Hip-Spine Interaction in Low Back Pain:

The Role of the Hip Extensors

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Abstract

Despite the high prevalence and global burden of low back pain (LBP), the pathogenesis is poorly understood. Without a better understanding of what structures are involved in the development and chronicity of LBP, the value and efficacy of clinical assessments and physical therapy interventions are limited. Although there is a clear link between the lumbar spine, pelvis and hip extensors during movement in both LBP and healthy subjects, there is limited evidence regarding whether it is passive or active components that are influenced. There is a need for improved prognostic evaluation of patients with LBP, including whether altered hip biomechanics are the result of structural, passive elements, or neuromuscular, active components of movement. Such evaluations will be beneficial for researchers, clinicians and physical therapists.

The purpose of the present investigation is initially to demonstrate how a handheld measuring device can be adapted for use in measuring passive hip moments during supine leg raising. Comparisons are then made between subjects with LBP and healthy controls. A validated dynamic biomechanical model is used to calculate passive hip moments at a variety of knee angles, from which a predictive equation is derived, which is specific to each subject. Following a gait analysis protocol, the predictive equation is used to calculate passive hip extensor moments during the hip flexion component of gait. Comparisons are made between passive hip extensor moments, total hip moments, power and work done, in subjects with and without LBP.

The present investigation demonstrated the high accuracy of a handheld force transducer for the measurement of passive hip moments. There were no

statistically significant differences in passive hip extensor biomechanical properties between subjects with LBP and healthy controls. However, assessment during walking demonstrated significant differences in passive hip extensor moments between subjects with LBP and controls. Further differences were identified in total hip moments, power and work done, despite no differences in gait parameters. It is plausible that the passive and active components of movement interact, although further research is required to determine whether such interactions are consistent and predictable.

It was observed that the passive contribution to hip biomechanics during the swing phase of gait is considerable, and should be incorporated into dynamic modelling. Differentiating between passive and active components may be particularly useful for researchers, clinicians and physical therapists, for evaluating which components are influenced by LBP and for assessing the efficacy of component-specific interventions. Future research should expand on this research to include a wider range of LBP patients, with different severity and disability of LBP, to develop a more complete range of data on how passive and active components are influenced and the range of interactions during common movements. Other research should attempt to determine which interventions are most appropriate for targeting changes to passive and active components independently, and in accordance with patient adaptations to LBP. The modelling, experimental procedures and customised equipment used in the present investigation are appropriate for use in assessing passive contributions to joint biomechanics during movement.

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Acknowledgements

It has been a life goal ever since early in my undergraduate degree to complete a doctorate, and the study of biomechanics and exercise physiology have been passions of mine for at least as long. When I first imagined what the doctoral research project would be like, I imagined a solitary journey, but the reality was so far from it, and so much more rewarding as a result.

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1. Introduction

1.1 Problem Statement

Low back pain (LBP) is one of the leading causes of disability globally, estimated to be responsible for 58.2 million disability-adjusted life years in 1990, and 83 million in 2010 (Buchbinder *et al.*, 2013). In a survey of the UK general population, 20% of individuals suffering with non-specific back pain reported it to be intense, disabling and chronic (Webb *et al.*, 2003).

Work absenteeism and productivity contribute the most to the economic burden of LBP (Maniadakis and Gray, 2000, Katz, 2006, Maetzel and Li, 2002, Dagenais *et al.*, 2008). Absenteeism represents 75% of the economic burden of LBP in Germany, where annual costs are approximately 7000 euros per patient (Juniper *et al.*, 2009). In Sweden, LBP accounts for 11% of illness-related work absenteeism, and costs approximately 1.86 billion euros a year, of which 84% is through absenteeism and lost productivity (Ekman *et al.*, 2005). In the U.S., the annual socioeconomic burden of LBP is estimated to be between US$100 billion and $200 billion, mostly through work absenteeism and reduced productivity (Katz, 2006). The same factors contribute to more than AU$9 billion of costs in Australia due to LBP (Walker *et al.*, 2003), and more than £12 billion in the UK (Maniadakis and Gray, 2000).

