EFFECTS OF A THREE-WEEK HAMSTRINGS STRETCH PROGRAM ON MUSCLE EXTENSIBILITY AND STRETCH TOLERANCE IN PATIENTS WITH CHRONIC MUSCULOSKELETAL PAIN

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Candidate's Declaration

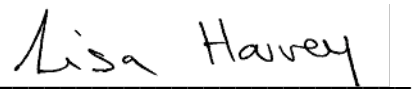
This is to certify that this thesis has not been submitted for a higher degree to any other university of institution. I hereby declare that this submission is entirely my own work and that it contains no material previously published or written by another person except where acknowledged in the text.

Roberta Law

(Master of Philosophy Candidate)

Supervisors' Statement

This is to certify that this thesis entitled "Effects of a three-week stretch program for patients with chronic musculoskeletal pain" submitted by Roberta Law in fulfilment of the requirements for the degree of Master of Philosophy in Medicine is suitable for examination.

Signed:  Date: 30-3-09

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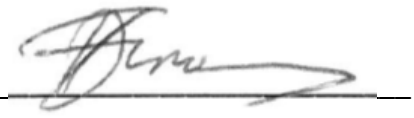
Signed:  Date: 2/04/2009

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Abstract

Background: Physical deconditioning is often associated with chronic pain and is believed to be a result of gradual movement inhibition and reduction of physical activities. It is common for chronic pain sufferers to present with limited muscle extensibility and poor tolerance to physical movement. Exercises are therefore prescribed to assist in regaining muscle extensibility, strength, fitness and endurance. Of particular interest is stretch, a type of exercise aimed at increasing muscle extensibility. Stretch is commonly prescribed as part of physical rehabilitation in pain management programs, yet little is known of its effectiveness in the chronic pain population.

Aim: The aim of this randomised controlled trial was to investigate the effects of a three-week stretch program on muscle extensibility and stretch tolerance in patients with chronic musculoskeletal pain.

Methods: Thirty adults with pain persisting for at least three months and limited hamstring muscle extensibility were recruited from patients enrolled in a multidisciplinary pain management program at a Sydney Hospital. A within-subject design was used, with one leg of each participant randomly allocated to an experimental (stretch) condition and the other to a control (no stretch) condition. The hamstring muscles of the experimental leg were stretched for one minute a day over a three-week period, whilst the hamstring muscles of the control leg were not stretched during this time. This intervention was embedded within a pain management program and supervised by physiotherapists. Primary outcome measures were muscle

extensibility and stretch tolerance, reflected by passive hip flexion angles produced with standardised and non-standardised torques, respectively. Initial measures were taken prior to the first stretch on day one and final measures were taken one to two days after the last stretch. A blinded assessor was used for all testing.

Results: After three weeks of intervention, stretch did not increase muscle extensibility (mean between-group difference in hip flexion was 1 degree; 95% CI -2 to 4 degrees) but did improve stretch tolerance (mean between-group difference in hip flexion was 8 degrees; 95% CI 5 to 10 degrees).

Conclusion: Three weeks of stretch increases tolerance to the discomfort associated with stretch but does not change muscle extensibility in patients with chronic musculoskeletal pain. This study provides support for the ongoing incorporation of stretch in pain management programs, where stretch may be conceptualised as a graded exposure to movement and assisting in the restoration of normal activity and function.

Chapter One: Introduction

1.1 Overview of the Introduction

This thesis investigates the role of stretch in chronic pain. The introduction is divided into two parts, providing a general overview of stretch and chronic pain. The first section focuses on stretch, its proposed mechanisms, and a review of current literature examining the efficacy of stretch in animals and humans for improving muscle extensibility. The second part of the introduction presents a review of chronic pain with an evaluation of different pain concepts and theories, followed by a brief overview of the assessment and management of chronic pain. Lastly, the role of stretch for the chronic musculoskeletal pain population is discussed.

1.2 Introduction to Stretch

1.2.1 Background

Muscle extensibility is an important aspect of normal human function. Limited extensibility has been associated with increased risk of injury and reduced levels of function and performance (Halbertsma *et al.*, 1999, Hartig and Henderson, 1999, Hreljac *et al.*, 2000). As a result, stretch exercises are used extensively in clinical rehabilitation and sports medicine in an attempt to improve and maintain muscle extensibility.

Stretch is used among a wide spectrum of people, ranging from recreational to competitive athletes, the disabled to the able-bodied, children and adolescents to adults. Yet despite the frequent use of stretch, its efficacy in increasing muscle extensibility has been controversial, with discrepancies between anecdotal evidence and good quality randomised controlled trials. Whilst the immediate (positive) effects of stretch have been well documented (Duong *et al.*, 2001, Herbert, 1993, Magnusson, 1998, Magnusson *et al.*, 2000, Magnusson *et al.*, 1996), the lasting effects of stretch are less convincing (Ada *et al.*, 2005, Ben *et al.*, 2005, Ben and Harvey, 2009, Folpp *et al.*, 2006, Halbertsma *et al.*, 1996, Harvey *et al.*, 2006, Harvey *et al.*, 2002, Harvey *et al.*, 2000, Harvey *et al.*, 2003, Harvey and Herbert, 2002, Lannin *et al.*, 2007, Lannin *et al.*, 2003, Turton and Britton, 2005).

Mounting evidence in recent years has highlighted the disparity between disabled and able-bodied populations on stretch efficacy, leading to further questions of the rationale behind stretch. Furthermore, it has been suggested that improvements in

range of motion shown in the able-bodied population following regular stretch are a result of apparent, rather than real, changes in muscle extensibility (Ben and Harvey, 2009, Bjorklund *et al.*, 2001, Chan *et al.*, 2001, Folpp *et al.*, 2006, Halbertsma and Goeken, 1994, Magnusson *et al.*, 1996). That is, regular stretch can alter and increase a person's tolerance to the uncomfortable stretch sensation and hence result in an apparent improvement in extensibility. This part of the introduction therefore aims to provide an insight into the body of work surrounding the topic of stretch and the important questions that remain to be answered.

1.2.2 Definitions

Discussions related to the topic of stretch will almost always include the following terms: stretch, stiffness, joint range of motion, flexibility, and extensibility.

Stretch, or stretching, is the act of applying tension to soft tissues with the aim of increasing muscle extensibility and hence improving joint range of motion (Harvey *et al.*, 2002). Stretch can be administered via several means depending on the desired duration. Shorter duration stretches can be either self-administered or manually applied by another person such as a treating therapist. Longer duration stretches are often administered via specially designed devices such as splints, casts and other equipments.

Stiffness is a term frequently used when referring to the passive mechanical properties of a muscle. Stiffness is defined as the rate of change in muscle tension (Newtons) with respect to the change in muscle length (millimetres) (Herbert, 1993). This relationship

can be represented by the length-tension curve (Figure 1). An accurate length-tension curve can only be attained if the muscle is isolated and measured *in vitro*. *In vivo*, this relationship is reflected by the rate of change in torque (Newton metres) with respect to the change in joint angle (degrees), as shown in Figure 2 (Herbert, 1993). Following the application of stretch, it is proposed that muscle stiffness is decreased through changes occurring in the passive viscoelastic properties of the muscle (Thacker *et al.*, 2004).

Joint range of motion (ROM) is measured by the number of degrees from the starting position of a body segment to its end position at full range of movement. Range of motion refers to the amount of movement attainable across a joint, and is therefore directly (but not solely) influenced by the soft tissues that cross the joint complex. The end position of a joint is often termed the "end of range", or "end range of motion". Range of motion is however a poorly defined term, as it depends on what is used to determine end of range. For example, some investigators use electromyographic (EMG) activity while others use patients' or therapists' perceptions of "pull". Other factors are also used to determine end of range; however, all will yield different results unless the torque is standardised.

The terms "flexibility" and "extensibility" are often used interchangeably. For the purposes of this thesis, the term "extensibility" will primarily be used. Many different interpretations of extensibility exist, but within the confines of this thesis, it will be used in reference to the passive mechanical properties of soft tissues spanning joints as reflected by either torque-angle or length-tension curves. Soft tissue extensibility is

dependent on the viscoelastic and mechanical properties of muscles, ligaments and other connective tissues. A change in extensibility in response to a stretch intervention must be accompanied by a change in the passive mechanical properties. This can only be verified by measures of joint angle taken with a standardised torque. There are two ways in which extensibility can change in response to stretch, as demonstrated in Figure 1. The left diagram (A) represents a real change in resting length with no change in stiffness (as indicated by the gradient), whilst the right diagram (B) represents a change in stiffness (gradient) with no change in resting muscle length. Changes to either of these factors will be reflected by an increase in joint angle with a standardised torque.

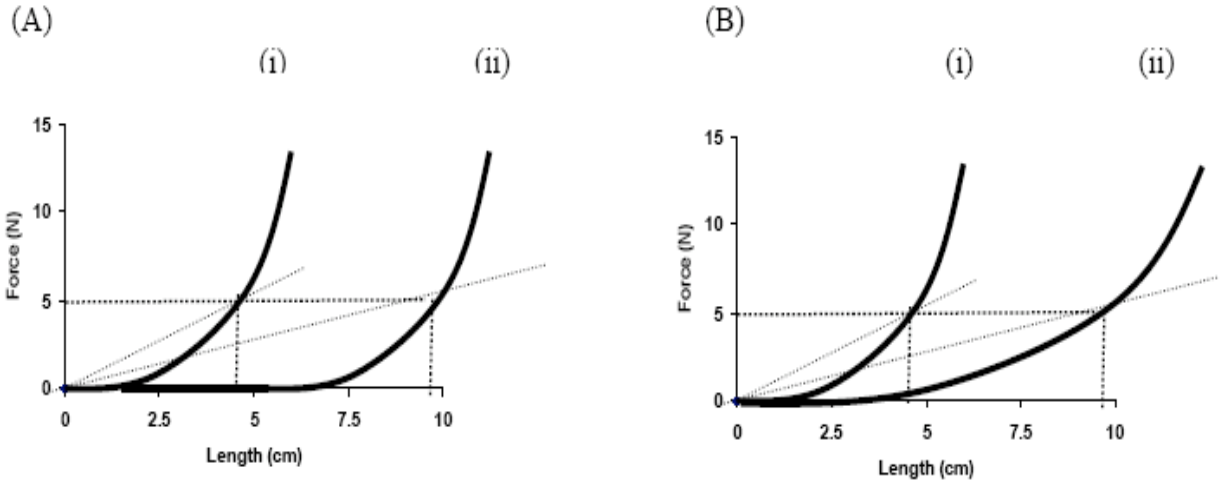

Figure 1. Length-tension curves. (A) represents a change in muscle length with no change in stiffness (gradient), (B) represents a change in stiffness (gradient) with no change in resting muscle length.

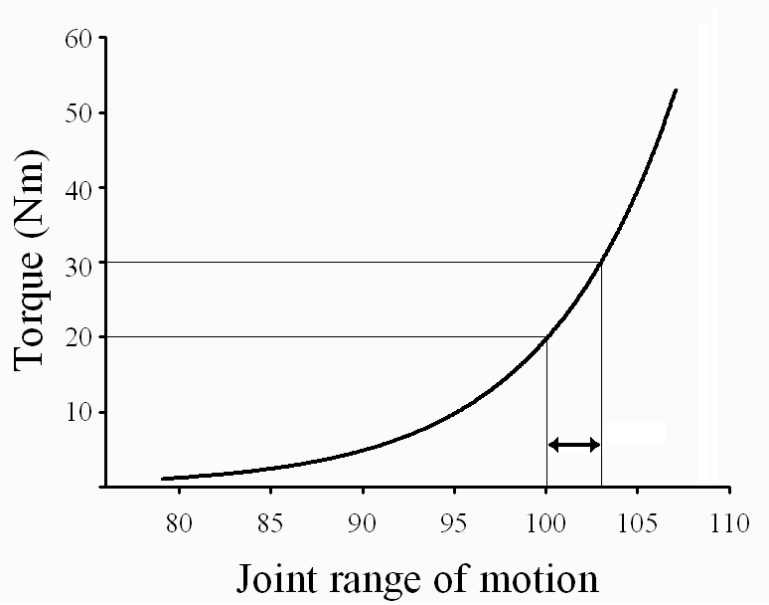

Figure 2. Torque-angle curve. The relationship between torque and angle is represented by the angle (joint range of motion) increasing along with the application of larger torques. The arrow indicates an *apparent* change in extensibility.

1.2.3 Types of Stretch

Numerous stretch techniques have been developed, applied and used by physiotherapists, coaches and trainers (Halbertsma *et al.*, 1999). These include isometric, ballistic, dynamic range of motion, proprioceptive neuromuscular facilitation, passive and static stretch (Bandy *et al.*, 1998).

Isometric stretch is similar to static stretch, comprising of sustained muscle lengthening with the addition of resistance against an immobile force. The opposing resistance can be provided by either an immobile object or another person. Ballistic stretch aims to provide rapid lengthening of the muscle by using jerky or bouncing movements. The dynamic range of motion stretch technique requires the antagonist muscle to contract, resulting in the joint crossed by the agonist (lengthening muscle) to move through the full range of motion at a slow, controlled speed. All movements are performed slowly and carefully (Bandy *et al.*, 1998). Proprioceptive neuromuscular

facilitation is a hold-relax stretch technique performed by another person (such as a therapist) together with the subject. Following a contraction of the antagonistic muscle, the partner performs a passive stretch on the subject.

The final two stretch methods are regularly administered and prescribed in both clinical and community settings. Both passive and static stretch techniques involve the performance of slow, sustained muscle lengthening. Passive stretches are performed by a person other than the subject, whereas static stretches are self-administered and performed by the subject independently. Depending on the situation and environment, passive stretches may be held for up to 30 to 60 minutes whilst static stretches are usually held for durations lasting between 15 to 60 seconds.

1.2.4 Effects of Stretch

Short-term effects

There is convincing evidence to show that stretch produces immediate increases in muscle extensibility due to the viscoelastic nature of soft tissues (Duong *et al.*, 2001, Herbert and Balnave, 1993, Magnusson, 1998, Magnusson *et al.*, 2000, Magnusson *et al.*, 1996). However, these short-term effects are transient and quickly dissipate. Upon the application of stretch to soft tissues over a period of seconds or minutes, the tissues undergo progressive deformation as a result of reduced resistance between collagen fibres. These fibres are therefore able to slide past each other more smoothly within the surrounding ground substance (Herbert, 1993). Subsequently, following viscoelastic deformation, soft tissues can be extended further with a constant force (the creep phenomenon) or the tissues exert less force when stretched to a constant

length (stress relaxation) (Taylor *et al.*, 1989).

Long-term effects

An increase in muscle extensibility is considered long-lasting if the effect remains for at least 24 hours upon removal of the last stretch (Harvey *et al.*, 2002). This lasting change in extensibility may be due to either changes in the passive mechanical properties of the muscle resulting in increased joint range of motion (indicating *real* extensibility), or changes in stretch tolerance resulting in improved range of motion without any underlying structural adaptations (indicating *apparent* extensibility). It is the long-term effects of stretch that are more controversial but arguably of more importance, particularly for patients with functional limitations, disabilities and chronic pain.

Despite the popularity and widespread implementation of stretch amongst the general community, sporting individuals and patients undergoing physical rehabilitation, considerable uncertainty remains surrounding its lasting effects. The controversy arises from the discrepancies between strong anecdotal and animal evidence supporting the effectiveness of stretch (Goldspink, 1977, Goldspink *et al.*, 1974, Williams and Goldspink, 1978), and high-quality randomised controlled trials indicating otherwise (Ada *et al.*, 2005, Ben *et al.*, 2005, Ben and Harvey, 2009, Folpp *et al.*, 2006, Halbertsma *et al.*, 1996, Harvey *et al.*, 2006, Harvey *et al.*, 2002, Harvey *et al.*, 2000, Harvey *et al.*, 2003, Harvey and Herbert, 2002, Lannin *et al.*, 2007, Lannin *et al.*, 2003, Turton and Britton, 2005).

The following section will present a literature review of the evidence surrounding stretch and its long-lasting effects on soft tissues in animal and human populations.

i) Evidence from Animal Studies

Research in animals has shown that soft tissues are highly adaptable and undergo structural remodelling in response to immobilisation in both shortened and lengthened positions. It is well known that muscles immobilised in the shortened position lead to a reduction in serial sarcomere number, although it is not clear how the sarcomeres are reduced (Coutinho *et al.*, 2004, Gomes *et al.*, 2004, Williams, 1988, 1990). A linear relationship between the position of immobilisation and the resting muscle length has been shown in rabbit soleus muscles (Herbert and Balnave, 1993). When a muscle is immobilised in a shortened position, physiological adaptations occur resulting in decreased muscle length and increased stiffness (Goldspink, 1977, Goldspink *et al.*, 1974, Gossman *et al.*, 1982, Herbert and Balnave, 1993, Tabary *et al.*, 1972, Williams and Goldspink, 1978). This response is believed to be due to the loss of sarcomeres in series within the muscle, causing a reduction in muscle fibre length. Several studies have shown that animal muscles can lose up to 40% of its sarcomeres in series following immobilisation in a shortened position (Goldspink *et al.*, 1974, Tabary *et al.*, 1972, Williams and Goldspink, 1973, 1978).

The reduction in muscle fibre length is accompanied by a loss in muscle extensibility. This is due to either or both of the following factors: 1) a shift of the length-tension curve to the left and/or 2) an increase in the slope of the curve. That is, the muscle becomes shorter and/or stiffer (Goldspink *et al.*, 1974, Williams and Goldspink, 1978).

However these changes in the length-tension curve appear to be reversible, with evidence showing that the curve returns to its normal shape four weeks following cast removal and restoration of normal activity (Tabary *et al.*, 1972).

Stiffness is represented by the slope of the length-tension curve (Figure 1), and is said to affect the passive viscoelastic properties of tissues (Thacker *et al.*, 2004). The increase in stiffness has been partly attributed to an increased amount of connective tissue (endomysium and perimysium) surrounding the muscle following periods of immobilisation (Cox *et al.*, 2000, Tabary *et al.*, 1972, Williams *et al.*, 1998, Williams and Goldspink, 1984). Cox *et al.* (2000) immobilised the rabbit latissimus dorsi muscle for three weeks and found a 4% increase in the amount of collagen around the intramuscular area. Additionally, realignment of collagen fibres after two weeks of immobilisation has been shown in the mouse soleus muscle, which may also affect muscle stiffness (Williams and Goldspink, 1984). However the role of connective tissue in post-immobilisation stiffness is unclear, and it is possible that other structures within the muscle fibres or soft tissues may be involved.

Periods of immobilisation in a shortened position also produce changes in tendons (Herbert and Crosbie, 1997). Herbert and Crosbie (1997) studied the soleus muscle-tendon unit of rabbits immobilised for 14 days. Following this period of immobilisation, the relative change in muscle length compared to tendon length was evaluated. Results showed that there was an overall reduction in the length of the muscle-tendon unit, and interestingly, the major contributing factor to the reduction in length was tendon rather than muscle. This highlights the possible involvement of

tendons (as part of the muscle-tendon unit) in structural adaptations and morphological changes following immobilisation. However the response of tendons to short duration stretches is unclear.

Similarly, structural adaptations in the muscle have also been shown to occur following immobilisation in the lengthened position. Evidence from animal studies have shown that the number of sarcomeres in series along the muscle fibre increases by up to 25%, with a corresponding decrease in resting sarcomere length (Goldspink *et al.*, 1974, Gossman *et al.*, 1982, Tabary *et al.*, 1972, Williams, 1990, Williams and Goldspink, 1973, 1978). The length of the actin and myosin filaments however remain unchanged, and therefore do not affect the amount of overlapping between the filaments (Tabary *et al.*, 1972). The increase in the number of sarcomeres in series leads to an increase in the length of the muscle fibre, and subsequently an overall increase in muscle extensibility. These adaptations are believed to be important for maintaining an optimal length-tension relationship.

In the clinical setting, it may be more relevant and worthwhile to consider the effects of shorter duration stretches on muscle extensibility. Intermittent static stretch has been shown to produce lasting structural changes in the rat soleus muscle (Williams, 1990). Williams (1990) demonstrated that 15 minutes of stretch performed every second day reduced the loss of sarcomeres, whereas 30 minutes of stretch every day was sufficient to prevent the loss of serial sarcomere number and maintain the range of motion in the rat soleus muscle. Furthermore, stretching for two hours every day increased the number of sarcomeres in series by 10%.

In summary, there is clear evidence to show that animal soft tissues are highly adaptable and undergo morphological remodelling in response to the position, duration, intensity and frequency of the immobilisation. Muscles immobilised in a shortened position lead to a reduction in its resting length, whereas muscles immobilised in a lengthened position result in an increase in its resting length. Even stretches of shorter durations (such as 15 minutes or less) may be sufficient to produce changes in muscle extensibility. There is however no distinct relationship between animal and human soft tissues, as human tissue morphology may be different to that of animals. The response of human tissues to stretch is discussed in the next section.

ii) Evidence from Human Studies

Clinical studies into the response of human tissue to stretch are less consistent. Many trials have investigated stretch in both the able-bodied and disabled populations, yet considerable controversy remains over the effectiveness of stretch. In human studies, it is difficult to isolate individual muscles to assess muscle morphology and extensibility. Instead, effects of stretch are often measured using joint range of motion (ROM) as a reflection of muscle extensibility.

The method through which joint ROM is measured plays a crucial role in the validity of the outcome. A *real* increase in muscle extensibility is demonstrated if the joint ROM is measured with a standardised torque. Without standardisation, changes in ROM can instead be influenced by the amount of torque applied and the amount of stretch tolerated. For example, the more tolerant an individual is to stretch, the larger the

torque applied (for example, by a therapist) and subsequently the greater the joint ROM. Hence measures of joint range with a non-standardised torque may merely reflect an *apparent* increase in extensibility, without any underlying change in the muscle properties themselves (as shown in Figure 2).

The second important issue when considering the methodology of human stretch studies is the timing of the final assessment. The effects of stretch are only considered long-lasting if the effects remain for at least 24 hours after the last stretch was administered (Harvey *et al.*, 2002). Initial short-term effects are due to viscoelastic deformation of tissues and any measurements made shortly after a stretch cannot provide an accurate indication of *real* muscle extensibility changes. It is therefore imperative that measurements of the final outcome are taken at least 24 hours after the most recent stretch application to reflect long-term changes. The failure to meet these two methodological criteria will affect the interpretation of the outcomes.

The following section reviews the current evidence surrounding stretch in:

1. Able-bodied populations, and
2. Disabled populations.

1. Able-bodied populations

Among physically active individuals in the general community, stretching before and after exercise has become routine practice. It is believed that stretch can improve flexibility, enhance performance and reduce injuries (Hartig and Henderson, 1999, Shrier, 2004, Thacker *et al.*, 2004). Strong anecdotal evidence exists demonstrating the

beneficial effects of stretch on extensibility, especially in sports such as gymnastics and dance where the muscles of these individuals are extremely extensible. This may be a response of soft tissues to the stretch stimulus and the constant exposure to it. Unfortunately, there remains a lack of strong evidence and high quality randomised controlled trials to support these anecdotal reports.

Two recent systematic reviews (Decoster *et al.*, 2005, Harvey *et al.*, 2002) reported initial evidence supporting the use of stretch in able-bodied individuals for improving muscle extensibility. Following specific inclusion and exclusion criteria, Decoster *et al.* (2005) identified 28 clinical trials (23 randomised and 5 non-randomised) and Harvey *et al.* (2002) identified 13 randomised controlled trials for review. The difference in the number of randomised controlled trials identified by the two authors reflects the differences in their inclusion criteria. Two independent assessors were used in both systematic reviews to critically evaluate the methodological qualities of the selected manuscripts according to the Physiotherapy Evidence Database (PEDro) (10-point) scale, which include criteria such as concealed allocation, blinding and intention-to-treat analysis (Maher *et al.*, 2003). PEDro scores of 7/10 or more were categorised as 'high' quality, scores of 5/10 or 6/10 were 'moderate', and scores of 4/10 or less were 'poor'. The PEDro scale is reported to have 'fair' to 'good' inter-rater reliability with a mean score of 0.55 (95% CI, 0.41 to 0.72) (Maher *et al.*, 2003).

