted[2].

Definitions range from pain: lasting longer than 7 – 12 weeks[3]; greater than 6 months duration without objective findings[4]; prolonged beyond three months[5]; defined by its length (more than six months) and its resistance to conventional therapies[6]. Estimates of incidence vary widely also, ranging from 0.1% to 105 of the population[7].The costs emotionally and psychosocially are incalculable[8]. Financial costs have been estimated at $100 Billion U.S. in the most up-to-date figures available[9;] it is estimated as being responsible for a major portion of Workcover costs in Australia[10].

It is recognized that there is only a weak correlation between demonstrated pathology and chronic back pain[4,11,12,13] that physical and laboratory measures of symptoms or function and commonly used physical tests of muscle strength and range of motion correlate poorly with actual patient behaviour[14]. Further the subjective experience of pain can poorly match quantifying criteria[15]. Various treatment approaches ranging from therapeutic exercise regimes[16,17] to surgery[18,19] have failed to show positive results in alleviating, or significantly altering chronic spinal pain.

Surgical intervention, in some instances, has been shown as counter-productive[20,21] with spinal fusion being complicated in 4 to 6% of cases by nerve root damage, usually as the result of pedicle screw malplacement[22]. Comparisons of spinal cord stimulation, versus spinal re-operation, (both invasive techniques) have demonstrated spinal cord stimulation is more effective in alleviating chronic spinal pain[23].

Five references, only, could be found suggesting any connection between low back pain, upper back, and cervical pain which implicated iliopsoas in this mechanism[24-29]. One study of six failed low back pain participants outlined improvement with injection of the IMC[29].

Rationale

	Skeletal muscle comprises 40% of the human body and yet still fails to attract attention as being a source of pain [24]. The existence of MTrPs, in skeletal muscle has been the source of contention[30]. Evidence with substantial research bases, now demonstrates the existence and effects of MTrPs, being the shortening, tightening and weakening of affected skeletal muscle/s[31-35] with the potential to cause pain directly, or indirectly. Two muscle complexes span the area from the occiput to the lesser trochanter of the femur[27]. The trapezius muscle attaches to the superior nuchal ridge, clavicle, acromion, spine of the scapula, and all vertebrae from cervical one to thoracic twelve. The psoas muscles attach to the vertebrae, and intervertebral discs of thoracic twelve to lumbar five. Its attachments are anterior to the axis of rotation of movement. The iliacus component, of the complex, arises from the superior inner surface of the iliac fossa. Conjointly, the psoas and ilacus insert on the lesser trochanter of the femur to form the IMC. The IMC is a mammalian muscle, functionally lengthened to position the hip joint at +/- 100º in all mammals excepting humans in which the functional length is 180º across the hip joint. This has created an increased susceptibility to overload and dysfunction, through maladaption[25,27]. The IMC participates in all static and dynamic postures with the exception of lying[24]. The sitting posture shortens the IMC[24]. The so described epidemic of back pain coincides with increased sitting times and activities. ▪ The effects of myofascial trigger point activity in the IMC result in:shortening of the muscle/s ▪ the lumbar spine being pulled anteriorly thus increasing compressive forces on the zygoaphysial joints and intervertebral discs ▪ ipsilateral concave scoliosis ▪ anterior and inferior rotation of the ilium ▪ superior movement of the sacral one to the lumbar five vertebrae ▪ rotation of the lumbar five=/- four vertebra/e. Successful outcomes have been achieved in chronic back pain treatment[24].

	A requirement for normal movement, is horizontal eye position; something not achievable in the presence of a lumbar scoliosis, unless there is a compensatory scoliosis in the contralateral thoracic spine. A counter rotation in the contralateral thoracic spine is necessary, also, to counteract pelvic rotation. This restores frontal positioning required for forward motion. These compensatory movements and postures reflect muscle contraction, hypothesised as being initiated by the IMC via its conjoint attachment to the thoracic twelve vertebra. It is further hypothesised that this is the cause chronic spinal pain due to compression and torsion of pain sensitive structures in the spine and the spread of pain from the low back upwards. **Objectives** The aim of this research project is to evaluate the effects on chronic spinal pain with treatment of the IMC. The project will focus on the objective and subjective experiences of the participants and the relationships of these to functionality.
	Study Design A randomised clinical trial involving 106 spinal pain participants 106 participants will be randomly assigned to one of two groups, treatment or control. The treatment group will receive twice weekly treatments to identified MTrPs in the IMC, along with a self-managed stretching regime. The control group to receive a self-managed stretching regime which will be taught after randomisation. **Duration of the study** Planning: 2 months Recruitment & Intervention: 6 months Statistical analyses and thesis writing: 3 months Total: approximately 11 months.
	Selection of participants Over approximately six months a total of 106 chronic spinal pain participants will be recruited from: ▪ an advertisement /editoral will be placed in the Messenger newspaper ▪ private allied health practitioners will be contacted via mail with a request to assist in recruitment for

	this study with a full explanation offered as to the aims (see Appendix 4). The inclusion and exclusion criteria will be supplied to private practitioners in order that they may identify potential participants (see Appendix 3). The researcher will further assess suitability and, when appropriate, invite the patient to participate in the study. Written consent will be obtained prior to patient participation (see Appendix 6). **Inclusion criteria** Chronic spinal pain • low back only • low back plus thoracic or cervical or both which has been • medically investigated • of six months or longer duration • non-responsive to conventional treatment/s Age range 18 to 65 years. **Exclusion criteria** • suspicion or diagnosis of osteoporosis • history of problems involving the aorta or vena cava • the taking of corticosteroids or any medication known to affect bone density • any other medical condition in which bone integrity may be compromised by the application of gentle isometric contraction, including spinal fusion. **Withdrawal criteria** Participants may withdraw from this study at any stage without prejudice to their ongoing care. **Statistical Analysis** a) Sample size = 106 b) Analysis of results: Two independent samples will be compared. Sample size calculations are based on an estimated mean difference of 1.8000 and a within group deviation of 3.250. The test of equality of means will be carried out at the .50 level of significance. A sample size of 53 per group gives a probability of .806 of rejecting the null hypothesis of equal means if the alternative holds.
6. Drug Profile	Not applicable
7. Procedures, including drug treatment involving the subject:	Drug treatment: Not applicable. As the inclusion criteria includes chronic spinal pain which has been resistant to conventional treatments, it is anticipated that concurrent treatment will consist of medically prescribed