Due to a lack of certainty regarding causal factors in LBP, selecting an appropriate treatment modality that is both effective and economical is problematic (North *et al.*, 2014, Pai and Sundaram, 2004). Physiotherapy is the most costly treatment modality, overall, contributing approximately 17% of total

LBP treatment costs. Other costs include inpatient care (~17%), pharmacy (~13%), primary care (~13%), outpatient services (~8%), diagnostic imaging (~7%), specialists (~7%), surgery (~5%), chiropractic and osteopathy (~5%), other services (~5%), complementary and alternative medicine (~2%), emergency department (~1%) and mental health (~1%) (Dagenais *et al.*, 2008). Although these values differ by country, the overall distribution of costs is similar (Maniadakis and Gray, 2000).

Studies comparing the effectiveness of different physiotherapy-based interventions show similar improvements at 18 months follow-up (Critchley *et al.*, 2007, Kominski *et al.*, 2005). This finding suggests a lack a patient-specificity of treatments, leading to moderate but not optimal improvements for the individual. However, some investigators have recommended against physiotherapy and chiropractic care, on the basis that costs are high and outcomes no better than standard care (pharmaceuticals and recommendations from a doctor on back care and physical activity) (Kominski *et al.*, 2005). It is plausible that the limitations in physiotherapy outcomes are related to the uncertainties in the cause of LBP, and how LBP manifests in terms of any structural and functional effects on the musculoskeletal and neuromuscular systems.

Risk factors for LBP include degeneration of lumbar discs and narrowing of disc spaces, although these cannot necessarily be regarded as the specific, underlying cause of LBP (Balague *et al.*, 2012). Obesity has been positively associated with LBP, although mechanical factors have not, such as occupational sitting, posture, standing and walking, manual handling, or movements such as pushing and pulling, bending and twisting (Balague *et al.*,

2012). Back pain is associated with increased age and disability (Webb *et al.*, 2003), although it can occur at any point in the lifespan. In adolescents, it has been associated with increased body mass index (BMI), reduced hamstring flexibility and reduced hip flexion (Sjolie, 2004).

LBP can only be attributed to a specific cause in 5-15% of cases, in which osteoporotic fracture, neoplasm or infection are regarded as responsible for the condition (Hoy *et al.*, 2010). There is a potential for LBP to arise from various anatomical structures, such as the bones, intervertebral discs, joints, ligaments, muscles, neural structures and blood vessels, but direct, causal associations remain rarely determined (Hoy *et al.*, 2010). Thus, in 85-95% of patients, LBP is considered non-specific (Hoy *et al.*, 2010, Balague *et al.*, 2012).

Kinematics of the lumbar spine and hips has previously been investigated in subjects with LBP during gait (Lamoth *et al.*, 2006a), sporting activity (Stuelcken *et al.*, 2010), sit-to-stand and stand-to-sit (Shum *et al.*, 2005a), and other activities of daily living (Shum *et al.*, 2005b). Individuals with LBP have been reported to have modifications in gait velocity, and sagittal-, transverse- and frontal-plane movements of the hips and spine, when compared with healthy controls (Sjolie, 2004, Wong and Lee, 2004, Lamoth *et al.*, 2006a). It has been hypothesised that these modifications may be long-term pain-avoidance, or pain-limiting adaptations, which are an effect of chronic, rather than acute, LBP (Lamoth *et al.*, 2004). However, the mechanisms responsible for pain-avoidance strategies remain to be elucidated (Tucker *et al.*, 2009).

Although hip flexion, stride velocity and stride length have been found to be reduced in individuals with LBP (Sjolie, 2004, Wong and Lee, 2004), there has been little investigation into whether these alterations derive from changes

in the structural materials of the muscle-tendon units (MTUs), or if they are neuromuscular alterations in motor control. Structural alterations are measured by their passive influence on joint moments, stiffness and strain energy, whereas motor control is measured via the active influence on joint moments. By differentiating between passive and active influences (structural and functional influences, respectively), it would be possible to improve our understanding of LBP and how the body adapts over time. This may also be useful in determining how preventative and rehabilitative programmes should be structured, and their effectiveness measured and monitored over time.

The majority of studies on LBP, in which gait and other activities of daily living (ADLs) are compared between LBP subjects and healthy controls, measure only the total joint moments and kinematics (Lamoth *et al.*, 2002, Lamoth *et al.*, 2004, Lamoth *et al.*, 2006a, Vogt *et al.*, 2001, Selles *et al.*, 2001, Huang *et al.*, 2011). A comparison of the passive-elastic component and passive stiffness of the hip joint found significant differences between LBP participants and healthy controls (Tafazzoli and Lamontagne, 1996). This difference in passive-elastic component is not in agreement with that of a similar study (Halbertsma *et al.*, 2001). The lack of agreement in findings, the paucity of research on the passive contributions, and the potential benefits to clinical practice of determining structural versus functional effects of LBP, all demonstrate a strong rationale for investigating this area further.

