THE RELATIONSHIPS BETWEEN STATURE RECOVERY, MUSCLE ACTIVITY AND PSYCHOLOGICAL FACTORS IN PATIENTS WITH CHRONIC LOW BACK PAIN

SANDRA LEWIS

THESIS SUBMITTED IN FULFILMENT OF THE

REQUIREMENT FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

MANCHESTER METROPOLITAN UNIVERSITY

January 2011

Abstract

It has previously been established that individuals with mild chronic low back pain (CLBP) have a delayed rate of post-exercise stature recovery compared to asymptomatic controls, and that this is associated with increased paraspinal muscle activity, pain and disability. The purpose of this thesis was to explore these relationships further in NHS patients with CLBP and to establish if links exist between these measures and a number of psychological factors known to play an important role in the development of the condition.

Forty seven patients were recruited from the waiting list for two physiotherapist-led rehabilitation programmes. Paraspinal muscle activity was assessed via surface EMG while standing at rest and stature recovery over a 40-minute unloading period was measured on a precision stadiometer. Self-report of pain was noted and patients were asked to complete a questionnaire booklet assessing disability, anxiety, depression, pain-related anxiety, fear of movement, self-efficacy, catastrophising and defensiveness. Where possible, patients returned for a second testing session after completing the rehabilitation programme (n = 23) and again after a further six months (n = 14). The effect of superficial heat treatment was additionally assessed via a similar testing session (n = 24), but on this occasion the participant put on a heat wrap two hours before the time of the appointment.

Significant correlations were found between baseline muscle activity and both pain and disability. Pain was a significant mediator in the relationship between muscle activity and disability. Muscle activity also demonstrated links with self-efficacy, depression, anxiety, pain-related anxiety and catastrophising and was a significant mediator in the relationship between self-efficacy and pain. Stature recovery was not significantly related to any of the other baseline measures, perhaps reflecting the heterogeneous nature of the patients involved. A high prevalence of defensive high anxious individuals was found in the patient group. Changes in stature recovery immediately following the programme were significantly linked to improvements in pain and disability, although a significant increase in stature recovery was only observed by the end of the follow-up period. Resting EMG was not reduced following the rehabilitation programme. The heat wrap resulted in a significant decrease in non-normalised EMG levels and a positive effect on self-report of disability, self-efficacy, catastrophising and pain-related anxiety.

In conclusion, the relationship between stature change and muscle activity appears to be more complex than originally hypothesized. However, six months after a rehabilitation programme, the rate of stature recovery had increased to levels similar to asymptomatic individuals, suggesting that the delayed recovery seen in CLBP patients is not primarily the result of pathology. The findings confirm that muscle activity plays an important role in CLBP, in particular as a pathway by which psychological factors may impact on clinical outcome. The role of muscle activity as a mediator between psychological factors and pain suggests that interventions that are able to reduce muscle activity may be of particular benefit to patients demonstrating characteristics such as low self-efficacy, which may help in the targeting of treatment for CLBP. The results also highlight that an immediate decrease in EMG levels following active treatment may not always be the optimal response for long-term improvements in clinical outcome and that a period of adaptation might be expected. The unexpectedly high prevalence of a defensive high anxious coping style suggests that this may represent a risk factor for CLBP, a predictor of poor outcome or an adaptation to a chronic condition.

Publications

Lewis, S., Fowler, N., Hindle, J., Woby, S., & Holmes, P. (Under review). The relationships between stature recovery, muscle activity and psychological factors in patients with chronic low back pain. *Manual Therapy.*

Based on research carried out within Study 1 of this thesis.

Lewis, S., Woby, S., Fowler, N., Hindle, J., & Holmes, P. (Under review). Defensive coping styles and chronic low back pain. *Physiotherapy*.

Based on research relating to coping styles carried out within Study 1 of this thesis.

Lewis, S., Fowler, N., Hindle, J., Woby, S., & Holmes, P. (Under review). The effect of an active rehabilitation programme on stature recovery, muscle activity and psychological factors for patients with chronic low back pain. *Disability and Rehabilitation.*

Based on research carried out within Study 2 of this thesis.

Lewis, S., Fowler, N., Hindle, J., Woby, S., & Holmes, P. (Under review). The short-term effect of superficial heat treatment on paraspinal muscle activity, stature recovery and psychological factors in patients with chronic low back pain. *Archives of Physical Medicine and Rehabilitation.*

Based on research carried out within Study 3 of this thesis.

Lewis, S. & Fowler, N. (2010). Evaluation of the loading response of intervertebral discs using measurements of stature change and Magnetic Resonance Imaging. *Journal of Bone and Joint Surgery*, *92B*, 233.

Orthopaedic Proceedings. Based on research carried out with the Director of Studies prior to commencement of the PhD, which underpinned the work in this thesis.

Lewis, S. & Fowler, N. (2009). Changes in intervertebral disk dimensions after a loading task and the relationship with stature change measurements. *Archives of Physical Medicine and Rehabilitation, 90,* 1795-1799.

Based on research carried out with the Director of Studies prior to commencement of the PhD, which underpinned the work in this thesis.

Conference presentations

Lewis, S., Fowler, N., Hindle, J., Woby, S., & Holmes, P. (2010, November). *The relationships between stature recovery, muscle activity and psychological factors in patients with chronic low back pain*. Poster session presented at the 7th Interdisciplinary World Congress on Low Back & Pelvic Pain, Los Angeles.

Based on research carried out within Study 1 of this thesis.

Lewis, S., Fowler, N., Hindle, J., Woby, S., & Holmes, P. (2010, April). *The relationships between stature recovery, muscle activity and psychological factors in chronic low back pain patients.* Poster session presented at the Annual Scientific Meeting of the British Pain Society, Manchester.

Based on research carried out within Study 1 of this thesis.

Lewis, S., Fowler, N., Hindle, J., Woby, S., & Holmes, P. (2009, October). *The relationship between muscle activity, stature recovery and clinical outcome in chronic low back pain.* Paper presented at the 6th annual meeting of the Postgraduate Researchers in Science Medicine (PRISM), Manchester.

Based on research carried out within Study 1 of this thesis.

Lewis, S. & Fowler, N. (2008, November). *Evaluation of the loading response of intervertebral discs using measurements of stature change and Magnetic Resonance Imaging*. Paper presented at the annual meeting of the Society for Back Pain Research, Keele.

Based on research carried out with the Director of Studies prior to commencement of the PhD, which underpinned the work in this thesis.

Lewis, S. & Fowler, N. (2008, April). *Evaluation of the loading response of intervertebral discs using measurements of stature change and MRI.* Poster session presented at the Annual Scientific Meeting of the British Pain Society, Liverpool.

Based on research carried out with the Director of Studies prior to commencement of the PhD, which underpinned the work in this thesis.

Contents

List of figures

List of plates

List of tables

Acknowledgments

Foremost, I would like to express my gratitude to Professor Neil Fowler, Dr Paul Holmes and Jackie Hindle at Manchester Metropolitan University and Dr Steve Woby at North Manchester General Hospital for their supervision as well as their support, expertise and guidance throughout this PhD.

I am also indebted to the staff in the Physiotherapy Department at North Manchester General Hospital and the Cornerstone Centre in Beswick for their advice and encouragement. In particular, I would like to thank them for allowing me to carry out the data collection in the Department and for making me feel very welcome.

I am most grateful to the MMU technical department, who constructed the stadiometer and provided ongoing technical support.

Warmest thanks go to all the people who gave up their time to participate in the studies and made data collection an interesting and enjoyable experience. It goes without saying that the research wouldn't have been possible without them.

Finally, thanks to my friends and family for their constant support and encouragement.

Declaration

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

Signed…….

Date…….

CHAPTER ONE

1. Introduction

Back pain is a widespread problem with a one-year prevalence of up to 65% (Walker, 2000) and a lifetime prevalence of up to 84% (Airaksinen *et al.*, 2006). In 1998 it was estimated that the direct health care cost of LBP in the UK was £1,632 million per annum. If the estimated production losses due to incapacity to work and informal care costs are included, the total figure was conservatively estimated at £6,650 million and may be as high as £12,300 million per annum (Maniadakis & Gray, 2000). LBP symptoms, pathology and radiological findings are poorly correlated and less than 15% of patients receive a specific diagnosis (Airaksinen *et al.*, 2006). This leads to many patients being classified as having 'non-specific LBP'. A wide range of different treatments exist for LBP, many of which result in reduced pain and/or disability for some patients, although perhaps due to the heterogeneity of the condition, there is no single treatment that is effective for all patients. This has led to increasing calls for research to aid in the identification of subgroups of LBP patients which will benefit most from specific interventions.

LBP patients often demonstrate altered muscle function compared to asymptomatic controls. This manifests in a number of ways. When dealing with internal or external perturbations to the body, people with LBP tend to exhibit delayed or reduced activation of the deep trunk muscles (Hodges & Moseley, 2003); during movement, LBP patients frequently have increased antagonistic muscle activity (e.g. Ahern, Follick, Council, Laser-Wolston, & Litchman, 1988); and in static postures, such as standing and full flexion, there is often hyperactivity of the superficial paraspinal muscles (e.g. Ambroz, Scott, Ambroz, & Talbott, 2000). Several hypotheses have been put forward to explain the presence of elevated trunk muscle activity in LBP patients. It has been suggested that hyperactivity may represent a spasm caused by pain (Travell, Rinzler, & Herman, 1942); a strategy to

splint the trunk and prevent further injury (Lund, Donga, Widmer, & Stohler, 1991); or compensation for spinal instability resulting from dysfunction or injury (Panjabi, 1992a). However, the heterogeneity of the changes in muscle activation between patients suggests that they may be more complicated than can be explained by any one of these models alone, leading to the popular current view that individuals develop unique muscle recruitment strategies aimed at maintaining homeostasis and minimising pain (Murray & Peck, 2007).

Linked to this elevated paraspinal muscle activity is the finding that LBP patients appear to exhibit increased loading of the intervertebral discs as manifested by delayed stature recovery after loading. Intervertebral discs lose height in response to compressive forces, due to a combination of fluid outflow and elastic deformation of both the disc and the vertebral endplates. When the spine is subsequently unloaded, these processes are reversed, leading to fluid inflow and disc height recovery (Koeller, Funke, & Hartmann, 1984). Changes in disc height lead to changes in overall body height, or stature. Hence stadiometry, which measures changes in body height, is often used as an indirect and non-invasive method for assessing changes in disc height.

People with LBP appear to lose stature at a similar rate to healthy controls in response to loading. However, they are significantly slower to recover this height when the spine is unloaded and this reduced stature recovery has been linked to increased paraspinal muscle activity (Healey, Fowler, Burden, & McEwan, 2005a). It is therefore hypothesized that the elevated muscle activity observed in people with LBP results in greater compressive loads on the spine that, in turn, prevent the intervertebral discs from regaining their initial height and consequently prolongs stature recovery (Healey *et al.*, 2005a). Intervertebral disc height loss may increase the risk of future back pain by compromising spinal stability

(Zhao, Pollintine, Hole, Dolan, & Adams, 2005) and has been associated with a decrease in the discs' shock absorbing properties, increased loading on spinal structures such as the facet joints and concentrations of compressive stress (Adams, Bogduk, Burton, & Dolan, 2002). Significant negative correlations between stature recovery and both pain and disability support the clinical relevance of this relationship (Healey *et al.*, 2005a). These findings imply that reducing paraspinal muscle activity may increase the rate of stature recovery and therefore have a beneficial effect on clinical outcome.

The argument for causality was supported by the findings of a subsequent study (Healey, Burden, McEwan, & Fowler, 2008) in which paraspinal muscle activity of asymptomatic participants was increased using functional electrical stimulation to a level intended to mimic that seen in patients with LBP and there was an associated reduction in stature recovery, similar to that observed in LBP patients. Although an important finding, this does not prove that elevated muscle activity is causal for reduced stature recovery in a clinical population. The link between muscle activity and stature recovery in patients with LBP may be due to a third factor such as disc degeneration, which is known to affect the loading response (Adams & Hutton, 1983). The pain resulting from degeneration and the accompanying reduced stature recovery may then lead to increased muscle activity. In this case, reducing muscle activity would not be expected to have a major impact on stature recovery. This possibility was considered when determining the study design for the thesis.

Psychological factors play an important role in LBP. Although they have been identified as potential causal factors of low back disorders (Adams, Mannion, & Dolan, 1999), they appear to have a greater impact in the development of chronic pain and disability (Adams *et al*., 2002) and are sometimes viewed as "obstacles to recovery" (e.g. Foster, Thomas, Bishop, Dunn, & Main, 2010, p.398). Catastrophizing, fear of movement (Picavet,

Vlaeyen, & Schouten, 2002), self-efficacy (Woby, Urmston, & Watson, 2007b), depression and anxiety (Linton, 2000) have all been identified as significant in the onset and/or perpetuation of LBP. It has been suggested that one of the ways psychological factors may affect the condition is via increased spinal loading resulting from altered paraspinal muscle activity. For example, psychological stress has been observed to increase spinal compression and lateral shear, with these increased spinal loadings traced to differences in muscle co-activation (Marras, Davis, Heaney, Maronitis, & Allread, 2000). Furthermore, LBP patients who have high levels of pain-related fear generally exhibit elevated paraspinal muscle activity compared to low fearful patients, especially when confronted with movements which they believe are harmful (Vlaeyen & Linton, 2000). It is proposed that pain-related fear may perpetuate pain and disability via this muscle guarding. Muscle activity may therefore be a contributory factor in the link between psychological factors and clinical outcome.

This thesis aims to investigate the two main general hypotheses which follow from the above research findings. Firstly, that reducing paraspinal muscle activity leads to an increase in the rate of stature recovery, and so has a beneficial impact on clinical outcome. Secondly, that certain psychological factors are linked to levels of muscle activity and that these associations may help to explain causes of, and changes in, pain and disability. If the results supported these hypotheses, it would suggest that interventions that were able to reduce paraspinal muscle activity and promote more rapid stature recovery may be more effective in improving clinical outcome than those that do not influence this primary load mechanism. Furthermore, it may be possible to identify subgroups of patients, with certain psychological characteristics, for whom muscle hyperactivity and delayed stature recovery appear to play a greater role. If so, this may help in the targeting of treatment for LBP.

1.1. Global aim:

To establish whether active rehabilitation programmes and superficial heat treatment result in reduced paraspinal muscle activity in chronic LBP (CLBP) patients and whether this affects an increase in the rate of stature recovery. If so, to investigate whether this has a beneficial effect on clinical outcome, or can help to identify any subgroups of patients for whom this approach might be most effective.

1.2. Specific aims:

(1) extend the current knowledge about the relationship between paraspinal muscle activity, stature recovery and levels of pain and disability in NHS patients with CLBP;

(2) assess the repeatability of inter-day stadiometer measurements in patients with CLBP;

(3) investigate the role of psychological factors on paraspinal muscle activity, stature recovery and clinical outcome;

(4) identify whether baseline levels of muscle activity, stature recovery or psychological factors, or changes in these variables following treatment, are associated with better clinical outcome;

(5) investigate the effect of superficial heat treatment on muscle activity and stature recovery.

1.3. Structure of the thesis

The structure of the thesis is as follows: a review of literature to determine current state of knowledge with regards to the area of study, followed by three experimental studies to test the research hypotheses. The main aim of Study 1 was to assess whether the established relationships between stature recovery, muscle activity, pain and disability exist in a more

clinically relevant population of NHS patients with CLBP, including individuals with more severe back pain than previously examined. In addition, this study furthered previous work by seeking to establish whether a range of self-report psychological factors demonstrated an association with muscle activity or stature change. An asymptomatic control group was also included to enable comparison of stature change and muscle activity levels between the two groups. Study 2 was longitudinal; patients were tested both before and after they participated in an active rehabilitation programme and at a six-month follow up session. Relationships between changes in the variables were examined and any characteristics associated with better clinical outcome were sought. For both ethical and practical reasons, a separate control group was not included in this study. However, the patients on the rehabilitation programme were all long-term sufferers of back pain and hence it was believed that the measures would be relatively stable over time. This was assessed (within Study 1), where possible, by means of two testing sessions held approximately a week apart prior to the participant commencing the rehabilitation programme. These additional visits also enabled repeatability of inter-day stadiometer measurements in patients with CLBP to be analysed, an area lacking in the literature. Study 3 investigated the effect of superficial heat treatment, which is thought to result in a short term reduction in muscle activity. If muscle activity could be temporarily reduced in this way, it would enable analysis of the acute effect of muscle relaxation on stature recovery. Since neither active rehabilitation programmes nor heat treatment would be expected to affect pathology, any changes in stature recovery following either intervention would help to address the suggestion that such factors are the primary cause of reduced stature recovery.

CHAPTER TWO

2. Literature review

2.1. Low back pain

Low back pain can be classified as acute, sub-acute and chronic. Definitions vary, but most commonly CLBP is classed as that lasting more than three months, with sub-acute covering durations of between six weeks and three months, and acute less than six weeks (Waddell, 2004). This can be misleading, however, because LBP is often a recurrent problem, with a previous history of back pain identified as the greatest risk factor for a new episode of LBP (Mannion, Dolan, & Adams, 1996).

LBP symptoms, pathology and radiological findings are poorly correlated and less than 15% of patients will have a specific diagnosis (Airaksinen *et al.*, 2006). This leads to many patients being diagnosed as having 'non-specific LBP', defined in the recent National Institute for Health and Clinical Excellence (NICE) guidelines (NICE, 2009, p.9) as "tension, soreness and/or stiffness in the lower back region for which it isn't possible to identify a specific cause of the pain". Pain and disability represent distinct aspects of the condition and are not as closely related as might be expected (Waddell, Newton, Henderson, Somerville, & Main, 1993). In fact, a number of researchers have found cognitive factors such as pain-related fear to be better predictors of self-reported disability levels than pain intensity or biomedical status (e.g. Crombez, Vlaeyen, Heuts, & Lysens, 1999). This illustrates the multifactorial nature of the condition, in which biomechanical, psychological and social factors have all been identified as important and are often thought to be inter-linked (Waddell, 2004).

2.2. Anatomy

2.2.1. *Anatomy of the vertebral column*

The main functions of the vertebral column are to support the trunk, allow movement and locomotion, and protect the spinal cord (Middleditch & Oliver, 2005). It typically consists of 24 separate vertebrae: 7 cervical (C1-C7) in the neck or cervical region, 12 thoracic (T1 – T12) connected with the ribs and 5 lumbar (L1-L5) in the lower back. Below this are five fused vertebrae that form the sacrum and usually four fused vertebrae that form the coccyx. There are intervertebral discs between the adjacent vertebral bodies, except for between C1 and C2.

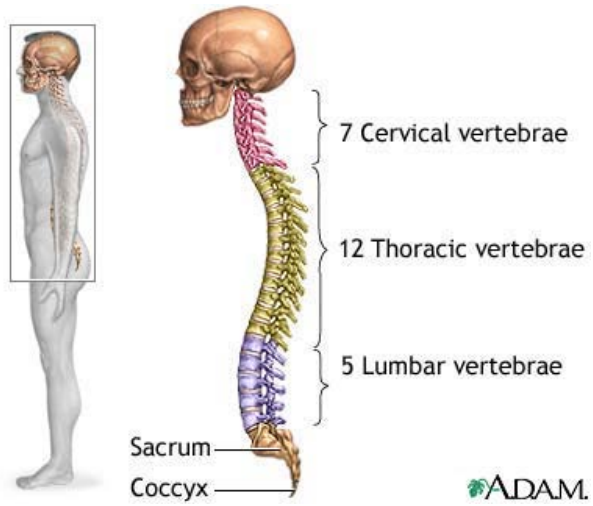


Figure 2.1 Lateral view of the vertebral column (A.D.A.M. Medical Encyclopedia, 2010)

The anterior pillar of the spine (including the intervertebral discs) has primarily weight-bearing and shock absorbing functions whereas the posterior pillar (including the apophyseal joints) controls movement and maintains stability (Adams *et al.*, 2002; Middleditch & Oliver, 2005). In-between is the vertebral canal, containing the spinal cord and associated structures. There are four curves in the vertebral column (one each of cervical, thoracic, lumbar and sacral) when in the upright posture, which also act in a shock-absorbing capacity.

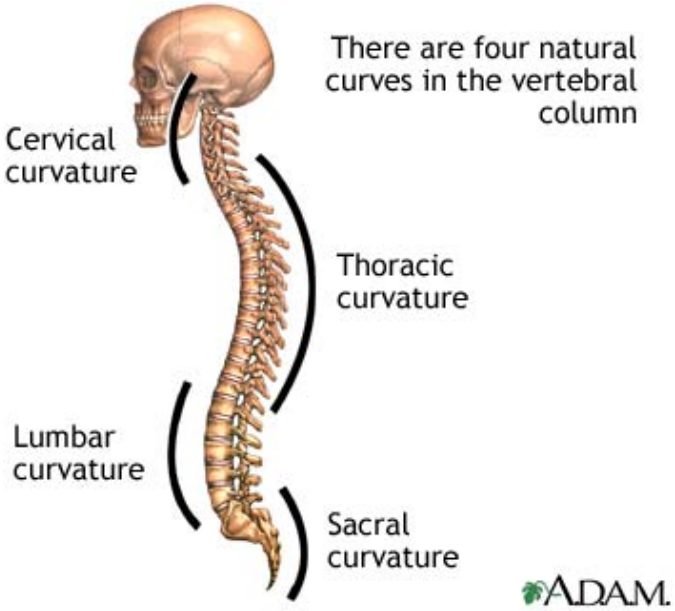


Figure 2.2 The curves in the vertebral column (A.D.A.M. Medical Encyclopedia, 2010)

2.2.2. *The motion segment*

A motion segment consists of two vertebrae and the intervening disc and ligaments (Adams *et al.*, 2002) and represents the smallest functional unit of the spine. Vertebrae consist of a roughly cylindrical vertebral body, from the posterior surface of which are two projections, or pedicles. These extend posteriorly to connect to the laminae. The pedicles and laminae form the vertebral or neural arch, and between this and the vertebral body is the vertebral foramen which encircles the spinal cord (Jenkins, 1998). Projecting laterally from the junction of the lamina and pedicle is a bony projection called the transverse process for the attachment of muscles and ribs (Jenkins, 1998; Adams *et al.*, 2002). The superior and inferior articular processes extend from the lateral corners of the lamina on either side and bear smooth facets for articulation with the vertebrae above and below to form the apophyseal joints (Jenkins, 1998; Adams *et al.*, 2002).

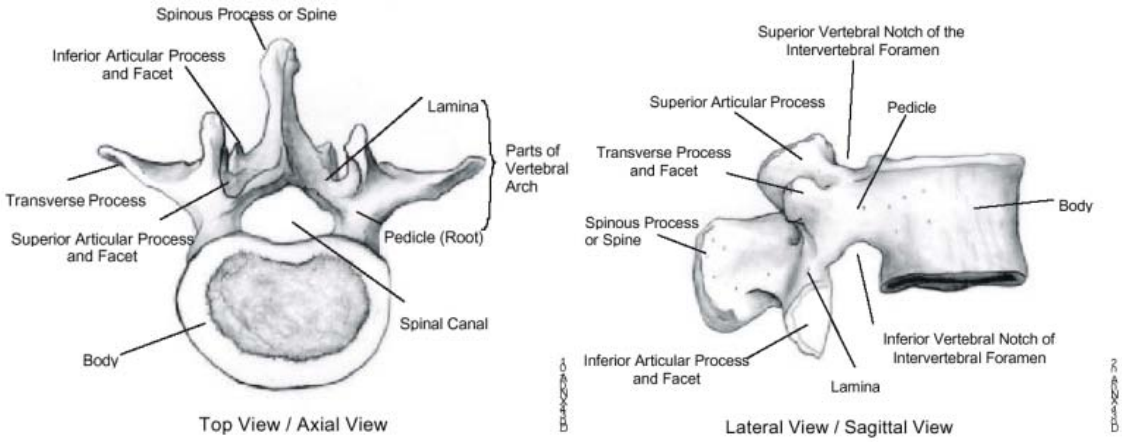


Figure 2.3. A typical vertebra, superior and lateral view (NCBI Bookshelf, 2001)

2.2.3. *Anatomy and function of the spinal joints*

There are three joints connecting the vertebrae: two are synovial gliding joints formed from the inferior articular processes of the upper vertebrae and the superior articular processes of the lower vertebrae. These are called apophyseal, zygapophysial or facet joints (Bogduk, 2005). The direction and range of motion at the joint is determined by the orientation of the articular surfaces. The apophyseal joints aid spinal stability, resist horizontal forces acting on the spine and protect the discs from excessive shear and torsion (Adams *et al.*, 2002). They are also weight-bearing, particularly following disc narrowing, although evidence regarding the extent to which this occurs has found fairly wide-ranging results (Middleditch & Oliver, 2005). The intervertebral joints are cartilaginous joints between the vertebral bodies, formed by the union of the intervertebral discs with the vertebral bodies. These are reinforced anteriorly and posteriorly by the longitudinal ligaments.

2.2.4. *Anatomy and function of the spinal ligaments*

The ligamentous structures of the spine contribute to its intrinsic stability (Bogduk, 2005). The intervertebral discs are reinforced and protected by the longitudinal ligaments, which

run the length of the vertebral column anteriorly and posteriorly and give it important support (Cailliet, 1991). Their function is to prevent excessive separation of the posterior and anterior ends of the vertebral bodies. The deepest fibres extend from one vertebra to the next while more superficial fibres extend over several vertebrae (Jenkins, 1998; Middleditch & Oliver, 2005).

The ligaments of the posterior elements are the ligamentum flavum, the interspinous ligament and the supraspinous ligament. The supraspinous ligaments join the tips of the spinous processes, the interspinous ligaments connect adjoining spinous processes and the ligamentum flavum is a short, thick ligament that joins the laminae of each consecutive vertebra (Middleditch & Oliver, 2005). It is thought that its elastic nature helps restore a flexed lumbar spine to its natural extended position (Bogduk, 2005). In addition, there are intertransverse ligaments which connect adjacent transverse processes and which are most prominent in the lumbar region (Jenkins, 1998).

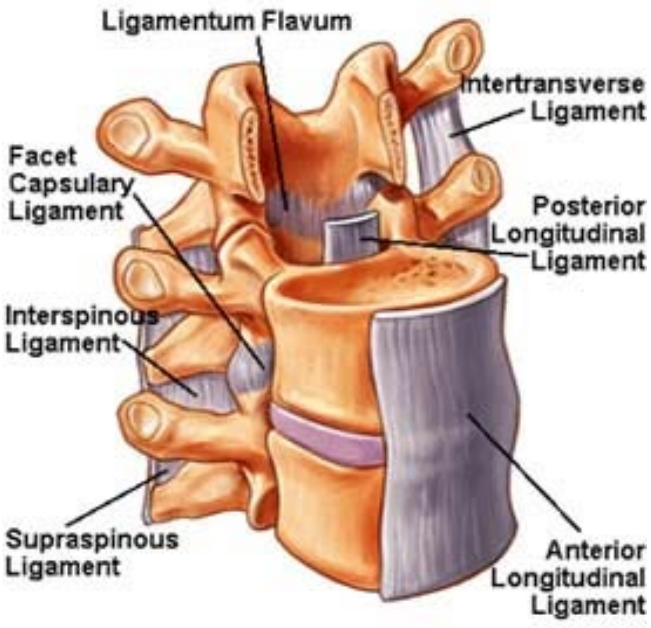


Figure 2.4. The ligaments of the spinal column (SpineUniverse, 2010)

2.2.5. *Type and function of the lumbar paraspinal muscles*

The lumbar muscles can be divided into three groups: the posterior, the intertransverse, and the anterolateral muscles (Adams *et al*., 2002).

Posterior muscles

The erector spinae is a large powerful musculotendinous mass, which gained its name due to its action in extending the vertebral column (Jenkins, 1998; Middleditch & Oliver, 2005). It consists of three muscles, which are (from lateral to medial): iliocostalis, longissimus and spinalis, only the first two of which are well developed. It extends the length of the spine, from skull to sacrum and lies lateral to multifidus. It acts bilaterally to extend the vertebral column and unilaterally to create lateral flexion (Middleditch & Oliver, 2005).

The multifidus is the largest and deepest of the lumbar back muscles (Bogduk, 2005) and is thought to be important for spinal stability. The muscle fibres arise from the sacrum and transverse processes of the lumbar and lower cervical vertebrae to insertions on the spinous processes (Jenkins, 1998). The arrangement of the fibres allows the multifidus to act on each spinous process individually and the muscle acts to pull downwards on the processes causing extension of the vertebral column, controlling flexion and acting as a stabiliser during rotation (Adams *et al.*, 2002; Middleditch & Oliver, 2005).

As their name suggests, the interspinales connect the edges of opposing spinous processes.

Intertransverse muscles

The intertransversarii span adjacent transverse processes. Along with the interspinales, these intersegmental muscles have a large number of muscle spindles, and hence are thought to have an important proprioceptive role (Adams *et al.*, 2002).

Anterolateral muscles

Quadratus lumborum is a wide, quadrilateral muscle, deep to erector spinae, that connects the lumbar transverse processes, the ilium and 12th rib. The exact function of this muscle is unclear, although it is thought to support the 12th rib during respiration and control the rate of descent during lateral flexion (Bogduk, 2005; Middleditch & Oliver, 2005).

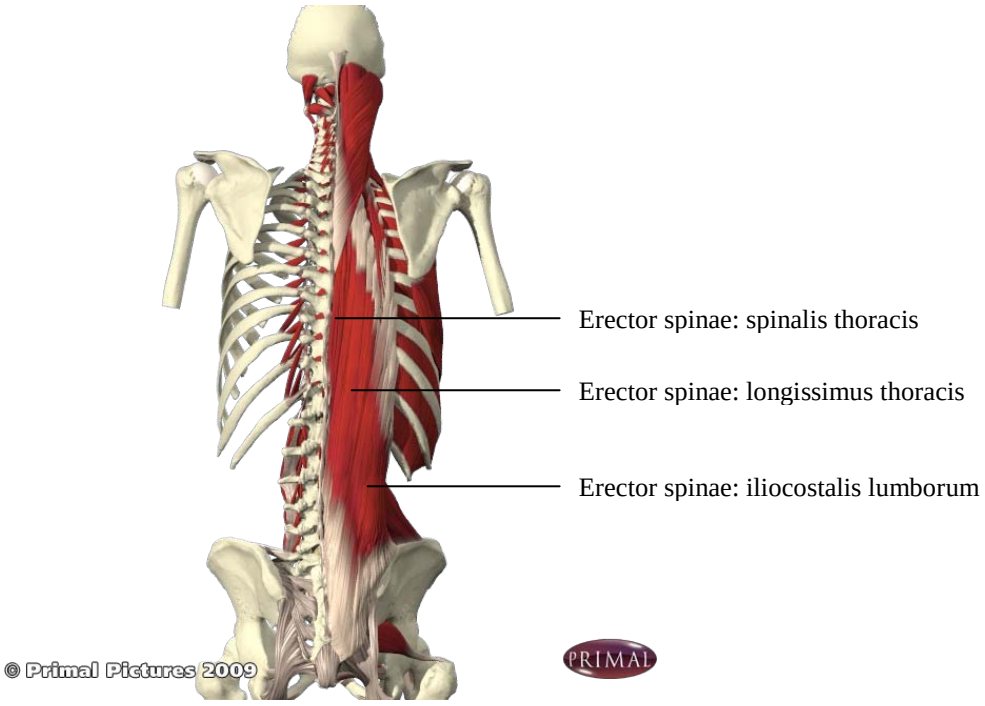


Figure 2.5. The erector spinae group, posterior view (Primal Pictures, 2009).

Psoas major is a long muscle which arises from the anterolateral aspect of the lumbar spine and inserts into the lesser trochanter of the femur. The lumbar vertebral column acts as a base as the muscle performs its principal action of flexing the hip, thought to cause very

large compressive forces on the lumbar discs. However, being too close to the axes of rotation of the lumbar vertebrae, the psoas is not able to exert a substantial moment on the lumbar spine with respect to flexion and extension (Adams *et al.*, 2002; Middleditch & Oliver, 2005).

Classification

The back muscles may be categorised into local, or deep, muscles (e.g. multifidi, interspinales, and intertransversarii) that connect individual lumbar segments, and global, or superficial, muscles (e.g. erector spinae) that don't attach directly to the spine and primarily act to generate movement (Bergmark, 1989). The intersegmental nature of the deep back muscles makes them well-suited for stabilisation of the vertebral column and 'fine-tuning' of movements (Middleditch & Oliver, 2005). Meanwhile, the superficial muscles, being larger in size and further from the centre of rotation, are better adapted to counterbalance external loads, and have the greatest potential to generate torque to move the trunk (Hodges & Moseley, 2003; Middleditch & Oliver, 2005).

2.2.6. *Structure and function of the intervertebral disc*

The intervertebral discs lie between the vertebral bodies, forming approximately one-third of the height of the spinal column (Reilly, Boocock, Garbutt, & Troup, 1988). The discs consist primarily of proteoglycans (large molecules that have the property of attracting and retaining water), collagen and water (Middleditch & Oliver, 2005). At the centre of each disc is a gelatinous mass, known as the nucleus pulposus, which consists of around 80 – 90% water (Park, 1997) and is primarily involved in weight-bearing (Bogduk, 2005). Surrounding this is the annulus fibrosus, which consists of 10-20 sheets of collagen, called lamellae, tightly packed together around the periphery of the disc (Adams *et al.*, 2002). This acts to restrain movement and aid stability (Bogduk, 2005). The third component of

the disc is the vertebral endplates; plates of cartilage that separate the discs from the adjacent vertebral bodies (Middleditch & Oliver, 2005). Disc height varies in the different regions of the spine, being thickest in the lumbar region. The discs are also wedge-shaped in the cervical and lumbar regions, helping to form the lordotic curves (Middleditch & Oliver, 2005). The discs provide flexibility, enabling bending, flexion and torsion. They also transmit loads arising from body weight and muscle activity through the spinal column (Urban & Roberts, 2003) and distribute compressive loading evenly onto the vertebral bodies (Adams, 2004).

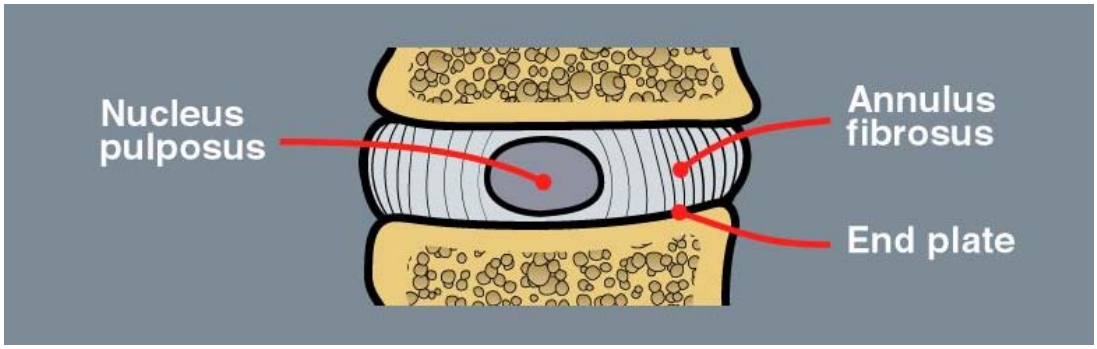


Figure 2.6. An intervertebral disc, consisting of a nucleus pulposus surrounded by an annulus fibrosus, both sandwiched between two cartilaginous vertebral endplates (Primal Pictures, 2009)

2.3. Stature loss and stadiometry

2.3.1. *Behaviour of the spinal motion segment when exposed to loading*

Intervertebral discs have been observed to demonstrate viscoelastic deformation in response to compressive forces. Over short periods of time, the disc behaves elastically in response to loading, but a slow compression of the disc known as ‘creep’ also occurs over longer periods (Smeathers, 1984). Two main causes of disc height loss have been identified. First, the disc contains a high percentage of water, which flows into and out of

the disc in response to loading until a new equilibrium is reached. Fluid loss that occurs in this way will lead to a reduction in disc volume and hence a decrease in disc height (Adams & Hutton, 1983). Second, height loss occurs due to elastic deformation of the disc and vertebral endplates. The annulus fibres bulge radially in response to loading (Heuer, Schmitt, Schmidt, Claes, & Wilke, 2007; Park, 1997) and the vertebral endplates bulge vertically into the vertebral bodies (Brinckmann, Frobin, Hierholzer, & Horst, 1983). When the spine is unloaded (typically overnight or when lying down), these processes are reversed, leading to fluid inflow and disc height recovery (Koeller *et al*., 1984). It is believed that this fluid flow induced by loading and unloading of the spine is important for the nutrition of the discs (Adams & Hutton, 1983).

Several *in vivo* studies have used Magnetic Resonance Imaging (MRI) in an attempt to directly measure diurnal changes in the lumbar spine and have observed mean disc height changes of 0.9-1.2mm (Boos, Wallin, Aebi, & Boesch, 1996; Botsford, Esses, & Ogilvie-Harris, 1994; Park, 1997). In addition, the volume of the three lower lumbar discs has been found to decrease by approximately 16-17% over the course of a day (Botsford *et al*., 1994; Roberts, Hogg, Whitehouse, & Dangerfield, 1998), supporting the fluid loss mechanism. The presence of radial bulging in these studies has been less clear, with Park (1997) reporting bulging of approximately 9%, whereas Botsford *et al.* (1994) conversely found a decrease in disc diameter. The low participant numbers in all of these studies, however, means that caution is needed in interpreting the results, none having more than ten participants. To date, the use of MRI to evaluate the effects of shorter-term loading appears to be limited, although Kimura, Steinbach, Watenpaugh and Hargens (2001) used MRI to scan the lumbar region of eight participants following five minutes of axial loading of 50% of body weight (to simulate upright posture). MRI was performed with the axial force still applied and a decrease in spine length (from T12 to S1) of 2.5mm was observed.

The difficulty in measuring individual disc dimensions *in vivo* has led to a number of *in vitro* experiments to investigate the effect of loading on cadaveric discs. However, the validity of these studies has been questioned due to the different physiologic and mechanical properties of discs in live compared to cadaver animals (Keller, Holm, Hansson, & Spengler, 1990) and the difficulty in replicating physiological forces (Botsford *et al*., 1994). Adams, McMillan, Green and Dolan (1996) found mean lumbar disc height loss of 1.2mm after two to three hours of loading (1-2kN) that simulated sustained manual labour and Adams and Hutton (1983) reported a decrease of approximately 1.5mm after four hours of creep loading at around 700N, which was accompanied by fluid loss of approximately 11%. The authors concluded that approximately two-thirds of the average height loss was due to fluid loss, the remaining one-third being due to creep deformation of the vertebrae and annulus fibrosus. The contribution of each mechanism is also thought to be time-dependent. This was supported by the results of a mechanical model of lumbar disc function, which showed that deformation of annulus fibres was the primary mechanism initially, but contributed only approximately one quarter of the height change obtained after several hours' normal activity (Broberg, 1993).

The magnitude of disc height loss varies greatly between individuals and has been found to be dependent on factors such as age, gender, disc cross-sectional area and disc degeneration (Althoff, Brinckmann, Frobin, Sandover, & Burton, 1992; Kanlayanaphotporn, Lam, Williams, Trott, & Fulton, 2001; Natarajan & Andersson, 1999). In particular, degenerated discs demonstrate reduced stiffness and lose more height in response to loading. However, due to reduced proteoglycan content, the fluid loss is relatively less than for non-degenerate discs (Adams & Hutton, 1983), implying that

degenerated discs have reduced stiffness of the annulus fibres (and possibly the endplate and underlying bone) (van Dieën & Toussaint, 1993).

Disc height loss results in a measurable decrease in stature, both in response to short term loading tasks and over the course of a day. This can be exacerbated by an increased lordosis following loading (De Puky, 1935; Kimura *et al.*, 2001). Mean circadian variation has been found to be of the order of 1.1% of stature, with approximately 50% of the diurnal loss of stature occurring in the first hour after rising and approximately 70% regained during the first half of the night (Tyrrell, Reilly, & Troup, 1985).

2.3.2. *Clinical relevance of disc height loss*

Decreased disc height affects spinal mechanics and causes increased loading on spinal structures such as the facet joints (Dunlop, Adams, & Hutton, 1984). A height loss of approximately 1mm was observed to increase the percentage of intervertebral compressive load that is borne by the apophyseal joints from 4% to 16% (Adams & Hutton, 1980). Within the disc, the associated water loss causes a transfer of load from the nucleus to the annulus, which can result in stress concentrations in the posterior annulus, possibly leading to pain and structural disruption (Adams *et al.*, 1996). At the same time, the space for the spinal nerve roots is reduced by the diminished height and diameter of the intervertebral foramen, thereby increasing the risk of nerve root impingement (Adams *et al.*, 2002). Furthermore, reduced disc height results in some slack in the annulus and intervertebral ligaments, allowing increased flexion, lateral bending and translation, and potentially comprising spinal stability (Zhao *et al.*, 2005). The risk of injury may also be increased due to the decreased shock absorbing properties of the disc (Koeller *et al.*, 1984) and as a result of delayed reflex activation of the back muscles, which has been observed following spinal creep (induced via sustained flexion) (Sánchez-Zuriaga, Adams, & Dolan, 2010). Some

LBP treatments have recognised the potential for pain generation resulting from reduced disc height. For example, spinal traction is a relatively popular procedure used to treat and alleviate the symptoms of LBP by the application of a force to temporarily increase intervertebral disc height and hence reduce pressure and overloading on spinal structures (Rodacki, Weidle, Fowler, Rodacki, & Persch, 2007). Non-invasive spinal decompression has also been associated with a significant reduction in LBP, which was correlated with an increase in disc height (Pergolizzi, Apfel, Florio, & Richmond, 2008), although further research would be required to prove a causative relationship.

As discussed in more detail below, LBP patients tend to demonstrate delayed or reduced recovery of stature following loading (Healey *et al.*, 2005a). This potentially exposes them to increased pain and a greater risk of further injury. It has been suggested that individuals with LBP may also exhibit reduced diurnal variation of stature. In support of this, Reilly *et al.*, (1988) reported a diurnal variation for five CLBP patients awaiting surgery that was approximately 40% of that of healthy participants (although the results were clearly based on a limited sample size). However, it was noted that the patients took periods of rest to alleviate their symptoms and hence the reduced variation may have been the result of lower levels of spinal loading throughout the day. Although little detail is given on the pain and disability levels of these patients, it is assumed that their back pain was relatively severe since they were awaiting surgery and the authors mention that difficulties arose during training “due to excessive pain”. A more recent study (Fowler & Healey, 2008) found no significant difference between the diurnal variation in stature for participants with mild CLBP and healthy controls. Interestingly, Hupli, Heinonen and Vanharanta (1997) found that an active rehabilitation programme increased both the morning and evening heights of patients with relatively mild CLBP, with an average morning increase of 7.2mm after 2.5 weeks. However, the daily variation did not change significantly.

2.3.3. *Stadiometry*

The stadiometer was popularised by Eklund and Corlett and was designed to accurately measure stature change or 'spinal shrinkage', which the authors suggested could be used to measure disc compression and hence quantify spinal loading (Eklund & Corlett, 1984). Most stadiometers are modifications of the one developed by Eklund and Corlett and generally consist of a frame that inclines the body backwards at an angle of between 5-15°, allowing the participant to adopt a relaxed position. A number of supports or markers control the position of the body, with various approaches adopted to control the head position. Participants are typically trained on the stadiometer beforehand until they are able to relax in the apparatus, as repeatability has been observed to improve following such familiarisation sessions. The criterion used for determining adequate training varies between researchers. Most commonly, training continues until the standard deviation (SD) of ten successive measurements is ≤ 0.5mm, the reasoning for this apparently being that it represents an achievable target, while being significantly less than the stature change observed in most stadiometer studies. A number of studies have involved measuring participants in the seated position, which has the potential advantage of reducing the extent to which height loss in areas other than the spine, such as the heel pad, are included. However, loss of stature measurements in the two postures has not been found to be significantly different (McGill, van Wijk, Axler, & Gletsu, 1996), supporting the argument that the majority of stature change is in fact due to spinal height changes. Furthermore, participants appear to require more training on the seated stadiometer in order to achieve adequate repeatability (Rodacki, Fowler, Rodacki, & Birch, 2001).

Reilly *et al.*, (1988) raised concern over the reliability of stature measurements of LBP patients. In observations of eight patients, the authors reported that excessive pain when trying to relax within the apparatus meant that only five were able to achieve the training

standard of SD ≤ 0.5mm over ten consecutive measurements. These patients were all awaiting surgery, however, and hence the results may not be generalisable to typical patients with non-specific mechanical LBP. A more recent study (Healey, Fowler, Burden, & McEwan, 2005b) reported that both CLBP patients and asymptomatic individuals were able to produce stature measurements with a good level of repeatability after appropriate prior practice and also that this ability was retained for at least two weeks. Since many stadiometer studies involve testing on more than one day, the day-to-day variation in stature change response to loading and unloading is also important, although research in this area is extremely scarce. Leivseth and Drerup (1997) measured the vertical spinal creep (VSC) response to work in standing and sitting postures using a standing stadiometer on two consecutive days and found low intra-individual variation (with reported means of SDs of 0.51mm and 0.64mm for the standing and sitting activities respectively). Kanlayanaphotporn, Williams, Fulton and Trott (2002) investigated the reliability of a VSC response over a 25-minute loading and unloading protocol on two consecutive days. Five patients with LBP and an asymptomatic group of ten individuals were included and the results demonstrated that both groups had good levels of repeatability. Although the response was found to vary between days, the variation (SEM and mean of SDs) was less than 2mm in both cases.

2.3.4. *Measurement of spinal loading*

The reduction of loading on the spine is considered to be beneficial in decreasing the incidence or severity of LBP. *In vivo* measurement of spinal loading is difficult, however, and has involved invasive techniques such as intradiscal pressure measurements (using transducer needles or an implanted transducer) (Sato, Kikuchi, & Yonezawa, 1999; Wilke, Neef, Caimi, Hoogland, & Claes, 1999*)* or surgically implanted spinal fixators equipped with load cells (Rohlmann *et al*., 2001). As a consequence, a number of researchers have

developed various biomechanical models, the most complex of which use EMG to determine the activity of each relevant muscle (e.g. the Marras group and the McGill group). This approach is validated, for example, by the findings of pressure-needle studies which have confirmed that most of the compressive force acting on the lumbar spine arises from muscle action (Adams & Dolan, 1995).

Since intervertebral disc height loss (and hence stature loss) has been shown to be greater for greater loads, spinal loading may alternatively be quantified by measuring changes in disc height (or stature). Radiography, ultrasound and MRI have all been used to image the spine to allow disc height measurement, or, more simply, total stature change has been assessed via stadiometry. This approach is supported by findings which have suggested a linear relationship between stature loss and static axial loading of the spine (e.g. Althoff *et al.*, 1992). Furthermore, a recent study identified a significant and very high ($r = 0.99$) correlation between spinal shrinkage and intradiscal pressure during static situations and walking (van Deursen, van Deursen, Snijders, & Wilke, 2005). Criterion validity is additionally demonstrated by the finding that spinal shrinkage is significantly related to biomarkers of type I collagen metabolism, which are used to assess the alterations of spinal tissues in response to physical loading (Kuiper, van Dieën, Everts, Verbeek, & Frings-Dresen, 2004). The apparent relationship between spinal shrinkage and perceived discomfort (Beynon & Reilly, 2001; Corlett, Eklund, Reilly, & Troup, 1987; Troup, Reilly, Eklund, & Leatt, 1985) also implies concurrent validity.

2.4. Paraspinal muscle activity

2.4.1. *Electromyography*

Electromyography (EMG) is a technique for assessing muscular activation by recording changes in the electrical potential of muscle fibres (Burden, 2008). The measurement can be taken from within the muscle using needle EMG, or via less invasive surface EMG. A review carried out by Mohseni-Bandpei, Watson and Richardson (2000) considered the use of surface EMG in assessing lumbar muscle activity in static and/or dynamic positions. Of the 14 studies included, all except two reported favourable results, leading the authors to conclude that surface EMG is a reliable technique for assessing lumbar muscle function and acceptable for clinical application in LBP populations.

Absolute EMG amplitudes are affected by a variety of factors. Those which have an elemental influence on the EMG signal, known as causative factors, have been classified by De Luca (1997) as either extrinsic or intrinsic. Extrinsic factors include the electrode structure and its orientation and location on the skin, which, in particular, will affect the cross-talk from neighbouring muscles. Intrinsic factors include "the physiological, anatomical and biochemical characteristics of the muscle" and include the thickness of the subcutaneous fat layer, the fibre type composition and their depth and location in respect to the electrodes and the blood flow in the muscle, which will be affected by muscle temperature. In addition, variations in temperature may influence the EMG signal via changes in sweat levels, which will alter the skin impedance. Many of these factors, which will affect the amplitude recorded, can not be controlled for and hence there is an argument that comparison of EMG amplitudes between individuals should not be carried out directly (Burden, 2008). In particular, when the comparison involves LBP patients and asymptomatic controls, differences in physical activity levels may lead to variations in

body fat (unless adequate matching is undertaken), and patient groups are often older than control groups due to the difficulty of recruiting older participants with no history of LBP.

The traditional approach to the issue of inter-individual differences is to normalise the EMG data relative to the amplitude achieved during a maximum voluntary contraction (MVC). However, this method is dependent on the motivation of the individual to perform a true maximal contraction and again this is a particular issue for LBP patients who have often proven unwilling or unable to perform a maximal contraction due to (fear of) pain/(re)injury (Al-Obaidi, Nelson, Al-Awadhi, & Al-Shuwaie, 2000). The generally accepted approach in this case is therefore to normalise the data relative to a sub-maximal reference voluntary contraction (RVC). Lehman (2002) considered between-day repeatability of EMG activity of the erector spinae muscle during quiet standing. Eight healthy participants were tested on three separate days. Both MVC and RVC normalisation techniques were observed to improve the repeatability of the EMG signal, although even non-normalised trials demonstrated excellent repeatability. Similar results were found by Healey (2005) who found good between-days repeatability of integrated EMG measurements during standing and a RVC for CLBP patients and controls. Normalising relative to a RVC was found to improve the level of repeatability for both groups. Dankaerts, O'Sullivan, Burnett, Straker and Danneels (2004) compared the reliability of surface EMG from both MVCs and RVCs in six healthy controls and five CLBP patients. Although excellent within-day reliability was observed for both contractions, the between-day results revealed good reliability for the RVC compared to poorer levels for the MVC. The observed pattern was consistent for both healthy controls and CLBP patients. The authors therefore recommended the use of a sub-maximal contraction for amplitude normalisation.

Conversely, van Dieën, Selen and Cholewicki (2003b) concluded that non-normalised EMG amplitudes are preferable for clinical studies. In addition to the problems of achieving a maximal contraction, the authors argue against the use of a RVC since the EMG activity during the reference contraction may be affected in the same manner as the levels during the experimental task. Instead they stress the importance of careful matching with respect to factors such as body mass, body mass index or skinfold thickness and the need for adequate group size to maintain power despite random variance in raw EMG amplitudes. A number of studies have adopted this approach of attempting to reduce inter-individual differences by controlling for confounding factors instead of (or as well as) normalising EMG data.

