Pilates can decrease chronic low back pain and related functional disability

Claire O’Brien

A research project submitted in partial fulfilment of the requirements for the degree of Master of Osteopathy, Unitec Institute of Technology, 2010.
Declaration

Name of candidate: Claire O’Brien

This Research Project entitled “Pilates can decrease chronic low back pain and related functional disability” is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

Candidate’s declaration:

I confirm that:

- This research project represents my own work.
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
- Research for this work has been conducted in accordance with the Unitec research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: 2009-923

Candidate Signature: Date:

Student ID: 1098583
Acknowledgments

Thank you to the Maree Seerden and the Pilates Body Studio for the use of the studio space and equipment.

Thank you to my supervisors Rob Moran, Craig Hilton, and Andy Stewart.

I would also like to thank all our participants for their interest and commitment.

To my family and friends and Nick, thank you for the encouragement and support.
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Overview

The following research project is divided into three sections:

1. The literature review, with emphasis on:
   - Low back pain prevalence and societal impact
   - Chronic low back pain and disability
   - Current treatments for chronic low back pain
   - The treatment of chronic low back pain with exercise
   - Pilates as an exercise intervention for low back pain

2. A manuscript in the format specified for submission to the Journal of Bodywork and Movement Therapies.

3. Appendices that include ethics approval, participant information sheet, screening questions, consent form, questionnaires, medical history form and the guidelines for authors to the Journal of Bodywork and Movement Therapies.
Section 1: Literature Review
Introduction

In modern western society low back pain (LBP) is a major health and economic problem. For a new episode of LBP the prognosis is generally considered to be good, with 90% of LBP resolving within 12 weeks. However, a small percentage will go on to develop a chronic LBP disorder. Chronic LBP is costly in terms of direct treatment costs; and indirect costs such as work days lost, and income compensation for permanent disability. In Section I of this thesis, the different models for diagnosis and classification of non-specific chronic low back pain are discussed, including the biopsychosocial model. It is well recognised that chronic LBP is a multidimensional problem, with mechanical, neurophysiological, psychological and social factors all influencing its presentation and progression. The focus and challenge of recent research has been finding effective treatments LBP with lasting effects. The last part of this Section briefly describes current treatments, and focuses on the use of exercise therapy and Pilates for management of chronic LBP.
Low Back Pain

Low back pain (LBP) is defined as pain that is perceived as arising in the region bounded by the 12th rib and the inferior gluteal folds; and may also be associated with or without leg pain (Krismer, van Tulder & Low Back Pain Group of the Bone and Joint Health Strategies for Europe Project, 2007). LBP is the most common of all musculoskeletal problems and it has been estimated that 70-85% of adults will suffer at least one episode of back pain at some time in life (Andersson, 1999). In the United States low back pain is the most common type of musculoskeletal complaint reported by adults (Deyo, Mirza & Martin, 2006). The prevalence of LBP is high in nearly every country in which data has been collected (Andersson, 1999;Deyo et al., 2006).

Low back pain and its associated disability poses an economic burden to society, mainly in terms of the large number of work days lost (indirect costs) and to a lesser extent by direct treatment costs (Krismer et al., 2007; Dagenais, Caro & Haldeman, 2008). In New Zealand it is estimated that 20 – 25% of all workplace injuries are related to LBP (Firth, Herbison, McBride & Feyer, 2002). With the total cost to New Zealand’s society (including indirect costs) estimated to be NZD $500 million annually (McBride, Begg, Herbison & Buckingham, 2004). In Australia, the total cost of LBP has recently been estimated to be more than AUD$9 billion per year, with a national prevalence of 65% annually (Dagenais et al., 2008).

There is a need to investigate new treatment and rehabilitation approaches for low back pain because such a large portion of the population is affected, and the problem has such a substantial economic and social impact on society. In contemporary healthcare, low back pain is generally considered as a multidimensional problem with a multicausal aetiology; due to the heterogeneity of the condition there is no standard intervention that ‘works’ for all individuals with LBP. Recently, researchers have become increasingly interested in
identifying different sub-groups of low back pain (Delitto, Erhard & Bowling, 1995; Kent & Keating, 2004; O'Sullivan, 2005), with the ultimate goal of matching the most appropriate therapy with the ‘type’ of presentation. It is believed that identification of subgroups of patients (with a particular type of low back pain) could improve outcomes and enhance treatment effects (Hicks, Fritz, Delitto & McGill, 2005; Fritz, Cleland & Childs, 2007; Dankaerts et al., 2009).

Low back pain can arise from a wide variety of causes, such as unaccustomed activity, trauma, stress or injury to the structural elements of the spine. Acute LBP occurs suddenly, either as a completely new presentation (first time ever) or, after a period of at least 6 months without LBP. Acute LBP is usually defined as pain that is present for less than 6 weeks after onset (Krismer et al., 2007). Most people with acute LBP will recover quickly without residual functional loss and 60-70% of people will recover from an acute episode of LBP by 6 weeks, with over 90% recovering within 3 months (Andersson, 1999). However, a smaller proportion, approximately 10-20%, will continue to experience low back pain on an ongoing basis leading to chronic or recurring LBP (Maher, 2004).

Chronic low back pain (CLBP) is a major health problem, as well as a social and economic burden. The population of people with chronic LBP is accountable for approximately 80% of all healthcare costs related to back pain (Maher, 2004; Waddell, 1998). It is difficult to compare the cost of chronic LBP in different countries, but to give an indication of the enormity of the issue, one study in the USA identified that only 4.6-8.8% of LBP cases lasted for more than 1 year but they accounted for 64.2-84.7% of the costs (Hashemi, Webster & Clancy, 1998). In New Zealand, the Accident Compensation Corporation (ACC), a government insurer for workplace and sporting injury, statistics over the period 2000–2001 reveal that 10,968 new claims were processed at a cost of $30 million, whilst the 6,660 ongoing claims cost over double that, totalling $68 million (ACC Injury Statistics 2001).
Non-Specific Low Back Pain

In a small proportion of cases LBP can be indicative of serious spinal pathology, nerve root compromise; or damage to a specific structure in the lumbar spine, however, a valid clinical diagnosis can be made in an estimated 5-10% of LBP cases (Krismer et al., 2007). Some specific causes of LBP include disc injury, spinal stenosis, inflammatory conditions, some degenerative conditions, infection or neoplasm, metabolic bone disease, referred pain, psychogenic pain, trauma and congenital disorders.

Current clinical guidelines recommend screening for signs of serious spinal pathology or ‘red flags’, and nerve root compression signs upon initial presentation (The New Zealand Acute Low Back Pain Guide, 1999; Waddell, 2004). The presence of red flags should direct care towards a specialist referral.

Where there is no evidence of any underlying pathology; detectable tissue damage; or nerve injury it has proven difficult to classify LBP. This has given rise to the heterogeneous diagnostic grouping of ‘non-specific low back pain’ (Field, 2009). Interestingly, objective indicators of pathology detected by magnetic resonance imaging (MRI) such as disc degeneration or herniation, and other degenerative changes in the lumbar spine are present in some asymptomatic individuals, this further confounds the task of clinical identification of the source of a patient’s LBP (Takatalo et al., 2009).

Approximately 90% of LBP (both acute and chronic) is considered non-specific (Krismer et al., 2007; Henchoz & Kai-Lik So, 2008). Non-specific LBP, also known as ordinary or “simple backache”, and “common” or “garden variety low back pain”, is mechanical low back pain of musculoskeletal origin in which symptoms vary with physical activities (Waddell, 2004). Non-specific LBP may be related to mechanical strain or dysfunction, although it often develops spontaneously, and can be painful and disabling, however the severity or intensity
of the pain tells the clinician very little about the source of pain. Back pain often spreads to one or both of the buttocks or thighs, and this is usually somatic referred pain and not a sign of nerve root compression (Bogduk, 2009).

**Chronic Low Back Pain and Disability**

Non-Specific Low Back Pain is often further subdivided based on duration of symptoms. Acute LBP lasts up to 6 weeks; sub-acute pain is identified as lasting 6 weeks to 3 months; chronic low back pain is often defined as having low back pain that lasts for longer than 12 weeks (Weiner & Nordin, 2010). However, the course of LBP is not always continuous and many patients experience multiple ‘flare-ups’ of acute pain interspersed with periods of remission (Pengel, Herbert, Maher & Refshauge, 2003). In light of this, a further patient population group with ‘recurrent’ non-specific LBP has been suggested (Stanton, Latimer, Maher & Hancock, 2009).

Most treatment trials study either acute or chronic (persistent) LBP populations, and it is often unclear when reading these studies if the authors have included and acknowledged people who experience recurrent episodes of pain. Others classify frequently recurring back pain as chronic pain since it intermittently affects an individual over long period (Andersson, 1999). Because of the long-term nature of recurrent LBP it may perhaps be classed as an intermittent form of chronic LBP. A review that found exercise to be beneficial for subacute, chronic and recurrent LBP, but not for acute LBP, implies that recurrent LBP behaves like an intermittent form of CLBP (Henchoz & Kai-Lik So, 2008).

Approximately 85% of chronic low back pain (CLBP) cases do not have a specific diagnosis (Dillingham, 1995); these are labelled non-specific chronic LBP disorders. It is thought that non-specific CLBP is not linked to any persisting tissue damage, however, it is thought by some that a large portion of the non-specific CLBP group consists of tissue sprains and
strains that have not resolved beyond normal tissue healing time (Abenhaim et al., 2000; NICE guidelines 2008). This group has been broadly classified based on the anatomical region in which pain is perceived as arising, however, this diagnostic/classification system is of little clinical value as it does not identify the underlying pain generating mechanism, and consequently it fails to provide clear direction for specific management (Padfield, Chesworth & Butler, 2002).

The overall objective of the early management of non-specific low back pain (lasting six weeks to one year) is to ensure that an episode of acute low back pain does not result in long-term withdrawal from normal activities, including sickness absence from paid employment. The relationship between disability (reduced ability to carry out activities of daily living) and the severity of LBP is weak (Bogduk, 2006). Waddell et al. (1993) has demonstrated that 40% of disability associated with low back pain can be explained by physical impairment, 23% by psychological distress, and 8% explained by illness behaviour. More severe pain and back pain-related disability, and psychological distress predict a poor long-term outcome for people with non-specific back pain (Pincus et al., 2008).
Models for Classification and Diagnosis of Low Back Pain

There are many theories about the physiological processes and biomechanical events that are operant in persistent low back pain. The pathoanatomical model, biomechanical/postural model, motor control and movement impairment models all attempt to identify a damaged or dysfunctional anatomical cause of pain. The psychosocial model focuses on the psychological and social factors and how they can modulate the pain response. The prevailing model of chronic musculoskeletal pain in western health care is the biopsychosocial model (first described by Engel in 1977), which classifies chronic low back pain disorders on the basis of the dominant feature, but without ignoring the other factors contributing to the condition (Waddell, 2004).

Pathoanatomical Model

The pathoanatomical model is best conceptualised as the traditional biomedical approach to the diagnosis of back pain; it attempts to identify a specific anatomical structure that can be implicated as the ‘pain generating structure’. As mentioned earlier, for a small number of cases (=10%), pain in the lower back is a symptom of more serious pathology. Attempts to classify the remaining 90% according to the anatomical structures causing pain has lead to an over emphasis being placed on diagnostic imaging findings. The findings of intervertebral disc (IVD) and facet joint degeneration, annular tears, IVD prolapse, spodylolisthesis, foraminal and spinal stenosis are commonly assumed to be ‘the cause’ of back pain (and in some cases associated nerve pain), however, studies have demonstrated that structural changes identified on imaging in a substantial proportion of people who are asymptomatic (Jensen et al., 1994).
Other authors have reported that the majority of CLBP is caused by disruption of the IVD (approximately 45% of CLBP), and the second most common pain generating structure is zygapophyseal joints (20%), followed by the sacro-iliac joint (15%) (Bogduk, 1995). This has lead to the development (Bogduk, 2004) and trial (Bogduk, 2002; Manchikanti, Singh, Falco, Cash & Pampati, 2010) of diagnostic nerve blocks to identify and subsequent denervation of the painful structure.

The use of routine X-rays in the diagnosis of LBP has fallen out of favour, and is not recommended in any current evidence based clinical guidelines. Often changes that show up on X-ray such as mild degeneration or slight loss of intervertebral disc space are due to normal aging and are not the cause of LBP. In the past X-rays have commonly been ordered as an attempt to reassure the patient that nothing serious was wrong with their back, but could end up having the complete opposite effect. Descriptions of degeneration due to ‘wear and tear’ in the radiologists report may actually reinforce the patient’s negative beliefs and cognitions about the serious nature of their back pain (Waddell, 2004). There is also an issue about exposure to ionizing radiation, and X-rays therefore need to be clinically justifiable on a risk: benefit basis.

In most cases of LBP, little clinically useful information is gained that would change management and the use of X-rays is now actively discouraged in most evidence-based guidelines (The New Zealand Acute Low Back Pain Guide, 1999). Treatment based on a pathoanatomical model tends not to address the psychological factors, beliefs and behaviours that are commonly present in people experiencing chronic pain disorders.
Movement Impairment Models

Postural/ Biomechanical Model

There is little doubt that back pain can start as a physical problem in the back. It has been argued that non-specific LBP arises from dysfunction or physiologic impairment (Waddell, 2004). Dysfunction depends on the level of demand or stress, and the capacity of the musculoskeletal system to respond to physiological and biomechanical demands or stresses. Any position that increases the physical stress to the joints may be called “faulty posture” (Kendall, McCreary & Provance, 1983).

Mechanical factors are frequently reported to be associated with the initial onset and recurrence of LBP (O'Sullivan, 2005). There are multiple risk factors associated with the occurrence of LBP, some of these factors are; repetitive motion; curvature and torsion of the spine; pushing and pulling activities; stumbles; falls; and static or sitting work posture (Cholewicki & McGill, 1996). However the presence of these does not necessarily lead to the occurrence of back pain, and absence of these factors does not necessarily prevent LBP from occurring.

There appears to be a commonly held belief amongst some practitioner groups that asymmetric or ‘imperfect’ posture is a chronic stressor and the origin of most noxious stimuli and that optimising posture could alleviate 70-90% of chronic pain (Irvin, Vleeming, Mooney & Stockhart, 2007). A ‘correct’ or ‘ideal’ posture has been described as the position in which minimal stress is applied at each joint (Kendall et al., 1983). In the spine this is often referred to as a ‘neutral position’ (McGill, 2007) (Wallden, 2009). Sustained deviations from neutral joint positions may cause uneven mechanical loading across articular surfaces, leading to increased compressive forces and shortened tissues on one side of the joint and relatively lengthened tissues and a distracted joint position on the opposite side (Wallden,
Under normal circumstances the nervous system would signal this position through mechanoreception and the musculature around the joint would respond by returning the joint to a more neutral position (Wallden, 2009). However, if such positions are maintained or frequently repeated then changes of musculoskeletal elements may occur due to uneven loading (Vleeming, Mooney & Stockhart, 2007). On the shortened side, tissues undergo dehydration, contracture, will shorten and become less able to translate loads, whereas the tractioned tissues will undergo dehydration, creep, will lengthen and will lose tensile strength (McGill, Grenier, Kavcic & Cholewicki, 2003). Observations of deviations from ‘ideal posture’ have formed the rationale for many manual therapies (Kendall et al., 1983; Sahrmann, 2002). These include stretching of short tight muscles; strengthening long weak muscles; and mobilisation or manipulation of restricted joints. There is little objective data that supports the relationship between postural symmetry or spinal curves and low back pain (Christensen & Hartvigsen, 2008; Levangie, 1999). However, there is evidence of a relationship between decreased lordosis in the neck, forward head posture and severity of disability in patients with neck pain (Yip, Chiu & Poon, 2008; McAviney, Schulz, Bock, Harrison & Holland, 2005).

Instability Model

Another proposed aetiology of CLBP that falls under the broader category of movement impairment is Panjabi’s model of ‘clinical spinal instability’ (Panjabi, 2003). Panjabi’s model theorises that laxity around the neutral position of the spinal segment referred to as the neutral zone, leads to an increased size of the neutral zone (Panjabi, 2003). One of the possible sub-groups of chronic low back pain population, are individuals who have an increased neutral zone (O’Sullivan, 2005). In pain free spinal biomechanics the neutral zone is within the pain free range of the total range of movement (ROM) for that spinal segment.
If the patient has an increased neutral zone, this means that the spinal motion more easily enters the pain generating range.

Instability can be the result of trauma causing ligamentous laxity, fracture, damage to the intervertebral disc, or deficits in neuromuscular control. Chronic low back pain occurs due to the repeated micro trauma of spinal motion entering the pain generating range (Hodges 2003a); therefore decreased intervertebral motion should result in a reduction in pain in the CLBP patient (Panjabi, 2003). This forms the basis for many treatments and therapies for low back pain including surgical fusion, muscle strengthening (stabilization exercise programmes) and motor control training.

**Motor Control Model**

Motor control is a dynamic strategy that refers to the generation of an appropriate sequence of movements that the motor control system determines the requirements for movement and stability and produces appropriate strategies to move the trunk and limbs in a balanced, efficient and coordinated way (Field, 2009).

It is well recognised that motor control impairments exist in the CLBP population (Hodges & Richardson, 1996; Hodges & Richardson, 1999a; Hodges, 1999; Hodges, 2001; O'Sullivan, Twomey & Allison, 1997; Hodges & Moseley, 2003; Jacobs, Henry & Nagle, 2009; van Dieën, Selen & Cholewicki, 2003). Recently there has been increased focus on the management of CLBP from a motor control perspective (Hodges 1996; Hodges, Hides & Richardson, 2004; Macedo, Maher, Latimer & McAuley, 2009; O'Sullivan, 2000; Jull & Richardson, 2000; Richardson & Jull, 1995).

It has been demonstrated that exercise interventions designed with the intent to retrain motor control of the trunk can decrease pain and improve levels of disability in CLBP population (Ferreira et al., 2007; Ferreira, Ferreira, Maher, Herbert & Refshauge, 2006;
Macedo 2008; Macedo et al., 2009) and appear to be particularly effective with those that have a radiographic diagnosis of instability related to spondyloisthesis or spondylolysis\(^1\) (O’Sullivan et al., 1997). For this precise type of exercise to be effective in reducing CLBP and disability, altered motor control has to be part of the pain generating mechanism and not a compensatory consequence of pain from a specific spinal pathology (O’Sullivan, 2005). It is unclear whether pain causes changes in motor control or whether motor control changes lead to pain, or both (Hodges 2003a). In other words, an important question that is being currently being addressed in the literature is whether altered motor control is a cause or an effect of pain (Hodges & Moseley, 2003; Hodges et al., 2004; Moseley & Hodges, 2005).

**Movement Impairment Classification (Muscle Guarding Model)**

Some CLBP disorders are thought to be associated with abnormally high levels of muscle guarding and co-contraction of muscles around the lumbar spine and pelvis, causing painful loss or impairment of normal (active and passive) physiological movement in one or more directions (Dankaerts et al., 2009). This muscle guarding appears to be driven by an exaggerated withdrawal motor response to pain (O’Sullivan, 2005) (Sahrmann, 2002). The co-contraction of the muscles surrounding the lumbar spine leads to increased compressive loading across the articulations of the lumbopelvic region (Moseley, Nicholas & Hodges, 2004), movement restrictions and rigidity (which could be considered to be ‘excess stability’), resulting in a mechanism for tissue strain and ongoing nociceptor sensitisation (O’Sullivan, 2005). This can be accompanied by an acute awareness of pain (hyper-vigilance) and attempts to avoid the pain provoking movements for fear that pain provocation is damaging. Ironically it can be the fear of pain with movement that makes the movement painful (O’Sullivan, 2005).

\(^1\) Clinical instability is confirmed by a radiographic diagnosis of spondyloisthesis or spondylolysis, as defined by Panjabi (Panjabi, 2003)
Psychosocial Model

It is clear from the literature that psychological and social factors can influence the experience and course of back pain (Dionne et al., 1997; Nachemson, 1999; Grotle, Vøllestad, Veierød & Brox, 2004; Bogduk, 2006). The presence of psychosocial risk factors or ‘yellow flags’ is known to be predictive of the development of chronic disability and time off work due to low back pain (Kendall, Linton & Main, 1997; Pincus, Burton, Vogel & Field, 2002). Negative attitudes, fear avoidance beliefs, depression, anxiety, distress, and related emotions, as well as a past history of sexual or physical abuse have all been implicated as risk factors for chronic back pain (Linton, 2002; Linton, Nachemson & Johnsson, 2000).

In patients with LBP fear avoidance beliefs, catastrophizing, hypervigilance and other negative coping strategies have been shown to be associated with high levels of pain, and disability (Walsh & Radcliffe, 2002; Grotle et al., 2004; Waddell, 2004; Woby, Roach, Urmston & Watson, 2007). The main factors that predict a poor outcome (prognostic factors) and represent potential barriers to recovery are distress or depression, (Dionne et al., 1997; Pincus et al., 2002); somatization (Dionne et al., 1997); catastrophizing (Severeijns, Vlaeyen, van den Hout & Weber, 2001); and inappropriate illness behaviors (Waddell, 2004).

Social factors such as compensation systems (Rasmussen, Leboeuf-Yde, Hestbaek & Manniche, 2009), work place disputes, work and family tensions (Schwartz, Slater & Birchler, 1996), and cultural beliefs can also be barriers to recovery and increase long term disability due to back pain (Nachemson, 1999; Pincus et al., 2002).

The success of interventions that solely targeted the psychological aspects of back pain (eg: pain education) have had limited effect on reported level of disability in CLBP population (Cherkin, Deyo, Street, Hunt & Barlow, 1996). Education about pain neurophysiology can
alter pain cognitions and physical performance but is insufficient by itself to obtain a change in perceived disability, but it has been suggested that pain neurophysiology education should be included in a wider pain management approach (Moseley et al., 2004).

**Biopsychosocial Model**

The prevailing model of contemporary healthcare for chronic pain is the biopsychosocial model. It is not a causal model, but rather a cross-section of the clinical presentation at one point in time. The model illustrates key psychological and behavioural factors that may help to understand the patient’s current level of pain and disability.

Engel was amongst the first to argue that the dichotomist approach to diagnosis of all disease (not just back pain) into either a problem of ‘the physical’ or ‘mental’ is clearly lacking (Engel, 1977). Engel (1977) appealed to the medical community to adopt a new medical model that would take into account the psychological, social and cultural influences on the patient’s health in addition to the biomedical (pathology and anatomy). Considerably later, it was Scottish orthopaedic surgeon and back pain specialist Gordon Waddell who popularised the biopsychosocial model with respect to low back pain (Waddell, 2004). The biophychosocial model is a framework that encompasses the pathoanatomical (biomedical) and psychosocial models.
It has been widely accepted that low back pain and disability is best understood and managed using a biopsychosocial model (Elvey & O'Sullivan, 2004; Waddell, 2004; Waddell & Burton, 2005; O'Sullivan, 2005). This model allows for the physical, psychological, and social factors to be considered as contributing factors to the chronic low back pain disorder. Which factors are more dominant will differ for each patient with chronic low back pain (O'Sullivan, 2005).

The biopsychosocial model has been applied and investigated more in the context of LBP than any other common health problem (Waddell & Burton, 2005). Interventions that combine physical and psychological therapy have been shown to be more effective for treatment of CLBP, than interventions that use either psychology or physical therapy alone (Bendix et al., 1996; Brox et al., 2008b; Demoulin et al., 2009; Savigny et al., 2009).

**Cognitive Behavioural Therapy**

Cognitive Behavioural Therapy (CBT) is a clinical psychology intervention that includes various combinations of creative visualization, imagery, progressive muscle relaxation techniques, and problem solving techniques. The goal is to have the patient understand, accept, and take control of their “back pain” by helping the patient develop adaptive coping behaviours and strategies, and thus empowering them. Cognitive behavioural therapy interventions are effective at reducing pain and disability (Woby, Watson, Roach & Urmston, 2004; Nagarajan & Nair, 2010), and can decrease fear of movement (fear avoidance), catastrophizing; and increase control over pain in patients with non-specific CLBP (Woby, Roach, Urmston & Watson, 2008 Brox et al., 2008a).

**Functional Restoration Programmes**

Functional Restoration Programmes are intensive, time-consuming and costly rehabilitation programs usually conducted in secondary or tertiary care settings full time for 3–6 weeks. They always contain intensive exercise component of daily exercise sessions associated with
cognitive and behavioural therapy and, for some, ergonomic or social interventions in the work place. They are designed to restore the physical, psychological, and social interactions of the patient through their active participation in treatment. Developed by Mayer et al. (1985) this multidisciplinary approach to rehabilitation typically involves physicians, a pain management specialist, occupational therapists, physiotherapists, and psychologists or psychiatrist (Mayer et al., 1985).

The evidence supports the use of multidimensional biopsychosocial interventions for the treatment of sub acute or early chronic LBP (Schonstein, Kenny, Keating & Koes, 2003). However, these interventions are expensive to implement and it has been recommended that Cognitive Behavioural Therapy (CBT), and Multidisciplinary Functional Restoration be reserved for more severe cases of LBP (Maher, 2004).

**Treatment based subgroups Model**

A treatment based classification approach, unlike the aforementioned models, does not focus on causes of LBP, but instead attempts to group patients’ according to the treatment most likely to be beneficial. Delitto et al. (Delitto et al., 1995) developed a system of classification designed to inform and direct physiotherapy management of patients with low back pain. There are four classification groups (manipulation, stabilisation, specific exercise; and traction) each with its own set of examination criteria and associated treatment strategy predicted to result in the best outcomes for the patient. The system is designed to classify patients with acute LBP or those experiencing an acute exacerbation of LBP requiring pain reduction. An apparent limitation is that the approach offers little guidance for the treatment of chronic low back pain. An obvious weakness is that the model does not include assessment of the psychosocial factors that are present in these disorders – a glaring
omission in light of the widespread acceptance of the biopsychosocial model. Since the
proposal of this classification system a substantial amount of research has been undertaken
(Fritz, Childs & Flynn, 2005; Fritz et al., 2007), including development of a clinical prediction
rule (Flynn et al., 2002; Hicks et al., 2005), and updating of the specific signs and symptoms
(criteria) used to identify patient subgroups and improving intervention protocols for each
classification (Fritz et al., 2007).
Changes that occur with chronic low back pain

It is well established that symptoms of back pain can arise from physical processes in the back. The development of chronic pain and disability are however attributable to a complex combination of factors that occur concurrently, these changes are outlined in the following section; this includes alteration in motor control of the lumbopelvic region; sensitisation of the nervous system; psychological and behavioural factors. The adaptation or reaction to persistent back pain will occur in varying degrees for each individual, despite all of them being classified under the broad heterogeneous grouping of “non-specific CLBP”, this highlights the difficulty of treating the CLBP population.