Decoster *et al.* (2005) used broad inclusion criteria in their systematic review. Details of the 28 studies are provided in Table 1. Both randomised (n = 23) and non-randomised (n = 5) studies were included, and the timing of the final measurement

ranged from immediate to two days. There was no recognition of the significance between using a standardised versus a non-standardised torque for joint ROM measurements. Additionally, the heterogeneous nature of the studies included did not allow for meta-analysis of the results or calculation of effect sizes for comparison. Results showed that the overall methodological quality of the studies was 'poor' with a mean (SD) PEDro score of 4.3 (1.6), ranging from 2 to 8. Of the 28 studies, 12 did not state the timing of the final ROM measurement (post-stretch intervention), 10 performed the final assessment within the same day (0 to 90 minutes post-stretch), and only 6 of the studies performed the final measurement 1 to 2 days post-intervention.

Two studies were of particular interest, as they highlight the general misunderstanding between real and apparent changes in muscle extensibility (structural adaptations versus tolerance), and the linear relationship between passive stiffness and joint ROM (Chan *et al.*, 2001, Reid and McNair, 2004).

Chan *et al.* (2001) investigated the effects of two different static stretching programs on the flexibility and passive resistance of the hamstring muscles in 40 healthy young adults. The two experimental groups performed static stretches on the hamstring muscles of their dominant leg, whilst the two control groups performed no stretch. Group 1 performed one set of 5 x 30s stretches, three times a week for a total of eight weeks duration. Group 2 performed two sets of 5 x 30s stretches, three times a week for a total of four weeks. Group 3 acted as control (no stretch) for group 1, whilst group 4 acted as control for group 2. End ROM was determined by each subject's

perception of “maximum stretch without pain” and measured with a fixed goniometer at the knee joint. At 24 hours after the last stretch intervention, mean knee ROM improved significantly ($p < 0.05$) for both groups. The mean between-group differences (95% CI) for the 8-week (Groups 1 and 3) and 4-week protocols (Groups 2 and 4) were 12 degrees (95% CI, 3 to 20) and 9 degrees (95% CI, 2 to 15) respectively.

The authors suggested two possible mechanisms for the demonstrated increase in hamstring muscle extensibility. Firstly, it could be a result of physiological adaptations occurring in the visco-elastic properties of the muscle itself (*real* change). Secondly, it could be from increased tolerance to the stretch sensation (*apparent* change). However this study did not use a standardised torque to measure ROM (which would distinguish *real* from *apparent* changes in extensibility). Hence although muscle extensibility appears to have improved in both the 4-week and 8-week training groups, no conclusion can be made about the mechanism behind the observed changes.

Chan *et al.* (2001) also measured the passive resistance of the hamstring muscles at maximal joint angle (end ROM as determined by “maximum stretch without pain”). No change was observed between Groups 1 and 3 (8-week protocol; mean between-group difference -1 Nm; 95% CI, -2 to 0), whereas Groups 2 and 4 (4-week protocol) demonstrated a mean increase of 4 Nm (95% CI, 1 to 7) in the passive resistance of the hamstring muscles at maximal joint angle. The authors believed that four weeks of stretch was insufficient for changes to occur in tissue morphology and consequently passive resistance increased. In contrast, eight weeks of stretch was sufficient to induce adaptations in the underlying soft tissue properties and hence resistance

decreased. This is an interesting observation, as in fact, passive tissue tension has been shown to increase exponentially as joint angle increases (Toft *et al.*, 1989). This is a normal phenomenon of soft tissue structures. It is therefore not surprising that after four weeks of stretch, the increased end ROM (maximal joint angle) was accompanied by an increase in the passive resistance of the hamstring muscles. Measurements were taken at different points of the same torque-angle curve, with the application of a non-standardised torque. On the other hand, although a significant increase in end ROM was demonstrated following eight weeks of stretch, no accompanying change in passive resistance of the hamstring muscles was found. The authors explained this finding by concluding that structural adaptations had occurred within the muscles. However, a change in tissue properties may only be concluded if a reduction in force was attained at the same joint angle, or if a greater joint angle was achieved with the same torque. Hence considering that measurements were once again taken at different points on the torque-angle curve, these results do not necessarily reflect any structural change in the mechanical properties of the muscle.

Similarly, Reid and McNair (2004) also failed to recognise this normal phenomenon of soft tissues. They conducted a randomised controlled trial to examine the effect of a six-week hamstring muscle stretching program on knee extension ROM, passive resistive forces and muscle stiffness. Passive muscle stiffness was measured over the last 10% of knee extension ROM. Significant improvements in all three outcome measures were observed in the stretch group, but not the control group. A positive effect for hamstring muscle extensibility was shown with a mean between-group treatment effect of 7 degrees (95% CI, 2 to 12). Increased passive stiffness was also

observed at greater ROM. The authors concluded from these results that structural changes had occurred in the stretched muscles. However, the increased passive stiffness associated with measuring at a greater ROM is a typical phenomenon of soft tissues and is not necessarily indicative of structural adaptations in the muscle.

In summary, Decoster *et al.* (2005) reported positive treatment effects from stretch interventions in able-bodied individuals. The authors concluded that stretch appears to increase ROM in the hamstring muscles with a variety of techniques, positions and durations. However due to the lack of consideration for several methodological issues, the interpretation of these findings are questioned.

Another systematic review by Harvey *et al.* (2002) used more rigid selection criteria. Details of the studies are shown in shown in Table 2. Only randomised controlled trials, and those that measured final joint ROM at least one day (24 hours) after the last stretch intervention, were included. Out of the 13 studies selected, nine were of 'poor' quality and four were of 'moderate' quality. Treatment effect sizes were determined using meta-analyses. Results showed that from the four 'moderate' quality studies, the mean increase in joint ROM (evident 24 hours after the cessation of stretch) was 8 degrees (95% CI, 6 to 9 degrees). Similarly, the nine 'poor' quality studies demonstrated a mean increase of 6 degrees in joint ROM (95% CI, 5 to 8 degrees).

It is unclear whether the results of this systematic review reflected changes in *apparent* or *real* muscle extensibility. The majority of the studies (10 out of 13) used

either the 'assessor's perception of tightness' or the 'subject's perception of pain/stretch' as an indication of the end joint ROM. In this way, there was no standardisation of the applied torque during stretch and hence ROM changes shown may instead reflect an improvement in subjects' tolerance to the discomfort associated with stretch (*apparent* extensibility), as opposed to physiological changes in the underlying tissue structures (*real* extensibility).

In addition, post-hoc analyses found that joints with limited ROM appeared to respond better to stretch intervention (mean increase in joint ROM 8 degrees; 95% CI, 6 to 9 degrees) than joints with normal ROM (mean increase in joint ROM 4 degrees; 95% CI, 2 to 7 degrees). In the same way, stretch interventions administered over a longer period of time (more than three weeks; mean increase in ROM 8 degrees; 95% CI, 6 to 10 degrees) were shown to be more effective than interventions of shorter periods (such as three to eight days; mean increase in ROM 5 degrees; 95% CI, 3 to 8 degrees).

There were however limitations to this review by Harvey *et al.* (2002). It may be possible that studies with significant findings were excluded, merely due to the failure of authors to provide adequate methodological details in their published papers. It is also possible that other relevant papers of interesting findings were not identified. Furthermore some articles may not have been found if they were written in a language other than English, or if ambiguous keywords were used which did not match the keywords used in the search.

Three studies did use standardised torques for ROM measurements; however, results

from these studies produced inconclusive findings (Bohannon, 1984, Magnusson *et al.*, 1996, Medeiros *et al.*, 1977). Bohannon (1984) investigated the effects of loading the hamstring muscles (*n* = 10) for eight minutes a day over three consecutive days compared to a control group (*n* = 10). Hip flexion angles were measured cinematographically for each subject at 8 minutes, 10 minutes and 24 hours post-stretch using a within-subject standardised torque. At 24 hours post-intervention, the between-group (95% CI) mean difference in hip flexion was 4 degrees (95% CI, -6 to 13). Although the result provides some evidence of the stretch effects on muscle extensibility, the quality of this study was 'poor', and the wide 95% CI reflects the uncertainty surrounding the results.

Medeiros *et al.* (1977) also examined the effects of stretch on the hamstring muscles. The experimental group (*n* = 10) performed hamstring stretches for one minute daily over eight consecutive days, whilst the control group (*n* = 10) did not stretch during this period. Passive hip flexion angles were assessed using a within-subject standardised torque. At 24 hours post-intervention, the mean treatment effect was found to be 5 degrees (95% CI, 3 to 8). Similar to Bohannon (1984), the methodology of this study was 'poor', with the 95% CI also indicating the inconclusiveness of the results.

In contrast, the third study using a standardised torque to measure ROM by Magnusson *et al.* (1996) showed hamstring muscle stretching to have no effect on muscle extensibility. Subjects in the experimental group (*n* = 7) performed five 45-second stretches, twice daily, for 20 consecutive days. The control group (*n* = 7) did

not undergo any stretch during this time. The extensibility of the hamstring muscles was determined by measuring the resistance to stretch (passive torque) during a passive knee extension manoeuvre (hip joint stabilised) at a constant slow velocity (0.87 rad^{-1} second). Measurements were taken with a standardised torque (on the same point of the torque-angle curve), ensuring that outcomes will reflect *real* changes in muscle extensibility. At 24 hours following the final stretch intervention, no significant changes were evident in either muscle stiffness (mean between-group difference 0 Nm rad^{-1}) or joint ROM (ROM measures not reported by authors).

Although all three studies recognised the importance of using standardised torque for ROM measurements (Bohannon, 1984, Magnusson *et al.*, 1996, Medeiros *et al.*, 1977), the overall methodological quality was 'poor' and results did not provide any conclusive evidence on the effect of stretch on *real* muscle extensibility.

More recent randomised controlled trials have also investigated the relationship between stretch and muscle extensibility in healthy able-bodied individuals, as demonstrated in Table 3 (Davis *et al.*, 2005, Gajdosik *et al.*, 2007, Gajdosik *et al.*, 2005, Winters *et al.*, 2004). PEDro scores for methodological quality are shown in Table 4. The results of all four studies favoured treatment, showing a positive effect of stretch on extensibility. The duration of stretch treatment ranged from 30 seconds to five minutes per day, for a total period of between four to eight weeks. The mean treatment effect reported in these four studies ranged from 1 to 21 degrees (95% CI range, 2 to 27; insufficient data presented in Winters *et al.*, 2004).

The four studies were of poor to moderate methodological quality (Table 4). Torque was not standardised for extensibility measurements and hence it was impossible to distinguish between *real* and *apparent* changes in extensibility. There was no recognition of the short-term visco-elastic deformations that occur within the first 24 hours post-stretch. The final assessments of joint ROM were measured either immediately after the last stretch (Davis *et al.*, 2005), or otherwise the timing was not clearly stated (Gajdosik *et al.*, 2007, Gajdosik *et al.*, 2005, Winters *et al.*, 2004). Although positive treatment effects were reported in all four studies, the wide 95% confidence intervals reflect the remaining uncertainty surrounding the results.

Not many studies have examined the influence of stretch on tendons. A recent randomised controlled trial investigated the effect of stretch training on the visco-elastic properties of human tendon structures in eight healthy men (Kubo *et al.*, 2002). Subjects acted as their own control, with both legs randomly allocated into either the treatment or control group. Stretch was administered to the calf muscles of the treatment leg in a standing position for 5 x 45 seconds twice a day, on a daily basis for 20 consecutive days (three weeks). Outcome measures of extensibility and the visco-elastic properties of tendon structures (stiffness and viscosity) were assessed by ultrasonography.

Following three weeks of stretch training, a significant decrease in passive torque values was observed at all ankle angles of the experimental legs. The authors concluded this to be reflective of increased extensibility of the plantarflexor muscles. The observed change in passive torque was reflected by a mean between-group

difference of -0.2 Nm/deg (95% CI, -0.4 to -0.1; $p < 0.05$) in the flexibility index value (defined by the slope of the portion of the passive torque-angle curve from 15 to 25 degrees). The authors believed that it is unlikely for any muscle activity to have contributed to the passive torque measurements, as electromyographic activities of the medial and lateral gastrocnemius, soleus and tibialis anterior muscles during stretch manoeuvres were found to be very small (1% of maximal voluntary contraction).

On the other hand, stretch training had no significant effect on passive stiffness of tendons (mean between-group change -1 N/mm; 95% CI, -2 to 0), whilst viscosity (as reflected by % change in hysteresis) was significantly decreased (mean between-group change -8%; 95% CI, -13 to -2). These findings suggest that stretch may have an effect on the viscosity of human tendon structures but not the elastic properties of tendons.

In summary, evidence from the able-bodied population seems to suggest that stretch has a small but positive effect on muscle extensibility. Yet the real mechanisms behind the observed stretch effects remain unclear. Unfortunately, most of the studies in the able-bodied population are of poor methodological quality, and fail to differentiate between *real* (structural adaptations in muscle length) and *apparent* muscle extensibility (changes in subjects' tolerance to the stretch sensation). Due to the lack of high-quality randomised controlled trials within the able-bodied population, no conclusive findings have been produced to date.

Table 1. Details of the 28 studies in able-bodied population that satisfied inclusion criteria (Decoster *et al.*, 2005). All data shown are directly taken from Decoster *et al.* (2005).

Study	PEDro Score	ROM Gain (deg)	Outcome	Position	Technique	Duration	Protocol	Warm-up	Comparison Groups	Time Elapsed Before Poststretching Measurement
de Weijer et al	8	14.0	KE	Supine	Static	3 x 30 s	Single session	Active	WU, str, control	0, 0.25, 1, 4, 24 h
Bandy and Irion	6	12.5	KE	Standing	Static	1 x 30 s	5/wk, 6 wk	No	15s, 60s, control	2 d
Bandy et al 1998	6	11.4	KE	Standing	Static	1 x 30 s	5/wk, 6 wk	No	Dynamic, control	2 d
Webright et al	6	10.2	KE	Seated	Active	1 x 30 s	2/d, 6 wk	Active	15s, control	Not stated
Wiemann and Hahn	6	8.4	SLR	Not clear	Ballistic	3 x 15-s reps	Single session	Active	15s static, WU, control	Immediate
Taylor et al	6	5.7	KE	Seated	Static	1 x 60 s	1/wk, 3 wk	Heat	Ice + str, str	Immediate
Nelson et al	5	12.0	KE	Standing	Static	1 x 30 s	3/wk, 6 wk	No	Eccentric strength / control	2 d
Funk et al	5	N/A*	KE	Seated	Static	3 x 30 s	1/wk, 2 wk	No	Heat	Not stated
Hubley et al	5	9.0	SLR	Not stated	Static	15 min	Single session	No	Cycling with and without str	15 min
Cipriani et al	5	28.0	SLR	Standing	Static	6 x 10 s	7/wk, 6 wk	No	2 x 30 s	Not stated
Sullivan et al	5	13.0	KE	Standing with APT	Static / PNF	1 x 30 s	4/wk, 2 wk	Active	Posterior pelvic tilt	Same day
Bandy et al 1997	5	11.5	KE	Standing	Static	1 x 30 s	5/wk, 6wk	No	3 x 30s, 3 x 60s, 1 x 60s	2 d
Chan et al	5	11.2	KE	Seated	Static	5 x 30 s	3/wk, 8wk	Active	4 wks, control	1 d
Worrell et al	5	9.5	KE	Standing with APT	PNF	4 x 20-s cycles	5/wk, 3 wk	No	Static x 15 s	Not stated

Table 1 (continued)

Study	PEDro Score	ROM Gain (deg)	Outcome	Position	Technique	Duration	Protocol	Warm-up	Comparison Groups	Time Elapsed Before Poststretching Measurement
Roberts and Wilson	5	8.5	KE	Not stated	Static active	3 x 15 s	3/wk, 5 wk	Active	9 x 5s, control	Not stated
Reid and McNair	4	10.0	KE	Standing	Static	3 x 30 s	5/wk, 6 wk	No	Control	Not stated
Wallin et al	4	8.5	SLR	Standing	PNF	5 x 19-s cycles	3/wk, 4 wk	No	Ballistic str	Not stated
Rowlands et al	4	33.6	SLR	Supine and seated	PNF	3 x 20-s cycles x 2	2/wk, 6 wk	Active	Various protocols, control	1 d
Willy et al	4	12.3	KE	Standing with APT	Static	2 x 30s	5/wk, 6 wk	Active	Control	Not stated
Spernoga et al	4	7.8	KE	Supine	PNF	5 x 26-s cycles	Single session	Active	Control	0-32 min
De Pino et al	4	7.0	KE	Standing with APT	Static	4 x 30 s	Single session	Active	Control	1 min
Moller et al	3	6.0	SLR	Standing	PNF	5 x 16-s cycles	Single session	Active	No comparison	90 min
Prentice	2	12.0	SLR	Supine	PNF	3 x 20 s cycles	3/wk, 10 wk	Not stated	Static	Not stated
Wiktorsson-Moller et al	2	9.0	SLR	Not stated	PNF	6 x 16-s cycles	Single session	Active	No comparison	Immediate
Halbertsma et al	2	8.9	SLR	Standing	Static	10 x 30s	Single session	No	Control	Immediate
Hartig and Henderson	2	7.0	KE	Standing	Static	5 x 30 s	4/d, 7 wk	No	Control	Not stated
Osternig et al	2	5.3	KE	Supine	PNF	1 x 80 s	Single session	Active	Static, control	Not stated
Halbertsma and Goeken	2	5.2	SLR	Seated	PNF	1 x 10 min	2/d, 4 wk	No	Control	Not stated

Abbreviations: APT, anterior pelvic tilt; KE, knee extension; PNF, propriceptive neuromuscular facilitation; Reps, repetitions; ROM, range of motion; SLR, straight leg raise; str, stretch; WU, warm-up.

* No pre-intervention measurement taken.

Table 2. Details of the 13 studies in disabled population that satisfied inclusion criteria (Harvey *et al.*, 2002). All data shown are directly taken from Harvey *et al.* (2002).

Reference (quality of trial and PEDro score)	Subjects	Number of subjects*	Mean age (SD)	Joint(s) or muscle(s) stretched	Intervention	Time elapsed after last stretch intervention**	Outcome measure	Outcomes by group^
Bandy et al, 1998 (moderate, 5)	Healthy subjects with 'short' hamstrings	19 19 20	26 (6)	Hamstrings	30s static stretch 30s dynamic stretch control x5/week for 6 weeks	2 days	Passive ROM End of range determined by assessor's perception of tightness Angle measured with a goniometer	11 (7) 4 (3) 1 (2)
Bandy et al, 1997 (moderate, 5)	Healthy subjects with 'short' hamstrings	18 19 18 18 20	26 (5)	Hamstrings	3min stretch 90s stretch 60s stretch 30s stretch control x5/week for 6 weeks	2 days	Passive ROM End of range determined by subject's perception of pain/stretch Angle measured with a goniometer	10 (8[a]) 10 (10[a]) 11 (8[a]) 12 (10[a]) 1 (8[a])
Bandy and Irion, 1994 (moderate, 5)	Healthy subjects with 'short' hamstrings	14 14 14 15	27 (5)	Hamstrings	5s stretch 30s stretch 60s stretch control x5/week for 6 weeks	2 days	Passive ROM End of range determined by subject's perception of pain/stretch Angle measured with a goniometer	4 (7[a]) 13 (10[a]) 11 (8[a]) 0 (7[a])
Starring et al, 1988 (moderate, 5)	Healthy subjects with 'short' hamstrings	22 21 22	NA (20-40)	Hamstrings	15min cyclic stretch 15min sustained stretch control x1/day for 5 days	7 days	Passive ROM End of range determined by assessor's perception of tightness Angle measured with a goniometer	10 (6)

Table 2 (continued)

Reference (quality of trial and PEDro score)	Subjects	Number of subjects*	Mean age (SD)	Joint(s) or muscle(s) stretched	Intervention	Time elapsed after last stretch intervention**	Outcome measure	Outcomes by group^
McCarthy et al, 1997 (poor, 4)	Healthy	21 19	23 (2)	Neck	exercise and stretch (NA) control x2/day for 7 days	7 days	Active ROM End of range determined by subject's perception of pain/stretch Angle measured with a cervicorometer	10[a] (15[a]) 7[a] (12[a])
Magnusson et al, 1996c (poor, 4)	Healthy	7 7	26 (6)	Hamstrings	225s of stretch control x2/day for 20 days	1 day	Passive ROM End of range determined by torque Angle measured with Kincom	NA^^
Godges et al, 1993 (poor, 4)	Healthy subjects with limited hip extension	9 7	21 (1)	Hip flexors	6min stretch control x2/week for 3 weeks	24-48 hrs	Passive ROM End of range determined by point at which pelvis lifted Angle measured with a goniometer	11[a] (6[a]) -2[a] (9[a])
Lentell et al, 1992 (poor, 4)	Healthy	18 16	24 (4)	Shoulders	15min stretch control x3 over 5 days	3-7 days	Passive ROM End of range determined by gravity	2 (7) 0 (4)
Bohannon, 1984 (poor, 4)	Healthy	10 10	24[a] (4[a])	Hamstrings	8min stretch control 3 over 5 days x1/day for 3 days	1 day	Passive ROM End of range determined by torque Angle measured with video	4[a] (11[a]) 1[a] (9[a])
Halbertsma and Goeken, 1994 (poor, 3)	Healthy subjects with 'short' hamstrings	9 9	27 (5[a])	Hamstrings	10min stretch control x2/day for 4 weeks	>12 hrs	Passive ROM End of range determined by onset of EMG Angle measured with an electrogoniometer	5 (6) 1 (2)

Table 2 (continued)

Reference (quality of trial and PEDro score)	Subjects	Number of subjects*	Mean age (SD)	Joint(s) or muscle(s) stretched	Intervention	Time elapsed after last stretch intervention**	Outcome measure	Outcomes by group^
Hardy, 1985 (poor, 3)	Healthy	7 7 7 7 7 7 7	NA (18-22)	Hip	90s stretch 90s stretch 90s stretch 90s stretch 90s stretch 90s stretch 90s stretch control x1/day for 6 days	1 day	Active ROM End of range determined by subject's perception of pain/stretch Angle measured with flexometer	5 (4) 13 (7) 14 (8) 11 (4) 7 (11) 8 (7) 0 (3)
Mederios et al, 1977 (poor, 3)	Healthy	10 10	26 (21-34)	Hamstrings	1min stretch control x1/day for 8 days	24 hours	Passive ROM End of range determined by torque Angle measured with a goniometer	6 (4) 1 (2)
Tanigawa, 1972 (poor, 3)	Healthy subjects with 'short' hamstrings	10 10	NA (20-48)	Hamstrings	< 1 min hold-relax procedure 15s static stretch control x2/week for 3 weeks	1 week	Passive ROM End of range determined by subject's perception of pain/stretch Angle reflected from distance between ankle and ground	10[a] (NA) 2[a] (NA) 0[a] (NA)

Abbreviations: ROM = range of motion. NA indicates that insufficient data were available. [a] indicates data were derived from data presented in the paper.

* Number of subjects allocated to each group. Number of subjects are reported for each group in the order that the interventions are presented in the intervention column. All meta-analyses were based on number of subjects that completed the study. ** Time between the last treatment and re-assessment.

^ The size of the treatment effect is reported as the mean (± SD) change in joint ROM for the different interventions (a positive value indicates an increase in ROM). In some cases the mean and SD of the change score were calculated from the means and SDs of pre-test and post-test as recommended by the Cochrane Collaboration (Mulrow and Oxman, 1997). When identical interventions were applied to right and left legs the mean result is reported. Outcomes are reported for each group in the order that the interventions are presented in the intervention column.