	medication or any other medically prescribed or recommended method for pain relief such as T.E.N.S. devices. The treatment group (n = 53) will have individually prescribed bilateral treatment of the IMC which will include: ▪ a coolant spray and gentle (10%) isometric exercise technique ▪ gentle acupressure ▪ a combination of the above ▪ + a self managed stretching regime (identical to that prescribed to the control group) which will be taught on the first treatment appointment. Instructions, and a diary to record daily details of stretching will be supplied at this time, also to allow an assessment of compliance (see Appendix 10) ▪ visits will be twice weekly for a total of six weeks: each visit will be of approximately half an hours' duration and will include treatment, administration of surveys and measurements.
8. Assessment of Participants	**Laboratory**: not applicable. **Radiological**: any recent radiological investigations (plain X-rays, CAT scans, MRI's) will be reviewed if available. No further radiological investigations will be requested. **Clinical**: Measurements to be utilised will be: ▪ Personalised questionnaire (see Appendix 7) ▪ Short Form of the McGill Pain Questionnaire at baseline and six weeks (see Appendix 8) ▪ Patient Specific Disability Measure administered weekly (see Appendix 9) ▪ Weight, and height measurements using a portable height stick will be recorded at baseline, pre and post treatment (for the treatment group) at six weeks ▪ Palpation of the IMC to ascertain the presence of MTrPs will be undertaken at baseline and six weeks. Spinal range of motion will not be evaluated due to the risk of exacerbation of patient symptoms. Blinded outcome assessment (at six weeks) will be conducted by a trained physiotherapist (see Appendix 11). **Monitoring adverse effects.** The treatment techniques to be utilised are non-invasive. Some discomfort, or shift in pain patterns, may occur due to alteration of biomechanics. The participants will be informed of this in the Participation Information Sheet (see Appendix 5). **Significant adverse effect/s.** While no significant adverse effect/s are anticipated, should this situation arise they will be reported to the FCREC.

9. Administrative Aspects.	**Source and details of funding.** No funding has been sought for this project at this stage however we anticipate that funding may be sought to cover the costs of the blinded outcome assessor. The coolant spray will be supplied at no charge by: Sun Medical, South Road, Clovelly Park. **Maintenance of records.** In accordance with sound clinical practice principles, records will be documented at each visit for the treatment group. These will include: ▪ Date of visit ▪ Reassessment details ▪ Other relevant information gathered. **Special facilities required.** No special facilities are required.
10. Indemnity	As a Flinders University student indemnity is offered by enrolment with this Institution. The student also has current Professional Indemnity insurance as a registered physiotherapist.
11. Consent Form.	The researcher will ensure a detailed explanation of the essence of the study. Written informed consent will be obtained from the participant prior to participation in this project (see Appendix 6).
12. Patient Information Sheet.	See Appendix 5.
13. Ethical Considerations.	**Benefits anticipated from the project.** This project will provide new knowledge as to the role of the IMC in chronic spinal pain. It is hoped that this will be the basis of further research into its role in acute pain and also significant secondary clinical problems arising from abnormalities in the IMC. **Risks.** While patient assessment procedures are non-invasive abdominal palpation of the IMC may be a source of discomfort to the patient. Discomfort, or alteration in pain patterns, may occur concomitant to alteration in biomechanics. **Research on people in Dependent Relationships.**

	No patient of the researcher will be admitted into the study. **Separation of Research and Clinical Responsibilities.** Following the Code of Ethics of the Physiotherapists Act 1991) and the NH&MRC *National Statement on Ethical Conduct in Research Involving Humans* the researcher acknowledges that clinical responsibilities override research responsibilities and will follow appropriate, professional procedures if these circumstances arise. Statement of compliance with NH&MRC *National Statement on Ethical Conduct in Research Involving Humans* has been accessed from the NH&MRC website at: http:www.nhmrc.health.gov.au/publications/pdfe35.pdf. The project complies with the NH&MRC National Statement on Ethical Conduct in Research Involving Humans.
	Source of Participants. Participants will be recruited by way of advertisement/editorial in the Messenger newspaper and via private practitioners (see Appendix 4). **Protection of privacy and preservation of confidentiality.** All information acquired during the project will be kept strictly confidential to be used only in accordance with the stated objectives. De-identified statistical data only will be supplied to a statistician for analyses. All information collected will be entered on a secure data base: hard copy will be secured in a locked filing cabinet in the Rehabilitation and Aging Studies Unit, Flinders University for a total of 15 years which is in accordance with NH&MRC guidelines.

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Appendix Two

Flinders Medical Centre
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Telephone (08) 8204 5511
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Flinders Clinical Research Ethics Committee
FWA00001785

Telephone (08) 8204 4507
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14 November 2002

MEMORANDUM

TO: Ms. A. Jefferis, c/- Ms. M. Miller, Rehabilitation & Aged Care, RGH

FROM: Ms. C. Hakof, Executive Officer, Flinders Clinical Research Ethics Committee

TOPIC: **Research Application 33/023**

I am pleased to advise that the Flinders Clinical Research Ethics Committee has approved your research application in accordance with the following extract from the Minutes of its meeting held on 11 November 2002.

5458 RESEARCH APPLICATION 33/023 – MS. A. JEFFERIS
The connection between the iliopsoas muscle complex and chronic spinal pain.
Reviewer: Dr. J. Walsh

This application was approved subject to amendments to the information sheet which have been conveyed to the investigator.

A progress report must be provided annually. Approval is given for a period of three (3) years only and, if the study is more prolonged than this, an updated submission will be required.

If conditional *('subject to' or 'in principle')* approval is granted, research involving human subjects **may proceed only after written acceptance of the conditions of approval** (including a copy of the modifications) has been received by the Committee.