Altered motor control

Many studies report poor postural control and changes in motor control in people with acute or chronic low back pain (Hodges 1999; Hodges 1996; O’Sullivan, 2000; Hodges & Moseley, 2003; Lamoth, Meijer, Daffertshofer, Wuisman & Beek, 2006). The principal muscles affected are those that have a role in movement and stability of the trunk and lumbopelvic region; transversus abdominis; internal and external obliques; lumbar multifidus; other lumbar erector spinae; and the muscles of the pelvic floor (Moseley & Hodges, 2005; Moseley, Hodges & Gandevia, 2002; van Dieën et al., 2003; Sapsford et al., 2001; Hodges & Moseley, 2003; Hodges & Richardson, 1999a; Hodges, 2001; Hodges et al., 2004; Macedo et al., 2008; Richardson & Jull, 1995). Unlike the muscles of the limbs, the muscles involved in lumbopelvic stability also perform a variety of essential homeostatic functions, such as breathing and continence, in addition to movement and control of the trunk (Hodges & Gandevia, 2000; McGill et al., 2003; Sapsford et al., 2001; Grimstone & Hodges 2003; Courtney, 2009; Sapsford, Richardson, Maher & Hodges, 2008).
Co-activation of the pelvic floor and deep abdominal muscles and diaphragm maintains intra-abdominal pressure, causing increased spinal stiffness and therefore enhanced stability (Hodges, Eriksson, Shirley & Gandevia, 2005). It has been demonstrated prospectively that pelvic floor (PF) muscle dysfunction may be linked to the development of lumbopelvic pain (Sapsford et al., 2001; Sapsford 2004), and recently it has been observed that women with LBP have decreased PF muscle function when compared with asymptomatic controls (Arab, Behbahani, Lorestani & Azari, 2010). Smith et al. (2006) observed a strong correlation between disorders of breathing and continence and incidence of LBP, suggesting that their presence is a stronger predictor for LBP than obesity and levels of physical activity.

Compared with healthy controls, CLBP subjects exhibit over activation of the more superficial larger muscles of the trunk and under activation of the inter-segmental ones (Hodges, 1996; van Dieën et al., 2003). Inter-segmental muscles are responsible for providing segmental stability and direct control over the position of the lumbar segments; they include the lumbar multifidus, quadratus lumborum, the lumbar parts of the iliocostalis and logiissimus, transversus abdominis (Hodges, 1999), the diaphragm and the posterior fibers of internal obliquus abdominis (O'Sullivan, 2000).

Contraction of the transversus abdominis and the lumbar multifidus, which normally occurs in preparation for subsequent movement of the extremities or the body in any direction (Hodges & Richardson, 1999b), has been shown to be delayed, or attenuated in those with LBP (Hodges & Moseley, 2003; Hodges & Richardson, 1996; Hodges, 1999; Moseley et al., 2002). Interestingly these same delays have been demonstrated with acute experimentally induced pain in subjects with no history of LBP (Hodges, Moseley, Gabrielsson & Gandevia, 2003). In some subjects these changes in trunk muscle activity persisted after the resolution of pain, consistent with observations of patients with recurrent LBP that are asymptomatic at the time (Hodges & Moseley, 2003; Hodges & Richardson, 1996; Hodges & Richardson, 1999a).
Studies have documented that the deep lumbar multifidus in patients with CLBP have a higher proportion of type II fibers (fast twitch, fatigable) as well as smaller fiber size (Hides, Stokes, Saide, Jull & Cooper, 1994; Käser et al., 2001; Wallwork, Stanton, Freke & Hides, 2009), at the affected painful site. Furthermore, atrophy of lumbar multifidus was not due to patient inactivity (Mazis et al., 2009), and these changes in morphology and function did not resolve with the natural remission of symptoms (MacDonald, Moseley & Hodges, 2009).

During postural and functional tasks the activity of the deep fibres of multifidus (DM) ordinarily precedes that of the superficial fibres (Moseley et al., 2002). This differential activity is consistent with the biomechanical data that suggests that the DM, which are situated close to the centre of rotation of the lumbar segments, generate compression and control intersegmental motion, whereas the superficial fibres have a larger moment arm over which to maintain and control the lumbar lordosis and counteract flexion torques (MacDonald, Moseley & Hodges, 2006). There is evidence to suggest that lumbar multifidus activation is delayed in patients with LBP (Leinonen et al., 2001), even when spinal loading (perturbation of the trunk) is predictable (Hodges & Moseley, 2003).

Importantly, it has been demonstrated that subjects with LBP can achieve improvements in motor control by specific training of the affected muscles (Tsao & Hodges, 2007; Tsao & Hodges, 2008; Tsao, Druitt, Schollum & Hodges, 2010). Furthermore, voluntarily contracting pelvic floor muscles has been shown to increase the thickness of transversus abdominis (Critchley, 2002), suggesting that PF should be considered part of the trunk stability mechanism (Arab et al., 2010). Interestingly, decreased LM activation but not decreased transversus abdominis activation was found to be predictive of clinical success with a stabilization exercise programme (Hebert, Koppenhaver, Magel & Fritz, 2010). However the study authors suggest that their finding should be interpreted with caution, because the
testing investigated activation of transversus abdominis, and did not establish if activation was feedforward (in preparation for movement) (Hebert et al., 2010).

**Decreased Variability of Motor Control**

Another difference that has been observed in subjects with CLBP is decreased variability of motor control patterns employed to recruit muscles that move and support the lumbopelvic region (Arendt-Nielsen, Graven-Nielsen, Svarrer & Svensson, 1996; Bruno & Bagust, 2007; Jacobs et al., 2009; Lamoth et al., 2004; Lamoth et al., 2006).

It has been reported that subjects with LBP have greater consistency in firing patterns of gluteals, hamstrings and erector spinae muscles in a prone hip extension exercise than non-LBP subjects (Bruno & Bagust, 2007). This decrease in variability means a reduction in the number of available motor patterns, and this is thought to translate to a decrease in adaptability (Jacobs et al., 2009). The availability of few motor patterns would decrease the ability of a system to adapt to unexpected stresses, such as slipping, sudden loading or rapid change in direction (Lamoth et al., 2004) – perhaps contributing to injury.

**Impaired function of Joints and Deficits in Proprioception**

In people with chronic low back pain the function of spinal joints may become impaired. This is thought to occur through local adhesions or due to altered muscular activity, and may contribute to the perpetuation of pain signalling (Lantz 1995). In people with LBP, proprioception can be altered including decreased joint position sense (ability to reposition a joint to a target position) and reduced ability to detect movement (kinaesthesia) (Field, 2009; Lantz, 1995).
Central and Peripheral Sensitisation of the Nervous System

Peripheral Sensitisation

Most peripheral sensitisation is short lasting and related directly to a stimulus or the effects of local inflammation. It may endure where a nerve axon is traumatised (nociceptive pain); or when ongoing irritation produces enduring sensitisation of the nerve either peripherally or centrally (Field, 2009).

Central Sensitisation

Central sensitisation is an increase in the excitability of neurons within the central nervous system (CNS) so that normal inputs begin to produce abnormal or exaggerated responses. This can occur in the spinal cord where prolonged or repetitive nociceptive input at a constant intensity can cause second order neurones in the dorsal horn to become sensitised (Mendell & Wall, 1965), or adjacent neurones to the receptors in the dorsal horn become sensitive to stimuli (noxious or not) to which they were previously unresponsive. For example, low threshold sensory fibres activated by light touch of the skin begin to activate neurones in the spinal cord that normally only respond to noxious stimuli, this means an input that would usually evoke an innocuous sensation is now perceived as being painful. The result of both of these scenarios is in an increased afferent signalling from the spinal cord to the mid brain and a heightened sense of pain perception by the person (Butler, Moseley & Harman, 2003; Field, 2009).

Overstimulation of sensory nerves results in enduring sensitivity to noxious sensation. Usually dorsal horn hypersensitivity is dampened by interneurones acting to inhibit nociceptive signalling; these are controlled in the most part by signals descending from the brainstem nuclei (nucleus raphe and reticular formation). The mechanisms for this are tonic inhibition which causes a general dampening, ensuring only persistent nociceptive signals get passed upwards, and the more profound ‘descending noxious inhibitory control’ (DNIC)
In DNIC, collaterals from ascending second-order nociceptors act in the brainstem producing descending inhibition throughout the system, other than at their level of activation. They therefore ensure that, when nociception arrives at the higher centres, it is carrying specific information about the origin of painful stimuli (Butler et al., 2003).

The descending pain control mechanisms primarily use opiate-based neurotransmitters. With enduring pain states, there is an increase in the gene expression of the neuropeptide cholecystokinin (CCK) and its receptor protein within the dorsal horn. CCK inhibits the effectiveness of opiates used by descending inhibitory pathways and thus promotes pain persistence by reducing second-order pain inhibition (Wiesenfeld-Hallin et al., 1997).

**Reorganisation of motor cortex**

Another change that occurs with CLBP is reorganisation of the somatosensory cortex and enlarged cortical representation of the painful area (ie low back) (Flor, Braun, Elbert & Birbaumer, 1997). It has also been observed that there is a relationship between increased cortical reactivity and chronicity (Flor et al., 1997).

**Altered Pathways within Higher Centres**

The limbic system is the final relay in the nociceptive pathways before the conscious perception of pain; it also acts in co-ordinating motor signals sent from higher centres downwards. It plays a role in gating pain, and is responsible for the generation of emotion and associated psychological changes. Importantly, through the cingulate cortex it ascribes suffering to pain (Butler & Matheson, 2000). From a neuro-anatomical perspective, the mind-body interaction may be considered to arise largely from the limbic system (Jones, Dilley, Drossman & Crowell, 2006).
Hyper-vigilance in the limbic system has been described as part of the explanation for chronic pain (Butler et al., 2003). Cognitive behavioural therapy for chronic pain patients has been shown to reduce electrical activity in the limbic system (Lackner et al., 2006). Where changes in activity of the limbic system are observed, they have been accompanied by significant improvements in pain and psychological functioning (e.g. reduction in anxiety and worry) (Lackner et al., 2006).

It has been demonstrated that patients with CLBP have a lower pain threshold because of increased attention to external stimulation and preoccupation with pain sensations (states that are mediated through the limbic system) (Giesecke et al., 2004).

Limbic dysfunction also manifests as an abnormal efferent innervation of musculature, both visceral and somatic. Fenton (2007) investigated limbic associations to chronic pelvic pain, and found that musculature undergoes tonic contraction as a result of the limbic efferent stimulation, which may generate further sensations of pain (Fenton, 2007). It has been postulated that the formation of enduring synaptic links, called long term potentiation (a form of neural memory), within the limbic system may be a new model for understanding central sensitisation related to chronic pain, as well as pain related cognitive emotional disorders (Zhuo & others, 2007).
Psychological & Behavioural Factors in Chronic Low Back Pain

Until the 1980s, back pain was considered to be largely a physical functioning problem but a growing body of published work began to illustrate the influence of non-physical factors on the outcome of treatment. It is now well accepted that psychological and social factors have a large part to play in CLBP (Linton et al., 2000; Pincus et al., 2002; Waddell & Burton, 2005; Woby et al., 2007). Illness behaviour is influenced by a person’s beliefs about pain, coping strategies, distress, and social interactions (Waddell, 2004).

Cognitions and Beliefs

How patients think (cognitions) and feel (emotions) about their back pain is pivotal in what actions they take and the influence of chronic pain in a person’s life. Cognitive processes translate nociceptive signals into perceptions of pain, and interpret them in terms of ‘threat value’ and potential necessity of action (Main, Foster & Buchbinder, 2010). Pain perception can be altered by beliefs, emotions, memories and previous experiences (Main et al., 2010). Beliefs about the extent to which pain can be controlled appear to be the most powerful determinates of adjustment to pain or the development of incapacity (Keefe, Rumble, Scipio, Giordano & Perri, 2004; Linton et al., 2000; Main et al., 2010).

Catastrophizing

Pain catastrophizing is the tendency to focus on pain and negatively evaluate one’s ability to deal with pain (Keefe et al., 2004). In patients with CLBP pain related fear and catastrophizing has been shown to be a stronger predictor of disability than activity intolerance or pain intensity (Keefe et al., 2004; Koleck, Mazaux, Rascle & Bruchon-Schweitzer, 2006; Thibault, Loisel, Durand, Catchlove & Sullivan, 2008). Patients that have persistent pain can become quite anxious and engage in fear avoidance behaviours (Vlaeyen & Linton, 2000).
Fear Avoidance Behaviour

Fear of pain (or reinjury) can lead to the avoidance of movements that are believed to be ‘dangerous’, this is known as fear avoidance behaviour (Waddell, Newton, Henderson, Somerville & Main, 1993). Fear of movement appears to develop from the patients’ initial experience of severe acute pain, as well as their beliefs (often reinforced by sympathetic family members and treatment providers) that pain is harmful. Movement related fear, hypervigilance, and anxiety associated with their pain reinforces the faulty cognitive coping strategies and beliefs, further amplifying the pain and increasing muscle guarding (O’Sullivan, 2005).

People with CLBP have been demonstrated to record higher levels of fear avoidance beliefs when compared to people with acute LBP (Grotle et al., 2004). One review concluded that high levels of fear avoidance was not indicative of a poor prognosis, but that it may have a role to play in pain that is persistent (Pincus, Vogel, Burton, Santos & Field, 2006). It has been argued that fear of pain and re-injury prevents normal return to activity, which leads to deconditioning and dysfunction, and can be a further barrier to recovery (Hodges & Moseley, 2003).

Distress and Depression

The emotional impact of pain can range from mildly distressing to overwhelming (Main et al., 2010). Distress can increase awareness of bodily sensations, lower pain tolerance, and make people more likely to seek health care (Waddell, 2004). In a systematic review, Pincus et al (2002) use the term ‘distress’ to encompass psychological distress, depressive symptoms, and depressive mood. Distress has been identified as a significant predictor of unfavourable outcomes, namely, the development of physical disability and CLBP (Pincus et al., 2002).
Self-Efficacy

Self-efficacy is the belief in one's ability to perform a task or activity, and appears to explain the relationship between fear-avoidance beliefs and disability (Woby et al., 2007). It has been recommended that self-efficacy should be measured in addition to fear-avoidance beliefs in chronic low back pain patients (Main et al., 2010). It has been demonstrated that self-efficacy beliefs are more important determinants of disability than fear-avoidance beliefs (Costa, Maher, McAuley, Hancock & Smeets, 2010). Maine et al. (2010) also argue that patient expectations of, and preference for, a particular treatment have an influence on the outcome. Therefore it seems sensible that patients be involved in selecting treatment approaches.
Management of Chronic Low Back Pain

The management of LBP has comprised a range of different intervention strategies, including surgery, drug therapy, spinal manipulation, exercise and other non-medical interventions (such as massage, physical therapy, electrotherapies, acupuncture). Currently research on the effectiveness of various treatments for non-specific chronic low back pain (NSCLBP) indicates that there are few effective treatments available. The reasons for this weak effectiveness becomes apparent when the different ‘types’ of back pain are considered; firstly a group of individuals with NSLBP is not a homogeneous group, there is wide variance in mode of onset, symptomatic presentation (distribution and intensity), and duration of LBP (persistent or recurrent, is often not defined). This has lead to the heterogeneous grouping of NSCLBP, where clinicians are often classifying symptoms rather than causes. Secondly LBP is just a feature of the disorder, pain itself is not a disorder (Elvey & O’Sullivan, 2004).

Manipulation and Massage

In heterogeneous CLBP populations spinal manipulative therapy (SMT) has been shown to provide greater improvements in functional disability and better short and long term-pain relief compared to either back school (exercise and education programme for LBP, delivered in small group classes), or individualised physiotherapy (Cecchi et al., 2010). SMT as also been shown to be more effective than general exercise at improving function and reducing pain (Ferreira et al., 2007). However passive treatments for sub-acute and chronic LBP should be used minimally due to lack of proven long-term effectiveness, cost, and lack of impact on functional outcomes and return to work (Weiner & Nordin, 2010). Manual therapy including manipulation is recommended under current clinical guidelines for management of NSLBP in adults, to provide short-term pain relief and facilitate increased activity (The New Zealand Acute Low Back Pain Guide, 1999) (Savigny et al., 2009). Massage
combined with exercises and education has been shown to be more effective than soft
tissue massage alone; remedial exercises and education only; and sham laser therapy (van
Tulder & Koes, 2007).

**Traction**

There is little to no evidence supporting the use of traction for treatment of chronic low
back pain, and the intervention is not recommended by systematic reviews or practice
guidelines (Clarke et al., 2006; Maher, 2004; Savigny et al., 2009).

**Injections**

The efficacy of spinal injections is limited (Deyo, Mirza, Turner & Martin, 2009) (Henschke et
al., 2010). Epidural corticosteroid injections may offer temporary relief for sciatica, but both
European and American guidelines, based on systematic reviews, conclude that they do not
decrease the rate of subsequent surgery (Muller, 2007; Deyo et al., 2009). Facet joint
injections with corticosteroids appear to be no more effective than sham injection with
saline (van Tulder & Koes, 2007).

In a recent randomised controlled trial (Peng, Pang, Wu, Zhao & Song, 2010) promising
results were reported using intradiscal injection of methylene blue (MB) as a treatment for
discogenic back pain (that had been confirmed by discography). The 36 patients that
received the intradiscal injection of MB showed a mean reduction in pain measured by 101-
point numerical rating scale (NRS) of 52.50, a mean reduction in Oswestry disability index
(ODI 0-100) scores of 35.58, compared with placebo treatment group (also 36 patients) NRS
6.91, ODI 1.68, (p< 0.001 and p< 0.001, respectively). Patients who respond to injection of
methylene blue typically obtain relief within 24 hours of receiving injection, due to gradual
denervation of the disc. This denervation appears to be enduring, for once established, the
relief persists for two years (ie the duration of follow up to date of publication in 2010). In
an editorial commentary spinal injection researcher Nikolai Bogduk (Bogduk, 2010) states that if the results of Peng et al. (2010) are true, this intervention has the potential to revolutionize the treatment of low back pain. However, Bugduk warns that credibility and reproducibility of the results are the main issue and replication studies are required.

**Back Schools**

Back school originated in Sweden in 1969 (Forsell, 1981). The Swedish back school consisted of information on spinal anatomy and physiology, biomechanics, optimal posture, and ergonomics. Patients were taught how to protect spinal structures in daily activities (Brox et al., 2008b). Later, back schools were incorporated in comprehensive multidisciplinary programmes or functional restoration. Now there is wide variation between different back schools, the duration, educational content and exercises taught are not at all standardised (Maher, 2004). In most back schools a combination of both biopsychosocial and biomedical/biomechanical educational approaches are used, making it difficult to determine if this has an effect on the outcomes (van Tulder & Koes, 2007). In an occupational setting, back schools are better than no treatment, with back school resulting in improved short-term pain and reduced disability (Brox et al., 2008b; van Tulder & Koes, 2007). However, when compared to other forms of exercise therapy there were no significant differences in effect on pain or disability in the short or long term (van Middelkoop et al., 2010).

**Brief Education**

Brief education with regard to low back pain generally consists of advice to the patient to stay active, and attempts to dispel any negative thoughts or self limiting beliefs the patient may have about LBP. These short educational interventions can be provided in person (eg by GP or physiotherapist), in written form using plain language in published written matter such as *The Back Book* (Roland et al., 1997). Two systematic reviews recommend brief education in the clinical setting (usually a general practitioner office) for improved return to
work and short-term reduction of pain (Brox et al., 2008a; Brox et al., 2008b). This is consistent with current guidelines for LBP that advocate the promotion of self-management and to stay active (Savigny et al., 2009). There is also no evidence that brief education provided as a back book improves rates of return to work and conflicting evidence that it decreases disability (Brox et al., 2008a). One study evaluated the effect of education about pain neurophysiology (Moseley et al., 2004). This approach had an effect on pain cognitions and physical performance, but did not change perceived level disability, and was deemed not be clinically meaningful by the investigators (Moseley et al., 2004).

**Cognitive Behavioural Therapy and Fear Avoidance Training**

Cognitive behavioural therapy appears to be effective for sub acute and chronic low back pain (van Tulder, Koes & Malmivaara, 2006). There is moderate to strong evidence that CBT should be used early if certain psychosocial ‘yellow flags’ are present, and there is strong evidence that CBT should be used in most chronic patients with a duration of NSLBP >12 weeks. In addition, there is evidence of no difference in clinical outcome between fear-avoidance training and spinal fusion in chronic low back pain (Brox et al., 2008b). The risks are clearly less for non-operative therapies; no complications have been reported with therapies including exercise or CBT (Weiner & Nordin, 2010). Further evidence suggests that CBT is cost effective, provides an additional 20% efficacy to usual rehabilitation and reduces the duration of recurrence (Weiner & Nordin, 2010).

A comprehensive Cochrane review (Schonstein et al., 2003) concluded that successful physical conditioning programmes that include a cognitive-behavioural approach plus intensive physical training (specific to the job or not) and are given and supervised by a physiotherapist or a multidisciplinary team, seem to be effective in reducing the number of sick days for workers with chronic back pain, when compared to usual care. However, they found no evidence of their efficacy for acute back pain. In a recent update to this review
(Schaafsma et al., 2010) found conflicting results when physical conditioning programmes were compared to other exercise therapy (6 studies) in reducing time lost from work in workers with chronic back pain. Furthermore the addition of cognitive behavioural therapy to physical conditioning programmes was not more effective than the physical conditioning alone (Schaafsma et al., 2010).

It is evidence unclear whether CBT adds benefit to physical conditioning programmes (active therapies). However, it does appear that CBT is effective in reducing rates of recurrence and long-term disability, and therefore decreases utilization of health care following the intervention (Linton & Andersson, 2000).

**Functional Restoration Programmes**

Functional restoration generally includes an active exercise routine applied using a sports medicine approach, and individualized goal oriented programme, and intensive psychological support (Waddell, 2004). A Cochrane review has found that functional restoration programmes (FRP) with a cognitive behavioural approach plus physical training for workers with chronic back pain reduced sick days (Schonstein et al., 2003). However FRP did not reduce the risk of being off work at 12 months when compared to management by general practitioner, or other interventions (Schonstein et al., 2003).

**Multidisciplinary Treatment Programmes**

Multidisciplinary treatment programmes are based on a biopsychosocial rehabilitation model, they enlist the involvement of several health professionals in a treatment programme, to manage the physical, psychological, social and occupational factors associated with chronic low back pain (Maher, 2004). Most programmes include a graded physical activity programme that incorporates exercise therapy sessions, a home exercise programme, and graded increase of functional tasks both at work and at home.
Programmes vary widely, but most are brief and intensive (e.g. full time over 3 weeks) (Maher, 2004).

Multidisciplinary programmes have been shown to positively influence pain and functional status and ability to work when implemented in the early chronic stages of LBP (Guzman et al., 2001). A Cochrane review concluded that multidisciplinary biopsychosocial rehabilitation with functional restoration was more effective than non-multidisciplinary outpatient rehabilitation or usual care, for treatment of chronic low back pain (Guzman et al., 2001).

**Exercise for Chronic Low Back Pain**

Exercise is one of the few clearly effective treatments for low back pain (Maher, 2004). The therapeutic use of exercise in the treatment of low back pain has been widely reported. Despite its effectiveness the use of exercise as a treatment for chronic low back pain is under utilised (Carey et al., 2009). There is strong evidence that exercise can decrease pain, disability, secondary physical deconditioning and reduce time off work for people with chronic low back pain (Henchoz & Kai-Lik So, 2008; Maher, 2004).

Most treatment trials study either acute or chronic (persistent) LBP populations, and it is often unclear if the authors have included and acknowledged people who experience recurrent episodes of pain. Others classify frequently recurring back pain as chronic pain since it intermittently affects an individual over a long period (Andersson, 1999). Because of the long-term nature of recurrent LBP it may perhaps be classed as an intermittent form of chronic LBP. A review that found exercise to be beneficial for subacute, chronic and recurrent LBP, but not for acute LBP, implies that recurrent LBP behaves like an intermittent form of CLBP (Henchoz & Kai-Lik So, 2008).
It is well established that staying active is more effective than resting in bed for treating acute back pain (Hagen, Hilde, Jamtvedt & Winnem, 2004). In another systematic review, exercise therapy was found to be more effective than no treatment and other conservative treatments in reducing pain and improving function in patients with chronic LBP (Hayden et al., 2005).

**General Exercise**

A systematic review concluded that exercise is effective for the primary and secondary prevention of chronic non-specific low back pain. Exercise is more effective in decreasing pain and disability from LBP than control treatments or physician consultation (Henchoz & Kai-Lik So, 2008).

Exercise programmes for chronic low back pain are usually designed to reverse de-conditioning or fear of movement associated with pain, or both. Such exercise programmes are often conducted in groups and typically include aerobic exercise such as walking or stationary cycling, as well as stretching and strengthening exercises (Hayden & others, 2005; Klaber Moffett, Frost & UK BEAM, 2000).

One review concluded that there was contradictory evidence that various general exercise/physical fitness programmes reduced future LBP and work loss, and that any effect size was modest (Waddell & Burton 2001, Occupational Health guidelines for the management of low back pain at work: evidence review).

**Specific Exercise**

The existence of subgroups of patients that respond to repeated end range movements was popularised by McKenzie in the 1980s (Fritz et al., 2007). The “centralisation phenomenon” describes the tendency for the location of symptoms to shift towards the centre of the body following repeated end range movement – this concept is most often applied in the lumbar
spine. In a specific exercise approach the direction of movement that causes “centralisation” is used to indicate the ‘direction of treatment’ (Hefford, 2008). Exercises are repeated in a specific direction of; flexion; extension; or a lateral shift. Extension is the most common direction of specific exercise prescription; patients perform a prone press up or back extension (similar to a ‘cobra’ position from yoga) type of exercise (Fritz et al., 2007). One systematic review referred to this specific type of exercise in a broader classification of unloaded exercise, which also included yoga, tai chi, and passive stretching (Slade & Keating, 2007). Slade et al. (2007) concluded that unloaded exercise improves pain and function for people with NSCLBP, and is more effective than no exercise. Another review of specific exercise (delivered according to McKenzie principles) found greater reductions in pain and disability in the short term, but the differences were small in magnitude and not significant at long term follow up (Machado, de Souza, Ferreira & Ferreira, 2006). However the studies in these reviews had broad inclusion criteria (i.e. NSLBP) and it is argued that this may explain the small treatment effect (Fritz et al., 2007).