1.2 Thesis Purpose

The purpose of the current research project is to improve understanding of any biomechanical adaptations to LBP at the hip joint. Importantly, the research will focus on passive hip extensor contributions to total hip joint moments during walking, so that it is possible to determine the passive contributions to total moments.

By evaluating passive contributions to total hip moments, in subjects with or without LBP, it will be possible to better understand whether any adaptations are a property of the passive or active joint moments. This, in turn, will show whether adaptations are affecting structural materials, such as the muscle and/or tendon directly, or if there are changes in the motor control of muscles during walking. Such an understanding will help direct physical therapists in their interventions to help treat LBP, and demonstrate a means of measuring the effectiveness of those treatment programmes.

1.3 Proposed Research

Complexities in how to reliably and accurately measure passive and active contributions to movement have limited the scope of research into LBP. Improvements in techniques and modelling, coinciding with further research into movement-related factors, should help establish correlations and potentially casual factors and effective treatment protocols.

In order to investigate passive and active contributions of the hip extensors in LBP subjects, research studies will be developed to compare LBP subjects and healthy control subjects, incorporating the following criteria:

- A handheld force transducer will be adapted to accurately and precisely measure hip extensor moments during passive leg raising.
- Leg raising tests will be performed utilising a range of hip and knee angles.
- A biomechanical model will be adapted for the calculation of hip extensor passive moments, stiffness and strain energy.
- A predictive model will be developed to determine passive moments as a product of hip and knee angle.
- Gait analysis will be performed to determine total hip moments, power and mechanical work done.
- The predictive model will be integrated into the gait analysis, to calculate the magnitude of passive hip extensor moments to total hip moments.

Comparisons between LBP subjects and healthy controls will help elucidate the role and interactions of passive and active components of movement. It is anticipated that the findings of such research studies may be useful in determining how LBP influences passive and active components of movement. This research approach may subsequently be incorporated into other research studies of LBP, and help to improve clinical assessments, with prospective studies being important for improving understanding of the pathogenesis of LBP. Such findings may further be integrated into research of the most appropriate and effective interventions to prevent and treat LBP.

1.4 Thesis Outline

The thesis will comprise a literature review, methodology, and chapters relating to specific studies conducted during the progression of this research. The thesis ends with a general discussion and conclusions.

The first chapter of this thesis is the current introduction, in which a case is presented for the importance of understanding more about back pain, its study and the potential benefits of this.

Chapter two is the literature review. The purpose of the literature review is to introduce and critically analyse various topics of direct relevance to the overall research project.

Chapter three presents information on the design and development of a novel force measuring device for the measurement of passive joint moments. The chapter includes details regarding how the transducer was constructed, its components, and how each component was tested for reliability and validity.

Chapter four is a presentation of the first study conducted within this research project. The chapter introduces the specific need to measure and compare joint passive moments in LBP and healthy subjects, and for the subsequent integration of this data into dynamic joint assessments. The methods section details the subjects, procedures and equipment involved in the testing of passive joint moments. The results section provides information on the reliability of the test measurements, and comparisons between groups and sub-groups. The discussion places this information in the context of published reports on the same and similar topics.

Chapter five provides a technical account of how passive hip joint moments can be modelled from measurements with various hip and knee angles, to develop a predictive equation. The predictive equation can subsequently be used to calculate passive joint moments during activities of daily living, such as walking. The importance of calculating passive joint moments for individual subjects is discussed, in preference to estimates based upon population means of equation coefficients.

Chapter six develops the predictive equation into calculations of passive hip extensor joint moments during walking, and their contribution to total hip moments. The passive contribution to total moments is often considered negligible, and modelled accordingly. This chapter shows how the predictive equation integrates passive hip moments into walking assessments, and demonstrates the importance of doing so. The results include an assessment of passive contributions to total hip moments. The discussion places these findings in the context of related research, and promotes the need for considering passive contributions to total moments, and the potential for this approach in studies of dysfunctions, injuries and chronic pain, including low back pain.