If patients are involved the chief investigator is responsible for the process of notification, seeking approval or permission of Departments, Divisions or individual consultants. ***A copy of the signed consent form is to be filed in the participant's medical record.*** Please note that if this trial involves normal volunteers it will be necessary for you to keep a record of their names and you may be required to supply this list with your annual report.

You are reminded that the Flinders Clinical Research Ethics Committee must approve the content and placement of advertisements for the recruitment of volunteers.

The Committee must be notified and approve any changes (e.g. additional procedures, modification of drug dosage, changes to inclusion or withdrawal criteria, changes in mode and content of advertising) in the investigational plan particularly if these changes involve human subjects.

The safe and ethical conduct of a trial is entirely the responsibility of the investigators. While the Flinders Clinical Research Ethics Committee takes care to review and give advice on the conduct of trials, approval by the Committee is not an absolute confirmation of safety, nor does approval alter in any way the obligations and responsibilities of investigators.

It is the duty of the chief investigator to give prompt notification to the Flinders Clinical Research Ethics Committee of matters which might affect continued ethical acceptability of the project, including:

1. Adverse effects of the project on participants, including the total number of participants recruited, and of steps taken to deal with these adverse effects.
2. Other unforeseen events.
3. A change in the base for a decision made by the Committee, e.g. new scientific information that may invalidate the ethical integrity of the study.

C. Hakof

The Flinders Clinical Research Ethics Committee is constituted and operates in accordance with the National Health and Medical Research Council's National Statement on Ethical Conduct in Research involving Humans (June 1999).

Appendix Three

Inclusion criteria:

- Chronic spinal pain: lumbar only; lumbar plus thoracic or cervical or both, being of six months, or greater duration; medically diagnosed; and with failure to respond to conventional therapy or therapies
- Age range 18 to 65.

Exclusion criteria:

- Outside the designated age range
- Diagnosis of osteoporosis
- The taking of corticosteroids (known to affect bone density)
- Any other medical condition in which bone integrity may be compromised by the application of gentle isometric contraction (including spinal fusion)
- A medical history of problems involving the aorta (for example an aneurysm) or the vena cava
- Pregnancy.

Withdrawal criteria:

Participants may withdraw from the study at any stage without prejudice to their ongoing care and/or treatment.

Appendix Four

Aileen S Jefferis
Rehabilitation & Ageing Studies Unit

Student in:
Masters Clinical Rehab (Research)

Contact Phone No: 0418-784-753

Dear Practitioner,

Re: Flinders University based Research project entitled:
"The role of the Iliopsoas muscle complex in chronic spinal pain"

As a component of my research based Masters of Clinical Rehabilitation I am undertaking a randomised controlled clinical trial as to the role that the Iliopsoas muscle complex may have in chronic spinal pain. This will involve a total of 106 patients randomly assigned to either a treatment or non-treatment group. I am seeking your assistance in identifying potential participants from your patient population who fulfil the inclusion criteria (as per attached sheet) and who may be willing to participate. A Patient Information sheet is attached to this letter for your reference.

This research project has gained Ethics approval from Flinders Clinical Research Ethics Committee.

If you would like further details or have a patient, or patients, fulfilling the criteria and may be willing to participate, please contact me on the above telephone number.

Thank you.

Yours sincerely,

Aileen S Jefferis

Encl.

Appendix Five

FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

PARTICIPANT INFORMATION SHEET

Study Title – "The role of the Iliopsoas muscle complex in chronic spinal pain".

Because your spinal pain has lasted for six months, or longer, and has not responded to conventional treatment/s or therapies your condition can be classified as chronic. For these reasons you are invited to participate in the research project to study a deep and forward placed group of muscles which attach to your back bone. These are called the Iliopsoas muscles.

The aim of this project is to look at the ways in which this muscle group may effect your ongoing pain/s. As the situation with any muscle of which you can control its movement/s, the Iliopsoas muscles can develop pressure points which have the effects of shortening, tightening and weakening the involved muscle/s. A total of eighty people suffering chronic spinal pain will be divided into two groups by a method of chance; for example, tossing a coin. Both groups will be asked to complete:

- three simple questionnaires, with the researchers involvement.
- have your height measured
- have your weight measured
- have your iliopsoas muscles examined through feeling in your tummy and upper thigh.

One group will then have treatment to the Iliopsoas muscle group. If you are in this group there will be a total of twelve treatments, two treatments per week, each lasting approximately half an hour. The treatment will be –

- height measurement before and after each treatment.
- filling in a short questionnaires every week.
- gentle pressure, the use of a coolant spray or both these treatment techniques combined to the pressure points.
- an exercise activity to stretch the Iliopsoas muscles which you will be taught how to do, asked to do at home and record this activity in a diary which will be provided.
- at the end of your twelve treatments you will be asked to fill in again one of the original questionnaires.

The non-treatment group will be asked to:

- fill in one of the very simple questionnaires once a week at home.

- an exercise activity to stretch the Iliopsoas muscles which you will be taught how to do, asked to do at home and record this activity in a diary which will be provided.
    - attend at the end of six weeks from when you first filled in the questionnaires and have your height and weight measured again and fill in one short questionnaire again.

While you may not directly benefit from this project it is hoped that very useful information regarding chronic spinal pain will be gathered. Using this information, further research is planned, also.

The people who are in the treatment group may experience some discomfort, and/or in your pain site/s as the pressure points in the Iliopsoas muscles are let go, the muscles get longer and the position of your back bone changes.

1. If a subject of this research suffers injury, compensation may, at the discretion of the researcher or sponsor of the research, be paid without litigation. However, compensation is not automatic and subjects may have to take legal action in order to receive payment.
2. Your participation in the study is entirely voluntary and you have the right to withdraw from the study at any time. If you decide not to participate in this study or if you withdraw from the study, you may do this freely without prejudice to any treatment at Flinders Medical Centre. The researcher is a student in the Masters of Clinical Rehabilitation (research based) at Flinders University and is a qualified physiotherapist of twenty-six years. No financial rewards, nor incentives, are offered or to be gained for the researcher in this project.
3. All records containing personal information will remain confidential and no information that could lead to your identification will be released. The results of this project will be submitted, for publication, in either the Australian Physiotherapy Journal or the Journal of the Australian Medical Association.
4. Should you require further information about the project, either before, during or after the study, you may contact: Aileen Jefferis, PO Box 510 Park Holme SA 5043 Ph 0418-784753.
5. This study has been reviewed by the Flinders Clinical Research Ethics Committee. Should you wish to discuss the project with someone not directly involved, in particular in relation to matters concerning policies, your rights as a participant, or should you wish to make a confidential complaint, you may contact the Administrative Officer – Research, Ms. Carol Hakof, on 8204-4507.