**Stabilization Exercise**

The term ‘stabilisation exercise’ is largely synonymous with ‘motor control exercise’. In current literature both terms are in use, ‘stabilisation’ is more popular amongst clinicians especially physiotherapists, and ‘motor control exercise’ appears to be the term preferred by researchers. Typically, stabilisation exercises are aimed at retraining transversus abdominis and lumbar multifidus (Maher, 2004). Patients are taught how to activate these muscles independently from the more superficial trunk muscles in isolation first, then during more functional tasks (Richardson & Jull, 1995). Pelvic floor activation and breathing control exercises are commonly included in these protocols (Maher et al., 2005).

O’Sullivan et al. (1997) demonstrated effectiveness of a specific stabilisation exercise approach in a CLBP population with a specific diagnosis of spondylolisthesis or spondylolysis.
Within the group who received specific exercise (SEG, specific exercise group) a significant reduction in pain intensity ($p=0.0006$, effect sizes: CG $d=0.21$ ‘trivial’; SEG $d=1.78$ ‘very large’) and functional disability levels (ODI) ($p=0.0001$, effect size CG $d=0.06$ ‘trivial’, SEG $d=0.88$ ‘large’) was observed, with maintenance of this effect at 30-month follow up. No significant changes were seen in a control group receiving usual care (O’Sullivan et al., 1997). However, despite being often cited this study has not been replicated to date.

**Motor Control Exercise**

It has been demonstrated that people with CLBP have altered motor control of their trunk muscles (Hodges & Richardson, 1996; Hodges & Richardson, 1999a; Hodges & Richardson, 1998; Hodges, 2001; McCook, Vicenzino & Hodges, 2009; MacDonald et al., 2009). Motor control exercise was developed based on the principle that individuals with LBP have a lack of control of trunk muscles (Richardson & Jull, 1995). The premise of the motor control approach is that simple functional exercise alone does not re-establish the coordination of the trunk muscles involved in lumbopelvic stability (Maher et al., 2005). A motor learning approach is used to retrain the optimal control and coordination of the spine (Macedo et al., 2009).

The protocol for retraining motor control of the trunk muscles and intrinsic muscles that stabilise the lumbar spine and contribute to lumbopelvic stability is outlined in Richardson (1995). The key feature of motor control exercise is training of the deep trunk muscles in isolation before progressing to demanding tasks that train coordination of the deep and superficial trunk muscles (Maher et al., 2005). In more recent times the use of real-time ultrasound (RTUS) to provide feedback has become widely utilized in motor control training (Ferreira et al., 2007; Macedo et al., 2008), although the use of ultrasound to provide feedback doesn’t appear to be common practice in routine private physiotherapy practices, this is probably due the cost of equipment and specialized training required. There has been
no research to date examining if outcomes from this form of exercise are superior when delivered using RTUS feedback.

There have been four systematic reviews comparing motor control exercise to other treatments or different types of exercise for chronic non-specific LBP. Two of these (Ferreira et al., 2006; Macedo et al., 2009) are considered to be of higher quality as they use meta-analysis / meta-regression (van Middelkoop et al., 2010), whereas the other two simply describe and compare the results of the studies included (Hauggaard & Persson, 2007; Rackwitz et al., 2006).

In the systematic review by Macedo et al. (2009) the outcome of motor control exercises for non-specific chronic LBP was found it to be superior to minimal intervention and confers benefit when added to another therapy for pain and for disability at short-term follow-up. However motor control exercise was not found more effective than manual therapy or other forms of exercise (Macedo et al., 2009). Ferreira et al. (2007) demonstrated that motor control exercise and spinal manipulative therapy produce a slightly larger decrease in functional disability measured using the Patient Specific Functional Scale (but not RMDQ), and global perceived effect of treatment (Global perceived effort scale, GPE) than general exercise following an 8-week intervention. However there were no differences between the groups with regard to reduction in pain intensity, and these differences were not significant in the medium and long-term follow up (Ferreira et al., 2007).

One interesting finding of the review by Macedo et al. (2009) was that a less complex form of exercise therapy that did not incorporate the retraining of specific muscles was as effective in reducing pain and increasing quality of life as motor control exercise (Critchley, Ratcliffe, Noonan, Jones & Hurley, 2007), which is often time consuming for therapists to deliver, as it has to be done 1-on-1 for adequate feedback, and can be costly for patients.
Pilates

Pilates is an exercise method that was first taught as “Contrology” by Joseph Pilates at his studio in New York during the late 1920s. The exercise system that Joseph Pilates developed merged the theories and movement styles of gymnastics, martial arts, yoga and dance. In the 1940s Pilates’s method became most popular with dancers, and he was well known for his apparent ability to return dancers to the stage following leg or back injuries (Latey, 2001).

Currently there are several different styles of Pilates, and these can be conveniently divided into two main schools: the repertory approach (sometimes labelled ‘traditional’ or ‘classical Pilates’), and ‘modern Pilates’ (Latey, 2001). The repertory approach follows closely the 34 traditional mat exercises described in Return to Life (Pilates, 1945). The exercises are vigorous with a fast, dynamic rhythm and are difficult to execute correctly, particularly for people with musculoskeletal impairments. To do these exercises requires substantial muscular strength and a “good to high level of flexibility” (Latey, 2002). The repertoire is designed to challenge and strengthen the abdominal or trunk muscles referred to as the “powerhouse”, by maintaining a “flat back” or ‘imprinted spine’, where the lumbar spinal curve is pressed to the floor, whilst locking or holding of the upper abdominals, hip flexor origins, and glutei muscles (Latey, 2002). This approach has been criticised (Latey, 2002) in light of research that has identified the muscle function and connections of the lower abdominal muscles and the pelvic floor.

Modern Pilates focuses on maintaining a ‘neutral spine’, pelvic and spinal stability, along with activation of transversus abdominis and pelvic floor muscles in combination with controlled breathing (Latey, 2002). The neutral spine position is typically defined as the position in which the natural spinal curves are maintained (lumbar and cervical lordosis, and thoracic kyphosis). To maintain a neutral spine requires a ‘neutral pelvis’. Neutral pelvis is
achieved when the anterior superior iliac spines (ASIS) are in the same transverse plane, and both ASIS and the pubic symphysis are in the same coronal plane (Sahrmann, 2002).

There are several published claims that Pilates exercises strengthens the ‘core’, increases flexibility, and improves posture and balance (Curnow, Cobbin, Wyndham & Boris Choy, 2009; Gladwell 2006. Furthermore, in people with CLBP, Pilates is sometimes promoted on the basis that it improves pain levels, flexibility, proprioception and the perception of positive general health. (Gladwell, Head, Haggar & Beneke, 2006; Johnson, Larsen, Ozawa, Wilson & Kennedy, 2007).

Pilates is typically delivered over a number of sessions in a graded way, and as the sessions progress, mobility and articulation of the spine is encouraged, along with development of abdominal muscle endurance (deep and superficial) (Levine, Kaplanek, Scufura & Jaffe, 2007). Pilates’ techniques began to gain popularity in the rehabilitation setting during the 1990s. Many practitioners were using the method in multiple fields of rehabilitation, including orthopaedic, geriatric, chronic pain, and neurological (Anderson & Spector, 2000). In the rehabilitation setting, exercises are performed on various apparatus (such as the ‘reformer’). The apparatus repertoire was evolved from the traditional Pilates mat exercises, which were difficult to perform under the influence of gravity. By using apparatus, springs and gravity are used to assist in the controlled completion of movements. By altering the amount of assistance and increasing the challenge of gravity, an individual can be progressed toward achieving functional movement (Anderson & Spector, 2000).

The Pilates reformer is the most widely used piece of Pilates equipment and consists of the spring loaded carriage that moves on rails in the horizontal plane. A participant sits, kneels or lies on the carriage and moves the carriage against resistance. The reformer allows load to be modified dependent on the anthropometric proportions of the participant. The
participant can be assisted (against gravity) or have additional resistance added using springs attached to the carriage. Exercises can be modified for the individual by either decreasing the amount of assistance, increasing the amount of resistance, changing the length of the lever or altering the base of support. (Anderson & Spector, 2000; La Touche, Escalante & Linares, 2008). Lying supine on the reformer with a neutral spine allows for disassociation\(^2\) of the hip (with the lumbar spine) and shoulder (with the upper back and neck) whilst exercising the legs, arms and abdominals (Anderson & Spector, 2000).

**Critical Review of Previous Research of Pilates for Treatment of LBP**

To date there have been four studies investigating the effect of Pilates on CLBP, and one review article in which the three studies were considered (La Touche et al., 2008). Published to date, there have been two randomised controlled trials (RCT) (Gladwell et al., 2006; Rydeard, Leger & Smith, 2006), one clinically controlled trial (Donzelli, Di Domenica, Cova, Galletti & Giunta, 2006), and one comparative trial of three different Pilates regimens (Curnow et al., 2009). This section will critique each of these studies; they are also reviewed in Table 1.

**Critique of Rydeard et al**

Rydeard et al. (2006) conducted a well designed and executed randomised controlled trial, investigating the effectiveness of Pilates based therapeutic exercise for the treatment of CLBP. This is the only published study involving the use of Pilates for treatment of LBP to incorporate the use of equipment (reformers) in their exercise programme; and the only study to deliver treatment in an individualised manner. Delivering the intervention in this way makes it the study more relevant to the clinical setting, as this is how most therapists who employ Pilates exercise would implement a specific exercise training intervention in

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\(^2\)Dissociation is the movement pattern where two adjacent joints (e.g., the lumbar spine and hips) are moved independently of each other. This basic movement pattern is often absent in those people with chronic low back pain. (Sahrmann, 2002)
clinical practice. To allow for easier replication and comparison with other studies more
description of the exercises that were taught to participants should have been reported.
Thirty-nine participants were randomly allocated into the Pilates group (specific exercise
training group) n=21, or into the control group (CG) n=18. The Pilates intervention was
administered over 4 weeks; participants attended three 1-hour sessions per week, and were
to complete 15-mins of home practice 6 days a week. Adherence by the participants to the
home programme was not monitored so it is impossible to estimate how much this
contributed to the treatment effect, however, home programmes are commonly used in
practice and the use here does represent the way the intervention is typically undertaken.
There were no dropouts during the 4-week trial period.

The outcome measures used were relevant to LBP, the Roland Morris Disability
Questionnaire Hong Kong version (RMDQ-HK) was used as a measure of functional disability
(scale of 0-24) and Numeric Rating Scale (NRS) as a measure of pain intensity (scale of 0-
100). The differences between the two groups at baseline were not significant for either
outcome measure (RMDQ-HK, p=0.14; and NRS p=0.56), but the Pilates group did have lower
mean scores for functional disability and pain intensity prior to the intervention.

Treatment efficacy was assessed immediately post intervention and showed significant
reduction in functional disability (p=0.23) and pain (p=0.002) in the Pilates group compared
with the CG. In the Pilates group the pretest adjusted mean (±SEM) was 3.1(0.6) RMDQ-HK
points, and the post-test the adjusted mean (±SEM) was 2.0(0.3) (95% CI, 1.3 to 2.7) RMDQ-
HK points. In contrast, the CG with a pre-test adjusted mean(SEM) in the CG of
4.2(3.6)RMDQ-HK points and post-test adjusted mean(SEM) of 3.2(0.4) (95% CI, 2.5 to
4.0)RMDQ-HK points. For LBP pain intensity (NRS) the Pilates group had a decrease in pain
intensity from pre-test adjusted mean(SEM)of 23.0(17.7) NRS points, to post-test 18.3(3.2)
(95% CI, 11.8 to 24.8) NRS points. When compared to the CG the Pilates group had an
<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Method &amp; Subjects</th>
<th>Intervention</th>
<th>Outcome</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rydeard et al. (2006)</td>
<td>RCT assessor blinded</td>
<td>EG: Pilates on the mat, progressing to the reformer. Three 1hr classes per week, plus home practice for 15 mins 6 days a week for 4 weeks</td>
<td>Decrease in RMDQ-HK in EG and CG.</td>
<td>100% of cohort complete trial</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CG: usual care consisted of consultation with health care professional as necessary.</td>
<td>Sig decrease for EG in RMDHK (p 0.023) and NRS (0.002) when compared to CG. Pain Intensity NRS(0-100) increased in CG. Treatment effect retained at 3, 6 and 12 month for EG.</td>
<td>Low levels of pain and disability at baseline: RMDHK (0-24) mean EG Pre 3.1 Post 2.0 CG Pre 4.2 Post 3.2 NRS (0-100) mean EG Pre 23.0 Post 18.3 CG Pre 30.4 Post 33.9</td>
</tr>
<tr>
<td></td>
<td>N=39 14M 25F</td>
<td>EG=21 8M 13F</td>
<td>CG=18 6M 12F</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age years(SD) 37(9)</td>
<td>34(8)</td>
<td>9 years (1-20)</td>
<td>5.5 years (0.5-27)</td>
</tr>
<tr>
<td>Gladwell et al. (2006)</td>
<td>RCT assessor blinded</td>
<td>EG: Pilates on mat One 1hr class plus 2x 30min unsupervised home practice per week, for 6 weeks</td>
<td>34 completed the trial</td>
<td>11 mat exercises introduced over 6 weeks.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CG: continue with normal activities and exercise</td>
<td>ODI improved more for CG than EG significantly (p&lt;0.05). Pain (RMVAS) decreased more for EG</td>
<td>Modifications offered progressions and regressions but no use of props to assist mentioned</td>
</tr>
<tr>
<td></td>
<td>N=49 15 dropout</td>
<td>EG=20 d/o= 5</td>
<td>CG=15 d/o= 10</td>
<td>Sig dif between mean age of groups at baseline.</td>
</tr>
<tr>
<td></td>
<td>Mean age years(SD) 36.9(8.1)* *sig</td>
<td>45.9(8.0)</td>
<td>9.6(8.4) years</td>
<td>11.6(12.3) years</td>
</tr>
<tr>
<td></td>
<td>Duration of symptoms median (range)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Donzelli et al. (2006)</td>
<td>Clinical Controlled Trial</td>
<td>EG: Pilates on mat</td>
<td>43 completed the trial</td>
<td>Poor compliance of home exercises 4.5% of CG and 9.5% of EG reported doing their exercises on a regular basis. Pilates EG had greater perceived benefit. Authors describe as RCT in title, but randomisation was not used.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CG: Back School</td>
<td>Both groups had a decrease in VAS and ODI at 1.3 and 6 months compared to baseline, but results only displayed graphically, with no statistical analysis to determine sig and there was no comparison between groups.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N=53 10 dropout</td>
<td>EG=22</td>
<td>CG=21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean age</td>
<td>50.8 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Duration of symptoms &gt;3months</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curnow et al. (2008)</td>
<td>Between subjects equivalent group experiment.</td>
<td>All exercise groups, Group A =CG Group A: did 4 exercises (ab curl, oblique ab curl, side lying double leg lift, prone Tsp extension) Group B: did the same as group A plus a relaxation posture. Group C: did the same as group B plus a ‘genie exercise’ (postural training). All groups were to do exercises 3 x p/wk for 6 weeks, with follow up at 2, 4, and 8 weeks</td>
<td>ODI total scores not reported. Pain frequency decreased in groups B &amp; C, but increased once exercising stopped. No significant differences between the groups.</td>
<td>Not really Pilates method of exercise prescription. A set of 4 or 5 exercises (20 – 40 reps each!) and a relaxation position. 3 groups doing similar exercises. No statistical analysis done within groups pre-post.</td>
</tr>
<tr>
<td></td>
<td>N=39,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Group A n=13 Group B n=14 Group C n=12</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>No other baseline data about participants was provided</td>
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</table>

Key: EG=Exercise Group CG= Control Group
increase in pain intensity from pre-test adjusted mean(SEM) 30.4(17.6) NRS points, to post-
test 33.9(3.5) (95% CI,26.9 to 41.0) NRS points  (Rydeard et al., 2006).

Follow up data was collected at 3, 6 and 12 months following the completion of the main
study. At three months, 3 people were lost to follow up, so 85% of the initial sample was
analyzed. There was further decrease in functional disability post intervention to 3-month
follow up to 0.9(0.4) RMDQ-HK points. This level appears to be maintained over the next 12-
months, however, this result should be interpreted with caution due to lower response rate
of 57% at 6-months and 62% at 12 months. The absence of knowledge about the outcomes
of those participants who were not able to be followed up limits how much weight should
be given to this finding, in actuality those who were not followed up could have had a much
poorer outcome that those who were.

A criticism of this study is the use of standard error of the mean (SEM) instead of Standard
Deviations (SD) when publishing results. The SEM describes the uncertainty of how the
sample mean represents the population mean. The SEM is always smaller than the SD and
it can mislead the reader into underestimating the variability between individuals in the
sample  (Nagele, 2003). For checklist of CONSORT statement criteria and other notes on this
study please consult Appendix C.


Another study investigating the effectiveness of Pilates was undertaken by Gladwell et al
(2006). They conducted a single blinded RCT using mat based Pilates exercise delivered in a
small group class setting as a treatment for CLBP. Forty-nine participants were randomized
into either a Pilates group (n=25); or a control group (n=24). All participants were
encouraged to make no changes to their normal exercise, activities, or drug therapy. The
Pilates group attended one 1-hour mat class (maximum class size of 12 participants), and
were directed to undertak two 30-minute home practice sessions per week for 6-weeks.
The CG was given no specific intervention, and was not able to receive physiotherapy or other therapy during the trial period, but they were allowed to continue with usual activities and pain medication. There were a substantial number of drop outs during the trial, with only 34 participants completing the trial; 20 in the Pilates group and 15 in the control group. The reasons for 30% of the original sample withdrawing from the study are not fully discussed, the dropouts from the Pilates group were unable to attend two or more classes, but no reasons were given for dropouts from the control group. The high number of dropouts compromises the ability to draw a conclusion.

Despite randomization the mean age of the Pilates group 36.9 years was significantly younger ($p<0.05$) than the CG = 45.9 years. The procedures of randomization for this study were not described. By chance (with randomisation), particularly in small trials, study groups may not be well matched for baseline characteristics, such as age and stage of disease. This weakens the trial’s internal validity, and the ability of the authors to make accurate comparisons between the two groups, because the treatment received is not the only difference between groups. On average the Pilates group had not had LBP for as long as the CG, although this difference was not significant. Data was not normally distributed in the majority of cases and therefore non-parametric tests were performed. Wilcoxon sign-ranked tests were used to identify any significant changes pre- and post- intervention within each group. Consequently there were no confidence intervals provided.

A large number of outcome measures were assessed pre and post intervention. Roland Morris VAS (RMVAS) was used to measured pain intensity (a scale of 0-10), Oswestry Disability Index (ODI, 0-100) for functional disability, The SF-12 as a generic measure of health, a sports functioning questionnaire, the stork stand and sit and reach test as objective measures of functional performance, and a pain diary was maintained by participants.

Results were described as significant if $p<0.05$, however, no exact $p$ values were given which limits the readers ability to draw their own conclusions about what is an acceptable ‘cut-off’
point to interpret as being statistically significant. In the Pilates group the authors reported there were “significant increases” in general health (SF-12) and sports functioning. There was a significant decrease in LBP pain intensity from pre intervention mean(SD) of 2.7(0.9) RMVAS points to post test 2.2(0.9) RMVAS points. However the authors fail to discuss whether these statistical differences are clinically meaningful. When compared to the CG pre intervention mean(SD) of 2.4 (0.9) to post test 2.4 (0.8) RMVAS points, on average there was no change, and there was no significant difference between the CG and the Pilates group (Ostelo & de Vet, 2005) (Gladwell et al., 2006).

Interestingly there was a significant decrease (but probably not a clinically meaningful decrease) in functional disability (ODI) in the control group but not the Pilates group following the intervention. Functional disability for the CG at pre intervention was mean(SD) 24.1(13.4) and post intervention 18.1(13.0) ODI points. For the Pilates group ODI scores were 19.7(9.8) and 18.1(11.2) pre and post intervention respectively. This finding is not highlighted in the abstract or in the conclusions.

The authors state that it is difficult to determine if the positive gains were solely dependent on Pilates or other aspects of the intervention (Gladwell et al., 2006). It is unclear what these other aspects are, but participants were encouraged to continue with normal activity and pain medication. The authors appropriately suggest that their findings be interpreted cautiously as individuals respond to differing extents to the Pilates intervention. Of course this will be true of any heterogeneous NSCLBP population and any intervention. The modest number of participants who completed the study, and the imbalance of the number of participants in the two groups due to the higher drop out rate in the control group are a weakness of this study. For checklist of CONSORT statement criteria and other notes on this study please consult Appendix D.

A third study by Donzelli et al.(2006) compared Pilates to back school. This study was of poor quality and the methodology used was not typical of a Pilates rehabilitation programme (Rydeard et al., 2006) (Gladwell et al., 2006) (La Touche et al., 2008). This trial is described in the title as a randomized controlled trial, but allocation into the two groups (Pilates group and Back School) is done according to the time of day the participant can attend classes (Donzelli et al., 2006). This is not random allocation and therefore this study should not be considered as an RCT. Both the Pilates intervention, and the Back School, was delivered in ten 1-hour small group classes (max 7 people) delivered over ten consecutive days. Participants were then provided with a booklet outlining the exercises done in class and were asked to continue to practice at home. The expectations of frequency and duration of home practice were not reported. Follow up was undertaken at 1, 3, and 6-months.

Primary outcome measures were pain intensity (measured by VAS, 0-10) and functional disability (ODI, 1-100). Secondary outcome measures were a subjective rating of the treatment satisfaction and perceived benefit.

No baseline or demographic data for comparison of the two groups is presented with the exception of the mean age of all participants is given 50.08 years, and the mean age of males 49years and females 50.65years. The lack of demographic data, limits the readers ability to determine whether comparisons are being made on a similar basis (“apples vs apples or apples vs pears”) and also limits the reader’s ability to determine whether the participants studied are similar to the reader’s own clinical setting. The number of participants allocated to each group was Pilates group n= 22; Back School n=21. There is no further information about the mean age, or the distribution of men and women in each group. Participants were blinded to the intervention they were receiving. Two different
physicians did the pre-treatment and follow up examinations although this can lead to problems with inter-examiner reliability. It has been demonstrated that inter-operator reliability is poor (McGregor, McCarthy & Hughes, 1995). It is not clear if the assessor was blinded to which intervention the participant was assigned to, in order to prevent bias.

Another weakness of this study is that it only compares results in a descriptive way. Surprisingly, there is no statistical analysis of the results of primary outcomes within groups (pre compared with post intervention) and there is no comparison of results between groups. ODI and VAS were analyzed using frequency tests, and there were no p values, confidence intervals, or estimated effect sizes published. The mean ODI scores and VAS score for the two groups were combined and presented along with the results of the individual groups. The reasons for this are not apparent, nor described in the text.

The authors claim that the Pilates group demonstrated better compliance and subjective feelings of satisfaction compared to the Back School group, however, it is not clear if these differences are clinically important when compared to the Back School group. Moreover, subjective feelings of satisfaction must be considered as ‘soft’ outcome measures and are prone to suggestion and social influence of the investigators over participants. It was suggested by the authors that the Pilates intervention was more easily modified or personalized for participants during the small group sessions, and that this lead to increased compliance of home programme and improvement of symptoms. However, the results for primary outcomes (pain intensity and functional disability) appear to be similar for both groups. The Pilates group may have had better self-reported compliance, but this did not result in significantly better outcomes than Back School. Furthermore, as the two interventions were delivered using similar methods, it is difficult to see how one is more easily modified because that would be largely dependent on the therapist teaching the class.

For checklist of CONSORT statement criteria and other notes on this study please consult Appendix E.
**Critique of Curnow et al (2009)**

The most recently published study to investigate Pilates, Curnow et al. (2009), was published after the review by La Touche et al., (2008). The objective of Curnow et al’s (2009) study was to compare the effects of three different exercise regimen. In reality the three different regimens were very similar, and this obvious similarity probably explains why no significant differences were seen between the groups. The study population was divided into 3 groups; Group A (n=13), Group B (n=14), Group C (n=12). The exercise protocols consisted of 4 to 5 exercises each performed for 20-40 repetitions. This prescription is not consistent with standard Pilates rehabilitation principles which tend to limit the number of repetitions each exercise is undertaken in a single session to between 6 to10 repetitions with an emphasis on form and quality of movement. This restriction is to limit the effect of muscular fatigue and encourage quality of movement and precision – both thought to be important factors in developing new, ‘correct’ movement patterns (Richardson & Jull, 1995).

An absence of description of the characteristics of participants, or of the eligibility criteria, makes it difficult to define the sample population, and limits the reader’s ability to generalize findings to their own clinical setting. The only results discussed were the differences between the groups post intervention, which were not significant and certainly not clinically meaningful. There is no discussion of the differences within the groups’ pre-post intervention, the mean values for each group pre and post intervention are displayed graphically but there is no statistical analysis of this data presented (Curnow et al., 2009).

For checklist of CONSORT statement criteria and other notes on this study please consult Appendix F.

**Critique of La Touche et al (2008)**

This is a systematic review of the studies published prior to 2008 that used the Pilates method as a treatment for non-specific chronic low back pain. Included in the review were
Rydeard et al. (2006), Donzelli et al (2006), and Gladwell et al. (2006). The PEDro and Jadad Scales were used to review the methodological quality of each study. The conclusions that La Touche et al. made were based on the PEDro and Jadad ratings and were (ranked from highest quality to lowest), Rydeard et al (2006), Gladwell et al. (2006), and Donzelli et al (2006). La Touche et al (2008) concluded that further studies must be carried out (La Touche et al., 2008).

The results of the primary outcomes for three previous studies on Pilates for CLBP that were reviewed by LaTouche et al (2008) are presented in Table 2. Along with the results of Ferreira et al. (2007) which is a high quality RCT with a low risk of bias that compared general exercise, motor control exercise and spinal manipulative therapy (van Middelkoop et al., 2010).