2.4.2. *Hyperactivity of muscle activity*

A considerable body of research has focused on the issue of whether superficial trunk muscle activity (as recorded by either surface or intramuscular EMG) is increased in individuals with LBP compared to asymptomatic controls during static postures. The findings have been conflicting and inconclusive. While a number of studies have seemingly demonstrated that patients with LBP have higher paraspinal muscle activity than healthy controls (e.g. Ambroz *et al.*, 2000; Flor, Birbaumer, Schugens, & Lutzenberger, 1992; Healey *et al.*, 2005a), others have found no difference or even the converse (e.g. Collins, Cohen, Naliboff, & Schandler, 1982; Glombiewski, Tersek, & Rief, 2008). In some cases, results have been mixed: for example, Kravitz, Moore and Glaros (1981) found no significance difference between groups at rest, but when contracting other muscles, the patients with LBP exhibited higher levels of lumbar muscle activity compared to controls. Differences in EMG technique may explain some of the discrepancies, one of the main issues being the normalisation of EMG data, as mentioned above. It is notable that two recent studies which controlled for potential confounding factors found a clear

difference between the EMG activity of CLBP patients and asymptomatic controls. Ambroz *et al.* (2000) included 30 CLBP patients and 30 controls matched for BMI, sex and age, and found static EMG activity to be three times greater for the CLBP patients than the controls. Healey *et al.* (2005a) considered 20 CLBP patients and 20 controls matched for body mass, physical activity level, sex and age and also normalised EMG levels to a RVC. Resting EMG levels were found to be significantly higher than for controls, despite the patient group only having mild back pain.

The apparent inconsistency of results may also be due to individual patterns of hyperactivity which only become evident in certain postures. For example, Sherman (1985) compared paraspinal EMG activity of LBP patients and controls during six positions (both static and dynamic) and found that each of the 83 LBP patients showed elevated muscle activity (exceeding two SDs above the control mean) during at least one position, with each individual having a consistent, unique profile. However, when analysing the data for each task, no differences between patients and controls were found. In a review paper, Roland (1986) concluded that, although LBP patients tend to have increased spinal muscular activity at rest or following exercise, in no study were patients with LBP found to have increased back muscle activity under all conditions. Subsequent reviews (Geisser *et al.*, 2005; van Dieën *et al.*, 2003b) have similarly found fairly strong evidence that increased muscle activation can occur at rest in the LBP patient population, but that this appears to be dependent on the posture considered, with the largest effect size found for standing.

Research findings may also be affected by the pain state at the time of the investigation. Often this is either not reported or participants are in remission during testing (Sherman, 1985), possibly affecting results. Sherman (1985) found that for nine of the eleven

participants who reported changes in pain intensity between recording sessions, there was a positive correlation between the reported intensity of pain and the level of contraction in the one position most different from the control group. Arena, Sherman, Bruno and Young (1991) recorded the muscle activity of 46 LBP patients and 20 asymptomatic controls during six positions and identified a non-significant trend for patients to have higher EMG levels during a higher pain state. However, pain state only became significant when considering the position that gave the highest EMG levels relative to the group mean for each individual during high pain and then comparing that position during low pain. The authors suggested that a time delay may exist between muscle activity levels and pain, although as yet, there is no conclusive evidence for either an immediate or a lagged relationship between EMG activity and pain in LBP patients.

Another issue that has attracted criticism is the tendency to combine all back pain patients into a single group. The potential disadvantage of this approach was highlighted by Arena, Sherman, Bruno and Young (1989) who sub-classified the back pain patients into a number of different diagnostic categories and observed that, in various positions, muscle activity differed significantly between subgroups. Similarly, in a study considering muscle activity during sitting, Dankaerts, O'Sullivan, Burnett and Straker (2006) demonstrated that when non-specific LBP patients were included as a single group, there was no difference in superficial trunk muscle activity compared to healthy controls. However, when the patients were then sub-classified into Flexion Pattern (FP) (patients position themselves near end-range flexion during sitting) and Active Extension Pattern (AEP) (patients hold themselves actively into hyperextension), the AEP subgroup exhibited significantly higher levels of muscle co-activation compared with the controls and FP groups. The authors concluded that when non-specific LBP patients are pooled, the findings in one subgroup of patients may be ‘washed-out’ by the opposite findings in another subgroup, potentially leading to

differences between patients and controls being overlooked. Key, Clift, Condie and Harley (2008) have suggested a similar classification system based on clinical observation. They report that back pain patients commonly demonstrate consistent underactivity of the deep system but show variable overactivity of the superficial system, with some patients having a tendency for low tone, while others demonstrate more hyperactive tone with general tension and stiffness. Both classification systems have therefore identified sub-groups of LBP patients that tend to exhibit superficial trunk muscle hyperactivity.

2.4.3. *Flexion-relaxation phenomenon (FRP)*

There is one posture in which LBP patients consistently demonstrate increased paraspinal muscle activity compared to healthy controls and that is in full flexion. As described by Floyd and Silver (1955), most healthy participants exhibit relative electrical silence in the paraspinal muscles in full flexion (known as the FRP). The required supporting moment is thought to be primarily generated by the passive tissues, particularly the spinal ligaments, although the extensor muscles may generate force elastically through passive stretching of the series elastic elements (McGill & Kippers, 1994) and some deep muscle activation has been demonstrated (Andersson, Oddsson, Grundström, Nilsson, & Thorstensson, 1996). The flexion-relaxation response has consistently been shown to be absent in patients with LBP, who instead continue to exhibit activation of superficial trunk muscles during full flexion (Watson, Booker, Main, & Chen, 1997b). This is illustrated by comparison of the flexion-relaxation ratio (FRR), which compares paraspinal muscle activity during forward flexion and the activity at full flexion and has been shown to reliably discriminate between LBP patients and healthy controls (Watson *et al.*, 1997b). The reason for this elevated muscle activity in patients is not known, although it has been suggested that it may be either a conscious or unconscious guarding action due to (fear of) pain or a reflex from injured spinal ligaments (Fryer, Morris, & Gibbons, 2004). However, it has also been

found to be predictive of future LBP in pregnant women (Sihvonen, Huttunen, Makkonen, & Airaksinen, 1998). The absence of flexion-relaxation in the lumbar paraspinal muscles correlates strongly with back pain (Sihvonen, Partanen, Hänninen, & Soimakallio, 1991) and a 'normal' response has been observed to return following treatment that consisted of exercise progression and disability management (Mayer, Neblett, Brede, & Gatchel, 2009; Neblett, Mayer, Gatchel, Keeley, Proctor, & Anagnostis, 2003). A significant increase in the FRR of LBP patients, towards that seen in healthy participants, was also observed after an intensive 12-week functional restoration programme of physical conditioning, working conditioning and work readiness (Mak *et al.*, 2010). Watson, Booker and Main (1997a) similarly reported that, following a pain management programme, CLBP patients demonstrated reduced muscle activity at full flexion and a significant increase in the FRR. Although paraspinal muscle activity during standing also showed a reduction, this did not reach significance, although this may have been because the initial static activity levels for this particular patient group were not significantly different to those of a matched control group. Mannion, Taimela, Muntener and Dvorak (2001b) conversely reported no significant change in EMG levels during flexion after any of three randomly assigned treatments (active physiotherapy, muscle reconditioning or low-impact aerobics). However, they did report altered back muscle activation during trunk extension exercises, which they suggest may be due to increased confidence in using the lumbar muscles and hence a decrease in guarding mechanisms.

Loss of flexion-relaxation has also been demonstrated following induced experimental pain, even when participants were instructed to maintain the same velocity and range of motion as in pain-free conditions, suggesting that the change was more than just a voluntary response (Zedka, Prochazka, Knight, Gillard, & Gauthier, 1999). The authors instead proposed that pain caused a change in the strategy of the central nervous system,

which resulted in the muscle working in a protective 'pain mode'. Similarly to the lack of flexion-relaxation, patients with LBP show significantly increased paraspinal EMG activity in the swing phase during gait; a phase where lumbar muscles are normally silent (Arendt-Nielsen, Graven-Nielsen, Svarrer, & Svensson, 1995; van der Hulst, Vollenbroek-Hutten, Rietman, & Hermens, 2010a). Similar EMG patterns have been found following induced pain (Arendt-Nielsen *et al.*, 1995; Lamoth *et al.*, 2004), but not fear of pain (Lamoth *et al.*, 2004), suggesting that again the altered gait is more complex than a voluntary guarding strategy.

2.4.4. *Altered muscle patterns*

Analysis of EMG levels during dynamic movements has led to mixed results, with some studies observing a greater tendency for reduced activity for LBP patients compared to controls (Roland, 1986). The clearest finding is that of reduced muscle activity during re-extension after full flexion (Geisser *et al.*, 2005), with the ratio of EMG activity during flexion and extension being significantly different between patients and controls (Sihvonen *et al.*, 1991). This was also observed following induced pain (Zedka *et al.*, 1999). In addition to variations in absolute levels of muscle activity, asymmetry of muscle activity and altered muscle recruitment patterns have been found to accompany LBP, both in static postures and during movements. Hoyt *et al.*, (1981) found that LBP patients showed significantly greater left-right erector spinae difference during standing than pain-free controls, although there was no significant difference in either the semi-Fowler's or sitting position. Using large-array surface EMG, Finneran, Mazanec, Marsolais, Marsolais and Pease (2003) demonstrated that not only did a LBP population of 28 patients have significantly higher EMG activity than 163 pain-free controls in static postures, but 93% of the controls had symmetrical back muscle function, whereas 89% of the LBP population had abnormal images, showing more asymmetrical and/or multifocal patterns of activity.

The images were found to return to normal in the three patients reporting full recovery from pain during the six-week follow up period. Extending this to dynamic lumbar motion, Hu, Siu, Mak and Luk (2010) used surface EMG topography to investigate patterns of muscle activity in 15 LBP patients and 20 healthy controls during flexion-extension. The dynamic topography of the healthy controls was found to have a consistent symmetric pattern whereas LBP patients had a significantly different topography with an obviously more asymmetric or disorganised pattern. After physiotherapy rehabilitation, the images of the LBP patients tended towards the normal pattern.

EMG activity has been shown to be generally higher for LBP patients than controls during upright isometric trunk extensions, with significant differences between the painful and painless side (Alexiev, 1994). Additionally, during rotation, patients have shown hyperactivity of the muscles contralateral to rotation direction (Ahern *et al.,* 1988), suggesting protective guarding. There are also reports that using EMG biofeedback to teach a more symmetrical EMG pattern to a patient results in pain reduction (e.g. Jones & Wolf, 1979).

Asymmetrical muscle activation may be the result of muscle underactivity, (e.g. due to pain, reflex inhibition or selective muscle atrophy) or hyperactivity (e.g. due to reflex spasm, as a splinting mechanism to protect a painful spinal segment, or as compensation for underactivity of other muscles) (Adams *et al.,* 2002). For example, LBP patients may attempt to avoid pain by avoiding activating the painful muscles or by stiffening the joints by activating the surrounding muscles, both of which will lead to abnormal muscle patterns (Hermens & Hutten, 2002). Asymmetrical muscle activation may be clinically significant, possibly leading to stress concentrations within the disc and neural arch. It is suggested that either left-right asymmetries, or flexor/extensor imbalances in the back muscles, could

potentially result in back pain if they are sufficiently marked to change the angulation of adjacent lumbar vertebrae by at least two degrees (Adams *et al.*, 2002).

2.4.5. *Pain-spasm-pain model*

Hyperactivity of the back muscles is consistent with the pain-spasm-pain model, formally proposed by Travell *et al.* (1942), who suggested that ischemic pain is the consequence of a sustained spasm of the skeletal musculature, and may itself cause reflex muscle spasm, leading to a self perpetuating, pain-producing cycle. There is evidence that pain and injury can lead to muscle hyperactivity. A number of studies have observed muscle spasms caused by stimulation or deformation of the annulus fibrosus or spinal ligaments in animals and this has also been demonstrated to a limited extent in humans (Indahl, Kaigle, Reikerås, & Holm, 1997; Solomonow, Zhou, Harris, Lu, & Baratta, 1998). Muscle spasm may result in reduced blood flow, leading to an accumulation of metabolic waste products and inflammatory substances in the muscles, which may increase pain sensitivity (Lundberg *et al.*, 2002). Consistent with the model, reduced tissue oxygenation in the lumbar extensor muscles has been shown to result from muscle contractions as low as 2% of MVC (McGill, Hughson, & Parks, 2000) and muscle relaxants have been shown to increase paraspinal muscle blood flow in LBP patients (Sakai *et al.*, 2008). Indeed, muscle hyperactivity and its clinical importance appear to be assumed in a number of both pharmacological (e.g. muscle relaxants) and psychophysiological (e.g. biofeedback, relaxation therapy) interventions which are aimed specifically at reducing paraspinal and/or overall body muscle tension (Arena *et al.*, 1991). Furthermore, a recent Cochrane review of muscle relaxants concluded that they were effective in the management of both acute and chronic LBP (van Tulder, Touray, Furlan, Solway, & Bouter, 2003). Despite this, the pain-spasm-pain model has lost popularity, partly due to the inability to consistently demonstrate elevated muscle activity in LBP patients and doubt as to whether

observed increases are of significant magnitude to be clinically relevant. Furthermore, it has become clear that, in some instances, LBP patients tend to show reduced muscle activation, a phenomenon which can not be explained by the model.

2.4.6. *Lack of muscle relaxation*

Perhaps answering one of these issues, there is a body of research (largely focused on work-related musculoskeletal pain), which argues that even low level, but sustained EMG hyperactivity can contribute to the development and maintenance of chronic pain. The lack of muscle relaxation characteristic of patients with chronic pain is suggested as a possible cause (Hägg & Åström, 1997; Hermens & Hutten, 2002). Hägg (1991) proposed the Cinderella principle (resulting from Henneman's principle of orderly recruitment of motor units) by which, even at low levels of muscle activity, single muscle fibres may be intensely active and therefore lack of sufficient relaxation may cause damage and prevent repair of damaged fibres, leading to pain. Following patient training, decreases in pain have been found to be related to an increased ability to relax but not to reductions in the absolute EMG levels (Vollenbroek-Hutten, Hermens, Voerman, Sandsjö, & Kadefors, 2006), suggesting that lack of relaxation may be even more important than the absolute level of muscle contraction. This, therefore, may be one mechanism by which even low level muscle activity can impact on chronic back pain.

2.4.7. *Pain-adaptation model*

A review carried out by Lund *et al*. (1991) concluded that, contrary to Travell *et al.*'s findings, the only situation in which EMG activity appeared to be higher than normal was when the muscle was acting as an antagonist. This led to the authors rejecting the pain-spasm-pain theory and instead proposing the pain-adaptation model. This model states that pain causes the output of agonist muscles to be decreased whilst the level of antagonist co-

contraction is increased, leading to a reduction in MVC and in the range and velocity of movement, thereby protecting the injured part. This concept of the muscles acting to splint or guard the injured part has gained support (Graven-Nielsen, Svensson, & Arendt-Nielsen, 2000), although van Dieën *et al.* (2003b) reviewed the evidence for both the pain-spasm-pain model and the pain adaptation model and concluded that neither was consistently supported by the literature. Although there was evidence for reduced activation in some cases, in line with the pain adaptation model (and contrary to the pain-spasm-pain model), they also found "fairly strong evidence" that increased muscle activation can occur at rest and in full flexion in patient groups and this was interpreted as being contrary to the pain-adaptation model. However, it should be noted that some researchers instead interpret these findings as supportive of the model and suggest that increased co-contraction and stiffening of muscles, even at rest, is part of a general splinting or guarding strategy which is consistent with the pain-adaptation model (Hodges & Moseley, 2003).

2.4.8. *Instability*

Panjabi (1992a) proposed that the spinal stabilising system consists of three interdependent subsystems: the passive system (vertebrae, ligaments and intervertebral discs), active system (muscles and tendons) and the neural control system. The three subsystems work together to provide spinal stability and, within certain limits, deficiency in one system can be compensated for by an increase in another. The 'neutral zone' was defined as that part of the range of motion within which there is minimal resistance to intervertebral motion, increased neutral zone being an indicator of clinical instability (Panjabi, 1992b). Dysfunction in one of the subsystems (caused for example by injury, disease or degeneration) may increase the neutral zone and cause strain on discs and ligaments, possibly leading to a compensatory increase in muscle co-contraction to restore stability. This was supported by the calculation that trunk muscle co-activation of around 1 -3%

MVC was required to maintain stability around a neutral spine, but that this increased to 3 – 6% MVC when the model was run with reduced passive spine stiffness to simulate an injury (Cholewicki, Panjabi, & Khachatryan, 1997). Alternatively, abnormal muscle activation may itself lead to spinal instability. For example, insufficient activation of the deep trunk muscles, such as multifidus and transversus abdominus (TrA), may reduce spinal stiffness, increasing the neutral zone and causing injury to discs and other soft tissues. Hence instability can be both the cause and the consequence of injury (McGill, Grenier, Kavcic, & Cholewicki, 2003). Panjabi's model suggests that alterations in muscle recruitment, in particular, increased muscle co-contraction, may therefore be functional to provide additional stabilisation of the spine. However, these changes may remain present after their functional significance has disappeared and injured structures have recovered (van Dieën *et al.*, 2003b).

Consistent with the instability hypothesis (Panjabi, 1992b), the presence of increased muscle co-activation in people who report LBP is a common finding (Nelson-Wong, Gregory, Winter, & Callaghan, 2008; Radebold, Cholewicki, Panjabi, & Patel, 2000) and a greater ratio of lumbar over thoracic activity has also been observed, a recruitment pattern thought to enhance spinal stability (van Dieën, Cholewicki, & Radebold, 2003a). The disadvantage of adopting a strategy of muscle co-activation to stabilise the spine, is that it also increases spinal load (Granata & Marras, 2000), with the greatest differences in spinal loading between LBP patients and controls being observed during the least taxing biomechanical conditions (Marras, Ferguson, Burr, Davis, & Gupta, 2004). Marras, Davis, Ferguson, Lucas and Gupta (2001) observed that LBP patients experienced 26% greater spine compression and 75% greater lateral shear than an asymptomatic group during controlled exertions, primarily as the result of increased trunk muscle co-activation. The additional force caused by excessive antagonistic muscle activity could exacerbate existing

injuries and cause pain (Adams *et al.*, 2002). Furthermore, during lifting exertions, the erector spinae muscles were found to activate significantly earlier and remained active significantly longer in LBP patients compared to healthy controls (Ferguson, Marras, Burr, Davis, & Gupta, 2004), increasing exposure to potentially injurious circumstances. Previously asymptomatic participants who developed LBP during prolonged standing have been shown to have greater muscle activity and co-activation levels (Gregory, Brown, & Callaghan, 2008; Nelson-Wong *et al.*, 2008) and these higher levels of muscle co-activation were demonstrated immediately on commencement of the standing protocol, prior to any subjective reports of LBP (Nelson-Wong & Callaghan, 2010). This suggests that muscle co-activation may not be entirely adaptive and may in fact be contributory to the problem and predispose some individuals to develop LBP (Nelson-Wong *et al.*, 2008).

The instability hypothesis assumes a relationship between abnormal intervertebral motion and LBP. The idea that decreasing intervertebral motion in a patient with LBP may result in reduced pain is in fact the basis for many low back treatments e.g. fusion, muscle strengthening (Panjabi, 2003). However, support for this concept is not universal. Mulholland (2008) suggests that the problems of patients with chronic back pain tend to be postural, rather than related to the process of movement and proposes that load transfer disturbance is the central issue in mechanical back pain, with abnormal loading pattern and high ‘spot-loading’ potentially causing pain. The author states that artificial discs, which alter load transmission, have had similar clinical success as spinal fusion, used to treat ‘instability’. Adams (2004) also suggests that back pain may arise from abnormal loading and proposes that stress concentrations within innervated tissues may still result in pain even if they are insufficient to cause physical disruption or obvious pathology.

2.4.9. *Integrated pain adaptation model*

van Dieën *et al.* (2003b) concluded that the observed changes in muscle activation in LBP patients are of a more complicated nature than suggested by either the pain-spasm-pain or pain-adaptation models. The authors support the hypothesis on instability suggested by Panjabi (1992b) and propose that the changes are functional adaptations aimed at reducing noxious tensile stresses in injured structures by limiting range of motion and providing stabilisation of the spine. This concept of functional alterations in trunk muscle recruitment which are both highly variable within individuals and task dependent has been largely accepted as the current view (Koumantakis, 2006) and has been formalised by Murray and Peck (2007) as the Integrated pain adaptation model, in which individuals develop unique muscle recruitment strategies aimed at maintaining homeostasis and minimising pain.

2.4.10. *Motor control and spinal reflexes*

The nervous system deals with internal or external perturbations to the body through two main mechanisms: anticipatory postural adjustments which occur before or up to around 70ms after the onset of activity in a prime mover muscle (feed-forward control) and reflex responses which occur after this time (feedback control) (Koumantakis, 2006). The clearest alteration in feed-forward control in patients with LBP occurs in the TrA muscle, the deepest of the abdominal muscles. In healthy participants, TrA is active prior to limb movement and onset is not influenced by the direction of movement, supporting the proposal that this muscle acts to increase the stiffness of the spine and hence oppose any perturbing force (Hodges & Richardson, 1996). In contrast, this onset of TrA is significantly delayed in patients with LBP and varies with the direction of limb movement, which the authors suggest indicates a fundamental problem of motor control of this muscle (Hodges & Richardson, 1996). Preliminary evidence has suggested that delayed activation of TrA is associated with reorganisation of trunk muscle representation at the motor cortex

in individuals with recurrent LBP (Tsao, Galea, & Hodges, 2008). This is thought to be reversible however: motor skill training involving isolated voluntary contractions of TrA resulted in a significant shift in motor cortical representation, towards that observed in healthy individuals, and this shift was associated with earlier postural activation of TrA (Tsao, Galea, & Hodges, 2010).

Delayed and/or reduced activation of TrA is found consistently in LBP patients and similar changes have also been observed following experimental induced pain (Hodges, Moseley, Gabrielsson, & Gandevia, 2003). This implies that the delays in anticipatory responses seen in LBP patients may be caused by pain, although the converse may also be true. Moseley (2004) found that patients and controls who showed an abnormal response on an abdominal drawing-in task (aimed at assessing postural control of TrA) were three to six times more likely to develop persistent or recurrent LBP, suggesting that trunk muscle dysfunction may be etiologic in the development of LBP. The association between pain and delayed TrA onset was further supported by the identification of a significant relationship, although of low to moderate strength, between self-reported pain and latency times during rapid limb movement (Marshall & Murphy, 2009). Despite these findings, concern has been raised as to whether timing differences of only a few tens of milliseconds could really be a primary cause of LBP (Hasan, 1997; Koumantakis, 2006).

It has been suggested that multifidus, the deepest back muscle in the lumbar region, may show similar changes in activity (Hodges & Moseley, 2003) and LBP patients have demonstrated reduced EMG activity of the multifidus muscle during co-ordination and strength exercises compared to controls (Danneels *et al.*, 2002). However, the evidence in this case appears less convincing; LBP patients unexpectedly demonstrated earlier activation of multifidus than controls during rapid shoulder abduction (Hodges &

Richardson, 1996) and experimental pain also led to earlier onset (Hodges *et al.*, 2003). Reduced activity of multifidus is consistent with changes in its morphology (Hodges & Moseley, 2003). Atrophy of the multifidus muscle is a common finding in LBP patients (Hides, Gilmore, Stanton, & Bohlscheid, 2008; Hides, Stokes, Saide, Jull, & Cooper, 1994), even being observed as soon as 24 hours after the onset of acute LBP (Hides *et al.*, 1994). Although premorbid atrophy has been suggested, the phenomenon of rapid, localised atrophy following injury was supported by the finding of significantly reduced cross-sectional area (CSA) three days after disc lesion in pigs (Hodges, Holm, Hansson, & Holm, 2006). The nature of the atrophy led the authors to suggest disuse following reflex inhibitory mechanisms as a cause. In addition to reduced CSA, patients with unilaterally distributed LBP also demonstrate asymmetry of the multifidus muscle, the painful side being smaller in each case (Hides *et al.*, 2008). Atrophy of the multifidus is likely to affect the control and stability of the spine (Hides, Richardson, & Jull, 1996), possibly contributing to a predisposition to further injury and recurrence of LBP (Hides *et al.*, 1994). Although recovery does not appear to occur spontaneously on resolution of pain and disability, training of multifidus has been reported to restore symmetry and muscle size (Hides *et al.*, 1996), and improve clinical outcome (Hides, Jull, & Richardson, 2001; O'Sullivan, Twomey, & Allison, 1997).

A recent study (Silfies, Mehta, Smith, & Karduna, 2009) investigated alterations in trunk muscle timing patterns in two subgroups of CLBP patients (segmental instability and non-instability), according to their diagnosis. Overall, the CLBP patients demonstrated significantly delayed feedforward activation of the trunk muscles compared to a healthy control group. However, within the CLBP patients, the instability group demonstrated significantly delayed onset of both erector spinae and lumbar multifidus compared to the

non-instability group, supporting the notion of meaningful subgroups within the CLBP population.

In general, spinal pain appears to be associated with hypoactivity of the deep trunk muscles, typically accompanied by hyperactivity of the superficial muscles (Hodges & Moseley, 2003), although the mechanism by which pain affects motor control, or vice versa, is unclear. The delay of anticipatory muscle activation in LBP patients may be because specific anticipation imposes a risk of further disturbing equilibrium and patients may therefore prepare by instead co-contracting muscles (van Dieën *et al.*, 2003b). Alternatively, the changes may reflect a central nervous system strategy to splint the trunk (Moseley, Nicholas, & Hodges, 2004a) via hyperactivity of the superficial muscles, possibly resulting in a reduced need for fine control and hence reduced activity of the deep trunk muscles (Hodges & Moseley, 2003). Although neither attention demanding nor stressful tasks were found to replicate the motor control changes observed following pain (Moseley *et al.*, 2004a), Hodges and Moseley suggest that fear of pain may play an important role. Anticipation or threat of back pain has been shown to result in increased reaction times, delayed activation of the deep trunk muscles, and an increase in superficial muscle activity, consistent with the activation patterns exhibited by LBP patients (Moseley, Brhyn, Ilowiecki, Solstad, & Hodges, 2003; Moseley, Nicholas, & Hodges, 2004b). The results of these studies suggest a link between psychosocial factors and physiological changes (Hodges & Moseley, 2003), a concept which is discussed further below. The strategy of reduced deep trunk muscle activity combined with increased superficial muscle activity and co-contraction may lead to greater spinal loading and reduced control, both of which are risk factors for further pain and injury (Hodges & Moseley, 2003).

Analysis of the feedback control system has also identified differences between LBP patients and controls. In response to sudden loading, patients with LBP demonstrate generally longer trunk muscle reaction times and, unlike the healthy controls, tend to keep their antagonistic muscles contracted as they activate the agonistic muscles (Radebold *et al.*, 2000; Reeves, Cholewicki, & Milner, 2005). LBP patients also tend to demonstrate loss of position sense, repositioning error, poorer balance and postural control (Brumagne, Janssens, Knapen, Claeys, & Suuden-Johanson, 2008; Luoto *et al.*, 1998; Taimela, Kankaanpää, & Luoto, 1999; O'Sullivan *et al.*, 2003), with proprioceptive deficiency thought to be a common underlying cause (Leinonen *et al.*, 2003). Such proprioception deficits may be the result of soft tissue injuries that have damaged receptors in ligaments, intervertebral discs, facet joints and muscles. A loss of proprioception may result in an altered muscle recruitment pattern and an increase in trunk muscle co-activation to compensate for the loss of stability (Radebold *et al.*, 2000). Panjabi (2006) has recently formalised this concept in a new hypothesis in which he suggests that subfailure injuries of ligaments (spinal ligaments, disc annulus and facet capsules) may disrupt and/or injure the embedded mechanoreceptors, resulting in corrupted transducer signals. These may then lead to a corrupted muscle response pattern, including co-activation of agonist and antagonist muscles to stabilise the spine. However, this altered muscle response pattern may result in abnormal stresses and strains in the ligaments, mechanoreceptors and muscles and excessive loading of the facet joints, leading to a vicious circle and the development of chronic back pain.

2.4.11. *Muscle fatigue and fibre type*

It has been suggested that LBP patients demonstrate lower levels of both strength and endurance than asymptomatic individuals, although the evidence to date has been conflicting and inconclusive. Common measurement techniques tend to rely on high

participant motivation and it has been argued that patients with LBP may simply be unwilling/unable to sustain a contraction for as long as healthy controls due to psychological factors such as (fear of) pain/(re)injury. Therefore a number of studies have instead focussed on the EMG power spectrum, since it is widely accepted that there is a shift towards lower frequencies as a muscle fatigues and median frequency decline has been found to correlate well with endurance time and subjective measures of fatigue for healthy participants (Dedering, Németh, & Harms-Ringdahl, 1999). Spectral parameters measured during fatiguing contractions from the paraspinal muscles have also been shown to discriminate LBP patients with a high degree of accuracy (Peach & McGill, 1998; Roy, De Luca, & Casavant, 1989). However, while earlier studies have tended to report that LBP patients show a greater decline in median frequency during isometric contractions, suggesting greater fatigue (Roy *et al.*, 1989; Tsuboi, Satou, Egawa, Izumi, & Miyazaki, 1994), later studies have tended to show either little difference or the converse (Elfving, Dedering, & Németh, 2003; Kramer *et al.*, 2005; Larivière, Arsenault, Gravel, Gagnon, & Loisel, 2003). Some of the discrepancies may be explained by the different approaches taken. In particular, while some have used a consistent load between groups, many have used a load proportional to MVC, with some authors subsequently suggesting that their results may have been affected by the underestimation of the MVC in the LBP patients (Larivière *et al*., 2003). The severity and duration of pain in the LBP group has also varied greatly between studies and potential confounding factors (e.g. gender, age and body fat) were often not controlled for. In addition, the presence of pain may lead to an inhibition of certain muscles (Elfving *et al*., 2003), resulting in altered recruitment patterns, which may help to explain some of the unexpected findings. For example, Mannion *et al.* (Käser *et al*., 2001; Mannion *et al*., 2001b) counter-intuitively found that therapy led to a greater decline in median frequency (implying greater fatigue) despite increased strength and endurance of LBP patients. The authors suggested that their findings may be due to greater muscle

activation and co-ordination and different recruitment patterns following treatment, perhaps as the result of less guarded movement.

van Dieën *et al.* (2003b) suggest that alterations in muscle recruitment and reduced ability to activate back muscles as a consequence of LBP may lead to changes in muscle fibres and hence greater fatigability. This might then create a vicious circle. Mannion, Dumas, Stevenson and Cooper (1998) considered the link between fatigability and muscle fibres and reported a highly significant relationship between the relative area of Type I (slow twitch) muscle area and median frequency decline, with a lower percentage of Type I fibre area being associated with greater fatigue, suggesting that fibre type distribution may predispose some individuals to LBP. In an earlier study (Mannion, Weber, Dvorak, Grob, & Müntener, 1997), it was determined that LBP patients had a lower relative area of Type I fibres than controls, suggesting greater fatigability. Patients, particularly those who had only experienced symptoms for a short period of time, also had more Type IIC 'intermediate' fibres reflecting ongoing fibre type transformation. The results suggested that the process is one of general disuse and that fibre type transformation rather then changes in fibre size are predominant changes in muscles of LBP patients.

2.4.12. *Muscle activity and stature change*

Back muscle activity exerts a high compressive force on the spine due to the longitudinal direction of the muscle action, which then raises the intradiscal pressure (Bogduk, 2005; Middleditch & Oliver, 2005). Adams *et al.* (2002) suggested that the high antagonistic muscle forces typical of people with back pain may subject the spine to high chronic loading, leading to accelerated loss of disc height and therefore high stress concentrations within the disc. Earlier studies support the concept that compressive loading resulting from muscle co-activity can affect intervertebral disc height. Spinal shrinkage observed during

deep water running was attributed to compressive forces resulting from the activity in the trunk muscles required to maintain an upright posture in the water (Dowzer, Reilly, & Cable, 1998) and a study to investigate the spinal shrinkage induced by torsion, lateral bending or flexion found that torsion, which involves the greatest muscular co-activity, also resulted in the greatest stature change (Au, Cook, & McGill, 2001).

In fact, analysis of stature change between LBP patients and healthy controls has indicated no significant difference in the magnitude of disc height loss following loading (Garbutt, Boocock, Reilly & Troup, 1990, Healey *et al.*, 2005a). Interestingly though, participants with LBP consistently appear unable to recover stature to the same extent as healthy controls (Fowler, Rodacki, & Rodacki, 2005; Healey *et al.*, 2005a; Rodacki, Fowler, Rodacki, & Birch, 2003). Healey *et al.* (2005a) observed that individuals with CLBP exhibited elevated muscle activity compared to asymptomatic controls and that paraspinal muscle activity correlated negatively with stature recovery. Significant negative correlations between stature recovery and levels of both perceived pain and disability suggest that the delayed recovery of height may be clinically important. Since disc degeneration is associated with greater disc height loss following mechanical loading (Adams & Hutton, 1983), the similar loading response in both the patient and asymptomatic groups implies an alternative cause of the recovery rate difference. The authors suggested that, instead, the prolonged and higher levels of paraspinal muscle activity in CLBP patients may result in greater compressive loads on the spine, which reduce the rate at which the intervertebral discs recover their initial height and consequently prolong stature recovery. This argument was supported by the findings of a subsequent study (Healey *et al*., 2008) in which paraspinal muscle activity of asymptomatic participants was increased using functional electrical stimulation to a level intended to mimic that seen in patients with CLBP and there was an associated reduction in

stature recovery, similar to that observed in CLBP patients. In addition, Hupli *et al.* (1997) found that an active back rehabilitation programme significantly increased the morning height of CLBP patients compared to those who received no treatment, with a reduction in muscle contraction suggested as a possible explanation for the greater height recovery. Again, the gain of height was significantly correlated with reduction of pain and decrease in disability. Importantly, however, the height gain did not correlate with the state of disc degeneration and neither did the MRI scans of the discs show any differences after treatment, supporting the view that the reduced stature recovery observed in CLBP patients is not simply the result of disc degeneration.

As discussed, reduced stature recovery may increase the risk of future back pain by increasing the size of the neutral zone and compromising spinal stability (Zhao *et al.*, 2005). It may also cause a decrease in the discs' shock absorbing properties, increased loading on spinal structures such as the facet joints and concentrations of compressive stress (Adams *et al.*, 2002). Therefore delayed stature recovery, itself a potential consequence of increased paraspinal muscle activity, may contribute to a vicious cycle via an increase in pain and a greater risk of further injury.

2.5. Psychological factors

2.5.1. *Role of psychological factors in low back pain*

LBP is not solely a physical problem. The importance and interdependence of physiological, psychological and social factors in the development and maintenance of the disorder are now accepted widely and formalised in the biopsychosocial model of LBP (Waddell, 2004).

A number of studies have considered whether psychological characteristics may be risk factors for both first time and subsequent episodes of LBP (e.g. Adams *et al*., 1999; Linton, 2000; Linton, 2005). Psychological distress (in terms of increased somatisation, depressive and anxiety symptoms) has been shown to be a significant predictor of first time pain (Adams *et al.*, 1999; Mannion *et al.*, 1996). However, in general, the role of psychological factors in first time onset appears limited, with Mannion *et al.* (1996) observing that scores on psychological questionnaires (measuring somatic anxiety, depression and beliefs about health) predicted less than 3% of first-time LBP. The argument regarding the recurrence of LBP is more convincing. In several population based studies, catastrophising (exaggerated negative rumination or worry), kinesiophobia (excessive fear of movement) (Picavet *et al*., 2002), depression (Carroll, Cassidy, & Côté, 2004) and psychological distress (Linton, 2005) were found to significantly increase the risk of future episodes of back pain. However, it is feasible that factors such as distress may also make individuals more likely to report symptoms (Waddell, 2004) and/or lead to a heightened sensitivity to back discomfort (Mannion *et al.*, 1996). Mannion *et al.* (1996) concluded that psychological variables are more important in explaining how people respond to back pain than in explaining the onset of pain and this is supported by recent research findings. Numerous studies have identified psychological factors as significant in the transition from acute to chronic pain and the perpetuation of pain and disability (e.g. Buer & Linton, 2002; Pincus,

Burton, Vogel, & Field, 2002; Woby *et al*., 2007b). In particular, self efficacy (Denison, Åsenlöf, & Lindberg, 2004; Woby *et al.*, 2007b;), catastrophising (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998; Picavet *et al.*, 2002), kinesiophobia, fear-avoidance beliefs (Crombez *et al*., 1999, Vlaeyen *et al*., 1999, Picavet *et al.*, 2002), depression, anxiety, distress and stress (Linton, 2000; Keogh, McCracken, & Eccleston, 2006) have all been shown to play a significant role in the development and/or perpetuation of LBP.

Psychological factors are also relevant in the treatment of LBP, and there is a complex interaction between such factors and clinical outcome (Woby, Watson, Roach, & Urmston, 2004). Firstly, not all patients benefit from treatment to the same extent and psychological variables can be viewed as predictors of treatment outcome. For example, patients who demonstrate high emotional distress, catastrophising and other forms of negative thinking in response to pain tend to derive less benefit from treatments than other patients (McCracken & Turk, 2002). More importantly, however, cognitive processes have been demonstrated to influence treatment outcome strongly (Woby *et al.*, 2004). In particular, pre- to post-treatment modifications in pain-related anxiety (McCracken, Gross & Eccleston, 2002), fear (avoidance) (Vlaeyen, de Jong, Geilen, Heuts, & van Breukelen, 2002; Woby, Roach, Urmston, & Watson, 2008), psychological distress (Grotle, Vøllestad, & Brox, 2006; Mannion, Junge, Taimela, Müntener, & Dvorak, 2001a), catastrophising and self-efficacy (Foster *et al*., 2010; Woby *et al.*, 2008) have all been identified as significant predictors of clinical outcome, thus supporting the role of such factors in the perpetuation of the condition.

Numerous models have been suggested that aim to explain the process by which psychological factors affect the development of and recovery from LBP. One of the most influential has been the fear-avoidance model, which is described on page 49. Fear-

avoidance refers to the "avoidance of movement or activities based on fear" (Vlaeyen & Linton, 2000, p.317).

2.5.2. *Fear-avoidance model*

The fear-avoidance model (proposed by Lethem, Slade, Troup, & Bentley (1983) and developed by Vlaeyen, Kole-Snijders, Boeren, & van Eek (1995)) is a widely influential cognitive-behavioural model that, for a subgroup of patients, aims to explain the transition from acute to CLBP. The model suggests two behavioural responses to acute pain. If the patient engages in catastrophic thinking, the consequence may be pain-related fear, avoidance behaviour and hypervigilance. These responses may be adaptive in the acute pain stage but, if they persist in the long term, may become dysfunctional and lead to a vicious cycle of disuse, depression and increased disability (Leeuw *et al*., 2007a). Alternatively, if acute pain is viewed as non-threatening, recovery may occur following the adaptive response of confrontation and continued engagement in daily activities. In essence, the model proposes that fear of movement/(re)injury mediates the relationship between catastrophising and functional disability (Leeuw *et al*., 2007b).

Numerous studies have supported the proposal that catastrophising and pain-related fear are significantly related to pain and disability (e.g. Grotle, Foster, Dunn, & Croft, 2010; Picavet *et al.*, 2002; Thomas & France, 2008; Woby *et al.*, 2004), and changes in catastrophising have been shown to mediate the reduction of disability and pain intensity (Smeets, Vlaeyen, Kester, & Knottnerus, 2006). Furthermore, prospective studies have found pain catastrophising to be predictive of pain-related fear (Leeuw *et al.*, 2007b) and fear of movement/(re)injury to be a powerful predictor of future perceived disability and, to a lesser extent, of participation in daily activities (Swinkels-Meewisse *et al.*, 2006b). Although prospective studies fully confirming the sequential relationships proposed by the

fear-avoidance model are lacking, a recent cross-sectional study involving sub-acute whiplash patients did show that fear of movement mediated the relationship between catastrophising and disability (Nieto, Miró, & Huguet, 2009).

Further support for the model is provided by the fact that pain-related fear and catastrophising are associated with hypervigilance to symptoms, greater attentional demand of pain and difficulty in disengaging from pain (Goubert, Crombez, Eccleston, & Devulder, 2004; Van Damme, Crombez, & Eccleston, 2004; Vlaeyen & Linton, 2000), which may be reflected in increased report of pain. LBP patients also demonstrate increased sensitivity to pressure-induced pain over the painful muscles, with highly significant correlations identified between pressure pain threshold scores and fear avoidance beliefs (Watson, 2002). This suggests that the increased responsiveness is distinct from central sensitisation, which is thought to be present in some cases of CLBP, and may result in generalised hypersensitivity to pain and increased sensitivity to a variety of stimuli including mechanical pressure (Nijs, Van Houdenhove, & Oostendorp, 2010).

One aspect of disuse is considered to be disturbed trunk muscle activity during movement (Leeuw *et al.*, 2007a). Pain-related fear has been associated with limited movement during lumbar flexion (Geisser, Haig, Wallbom, & Wiggert, 2004; Thomas & France, 2008), alternative movement strategies during reaching tasks (Thomas & France, 2007) and reduced activation of certain trunk muscles during maximal isometric exertions (Thomas, France, Sha, & Vander Wiele, 2008). It is also proposed that avoidance behaviour may lead to decreased physical activity and hence physical deconditioning, which may be measured as either a reduced level of aerobic fitness or a decrease in muscle strength (Leeuw *et al.*, 2007a). Studies investigating the muscle strength of LBP patients have indeed shown correlations between fear of movement and reduced performance in lifting

tasks, isokinetic extension and flexion tasks and isometric strength tests (Al-Obaidi *et al.*, 2000; Crombez *et al.*, 1999; Swinkels-Meewisse, Roelofs, Oostendorp, Verbeek, & Vlaeyen, 2006a; Vlaeyen *et al.*, 1995), although Huijnen, Verbunt, Peters and Seelen (2010) only found fear of movement to be predictive of performance in patients who also persistently over-predicted activity-related pain. A consistent problem with studies involving maximal strength testing is determining whether fearful patients are simply displaying sub-maximal physical performance during the task due to fear of pain (Al-Obaidi *et al.*, 2000; Crombez *et al.*, 1999; Vlaeyen *et al.*, 1995). For example, Verbunt *et al.* (2005a) measured muscle strength of the quadriceps muscle on a dynamometer. During peak torque, electrical stimulation was applied to the muscle, which generated additional twitch torque in the case of sub-maximal performance. A higher additional twitch torque therefore represented a lower performance level. Using this approach, it was observed that CLBP patients had a significantly lower performance level than healthy controls. In particular, CLBP patients who reported increased psychological distress and a higher current level of pain tended to show increased inhibition of muscle activity, leading to sub-optimal performance. However, no association was found with either catastrophising or fear of injury in this case, suggesting that levels of distress and pain actually have a greater influence on underperformance.

It has been found that fear avoidance beliefs more than double the risk of lower levels of activity (Buer & Linton, 2002) and that increases in physical activity appear to accompany decreases in pain-related fear (Vlaeyen *et al.*, 2002). However, research which aimed to demonstrate that LBP patients have lower aerobic fitness than matched healthy counterparts has produced largely inconclusive results. While Schmidt (1985) did find lower treadmill endurance of CLBP patients compared to controls, the patients actually showed lower levels of physiological demand and appeared to stop because of over-

estimate of exertion rather than increased pain. When CLBP patients and controls were matched for level of sport activity as well as age and sex, CLBP patients were found to have significantly lower aerobic fitness than their matched controls (Smeets, van Geel, & Verbunt, 2009). This lower level of aerobic fitness was significantly associated with lower levels of leisure and work activity but, in line with another cross-sectional study (Verbunt, Seelen, Vlaeyen, van der Heijden, & Knottnerus, 2003b), was not associated with pain-related fear or catastrophising. In a study involving over 100 LBP patients, Verbunt *et al.* (2005b) found that activity decline was correlated with disability, fear of movement/(re)injury, catastrophising and pain intensity. Consistent with the fear-avoidance model, activity decline also appeared to play a mediating role between fear of movement/(re)injury and disability. However, this study was limited by being cross-sectional in design and therefore relied on self-report of past physical activity levels, possibly suggesting that it is perceived rather than actual loss of physical activity levels that is disabling (Verbunt *et al.*, 2005b). Two more recent longitudinal studies have, in fact, not supported the link between pain-related fear and physical activity levels (Bousema, Verbunt, Seelen, Vlaeyen, & Knottnerus, 2007; Leonhardt *et al.*, 2009). In a study involving over 100 patients with sub-acute LBP, only a subgroup of patients showed a decrease in physical activity levels (PALs) (measured using an accelerometer over a seven-day period) over the following year and neither fear of movement nor catastrophising were found to be significant predictors (Bousema *et al.*, 2007). Furthermore, no signs of physical deconditioning were found in the patients (although this was only assessed via changes in strength and lean body mass and weight, rather than changes in aerobic fitness). These findings were supported by a more recent questionnaire based, longitudinal study (Leonhardt *et al.*, 2009) which followed 787 acute and chronic LBP patients over a year. Physical activity levels were found to increase rather than decrease over the year and initial fear avoidance beliefs did not significantly predict

physical activity levels at the end of the year. The authors suggested that patients with high fear avoidance beliefs were only avoiding specific activities which they believed to be dangerous rather than reducing their general level of activity. This is supported by the observation that performance in tasks chosen to resemble typical daily activities (such as walking, climbing stairs and reaching) was not related to fear of movement in a study involving over 200 CLBP patients (Smeets, Van Geel, Kester, & Knottnerus, 2007).

Research findings may have been affected by patients who cope with pain using endurance strategies, as described in the avoidance-endurance model (Hasenbring, Hallner, & Klasen, 2001; Bousema *et al.*, 2007). It has been suggested that, in addition to the adaptive coping and avoidance behaviours described by the fear-avoidance model, there may also be patients who tend to suppress or ignore pain (Hasenbring *et al.*, 2001). Extreme suppressive behaviour may lead to patients overloading their muscles, leading to muscular hyperactivity and pain. These patients may be expected to have a cyclic pattern of over and underactivity, as the pain caused by overloaded muscles, bones and discs leads to periods of rest in order to recover. In the long run, both strategies would be expected to result in a decline in PALs and physical deconditioning (Verbunt *et al.*, 2003a), but it has been suggested that fluctuations in activity levels may actually have a greater association with disability than mean activity level over time (Huijnen, Verbunt, Roelofs, Goossens, & Peters, 2009).

There are undoubtedly some results which appear inconsistent with the relationships proposed in the fear avoidance model. For example, Sieben *et al.* (2005) found that pain-related fear is not independently predictive of future disability. In a recent prospective study including over 400 participants with back pain, Gheldof *et al*. (2010) also found that pain-related fear at baseline did not directly affect pain-severity at 18-month follow-up and

the direct effect on disability was significant but of small effect size. Furthermore, the model in which pain-related fear is a consequence of pain severity was, unexpectedly, found to be a better fit than the model in which pain-related fear is an antecedent of pain severity, as would be predicted by the fear avoidance model, although the relatively low level of disability of the participants in this study may have affected these results. Other researchers have reported that distress (Grotle *et al.*, 2006), depression (Boersma & Linton, 2005) and self-efficacy (Denison *et al.*, 2004; Woby *et al.*, 2007b) are stronger predictors of disability then fear avoidance. In two review articles, Pincus *et al*. (Pincus *et al.*, 2002; Pincus, Vogel, Burton, Santos, & Field, 2006) concluded that there was little evidence for the role of fear avoidance as a risk factor for poor outcome and suggested other pathways, such as social factors and depression, to reduced activity and hence poor outcome. One explanation suggested for their findings is that fear plays an important role only in later stages of pain. In support of this, Boersma and Linton (2005) found that the relationship between function and fear of movement was moderated by the stage of chronicity and that fear of movement did not emerge as a significant predictor for function until after one year of pain duration, although this may also be interpreted as inconsistent with the fear-avoidance model.

A recent study (Wideman, Adams, & Sullivan, 2009) investigated the sequential relationships proposed by the fear avoidance model in over 100 patients with musculoskeletal injuries during a ten-week disability management intervention and at a four-week follow-up. Although changes in catastrophising and fear of movement were shown to predict return to work at follow-up, the authors found that early changes (up to the mid-point of the intervention) in catastrophising were not correlated with late changes (after the mid-point of the intervention) in fear of movement and pain. They therefore concluded that their findings did not support the sequential components of the fear-

avoidance model, although acknowledged that changes in fear of movement may have followed changes in catastrophising too closely for this to be detected via the five-week testing sessions. Furthermore, this study was in fact investigating the relationships involved in the treatment and recovery of CLBP rather than its development, which may not necessarily follow the same pattern. In a response to Wideman *et al.*'s study, Vlaeyen, Crombez and Linton (2009, p.222) state that the fear-avoidance model was intended as a "theory-based heuristic", rather than a "final model to be unconditionally embraced or falsified". Despite conflicting findings, the model remains popular and still constitutes a useful model of chronic pain for at least a proportion of patients (McCracken & Samuel, 2007).

2.5.3. *Self-efficacy*

Functional self-efficacy refers to the confidence that an individual has in his or her own ability to accomplish certain functional tasks, such as household and social activities (Woby *et al*., 2007b). Self-efficacy was not included in the original fear-avoidance model, and this variable has also been excluded from many of the studies that reported a strong association between pain-related fear and disability (Woby, Roach, Urmston, & Watson, 2007a). Woby *et al.* (2007b) found that functional self-efficacy mediated the relationship between pain-related fear and both pain and disability. The authors therefore suggested a modification to the fear-avoidance model, such that high self-efficacy may enable even patients with high pain-related fear to become 'confronters' and maintain physical activity levels. A number of studies have highlighted the importance of self-efficacy (e.g. Denison *et al.*, 2004), observing that it significantly predicts physical performance (Lackner & Carosella, 1999) and mediates the relationship between pain intensity and both pain related disability and depression (Arnstein, Caudill, Mandle, Norris & Beasley, 1999; Costa, Maher, McAuley, Hancock & Smeets, in press; Woby *et al.*, 2007b). Costa *et al.* (in press)

found that both self-efficacy and fear of movement were partial mediators in the relationship between pain and disability, but in a longitudinal analysis, only improvements in self-efficacy beliefs partially mediated the relationship between changes in pain and disability over a 12-month period, thereby supporting the potential importance of interventions that are able to improve self-efficacy. They also note the common limitation of these studies, however; that there may be overlap in self-report of disability and self-efficacy as patients may find it difficult to distinguish between the constructs.