Chapter seven demonstrates how the predictive equation for passive joint moments is integrated within a walking assessment of subjects with and without LBP. The findings help to elucidate whether adaptations to LBP are structural changes in the material properties, neural alterations in motor control, or neither. The conclusions of this chapter are therefore of relevance to our understanding of any adaptations of the hip joint to LBP.

Chapter eight is the general discussion and findings. The importance, relevance and usefulness of the research is discussed, together with recommendations for further research in this and related areas.

Chapter nine summarises the thesis and draws conclusions from the discussion chapter.

2. Literature Review

2.1 Introduction

Due to the high prevalence of LBP there is an urgent need to improve understanding of the occurrence, course, impact, risk factors and potential interventions for LBP patients worldwide (Buchbinder *et al.*, 2013). The purpose of this chapter is to evaluate what is currently understood regarding LBP, the related biomechanics, population differences, and to summarise the effectiveness of techniques to influence biomechanical factors. In doing so, it should become evident that research is needed to separate passive and active contributions to movement, which can subsequently be used by researchers and clinical therapists in their analysis and treatment of patients with LBP.

2.1.1 Low Back Pain across Life Course

In three best practice reviews of LBP (Hoy *et al.*, 2010, Axen and Leboeuf-Yde, 2013, Dunn *et al.*, 2013), the authors recommended that back pain be considered a long-term, or lifelong condition, rather than in the more traditional view of potentially recurring but single events. It was stated that individuals with LBP might experience it 'on or off' across many years, whereas those without LBP do not suddenly develop long-lasting pain, but rather a few acute bouts, perhaps becoming chronic over time (Hoy *et al.*, 2010, Axen and Leboeuf-Yde, 2013, Dunn *et al.*, 2013) .

LBP can be considered in terms of its two components: the long-term condition in which the sufferer is prone to LBP, and the acute episode (relapse or exacerbation) when the pain appears, reappears or worsens. It is plausible that the underlying mechanisms responsible for the long-term condition, may differ from the 'triggers' of the acute or exacerbated state (Axen and Leboeuf-Yde, 2013), although further research is required to establish such an hypothesis.

2.1.2 Prevalence

Out of a UK population of 14,680 individuals aged 25 years and older, the one-month prevalence of any back pain lasting at least a day was 63.1%, and for the lower back was 28.5% (Macfarlane *et al.*, 2012). Back pain peaked at 41-50 years, and was reported in 25% of individuals aged 80 years and over, with the severity of LBP increasing with age (Macfarlane *et al.*, 2012). LBP was different between genders, reported in 29.3% of females and 27.5% of males. Many of those reporting LBP also reported pain at other sites, including at the hip (32.0%), shoulder (24.8%) and knee (24.6%) (Macfarlane *et al.*, 2012). These results are approximately in agreement with two earlier, smaller surveys of the UK population, which reported prevalence of LBP as 39% (42% of females, 35% of males) for a study by Papageorgiou *et al*. (1995) , and 22.7% (24.5% of females, 21.3% of males) for a study by Webb *et al*., (2003).

In the survey of 4,515 UK-based individuals by Webb *et al*. (2003), 1,481 reported either back or neck pain. In agreement with the study by MacFarlane *et al*. (2012), the majority of individuals who suffered with back pain (74.8%) or

neck pain (88.7%), also reported pain at other sites. Of those reporting back pain, the most common other sites of pain were the knee, shoulder and neck. Back and neck pain were univariately associated with female gender, BMI indicating either overweight or underweight, increasing material deprivation and living alone (Webb *et al.*, 2003). However, following model adjustment, female gender only predicted neck pain, rather than back pain (Webb *et al.*, 2003).

Of those reporting back pain, 12.7% of women experienced intense back pain, 10.7% disabling back pain, 12.3% chronic back pain and 6.2% intense, disabling and chronic back pain. The figures for men were 9.4%, 7.3%, 10.5% and 3.9%, respectively (Webb et al., 2003). Women were 4.5 times more likely to experience disabling back or neck pain in those aged 75 and older, compared with women aged 16 to 44 years. Men were 3.5 times more likely at 75 and above, compared with the younger age group (Webb *et al.*, 2003).

MacFarlane *et al*. (2012), reported that older individuals with LBP were more likely to seek medical attention. Management of LBP by practitioners was found to be influenced by age, with older patients more likely to be prescribed analgesics, and less likely to be prescribed physiotherapy or exercise, or referred to a specialist. This may partly be explained by older LBP sufferers having received physical therapy previously, and, finding it was not helpful for their symptoms, became reluctant for such referrals (Macfarlane *et al.*, 2012).