Summary of primary studies investigating Pilates for treatment of CLBP

In conclusion it appears that Pilates is more effective in reducing pain and disability in patients with non-specific CLBP than usual care (Rydeard et al., 2006) (Gladwell et al., 2006) (La Touche et al., 2008). Although reductions in symptoms have been observed in previous studies, these improvements are not always of sufficient magnitude to be considered clinically important change (Curnow et al., 2009) (Gladwell et al., 2006) (Maughan & Lewis, 2010) (Ostelo & de Vet, 2005).

The superior result of Rydeard et al. (2006) when compared to Gladwell et al. (2006) raises many questions about the ideal application of Pilates based therapeutic exercise and its application as a therapy for CLBP populations. Firstly it is unclear from the current evidence whether Pilates based exercise undertaken on the mat, or the reformer, or a combination of both produces better results. Secondly the ideal frequency and duration of a Pilates based intervention is yet to be determined. And thirdly, it is unclear what contribution, if any, the
performance of home exercise adds to the effectiveness of a program of supervised intervention.

**Conclusion**

Low back pain is very common. Chronic low back pain and its associated disability are a major health problem and pose an economic burden to society. The majority of chronic low back pain (CLBP) is referred to as non-specific, and does not have a specific pathoanatomical diagnosis. There are many factors that contribute to the maintenance and persistence of LBP, and classification under the biopsychosocial model is necessary to acknowledge all contributing factors of the condition. Research on the effectiveness of various treatments for non-specific chronic low back pain indicates that there are few effective treatments available. This is, at least in part, due to patients with non-specific CLBP being part of a large heterogeneous group. Active therapies are recommended for the treatment of low back pain and the prevention of disability. There is some evidence that motor control exercises are effective in decreasing pain and functional disability in CLBP populations and the proposed mechanism of action for Pilates based therapeutic exercise is retraining motor control of the muscles around the lumbopelvic region. The benefits of private or individualised Pilates sessions compared to small group classes, as well as cost benefit ratio of each also need to be addressed in future work and the efficacy of Pilates compared to other treatment modalities for CLBP has yet to be evaluated. Research on the effectiveness of Pilates as a treatment option for CLBP is still in the early days, however, presently it appears that Pilates is a worthwhile treatment option for CLBP that warrants further investigation.

Section two of this thesis reports on the investigation of the effectiveness of a 6 week Pilates programme for adults with chronic low back pain.
References


Section 2: Manuscript
Pilates can decrease chronic low back pain and related functional disability
Abstract

Background

Recently, the popularity of Pilates has increased with both the general public and clinicians utilising therapeutic approaches developed from the Pilates method. The claimed benefits of Pilates include strengthening the ‘core’ musculature, decreasing low back pain, improving flexibility, and improved posture. To date there have been few studies undertaken to investigate the merit of these claims.

Objectives

The objective of this study was to evaluate changes in pain, functional disability and flexibility in adults with chronic low back pain following a 6-week Pilates exercise programme.

Methods and Measures

A pretest-post test single group study design was used. Data from 47 participants (n=31 females, n=16 males), between 25 – 65 years of age (mean age 41.25 years) was analysed. Participants attended a total of 12 Pilates sessions over a 6 week period arranged as two reformer and one mat sessions per week. Primary outcome measures were the 11-point numeric pain scale (NPRS, 0-10) to measure low back pain (LBP) intensity and any associated leg pain. The Patient Specific Functional Scale (PSFS, 0-10), and the Oswestry Disability Questionnaire (ODQ, 0-100) were used to assess levels of functional disability. Secondary outcome measures were the Troublesomeness Scale; and Schober’s Index, and Fingertip to Floor Test as an objective measure of flexibility of the lumbar spine, hips and hamstrings.
Results

After the intervention there was a significantly lower level of functional disability as measured by both PSFS and ODI (p≤0.001), and a decrease in average pain intensity (NPRS).

PSFS (0-10) score mean was 3.57±1.28 pre-intervention and 6.38±1.87 post-intervention (mean difference 2.81, p≤0.001, d=0.81). 63% of participants (n=30) experienced an improvement in PSFS scores of at least the minimum clinically important difference (MCID ≥ 2 points). The mean LBP intensity (0-10) was 4.73±1.75 at pre-intervention and decreased to 3.11±1.19 post-intervention (z=3.85, p≤0.001, d=0.55). Over half the participants (n=26) achieved a clinically meaningful outcome (achieved the MCID) for low back pain intensity.

Results also showed significant decrease in LBP troublesomeness (0-5), pre-intervention mean of 3.21±0.81, improving to a post intervention mean of 2.28±0.90, with a mean difference of 0.96 (95% CI: 0.64 to 1.23, p≤0.001).

Conclusion

These findings indicate that Pilates exercise is an effective treatment option in improving functional disability and decreasing pain and troublesomeness in adults with chronic non-specific low back pain. Further research should include comparing Pilates exercise with other interventions; and also compare the efficacy of group classes to more individualised one-on-one sessions with a instructor. The ideal frequency of classes and duration of trial period has yet to be determined, future research should also include an intervention over longer duration and longer term follow up periods.

Key words: Low back pain; chronic pain; Pilates; motor control; Patient Specific Functional Scale (PSFS); exercise therapy
INTRODUCTION

Low back pain (LBP) is a common condition with a lifetime prevalence of up to 90%, that is, up to 90% of all people will experience low back pain in their lifetime (Andersson, 1999). Low back pain is costly to workers compensation authorities (Firth, Herbison, McBride & Feyer, 2002). In New Zealand alone, the cost to the economy is estimated to be NZD$500 million per annum (McBride, Begg, Herbison & Buckingham, 2004). It is worth investigating new treatments and rehabilitation methods for lower back pain considering such a large proportion of the population are affected and its substantial economic impact in society. The prognosis for an initial onset of LBP is good, with the 90% of patients recovering within 12 weeks (Weiner & Nordin, 2010). The standard recommendation for people experiencing an episode of acute LBP is to stay active (Henchoz & Kai-Lik So, 2008), as lack of movement can lead to further loss of function and may lead to the development of chronic lower back pain (Henchoz & Kai-Lik So, 2008). It is this chronic population that accounts for a large proportion of the healthcare costs associated with back pain and it is therefore chronic back pain that has been the focus of most research in this area (Maher, 2004).

There is strong evidence that exercise can decrease pain, disability, secondary physical deconditioning and reduce time off work for people with chronic lower back pain (Henchoz 2008). Exercise therapy has been shown to be more effective than ‘usual care’ by a general practitioner (which includes staying active and taking analgesics as required), and just as effective as conventional physiotherapy (van Tulder, Malmivaara, Esmail & Koes, 2000). The use of graded exercise for treatment of chronic lower back pain has been demonstrated to decrease pain and improve functional ability by positively reinforcing healthy behaviors and activity levels (Leeuw et al., 2008). Graded exercise is when the amount of activity is gradually increased so that symptom aggravation is avoided. In graded Pilates exercise, once the participant has achieved a successful movement pattern without pain, the exercise
is progressed by decreasing the assistance and challenging the base of support (Anderson & Spector, 2000).

There are several published claims that Pilates exercises strengthen the ‘core’, increase flexibility, improve posture, and balance (Curnow, Cobbin, Wyndham & Boris Choy, 2009; Gladwell, Head, Haggar & Beneke, 2006). Furthermore, in people with CLBP, Pilates can improve pain levels, flexibility, proprioception and the perception of positive general health (Gladwell et al., 2006; Johnson, Larsen, Ozawa, Wilson & Kennedy, 2007). Initially, Pilates focuses on maintaining a ‘neutral spine’, along with activation of Transversus Abdominis (TrA) and pelvic floor muscles in combination with controlled breathing (Latey, 2002). The neutral spine position is when all the natural spinal curves are maintained (lumbar and cervical lordosis, and thoracic kyphosis) (Wallden, 2009). To maintain a neutral spine requires a ‘neutral pelvis’. Neutral pelvis is achieved when the anterior superior iliac spines (ASIS) are in the same transverse plane, and both ASIS and the pubic symphysis are in the same coronal plane (Sahrmann, 2002). As the sessions progress, mobility and articulation of the spine is encouraged, along with development of abdominal muscle endurance (deep and superficial) (Levine, Kaplanek, Scafura & Jaffe, 2007).

Studies have shown that people with chronic lower back pain (CLBP) do not recruit or properly engage the muscles that stabilise the lumbar spine and pelvis when performing movement tasks (Hodges, 1999a; Hodges 1999). Several muscles have been identified that act to stabilise the lumbar spine and pelvis. These include transversus abdominis (TrA), internal and external oblique, rectus abdominis (RA), lumbar multifidus, and muscles of the pelvic floor. Fitness and exercise industry professionals sometimes refer to these muscles collectively as ‘the core’, or in traditional Pilates, as ‘the powerhouse’ (Muscolino & Cipriani, 2004a; Muscolino & Cipriani, 2004b)
The aim of this study was to investigate the effectiveness of a 6-week programme of Pilates exercise in a sample of people with chronic low back pain (CLBP). The objectives of the study were to i) evaluate self-reported changes in low back pain intensity, functional disability, and troublesomeness in CLBP sufferers following a 6-week Pilates exercise programme; and ii) examine objective changes in flexibility in lumbar spine, hips, and hamstrings in adults with CLBP following a 6-week Pilates exercise programme.

METHODS

Participants

Participants were recruited over a 4½-month period, through word of mouth and flyers posted on public notice boards, in sports clubs, and on shop windows. In order to increase the homogeneity of the sample participants were required to be between 25 and 65 years of age with current low back pain of at least 6 months duration or current low back pain of less than 6-months duration with repeated pain episodes in the last year. For the purpose of this study CLBP was defined as the presence of low back pain for a duration of at least 6-months, or recurring low back pain occurring in multiple episodes over the previous 12-months (Stanton, Latimer, Maher & Hancock, 2009).

Participants who responded to advertising were sent an information sheet about the study. After reading the information those interested in taking part were invited to email or phone to confirm their interest. A screening interview over the phone was then conducted to determine eligibility to take part. Exclusion criteria were: nerve root compression signs including leg pain to the knee or below; recent spinal fracture, tumour, or infection; abdominal or spinal surgery in the 12 months prior; osteoporosis; pregnancy (known or expected); co-morbidities or contraindications to exercise.
Participants also had to understand written and spoken English; be available to attend two 1-hour classes per week for 6-week intervention period; and have no previous experience with the Pilates method. If the participant was eligible then an appointment for pre-intervention data collection was made. Baseline measures were taken during the week prior to commencement of the intervention.

**Study Design**

A pre-test – post-test single group design was used.

**Assessments**

In the week prior to the commencement of the intervention all participants were required to attend an appointment for a physical examination (including neurological screening), and pre-intervention data collection. At this appointment all demographic and anthropomorphic information was gathered including age, gender, height and weight (BMI was calculated later). Schober’s index and fingertip to floor test was measured as an objective indication of lumbar spine, hip and hamstring flexibility. Pain intensity, functional disability, and troublesomeness were all measured using standardised questionnaires.

**Outcome Measures**

All outcomes were measured pre and post intervention. All post intervention measures were undertaken one week following the completion of the intervention.

**Pain Intensity**

*11-point Numeric Pain Rating Scale (NPRS)*

The NPRS was used to measure low back and leg pain intensity. The NPRS ranges from 0 being equal to no pain, and 10 being the worst pain imaginable (Farrar, Young, LaMoreaux, Werth & Poole, 2001). Participants were asked to rate how ‘bothersome’ their pain had
been in the past week by circling a number on the scale of 0 – 10. The minimum clinically important difference (MCID) for NPRS is change of ≥2 points (Childs, Piva & Fritz, 2005; Farrar et al., 2001).

**Troublesomeness Scale**

Troublesomeness was used as an overall indicator of pain and how bothersome it was to the subject, in all regions of the body including the lower back. There are 13 regions of the body represented on the scale, ranging from headache or neck pain to ankle/foot pain and everything in between (Parsons et al., 2006). The scale extends from 0 being ‘no pain experienced’, to 5 being ‘extremely troublesome’. The Troublesomeness Scale provides a total (whole body) troublesomeness score out of 65, and a LBP troublesomeness score out of 5. The minimum clinically important difference (MCID) for troublesomeness has not yet been determined.

**Functional Disability**

**Patient Specific Functional Scale**

Patient specific Functional Scale (PSFS) was used to measure LBP related disability. The PSFS asks participants to identify at least three, and up to five, important activities that they are unable to do or have difficulty doing as a result of their LBP. They are then asked to score on a scale of 0-10 how easily they can perform the activities. Where 0 = unable to perform activity, and 10 = able to perform the activity as before injury or problem. The scores of the selected activities were then averaged to calculate a mean score. The minimal detectable change for PSFS mean is 2 points, and for a single PSFS activity, 3 points Stratford 1995. The MCID for mean PSFS is reported to be 2 points (Maughan & Lewis, 2010). The MCID for a single PSFS activity has yet to be determined. To allow comparison with previous studies (eg Ferreira (2007)) a total score out of 30 was calculated, where three activities were totalled.
For those participants that selected more than 3 activities a best and worst case was determined for data analysis.

**Modified Oswestry Disability Index**

The Modified Oswestry Disability Index (ODI) is another measure of functional disability (Fairbank & Pymsent, 2000). The ODI questionnaire is designed to show how the subject’s low back pain affects their ability to carry out activities of daily living (ADLs). Subjects are asked to choose the statement that best describes their condition today. There are 10 questions about different ADLs, subjects can select from 6 statements to describe how their LBP has affected each activity. The version of Oswestry used was also utilised in (Fritz & Irrgang, 2001). Each question is scored from 0 to 5 giving a total score out of 50; the score is then doubled to give a score out of 100 or a percentage. The MCID for the ODI is 10 points (Ostelo & de Vet, 2005) (Ostelo et al., 2008). A change of 50% has also been suggested as an MCID for a chronic low back pain population (Fritz, Hebert, Koppenhaver & Parent, 2009).

**Flexibility**

**Fingertip to Floor**

Fingertip to floor (FTF) is a reliable and accurate measure of gross lumbar spine, hip, and hamstring flexibility (Perret et al., 2001). Participants stand on a 20cm box with feet together and legs straight and reach as towards the ground, the distance between the middle finger and the floor is measured as interpreted as: < 20cm = below ground level, 20cm = ground level, >20cm = number of centimetres distance from the ground + 20cm. The minimum clinically important difference for FTF has not been reported in the literature.

**Schober’s Index**

The Schober’s Index is used to indicate flexibility of forward being of the lumbar spine. A mark is made in the midline 5cm below the level of the posterior inferior iliac spine (PSIS) and another 10cm above the level of the PSIS. The subject is then instructed to flex
forwards as if to touch ones toes, and the distance between the marks is measured. Normal is considered to be 20cm when in full lumbar spine flexion, less than this is an indication of decreased range of motion or stiffness in the lumbar spine (Waddell et al., 1982). Fifteen centimetres (the original distance of the marks) is subtracted from the distance between the two marks when in full flexion, to give a value for flexibility of the lumbar spine in centimetres (Perret et al., 2001).

**Intervention**

The intervention was designed so that each subject would take part in one mat class and one reformer class each week, for 6 weeks. Wherever possible, participants were scheduled to attend sessions at intervals of at least one day, and no more than 4 days between classes. Occasionally it was impossible for the subject to have a gap day in between classes, and in very rare circumstances some subjects participated in 2 classes (mat and reformer) on the same day. If for some reason subjects were unable to take part in two classes in one week, they were scheduled to make up this class in the following week. All participants completed the 12 classes required, with the desired ratio of mat to reformer classes. The majority of participants completed in six weeks, with 6 participants taking 7 weeks and 1 participant taking part in 12 classes over a shorter period of 5 weeks.

Every participant attended one mat class and one ‘reformer’ class per week. The mat classes were performed on exercise mats on the floor using supine, side lying, or 4-point kneeling positions. Basic ‘props’ including cushions, resistance circle, balls, resistance bands, gravity and the participant’s own body weight were used to provide resistance and/or assistance (if required) for the performance of mat exercises and stretches. The ‘reformer’ is a spring-loaded carriage that can be used either to support or to challenge the subject during exercises (Figure 1). The size of the classes ranged from (min-max) 2 to 7 for the reformer classes to 5 to16 for the mat classes.
The exercises were graded for difficulty and were progressed across weeks 1 to 6. The intended purpose of the exercise intervention was to improve control of movement and body awareness. Exercises were progressed from initial body awareness in a supine position identifying a neutral spine position and contracting pelvic floor and deep abdominal muscles in isolation, to maintaining control of a ‘neutral spine position’ and perceived activation of deep abdominal wall muscles whilst performing more dynamic tasks that involved the larger more superficial global muscle groups. Exercises that were intended to promote controlled spinal mobility, and improve flexibility of the hips and adjacent muscles were included in all sessions.

The exercises on the reformer were predominately in the sagittal plane, with flexion of the hip, knee, ankle, trunk and shoulder all practiced whilst maintaining a neutral spine. More challenging exercises that incorporated control of a neutral spine in a seated and standing position were also practiced on the reformer in the latter stages of the intervention. In the mat class, exercises that encouraged gentle flexion, rotation and extension of the lumbar and thoracic spine were included.
Statistical Analysis

Data was analysed using SPSS version 17 (SPSS Inc, Chicago, IL). The data for all outcome measures was explored for normality by inspecting plots of raw data and by calculating measures of skewness and kurtosis; and the Shapiro-Wilk statistic. Oswestry Disability Index, LBP Troublesomeness, Leg Pain, and Fingertip to Floor were all normally distributed. PSFS, LBP, Total Troublesomeness, and Schober’s Index were not normally distributed. Pre and post intervention scores for normally distributed data were analysed using paired sample t-tests. Non-normally distributed data was analysed using Wilcoxon Signed Ranks Test. Effect sizes for all contrasts of normally distributed variables were calculated using Cohen’s $d$ (Cohen, 1988), and 95% confidence intervals for mean differences were calculated. Effect sizes for non-parametric data were calculated using $r = z/\sqrt{n}$. All effect sizes were interpreted using Cohen’s suggested descriptors (Cohen, 1988). Throughout the text means are presented as mean (±SD). Post-hoc power analysis was undertaken using G*Power v3.0 (Erdfelder, Faul & Buchner, 1996)
RESULTS

Of the 66 people interviewed, 59 were eligible to take part in the study. 3 people did not commence the intervention; therefore 56 participants were enrolled in the study. For logistical reasons the participants received the intervention in three groups. Of the 56 who commenced the intervention, 6 dropped out leaving datasets for 50 participants. Of these 50, 3 datasets were excluded from analysis (1 because they had no back pain at pre-intervention and were incorrectly enrolled; 1 because their back pain was attributable to pelvic pathology subsequently confirmed by surgery; and 1 because of spoiled data collection sheets), therefore 47 datasets were analysed (n=31 females; n=16 males). The flow of participants through the study is illustrated in figure 2. For information about the study population see Tables 1 and 2.

Table 1. Baseline Characteristics of Study Population

<table>
<thead>
<tr>
<th></th>
<th>Participants n=47</th>
<th>Range (min – max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) mean (sd)</td>
<td>41.25 (10.70)</td>
<td>25 – 65 years</td>
</tr>
<tr>
<td>Gender N (%)</td>
<td>16 (34%) Male</td>
<td>31 (66%) Female</td>
</tr>
<tr>
<td>Body Mass Index (sd) kg/m2</td>
<td>26.2 (4.6)</td>
<td>20.3 – 41.6 kg/m2</td>
</tr>
<tr>
<td>Duration of LBP (years) mean (sd)</td>
<td>9.0 (7.44)</td>
<td>0.5 – 30 years</td>
</tr>
<tr>
<td>6-12 months N (%)</td>
<td>6 (12.8%)</td>
<td>6 – 360 months</td>
</tr>
<tr>
<td>13-36 months</td>
<td>8 (17.0%)</td>
<td>(median96 months)</td>
</tr>
<tr>
<td>&gt; 36 months</td>
<td>33 (70.2%)</td>
<td></td>
</tr>
<tr>
<td>Current LBP NPRS (0-10) mean (sd)</td>
<td>4.66 (1.75)</td>
<td>1 – 8 NPRS</td>
</tr>
<tr>
<td>Associated Leg Pain N (%)</td>
<td>12 (25.5%)</td>
<td></td>
</tr>
<tr>
<td>Associated Leg Pain NPRS mean (sd)</td>
<td>0.85 (1.77)</td>
<td>0 – 9.0 (median 0)</td>
</tr>
<tr>
<td>Pain Medication Yes N (%)</td>
<td>6 (12.7%)</td>
<td></td>
</tr>
<tr>
<td>Education Level Completed N (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High School (or equivalent)</td>
<td>15 (31.25%)</td>
<td></td>
</tr>
<tr>
<td>Tertiary degree or diploma</td>
<td>23 (48.9%)</td>
<td></td>
</tr>
<tr>
<td>Postgraduate degree completed</td>
<td>9 (18.75%)</td>
<td></td>
</tr>
<tr>
<td>Work Status N (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full-time</td>
<td>31 (66%)</td>
<td></td>
</tr>
<tr>
<td>Part-time</td>
<td>8 (16.6%)</td>
<td></td>
</tr>
<tr>
<td>Not working</td>
<td>8 (16.6%)</td>
<td></td>
</tr>
</tbody>
</table>
**Table 2. Study Population**

<table>
<thead>
<tr>
<th></th>
<th>Total Participants (n)</th>
<th>Drop outs</th>
<th>Excluded Post Intervention</th>
<th>Successfully Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females</td>
<td>39</td>
<td>5</td>
<td>3</td>
<td>31</td>
</tr>
<tr>
<td>Males</td>
<td>17</td>
<td>1</td>
<td>0</td>
<td>16</td>
</tr>
<tr>
<td>Total</td>
<td>56</td>
<td>6</td>
<td>3</td>
<td>47</td>
</tr>
</tbody>
</table>

**Figure 2. Subject flow during the study**

- **Potential participants n=66**
  - Excluded n=7 (Did not meet criteria)
- **Eligible n=59**
  - Dropped out n=3 (Prior to intervention commencing)
- **Participants n=56**
  - Dropped out n=6
- **Completed Intervention n=50**
  - Excluded n=3 (Did not meet criteria)
- **Analysed n=47**
**Power**

Post hoc analysis to identify achieved power for the primary outcome of ODI revealed an achieved power of 0.94 (tails = 2; observed effect size = 0.53; \( \alpha = 0.05 \); \( n = 47 \)).

The means and standard deviations for all outcome measures pre and post-intervention are presented in Table 3 and 4.

**Table 3. Pre – post changes for normally distributed data**

<table>
<thead>
<tr>
<th></th>
<th>Mean Pre</th>
<th>SD Pre</th>
<th>Mean Post</th>
<th>SD Post</th>
<th>Mean Difference</th>
<th>95% Confidence Intervals</th>
<th>P-value</th>
<th>Effect Size</th>
<th>Descriptors</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBP Troublesomeness</td>
<td>3.21</td>
<td>0.81</td>
<td>2.28</td>
<td>0.90</td>
<td>0.96</td>
<td>0.64 - 1.23</td>
<td>≤0.001</td>
<td>1.09</td>
<td>Large</td>
</tr>
<tr>
<td>Oswestry (ODI)</td>
<td>19.36</td>
<td>10.86</td>
<td>13.79</td>
<td>10.04</td>
<td>5.58</td>
<td>3.28 - 7.86</td>
<td>≤0.001</td>
<td>0.53</td>
<td>Moderate</td>
</tr>
<tr>
<td>Fingertip to Floor</td>
<td>22.19</td>
<td>12.27</td>
<td>18.57</td>
<td>10.31</td>
<td>3.62</td>
<td>1.95 - 5.28</td>
<td>≤0.001</td>
<td>0.37</td>
<td>Small</td>
</tr>
<tr>
<td>Leg Pain (NPRS)</td>
<td>0.85</td>
<td>1.77</td>
<td>0.98</td>
<td>1.91</td>
<td>-0.13</td>
<td>-0.64 - 0.39</td>
<td>≤0.001</td>
<td>0.07</td>
<td>Trivial</td>
</tr>
</tbody>
</table>

\( a \) Effect sizes for parametric data were calculated using the Cohen statistic \( d = \frac{\text{mean}_a - \text{mean}_b}{\text{SD}} \) (Cohen, 1988).

Descriptors for magnitudes of effect are based on those described by Cohen (Cohen, 1988)

**Table 4. Pre – post changes for non-normally distributed data**

<table>
<thead>
<tr>
<th></th>
<th>Mean Pre</th>
<th>Median Pre</th>
<th>SD Pre</th>
<th>Mean Post</th>
<th>Median Post</th>
<th>SD Post</th>
<th>P-Value</th>
<th>Z</th>
<th>Effect Size</th>
<th>Descriptors</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSFS Average</td>
<td>3.57</td>
<td>3.5</td>
<td>1.28</td>
<td>6.38</td>
<td>6.0</td>
<td>1.87</td>
<td>≤0.001</td>
<td>-5.58</td>
<td>0.81</td>
<td>Large</td>
</tr>
<tr>
<td>LBP (NPRS)</td>
<td>4.66</td>
<td>4.0</td>
<td>1.75</td>
<td>3.11</td>
<td>2.0</td>
<td>1.91</td>
<td>≤0.001</td>
<td>-3.76</td>
<td>0.55</td>
<td>Moderate</td>
</tr>
<tr>
<td>Total Troublesomeness</td>
<td>15.57</td>
<td>16.0</td>
<td>8.16</td>
<td>11.60</td>
<td>10.0</td>
<td>7.37</td>
<td>≤0.001</td>
<td>-4.12</td>
<td>0.610</td>
<td>Moderate</td>
</tr>
<tr>
<td>Schober’s Index</td>
<td>5.62</td>
<td>6.0</td>
<td>1.28</td>
<td>5.28</td>
<td>6.0</td>
<td>1.37</td>
<td>0.144</td>
<td>-1.45</td>
<td>0.21</td>
<td>Small</td>
</tr>
</tbody>
</table>

\( a \) based on negative ranks
\( b \) based on positive ranks

Effect sizes for non-parametric data were calculated using \( r = \frac{z}{\sqrt{N}} \) where \( n = 47 \).