^^ This paper measured outcomes in terms of stiffness and torque at a target angle. Data were only presented graphically.

Table 3. Details of randomised controlled trials investigating stretch in able-bodied individuals. These are more recent trials published since the two systematic reviews. Data derived from results presented in the individual papers.

Reference (PEDro score)	*Subjects*	*Mean age (SD)*	*Muscles/Joints Stretched*	*Stretch intervention*	*Outcome measure*	*Time elapsed after last stretch*	*Between-group Difference (95% CI)*
Davis *et al.* (2005)	19 young adults with tight	23	Hamstrings	1) Active self-stretch	Knee Extension Angle	Immediate	Static Stretch vs
(3/10)	hamstrings	(12)		2) Passive static stretch	End ROM determined by		Control
				3) PNF-R	subject's perception of a "strong, but		21°
				4) Control (no stretch)	tolerable stretch", measured with a		(14° to 27°)
				30s stretch each leg	fixed inclinometer		
				3 x / week for 4 weeks			
Gajdosik *et al.* (2005)	19 older women with limited	74	Ankle	1) Static stretch	Ankle Dorsiflexion Angle	Not stated	5°
(6/10)	ankle dorsiflexion	(8)	dorsiflexors	10 x 15s each leg	End ROM determined by subject's		(2° to 8°)
	Stretch (n=10); Control (n=9)			3 x / week for 8 weeks	perception of maximum tolerated		
					passive stretch, measured with an		
				2) Control (no stretch)	ankle-foot apparatus		
Winters et al (2004)	45 healthy subjects	24	Hip flexors	1) Active stretch 30s	Modified Thomas Test	Not stated	1°*
(5/10)	(22 males and 23 females)	(5)		2) Passive stretch 30s	ROM measued with goniometer		(insufficient data)
	with limited hip extension			x 10 daily for 6 weeks			
Gajdosik *et al.* (2007)	12 unconditioned younger	22	Ankle	1) Static stretch	Ankle Dorsiflexion Angle	Not stated	7°
(6/10)	Women	(3)	dorsiflexors	10 x 15s each leg	End ROM determined by subject's		(2° to 12°)
				5 x / week for 6 weeks	perception of maximum tolerated		
					passive stretch, measured with an		
				2) Control (no stretch)	ankle-foot apparatus		

* No non-stretch control. 14° increase in ROM reported for active stretch group. 15° increase in ROM reported for passive stretch group. No measure of variability reported.

† Insufficient data presented. Data derived from estimated values from visual inspection of figures.

Table 4. PEDro scores of randomised controlled trials investigating stretch in able-bodied individuals.

Author	Random allocation	Concealed allocation	Baseline comparability	Blind subjects	Blind therapists	Blind assessors	Adequate follow-up	Intention to-treat analysis	Between-group comparisons	Point estimates & variability	PEDro score	Quality
Davis et al. (2005)	Y	N	N	N	N	Y	N	N	Y	N	3	Poor
Winters et al. (2004)	Y	N	N	N	N	Y	N	Y	Y	Y	5	Moderate
Gajdosik et al. (2005)	Y	Y	Y	N	N	N	Y	N	Y	Y	6	Moderate
Gajdosik et al. (2007)	Y	Y	Y	N	N	N	Y	N	Y	Y	6	Moderate

2. Disabled Populations

The aim of stretch in people with disability is to maintain, increase, and/or prevent the loss of tissue extensibility. In contrast to animal, anecdotal and human able-bodied evidence, trials in the disabled population have found consistent evidence showing that stretch produces no lasting effect on muscle extensibility. Interestingly, the stretch interventions used in studies of disabled individuals were of considerably longer durations (20 minutes to 24 hours a day; 4 to 12 weeks) than those typically administered in the clinical settings of the able-bodied population (30 seconds to 15 minutes a day; 5 days to 6 weeks).

Studies in the disabled population include conditions such as spinal cord injuries (Ben *et al.*, 2005, Harvey *et al.*, 2007, Harvey *et al.*, 2006, Harvey *et al.*, 2000, Harvey *et al.*, 2003), stroke (Ada *et al.*, 2005, Dean *et al.*, 2000, Harvey *et al.*, 2006, Lannin *et al.*, 2007, Turton and Britton, 2005), traumatic brain injuries (Harvey *et al.*, 2006, Lannin *et al.*, 2003, Moseley, 1997), Charcot-Marie-Tooth disease (Refshauge *et al.*, 2006), and post ankle fracture (Moseley *et al.*, 2005). Stretch interventions in these trials were administered to the ankle (Ben *et al.*, 2005, Harvey *et al.*, 2000, Moseley, 1997, Moseley *et al.*, 2005, Refshauge *et al.*, 2006), hamstrings (Harvey *et al.*, 2003), hand (Harvey *et al.*, 2007, Harvey *et al.*, 2006, Lannin *et al.*, 2007, Lannin *et al.*, 2003, Turton and Britton, 2005) and shoulder muscles (Ada *et al.*, 2005, Dean *et al.*, 2000, Turton and Britton, 2005). The details and PEDro scores for each of the above-mentioned studies are presented in Table 5 and Table 6 respectively.

In general, the methodological quality of these studies is higher than those in the able-bodied population. In addition, nine of the 13 studies assessed joint ROM with a standardised torque, thus ensuring the distinction between *real* and *apparent* muscle extensibility (Ben *et al.*, 2005, Harvey *et al.*, 2007, Harvey *et al.*, 2006, Harvey *et al.*, 2000, Harvey *et al.*, 2003, Lannin *et al.*, 2007, Lannin *et al.*, 2003, Moseley *et al.*, 2005, Refshauge *et al.*, 2006). Of these nine studies, eight (Refshauge *et al.*, 2006) were rated as being of high methodological quality with scores of 7/10 or greater on the PEDro scale. Interestingly, all eight of these high quality trials reported no lasting treatment effects in their results (mean treatment effect ranged from 0 to 4 degrees; 95% CI range, -6 to 8 degrees). These studies provide strong evidence that stretching in people with neurological conditions and disabilities does not induce changes in the mechanical properties of the muscle, and hence does not increase *real* muscle extensibility.

Only two studies reported positive findings (Ada *et al.*, 2005, Moseley, 1997), however methodological issues were noted in both. Ada *et al.* (2005) investigated the effects of stretch in preventing shoulder muscle contractures in stroke patients. Stretch was administered to the shoulder in maximum external rotation and 90° of flexion for two 30-minute sessions daily, 5 days a week, for a total of 4 weeks. Results showed that stretch prevented the development of shoulder external rotation contractures with a mean treatment effect of 12 degrees (95% CI, 0 to 24 degrees), but did not prevent shoulder flexion contractures (mean treatment effect -3 degrees; 95% CI, -12 to 7 degrees). However these results may not reflect *real* changes in tissue extensibility. The assessment of maximal passive ROM was determined by gravity acting on the

weight of the limb. This does not solely reflect underlying changes in tissue extensibility as gravity cannot be used in this way to standardise torque, since the gravity-dependent torque and the weight of the limb changes with joint angle.

The second study by Moseley (1997) examined the effect of prolonged stretching (via casting) for seven days on passive ankle dorsiflexion in adults with traumatic head injuries. A mean treatment effect of 15 degrees was reported (95% CI, -2 to 12). Although a standardised torque was used when measuring passive ankle dorsiflexion, the final assessments were conducted immediately after the removal of casts. These results may therefore merely reflect the short-term effects of stretch due to the viscous deformation of tissues. Therefore the positive findings of Moseley (1997) may primarily reflect short-term increases in tissue extensibility, as opposed to *real* structural adaptations of the muscle.

In summary, high-quality randomised controlled trials in people with neurological impairment and disability consistently fail to find long-lasting effects on muscle extensibility from stretch interventions. This presents an interesting contrast to the findings of animal, anecdotal and human able-bodied evidence. The next section of the thesis attempts to explain this disparity.

Table 5. Details of randomised controlled trials investigating stretch in the disabled population. Data derived from results presented in the individual papers.

Reference (PEDro score)	Subjects	Mean age (SD)	Muscles/Joints Stretched	Stretch intervention	Outcome measure	Time elapsed after last stretch	Between-group difference (95% CI)
Ada *et al* (2005)	36 subjects < 20 days post	68	Shoulder	2 x 30min stretch 5 x / week	Maximum passive ROM	Not stated	12°
(7/10)	stroke	(8)	internal rotators	Control	End range determined by		(0° to 24°)
			and extensors	2 x 30min stretch 5 x / week	force of gravity on limb		-3
				Control			(-12° to 7°)
				Duration 4 weeks			
Ben *et al* (2005)	20 patients with SCI	34	Ankle	30 minutes 5 x / week	Torque-controlled	24 hours	4°
(8/10)		(15)	plantarflexors	Duration 12 weeks	passive ROM		(2° to 6°)
Dean *et al*	28 inpatients <10 weeks	58	shoulder	20 minutes 5 x / week	Passive ROM (goniometer)	Not stated	3°
(2000)	post stroke	(12)		(+ usual multidisciplinary	End ROM determined by		(insufficient
(7/10)				rehabilitation)	subject's perception of		data)
				Control (multidisciplinary	'pain'		
				rehabilitation only)			
				Duration 6 weeks			
Harvey *et al*	14 patients with spinal	36	Ankle	30 minutes 5 x / week	Torque-controlled	1 week	0°
(2000)	cord injury (SCI)	(16)	pantarflexors	Control (no stretch)	passive ROM		(-3 to 3)
(8/10)				Duration 4 weeks			
Harvey *et al*	16 patients with SCI	33	Hamstrings	30 minutes 5 x / week	Torque-controlled	24 hours	1°
(2003)		(15)		control (no stretch)	passive ROM		(-2° to 5°)
(7/10)				Duration 4 weeks			
Harvey *et al*	44 community-dwelling	54	Thumb	1) 8 hrs daily night splinting	Torque-controlled passive	24 hours	1°
(2006)	patients with neurological	range	webspace	2) Control (no splint)	ROM of carpometacarpal		(-1° to 2°)
(8/10)	condition (14 stroke, 7 TBI,	43-65		Duration 12 weeks	joint angle		
	23 SCI) with uni/bilateral						
	thumb contractures						
Harvey *et al*	20 subjects with	37	Thumb flexor	1) Daily night splinting on	Torque-controlled	At least 24 hours	0°
(2007)	tetraplegia and bilateral	range	pollicus longus	experimental thumb	passive ROM of thumb	post removal of	(-6° to 6°)
(8/10)	paralysis of all thumb	31-47		2) Control (no splint)	carpometacarpal joint	splint	
	muscles			Duration 3 months	extension		

Table 5 (continued). Details of randomised controlled trials investigating stretch in the disabled population.

Reference (PEDro score)	Subjects	Mean age (SD)	Muscles/Joints Stretched	Stretch intervention	Outcome measure	Time elapsed after last stretch	Between-group difference (95% CI)
Lannin *et al.* (2003) (8/10)	28 subjects with acquired brain injury (with no active wrist extension)	67 (12)	Wrist and finger flexors	Maximum 12-hour stretch via splint (+ usual treatment) Control (usual treatment) Duration 4 weeks	Torque-controlled passive ROM	1 week	1° (-4° to 6°)
Lannin *et al* (2007) (8/10)	63 subjects < 8 weeks post stroke	71 (12)	Wrist and long finger flexors	1) Neutral splint 2) Extended splint 3) Control (no splint) 9-12 hours daily for 4 weeks	Torque-controlled passive ROM	12 to 24 hours post removal of splint	Neutral 1° (-5° to 8°) Extended 1° (-5° to 2°)
Moseley *et al* (1997) (4/10)	10 subjects with traumatic brain injury (TBI)	29 (11)	Ankle plantarflexors	7 days of cast immobilisation 7 days without casting or stretching Over 2 week period	Torque-controlled passive ROM	Immediate	15° (-2° to 32°)
Moseley *et al* (2005) (8/10)	150 subjects with plantarflexion contracture after cast immobilisation	46 (15)	Ankle plantarflexors	30 minutes daily (+ exercises) 6 minutes daily (+ exercises) Control (exercises) Duration 4 weeks	Torque-controlled passive ROM	Immediate	1°* (-1° to 4°)
Refshauge *et al* (2006) (6/10)	14 subjects with Charcot-Marie-Tooth disease (type 1A) and ≤ 15 degrees of ankle dorsiflexion	15 (8)	Ankle plantarflexors	1) Daily night splinting (leg A), control (leg B) for 6 weeks, followed by; 2) Daily night splinting (leg B), control (leg A) for 6 weeks	Passive dorsiflexion ROM using Lidcombe template with standardised force (69 N if ≤ 12 years old, 118 N if > 12 years old)	Within 24 hours after removal of splint	1° (-3° to 4°)
Turton & Britton (2005) (5/10) (insufficient statistical power)	29 patients with stroke	68 (12)	(a) Shoulder external rotators (b) Wrist and finger flexors	(a) 2 x 30-minute daily stretch daily (+ usual care) Control (usual care) (b) 2 x 30-minute daily stretch daily (+ usual care) Control (usual care) Duration 12 weeks	Torque-controlled passive ROM (End ROM determined by pain if standardised torque could not be tolerated)	Not stated	(a) -1.1° (-14° to 16°) (b) -5.6° (-9° to 20°)

NB. All angle data are rounded to the nearest degree. *data presented is ROM measured with the knee extended.

Table 6. PEDro scores of randomised controlled trials investigating stretch in the disabled population.

Author	Random allocation	Concealed allocation	Baseline comparability	Blind subjects	Blind therapists	Blind assessors	Adequate follow-up	Intention to-treat analysis	Between-group comparisons	Point estimates & variability	PEDro score	Quality
Ada *et al.* (2005)	Y	N	Y	N	N	Y	Y	Y	Y	Y	7	***High***
Ben *et al.* (2005)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***High***
Dean *et al.* (2000)	Y	Y	Y	N	N	Y	N	Y	Y	Y	7	***High***
Harvey *et al.* (2000)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***High***
Harvey *et al.* (2003)	Y	Y	N	N	N	Y	Y	Y	Y	Y	7	***High***
Harvey *et al.* (2006)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***Moderate***
Harvey *et al.* (2007)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***High***
Lannin *et al.* (2003)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***High***
Lannin *et al.* (2007)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***High***
Moseley *et al.* (1997)	Y	N	N	N	N	N	Y	N	Y	Y	4	***Poor***
Moseley *et al.* (2005)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8	***High***
Refshauge *et al.* (2006)	Y	N	Y	N	N	Y	Y	N	Y	Y	6	***Moderate***
Turton and Britton (2005)	Y	Y	Y	N	N	Y	N	N	Y	Y	6	***Moderate***

iii) Apparent versus Real Increase in Muscle Extensibility

One possible reason for the conflicting findings between disabled and able-bodied populations is that the able-bodied findings reflect *apparent*, not *real*, changes in muscle extensibility. Some investigators believe that what appears to be lasting changes in extensibility are in fact changes in people's tolerance to the discomfort associated with stretch over time (Ben and Harvey, 2009, Bjorklund *et al.*, 2001, Chan *et al.*, 2001, Folpp *et al.*, 2006, Halbertsma and Goeken, 1994, Magnusson *et al.*, 1996). For example, following a stretch program for the hamstring muscles an individual may touch their toes more easily (that is, more flexible). This may not be due to any underlying structural change in the hamstring muscles but instead may be due to the direct relationship between applied stretch torque and resultant joint angle. That is, the harder an individual leans forward, the further they can reach down towards their toes. The ability to reach further into range is due to altered perceptions and increased tolerance to the discomfort associated with stretch.

Halbertsma and Goeken (1994, 1996) and Magnusson *et al*. (1996) were amongst the first to propose this phenomenon. Magnusson *et al*. (1996) investigated the effect of a three-week stretch regime, with participants performing two 45-second stretches on the hamstring muscles daily. No change was observed in the tissue properties of the hamstring muscles after the intervention period, but interestingly both the final joint ROM and corresponding passive torque were improved. As the final joint ROM was not measured with a standardised torque, it is important to note that this result reflects an improvement in *apparent* muscle extensibility. The authors subsequently proposed that the improved joint ROM is a result of increased stretch tolerance rather than any

change in the mechanical or viscoelastic properties of the muscle.

Similarly, Halbertsma and Goeken (1996) examined the effect of one stretch session, in which subjects performed ten 30-second stretches on the hamstring muscles over a period of ten minutes. No change in passive muscle stiffness was noted, however a significant increase in joint ROM (and hence hamstring muscle extensibility) was shown. As the torque was not standardised, the improved ROM following stretch is once again a likely indication of *apparent* changes in muscle extensibility.

More recently, two randomised controlled trials (Ben and Harvey, 2009, Folpp *et al.*, 2006) have made further attempts to distinguish between real and apparent changes in muscle extensibility by using both standardised and non-standardised torques in their methodology. Both studies examined the effects of stretch on muscle extensibility and stretch tolerance in able-bodied individuals. Folpp *et al.* (2006) recruited 20 healthy able-bodied individuals with limited hamstring muscle extensibility. Stretch was self-administered for 20 minutes a day, five days a week, for four weeks. Outcome measures were hamstring muscle extensibility (angle of hip flexion measured with a standardised torque) and stretch tolerance (angle of hip flexion measured with a non-standardised, maximal tolerated torque). After the four-week intervention, there was no change in the extensibility of the hamstring muscles (mean change in hip flexion -1 degree, 95% CI -4 to 3), whereas stretch tolerance was improved (mean change in hip flexion 8 degrees, 95% CI 5 to 12).

The stretch protocol implemented by Ben and Harvey (2009) was of a slightly longer

duration. Their stretch intervention was administered for 30 minutes a day, five days a week, for six weeks in the hamstring muscles of 60 healthy able-bodied individuals. Outcome measures were identical to Folpp *et al.* (2006), assessing hamstring muscle extensibility and stretch tolerance. Results showed that six weeks of 30-minute stretches produced no effect on hamstring muscle extensibility (as measured with a standardised torque) with a mean treatment effect of -1 degree (95% CI, -3 to 2). On the other hand, the six-week intervention increased stretch tolerance (maximal joint angle measured with a non-standardised stretch torque) with a mean treatment effect of 10 degrees (95% CI, 6 to 14).

The evidence produced from these two randomised controlled trials (Ben and Harvey, 2009, Folpp *et al.*, 2006) indicate clearly that there is an important distinction between *real* and *apparent* changes in muscle extensibility which must be recognised when examining the efficacy of stretch. It is possible that increases in muscle extensibility are merely due to an improved tolerance to the uncomfortable stretch sensation without any structural changes in the muscle properties itself. These findings therefore add further support to the initial suggestions of Halbertsma and Goeken (1994, 1996) and Magnusson *et al*. (1996) that stretch tolerance may play a crucial role in tissue extensibility.

1.2.5 Summary of Stretch

In conclusion, both anecdotal and animal evidence strongly support the use of stretch for producing long-lasting changes in muscle extensibility. Positive stretch effects have also been shown in the human able-bodied literature, although most of the studies

reviewed were of poor quality leading to inconclusive findings. The strong evidence from the human disabled population, clearly indicating that stretch does not change muscle extensibility, has subsequently led to many questions surrounding this topic.

Furthermore, recognising the distinction between *real* (structural adaptations) and *apparent* (stretch tolerance) changes in muscle extensibility has led to important improvements in methodology when assessing the efficacy of stretch. With increasing evidence in the able-bodied population suggesting that muscle extensibility changes are due to improvements in an individual's tolerance to the stretch sensation, it is therefore of interest to further investigate this phenomenon in other population groups where movement tolerance presents as an important issue, such as people with chronic pain.

1.3 Introduction to Pain

1.3.1 Background and Definitions

Pain is a complex multidimensional experience comprising biological, psychological and social factors. Due to the complexity of this experience, both its assessment and management must be considered in the context of a multifactorial framework where each component is equally acknowledged.

The most currently accepted and used definition of pain as defined by the International Association for the Study of Pain (IASP) (1979), states that "pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey and Bogduk, 1994). It acknowledges that the experience of pain is subjective and appreciates the contributions of biological, psychological and environmental factors.

Pain is categorised into two phases according to symptom duration. Pain that is present for less than three months is termed "acute", and pain that persists for greater than three months is considered "chronic" (Merskey and Bogduk, 1994, Scheer *et al.*, 1997). Acute pain is the most common form and is usually associated with injury or trauma. This phase typically subsides within two to four weeks (Merskey and Bogduk, 1994). However in some individuals, pain persists beyond the expected time frame and becomes chronic. In these cases, the impact of pain can become significant on many aspects of life, and it is therefore important that the experience is appreciated from a multidimensional perspective. More detailed explanations of acute and chronic pain are provided in the following paragraphs.

i) Acute Pain

Acute pain is experienced for a short time and usually has a specific cause (such as physical injury to body tissue) and a specific purpose (such as immediate response by the appropriate body systems to reduce further injury) (Adams and Bromley, 1998). Although acute pain is typically related to physical trauma or injury, it is now recognised that many psychological and social factors also play a fundamental role in the experience of pain. This is consistent with Loeser's multidimensional model of pain as shown in Figure 3 (Loeser, 1982), where there is an integration of the different components of pain including nociception, pain perception, suffering, pain behaviour and environment.

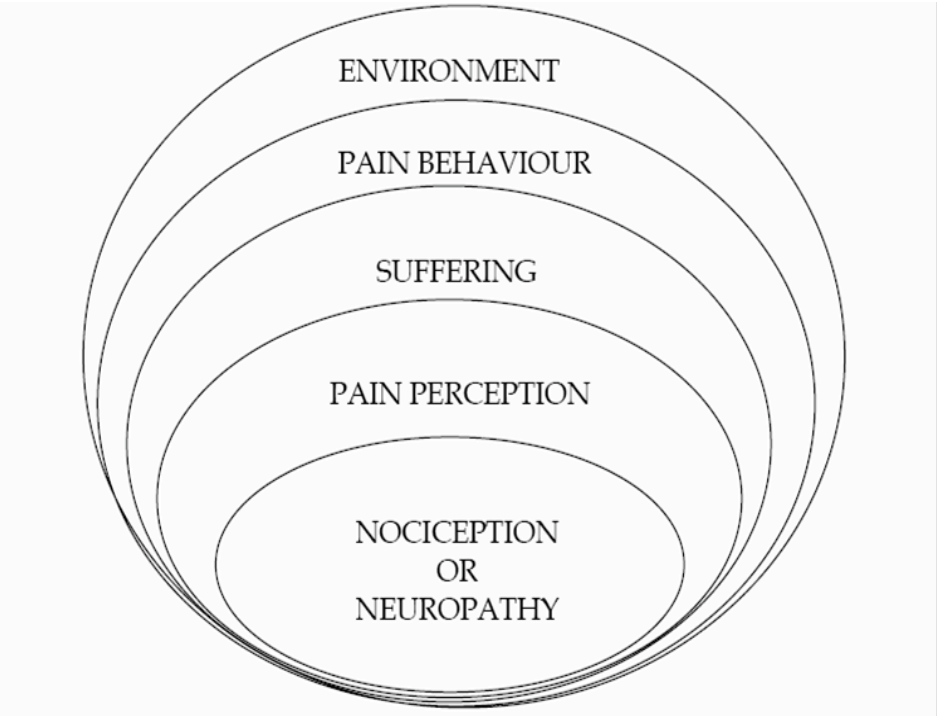

Figure 3. Loeser's multidimensional model of the components of pain. This diagram depicts the multidimensional nature of pain and the various influences that may contribute to the chronic pain experience. This model highlights the fact that tissue damage and subsequent nociception or neuropathy (pain signals) are only a few of the many factors determining how much pain will be experienced. This pain system model is particularly useful in the management of chronic pain.

ii) Chronic Pain

The International Association for the Study of Pain conceptualises chronic pain as "pain that persists beyond the normal time of healing" (International Association for the Study of Pain, 1979). In this phase, pain has no time limit and therefore can persist for months and years. Chronic pain places a significant impact on an individual's quality of life with multiple potential contributions from physical, psychological and environmental factors. For example, the overall pain experience can cause individuals to progressively develop physical deconditioning, functional disability, emotional instability and social difficulty.