Lackner and Carosella (1999) found self-efficacy to be a better predictor of performance in a lifting task than either perceptions of pain control or psychological distress. In a recent study involving over 800 patients with LBP in primary care and assessing 20 psychological factors, pain self-efficacy was one of four factors found to be most predictive of outcome at six months (the others being: patients' perceptions that the problem will last well into the future, that many symptoms are related to their back problem and weak beliefs about personal controllability) (Foster *et al.*, 2010). Interestingly, neither catastrophising nor fear-avoidance were found to be significant independent predictors of outcome, the authors suggesting that this may be because some factors are important earlier in the LBP episode. Despite these positive findings regarding the importance of self-efficacy, Mannion *et al.* (2001a) reported that self-efficacy beliefs correlated significantly with disability in women only and overall, their influence on disability was less than that of other psychological factors and pain. However, the participants in this study were recruited via advertisements in the local media and hence may not have been representative of the typical patient population. In particular, participants who volunteer for such a study are probably less likely to report low self-efficacy than patients recruited from clinical practice.

2.5.4. *Depression*

Depression is characterised by a pervasive low mood, loss of interest in usual activities and diminished ability to experience pleasure (Woo, 2010). It is well known that depression and chronic pain often co-exist and that physiologically similar responses can exist between the two (Woo, 2010); however, the mechanism to explain why so many chronic pain patients experience depression and even the direction of the cause-effect relationship remains unclear. Psychological distress (in terms of increased somatisation, depressive and anxiety symptoms) and depression have been shown to significantly increase the risk of first and future episodes of back pain (Mannion *et al.*, 1996; Adams *et al.*, 1999; Carroll *et al.*, 2004; Linton, 2005). Furthermore, the presence of depression is associated with a worse prognosis (Bair, Robinson, Katon, & Kroenke, 2003). In particular, depression was found to be a significant predictor of poor outcome both seven weeks and one year after an initial visit to a primary care clinic for an episode of low back pain (Cherkin, Deyo, Street, & Barlow, 1996). However, there has been growing acceptance of the notion that pain generally precedes depression, rather than *vice versa* (Arnstein *et al.*, 1999), although it has been suggested that depression is not a direct consequence of pain. Instead, pain intensity may be related to depressive symptoms via the mediating role of interference with activities (Woby *et al.*, 2008). This is supported by the finding that changes in disability, rather than pain intensity, are strongly linked to changes in depression (Woby *et al.*, 2008).

Boersma and Linton (2005) separated nearly 200 chronic pain patients into three groups depending on the duration of their pain (< 1 year, 1-3 years and > 3 years) and found depression and function to be strongly correlated at all three stages (Boersma & Linton, 2005). Following suggestions that depression may lead to self-report bias of function, Alschuler, Theisen-Goodvich, Haig and Geisser (2008) assessed disability via physical performance on a lifting task and found a relationship of similar magnitude as when using

a self-report measure, suggesting that self-report bias was not an issue. However, physiologic effort (as assessed via maximum heart rate) partially mediated the relationship between depression and physical performance, supporting the idea that function is at least partly influenced by reduced physiologic effort.

The fear avoidance model predicts that depression may follow catastrophising and pain-related fear (Leeuw *et al.*, 2007a). Consistent with this, reductions in catastrophising following treatment were reported to mediate reductions in depression (Spinhoven *et al.*, 2004). It has also been suggested that depression may play a greater role than previously thought in the early stages of back pain (Pincus *et al*., 2006). Following a review of the fear avoidance model, Pincus *et al*., (2006) suggested further investigation into the individual contribution of the components of depression (cognitions, negative mood and somatic symptoms) and their interaction with fear and anxiety in the development of disuse and avoidance behaviours. This proposal is supported by the recent observation that CLBP patients classified as 'distressed' spent one hour and thirty minutes less time upright (as measured with an accelerometer) over an average day than a 'non-distressed' group, with depressive symptoms emerging as the only psychosocial variable measured which was a significant independent predictor of time spent upright (Ryan, Gray, Newton, & Granat, 2010). In particular, contrary to the theory of the fear-avoidance model, fear of movement was not found to be a significant predictor of physical activity levels. The cross-sectional nature of the study meant that causal relationships could not be explored and, therefore, it may also be possible that lack of physical activity contributes to any increased depressive symptoms.

2.5.5. *Anxiety*

Anxiety and depression often co-occur in clinical populations, possibly representing characteristics of a broader negative affect dimension, or reflecting a common origin (Rutledge *et al*., 2009). Anxiety is a psychophysiological state characterised by cognitive, somatic, emotional and behavioural components producing fear and worry (Woo, 2010). Anxiety disorders are common in chronic pain populations and, in fact, depression is the only more frequent psychological co-morbidity (Woo, 2010). Many markers of anxiety exist. For chronic pain populations, more specific markers of anxiety related to pain are often used, such as the Pain Anxiety Symptoms Scale (PASS), which acts as a marker of behavioural responses to pain.

In the literature, the term pain-related fear is often used as a general term to cover several aspects of pain-related fear and anxiety, including fear of movement and (re)injury. However, evidence supports the independence of pain-related fear (fear of current pain) and pain-related anxiety (fear of the possibility of pain), in particular, as related but distinct constructs (Carleton & Asmundson, 2009). In a study comparing a number of questionnaires assessing pain-related fear and anxiety, Crombez *et al*. (1999) also concluded that they appeared to measure unique constructs. The subscales of the questionnaires assessing fear-avoidance beliefs (Fear-Avoidance Beliefs Questionnaire) and fear of movement/(re)injury (Tampa Scale for Kinesiophobia) were superior in predicting disability and poor behavioural performance, whereas the questionnaire designed to assess pain-related anxiety (Pain Anxiety Symptoms Scale) appeared more strongly correlated with catastrophising and negative affect.

Asmundson, Norton, and Vlaeyen (2004) presented an updated fear-anxiety-avoidance model which differentiates between fear and anxiety. They argue that anxiety typically has

a greater cognitive component and a more suppressed physiological component than fear and that different behavioural responses also exist. Fear occurs in response to a perceived immediate threat, and can lead to escape and defensive behaviours. In contrast, anxiety occurs in response to anticipated threats that are often more uncertain in nature and it is this pain-related anxiety which can lead to avoidance and preventative behaviours. In light of this, Asmundson *et al.* (2004) added an anxiety pathway in addition to the existing fear pathway but suggested that both may trigger each other in a mutually reinforcing fashion.

Eysenck (1997) proposed a cognitive theory of anxiety. Central to the theory is the suggestion that the emotional experience of anxiety depends on an individual's attentional and interpretive processing of four factors: (1) the situation; (2) their own physiological activity; (3) negative cognitions about possible future events (worries); and (4) their own behaviour. Evidence suggests that high-anxious individuals demonstrate an attentional bias to attend to threat related rather than neutral stimuli and an interpretive bias to interpret ambiguous stimuli in a threatening rather than a neutral fashion (Eysenck, 2000). In general, low-anxious individuals are not thought to exhibit cognitive biases (Eysenck, 2000).

2.5.6. *Coping styles*

Research into coping has conceptualised it in a variety of ways, including viewing coping both as a trait like disposition and a dynamic process (transactional approach). Within the transactional approach, two major functions of coping were identified: to manage or alter the problem causing the distress, and to regulate emotional response to the problem (Lazarus & Folkman, 1984). Coping strategies were subsequently grouped into three main categories: problem focused, emotion focused and avoidance, which may involve behavioural (e.g. physically removing oneself from the situation) and/or cognitive

avoidance (e.g. mentally blocking out stressors) (Endler & Parker, 1990; Lazarus & Folkman, 1984). In general, problem focused coping is believed to be the most efficacious strategy, associated with more positive outcomes, whereas avoidance is often considered a maladaptive coping strategy (Richards & Steele, 2007).

The term 'coping style' refers to a more trait like disposition that influences the coping strategies that an individual might use and their response to a stressor (Prasertsri *et al.,* in press). One approach to classifying coping styles is on the basis of scores obtained on measures of defensiveness such as the Marlowe-Crowne Social Desirability Scale (MC-SD) (Crowne & Marlowe, 1960). The MC-SD was originally developed to measure the extent to which an individual will respond in a culturally acceptable manner in order to gain social approval (and hence has been used to adjust for the influence of response bias for example), but has also been widely adopted as a measure of psychological defensiveness (with a high score reflecting greater defensiveness). A high MC-SD score is thought to reflect a tendency to over-report socially desirable and under-report socially undesirable personal information, although it is possible that people who score highly on the MC-SD may actually behave in an altruistic manner (Johnson & Fendrich, 2002). Women typically score higher than men on social desirability, with less educated and older respondents also reportedly scoring higher (Burris, Johnson, & O'Rouke, 2003). Defensiveness is generally assumed to be a relatively stable personality trait, although it has also been suggested that it includes both self-deception and impression management, with the latter being situational (Burris *et al.,* 2003).

Weinberger, Schwartz and Davidson (1979) classified individuals based on self-report scores of trait anxiety and defensiveness. Four groups of individuals were identified: high-anxious, low-anxious, defensive high-anxious (DHA) and repressors, who score low on

anxiety measures, but high on defensiveness. According to the four-factor theory (Eysenck, 1997), repressors exhibit opposite attentional and interpretive biases to high-anxious individuals, leading them to avoid attending to threat related stimuli, and interpreting ambiguous stimuli as non-threatening. Experimental evidence that has attempted to detect these predicted biases has generally been most successful in cases where the situation or stimuli was personally relevant to the individual (Eysenck, 2000). Despite the belief that a repressive coping style is associated with avoidant strategies, repressors themselves tend to report more adaptive or problem focused strategies, perhaps distorting the true situation in order to present a view of more socially desirable methods of coping (Richards & Steele, 2007). This is in contrast to high anxious individuals who often score high on avoidance and low on problem focused coping, and DHA individuals who report a variety of coping strategies (Richards & Steele, 2007).

Although reporting low anxiety, repressors tend to exhibit elevated behavioural and psychophysiological responses, similar to high-anxious individuals, in stressful situations (Eysenck, 2000). In order to explain this apparent discrepancy, Derakshan, Eysenck and Myers (2007) proposed the vigilance-avoidance theory, which suggests that, in response to self-relevant threats, repressors have an initial rapid vigilant response triggering behavioural and physiological responses. This is then followed by an avoidance stage involving avoidant cognitive biases that produces low levels of experienced anxiety (Derakshan *et al.*, 2007). There is evidence that, in line with the theory, repressors do show elevated responses in cardiovascular, electrodermal and muscular systems (Burns, 2000a). It has additionally been suggested that in patient groups, repressors may exacerbate chronic pain via physiological reactivity e.g. stress-induced muscle activity, but to date this remains largely speculative (Burns, 2000a). This discrepancy between physiological activity and self-report measures of distress and anxiety may lead to relationships between

psychological and physiological variables being overlooked if repressors are not accounted for in research and rehabilitation/therapy. Although reporting low distress, repressors appear willing to report high levels of pain, suggesting that they may be suppressing negative emotions associated with the pain rather than all pain-related thoughts (Elfant, Burns, & Zeichner, 2008).

Repressors are relatively common, accounting for between 10 and 20% of non-clinical populations and up to 50% in various clinical and elderly groups (Myers, 2010). Certain chronic illness populations, such as those with heart disease, cancer and asthma, appear to have an increased prevalence of repressive coping (Myers, 2010; Phipps & Steele, 2002), which questions whether this coping style may be a risk factor for chronic conditions, or whether a shift towards increased defensiveness and more repressive styles of adaptation is a response to the threat of serious illness (Phipps & Steele, 2002). The results of a study of 646 women referred to mammographic examination for breast cancer (Zachariae *et al.*, 2004) suggest the latter. Four weeks after diagnosis, increased repression was found in the group of 71 women diagnosed with breast cancer, but not in those without cancer. The change was primarily found to be due to an increase in defensiveness (Zachariae *et al.*, 2004).

Within chronic illness populations, repressive coping appears to be associated with poor prognosis and a higher rate of mortality (Myers, 2010). In particular, repressors do not appear to respond well when given ‘high-risk’ news about their medical condition (Myers, 2010), which perhaps follows from their general tendency to avoid negative personal information. Repressors are also better at undertaking self-care health behaviours, compared to those which they perceive as being outside their control (Myers, 2010), and may be reluctant to seek social support or engage effectively in psychotherapy (Phipps &

Steele, 2002). Indeed, among chronic pain patients, a repressive coping style is associated with poorer long-term adjustment and a poor response to a multidisciplinary rehabilitation programme (Burns, 2000b, Elfant *et al*., 2008). Repressors also tend to be overly optimistic, both in terms of interpretation bias (Eysenck, 2000), and in predicting their future performance (Jones, Smith, & Holmes, 2004). This may have an impact on pacing, potentially leading to task persistence and endurance behaviour, but to date this has not been investigated. Although repressors show short-term tolerance of acute pain, this is thought to lead to long-term sensitivity to pain (Elfant *et al*., 2008), suggesting that, although it is associated with lower reports of depression, distress and catastrophising (Prasertsri *et al*., in press), this is a maladaptive long-term coping style.

In addition to the evidence regarding repressors, high defensiveness in general, incorporating both the repressive (appearing as defensive low-anxious) and DHA coping styles, may be associated with negative outcomes. Defensive patients may not respond well to treatment, perhaps being unwilling to acknowledge issues and challenge beliefs (Burns, 2000a) and it has been suggested that a DHA coping style may also have a detrimental effect on health, particularly via immunological functioning (Creswell & Chalder, 2001). Consistent with this, Jamner and Leigh (1999) observed a significant positive correlation between resting plasma β-endorphin (a powerful immunoregulator) levels and defensiveness in men (although not women), and the correlation was strengthened following the presentation of painful stimuli. Similarly, Brody *et al.* (2000) observed that defensiveness (Marlowe-Crowne Social Desirability) scores were associated with higher cortisol levels in 60 firefighters under the age of 45, although, interestingly, the same relationship was not apparent in the older firefighters, where there was a trend in the opposite direction. The authors conclude that the higher cortisol levels suggest “poorer stress coping, greater depression and greater risk for long-term neuronal and immunologic

damage" (Brody *et al.*, 2000, p.228). Due to the low prevalence of the DHA coping style in the general population, research into the area has been scarce. It has been reported, however, that certain chronic pain populations contain an unusually high proportion of DHA patients. Creswell and Chalder (2001) found that 46% of participants with chronic fatigue syndrome (CFS) were classified as DHA, compared to 17% of asymptomatic controls and 10% of diabetes. The inclusion of the diabetes group confirmed that the result was not simply caused by the anxiety associated with having a chronic condition (Creswell & Chalder, 2001). This finding for CFS is particularly interesting given that a cognitive-behavioural model of CFS proposed that it is characterised by low levels of self-esteem accompanied by rigid defence mechanisms (Surawy, Hackmann, Hawton, & Sharpe, 1995). In addition to the effect of high defensiveness, high levels of anxiety may also have an adverse effect on clinical outcome, perhaps resulting in avoidance of physical and functional restoration activities (Burns, 2000a).

To date, the effect of coping styles, or even the prevalence of different coping styles, within the CLBP population, has not been addressed. It may be expected that, despite reporting low anxiety and disability, an initial vigilant response will cause repressors to exhibit similar physiological reactivity (such as elevated muscle activity) as high anxious individuals in stressful situations. Additionally, by attempting to suppress pain, they may actually experience a high sensitivity to pain in the long term. Repressors may also be less willing to seek or accept treatment and may be over-optimistic about their ability to perform tasks, resulting in persistence behaviours and a cycle of over and under-activity. Therefore the theory suggests that a repressive coping style in patients with CLBP will be associated with a poor long term outcome. The information on the effect of a DHA coping style and the cognitive biases associated with it are more limited and it is not clear how such a coping style would affect clinical outcome in the case of CLBP. In general it is

assumed that DHA individuals act in a similar manner to those who are high anxious, although it is apparent that important differences do exist. For example, DHA individuals were found to spend less time worrying about future negative events (Eysenck & Derakshan, 1997) and were less vigilant for threat (Ioannou, Mogg, & Bradley, 2004) than high anxious individuals, which suggests a defensive coping style may have some benefits. High prevalence in certain chronic conditions, however, suggests an overall negative impact of the DHA coping style. Given the issues associated with repressors and DHA patients, the presence of both should be considered in CLBP patients, particularly when investigating links between psychological and physiological factors.

2.6. Psychological factors and muscle activity

2.6.1. *Muscle guarding*

LBP patients who have high levels of pain-related fear generally exhibit greater paraspinal EMG readings compared to low fearful patients, especially when confronted with movements which they believe are harmful (Vlaeyen & Linton, 2000). Fear-induced increase in paraspinal muscle activity has also been demonstrated to predict greater pain during a subsequent physical performance test (Vlaeyen *et al.*, 1999). Vlaeyen *et al.* therefore propose that pain-related fear perpetuates pain and disability through muscular reactivity, implying that reduction in fear-avoidance beliefs may reduce muscular reactivity, and thereby disability. In support of this notion, fearful patients demonstrate less relaxation at the end of trunk flexion (Geisser *et al.*, 2004) than non-fearful patients and controls, leading to a significant, inverse relationship between pain-related fear and the flexion-relaxation ratio (FRR) (Watson *et al.*, 1997a). Significant correlations have also been found between reductions in fear-avoidance beliefs, increases in pain self-efficacy beliefs and increased FRRs following a pain management programme (Watson *et al.*, 1997a). Changes in range of motion or self-report of pain were not related to changes in

the FRR. This muscle guarding is thought to continue despite the resolution of the physiological cause and conditioning might mean that simply anticipating a movement results in a muscular guarding response (Main & Watson, 1999). Over time, abnormal motion and a transfer of loads to other structures of the musculoskeletal system may contribute to the development of disuse and deconditioning (Verbunt *et al.*, 2003a). This may therefore represent one pathway by which fear avoidance leads to increased disability as suggested by the fear-avoidance model. Support for the role of conditioned muscular responses in chronic pain was provided by a study investigating muscular factors in relation to both conditioned and unconditioned pain stimuli. The chronic pain patient groups had both greater muscular responses to the unconditioned pain stimulus and also a higher number of conditioned responses compared to healthy controls. Furthermore, a significant relationship was found between muscular responses and the experience of pain the following day (Klinger *et al*., 2010).

2.6.2. *Catastrophising and muscle activity*

van der Hulst, Vollenbroek-Hutten, Rietman and Hermens (2010b) responded to the suggestion in pain-models such as the fear-avoidance model, that coping strategies may play a role in the 'chronification' of pain via changes in physical activity. They investigated the relationships between three coping strategies (labelled 'catastrophising', 'distraction', and 'persistence and control') and lumbar muscle activity during walking in 63 patients with CLBP. Catastrophising was significantly related to increased lumbar muscle activity during all periods of stride, and distraction was found to be related to increased relative relaxation during the swing phase. Catastrophising is associated with both avoidance behaviour and difficulty in disengaging from pain (Van Damme *et al.*, 2004) and the higher muscle activity associated with catastrophising would support the view that this is a maladaptive coping strategy (although it has also been suggested that

catastrophising may more closely reflect emotional distress than a coping strategy (van der Hulst *et al.*, 2010b)). As alternation between relaxation and exertion is thought to reflect a healthy pattern of muscle activity during walking, this may suggest that distraction is an adaptive strategy. However, this study does not enable analysis of any long term effects. In particular, the use of distraction to control pain has been associated with greater pain once distraction ceases (Goubert *et al.*, 2004). Distraction also represents a form of avoidance, which is often considered a maladaptive long-term coping strategy (Richards & Steele, 2007).

2.6.3. *Diathesis-stress*

In a widely reported series of studies, Flor *et al.* (Flor, Turk, & Birbaumer, 1985; Flor *et al.*, 1992) found that EMG amplitudes in the lumbar paraspinal muscles increased for CLBP patients compared to controls when they were exposed to personally relevant stressful situations, although the muscle activity levels of more distal sites were not significantly different between groups. The patients with high paraspinal reactivity appeared to be those who were most depressed, worried and emotionally affected by their pain problem. The authors therefore proposed a diathesis-stress model in which they suggest that CLBP may be the consequence of extensive and sustained reactions of the back muscles resulting from the interaction of personally-relevant stressful events with a predisposing organic or psychological condition. Increases in muscle activity might lead to ischemia, reflex muscle spasms, oxygen depletion and the release of pain-eliciting substances, with the subsequent pain itself potentially a further stressor (Flor *et al.*, 1985). The appeal of this model over the pain-spasm-pain model is that, since the muscle hyperactivity is dependent on the presence of a relevant stressor, it would explain the apparent inconsistent muscle activity findings for CLBP patients. Other studies have supported this model and also demonstrated symptom specific muscular hyper-reactivity to

a wider range of stressors, including personal, cognitive and social stressors (DeGood, Stewart, & Adams, 1994; Glombiewski *et al.*, 2008), anger (Burns, 2006), and pain (Watson, 2002), with Burns (2006) finding that paraspinal reactivity correlated significantly with everyday pain severity. CLBP patients have also shown slower recovery in muscle activity levels than controls following stress (Burns, 2006; Flor *et al.*, 1985), possibly related to the underestimation of high muscle activity that has been observed in CLBP patients (Flor, Fürst, & Birbaumer, 1999). It is not known why CLBP patients exhibit this symptom specific response. Knost, Flor, Birbaumer and Schugens (1999) suggest that chronic pain patients might have learned to increase their muscle activity as a pain inhibitory mechanism (via increased non-nociceptive input and diversion of attention) when anticipating stress/pain. When maintained, however, this strategy may itself induce pain via ischemia and hypoxia.

2.6.4. *Stress, spinal loading and muscle relaxation*

Marras *et al*. (2000) demonstrated that psychosocial stress is associated with mean activity increases of between 3.5% and 6.5% in trunk muscles and a 12% increase in lateral shear, with results varying markedly between individuals. The increased spine loadings were traced to greater trunk muscle co-activity. This study provides an indication for the first time that there is a biomechanical pathway to spinal loading associated with psychosocial stress. A subsequent study (Davis, Marras, Heaney, Waters, & Gupta, 2002) also found that mental stress led to poorer co-ordination and greater trunk muscle co-activation, resulting in increased spinal loading. Although both studies identified that certain personality types responded with significantly greater spinal loading, the traits that appeared to make patients more susceptible were different in each case, perhaps reflecting the way different pressures affect different personality types (Davis *et al.*, 2002).

Recent research has identified a significant correlation between perceived psychosocial work stress and muscle activity. A significant negative correlation has also been observed between perceived negative stress and the amount of muscle rest time (Rissén, Melin, Sandsjö, Dohns, & Lundberg, 2000). Lack of muscle relaxation time in particular has been suggested as a contributory factor in the development and perpetuation of chronic pain (Hägg & Åström, 1997; Hermens & Hutten, 2002), suggesting that stress may play an important role in the maintenance of chronic pain.

2.7. Management of low back pain

Approximately four million people see their GP with back pain each year (Waddell, 2004), with medication the most commonly prescribed treatment (Airaksinen *et al.*, 2006). Although there is no cure for LBP, a wide range of different treatments exist. Broadly, these can be categorised into the following areas: education/information, exercise, manual therapies (including manipulation, massage, mobilisation), other non-pharmacological interventions (including transcutaneous electrical nerve stimulation (TENS), traction, heat/cold), psychological interventions (including cognitive behavioural therapy (CBT), graded exposure), combined physical and psychological interventions (including multidisciplinary rehabilitation programmes), pharmacological interventions (including anti-depressants, opioids, muscle relaxants and non-steroidal anti-inflammatory drugs (NSAIDS)), invasive procedures (including acupuncture), and surgical referral (NICE, 2009). The recent NICE guidelines for LBP promote patient education and state that the key priorities are to initially offer patients a structured exercise programme, a course of manual therapy or a course of acupuncture. Patients who have received at least one less intensive treatment and have high disability and/or significant psychological distress should then be considered for referral for a combined physical and psychological treatment

comprising around 100 hours. Only after this should surgery be considered (NICE, 2009). A number of the available treatments for LBP are discussed in more detail below.

2.7.1. *Exercise*

Exercise has become an increasingly popular treatment choice for LBP and there is some evidence to suggest that regular exercise also decreases the risk of recurrence (Rainville *et al.*, 2004). 'Exercise therapy' encompasses a wide range of different programmes, although there appears to be little difference between aerobic exercise, muscle reconditioning or physiotherapy exercises in relation to their effect on pain and disability up to twelve months after treatment (Airaksinen *et al.*, 2006; Mannion, Müntener, Taimela, & Dvorak, 1999). Although there is 'strong' evidence that exercise therapy is more effective in reducing pain and/or disability than 'GP care', the "European Guidelines for the management of chronic non-specific LBP" (the European Guidelines) (Airaksinen *et al.*, 2006) also conclude that there is also strong evidence that these changes in pain and disability are not related to changes in any aspect of physical performance capacity. Perhaps then, exercise may (to some extent) improve outcome via its effect on pain-related fears and beliefs and by changing behaviours (Mannion *et al.*, 1999; Rainville *et al.*, 2004). Exercise therapy may additionally help to reduce depression and also lead to a greater feeling of well-being through achieving an increase in physical fitness (Mannion *et al.*, 1999). Overall, it may therefore play an important role in breaking the cycle of deconditioning, disability and depression suggested by the fear-avoidance model (Vlaeyen *et al.*, 1995).

2.7.2. *Manual therapies*

Evidence regarding the use for manual therapies (manipulation, mobilisation, massage) has been promising (Airaksinen *et al.*, 2006; Furlan, Imamura, Dryden, & Irvin, 2009; Koes,

Assendelft, Van der Heijden, & Bouter, 1996; NICE, 2009). Spinal manipulation is defined as a high velocity thrust to a joint beyond its restricted range of movement. Spinal mobilisation involves low-velocity, passive movements within or at the limit of joint range (Koes *et al.*, 1996). It has been suggested that one of the ways that both techniques reduce pain may be via altered paraspinal muscle activity, although the results appear inconsistent, with both increased and decreased activity of the paraspinal muscles being reported (Ferreira, Ferreira, & Hodges, 2007). Krekoukias, Petty and Cheek (2009) recently observed a significant decrease in the EMG activity of erector spinae following mobilisation in an asymptomatic population, but the ability to generalise to a clinical population may be limited. For example, spinal manipulation was shown to increase the activity of the oblique abdominal muscles in LBP patients, but not controls (Ferreira *et al.*, 2007). In this study, postural activity of the trunk muscles of LBP patients was modified by spinal manipulation, but it did not appear to affect the postural response of TrA. DeVocht, Pickar and Wilder (2005) reported that in 27 of 31 paraspinal muscle sites, muscle activity was reduced following spinal manipulation (and by at least 25% in 24 of these sites), but in four cases EMG activity was higher. Lumbar spine manipulation on LBP patients has also been shown to have an immediate effect of reducing abnormally high EMG activity of paraspinal muscles during full flexion and hence increasing the FRR (Bicalho, Setti, Macagnan, Cano, & Manffra, 2010; Lalanne, Lafond, & Descarreaux, 2009).

2.7.3. *Superficial heat therapy*

Superficial heat therapy is commonly used to treat LBP, both by practitioners and by patients at home. It is generally accepted that heat increases the extensibility of collagen tissue, decreases joint stiffness, relieves muscle spasm, produces pain relief, and increases both blood flow and metabolism (Lehmann & Lateur, 1990). Although it is commonly

observed that muscle spasms can be reduced through the use of heat, the mechanisms that cause this are still not fully understood. It is thought that there is a selective cessation of firing of secondary spindle afferent endings which reduce muscle tone and this may be supplemented by greater inhibitory impulses from the Golgi tendon organs (Lehmann & Lateur, 1990). Heat is also widely used to relieve pain, although again the physiological data underpinning this is limited. It has been suggested that the thermal stimulus affects the pain sensation in line with the "gate theory" of Melzack and Wall (1965). Pain relief may also occur through the action of endorphins or via increased blood flow 'washing out' pain provoking metabolites (Low & Reed, 2000). Additionally, the interdependence of pain and muscle spasm implies that a reduction in one may cause a reduction in the other (Low & Reed, 2000). An animal study reported that superficial heat treatment also decreases pain-related behaviours. The effects of heat treatment were investigated on rats with knee joint inflammation. After treatment with heat, the rats reduced guarding behaviour and increased weight bearing on the inflamed limb (Sluka, Christy, Peterson, Rudd, & Troy, 1999).

A number of studies have considered the effect of heat on muscle activity. In general, the results have shown that the EMG amplitude during isometric contractions is unaffected by heating up to temperatures in the region of 40 - 45°C (Holewijn & Heus, 1992; Krause *et al.*, 2001; Mito *et al*., 2007; Petrofsky & Laymon, 2005), although changes in the mean frequency of the signal have been noted (Krause *et al*., 2001; Petrofsky & Laymon, 2005).

A Cochrane review (French, Cameron, Walker, Reggars, & Esterman, 2006) concludes that there is moderate evidence that heat therapy reduces short-term pain and disability for acute back pain, but that there is insufficient evidence regarding the effect of heat for CLBP. Most of the existing studies have been carried out by two research teams, both assessing the effect of the same disposable lumbar heat wrap (Thermacare heat wrap)

(Mayer *et al.*, 2005; Mayer *et al.*, 2006; Nadler *et al.*, 2002; Nadler *et al.*, 2003a; Nadler *et al.*, 2003b). The wrap heats to 40°C within 30 minutes of exposure to air and maintains this temperature for eight hours of continuous wear (Nadler *et al.*, 2003a).

Four of the five studies compared the heat wrap with various different interventions for patients with acute LBP (Mayer *et al.*, 2005; Nadler *et al.*, 2002; Nadler *et al.*, 2003a; Nadler *et al.*, 2003b). For the three Nadler studies, treatments were administered for either two or three days or nights, followed by two days of follow-up. By the end of the follow-up period, patients using the heat wrap had greater pain relief, reduced disability, less muscle stiffness and increased flexibility compared to those taking an oral placebo (Nadler *et al.*, 2003a) and either ibuprofen or acetaminophen (Nadler *et al.*, 2002). Overnight use of the wrap also provided effective pain relief throughout the next day, reduced muscle stiffness and disability and improved trunk flexibility compared to an oral placebo (Nadler *et al.*, 2003b). Mayer *et al*. (2005) investigated the efficacy of the wrap alone and combined with active exercise, versus exercise alone and a control (which in this case was an educational booklet) over five consecutive days. Patients receiving the combined heat and exercise treatment had significantly improved function and reduced disability compared to the other groups and pain relief was significantly greater than for exercise alone or the control group. These differences were most apparent at the end of the two-day follow-up period. It should be noted, however, that the participants in the combined group received approximately twice the amount of intervention and attention as either treatment alone, which may have affected the results.

A second study by Mayer *et al.* (2006) examined the efficacy of heat treatment in both preventing and treating delayed-onset muscle soreness (DOMS) in previously asymptomatic individuals. Vigorous eccentric exercise was performed to induce DOMS. In

the prevention sub-study participants who wore a heat wrap (for four hours before the exercise and four hours afterwards) had significantly less pain intensity, disability and deficits in function after 24 hours than the control group. The results of the treatment study were less convincing. Although pain relief was significantly less with heat treatment than with the cold pack after 24 hours, there was no difference in self-reported physical function or disability. It should also be noted that the heat therapy group had a longer treatment time than the cold therapy group. Furthermore participants were able to remain active while wearing the heat wrap, in contrast to the cold pack which was applied with the participant either prone or supine. Overall, however, the results suggest that heat therapy is beneficial in the prevention and early phase treatment of low back DOMS, contrary to the traditional practice of applying cold therapy for acute muscular pain.

Nuhr *et al*. (2004) carried out a study involving 100 patients with acute back pain during emergency transfer to hospital. Patients were randomly assigned to two groups: active warming with an electric heated blanket or passive warming with a woollen blanket. Those patients who received active heating had significantly lower pain and anxiety scores and a lower heart rate on arrival at hospital compared to those who received passive warming. The changes observed in the pain and anxiety scores were large enough to be clinically relevant as well as statistically significant. The authors suggest that as well as the physiological benefits, the psychological benefits should be considered such as increased central relaxation and increased feelings of well-being.

2.7.4. *Cognitive and behavioural treatments*

The main assumption of a behavioural approach is that pain and disability are not only influenced by pathology, but also by psychological and social factors (van Tulder *et al*., 2000). There are three general behavioural treatment approaches: operant treatments

include positive reinforcement of healthy behaviours and withdrawal of attention from pain behaviour; cognitive treatments are designed to modify patients' cognitions regarding their pain and disability e.g. by modification of maladaptive thoughts and beliefs; and respondent treatments aim to directly modify the physiologic response system e.g. by reduction of muscle activity via techniques such as relaxation or EMG biofeedback (van Tulder *et al.*, 2000). Pain Management Programmes, based on cognitive-behavioural principles, are becoming increasingly popular and are described as the treatment of choice for people with persistent pain by the British Pain Society (British Pain Society, 2007). Pain Management Programmes are delivered in a group format and consist of education and guided practice, typically involving goal-setting and exercise (British Pain Society, 2007). There is strong evidence for the effectiveness of behavioural treatments for LBP and CBT is therefore recommended for patients by the European Guidelines (Airaksinen *et al.*, 2006). It is also noted, however, that some patients appear to improve more than others and hence more research is needed on possible underlying mechanisms to define any subgroups which may benefit most from behavioural treatment (Airaksinen *et al.*, 2006).

2.7.5. *Graded exposure in vivo*

Graded exposure is a recently developed intervention aimed at chronic pain patients with high levels of fear and avoidance (Boersma *et al.*, 2004). It is based on the principles of systematic desensitisation and involves a gradual confrontation with feared activities, thereby challenging and disconfirming catastrophic expectations (Lohnberg, 2007). Exposure-based interventions have proved to be effective in significantly reducing fear beliefs, avoidance behaviour and pain-related anxiety, and in improving self-efficacy and function (Boersma *et al.*, 2004; Lohnberg, 2007; Woods & Asmundson, 2008). However, perhaps contrary to expectations, exposure has been shown to be effective only for patients

with low or moderate pre-treatment levels of catastrophising (Flink, Boersma, & Linton, 2010).

2.7.6. *Multidisciplinary rehabilitation programmes*

Multidisciplinary treatment programmes are based on the biopsychosocial model of illness (Karjalainen *et al.*, 2001) and usually consist of an extensive combination of physical, vocational and behavioural components, and the modification of medication use. True multidisciplinary treatment programmes have to be provided at least by three health care professionals with different clinical backgrounds (Airaksinen *et al.*, 2006). They are often carried out over a considerable number of hours and require substantial staff and financial resources (Karjalainen *et al*., 2001). There is strong evidence in their favour (Airaksinen *et al.*, 2006) however, with the NICE guidelines concluding that the best evidence for effectiveness exists for those programmes of over 100 hours of exposure (NICE, 2009).

2.7.7. *Antidepressants/muscle relaxants*

There is strong evidence that antidepressants are effective in relieving pain in CLBP patients, but they do not appear to improve function. The benefit also appears to be independent of depression status (Airaksinen *et al.*, 2006).

The European Guidelines concluded that, while there was some evidence that muscle relaxants were effective for the short-term relief of pain, in the studies included in the review, this did not always appear to occur via the reduction of muscle spasm (Airaksinen *et al.*, 2006).

2.8. Summary

It appears that (at least certain subgroups of) LBP patients tend to exhibit altered patterns of muscle activity and increased co-contraction, whether this is in response to a loss of stability, damage to ligaments and other soft tissues, or abnormal loading on spinal structures. Activity of the deep trunk muscles tends to be delayed and/or reduced, and is associated with hyperactivity of the superficial trunk muscles. The result is less control over the trunk and a reduction in position sense. Increased superficial muscle activity and co-contraction increase spinal compression and are linked to slower recovery of intervertebral disc height following loading, which may exacerbate the condition.

It is clear that psychological factors such as pain-related fear, catastrophising, self-efficacy and stress can play an important role in the development and perpetuation of LBP. The fear-avoidance model details one potential pathway by which such factors can result in an outcome of disuse and disability. Even here, disuse is taken not just to mean the avoidance of activity, but the abnormal motion or muscular guarding which is a recognised accompaniment to high levels of pain-related fear and which may persist beyond the resolution of the physical cause. This supports the proposition that altered muscle activity may be a contributory factor in the link between psychological factors and pain and disability.

Although much research has focussed on either the biomechanical or psychological aspects of LBP, a number of researchers have linked the two strands of research. In particular, fear-avoidance beliefs have been associated with muscle guarding (Vlaeyen *et al.*, 1999), and especially loss of the FRP (Geisser *et al.*, 2004); pain-related fear has been demonstrated to replicate the delayed activation of deep trunk muscles and increase in superficial muscle activity observed in LBP patients (Moseley *et al.*, 2003); catastrophising

is associated with higher lumbar muscle activity during walking (van der Hulst *et al.*, 2010a); and stress has been linked to symptom specific muscular hyper-reactivity (Flor *et al.*, 1992), lack of muscle relaxation (Rissén *et al.,* 2000) and an increase in spinal loading via greater trunk muscle co-activity (Marras *et al.,* 2000). Altered muscle activity therefore represents a common factor in both biomechanical and psychological models and may be a cause of the persistence or recurrence of the condition. One of the mechanisms by which this may occur is via delayed recovery of intervertebral disc height (and hence stature) following loading. However, research in this area is scarce and it remains to be seen if improved stature recovery, via reduced muscle activity, improves clinical outcome. In addition, further research is required to investigate the relationship between altered muscle activity, psychological factors and pain and disability in patients with LBP.

CHAPTER THREE

3. General methods

NHS patients

All three studies involved NHS patients with CLBP. The self-reported duration of back pain varied from 3 months to 40 years. Patients were recruited from the waiting list for two physiotherapist-led rehabilitation programmes in North Manchester. In the case of Studies 1 and 3, participants also included patients who had attended one of the programmes during the previous two years. All participants of both the longitudinal and heat treatment investigations for Studies 2 and 3 respectively were also included for baseline analysis in Study 1 and some patients took part in all three studies. Potential participants identified from the waiting lists were sent information about the study in the post (Appendix 1) and asked to return a reply slip if they were willing to take part. A diagram of the procedure for those who were recruited from the waiting lists is given in Figure 3.1 on page 82.

In addition, to increase participant numbers for Study 3, patients who had been through the rehabilitation programmes in the last two years were contacted and invited to take part in the study (Appendix 2). These participants completed two sessions only, one with a heat wrap and one without. The results for the sessions without a heat wrap were also included in Study 1. The numbers of participants included in each study are shown in Figure 3.2 on page 83. All testing of NHS patients took place at North Manchester General Hospital.

Figure 3.1. Procedure for patients recruited from waiting lists

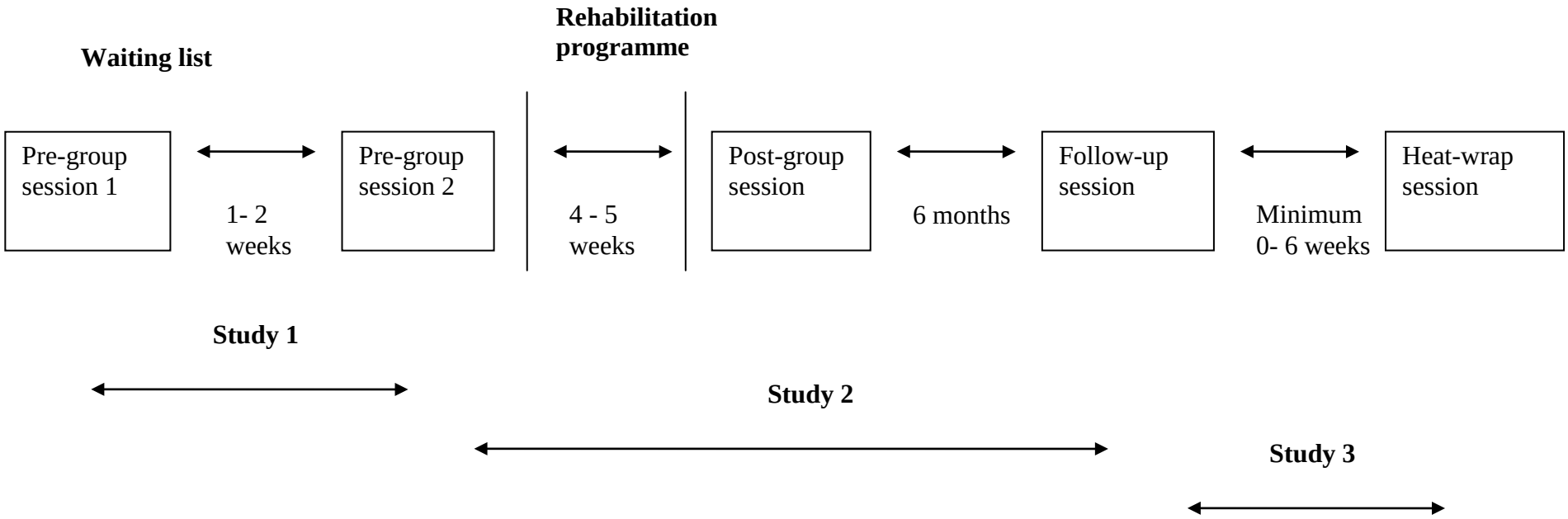
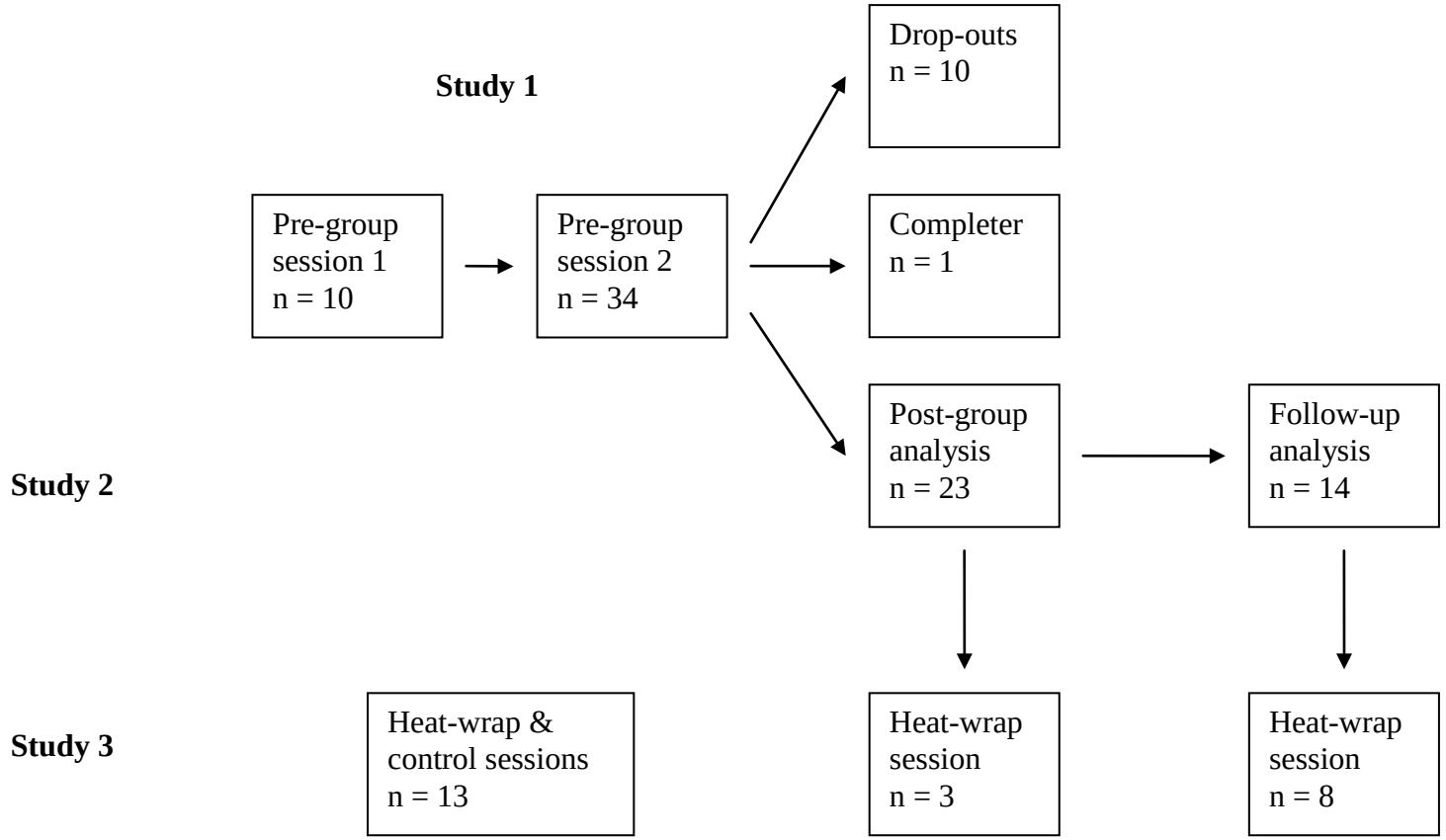


Figure 3.2. Numbers of patients recruited to each study



Completer: completed the rehabilitation programme but did not return for the post-group session

Exclusion criteria were nerve root compression, central nervous system impairment, progressive motor deficit, sphincter impairment from neurologic cause and presence of 'red flags' (e.g. unexplained weight loss, recent urinary tract infection, history of intravenous drug use). Many of the patients were taking pain-killers for their back pain as it was not considered practical to exclude those on medication. Participants were offered £12.50 for each session they attended to cover travel and parking expenses. Ethical approval was granted by the North Manchester NHS ethics committee and local NHS permission was granted by Pennine Acute NHS Trust. All participants provided written informed consent (Appendix 3).

Asymptomatic participants

Candidates were excluded from the asymptomatic group if they had, at any time; experienced recurring or persistent back pain; lost a working day because of back pain; or had consulted a physician about back pain within the last 15 years. Participants were people known personally to the author and were not directly matched to the patient group.

The stadiometer

Changes in stature were measured with a standing stadiometer, which consisted of a rigid frame, mounted at a right angle to a base plate and inclined backward 15° from the vertical. Four anatomical points were identified and then supported by the frame to maintain the natural contours of the head and spine. These points were (1) the most posterior distension of the head at the occipitut, (2) the deepest point of cervical lordosis approximate to C4, (3) the most prominent point of the thoracic kyphosis approximate to the inferior angle of the scapula (T7), and (4) the deepest point of lumbar lordosis approximate to L3. The position of the feet was marked and head position was controlled by the use of spectacle frames with attached lasers. These were aligned with two movable targets above the participant's

head. A high-resolution linear variable displacement transducer (LVDT) was used to detect changes in stature by measuring vertical displacement with an accuracy of approximately 0.1mm. The LVDT was fixed to the top of the stadiometer and positioned to lie directly above the apex of each participant's head. The information was observed graphically on a laptop computer at the time of collection and stored digitally for later analysis at a sampling rate of 100Hz.

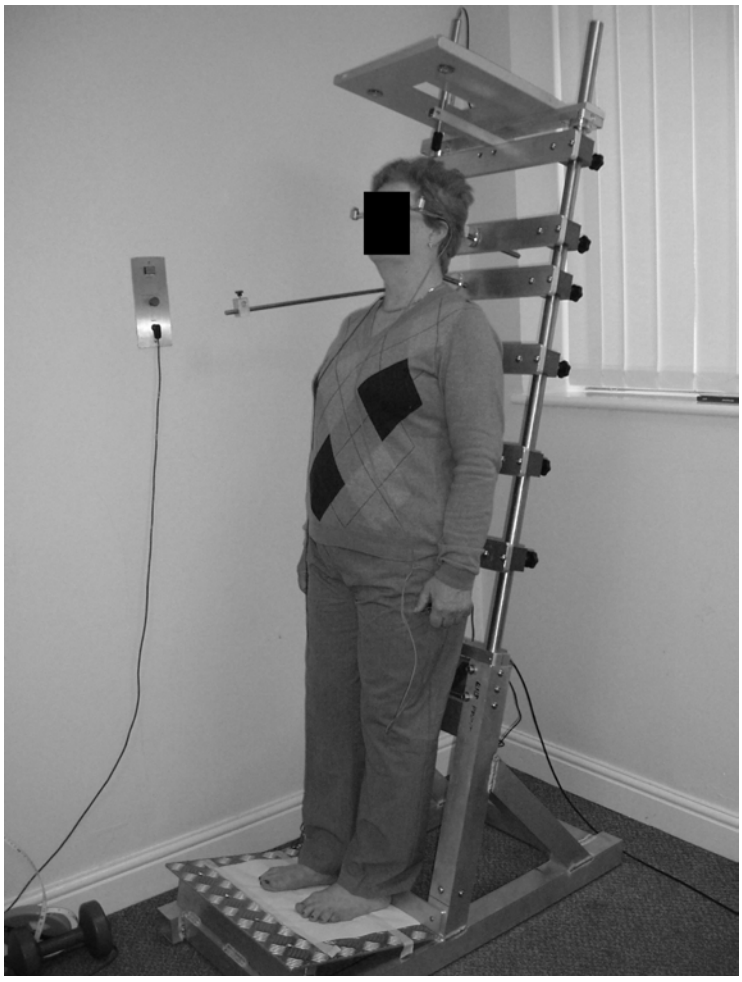


Plate 3.1. Participant in position in the stadiometer

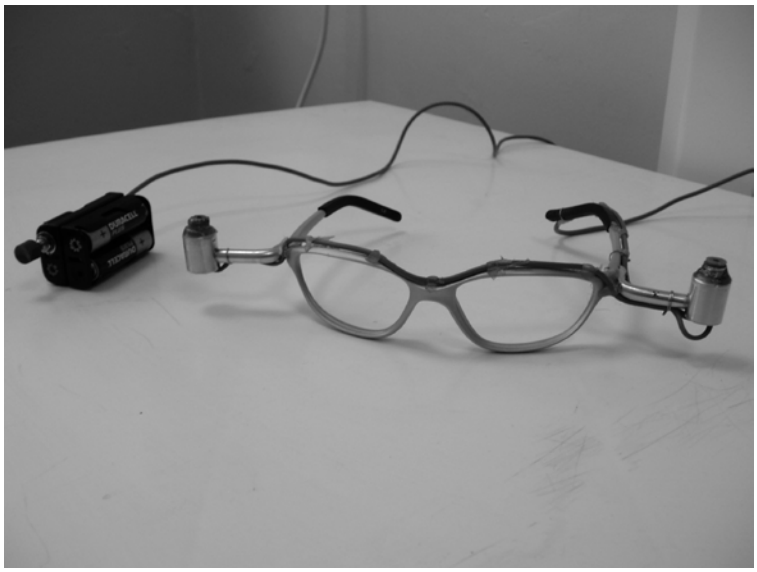


Plate 3.2. Spectacle frames with lasers

All participants initially undertook a brief familiarisation session on the stadiometer to enable them to practice the adoption of a repeatable and comfortable posture. This consisted of five recordings, between which the participant was asked to lean forward and break contact with the postural controls before resuming their position for the next measurement. A pilot study demonstrated that this approach was sufficient to produce reliable stadiometer readings (average SD 1.0mm, SEM 0.8mm).