Appendix Six

FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

CONSENT TO PARTICIPATION IN RESEARCH

I, _____ give consent to my involvement in the
(first or given name) (Surname)
research project:

"The role of the Iliopsoas muscle complex in chronic spinal pain".

I have been provided with a Patient Information Sheet which I have read and understood regarding the nature and purpose of the research project. Any questions regarding the research which I had have been answered to my satisfaction by: _____
(first or given name) (Surname)

My consent is given voluntarily.

I acknowledge that as a participant I will be assigned to one of two groups by a method of chance.

1. Treatment group.	2. Control group.
a) an initial interview of forty-five minutes at which time three short questionnaires will be completed, X-rays/scans will be reviewed, height and weight measurements will be recorded and iliopsoas muscles assessed. b) a self-managed stretching regime will be demonstrated and you will be expected to complete this regime in your own home twice daily for 6 weeks. A description of this regime will be provided to you in sheet form along with a diary to record how often you perform this regime. c) Twice weekly attendance for six weeks = total of twelve visits. Height measurements will be recorded before and after each treatment. These will be approximately 30 minutes in length and will consist of either gentle acupressure, the application of coolant spray and gentle exercises or a combination of the above. This may result in some discomfort, or pain, as a result of altered body function and/or stresses. d) One very short questionnaire to assess your function will be required to be completed weekly for 6 weeks. e) an interview of forty-five minutes at six weeks will be conducted. We will again measure height and weight and ask you to complete a short questionnaire to reassess your pain level/s.	a) an initial interview of approximately forty-five minutes at which time three short questionnaires will be completed, X-rays/scans will be reviewed, height and weight measurements will be recorded and the iliopsoas muscles assessed. b) a self-managed stretching regime will be demonstrated and you will be expected to complete this regime in your own home twice daily for 6 weeks. A description of this regime will be provided to you in sheet form along with a diary to record how often you perform this regime. c) One very short questionnaire to assess your function will be required to be completed weekly for 6 weeks. d)an interview of forty-five minutes at six weeks will be conducted. We will again measure height and weight and ask you to complete a short questionnaire to reassess your pain level/s.

I understand that my involvement with consent in this research project, whether I am assigned to the treatment or control group, may not be of direct benefit to me.

I have been informed, also, that I may withdraw my consent to participate in this research project without affecting my rights including those of on-going care and/or treatment/s.

Appendix Seven

FLINDERS MEDICAL CENTRE
FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

PATIENT HISTORY QUESTIONNAIRE

Date: .....

Patient Code : .....

D.O.B : ..... Gender : .....

1. When did you first develop pain in your spine?
   .....
2. Where did this pain start?
   .....
3. Was the pain sudden or gradual?
   .....
4. What were you doing at the time?
   .....
5. Has the pain spread?
   .....
6. If yes to Q5 – where to?
   .....
7. If yes to Q5 & Q6 – how long after the beginning of your first pain did the other pain, or pains, occur?
   .....
8. Are you receiving any treatment, or treatments, currently? .....
   If yes – details of these .....
   .....
9. What treatment or treatments have you received for your pain or pains? .....
   .....
   .....
10. For how long did you receive the treatment, or treatments? .....
    .....

Appendix Eight

Short-Form McGill Pain Questionnaire as formulated by Robert Melzack (1984)

Patient name Date of assessment

Pain description	Score 0 (none)	Score One (mild)	Score 2 (moderate)	Score 3 (severe)
Throbbing				
Shooting				
Stabbing				
Sharp				
Cramping				
Gnawing				
Hot-Burning				
Aching				
Heavy				
Tender				
Splitting				
Tiring-Exhausting				
Sickening				
Fearful				
Punishing-Cruel				

Please mark the category that best describes your pain currently

0 = No Pain

1 = Mild Pain

2 = Discomfort

3 = Distressing

4 = Horrible

5 = Excruciating

Please mark on the line where you feel your pain to be currently

Appendix Nine

Patient Specific Disability Measure. (Stratford et al 1995)

Instructions:

Clinician to read and fill in, please complete at the end of the history and prior to the physical

Read at baseline assessment
I'm going to ask you to identify up to 5 important activities that you are unable to do or have difficulty performing as a result of your problem. Today how difficult is it to perform activity: 1(have patient score this activity). 2(have patient score this activity). 3 etc..

Read at follow-up visits:
When I assessed you on (state previous assessment date), you told me that you had difficulty performing these activities (read1,2,3,4,5, from list). Today do you still have difficulty with activity: 1(have patient score this activity). 2(have patient score this activity). 3 etc..

Scoring scheme (show patient scale):

0 1 2 3 4 5 6 7 8 9 10

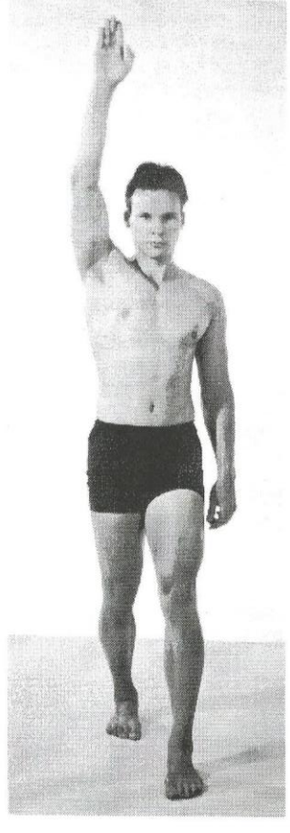
0 unable to perform activity

10 able to perform activity at pre-injury level

	Date/Score								
Activity									
1									
2									
3									
4									
5									
Additional									
Additional									
Average score									

Instructions:
Position right leg behind with foot pointing straight ahead, raise right arm, look straight ahead, squeeze buttocks.