Physical deconditioning is often associated with chronic pain (Bousema *et al.*, 2007, Verbunt *et al.*, 2003) and is believed to be a result of gradual movement inhibition and reduction of physical activities (Smeets *et al.*, 2006). Patients often enter a physical deconditioning/disuse cycle in which the pain experience results in a growing fear of movement/re-injury. This subsequently leads to further reductions in movement, physical deconditioning and associated emotional changes. This cycle is demonstrated in Figure 4.

Figure 4. Physical deconditioning cycle. Development of the disuse syndrome and physical/mental deconditioning due to fear of injury and pain. The cycle causes more and more pain to occur as can be seen in the illustration.

In a recent prevalence study conducted amongst 17,543 Australian adults (Blyth *et al.*, 2001), 17% of males and 20% of females reported the presence of chronic pain. Pain prevalence was highest in the 65-69 age group for males and the 80-84 age group for females. Chronic pain was significantly associated with older age, poor self-rated health, psychological distress and poor socioeconomic status such as low level of education, unemployment, and absence of private health insurance. Interference with normal activities of daily living caused by pain was reported in 11% of males and 14% of females and was significantly associated with younger age, female gender, and the absence of private health insurance. There were also strong associations with receiving disability benefits and being unemployed due to health reasons. These results demonstrate that chronic pain has a huge impact on the Australian community individuals of all ages.

The following part of the introduction will explain the different concepts of chronic pain. The assessment and management of the various dimensions of chronic pain are discussed in further details later on in this chapter (sections 1.3.3 and 1.3.4 respectively).

1.3.2 Concepts

Different historical perspectives on pain have led to the development of several conceptualisations. These include the specificity theory, the gate control theory, and the biopsychosocial framework. More modern perspectives (such as the gate control theory) see the pain concept from a viewpoint that considers the involvement of central processing in the pain experience, whilst earlier theories (such as the specificity theory) were more focused on physical tissue damage being the only direct cause of pain. The biopsychosocial model provides a framework for understanding the interaction between biological, psychological and social variables within the pain experience. Overall, these conceptualisations help to provide an insight into what pain is, how it originates and why one experiences pain.

i) Specificity Theory

One of the earliest theories of pain was the specificity theory, developed by a French philosopher named René Descartes in the 17th century (Melzack and Wall, 1996). The specificity theory was based on a biomedical model, proposing that injury or damage to body tissue causes a noxious stimulus which activates specific pain receptors and fibres. Direct pain messages are sent through a spinal pain pathway to a pain centre in the brain, which subsequently causes the individual to experience pain (Melzack,

1999) (Figure 5-A). Descartes viewed the pain system as a specific, hard-wired sensory projection channel that runs from the skin to the brain and operates the bell alarm in the brain. This hard-wired concept is illustrated in Figure 6. Descartes believed that a direct link exists between injury and pain, and the amount of pain experienced by the individual is determined by the severity of the peripheral injury or pathology (Brannon and Feist, 2000). The specificity theory however failed to consider any individual differences in the perception of pain, or possible psychological contributions to the pain experience.

In summary, the specificity theory failed to explain the complete pain experience, as its biomedical perspective did not consider possible psychological and environmental aspects of pain. The role of central processing was not acknowledged during the two hundred years in which this theory stood, apart from being seen as a passive receiver of pain signals.

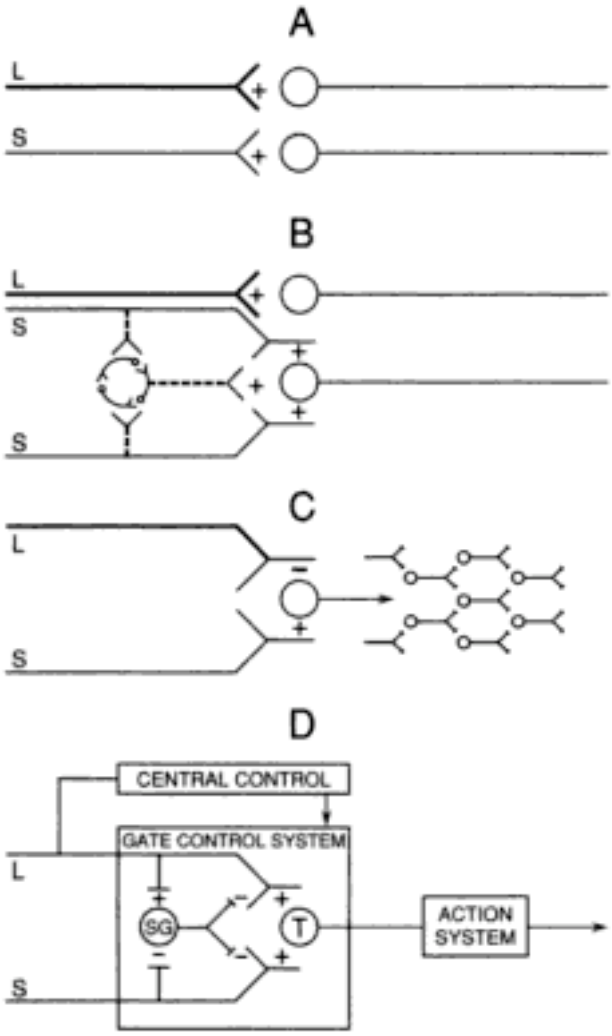

Figure 5. Schematic representation of conceptual models of pain mechanisms. (A) Specificity theory: Large (L) and small (S) fibres are assumed to transmit touch and pain impulses, respectively, in separate, specific, straight-through pathways to touch and pain centres in the brain. (B) Goldscheider's (1894) summation theory, showing convergence of small fibres onto a dorsal horn cell. The central network projecting to the central cell represents Livingston's (1943) conceptual model of reverberatory circuits underlying pathological pain states. Touch is assumed to be carried by large fibres. (C) Sensory interaction theory, in which large (L) fibres inhibit (-) and small (S) fibres excite (+) central transmission neurons. The output projects to spinal cord neurons, which are conceived by Noordenbos (1959) to comprise a multisynaptic afferent system. (D) Gate control theory: The large (L) and small (S) fibres project to the substantia gelatinosa (SG) and first central transmission (T) cells. The central control trigger is represented by a line running from the large fibre system to central control mechanisms, which, in turn, project back to the gate control system. The T-cells project to the entry cells of the action system. +, excitation; -, inhibition. (Melzack and Katz, 2006).

Figure 6. Descartes' specificity theory. This illustration depicts Descartes' theory that the pain system is a straight channel which runs from the skin to the brain and operates the bell alarm in the brain.

ii) Gate Control Theory

In the 20th century, researchers set out to analyse and understand pain more as a complex and multidimensional problem. This led to the development of the gate control theory of pain by Melzack and Wall in 1965 (Melzack and Wall, 1965). This is the first and only theory that has incorporated the central control processes of the brain, as well as taking into consideration the psychological factors of the pain experience.

The gate control theory proposed the idea that pain perception is not only influenced by the direct activation of nociceptors at the peripheral level, but is also affected by modulations occurring in the central nervous system. It is hypothesised that the transmission of nerve impulses from afferent fibres (large-diameter touch fibres and small-diameter pain fibres) to spinal cord transmission cells is modulated by a gating mechanism in the spinal dorsal horn. This is demonstrated in Figure 5-D. The gate is controlled by the amount of activity in the large and small diameter fibres; large diameter fibres tend to inhibit transmission (close the gate) and small diameter fibres

tend to facilitate transmission (open the gate). The theory asserts that activation of nerve fibres that do not transmit pain signals (large fibres) can interfere with signals from pain fibres, subsequently inhibiting an individual's perception of pain. For example, it is common for individuals to lightly rub an injured area in order to experience some pain relief.

In summary, the gate control theory appreciates the roles of the peripheral, spinal and higher central nervous system inputs in the final experience of pain.

iii) Biopsychosocial Framework

The evolution of the gate control theory has led to an increasingly multidimensional approach to pain assessment and management. Known as the biopsychosocial model, it is based on the concept that the overall pain experience is a function of physical, psychological and social/environmental factors operating in concert with each other (Engel, 1977). The model explains that “biological factors may initiate, maintain, and modulate physical perturbations; psychological factors influence the appraisal and perception of internal physiological signs; and social factors shape the behavioural response of patients to the perception of their physical perturbations” (Turk, 1996). The interaction of these components is demonstrated in Figure 7.

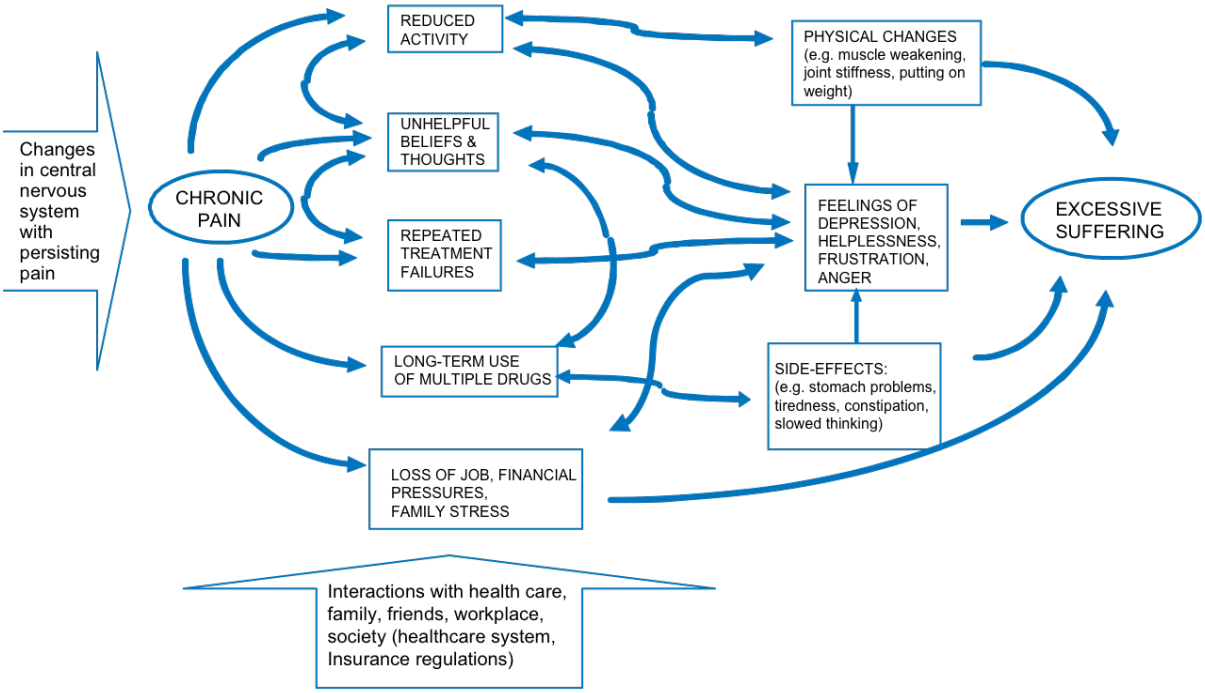

Figure 7. The complexity of chronic pain. The biological, psychological, and social variables which all interact to influence how an individual responds to pain (Nicholas, 2008).

The emergence of the biopsychosocial framework correlated with further understandings of the pain experience from other researchers. For example, Price (2000) recognised the importance of the affective component of pain, stating that pain is an experience which "contains both sensory and affective dimensions and is often accompanied by desires to terminate, reduce, or escape its presence" (Price, 2000). Emotional feelings relating to the present, short-term or long-term future such as distress or fear, can affect the way an individual experiences and responds to pain.

In contrast to older concepts of pain, the biopsychosocial framework recognises that psychosocial factors such as beliefs, attitudes, emotions, and the social and environmental contexts surrounding an individual can also impact the way they respond to and experience pain (Flor and Hermann, 2004, Turk and Flor, 1999, Turk

and Okifuji, 2002). This provides quite a distinct contrast to previous biomedical models (Melzack and Wall, 1996), which focused primarily on the disease process and hence addressed only the pathophysiological component of pain.

1.3.3 Assessment of Pain

As it is now understood that pain is a complex multidimensional experience, the assessment of pain must reflect this through the use of a variety of examination methods and measurement tools to evaluate and quantify the different pain dimensions. Of course, it is important not to diminish the value of a careful history, examination and investigations to explore specific underlying pathology, as well as self-report measures such as questionnaires. Many self-report assessments exist, ranging from simple pain rating scales to more complex, professionally administered questionnaires. Selection of the type of measurement tool to be used should be determined by the purpose of the assessment, depending on the particular dimension(s) of pain experience that is deemed relevant to the individual (Brown *et al.*, 1998).

The following section will provide an overview of various measurement tools that are commonly used in multidisciplinary pain management programs and which play a crucial role in the assessment and management of chronic pain. These measures are all self-report scales that encompass the first three core outcome domains in chronic pain, as recommended by the IMMPACT (Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials) groups (Dworkin *et al.*, 2005, Turk *et al.*, 2003). All dimensions of the pain experience are accounted for, including assessments of pain

severity and intensity, physical functioning (disability), emotional functioning (mood), quality of life, cognitions (beliefs) and multidimensional aspects of pain.

i) Pain Severity

Numerical Rating Scale (NRS)

On a scale out of 10, with 0 being "no pain" and 10 being "pain as bad as it could be", patients are asked to rate their current pain intensity experienced at the time of recording, as well as their highest, lowest, and usual pain intensity over the last week. Numerical rating scales are sensitive to change and are reported to have high validity and reliability (Jensen *et al.*, 1986, Jensen *et al.*, 1989, Lundeberg *et al.*, 2001). They are simple to administer and score (Jensen *et al.*, 1986), rating is instant and does not have the potential for measurement error as in the visual analogue scale (Price and Harkins, 1992).

ii) Physical Functioning (Disability)

Roland and Morris Disability Questionnaire (RMDQ)

This questionnaire is used to measure the impact of pain on functional activities and physical disability in relation to low back pain (Roland and Morris, 1983). The original RMDQ contains 24 yes/no items, in which patients are asked whether each statement applies to them on that particular day (last 24 hours). For example, "I stay at home most of the time because of my back". The final score is determined by the number of "yes" items, and can range from 0 (no disability) to 24 (maximum disability). Other versions of the RMDQ have also been used and reported to have good psychometric properties (Asghari and Nicholas, 2001, Jensen *et al.*, 1992). These include the

modified RMDQ (Patrick *et al.*, 1995) which contains 23 items, and the short RMDQ (Stratford and Binkley, 1997) which includes 18 items.

Oswestry Disability Index (ODI)

This questionnaire comprises of ten sections with statements relating to activities of daily living and how much those activities have been affected by back pain (Fairbank *et al.*, 1980, Fairbank and Pynsent, 2000). Each section consists of six statements with scores ranging from 0 to 5; the first statement is scored 0, the last statement is scored 5. For example, the following statements are used in section 4 (walking): "pain does not prevent me walking any distance" (score 0), and "I am in bed most of the time and have to crawl to the toilet" (score 5). The total possible score of the ODI (ten sections) can therefore range from 0 to 50. The higher the score, the greater the amount of physical disability and functional limitation experienced by the patient. The ODI has been shown to be a sensitive measure for identifying activity intolerances (Baker *et al.*, 1989).

iii) Emotional Functioning (Mood)

Depression Anxiety and Stress Scales (DASS)

The DASS is a set of three self-report scales designed to measure the negative emotional states of depression, anxiety and stress (Lovibond and Lovibond, 1995). Importantly this scale does not comprise of somatic items; this is particularly useful in medical conditions whereby somatic symptoms may potentially inflate the score if assessed on scales that do include somatic items such as the Beck Depression Inventory (BDI) (Beck et al., 1979). The overall questionnaire comprises of 42 items,

which are statements related to how the patient feels about particular aspects of their mood. Patients are asked to use a 4-point severity/frequency scale (0 to 3) to rate the extent to which they have experienced each state over the past week. For example, 0 represents that the statement "did not apply to me at all", whilst 3 represents that the statement "applied to me very much". Scores for each sub-scale: depression, anxiety and stress, are subsequently calculated by summing the scores for the relevant items. The higher the score, the higher the level of severity. The depression scale assesses feelings such as dysphoria, hopelessness and inertia; for example, "I couldn't seem to experience any positive feeling at all". The anxiety scale measures symptoms of fear, autonomic arousal and situational anxiety; for example, "I experienced breathing difficulty". The stress scale assesses symptoms such as tension, agitation and irritation; for example, "I found myself getting upset by quite trivial things". The DASS has been shown to have good reliability (Antoney et al., 1998) and strong psychometric properties (Nicholas et al., 2008).

Beck Depression Inventory (BDI)

The original BDI is a multiple-choice self-report inventory comprising of 21 categories (Beck *et al.*, 1979). It is one of the most widely used instruments for measuring mood, in particular the severity of depression, in chronic pain populations (Morley *et al.*, 2002). Each question has a set of four possible answers, ranging in intensity from 0 (for example, "I do not feel sad") to 3 (for example, "I am so sad or unhappy that I can't stand it"). For every item, the patient will assign a score ranging from 0 to 3 and the total score (0 to 63) is subsequently calculated to determine the severity of depression. A score of 0 – 9 indicates that a person is not depressed, 10 – 18 indicates

mild-moderate depression, 19 – 29 indicates moderate-severe depression and 30 – 63 indicates severe depression. The higher the total score, the more severe the depression symptoms. There are three versions of the BDI. The most current version of the BDI is designed for individuals aged 13 and over. It includes items relating to depression symptoms such as hopelessness and irritability, cognition such as guilt or feelings of punishment, and physical symptoms such as fatigue and weight loss. This questionnaire has been shown to have good reliability and validity (Morley *et al.*, 2002).

iv) Quality of Life

SF-36 Health Survey Questionnaire

The SF-36 Health Survey Questionnaire (Ware and Sherbourne, 1992) is a generic, multi-purpose, health-related quality of life measure consisting of 36 items (eight scales), with scores ranging from 0 to 100 for each of the scales. The eight scales encompass the following areas: physical functioning, role function-physical aspects, bodily pain, general health, vitality, social functioning, role function-emotional aspects, and mental health. The higher the score, the better the patient's condition. The SF-36 has been reported to have good reliability, validity and responsiveness (Kavien *et al.*, 1998, Schlenk *et al.*, 1998).

v) Cognition (Self-Efficacy Beliefs)

Pain Self-Efficacy Questionnaire (PSEQ)

The PSEQ (Nicholas, 1989) is a 10-item questionnaire which assesses self-efficacy beliefs in people with chronic pain. Based on Bandura's concept of self-efficacy

(Bandura, 1977), this questionnaire measures a patient's level of self-confidence and belief in his/her ability to perform a range of activities despite the pain. The activities referred to in each item are purposely described in general terms, for example, "I can enjoy things, despite the pain". This is such that the measure can be easily administered across a range of people with chronic pain individuals. Each item is rated on a 7-point scale (0 to 6), with 0 being "not at all confident" and 6 being "completely confident". The final score can therefore range from 0 to 60, and is determined by adding up the individual scores for each of the ten items. Higher scores represent stronger self-efficacy beliefs. The PSEQ consists of strong psychometric properties, and is reported to have high reliability and validity (Asghari and Nicholas, 2001, Nicholas, 2007).

vi) Cognition (Behaviours)

Tampa Scale of Kinesiophobia (TSK)

The TSK is a 17-item questionnaire used to assess fear of movement/(re) injury in individuals with pain (Kori *et al.*, 1990). Individuals are asked to rate on a 4-point rating scale the extent to which they agree with each of the 17 statements, where 1 represents "strongly disagree" and 4 represents "strongly agree". An example of a statement is: "I'm afraid that I might injure myself if I exercise". The interesting part of the TSK is that four of the items are reverse-scored (items 4, 8, 12 and 16) in which positive self-coping statements are used. For example, "my pain would probably be relieved if I were to exercise". After inversion of the individual scores of the reverse-scored items, a total score is then calculated by summing up all the scores of each 17 statements. Higher scores indicate a greater sense of fear of movement/(re) injury.

The TSK has been reported to have good reliability and validity (Vlaeyen *et al.*, 2002, Vlaeyen *et al.*, 1995).

vii) Multidimensional Pain Assessment

The McGill Pain Questionnaire (MPG)

The MPQ (Melzack, 1975) provides a multidimensional assessment of pain, measuring the sensory, affective and evaluative dimensions of the pain experience. The sensory component comprises of 42 items, with words describing the sensory qualities of the experience in terms of temporal, spatial, pressure, thermal and other properties. The affective aspect consists of 14 items, describing affective qualities of pain in relation to tension, fear and autonomic properties that are part of the pain experience. The evaluative component includes 5 items with words that describe the overall intensity of the total pain experience. The MPQ can be scored in various ways. The Pain Rating Index (PRI) is commonly used, and is scored according to the rank values allocated to different words. For example, the word implying the least pain is given a value of 1, the next word is given a value of 2, and so on. The rank values are then added together to obtain a score for each category and a final score for the overall questionnaire. Another scoring method is the Present Pain Intensity (PPI), to assess the level of pain experienced by the subject at the time of administration of the questionnaire. For example, in response to the question "which word describes your pain right now?", patients are required to choose from the following list of words: mild (1), discomforting (2), distressing (3), horrible (4), and excruciating (5). The PPI is therefore scored according to the word selected. Higher scores represent more severe pain. The

reliability and validity of the MPQ have been supported in the literature (Melzack and Katz, 2001).

1.3.4 Management of Chronic Pain – The Biopsychosocial Model

Within the biopsychosocial framework of pain, it is now understood that although some form of tissue damage or pathology typically initiates the pain process, the physical component is only one of many variables that can influence an individual's processing of and response to pain. Psychological influences such as catastrophising (Sullivan *et al.*, 2005), negative thoughts and attitudes, fear-avoidance beliefs and behaviours (Leeuw *et al.*, 2007), and passive coping strategies (Blyth *et al.*, 2005, Keefe *et al.*, 2004), are some variables that have been shown to negatively impact how an individual views their pain. On the other hand, positive self-efficacy beliefs (Nicholas, 2007), acceptance of the illness (McCracken and Eccleston, 2005), and the implementation of self-management/active coping strategies (Blyth *et al.*, 2005, Keefe *et al.*, 2004) are shown to result in better outcomes and improved adaptation to pain.

In addition to psychological factors, the social/environmental contexts surrounding an individual also play a significant part in affecting their response to the illness. For example, social support from family and friends that encourages self-management and independence (Goldberg *et al.*, 1993) are far more beneficial than excessive sympathy and over-protectiveness, which in turn leads to further dependence and inactivity (Newton-John, 2002). Similarly, job dissatisfaction, unemployment, financial pressures, family conflict and stress, lack of social support, and poor quality of life are examples of unhelpful social/environmental influences that could result in maladaptive pain

behaviours and further negativity (Elliott *et al.*, 1999, Harris *et al.*, 2005, Martikainen *et al.*, 2002).

The biopsychosocial model of pain therefore provides a framework that enables health professionals and patients alike to understand the dynamic interactions between biological, psychological and social variables. The model demonstrates that pain is an extremely complex phenomenon (Figure 7); and in particular, it highlights the need for a multidimensional approach to both the assessment and management of chronic pain, whereby each of the three components of pain (physical, psychological and social) are addressed in accordance to its relative contributions (Nicholas, 2008).