Testing was carried out in both the morning and afternoon as research has indicated that this does not significantly affect measurements of stature change (Puntumetakul, Trott, Williams, & Fulton, 2008). However, participants who attended multiple testing sessions attended at the same time of day each time (or within one hour of the previous time), except in one case as noted within Study 2 when, due to work commitments, the patient attended in the morning for the first visit and the afternoon for the two subsequent visits. Participants remained in position for a period of 20 seconds and the stature value used was the mean reading over the final 10 seconds. This approach was based on the findings of a pilot study which confirmed that readings measured in this way were not significantly different to averaging over a 30 second period, whilst reducing the amount of time that the

participant was required to remain in position. All measures were recorded as changes from the baseline as this first measure was arbitrary and dependent on the initial location of the LVDT.

Muscle Activity Measurement

Raw electromyographic signals were recorded using a DELSYS EMG system (Delsys Inc. Boston, MA, USA). Single differential surface electrodes consisting of two silver bars with an inter-electrode spacing of 10mm were used. Signals were band-pass filtered between 20 and 450 Hz with a sampling frequency of 1000Hz. The raw EMG signal was then rectified, integrated and normalised to the reference.

Dry shaving, skin abrasion and cleaning with alcohol preceded electrode placement. Electrodes were placed over the erector spinae muscle at the L1-2 and L4-5 interspaces, approximately 3cm from the midline. These landmarks were the same as those used by Healey *et al.* (2005a) and were chosen to avoid unwanted signal effects related to the innervation zones and to minimise cross-talk (Healey *et al.*, 2005a). After the same skin preparation, the reference electrode was placed on the right iliac crest. Participants then assumed a standing posture for ten seconds while a recording was taken.

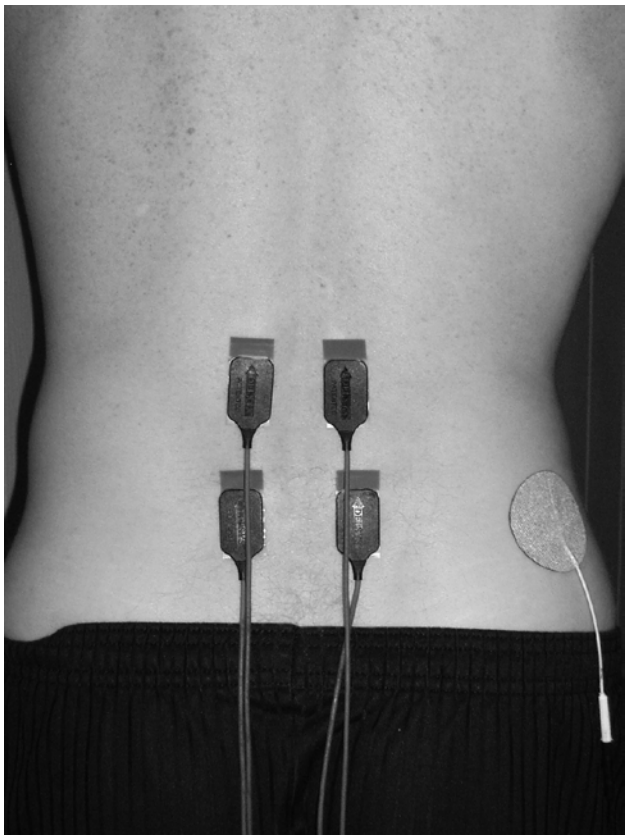


Plate 3.3. EMG electrode placement

The electromyographic data were normalised relative to a reference voluntary contraction (RVC). This reference task required each participant to stand while holding a specific mass (0.5kg) in each hand with arms bent (upper arms horizontal, lower arms vertical) for ten seconds (shown in Plate 3.4).

Plate 3.4. Participant performing a reference voluntary contraction (RVC)

The raw electromyograms (EMGs) were rectified and integrated over a period of five seconds. This approach was the same as that used by Healey *et al.* (2005a) and is recognised as an appropriate processing method (Burden, 2008). The EMG reading and the RVC were both taken to be the average of the three readings recorded during the session. All processed integrated EMGs recorded throughout the testing session were expressed as a percentage of the integrated EMG from the RVC.

Normalising relative to a sub-maximal RVC has been shown to improve the repeatability of the EMG signal (Lehman, 2002) and this is the most common approach when testing LBP patients who may be unwilling or unable to perform a maximal contraction due to

(fear of) pain/(re)injury. However, it has also been suggested that non-normalised values are preferable for clinical studies since the EMG activity during the reference contraction may be affected in the same manner as the levels during the experimental task (van Dieën *et al*., 2003b). Therefore non-normalised values were also subject to analysis.

Measures

Patients completed a series of self-report measures following each visit (Appendix 4). The patients were asked about their pain intensity during the past 24 hours at the end of the testing session. Although some patients completed the remaining questionnaire booklet immediately, the majority completed it at home and returned it either by post or on their subsequent visit. A self-report measure was classified as incomplete if there were three or more data items missing. If there were either one or two items missing, then these were assumed to be the same as the answers filled in by the same participant on the previous or subsequent questionnaire (as applicable). If only one questionnaire had been completed, the mean score from the completed items on that measure was used.

Pain intensity

A numerical rating scale (NRS) (see Appendix 4) was employed to assess pain intensity. Participants were asked to rate their pain during the past 24 hours on a scale ranging from (0) “no pain” to (10) “worst possible pain”. Research supports the reliability and validity of numerical rating scales of pain intensity (Jensen, 2003) and the 11-point NRS was recently recommended by the Initiative on Methods, Measurement and Pain Assessment in Clinical Trials (IMMPACT) to assess chronic pain intensity (Dworkin *et al.*, 2005).

Disability

The Roland Disability Questionnaire (RDQ; Roland & Morris, 1983) (see Appendix 4b) is a 24-item self-report measure that assesses disability due to back pain. Patients are asked to select which statements, related to perceived limitations in typical daily activities, apply to them. The score is calculated by adding up the number of selected items, so that a higher number represents greater perceived disability. The RDQ has excellent reliability, validity and responsiveness (Roland & Fairbank, 2000; Turner, Fulton-Kehoe, Franklin, Wickizer, & Wu, 2003) and is widely used to sample within the specific population for this study.

Anxiety and Depression

The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) (see Appendix 4a) is a widely used measure, designed for use with general medical outpatient populations. It consists of anxiety and depression subscales, both of which have seven items. Each item is rated on a four-point scale from 0 to 3, determining the extent to which the individual feels that each statement applies to them. A higher score reflects greater levels of anxiety or depression. Both subscales of the HADS have established validity and reliability in a clinical population (Johnston, Pollard, & Hennessey, 2000; Pallant & Bailey, 2005; Bjelland, Dahl, Haug, & Neckelmann, 2002).

Functional self-efficacy

Functional self-efficacy refers to the confidence that an individual has in their ability to successfully accomplish functional activities. The functional subscale of the Chronic Pain Self-Efficacy Scale (CPSS-PF; Anderson, Dowds, Pelletz, Edwards, & Peeters-Asdourian, 1995) (see Appendix 4c) was used to assess functional self-efficacy. The original CPSS-PF, which has been shown to be reliable and valid (Anderson *et al.*, 1995), is scored on a ten-point Likert scale, with higher scores reflecting higher levels of self-efficacy. This

study used a nine-point Likert scale because it provides patients with a mid-point option. The nine-point version has also been shown to have excellent internal consistency and test-retest reliability with CLBP patients (Woby *et al.*, 2007a).

Fear of movement/(re)injury

The Tampa Scale of Kinesiophobia (TSK; Kori, Miller, & Todd, 1990) (see Appendix 4e) aims to measure fear of movement/(re)injury in individuals with pain. It consists of 17 items, for which patients rate themselves on a four-point Likert scale ranging from "strongly disagree" to "strongly agree". Four of the items are inversely scored. Scores range from 17 to 68, with higher scores representing greater fear of movement/(re)injury. The English version of the TSK has been found to be valid and reliable (Woby, Roach, Urmston, & Watson, 2005; French, France, Vigneau, French & Evans, 2007).

Catastrophising

The Pain Catastrophising Scale (PCS; Sullivan, Bishop, & Pivik, 1995) (see Appendix 4f) consists of 13 items describing thoughts or feelings that may be experienced when in pain. For each statement, participants rate the extent to which they have the feeling when experiencing pain on a five-point Likert scale ranging from (0) "not at all" to (4) "all the time". A higher score indicates greater levels of pain catastrophising. In addition to a total score reflecting the level of catastrophising, there are three subscales assessing rumination, magnification and helplessness. The PCS has been shown to have good reliability and validity (Sullivan *et al.*, 1995; Osman *et al.*, 1997; Osman *et al.*, 2000).

Pain-related anxiety

The Pain Anxiety Symptoms Scale-20 (PASS-20) (see Appendix 4d) is a shortened 20-item version of the original Pain Anxiety Symptoms Scale (PASS; McCracken, Zayfert, &

Gross, 1992), a 40-item measure developed to assess pain-related anxiety in individuals with chronic pain. Higher scores represent higher levels of pain-related anxiety. Both the PASS and the PASS-20 consist of four subscales that assess fearful appraisal of pain, cognitive anxiety, physiological anxiety and escape/avoidance behaviour. The PASS-20 has been shown to have good factorial validity, internal consistency and close association to the original 40-item version (Coons, Hadjistavropoulos, & Asmundson, 2004; Roelofs *et al.*, 2004).

General procedure

On first attending, each participant was verbally informed about the testing procedures and requirements of the study and signed an informed consent form. Height and body mass were recorded. Participants then undertook a brief familiarisation session on the stadiometer as described above.

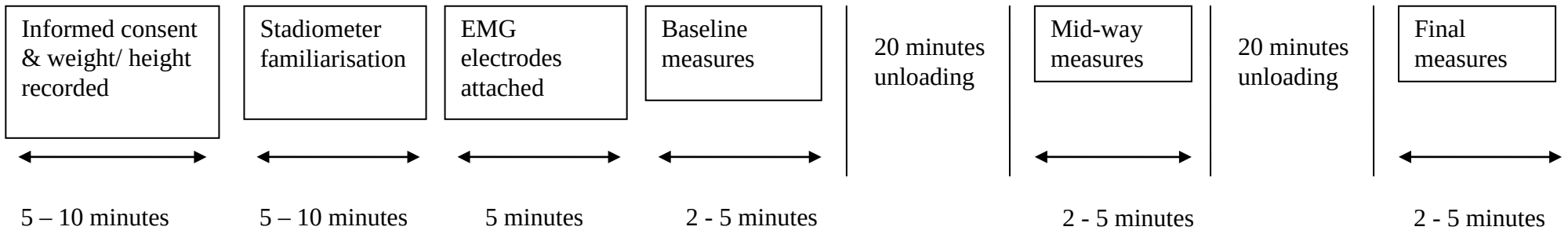
After this familiarisation period, electrodes were attached to the skin overlying the paraspinal muscles and initial EMG readings (at rest and during the RVC) were taken. Since these were performed in a standing position, the time taken for the EMG readings also allowed soft tissue deformation to stabilise, which reduced the likelihood of it influencing the ensuing stature measurements. Participants then assumed the correct position within the stadiometer and a baseline stature measurement was recorded.

Following these baseline measurements, participants assumed an unloading position on a physiotherapy bed for 20 minutes. This was either a side-lying or a prone position, whichever the participant felt was the most comfortable. After 20 minutes, the participants stood up and performed the same EMG and stadiometer measurements, before assuming the same unloading position for a further 20 minutes. The same measurements were then

taken for a final time. The protocol for an initial visit, with approximate timings, is shown in Figure 3.3 on page 95.

The protocol involved measurement of stature recovery rather than stature loss, which has been covered more widely in the literature. This decision followed the findings of Healey *et al.* (2005a), who reported that, although stature loss occurred at the same rate for individuals with CLBP as for asymptomatic controls, stature recovery was significantly delayed in the CLBP group. The unloading period of 40 minutes was also based on the study by Healey *et al.* (2005a), who similarly used a 40-minute unloading period, although in that case it was preceded by a 20-minute loading task. The severity of the back pain experienced by some of the patients in all three studies meant that a similar weight-bearing task, or even a simple walking task, was not considered appropriate for this thesis. However, a pilot study confirmed that even in the absence of an initial loading task, a significant height gain was still demonstrated after 40 minutes of unloading.

Figure 3.3. Protocol for initial testing session



Sample size

Healey *et al*. (2005a) reported significant negative correlations between stature recovery and each of muscle activity (r = -0.68), perceived pain (r = -0.45) and perceived disability (r = -0.43). Power analysis (Cohen, 1988) based on these correlation results suggested a sample size of 11 would be required to achieve power of 80% in detecting a link between baseline measures of stature recovery and muscle activity. Similarly, the sample sizes required for detecting the expected correlations with pain and disability were 29 and 32 respectively. On this basis, a minimum sample size of 40 patients was selected for Study 1. No studies had been published concerning the relationship between changes in muscle activity and stature recovery following either a CLBP rehabilitation programme or superficial heat treatment. As such, *a priori* power analysis was not possible for Studies 2 and 3. Most published studies assessing stature change had used sample populations of approximately 20 participants in each group. Studies of clinical outcome have varied greatly in size; therefore, an initial analysis of approximately 20 participants was performed to establish the basis for a power analysis, with the results discussed in the General Discussion section.

Statistical analysis

Data analysis was conducted using SPSS software (version 16.0). Parametric tests were employed provided that all assumptions were met. In all cases a probability of $p < 0.05$ was considered to be significant. Pearson's product moment and regression analyses were implemented to assess the relationships between the data sets obtained. Reliability was assessed via the intraclass correlation coefficient and the standard error of measurement.

Limitations

The thesis is inevitably affected by the heterogeneous nature of CLBP, with specific symptoms varying between patients. This is a common issue in research of this kind. This heterogeneity, however, also enabled analysis of the data with the aim of identifying meaningful subgroups within the patient population. The patient group also varied in the severity of their back pain with some patients having only mild levels of pain and disability.

Many of the patients were taking pain-killing medication and were not asked to cease this for the purposes of the study. This may have affected some of the measures, particularly the self-report of pain. For Study 2, some patients also changed their use of medication during the course of the study. One reason for this was that effective medication use was one of the topics covered in the rehabilitation programmes.

The patient group covered a wide range of ages, heights and weights, all of which are known to affect the stature change response (Althoff *et al*., 1992; Rodacki, Fowler, Provensi, Rodacki, & Dezan, 2005), although allowance was made for this in the data analysis. Although no patients reported that standing in the stadiometer was painful, a few patients did report mild discomfort, either when in position or when stepping in or out. This may have affected their ability to relax and hence the reliability of the subsequent measurements. However, all patients were able and willing to complete all the stadiometer measurements. Ideally all testing would have taken place at the same time of day, although this is not thought to significantly affect stature change results (Puntumetakul *et al.*, 2008). It was also not possible to control the spinal loading that occurred prior to the participants attending each testing session. This may therefore have varied between both participants and visits.

The method of assessing muscle activity has its limitations. Absolute EMG amplitudes are affected by a variety of factors such as electrode position, skin and muscle temperature and thickness of the subcutaneous fat layer, leading to the argument that comparison of EMG amplitudes between individuals should not be carried out directly (Burden, 2008). A normalised value was therefore used, but, as reported by van Dieën *et al.* (2003b), the EMG activity during the reference contraction may also be altered in CLBP patients. Additionally, a few patients found even the relatively easy RVC used in this thesis caused slight discomfort, which may have affected the readings.

Some patients completed the questionnaire immediately, but many took it away with them and, in some cases, may have filled it in some time later, possibly affecting the answers given. Although all the questionnaires used in this thesis have been found to be reliable and valid, they relied on patients' self-report, rather than any objective measure. Anecdotally, it was noted in particular that some patients verbally reported feeling better and in less pain on their return visit, but completed the pain NRS to show the same or a higher pain level than previously.

CHAPTER FOUR

4. Study 1: The relationships between baseline measures of stature recovery, muscle activity and psychological factors in chronic low back pain patients

4.1. Introduction

Although conflicting results have been reported, individuals with CLBP have often been found to exhibit hyperactivity of the superficial paraspinal muscles during static postures such as standing (e.g. Ambroz *et al*., 2000). Linked to this elevated paraspinal muscle activity is the finding that individuals with CLBP appear to exhibit increased loading of the intervertebral discs as manifested by delayed stature recovery after loading (Healey *et al.*, 2005a). Healey *et al*. (2005a) observed significantly reduced stature recovery in a group of individuals with mild CLBP (who all self-managed their pain) compared to a control group, with stature recovery negatively correlated with paraspinal muscle activity. Healey *et al*. (2005a) hypothesized that the elevated muscle activity observed in the CLBP group resulted in greater compressive loads on the spine that, in turn, prevented the intervertebral discs from regaining their initial height and consequently prolonged stature recovery. Reduced stature recovery may increase the risk of future back pain and increase loading on spinal structures such as the facet joints (Adams *et al*., 2002). Significant negative correlations between stature recovery and both pain and disability support the clinical relevance of this relationship (Healey *et al.*, 2005a).

As discussed in Chapter 2, psychological factors play an important role in LBP, possibly via increased spinal loading resulting from altered paraspinal muscle activity. Muscle activity may, therefore, be a contributory factor in the link between psychological factors and clinical outcome.

The aim of this study was to extend the findings of Healey *et al.* (2005a) by analysing the relationship between stature recovery, muscle activity, pain and disability in a more clinically relevant population of NHS patients with CLBP, including individuals with more severe back pain than previously examined. In addition, the study furthered previous work by seeking to establish whether a range of self-report psychological factors were linked to muscle activity or stature change in this group. An asymptomatic control group was also included to enable comparison of stature change and muscle activity levels between the two groups. It was hypothesized that, consistent with the findings of Healey *et al.* (2005a), the patients with CLBP would have higher muscle activity and reduced stature recovery compared to the asymptomatic group and that stature recovery would be negatively related to each of muscle activity, pain and disability. Although research into the area is limited, there has been support for associations between paraspinal muscle activity and psychological factors such as pain-related fear (Vlaeyen *et al.*, 1999), stress (Flor *et al.*, 1992) and catastrophising (van der Hulst *et al.*, 2010b). It was therefore expected that muscle activity would be correlated with the psychological factors considered in the current study. It was also thought that such factors may impact on stature recovery via their influence on muscle activity and hence lead to observed correlations between stature change and a range of psychological variables.

The specific hypotheses addressed by this study were as follows:

(1) patients with CLBP demonstrate acceptable levels of repeatability for inter-day stadiometer measurements;

(2) patients with CLBP have higher muscle activity and reduced stature recovery compared to asymptomatic individuals;

(3) stature recovery for patients with CLBP is negatively related to each of muscle activity, pain and disability;

(4) muscle activity for patients with CLBP is negatively related to pain and disability; (5) psychological factors are associated with paraspinal muscle activity and stature recovery for patients with CLBP.

4.2. Methods

Participants

Data were collected from 47 patients with CLBP (age, 46.6 ± 11.1y; height, 166.4 ± 7.5cm; body mass, 79.3 ± 18.8kg) who had been referred to a physiotherapist-led rehabilitation programme in North Manchester and 18 asymptomatic controls (age, 44.6 ± 13.3y; height, 169.0 ± 10.3cm; body mass, 70.8 ± 12.7kg). The CLBP group was mixed (18 men, 29 women), between the ages of 23 and 70 years. The control group consisted of nine men and nine women, between the ages of 25 and 64 years. Further details of the patients are provided in the General Methods section. Candidates were excluded from the control group if they had, at any time; experienced recurring or persistent back pain; lost a working day because of back pain; or had consulted a physician about back pain within the last 15 years.

Procedure

The majority of patients attended the testing session shortly before starting the rehabilitation programme, although six patients attended shortly after the programme commenced and the study also included 13 patients who had completed the programme within the last two years. Where possible, patients attended twice before the commencement of the programme to enable test-retest analysis. Healthy controls completed one testing session each and completed only the anxiety and depression (HADS) and defensiveness questionnaires (see page 103). Testing was carried out in morning and afternoon sessions. Patients who attended twice came at the same time (or

within one hour of the previous time) for both visits. Paraspinal muscle activity was assessed via surface electromyography (EMG), whilst both standing at rest and during a reference voluntary contraction (RVC). Stature recovery over a 40-minute unloading period was measured on a standing stadiometer. Details of the protocol are provided in the "General Methods" section. Patients also completed a series of self-report measures following their visit. In addition to the measures described in the "General Methods" section, an additional questionnaire to assess defensiveness was used for this study.

Defensiveness

The ten item short form Marlowe-Crowne Social Desirability Scale (MC-SD) (see Appendix 4g) was used to assess defensiveness. Participants answer "True" or "False" to each of ten statements concerning personal attitudes and traits e.g. "I have never intensely disliked anyone". The items are thought to reflect culturally approved behaviours but behaviours believed unlikely to occur. A correlation coefficient $r > 0.9$ between the 10-item version and the original 33-item scale (Strahan & Gerbasi, 1972) and an acceptable level of reliability (alpha coefficient = 0.66) have been reported (Reynolds, 1982).

Identifying coping style with HADS and MC-SD

The anxiety scale of the HADS and the MC-SD were used to classify participants as high-anxious, low-anxious, defensive high-anxious and repressors. Methods of classifying the different coping styles vary. For this study, tertiary splits based on normative data for the HADS (Crawford, Henry, Crombie, & Taylor, 2001) was used with low anxious classified as a score of four or below and high anxious as eight and above. Similar normative data was not available for the specific short form Marlowe-Crowne scale used in this study. However, two population studies based in Chicago reported a mean score of 5.4 (standard deviation of 2.2, mode of 5) for 614 randomly selected residents aged between 18 and 40

(Johnson & Fendrich, 2002), and a mean score of 6.8 (standard deviation of 2.3, mode of 7) for 588 randomly selected women aged 50 and over (Burris *et al*., 2003). Both studies used the short version of the MC-SD with a highest possible score of 10. It is observed both that women typically score higher than men and that the older the respondent, the more likely they are to score highly (Burris *et al.,* 2003), which may explain the different findings from the two studies. Based on these average results, a score of eight and above was considered to be high-defensive.

Analysis

For the patients who attended two testing sessions prior to the programme, reliability of measures, particularly the inter-day stature recovery measurements, was assessed via the intraclass correlation coefficient (ICC), the standard error of measurement (SEM) and the mean of the standard deviations (SDs). To compare the data between the CLBP patients and the asymptomatic controls, one-tailed t-tests were calculated to examine for differences in levels of muscle activity, stature recovery, anxiety, depression and defensiveness. For the CLBP patients, Pearson's correlation coefficient was calculated to investigate the relationships between baseline measures of stature recovery, muscle activity, pain, disability and the range of psychological factors considered.

Hierarchical multiple regression analysis was performed to investigate the extent to which the variance in disability could be explained. Age and sex were entered in Step 1 of the analysis, pain was entered in Step 2, and self-efficacy and a combined anxiety and depression score (represented by the total HADS score) were entered as Step 3. The approach taken for Steps 1 and 2 was based on a similar regression analysis by Woby *et al.* (2007a), in which demographics were included as covariates in Step 1, and then, because previous research has shown it to be a strong predictor of disability, pain was included as

an additional covariate. The factors selected for Step 3 were those that had the strongest relationships with disability based on the results of the zero-order correlational analysis. Due to the common co-occurrence of anxiety and depression in clinical populations and overlap of symptoms (Rutledge *et al*., 2009), a composite measure of anxiety and depression was included in the analysis. The predictor variables all had variance inflation factors below ten and tolerance levels higher than 0.2. It was also verified that there were no cases with a Cook's distance of greater than one nor leverage values that were three times the average value. Furthermore less than 5% of cases had standardised residuals greater than two.

Mediational analysis, including Sobel's test of mediation, was carried out to investigate whether muscle activity acted as a partial mediator between a range of psychological factors and either pain or disability. In each case, three linear regressions were performed. In Analysis 1, either pain or disability was the dependent variable and the psychological factor was the independent variable. In Analysis 2, muscle activity was the dependent variable and the psychological factor was again the independent variable. In the third analysis, either pain or disability was the dependent variable and the psychological factor and muscle activity were the independent variables. If muscle activity did mediate the relationship between the psychological factor and either pain or disability then the relationship with muscle activity should remain significant after controlling for the psychological factor, whereas the impact of the psychological factor should be significantly less.

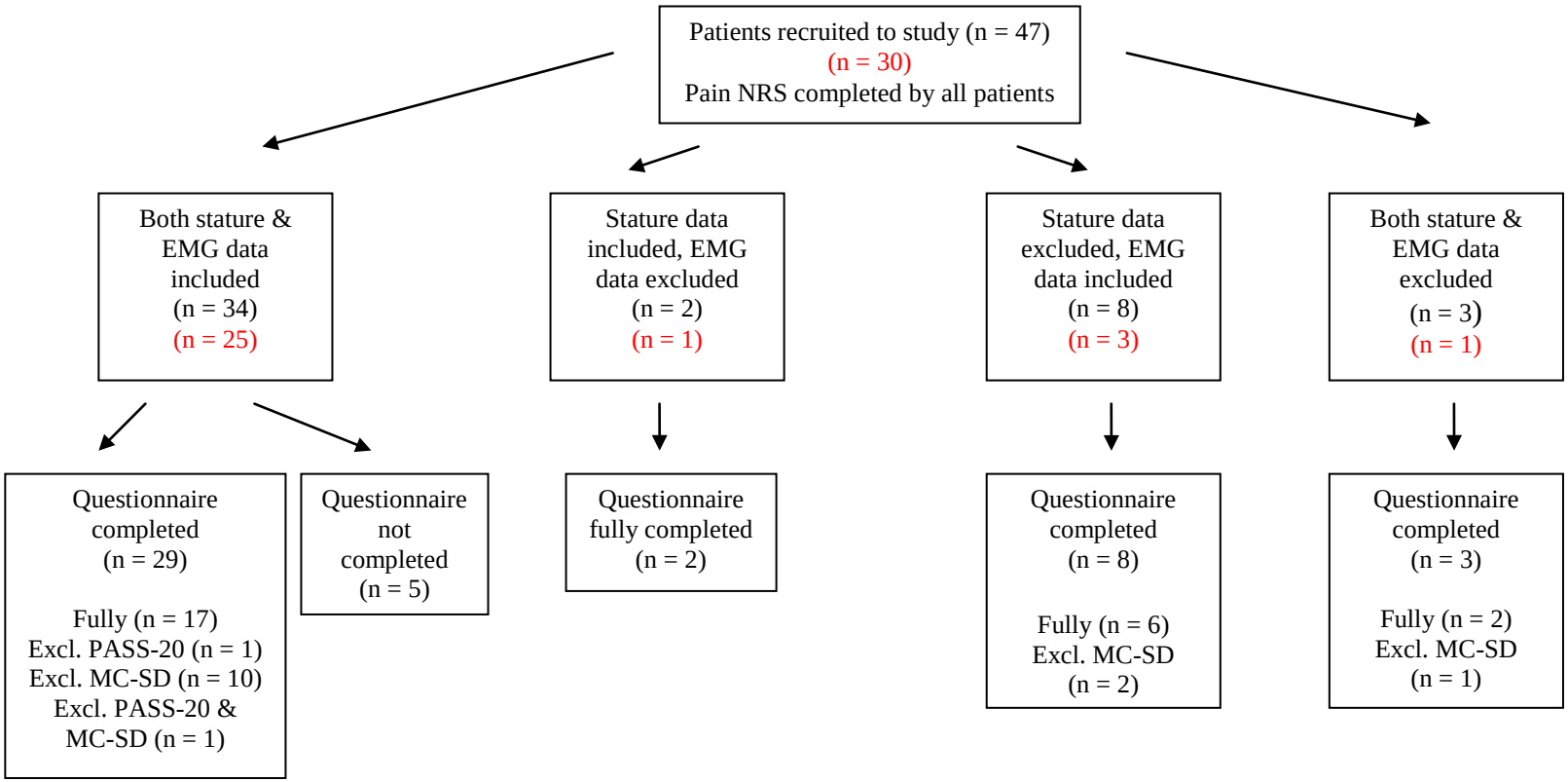
Similar mediational analysis was carried out to investigate whether pain (or disability) mediated the relationship between muscle activity and disability (or pain) and also whether muscle activity acted as a mediator between disability and pain.

In each case, a one-tailed Sobel test was calculated to determine the statistical significance of any mediational effect. It was verified that there were no cases with a Cook's distance of greater than one nor leverage values that were three times the average value and, to control for multi-collinearity, variance inflation factors had to be below ten.

4.3. Results

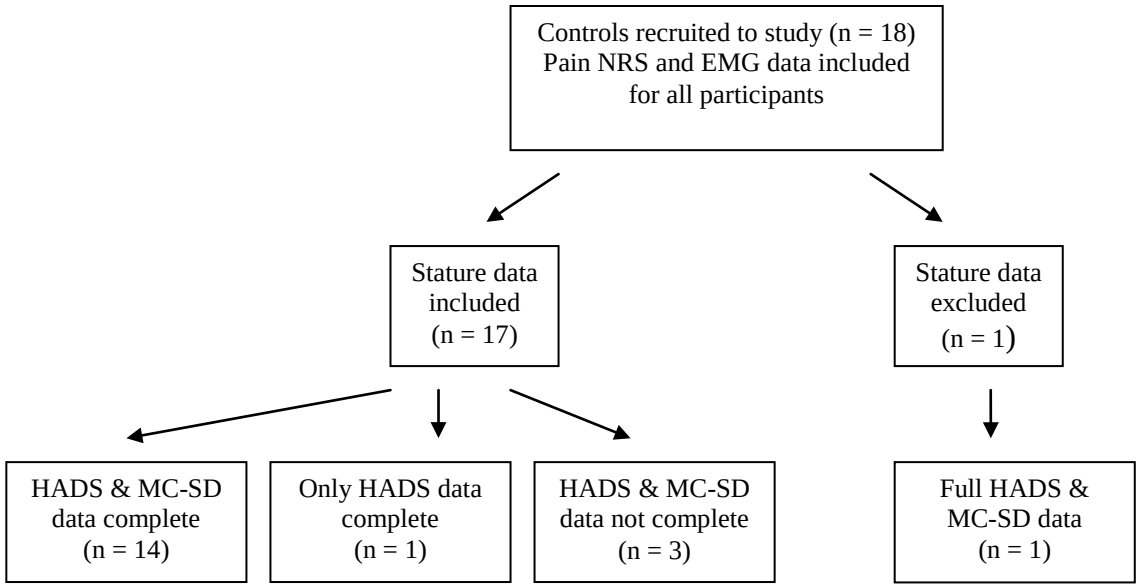
Parametric tests were used, based on the results of Kolmogorov-Smirnov and Shapiro-Wilk tests of normality. As can be seen in Table 4.2 on page 113, the sample represented moderate levels of pain and disability. The mean duration of pain was 7.2 years and the median was 2 years (range: 3 months – 40 years). 42 patients and 15 asymptomatic participants completed the questionnaires. Some participants found it difficult to maintain a consistent posture in the stadiometer. Eleven patients and one control group participant were excluded from the stature recovery data as the standard deviation (SD) of the five familiarisation readings was considered to be too high. For this purpose a SD of 1.7mm was taken as the cut-off point as it marked a clear gap between the majority of participants and the relatively small number with large SD values. The remaining patients had an average SD of 1.0mm over the five familiarisation readings, which compared to an average SD of 1.0mm for the control group. Technical problems with the EMG system at the start of the study meant that EMG data were not recorded for two patients. In addition, one patient was excluded from the EMG data due to high noise levels in the signal (possibly as this patient asked not to be shaved at the electrode site), which meant that the values were considered unreliable. The EMG data of two further patients were excluded from the analysis as they were materially different to the values of all other patients (muscle activity was < 30% of RVC) and they were therefore considered to be outliers. The numbers of patients and controls whose data was included in the analyses are set out in Figures 4.1 on page 107 and Figure 4.2 on page 108 respectively.

Figure 4.1. Numbers of patients included in the analyses (figures in red reflect the numbers included in the matched comparison of baseline factors)



Fully: All sections of the questionnaire were completed; Excl. PASS-20: the questionnaires were all completed with the exception of the Pain Anxiety Symptoms Scale-20; Excl. MC-SD: the questionnaires were all completed with the exception of the Marlowe-Crowne Social Desirability Scale; Excl. PASS-20 & MC-SD: the questionnaires were all completed with the exception of the Pain Anxiety Symptoms Scale-20 and the Marlowe-Crowne Social Desirability Scale

Figure 4.2. Numbers of controls included in the analyses

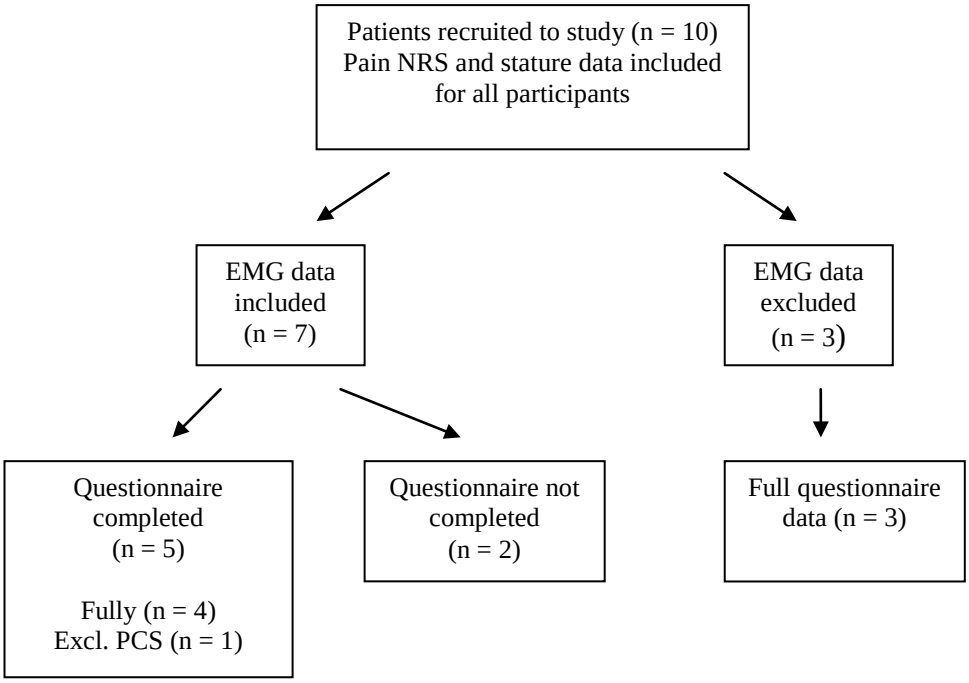


HADS: The Hospital Anxiety and Depression Scale; MC-SD: the Marlowe-Crowne Social Desirability Scale

Repeatability of measures

Ten patients attended two testing sessions before commencement of the programme, and eight of these completed a questionnaire each time. Due to technical problems with the EMG system, only seven had complete EMG readings for both visits. The participant numbers are detailed in Figure 4.3.

Figure 4.3. Numbers of patients included in the repeatability study



Fully: All sections of the questionnaire were completed on both visits; Excl. PCS: the questionnaires were all completed on both visits with the exception of the Pain Catastrophising Scale

The results from the two visits are shown in Table 4.1 on page 110. The average SD of the two stadiometer readings was 1.4mm, ICC was 0.72 and the SEM was 1.4mm. Additionally, some of this variation may have been expected due to a difference in pain scores between the two sessions. Therefore, it was concluded that the stadiometer readings reached acceptable levels of repeatability.

Table 4.1. Repeatability of measures

Variable	Visit 1 Mean (± SD)	Visit 2 Mean (± SD)	Average SD	ICC	SEM
Stature change (mm)	1.8 (± 3.5)	1.7 (± 1.9)	1.4	0.72	1.4
Muscle activity (% of RVC)	77.1 (± 8.4)	79.5 (± 6.9)	7.9	*	16.2
Disability	12.1 (± 4.8)	11.8 (± 5.5)	1.5	0.92	1.4
Pain NRS	5.4 (± 2.8)	6.1 (± 2.7)	0.7	0.93	0.7
Anxiety	10.3 (± 4.2)	10.0 (± 4.2)	1.4	0.89	1.4
Depression	8.1 (± 1.9)	8.4 (± 2.2)	0.9	0.75	1.0
Functional self-efficacy	49.5 (± 13.2)	41.8 (± 15.6)	5.8	0.97	2.7
Pain-related anxiety	41.4 (± 21.7)	44.4 (± 27.9)	6.5	0.95	5.6
Catastrophising	21.3 (± 12.4)	23.6 (± 13.6)	5.7	0.85	5.0
Fear of movement	37.5 (± 7.3)	39.1 (± 7.6)	3.0	0.80	3.4

* The ICC in respect of muscle activity has been excluded because it resulted in a negative figure (as inter-individual differences between patients were small, but two patients had large variations between visits).

Relationships between changes in outcome measures over the two visits prior to commencement of the rehabilitation programme

Correlational analysis was carried out on the changes in each of the outcome measures over the two visits, although the limited participant numbers involved in this analysis should be noted. There was a significant correlation between changes in stature recovery and changes in each of muscle activity ($r = -0.68$, $p = 0.05$), disability ($r = -0.66$, $p = 0.04$), and anxiety ($r = -0.73$, $p = 0.02$). There was also a significant correlation between changes in muscle activity and changes in self-efficacy ($r = -0.96$, $p < 0.01$), although this was based on the results for five patients only.

Baseline characteristics

The characteristics of the patient and control groups (where applicable) are given in Table 4.2 on page 113. As the location of patients' pain (e.g. right or left side) was not recorded, the four EMG electrode sites were not considered separately and the muscle activity figures presented are the average of the four electrode sites used.

As shown in Table 4.2, the control group had significantly greater stature recovery and less muscle activity (as a % of the RVC) compared to the patient group. However, the control group had significantly lower body mass, a factor known to affect stature change. Any differences in body fat would also be expected to affect EMG readings. The patient group was additionally shorter in stature on average and although the difference in body height did not reach significance, it may have influenced the stature recovery results. Therefore, the control group was also compared with a matched (age, sex, height, weight) subset of the patient group (as shown in Table 4.3 on page 114). In this case, there was a trend for reduced stature recovery and elevated muscle activity in the patient group compared to the control group, but it no longer reached significance.

The baseline characteristics have also been shown separately for the 30 patients who attended the BEG and the 17 patients who attended the WBTL group in Table 4.4 on page 115. On average, the patients in the WBTL group were more disabled than the BEG group in terms of pain, disability, anxiety, depression, self-efficacy, pain-related anxiety and catastrophising. Muscle activity was also significantly higher for the WBTL group, but there was no difference in stature recovery.

Table 4.2. Baseline characteristics

Variable	Patient group Mean (± SD)	Control group Mean (± SD)	Significance	Effect size	Possible range
Body mass (kg)	79.3 ± 18.8	70.8 ± 12.7*	p = 0.04	0.45	
Stature change (mm)	2.6 (± 2.2)	3.7 (± 1.4)*	p = 0.03	0.50	
Muscle activity (% of RVC)	75.5 (± 12.2)†	66.8 (± 15.8)*	p = 0.01	0.71	
Disability	11.5 (± 5.9)				0-24
Pain NRS	4.8 (± 2.2)				0-10
Anxiety	8.5 (± 3.7)	5.2 (± 2.8)**	p < 0.01	0.88	0-21
Depression	6.3 (± 3.4)	3.5 (± 2.1)**	p < 0.01	0.80	0-21
Functional self-efficacy	45.5 (± 18.3)				0-72
Pain-related anxiety	42.4 (± 23.9)				0-100
Catastrophising	21.1 (± 12.8)				0-52
Fear of movement	36.7 (± 9.3)				17-68
Defensiveness	7.8 (± 1.9)	4.9 (± 1.3)**	p < 0.001	1.47	0-10

† The muscle activity details for the patient group exclude two outliers. If these two patients' details are included, the mean (± SD) would be 73.1% (± 16.3%), p = 0.09, effect size = 0.38.

* p < 0.05, ** p < 0.01

Table 4.3. Baseline characteristics of matched patient and control groups

Variable	Patient group Mean (± SD)	Control group Mean (± SD)	Significance	Effect size
Age (yrs)	43.8 (± 10.6)	44.6 (± 13.3)		
Height (cm)	167.2 (± 6.8)	169.0 (± 10.3)		
Body mass (kg)	71.6 (± 11.9)	70.8 (± 12.7)		
Disability	10.3 (± 5.6)			
Pain NRS	4.4 (± 2.1)			
Stature change (mm)	2.7 (± 2.4)	3.7 (± 1.4)	p = 0.06	0.44
Muscle activity (% of RVC)	72.8 (± 13.0)	66.8 (± 15.8)	p = 0.09	0.42

Table 4.4. Baseline characteristics for the BEG and WBTL groups

Variable	BEG Mean (± SD)	WBTL Mean (± SD)	Significance	Effect size
Body mass (kg)	82.3 (± 13.2)	78.9 (± 26.7)		-0.26
Stature change (mm)	2.7 (± 2.2)	2.4 (± 2.2)		-0.18
Muscle activity (% of RVC)	72.6 (± 12.5)	81.2 (± 9.7)*	p = 0.02	0.69
Disability	9.7 (± 5.2)	15.1 (± 5.6)**	p < 0.01	1.05
Pain NRS	4.3 (± 2.1)	5.8 (± 2.1)*	p = 0.01	0.70
Anxiety	7.2 (± 2.8)	10.8 (± 4.2)**	p < 0.01	1.31
Depression	5.0 (± 2.6)	8.5 (± 3.5)**	p < 0.01	1.34
Functional self-efficacy	53.4 (± 14.7)	31.3 (± 15.6)**	p < 0.01	-1.49
Pain-related anxiety	35.2 (± 19.0)	57.3 (± 26.8)**	p < 0.01	1.16
Catastrophising	17.8 (± 10.3)	27.0 (± 14.9)*	p = 0.01	0.89
Fear of movement	35.6 (± 9.0)	38.8 (± 9.6)		0.35
Defensiveness	7.7 (± 1.9)	7.9 (± 2.1)		0.13

* $p < 0.05$, ** $p < 0.01$

Correlational analysis: muscle activity

When analysing the baseline measures for the patients with CLBP, significant correlations were found between muscle activity and both pain ($r = 0.48$, $p < 0.01$) (Figure 4.4) and disability ($r = 0.43$, $p < 0.01$) (Figure 4.5).

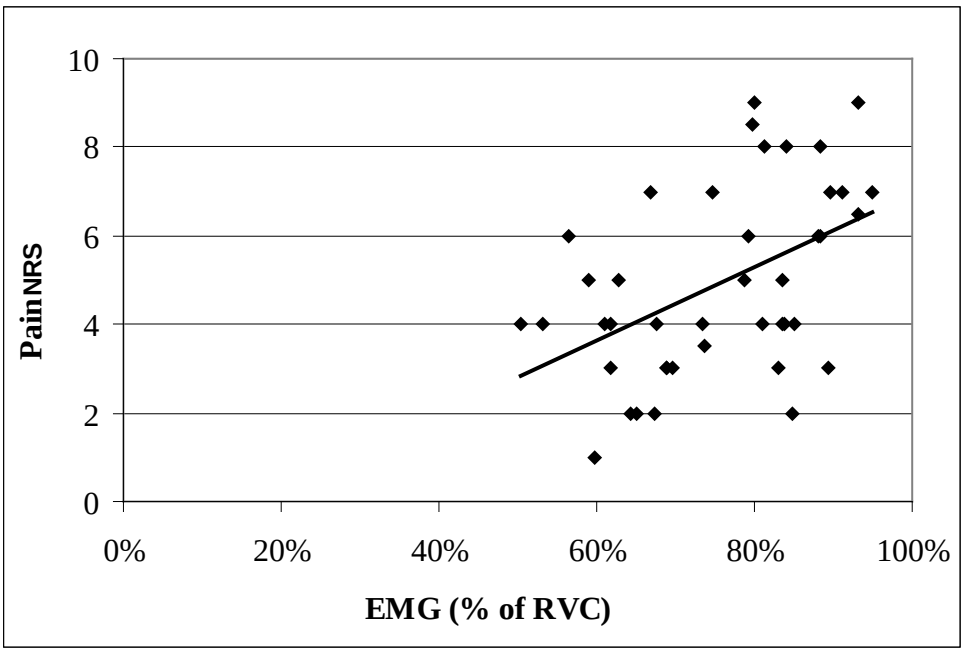


Figure 4.4. Relationship between pain and muscle activity

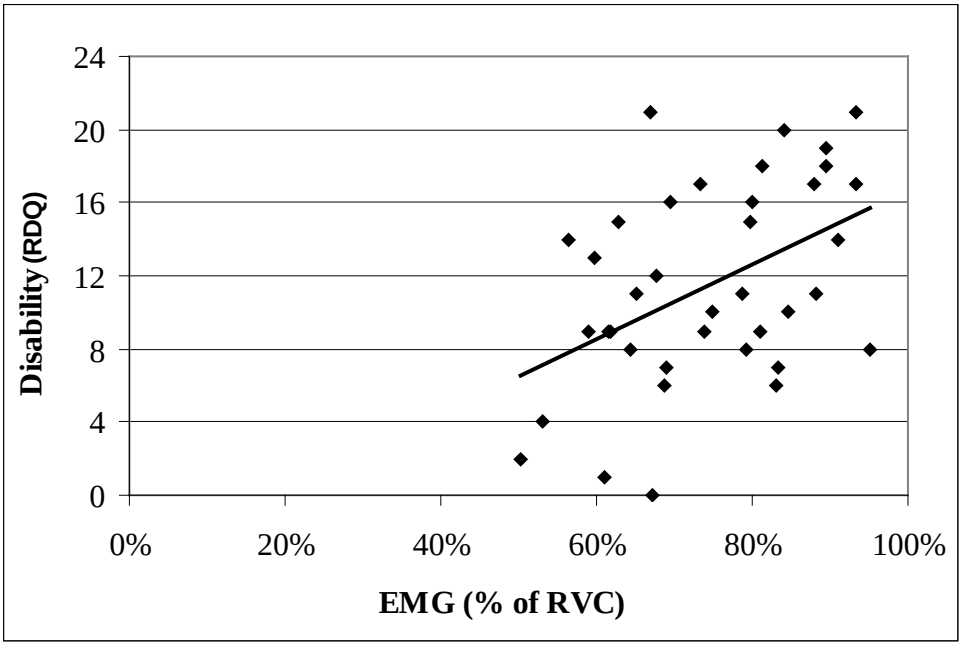


Figure 4.5. Relationship between disability and muscle activity

From the psychological factors considered, muscle activity was correlated with functional self-efficacy ($r = -0.45$, $p < 0.01$) (Figure 4.6), depression ($r = 0.33$, $p = 0.03$) (Figure 4.7), anxiety ($r = 0.31$, $p = 0.03$), pain-related anxiety ($r = 0.29$, $p = 0.05$) and catastrophising ($r = 0.29$, $p = 0.04$). There was also a trend for a negative link with defensiveness ($r = -0.28$, $p = 0.07$).

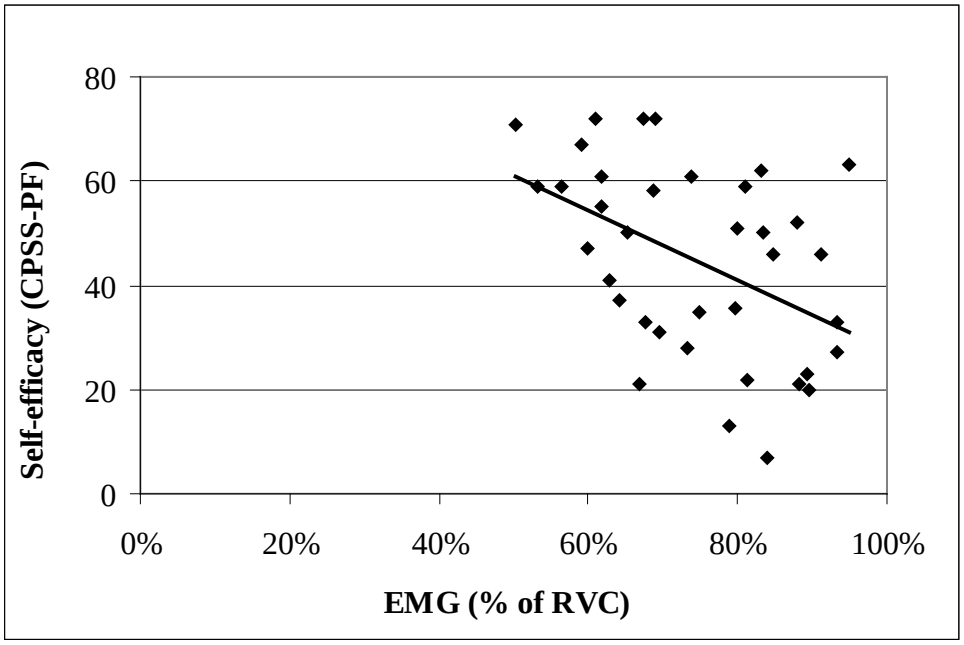


Figure 4.6. Relationship between self-efficacy and muscle activity

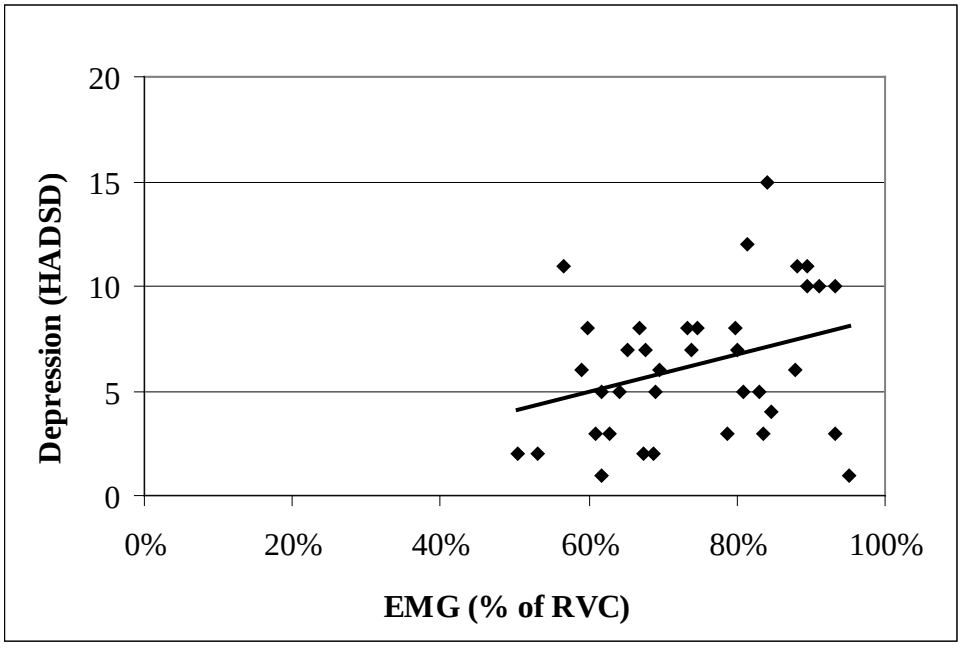


Figure 4.7. Relationship between depression and muscle activity

When the subscales of the PASS-20 and PCS were considered separately, muscle activity was correlated with both cognitive anxiety and helplessness.