Descriptors for magnitudes of effect are based on those described by Cohen (Cohen, 1988)
**Functional Disability**

The Oswestry Disability Index (ODI, 0-100) and Patient Specific Functional Scale (PSFS) are both measures of functional disability. For ODI the pre and post-intervention means (SD) were 19.36(10.86) and 13.79(10.04) respectively, this demonstrates on average a decrease in the level of functional disability, with a mean difference of 5.58 (95% CI = 3.28 to 7.86; p≤0.001; \(d=0.53\) “moderate effect”). The minimum clinically important difference (MCID) for ODI is 10 points (Ostelo & de Vet, 2005), or 50% change for a more chronic population (Fritz et al., 2009). Fourteen participants achieved a decrease of 10 points or more, and sixteen participants demonstrated a ≥50% decrease in ODI score between baseline and at 7 weeks (1 week post-intervention).

At baseline the mean (SD) PSFS score was 3.5(1.3) and improved to 6.3(2.0) following the intervention (Wilcoxon sign rank test \(p \leq 0.001\)). Thirty participants demonstrated a clinically important improvement (≥2 points) in average PSFS score, 17 participants had change in PSFS scores of ≤2 points. The number of participants who achieved MCID in single activities is presented in Table 5. Twenty-six participants achieved MCID (≥3 points) improvement in 3 or more activities, 7 participants in 2 activities, and 5 people with 1 activity. There were 9 people who did not experience a clinically important change in any activity.

**Table 5. Minimum Detectable Change for PSFS individual activities**

<table>
<thead>
<tr>
<th>Change ≥ 3 points PSFS Single activity (MCID)</th>
<th>Number of Participants</th>
<th>% of Study Population (n=47)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 activities</td>
<td>26 people</td>
<td>55.32%</td>
</tr>
<tr>
<td>2 activities</td>
<td>7 people</td>
<td>14.89%</td>
</tr>
<tr>
<td>1 activity</td>
<td>5 people</td>
<td>10.64%</td>
</tr>
<tr>
<td>0 activities</td>
<td>9 people</td>
<td>19.15%</td>
</tr>
</tbody>
</table>

MCD = minimal detectable change; PSFS = Patient Specific Functional Scale
When the scores of three activities are totalled this gives a PSFS value of between 3-30 (Ferreira et al., 2007). If the participants selected more than three activities then a best and worst case scenario was calculated. The best-case scenario represented the greatest magnitude of positive change, and the worst case the least magnitude of positive change or negative change. In the best case the average pre intervention PSFS score was 10.66(4.26) this increased to 20.30(5.68) post-intervention (mean difference 9.64. 95% CI: 7.61-11.67; p= ≤0.001, d=1.93), for the worst case the PSFS score was 10.96(3.88) pre intervention and 18.36(6.06) post intervention (mean difference 7.40, 95%CI: 5.44-9.37; p= ≤0.001, d=1.49). In both cases a larger post intervention PSFS score indicates an improvement in functional disability, and a ‘large’ effect.

Table 6. Pre – post changes for PSFS Total

<table>
<thead>
<tr>
<th></th>
<th>Mean Pre</th>
<th>SD Pre</th>
<th>Mean Post</th>
<th>SD Post</th>
<th>Mean Difference</th>
<th>95% Confidence Intervals</th>
<th>P-value</th>
<th>Effect Size</th>
<th>Descriptors</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSFS Total Best Case</td>
<td>10.66</td>
<td>4.26</td>
<td>20.30</td>
<td>5.68</td>
<td>9.64</td>
<td>7.61-11.67</td>
<td>≤0.001</td>
<td>1.93</td>
<td>Large</td>
</tr>
<tr>
<td>PSFS Total Worst Case</td>
<td>10.96</td>
<td>3.88</td>
<td>18.36</td>
<td>6.06</td>
<td>7.40</td>
<td>5.44-9.37</td>
<td>≤0.001</td>
<td>1.49</td>
<td>Large</td>
</tr>
</tbody>
</table>

a Effect sizes for parametric data were calculated using the Cohen statistic $d = \text{mean}_\alpha - \text{mean}_\beta / \text{SD}$. Descriptors for magnitudes of effect are based on those described by Cohen (Cohen, 1988)

**Troublesomeness**

A comparison of the mean LBP Troublesomeness (0-5) scores between pre and post-intervention measures for all participants (n=47) revealed a substantial decrease in LBP Troublesomeness, from pre-intervention mean(SD) 3.21(0.81) points compared to post-intervention 2.28(0.90) points. The difference between means was 0.96 points (out of maximum =5) (95% CI for difference = 0.64 to 1.23; paired t-test; p≤0.001; d=1.09 “large effect”).
**Pain Intensity**

LBP intensity measured by NPRS (0-10) was lower post-intervention (mean 3.17, med 2.00) compared with pre-intervention scores (mean of 4.73, med 4.0), $z=3.85$ ($p≤0.001$). This demonstrates a decrease in LBP intensity and a ‘moderate’ effect ($d=0.55$). Twenty-six subjects achieved a change at the level of the MCID (2 NPRS points) or better. The post intervention level of LBP intensity (NPRS), along with a tally of participants that had a clinically meaningful change over the course of the intervention is presented in Table 7.

**Table 7. Low Back Pain Intensity following a 6-week Pilates Intervention**

<table>
<thead>
<tr>
<th>Post Intervention NPRS Score (0-10)</th>
<th>Number of Participants</th>
<th>Number of Participants who achieved MCID</th>
<th>% of Study Population (n=47)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>6</td>
<td>6</td>
<td>12.7%</td>
</tr>
<tr>
<td>2-3</td>
<td>28</td>
<td>19</td>
<td>40.4%</td>
</tr>
<tr>
<td>4-5</td>
<td>5</td>
<td>1</td>
<td>2.1%</td>
</tr>
<tr>
<td>≥ 6</td>
<td>8</td>
<td>0</td>
<td>0.0%</td>
</tr>
<tr>
<td>Totals</td>
<td>47</td>
<td>26</td>
<td>55.3%</td>
</tr>
</tbody>
</table>

MCID = a decrease of ≥2 NPRS Points, NPRS= Numeric Pain Rating Scale

**Leg Pain**

There is a 95% chance that true mean difference for leg pain (0-10) in the general population mean lies between -0.64 and 0.39 NPRS points (CI). The confidence interval for mean change spans zero indicating a range of possible responses.

Of the 18 people that reported having leg pain pre-intervention, not all of them had actual leg pain (outside of the LBP area). On inspection of pain drawings on body diagram at enrolment it was revealed that; 6 people did not have leg pain that was related to their back pain; 2 people had possible signs of nerve root irritation (but no weakness in the lower
extremity was present); and 10 people did have leg pain. The results for leg pain are presented in Table 8.

**Table 8. Pre – post changes for Leg Pain (NPRS)**

<table>
<thead>
<tr>
<th>n=10</th>
<th>Mean Pre</th>
<th>SD Pre</th>
<th>Mean Post</th>
<th>SD Post</th>
<th>Mean Difference</th>
<th>95% Confidence Intervals</th>
<th>P-value</th>
<th>Effect Size</th>
<th>Descriptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leg Pain (NPRS)</td>
<td>3.5</td>
<td>2.17</td>
<td>2.7</td>
<td>2.71</td>
<td>0.8</td>
<td>-1.07</td>
<td>2.67</td>
<td>0.36</td>
<td>0.32</td>
</tr>
</tbody>
</table>

Table 8. Pre – post changes for Leg Pain (NPRS)

Of the participants that did have leg pain at baseline (but no neurological signs), n=10. Five people reported a clinically important decrease in leg pain post (≥ 2 NPRS points), and 3 of them reported no leg pain post intervention (0 NPRS points). Three people had minimal improvement or had no change, and 2 people reported an increase in leg pain post.

Of the people that reported no leg pain at the start of the trial (n=37), seven people reported having leg pain after the intervention. However, a body diagram did not accompany the follow up question regarding the presence of leg pain so verification of location of pain was not possible. One participant experienced a traumatic onset of leg pain (whilst playing squash) two days prior to measurement of final outcomes. In this case leg pain = 6 NPRS points, and is thought to not be related to the participation in the intervention. The range for all cases of new leg pain post intervention was 1-6 NPRS points.

**Flexibility**

Results showed significant improvement (P<0.001) in all outcome measures except Leg Pain and Schober’s Index. This is not surprising considering that leg pain (to or below the knee) was one of the exclusion criteria, and that the mean score for Schober’s Index pre and post intervention was within the normal range, which is anything over 5cms. There were 11
individuals that were outside the normal range, but 10 of them (over 90%) were within 10% (2 cm) of the norm. Over 70.0% (8 people) of those outside the normal range for Schober’s Index demonstrated improved lumbar spine flexion following the intervention, with 4 people scoring within the normal range (over 5cm) post intervention. Three participants (27.27%) that had pre scores of 4.0 – 4.5 cms experienced no change, a slightly higher rate than the total population where 22.9% experienced no change in Schober’s Index. Two participants experienced a decrease in Schober’s Index score; one of these participants had possible nerve root compression signs pre-intervention and probably should have been excluded, however, they did experience a decrease in their leg pain score post intervention. The other suffered an injury playing squash in the week between the intervention and the re-examination of outcome measures leading to an acute aggravation of symptoms.

The effect of the intervention on forward bending flexibility was measured using the ‘Fingertip to floor’ procedure (gross measure of hamstring, hip and lumbar spine flexibility) was small ($d=0.36$) but significant ($P≤0.001$). The mean pre-intervention score was 22cm from the ground, but participants were standing on a 20cm box, so it is actually 2cm above ground level. The effect on flexibility of the lumbar spine as measured by the Schober’s Index and leg pain (measured by NPRS) by the intervention was ‘trivial’ ($d≤ 0.2$), and not significant ($P=0.58$ and 0.20 respectively).
DISCUSSION

The aim of this study was to investigate the effect of a 6-week Pilates based exercise programme on pain and functional disability in a chronic non-specific low back pain population. To date there have been no other studies evaluating the outcome of a Pilates programme delivered in small group classes that utilised a combination of both Pilates equipment (such as the reformer) and mat based exercises.

Main findings

The main finding of this study was a substantial decrease in low back pain and functional disability following a 6-week programme of Pilates exercise in adults with chronic low back pain. This is consistent with the outcomes of other studies that have trialled Pilates (Gladwell et al., 2006) (Rydeard, Leger & Smith, 2006), and other types of therapeutic exercise (Ferreira et al., 2007) in non-specific chronic low back pain populations. The results of this study are also in agreement with a systematic review of the literature (van Tulder et al., 2000).

In most cases participants completed the 12 sessions in the 6-week period allocated. Ideally they participated in 2 classes per week, one of each type (mat and reformer). If for whatever reason they were unable to attend a class they were encouraged to do a make up class within the 6 weeks. This was not possible for seven participants who took at most four days to complete the 12 sessions. This was probably related to an increased incidence of illness during the winter months of the data collection period. One participant completed the intervention in 5 weeks, as they had to undergo surgery (unrelated to LBP) in the sixth week.
**Comparison with previous similar studies**

In comparison to two other studies (both RCTs) that have investigated the efficacy of Pilates in a CLBP population, the mean age of the current study population was slightly older (the inclusion criteria for Rydeard et al (2006) was age 20-55, the maximum age is 10 years younger than the oldest participant included in our sample). The mean duration of LBP was similar to that of the population in the study by Gladwell et al. (2006) and the control group in Rydeard et al. (2006). The range for duration of symptoms was similar in our group to the Pilates group in Rydeard et al’s study, highlighting the wide variance/ heterogeneity of the non-specific CLBP population. The current study had slightly higher mean pain intensity pre intervention, and a larger sample size that received the intervention (n=47). In the sample population there were more females than males (31 females, and 16 males), and the majority of participants had a higher level of education (all graduated high school or equivalent) compared to Rydeard et al.’s study.

The results for the current study showed greater improvements in pain intensity and disability (ODI) than the Gladwell Pilates group who reported an increase in mean ODI post intervention. Both studies were for a 6-week duration, however, the Gladwell group completed half as many classes in that time (6 classes), and practiced only mat exercises, in class and at home.

The changes seen in the current study are difficult to compare to the study by Rydeard et al. (2006) because of the different outcome measures used. Their chosen outcome for LBP related disability was the Roland Morris Disability Questionnaire (RMD-HK), and for LBP intensity the NPS (0-100). They observed a decrease in both outcomes; RMD-HK pre-intervention mean(SEM) 3.1(0.6) post 2.0(0.3) p=0.023; NRS pre-intervention 23.0(3.9) post 18.3(3.2) p=0.002 (n=21). Their sample population was also in the lowest strata for functional disability (RMD-HK). It has been suggested that the MCID for patients in the
lowest strata is 1-2 RMDQ points, so these results may indicate a clinical meaningful change (Stratford, Binkley, Riddle & Guyatt, 1998) although most authors consider that the MCID is 4-5 RMDQ points for higher levels of disability. All participants completed the 4-week trial of three 1-hour Pilates sessions per week (total = 12 sessions), plus home practice (Rydeard et al., 2006).

Ferriera et al. (2007) carried out a RCT that compared general exercise (GE), to motor control exercise (MCE), to spinal manipulative therapy (SMT), 12 sessions delivered over 8 weeks. Their results favoured MCE and SMT in the short term, but the authors concluded that there was no significant difference between the 3 groups at 6 and 12 months, and therefore there was no long-term benefit in choosing one treatment option over another (Ferreira et al., 2007). The results of the current study were slightly better in terms of improvement in functional disability as measured by PSFS. Pre intervention the PSFS Total scores (0-30) for MCE, SMT, and Pilates (best and worst case), were all within the range of 10.7-11.2 PSFS points. Post intervention (short term follow up) the Pilates group had greater improvement than MCE (17.7±6.2) and SMT (17.5±6.2) PSFS post intervention. An explanation for this could be that only 73 and 77 participants completed the trial for MCE and SMT respectively but 80 data sets were analysed for each group. The analysis approach that Ferriera et al. (2007) used was to carry forward the baseline measure and use it as the post score also, this would result in less change in the mean score, and therefore these results can be seen as a conservative representation of the effect of these treatments. At short term follow up the MCE and SMT group had greater decrease in pain intensity (mean difference) when compared to the current study. However, this could be due to higher levels of pain at baseline and possibly greater potential to change. The average level of pain intensity for the current study was lower than that of all groups of the Ferreira et al. (2007) trial (pre and post intervention).
**Study strengths**

The participants of this study could be considered typical of a non-specific chronic low back pain population. The heterogeneity of the group including a wide variation of duration, onset, and intensity of low back pain between participants is to be expected in a community sample of NSCLBP sufferers. This group may be typical of the type of patients that would present to private practitioners (eg physiotherapy, chiropractic, osteopathy) for treatment of disability and pain, despite having returned to work and most of their normal activities following an acute episode.

The use of two outcomes measures for evaluating changes in self-reported functional disability, enables comparison of the results of the current study to larger randomised controlled trials, and through use of the PSFS allows detection of clinically meaningful change in individual participants.

The Oswestry Disability Index is one of the most commonly used outcome measures in low back pain research, and has good reliability and validity (ICC >0.8) (Davidson & Keating, 2002). The minimum clinically important difference (MCID) for ODI is reported to be between 4 and 16 points (Lauridsen, Hartvigsen, Manniche, Korsholm & Grunnet-Nilsson, 2006). Recently a panel of experts in the field of back pain research proposed the MCID for ODI as 10-points, this is in agreement with and fits in the range reported by Lauridsen et al. (2006) of between 8 and 11 ODI points for LBP patients with and without leg pain (Ostelo et al., 2008; Ostelo & de Vet, 2005). Most recently a change of 50% (from baseline score) was suggested as MCID for a chronic back pain population (Fritz et al., 2009).

One of the main criticisms of ODI is that it may not be sensitive enough to detect change in populations with lower levels of disability (Dawson, Steele, Hodges & Stewart, 2009). For this reason others have recommended the use of Roland Morris Disability Questionnaire (RMDQ), which is thought to be slightly more sensitive (Müller, Röder & Greenough, 2006;
Pincus et al., 2008). Another common criticism of ODI and other disability measures is that they are generic and may not be relevant to the individual patient. Therefore the PSFS has been developed to enable the use of context specific measures for individual participants. Scores obtained from the PSFS correlate well with those of the RMDQ, indicating good construct validity as a outcome questionnaire for LBP (Stratford, 1995; Vaughan & DiVenuto, 2004). The current study used PSFS to detect change in patient functioning that may be deemed more relevant to each participant’s own context than the activities included in the ODI or RMDQ.

The majority of participants in this study fell within the lowest strata of ODI scores from 0-20 (minimally disabled) and there were eight people who scored below the MCID of 10 ODI points at baseline. The inclusion of PSFS made it possible to detect clinically important change in a minimally disabled population of people with CLBP.

The fact that there were significant differences (p≤0.001) in the levels of LBP troublesomeness and to a lesser extent total troublesomeness (full body), and not a significant decrease in leg pain (p=0.62) indicates two things: Firstly, that the intervention targeted LBP; and secondly that the population sampled were mainly troubled by their LBP and largely did not suffer from other more generalised chronic pain syndromes.

**Study weaknesses**

It is uncertain how many people that reported having leg pain post intervention actually had leg pain post intervention. The reason for this is that a pain diagram was not used post intervention to clarify the distribution of symptoms as had been done pre-intervention. Despite use of a pain diagram two participants were incorrectly enrolled that had signs of neurological irritation. One had an area of changed sensation over the posterior aspect of the thigh and calf (to the heel), and the other reported pain down the posterior aspect of the leg and numbness along the lateral border of the foot, but neither had any objective
weakness of the lower extremity when tested. Both reported improvement in leg pain post intervention, from 3 to 2, and 2 to 0 NPRS points respectively. The finding of the current study was that the affect of the intervention on leg pain was trivial, and at least in these two participants with leg pain didn’t appear to exacerbate leg pain.

**Limitations of the study**

The major limitation of this study is the lack of a control group, however, it is unlikely that the improvements noted are explained simply by the passage of time (‘maturation bias), because of the long duration of back pain that the majority of participants in our sample reported prior to starting the trial (mean (SD) duration of LBP 9.0(7.4) years)

A wait-list control design was considered, however, the logistics and time constraints for completion of the study lead to the use of a single group design. This type of study is considered to be the weakest for assessing change, because there is no group for comparison (Stratford et al., 1996). However, considering the aim of the study was to examine if this therapy was effective, not necessarily if it was more effective than another type of therapy, this design was adequate.

**Clinical implications**

The criteria for inclusion and exclusion in the study selected a community based sample that are typical of non-specific CLBP sufferers who present to health care professionals for treatment for ongoing back pain.

Most participants had a low level of disability, but over a long duration. For this type of patient ‘active therapy’ is recommended for the management of their CLBP. The participants included in the study are probably typical of the type of patient that would receive this type of therapy in clinical practice, or be referred by their clinician to a certified Pilates instructor. Therefore the current findings should only be considered in the context of
a population with minimal disability. Future studies should investigate the use of Pilates as a treatment option for more severely disabled non-specific CLBP sufferers. The same results may not be observed in a population with higher levels of functional disability.

There were no attempts to identify sub-groups of participants that responded more favourably to the intervention, however, this analysis is published elsewhere (Okyay, 2010). Okyay (2010) used regresional analysis to identify factors that predicted a favourable or poor outcome and create a clinical prediction rule (CPR) for use of Pilates based therapeutic exercise for management of CLBP.

**Recommendations for further work**

Further research should continue to evaluate the effectiveness of Pilates, and the efficacy of Pilates compared to other types of exercise and treatments for CLBP. The ideal duration and frequency of a Pilates programme has not been established, but the current research shows a trend toward better results with two or more classes per week. In many trials of exercise therapy for CLBP the results show an immediate improvement following the intervention, but the results are not maintained at medium or long term follow up. In these cases the authors often conclude that there is no long-term benefit, but to say there is no long-term benefit in doing any type of exercise may be an overstatement, as these claims are made based on research exercise interventions often ranging from 4 – 8 weeks, and up to a maximmm of 12 weeks duration. With all training there is a point where performance will begin to plateau, but there is little research that has yet investigated the length of time necessary for the participant to achieve the most improvement. More longitudinal studies are needed to assess whether more prolonged intervention periods add any additional benefits. A study design in which all participants are assigned to carry out a 6-week intervention and then 50% continue for another six months, might start to reveal if there is any benefit in continuing to participate in longer term exercise regimens. There are many
anecdotal accounts of general members of the public that participate in Pilates, continuing to experience improvement in physical functioning and reduction in pain over a prolonged period of consistent practice.

Further investigation should be undertaken to evaluate whether there is any advantage in implementing closer supervision (individualised instruction) Pilates sessions compared to small group classes. Individualised Pilates is already used in the clinical setting for rehabilitation of patients with CLBP. More research should be done to validate the effectiveness of individualised Pilates, especially as a treatment option for more severely disabled patients with CLBP. The cost benefit ratio and other economic analysis also needs to be addressed in future work.

Patients with CLBP have been shown to have deficits in motor control of the lumbopelvic stabilising muscles, as well as the presence of psychosocial risk factors. Part of the proposed mechanism of action for Pilates exercise is re-training of motor control of the deep lumbopelvic stabilising muscles. Future studies should objectively evaluate whether these changes occur in CLBP population following a Pilates training programme. Finally, future research should determine if there are any effects on psychosocial and psychological factors that are associated with CLBP, such as fear avoidance beliefs, kinesiophobia, distress and depression. In contrast the effect psychosocial factors play in influencing the outcomes of a Pilates intervention should also be evaluated, recently it has been suggested that self-efficacy beliefs have an important role to play in recovery (Costa, Maher, McAuley, Hancock & Smeets, 2010).
CONCLUSIONS

The results of the current study support the use of Pilates for the management of non-specific CLBP. A 6-week Pilates intervention was able to decrease the amount of functional disability, low back pain intensity, and LBP troublesomeness in adults with non-specific CLBP.
REFERENCES


Okyay, L. (2010). *Predictors of functional improvement in people with chronic low back pain following a graded programme of movement control exercises*. Thesis,


Section 3: Appendices
Appendix A: Example of Graded Exercise Progression for Classes
# Example of Graded Exercise Progression for Mat Classes

<table>
<thead>
<tr>
<th>Mat Week 1</th>
<th>Mat Week 3</th>
<th>Mat Week 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finding Neutral Spine</td>
<td>Finding Neutral Spine</td>
<td>Lying on the ½ or full roller:</td>
</tr>
<tr>
<td>Breathing Practice</td>
<td>Breathing Practice</td>
<td>• Breathing Practice</td>
</tr>
<tr>
<td>• Engaging pelvic floor and Transversus Abdominis</td>
<td>Bent Knee Fall Out</td>
<td>• Bent Knee Fall Out</td>
</tr>
<tr>
<td>• Inhale through nose</td>
<td>Dead Bugs/ Single Knee Float</td>
<td>• Dead Bugs/ Single Knee Float</td>
</tr>
<tr>
<td>• Exhale through mouth</td>
<td>Chest Lift (Curl Ups) arms reaching</td>
<td>• Snow Angels</td>
</tr>
<tr>
<td>• Elongating exhalation, and using this to encourage relaxation of areas</td>
<td>Pelvic Tilting</td>
<td>• Arm arcs</td>
</tr>
<tr>
<td>of tension in the body</td>
<td>Bridging</td>
<td>• On the Mat</td>
</tr>
<tr>
<td>Bent Knee Fall Out</td>
<td>Hamstring Stretch Supine with resistance-band</td>
<td>• Bent Knee Fall Out</td>
</tr>
<tr>
<td>Pelvic Tilting</td>
<td>Side to Side (Hip Rolls) with feet on the floor</td>
<td>• Dead Bugs/ Single Knee Float</td>
</tr>
<tr>
<td>Bridging ½, ¾, full</td>
<td>Single → Double dead bug (up, up, down, down)</td>
<td>• Pelvic Tilting &amp; Bridging</td>
</tr>
<tr>
<td>Supine Hamstring Stretch <em>with resistance-band or yoga strap</em></td>
<td>Side to Side with knees and hips bent at 90° (legs at tabletop)</td>
<td>• Stretching Gluteals Supine (fig 4 stretch)</td>
</tr>
<tr>
<td>Clams</td>
<td>Kneeling Hip Flexor &amp; Adductor Stretch</td>
<td>Chest Lift with arms reaching &amp; arms behind head</td>
</tr>
<tr>
<td>Magic Circle Squeeze</td>
<td>Assisted Roll Ups</td>
<td>Oblique Chest Lift arms behind head</td>
</tr>
<tr>
<td>Stretching Gluteals supine (fig 4 stretch)</td>
<td>Clams</td>
<td>Side to Side feet on the floor &amp; legs at tabletop (90°)</td>
</tr>
<tr>
<td>Chest Lift (Curl Ups) arms reaching</td>
<td>Magic Circle Squeeze</td>
<td>Assisted Roll Ups</td>
</tr>
<tr>
<td>Dead Bugs/ Single Knee Float</td>
<td>Stretching Gluteals Supine (fig 4 stretch)</td>
<td>Hamstring Stretch Supine with resistance-band</td>
</tr>
<tr>
<td>Side to Side (Hip Rolls) with feet on the floor</td>
<td>Chest Lift legs at tabletop (90°)</td>
<td>Side to Side Lying Single → Double Leg Lift</td>
</tr>
<tr>
<td>Prone Breathing</td>
<td>100s legs at tabletop (90°)</td>
<td>Clams</td>
</tr>
<tr>
<td>Prone Thoracic Extension</td>
<td>Kneeling Hip Flexor &amp; Adductor Stretch</td>
<td>• Oblique Chest Lift arms behind head</td>
</tr>
<tr>
<td>Child Pose/ Rest Position</td>
<td>Quadraposition</td>
<td>• Kneeling Hamstring Stretch</td>
</tr>
<tr>
<td>Kneeling Hip Flexor &amp; Adductor Stretch</td>
<td>• Single Arm Lift</td>
<td>Quadraposed Position</td>
</tr>
<tr>
<td>Cat Stretch</td>
<td>• Single Leg Lift</td>
<td>• Single Arm Lift &amp; Single Leg Lift</td>
</tr>
<tr>
<td>Assisted Roll Up</td>
<td>• Opposite Arm and Leg Lift</td>
<td>• Opposite Arm and Leg Lift</td>
</tr>
<tr>
<td>Side Lying Single Leg Lift</td>
<td>Cat Stretch</td>
<td>Cat Stretch</td>
</tr>
<tr>
<td>Prone leg Lift</td>
<td>Prone Breathing</td>
<td>Chest Lift legs at tabletop (90°)</td>
</tr>
<tr>
<td>Child Pose/ Rest Position</td>
<td>Prone Quadriceps Stretch</td>
<td>Knee Hug (Supine Rest Position)</td>
</tr>
<tr>
<td>Quadraposition</td>
<td>Prone Single Leg Lift</td>
<td>Single Leg Stretch</td>
</tr>
<tr>
<td>• Single Arm Lift</td>
<td>Child Pose/ Rest Position</td>
<td>100s legs at tabletop (90°)</td>
</tr>
<tr>
<td>• Single Leg Lift</td>
<td>Book Openings/ Side Lying Rotation</td>
<td>Prone Quadriceps Stretch</td>
</tr>
<tr>
<td>Standing Roll Down</td>
<td>Standing Roll Down</td>
<td>Prone Single Leg Lift</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prone Thoracic Extension → Dart</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Child Pose/ Rest Position</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Book Openings/ Side Lying Rotation</td>
</tr>
</tbody>
</table>
### Reformer Week 1