10



Compliance Record

Name _____

Start Date _____

Put a "✓" in each box as appropriate.

	Week 1 am pm	Week 2 am pm	Week 3 am pm	Week 4 am pm	Week 5 am pm	Week 6 am pm
MONDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐
TUESDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐
WEDNESDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐
THURSDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐
FRIDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐
SATURDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐
SUNDAY	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐	☐ ☐

Appendix Eleven

Blinded Outcome Assessment

Date of re-assessment: _____

Patient Code: _____

Data File No: _____

Trigger point assessment scores:

Active = 1
Latent = 2
Absent = 3

Trigger point assessment:

R)		L)	
	1		1
	2		2
	3		3
	4		4
	5		5

Were you made aware of which group the participant was allocated to: Yes ___ No___

If yes: which group was specified: _____

This examination has led me to believe this participant was allocated to the:

a) Treatment group _____

b) Control group _____

Signed: _____

Qualification: _____

Appendix Twelve

FLINDERS MEDICAL CENTRE/FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

FLINDERS CLINICAL RESEARCH ETHICS COMMITTEE

1. Project Title:	"The role of the iliopsoas muscle complex and chronic spinal pain and, secondary associated signs and symptoms".
2 Investigator Details:	Ms Aileen Jefferis Flinders University Telephone (08) 83774585 Qualifications Diploma of Physiotherapy (NZ), Otago Polytechnic, 1976. Graduate Diploma in Social Science (Rehabilitation), University of South Australia, 2000. Professor Neil Piller Dept of Surgery School of Medicine Flinders Medical Centre Bedford Park S A 5042 Assoc Professor Michael Tyler Dept of Zoology Adelaide University S A 5000
3. List of places research is being undertaken:	The project is a study which will be undertaken by a PhD student in: • The Lymphoedema assessment Clinic, Department of Surgery, Flinders University • Private physiotherapy and massage practices in metropolitan Adelaide • Private homes.
4. Project details:	**Explanations** The Iliopsoas muscle complex is herein referred to as the IMC. **Background** Previous ethics approval was sought, and gained (Register no. 33/023 on 11/11/2002) to investigate the role of the IMC in chronic spinal pain. As a continuum to the above, further ethics approval is now sought to research some of the secondary signs and symptoms reported by chronic spinal pain patients who have dysfunction of the IMC. Commonly, chronic spinal pain sufferers report a plethora of seemingly unrelated signs and symptoms as their history progresses.[1] Anatomically, the IMC is in contact with, or in close proximity to, structures and organs which have the capacity to cause these secondary signs and symptoms; most significant to this research are the sympathetic nerves of the thoraco-lumbar plexus and, the colon.[2] **Rationale** The sympathetic thoraco-lumbar plexus lies adjacent to, and in direct contact with, the IMC.[2] The presence of myofascial trigger points in the IMC (the acknowledged effects of these being shortening, tightening and weakening[3,4]) creates the potential for disturbance of: Sympathetic nerve function ↓ Adrenal overload ↓ Zinc deficiency.

There are thirty listed signs of zinc deficiency [5] with levels/status tested by various methods including:

- Levels of zinc in leukocytes
- Hair analysis
- Administration of the Zinc Taste Tally Test [5]

Zinc deficiency and/or depletion can occur for many differing reasons including mental and physical stress and is implicated in a multitude of disease processes and syndromes. [5]
Zinc is an essential trace element, important in many bodily functions including; metabolism, cellular growth and collagen synthesis.[5, 6] Second only in concentration to iron, 30% of the body's zinc is stored in bones and 60% in muscles.[7] Zinc has been identified in over 300 catalytically active metallo-proteins and in excess of 2000 dependent transcription factors, reflecting its key role in many vital functions of the human body.[8]
As previously noted, zinc plays a vital role in many bodily functions, including collagen synthesis. A study conducted on chicks confirmed that zinc deficiency markedly reduced collagen synthesis and turnover in only 8 days.[9] Collagen abnormalities were reported in biopsies of muscle affected by congenital and idiopathic scoliosis and these were considered to be secondary to a primary, but unknown, causation.[10]
Reported systemic abnormalities in idiopathic scoliosis appeared to be related to collagen and proteoglycan synthesis, and could not be attributed to biomechanical effects.[11]
Decreased zinc levels in found in the sacrospinalis muscles of 50 patients (control n = 20) undergoing Harrington rod fusion; the conclusion drawn from this study was that the zinc deficiency in these muscles was a secondary feature arising from the primary scoliotic condition.[12]
Chronic spinal pain patients who have undergone alteration in their biomechanics may have, therefore, the compounding factor of being collagen weak as a result of a zinc deficiency; this may contribute to, or be a perpetuating factor in their musculo-skeletal syndrome.

The IMC is positioned posteriorly on the right to the ascending colon and, on the left to the descending colon. Faecal material, in constipated patients, has been reported to evoke pain via compression on psoas trigger points.[4]
Hypertrophy of the IMC in athletes has been shown to impinge on the large intestine.[13]
The researcher hypothesises that trigger points in the IMC may, in fact, be the cause of reported bowel and urinary dysfunction in chronic spinal pain patients.

Objectives

The aim of this research project is to investigate the role of the IMC in causing secondary signs and symptoms including:

- Sympathetic nervous system overactivity
- The relationship of the above has to zinc status and, therefore, collagen integrity.
- Large intestine dysfunction
- Urinary dysfunction.

The project will focus on objective measurements and a qualitative questionnaire to ascertain reported signs and symptoms of the above in participants; the relationship of these with their chronic spinal problems and other commonly reported complaints.