A typical multidisciplinary pain rehabilitation program would incorporate the use of physical techniques, psychological techniques, and social/environmental interventions to provide a more holistic approach to the management of chronic pain. Details of each intervention component are summarised in Table 7. Physical deconditioning and functional limitations are addressed through graded reactivation programs and physiotherapy (strengthening and stretching exercises) to facilitate the return to normal movement, activity and function. Negative psychological states are addressed through the implementation of advice, cognitive-behavioural therapy, relaxation, attentional techniques and education to modify unhelpful cognitions and develop self-coping strategies. Cognitive-behavioural therapy is one of the most significant components of the program, providing the mechanisms and principles by which all other aspects of the program are dependent upon (Nicholas, 2008). Importantly, cognitive-behavioural therapy is not a form of treatment that is only implemented by

the psychologist, but rather all multidisciplinary staff must also use it during their interactions with patients. Social/environmental problems are also addressed in the program by promoting better support from the family, and providing assistance and education as necessary on any workplace or return to work issues.

Table 7. Multidisciplinary Pain Management Program. Core components forming the biopsychosocial approach in the management of chronic pain, as used in the ADAPT program.

Component	Technique	Specific Interventions
PHYSICAL	*Exercise & Activity*	• Graded reactivation / functional restoration programs • Resume normal daily activities • Physiotherapy (strengthening & flexibility)
PSYCHOLOGICAL	*Advice*	• Active listening • Advise on improved understanding of pain • Assurance/differentiation between hurt versus harm
	Cognitive Behavioural Therapy	• Teach identification of unhelpful thought and behaviour patterns, • Encourage alternative, more helpful responses • Differential staff reinforcement of practice of exercises and appropriate self-management strategies • Teach better coping strategies • Encourage self-reliance through problem solving and self-reinforcement for gains
	Relaxation & Attentional Techniques	• Decrease threat value of pain • Teach pain desensitising technique for application whenever pain troubling
	Education/ Information Provision	• Interactive sessions to educate patients on why they experience pain, answer their questions, implement self-management approach etc. • Education on helpful exercises • Advice on coping strategies on home rehabilitation
SOCIAL / ENVIRONMENTAL	*Support Groups*	• Community support groups • Promote family education and support
	Work Modifications	• Address any specific issues/conflicts between employer and patient • Facilitate a graduated return to work • Education

1.3.5 Summary of Pain

In conclusion, the understanding of the pain phenomenon has moved from a unidimensional (pathophysiological) to a multidimensional (biopsychosocial) perspective. Pain is a multifactorial experience that comprises of biological processes at numerous levels (peripheral, spinal and central), psychological factors and social/environmental influences. The biopsychosocial model of pain therefore provides a useful framework for the assessment and management of chronic pain, taking into account the physical, psychological and social/environmental aspects of the overall pain experience.

1.4 Role of Stretch in Chronic Musculoskeletal Pain

Limited extensibility is a common problem in people with chronic musculoskeletal pain. It is undesirable for many reasons, but in particular, because it limits the ability to perform normal functional activities. This leads to physical deconditioning which in turn causes adverse changes in fitness, strength, endurance and psychological well-being (Blyth *et al.*, 2007, Greenleaf, 1997, Musacchia, 1988, Smeets *et al.*, 2006). The importance of addressing physical deconditioning and function in chronic pain is therefore well recognised (Hayden *et al.*, 2005, Schonstein *et al.*, 2003, Van Tulder *et al.*, 2000). A graded functional restoration/reactivation program is often recommended to reverse the effects of deconditioning (Nicholas, 2008).

Multidisciplinary pain management programs typically consist of a physical rehabilitation component, which aims to assist individuals regain movement and strength, and facilitate their return to normal activities (Becker *et al.*, 2000, Jensen *et al.*, 2005, Nicholas *et al.*, 1992, Skouen *et al.*, 2006). Exercises, usually prescribed by physiotherapists within the pain management programs, are implemented with the aims of increasing extensibility, strength, fitness and endurance (Nicholas *et al.*, 2000).

Stretch exercises are commonly prescribed as part of these programs. It is believed that stretch increases muscle extensibility (Harvey *et al.*, 2002) and therefore improves joint range of motion, movement and function (Nicholas *et al.*, 2000, Williams *et al.*, 1996). It is for these reasons that stretch regimes are routinely incorporated into chronic pain management programs. For example, stretch of the hamstring muscles is a commonly prescribed exercise. Stretch is administered to these muscles, which span

across the back of the hip and knee, with the aim of improving the ability to reach down and touch the toes.

The amount of stretch that an individual can endure is dependent on two things: muscle extensibility and tolerance to stretch. Muscle extensibility, as defined earlier in Section 1.2.2 of Chapter One, refers to the "change in passive mechanical properties of a muscle". Tolerance to stretch can be defined as "the greatest level of stretch (discomfort) which an individual is able to tolerate". This tolerance level is the subjective experience of the individual; and in the case of chronic pain, tolerance may be affected by an array of factors that are influential in the processing of pain.

Individuals often describe the stretch sensation as a "discomfort" or an "uncomfortable" sensation. Referring to the physical deconditioning cycle (Figure 4), individuals with chronic pain and fear of movement are more likely to develop poor tolerance to physical activities and inhibitive movement patterns. It is therefore believed that stretch exercises can address the muscles that are shortened and less extensible by improving an individual's tolerance to the movements that may cause discomfort, and thereby reversing the effects of physical deconditioning. However, it is interesting to observe how stretch has become such a regular component of physical rehabilitation programs in chronic pain management, yet its ability to improve muscle extensibility and movement tolerance has never been empirically examined in the chronic pain population.

1.5 Aim of the Current Study

Muscle extensibility and tolerance to stretch and movement are of particular importance for people with chronic musculoskeletal pain. It is common for chronic pain sufferers to present with limited muscle extensibility and poor tolerance to movement and therefore it was considered to be worthwhile and interesting clinical research to explore whether stretch affects one or both of these factors. The response of chronic pain individuals to stretch may differ to the response of others. It is possible that those with chronic pain have a heightened sensitivity to movement due to their hypervigilance and threat perception in relation to physical activity and pain, subsequently predisposing these individuals to limited extensibility (Leeuw *et al.*, 2007). In addition, there may be increased sensitivity due to central pain processing. To date, outcomes following stretch regimes in people with chronic pain (performed as part of the physical component in a multidisciplinary pain management program) have not been clinically investigated in a randomised controlled trial.

The aims therefore of this study were:

1. To determine the effect of a three-week stretch program on hamstring muscle extensibility in people with chronic musculoskeletal pain;
2. To determine the effect of a three-week stretch program on stretch tolerance in people with chronic musculoskeletal pain.

Chapter Two: Methods

The study protocol was registered with the Australian Clinical Trials Registry (ACTRN012607000299404) and approved by the Human Research Ethics Committees of the Northern Sydney Central Coast Area Health Service and the University of Sydney.

2.1 Experimental Design

A randomised controlled trial using a within-subject design was undertaken. One leg of each participant was randomly allocated to an experimental condition and the other to a control condition. To ensure concealed allocation, a blocked random allocation schedule with equal number of right and left legs was generated by computer and placed in a series of consecutively numbered, opaque envelopes by a person external to the study. The envelopes were opened after each participant's initial assessment, indicating his/her inclusion into the trial.

2.2 Participants

Thirty adult participants were recruited from patients enrolled in a multidisciplinary pain management program based at a Sydney hospital. All participants were English-speaking. To be eligible for inclusion, patients had to have pain of musculoskeletal origin persisting for at least three months; be likely to participate in a hamstring muscle stretch regimen as part of the pain management program; and be over 18 years of age. They were excluded prior to randomisation if they were unable to tolerate the testing procedure (that is, unable to tolerate the passive hip flexion movement produced by the measurement device); unable to perform the stretch task

(that is, unable to sit on the ground with full knee extension); had excessive hamstring muscle extensibility (able to place palms on the floor in a standard toe-touch test) (Gauvin *et al.*, 1990); required further medical, surgical or psychiatric investigations and/or interventions; or had a history of drug or alcohol abuse.

A clinically worthwhile treatment effect of five degrees in passive hip flexion angle was determined *a priori* as recommended by others (Ben and Harvey, 2009, Folpp *et al.*, 2006, Harvey *et al.*, 2003). It was estimated that 30 participants would provide a 95% probability of detecting a between-group difference of five degrees. This power calculation was based on a predicted standard deviation of five degrees (Folpp *et al.*, 2006), a drop-out rate of 15% and an alpha of 0.05.

2.3 Intervention

The stretch intervention was supervised by trained physiotherapists and embedded within a multidisciplinary pain management program (ADAPT). This program is a well-established method of chronic pain management utilising a multidisciplinary team approach (Nicholas *et al.*, 2000). Cognitive-behavioural principles form the basis of the program, incorporating the following components: exercise and stretch, pacing, education, drug reduction, relaxation, sleep management, relapse prevention and family involvement (Williams *et al.*, 1996). The program is run over three weeks with at least three hours of physical rehabilitation daily aimed at improving muscle extensibility, fitness, strength and posture. Physiotherapists supervise exercise sessions and use cognitive-behavioural strategies throughout to provide education on behavioural modifications and other issues (Finniss *et al.*, 2006).

2.3.1 Stretch Treatment

The hamstring muscles of the experimental leg were stretched for one minute per day over eighteen consecutive days, whilst the hamstring muscles of the control leg received no specific stretch treatment during this period. This allowed the non-treated leg to act as a control for the treated leg. Stretches were self-administered. Participants sat on the ground, reaching forwards with both hands over the experimental leg whilst maintaining full knee extension (Figure 8).

Figure 8. Stretch treatment. The stretch was administered to the hamstring muscles of the experimental leg only. Participants sat on the ground, reaching forwards with both hands over the experimental leg whilst maintaining full knee extension. *Image copied with permission from www.physiotherapyexercises.com*

Participants were instructed to refrain from stretching the hamstring muscles of the control leg during the course of the study. Fourteen of the 18 stretch sessions were performed within the pain management program during weekdays and supervised by physiotherapists. The other four stretch sessions were unsupervised and performed independently by participants on weekends. Compliance to the intervention was carefully monitored and all stretch sessions were recorded on an exercise sheet. This was reviewed at each subsequent supervised stretch session.

2.4 Outcome Measures

The two primary outcome measures were hamstring muscle extensibility and stretch tolerance. These were assessed on both legs of each participant before and after the three-week stretch intervention. Extensibility was reflected by the angle of passive hip flexion with the application of a *standardised* torque. Stretch tolerance was reflected by the angle of passive hip flexion with the application of a *non-standardised* torque, that is, one which corresponded to the largest stretch torque participants were willing to tolerate.

2.4.1 Measurement Device

A device specifically designed to measure passive hip flexion and hip flexor torque was used (Harvey *et al.*, 2003). The device consisted of a wheel connected to the side of a physiotherapy bed (Figure 9). A leg splint was attached to the wheel and both components rotated simultaneously. The leg splint ensured full knee extension and restricted any hip abduction or rotation. Adjustable counterweights attached to a long rod were used to counteract the torque produced by the weight of the leg and splint.

The long rod was connected to the wheel apparatus and extended proximally from the splint towards the head of the participant. Weights were hung tangentially from the rim of the wheel. The weights generated a hip flexor torque that rotated the leg splint and leg together. The torque was a product of the mass of the weights and the radius of the wheel (28 cm). The device has been reported to have good reliability (ICC = 0.97; 95% CI, 0.96 to 0.98; Harvey *et al.*, 2003).

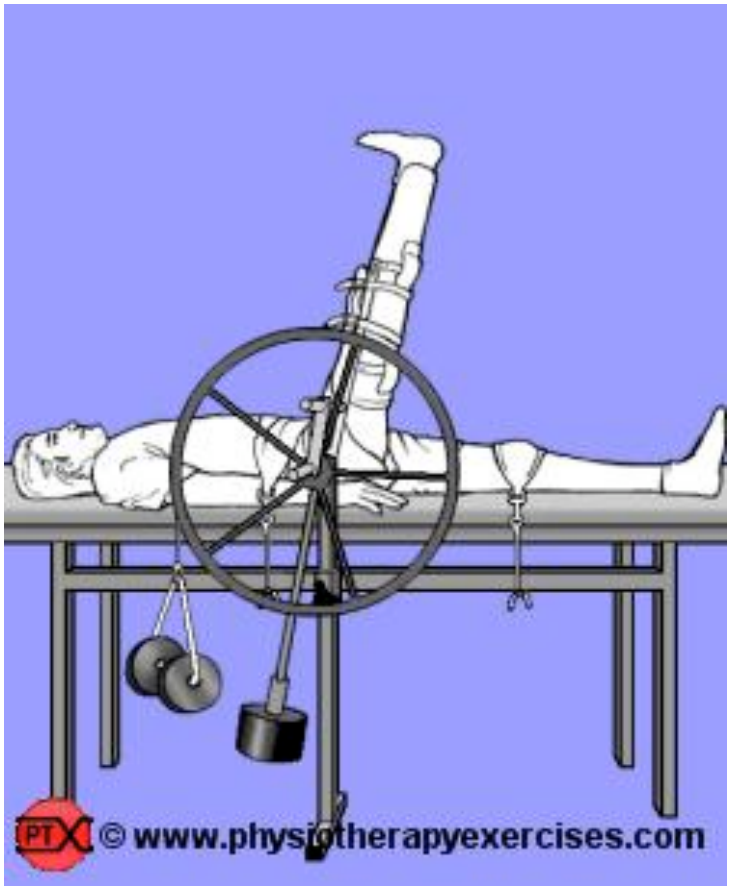

Figure 9. The testing device. The wheel, leg splint and rod rotate simultaneously. *Image copied with permission from www.physiotherapyexercises.com*

2.4.2 Testing Procedure

Participants attended a trial session at least three days prior to the commencement of the study to allow for familiarisation with the testing procedure. Initial measures were taken on day one of the three-week program prior to the first stretch, upon

completion of the eligibility assessment, recruitment and informed consent. Final measures were taken at least 24 hours and no more than 48 hours after the last stretch to ensure that the results did not merely reflect the transient effects of stretch (Duong et al., 2001, Magnusson et al., 1996). The same blinded assessor was used for all testing and participants were instructed not to discuss any aspect of the intervention with assessors. The success of blinding was confirmed by asking the assessor to guess the treatment allocation for each participant.

All testing was performed in the same format, with measurements of the right leg taken prior to the left leg. Participants were positioned supine on a physiotherapy bed with the measuring leg strapped to a splint. The contralateral leg and pelvis were stabilised with straps. Participants were positioned with their hip joint aligned to the centre of the wheel. Passive hip flexion angle was measured with a digital inclinometer aligned on the long axis of the leg splint. All verbal instructions and explanations were standardised between participants.

2.4.3 Measure of Muscle Extensibility

The extensibility of the hamstring muscles was reflected by the angle of passive hip flexion with the application of a *standardised* torque. This standardised torque corresponded to the highest torque tolerated by each participant on both legs at both pre- and post-assessments. In this way, the standardised testing torque was the same for each individual participant, but not across participants. This was appropriate because the effects of the stretch intervention were being compared within, rather than between, participants.

Prior to measurements of each leg, a stretch torque of 18 Nm was applied for two minutes. This two-minute pre-stretch exhausted most viscous deformation, reducing the effect of "creep" on subsequent measures and diminishing any reflex muscle activity around the hip and knee joints (Bandy *et al.*, 1997, Bohannon, 1984, Duong *et al.*, 2001, Magnusson *et al.*, 1995, Magnusson *et al.*, 1996).

2.4.4 Measures of Stretch Tolerance

Stretch tolerance was reflected by the angle of passive hip flexion at the highest torque tolerated by the participant. This torque was *not standardised* but instead varied between legs, testing sessions and participants. Different stretch torques were applied to determine stretch tolerance. A gradually increasing hip flexor torque was applied at increments of 6.1 Nm every 30 seconds. Hip flexion at each increment was measured with a digital inclinometer.

Participants were asked to indicate when the stretch felt "very uncomfortable'. At this point, the torque was slightly reduced. However, the torque was then again further increased but at smaller increments of 1.53 Nm and at a faster rate. This continued until the participant indicated a second time that the stretch was "very uncomfortable". The hip flexion angle was then measured at this point.

Pain intensity was also used to reflect stretch tolerance. At the second indication of a "very uncomfortable" stretch, participants were also asked to rate their pain on an 11-point numerical rating scale (with zero being 'no pain', and 10 being the 'worst pain you can imagine'). Numerical rating scales are sensitive to changes in pain and have

high validity and reliability (Jensen *et al.*, 1986, Jensen *et al.*, 1989, Lundeberg *et al.*, 2001). Participants were blindfolded throughout testing to minimise the influence of visual feedback on their tolerance to stretch.

2.5 Statistical Analysis

Changes in hip flexion angles between initial to final measures *with* and *without* a standardised torque were calculated for both the experimental (stretch) and control (no stretch) legs. Mean between-group differences and their corresponding 95% confidence intervals were then calculated. A positive change in hip flexion angle with a standardised torque reflected an increase in hamstring muscle extensibility following the stretch intervention. Positive changes in hip flexion angle and pain rating score at the highest-tolerated torque reflected an increase in stretch tolerance. In addition, paired-samples T-tests were performed on data collected from four pain questionnaires routinely used in the ADAPT pain management programs. These questionnaires included the Depression Anxiety and Stress Scales (Lovibond, 1998), the modified Roland and Morris Disability Questionnaire (Asghari and Nicholas, 2001), the Multidimensional Pain Inventory (Kerns *et al.*, 1985), and the Pain Self-Efficacy Questionnaire (Nicholas, 2007). A p-value of <0.05 was considered significant. Data were analysed by intention-to-treat (Pocock, 1983).

Chapter Three: Results

3.1 Participant Characteristics

All 30 participants completed the study with no dropouts or withdrawals (Figure 10). The demographic characteristics of the participants are shown in Table 8. The mean (SD) age, pain duration, height, weight and toe-touch distance (fingers to floor) were 43 (12) years, 6 (4) years, 173 (10) cm, 79 (18) kg and 23 (16) cm, respectively. 63% of participants exercised less than five hours per week and 67% reported walking as their main form of exercise. 43% of participants did not perform any stretch exercises. The site of pain was diverse, affecting the neck (23%), back (77%), arms (40%), and legs (53%). Over half (60%) reported pain in two or more major sites, and 43% experienced pain in both the back and legs. Mean (SD) initial values on the Depression Anxiety and Stress Scales (Lovibond, 1998), the modified Roland and Morris Disability Questionnaire (Asghari and Nicholas, 2001), the Multidimensional Pain Inventory (Kerns *et al.*, 1985), and the Pain Self-Efficacy Questionnaire (Nicholas, 2007) were 16 (12), 12 (5), 4 (1), and 24 (12), respectively. These scores are consistent with some recently developed normative data in the chronic pain population (Nicholas *et al.*, 2008), indicating that our sample group was representative of this population.

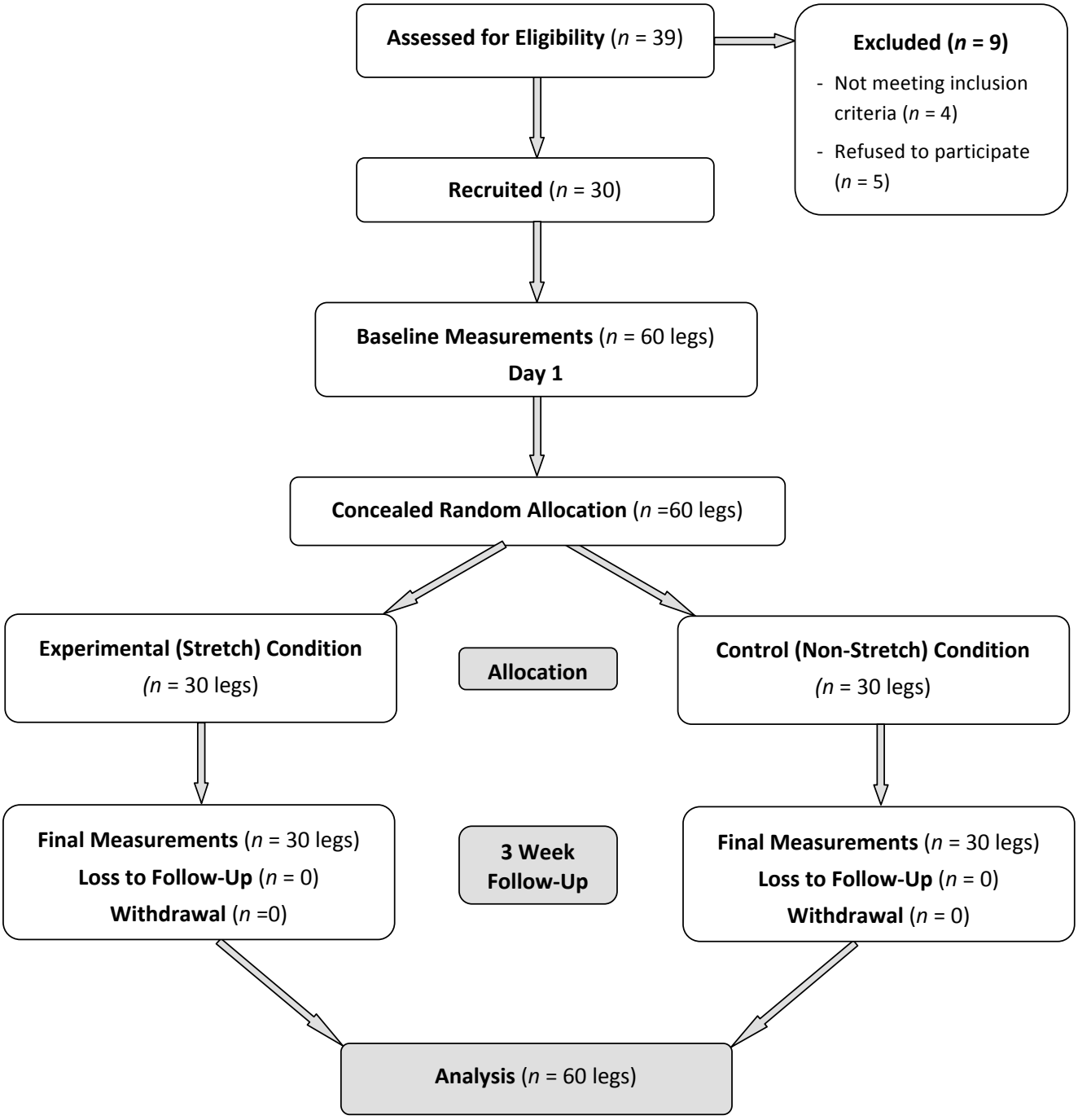

Figure 10. Flow of participants through the trial. Primary outcomes measured on the first and final day of the 3-week program.

Table 8. Demographic characteristics of participants.

Demographic Characteristics	**(n = 30)**
Gender	*n* (%)
Male	15 (50)
Female	15 (50)
Mean age (SD), *years*	43 (12)
Mean duration of pain (SD), *years*	6 (4)
Mean height (SD), *cm*	173 (10)
Mean weight (SD), *kg*	79 (18)
Mean toe-touch distance (SD), *cm*	23 (16)
Physical activity	*n* (%)
0 to 5 hours per week	19 (63)
6 to 10 hours per week	6 (20)
11 + hours per week	5 (17)
Stretch exercises	*n* (%)
None	13 (43)
1 to 2 hours per week	13 (43)
3 + hours per week	4 (13)
Pain site	*n* (%)
Neck	7 (23)
Back	23 (77)
Arms	12 (40)
Legs	16 (53)
Neck and arms	6 (20)
Back and legs	13 (43)
Multiple (2 or more major pain sites)	18 (60)
Pain questionnaires	*Mean (SD)*
Depression (DASS)[a] (0-42)	16 (12)
Physical disability (RMDQ)[b] (0-24)	12 (5)
Pain intensity (MPI)[c] (0-6)	4 (1)
Pain self-efficacy (PSEQ)[d] (0-60)	24 (12)

[a] DASS, Depression Anxiety and Stress Scales
[b] RMDQ, Roland and Morris Disability Questionnaire
[c] MPI, Multidimensional Pain Inventory
[d] PSEQ, Pain Self-Efficacy Questionnaire

3.2 Adherence to Protocol

The protocol required participants to perform 18 stretch sessions over 18 consecutive days with 14 supervised and four unsupervised. There were some minor deviations from the study protocol with a total of 12 stretches missed out of a total of 540 (2%) for all participants.