**Lying Supine**  
Neutral Spine  
Breathing

**Leg and Footwork**  
- Double Leg Presses:  
  - in parallel on the heels and toes  
  - in turn out on the toes/or heels (Pilates V)  
  - out wide on the heels and toes (2nd position)  
  - Tendon Stretches (calf raises)  
  - Prancing  
  - Calf Stretch

**No Straps, Lying supine on carriage**  
- Chest Lift (calf raises)  
- Oblique Chest Lift  
- Dead Bugs/Single Knee Float (from the foot bar)  
- Single → Double dead bug (up,up,down,down)  
- Holding legs at tabletop for 5 breaths

**Legs in Straps**  
- Hamstring Press  
- Bend and stretch  
- Frogs  
- Circles  
- Adductor Stretch

**Feet on Footbar**  
- Pelvic tilting  
- Bridging  
- Stretching Gluteals supine (fig 4 stretch)

**Hands on the foot-bar**  
- Standing Hip Stretch (knee resting on carriage)  
- Standing on floor  
- Roll Down

### Reformer Week 3

**Lying Supine**  
Neutral Spine  
Breathing

**Leg and Footwork**  
- Double Leg Presses:  
  - in all 5 positions  
  - Tendon Stretches & Prancing  
  - Calf Stretch  
- Single Leg Presses:  
  - Heels & Toes (other leg at table top)  

**Lying supine on carriage no straps**  
- Chest lift legs at tabletop  
- 100s leg at tabletop

**Lying supine on carriage hands in straps**  
- Supine Arm Arcs  
- Chest Lift  
- Oblique Chest Lift

**Legs in Straps**  
- Hamstring Press  
- Frogs  
- Circles  
- Openings  
- Adductor Stretch

**Footbar down hands on the platform**  
- Quadraped  
- Hands on the foot-bar  
- Scooter  
- Standing Hip Stretch (knee resting on carriage)  
- Knee Stretch

**Seated on the Box**  
- Lat Pulls & Bicep Curls  
- Standing Abduction  
- Skating Prep  
- Supine  
- MC Squeeze  
- Gluteal Stretch

### Reformer Week 6

**Lying Supine**  
Neutral Spine  
Breathing

**Leg and Footwork**  
- Double Leg Presses in all 5 positions  
- Tendon Stretches, Prancing & Calf Stretch  
- Single Leg Presses:  
- Heels & Toes (other leg at table top)  

**Facing the headrest holding the tracks**  
- Reverse Abs  
- Footbar down hands on the platform  
- Quadraped

**Lying supine on carriage hands in straps**  
- Supine Arm Arcs  
- Chest Lift  
- Oblique Chest Lift  
- 100s leg at tabletop (can be done without straps)

**Legs in Straps**  
- Hamstring Press  
- Frogs  
- Circles  
- Openings  
- Adductor Stretch

**Hands on the foot-bar**  
- Scooter  
- Standing Hip Stretch (knee resting on carriage)  
- Knee Stretch

**Seated on the Box**  
- Lat Pulls & Bicep Curls  
- Standing Abduction  
- Skating Prep  
- Standing Adduction  
- Pigeon Stretch (gluteal stretch over Trapeze Table)
Appendix B: Exercise Repertoire
Appendix B: Exercise Repertoire

All exercises were completed with emphasis on traditional Pilates principles; breathing; precision; core control (centering); flowing and efficient movement. Breathing for all exercises was cued via verbal instruction to encourage feed forward activation of the pelvic floor and deep abdominals with exhalation, before movement of the limbs or trunk, to enhance lumbopelvic stability. All exercises were performed for between 6 - 10 repetitions (unless otherwise stated), and all stretches were held for 30 seconds. All exercises are modified from the original Pilates repertoire in accordance with the Pilates Method Alliance Study Guide.

Mat Exercises

Breathing Practice

Lying supine in neutral spine position
Inhale through nose, exhale through mouth
Engaging pelvic floor (PF) and Transversus Abdominis (TrA) with every exhalation
Elongate the exhalation phase of the breath, and using this to encourage relaxation of areas of tension in the body
Progress to maintaining low level contraction of TrA and PF during inhalation

Bridging

Starting in neutral spine position. Exhale to posteriorly tilt the pelvis.
Gently articulate the spine into flexion as hips move into extension.
Inhale pause with hips up. Exhale to articulate through the spine and return to neutral.
Aim is to increase spinal mobility (articulation) into flexion, and hip extension.
Bent Knee Fall Out

Lying supine in a neutral spine position, knees bent feet flat on the floor
Exhale to open one knee out to the side, inhale to return to start position
Challenge to pelvic stability, aim to prevent rotation of lumbar spine

Dead Bugs

Lying supine in a neutral spine position, knees bent feet flat on the floor
Exhale to float one leg up to table top (knee and hip flexed at 90°)
Inhale hold in this position, exhale lower leg and return to start position
The pelvis and lumbar spine should remain still in the neutral position
This exercise can be progressed to the Double Dead Bug (see below)
Chest Lift

Abdominal strengthening exercise, contraction of TrA and PF is prior to trunk flexion and contraction of the more superficial abdominals. Pelvis remains in neutral.

This exercise can be progressed by:
- bringing the hands behind the head (or to provide support for the cervical spine)
- rotating the thorax for added challenge to the obliques
- bring the legs into tabletop

Single Leg Stretch

Abdominal strengthening exercise. Maintaining chest lift position throughout, Exhale to stretch one leg away from the body (whilst maintaining neutral spine and pelvis), Inhale return leg to tabletop position, Repeat with other leg.

100s legs at tabletop (90°)

Abdominal strengthening exercise. Chest lift position held for 10 elongated breaths, with emphasis on maintaining activation of deep abdominals and pelvic floor on inhalation. One set of 10 breaths.
Knee Hug (Supine Rest Position)

Side to Side (Hip Rolls)

Controlled rotation of the spine remaining within a range of motion where a neutral lumbar lordosis is maintained (avoiding going into flexion or excessive extension). Shoulders remain fixed on the floor, but avoid pushing or gripping with arms. To progress this exercise the legs can be at tabletop (90°), see below.

Assisted Roll Up

Controlled spinal flexion using the abdominals to articulate the spine away from the floor, maintaining a C-curve shape to the spine until the feet touch the ground. In seated the spine is lengthened vertically into a neutral spine position. To roll down the spinal flexion is initiated from a posterior pelvic tilt on the exhalation.
Side Lying Leg Lift

All side lying exercises are a challenge to pelvic stability and lateral stabilising muscles.

Clams

Challenge to pelvic stability and gluteal strength.
Side Lying, with pelvis and Lsp in neutral, soles of feet in line with sacrum.
Exhale to lift knee, keeping feet together. Inhale to lower.

Side Lying Leg Kick

Exhale to swing top leg forward. Inhale to bring it back in line with body.
This exercise is progressed by lifting and lowering leg whilst in forward kick position.

Magic Circle Squeeze

Lying supine with neutral spine and pelvis
Circle positioned along the joint line of the knee
Exhale to press in using adductors
Inhale to control the release
Progression: legs at tabletop
Stretching Gluteals Supine (fig 4 stretch)

All stretches held for 30 seconds each side

Supine Hamstring Stretch with Resistance Band

Kneeling Hip Flexor & Adductor Stretch

Kneeling Hamstring Stretch

Quadruped Position

Breathing practice engaging pelvic floor and deep abdominal muscles, whilst maintaining neutral spine in quadruped position.

To challenge coordination and balance a single arm or leg is lifted off the floor, whilst maintaining neutral spine and shoulder girdle and pelvis level with the floor. This exercise can then be progressed to lifting opposite arm and leg as pictured below.

Opposite Arm and Leg Lift
Cat Stretch

Starting in a neutral position. Exhale to flex the lumbar spine, inhale and pause. Exhale and return to neutral spine, inhale and pause in start position.

Prone Breathing

Breathing practice in a prone position.

Prone Thoracic Extension

Exhale to articulate thoracic spine only into extension, lumbar spine remains neutral.

Prone Single Leg Lift

Prone hip extension with lumbar spine maintained in neutral lordosis, avoiding excessive lumbar spine extension. Pelvis should remain stable, avoid rotation and anterior tilt.

Prone Quadriceps Stretch

Stretch held for 30 seconds each side

Child Pose/ Rest Position

Position held for 10-30 seconds
Book Openings/ Side Lying Rotation

Increase spinal mobility, movement starts in the thoracic spine and rotation occurs sequentially down the spine into the lumbar spine. Pelvic stability should be maintained, but not if this causes excessive muscular tension around the lumbar spine. This should be thought of as a release exercise or slight stretch.

Standing Roll Down

Starting in standing, this is a more functional position because the body is in a familiar orientation to gravity. Exhale to articulate into flexion down the spine. Inhale take a breath when flexed all the way forward (picture far right). Exhale to engage pelvic floor and restack the spine into a neutral erect posture.
Appendix B continued: Reformer Exercises

Bridging

Supine Gluteal Stretch (Figure 4 stretch)

Leg and Footwork

Tendon Stretches and Prancing

Maintain neutral spine and pelvis as the carriage is pressed out with hip extension on exhalation, and controlled back in as hips flex on inhalation. Five leg and foot positions were practiced.
Single Leg Press

Abdominal Exercises

Supine Arm Arcs

Chest Lift

In neutral spine and pelvis, legs at tabletop. Exhale to perform a chest lift (as on the mat) whilst pulling the straps down. Inhale to control the curl down and return carriage to start position. This exercise can be regressed by having the feet resting on the footbar and not using the straps.

Oblique Chest Lift

In neutral spine and pelvis, legs at tabletop knees apart, and feet together. Exhale to curl up and press one hand between the legs, the other just to the outside of the thigh. Inhale return to start position with control, and repeat on the other side. This exercise can be regressed by resting the feet on the footbar and not using the straps (see below).

Hundreds legs at tabletop

Abdominal endurance exercise. Chest lift position held for 10 breaths, with emphasis on maintaining activation of deep abdominals and pelvic floor on inhalation. One set of 10 breaths
*Legs in Straps*

The challenge of all exercises with legs in straps is to maintain pelvic stability and neutral spine.

**Hamstring Press**

**Frogs**

**Circles**

**Quadraped**

Maintain neutral spine throughout. Exhale press carriage out, and hips into extension. Inhale return the carriage. Shoulders remain above the wrists.

**Knee Stretch**

Maintain neutral spine throughout. Inhale press carriage out. Exhale to flex at the hips and draw the carriage in. This exercise encourages hip disassociation.

**Scooter**

Standing on one leg, with pelvis level. Maintain neutral spine and pelvis throughout. Inhale to press carriage out. Exhale to flex the hip and control the carriage in. This exercise encourages hip disassociation.
Standing Hip Stretch (knee resting on carriage)

Arms Seated on the box (Lat Pulls & Bicep Curls)

Maintaining a neutral spine while seated on the box.
Exhale pull back on the straps, inhale to control the return to the start position.

Standing Abduction & Adduction

Standing with one foot on stable platform and one foot on moving carriage.
Upright neutral posture, hips level.
Exhale to press the carriage out, maintaining lumbopelvic stability.
Inhale to return the carriage.
Repeat facing the other direction.

Skating Prep

Gluteal strengthening exercise.
Body position is slightly pitched forward from the hips (hip flexion), spine and pelvis remain in neutral throughout.
Exhale to press the inside leg to straight, inhale to control the return.

Reverse Abs

Abdominal and hip flexor strengthening exercise.
Start in quadraped position, shoulders vertically above wrists throughout.
Exhale to flex hips whilst maintaining neutral spine,
Inhale to control the return of the carriage.
Light springs.
<table>
<thead>
<tr>
<th>Section/Topic</th>
<th>Item No</th>
<th>Checklist item</th>
<th>Reported on page No</th>
<th>Notes</th>
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<tbody>
<tr>
<td><strong>Title and abstract</strong></td>
<td></td>
<td></td>
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<tr>
<td>Pilates-Based Therapeutic Exercise: Effect on Subjects With Nonspecific Chronic Low Back Pain and Functional Disability: A Randomized Controlled Trial</td>
<td></td>
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<tr>
<td>Rochenda Rydeard, PT, MSc</td>
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<tr>
<td>Andrew Leger, PT, PhD</td>
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<tr>
<td>Drew Smith, PhD</td>
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<tr>
<td>J Orthop Sports Phys Ther • Volume 36 • Number 7 • July 2006</td>
<td></td>
<td></td>
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<tr>
<td>1a</td>
<td>Identification as a randomised trial in the title</td>
<td>472</td>
<td>Yes</td>
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<tr>
<td>1b</td>
<td>Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)</td>
<td>472</td>
<td>Yes</td>
<td></td>
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<tr>
<td><strong>Introduction</strong></td>
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<td>Background and objectives</td>
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<td>2a</td>
<td>Scientific background and explanation of rationale</td>
<td>473</td>
<td>Yes</td>
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<td>2b</td>
<td>Specific objectives or hypotheses</td>
<td>473 - 474</td>
<td>Yes</td>
<td></td>
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<tr>
<td><strong>Methods</strong></td>
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<tr>
<td>Trial design</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3a</td>
<td>Description of trial design (such as parallel, factorial) including allocation ratio</td>
<td>474 - 475</td>
<td>A pre-postest design, with follow up immediately after intervention plus at 3, 6, and 12 months. CG parallel for intervention period then had the option to have the intervention. Allocation ratio described</td>
<td></td>
</tr>
<tr>
<td>3b</td>
<td>Important changes to methods after trial commencement</td>
<td>474</td>
<td>Methods</td>
<td>None</td>
</tr>
</tbody>
</table>
Participants

4a Eligibility criteria for participants

Well described: adults aged 20 – 55 years, had to be physically active 3 x 30 min sessions p/wk min, with LBP > 6 weeks or recurrent LBP with 2 painful episodes per year. Have evidence of neuromuscular dysfunction: 1. strength of gluteus max less than 4/5, and 2. altered recruitment of gluteus max in prone hip extension. Subjects meeting this criteria were selected as they were thought to be more likely to respond to this treatment.

4b Settings and locations where the data were collected

Testing was performed at the Hong Kong Polytechnic University. Clinical intervention conducted at a private physiotherapy clinic in Hong Kong.

Interventions

5 The interventions for each group with sufficient details to allow replication, including how and when they were actually administered

Specific exercise intervention not described in enough detail with regard to the types of exercises taught. Administered in an individualised manner. Static postures on mat first, followed by a variety of movement patterns to stress the lumbar-pelvic region and involving hip extension. Then progressed to reformer.

Outcomes

6a Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed

Yes

6b Any changes to trial outcomes after the trial commenced, with reasons

None

Sample size

7a How sample size was determined

It is unclear

7b When applicable, explanation of any interim analyses and stopping guidelines

4 week trial period completed

Randomisation:

Sequence

8a Method used to generate the random allocation sequence

Subjects randomly pulled a card from a box, cards marked specific exercise group or control group
**Generation**

Type of randomisation; details of any restriction (such as blocking and block size)

- **Allocation concealment mechanism**: No restriction, small sample size. Unclear if there were a certain number of cards with the 2 options, or only 2 cards so participants would have a 50% chance of choosing each intervention.

- **Implementation**: Subjects randomly pulled a card from a box, cards marked specific exercise group or control group. Randomisation was administered by independent office staff.

- **Blinding**: Subjects randomly pulled a card from a box, cards marked specific exercise group or control group. Randomisation was administered by independent office staff.

- **Statistical methods**: Subjects randomly pulled a card from a box, cards marked specific exercise group or control group. Randomisation was administered by independent office staff.

**Results**

For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the

- **Participant flow**: Details of inclusion exclusion and numbers excluded pre intervention Flow Chart with numbers of
<table>
<thead>
<tr>
<th>Section</th>
<th>Description</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>13b</td>
<td>For each group, losses and exclusions after randomisation, together with reasons</td>
<td>475 participants, and numbers of and reasons for drop outs</td>
</tr>
<tr>
<td>Recruitment</td>
<td>Dates defining the periods of recruitment and follow-up</td>
<td>No one excluded after beginning intervention, losses to follow up detailed in flow chart</td>
</tr>
<tr>
<td>14a</td>
<td>Why the trial ended or was stopped</td>
<td>4 month period for recruitment — no dates given</td>
</tr>
<tr>
<td>14b</td>
<td>Dates defining the periods of recruitment and follow-up</td>
<td>4 week trial period completed</td>
</tr>
<tr>
<td>Baseline data</td>
<td>A table showing baseline demographic and clinical characteristics for each group</td>
<td>Yes. But some information displayed in wrong column (not sure how much possibly just male and female), outcome measures are in correct columns.</td>
</tr>
<tr>
<td>Baseline data</td>
<td>A table showing baseline demographic and clinical characteristics for each group</td>
<td>Yes, but in flow diagram and table 2 CG n=18, SETG n=21 however in table 1 (baseline characteristics) CG n=21, SETG n=18. Confusing! CG= control group SETG= specific exercise training group</td>
</tr>
<tr>
<td>Numbers analysed</td>
<td>For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups</td>
<td>95% CI, but no estimated effect size, but enough information provided so effect size could be calculated. Pre and post mean values for RMD-HK and NRS presented in table.</td>
</tr>
<tr>
<td>Outcomes and estimation</td>
<td>For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)</td>
<td></td>
</tr>
<tr>
<td>Ancillary analyses</td>
<td>Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory</td>
<td><em>Would have been interesting to see any of the CG that received the intervention (if any) after the 4-week trial period analyzed separately.</em></td>
</tr>
</tbody>
</table>
| Harms | All important harms or unintended effects in each group (for specific guidance see CONSORT for harms) | No increase in functional disability reported in SE treatment group (only those that fall in lowest strata of RMD). It is unclear if anyone in the study had an increase in pain or disability at 4-week follow up. Pain intensity not mentioned. 100% completed trial (no drop...
Discussion

Limitations

<table>
<thead>
<tr>
<th>Page</th>
<th>Description</th>
</tr>
</thead>
</table>
| 480-483 | Small sample size SETG n=21, CG n=18, very specific inclusion criteria (pg 481) source of bias? Although not significant differences between the CG and SETG, the SETG had lower levels of functional disability and pain, and had less duration of LBP (median 5.5 years compared to 9 years for CG)

Possible that the control group intentionally reported increase in pain in order to receive treatment after wait period? Used SEM instead of SD. Multiplicity of analysis for retention of treatment effect robustness (pg 482)

Generalisability

<table>
<thead>
<tr>
<th>Page</th>
<th>Description</th>
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</thead>
</table>
| 481 | Authors do state that results may not be applicable to those with acute or very disabling LBP as the sample population had low scores for functional disability (pg 481). Trail conducted in Hong Kong, at a private physiotherapy practice, the Pilates method was not well known by the general public in HK at the time (pg 474), this would have reduced preconceived expectations of the sample population. How reproducible these results are in other populations (countries) is yet to be seen.

474 | Participants received individualised treatment/ exercise programmes – this may affect generalisability.

476 | This LBP population had dysfunction of gluteus max, so the finding can’t be extrapolated to CLBP with out this.

481 | |
<table>
<thead>
<tr>
<th>Item</th>
<th>Section</th>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interpretation</td>
<td>22</td>
<td>Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence</td>
<td>483</td>
</tr>
<tr>
<td>Other information</td>
<td></td>
<td>Interpretation consistent with results. Statistically significant differences in No harms reported, perhaps no one got worse.</td>
<td></td>
</tr>
<tr>
<td>Registration</td>
<td>23</td>
<td>Registration number and name of trial registry</td>
<td>474</td>
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<tr>
<td>Protocol</td>
<td>24</td>
<td>Registration of trial not mentioned.</td>
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<tr>
<td>Funding</td>
<td>25</td>
<td>Ethics approval granted from The Faculty of Health and Science Research Ethics Board, Queens University, Kingston, Ontario and the Hong Kong Polytechnic University Human Subjects Ethics Subcommittee, Hong Kong Special Administrative Region.</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Not mentioned.</td>
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<tr>
<td></td>
<td></td>
<td>Not mentioned.</td>
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</table>

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see [www.consort-statement.org](http://www.consort-statement.org).*
## CONSORT 2010 checklist of information to include when reporting a randomised trial*

<table>
<thead>
<tr>
<th>Section/Topic</th>
<th>Item No</th>
<th>Checklist item</th>
<th>Reported on page No</th>
<th>Comment</th>
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<tr>
<td>Title and abstract</td>
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<tr>
<td><strong>Does a Program of Pilates Improve Chronic Non-Specific Low Back Pain?</strong></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td><strong>Valerie Gladwell, Samantha Head, Martin Haggar, and Ralph Beneke</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>Introduction</strong></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Background and objectives</td>
<td>2a</td>
<td>Scientific background and explanation of rationale</td>
<td>338 - 339</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>2b</td>
<td>Specific objectives or hypotheses</td>
<td>339</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
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<td></td>
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<td></td>
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<tr>
<td>Trial design</td>
<td>3a</td>
<td>Description of trial design (such as parallel, factorial) including allocation ratio</td>
<td>340</td>
<td>Allocation ratio not described. “Randomly” is the only description of allocation.</td>
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<tr>
<td></td>
<td>3b</td>
<td>Important changes to methods after trial commencement</td>
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<td>None</td>
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<tr>
<td>Participants</td>
<td>4a</td>
<td>Eligibility criteria for participants</td>
<td>341</td>
<td>Well described.</td>
</tr>
<tr>
<td>Section</td>
<td>Description</td>
<td>Page(s)</td>
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<tr>
<td>4b</td>
<td>Settings and locations where the data were collected</td>
<td>340</td>
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<td></td>
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<tr>
<td>5</td>
<td>The interventions for each group with sufficient details to allow replication, including how and when they were actually administered</td>
<td>340 - 341</td>
<td></td>
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<tr>
<td>6a</td>
<td>Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed</td>
<td>342-344</td>
<td></td>
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<tr>
<td>6b</td>
<td>Any changes to trial outcomes after the trial commenced, with reasons</td>
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<td></td>
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<tr>
<td>7a</td>
<td>How sample size was determined</td>
<td>340</td>
<td></td>
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<tr>
<td>7b</td>
<td>When applicable, explanation of any interim analyses and stopping guidelines</td>
<td>N/A – 6 week trial period completed</td>
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<tr>
<td>8a</td>
<td>Method used to generate the random allocation sequence</td>
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<tr>
<td>8b</td>
<td>Type of randomisation; details of any restriction (such as blocking and block size)</td>
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<tr>
<td>9</td>
<td>Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned</td>
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<tr>
<td>10</td>
<td>Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions</td>
<td>340</td>
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</tbody>
</table>

**Inclusion**

Age 18-60 years, CLBP >12wks, otherwise

Participants recruited from Colchester Region. Intervention setting not described.

Pilates exercise intervention was described in enough detail. Control group continued with their normal activities and pain relief. All participants were not undergoing any regular physiotherapy or osteopathy treatment.

Yes

None

Unsure

Unsure

Unsure
If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how

Assessor blinded

If relevant, description of the similarity of interventions

Interventions not similar

Statistical methods

Statistical methods used to compare groups for primary and secondary outcomes

Data were tested for normality using Kolmogorov-Smirnov. Data was found to be not normally distributed in the majority of cases and therefore non-parametric tests were performed. Wilcoxon sign ranked tests were used to identify any significant changes pre- and post- intervention within each group. Differences in baseline data and post- intervention changes between the groups were analyzed using either Mann-Whitney U or Chi-squared. All values given are two-tailed.

Methods for additional analyses, such as subgroup analyses and adjusted analyses

No sub-group analysis done
<table>
<thead>
<tr>
<th><strong>Results</strong></th>
<th><strong>Participant flow (a diagram is strongly recommended)</strong> 13a</th>
<th>For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome</th>
<th>344-345</th>
<th>No flow chart. 10 dropouts from control group, no reasons given. From Pilates group 5 people were unable to attend 2 or more classes so were withdrawn from the trial. No long term follow up.</th>
</tr>
</thead>
<tbody>
<tr>
<td>13b</td>
<td>For each group, losses and exclusions after randomisation, together with reasons</td>
<td>344-345</td>
<td>No reasons given for CG dropouts. It seems all of the Pilates group dropouts were due to time constraints or other commitments meaning participants were unable to attend classes.</td>
<td></td>
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<tr>
<td><strong>Recruitment</strong></td>
<td>14a</td>
<td>Dates defining the periods of recruitment and follow-up</td>
<td>340</td>
<td>6 week trial period completed</td>
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<tr>
<td>14b</td>
<td>Why the trial ended or was stopped</td>
<td>345</td>
<td>Yes. Significant difference in age CG older. CG on average had back pain for longer duration than Pilates group, but this was not significant.</td>
<td></td>
</tr>
<tr>
<td><strong>Baseline data</strong></td>
<td>15</td>
<td>A table showing baseline demographic and clinical characteristics for each group</td>
<td>346 - 347</td>
<td>No</td>
</tr>
<tr>
<td><strong>Numbers analysed</strong></td>
<td>16</td>
<td>For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups</td>
<td>346 - 347</td>
<td>No 95% CI, because data was non-normal and analysed using non-parametric tests. Pre and post mean (SD) for primary (RMVAS and ODI) and secondary outcomes are presented in table 4. Estimated P values are given (P&lt;0.05) instead of exact values.</td>
</tr>
<tr>
<td><strong>Outcomes and estimation</strong></td>
<td>17a</td>
<td>For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)</td>
<td>346 - 347</td>
<td>No</td>
</tr>
<tr>
<td>17b</td>
<td>For binary outcomes, presentation of both absolute and relative effect sizes is recommended</td>
<td>N/A</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td><strong>Ancillary analyses</strong></td>
<td>18</td>
<td>Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory</td>
<td>N/A</td>
<td>Unsure of any adverse effects. This could have been reason for high drop out rate in CG.</td>
</tr>
<tr>
<td><strong>Harms</strong></td>
<td>19</td>
<td>All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Discussion
Limitations
Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses

20 Small sample size EG n=20, CG n=14. Using non-parametric tests give less power to results? Only 15% of the Pilates group and 29% of CG was male. Pilates group was younger and had not had LBP for as long as CG on average. The CG (the 14 that completed the trial) experienced a significant improvement in ODI without doing anything differently. This could be that all those in the CG that had an increase in pain or disability dropped out of the trial so they could seek some treatment, and those in the Pilates group stayed in because they felt that they were doing something for their back.