5. Proposed Methods

Study Design

This study will comprise the following:

- A questionnaire to ascertain signs and symptoms regarding sympathetic dysfunction, zinc deficiency and bowel function in chronic spinal pain patients and, a control group who have no history

	of spinal pain (see Appendix one) • The administration of the Zinc Taste Tally Test in all participants to ascertain zinc status. **Duration of the study** It is anticipated that nine months will be required to assess 116 participants, with a further three months for statistical analyses to be conducted on the collected data. **Selection of participants** The selection of participants will be on the following: **Inclusion criteria** • Chronic spinal pain which has been medically investigated of six months or longer duration • No history of any spinal pain • Age range 18 to 65 years. **Exclusion criteria** • Outside age range • Acute, or subacute, back pain • The taking of supplements containing calcium and/or zinc (see Appendix two). **Withdrawal criteria** Participants may withdraw from the study at any stage without prejudice to their ongoing care and/or treatment. **Statistical Analysis** Assuming an incidence of severe zinc deficiency in the control group of 25%, an incidence of 50% in the case group will be detected as statistically significant at alpha=0.05 with power 0.80 with n=58 people in each group (116 total).
6. Drug Profile:	One of the evaluations to be administered is the Zinc Taste Tally Test (which does not have to be swallowed). The British Pharmacopoeia approved this test in 1988, as a valid method of testing zinc status.[14] This product is categorised as a dietary supplement.
7. Procedures, including drug treatment involving the subject:	The administration of the Zinc Taste Tally Test requires the participant to hold 10mls.of this liquid in their mouth for 10 seconds to ascertain taste perception, if any during this time. This product is categorised as a dietary supplement, also.
8. Assessment of Patients.	Laboratory: nil. Radiological: nil. Clinical: Measurements to be utilised: • A questionnaire listing the 30 symptoms of zinc deficiency and, questions relating to bowel and bladder function, to be administered by the researcher (see Appendix one) • The administration of the Zinc Taste Tally Test (as described in **6** and **7** above). • Palpation of the IMC trigger points of which there are five on each side, in all participants. Significant adverse effect/s: While no significant adverse effect/s are anticipated, should this arise, it, or they will be reported to the FCREC.
9. Administrative Aspects:	Source and details of funding. Application has been made for research maintenance for travelling, phone and photocopying costs.
10. Indemnity:	As a Flinders University student indemnity is offered by enrolment with this

	Institution. The student has current professional indemnity as a registered Physiotherapist, also.
11. Consent Form:	The researcher will ensure a detailed explanation of the essence of the study. Written, informed consent will be obtained from the patient prior to participation in this project. Consent Form: (see Appendix three).
12. Patient Information Sheet:	See Appendix four.
13. Ethical Considerations:	**Benefits anticipated from the Project** This project will provide new knowledge as to a causation role of the IMC into some of the secondary associated signs and symptoms in chronic spinal pain patients, becoming the basis for the development of new strategies and treatments in this currently perplexing and costly condition. It is also envisaged that this research will lead to research into the role of the IMC in acute back pain and, then the prevention of spinal pain. **Risks** There are no known risks in the administration of the Zinc Taste Tally Test for the participants. **Research on People in Dependent Relationships** No relative of the researcher will be admitted into the study. **Separation of Research and Clinical Responsibilities** Following the Code of Ethics of the Physiotherapists Act (1991) and the NH&MRC *National Statement on Ethical Conduct in Research Involving Humans* the researcher acknowledges that clinical responsibilities override research responsibilities and will follow appropriate, professional procedures if these circumstances arise. Statement of compliance with NH&MRC *National Statement on Ethical Conduct in Research Involving Humans* has been accessed from the NHMRC Website at: http://www.nhmrc.health.gov.au/publications/pdf/e35.pdf. The project complies with the NH&MRC National Statement on Ethical Conduct in Research Involving Humans. **Source of Participants** Participants will be recruited by way of: • Patients from the researchers private practice • Invitation to participate from other private practitioners patients (see Appendix five) • Word of mouth • Previous participants **Protection of privacy and preservation of confidentiality.** All information acquired during this project will be kept strictly confidential to be used only in accordance with the stated objectives. De-identified statistical data only will be supplied to a statistician for analyses. All information collected will be entered on a secure data base; hard copy will be secured in a locked filing cabinet at Flinders University for a total of 15 years which is in accordance with NHMRC guidelines.

References

[1] Jefferis, A. 2001. *'Front to Back'*, p.p.12-13, Gillingham Printers, Adelaide.
[2] Bogduk, N.1997. '*Clinical Anatomy of the Lumbar Spine'*, p.138, Churchill Livingstone, Edinburgh.
[3] Travell, J. & Simons, D. 1983. '*Myofascial Pian and Dysfunction'*, Williams and Wilkins, Baltimore.
[4] Travell, J. & Simons, D. 1992. '*Myofascial Pian and Dysfunction'*, vol. 2 Williams and Wilkins, Baltimore.
[5] Favier, A. 1993. 'Current aspects about the role of zinc in nutrition', *Rev. Prat,* vol.2. p.p.146-51
[6] Bryce-Smith, D. & Hodgkinson, L. 1986. '*The Zinc Solution'*, Century Arrow, London.
[7] Life Extension Foundation
[8] Prasad, A. 2003. 'Zinc Deficiency', *The British Medical Journal*, vol. 326: p.p. 409-410.
[9] Starcher, B. Hill, C. & Madaras, J. 1980. ' Effect of zinc deficiency on collagenase and collagen turnover'. *Journal of Nutrition,* vol. 2 p.p. 2095-102.
[10] Uden, A. Nilssson, I. & Willner, S. 1980. ' Collagen changes in congenital and idiopathic scoliosis'. *Acta Orthopedia Scandavia,* vol. 51(2):p.p. 271-4.
[11] Worthington, V. & Shambaugh, P. 1991. 'Systemic abnormalities in idiopathic scolosis'. Vol. 14(8): p.p.467-71.
[12] Dastych, M. & Vlach, O. 1990. 'Zinc status in patients with idiopathic scoliosis'. *Spine,* vol.2 p.p. 65-66.
[13]
[14] British Pharmacopoeia,CRC Press: Boca Ration (FL) (1988), p315, section9.5.4. "Zinc".