3.3 Effects of Intervention

At the commencement of the trial, there was no difference in the extensibility of the hamstring muscles (as reflected by hip flexion angles with a standardised torque) between the experimental and control legs. The initial mean (SD) hip flexion angles with the application of an 18 Nm torque for the experimental and control legs were 47 (12) and 47 (13) degrees, respectively. Similarly, there was no difference in participant's tolerance to the discomfort associated with stretch between their experimental and control legs. The initial mean (SD) hip flexion angles at the highest tolerated torque were 69 (18) and 69 (19), respectively (as shown in Table 9).

Primary Analyses

3.3.1 Muscle Extensibility

Muscle extensibility was reflected by the angle of hip flexion with a standardised torque for each individual. The mean (SD) torque applied was 26 (8) Nm. The between-group (95% CI) mean difference in hip flexion was 1 degree (95% CI, -2 to 4; p = 0.386; Table 9). This result indicates that three weeks of stretch did not increase hamstring muscle extensibility. The pre-post changes in mean hip flexion

angles for the experimental and control groups, as measured with a standardised torque, are represented in Figure 11.

3.3.2 Stretch Tolerance

Stretch tolerance was reflected by the angle of hip flexion at the highest tolerated torque. The between-group (95% CI) mean difference in hip flexion with a non-standardised torque was 8 degrees (95% CI, 5 to 10; $p < 0.001$; Table 9). The corresponding between-group (95% CI) mean difference in torque tolerated by participants was 8 Nm (95% CI, 4 to 11; $p < 0.001$; Table 9). However, there was no difference in the amount of pain reported by participants at the highest tolerated torque between the beginning and end of the study, or between the experimental and control legs. The between-group (95% CI) mean difference in pain intensity score was 0 points (95% CI, -1 to 0; $p = 0.316$; Table 9). These results indicate that the stretch intervention was associated with an improvement in the participants' tolerance to a greater stretch for the same level of pain in the experimental leg. The pre-post changes in mean hip flexion angles for the experimental and control groups, as measured with a non-standardised torque, are represented in Figure 12.

Table 9. Mean (SD) hamstring muscle extensibility and stretch tolerance before and after three weeks of stretch.

	Control			Experimental			Between-group difference
	Pre	**Post**	**Change**	**Pre**	**Post**	**Change**	**Mean (95% CI)**
Hamstring Extensibility (standardised torque)							
Hip flexion (degrees)	58 (17)	60 (16)	2 (7)	58 (19)	61 (16)	3 (8)	**1 (-2 to 4)**
Stretch Tolerance (highest tolerated torque)							
Hip flexion (degrees)	69 (19)	72 (16)	2[a] (10)	69 (18)	79 (15)	10 (11)	**8* (5 to 10)**
Highest tolerated torque (Nm)	36 (14)	37 (13)	1 (6)	36 (12)	44 (16)	8 (9)	**8*[a] (4 to 11)**
Pain Intensity (0 to 10)	7 (2)	7 (1)	1[a] (2)	7 (2)	7 (2)	0 (2)	**0[a] (-1 to 0)**

Note: This table shows the mean (SD) change in hip flexion (degrees) with a standardised and non-standardised (highest tolerated) torque following three weeks of stretch. Mean (SD) changes in highest tolerated torque (Nm) and pain intensity (numerical rating scale out of 10), and mean (95% CI) overall effects are also shown.

* $p < 0.05$

[a] apparent error due to effects of rounding

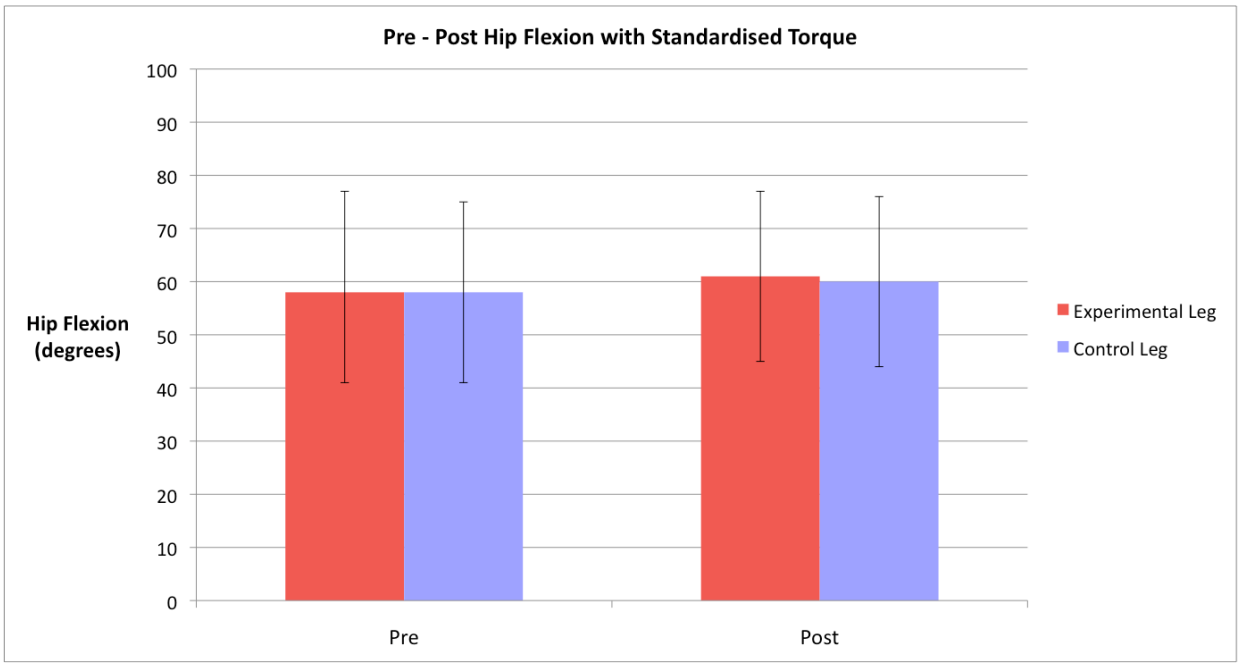

Figure 11. Pre-post hip flexion with standardised torque. Data are means ± standard deviations. The horizontal axis represents time of assessment pre- and post-intervention. The vertical axis represents hip flexion angles in degrees. The mean pre-post hip flexion angles for the experimental group are represented by the red columns; the mean pre-post hip flexion angles for the control group are represented by the blue columns.

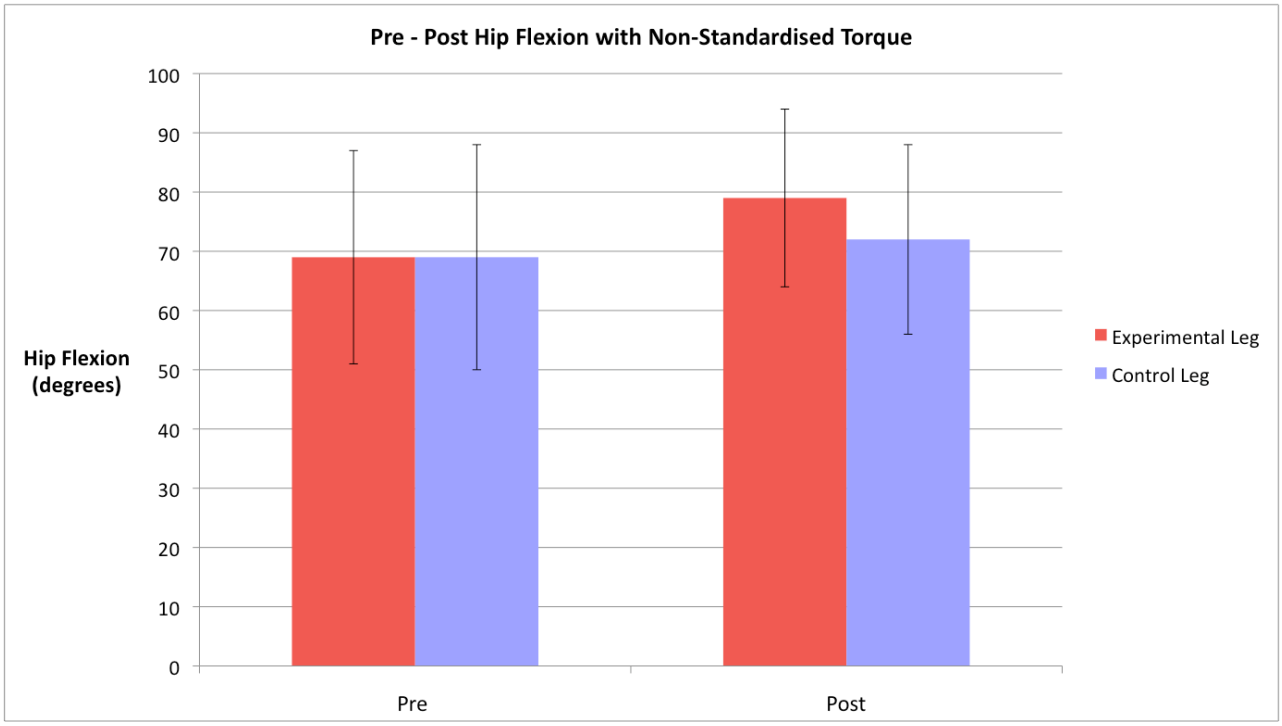

Figure 12. Pre-post hip flexion with non-standardised torque. Data are means ± standard deviations. The horizontal axis represents time of assessment pre- and post-intervention. The vertical axis represents hip flexion angles in degrees. The mean pre-post hip flexion angles for the experimental group are represented by the red columns; the mean pre-post hip flexion angles for the control group are represented by the blue columns.

Chapter Four: Discussion

4.1 Effects of Intervention

This trial investigated the effects of stretch on muscle extensibility and stretch tolerance in patients with chronic pain. The results indicate an *apparent* but not *real* increase in extensibility following a three-week hamstring muscle stretch program. The *apparent* increase was due to participants' improved tolerance to the discomfort associated with stretch, rather than any underlying structural change in muscle extensibility. Hence at the end of the stretch program, participants were willing to tolerate larger testing torques on the experimental (stretch) leg compared to the control (no stretch) leg. This corresponded with an increase in hip flexion when using a non-standardised testing torque. However, there was no change in hip flexion when a standardised testing torque was used. These results suggest that long-lasting structural changes did not occur in the hamstring muscles despite the observed increase in hip flexion due to improved tolerance.

Important design features were included in this study to minimise bias. These included the use of randomisation, concealed allocation and consistent verbal instructions. Bias is a real problem in trials of this kind due to the potential expectations of participants, researchers and assessors in regards to the effects of intervention. As participants could not be blinded, it was possible that placebo or Hawthorne effects may have caused bias. Perhaps the increased tolerance to stretch reflected the use of unblinded participants with strong expectations about the therapeutic benefits of stretch. That is, participants anticipated that the stretch

intervention would improve muscle extensibility and, therefore, inadvertently tolerated larger torques during the final testing of their experimental legs, leading to an apparent increase in ROM. We attempted to minimise participants' expectations by blindfolding participants during testing and thereby blocking visual cues to leg position. Nevertheless, it was difficult to completely blind participants in studies of this kind.

The timing of the final assessment was another important consideration of the methodology. In this study, final outcome measurements were taken between 24 to 48 hours following the last stretch treatment. This was crucial in order to distinguish between short-term and long-term effects of stretch. Immediate short-term effects are due to viscoelastic deformation of tissues, whilst long-term effects are resultant of underlying physiological adaptation of tissues. Therefore, only measurements that are taken at least 24 hours after the last stretch can be used to indicate long-lasting effects of the intervention (Harvey *et al.*, 2002).

The participants of this study were all adults aged between 19 and 68 years with chronic musculoskeletal pain originating from a variety of different sources. The most common location of pain was the back (77% of the sample group), followed by the legs (53% of the sample group). Nearly two-thirds (60%) of the sample group complained of persisting pain in multiple (two or more) major sites. This study however examined the effects of stretch to the hamstring muscles, regardless of the participant's site of pain. It is possible that those with leg pain responded differently to hamstring muscle stretches than those with arm or neck pain. There were

unfortunately insufficient participants to explore this possibility in sub-group analyses, although the narrow 95% CI associated with the between-group difference suggests that all participants responded to the stretch intervention in a consistent way.

Numerous stretch techniques have been developed, applied and used by physiotherapists, coaches and trainers (Halbertsma *et al.*, 1999). These include isometric, ballistic, dynamic range of motion, proprioceptive neuromuscular facilitation as well as passive and static stretches (Bandy *et al.*, 1998). A static stretch is a slow, sustained muscle lengthening that can easily be self-administered and commonly used amongst clinical and community settings. Static stretches are usually held for durations lasting between 15 to 60 seconds. We chose a one-minute static stretch for our intervention protocol, as this duration and type of stretch is typical and representative of current clinical practice within pain management programs. The time course of three weeks was also selected on the basis of current clinical practice in hospital pain management programs.

In Chapter One of this thesis, possible mechanisms underlying long-lasting changes in muscle extensibility following stretch were reviewed. In animal studies, this has clearly been attributed to the highly adaptable properties of soft tissues, which undergo structural remodelling in response to a stretch stimulus. Evidence from human studies are however less convincing. They invariably rely on measures of joint range of motion without the standardisation of torque. In this situation,

improvements in joint range of motion can be attributed to one of two factors, namely: 1) changes in muscle properties resulting in increased resting muscle length (indicating *real* extensibility), or 2) changes in stretch tolerance resulting in improved range of motion without any underlying structural adaptations (indicating *apparent* extensibility).

The following paragraphs will explore possible explanations for our two main findings: 1) why *real* muscle extensibility did not change with stretch, and 2) why stretch tolerance improved with stretch. In addition, the significance and implication of these findings in relation to patients with chronic musculoskeletal pain will be discussed.

4.2 Muscle Extensibility

The present study has demonstrated that three weeks of stretch performed one minute a day does not increase hamstring muscle extensibility (mean difference in hip flexion 1 degree; 95% CI, -2 to 4; $p = 0.386$). Final measurements were taken at 24 to 48 hours following the last stretch treatment, using a within-subject standardised torque.

A standardised torque was used to ensure that measurements solely reflected long-lasting structural changes in muscle extensibility (if any), rather than other psychologically or neurophysiologically mediated variables (such as participant's own perception of discomfort and/or willingness to tolerate stretch). A treatment effect of five degrees change in hip flexion was determined *a priori* to be clinically

worthwhile, based on recommendations from previous researchers who also investigated the efficacy of stretch interventions (Ben and Harvey, 2009, Folpp *et al.*, 2006, Harvey *et al.*, 2003, Lannin *et al.*, 2007, Moseley *et al.*, 2005).

The outcomes of this study contradict anecdotal evidence and the results of some clinical trials indicating the effectiveness of stretch for increasing extensibility in able-bodied individuals (Bandy *et al.*, 1997, 1998, Bonnar *et al.*, 2004, Davis *et al.*, 2005, Gajdosik *et al.*, 2005, Reid and McNair, 2004, Youdas *et al.*, 2003). Our findings however, were consistent with those reported in two similar studies of our own in healthy able-bodied individuals (Ben and Harvey, 2009, Folpp *et al.*, 2006). They were also comparable with multiple clinical trials involving people with neurological disabilities (Ben *et al.*, 2005, Harvey *et al.*, 2007, Harvey *et al.*, 2006, Harvey *et al.*, 2000, Harvey *et al.*, 2003, Lannin *et al.*, 2007, Lannin *et al.*, 2003, Moseley, 1997, Refshauge *et al.*, 2006, Turton and Britton, 2005). Studies involving people with neurological disabilities examined stretch interventions that were administered for much longer than a few minutes a day, yet still failed to demonstrate any *real* changes in muscle extensibility despite good statistical power. It is of course possible that the muscles of people with neurological disabilities respond differently to stretch than the muscles of healthy able-bodied individuals. Nevertheless these trials increasingly support the view that stretch as typically administered in the clinical setting, does not induce long-lasting structural adaptations in the muscles of human and therefore does not increase *real* muscle extensibility.

A possible explanation for the failure to increase *real* muscle extensibility is that one minute of stretch daily may be an inadequate stimulus for structural adaptations to occur in the passive mechanical properties of the muscle and surrounding soft tissue. Perhaps if each stretch was administered for a longer duration of time (that is, greater than one minute a day), the results may have been different. Similarly, it is possible that three weeks of stretch was insufficient to induce any mechanical change in the muscle. Perhaps if the stretch intervention was performed over a longer period of time (that is, greater than three weeks), different outcomes may have been attained. Nevertheless, the dosage of stretch used in this trial is similar to those typically applied in the clinical practice of pain management (Nicholas *et al.*, 2000). It is unlikely that patients with chronic pain would tolerate more intensive stretch regimes, where stretch is administered for longer than a few minutes per muscle per day. In addition, recent evidence in healthy able-bodied individuals have indicated that there is no added benefit from stretch administered for up to 20-30 minutes a day (Ben and Harvey, 2009, Folpp *et al.*, 2006).

The failure to detect a treatment effect on *real* muscle extensibility was not due to insufficient sample size. A clinically worthwhile treatment effect of five degrees change in hip flexion was determined prior to the commencement of the trial. A power calculation was performed to estimate the required sample size based on a predicted standard deviation of five degrees, a drop-out rate of 15% and an alpha of 0.05. It was therefore estimated that a sample size of 30 would provide a 95% probability of detecting a between-group difference of five degrees.

4.3 Stretch Tolerance

In contrast, our results have shown that three weeks of stretch performed one minute a day leads to an improvement in stretch tolerance as reflected by an *apparent* increase in muscle extensibility (mean difference in hip flexion was 8 degrees; 95% CI, 5 to 10; $p < 0.001$). That is, participants were able to tolerate greater stretch torques resulting in a larger ROM. Final measurements were taken at 24 to 48 hours following the last stretch treatment, using a non-standardised (highest tolerated) torque. This torque differed between legs and participants, and reflected the highest amount of stretch that participants tolerated on the hamstring muscles of each leg.

Our findings suggest that stretch tolerance may be an important mechanistic factor behind apparent changes in extensibility. Previous researchers have suggested that what appear to be lasting changes in muscle extensibility are in fact changes in people's tolerance to the stretch discomfort over time (Ben and Harvey, 2009, Bjorklund *et al.*, 2001, Chan *et al.*, 2001, Folpp *et al.*, 2006, Halbertsma and Goeken, 1994, Magnusson *et al.*, 1996). The ability to stretch further into range could be a result of altered perceptions of stretch and an increased tolerance to the discomfort associated with stretch. Hence the effect on extensibility is not related to any structural adaptations in the muscle itself, but instead may be a consequence of the direct relationship between applied stretch torque and resultant joint movement.

The exact mechanisms behind participants' increased stretch tolerance remain unclear. It is possible that physiological processes are responsible for the reduced

sensation of discomfort and improved tolerance following stretch; however the precise underlying explanation is uncertain. Nociceptive nerve endings in the joint and muscle have been suggested as possible structures contributing to the altered stretch tolerance (Magnusson et al., 1996). Other possible contributors are mechanoreceptors and proprioceptors, which have demonstrated reduced activity following stretch exercises (Proske *et al.*, 1993).

It is possible that the observed changes in stretch tolerance may be due to an underlying neurophysiological mechanism. For example, stretch interventions may influence sensory neural pathways (Laessoe and Voigt, 2003, Magnusson *et al.*, 1996) and alter the processing of the stretch stimulus. Afferent input from muscles and joints during a stretch manoeuvre may therefore interfere with signals from nociceptive fibres (discomfort sensations from the stretch), subsequently inhibiting an individual's perception of pain. This is consistent with the hypothesis behind the gate control theory (Melzack and Wall, 1965). Halbertsma and Goeken (1994) and Magnusson et al. (1996) reported initial evidence of altered stretch tolerance and sensory adaptations taking place following stretch. Both studies investigated the hamstring muscles with relatively intensive regimes comprising of ten minutes stretch, twice a day for four weeks (Halbertsma and Goeken, 1994), and 225 seconds of stretch, twice a day for 20 days (Magnusson *et al.*, 1996). Similarly, Bjorklund *et al.* (2001) provided evidence of sensory adaptations occurring after a two-week stretch protocol of the rectus femoris muscle. It was interesting that although Bjorklund *et al.* (2000) used a relatively mild stretch regime (80 seconds stretch, four times a week for two weeks) compared to the two previous studies, the stretch was still of a

sufficient stimulus to alter stretch tolerance. More recent evidence from Folpp *et al.* (2006) and Ben and Harvey (2009) add further support for the effectiveness of stretch in improving muscle extensibility via increased stretch tolerance, using fairly intensive stretch protocols (20 minutes a day, five days a week for four weeks; and 30 minutes a day, five days a week for six weeks respectively). The results of this study have demonstrated that even a much shorter duration and intensity of stretch (one minute per day over three weeks) can effectively increase stretch tolerance and improve *apparent* muscle extensibility. This has important implications for clinical practice where the prime aim of stretch is to achieve a greater joint range of motion, regardless of the underlying mechanism. For example, stretch is applied to increase hip flexion even if it is attained solely by an increased tolerance to the stretch discomfort. If this is the case, then even stretches of short duration (such as one minute) can be effectively and appropriately used for this purpose.

Alternatively, changes in stretch tolerance may be psychologically mediated. It is commonly believed by the general public that stretch provides a therapeutic effect. Therefore it is possible that participants anticipated the positive effects of stretch, and hence their perception of the discomfort associated with stretch was dampened. According to the gate control theory (Melzack and Wall, 1965), sensations of pain and discomfort are affected by descending modulatory influences from higher centres. Prior pain experiences, motivation, emotional states, and self-efficacy beliefs are some psychological factors that can influence the outcome of an experience. For example, prior experiences of the stretch discomfort, motivation to perform the stretch exercises daily (with supervision from physiotherapists), and

elevated mood and confidence through expectations of exercise benefits, are all potential psychological contributors explaining for participants' altered perception of the stretch discomfort and ability to tolerate a greater stretch over time.

It is interesting to note that although participants tolerated a larger testing torque on their experimental (stretch) leg following three weeks of stretch, there were no changes in their pre- and post-pain intensity scores at the highest-tolerated torque (as rated on an 11-point numerical rating scale). Similarly, there were no changes in pre- and post-pain scores on the Multidimensional Pain Inventory (Kerns *et al.*, 1985) routinely collected in the ADAPT pain management program. The mean (SD) change in the Multidimensional Pain Inventory was 0 (1; $p = 0.158$). From these results, it appears that there was in fact no change in the intensity of perceived pain, even though the amount of stretch torque applied was increased. Perhaps repeated exposure to stretch produces a desensitising effect, allowing the individual to become accustomed to the stretch sensation. Consequently, participants progressively tolerated larger stretch torques for the same level of experienced pain. This is consistent with the concepts of pain acceptance (McCracken *et al.*, 2007) and self-efficacy (Nicholas, 2007), both commonly referred to when discussing coping strategies in the management of chronic pain. Interestingly, there was a significant improvement in mean (SD) Pain Self-Efficacy Questionnaire (Nicholas, 2007) ratings during the program from 24 (12) to 40 (10) ($p < 0.001$). As participants' acceptance of the stretch discomfort progressively improved over the three weeks, they began to feel more confident in their ability to perform the stretch. This was reflected in a greater tolerance of the initially uncomfortable movement.