Authors state that it is difficult to determine if the positive gains were solely dependent on Pilates or other aspects of the intervention. It is unclear what these other aspects are. Perhaps it was that participants were able to take medication, and continue with normal levels of physical activity?

Generalisability
Generalisability (external validity, applicability) of the trial findings

21 Findings should be interpreted with caution as individuals respond to differing extents to the Pilates intervention, but this will be typical of any back pain population and any intervention. The data for each group at the start of the study were found to be similar. The study is limited by the modest number of participants who completed the study, and the imbalance of the number of participants in the two groups due to higher drop out rate from the control group.
Interpretation
Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence

Registration
Registration number and name of trial registry

Protocol
Where the full trial protocol can be accessed, if available

Funding
Sources of funding and other support (such as supply of drugs), role of funders

Not compared to previous Pilates research, as it was the first to study the effects of Pilates in a CLBP population. Studies that advocate general exercise for CLBP are mentioned in discussion, but not the result of significant decrease in ODI score for CG, 64% of whom were undertaking regular physical activity. It is also proposed that the ODI might not be sensitive enough to determine changes in individuals with mild disability or slight functional changes.

Registration of trial not mentioned.

Ethics approval granted from The University of Essex ethics committee.

Not mentioned.

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.*
### Title and Abstract

Two different techniques in the rehabilitation treatment of low back pain: A randomized controlled trial.

Donzelli, S., Di Domenica, E., Cova, A. M., Galletti, R., & Giunta, N.

*Europa Medicophysica, 42*(3), 205-10 (2006)

<table>
<thead>
<tr>
<th>Item No</th>
<th>Checklist item</th>
<th>Reported on page No</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>Identification as a randomised trial in the title</td>
<td>205</td>
<td>Yes</td>
</tr>
<tr>
<td>1b</td>
<td>Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)</td>
<td>205</td>
<td>Randomisation and blinding not detailed. Number of participants in each group not specified. No actual results for primary outcomes with C.Is published, only described as a significant reduction in pain intensity and disability observed across entire sample (ie.both groups CG and Pilates group). Total number of participants that completed the study (analysed) was mentioned (43) but not number for each group. No harms reported. Conclusion based on what? Results were vague.</td>
</tr>
</tbody>
</table>

### Introduction

**Background and objectives**

<table>
<thead>
<tr>
<th>Item No</th>
<th>Checklist item</th>
<th>Reported on page No</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>2a</td>
<td>Scientific background and explanation of rationale</td>
<td>205</td>
<td>Talks about Pilates being in Australia for over 30 years, then about numerous scientific studies proving its usefulness – none of them done in Australia or high quality scientific studies one was observational, and one a narrative (no experiment). Rational for study appears to be to give Cova Tech Pilates credibility as a treatment for CLBP like back school. Back School has systematic review reference.</td>
</tr>
<tr>
<td>2b</td>
<td>Specific objectives or hypotheses</td>
<td>206</td>
<td>To see weather a system of Pilates Cova-Tech Pilates is a valid rehabilitative treatment for CLBP, and to compare ‘Cova Tech Pilates” to Back School (which would mean it would be easily adaptable to the National Health Service) in Italy.</td>
</tr>
</tbody>
</table>
**Methods**

<table>
<thead>
<tr>
<th>Trial design</th>
<th>3a</th>
<th>Description of trial design (such as parallel, factorial) including allocation ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3b</td>
<td>Important changes to methods after trial commencement</td>
</tr>
</tbody>
</table>

**Participants**

<table>
<thead>
<tr>
<th>4a</th>
<th>Eligibility criteria for participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>4b</td>
<td>Settings and locations where the data were collected</td>
</tr>
</tbody>
</table>

**Interventions**

| 5 | The interventions for each group with sufficient details to allow replication, including how and when they were actually administered |

**Outcomes**

<table>
<thead>
<tr>
<th>6a</th>
<th>Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed</th>
</tr>
</thead>
<tbody>
<tr>
<td>6b</td>
<td>Any changes to trial outcomes after the trial commenced, with reasons</td>
</tr>
</tbody>
</table>

**Sample size**

<table>
<thead>
<tr>
<th>7a</th>
<th>How sample size was determined</th>
</tr>
</thead>
<tbody>
<tr>
<td>7b</td>
<td>When applicable, explanation of any interim analyses and stopping guidelines</td>
</tr>
</tbody>
</table>

**Randomisation:**

<table>
<thead>
<tr>
<th>206</th>
<th>Type of trial not emphasised.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CG: 22 participants Back School</td>
</tr>
<tr>
<td></td>
<td>EG: 21 participants Cova Tech Pilates</td>
</tr>
</tbody>
</table>

| 206 | No changes reported |

| 206 | Lasegue's test = SLR (why both?), Wassermann's Test = for syphilis. Age limits not mentioned in eligibility criteria, but range given for sample population 20-65 years. Chronic LBP = >3mo persistent or recurrent not specified. |

| 206 | Patients were recruited from the outpatient departments of G. Pini Orthopaedic Institute. The classes were carried out at “Ollstica Salus” gym (I think) as they are mentioned in the acknowledgements. |

| 207 | No. Back School protocol was changed to leave out exercises that were too similar to ones in Pilates protocol, but not sure which ones. Back school protocol probably described in references but not in English. Description of Pilates protocol was ‘Basic’ level of mat4me Cova Tech Pilates exercises, and aim of exercises described but not actual exercises. |

| 206-207 | Yes |

| None |

| 208 | By the number of drop outs before the first treatment session |

<p>| N/A |</p>
<table>
<thead>
<tr>
<th>Sequence generation</th>
<th>8a</th>
<th>Method used to generate the random allocation sequence</th>
<th>206</th>
<th>Patients allocated to different therapies dependent on what time of day they could attend classes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>8b</td>
<td>Type of randomisation; details of any restriction (such as blocking and block size)</td>
<td></td>
<td></td>
<td>Not really random allocation, selection based on patient convenience. Blocking not detailed</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Allocation concealment mechanism</td>
<td>9</td>
<td>Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned</td>
<td>206</td>
<td>Not properly randomised.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Implementation</td>
<td>10</td>
<td>Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions</td>
<td>206</td>
<td>Office clerk assigned groups dependent on what time of day they could attend classes.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blinding</td>
<td>11a</td>
<td>If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how</td>
<td>206</td>
<td>Patient blinded as to what treatment they were receiving. Two different physicians performed the pre-treatment and the follow-up examinations; this could lead to increased examiner error. Assessor blinded</td>
</tr>
<tr>
<td></td>
<td>11b</td>
<td>If relevant, description of the similarity of interventions</td>
<td>207</td>
<td>The similarity of the aims described but not in much detail. The Back School was modified slightly to make it comparable with the Cova Tech Pilates method – but how? The Back School protocol also omitted exercises that were similar to the Pilates exercises – not sure why.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statistical methods</td>
<td>12a</td>
<td>Statistical methods used to compare groups for primary and secondary outcomes</td>
<td>208-209</td>
<td>SPSS used to evaluate whether the Pilates group or Cg were significantly different from each other in age, sex, and occupational risk factors. ODI and VAS were analysed using ‘frequency tests’. I’m not sure what a frequency test is but there are no p values, confidence intervals or estimated effect size published for the pre – post data for either outcome measure. The average results for ODI and VAS for both groups and the groups combined (why?) is graphically represented on pg 208, showing the baseline, and 1, 3, and 6 months post.</td>
</tr>
<tr>
<td></td>
<td>12b</td>
<td>Methods for additional analyses, such as subgroup analyses and adjusted analyses</td>
<td>208</td>
<td>Sub group analysis not done. They could have looked at sub groups, how occupational risk factors affected outcomes, but did not. Subjective opinion of participants noted in Table 1. If they felt worse, same or better? At 1, 3, and 6 months.</td>
</tr>
<tr>
<td></td>
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</tr>
<tr>
<td>Category</td>
<td>Section</td>
<td>Description</td>
<td>Page(s)</td>
<td>Notes</td>
</tr>
<tr>
<td>--------------------------</td>
<td>---------</td>
<td>------------------------------------------------------------------------------------------------</td>
<td>---------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Results</td>
<td>13a</td>
<td>For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome</td>
<td>208-209</td>
<td>Flow diagram not included. The number of participants in Table 1 does not add up. In the Back School group 22, Pilates 21, Total 40! Should be 43. No explanation for the discrepancy. Did they really have 100% response to follow up?</td>
</tr>
<tr>
<td></td>
<td>13b</td>
<td>For each group, losses and exclusions after randomisation, together with reasons</td>
<td>208</td>
<td>10 participants dropped out before attending their first session, reasons detailed in results section. No one excluded after beginning intervention.</td>
</tr>
<tr>
<td>Recruitment</td>
<td>14a</td>
<td>Dates defining the periods of recruitment and follow-up</td>
<td>206</td>
<td>Yes. October 2003 – March 2004</td>
</tr>
<tr>
<td></td>
<td>14b</td>
<td>Why the trial ended or was stopped</td>
<td>206</td>
<td>10 consecutive lessons over 10 days, completed.</td>
</tr>
<tr>
<td>Baseline data</td>
<td>15</td>
<td>A table showing baseline demographic and clinical characteristics for each group</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Numbers analysed</td>
<td>16</td>
<td>For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups</td>
<td>No denominator displayed. No analysis done for 2 groups, results only displayed in graph pg 208.</td>
<td></td>
</tr>
<tr>
<td>Outcomes and estimation</td>
<td>17a</td>
<td>For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)</td>
<td>N/A</td>
<td>No statistical comparison within groups (pre-post) or between groups EG and CG.</td>
</tr>
<tr>
<td></td>
<td>17b</td>
<td>For binary outcomes, presentation of both absolute and relative effect sizes is recommended</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Ancillary analyses</td>
<td>18</td>
<td>Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory</td>
<td>None</td>
<td>None. They could have done sub group analysis of the primary outcomes based on level of compliance, as they had compliance data.</td>
</tr>
<tr>
<td>Harms</td>
<td>19</td>
<td>All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)</td>
<td>209</td>
<td>Subjective status of worse reported in table 1. At 1 month post intervention 5 out of Back School (n=22) and 3 out of CovaTech Pilates (n=21) reported being worse. But this may not be due to participation in the intervention as compliance was so poor; it could have been due to not doing exercises. No attempt made to clarify the relationship between compliance and primary outcomes.</td>
</tr>
<tr>
<td>Discussion</td>
<td>Limitations</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>-------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>209</td>
<td>Limitations of the study not addressed adequately. The benefit of Pilates comes from the gradual progression from easy to more challenging exercises under the guidance of an instructor over a prolonged period of time. The way this Pilates intervention was delivered was more like a Back School protocol 10 x 1 hr lessons over 10 days then sent away to practice at home (the expected frequency and duration of home practice is not stated). This is perhaps why the results achieved by the Back School and Pilates intervention were so similar. Difficult to comment on precision with no C.I.s published. No mention of MCID.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>205 - 206</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Generalisability</th>
<th>21</th>
<th>Generalisability (external validity, applicability) of the trial findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>206</td>
<td>No idea how many males or females were included in the sample. 53 participants in total, with a mean age of 50.08, the mean age of males 49, and females 50.65 so I guess there were more females than males in the sample.</td>
<td></td>
</tr>
<tr>
<td>209</td>
<td>Not compared to any other trials, but states that results achieved with back school intervention confirm results of previous trials referenced. Authors claim better compliance and subjective feeling of satisfaction with Pilates treatment, but we don't know if these differences are statistically significant when compared to Back school group.</td>
<td></td>
</tr>
</tbody>
</table>
Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence.

Results not discussed in relation to other studies. Discussion of results not systematic. Interpretation of the Pilates intervention being more easily modified or personalised for the participant in the small group session leading to its increased compliance and improvement of symptoms is a tenuous link considering the results for primary outcomes in both groups were so similar.

<table>
<thead>
<tr>
<th>Other information</th>
<th>23</th>
<th>Registration number and name of trial registry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol</td>
<td>24</td>
<td>Where the full trial protocol can be accessed, if available</td>
</tr>
<tr>
<td>Funding</td>
<td>25</td>
<td>Sources of funding and other support (such as supply of drugs), role of funders</td>
</tr>
</tbody>
</table>

Registration of trial not mentioned.

Not mentioned.

Not mentioned.

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.

dealing with a not very disabled group (like me)
**CONSORT 2010 checklist of information to include when reporting a randomised trial**

<table>
<thead>
<tr>
<th>Section/Topic</th>
<th>Item No</th>
<th>Checklist item</th>
<th>Reported on page No</th>
<th>Reason</th>
</tr>
</thead>
<tbody>
<tr>
<td>Title and abstract</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1a</td>
<td>Identification as a randomised trial in the title</td>
<td>1</td>
<td>No, not an RCT. Described as a comparative study</td>
</tr>
<tr>
<td></td>
<td>1b</td>
<td>Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)</td>
<td>1</td>
<td>No structure to abstract. No mention of number of participants, methods. Results described but not backed up with any data.</td>
</tr>
<tr>
<td>Introduction</td>
<td>2a</td>
<td>Scientific background and explanation of rationale</td>
<td>1-2</td>
<td>Rational for all groups receiving exercise is that in studies comparing exercise to no exercise, the results have generally had favoured exercise for CLBP. No rational provided as to why the researcher would be interested in load transfer through the pelvis in patients with CLBP.</td>
</tr>
<tr>
<td></td>
<td>2b</td>
<td>Specific objectives or hypotheses</td>
<td>1</td>
<td>The objective stated in the summary was to compare the effects of 3 different Pilates regimes (in reality they are very similar).</td>
</tr>
<tr>
<td>Methods</td>
<td>3a</td>
<td>Description of trial design (such as parallel, factorial) including allocation ratio</td>
<td>2</td>
<td>The study design was between subjects’ equivalent group experiment with the independent variable being the type of exercise (3 groups) and the dependent variable being the load transfer through the pelvis and low back pain symptoms. What would be of interest would be the difference in these outcomes.</td>
</tr>
</tbody>
</table>

**Altered motor control, posture and the Pilates method of exercise prescription**

Dorothy Curnow, MA. Deirdre Cobbin, PhD, PhD, Jennifer Wyndham, BSc, MSc, MPH, GCHE, S.T.Boris Choy, BSc, MPhil, PhD

*Journal of Bodywork and Movement Therapies (2008)*
measures pre-post intervention, rather than the difference between the 3 groups, but this is not in the study design. Allocation into 3 groups occurred after all were taught the same 4 exercises.

### Participants
- **4a** Eligibility criteria for participants
  - Not described at all. Unclear what constituted chronic LBP?

### Interventions
- **5** The interventions for each group with sufficient details to allow replication, including how and when they were actually administered
  - “Pilates” exercise intervention was described in enough detail. However not true Pilates as 6-10 repetitions of any one exercise is usual practice, not 40 repetitions.

### Outcomes
- **6a** Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed
  - Outcome measures poorly described, and how and when they were assessed is presented on flow chart pg4.
  - ODI not described, how frequency and intensity of pain was measured (on what scale) is not described.
  - None

### Sample size
- **7a** How sample size was determined
  - Unsure

### Randomisation:
- **8a** Method used to generate the random allocation sequence
  - Unsure of randomisation procedure (not described)
<table>
<thead>
<tr>
<th>Section</th>
<th>Details</th>
<th>Rating</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>generation</td>
<td>Type of randomisation; details of any restriction (such as blocking and block size)</td>
<td>4</td>
<td>Unsure of randomisation procedure. Groups of n = 13, 14, 12</td>
</tr>
<tr>
<td>Allocation concealment mechanism</td>
<td>Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned</td>
<td>2</td>
<td>Unsure of randomisation procedure.</td>
</tr>
<tr>
<td>Implementation</td>
<td>Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions</td>
<td>2</td>
<td>Unsure</td>
</tr>
<tr>
<td>Blinding</td>
<td>If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how</td>
<td></td>
<td>It is unclear whether the assessor was blinded</td>
</tr>
<tr>
<td></td>
<td>If relevant, description of the similarity of interventions</td>
<td>2</td>
<td>Interventions and control very similar.</td>
</tr>
</tbody>
</table>
| Statistical methods           | Statistical methods used to compare groups for primary and secondary outcomes                                                                                                                                                                                                                                                        | 3 - 7  | Many different tests were used for different outcome measures.  
For ODI Wilcoxon test used. However analysed individual questions pre - post is not the norm for ODI.  
For Frequency of Pain Fisher, and Scheffe Testes were used. Duration of pain it is unclear which test was used but the differences between the groups were apparently insignificant.  
Intensity of pain Krusal Wallis Test.  
Stork Test Wilcoxn (but only for group B?)  
No pre post analysis of individual groups was done |
<p>|                               | Methods for additional analyses, such as subgroup analyses and adjusted analyses                                                                                                                                                                                                                                                  |        |                                                                                                                                                                                                       |</p>
<table>
<thead>
<tr>
<th>Results</th>
<th>13a</th>
<th>13b</th>
<th>14a</th>
<th>14b</th>
<th>15</th>
<th>16</th>
<th>17a</th>
<th>17b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant flow (a</td>
<td></td>
<td></td>
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<tr>
<td>diagram is strongly</td>
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<td>recommended)</td>
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<tr>
<td>For each group, the</td>
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<tr>
<td>numbers of participants</td>
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<tr>
<td>who were randomly</td>
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<tr>
<td>assigned, received</td>
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</table>
Ancillary analyses 18 Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory

Harms 19 All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)

Discussion

Limitations 20 Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses

<table>
<thead>
<tr>
<th>Limitations</th>
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<th>Discussion</th>
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Generalisability  

21 Generalisability (external validity, applicability) of the trial findings

No description of the characteristics of participants, the trial setting, or the eligibility criteria make it very difficult to envisage the sample population. Only results talked about were the differences between the groups (which were usually not significant), and not the differences pre – post intervention, which can be seen on the graphs pg 6 but there is no statistical analysis to accompany these results, they are only interested in presenting inter group comparisons.

Interpretation  

22 Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence

Not compared to previous Pilates research. To be fair this is not Pilates. The authors’ justification for using so few exercises in the intervention is so that it would not be difficult to discern which exercises were effective.

In the conclusions it is stated that all groups experienced a reduction in the mean number of days in pain, duration and intensity of pain each week. It is unclear if this is in relation to the pre intervention measures, or the previous week (which would be untrue for group A at least). If these results were statistically significant within groups as stated, why not present this data? The differences between groups were not significant.

Groups B and C achieved greater reduction in symptoms but since we don’t know what scale the reduction is measured on it is unclear whether this reduction is significant or coincidence.
<table>
<thead>
<tr>
<th>Registration</th>
<th>23</th>
<th>Registration number and name of trial registry</th>
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<tbody>
<tr>
<td>Protocol</td>
<td>24</td>
<td>Where the full trial protocol can be accessed, if available</td>
</tr>
<tr>
<td>Funding</td>
<td>25</td>
<td>Sources of funding and other support (such as supply of drugs), role of funders</td>
</tr>
</tbody>
</table>

Registration of trial or Ethics approval not mentioned.

Not mentioned

Not mentioned.

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see [www.consort-statement.org](http://www.consort-statement.org).
Appendix G: Outcome Measures
<table>
<thead>
<tr>
<th>Variable</th>
<th>Explanation</th>
<th>Measurement Tool</th>
<th>Reliability</th>
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</thead>
<tbody>
<tr>
<td><strong>Demographic and Anthropomorphic</strong></td>
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<td></td>
</tr>
<tr>
<td>Age</td>
<td>ranging from 25-65</td>
<td>single question</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>male or female</td>
<td>single question</td>
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<tr>
<td>Body Mass Index</td>
<td>weight(kg)/height(m)^2</td>
<td>scales and standing height measurement</td>
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<tr>
<td>Education Level</td>
<td>7 categories ranging from ‘no formal schooling’ to ‘postgraduate degree completed’</td>
<td>As recommended by Pincus et al. (2008)</td>
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<tr>
<td>Work Status</td>
<td>9 categories including full-time, part-time or reason for not working</td>
<td>As recommended by Pincus et al. (2008)</td>
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<td><strong>Characteristics of pain</strong></td>
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<tr>
<td>LBP intensity</td>
<td>11-point numeric rating scale question regarding bothersomeness of LBP over the past week</td>
<td>11-point numeric rating scale (Farrar et al. 2001)</td>
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<tr>
<td>Leg pain intensity</td>
<td>11-point numeric rating scale question regarding bothersomeness of leg pain over the past week</td>
<td>11-point numeric rating scale (Farrar et al. 2001)</td>
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<tr>
<td>Mode of onset of LBP</td>
<td>gradual or sudden</td>
<td>verbal history</td>
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<td>History of traumatic onset of LBP</td>
<td>yes or no</td>
<td>verbal history</td>
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<tr>
<td>Duration of LBP</td>
<td>Initial onset of LBP (in years)</td>
<td>verbal history</td>
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<tr>
<td>Troublesomeness of LBP</td>
<td>5-point Likert scale rating troublesomeness of LBP</td>
<td>Troublesomeness Questionnaire (Parsons et al. 2006)</td>
<td>ICC=.59-.91</td>
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<td><strong>Functional Disability</strong></td>
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<tr>
<td>Disability as a result of LBP</td>
<td>Measures perceived restriction in common activities of daily living as a result of LBP</td>
<td>Oswestry Disability Questionnaire (Davidson and Keating 2002)</td>
<td>ICC=.80</td>
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<tr>
<td>Patient-specific disability rating</td>
<td>Rating of functional status of 3-5 self-selected activities affected by LBP</td>
<td>Patient-Specific Functional Scale (Stratford et al. 1995)</td>
<td>ICC=.97</td>
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<tr>
<td><strong>Psychosocial Factors (not directly related to activity)</strong></td>
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<tr>
<td>Troublesomeness of body pains</td>
<td>Total score of troublesomeness rating of pain in 12 other body regions (not low back)</td>
<td>Troublesomeness Questionnaire (Parsons et al. 2006)</td>
<td>ICC=.59-.91</td>
</tr>
</tbody>
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Note: LBP = low back pain
Appendix H: Ethics approval for this study
Claire O'Brien and Leyla Okyay
12 Powell Street
Avondale
Auckland

30 March 2009

Dear Claire and Leyla

Your file number for this application: 2009-923

**Title:** This is a joint project entitled “Pilates exercise for low back pain”. The individual project titles are: The effectiveness of a 6 week Pilates exercise programme for adults with chronic non-specific low back pain 2) Development of a clinical prediction rule to determine successful outcome following a Pilates-based exercise programme in adults with non-specific chronic low back pain.

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

- Start date: 3 April 2009
- Finish date: 2 April 2010

Please note that:
1. the above dates must be referred to on the information AND consent forms given to all participants
2. you must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

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

Yours sincerely

[Signature]

Deborah Rolland
Deputy Chair, UREC

cc: Rob Moran
Appendix I: Information Sheet for Participants
Pilates Exercise for Chronic Low Back Pain

About this research
You are invited to take part in a research project that is being undertaken as part of the Unitec Master of Osteopathy degree. We will be studying the effects of a 6-week Pilates programme on chronic low back pain. The research will also seek to identify factors that may predict a reduction in low back pain.

The Researchers
The researchers are Claire O’Brien and Leyla Okyay, both in their final year of the Master of Osteopathy degree. Claire O’Brien will be your Pilates instructor. She has been teaching Pilates for 4 years and is certified through Pilates International, Australia. The research is supervised by Rob Moran, Associate Professor Andrew Stewart and Senior Lecturer Craig Hilton of the School of Health Sciences.

What will participation involve?
Once you have agreed to participate in the project and have signed the consent form, you will need to attend an initial appointment at the Unitec Osteopathy Clinic (Building 41, Entry 3, Carrington Rd, Mt Albert). The appointment will take about one hour and will involve the completion of some questionnaires, a brief interview and a physical assessment. These will help us to collect detailed information about your low back pain, medical history and daily activities. At this point we will make sure that there is nothing preventing you from participating in the Pilates programme. For the physical assessment you will be required to undress down to your underwear. We will provide you with loose-fitting shorts if you require. The physical examination might cause some discomfort but should be no more painful than activities you perform everyday.

You will then need to attend a Pilates programme consisting of 2 classes per week for 6 consecutive weeks. The classes are 1 hour long and held at the Pilates Body Studio, 2/141 Wellesley Street West, Freemans Bay, Auckland. You will need to arrange your own transport to the studio, but you will be provided with a $20 petrol voucher to cover some of the transport cost. Parking outside the studio costs $1 per hour, you will receive $12 to cover parking costs. The classes carry no charge. Class sizes may vary from 5-12 people and include both men and women. One week after the Pilates programme you will need to complete a final questionnaire and a flexibility test at the Unitec Osteopathy Clinic which will take about 20 minutes. The researchers may contact you between 3 and 12 months after completion of the programme for a short telephone follow-up about your low back pain.

Your involvement in this research will help to determine whether Pilates is an effective treatment for low back pain and if there are indicators that may predict a decrease in pain and disability. This information will be useful to doctors, therapist and patients in choosing treatments for back pain.