Appendix Thirteen

Flinders Medical Centre
Bedford Park South Australia 5042

Telephone (08) 8204 5511
International 618 8204 5511

Flinders Clinical Research Ethics Committee
FWA00001785

Telephone (08) 8204 4507
Facsimile (08) 8204 5834
email: Carol.Hakof@fmc.sa.gov.au

16 August 2004

MEMORANDUM

TO: Ms. A. Jefferis, PO Box 510, PARK HOLME SA 5043

FROM: Ms. C. Hakof, Executive Officer, Flinders Clinical Research Ethics Committee

TOPIC: **Research Application 19/045**

I am pleased to advise that the Flinders Clinical Research Ethics Committee (FCREC) has approved your research application in accordance with the following extract from the Minutes of its meeting held on 9 August 2004.

5999 RESEARCH APPLICATION 19/045 – MS. A. JEFFERIS
The role of the iliopsoas muscle complex and chronic spinal pain and secondary associated signs and symptoms.
Reviewer: Dr. J. Walsh

This application was approved.

A progress report must be provided annually. Approval is given for a period of three (3) years only and, if the study is more prolonged than this, an updated submission will be required.

If conditional *('subject to' or 'in principle')* approval is granted, research involving human subjects **may proceed only after written acceptance of the conditions of approval** (including a copy of the modifications) has been received by the Committee.

If patients are involved the chief investigator is responsible for the process of notification, seeking approval or permission of Departments, Divisions or individual consultants. ***A copy of the signed consent form is to be filed in the participant's medical record.*** Please note that if this trial involves normal volunteers it will be necessary for you to keep a record of their names and you may be required to supply this list with your annual report.

You are reminded that the FCREC must approve the content and placement of advertisements for the recruitment of volunteers.

The Committee must be notified and approve any changes (e.g. additional procedures, modification of drug dosage, changes to inclusion or withdrawal criteria, changes in mode and content of advertising) in the investigational plan particularly if these changes involve human subjects.

The safe and ethical conduct of a trial is entirely the responsibility of the investigators. While the FCREC takes care to review and give advice on the conduct of trials, approval by the Committee is not an absolute confirmation of safety, nor does approval alter in any way the obligations and responsibilities of investigators.

It is the duty of the chief investigator to give prompt notification to the FCREC of matters which might affect continued ethical acceptability of the project, including:

1. Adverse effects of the project on participants, including the total number of participants recruited, and of steps taken to deal with these adverse effects.
2. Other unforeseen events.
3. A change in the base for a decision made by the Committee, e.g. new scientific information that may invalidate the ethical integrity of the study.

Appendix Fourteen

FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

"The role of the Iliopsoas muscle complex in CLBP/chronic spinal pain, and some of the secondary associated signs and symptoms"

Criteria

Inclusion criteria:

- CLBP/chronic spinal pain that has been medically investigated, of 6 months or longer duration

  or

- No history of any spinal pain
- Age range 18-65 years.

Exclusion criteria

- Outside the age range
- Acute, or subacute, spinal pain
- Suspicion or diagnosis of osteoporosis
- History of problems involving the aorta or vena cava
- The taking of corticosteroids or any medication known to affect bone density
- Pregnancy
- The taking of supplements containing calcium and/or zinc.

Withdrawal criterion

- Participants may withdraw from the study at any stage without prejudice to their ongoing care and/or treatment.

Appendix Fifteen

Aileen S Jefferis
Department of Surgery
Flinders University

PhD. student
Contact Phone No: 0418-784-753

Dear Practitioner,

Re: Flinders University based Research project entitled:
"The role of the Iliopsoas muscle complex in chronic spinal pain and, secondary associated signs and symptoms"

As the second component of my research, I am undertaking a clinical trial to ascertain whether the Iliopsoas muscle complex causes secondary associated signs and symptoms in chronic spinal pain patients.
This will involve a total of 106 patients, both chronic spinal pain patients, and patients who have never suffered any form of spinal pain.
I am seeking your assistance in identifying potential participants from your patient population who fulfil the inclusion criteria (as per attached sheet) and who may be willing to participate. A Patient Information sheet is attached to this letter for your reference.

This research project has gained Ethics approval from Flinders Clinical Research Ethics Committee.

If you would like further details or have a patient, or patients, fulfilling the criteria and may be willing to participate, please contact me on the above telephone number.

Thank you.

Yours sincerely,

Aileen S Jefferis

Encl.

Appendix Sixteen

FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

PARTICIPANT INFORMATION SHEET

Study Title

" The role of the Iliopsoas muscle complex in chronic spinal pain and some of the secondary associated signs and symptoms"

The aim of this project is to look at the ways in which the iliopsoas muscle complex may cause, or play a role in, the secondary associated problems that long-term sufferers of chronic spinal pain report.

If your spinal pain has lasted for six months, or longer, and has not responded to conventional treatment/s or therapies your condition can be classified as chronic. For these reasons you are invited to participate in the research project to study the secondary associated problems that long-term sufferers of chronic spinal pain experience.

Equally, if you have never experienced **any** form of pain in your low or middle back, neck pain or headaches then you may be eligible to participate as part of the comparison group.

Any muscle you can control its movement/s, the iliopsoas muscles can develop pressure points which have the effects of shortening, tightening and weakening the involved muscle/s. Closely ssociated with the iliopsoas muscle complex are a group of nerves called the autonomic ("automatic") nerves and these have the potential to cause directly or indirectly some of the problems that longstanding spinal pain sufferers report: some of these problems, at least, may be caused by these nerves depleting the body of zinc.

The aim of the study is to research these secondary problems affecting spinal pain patients by asking questions which look at the role the iliopsoas muscle complex may play in these. This is to ascertain some of the secondary associated problems including zinc deficiency, bladder and bowel problems.

A total of 58 people suffering chronic spinal pain in the low or middle back, neck pain or headaches will be asked to undertake:

- One questionnaire with the researcher's involvement
- Have your iliopsoas muscles examined by feeling the tummy and upper thigh
- A Zinc Tally Taste Test which involves holding 10mls of a solution in the mouth for 10 seconds and noting any taste sensation during this time.

You are requested not to eat, drink or smoke cigarettes for one hour prior to this test being given.

A total of 58 people who have never experienced **any** form of pain in your low or middle back, neck pain or headaches will be asked to undertake:

- One questionnaire, with the researchers involvement
- Have their iliopsoas muscles examined by feeling the tummy and upper thigh
- A Zinc Taste Tally Test, which involves holding 10 mls. of a solution in the mouth for 10 seconds and noting any taste sensation during this time. You are requested not to eat, drink or smoke cigarettes for one hour prior to this test being given.