Correlational analysis was also carried out to investigate links with the non-normalised, integrated EMG, rather than the value normalised relative to the RVC. Non-normalised muscle activity was found to be significantly related to fear of movement ($r = 0.34$, $p = 0.02$) and catastrophising ($r = 0.31$, $p = 0.03$) and there was a negative correlation with defensiveness ($r = -0.46$, $p = 0.01$).

Correlational analysis: stature recovery

The relationship between the baseline measures of stature recovery and pain for the CLBP patients is shown in Figure 4.8.

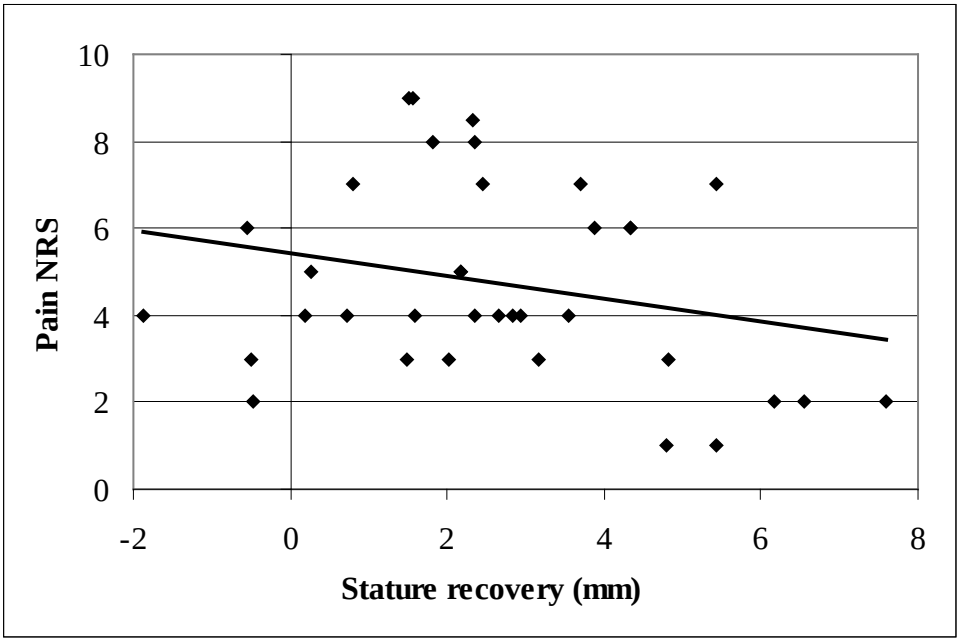


Figure 4.8. Relationship between pain and stature recovery

There were trends for links between stature recovery and pain ($r = -0.23$, $p = 0.09$) and depression ($r = 0.25$, $p = 0.08$). Stature recovery was not significantly related to disability or any of the psychological factors.

Relationship between muscle activity and stature recovery

The relationship between muscle activity and stature recovery (as shown in Figure 4.9) was not found to be significant.

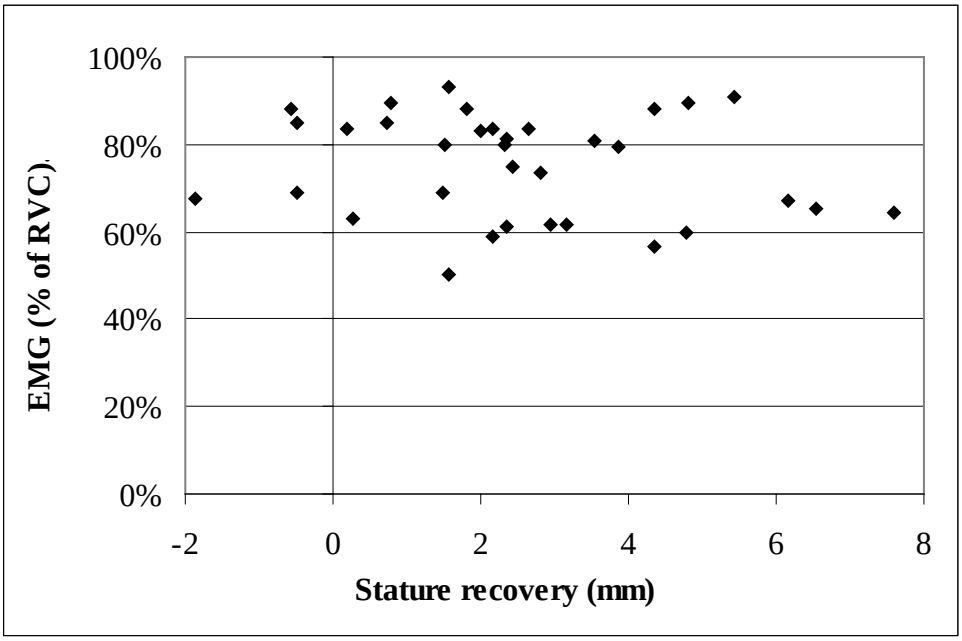


Figure 4.9. Relationship between muscle activity and stature recovery

The correlations between all the measures are shown in Table 4.5 on page 120.

Table 4.5. Correlation coefficients between outcome measures

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Stature change	-										
2. Muscle activity	-0.18	-									
3. Disability	-0.09	0.43**	-								
4. Pain	-0.23	0.48**	0.57**	-							
5. Anxiety	0.10	0.31*	0.55**	0.42**	-						
6. Depression	0.25	0.33*	0.57**	0.34*	0.68**	-					
7. Self-efficacy	0.00	-0.45**	-0.73**	-0.49**	-0.47**	-0.57**	-				
8. Pain-related anxiety	0.13	0.29*	0.54**	0.37*	0.67**	0.57**	-0.56**	-			
9. Catastrophising	0.13	0.29*	0.41**	0.38**	0.59**	0.63**	-0.45**	0.81**	-		
10. Fear of movement	0.12	0.20	0.39**	0.27*	0.57**	0.44**	-0.25	0.63**	0.65**	-	
11. Defensiveness	0.21	-0.28	-0.27	-0.08	-0.15	-0.10	0.02	-0.14	-0.12	-0.19	-

* $p < 0.05$, ** $p < 0.01$

Correlational analysis: BEG and WBTL

Patients in the WBTL group were more disabled than those in the BEG on most of the baseline measures considered. Therefore separate correlational analysis between stature recovery and muscle activity and the other outcome measures was carried out for the two groups to investigate whether the results differed.

Table 4.6. Correlation coefficients between stature recovery, muscle activity and the other outcome measures for the BEG and WBTL groups

	Stature recovery		Muscle activity	
	BEG (n = 22)	WBTL (n = 9)	BEG (n = 25)	WBTL (n = 12)
1. Stature change	-	-		
2. Muscle activity[†]	-0.45*	0.76*	-	-
3. Disability	-0.25	0.40	0.31	0.36
4. Pain	-0.39*	0.25	0.45*	0.37
5. Anxiety	-0.13	0.71*	-0.10	0.78**
6. Depression	0.23	0.51	-0.07	0.67*
7. Self-efficacy	0.02	-0.26	-0.34	-0.31
8. Pain-related anxiety	0.16	0.33	0.03	0.65*
9. Catastrophising	0.10	0.25	0.00	0.61*
10. Fear of movement	0.07	0.27	-0.03	0.65*
11. Defensiveness	0.18	0.48	-0.28	-0.41

[†] n = 24 for BEG, n = 10 for WBTL

As shown in Table 4.6, the relationships between muscle activity and psychological factors were notably stronger for the more disabled WBTL group than the BEG. For the BEG, stature recovery was negatively correlated with muscle activity and pain. In contrast, for the WBTL group, stature recovery showed positive correlations with muscle activity and

anxiety. However, it should be noted that participant numbers were limited for the stature recovery analysis for the WBTL group and the more disabled patients also tended to be those who were less reliable on the stadiometer, as confirmed by the trend for a greater SD on the familiarisation readings ($p = 0.06$) for these patients compared to the BEG.

Regression analysis

The results of the overall correlational analysis were used to guide selection in a stepwise multiple regression analysis to investigate the extent to which the variance in disability could be accounted for by self-efficacy and a combined anxiety and depression score (represented by the total HADS score). The results are shown in Table 4.7.

Table 4.7. Hierarchical regression analysis with disability as the outcome measure

Step and variable	R^2	R^2 change	Standardised β
Step 1	0.01	0.01	
Age			0.11
Sex			0.20
Step 2	0.34	0.33	
Pain			0.28*
Step 3	0.68	0.34	
Self-efficacy (CPSS)			-0.55**
Anxiety & Depression (HADS)			0.20

The β weights are from the final regression equation.
* $p < 0.05$, ** $p < 0.01$

Therefore pain intensity explained 33% of the variance in disability beyond that explained by age and sex. A further 34% of the variance in disability could be accounted for by self-efficacy and a combined anxiety and depression score. Overall, the model explained 68% of the total variance in disability.

Muscle activity as a mediator in the relationship between psychological factors and clinical outcome

On the basis of the strong correlations found with muscle activity, mediational analysis was performed to investigate whether muscle activity acted as a partial mediator between any of the psychological factors and either pain or disability. The results (Table 4.8) showed that muscle activity was a significant mediator in the relationship between self-efficacy and pain ($z = -1.92$, $p = 0.028$).

Table 4.8. Muscle activity as a mediator in the relationship between self-efficacy and pain

Dependent variable	Independent variable	R^2	Significance
Pain (Analysis 1)	Self-efficacy	0.24	0.001
Muscle activity (Analysis 2)	Self-efficacy	0.21	0.005
Pain (Analysis 3)	Muscle activity	0.29	0.019
	Self-efficacy		0.169

Similar analysis showed that there was a trend for muscle activity to also be a partial mediator in the relationship between anxiety ($p = 0.056$), depression ($p = 0.051$), pain-related anxiety ($p = 0.061$) and catastrophising ($p = 0.061$) and pain. Muscle activity was not found to be a mediator in the relationships between any of the psychological factors and disability.

Pain as a mediator in the relationship between muscle activity and disability

Mediational analysis (Table 4.9 on page 124) demonstrated that pain was a significant mediator in the relationship between muscle activity and disability ($z = 2.13$, $p = 0.017$). Similar analysis also indicated both that disability acted as a partial mediator between

muscle activity and pain (z = 1.95, p = 0.025) and that muscle activity acted as a partial mediator between disability and pain (z = 1.77, p = 0.038).

Table 4.9. Pain as a mediator in the relationship between muscle activity and disability

Dependent variable	Independent variable	R^2	Significance
Disability (Analysis 1)	Muscle activity	0.18	0.007
Pain (Analysis 2)	Muscle activity	0.23	0.001
Disability (Analysis 3)	Pain	0.32	0.011
	Muscle activity		0.198

Defensiveness

The anxiety and defensiveness results for both the patients with CLBP and the asymptomatic controls are shown in Figure 4.10.

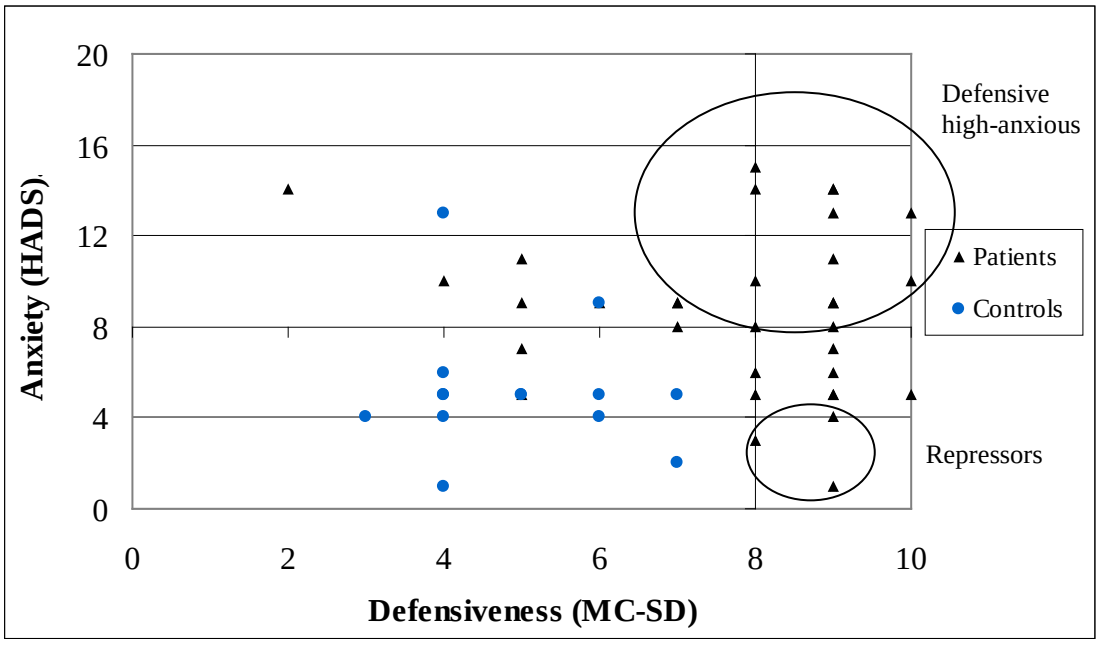


Figure 4.10. Anxiety and defensiveness for patients and controls

The CLBP patients scored significantly higher than the control group on the MC-SD, suggesting that CLBP patients are typically more defensive than asymptomatic individuals.

Of the 33 patients who had full HADS and MC-SD data, three were classified as repressors and 13 as defensive high-anxious (DHA). None of the controls were classed as high-defensive (either DHA or repressors).

As reported above, defensiveness was found to be significantly negatively related to non-normalised EMG, with a trend for a similar negative link with normalised EMG. However, further investigation of the data revealed that the results were strongly influenced by the data for just three patients, all with low reported defensiveness, but relatively high levels of both normalised and non-normalised EMG. If these three patients were excluded, none of the relationships remained significant. Care should therefore be taken in interpreting these results.

As defensiveness has been reported to vary by age and by gender, the participants were separated by gender and additionally into those above and below age 45. In neither case was the difference significant. For both young and old groups the average defensiveness score was very similar (7.7 and 7.8 for those under and over 45 respectively) and the average score for women was only slightly higher than for men (7.9 versus 7.5).

4.4. Discussion

In line with previous research, there was a trend for patients with CLBP to have higher muscle activity and delayed stature recovery compared to asymptomatic individuals, although the effect size of 0.42 for the comparison of muscle activity (0.71 for the comparison with the total, unmatched, patient group) was less than the average effect size of 1.14 during standing reported in a recent meta-analysis of 20 studies (Geisser *et al.*, 2005). In the current study, many of the patients with the greatest body mass were also those who were most disabled by their back pain. Excluding these in order to match with

the asymptomatic group also had the effect of reducing the average pain and disability scores of the patient group, which may have affected the results. The patient group also scored significantly higher on anxiety, depression and defensiveness and consisted of a greater proportion of DHA individuals.

The results confirm that patients with greater self-report of pain and disability exhibit elevated paraspinal muscle activity compared to those with lower self-report scores. There was also a trend for those with higher pain to demonstrate reduced stature recovery (and this became significant when considering the BEG only). Muscle activity was significantly correlated with self-efficacy, depression, anxiety, pain-related anxiety and catastrophising and was found to be a partial mediator in the relationship between self-efficacy and pain. This is an important finding which verifies the link between psychological and biomechanical factors in CLBP and confirms the role of muscle activity as a pathway by which psychological factors may affect clinical outcome.

Although muscle activity was significantly associated with a range of psychological factors, it was not found to be related to fear of movement (except when considering the WBTL group only), which appears contrary to current literature relating to the fear-avoidance model and muscle guarding. However, this may be because muscle activity was only measured during relaxed standing whereas hyperactivity due to muscle guarding, for example, may become more apparent in certain postures or during movements perceived as threatening/harmful.

The results of the mediational analysis suggest that muscle activity may affect disability via its influence on pain, further confirming the importance of muscle activity within LBP. However, further analysis indicated that the relationship between these three variables is

likely to be more complicated than a single pathway and that, within their impact on pain, both muscle activity and disability may separately play a mediating role.

Although there was a trend for a negative link between pain and stature recovery, in general the baseline data did not support the hypothesized relationships between stature recovery and the other factors considered. In particular, the results did not support the hypothesized negative link between muscle activity and stature recovery, which is in contrast to the findings of Healey *et al*. (2005), who did establish such a relationship in individuals with mild LBP (RDQ: 5.6 ± 2.9 compared to 11.5 ± 5.9 for the current study). The current study had the advantage of deriving data from a clinical sample with moderate levels of pain and disability and the results suggest that the relationship between muscle activity and stature recovery within this patient population may be more complex than originally thought. However, the results may also reflect the heterogeneity typical of such a clinical population and the existence of sub-groups within the patient group who exhibit different patterns of muscular activity and/or stature recovery. The participant numbers were unfortunately insufficient to test for this. It was interesting to note, however, that when considering the patients who attended the BEG only, significant negative relationships were observed between stature recovery and both pain and muscle activity. In addition, it is possible that the stature change measurements were influenced by patients attending the testing session at different times of day, whereas Healey *et al*. (2005) restricted the testing sessions to approximately one hour after rising in the morning.

The repeatability data indicated that even patients with moderate CLBP are able to achieve reliable readings in the stadiometer, and the inter-day variation was similar to that reported in a previous repeatability study (Kanlayanaphotporn *et al.*, 2002). The ICCs also confirmed that pain, disability and the psychological factors considered remained relatively

stable over time. The finding of a significant correlation between changes in stature recovery and muscle activity in the seven patients who attended twice before starting the rehabilitation programme (and who had full EMG data) supports the hypothesized relationship between the two measures. It is also consistent with the suggestion that inter-individual differences were the reason that this relationship was not observed within the full baseline data. The results from the two pre-measures additionally highlighted a significant link between changes in stature recovery and changes in both disability and anxiety. Despite limited patient numbers, these results also showed a significant correlation between changes in muscle activity and changes in self-efficacy, further supporting the relationship between these two measures.

The regression analysis revealed that, together, self-efficacy and a combined anxiety and depression measure accounted for 34% of the variance in disability, beyond that explained for by age, sex and pain. Overall, the model explained 68% of the total variance in disability, with pain and self-efficacy both uniquely contributing to the prediction of outcome. This compares to a figure of 61% of the variance in disability accounted for by the predictors in a regression analysis performed by Woby *et al.* (2007a). Although a greater number of cognitive factors were included in this earlier study, pain, self-efficacy and depression were found to be the sole factors to contribute unique variance to the prediction of disability. A possible limitation of both these studies is that the disability measure asks patients to rate the extent to which their back pain interferes with daily activities, and the self-efficacy measure asks patients to rate their confidence in performing activities in spite of pain. Although these questions are conceptually different, it is possible that patients may not have made the distinction and hence that the two measures may not be entirely independent.

Patients were significantly more defensive than controls and the high proportion of DHA patients compared to the asymptomatic group is consistent with similar findings observed in a study involving patients with chronic fatigue syndrome (CFS) (Creswell & Chalder, 2001). Although classification of patient groups is still relatively limited, in general chronic illnesses have been associated with high prevalence of a repressive, rather than DHA, coping style. This therefore makes the high number of DHA patients in the current study an interesting finding, particularly as CFS shares a number of similarities with CLBP (such as a cycle of depression, avoidance and de-conditioning) and (as proposed by a cognitive behavioural model) defence mechanisms are thought to play an important role in the development of CFS (Surawy *et al.,* 1995). To date, however, the effect of defensiveness within CLBP remains unclear. Defensiveness is generally assumed to be a relatively stable trait (Burns, 2000), and hence the high prevalence suggests that it may be a risk factor for CLBP (and possibly chronic illness in general), or associated with poor outcome, although this remains to be investigated. Of course, it should be remembered that certain coping styles may make certain individuals more likely to attend an active rehabilitation programme and then to volunteer for a research study, which is likely to have influenced the prevalence observed in the current study. In particular, repressors are thought to be reluctant to seek medical support and hence may be less likely to attend such a programme. Additionally, responses to the MC-SD may have been affected by patients meeting (and expecting to meet again) the researcher, as compared to anonymous completion by post, for example. It may have been expected that high defensiveness would be associated with elevated muscle activity. In fact, this was not found to be the case and there was a trend for a negative relationship between the two variables. Unfortunately, as there were only three patients classified as repressors in the current study, it was not possible to analyse any links between this coping style and either of the biomechanical measures.

4.5. Conclusions

Patients who demonstrated higher paraspinal muscle activity were those with more severe CLBP and the mediational analysis also indicated that muscle activity may affect disability via its influence on pain. The results therefore support the clinical relevance of this measure and suggest that treatments that reduce muscle activity may improve outcome. In addition, muscle activity was significantly correlated with a number of psychological factors and was found to act as a partial mediator between self-efficacy and pain, confirming the link between psychological and biomechanical factors in CLBP. Furthermore, it suggests that there may be particular benefit in reducing muscle activity in those with low self-efficacy.

The conclusions to the specific hypotheses addressed by this study were as follows:

(1) Hypothesis: patients with CLBP demonstrate acceptable levels of repeatability for inter-day stadiometer measurements.

Conclusion: patients with CLBP do demonstrate acceptable levels of repeatability for inter-day stadiometer measurements.

(2) Hypothesis: patients with CLBP have higher muscle activity and reduced stature recovery compared to asymptomatic individuals.

Conclusion: there was a trend for patients with CLBP to have higher muscle activity and reduced stature recovery compared to asymptomatic individuals.

(3) Hypothesis: stature recovery for patients with CLBP is negatively related to each of muscle activity, pain and disability.

Conclusion: there was a trend for patients' stature recovery to be negatively related to pain, but there was no link with muscle activity or disability. However, when considering the

BEG only, there was a negative relationship between stature recovery and both muscle activity and pain. For the patients who attended twice before starting the rehabilitation programme, there was a significant correlation between changes in stature recovery and changes in both muscle activity and disability.

(4) Hypothesis: muscle activity for patients with CLBP is related to pain and disability.
Conclusion: significant correlations supported the hypothesis that patients' muscle activity is related to both pain and disability.

(5) Hypothesis: psychological factors are associated with paraspinal muscle activity and stature recovery for patients with CLBP.
Conclusion: muscle activity was significantly related to self-efficacy, depression, anxiety, pain-related anxiety and catastrophising and was a partial mediator in the relationship between self-efficacy and pain. Psychological factors were not found to be related to stature recovery, although changes in stature recovery were linked to changes in anxiety for the patients who attended twice before starting the programme.

CHAPTER FIVE

5. Study 2: the effect of a CLBP rehabilitation programme on stature recovery, muscle activity and psychological factors

5.1. Introduction

Individuals with CLBP often exhibit hyperactivity of the superficial paraspinal muscles during static postures such as standing (e.g. Ambroz *et al.*, 2000) or full flexion (e.g. Watson *et al.*, 1997b). Linked to this elevated muscle activity is the finding that patients with mild CLBP appear to exhibit increased loading of the intervertebral discs as manifested by delayed stature recovery after loading (Healey *et al*., 2005a). Altered paraspinal muscle activity has also been linked to various psychological factors which have been shown to play an important role in CLBP, and this is further supported by the results of Study 1. This altered or elevated muscle activity has been suggested as a potential pathway by which these factors may affect the development and perpetuation of the condition.

Studies investigating the effect of treatment programmes on both abnormal superficial back muscle activity and stature recovery are limited. Hupli *et al.* (1997) found that an intense physical exercise rehabilitation programme significantly increased the morning height of CLBP patients (by 7.2mm on average) after two and a half weeks compared to those who received no treatment. The gain of height was significantly correlated with reduction of pain and decrease in disability. The MRI scans of the discs did not show any differences after treatment and therefore a reduction in muscle contraction was suggested as a possible explanation for this increase.

It has been shown that an absence of flexion-relaxation can be corrected with treatment that consists of exercise progression and disability management (Neblett *et al*., 2003;

Mayer *et al*., 2009). Mak *et al.* (2010) also reported a significant increase in the flexion-relaxation ratio (FRR) of LBP patients, towards that seen in healthy participants, after an intensive 12-week functional restoration programme of physical conditioning, working conditioning and work readiness. Watson *et al*. (1997a) reported that, following a pain management programme, CLBP patients demonstrated reduced muscle activity at full flexion and a significant increase in the FRR. This was correlated with reductions in fear avoidance beliefs and increased self-efficacy beliefs, supporting the link between psychological factors and elevated muscle activity.

Research into the effect of rehabilitation programmes on psychological factors is more common and it has been shown that such programmes can be effective in positively influencing the psychological status of CLBP patients (e.g. Woby *et al.*, 2008). Cognitive processes have also been demonstrated to strongly influence treatment outcome (Woby *et al.*, 2004). In particular, pre- to post-treatment modifications in pain-related anxiety (McCracken *et al.*, 2002), fear (avoidance) (Vlaeyen *et al.*, 2002; Woby *et al*., 2008), psychological distress (Grotle *et al.*, 2006; Mannion *et al.*, 2001a), catastrophising and self-efficacy (Foster *et al.*, 2010; Woby *et al.*, 2008) have all been identified as significant predictors of clinical outcome, although it is not known specifically how these cognitive processes lead to improvements in pain and/or disability.

The aim of this study was to investigate the effect of an active physiotherapist-led rehabilitation programme for patients with CLBP. Stature recovery, static paraspinal muscle activity and a range of psychological factors were measured both before and after the programme and at a six-month follow-up session in order to investigate any relationships between the variables and to identify any factors associated with clinical outcome. The results of Study 1 highlighted strong links between paraspinal muscle

activity and pain, disability, and a number of psychological factors such as self-efficacy and depression. It was therefore hypothesized that muscle activity would be reduced following the programme and that changes in muscle activity would be associated with improvements in pain and disability and cognitive processes. The failure to find relationships between stature recovery and the other outcome measures in Study 1 was attributed largely to inter-individual differences and hence it was hypothesized that stronger links may be observed between changes in the variables in this longitudinal study. In line with previous research (McCracken & Turk, 2002), it was expected that patients who had high baseline levels of factors such as anxiety, depression and catastrophising would tend to derive less benefit from treatment and hence that these factors would be associated with poor clinical outcome. Furthermore, since these factors were shown to be linked to levels of muscle activity in Study 1, it was hypothesized that high baseline levels of muscle activity would also be related to poor clinical outcome.

The specific hypotheses addressed by this study were as follows:

(1) following the rehabilitation programme, patients with CLBP demonstrate improvements in pain, disability and psychological factors;

(2) paraspinal muscle activity of CLBP patients is reduced following the rehabilitation programme, and this improvement is linked to improvements in pain, disability and psychological factors;

(3) stature recovery of CLBP patients is increased following the rehabilitation programme, and this improvement is linked to reductions in muscle activity and improvements in pain, disability and psychological factors;

(4) patients with low baseline levels of both muscle activity and psychological factors, such as anxiety, depression and catastrophising, demonstrate better clinical outcome than those with high levels.

5.2. Methods

Participants

Patients were recruited from the waiting list for both the Back Exercise Group (BEG) and the Work Back to Life Group (WBTL), both of which are run in North Manchester. Data were collected from 23 patients with CLBP (age, 47.7 ± 11.7y; height, 165.4 ± 7.5cm; body mass, 75.1 ± 23.0kg). The group was mixed (8 men, 15 women), between the ages of 23 and 70 years. Fourteen (six men, eight women) of these patients (age, 51.6 ± 8.3y; height, 165.2 ± 8.7cm; body mass, 71.9 ± 15.5kg) went on to complete the six-month follow-up session. The follow-up group did not differ significantly in terms of age, height or body mass, from either the 23 patients who participated after the programme or the total patient population included in Study 1. Further details of the patients are provided in the General Methods section.

Intervention

Patients were referred to the rehabilitation programmes via their physiotherapist. The BEG involved four sessions (one a week). The first and last sessions were two hours in duration and consisted of exercise and education. The middle two sessions were one hour of exercise only. The exercise facet of the programme consisted of specific stretching and strengthening exercises and became progressively more difficult over the four weeks. Patients were also encouraged to exercise daily at home. The educational component included: medical terms used to describe back pain, the physical and psychological benefits of participating in physical activity and the role of medication in the management of back pain. The WBTL group included five sessions (one a week), each of three and a quarter hours in duration. This programme included the exercise and education components that were in the BEG, but was based more on cognitive-behavioural principles. In particular, the WGTL group included individual goal setting aimed at

returning patients to activities and tasks that they had stopped doing because of their back pain. These were followed up each week. Patients were allocated to either the BEG or the WBTL based on the results of TSK and RDQ questionnaires. The WBTL group was intended for those patients who were more severely disabled and had a score on the TSK-11 (a shortened version of the TSK) of at least 26. Further details on the WBTL programme are given in the study by Woby *et al.* (2008).

Procedure

Patients attended the testing session before starting the rehabilitation programme (except for five patients who attended shortly after the programme commenced). Patients then returned for another testing session shortly after completing the rehabilitation group and then again six months later, where possible. (For one of the patients, the follow-up session was carried out nine months later as she was involved in a minor traffic accident just before she was due to come back for her six-month follow-up session.) Patients came at the same time for each visit (or within one hour of the previous time), except in one case when due to work commitments, the patient attended in the morning for the first visit and the afternoon for the two subsequent visits. Paraspinal muscle activity was assessed via surface electromyography (EMG), whilst both standing at rest and during a RVC. Stature recovery over a 40-minute unloading period was measured on a standing stadiometer. Patients also completed a series of self-report measures following their visit. Details of both the protocol and the measures used are provided in the "General Methods" section.

The procedure, and numbers of patients at each stage, are shown in Figures 5.1 and 5.2. The numbers for the BEG and the WBTL groups are shown separately , although the limited participant numbers for the WBTL group meant that separate analysis was not performed for the two groups.

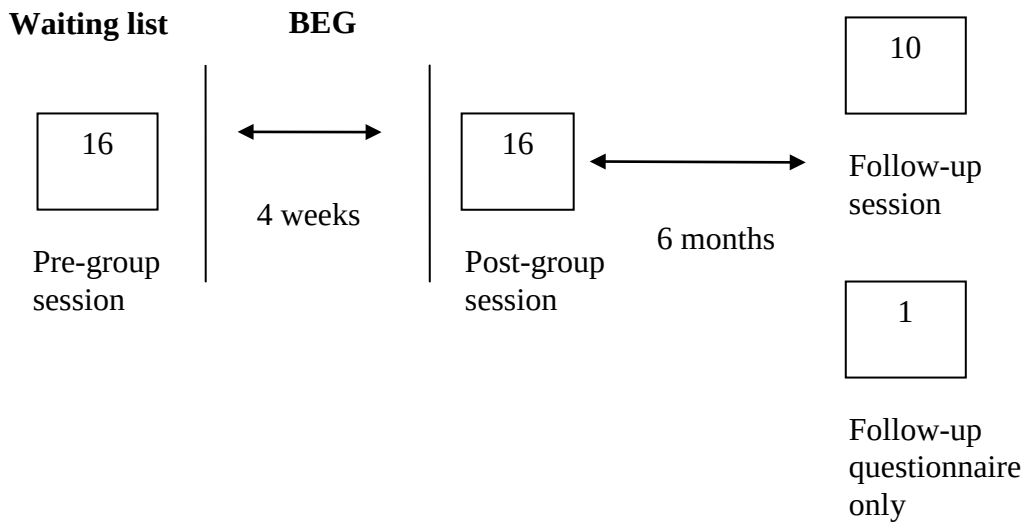


Figure 5.1. Procedure and patient numbers for the BEG

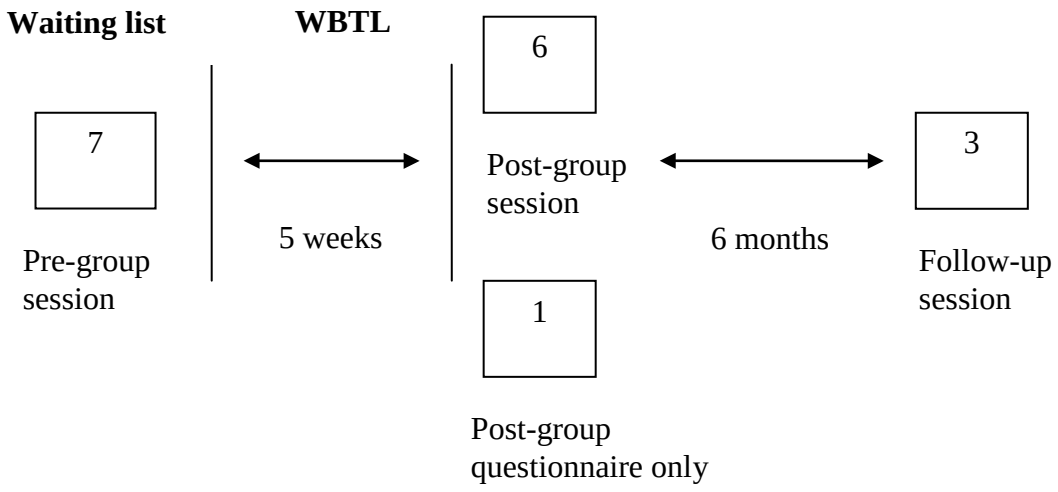


Figure 5.2. Procedure and patient numbers for the WBTL group

Analysis

A number of key analyses were carried out. Firstly, a series of t-tests were performed to explore whether the baseline characteristics of patients who dropped out of the intervention differed from those who completed it. Secondly, one-tailed t-tests were calculated to determine whether pre- to post- treatment improvements occurred in the outcome measures (i.e. stature recovery, muscle activity, pain, disability, anxiety, depression, functional self-efficacy, pain-related anxiety, catastrophising and fear of movement). For those patients who completed the six-month follow-up, Kolmogorov-Smirnov tests indicated that the baseline data for disability and anxiety were not normally distributed and hence Wilcoxon signed rank tests were instead used for these variables. Parametric analyses were used in respect of the change scores for each of the outcome measures, based on the results of Kolmogorov-Smirnov and Shapiro-Wilk tests of normality. Pearson's correlation coefficient was therefore implemented to show the inter-relations that existed between the changes that occurred in these measures (both over the duration of the programme and up to the end of the six-month follow-up period). Finally, two-tailed correlation coefficients were employed to investigate the extent to which any of the measures at baseline were linked to overall levels of improvement in pain, disability, muscle activity and stature recovery.

5.3. Results

Experimental mortality

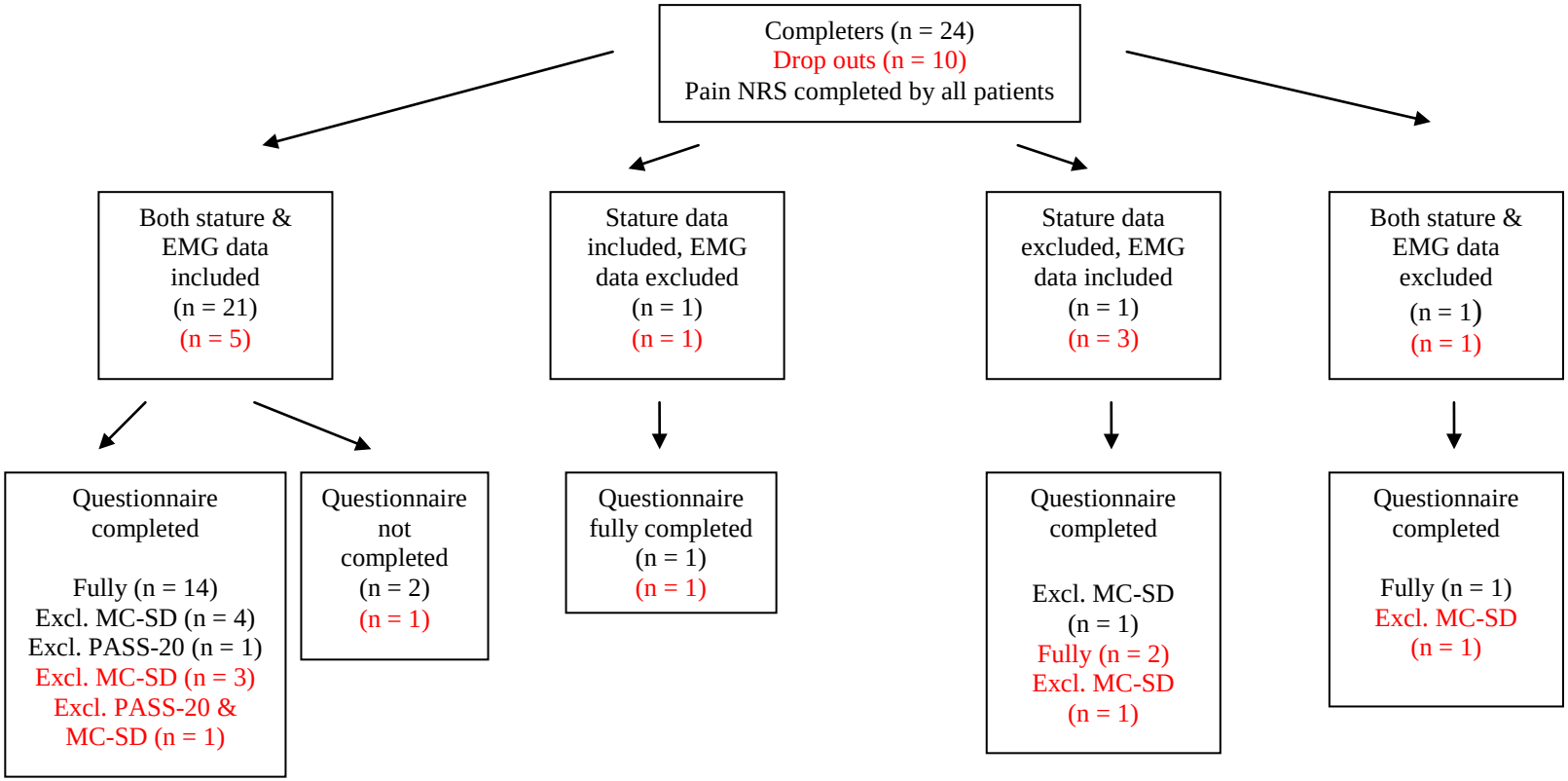
Of the 34 patients who participated in Study 1 before starting the rehabilitation programme (or shortly after the programme commenced), 10 dropped out (which in this case is taken to mean that they attended a maximum of two sessions of the programme). A number of differences on the self-report measures were observed between those patients who dropped out and those who completed the programme as shown in Table 5.1 on page 142. It should

be noted that although stature recovery was greater for ‘drop-outs’ than ‘completers’, the data for the drop-out group is limited because four of this group were considered unreliable on the stadiometer and therefore their stature recovery results have been excluded. The numbers of patients whose data was included in the analysis is shown in Figure 5.3 on page 141. In particular, patients who dropped out had lower baseline levels of self-efficacy than those who completed the programme. Pain-related anxiety, depression and catastrophising were significantly higher for drop-outs than completers.

Pre-post analysis

As can be seen in Table 5.2 on page 144, the sample represented patients with moderate levels of pain and disability. The mean duration of pain was 6.2 years and the median was 2 years (range: 3 months – 25 years). Twenty two patients attended sessions both before and after the programme and one further patient attended before the programme and then completed a questionnaire afterwards (and hence did not have full stature or EMG data). Twenty patients completed the questionnaire both before and after the programme. Some patients found it difficult to maintain a consistent posture in the stadiometer and three patients were excluded from the stature recovery data as the standard deviation (SD) of the five familiarisation readings was considered to be too high. As for Study 1, a SD of 1.7mm was taken as the cut-off point. The remaining patients had an average SD of 1.1mm over the five familiarisation readings. Technical problems with the EMG system at the start of the study meant that two patients did not have EMG data both before and after the programme. As for Study 1, the EMG data of one patient was excluded from the analysis as they were considered to be an outlier (muscle activity was < 30% of RVC). The numbers of patients included in the analyses are shown in Figure 5.3 on page 141.

Figure 5.3. Numbers of patients included in the comparison of baseline measures for those who completed the programme (shown in black) and those who dropped out (shown in red)



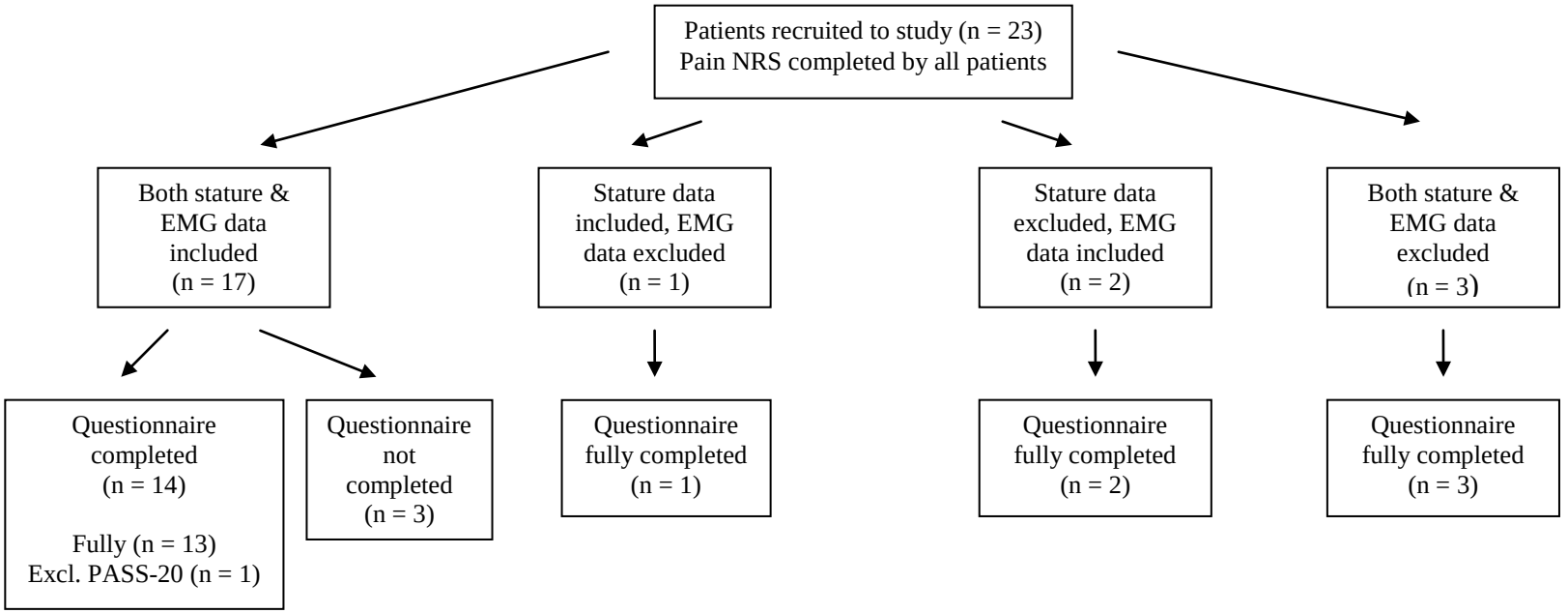
Fully: All sections of the questionnaire were completed; Excl. PASS-20: the questionnaires were all completed with the exception of the Pain Anxiety Symptoms Scale-20; Excl. MC-SD: the questionnaires were all completed with the exception of the Marlowe-Crowne Social Desirability Scale; Excl. PASS-20 & MC-SD: the questionnaires were all completed with the exception of the Pain Anxiety Symptoms Scale-20 and the Marlowe-Crowne Social Desirability Scale

Table 5.1. Baseline measures for those who completed the programme and those who dropped out

Variable	Completers Mean (± SD)	Drop-outs Mean (± SD)	Effect size
Stature change (mm)	2.4 (± 2.0)	4.4 (± 2.7)*	0.74
Muscle activity (% of RVC)	73.8 (± 13.1)	79.0 (± 10.4)	0.50
Disability	11.5 (± 5.9)	12.1 (± 5.9)	0.10
Pain NRS	4.8 (± 2.2)	5.0 (± 2.6)	0.07
Anxiety	8.3 (± 3.5)	10.1 (± 4.3)	0.43
Depression	5.9 (± 3.5)	8.8 (± 3.5)*	0.81
Functional self-efficacy	49.3 (± 17.0)	31.8 (± 18.0)**	-0.98
Pain-related anxiety	34.9 (± 21.1)	58.0 (± 27.9)*	0.83
Catastrophising	18.5 (± 13.3)	28.7 (± 13.2)*	0.76
Fear of movement	36.8 (± 7.8)	40.6 (± 6.6)	0.58
Defensiveness	8.2 (± 1.5)	7.3 (± 3.5)	-0.26

* $p < 0.05$, ** $p < 0.01$

Figure 5.4. Numbers of patients included in the pre-post analyses



Fully: All sections of the questionnaire were completed (MC-SD was not included in this analysis); Excl. PASS-20: the questionnaires were all completed with the exception of the Pain Anxiety Symptoms Scale-20

Outcome measures pre- and post-treatment

For those patients who completed the programme and returned for a second testing session, a summary of the outcome measures before and immediately following treatment are given in Table 5.2.

Disability, pain, fear of movement, catastrophising and functional self-efficacy were all significantly improved immediately following the rehabilitation programmes. It is noteworthy that stature recovery was, on average, unchanged, and that the majority of patients actually demonstrated no change or a slight increase in muscle activity following the programme (although the mean change was not significant).

Table 5.2. Outcome measures pre- and post-treatment

Variable	Pre Mean (± SD)	Post Mean (± SD)	Effect size
Stature change (mm)	2.5 (± 2.0)	2.7 (± 2.5)	0.10
Muscle activity (% of RVC)	73.9 (± 13.2)	75.5 (± 11.1)	0.12
Disability	11.4 (± 5.9)	9.2 (± 7.1)**	-0.37
Pain NRS	4.7 (± 2.2)	3.4 (± 2.5)**	-0.58
Anxiety	8.4 (± 3.5)	7.6 (± 3.3)	-0.24
Depression	5.9 (± 3.5)	5.7 (± 4.0)	-0.06
Functional self-efficacy	50.7 (± 16.5)	57.0 (± 13.5)*	0.38
Pain-related anxiety	32.3 (± 18.9)	28.1 (± 20.4)	-0.22
Catastrophising	17.4 (± 12.0)	13.2 (± 10.1)*	-0.35
Fear of movement	36.3 (± 7.3)	32.8 (± 8.2)**	-0.47

One outlier excluded from the stature change data as explained on page 145
* $p < 0.05$, ** $p < 0.01$

Correlational analysis: changes in stature recovery

There was a significant relationship between changes in stature recovery and pain such that those patients with reduced pain after the programme also tended to have increased stature recovery ($r = -0.48$, $p = 0.02$).

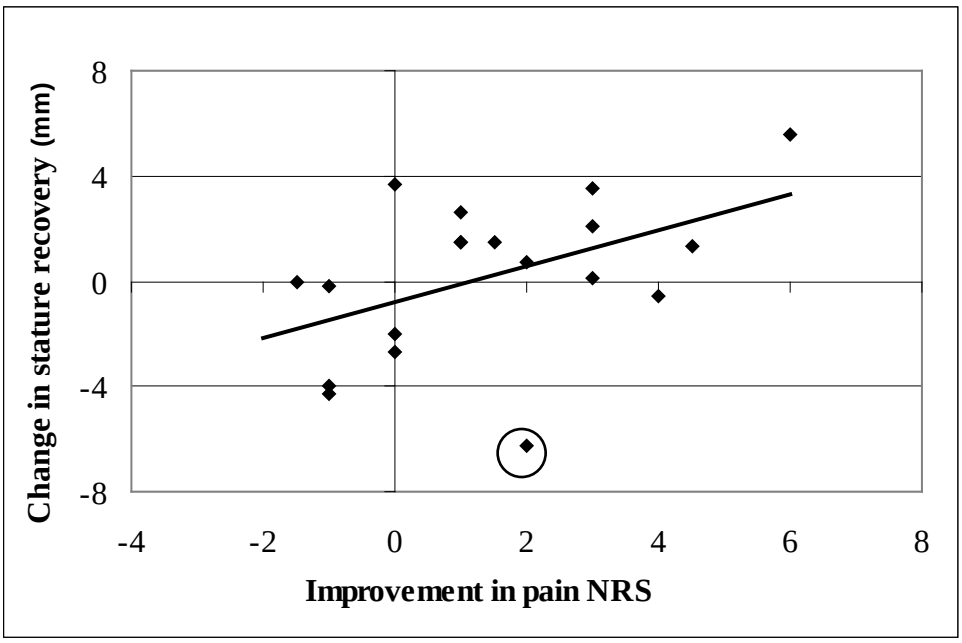


Figure 5.5. Relationship between changes in stature recovery and changes in pain. (A positive improvement in pain on the graph represents a reduction in the pain score following the programme.) Outlier circled

There was an outlier in the stature recovery data for this study (circled in Figures 5.5 and 5.6). This was a patient who unexpectedly had a materially negative stature recovery (< -3mm) on his return visit (and thus distorted the results). As the SD of his familiarisation readings was just below the cut-off point, it is likely that this patient found it difficult to maintain a consistent posture on the stadiometer. This patient has therefore been excluded from the correlation table on page 147. With this outlier excluded, the link with pain becomes highly significant ($r = -0.60$, $p < 0.01$) and the relationships between changes in stature recovery and changes in both disability ($r = -0.46$, $p = 0.04$) and catastrophising ($r = -0.59$, $p = 0.01$) also become significant. In addition, there was a trend for reductions in both anxiety ($r = -0.35$, $p = 0.10$) and depression ($r = -0.41$, $p = 0.07$) to be associated with increased stature recovery.

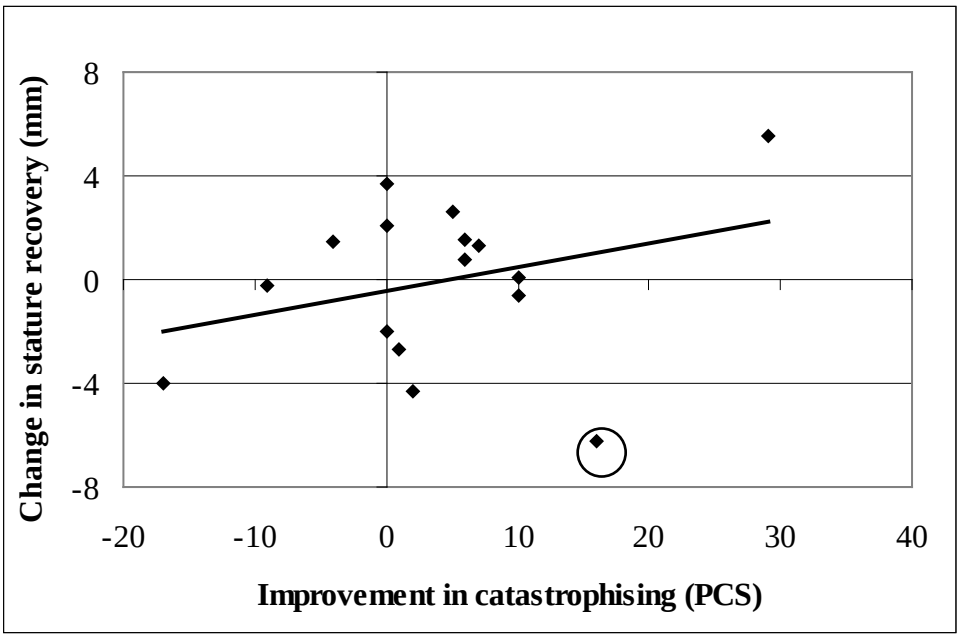


Figure 5.6. Relationship between changes in stature recovery and changes in catastrophising. (A positive improvement in catastrophising on the graph reflects a reduction in the score following the programme.) Outlier circled

Changes in stature recovery were significantly linked to all three subscales (Rumination, Magnification and Helplessness) of the catastrophising questionnaire and there was a highly significant relationship with the Magnification subscale ($r = -0.73$, $p < 0.01$).