Selection of Participants
In order to participate you need to meet the following criteria:

- Be between 25-65 years of age
- Currently experience low back pain, and have had persistent or frequently recurring back pain for at least 6 months
- Are able to undertake non-vigorous exercise

You can not participate if:
You are already involved in regular Pilates classes, or a rehabilitative exercises programme

- You are pregnant (or suspect that you might be)
- You have been diagnosed with osteoporosis
- You have had any of the following in the last 12 months: a spinal fracture, spinal tumour, spinal infection, surgery to your spine, or abdominal surgery

Potential Risks to Participants
Any form of exercise carries the risk of potential injury. To minimise harm we will screen for any medical problems that may make participation in physical exercise inappropriate. All exercises in this programme are designed for people with low back pain and will be individualised to suit your level of ability. The Pilates exercises should cause you no pain. However, if you are uncomfortable with performing any of the exercises you need to inform the Pilates instructor immediately. The instructor will provide you with an alternative exercise or give you some time to rest. You can withdraw from the programme at any time, for any reason.

Confidentiality
Confidentiality and your anonymity will be protected in the following ways:

- As many of the questions are of a personal nature we ask that you do not write your name anywhere on the questionnaire. This is important to protect your anonymity.
- You will be given an ID number on enrolment in the study, which is printed on the questionnaires. This is so that we can compare your pain measurement at the beginning and end of the Pilates programme with your answers to the questions. Your name, or any other information that could identify you, will be stored separately.
- The completed questionnaires will be seen only by the researchers.
- Once the research has been completed, your name and your questionnaire number will be deleted from all records so that you cannot be identified. All computer records will only be accessible by passwords held by the researchers. All hard copies will be stored in a locked file, accessible only by the researchers.
- Information gathered during this research will be held for 5 years before being destroyed.

You have the right to withdraw your data from this research project at any time within 1 week of your final data collection (1 week after the final interview). This can be done by contacting one of the researchers listed below.

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

Information and concerns
If you require any further information about the project please contact us by phone or email:

Claire O’Brien  Leyla Okyay  Rob Moran
Tel.: 09 550 3212  Tel.: 09 550 3212  Tel.: 09 815 4321 ext 8642
Senior
Tel.: 021 55 84 55  Mob.: 021 142 42 61  Tel.: 09 815 4321 ext 8642
Pilates.research@gmail.com  Pilates.research@gmail.com  rmoran@unitec.ac.nz

Finally, we would like to thank you for your interest in contributing to this research project.
UREC REGISTRATION NUMBER: 2009-923
This study has been approved by the UNITEC Research Ethics Committee from 3rd April 2009 to 2nd April 2010. If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the Secretary (ph: 09 815-4321 ext 7248). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.
Appendix J: Telephone Screen for Eligibility
TELEPHONE SCREENING QUESTIONS

Hi, Who am I speaking with? Name ____________________________ Age? ________

Blurb about research: Pilates for low back pain by Unitec Master of Osteopathy students. Part of a research team trying to find out if Pilates is able to decrease low back pain....

Interested? Great, can I ask you a few questions to make sure that you can take part in the study?

1. Do you currently have LBP? Yes go to Q4
2. When was the last time you had LBP?
3. How long did it last (duration of episode)?
4. How long have you had LBP? < 6 months - exclude
5. Would you be willing to commit to attending 2x 1hr sessions per week for 6 weeks in Freemans Bay, Auckland (near Victoria Park Market)?
6. Are you currently participating in regular Pilates classes? Or having treatment/doing exercises specifically for low back pain? Yes, explain? __________How often? _______Exclude if treatment more than 1/month
7. Women only: Are you currently pregnant or is there a possibility that you might be pregnant or are planning on becoming pregnant during the next 2 months? Yes - exclude
8. Has your doctor recommended that you abstain from participating in physical activity? Yes - why? _______________________________Exclude if yes for non-vigourous exercise
10. Has there been any concern about your bone density? Yes - exclude
11. What effect does coughing/ sneezing/ straining have on the pain? ____________Active Disc Herniation
12. Recently, have you experienced any symptoms in your legs - like pain, weakness, stiffness or numbness? Yes, please explain _______________________________Stenosis, Nerve root compression, Rheumatologic
13. Do you have stiffness or pain in the morning when you wake up? If yes, duration? _______________________________ > 1 hour - exclude
14. Do you have any numbness or tingling in your groin/inside thighs? Yes - exclude
15. Is your pain better, worse or unchanged for activity_______________
16. Is your pain better, worse or unchanged for rest_____________________
17. Do you have trouble urinating or controlling your bowel and bladder? Yes – exclude
18. Do you have unrelenting pain at night? recent unplanned weightloss? Yes - exclude
19. Have you ever be diagnosed with cancer, including skin cancer? Type/ Location? ____________ Yes - exclude
20. Do you have a history of psoriasis, diarrhoea, eye trouble, or severe pain in the joints of hands or feet joints? ______________________________Psoriatic arthritis, Reiters.
21. Have you recently been feeling unwell? Details
Appendix K: Consent Form
Pilates Exercise for Chronic Low Back Pain

This research project examines the effects of a 6-week Pilates programme on chronic low back pain, and will determine what indicators might predict a successful outcome. The research is being conducted by Claire O’Brien and Leyla Okyay, Master of Osteopathy students at Unitec, and will be supervised by Rob Moran, Associate Professor Andrew Stewart and Senior Lecturer Craig Hilton.

Name of Participant:……………………………………………………………………

I have seen the Information Sheet for participants in the project titled “Pilates Exercise for Chronic Low Back Pain”. I have had the opportunity to read the contents of the information sheet and to discuss the project with a member of the research team and I am satisfied with the explanations I have been given. I understand that taking part in this project is voluntary (my choice) and that I may withdraw from the project at any time (refer below) and this will in no way affect my access to the services provided by the Unitec Osteopathy Clinic or Unitec NZ.

I understand that I can withdraw from the study, for any reason, up to 1 week after the last data collection, but no later.

I understand that my participation in this project is confidential and that no material that could identify me will be used in any reports on this project.

I have had enough time to consider whether I want to take part.

I know whom to contact if I have any questions or concerns about the project.

The researchers for this project are:
Claire O’Brien            Leyla Okyay            Rob Moran
Tel.: 09 550 3212            Tel.: 09 550 3212            Senior
Lecturer - Osteopathy
Mob.: 021 55 84 55            Mob.: 021 142 42 61            Tel.: 09 815 4321 ext 8642
Pilates.research@gmail.com    Pilates.research@gmail.com    rmoran@unitec.ac.nz

Participant Signature…………………………………………             ………....(date)

Project explained by…………………………………………………………

Researcher Signature……………………………………………………………             ………....(date)

The participant should retain a copy of this consent form.

UREC REGISTRATION NUMBER: 2009-923
This study has been approved by the UNITEC Research Ethics Committee from 3rd April 2009 to 2nd April 2010. If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the Secretary (ph: 09 815-4321 ext 7248).
Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.
Appendix L: Pre-intervention Questionnaire
QUESTIONNAIRE

Pilates Exercise for Chronic Low Back Pain

Welcome and thank you for participating in this study.

Please take time to read the questions carefully and answer them truthfully. If you are not sure how to answer a question, please mark it with a question mark (?) and we will clarify during the interview.

To protect your anonymity please DO NOT write your name anywhere on the questionnaire.

Section 1:

Date of Birth: _______ / _______ / _______  Gender: Male ☐ Female ☐

1. What is the highest level of education that you have completed?
   - No formal schooling
   - Less than primary school
   - Primary school completed
   - Intermediate school completed
   - High School (or equivalent) completed
   - Tertiary degree or diploma completed
   - Postgraduate degree completed

2. At present are you working?
   - Yes, full-time
   - Yes, part-time
   - Not working, reason:
     - Homemaker/ caring for family
     - Looked but can’t find a job
     - Doing unpaid work/ voluntary activities
     - Studies/ training
     - Retired/ too old to work
     - Ill health
     - Other (please state) __________________________
3. During the last 12 months what has been your main occupation?

- Legislator/ Senior official/ Manager.
- Professional (engineer, doctor, teacher, clergy, etc).
- Technician/ Associate Professional (inspector, finance, dealer, etc).
- Clerk (secretary, cashier, etc).
- Service/ Sales worker (cook, travel guide, shop salesperson, etc).
- Agriculture or Fishery worker (vegetable grower, livestock producer, etc).
- Craft or Trades worker (carpenter, painter, jewellery worker, butcher, etc).
- Plant /Machine Operator or Assembler (equipment assembler, sewing machine operator, driver, etc).
- Elementary worker (street food vendor, shoe cleaner, etc).
- Armed Forces (government military)

4. How satisfied are you with your work in general?

Extremely dissatisfied 0  1  2  3  4  5  6 Extremely satisfied

5. Medical History:

Do you currently have or have you ever been diagnosed with any of the following?

- Arthritis
- Asthma
- Anaemia
- Bowel/Bladder Changes
- Balance Problems
- Bursitis
- Cancer
- Diabetes
- Dizziness
- Fainting
- Disc bulge (herniated)
- Epilepsy
- Gynaecological problems
- Heart Attack
- Heart Palpitations
- Heart Disease
- Hyperglycemia
- Hypoglycemia
- High Blood Pressure
- Low Blood Pressure
- Numbness/weakness
- Osteoarthritis
- Osteoporosis
- Osteopenia
- Migraines
- Shortness of Breath
- Stenosis
- Thyroid Disorder
- Kidney Disorder
- Visual Disturbances

6. Are you currently taking any medication?

- No
- Yes (please state)

7. Are you currently receiving professional health care services? (eg. Osteopathy, Physiotherapy, Chiropractic, Massage, Medical Treatment)

For low back pain

- No
- Yes (please explain)

For another condition

- No
- Yes (please explain)
Section 2:
1. Do you get leg pain below the knee? Yes ☐ No ☐

2. In the past week how bothersome have the following symptoms been?
   (0–10, where 0 = no pain, 10 = worst pain imaginable)
   a. Lower Back Pain
      
      | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
      |---|---|---|---|---|---|---|---|---|---|----|
   b. Leg Pain
      
      | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
      |---|---|---|---|---|---|---|---|---|---|----|

3. I am going to ask you to identify at least three important activities that you are unable to do or are having difficulty with as a result of your low back pain. Please choose at least three activities and write them in the chart below, then score each activity from 0-10 according to the scale shown.

   Examples of activities: running, playing squash, getting out of bed, vacuuming, sitting for longer than 1 hour, playing soccer with your children, gardening, bending down to tie your shoe laces.

<table>
<thead>
<tr>
<th>Unable to perform activity</th>
<th>Able to perform activity at the same level as before injury or problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

   Activity | Score
   ---------------
   1.
   2.
   3.
   4.
   5.

   Average Score (we will calculate this)
Section 3:
1. During the past week, how troublesome have each of the following symptoms been? (Please put a cross (x) in the appropriate box on each row for each area that you have pain)

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No pain experienced</th>
<th>Not at all troublesome</th>
<th>Slightly troublesome</th>
<th>Moderately troublesome</th>
<th>Very troublesome</th>
<th>Extremely troublesome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>□</td>
<td>□</td>
<td>X</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Neck pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Shoulder pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Elbow pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Wrist/hand pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Chest pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Upper back pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Lower back pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Hip/thigh pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Knee pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Ankle/foot pain</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Other pains</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>
2. Location and Distribution of Symptoms

Please indicate where on your body you feel these sensations. Use the symbols below and please mark ALL areas.

Pins and needles: ooooo

Ache: xxxxx

Numbness: -----

Pain: ////

© NOI Australasia
**Section 4:**

This questionnaire is designed to give us information as to how your back pain has affected your ability to manage in everyday life. Please answer every question by placing a cross in the one box that best describes your condition today. We realize that you may feel that 2 of the statements may describe your condition, but please mark only the box that most closely describes your current condition.

<table>
<thead>
<tr>
<th>Pain Intensity</th>
<th>Standing</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ I can tolerate the pain I have without having to use pain medication.</td>
<td>□ I can stand as long as I want without increased pain.</td>
</tr>
<tr>
<td>□ The pain is bad, but I can manage without having to take pain medication.</td>
<td>□ I can stand as long as I want, but it increases my pain.</td>
</tr>
<tr>
<td>□ Pain medication provides me with complete relief from pain.</td>
<td>□ Pain prevents me from standing more than 1 hour.</td>
</tr>
<tr>
<td>□ Pain medication provides me with moderate relief from pain.</td>
<td>□ Pain prevents me from standing more than ½ hour.</td>
</tr>
<tr>
<td>□ Pain medication provides me with little relief from pain.</td>
<td>□ Pain prevents me from standing more than 10 minutes.</td>
</tr>
<tr>
<td>□ Pain medication provides me with no effect on my pain.</td>
<td>□ Pain prevents me from standing at all.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Personal Care (eg. Washing, Dressing)</th>
<th>Sleeping</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ I can take care of myself normally without causing increased pain.</td>
<td>□ Pain does not prevent me from sleeping well.</td>
</tr>
<tr>
<td>□ I can take care of myself normally, but it increases my pain.</td>
<td>□ I can sleep well only by using pain medication.</td>
</tr>
<tr>
<td>□ It is painful to take care of myself, and I am slow and careful.</td>
<td>□ Even when I take pain medication, I sleep less than 6 hours.</td>
</tr>
<tr>
<td>□ I need help, but I am able to manage most of my personal care.</td>
<td>□ Even when I take pain medication, I sleep less than 4 hours.</td>
</tr>
<tr>
<td>□ I need help everyday in most aspects of my care.</td>
<td>□ Even when I take pain medication, I sleep less than 2 hours.</td>
</tr>
<tr>
<td>□ I do not get dressed, wash with difficulty, and stay in bed.</td>
<td>□ Pain prevents me from sleeping at all.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lifting</th>
<th>Social Life</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ I can lift heavy weights without increased pain.</td>
<td>□ My social life is normal and does not increase my pain.</td>
</tr>
<tr>
<td>□ I can lift heavy weights, but it causes increased pain.</td>
<td>□ My social life is normal, but it increases my level of pain.</td>
</tr>
<tr>
<td>□ Pain prevents me from lifting heavy weights off the floor, but I can manage if the weights are conveniently positioned (eg. on a table)</td>
<td>□ Pain prevents me from participating in more energetic activities (eg. sports, dancing).</td>
</tr>
<tr>
<td>□ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.</td>
<td>□ Pain prevents me from going out very often.</td>
</tr>
<tr>
<td>□ I can lift only very light weights.</td>
<td>□ Pain has restricted my social life to my home.</td>
</tr>
<tr>
<td>□ I cannot lift or carry anything at all.</td>
<td>□ I have hardly any social life because of my pain.</td>
</tr>
<tr>
<td>Walking</td>
<td>Travelling</td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td>------------------------------------------------------</td>
</tr>
<tr>
<td>□ Pain does not prevent me from walking any distance.</td>
<td>□ I can travel anywhere without increased pain.</td>
</tr>
<tr>
<td>□ Pain prevents me from walking more than 1.6km.</td>
<td>□ I can travel anywhere, but it increases my pain.</td>
</tr>
<tr>
<td>□ Pain prevents me from walking more than 800m.</td>
<td>□ My pain restricts my travel over 2 hours.</td>
</tr>
<tr>
<td>□ Pain prevents me from walking more than 400m.</td>
<td>□ My pain restricts my travel over 1 hour.</td>
</tr>
<tr>
<td>□ I can only walk with crutches or a cane.</td>
<td>□ My pain restricts my travel to short necessary journeys under ½ hour.</td>
</tr>
<tr>
<td>□ I am in bed most of the time and have to crawl to the toilet.</td>
<td>□ My pain prevents all travel except for visits to the doctor/therapist or hospital.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sitting</th>
<th>Employment/Home making</th>
</tr>
</thead>
<tbody>
<tr>
<td>□ I can sit in any chair as long as I like</td>
<td>□ My normal homemaking/job activities do not cause pain.</td>
</tr>
<tr>
<td>□ I can only sit in my favourite chair as long as I like.</td>
<td>□ My normal homemaking/job activities increase my pain, but I can still perform all that is required of me.</td>
</tr>
<tr>
<td>□ Pain prevents me from sitting for more than 1 hour</td>
<td>□ I can perform most of my homemaking/job duties, but pain prevents me from performing more physically stressful activities (eg. lifting, vacuuming)</td>
</tr>
<tr>
<td>□ Pain prevents me from sitting for more than ½ hour.</td>
<td>□ Pain prevents me from doing anything but light duties.</td>
</tr>
<tr>
<td>□ Pain prevents me from sitting for more than 10 minutes.</td>
<td>□ Pain prevents me from doing even light duties.</td>
</tr>
<tr>
<td>□ Pain prevents me from sitting at all.</td>
<td>□ Pain prevents me from performing any job or homemaking chores.</td>
</tr>
</tbody>
</table>
Appendix M: History and Physical Examination Form
History of Lower Back Pain

<table>
<thead>
<tr>
<th>Mode of onset</th>
<th>Gradual / Sudden / Traumatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>When did it start?</td>
<td>How?</td>
</tr>
<tr>
<td>Frequency of episodes</td>
<td></td>
</tr>
<tr>
<td>How has it progressed?</td>
<td></td>
</tr>
<tr>
<td>Duration of symptoms.</td>
<td></td>
</tr>
<tr>
<td>How long does the pain last?</td>
<td></td>
</tr>
<tr>
<td>Daily Pattern</td>
<td></td>
</tr>
<tr>
<td>Aggravating Factors</td>
<td></td>
</tr>
<tr>
<td>Relieving Factors</td>
<td></td>
</tr>
<tr>
<td>Response to prior treatments</td>
<td></td>
</tr>
<tr>
<td>Associated Symptoms</td>
<td></td>
</tr>
<tr>
<td>Saddle anaesthesia?</td>
<td>Incontinence?</td>
</tr>
</tbody>
</table>

Height _________ cm

Weight _________ kg

Finger tip to floor _________ cm
Standing

**Aberrant motions with flexion**

☐ Painful arc
☐ Painful arc on return
☐ Gower’s Sign
☐ Instability catch
☐ Reverse Lumbopelvic Rhythm
☐ *Hands flat on floor (LLS)*

Shober Index ___________ cm

**Supine**

**Leg Length** *(ASIS to med malleolus)*

Right ___________ cm
Left ___________ cm

<table>
<thead>
<tr>
<th>SLR</th>
<th>Passive</th>
<th>Active</th>
<th>Pain? A/P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reflexes</th>
<th>L</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patella</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Achilles</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Myotomes</th>
<th>Flex</th>
<th>Ext</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip</td>
<td>R L</td>
<td>R L</td>
</tr>
<tr>
<td>Knee</td>
<td>R L</td>
<td>R L</td>
</tr>
<tr>
<td>Ankle</td>
<td>R L</td>
<td>R L</td>
</tr>
</tbody>
</table>

**Dermatomes**

Light touch / Sharp touch

Seated

**Ligamentous Laxity Scale**

<table>
<thead>
<tr>
<th>Hyperextension</th>
<th>Left</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow &gt;10°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Little finger &gt;90°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thumb to wrist</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee &gt;10°</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Movement Control Tests**

Sitting knee extn ☐

4 point kneeling ☐Rocking back ?F
☐Rocking fwd ?E

Prone knee bend ☐Flexion
☐Rotation

**Prone**

**Segmental Mobility / Prone Instab**

<table>
<thead>
<tr>
<th></th>
<th>↑ Norm</th>
<th>↓ Pain</th>
<th>P.I +ve</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LS</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Endurance Tests**

Extensor ___________ mins/secs
L Lateral ___________ mins/secs
R Lateral ___________ mins/secs
Flexor ___________ mins/secs
Appendix N: Post-intervention Questionnaire
Pilates Exercise for Chronic Low Back Pain

Please take time to read the questions carefully and answer them truthfully. If you are not sure how to answer a question, please mark it with a question mark (?) and we will clarify during the interview. To protect your anonymity please DO NOT write your name anywhere on the questionnaire.

Section 1:
1. Do you get leg pain below the knee?   Yes ☐    No ☐

2. In the past week how bothersome have the following symptoms been?
   (0–10, where 0 = no pain, 10 = worst pain imaginable)
   a. Lower Back Pain

   0 1 2 3 4 5 6 7 8 9 10

   b. Leg Pain

   0 1 2 3 4 5 6 7 8 9 10

Section 2:
1. When we assessed you initially, you told us that you had difficulty with the activities listed below. Today, do you still have difficulty with these activities?

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unable to perform activity</td>
<td>Able to perform activity at the same level as before injury or problem</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Activity | Score
---|---
1. | 
2. | 
3. | 
4. | 
5. | 
Average Score | 
**Section 3:**
During the past week, how troublesome have each of the following symptoms been? (Please put a cross (x) in the appropriate box on each row for each area that you have pain)

<table>
<thead>
<tr>
<th>Pain Location</th>
<th>No pain experienced</th>
<th>Not at all troublesome</th>
<th>Slightly troublesome</th>
<th>Moderately troublesome</th>
<th>Very troublesome</th>
<th>Extremely troublesome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head ache</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Neck pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Shoulder pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Elbow pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Wrist / hand pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Chest pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Upper back pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Lower back pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Hip/thigh pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Knee pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Ankle/foot pain</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
<tr>
<td>Other pains</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
<td>[]</td>
</tr>
</tbody>
</table>
**Section 4:**
This questionnaire is designed to give us information as to how your back pain has affected your ability to manage in everyday life. Please answer every question by placing a cross in the one box that best describes your condition today. We realize that you may feel that 2 of the statements may describe your condition, but please mark only the box that most closely describes your current condition.

<table>
<thead>
<tr>
<th>Pain Intensity</th>
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</tr>
</thead>
<tbody>
<tr>
<td>☐ I can tolerate the pain I have without having to use pain medication.</td>
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<tr>
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<tr>
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<tr>
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<td>☐ Pain prevents me from standing more than ½ hour.</td>
</tr>
<tr>
<td>☐ Pain medication provides me with little relief from pain.</td>
<td>☐ Pain prevents me from standing more than 10 minutes.</td>
</tr>
<tr>
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<td>☐ Pain prevents me from standing at all.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Personal Care (eg. Washing, Dressing)</th>
<th>Sleeping</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ I can take care of myself normally without causing increased pain.</td>
<td>☐ Pain does not prevent me from sleeping well.</td>
</tr>
<tr>
<td>☐ I can take care of myself normally, but it increases my pain.</td>
<td>☐ I can sleep well only by using pain medication.</td>
</tr>
<tr>
<td>☐ It is painful to take care of myself, and I am slow and careful.</td>
<td>☐ Even when I take pain medication, I sleep less than 6 hours.</td>
</tr>
<tr>
<td>☐ I need help, but I am able to manage most of my personal care.</td>
<td>☐ Even when I take pain medication, I sleep less than 4 hours.</td>
</tr>
<tr>
<td>☐ I need help everyday in most aspects of my care.</td>
<td>☐ Even when I take pain medication, I sleep less than 2 hours.</td>
</tr>
<tr>
<td>☐ I do not get dressed, wash with difficulty, and stay in bed.</td>
<td>☐ Pain prevents me from sleeping at all.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lifting</th>
<th>Social Life</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ I can lift heavy weights without increased pain.</td>
<td>☐ My social life is normal and does not increase my pain.</td>
</tr>
<tr>
<td>☐ I can lift heavy weights, but it causes increased pain.</td>
<td>☐ My social life is normal, but it increases my level of pain.</td>
</tr>
<tr>
<td>☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if the weights are conveniently positioned (eg. on a table)</td>
<td>☐ Pain prevents me from participating in more energetic activities (eg. sports, dancing).</td>
</tr>
<tr>
<td>☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.</td>
<td>☐ Pain prevents me from going out very often.</td>
</tr>
<tr>
<td>☐ I can lift only very light weights.</td>
<td>☐ Pain has restricted my social life to my home.</td>
</tr>
<tr>
<td>☐ I cannot lift or carry anything at all.</td>
<td>☐ I have hardly any social life because of my pain.</td>
</tr>
<tr>
<td>Walking</td>
<td>Travelling</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>------------------------------------------------</td>
</tr>
<tr>
<td>Pain does not prevent me from walking any distance.</td>
<td>I can travel anywhere without increased pain.</td>
</tr>
<tr>
<td>Pain prevents me from walking more than 1.6km.</td>
<td>I can travel anywhere, but it increases my pain.</td>
</tr>
<tr>
<td>Pain prevents me from walking more than 800m.</td>
<td>My pain restricts my travel over 2 hours.</td>
</tr>
<tr>
<td>Pain prevents me from walking more than 400m.</td>
<td>My pain restricts my travel over 1 hour.</td>
</tr>
<tr>
<td>I can only walk with crutches or a cane.</td>
<td>My pain restricts my travel to short necessary journeys under ½ hour.</td>
</tr>
<tr>
<td>I am in bed most of the time and have to crawl to the toilet.</td>
<td>My pain prevents all travel except for visits to the doctor/therapist or hospital.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sitting</th>
<th>Employment/Homemaking</th>
</tr>
</thead>
<tbody>
<tr>
<td>I can sit in any chair as long as I like</td>
<td>My normal homemaking/job activities do not cause pain.</td>
</tr>
<tr>
<td>I can only sit in my favourite chair as long as I like.</td>
<td>My normal homemaking/job activities increase my pain, but I can still perform all that is required of me.</td>
</tr>
<tr>
<td>Pain prevents me from sitting for more than 1 hour</td>
<td>I can perform most of my homemaking/job duties, but pain prevents me from performing more physically stressful activities (e.g. lifting, vacuuming)</td>
</tr>
<tr>
<td>Pain prevents me from sitting for more than ½ hour.</td>
<td>Pain prevents me from doing anything but light duties.</td>
</tr>
<tr>
<td>Pain prevents me from sitting for more than 10 minutes.</td>
<td>Pain prevents me from doing even light duties.</td>
</tr>
<tr>
<td>Pain prevents me from sitting at all.</td>
<td>Pain prevents me from performing any job or homemaking chores.</td>
</tr>
</tbody>
</table>

Section 5:
(We will complete this)

Fingertip to floor _____________cm

Schober Index _______________cm
Appendix O: Author Guide for Submission to the Journal of Bodywork and Movement Therapies
Journal of Bodywork and Movement Therapies

Guide for Authors

Official journal of the:

- Association of Neuromuscular Therapists
- Australian Pilates Method Association
- National Association of Myofascial Trigger Point Therapists, USA

Now indexed in Medline

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Acronyms used within the text are spelled out at the first location of usage and used as the acronym thereafter. For example, 'The location of a central trigger point (CTrP) is central to a taut fiber. The CTrP is palpated by....'

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