The results will then analysed to investigate any differences between the two groups.

While you may not directly benefit from this project it is hoped that very useful information regarding chronic spinal pain will be gathered. Using this information, further research is planned, also.

If a subject of this research suffers injury, compensation may, at the discretion of the researcher or sponsor of the research, be paid without litigation. However, compensation is not automatic and subjects may have to take legal action in order to receive payment.

Your participation in the study is entirely voluntary and you have the right to withdraw from the study at any time. Your decision to participate in this study is entirely voluntary and so if you withdraw from the study you are free to do this without prejudice to any treatment at Flinders Medical Centre.

The researcher is a PhD. student at Flinders University and is a qualified physiotherapist of twenty-eight years.

No financial rewards, nor incentives, are offered or to be gained for the researcher in this project.

All records containing personal information will remain confidential and no information that could lead to your identification will be released. The results of this project will be submitted, for publication, in an appropriate journal.

Should you require further information about the project, before, during or after the study, you may contact:
Aileen Jefferis,
PO Box 510
Park Holme SA 5043
Ph 0418-784753.

The Flinders Clinical Research Ethics Committee has reviewed this study. Should you wish to discuss the project with someone not directly involved, in particular in relation to matters concerning policies, your rights as a participant, or should you wish to make a confidential complaint, you may contact:
The Administrative Officer – Research,
Ms. Carol Hakof
Ph: 8204-4507.

Appendix Seventeen

FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

CONSENT TO PARTICIPATION IN RESEARCH

I, _____ give consent to my
(First or given name) (Surname)
Involvement in the research project:

"The role of the Iliopsoas muscle complex in chronic spinal pain and secondary associated signs and symptoms"

I have been provided with a Patient Information Sheet, which I have read ad understood regarding the nature and purpose of the research project. Any questions regarding the research, which I had, have been answered to my satisfaction by:

_____
(First or given name) (Surname)

My consent is given voluntarily.

I acknowledge that as a participant I will be participating in this research as a member of one of the two groups. This requires me to participate in the research as follows:

1. Chronic spinal pain group.	2. Never experienced spinal pain group.
a) an interview of thirty minutes at which time one questionnaire will be completed with the researcher b) your iliopsoas muscle will be assessed by the method of gentle acupressure to five points on each side of your tummy and upper thigh.	a) an interview of thirty minutes at which time one questionnaire will be completed with the researcher b) your iliopsoas muscle will be assessed by the method of gentle acupressure to five points on each side of your tummy and upper thigh.

I understand that my involvement with consent in this research may not be of direct benefit to me.

I have been informed, also, that I may withdraw my consent to participate in this research project without affecting my rights including those of on-going care and/or treatment/s.

I acknowledge that I have been informed that should I receive an injury, as a result of taking part in this study, I may be required to start legal action in order to receive compensation.

Signature of research participant: _____ Date: _____

Signature of witness: _____

Date: _____

Printed Name of witness: _____

I, _____ have described to _____

the research project, outlined above; the nature and effects of the procedures involved. In my opinion she/he understands my explanations and has given her/his consent, freely and with due knowledge.

Signature: _____

Date: _____

Status in project: _____

Appendix Eighteen

FLINDERS MEDICAL CENTRE
FLINDERS UNIVERSITY OF SOUTH AUSTRALIA

PATIENT HISTORY QUESTIONNAIRE

Date: .....

Patient code: .....

D.O.B: .....

Gender: .....

Employed: .....

1. Have you experienced pain in your spine? .....
2. If yes, how long have you experienced this pain? .....
3. If yes to Q1, where did this pain start? .....
4. If yes to Q1, has the pain spread and to where? .....
5. Do you know what started it? .....
6. Do you suffer from any of the following problems?

   Impaired sense of taste .....
   Impaired sense of smell .....
   Depression .....
   Mood changes .....
   Impaired concentration .....
   "Jitteriness" .....
   Speech problems .....
   Stretch marks on the skin .....
   Skin problems including acne .....
   Dry skin .....
   Dry hair or excessive hair loss .....
   Brittle nails .....
   White spots in your nails .....

Vertical ridges in your nails.....

Sensitivity to light or night blindness.....

Afternoon fatigue.....

Sugar and/or chocolate cravings.....

6. Do you smoke cigarettes?..... If yes, how many per day?.....

7. Do you drink alcohol regularly i.e. on a daily basis?

8.Are you a vegetarian?.....If yes, ovolacto.....or vegan?.....

9. Do you eat food regularly originating from a can i.e. on a daily basis?

10. Do you eat high fibre foods regularly?.....

11. If so how much and how often?.....

12. How much milk would you consume in a day?.....

13. Do you eat only organically grown and raised foodstuffs?

14. Are there any significant stresses in your life (such as financial; social; work; family; personal): current ..... or in the last 6 months.....?

15.Do you take any of the following?

Steroids.....

Diuretics.....

Antacids.....

Oral contraception.....

Hormone replacement therapy.....

Laxatives.....

Anticonvulsant drugs.....

Q16) Do you have any problems with your bladder function? If you are in Group1 i.e. the chronic spinal pain group and you answered yes, do you associate these changes with the onset of your spinal pain?

Q)17. If yes, have you consulted a doctor or health practitioner for this ? .....

Q18) If yes, was any treatment or advice given to you?.....

Q19). If yes, what was advised.....?

Q20). If yes, did this assist you?.....?

Q21) Do you have any problems with your bowel function? If you are in Group1 i.e.

the chronic spinal pain group and you answered yes, do you associate these changes with the onset of your spinal pain ?

.....

22. If yes, have you consulted a doctor or health professional for this? .....

23. If yes, was any treatment or advice given to you? .....

24. If yes, what was advised? .....

25. If yes, did this assist you? .....

26. Zinc Tally Taste Test administered? .....

Category (1-4) .....

Trigger point record

R) 0 1 2

1

2

3

4

5

L) 0 1 2

1

2

3

4

5