Correlational analysis: changes in muscle activity

The patients with increased EMG levels after the programme tended to be those with higher baseline self-efficacy ($r = 0.53$, $p = 0.03$). Additionally, there was a trend for a negative link between changes in both muscle activity and self-efficacy ($r = -0.38$, $p = 0.08$). This indicates that those patients who did have reduced EMG levels following the programme were generally those who showed a greater increase in self-efficacy. This perhaps reflects the lower initial levels of self-efficacy for these patients.

The correlations between changes in the factors from pre- to post-therapy are shown in Table 5.3 on page 147.

Table 5.3. Correlation coefficients between changes in outcome measures

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Stature change	-									
2. Muscle activity	-0.07	-								
3. Disability	-0.46*	0.17	-							
4. Pain	-0.60**	0.07	0.47*	-						
5. Anxiety	-0.35	-0.18	0.51*	0.44*	-					
6. Depression	-0.41	-0.02	0.56**	0.66**	0.78**	-				
7. Self-efficacy	0.32	-0.38	-0.60**	-0.50*	-0.31	-0.54**	-			
8. Pain-related anxiety	-0.24	0.24	0.73**	0.47*	0.29	0.46*	-0.63**	-		
9. Catastrophising	-0.59*	-0.24	0.56**	0.45*	0.43*	0.64**	-0.52**	0.55**	-	
10. Fear of movement	-0.18	0.21	0.48*	0.38*	0.20	0.40*	-0.60**	0.67**	0.64**	-

One outlier excluded from the stature change data as explained on page 145

* $p < 0.05$, ** $p < 0.01$

Reductions in pain-related anxiety and depression had the strongest relationships with improvements in disability and pain respectively, although reductions in both pain and disability were significantly correlated with improvements in each of the psychological factors considered.

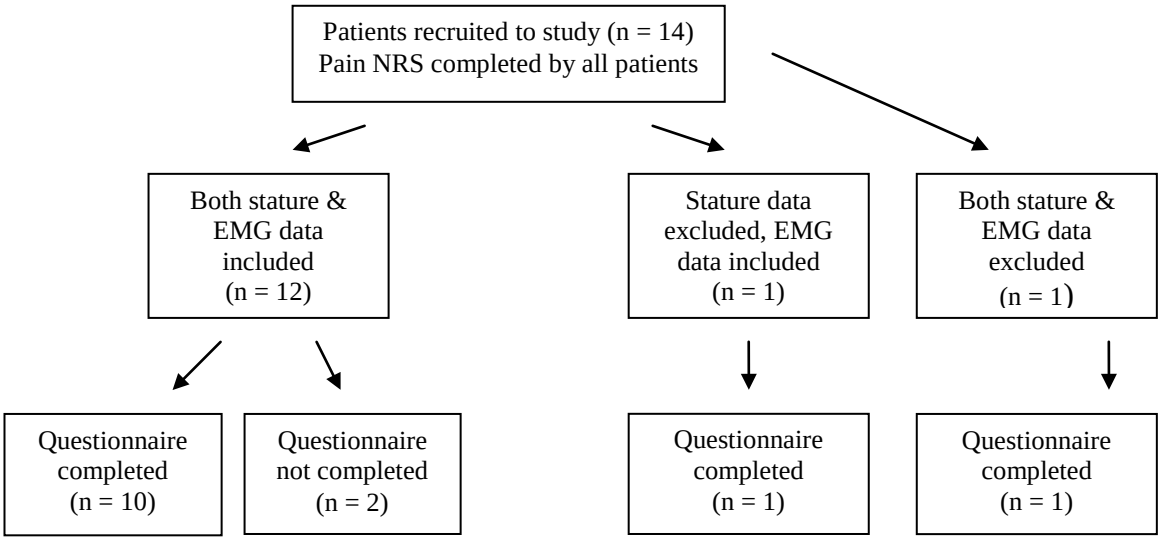
Relationships between baseline factors and clinical outcome

There was a correlation between reductions in pain immediately following the programme and high initial levels of pain ($r = -0.55$, $p < 0.01$). There was also a trend for a decrease in pain to be associated with high baseline levels of pain-related anxiety ($r = -0.43$, $p = 0.07$). No baseline factors were found to be related to a reduction in disability.

Follow-up data

Thirteen patients attended all three sessions, including the six-month follow-up, and eleven of these completed a questionnaire on each visit. One patient was considered to be unreliable on the stadiometer and hence his stature recovery data were omitted from the analysis. One further patient attended pre- and post-treatment sessions and completed a questionnaire after a following six months. The numbers of patients included in the analyses are shown in Figure 5.7 on page 149.

Figure 5.7. Numbers of patients included in the analyses of the follow-up data



There was a trend for the patients who completed the follow-up sessions to have lower initial self-reported pain-related anxiety compared to the total patient population who attended for Study 1 (30.4 ± 12.8 vs 42.4 ± 23.9, p = 0.05). The results for the patients who completed the follow-up are given in Table 5.4 on page 149.

By the end of the follow-up period, there was a significant reduction in disability, anxiety, depression and fear of movement. There were trends for reductions in pain (p = 0.06) and catastrophising (p = 0.08). Stature recovery and self-efficacy were significantly higher.

Table 5.4. Outcome measures pre- and post- treatment for patients who completed the follow-up

Variable	Pre Mean (± SD) A	Post Mean (± SD) B	Effect size	Follow-up Mean (± SD) C	Overall effect size	Post – follow-up effect size	p
Stature change (mm)	2.6 (± 1.6)	3.3 (± 1.7)	0.42	4.5 (± 2.3)	1.18	0.72	A < C**
Muscle activity (% of RVC)	72.6 (± 14.1)	74.0 (± 9.7)	0.10	69.8 (± 11.4)	-0.20	-0.43	
Disability	12.2 (± 5.1)	9.9 (± 7.6)	-0.44	8.6 (± 7.8)	-0.70	-0.18	A > C*
Pain NRS	4.9 (± 2.0)	3.5 (± 2.3)	-0.71	3.5 (± 2.4)	-0.71	0.00	A > B**
Anxiety	8.9 (± 2.7)	7.6 (± 3.9)	-0.49	6.4 (± 4.3)	-0.92	-0.30	A > C**, B > C*
Depression	6.2 (± 3.4)	6.1 (± 4.6)	-0.02	4.8 (± 4.3)	-0.42	-0.29	A > C*, B > C**
Functional self-efficacy	52.5 (± 14.0)	56.0 (± 13.9)	0.25	58.6 (± 14.2)	0.43	0.19	A < C*
Pain-related anxiety	30.4 (± 12.8)	28.5 (± 19.0)	-0.15	26.3 (± 19.3)	-0.32	-0.12	
Catastrophising	15.7 (± 9.6)	13.8 (± 12.3)	-0.36	11.6 (± 10.7)	-0.43	-0.18	
Fear of movement	37.7 (± 6.4)	35.4 (± 8.6)	-0.36	32.1 (± 13.5)	-0.88	-0.38	A > C*

* $p < 0.05$, ** $p < 0.01$

Relationships between overall changes in outcome measures

The relationships between changes in the factors from baseline to follow-up are shown in Table 5.5 on page 152. Reductions in disability were significantly correlated with improvements in all the psychological factors considered and improvements in pain were associated with reductions in catastrophising. There was also a trend for overall decreases in EMG levels to be associated with improvements in depression ($p = 0.06$) and self-efficacy ($p = 0.07$).

Relationships during the follow-up period

When considering the period from immediately after the programme to follow-up, decreases in muscle activity were associated with changes in anxiety ($r = 0.78$, $p = 0.04$). No correlation was found between those patients who had a short term increase in muscle activity levels immediately following the programme and clinical outcome.

Relationships between baseline factors and clinical outcome at follow-up

A reduction in pain by the end of the six-month follow-up period was significantly linked to high baseline levels of pain ($r = -0.60$), catastrophising ($r = -0.66$) and defensiveness ($r = -0.79$). An overall reduction in EMG was correlated with high initial levels of muscle activity ($r = -0.60$). There was also a trend for overall increases in stature recovery to be linked to low initial self-efficacy ($r = -0.56$).

Table 5.5. Correlation coefficients between overall changes in outcome measures for patients who completed the follow-up

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Stature change	-									
2. Muscle activity	0.24	-								
3. Disability	0.29	0.29	-							
4. Pain	-0.08	0.05	0.46	-						
5. Anxiety	0.06	-0.06	0.52*	0.17	-					
6. Depression	0.47	0.51	0.63*	0.12	0.61*	-				
7. Self-efficacy	0.08	-0.49	-0.75**	-0.46	-0.43	-0.55*	-			
8. Pain-related anxiety	0.38	0.23	0.58*	0.11	0.30	0.58*	-0.26	-		
9. Catastrophising	-0.18	0.22	0.75**	0.63*	0.37	0.30	-0.63*	0.52	-	
10. Fear of movement	-0.14	0.17	0.74**	0.37	0.76**	0.58*	-0.75**	0.44	0.59*	-

* $p < 0.05$, ** $p < 0.01$

5.4. Discussion

Improvements in both pain and disability were observed in patients who completed the rehabilitation programmes. Average pain remained the same at the six-month follow-up, but disability continued to decrease. Consistent with previous research, improvements in clinical outcome were significantly related to cognitive processes (e.g. Woby *et al*., 2008), and depression and pain-related had the strongest relationships with improvements in pain and disability respectively immediately following the programme. Reduced disability at the end of the six-month follow-up period was significantly related to improvements in all of the psychological factors considered.

Contrary to expectations, immediately following the rehabilitation programme, most patients demonstrated either no change or an increase in resting EMG. However, at the six-month follow-up, EMG levels had reduced to below the initial value (although this change was not significant). This pattern existed in both absolute and normalised EMG levels and therefore the initial increase was not simply due to a reduction in RVC values. It perhaps indicates an adaptation period following a programme of increased activity and exercise, as the muscles compensate for increased demands in the context of pre-existing instability and it is thus, only after an extended period of time that resting EMG levels achieve the expected decrease. Interestingly, the increased EMG levels were associated with higher initial self-efficacy, perhaps because this reflects greater participation in the daily exercise and stretching recommended in the programmes (although unfortunately physical activity was not measured in this study). This is not the first study to find that EMG levels do not make an immediate return to more 'healthy' patterns of activity. Mannion *et al*. (2001b) reported that EMG levels for CLBP patients during full flexion were not reduced following active therapy and initial EMG levels during isometric exercises were also increased. Additionally an increased rate of decline in EMG median

frequency, suggesting increased muscle fatigability, was observed during the post-therapy dynamic fatigue test. The authors suggested that patients may be employing different motor control/recruitment patterns, perhaps as a result of less guarding mechanisms after therapy (Mannion *et al*., 2001b). This may also be a possible explanation in the current study, such that patients are using painful lumbar muscles to maintain upright posture to a greater extent than previously, resulting in higher EMG levels. Alternatively, patients may be adopting an altered posture, such as a more neutral spine, following the programme, leading to changes in muscle activation patterns. This short term effect on muscle activity suggests that increases in EMG may not always be detrimental, but may reflect a positive adjustment and thus techniques such as EMG biofeedback that aim to decrease EMG levels may not be appropriate in such situations.

Perhaps due to this short-term effect of the programme on EMG levels, changes in muscle activity were not found to be significantly related to overall changes in any of the other outcome measures, although there was a trend for links with improvements in depression and self-efficacy by the end of the six-month period. During the follow-up period only, changes in muscle activity were correlated with changes in anxiety.

Excluding one outlier, there was a significant negative correlation between changes in pain and stature recovery immediately following the programme, confirming the relationship between pain and stature recovery identified by Healey *et al.* (2005a). Although there was a trend for this association to be present in the baseline factors analysed in Study 1, the relationship between changes in the factors in this longitudinal study is stronger, probably due to the removal of the effect of inter-individual differences. Despite the correlation with changes in pain, stature recovery was only significantly higher at the follow-up session and not immediately following the programme. Immediate increases in stature recovery were

also associated with improvements in disability and catastrophising (with a trend for links with improvements in anxiety and depression), although changes in muscle activity and stature recovery were not found to be related. Furthermore, when considering the overall changes up to the six-month follow-up, changes in stature recovery were not linked to changes in any of the other outcome measures, possibly as a result of the limited numbers for this section of the study.

Although patients were encouraged to continue exercising on completion of the programme, this was not monitored, either by the physiotherapists or as part of the current study. It is therefore unknown whether the changes observed at follow-up were the result of increased physical activity levels. Anecdotally, many patients reported that they had continued exercising, but at a reduced level since completing the programme.

The effect sizes observed immediately following the programmes can be compared to the results of a study by Woby *et al.* (2008), who also recruited patients from the WBTL programme for their investigation into the role of cognitive processes in clinical outcome (although the programme was slightly different at this time. Most notably the final session was held two weeks after the fourth session rather than the following week as currently). One hundred and thirty seven patients were included, and unsurprisingly, the baseline characteristics reveal patients with higher initial levels of disability, fear of movement and depression, and lower self-efficacy than the current study, which involved mainly less disabled BEG patients. Despite this, mean pain levels actually appear higher in the current study (although a visual analogue scale (VAS) was used for the Woby *et al.* study which prevents a direct comparison). Woby *et al.* reported effect sizes for pre- to post-treatment changes of 0.56 for disability and 0.04 for pain, which compares to 0.37 and 0.58 for disability and pain respectively in the current study. Woby *et al.* also reported much greater

effect sizes for the psychological factors (depression, fear of movement, functional self-efficacy and catastrophising) than the current study. Along with the greater effect size for disability, this is probably partly a result of the higher baseline levels and also because the WBTL group aims to tackle psychosocial issues to a greater extent than the BEG.

Patients who dropped out of the rehabilitation programme tended to report higher depression, pain-related anxiety and catastrophising and lower self-efficacy than patients who completed the programme. For those who did complete both the programme and the six-month follow-up, a reduction in pain by the end of this period was significantly linked to high baseline levels of pain, catastrophising and defensiveness. This appears contrary to previous research which has tended to show that patients who demonstrate high emotional distress, catastrophising and other forms of negative thinking in response to pain tend to derive less benefit from treatments (McCracken & Turk, 2002). However, it should be remembered that the number of patients who completed the follow-up session in this study was limited. Furthermore, patients attending the follow-up had volunteered for a minimum of three testing sessions in addition to completing the rehabilitation programme and there is therefore likely to be an element of self-selection. In particular, those patients with the highest baseline levels of depression, pain-related anxiety and catastrophising and lowest self-efficacy tended not to complete the rehabilitation programme or the follow-up session and therefore were excluded from this analysis as their clinical outcome was unknown. This led to a trend for a lower initial level of pain-related anxiety in the patients completing the follow-up compared to the total patient group included in Study 1. Therefore caution should be exercised in generalising these results.

5.5. Conclusions

Improvements in both pain and disability were observed in patients who completed the rehabilitation programmes. The important role of cognitive processes was highlighted by the significant correlations between reductions in disability and improvements in all of the psychological factors considered, both immediately following the programme and at the six-month follow-up. Significant relationships between changes in stature recovery and in pain and disability immediately following the programme supported the hypothesized link between these factors, although a significant increase in stature recovery was only observed by the end of the six-month follow-up period.

For many of the patients, muscle activity did not decrease immediately following the programme, despite improvements in pain and disability, perhaps reflecting a period of adaptation or altered patterns of motor control. However, by the end of the six-month follow-up, there was a (non-significant) reduction in muscle activity levels, suggesting that more 'normal' patterns of activity may accompany improved clinical outcome over the longer term.

The conclusions to the specific hypotheses addressed by this study were as follows:

(1) Hypothesis: following the rehabilitation programme, patients with CLBP demonstrate improvements in pain, disability and psychological factors.
Conclusion: by the end of the six-month follow-up period, CLBP patients demonstrated improvements in disability, self-efficacy, anxiety, depression and fear of movement, with trends for improvements in pain and catastrophising.

(2) Hypothesis: paraspinal muscle activity of CLBP patients is reduced following the rehabilitation programme, and this improvement is linked to improvements in pain, disability and psychological factors.
Conclusion: for many of the patients, muscle activity was unexpectedly increased immediately following the programme. Levels were reduced by the end of the six-month follow-up period, but this was not significant. There was a trend for overall decreases in EMG levels to be associated with improvements in depression and self-efficacy, and changes in the follow-up period only were related to changes in anxiety.

(3) Hypothesis: stature recovery of CLBP patients is increased following the rehabilitation programme, and this improvement is linked to reductions in muscle activity and improvements in pain, disability and psychological factors.
Conclusion: stature recovery was significantly increased by the end of the six-month follow-up period, but not immediately following the rehabilitation programme. Initial changes in stature recovery were linked to changes in pain, disability and catastrophising. There was also a trend for reductions in both anxiety and depression to be associated with increased stature recovery.

(4) Hypothesis: patients with low baseline levels of both muscle activity and psychological factors, such as anxiety, depression and catastrophising, demonstrate better clinical outcome than those with high levels.
Conclusion: contrary to expectations, for those patients who completed the programme, and returned for a six-month follow-up, a reduction in pain by the end of this period was significantly linked to high baseline levels of pain, catastrophising and defensiveness. Participant numbers were limited, however, and self-selection may have influenced the results. Initial muscle activity levels were not related to clinical outcome.

CHAPTER SIX

6. Study 3: The effect of superficial heat treatment on paraspinal muscle activity and stature recovery

6.1. Introduction

Superficial heat therapy is commonly used to treat CLBP, both by practitioners and by patients at home. It is generally accepted that heat increases the extensibility of collagen tissue, decreases joint stiffness, relieves muscle spasm, produces pain relief, and increases both blood flow and metabolism (Lehmann & Lateur, 1990). Additionally, the interdependence of pain and muscle spasm implies that a reduction in one may cause a reduction in the other (Low & Reed, 2000). A Cochrane review (French *et al*., 2006) concluded that there is moderate evidence that heat therapy reduces short-term pain and disability for acute back pain, but that there is insufficient evidence regarding the effect of heat for CLBP.

Most of the existing studies on superficial heat therapy for LBP have been carried out by two research teams, both assessing the effect of the same disposable lumbar heat wrap (Thermacare heat wrap) (Mayer *et al.*, 2005; Mayer *et al.*, 2006; Nadler *et al.,* 2002; Nadler *et al.,* 2003a; Nadler *et al.,* 2003b). The results of the five studies were encouraging. Four of the studies compared the heat wrap with various different interventions for patients with acute LBP (Mayer *et al.*, 2005; Nadler *et al.,* 2002; Nadler *et al.,* 2003a; Nadler *et al.,* 2003b). For the three Nadler studies, treatments were administered for either two or three days or nights, followed by two days of follow-up. By the end of the follow-up period, patients using the heat wrap had greater pain relief, reduced disability, less muscle stiffness and increased flexibility compared to both those taking an oral placebo (Nadler *et al.,* 2003a) and either ibuprofen or acetaminophen (Nadler *et al.,* 2002). Overnight use of the wrap also provided effective pain relief

throughout the next day, reduced muscle stiffness and disability and improved trunk flexibility compared to an oral placebo (Nadler *et al.*, 2003b). Mayer *et al*. (2005) reported that patients receiving combined heat and exercise treatment had significantly improved function and reduced disability compared to either exercise or heat treatment alone or a control group, and pain relief was significantly greater than for exercise alone or the control group. A second study by Mayer *et al.* (2006) showed heat therapy to be beneficial in the prevention and early phase treatment of low back delayed-onset muscle soreness (DOMS).

In a study involving 100 patients with acute back pain during emergency transfer to hospital, patients were randomly assigned to two groups: active warming with an electric heated blanket or passive warming with a woollen blanket (Nuhr *et al*., 2004). Those patients who received active heating had significantly lower pain and anxiety scores and a lower heart rate on arrival at hospital compared to those who received passive warming.

The effect of heat treatment on muscle activity or spasm is of particular importance for LBP since patients often demonstrate hyperactivity of the superficial paraspinal muscles (e.g. Ambroz *et al*., 2000). This elevated paraspinal muscle activity has also been linked to delayed stature recovery in people with mild CLBP (Healey *et al*., 2005a). Similarly, the impact of heat treatment on psychological factors is highly relevant as such factors are known to play an important role in the condition and have been linked to the perpetuation of pain and disability (Woby *et al*., 2007a), with altered paraspinal muscle activity suggested as a possible pathway by which this may occur.

This study aimed to investigate the effect of superficial heat therapy on both paraspinal muscle activity and stature recovery in patients with CLBP, and any relationship between

changes in these two factors. In addition, the study aimed to establish whether the heat wrap had any effect on the self-report of a range of psychological factors and, if so, whether these changes were linked to changes in muscle activity and/or stature recovery. As it is generally accepted that heat therapy reduces muscle spasm, it was hypothesized that the heat wraps would have the effect of decreasing muscle activity.

Studies 1 and 2 were unable to show the expected relationship between muscle activity and stature recovery. For Study 1, this may have been due to pathological differences between patients which impacted on stature change results and, in Study 2, relationships with muscle activity may have been obscured by a short-term adaptation period immediately following the rehabilitation programme. Without these issues, it was hypothesized that the previously established link between muscle activity and stature recovery (in patients with mild CLBP) would be observed in the CLBP patients included in Study 3. Hence, if the heat wrap reduced muscle activity as anticipated, it was expected that this would be accompanied by increased stature recovery and that there would be a negative link between changes in stature recovery and muscle activity.

Heat therapy has been shown to increase feelings of well-being and reduce anxiety (Nuhr *et al*., 2004). It was therefore expected that wearing the heat wrap would have a short-term beneficial impact on the self-report scores on the psychological measures considered. Studies 1 and 2 identified correlations between these self-report measures and both stature recovery and muscle activity; the results of Study 1 showed strong relationships between baseline levels of psychological factors and muscle activity and Study 2 highlighted that changes in stature recovery were related to changes in catastrophising, with a trend for similar associations with changes in anxiety and depression. Therefore it was hypothesized that even short term changes in self-report of psychological factors resulting from wearing

the heat wrap may be linked to changes in both paraspinal muscle activity and stature recovery in the current study.

The specific hypotheses addressed by this study were as follows:
(1) superficial heat treatment would result in a reduction in muscle activity and an increase in stature recovery for CLBP patients and that changes in these two factors would be related;
(2) superficial heat treatment would have a short term beneficial effect on the self report levels of the psychological factors considered and these changes would be linked to changes in both muscle activity and stature recovery.

6.2. Methods

Participants

Participants were CLBP patients who had completed either the Back Exercise Group (BEG) or the Work Back to Life Group (WBTL) within the previous two years. Data were collected from 24 patients with CLBP (age, 48.0 ± 9.0y; height, 166.6 ± 7.3cm; body mass, 80.2 ± 12.9kg). The group was mixed (9 men, 15 women), between the ages of 30 and 61 years. There was also a control group of 11 asymptomatic participants (age, 47.9 ± 15.4y; height, 168.7 ± 11.6cm; body mass, 69.3 ± 13.1kg). Further details in respect of the patients are provided in the General Methods section. Candidates were excluded from the control group if they had ever experienced recurring or persistent back pain, if they had ever lost a working day because of back pain, or had consulted a physician about back pain within the last 15 years. Participants were offered £12.50 for each session they attended to cover travel and parking expenses and CLBP patients were additionally offered two free heat wraps.

Procedure

All participants attended two testing sessions on separate days, both at the same time of day. On the second occasion, a heat wrap was given to the participant in advance, with instructions to put it on two hours before the time of the appointment. The heat wrap was then continued to be worn throughout the testing session, except for when it was briefly removed to apply the EMG electrodes. This heat wrap is made of cloth-like material which wraps around the lumbar region. It heats to 40C within 30 minutes of exposure to air and maintains this temperature for eight hours of continuous wear.

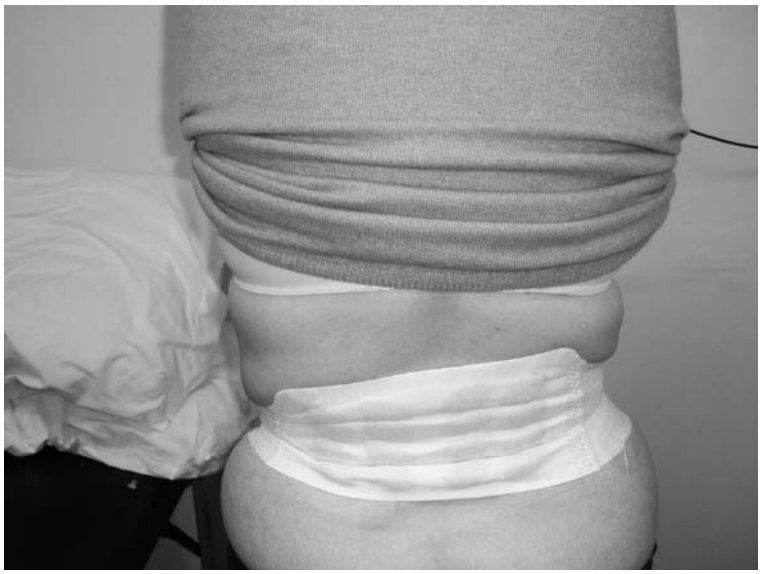


Plate 6.1. Participant wearing the heat wrap

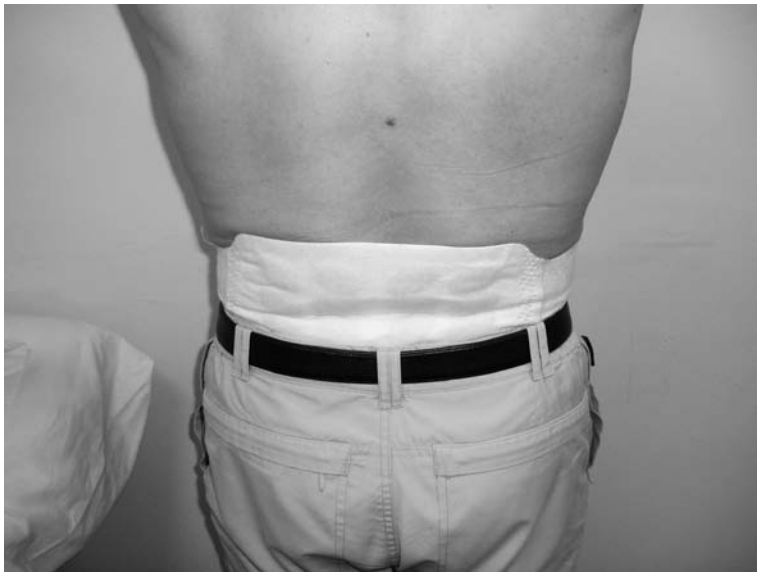


Plate 6.2. Participant wearing the heat wrap

Paraspinal muscle activity was assessed via surface electromyography (EMG), whilst both standing at rest and during a RVC. The signal was rectified and integrated over a five second period to give the integrated EMG (IEMG) value. Stature recovery over a 40-minute unloading period was measured on a standing stadiometer.

Patients were verbally asked about their pain intensity during the past 24 hours at the end of the testing session and completed a series of self-report measures following their visit. Following the session with the heat wrap they were asked, if possible, to complete the remaining questionnaire booklet in the evening after wearing the wrap all day. Details of both the protocol and the measures used are provided in the "General Methods" section.

Analysis

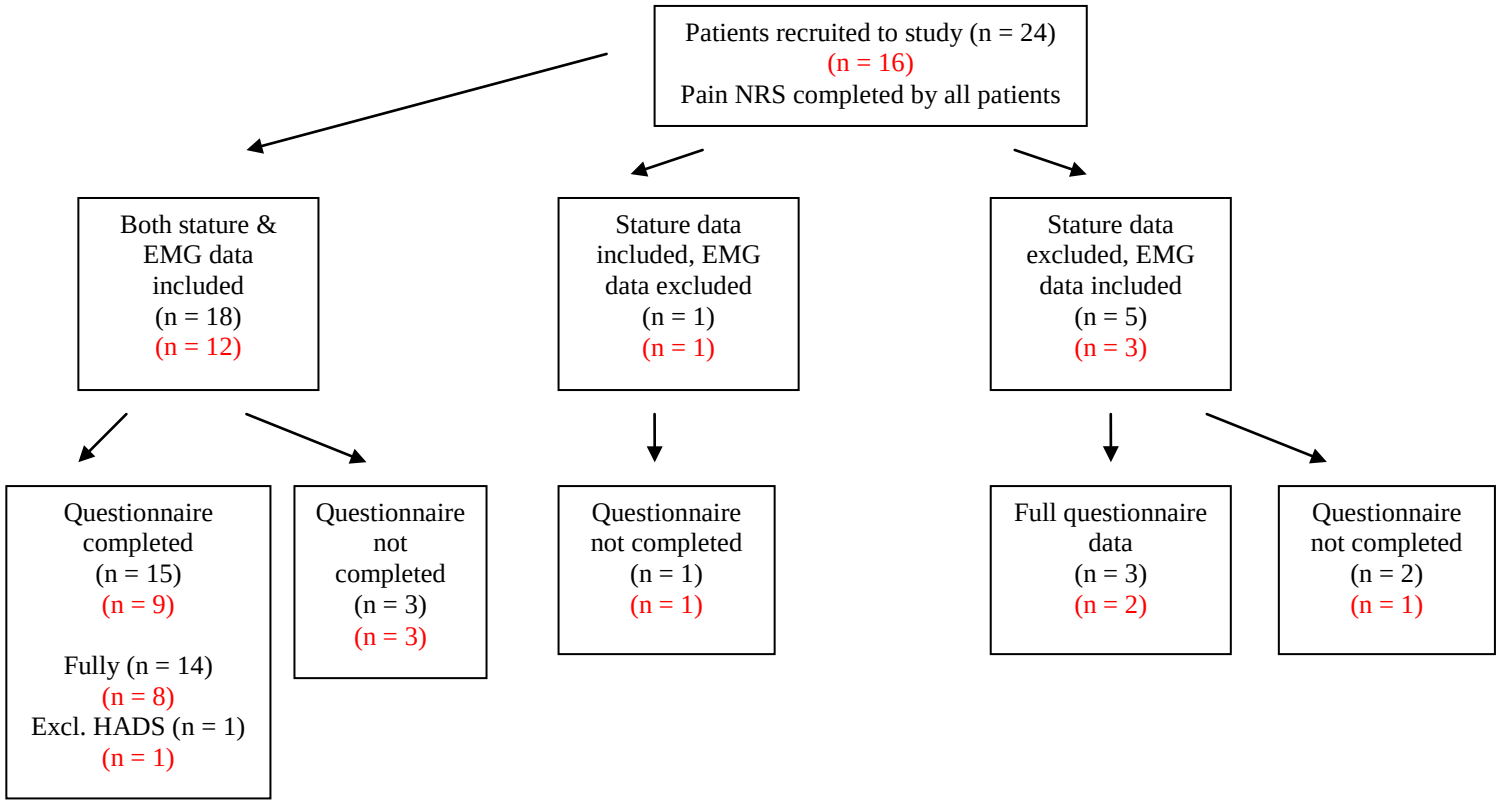
For the majority of outcome measures, one-tailed dependent t-tests were calculated to examine whether there were any differences between the two visits (one with a heat wrap and one without). However, for the non-normalised EMG data, a Kolmogorov-Smirnov test indicated that the baseline data were not normally distributed and hence a Wilcoxon signed rank test was used for this variable. Pearson's correlation coefficient was used to determine whether changes in stature recovery were associated with changes in muscle activity. For the self-report measures, Kolmogorov-Smirnov tests indicated that a number of the change scores were not normally distributed and Spearman's correlation coefficient was therefore implemented to analyse the change scores for these measures. Two-tailed Pearson correlation coefficients (or Spearman's correlation coefficient for non-normalised EMG) were employed to investigate the extent to which any of the measures at baseline were linked to reductions in muscle activity.

6.3. Results

It was notable that the patients who participated in this study had significantly lower levels of pain and disability and were also psychologically less impaired than the total patient group which took part in Study 1. This may have been because Study 1 mainly involved patients who were about to commence the rehabilitation programme and hence were likely to be experiencing more severe symptoms at that time than the patients in the current study who had all completed the programme at some point within the previous two years.

The mean duration of pain was 8.8 years and the median was 2 years (range: 4 months – 40 years). Eighteen patients completed the questionnaire on both visits. Some patients found it difficult to maintain a consistent posture in the stadiometer and three patients were excluded from the stature recovery data as the standard deviation (SD) of the five familiarisation readings was considered to be too high. As for Study 1, a SD of 1.7mm was taken as the cut-off point. The remaining patients had an average SD of 1.0mm over the five familiarisation readings. The patient who was identified as an outlier in Study 2 was again excluded from the stature recovery data. In addition, one patient was having a flare-up on his second visit and found lying down was causing him pain. The session was therefore terminated early and hence he did not have a stature recovery measurement for the heat wrap visit. As for Study 1, one patient was excluded from the EMG data as the signal contained high levels of noise and hence was considered unreliable. The numbers of patients included in the analyses are shown in Figure 6.1 on page 167.

Figure 6.1. Numbers of patients included in the analyses (figures in red reflect the numbers when the patients with differences in pain ratings of 3 or more are excluded)



Fully: All sections of the questionnaire were completed; Excl. HADS: the questionnaires were all completed with the exception of the Hospital Anxiety and Depression Scale

The results of the two visits are shown in Table 6.2 on page 169. Unfortunately the results were affected by the naturally fluctuating nature of CLBP and hence a number of patients had a notable difference between their pain ratings on the two visits (taken to be a difference of at least three on the pain NRS). The pain ratings were based on the pain levels over the previous 24 hours, rather than specifically at the time of the visit and the differences were not thought to be caused in any way by the wearing of the heat wrap. In fact, no patients reported that the heat wrap caused them pain. Six patients had notably higher pain ratings at the time of their second visit (wearing the heat wrap) compared to their visit without the wrap and two patients had a notably lower pain score. Overall this led to higher pain ratings for the heat wrap sessions. Since this was thought to affect the results, the same table has been shown (Table 6.3 on page 170), but excluding all patients with a difference of at least three in pain ratings between the two visits.

The asymptomatic participants did not exhibit any significant differences in ether stature recovery or muscle activity levels between the two visits. Eleven participants were included in the analysis but the stature recovery data for one individual was excluded as the standard deviation (SD) of the five familiarisation readings was considered to be too high. The results for the asymptomatic participants are given in Table 6.1.

Table 6.1. Outcome measures for the asymptomatic participants with and without the heat wrap

Variable	Without heat wrap Mean (± SD)	With heat wrap Mean (± SD)
Stature change (mm)	3.7 (± 1.4)	4.2 (± 2.4)
Muscle activity (% of RVC)	68.7 (± 14.7)	67.5 (± 19.9)
Muscle activity (IEMG) (μV.s)	23.4 (± 13.5)	24.4 (± 17.7)

Table 6.2. Outcome measures with and without the heat wrap

Variable	Without heat wrap Mean (± SD)	With heat wrap Mean (± SD)	Significance	Effect size
Stature change (mm)	2.9 (± 2.5)	2.8 (± 1.8)		
Muscle activity (% of RVC)	73.3 (± 11.3)	71.9 (± 13.0)		
Muscle activity (IEMG) (μV.s)	22.9 (± 8.9)	20.1 (± 7.0)*	p = 0.02	0.31
Pain during past 24 hours	3.8 (± 2.2)	4.5 (± 2.6)	p = 0.08	
Disability	7.7 (± 5.9)	7.9 (± 5.5)		
Anxiety	6.4 (± 3.5)	6.1 (± 3.7)		
Depression	4.0 (± 2.4)	4.0 (± 2.7)		
Functional self-efficacy	59.7 (± 10.8)	60.6 (± 10.3)		
Pain-related anxiety	28.7 (± 20.9)	25.8 (± 19.7)	p = 0.11	
Catastrophising	13.5 (± 10.5)	13.4 (± 12.2)		
Fear of movement	30.1 (± 12.7)	31.2 (± 12.6)		

* $p < 0.05$

The reduction in non-normalised muscle activity represents an average decrease of 9.3% compared to the visit without the heat wrap.

Table 6.3. Outcome measures with and without the heat wrap, excluding patients with difference in pain ratings of 3 or more

Variable	Without heat wrap Mean (± SD)	With heat wrap Mean (± SD)	Significance	Effect size
Stature change (mm)	3.2 (± 2.4)	3.2 (± 1.6)		
Muscle activity (% of RVC)	74.6 (± 12.5)	74.4 (± 11.3)		
Muscle activity (IEMG) (μV.s)	22.5 (± 7.1)	19.7 (± 6.8)*	$p = 0.05$	0.40
Pain during past 24 hours	3.9 (± 2.6)	3.9 (± 2.5)		
Disability	7.7 (± 6.5)	6.3 (± 6.5)**	$p < 0.01$	0.22
Anxiety	4.9 (± 3.3)	4.2 (± 3.5)		
Depression	2.7 (± 2.1)	2.6 (± 2.4)		
Functional self-efficacy	57.6 (± 12.3)	61.8 (± 10.2)**	$p < 0.01$	0.34
Pain-related anxiety	24.7 (± 19.8)	18.5 (± 15.8)*	$p = 0.04$	0.32
Catastrophising	11.5 (± 9.9)	8.6 (± 9.6)*	$p = 0.04$	0.29
Fear of movement	27.8 (± 12.6)	29.1 (± 12.9)		

* $p < 0.05$, ** $p < 0.01$

The reduction in non-normalised muscle activity represents an average decrease of 10.4% compared to the visit without the heat wrap.

Relationship between muscle activity and stature recovery

Including all patients, changes in stature recovery were significantly related to changes in non-normalised muscle activity ($r = -0.56$, $p < 0.01$), as shown in Figure 6.2.

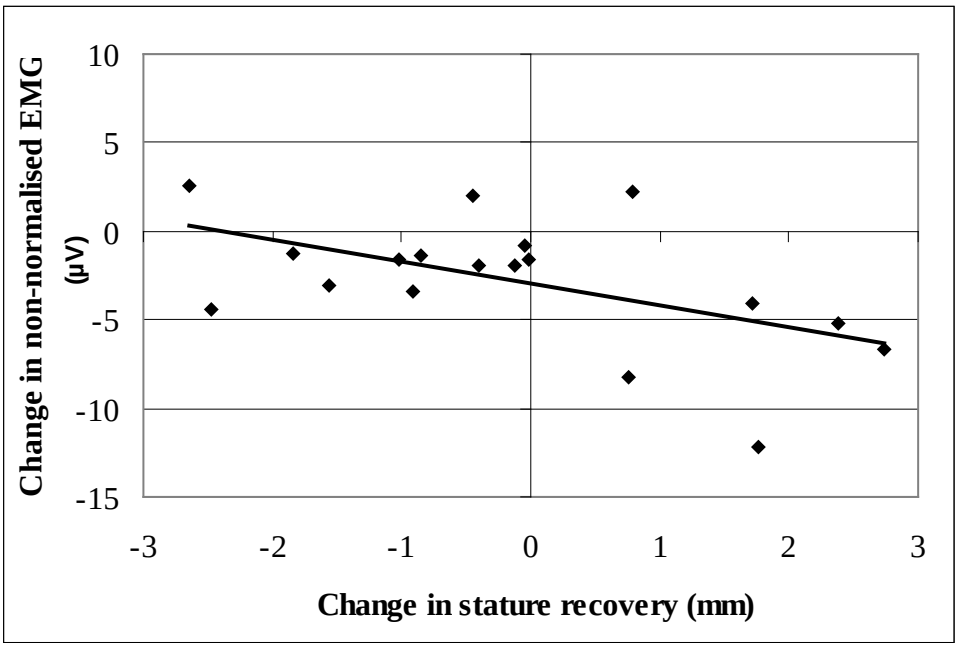


Figure 6.2. Relationship between changes in stature recovery and changes in non-normalised EMG

Correlational analysis: changes in stature recovery

Including all patients, changes in stature recovery were significantly related to changes in each of disability ($r = -0.70$, $p < 0.01$) (as shown in Figure 6.3 on page 172), fear of movement ($r = -0.53$, $p = 0.02$) (as shown in Figure 6.4 on page 172), pain ($r = -0.43$, $p = 0.03$), anxiety ($r = -0.51$, $p = 0.03$), pain-related anxiety ($r = -0.48$, $p = 0.04$) and catastrophising ($r = -0.49$, $p = 0.04$).

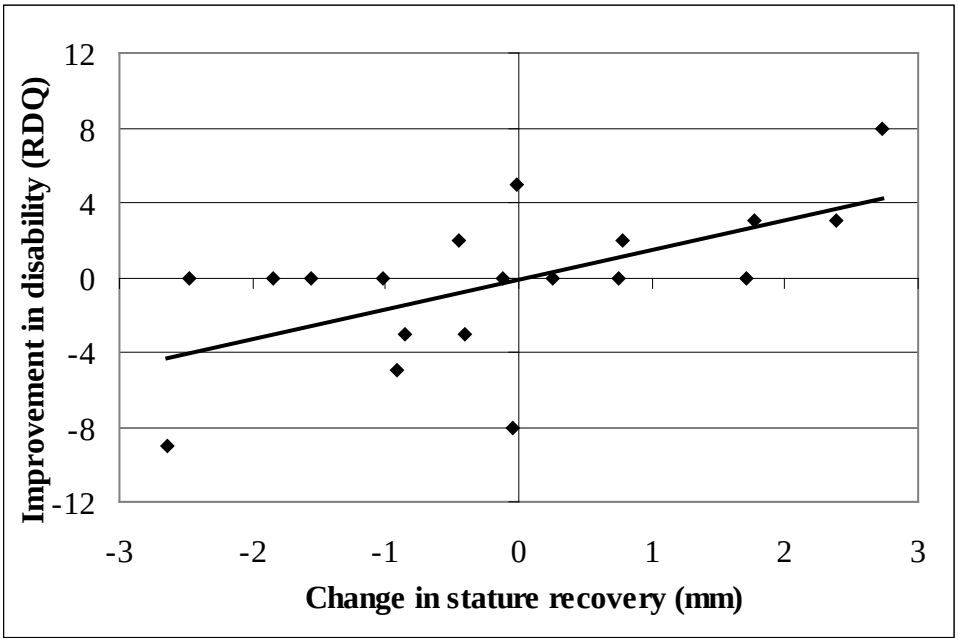


Figure 6.3. Relationship between changes in stature recovery and changes in disability. (A positive improvement in disability on the graph reflects a reduction in the score when wearing the wrap.)

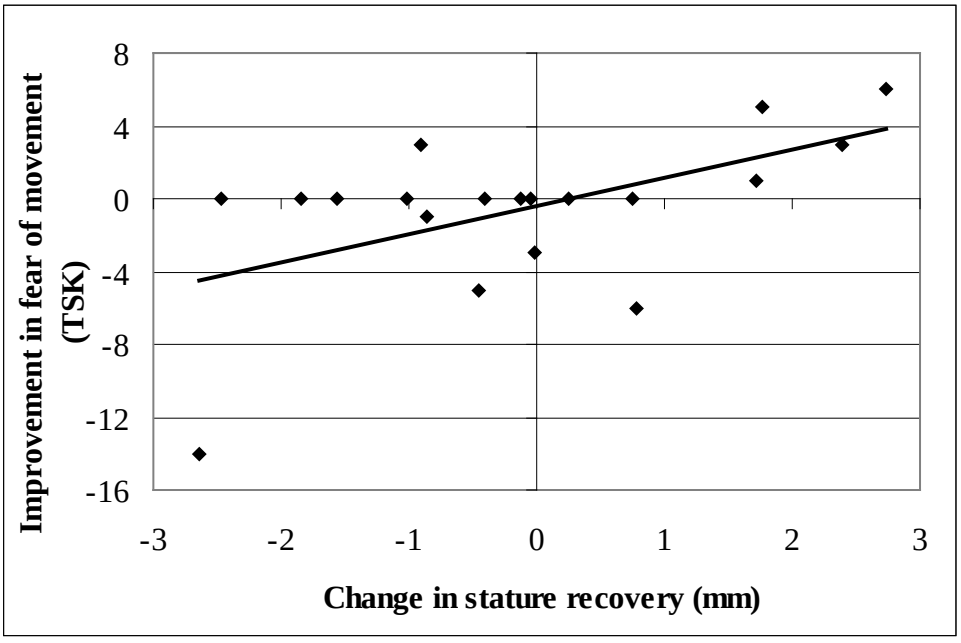


Figure 6.4. Relationship between changes in stature recovery and changes in fear of movement.
(A positive improvement in fear of movement on the graph reflects a reduction in the score when wearing the wrap.)

Correlational analysis: changes in muscle activity

Similarly, including all patients, changes in non-normalised muscle activity had significant correlations with changes in anxiety ($r = 0.79$, $p < 0.01$) (as shown in Figure 6.5) and fear of movement ($r = 0.78$, $p < 0.01$) (as shown in Figure 6.6).

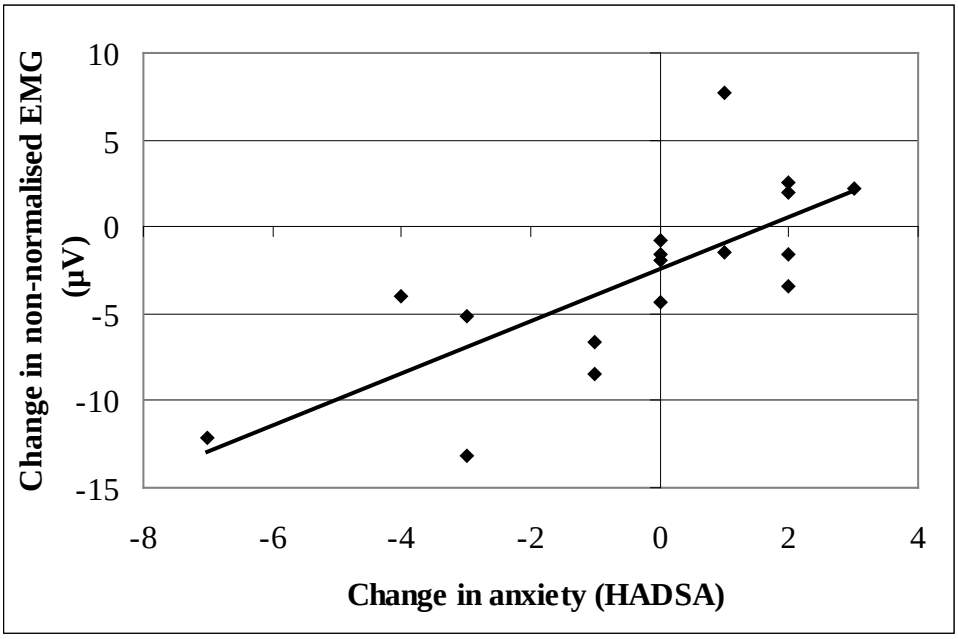


Figure 6.5. Relationship between changes in muscle activity and changes in anxiety

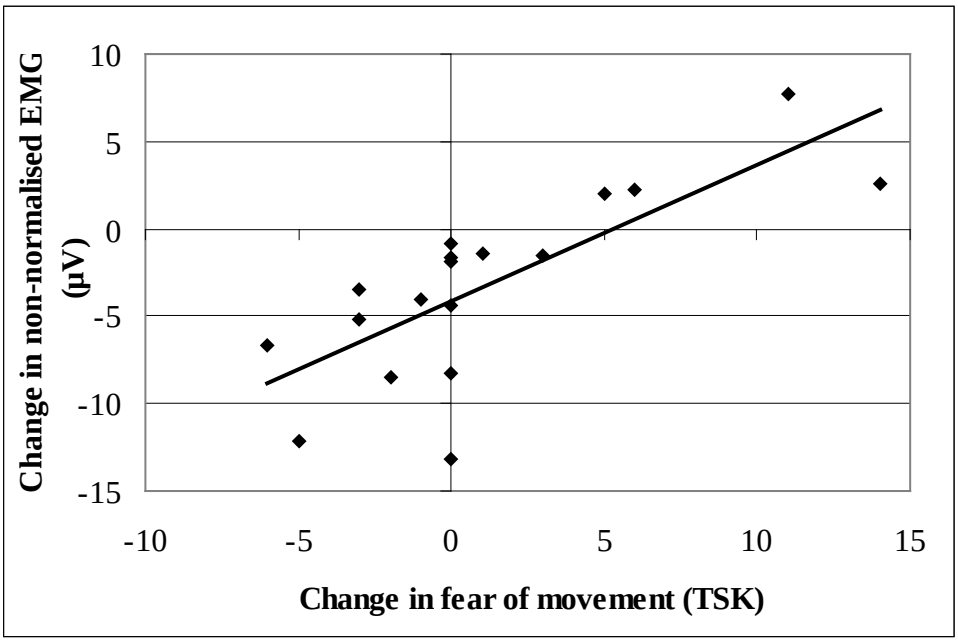


Figure 6.6. Relationship between changes in muscle activity and changes in fear of movement

There was also a trend for an association with changes in both pain-related anxiety ($r = 0.36$, $p = 0.07$) and self-efficacy ($r = -0.37$, $p = 0.06$).

Interestingly, changes in both anxiety and fear of movement were significantly linked to changes in both stature recovery and non-normalised muscle activity. However, analysis did not support changes in muscle activity as either a partial mediator or a moderator in the relationship between changes in any of the psychological factors and changes in stature recovery.

Relationships between baseline factors and outcome

Two-tailed tests revealed that patients who demonstrated reduced muscle activity when wearing the heat wrap were those with high baseline levels of both normalised ($r = -0.59$, $p < 0.01$) and non-normalised muscle activity ($r = -0.62$, $p < 0.01$) and low initial defensiveness ($r = 0.50$, $p = 0.02$). There were also trends for associations with high baseline levels of both fear of movement ($r = -0.42$, $p = 0.06$) and anxiety ($r = -0.42$, $p = 0.07$). For the patients who took part in this study, high initial levels of non-normalised muscle activity were significantly linked to high initial levels of fear of movement ($r = 0.50$, $p = 0.02$) and low defensiveness ($r = -0.51$, $p = 0.02$), but no other baseline factor.

Including all patients, the correlations between changes in the factors with and without the heat wrap are shown in Table 6.4 on page 175. Changes in disability were significantly related to changes in pain, catastrophising and self-efficacy, and changes in pain were also associated with changes in pain-related anxiety.