Participants' increased tolerance to stretch was found to be specific to the experimental (stretch) leg and did not crossover to the control (no stretch) leg. That is, administering stretch on one leg did not produce any corresponding effect on the opposite control leg. This was an interesting finding which merits further investigation. Furthermore, the effects of stretch on the experimental leg occurred on top of general improvements in the participants' overall depression, anxiety and physical disability. For example, the mean (SD) changes between the initial and final scores on the Depression and Anxiety Stress Scales (Lovibond, 1998) and the modified Roland and Morris Disability Questionnaire (Asghari and Nicholas, 2001) (collected before and after the ADAPT program) were 5 (6) (p = 0.002) and 3 (4) (p = 0.002), respectively. The overall improvements in mood and physical disability were presumably influenced by other components of the ADAPT program. These findings together suggest that the stretch intervention itself did not provide a generalised benefit. Rather, the intervention effects were specific to the leg that was stretched. This is consistent with reports on fear-avoidance interventions in chronic pain patients, showing that exposure to one particular movement produces specific improvements in that movement only, and does not generalise towards a different movement (Vlaeyen *et al.*, 2002).

4.4 Clinical Implications

This study provides support for the ongoing incorporation of stretch into pain management programs, provided the aim of stretch is to improve joint range of motion via increased tolerance to the discomfort associated with stretch, rather than via structural changes in the mechanical properties of the tissue. Stretch may

therefore be conceptualised as a graded exposure to movement, increasing tolerance to stretch and resultant movement. This is particularly relevant in the rehabilitation of chronic pain patients, who may suffer from physical deconditioning due to their constant fear of movement/re-injury, causing further reductions in physical activity, movement and function.

Our findings also provide implications for other movement-sensitive conditions where methods of desensitisation, such as through the increased exposure to stretch-induced discomfort, may be useful. If stretch exercises can help to diminish chronic pain individuals' perception of discomfort towards particular movements and improve their tolerance to certain activities, then stretch regimes may be an important and effective way to target specific movement limitations where sensations of discomfort may be involved. Through the regular application of stretch, individuals with chronic pain can therefore become desensitised and accustomed to movement-related sensations of discomfort, thus facilitating their acceptance of pain and increasing their tolerance to perform normal functional tasks.

Stretch should therefore become an essential component of physical rehabilitation in all multidisciplinary pain management programs, to assist in the restoration of movement and return to normal activity. Regardless of the underlying physiological and/or psychological mechanism behind changes in stretch tolerance, our study has provided evidence for the importance of stretch exercises in the rehabilitation and management of patients with chronic musculoskeletal pain.

4.5 Directions for Future Research

Further research is required to investigate the mechanisms underlying changes in tolerance and perception of discomfort associated with stretch. This study raised questions about the possible contributions of neurophysiological and psychological factors accounting for the observed increase in stretch tolerance, and hence further investigations are required to address this issue more closely.

Future studies should also compare the efficacy of different stretch protocols in order to establish whether there is an optimal frequency, duration and intensity of stretch that is most effective in improving stretch tolerance in people with chronic pain. Perhaps there is a minimal level of stretch intensity that could still provide a stimulus sufficient enough to induce clinically worthwhile improvements in *apparent* muscle extensibility via increased stretch tolerance. Furthermore, an examination of the duration of benefit beyond two days would also be of value. Such investigations may assist in the prescription of a maintenance program for people with chronic pain. These questions remain yet to be answered.

Additionally, further investigations should be directed at ascertaining the relative merits of targeting stretch to specific muscle groups and joints in patients with musculoskeletal pain originating from different parts of the body. It is possible that patients with shoulder pain will respond better to specific shoulder stretches, as opposed to a generic hamstring stretch. Similarly, patients with low back pain may respond significantly better to hamstring stretches (as in this study), compared to patients complaining of neck pain. It is therefore important to prescribe specific

stretches to target specific muscles and joints. The effectiveness of specific stretch interventions to target specific areas therefore needs to be established by further research.

Chapter Five: Conclusion

Adequate muscle extensibility is essential for normal movement and function. Limited extensibility presents as a common problem for many people, including able-bodied and disabled individuals, as well as those suffering from chronic pain. In particular, for people with chronic musculoskeletal pain, poor extensibility may affect their tolerance to movement, resulting in reduced functional capabilities and physical deconditioning. Stretch exercises are frequently used in clinical rehabilitation settings such as multidisciplinary pain management programs in the belief that improvements in muscle extensibility can be attained. However, there is a lack of scientific evidence to support their clinical efficacy in the chronic pain population.

The findings of this study suggest that three weeks of stretch improves *apparent* muscle extensibility as a result of increased tolerance to the stretch sensation, while three weeks of stretch has no effect on the underlying structural properties of the muscle with no change in *real* muscle extensibility.

Stretch exercises should continue to be implemented into multidisciplinary pain management programs. By providing a graded exposure to movement, stretch therefore plays a vital role in the physical reconditioning of patients with chronic musculoskeletal pain.

References

Ada L, Goddard E, McCully J, Stavrinos T, Bampton J. Thirty minutes of positioning reduces the development of shoulder external rotation contracture after stroke: a randomized controlled trial. Archives of Physical Medicine and Rehabilitation. 2005;86:230-234.

Adams B, Bromley B. Psychology for Health Care: Key Terms and Concepts. USA: Macmillan Press Limited; 1998.

Antoney MM, Bieling PJ, Cox BJ, Enns MW, Swinson RP. Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety and Stress scales in Clinical gorup and a community sample. Psychological Assessment. 1998;10:176-181.

Asghari A, Nicholas MK. Pain self-efficacy beliefs and pain behaviour: A prospective study. Pain. 2001;94:85-100.

Baker CD, Pynsent PB, Fairbank JC. The Oswestry Disability Index revisited: its reliability, repeatability and validity, and a comparison with the St. Thomas's Disability Index. In: Roland MO, Jenner JR, editors. Back Pain: New Approaches to Education and Rehabilitation. Manchester: University Press; 1989. p. 174-186.

Bandura A. Self-efficacy: toward a unifying theory of behavioural change. Psychological Review. 1977;84:191-215.

Bandy WD, Irion JM, Briggler M. The effect of time and frequency of static stretching on flexibility of the hamstring muscles. Physical Therapy. 1997;77(10):1090-1096.

Bandy WD, Irion JM, Briggler M. The effect of static stretch and dynamic range of motion training on flexibility of the hamstring muscles. Journal of Orthopaedic and Sports Physical Therapy. 1998;27:295-300.

Beck AT, Rush AJ, Shaw BF, Emery G. Cognitive therapy of depression. New York: Guilford Press; 1979.

Becker N, Sjogren P, Bech P, Olsen AK, Eriksen J. Treatment outcome of chronic non-malignant pain patients managed in a Danish multidisciplinary pain centre compared to general practice: A randomised controlled trial. Pain. 2000;84:203-211.

Ben M, Harvey L, Denis S, Glinsky J, Goehl G, Chee S, Herbert RD. Does 12 weeks of regular standing prevent loss of ankle mobility and bone mineral density in people with recent spinal cord injuries? Australian Journal of Physiotherapy. 2005;51:251-256.

Ben M, Harvey LA. Regular stretch does not increase muscle extensibility: a randomized controlled trial. Scandinavian Journal of Medicine and Science in Sports. 2009;Published online:DOI: 10.1111/j.1600-0838.2009.00926.x.

Bjorklund M, Hamberg J, Crenshaw AG. Sensory adaptation after a 2-week stretching regimen of the rectus femoris muscle. Archives of Physical Medicine and Rehabilitation. 2001;82:1245-1250.

Blyth FB, Macfarlane GJ, Nicholas MK. The contribution of psychosocial factors to the development of chronic pain: The key to better outcomes for patients? Pain. 2007;129:8-11.

Blyth FB, March LM, Brnabic AJM, Jorm LR, Williamsom M, Cousins MJ. Chronic pain in Australia: A prevalence study. Pain. 2001;89:127-134.

Blyth FB, March LM, Nicholas MK, Cousins MJ. Self-management of chronic pain: A population-based study. Pain. 2005;113:285-292.

Bohannon RW. Effect of repeated eight-minute muscle loading on the angle of straight-leg raising. Physical Therapy. 1984;64:491-497.

Bonnar BP, Deivert RG, Gould TE. The relationship between isometric contraction durations during hold-relax stretching and improvement of hamstring flexibility. Journal of Sports Medicine and Physical Fitness. 2004;44:258-261.

Bousema EJ, Verbunt JA, Seelen HAM, Vlaeyen JWS, Knottnerus JA. Disuse and physical deconditioning in the first year after the onset of back pain. Pain. 2007;130:279-286.

Brannon L, Feist J. Health Psychology: An Introduction to Behaviour and Health. 4th ed. USA: Brooks/Cole; 2000.

Brown SE, Atchison JW, Gnatz SM, Cifu DX. Pain rehabilitation. 2. Documentation of acute, subacute, and chronic pain. Archives of Physical Medicine and Rehabilitation. 1998;79, Supplement 1:S54-S59.

Chan SP, Hong Y, Robinson PD. Flexibility and passive resistance of the hamstrings of young adults using two different static stretching protocols. Scandinavian Journal of Medicine and Science in Sports. 2001;11:81-86.

Coutinho EL, Gomes ARS, Franca CN, Oishi J, Salvini TF. Effect of passive stretching on the immobilized soleus muscle fiber morphology. Brazilian Journal of Medical and Biological Research. 2004;37:1853-1861.

Cox VM, Williams PE, Wright H, James RS, Gillott KL, Young IS, Goldspink DF. Growth induced by incremental static stretch in adult rabbit latissimus dorsi muscle. Experimental Physiology. 2000;85:193-202.

Davis DS, Ashby PE, McCale KL, McQuain JA, Wine JM. The effectiveness of 3 stretching techniques on hamstring flexibility using consistent stretching parameters. Journal of Strength and Conditioning Research. 2005;19(1):27-32.

Dean C, Mackey F, Katrak P. Examination of shoulder positioning after stroke: A randomized controlled pilot trial. Australian Journal of Physiotherapy. 2000;46:35-39.

Decoster LC, Cleland J, Altieri C, Russell P. The effects of hamstring stretching on range of motion: a systematic literature review. Journal of Orthopaedic and Sports Physical Therapy. 2005;35:377-387.

Duong B, Low M, Moseley AM, Lee RY, Herbert RD. Time course of stress relaxation and recovery in human ankles. Clinical Biomechanics. 2001;16:601-607.

Dworkin RH, Turk DC, Farrar JT, Haythornthwaite JA, Jensen MP, Katz NP, Kerns RD, Stucki G, Allen RR, Bellamy N, Carr DB, Chandler J, Cowan P, Dionne R, Galer BS, Hertz S, Jadad AR, Kramer LD, Manning DC, Martin S, McCormick CG, McDermott MP, McGrath P, Quessy S, Rappaport BA, Robbins W, Robinson JP, Rothman M, Royal MA, Simon L, Stauffer JW, Stein W, Tollett J, Wernicke J, Witter J. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. Pain. 2005;113:9-19.

Elliott AM, Smith BH, Penny K, Smith WC, Chambers WA. The epidemiology of chronic pain in the community. Lancet. 1999;354:1248-1252.

Engel GL. The needs for a new medical model: A challenge for biomedicine. Science. 1977;196:129-136.

Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry low back pain questionnaire. Physiotherapy. 1980;66:271-273.

Fairbank JC, Pynsent PB. The Oswestry disability index. Spine. 2000;25:2940-2952.

Finniss DG, Murphy PM, Brooker C, Nicholas MK, Cousins MJ. Complex regional pain syndrome in children and adolescents. European Journal of Pain. 2006;10:767-770.

Flor H, Hermann C. Biopsychosocial models of pain. In: Dworkin RH, Breitbart WS, editors. Psychosocial Aspects of Pain: A Handbook for Health Care Providers Progress in Pain Research and Management Seattle: IASP Press; 2004. p. 47-75.

Folpp H, Deall S, Harvey LA, Gwinn T. Can apparent increases in muscle extensibility with regular stretch be explained by changes in tolerance to stretch? Australian Journal of Physiotherapy. 2006;52:45-50.

Gajdosik RL, Allred JD, Gabbert HL, Sonsteng BA. A stretching program increases the dynamic passive length of and passive resistive properties of the calf muscle-tendon unit of unconditioned younger women. European Journal of Applied Physiology. 2007;99:449-454.

Gajdosik RL, Linden DWV, McNair PJ, Williams AK, Riggin TJ. Effects of an eight-week stretching program on the passive-elastic properties and function of the calf muscles of the older women. Clinical Biomechanics. 2005;20:973-983.

Gauvin MG, Riddle DL, Rothstein JM. Reliability of clinical measurements of forward bending using the modified fingertip-to-floor method. Physical Therapy. 1990;70:443-447.

Goldberg GM, Kerns RD, Rosenberg R. Pain-relevant support as a buffer from depression among chronic pain patients low in instrumental activity. The Clinical Journal of Pain. 1993;9:33-40.

Goldspink DF. The influence of immobilization and stretch on protein turnover of rat skeletal muscle. Journal of Physiology. 1977;264:267-282.

Goldspink G, Tarbary C, Tarbary JC, Tardieu C, Tardieu G. Effect of denervation on the adaptation of sarcomere number and muscle extensibility to the functional length of the muscle. Journal of Physiology. 1974;236:733-742.

Gomes ARS, Coutinho EL, Franca CN, Polonio J, Salvini TF. Effect of one stretch a week applied to the immobilized soleus muscle on rat muscle fiber morphology. Brazilian Journal of Medical and Biological Research. 2004;37:1473-1480.

Gossman MR, Sahrmann SA, Rose SJ. Review of length-associated changes in muscle: experimental evidence and clinical implications. Physical Therapy. 1982;62(12):1799-1807.

Greenleaf JE. Intensive exercise training during bed rest attenuates deconditioning. Medicine and Science in Sports and Exercise. 1997;29:207-215.

Halbertsma JPK, Goeken LNH. Stretching exercises: Effect on passive extensibility and stiffness in short hamstrings of healthy subjects. Archives of Physical Medicine and Rehabilitation. 1994;75:976-981.

Halbertsma JPK, Mulder I, Goeken LNH, Eisma WH. Repeated passive stretching: Acute effects on the passive muscle moment and extensibility of short hamstrings. Archives of Physical Medicine and Rehabilitation. 1999;80:407-414.

Halbertsma JPK, Van Bolhuis AI, Goeken LNH. Sport stretching: Effect on passive muscle stiffness of short hamstrings. Archives of Physical Medicine and Rehabilitation. 1996;77:688-692.

Harris I, Mulford J, Solomon M, van Gelder JM, Young J. Association between compensation status and outcome after surgery a meta-analysis. Journal of American Medical Association. 2005;293:1644-1652.

Hartig DE, Henderson JM. Increasing hamstring flexibility decreases lower extremity overuse injuries in military basic trainees. American Journal of Sports Medicine. 1999;27:173-176.

Harvey L, Baillie R, Ritchie B, Simpson D, Pironello D, Glinsky J. Does three months of nightly splinting reduce the extensibility of the flexor pollicis longus muscle in people with tetraplegia? Physiotherapy Research International. 2007;12(1):5-13.

Harvey L, De Jong I, Goehl G, Mardwedel S. Twelve weeks of nightly stretch does not reduce thumb web-space contractures in people with a neurological condition: a randomised controlled trial. Australian Journal of Physiotherapy. 2006;52(4):251-258.

Harvey L, Herbert R, Crosbie J. Does stretching induce lasting increases in joint ROM? A systematic review. Physiotherapy Research International. 2002;7:1-13.

Harvey LA, Batty J, Crosbie J, Poulter S, Herbert RD. A randomized trial assessing the effects of 4 weeks of daily stretching on ankle mobility in patients with spinal cord injuries. Archives of Physical Medicine and Rehabilitation. 2000;81:1340-1347.

Harvey LA, Byak AJ, Ostrovskaya M, Glinsky J, Katte L, Herbert RD. Randomised trial of the effects of four weeks of daily stretch on extensibility of hamstring muscles in people with spinal cord injuries. Australian Journal of Physiotherapy. 2003;49:176-181.

Harvey LA, Herbert RD. Muscle stretching for treatment and prevention of contracture in people with spinal cord injury. Spinal Cord. 2002;40:1-9.

Harvey LA, McQuade L, Hawthorne S, Byak A. Quantifying the magnitude of torque physiotherapists apply when stretching the hamstring muscles of people with a spinal cord injury. Archives of Physical Medicine and Rehabilitation. 2003;84:1072-1075.

Hayden JA, van Tulder MW, Malmivaara AV, Koes BW. Meta-analysis: Exercise therapy for nonspecific low back pain. Annals of Internal Medicine. 2005;142:765-775.

Herbert RD. Preventing and treating stiff joints. In: Crosbie J, McConnell J, editors. Key Issues in Musculoskeletal Physiotherapy. Boston: Butterworth Heinemann; 1993. p. 114-141.

Herbert RD, Balnave RJ. The effect of positioning of immobilisation on resting length, resting stiffness, and weight of the soleus muscle of the rabbit. Journal of Orthopaedic Research. 1993;11:358-366.

Herbert RD, Crosbie J. Rest length and compliance of non-immobilised and immobilised rabbit soleus muscle and tendon. European Journal of Applied Physiology and Occupational Physiology. 1997;76:472-479.

Hreljac A, Marshall RN, Hume PA. Evaluation of lower extremity overuse injury potential in runners. Medicine and Science in Sports and Exercise. 2000;32:1635-1641.

International Association for the Study of Pain. Pain terms: A list with definitions and notes on usage. Pain. 1979;6:249-252.

Jensen IB, Bergstrom G, Ljungquist T, Bodin L. A 3-year follow-up of a multidisciplinary rehabilitation programme for back and neck pain. Pain. 2005;115:273-283.

Jensen MP, Karoly P, Braver S. The measurement of clinical pain intensity: A comparison of six methods. Pain. 1986;27:117-126.

Jensen MP, Karoly P, O'Riordan EF, Bland F, Burns RS. The subjective experience of acute pain: An assessment of the utility of 10 indices. The Clinical Journal of Pain. 1989;5:153-159.

Jensen MP, Storm SE, Turner JA, Romano JM. Validity of Sickness Impact Profile Roland scale as a measure of dysfunction in chronic pain patients. Pain. 1992;50:157-162.

Kavien TK, Kaasa S, Smedstad LM. Performance of the Norwegian SF-36 Health Survey in patient with rheumatoid arthritis. II a comparison of SF-36 with disease specific measures. Journal of Clinical Epidemiology. 1998;51:1077-1086.

Keefe FJ, Rumble ME, Scipio CD, Giordano LA, Peri LM. Psychological aspects of persistent pain: current state of the science. The Journal of Pain. 2004;5:195-211.

Kerns RD, Turk DC, Rudy TE. The West Haven-Yale Multidimensional Pain Inventory (WHYMPI). Pain. 1985;23:345-356.

Kori SH, Miller RP, Todd DD. Kinesiophobia: a new view of chronic pain behaviour. Pain Management. 1990;3:35-43.

Kubo K, Kanehisa H, Fukunaga T. Effect of stretching training on the viscoelastic properties of human tendon structures in vivo. Journal of Applied Physiology. 2002;92:595-601.

Laessoe U, Voigt M. Modification of stretch tolerance in a stooping position. Scandinavian Journal of Medicine and Science in Sports. 2003;14:239-244.

Lannin NA, Cusick A, McCluskey A, Herbert RD. Effects of splinting on wrist contracture following stroke: A randomized controlled trial. Stroke. 2007;38(1):111-116.

Lannin NA, Horsley SA, Herbert RD, McCluskey A, Cusick A. Splinting the hand in the functional position after brain impairment: A randomized, controlled trial. Archives of Physical Medicine and Rehabilitation. 2003;84:297-301.

Leeuw M, Goossens MEJB, Linton SJ, Crombez G, Boersma K, Vlaeyen JWS. The fear-avoidance model of musculoskeletal pain: Current state of scientific evidence. Journal of Behavioral Medicine. 2007;30:77-94.

Loeser JD. Concepts of pain. In: Stanton-Hicks M, Boas R, editors. Chronic Low Back Pain. New York: Raven press; 1982. p. 145-148.

Lovibond PF. Long-term stability of depression, anxiety, and stress syndromes. Journal of Abnormal Psychology. 1998;107:520-526.

Lovibond PF, Lovibond SH. The structure of negative emotional states: comparison of the depression anxiety stress scales (DASS) with the beck depression and anxiety inventories. Behaviour Research and Therapy. 1995;33:335-343.

Lundeberg T, Lund I, Dahlin L, Borg E, Gustafsson C, Sandin L, Rosen A, Kowalski J, Eriksson SV. Reliability and responsiveness of three different pain assessments. Journal of Rehabilitation Medicine. 2001;33:279-283.

Magnusson SP. Passive properties of human skeletal muscle during stretch maneuvers. A review. Scandinavian Journal of Medicine and Science in Sports. 1998;8:65-77.

Magnusson SP, Aagaard P, Simonsen E, Bojsen-Moller F. Passive tensile stress and energy of the human hamstring muscle in vivo. Scandinavian Journal of Medicine and Science in Sports. 2000;10:351-359.

Magnusson SP, Simonsen EB, Aagaard P, Gleim GW, McHugh MP, Kjaer M. Viscoelastic response to repeated static stretching in the human hamstring muscle. Scandinavian Journal of Medicine and Science in Sports. 1995;5:342-347.

Magnusson SP, Simonsen EB, Aagaard P, Kjaer M. Biomechanical responses to repeated stretches in human hamstring muscle in vivo. American Journal of Sports Medicine. 1996;24:622-628.

Magnusson SP, Simonsen EB, Aagaard P, Sorensen H, Kjaer M. A mechanism for altered flexibility in human skeletal muscle. Journal of Applied Physiology. 1996;497:291-298.

Maher CG, Sherrington C, Herbert RD, Moseley AM, Elkins M. Reliability of the PEDro scale for rating quality of randomized controlled trials. Physical Therapy. 2003;83:713-721.

Martikainen P, Bartley M, Lahelma E. Psychosocial determinants of health in social epidemiology. International Journal of Epidemiology. 2002;31:1091-1093.

McCracken LM, Eccleston C. A prospective study of acceptance of pain and patient functioning with chronic pain. Pain. 2005;118:164-169.

McCracken LM, Vowles KE, Gauntlett-Gilbert J. A prospective investigation of acceptance and control-oriented coping with chronic pain. Journal of Behavioral Medicine. 2007;30:339-349.

Medeiros JM, Smidt GL, Burmeister LF, Soderberg GL. The influence of isometric exercise and passive stretch on hip joint motion. Physical Therapy. 1977;57:518-523.

Melzack R. The McGill pain questionnaire: major properties and scoring methods. Pain. 1975;1:277-299.

Melzack R. Pain - An overview. Acta Anaesthesiologica Scandinavica. 1999;43:880-884.

Melzack R, Katz J. The McGill Pain Questionnaire appraisal and current status. In: Turk DC, Melzack R, editors. Handbook of Pain Assessment. 2nd ed. New York: Guilford Press; 2001. p. 35-52.

Melzack R, Katz J. Pain in the 21st century: The neuromatrix and beyond. In: Young G, Kane AW, Nicholson K, editors. Psychological Knowledge in Court: PTSD, Pain, and TBI. US: Springer; 2006. p. 129-148.

Melzack R, Wall PD. Pain mechanisms: A new theory. Science. 1965;150:171-179.

Melzack R, Wall PD. The Challenge of Pain. 2nd ed. London: Penguin Books; 1996.

Merskey H, Bogduk N. Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms, 2nd ed. Seattle: IASP Press; 1994.

Merskey H, Bogduk N, editors. Classification of Chronic Pain: Task Force on Taxonomy. 2nd ed. Seattle: IASP Press; 1994.

Morley S, Williams A, Cde C, Black S. A confirmatory factor analysis of the Beck Depression Inventory in chronic pain. Pain. 2002;99:289-298.