Table 6.4. Correlation coefficients between changes in outcome measures

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Stature change	-										
2. Muscle activity (% of RVC)	-0.09	-									
3. Muscle activity (non-normalised)	-0.56**	0.52**	-								
4. Disability	-0.70**	-0.21	0.25	-							
5. Pain	-0.43*	-0.00	0.21	0.42*	-						
6. Anxiety	-0.51*	0.51*	0.79**	0.19	0.22	-					
7. Depression	-0.26	0.05	0.21	0.10	0.13	0.61**	-				
8. Self-efficacy	0.33	-0.12	-0.37	-0.44*	-0.33	-0.27	-0.33	-			
9. Pain-related anxiety	-0.48*	0.11	0.36	0.30	0.43*	0.55*	0.71**	-0.48*	-		
10. Catastrophising	-0.49*	-0.40	-0.08	0.67**	0.08	0.13	0.08	-0.27	0.09	-	
11. Fear of movement	-0.53*	0.59**	0.78**	0.21	0.17	0.72**	0.16	-0.20	0.37	-0.00	-

* $p < 0.05$, ** $p < 0.01$

6.4. Discussion

Muscle activity expressed as a percentage of the RVC was not significantly different between the two visits. However, when the non-normalised values were considered, the values for the visit with the heat wrap were significantly lower, even when the patients in more pain were included. This therefore suggests that the heat wrap has the effect of reducing muscle activity at both rest and during the RVC. The average reduction of all patients was approximately 10%, although the effect size was only small to medium.

Normalised EMG values were used in Studies 1 and 2, and this is the typical approach when comparing EMG amplitudes. However, it is reasonable to assume that the heat wrap would have a similar effect on the muscle activity during an RVC as at rest and therefore the use of non-normalised values was considered appropriate in this case. This is consistent with the approach that has been taken by previous researchers when investigating the effect of temperature on the EMG signal (e.g. Mito *et al.*, 2007, who also carried out their testing on different days). There are issues around the inter-day comparison of non-normalised values e.g. the skin preparation and exact placement of the electrodes may differ, meaning that some variation between visits may be expected. This was seen in the inter-day variation during repeated visits in Study 1 and is likely to have contributed to the large standard deviation of the reductions seen in the current study. However, the variation in intrinsic factors, such as the thickness of the subcutaneous fat layer, is reduced due to the repeated-measures design.

Application of heat may potentially affect the EMG signal via changes in the amount of sweat and hence skin impedance. However, this was not thought to be the primary cause of the observed reduction in muscle activity when patients wore the heat wrap, since a similar decrease was not observed for the asymptomatic participants. This is consistent with

previous studies involving healthy volunteers participants, which have also tended to report that EMG amplitude is unaffected following application of heat (Holewijn & Heus, 1992; Krause *et al.,* 2001; Mito *et al.,* 2007; Petrofsky & Laymon, 2005).

Although a significant reduction in muscle activity was observed, this does not necessarily mean that the decrease was of clinical relevance. It has been reported, however, that even low level, but sustained EMG hyperactivity can contribute to the development and maintenance of chronic pain via the intense activity of single muscle fibres (Hägg, 1991). Therefore lack of sufficient relaxation may cause damage and prevent repair of damaged fibres, leading to pain. This suggests that, even if the heat wrap is able to achieve a small decrease in muscle activity, this may have a positive influence on pain in the short-term. In fact, in daily life, patients are more likely to wear the wrap when their back pain is most severe, which may mean that the true benefit is greater than was measured in the current study when patients wore the wrap on a pre-set date. This is supported by the fact that the greatest decrease in muscle activity was seen in those with high initial levels of both normalised and non-normalised muscle activity. The results also suggested that there may particularly be a benefit of heat therapy for those patients with low defensiveness or high initial levels of fear of movement or anxiety, as these were associated with greater reductions in EMG. It is important to note that the heat wrap remains warm for eight hours and is designed to be worn all day, whereas, for practical reasons, the testing session in the current study took place two hours after the wrap was put on. Therefore, it may be expected that a greater effect may be seen after a longer period of time with the wrap in place.

Although, stature recovery was not significantly different when wearing the heat wrap, there was a significant relationship between the change in non-normalised muscle activity

and the change in stature recovery. There was no link with changes in normalised EMG levels. Changes in stature recovery were also significantly related to changes in self-report of disability and pain. The results therefore generally support the hypothesized link between stature recovery and each of muscle activity, pain and disability as previously reported by Healey *et al.* (2005a).

Patients were asked to fill in the questionnaire booklets, if possible, at the end of the day (after wearing the heat wrap for the recommended eight hours) to investigate whether any differences in the psychological factors were reported. Excluding the patients with large differences in pain, self-report of functional self-efficacy was increased and self-report of disability, pain-related anxiety and catastrophising were decreased when wearing the heat wrap. It should be remembered that, by definition, self-report measures are not objective and may be subject to demand characteristics. In particular, patients might have felt that it was expected that they would answer the questionnaire in a more positive manner when wearing the heat wrap than without. In general, however, this was not thought to be the case because improvements were not reported on all the outcome measures. Therefore it was believed that the heat wrap had a positive short term effect on certain aspects of 'well-being'. In particular, the wrap appeared to have the effect of making patients feel that they were more able to continue with daily tasks, as shown in the decreased disability and increased self-efficacy scores. This is consistent with patients' comments on attending when wearing the wrap. Almost all the patients reported the wrap to be pleasant and several thought it was helping to reduce stiffness and/or possibly helping with the pain. Only two or three patients made slightly negative comments about wearing the wrap, as they found the heat to be an annoyance, although this may be partly because this study was carried out over the summer and some patients attended on hot days. None of the

participants reported that the wrap caused pain and nearly all the patients said that they would continue to wear it for the rest of the day after completing the study.

Changes in muscle activity were found to be correlated with changes in self-report of anxiety and fear of movement. This supports the strong relationships between muscle activity and psychological factors identified in Study 1, and also suggests that changes in psychological factors may have mediated the observed reductions in muscle activity. In addition, changes in stature recovery were related to changes in anxiety, pain-related anxiety, fear of movement and catastrophising. It is of interest that changes in anxiety and fear of movement were associated with changes in both muscle activity and stature recovery, which would be consistent with the original hypothesis of muscle activity as a pathway by which such factors affect stature recovery. However, analysis did not support either partial mediation or moderation in this case, although this may be the result of limited participant numbers. Additionally, only minimal changes in stature recovery were observed following use of the heat wrap, meaning that confounding factors and inter-day variation may obscure any relationships.

6.5. Conclusions

The effect of the heat wrap varied between individuals, and appeared to have particular benefit for those with high initial levels of muscle activity and low defensiveness, but overall, two hours of wear resulted in a significant decrease in non-normalised EMG levels. Care should be taken in generalising these findings due to the small to medium effect size and the well-reported issues of comparing non-normalised values for between-day visits. Although, stature recovery was not significantly different when wearing the heat wrap, there was a significant relationship between the change in non-normalised muscle

activity and the change in stature recovery. Changes in stature recovery were also significantly related to changes in self-report of disability and pain.

The wrap appeared to have a positive short-term effect on self-report of disability, self-efficacy, catastrophising and pain-related anxiety. Changes in both stature recovery and muscle activity were found to be related to changes in many of the psychological factors, again supporting the results of Studies 1 and 2 and confirming the link between the biomechanical and psychological outcome measures.

The conclusions to the specific hypotheses addressed by this study were as follows:

(1) Hypothesis: superficial heat treatment would result in a reduction in muscle activity and an increase in stature recovery for CLBP patients and that changes in these two factors would be related.
Conclusion: superficial heat treatment resulted in a reduction in muscle activity for CLBP patients, both at rest and during the RVC. Stature recovery was not significantly reduced, although changes in non-normalised muscle activity were negatively correlated with changes in stature recovery as hypothesized.

(2) Hypothesis: superficial heat treatment would have a short term beneficial effect on the self report levels of the psychological factors considered and these changes would be linked to changes in both muscle activity and stature recovery.
Conclusion: superficial heat treatment had a short-term beneficial effect on self-report levels of disability, self-efficacy, pain-related anxiety and catastrophising. Changes in stature recovery were significantly related to changes in self-report of disability, pain, anxiety, pain-related anxiety, fear of movement and catastrophising. Changes in non-

normalised muscle activity had significant correlations with changes in anxiety and fear of movement.

CHAPTER SEVEN

7. Epilogue: General discussion, conclusions and directions for future research

7.1. General discussion

This thesis aimed to investigate two main hypotheses. Firstly, that reducing paraspinal muscle activity leads to an increase in the rate of stature recovery, and so has a beneficial impact on clinical outcome. Secondly, that certain psychological factors are linked to levels of muscle activity and that these associations may help to explain causes of, and changes in, pain and disability.

The relationship between muscle activity and stature recovery

The association between paraspinal muscle activity and stature recovery was investigated in all three of the studies. In general, the results of the first two studies were unable to support the hypothesis of a significant correlation between these factors in NHS patients with CLBP, which appears to be in contrast to the findings of Healey *et al*. (2005a), who did establish such a relationship in a group of individuals with mild CLBP, who all self-managed their pain. The studies within this thesis had the advantage of deriving data from a clinical sample with moderate levels of pain and disability and the results suggest that the relationship between muscle activity and stature recovery within this patient population may be more complex than originally thought. In particular the results of Study 1 may reflect the heterogeneity typical of such a clinical population and the existence of sub-groups within the patient group who exhibit different patterns of muscular activity and/or stature recovery. In addition, it is possible that the stature change measurements were influenced by patients attending the testing session at different times of day, whereas Healey *et al*. (2005) restricted the testing sessions to approximately one hour after rising in the morning. These issues were addressed by the longitudinal nature of Study 2, which measured changes in both stature recovery and muscle activity before and after a

rehabilitation programme. In this second study, changes in stature recovery were found to be related to changes in pain and disability (and also catastrophising), but not to changes in muscle activity.

Despite these results, there was some evidence for the hypothesized relationship between stature recovery and muscle activity. In Study 1, when considering the patients who attended the BEG only, a significant negative relationship was observed between stature recovery and muscle activity. In addition, a limited number of patients attended two testing sessions before starting the rehabilitation programme and, for the seven patients who had full EMG and stadiometer data, there was a significant negative correlation between changes in stature recovery and changes in muscle activity. Similarly, Study 3 identified a significant negative link between changes in stature recovery and short-term changes in non-normalised muscle activity after wearing a heat wrap (despite no overall change in stature recovery). It is important to note that all three studies only measured superficial muscle activity as recorded by surface EMG and hence no conclusions can be drawn about the role of deeper muscles and their effect on stature recovery.

Delayed stature recovery in patients with CLBP

Although superficial muscle activity was reduced when wearing the heat wrap, the wrap did not appear to affect stature recovery. However, Study 2 demonstrated that six months after a rehabilitation programme, stature recovery was significantly increased. Since an active rehabilitation programme would not be expected to affect pathology, this suggests that the delayed stature recovery observed in CLBP patients compared to asymptomatic individuals is not primarily the result of pathology. In fact, the stature recovery exhibited by the patients at the follow-up session was similar to that shown by the control group in Study 1. Study 2 showed that immediate increases in stature recovery following

rehabilitation were linked to changes in pain and disability, which supports the clinical relevance of the reduced stature recovery observed in CLBP patients, although the study did not aim to prove causality. The link between changes in stature recovery and changes in self-reported disability was confirmed in both Studies 1 and 3, with Study 3 also showing that changes in stature recovery were related to changes in pain.

Links with psychological factors

Study 1 identified significant correlations between muscle activity and both pain and disability. Relationships were also established between muscle activity and a number of psychological factors, namely self-efficacy, depression, anxiety, pain-related anxiety and catastrophising. Furthermore muscle activity was found to partially mediate the link between self-efficacy and pain. This is an important finding which confirms the importance of paraspinal muscle activity as a pathway by which psychological factors affect clinical outcome. Although it is widely accepted that psychological factors such as self-efficacy have an impact on clinical outcome and there is also a limited body of research which has reported correlations between such factors and muscle activity, this is one of the first studies to show that muscle activity acts as a partial mediator in this way.

Although muscle activity was significantly associated with a range of psychological factors, it was not found to be related to fear of movement (except when considering the WBTL group only), which appears contrary to current literature relating to the fear-avoidance model and muscle guarding. However, this may be because muscle activity was only measured during relaxed standing whereas hyperactivity due to muscle guarding, for example, may become more apparent in certain postures or during movements perceived as threatening/harmful.

Following use of a heat wrap, changes in stature recovery were significantly related to changes in fear of movement, anxiety, catastrophising and pain-related anxiety. The repeatability data in Study 1 (based on two visits before commencing the programme) also highlighted a correlation between changes in both stature recovery and catastrophising, and following the rehabilitation programme, changes in stature recovery were significantly related to changes in catastrophising (with a trend for links with anxiety and depression). As there was no observed link with muscle activity in this second study, it suggests that there may be an alternative mechanism by which such factors are associated with stature recovery. Alternatively, the common link may be an aspect of muscle activity that was not measured within these studies. For example, any causal changes in the patterns or levels of paraspinal muscle activity may be more complicated or perhaps occurring at a deeper level than could be detected by four sites of surface EMG. Additionally, the rate at which stature recovery occurs may be associated with the ability to relax during the 40-minute unloading period. This could conceivably be associated with factors such as pain or anxiety, but would not necessarily be reflected in the subsequent EMG measurement during standing. In hindsight, it would have therefore been informative to also record the EMG levels during the unloading period. The actual EMG value used was calculated as the average of the measurements taken at the beginning, middle and end of the 40-minute unloading period and it was observed that some patients did demonstrate a reduction in EMG levels over the three readings; however this reduction in muscle activity was not found to be related to stature recovery.

Muscle activity, disability and pain

The results of the mediational analysis suggest that muscle activity affects disability via its influence on pain. However, the relationship between these three variables is likely to be more complicated than a single pathway and, within their impact on pain, both muscle

activity and disability may separately play a mediating role. This potential interaction between muscle activity, disability and pain further supports the importance of muscle activity in LBP and implies that reducing muscle activity may have a beneficial impact on clinical outcome.

This notion of an interaction between muscle activity, disability and pain appears broadly consistent with the biomedical/biomechanical models of LBP such as the pain-spasm-pain, pain adaptation and instability models. Although these models generally suggest pain as an initial trigger for increased muscle activity, once the condition has become chronic, the increased muscle activity itself is thought to then cause a cycle of increased pain and disability via hyperactivity of painful muscles, lack of muscle relaxation, altered patterns of movement and increased and abnormal spinal loading. The results could also be argued to be consistent with the fear avoidance and muscle guarding theories. These propose that, although initially triggered by pain, fear of pain/movement can subsequently lead to increased levels of muscle activity, pain and disability and a vicious cycle of fear of pain/movement, increased pain and disability, deconditioning and muscle hyperactivity.

Relationships between baseline factors and outcome and sub-groups

Patients with high baseline levels of anxiety, pain-related anxiety and catastrophising and low levels of self-efficacy were those least likely to complete the programme. Patients who did complete, however, showed improvements in pain, disability and self-report measures that persisted to at least the six-month follow-up session. From the self-selected group who participated in the follow-up, high initial levels of pain, catastrophising and defensiveness were associated with greater overall reductions in pain. To some extent, this may just reflect the fact that the most anxious patients had dropped out before this stage, as well as the fact that the patients in the most pain had the most to gain. However, it also emphasises

the importance of encouraging attendance and completion of the programme, as those most likely to drop out are potentially those who would benefit most.

The results of Study 1 highlight that muscle hyperactivity plays an important role, particularly for those patients with low self-efficacy and/or high levels of depression, anxiety, pain-related anxiety and catastrophising. As muscle activity acts as a partial mediator in the link between back pain and self-efficacy (with a trend for a similar role in the link with depression, anxiety, pain-related anxiety and catastrophising), a reduction in muscle activity may be expected to help reduce the effect of these factors on pain. This therefore suggests that interventions that are able to reduce muscle activity may be of particular benefit to patients demonstrating these characteristics, which may help in the targeting of treatment for LBP.

The patients who appeared to benefit most from wearing the heat wrap (in terms of reductions in muscle activity) were those with high initial levels of both normalised and non-normalised muscle activity, low baseline defensiveness and high initial fear of movement and anxiety. This may also suggest that such patients may benefit from other interventions aimed at reducing muscle activity, such as massage.

Coping styles

The results of Study 1 highlighted that a high proportion of the patients were classified as defensive high-anxious (DHA). This is in contrast to a number of studies which have instead found the repressive coping style to be prevalent in patients with chronic illnesses, although a similar finding was reported for chronic fatigue syndrome. The role of defensiveness within CLBP is not an area widely covered by the literature, although the current findings suggest that it may be a risk factor for either initial onset or poor clinical

outcome. It may have been expected that high defensiveness would be associated with elevated muscle activity, particularly given the strong positive relationships between muscle activity and the other psychological factors considered. In fact, this was not found to be the case and there was a trend for a negative relationship between the two variables. It is not clear why this should be and it would be interesting to investigate this further.

As defensiveness was assumed to be a stable trait, patients were only asked to fill in the MC-SD once. The decision to include the MC-SD was based on the preliminary findings of Studies 1 and 2, which highlighted anxiety as a potentially important factor, justifying further sub-classification of patients within the low/high anxious groups. As it was included at a later date, the MC-SD was completed at varying points within the treatment process, which may have affected results if, in fact, defensiveness is affected by factors such as clinical outcome. In hindsight, it would have been informative to administer the MC-SD more than once to assess whether defensiveness did remain stable throughout the rehabilitation programme and six-month follow-up period. The answer to this question is particularly pertinent since a reduction in pain by the end of the six-month follow-up was observed to be linked to high baseline levels of defensiveness, but it is unknown whether reductions in pain were linked to changes in defensiveness. If this were the case, it may encourage interventions that aim to reduce defensiveness. A positive conclusion, however, is that defensiveness did not appear to be a barrier to improvement during rehabilitation.

Repressors tend to be overly positive, both in terms of interpretation bias (Eysenck, 2000), and in predicting their future performance (Jones *et al*., 2004). This may have an impact on pacing, potentially leading to task persistence and endurance behaviour. In fact, Hasenbring, Hallner and Rusu (2009) describe an endurance subgroup characterised by task persistence behaviour linked with marked positive mood despite pain, which may

overlap with the group of patients classified as repressors. Unfortunately, given the very small number of repressors identified, it was not possible to investigate whether a repressive coping style was associated with elevated muscle activity, as would be consistent with the increased physiological reactivity predicted by the theory. In fact, the number of repressors identified in Study 1 is slightly fewer than might be expected in a typical non-clinical population, probably reflecting the reluctance of individuals with this coping style to initially seek medical support and then to persist with a additional rehabilitation programme. Screening patients for a repressive coping style may help during their subsequent treatment as likely effects on pacing and under-reporting of 'true' levels of distress could be acknowledged and allowed for.

Superficial heat treatment

The effect of the heat wrap varied between individuals, and appeared to have particular benefit for those with high initial levels of muscle activity and low defensiveness, but overall, it was associated with a significant decrease in non-normalised EMG levels. This may have a positive influence on pain in the short-term. Although the reduction in muscle activity only represented a small to medium effect size, this result was based on two hours of wear, whereas the wrap is actually designed to be worn for eight hours. Additionally, in daily life, patients may be more likely to wear the wrap during periods of higher pain and possible higher muscle activity, which may lead to a greater benefit than was measured in the current study. The wrap appeared to have a positive short-term effect on self-report of disability, self-efficacy, catastrophising and pain-related anxiety and, in particular, there were significant correlations between reductions in anxiety and fear of movement and decreases in muscle activity. It is therefore possible that the beneficial effects of the heat wrap on muscle activity occurred, to some extent, via its effect on psychological factors.

Power analysis

Power analysis based on the correlations between stature recovery and each of muscle activity, pain and disability reported by Healey *et al*. (2005a) suggested that a sample size of 32 would be sufficient to achieve power of 80% in detecting a link between these baseline measures. For reasons stated above however, any associations with stature recovery may be weaker for the patients included in the current study. In fact 47 patients participated in Study 1, although 13 were excluded from the stadiometer and/or EMG data (due to technical problems or because their data were considered unreliable), but a post-hoc power analysis for the stature recovery and pain data indicated power of only 39%. A *priori* power analysis was not carried out for Studies 2 and 3 and the lower participant numbers for these studies (particularly for the follow-up data in Study 2) inevitably resulted in reduced power, which may have led to correlations being overlooked. The results of these studies should therefore be interpreted with care with this in mind. The results of these studies should therefore be interpreted with this in mind. As is often the case, greater participant numbers would have been preferred. However, even where associations were not found to be significant, the results still form a useful basis for further research.

7.2. Conclusions and directions for future research

The conclusions to the specific hypotheses addressed by each study were as follows:

Study 1

(1) Hypothesis: patients with CLBP demonstrate acceptable levels of repeatability for inter-day stadiometer measurements.

Conclusion: patients with CLBP do demonstrate acceptable levels of repeatability for inter-day stadiometer measurements.

(2) Hypothesis: patients with CLBP have higher muscle activity and reduced stature recovery compared to asymptomatic individuals.
Conclusion: there was a trend for patients with CLBP to have higher muscle activity and reduced stature recovery compared to asymptomatic individuals.

(3) Hypothesis: stature recovery for patients with CLBP is negatively related to each of muscle activity, pain and disability.
Conclusion: there was a trend for patients' stature recovery to be negatively related to pain, but there was no link with muscle activity or disability. However, when considering the BEG only, there was a negative relationship between stature recovery and both muscle activity and pain. For the patients who attended twice before starting the rehabilitation programme, there was a significant correlation between changes in stature recovery and changes in both muscle activity and disability.

(4) Hypothesis: muscle activity for patients with CLBP is related to pain and disability.
Conclusion: significant correlations supported the hypothesis that patients' muscle activity is related to both pain and disability.

(5) Hypothesis: psychological factors are associated with paraspinal muscle activity and stature recovery for patients with CLBP.
Conclusion: muscle activity was significantly related to self-efficacy, depression, anxiety, pain-related anxiety and catastrophising and was a partial mediator in the relationship between self-efficacy and pain. Psychological factors were not found to be related to stature recovery, although changes in stature recovery were linked to changes in anxiety for the patients who attended twice before starting the programme.

Study 2

(1) Hypothesis: following the rehabilitation programme, patients with CLBP demonstrate improvements in pain, disability and psychological factors.

Conclusion: by the end of the six-month follow-up period, CLBP patients demonstrated improvements in disability, self-efficacy, anxiety, depression and fear of movement, with trends for improvements in pain and catastrophising.

(2) Hypothesis: paraspinal muscle activity of CLBP patients is reduced following the rehabilitation programme, and this improvement is linked to improvements in pain, disability and psychological factors.

Conclusion: for many of the patients, muscle activity was unexpectedly increased immediately following the programme. Levels were reduced by the end of the six-month follow-up period, but this was not significant. There was a trend for overall decreases in EMG levels to be associated with improvements in depression and self-efficacy, and changes in the follow-up period only were related to changes in anxiety.

(3) Hypothesis: stature recovery of CLBP patients is increased following the rehabilitation programme, and this improvement is linked to reductions in muscle activity and improvements in pain, disability and psychological factors.

Conclusion: stature recovery was significantly increased by the end of the six-month follow-up period, but not immediately following the rehabilitation programme. Initial changes in stature recovery were linked to changes in pain, disability and catastrophising. There was also a trend for reductions in both anxiety and depression to be associated with increased stature recovery.

(4) Hypothesis: patients with low baseline levels of both muscle activity and psychological factors, such as anxiety, depression and catastrophising, demonstrate better clinical outcome than those with high levels.

Conclusion: contrary to expectations, for those patients who completed the programme, and returned for a six-month follow-up, a reduction in pain by the end of this period was significantly linked to high baseline levels of pain, catastrophising and defensiveness. Participant numbers were limited, however, and self-selection may have influenced the results. Initial muscle activity levels were not related to clinical outcome.

Study 3

(1) Hypothesis: superficial heat treatment would result in a reduction in muscle activity and an increase in stature recovery for CLBP patients and that changes in these two factors would be related.

Conclusion: superficial heat treatment resulted in a reduction in muscle activity for CLBP patients, both at rest and during the RVC. Stature recovery was not significantly reduced, although changes in non-normalised muscle activity were negatively correlated with changes in stature recovery as hypothesized.

(2) Hypothesis: superficial heat treatment would have a short term beneficial effect on the self report levels of the psychological factors considered and these changes would be linked to changes in both muscle activity and stature recovery.

Conclusion: superficial heat treatment had a short-term beneficial effect on self-report levels of disability, self-efficacy, pain-related anxiety and catastrophising. Changes in stature recovery were significantly related to changes in self-report of disability, pain, anxiety, pain-related anxiety, fear of movement and catastrophising. Changes in non-

normalised muscle activity had significant correlations with changes in anxiety and fear of movement.

The overall aim of the thesis was:

Global aim: To establish whether active rehabilitation programmes and superficial heat treatment result in reduced paraspinal muscle activity in CLBP patients and whether this affects an increase in the rate of stature recovery. If so, to investigate whether this has a beneficial effect on clinical outcome, or can help to identify any subgroups of patients for whom this approach might be most effective.

In conclusion, there was limited evidence for a link between muscle activity and stature recovery within the CLBP patient population, but there was no evidence that reducing paraspinal muscle activity directly affected an increase in the rate of stature recovery. However, stature recovery was significantly increased to levels similar to an asymptomatic control group following an active rehabilitation programme, suggesting that the delayed recovery in CLBP patients is not primarily caused by pathology. The link between initial changes in stature recovery and clinical outcome supports the argument that the increase is of clinical relevance. Future investigations with a more intensive heat treatment (as well as other interventions aimed at muscle relaxation) may be expected to lead to a more marked reduction in muscle activity and hence provide further information regarding the association between changes in muscle activity and stature recovery. Overall, the results indicated that stature recovery measurements were less useful than expected for comparisons between patients with back pain and should not be used as a proxy indicator of absolute levels of pain or disability, or of the impact of psychological factors. In contrast, when considering intra-individual changes in stature recovery over time, and following rehabilitation or heat treatment, the results were more positive. Changes in

stature recovery were linked to changes in pain, disability and a number of psychological factors, both in Studies 1 and 3, where the measurements were up to two weeks apart and also in Study 2, where the measurements were up to six weeks apart. However, no correlations were found in the follow-up data for Study 2, suggesting that comparisons of stadiometer readings taken six months apart may be less reliable. Nevertheless, over periods of up to six weeks, stature recovery measurements could potentially be used as a proxy indicator of changes in clinical outcome.

There were a number of important findings regarding the role of muscle activity in CLBP. Study 1 identified significant correlations between muscle activity and both pain and disability and indicated that muscle activity affects disability via its influence on pain. Relationships were also established with each of self-efficacy, depression, anxiety, pain-related anxiety and catastrophising. Furthermore muscle activity was found to partially mediate the link between self-efficacy and pain. By measuring EMG levels, it was observed that there appears to be an adaptation period immediately following the rehabilitation programme, during which levels of muscle activity remain broadly the same, or even increase, before eventually reducing as expected. These findings confirm that muscle activity plays an important role in CLBP, particularly as a pathway by which psychological factors may impact on clinical outcome. This is despite EMG readings only being taken during relaxed standing at rest. More information may be gained by examining muscle activity levels in different postures or during movements perceived as threatening/harmful.

The role of muscle activity as a mediator between certain psychological factors and pain suggests that interventions that are able to reduce muscle activity may be of particular benefit to patients demonstrating these characteristics, which may help in the targeting of

treatment for LBP. For example, scores which exceed pre-set levels on psychological questionnaires could act as triggers to indicate that a patient is likely to demonstrate elevated muscle activity and that interventions, such as massage or relaxation techniques which are focused on reducing that hyperactivity, may be appropriate. These patients may also benefit simply by receiving education regarding the relationships between certain psychological factors and muscle activity and learning techniques to relax their back muscles during times of high anxiety, for example. Alternatively, high levels of muscle activity might indicate that a patient should be screened for the presence of psychological factors as a priority and that they may benefit from receiving treatments such as CBT, which aim to tackle psychosocial issues. However, the observations following the rehabilitation programme demonstrate that an immediate decrease in EMG levels may not always be the optimal result for long-term improvements in clinical outcome and that a period of adaptation might be expected. Research is needed to measure EMG levels following a cognitive therapy that does not include an exercise component to see whether a similar adaptation period is observed, or whether this is an adaptation to increased levels of physical activity only.

The high proportion of defensive high-anxious (DHA) individuals within the CLBP population is an interesting finding. Longitudinal studies are needed to clarify whether this coping style is a risk factor for initial onset of LBP, whether it is associated with poor outcome, or even whether a more defensive coping style tends to be adopted in response to a chronic condition, as has been indicated by some of the initial research in this area. It is important to understand the role that coping styles play within the development of CLBP and hence whether interventions aimed specifically at reducing defensiveness might be associated with improved outcome for CLBP patients. High defensiveness, however, did not appear to be a barrier to benefiting from treatment as reductions in pain following

rehabilitation were linked to high baseline levels of defensiveness. Information regarding the DHA coping style is limited due to its scarcity and hence little is known about associated cognitive biases or coping strategies and their effect on treatment effectiveness. The observation of a high prevalence of DHA individuals within the CLBP population provides an opportunity to study this coping style and its impact in more detail.

Only a limited number of patients were identified as repressors, although this may be because repressors would be less likely to participate in such a study. It would therefore be useful to establish the prevalence of this coping style within CLBP, whether it is in fact maladaptive within this condition and whether repressors do display the elevated muscle activity that might be anticipated from the theory. In general, classifying coping style may help identify vulnerable patients, highlight possible under-reporting of distress (in the case of repressors) and help with more effective targeting of treatments. For example, treatments that give repressors a greater feeling of control may encourage participation. In addition, the tendency to be overly positive may have an impact on pacing, potentially leading to task persistence despite pain, in which case education regarding effective pacing may be beneficial. Further studies are needed to investigate the link between coping style and pacing and whether a repressive coping style is associated with an endurance-related pattern of high persistence behaviour. To date, the effect of coping styles within CLBP, or even the prevalence of different coping styles with the CLBP population, has not been addressed.

To summarise, the three studies were unable to unequivocally confirm that elevated paraspinal muscle activity and stature recovery were related in patients with CLBP and that reducing muscle activity would necessarily lead to improved stature recovery. However, the results do indicate that both stature recovery and muscle activity play an important role

within CLBP and the management of the condition. The key findings from the thesis with respect to muscle activity were that significant correlations support the hypothesis that patients' muscle activity is negatively related to both pain and disability and that mediational analysis suggests that muscle activity affects disability via its influence on pain. Muscle activity is significantly related to self-efficacy, depression, anxiety, pain-related anxiety and catastrophising and is a partial mediator in the relationship between self-efficacy and pain. In addition, there appears to be an adaptation period such that muscle activity is not reduced immediately following a rehabilitation programme. The principal results in respect of stature recovery were that changes in stature recovery are linked to changes in both clinical outcome and psychological factors and that stature recovery is significantly increased six months after a rehabilitation programme. The thesis also made the important finding of a high prevalence of the DHA coping style within the CLBP population.

There are a number of clinical implications which follow from the findings in the thesis. The results generally support the importance of muscle activity in LBP and indicate that reducing muscle activity may have a beneficial impact on clinical outcome. The role of muscle activity as a mediator between psychological factors and pain suggests that interventions that are able to reduce muscle activity may be of particular benefit to patients demonstrating characteristics such as low self-efficacy. Furthermore, the presence of elevated muscle activity might indicate that a patient should be screened for the presence of psychological factors as a priority and conversely, high scores on certain psychological questionnaires might act as triggers to indicate that a patient is likely to demonstrate elevated muscle activity. Superficial heat treatment was associated with both a short-term decrease in muscle activity and a positive effect on self-report of disability, self-efficacy, catastrophising and pain-related anxiety. However, the results of the longitudinal study

also highlight that an immediate decrease in EMG levels following active treatment may not always be the optimal response for long-term improvements in clinical outcome and that a period of adaptation might be expected. The relationship between stature change and muscle activity appears to be more complex than originally hypothesized. Six months after a rehabilitation programme, however, the rate of stature recovery might be expected to have increased to levels similar to asymptomatic individuals, suggesting that the delayed recovery seen in CLBP patients is not primarily the result of pathology. The results support the use of changes in stature recovery measurements as a proxy indicator of changes in clinical outcome over periods of up to six weeks, but measurements should not be used as indicators of absolute levels of pain or disability, or of the impact of psychological factors. A high prevalence of patients classified as DHA was identified within the patient group attending the outpatient rehabilitation programme and therefore treatments may be more effective if targeted at this particular coping style. In contrast, the number of repressors was lower than might be expected in the general population and hence tailoring interventions to better suit this coping style (for example, by giving patients a greater sense of control) may encourage attendance.

REFERENCES

References

A.D.A.M. Medical Encyclopedia (2010). Retrieved May14, 2011 from http://www.nlm.nih.gov/medlineplus/ency/imagepages.

Adams, M. A. (2004). Biomechanics of back pain. *Acupuncture in Medicine, 22*, 178-188.

Adams, M. A., Bogduk, N., Burton, K., & Dolan, P. (2002). *The Biomechanics of Back Pain*. Edinburgh: Churchill Livingstone.

Adams, M. A., & Dolan, P. (1995). Recent advances in lumbar spinal mechanics and their clinical significance. *Clinical Biomechanics, 10,* 3-19.

Adams, M. A., & Hutton, W. C. (1980). The effect of posture on the role of the apophysial joints in resisting intervertebral compressive forces. *The Journal of Bone and Joint Surgery, 62*, 358-362.

Adams, M. A., & Hutton, W. C. (1983). The effect of posture on the fluid content of lumbar intervertebral discs. *Spine, 8*, 665-671.

Adams, M. A., Mannion, A. F., & Dolan, P. (1999). Personal risk factors for first-time low back pain. *Spine, 24*, 2497-2505.

Adams, M. A., McMillan, D. W., Green, T. P., & Dolan, P. (1996). Sustained loading generates stress concentrations in lumbar intervertebral discs. *Spine, 21*, 434-438.

Ahern, D. K., Follick, M. J., Council, J. R., Laser-Wolston, N., & Litchman, H. (1988). Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and non-patient controls. *Pain, 34*, 153-160.

Airaksinen, O., Brox, J. I., Cedraschi, C., Hildebrandt, J., Klaber-Moffett, J., Kovacs, F., *et al*. (2006). European guidelines for the management of chronic non-specific low back pain. *European Spine Journal, 15,* S192-300.

Alexiev, A. R. (1994). Some differences of the electromyographic erector spinae activity between normal subjects and low back pain patients during the generation of isometric trunk torque. *Electromyography and Clinical Neurophysiology, 34*, 495-499.

Al-Obaidi, S. M., Nelson, R. M., Al-Awadhi, S., & Al-Shuwaie, N. (2000). The role of anticipation and fear of pain in the persistence of avoidance behaviour in patients with chronic low back pain. *Spine, 25*, 1126-1131.

Alschuler, K. N., Theisen-Goodvich, M. E., Haig, A. J., & Geisser, M. E. (2008). A comparison of the relationship between depression, perceived disability, and physical performance in persons with chronic pain, *European Journal of Pain, 12*, 757-764.

Althoff, I., Brinckmann, P., Frobin, W., Sandover, J., & Burton, K. (1992). An improved method of stature measurement for quantitative determination of spinal loading: application to sitting postures and whole body vibration. *Spine, 17*, 682-693.

Ambroz, C., Scott, A., Ambroz, A., & Talbott, E. O. (2000). Chronic low back pain assessment using surface electromyography. *Journal of Occupational and Environmental Medicine, 42*, 660-669.

Anderson, K. O., Dowds, B. N., Pelletz, R. E., Edwards, W. T., & Peeters-Asdourian, C. (1995). Development and initial validation of a scale to measure self-efficacy beliefs in patients with chronic pain. *Pain, 63*, 77-84.

Andersson, E. A., Oddsson, L. I. E., Grundström, H., Nilsson, J., & Thorstensson, A. (1996). EMG activities of the quadratus lumborum and erector spinae muscles during flexion-relaxation and other motor tasks. *Clinical Biomechanics, 11*, 392-400.

Arena, J. G., Sherman, R. A., Bruno, G. M., & Young, T. R. (1989). Electromyographic recordings of 5 types of low back pain subjects and non-pain controls in different positions. *Pain, 37*, 57-65.

Arena, J. G., Sherman, R. A., Bruno, G. M., & Young, T. R. (1991). Electromyographic recordings of low back pain subjects and non-pain controls in six different positions: Effect of pain levels. *Pain, 45*, 23-28.

Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H., & Svensson, P. (1995). The influence of low back pain on muscle activity and coordination during gait: A clinical and experimental study. *Pain, 64*, 231-240.

Arnstein, P., Caudill, M., Mandle, C. L., Norris, A., & Beasley, R. (1999). Self efficacy as a mediator of the relationship between pain intensity, disability and depression in chronic pain patients. *Pain, 80*, 483-491.

Asmundson, G. J., Norton, P. J., & Vlaeyen, J. W. S. (2004). Fear-avoidance models of chronic pain: an overview. In: Asmundson, G. J., Vlaeyen, J. W. S., & Crombez, G. (Eds). *Understanding and treating fear of pain*. Oxford: Oxford University Press, pp3-24.

Au, G., Cook, J., & McGill, S. M. (2001). Spinal shrinkage during repetitive controlled torsional, flexion and lateral bend motion exertions. *Ergonomics, 44*, 373-381.

Bair, M. J., Robinson, R. L., Katon, W., & Kroenke, K. (2003). Depression and pain comorbidity, *Archives of Internal Medicine, 163*, 2433-2445.

Bergmark, A. (1989). Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthopaedica Scandinavica Supplementum, 230*, 20-24.

Beynon, C., & Reilly, T. (2001). Spinal shrinkage during a seated break and standing break during simulated nursing tasks. *Applied Ergonomics, 32*, 617-622.

Bicalho, E., Setti, J. A. P., Macagnan, J., Cano, J. L. R., & Manffra, E. F. (2010). Immediate effects of a high-velocity spine manipulation in paraspinal muscles activity of nonspecific chronic low-back pain subjects. *Manual Therapy, 15*, 469-475.

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale: an updated literature review. *Journal of Psychosomatic Research, 52*, 69-77.

Boersma, K., & Linton, S. J. (2005). How does persistent pain develop? An analysis of the relationship between psychological variables, pain and function across stages of chronicity. *Behaviour Research and Therapy, 43*, 1495-1507.

Boersma, K., Linton, S., Overmeer, T., Jansson, M., Vlaeyen, J., & de Jong, J. (2004). Lowering fear-avoidance and enhancing function through exposure *in vivo*. A multiple baseline study across six patients with back pain. *Pain, 108*, 8-16.

Bogduk, N. (2005). *Clinical Anatomy of the Lumbar Spine and Sacrum* (4th ed.). Elsevier: Edinburgh.

Boos, N., Wallin, A., Aebi, M., & Boesch, C. (1996). A new magnetic resonance imaging analysis method for the measurement of disc height variations. *Spine, 21*, 563-570.

Botsford, D. J., Esses, S. I., & Ogilvie-Harris, D. J. (1994). In vivo diurnal variation in intervertebral disc volume and morphology. *Spine, 19*, 935-940.

Bousema, E. J., Verbunt, J. A., Seelen, H. A. M., Vlaeyen, J. W. S., & Knottnerus, J. A. (2007). Disuse and physical deconditioning in the first year after the onset of back pain. *Pain, 130*, 279-286.

Brinckmann, P., Frobin, W., Hierholzer, E., & Horst, M. (1983). Deformation of the vertebral end-plate under axial loading of the spine. *Spine, 8*, 851-856.

British Pain Society. (2007, April). *Recommended Guidelines for Pain Management Programmes for Adults.* Retrieved February 2, 2008 from http://www.britishpainsociety.org/pub_professional.htm#pmp

Broberg, K. B. (1993). Slow deformation of intervertebral discs. *Journal of Biomechanics, 26*, 501-512.

Brody, S., Wagner, D., Heinrichs, M., James, A., Hellhammer, D., & Ehlert, U. (2000). Social desirability scores are associated with higher morning cortisol levels in firefighters. *Journal of Psychosomatic Research, 49*, 227-228.

Brumagne, S., Janssens, L., Knapen, S., Claeys, K., & Suuden-Johanson, E. (2008). Persons with recurrent low back pain exhibit a rigid postural control strategy. *European Spine Journal, 17*, 1177-1184.

Buer, N., & Linton, S. J. (2002). Fear-avoidance beliefs and catastrophizing: occurrence and risk factor in back pain and ADL in the general population. *Pain, 99*, 485-491.

Burden, A. (2008). Surface electromyography. In Payton, C., & Bartlett, R. (Eds.), *Biomechanical Evaluation of Movement in Sport and Exercise: The British Association of Sport and Exercise Sciences Guide* (pp. 77-102). Abingdon: Routledge.

Burns, J. W. (2000a). Repression in chronic pain: an idea worth recovering? *Applied & Preventive Psychology, 9*, 173-190.

Burns, J. W. (2000b). Repression predicts outcome following multi-disciplinary treatment of chronic pain. *Health Psychology, 19*, 75-84.

Burns, J. W. (2006). Arousal of negative emotions and symptom-specific reactivity in chronic low back pain patients. *Emotion, 6*, 309-319.

Burris, J. E., Johnson, T. P., & O'Rourke, D. (2003). Validating Self-Reports of Socially Desirable Behaviors. Paper presented at: 58th Annual Conference of the American Association for Public Opinion Research, Nashville, TN; May 15-18, 2003. Available from: http://www.amstat.org/sections/srms/Proceedings/y2003/ Files/JSM2003-000914.pdf.

Cailliet, R. (1991). *Low Back Pain Syndrome:* 4th edition. Philadelphia: FA Davis Company.

Carleton, R. N., & Asmundson, G. J. G. (2009). The multidimensionality of fear of pain: construct independence for the Fear of Pain Questionnaire-Short Form and the Pain Anxiety Symptoms Scale-20, *The Journal of Pain, 10*, 29-37.

Carroll, L. J., Cassidy, J. D., & Côté, P. (2004). Depression as a risk factor for onset of an episode of troublesome neck and low back pain. *Pain, 107*, 134-139.

Cherkin, D. C., Deyo, R. A., Street, J. H. (1996). Predicting poor outcomes for back pain seen in primary care using patients' own criteria, *Spine, 21*, 2900-2907.

Cholewicki, J., Panjabi, M., & Khachatryan, A. (1997). Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. *Spine, 22*, 2207-2212.

Cohen, J. (1988). *Statistical power analysis for behavioural science* (2nd ed.) New Jersey: Lawrence Erlbaum Associates.

Collins, G. A., Cohen, M. J., Naliboff, B. D., & Schandler, S. L. (1982). Comparative analysis of paraspinal and frontalis EMG, heart rate and skin conductance in chronic low back pain patients and normals to various postures and stress. *Scandinavian Journal of Rehabilitation Medicine, 14*, 39-46.

Coons, M. J., Hadjistavropoulos, H. D., & Asmundson, G. J. G. (2004). Factor structure and psychometric properties of the Pain Anxiety Symptoms Scale-20 in a community physiotherapy clinic sample. *European Journal of Pain, 8*, 511-516.

Corlett, E. N., Eklund, J. A. E., Reilly, T., & Troup, J. D. G. (1987). Assessment of workload from measurements of stature. *Applied Ergonomics, 18*, 65-71.

Costa, L. da C. M., Maher, C. G., McAuley, J. H., Hancock, M. J., & Smeets, R. J. E. M. (in press). Self-efficacy is more important than fear of movement in mediating the relationship between pain and disability in chronic low back pain. *European Journal of Pain.*

Crawford, J. R., Henry, J. D., Crombie, C., & Taylor, E. P. (2001). Normative data for the HADS from a large non-clinical sample. *British Journal of Clinical Psychology, 40*, 429-434.

Creswell, C., & Chalder, T. (2001). Defensive coping styles in chronic fatigue syndrome. *Journal of Psychosomatic Research, 51*, 607-610.

Crombez, G., Vlaeyen, J. W. S., Heuts, P. H. T. G., & Lysens, R. (1999). Pain-related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability. *Pain, 80*, 329-339.

Crowne, D. P., & Marlowe, D. (1960). A new scale of social desirability independent of psychopathology. *Journal of Consulting Psychology, 24*, 349-354.

Dankaerts, W., O'Sullivan, P., Burnett, A., & Straker, L. (2006). Altered patterns of superficial trunk muscle activation during sitting in nonspecific chronic low back pain patients: Importance of subclassification. *Spine, 31*, 2017-2023.

Dankaerts, W., O'Sullivan, P. B., Burnett, A. F., Straker, L. M., & Danneels, L. A. (2004). Reliability of EMG measurements for trunk muscles during maximal and sub-maximal voluntary isometric contractions in healthy controls and CLBP patients. *Journal of Electromyography and Kinesiology, 14*, 333-342.

Danneels, L. A., Coorevits, P. L., Cools, A. M., Vanderstraeten, G. G., Cambier, D. C., Witvrouw, E. E., *et al.* (2002). Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain. *European Spine Journal, 11*, 13-19.

Davis, K. G., Marras, W. S., Heaney, C. A., Waters, T. R., & Gupta, P. (2002). The impact of mental processing and pacing on spine loading. *Spine, 27*, 2645-2653.

Dedering, Å., Németh, G., & Harms-Ringdahl, K. (1999). Correlation between electromyographic spectral changes and subjective assessment of lumbar muscle fatigue in subjects without pain from the lower back. *Clinical Biomechanics, 14*, 103-111.

DeGood, D. E., Stewart, W. R., & Adams, L. E. (1994). Paraspinal EMG and autonomic reactivity of patients with back pain and controls to personally relevant stress. *Perceptual and Motor Skills, 79*, 1399-1409.

De Luca, C. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics*, 13, 135-163.

Denison, E., Åsenlöf, P., & Lindberg, P. (2004). Self-efficacy, fear avoidance, and pain intensity as predictors of disability in subacute and chronic musculoskeletal pain patients in primary health care. *Pain, 111*, 245-252.

De Puky, P. (1935). The physiological oscillation of the length of the body. *Acta Orthop, 6*, 338-347.

Derakshan, N., Eysenck, M. W., & Myers, L. B. (2007). Emotional information processing in repressors: the vigilance-avoidance theory. *Cognition & Emotion, 21*, 1585-1614.

DeVocht, J. W., Pickar, J G., & Wilder, D. G. (2005). Spinal manipulation alters electromyographic activity of paraspinal muscles: a descriptive study. *Journal of Manipulative and Physiological Therapeutics, 28*, 465-471.

Dowzer, C. N., Reilly, T., & Cable, N. T. (1998). Effects of deep and shallow water running on spinal shrinkage. *British Journal of Sports Medicine, 32*, 44-48.

Dunlop, R. B., Adams, M. A., & Hutton, W. C. (1984). Disc space narrowing and the lumbar facet joints. *The Journal of Bone and Joint Surgery, 66*, 706-710.

Dworkin, R. H., Turk, D. C., Farrar, J. T., Haythornthwaite, J. A., Jensen, M. P., Katz, N. P., *et al.* (2005). Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. *Pain, 113*, 9-19.

Eklund, J. A. E., & Corlett, E. N. (1984). Shrinkage as a measure of the effect of load on the spine. *Spine, 9*, 189-194.

Elfant, E., Burns, J. W., & Zeichner, A. (2008). Repressive coping style and suppression of pain-related thoughts: effects on responses to acute pain induction. *Cognition & Emotion, 22*, 671-696.

Elfving, B., Dedering, A., & Németh, G. (2003). Lumbar muscle fatigue and recovery in patients with long-term low-back trouble – electromyography and health-related factors. *Clinical Biomechanics, 18*, 619-630.

Endler, N. S., & Parker, J. D. (1990). Multidimensional assessment of coping: a critical evaluation. *Journal of Personality and Social Psychology, 58*, 844-854.

Eysenck, M. W. (1997). *Anxiety and Cognition: A Unified Theory*. Hove: Psychology Press.

Eysenck, M. W. (2000). A cognitive approach to trait anxiety. *European Journal of Personality, 14*, 463-476.

Eysenck, M. W., & Derakshan, N. (1997). Cognitive biases for future negative events as a function of trait anxiety and social desirability. *Personality and Individual Differences, 22*, 597-605.

Ferguson, S. A., Marras, W. S., Burr, D. L., Davis, K. G., & Gupta, P. (2004). Differences in motor recruitment and resulting kinematics between low back pain patients and asymptomatic participants during lifting exertions. *Clinical Biomechanics, 19,* 992-999.

Ferreira, M. L., Ferreira, P. H., & Hodges, P. W. (2007). Changes in postural activity of the trunk muscles following spinal manipulative therapy. *Manual Therapy, 12,* 240-248.

Finneran, M. T., Mazanec, D., Marsolais, M. E., Marsolais, E. B., & Pease, W. S. (2003). Large-array surface electromyography in low back pain. *Spine, 28,* 1447-1454.

Flink, I. K., Boersma, K., & Linton, S. J. (2010). Catastrophizing moderates the effect of exposure in vivo for back pain patients with pain-related fear. *European Journal of Pain, 14,* 897-892.

Flor, H., Birbaumer, N., Schugens, M. M., & Lutzenberger, W. (1992). Symptom-specific psychophysiological responses in chronic pain patients. *Psychophysiology, 29,* 452-460.

Flor, H., Fürst, M., & Birbaumer, N. (1999). Deficient discrimination of EMG levels and overestimation of perceived tension in chronic pain patients. *Applied Psychophysiology and Biofeedback, 24,* 55-66.

Flor, H., Turk, D. C., & Birbaumer, N. (1985). Assessment of stress-related psychophysiological reactions in chronic back pain patients. *Journal of Consulting and Clinical Psychology, 53,* 354-364.

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

Foster, N. E., Thomas, E., Bishop, A., Dunn, K. M., & Main, C. J. (2010). Distinctiveness of psychological obstacles to recovery in low back pain patients in primary care. *Pain, 148,* 398-406.

Fowler, N. E., Rodacki, C. d. L., & Rodacki, A. L. (2005). Spinal shrinkage and recovery in women with and without low back pain. *Archives of Physical Medicine and Rehabilitation, 86,* 505-511.

Fowler, N. E., & Healey, E. (2008). Diurnal stature changes in individuals with and without chronic low back pain. *Journal of Bone and Joint Surgery Br, 90,* 221.

French, S. D., Cameron, M., Walker, B. F., Reggars, J. W., & Esterman, A. J. (2006). A Cochrane review of superficial heat or cold for low back pain. *Spine, 31,* 998-1006.

French, D. J., France, C. R., Vigneau, F., French, J. A., & Evans, R. T. (2007). Fear of movement/(re)injury in chronic pain: a psychometric assessment of the original English version of the Tampa scale for kinesiophobia (TSK). *Pain, 127,* 42-51.

Fryer, G., Morris, T., & Gibbons, P. (2004). Paraspinal muscles and intervertebral dysfunction: Part one. *Journal of Manipulative and Physiological Therapeutics, 27,* 267-274.

Furlan, A. D., Imamura, M., Dryden, T., & Irvin, E. (2009) Massage for low back pain: an updated systematic review within the framework of the Cochrane Back Review Group. *Spine, 34,* 1669-1684.

Garbutt, G., Boocock, M. G., Reilly, T., & Troup, J. D. G. (1990). Running speed and spinal shrinkage in runners with and without low back pain. *Medicine and Science in Sports and Exercise, 22,* 769-772.

Geisser, M. E., Haig, A. J., Wallbom, A. S., & Wiggert, E. A. (2004). Pain-related fear, lumbar flexion, and dynamic EMG among persons with chronic musculoskeletal low back pain. *Clinical Journal of Pain, 20,* 61-69.

Geisser, M. E., Ranavaya, M., Haig, A. J., Roth, R. S., Zucker, R., Ambroz, C., *et al.* (2005). A meta-analytic review of surface electromyography among persons with low back pain and normal, healthy controls. *The Journal of Pain, 6,* 711-726.

Gheldof, E. L. M., Crombez, G., Van den Bussche, E., Vinck, J., Van Nieuwenhuyse, A., Moens, *et al*. (2010). Pain-related fear predicts disability, but not pain severity: a path analytic approach of the fear-avoidance model. *European Journal of Pain, 14,* 870.e1–870.e9.

Glombiewski, J. A., Tersek, J., & Rief, W. (2008). Muscular reactivity and specificity in chronic back pain patients. *Psychosomatic Medicine, 70,* 125-131.

Goubert, L., Crombez, G., Eccleston, C., & Devulder, J. (2004). Distraction from chronic pain during a pain-inducing activity is associated with greater post-activity pain. *Pain, 110,* 220-227.

Granata, K. P., & Marras, W. S. (2000). Cost-benefit of muscle cocontraction in protecting against spinal instability. *Spine, 25,* 1398-1404.

Graven-Nielsen, T., Svensson, P., & Arendt-Nielsen, L. (2000). Effect of muscle pain on motor control: A human experimental approach. *Advances in Physiotherapy, 2,* 26-38.

Gregory, D. E., Brown, S. H. M., & Callaghan, J. P. (2008). Trunk muscle responses to suddenly applied loads: Do individuals who develop discomfort during prolonged standing respond differently? *Journal of Electromyography and Kinesiology, 18,* 495-502.

Grotle, M., Foster, N. E., Dunn, K. M., & Croft, P. (2010). Are prognostic indicators for poor outcome different for acute and chronic low back pain consulters in primary care? *Pain, 151,* 790-797.

Grotle, M., Vøllestad, N. K., & Brox, J. I. (2006). Clinical course and impact of fear-avoidance beliefs in low back pain. *Spine, 31,* 1038-1046.

Hägg, G. M. (1991). Static workload and occupational myalgia – a new explanation model. In Anderson, P. A., Hobart, D. J., Danoff, J. V. (Eds). *Electromyographical Kinesiology*. Amsterdam: Elsevier, pp141-144.

Hägg, G. M., & Åström, A. (1997). Load pattern and pressure pain threshold in the upper trapezius muscle and psychosocial factors in medical secretaries with and without shoulder/neck disorders. *International Archives of Occupational and Environmental Health, 69,* 423-432.

Hasan, Z. (1997). Invited commentary. *Physical Therapy, 77,* 142-143.

Hasenbring, M., Hallner, D., Klasen, B. (2001). Psychologische mechanismen im prozess der schmerzchronifizierung. *Schmerz, 15*, 442-447.

Hasenbring, M. I., Hallner, D., Rusu, A. C. (2009). Comment on: chronic pain: avoidance or endurance? By Petra Karsdorp and Johan Vlaeyen. *European Journal of Pain, 13,* 662-663.

Healey, E. L. (2005). *The effect of paraspinal muscle activity upon stature change post-exercise and its association with chronic low back pain*. Doctoral thesis, Manchester Metropolitan University, Cheshire, England.

Healey, E. L., Burden, A. M., McEwan, I. M., & Fowler, N. E. (2008). The impact of increasing paraspinal muscle activity on stature recovery in asymptomatic people. *Archives of Physical Medicine and Rehabilitation, 89*, 749-753.

Healey, E. L., Fowler, N. E., Burden, A. M., & McEwan, I. M. (2005a). Raised paraspinal muscle activity reduces rate of stature recovery after loaded exercise in individuals with chronic low back pain. *Archives of Physical Medicine and Rehabilitation, 86*, 710-715.

Healey, E. L., Fowler, N. E., Burden, A. M., & McEwan, I. M. (2005b). Repeatability of stature measurements in individuals with and without chronic low-back pain. *Ergonomics, 48*, 1613-1622.

Hermens, H. J., & Hutten, M. M. R. (2002). Muscle activation in chronic pain: its treatment using a new approach of myofeedback. *International Journal of Industrial Ergonomics, 30*, 325-336.

Heuer, F., Schmitt, H., Schmidt, H., Claes, L., & Wilke, H-J. (2007). Creep associated changes in intervertebral disc bulging obtained with a laser scanning device. *Clinical Biomechanics, 22*, 737-744.

Hides, J. A., Gilmore, C., Stanton, W., & Bohlscheid, E. (2008). Multifidus size and symmetry among chronic LBP and healthy asymptomatic subjects. *Manual Therapy, 13*, 43-49.

Hides, J. A., Jull, G. A., & Richardson, C. A. (2001). Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine, 26*, e243-e248.

Hides, J. A., Richardson, C. A., & Jull, G. A. (1996). Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine, 21*, 2763-2769.

Hides, J. A., Stokes, M. J. P., Saide, M., Jull, G. A., & Cooper, D. H. (1994). Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine, 19*, 165-172.

Hodges, P., Holm, A. K., Hansson, T., & Holm, S. (2006). Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine, 31*, 2926-2933.

Hodges, P., Moseley, G. L., Gabrielsson, A., & Gandevia, S. (2003). Experimental muscle pain changes feedforward postural responses of the trunk muscles. *Experimental Brain Research, 151*, 262-271.

Hodges, P. W., & Moseley, G. L. (2003). Pain and motor control of the lumbopelvic region: Effect and possible mechanisms. *Journal of Electromyography and Kinesiology, 13*, 361-370.

Hodges, P. W., & Richardson, C. A. (1996). Inefficient muscular stabilization of the lumbar spine associated with low back pain: A motor control evaluation of transversus abdominis. *Spine, 21*, 2640-2650.

Holewijn, M., & Heus, R. (1992). Effects of temperature on electromyogram and muscle function. *European Journal of Applied Physiology*, 65, 541-545.

Hoyt, W. H., Hunt, H. H., de Pauw, M. A., Bard, D., Shaffer, F., Passias, J. N., *et al.* (1981). Electromyographic assessment of chronic low-back pain syndrome. *Journal of the American Osteopathic Association, 80*, 728-730.

Hu, Y., Siu, S. H. F., Mak, J. N. F., Luk, K. D. K. (2010). Lumbar muscle electromyographic dynamic topography during flexion-extension. *Journal of Electromyography and Kinesiology, 20,* 246-255.

Huijnen, I. P. J., Verbunt, J. A., Peters, M., & Seelen, H. A. M. (2010). Is physical functioning influenced by activity-related pain prediction and fear of movement in patients with subacute low back pain? *European Journal of Pain, 14,* 661-666.

Huijnen, I. P. J., Verbunt, J. A., Roelofs, J., Goossens, M., & Peters, M. (2009). The disabling role of fluctuations in physical activity in patients with chronic low back pain. *European Journal of Pain, 13,* 1076-1079.

Hupli, M., Heinonen, R., & Vanharanta, H. (1997). Height changes among chronic low back pain patients during intense physical exercise. *Scandinavian Journal of Medicine & Science in Sports, 7,* 32-37.

Indahl, A., Kaigle, A. M., Reikerås, O., & Holm, S. H. (1997). Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine, 22,* 2834-2840.

Ioannou, M. C., Mogg, K., & Bradley, B. P. (2004). Vigilance for threat: effects of anxiety and defensiveness. *Personality and Individual Differences, 36,* 1879-1891.

Jamner, L. D., & Leigh, H. (1999). Repressive/defensive coping, endogenous opioids and health: how a life so perfect can make you sick. *Psychiatry Research, 85,* 17-31.

Jenkins, D. B. (Ed). (1998). *Hollinshead's Functional Anatomy of the Limbs and Back: 7th Edition*. Philadelphia: W. B. Saunders.

Jensen, M. P. (2003). The validity and reliability of pain measures for use in clinical trials in adults: review paper written for the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT) meeting, April 12–13, 2003. IMMPACT-II.

Johnson, T. P., & Fendrich, M. (2002). A validation of the Crowne-Marlowe Social Desirability Scale. Paper presented at: American Association for Public Opinion Research, St Petersburg, FL, May 16-19, 2002. Available from: http://www.srl.uic.edu/publist/Conference/crownemarlowe.pdf.

Johnston, M., Pollard, B., & Hennessey, P. (2000). Construct validation of the hospital anxiety and depression scale with clinical populations. *Journal of Psychosomatic Research, 48,* 579-584.

Jones, K. A., Smith, N. C., & Holmes, P. S. (2004). Anxiety symptom interpretation and performance prediction in high-anxious, low-anxious and repressor sport performers. *Anxiety, Stress & Coping, 17,* 187-199.

Jones, A. L., & Wolf, S. L. (1980). Treating chronic low back pain: EMG biofeedback training during movement. *Physical Therapy, 60,* 58-63.

Kanlayanaphotporn, R., Williams, M., Fulton, I., & Trott, P. (2002). Reliability of the vertical spinal creep response measured in sitting (asymptomatic and low-back pain subjects). *Ergonomics, 45,* 240-247.

Kanlayanaphotporn, R., Lam, L., Williams, M., Trott, P., & Fulton, I. (2001). Adolescent versus adult responses to vertical spinal loading. *Ergonomics, 44,* 1384-1391.

Karjalainen, K., Malmivaara, A., van Tulder, M., Roine, R., Jauhiainen, M., Hurri, H., *et al.* (2001). Multidisciplinary biopsychosocial rehabilitation for subacute low back pain in

working-age adults: a systematic review within the framework of the Cochrane Collection Back Review Group. *Spine, 26*, 262-269.

Käser, L., Mannion, A. F., Rhyner, A., Weber, E., Dvorak, J., & Müntener, M. (2001). Active therapy for chronic low back pain. Part 2. Effects on paraspinal muscle cross-sectional area, fiber type size, and distribution. *Spine, 26*, 909-919.

Keller, T. S., Holm, S. H., Hansson, T. H., & Spengler, D. M. (1990). The dependence of intervertebral disc mechanical properties on physiological conditions. *Spine, 15*, 751-761.

Keogh, E., McCracken, L. M., & Eccleston, C. (2006). Gender moderates the association between depression and disability in chronic pain patients. *European Journal of Pain, 10*, 413-422.

Key, J., Clift, A., Condie, F., & Harley, C. (2008). A model of movement dysfunction provides a classification system guiding diagnosis and therapeutic care in spinal pain and related musculoskeletal syndromes: A paradigm shift--part 1. *Journal of Bodywork and Movement Therapies, 12*, 7-21.

Kimura, S., Steinbach, G. C., Watenpaugh, D. E., & Hargens, A. R. (2001). Lumbar spine disc height and curvature responses to an axial load generated by a compression device compatible with magnetic resonance imaging. *Spine, 26*, 2596-2600.

Klinger, R., Matter, N., Kothe, R., Dahme, B., Hofmann, U. G., & Krug, F. (2010). Unconditioned and conditioned muscular responses in patients with chronic back pain and chronic tension-type headaches and in healthy controls. *Pain, 150*, 66-74.

Knost, B., Flor, H., Birbaumer, N., & Schugens, M. M. (1999). Learned maintenance of pain: muscle tension reduces central nervous system processing of painful stimulation in chronic and subchronic pain patients. *Psychophysiology, 36*, 755-764.

Koeller, W., Funke, F., & Hartmann, F. (1984). Biomechanical behavior of human intervertebral discs subjected to long lasting axial loading. *Biorheology, 21*, 675-686.

Koes, B., Assendelft, W. J. J., Van der Heijden, G. M. G., & Bouter, L. M. (1996). Spinal manipulation for low back pain: an updated systematic review of randomized clinical trials. *Spine, 21*, 2860-2871.

Kori, S. H., Miller, R. P., & Todd, D. D. (1990). Kinisophobia: a new view of chronic pain behaviour. *Pain Management, 3*, 35-43.

Koumantakis, G. A. (2006). Muscle activity and back pain. In L. Gifford (Ed.), *Topical issues in pain 5* (pp. 341-377). Falmouth: CNS Press.

Kramer, M., Ebert, V., Kinzl, L., Dehner, C., Elbel, M., & Hartwig, E. (2005). Surface electromyography of the paravertebral muscles in patients with chronic low back pain. *Archives of Physical Medicine and Rehabilitation, 86*, 31-36.

Krause, K-H., Magyarosy, I., Gall, H., Ernst, E., Pongratz, D., & Schoeps, P. (2001). Effects of heat and cold application on turns and amplitude in surface EMG. *Electromyography and clinical Neurophysiology*, 41, 67-70.

Kravitz, E., Moore, M. E., & Glaros, A. (1981). Paralumbar muscle activity in chronic low back pain. *Archives of Physical Medicine and Rehabilitation, 62*, 172-176.

Krekoukias, G., Petty, N. J., & Cheek, L. (2009). Comparison of surface electromyographic activity of erector spinae before and after the application of central

posteroanterior mobilisation on the lumbar spine. *Journal of Electromyography and Kinesiology, 19*, 39-45.

Kuiper, J. I., van Dieën, J. H., Everts, V., Verbeek, J. H. A. M., & Frings-Dresen, M. H. W. (2004). Associations between serum markers of collagen metabolism and spinal shrinkage. *Clinical Biomechanics, 19*, 209-212.

Lackner, J. M., & Carosella, A. M. (1999). The relative influence of perceived pain control, anxiety, and functional self-efficacy on spinal function among patients with chronic low back pain. *Spine, 24*, 2254-2261.

Lalanne, K., Lafond, D., & Descarreaux, M. (2009). Modulation of the flexion-relaxation response by spinal manipulative therapy: a control group study. *Journal of Manipulative and Physiological Therapeutics, 32*, 203-209.

Lamoth, C. J. C., Daffertshofer, A., Meijer, O. G., Moseley, G. L., Wuisman, P. I. J. M., & Beek, P. J. (2004). Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking. *Clinical Biomechanics, 19*, 551-563.

Larivière, C., Arsenault, A. B., Gravel, D., Gagnon, D., & Loisel, P. (2003). Surface electromyography assessment of back muscle intrinsic properties. *Journal of Electromyography and Kinesiology, 13*, 305-318.

Lazarus, R. S., & Folkman, S. (1984). *Stress, Appraisal, and Coping*. Springer: New York.

Leeuw, M., Goossens, M. E. J. B., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. S. (2007a). The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *Journal of Behavioral Medicine, 30*, 77-94.

Leeuw, M., Houben, R. M. A., Severeijns, R., Picavet, S. J., Schouten, E. G. W., & Vlaeyen, J. W. S. (2007b). Pain-related fear in low back pain: a prospective study in the general population. *European Journal of Pain, 11*, 256-266.

Lehman, G. J. (2002). Clinical considerations in the use of surface electromyography: three experimental studies. *Journal of Manipulative and Physiological Therapeutics, 25*, 293-299.

Lehmann, J. F., & Lateur, J. B. (1990). Therapeutic heat. In Lehmann J. F. (ed): Therapeutic Heat and Cold, 4th edition. Baltimore: Williams and Wilkins.

Leinonen, V., Kankaanpää, M., Luukkonen, M., Kansanen, M., Hänninen, O., Airaksinen, O., *et al.* (2003). Lumbar paraspinal muscle function, perception of lumbar position, and postural control in disc herniation-related back pain. *Spine, 28*, 842-848.

Leivseth, G., & Drerup, B. (1997). Spinal shrinkage during work in a sitting posture compared to work in a standing posture. *Clinical Biomechanics, 12*, 409-418.

Leonhardt, C., Lehr, D., Chenot, J-F, Keller, S., Luckmann, J., Basler, H-D. *et al.* (2009). Are fear–avoidance beliefs in low back pain patients a risk factor for low physical activity or vice versa? A cross-lagged panel analysis. *GMS Psychosocial Medicine, 6.*

Lethem, J., Slade, P. D., Troup, J. D. G., & Bentley, G. (1983). Outline of a fear-avoidance model of exaggerated pain perception – I. *Behaviour Research and Therapy, 21*, 401-408.

Linton, S. J. (2000). A review of psychological risk factors in back and neck pain. *Spine, 25*, 1148-1156.

Linton, S. J. (2005). Do psychological factors increase the risk for back pain in the general population in both a cross-sectional and prospective analysis? *European Journal of Pain, 9*, 355-361.

Lohnberg, J. A. (2007). A review of outcome studies on cognitive-behavioral therapy for reducing fear-avoidance beliefs among individuals with chronic pain. *Journal of Clinical Psychology in Medical Settings, 14*, 113-122.

Low, J., & Reed, A. (2000). *Electrotherapy explained: principles and practice*. (3rd ed.). Oxford: Butterworth Heinemann.

Lund, J. P., Donga, R., Widmer, C. G., & Stohler, C. S. (1991). The pain-adaptation model: A discussion of the relationship between chronic musculoskeletal pain and motor activity. *Canadian Journal of Physiology and Pharmacology, 69*, 683-694.

Lundberg, U., Forsman, M., Zachau, G., Eklöf, M., Palmerud, G., Melin, B., *et al.* (2002). Effects of experimentally induced mental and physical stress on motor unit recruitment in the trapezius muscle. *Work & Stress, 16*, 166-178.

Luoto, S., Aalto, H., Taimela, S., Heikki, H., Ilmari, P., & Hannu, A. (1998). One-footed and externally disturbed two-footed postural control in patients with chronic low back pain and healthy control subjects: A controlled study with follow-up. *Spine, 23*, 2081-2089.

Main, C. J., & Watson, P. J. (1999). Psychological aspects of pain. *Manual Therapy, 4*, 203-215.

Mak, J. N. F., Hu, Y., Cheng, A. C. S., Kwok, H. Y., Chen, Y. H., & Luk, K. D. K. (2010). Flexion-relaxation ratio in sitting. *Spine, 35*, 1532-1538.

Maniadakis, N., & Gray, A. (2000). The economic burden of back pain in the UK. *Pain, 84*, 95-103.

Mannion, A. F., Dolan, P., & Adams, M. (1996). Psychological questionnaires: do "abnormal" scores precede or follow first-time low back pain? *Spine, 21*, 2603-2611.

Mannion, A. F., Dumas, G. A., Stevenson, J. M. & Cooper, R. G. (1998). The influence of muscle fiber size and type distribution on electromyography measures of back muscle fatigability. *Spine, 23*, 576-584.

Mannion, A. F., Junge, A., Taimela, S., Müntener, M., Lorenzo, K., & Dvorak, J. (2001a). Active therapy for chronic low back pain. Part 3. Factors influencing self-rated disability and its change following therapy. *Spine, 26*, 920-929.

Mannion, A. F., Müntener, M., Taimela, S., Dvorak, J. (1999). A randomised clinical trial of three active therapies for chronic low back pain. *Spine, 24*, 2435-2448.

Mannion, A. F., Taimela, S., Müntener, M., & Dvorak, J. (2001b). Active therapy for chronic low back pain: Part 1. Effects on back muscle activation, fatigability, and strength. *Spine, 26*, 897-908.

Mannion, A. F., Weber, B. R., Dvorak, J., Grob, D., & Müntener, M. (1997). Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. *Journal of Orthopaedic Research, 15*, 881-887.

Marras, W. S., Davis, K. G., Ferguson, S. A., Lucas, B. R., & Gupta, P. (2001). Spine loading characteristics of patients with low back pain compared with asymptomatic individuals. *Spine, 26*, 2566-2574.

Marras, W. S., Davis, K. G., Heaney, C. A., Maronitis, A. B., & Allread, W. G. (2000). The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. *Spine, 25*, 3045-3054.

Marras, W. S., Ferguson, S. A., Burr, D., Davis, K. G., & Gupta, P. (2004). Spine loading in patients with low back pain during asymmetric lifting exertions. *The Spine Journal, 4*, 64-75.

Marshall, P., & Murphy, B. (2009). Delayed abdominal muscle onsets and self-report measures of pain and disability in chronic low back pain. *Journal of Electromyography and Kinesiology, 20*, 833-839.

Mayer, J. M., Mooney, V., Matheson, L. N., Erasala, G.N., Verna, J. L., Udermann, B. E., *et al*. (2006). Continuous low-level heat wrap therapy for the prevention and early phase treatment of delayed-onset muscle soreness of the low back: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation, 87*, 1310-1317.

Mayer, J. M., Ralph, L., Look, M., Erasala, G.N., Verna, J. L., Matheson, L. N., *et al*. (2005). Treating acute low back pain with continuous low-level heat wrap therapy and/or exercise: a randomized controlled trial. *The Spine Journal, 5*, 395-403.

Mayer, T. G., Neblett, R., Brede, E., & Gatchel, R. J. (2009). The quantified lumbar flexion-relaxation phenomenon is a useful measurement of improvement in a functional restoration program. *Spine, 34*, 2458-2465.

McCracken, L. M., Gross, R. T., & Eccleston, C. (2002). Multimethod assessment of treatment process in chronic low back pain: comparison of reported pain-related anxiety with directly measured physical capacity. *Behaviour Research & Therapy, 40*, 585-594.

McCracken, L. M., & Samuel, V. M. (2007). The role of avoidance, pacing, and other activity patterns in chronic pain. *Pain, 130*, 119-125.

McCracken, L. M., & Turk, D. C. (2002). Behavioral and cognitive-behavioral treatment for chronic pain. *Spine, 27*, 2564-2573.

McCracken, L. M., Zayfert, C., & Gross, R. T. (1992). The Pain Anxiety Symptoms Scale: development and validation of a scale to measure fear of pain. *Pain, 50*, 67-73.

McGill, S. M., Grenier, S., Kavcic, N., & Cholewicki, J. (2003). Coordination of muscle activity to assure stability of the lumbar spine. *Journal of Electromyography and Kinesiology, 13*, 353-359.

McGill, S. M., Hughson, R. L., & Parks, K. (2000). Lumbar erector spinae oxygenation during prolonged contractions: Implications for prolonged work. *Ergonomics, 43*, 486-493.

McGill, S. M., & Kippers, V. (1994). Transfer of loads between lumbar tissues during the flexion-relaxation phenomenon. *Spine, 19*, 2190-2196.

McGill, S. M., van Wijk, M. J., Axler, C. T., & Gletsu, M. (1996). Studies of spinal shrinkage to evaluate low-back loading in the workplace. *Ergonomics, 39*, 92-102.

Melzack, R., & Wall, P. D. (1965). Pain mechanisms: a new theory. *Science, 150*, 971-979.

Middleditch, A., & Oliver, J. (2005). *Functional Anatomy of the Spine*: *Second Edition*. Elsevier: Oxford.

Mito, K., Kitahara, S., Tamura, T., Kaneko, K., Sakamoto, K., & Shimizu, Y. (2007). Effect of skin temperature on RMS amplitude of electromyogram and mechanomyogram

during voluntary isometric contraction. *Electromyography and Clinical Neurophysiology*, 47, 153-160.

Mohseni-Bandpei, M. A., Watson, M. J., & Richardson, B. (2000). Application of surface electromyography in the assessment of low back pain: a literature review. *Physical Therapy Reviews, 5*, 93-105.

Moseley, G. L. (2004). Impaired trunk muscle function in sub-acute neck pain: Etiologic in the subsequent development of low back pain? *Manual Therapy, 9*, 157-163.

Moseley, G. L., Brhyn, L., Ilowiecki, M., Solstad, K., & Hodges, P. W. (2003). The threat of predictable and unpredictable pain: Differential effects on central nervous system processing? *Australian Journal of Physiotherapy, 49*, 263-267.

Moseley, G. L., Nicholas, M. K., & Hodges, P. (2004a). Pain differs from non-painful attention-demanding or stressful tasks in its effect on postural control patterns of trunk muscles. *Experimental Brain Research, 156*, 64-71.

Moseley, G. L., Nicholas, M. K., & Hodges, P. W. (2004b). Does anticipation of back pain predispose to back trouble? *Brain, 127*, 2339-2347.

Mulholland, R. C. (2008). The myth of lumbar instability: The importance of abnormal loading as a cause of low back pain. *European Spine Journal, 17*, 619-625.

Murray, G. M., & Peck, C. C. (2007). Orofacial pain and jaw muscle activity: A new model. *Journal of Orofacial Pain, 21*, 263-278.

Myers, L. B. (2010). The importance of the repressive coping style: findings from 30 years of research. *Anxiety, Stress & Coping, 23*, 3-17.

Nadler, S. F., Steiner, D. J., Erasala, G. N., Hengehold, D. A., Abeln, S. B., & Weingand, K. W. (2003a). Continuous low-level heatwrap therapy for treating acute nonspecific low back pain. *Archives of Physical Medicine and Rehabilitation, 84*, 329-334.

Nadler, S. F., Steiner, D. J., Erasala, G. N., Hengehold, D. A., Hinkle, R. T., Goodale, M. B., *et al.* (2002). Continuous low-level heat wrap therapy provides more efficacy than ibuprofen and acetaminophen for acute low back pain. *Spine, 27*, 1012-1017.

Nadler, S. F., Steiner, D. J., Petty, S. R., Erasala, G. N., Hengehold, D. A., & Weingand, K. W. (2003b). Overnight use of continuous low-level heatwrap therapy for relief of low back pain. *Archives of Physical Medicine and Rehabilitation, 84*, 335-342.

Natarajan, R. N., & Andersson, G. B. J. (1999). The influence of lumbar disc height and cross-sectional area on the mechanical response of the disc to physiologic loading. *Spine, 24*, 1873-1881.

National Institute for Health and Clinical Excellence (NICE). (2009, May). *Low back pain: early management of persistent non-specific low back pain*. Retrieved November 12, 2009 from http://guidance.nice.org.uk/CG88

NCBI (National Center for Biotechnology) Bookshelf (2001). Images retrieved May14, 2011 from http://www.ncbi.nlm.nih.gov/books.

Neblett, R., Mayer, T. G., Gatchel, R. J., Keeley, J., Proctor, T., & Anagnostis, C. (2003). Quantifying the lumbar flexion-relaxation phenomenon: Theory, normative data, and clinical applications. *Spine, 28*, 1435-1446.

Nelson-Wong, E., Gregory, D. E., Winter, D. A., & Callaghan, J. P. (2008). Gluteus medius muscle activation patterns as a predictor of low back pain during standing. *Clinical Biomechanics, 23*, 545-553.

Nelson-Wong, E., & Callaghan, J. P. (2010). Is muscle co-activation a predisposing factor for low back pain development during standing? A multifactorial approach for early identification of at-risk individuals. *Journal of Electromyography and Kinesiology, 20,* 256-263.

Nieto, R., Miró, J., & Huguet, A. (2009). The fear-avoidance model in whiplash injuries. *European Journal of Pain, 13*, 518-523.

Nijs, J., Van Houdenhove, B., & Oostendorp, R. A. B. (2010). Recognition of central sensitization in patients with musculoskeletal pain: application of pain neurophysiology in manual therapy practice. *Manual Therapy, 15*, 135-141.

Nuhr, M., Hoerauf, K., Bertalanffy, A., Bertalanffy, P., Frickey, N., Gore, *et al*. (2004). Active warming during emergency transport relieves acute low back pain. *Spine, 29,* 1499-1503.

Osman, A., Barrios, F. X., Gutierrez, P. M., Kopper, B. A., Merrifield, T., & Grittmann, L. (2000). The Pain Catastrophizing Scale: further psychometric evaluation with adult samples. *Journal of Behavioral Medicine, 23*, 351-365.

Osman, A., Barrios, F. X., Kopper, B. A., Hauptmann, W., Jones, J., & O'Neill, E. (1997). Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *Journal of Behavioral Medicine, 20*, 589-605.

O'Sullivan, P. B., Burnett, A., Floyd, A. N., Gadsdon, K., Logiudice, J., Miller, D., *et al.* (2003). Lumbar repositioning deficit in a specific low back pain population. *Spine, 28,* 1074-1079.

O'Sullivan, P. B., Twomey, L. T., & Allison, G. T. (1997). Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine, 22*, 2959-2967.

Pallant, J. F. & Bailey, C. M. (2005). Assessment of the structure of the Hospital Anxiety and Depression Scale in musculoskeletal patients. *Health and Quality of Life Outcomes, 3.*

Panjabi, M. (1992a). The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders, 5*, 383-389.

Panjabi, M. (1992b). The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. *Journal of Spinal Disorders, 5*, 390-397.

Panjabi, M. M. (2003). Clinical spinal instability and low back pain. *Journal of Electromyography and Kinesiology, 13*, 371-379.

Panjabi, M. M. (2006). A hypothesis of chronic back pain: Ligament subfailure injuries lead to muscle control dysfunction. *European Spine Journal, 15*, 668-676.

Park, C. O. (1997). Diurnal variation in lumbar MRI. Correlation between signal intensity, disc height, and disc bulge. *Yonsei Medical Journal, 38*, 8-18.

Peach, J. P., & McGill, S. M. (1998). Classification of low back pain with the use of spectral electromyogram parameters. *Spine, 23*, 1117-1123.

Pergolizzi, J. V., Apfel, C., Florio, F., Richmond, C. (2008). Decreased lower back pain after non-invasive spinal decompression may be due to restored disc height. *Archives of Physical Medicine and Rehabilitation, 89,* E34.

Petrofsky, J., & Laymon, M. (2005). The relationship between muscle temperature, MUAP conduction velocity and the amplitude and frequency components of the surface EMG during isometric contractions. *Basic and Applied Myology*, 15, 61-74.

Phipps, S., & Steele, R. (2002). Repressive adaptive style in children with chronic illness. *Psychosomatic Medicine*, 64, 34-42.

Picavet, H. S. J., Vlaeyen, J. W. S., & Schouten, J. S. A. G. (2002). Pain catastrophizing and kinesiophobia: predictors of chronic low back pain. *American Journal of Epidemiology, 156*, 1028-1034.

Pincus, T., Burton, A. K., Vogel, S., & Field, A. P. (2002). A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine, 27*, E109-E120.

Pincus, T., Vogel, S., Burton, A. K., Santos, R., & Field, A. P. (2006). Fear avoidance and prognosis in back pain: A systematic review and synthesis of current evidence. *Arthritis & Rheumatism, 54*, 3999-4010.

Prasertsri, N., Holden, J., Keefe, F.J., & Wilkie, D. J. (in press). Repressive coping style: relationships with depression, pain and pain coping strategies in lung cancer out patients. *Lung Cancer.*

Primal Pictures (2009). Images retrieved May 14, 2011 from http://www.anatomy.tv

Puntumetakul, R., Trott, P., Williams, M., & Fulton, I. (2008). Effect of time of day on the vertical spinal creep response. *Applied Ergonomics, 40*, 33-38.

Radebold, A., Cholewicki, J., Panjabi, M. M., & Patel, T. (2000). Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine, 25*, 947-954.

Rainville, J., Hartigan, C., Martinez, E., Limke, J., Jouve, C., & Finno, M. (2004). Exercise as a treatment for chronic low back pain. *The Spine Journal, 4,* 106-115

Reeves, N. P., Cholewicki, J., & Milner, T. E. (2005). Muscle reflex classification of low-back pain. *Journal of Electromyography and Kinesiology, 15*, 53-60.

Reilly, T., Boocock, M. G., Garbutt, G., & Troup, J. D. G. (1988). Shrinkage in total body length: its measurement and application. *Humanbiologica Budapestinensis, 18,* 183-191.

Reynolds, W. M. (1982). Development of reliable and valid short forms of the Marlowe-Crowne Social Desirability Scale. *Journal of Clinical Psychology, 38*, 119-125.

Richards, M. M., & Steele, R. G. (2007). Children's self-reported coping strategies: the role of defensiveness and repressive adaptation. *Anxiety, Stress, and Coping, 20*, 209-222.

Rissén, D., Melin, B., Sandsjö, L., Dohns, I., & Lundberg, U. (2000). Surface EMG and psychophysiological stress reactions in women during repetitive work. *European Journal of Applied Physiology, 83*, 215-222.

Roberts, N., Hogg, D., Whitehouse, G. H., & Dangerfield, P. (1998). Quantitative analysis of diurnal variation in volume and water content of lumbar intervertebral discs. *Clinical Anatomy, 11,* 1-8.

Rodacki, A. L. F., Fowler, N. E., Provensi, C. L. G., Rodacki, C. L.N., & Dezan, V. H. (2005). Body mass as a factor in stature change. *Clinical Biomechanics, 20,* 799-805.

Rodacki, A. L. F., Weidle, C. M., Fowler, N. E., Rodacki, C. L.N., & Persch, L. N. (2007). Changes in stature during and after spinal traction in young male subjects. *Brazilian Journal of Physical Therapy, 11,* 63-71.

Rodacki, C. L., Fowler, N. E., Rodacki, A. L., & Birch, K. (2001). Repeatability of measurement in determining stature in sitting and standing postures. *Ergonomics, 44,* 1076-1085.

Rodacki, C. L., Fowler, N. E., Rodacki, A. L., & Birch, K. (2003). Stature loss and recovery in pregnant women with and without low back pain. *Archives of Physical Medicine and Rehabilitation, 84,* 507-512.

Roelofs, J., McCracken, L., Peters, M. L., Crombez, G., van Breukelen, G., & Vlaeyen, J. W. S. (2004). Psychometric evaluation of the Pain Anxiety Symptoms Scale (PASS) in chronic pain patients. *Journal of Behavioral Medicine, 27,* 167-183.

Rohlmann, A., Claes, L. E., Bergmann, G., Graichen, F., Neef, P., & Wilke, H-J. (2001). Comparison of intradiscal pressures and spinal fixator loads for different body positions and exercises. *Ergonomics, 44,* 781-794.

Roland, M. (1986). A critical review of the evidence for a pain-spasm-pain cycle in spinal disorders. *Clinical Biomechanics, 1,* 102-109.

Roland, M., & Fairbank, J. (2000). The Roland-Morris Disability Questionnaire and the Oswestry Disability Questionnaire. *Spine, 25,* 3115-3124.

Roland, M., & Morris, R. (1983). A study of the natural history of low back pain: part 1. Development of a reliable and sensitive measure of disability in low-back pain. *Spine, 8,* 141-144.

Roy, S. H., De Luca, C. J., & Casavant, D. A. (1989). Lumbar muscle fatigue and chronic low back pain. *Spine, 14,* 992-1001.

Rutledge, T., Linke, S. E., Krantz, D. S., Johnson, B. D., Bittner, V., Eastwood, J-A. (2009). Comorbid depression and anxiety symptoms as predictors of cardiovascular events: results from the NHLBI-sponsored Women's Ischemia Syndrome Evaluation (WISE) study. *Psychosomatic Medicine, 71,* 958-964.

Ryan, C. G., Gray, H. G., Newton, M., & Granat, M. H. (2010). The relationship between psychological distress and free-living physical activity in individuals with chronic low back pain, *Manual Therapy, 15,* 185-189.

Sakai, Y., Matsuyama, Y., Nakamura, H., Katayama, Y., Imagama, S., Ito, Z., *et al.* (2008). The effect of muscle relaxant on the paraspinal muscle blood flow: A randomized controlled trial in patients with chronic low back pain. *Spine, 33,* 581-587.

Sánchez-Zuriaga, D., Adams, M. A., & Dolan, P. (2010). Is activation of the back muscles impaired by creep or muscle fatigue? *Spine, 35,* 517-525.

Sato, K., Kikuchi, S., & Yonezawa, T. (1999). *In vivo* intradiscal pressure measurement in healthy individuals and in patients with ongoing back problems. *Spine, 24,* 2468-2474.

Schmidt, A. J. M. (1985). Cognitive factors in the performance level of chronic low back pain patients. *Journal of Psychosomatic Research, 29,* 183-189.

Sherman, R. A. (1985). Relationships between strength of low back muscle contraction and reported intensity of chronic low back pain. *American Journal of Physical Medicine, 64*, 190-200.

Sieben, J. M., Vlaeyen, J. W. S., Portegijs, P. J. M., Verbunt, J. A., van Riet-Rutgers, S., Kester, A. D. M., *et al.* (2005). A longitudinal study on the predictive validity of the fear-avoidance model in low back pain. *Pain, 117*, 162-170.

Sihvonen, T., Huttunen, M., Makkonen, M., & Airaksinen, O. (1998). Functional changes in back muscle activity correlate with pain intensity and prediction of low back pain during pregnancy. *Archives of Physical Medicine and Rehabilitation, 79*, 1210-1212.

Sihvonen, T., Partanen, J., Hänninen, O., & Soimakallio, S. (1991). Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Archives of Physical Medicine and Rehabilitation, 72*, 1080-1087.

Silfies, S. P., Mehta, R., Smith, S. S., & Karduna, A. R. (2009). Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. *Archives of Physical Medicine and Rehabilitation, 90*, 1159-1169.

Sluka, K. A., Christy, M. R., Peterson, W. L., Rudd, S. L., & Troy, S. M. (1999). Reduction of pain-related behaviors with either cold or heat treatment in an animal model of acute arthritis. *Archives of Physical Medicine and Rehabilitation, 80*, 313-317.

Smeathers, J. E. (1984). Some time dependent properties of the intervertebral joint when under compression. *Engineering in Medicine, 13*, 83-87.

Smeets, R. J., Van Geel, A. C., Kester, A. D., & Knotterus, J. A. (2007). Physical capacity tasks in chronic low back pain: what is the contributing role of cardiovascular capacity, pain and psychological factors? *Disability and Rehabilitation, 29*, 577-586.

Smeets, R. J., Van Geel, K. D., & Verbunt, J. A. (2009). Is the fear avoidance model associated with the reduced level of aerobic fitness in patients with chronic low back pain? *Archives of Physical Medicine and Rehabilitation, 90*, 109-117.

Smeets, R. J. E. M., Vlaeyen, J. W. S., Kester, A. D. M., & Knotterus, J. A. (2006). Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-behavioural treatment in chronic low back pain. *The Journal of Pain, 7*, 261-271.

Solomonow, M., Zhou, B-H., Harris, M., Lu, Y., & Baratta, R. V. (1998). The ligamento-muscular stabilizing system of the spine. *Spine, 23*, 2552-2562.

SpineUniverse (2010). Image retrieved January 10, 2011 from http://www.spineuniverse.com/ anatomy/spinal-ligaments-tendons

Spinhoven, P., ter Kuile, M., Kole-Snijders, A. M. J., Hutten Mansfield, M., den Ouden, D-J., Vlaeyen, J. W. S. (2004). Catastrophizing and internal pain control as mediators of outcome in the multidisciplinary treatment of chronic low back pain. *European Journal of Pain, 8*, 211-219.

Strahan, R., & Gerbasi, K. C. (1972). Short, homogenous versions of the Marlow *[sic]*-Crowne Social Desirability Scale. *Journal of Clinical Psychology, 28*, 191-193.

Sullivan, M. J. L., Bishop, S. R., & Pivik, J. (1995). The Pain Catastrophizing Scale: development and validation. *Psychological Assessment, 7*, 524-532.

Sullivan, M. J. L., Stanish, W., Waite, H., Sullivan, M., & Tripp, D. A. (1998). Catastrophizing, pain, and disability in patients with soft-tissue injuries. *Pain, 77*, 253-260.

Surawy, C., Hackmann, A., Hawton, K., & Sharpe, M. (1995). Chronic fatigue syndrome: a cognitive approach. *Behaviour Research and Therapy, 33*, 535-544.

Swinkels-Meewisse, I. E. J., Roelofs, J., Oostendorp, R. A. B., Verbeek, A. L. M., & Vlaeyen, J. W. S. (2006a). Acute low back pain: pain-related fear and pain catastrophizing influence physical performance and perceived disability. *Pain, 120*, 36-43.

Swinkels-Meewisse, I. E. J., Roelofs, J., Schouten, E. G. W., Verbeek, A. L. M., Oostendorp, R. A. B., & Vlaeyen, J. W. S. (2006b). Fear of movement/(re)injury predicting chronic disabling low back pain: a prospective inception cohort study. *Spine, 31*, 658-664.

Taimela, S., Kankaanpää, M., & Luoto, S. (1999). The effect of lumbar fatigue on the ability to sense a change in lumbar position: A controlled study. *Spine, 24*, 1322-1327.

Thomas, J. S., & France, C. R. (2007). Pain-related fear is associated with avoidance of spinal motion during recovery from low back pain. *Spine, 32*, E460-E466.

Thomas, J. S., & France, C. R. (2008). The relationship between pain-related fear and lumbar flexion during natural recovery from low back pain. *European Spine Journal, 17*, 97-103.

Thomas, J. S., France, C. R., Sha, D. H., Vander Wiele, N. (2008). The influence of pain-related fear on peak muscle activity and force generation during maximal isometric trunk exertions. *Spine, 33*, E342-E348.

Travell, J., Rinzler, S., & Herman, M. (1942). Pain and disability of the shoulder and arm. *Journal of American Medical Association, 120*, 417-422.

Troup, J. D. G., Reilly, T., Eklund, J. A. E., & Leatt, P. (1985). Changes in stature with spinal loading and their relation to the perception of exertion or discomfort. *Stress Medicine, 1*, 303-307.

Tsao, H., Galea, M. P., & Hodges, P. W. (2008). Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. *Brain, 131*, 2161-2171.

Tsao, H., Galea, M. P., & Hodges, P. W. (2010). Driving plasticity in the motor cortex in recurrent low back pain. *European Journal of Pain, 14*, 832-839.

Tsuboi, T., Satou, T., Egawa, K., Izumi, Y., & Miyazaki, M. (1994). Spectral analysis of electromyogram in lumbar muscles: fatigue induced endurance contraction. *European Journal of Applied Physiology, 69*,361-366.

Turner, J. A., Fulton-Kehoe, D., Franklin, G., Wickizer, T. M., & Wu, R. (2003). Comparison of the Roland-Morris Disability Questionnaire and generic health status measures: a population-based study of workers' compensation back injury claimants. *Spine, 28*, 1061-1067.

Tyrrell, A. R., Reilly, T., & Troup, J. D. G. (1985). Circadian variation in stature and the effects of spinal loading. *Spine, 10*, 161-164.

Urban, J. P. G., & Roberts, S. (2003). Degeneration of the intervertebral disc. *Arthritis Research & Therapy, 5*, 120-130.

Van Damme, S., Crombez, G., & Eccleston, C. (2004). Disengagement from pain: the role of catastrophic thinking about pain. *Pain, 107*, 70-76.

van der Hulst, M., Vollenbroek-Hutten, M. M., Rietman, J. S., & Hermens, H. J. (2010a). Lumbar and abdominal muscle activity during walking in subjects with chronic low back pain: support of the "guarding" hypothesis? *Journal of Electromyography and Kinesiology, 20*, 31-38.

van der Hulst, M., Vollenbroek-Hutten, M. M., Schreurs, K.M., Rietman, J. S., & Hermens, H. J. (2010b). Relationships between coping strategies and lumbar muscle activity in subjects with chronic low back pain. *European Journal of Pain, 14*, 640-647.

van Deursen, L. L., van Deursen, D. L., Snijders, C. J., & Wilke, H. J. (2005). Relationship between everyday activities and spinal shrinkage. *Clinical Biomechanics, 20*, 547-550.

van Dieën, J. H., Cholewicki, J., & Radebold, A. (2003a). Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine, 28*, 834-841.

van Dieën, J. H., Selen, L. P. J., & Cholewicki, J. (2003b). Trunk muscle activation in low-back pain patients, an analysis of the literature. *Journal of Electromyography and Kinesiology, 13*, 333-351.

van Dieën, J. H., & Toussaint, H. M. (1993). Spinal shrinkage as a parameter of functional load. *Spine, 18*, 1504-1514.

van Tulder, M. W., Ostelo, R., Vlaeyen, J. W. S., Linton, S. J., Morley, S. J., & Assendelft, W. J. J. (2000). Behavioral treatment for chronic low back pain: a systematic review within the framework of the Cochrane Collection, *Spine, 26*, 270-281.

van Tulder, M. W., Touray, T., Furlan, A. D., Solway, S., & Bouter, L. M. (2003). Muscle relaxants for non-specific low back pain: a systematic review within the framework of the Cochrane Collection, *Spine, 28*, 1978-1992.

Verbunt, J. A., Seelen, H. A., Vlaeyen, J. W., Bousema, E. J., van der Heijden, G. J., Heuts, P. H., *et al*. (2005a). Pain-related factors contributing to muscle inhibition in patients with chronic low back pain: an experimental investigation based on superimposed electrical stimulation. *The Clinical Journal of Pain, 21*, 232-240.

Verbunt, J. A., Sieben, J. M., Seelen, A. M., Vlaeyen, J. W. S., Bousema, E. J., van der Heijden, G. J., *et al*. (2005b). Decline in physical activity, disability and pain-related fear in sub-acute low back pain. *European Journal of Pain, 9*, 417-425.

Verbunt, J. A., Seelen, H. A., Vlaeyen, J. W., van der Heijden, G. J., Heuts, P. H., Pons, K., *et al*. (2003a). Disuse and deconditioning in chronic low back pain: concepts and hypotheses on contributing mechanisms. *European Journal of Pain, 7*, 9-21.

Verbunt, J. A., Seelen, H. A., Vlaeyen, J. W., van der Heijden, G. J., & Knottnerus, J. A. (2003b). Fear of injury and physical deconditioning in patients with chronic low back pain. *Archives of Physical Medicine and Rehabilitation, 84*, 1227-1232.

Vlaeyen, J. W. S., Crombez, G., Linton, S. (2009). The fear-avoidance model of pain: we are not there yet. Comment on Wideman et al. "A prospective sequential analysis of the fear-avoidance model of pain" [Pain, 2009] and Nicholas "First things first: reduction in catastrophizing before fear of movement" [Pain, 2009]. *Pain, 146*, 222-223.

Vlaeyen, J. W. S., de Jong, J., Geilen, M., Heuts, P. H. T. G., & van Breukelen, G., (2002). The treatment of fear of movement/(re)injury in chronic low back pain: further evidence on the effectiveness of exposure in vivo. *The Clinical Journal of Pain, 18*, 251-261.

Vlaeyen, J. W. S., Kole-Snijders, A. M. J., Boeren, R. G. B., & van Eek, H. (1995). Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. *Pain, 62,* 363-372.

Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain, 85,* 317-332.

Vlaeyen, J. W. S., Seelen, H. A. M., Peters, M. L., de Jong, P., Aretz, E., Beisiegel, E., *et al.* (1999). Fear of movement/(re)injury and muscular reactivity in chronic low back pain patients: an experimental investigation. *Pain, 82,* 297-304.

Vollenbroek-Hutten, M., Hermens, H., Voerman, G., Sandsjö, L., & Kadefors, R. (2006). Are changes in pain induced by myofeedback training related to changes in muscle activation patterns in patients with work-related myalgia? *European Journal of Applied Physiology, 96,* 209-215.

Waddell, G. (2004). *The Back Pain Revolution*. London: Elsevier.

Waddell, G., Newton, M, Henderson, I., Somerville, D., & Main, C. J. (1993). A fear-avoidance beliefs questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain, 52,* 157-168.

Walker, B. F. (2000). The prevalence of low back pain: a systematic review of the literature from 1966 to 1998. *Journal of Spinal Disorders, 13,* 205-217.

Watson, P. J. (2002). Psychophysiological models of pain. In Gifford, L. (Ed.), *Topical issues in pain 4* (pp. 181-198). Falmouth: CNS Press.

Watson, P. J., Booker, C. K., & Main, C. J. (1997a). Evidence for the role of psychological factors in abnormal paraspinal activity in patients with chronic low back pain. *Journal of Musculoskeletal Pain, 5,* 41-56.

Watson, P. J., Booker, C. K., Main, C. J., & Chen, A. C. N. (1997b). Surface electromyography in the identification of chronic low back pain patients: the development of the flexion relaxation ratio. *Clinical Biomechanics, 12,* 165-171.

Weinberger, D. A., Schwartz, G. E., & Davidson, J. R. (1979). Low-anxious, high-anxious, and repressive coping styles: psychometric patterns and behavioural and physiological responses to stress. *Journal of Abnormal Psychology, 88,* 369-380.

Wideman, T. H., Adams, H., & Sullivan, M. J. L. (2009). A prospective sequential analysis of the fear-avoidance model of pain. *Pain, 145,* 45-51.

Wilke, H-J., Neef, P., Caimi, M., Hoogland, T., & Claes, L. E. (1999). New in vivo measurements of pressures in the intervertebral disc in daily life. *Spine, 24,* 755-762.

Woby, S. R., Roach, N. K., Urmston, M., & Watson, P. J. (2005). Psychometric properties of the TSK-11: a shortened version of the Tampa Scale for Kinesiophobia. *Pain, 117,* 137-144.

Woby, S. R., Roach, N. K., Urmston, M., & Watson, P. J. (2007a). The relation between cognitive factors and levels of pain and disability in chronic low back pain patients presenting for physiotherapy. *European Journal of Pain, 11,* 869-877.

Woby, S. R., Roach, N. K., Urmston, M., & Watson, P. J. (2008). Outcome following a physiotherapist-led intervention for chronic low back pain: the important role of cognitive processes. *Physiotherapy, 94,* 115-124.

Woby, S. R., Urmston, M., & Watson, P. J. (2007b). Self-efficacy mediates the relation between pain-related fear and outcome in chronic low back pain patients. *European Journal of Pain, 11*, 711-718.

Woby, S. R., Watson, P. J., Roach, N. K., & Urmston, M. (2004). Are changes in fear-avoidance beliefs, catastrophizing, and appraisals of control, predictive of changes in chronic low back pain and disability? *European Journal of Pain, 8*, 201-210.

Woo, A. K. M. (2010). Depression and anxiety in pain. *Reviews in Pain, 4*, 8-12.

Woods, M. P., & Asmundson, G. J. G. (2008). Evaluating the efficacy of graded *in vivo* exposure for the treatment of fear in patients with chronic back pain: a randomised controlled clinical trial. *Pain, 136*, 271-280.

Zachariae, R., Jensen, A. B., Pedersen, C., Jørgensen, M. M., Christensen, S., Lassesen, B., *et al*. (2004). Repressive coping before and after diagnosis of breast cancer. *Psycho-Oncology, 13*, 547-561.

Zedka, M., Prochazka, A., Knight, B., Gillard, D., & Gauthier, M. (1999). Voluntary and reflex control of human back muscles during induced pain. *Journal of Physiology, 520*, 591-604.

Zhao, F., Pollintine, P., Hole, B. D., Dolan, P., & Adams, M. A. (2005). Discogenic origins of spinal instability. *Spine, 30*, 2621-2630.

Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica, 67*, 361-370.