Moseley AM. The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. Physical Therapy. 1997;77(3):240-247.

Moseley AM, Herbert RD, Nightingale EJ, Taylor DA, Evans TM, Robertson GJ, Gupta SK, Penn J. Passive stretching does not enhance outcomes in patients with plantarflexion contracture after cast immobilization for ankle fracture: A randomized controlled trial. Archives of Physical Medicine and Rehabilitation. 2005;86(6):1118-1126.

Musacchia XJ. Disuse atrophy of skeletal muscle animal models. Exercise and Sport Sciences Reviews. 1988;16:61-87.

Newton-John TRO. Solicitousness and chronic pain: a critical review. Pain Reviews. 2002;9:7-27.

Nicholas M. Pain management in musculoskeletal conditions Best Practice and Research Clinical Rheumatology. 2008;22:451-470.

Nicholas M, Molloy A, Tonkin L, Beeston L. Manage Your Pain. Sydney: ABC Books; 2000.

Nicholas M, Molloy A, Tonkin L, Beeston L. Stretching and exercising. In: Nicholas M, Molloy A, Tonkin L, Beeston L, editors, editors. Manage Your Pain. Sydney: ABC Books; 2000. p. 98-127.

Nicholas MK. Self-efficacy and chronic pain. Paper presented at the annual conference of the British Psychological Society; St. Andrews,1989.

Nicholas MK. The pain self-efficacy questionnaire: Taking pain into account. European Journal of Pain. 2007;11:153-163.

Nicholas MK, Asghari A, Blyth FM. What do the numbers mean? Normative data in chronic pain measures. Pain. 2008;134:158-173.

Nicholas MK, Wilson PH, Goyen J. Comparison of cognitive-behavioural group treatment and an alterative non-psychological treatment for chronic low-back pain. Pain. 1992;48:339-347.

Patrick DL, Deyo RA, Atlas SJ, Singer DE, Chapin A, Keller RB. Assessing health-related quality of life in patients with sciatica. Spine. 1995;20:1899-1908.

Pocock SJ. Clinical Trials. A Practical Approach. Chichester: Wiley; 1983.

Price DD. Psychological and neural mechanisms of the affective dimension of pain. Science. 2000;288:1769-1772.

Price DD, Harkins SW. Psychophysical approaches to pain measurement and assessment. In: Turk DC, Melzack R, editors. Handbook of Pain Assessment. New York: Guilford; 1992. p. 111-134.

Proske U, Morgan DL, Gregory JE. Thixotropy in skeletal muscle and in muscle spindles: a review. Progress in Neurobiology. 1993;41:705-721.

Refshauge K, Raymond J, Nicholson G, van den Dolder PA. Night splinting does not increase ankle range of motion in people with Charcot-Marie-Tooth disease: A randomised, cross-over trial. Australian Journal of Physiotherapy. 2006;52:193-199.

Reid DA, McNair PJ. Passive force, angle, and stiffness changes after stretching of hamstring muscles. Medicine and Science in Sports and Exercise. 2004;36:1944-1948.

Reid DA, McNair PJ. Passive force, angle, and stiffness changes after stretching of hamstring muscles. Medicine and Science in Sports and Exercise. 2004;36(11):1944-1948.

Roland M, Morris S. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. Spine. 1983;8:141-144.

Scheer SJ, Watanabe TK, Radack KL. Randomized controlled trials in industrial low back pain. Part 3. Subacute/chronic pain interventions. Archives of Physical Medicine and Rehabilitation. 1997;78:414-423.

Schlenk EA, Erlen JA, Dunbar-Jacob J, Engberg S, Serika SA, Rohay JM, Bernier MJ. Health related quality of life in chronic disorder. A comparison across studies using the Mos SF-36. Quality of Life Research. 1998;7:57-65.

Schonstein E, Kenny DT, Keating J, Koes BW. Work conditioning, work hardening and functional restoration for workers with back and neck pain. Cochrane Database of Systematic Reviews. 2003:Issue 3.

Shrier I. Does stretching improve performance? A systematic and critical review of the literature. Clinical Journal of Sport Medicine. 2004;14:267-273.

Skouen JS, Grasdal A, Haldorsen EMH. Return to work after comparing outpatient multidisciplinary treatment programs versus treatment in general practice for patients with chronic widespread pain. European Journal of Pain. 2006;10:145-152.

Smeets RJ, Wittink H, Hidding A, Knottnerus JA. Do patients with chronic low back pain have a lower level of aerobic fitness than healthy controls? Are pain, disability, fear of injury, working status, or level of leisure time activity associated with the difference in aerobic fitness level? Spine. 2006;31:90-97.

Smeets RJEM, Wade D, Hidding A, Van Leeuwen PJCM, Vlaeyen JWS, Knottnerus JA. The association of physical deconditioning and chronic low back pain: A hypothesis-oriented systematic review. Disability and Rehabilitation. 2006;28(11):673-693.

Stratford PW, Binkley JM. Measurement properties of the RM-18: a modified version of the Roland-Morris Disability Scale. Spine. 1997;22:2416-2421.

Sullivan MJL, Lynch ME, Clark AJ. Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions. Pain. 2005;113:310-315.

Tabary JC, Tabary C, Tardieu C, Tardieu G, Goldspink G. Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. Journal of Physiology. 1972;224:231-244.

Taylor D, Dalton JD, Seaber AV, Garrett WE. Viscoelastic properties of muscle-tendon units. The biomechanical effects of stretching. American Journal of Sports Medicine. 1989;18:300-309.

Thacker SB, Gilchrist J, Stroup DF, Kimsey CD. The impact of stretching on sports injury risk: A systematic review of the literature. Medicine and Science in Sports and Exercise. 2004;36:371-378.

Toft E, Espersen GT, Kålund S, Sinkjær T, Hornemann BC. Passive tension of the ankle before and after stretching. American Journal of Sports Medicine. 1989;17:489-494.

Turk DC. Biopsychosocial perspective on chronic pain. In: Gatchel RJ, Turk DC, editors. Psychosocial Factors in Pain: Critical Perspective. New York: Guilford Press; 1996. p. 18-34.

Turk DC, Dworkin RH, Allen RR, Bellamy N, Brandenburg N, Carr DB, Cleeland C, Dionne R, Farrar JT, Galer BS, Hewitt DJ, Jadad A, Katz NP, Kramer LD, Manning DC, McCormick CG, McDermott M, McGrath P, Quessy S, Rappaport BA, Robinson JP, Royal MA, Simon L, Stauffer JW, Stein W, Tollett J, Witter J. Core outcome domains for chronic pain clinical trials: IMMPACT recommendations. Pain. 2003;106:337-345.

Turk DC, Flor H. Chronic pain: A biobehavioral perspective. In: Gatchel RJ, Turk DC, editors. Psychosocial Factors in Pain: Critical Perspectives. New York: Guilford Press; 1999. p. 18-34.

Turk DC, Okifuji A. Psychological factors in chronic pain: Evolution and revolution. Journal of Consulting and Clinical Psychology. 2002;70:678-690.

Turton A, Britton E. A pilot randomized controlled trial of a daily muscle stretch regime to prevent contractures in the arm after stroke. Archives of Physical Medicine and Rehabilitation. 2005;19:600-612.

Van Tulder M, Malmivaara A, Esmail R, Koes B. Exercise therapy for low back pain. Spine. 2000;25(21):2784-2796.

Verbunt JA, Seelen HAM, Vlaeyen JWS, van de Heijden GJ, Heuts PH, Pons K, Knottnerus JA. Disuse and deconditioning in chronic low back pain: Concepts and hypotheses on contributing mechanisms. European Journal of Pain. 2003;7:9-21.

Vlaeyen JWS, de Jong J, Geilen M, Heuts PHTG, van Breukelen G. The treatment of fear of movement/(re)injury in chronic low back pain: Further evidence on the effectiveness of exposure in vivo. The Clinical Journal of Pain. 2002;18:251-261.

Vlaeyen JWS, Kole-Snijders AM, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioural performance. Pain. 1995;62:363-372.

Ware JE, Sherbourne CD. The MOS 36-item Short-Form Health Survey (SF-36): 1 Conceptual framework and item selection. Medical Care. 1992;30:473-483.

Williams A, Richardson PH, Nicholas MK, Pither CE, Harding VR, Ridout KL, Ralphs JA, Richardson IH, Justins DM, Chamberlain JH. Inpatient vs. outpatient pain management: Results of a randomised controlled trial. Pain. 1996;66:13-22.

Williams P, Kyberd P, Simpson H, Kenwright J, Goldspink G. The morphological basis of increased stiffness of rabbit tibalis anterior muscle during surgical limb-lengthening. Journal of Anatomy. 1998;193:131-138.

Williams PE. Effect of intermittent stretch on immobilised muscle. Annals of Rheumatic Diseases. 1988;47:1014-1016.

Williams PE. Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. Annals of Rheumatic Diseases. 1990;49:316-317.

Williams PE, Goldspink G. The effect of immobilization on the longitudinal growth of striated muscle fibres. Journal of Anatomy. 1973;116:45-55.

Williams PE, Goldspink G. Changes in sarcomere length and physiological properties in immobilized muscle. Journal of Anatomy. 1978;127:459-468.

Williams PE, Goldspink G. Connective tissue changes in immobilised muscle. Journal of Anatomy. 1984;138:343-350.

Winters MV, Blake CG, Trost JS, Marcello-Brinker TB, Lowe L, Garber MB, Wainner RS. Passive versus active stretching of hip flexor muscles in subjects with limited hip extension: A randomized clinical trial. Physical Therapy. 2004;84:800-807.

Youdas JW, Krause DA, Egan KS. The effect of static stretching of the calf muscle-tendon unit on active ankle dorsiflexion range of motion. Journal of Orthopaedic and Sports Physical Therapy. 2003;33:408-417.

Appendices

A. Subject Information Sheet

	Pain Management and Research Centre Department of Anaesthesia & Pain Management University of Sydney at Royal North Shore Hospital	

Protocol No. 0603-035M
Version # 3 (29/01/07)

PARTICIPANT INFORMATION SHEET

The effects of stretching for patients with persisting pain – a randomised controlled trial

You are being asked to participate in this research project.

This Participant Information Sheet contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it. Please read this Participant Information Sheet carefully. Feel free to ask questions about any information in the document. Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Participant Information Sheet and Consent Form to keep as a record.

The study staff will answer any questions at any time about this research study or your participation in the study.

This study was reviewed by the Northern Sydney Central Coast Health Human Research Ethics Committee. The Ethics Committee is independent of the study investigators. The purpose of the Ethics Committee is to protect the rights and safety of people who volunteer to take part in research studies.

Study Description: This study looks at the effects of stretching for patients suffering from persisting (chronic) pain. The study will involve thirty patients attending the ADAPT pain management program.

The aim of this study is to determine the effects of regular stretching on the extensibility of your hamstring muscles. We hope to learn whether regular stretching increases muscle extensibility. You have been selected as a possible participant in this study because your hamstring muscles have limited extensibility.

If you decide to participate, you will be asked to stretch the hamstring muscles of one leg for one minute per day for three weeks during the ADAPT program. This will be in addition to participating in other components of the ADAPT program. You will not be able to choose which leg is stretched. Your stretching will be supervised each day in a group

session during the program. The physiotherapist will show you how to perform the hamstring stretch correctly. You may experience some mild discomfort with the stretches. If you are concerned at any stage during each session, you can discuss your concerns with the supervising physiotherapist.

The extensibility of your hamstring muscles will be tested at the beginning of the study and at the completion of the three-week ADAPT program. Measurements will be taken with a device specifically designed for this purpose. The device will hold your knee straight and raise your leg up above the bed. A small device will then be used to measure the deviation of your leg from horizontal. In addition, an increasingly large stretch will be applied to your hamstring muscles. You will be asked to indicate to the investigator when you "first feel the stretch" and again when the stretch is "as much as you can tolerate". The stretch will be removed immediately at this point.

In order to be confident that any changes in the extensibility of your hamstring muscles are solely due to the effects of the stretch intervention, we request that you do not stretch the hip or knee muscles of either leg for the duration of the study. However, other than this, you may participate in all other components of the ADAPT program and your normal activities.

There is a possibility that the hamstring muscles of your untreated leg will become less extensible than the hamstring muscles of your treated leg. However, as soon as the project is completed you may recommence your usual activities and stretches. In this way, any differences should be quickly reversed.

Benefits of Participation: We cannot and do not hold out that you will receive any benefits from this study.

Termination of the Study: You are able to withdraw your participation from the study at any time with no effect on your ongoing care at the centre. You will still be able to participate in the ADAPT program. The data we collect from you will also not be used.

What if something goes wrong: If something goes wrong or you have any queries about the way in which the study is conducted and you do not feel comfortable contacting the research staff, you may contact the Patient Representative at Royal North Shore Hospital who is an independent person on 99267612.

Confidentiality: Any information about you that is obtained in connection with this study will remain confidential and will be disclosed only with your written permission. However, the results of the study may be published or disclosed to other people in a way that will not identify you. All data will be coded and stored on computer discs by the researcher for 7 years, at which time the files will be disposed of.

Who should I contact for further information: For further information regarding the study, please **contact Roberta Law on 99267556** or Damien Finniss on 99266377. **This study will form the basis of a Master of Research Degree for Roberta Law (Physiotherapy Department, Royal North Shore Hospital).**

Results of the Study: At the conclusion of the study you may ask one of the researchers about the results of the study.

B. Informed Consent Form

	Pain Management and Research Centre Department of Anaesthesia & Pain Management University of Sydney at Royal North Shore Hospital	

Consent Form to Participate in a Research Project

I, _____

(name of participant)

of_____

(street) (suburb/town) (state & postcode)

have been invited to participate in a research project entitled:

The effects of stretching for patients with persisting pain – a randomised controlled trial

In relation to this project I have read the Participant Information Sheet and have been informed of the following points:

1. Approval for the protocol has been given by the Human Research Ethics Committee (HREC) of Northern Sydney Health.

2. The aim of the project is to investigate the effects of stretching for patients suffering from persisting (chronic) pain.

3. The results obtained from the study will not be of direct benefit to my medical management.

4. The study will involve stretching one leg only for a period of three weeks, and that there may possibly be some short-term loss of extensibility in the leg that is not stretched.

5. My involvement in this project may be terminated if any of the following circumstances develop: My Doctor feels that I may no longer gain benefit from participation, the study is terminated or I wish to no longer participate in the study.

6. Should I develop a problem which I suspect may have resulted from my involvement in this project, I am aware that I may contact Damien Finniss on 9926 6377.

Date: _____ Witness: _____

(Please print name)

Signature: _____ Signature:_____

(of participant/volunteer) (of witness)

7. Should I have any problems or queries about the way in which the study was conducted, and I do not feel comfortable contacting the research staff, I am aware that I may contact the Patient Representative who is an independent person within the Hospital on 99267612

8. I can refuse to take part in this project or withdraw from it at any time without affecting my medical care.

9. I understand that participating in this Clinical Trial may or may not benefit my medical treatment directly however my participation may assist in the development of treatments and/or procedures for the future.

10. Participation in this project will not result in any extra medical and hospital costs to me.

11. I understand that my research records will be stored in the following manner: they will be locked in a filing cabinet in a secure office. The research team, authorised personnel, and regulatory entities may have access to my study records to protect my safety and welfare.

12. I understand that my initials and date of birth and a unique study number will identify my medical information. This information is potentially identifiable but all precautions will be taken by the clinical staff to ensure the information will be kept confidential.

13. If the results of my tests or information regarding my medical history are published, my identity will not be revealed.

14. While participating in this study, I should not take part in any other research project without approval from all of the investigators.

15. I declare that I am over the age of 18 years.

After considering all these points, I accept the invitation to participate in this project.

I am aware that I will be given a copy of the Participant Information Sheet and Consent Form.

I also state that I have/have not participated in any other research project in the past 3 months.

If I have, the details as follows: _____

Dr _____ on: _____
(phone and page numbers)

Date:_____ Witness:_____
(Please print name)

Signature:_____ Signature: _____
(of participant/volunteer) (of witness)

Investigators' confirming statement:

I have given this research subject information on the study, which in my opinion is accurate and sufficient for the subject to understand fully the nature, risks and benefits of the study, and the rights of a research subject. There has been no coercion or undue influence. I have witnessed the signing of this document by the subject.

Date: _____

Investigator's Name: _____

Investigator's Signature: _____

Withdrawal from Participation

Protocol Title: **The effects of stretching for patients with persisting pain – a randomised controlled trial**

An option should I wish to withdraw my consent to participate in the research protocol entitled above is to contact the researcher and return this slip. I understand that if I withdraw from the research protocol my medical care, my relationship with the Hospital and medical attendants will not be affected.

Patient's Name: _____

Patient's Signature: _____

Date: _____

Please detach the Withdrawal of Participation Section and send to **Mr Damien Finniss, Pain Management and Research Centre, 9C, Royal North Shore Hospital, St Leonards, 2065** or if I would like to speak to a member of the study investigation team, I may contact **Roberta Law on 9926 7556** or Damien Finniss on 99266377.

C. Raw Outcome Data

1. MUSCLE EXTENSIBILITY

Hip Flexion Angle (degrees) with a Standardised Torque

Subject	CONTROL LEG			EXPERIMENTAL LEG			*Grand Difference*	*Standardised Torque (Nm)*
	Pre	Post	*Change*	Pre	Post	*Change*		
1	47	45	-2	54	50	-4	-2	24
2	57	53	-4	67	57	-10	-6	18
3	68	73	5	65	72	7	2	24
4	89	86	-3	80	75	-5	-2	43
5	73	86	13	80	85	5	-8	31
6	64	61	-3	61	70	9	12	18
7	56	60	4	61	60	-1	-5	31
8	60	55	-5	49	53	4	9	18
9	62	69	7	65	65	0	-7	37
10	78	77	-1	78	69	-9	-8	31
11	23	25	2	17	30	13	11	18
12	66	63	-3	66	67	1	4	31
13	51	52	1	51	56	5	4	18
14	46	56	10	43	39	-4	-14	24
15	35	32	-3	28	41	13	16	18
16	32	57	25	40	61	21	-4	18
17	85	81	-4	100	84	-16	-12	49
18	73	78	5	76	78	2	-3	24
19	73	81	8	67	74	7	-1	31
20	57	58	1	54	55	1	0	31
21	46	44	-2	46	46	0	2	18
22	78	74	-4	69	76	7	11	31
23	48	45	-3	41	47	6	9	31
24	90	91	1	99	103	4	3	43
25	47	57	10	53	57	4	-6	24
26	60	65	5	58	68	10	5	24
27	48	44	-4	38	52	14	18	24
28	37	51	14	37	51	14	0	18
29	58	45	-13	51	52	1	14	18
30	43	48	5	45	48	3	-2	24
MEAN	58	60	2	58	61	3	**1**	26
STDEV	17	16	7	19	16	8	**8**	8
95%CI							**3**	
						Le CI	**-2**	
						Ue CI	**4**	

2. STRETCH TOLERANCE

Hip Flexion Angle (degrees) with a Non-Standardised Torque

Subject	CONTROL LEG			EXPERIMENTAL LEG			*Grand Difference*
	Pre	**Post**	***Change***	**Pre**	**Post**	***Change***	
1	61	60	-1	64	62	-2	-1
2	69	60	-9	89	70	-19	-10
3	82	76	-6	78	91	13	19
4	94	94	0	90	101	11	11
5	96	96	0	98	99	1	1
6	70	64	-6	72	78	6	12
7	67	65	-2	69	71	2	4
8	72	77	5	57	58	1	-4
9	72	78	6	68	86	18	12
10	94	91	-3	78	83	5	8
11	36	56	20	27	63	36	16
12	74	66	-8	71	82	11	19
13	61	63	2	53	58	5	3
14	65	81	16	64	78	14	-2
15	44	54	10	44	64	20	10
16	47	59	12	64	75	11	-1
17	100	95	-5	105	105	0	5
18	77	91	14	80	100	20	6
19	112	109	-3	74	84	10	13
20	61	59	-2	59	67	8	10
21	46	47	1	46	55	9	8
22	83	85	2	87	99	12	10
23	48	54	6	54	72	18	12
24	99	94	-5	104	106	2	7
25	61	64	3	64	64	0	-3
26	71	73	2	80	91	11	9
27	61	61	0	60	74	14	14
28	37	75	38	47	87	40	2
29	63	50	-13	60	69	9	22
30	61	58	-3	52	66	14	17
MEAN	69	72	2	69	79	10	**8**
STDEV	19	16	10	18	15	11	**8**
95%CI							**3**
						Le CI	**5**
						Ue CI	**10**

3. STRETCH TOLERANCE

Highest Tolerated Torque (Nm) with a Non-Standardised Torque

Subject	CONTROL LEG			EXPERIMENTAL LEG			*Grand Difference*
	Pre	Post	*Change*	Pre	Post	*Change*	
1	34	38	5	31	34	3	-2
2	28	23	-5	23	23	0	5
3	40	31	-9	35	60	25	34
4	61	60	-2	75	90	15	17
5	50	43	-8	55	43	-12	-5
6	21	21	0	28	28	0	0
7	43	38	-5	38	46	8	12
8	31	34	3	23	23	0	-3
9	46	47	2	41	66	24	23
10	41	46	5	32	47	15	11
11	29	37	8	28	41	14	6
12	30	35	5	38	47	9	4
13	24	24	0	20	21	2	2
14	41	41	0	37	55	18	18
15	26	32	6	29	37	8	2
16	31	23	-8	31	31	0	8
17	58	70	12	58	80	21	9
18	29	35	6	29	47	18	12
19	79	72	-7	37	50	13	20
20	32	32	0	35	40	5	5
21	18	20	2	18	26	8	6
22	35	37	2	41	47	6	5
23	32	44	12	44	57	12	0
24	55	47	-8	47	47	0	8
25	31	32	2	34	29	-5	-6
26	29	29	0	41	43	2	2
27	32	35	3	35	44	9	6
28	18	31	13	29	52	23	10
29	25	23	-2	25	28	3	5
30	40	34	-6	32	43	11	17
MEAN	36	37	1	36	44	8	**8**
STDEV	14	13	6	12	16	9	**9**
95%CI							**3**
						Le CI	**4**
						Ue CI	**11**

4. STRETCH TOLERANCE

Pain Intensity (Numerical Rating Scale 0-10) with a Non-Standardised Torque

Subject	CONTROL LEG			EXPERIMENTAL LEG			*Grand Difference*
	Pre	**Post**	***Change***	**Pre**	**Post**	***Change***	
1	9	10	1	9	9	0	-1
2	9	9	0	10	10	0	0
3	7	8	1	7	8	1	0
4	6	8	2	6	7	1	-1
5	1	7	6	1	5	4	-2
6	10	10	0	10	10	0	0
7	4	3	-1	6	5	-1	0
8	9	9	0	9	10	1	1
9	9	7	-2	8	6	-2	0
10	8	7	-1	8	5	-3	-2
11	8	9	1	6	8	2	1
12	8	6	-2	7	6	-1	1
13	6	8	2	8	5	-3	-5
14	4	6	2	5	4	-1	-3
15	7	8	1	7	8	1	0
16	7	7	0	7	7	0	0
17	6	6	0	7	7	0	0
18	7	6	-1	6	7	1	2
19	8	7	-1	8	8	0	1
20	7	6	-1	7	6	-1	0
21	8	8	0	8	8	0	0
22	6	9	3	7	9	2	-1
23	7	7	0	2	6	4	4
24	5	8	3	8	8	0	-3
25	8	7	-1	8	9	1	2
26	7	6	-1	8	7	-1	0
27	7	8	1	6	8	2	1
28	6	6	0	7	4	-3	-3
29	4	7	3	6	7	1	-2
30	7	8	1	7	8	1	0
MEAN	7	7	1	7	7	0	**0**
STDEV	2	1	2	2	2	2	**2**
95%CI							**1**
						Le CI	**-1**
						Ue CI	**0**