2.1.3 Risk Factors

The pathogenesis of LBP is poorly understood. Nociceptive factors will be of greatest importance during acute phases of LBP, but the origin of pain has

proved complex to determine, and severity a product of causative factors in addition to anthropometrical (i.e. BMI), psychosocial and behavioural factors (Balague *et al.*, 2012).

Risk factors for LBP include degeneration of lumbar discs and narrowing of disc spaces, although these cannot necessarily be regarded as the specific, underlying cause of LBP (Balague *et al.*, 2012). Obesity has been positively associated with LBP, although postural and movement-related factors have not, such as occupational sitting, posture, standing and walking, manual handling, or movements such as pushing and pulling, bending and twisting (Balague *et al.*, 2012). LBP can only be attributed to a specific cause in 5-15% of cases, in which osteoporotic fracture, neoplasm or infection are regarded as responsible for the condition (Hoy *et al.*, 2010). There is a potential for LBP to arise from various anatomical structures, such as the bones, intervertebral discs, joints, ligaments, muscles, neural structures and blood vessels, but direct, causal associations remain rarely determined (Hoy *et al.*, 2010). Hence, LBP is considered non-specific in 85-95% of patients (Hoy *et al.*, 2010, Balague *et al.*, 2012).

2.1.4 Summary

Due to the high prevalence of LBP in those aged 41-50 years, and the increasing severity of LBP with age, it is important that optimal pharmacological and non-pharmacological interventions are determined and prescribed (Macfarlane *et al.*, 2012). Overall, there is a need to improve and develop better tools for prognostic analysis across the trajectory of chronic LBP, as well

as for testing and implementation of population-based interventions, potentially utilising novel technologies (van der Windt and Dunn, 2013).

The pathogenesis of LBP is poorly understood, and is only associated with a specific, predisposing condition in 5-15% of cases. Thus, the majority of chronic LBP is referred to as non-specific. As a consequence, there is a lack of consensus regarding risk factors and appropriate clinical assessments. Amongst potential biomechanical factors, there has been some interest in an association between hip extensor function and LBP, although the specific nature of any such interaction, and what might be cause or effect, remains to be elucidated.

2.2 The Hip Extensors and Low Back Pain

The functional capacity of the hip extensors is commonly assessed in relation to hip flexibility (joint range of motion, ROM) and extensibility (muscle lengthening), via knee extension angle, sacral angle, sit and reach test (Davis *et al.*, 2008), and variations of the straight leg raise test (SLR), whether active, passive, manual or instrumental (Davis *et al.*, 2008, Ylinen *et al.*, 2010). Findings from such clinical assessments are often extrapolated and used to evaluate conditions such as LBP (Ekedahl *et al.*, 2010), in addition to peripartum pelvic pain (Mens *et al.*, 1999), general hip function (Martin *et al.*, 2010a, Martin *et al.*, 2010b), and risk of sports injury in athletes (Scher *et al.*, 2010, Bradley and Portas, 2007, Verrall *et al.*, 2007).

Despite this, relationships between hip ROM and injury or dysfunction are poorly understood, with some investigators reporting no association

(Hennessey and Watson, 1993), and a lack of reliability of some tests (Ekedahl *et al.*, 2010, Ylinen *et al.*, 2010, Hunt *et al.*, 2001). Similar conclusions have been reported on the lack of reliability of back ROM testing for patients with LBP (Nattrass *et al.*, 1999, Nitschke *et al.*, 1999).

From the literature, it is unclear whether any reduced extensibility of the hip extensors is a cause or effect of LBP (Johnson and Thomas, 2010, Marshall *et al.*, 2009, Sjolie, 2004, Wong and Lee, 2004). In a study by Marshall *et al.* (2009), it was concluded that impaired stretch tolerance in patients with LBP was associated with mechanical muscular restrictions, rather than pain or fear-avoidance. Further, other investigators (Johnson and Thomas, 2010, Shum *et al.*, 2005a, Shum *et al.*, 2007a) have reported differences in performance measures in people with and without LBP, although underlying mechanisms and any associations between pain, function and active and passive muscle properties remain to be determined. There is a need to better understand hip-spine interaction in LBP, and, more specifically, to determine whether measured alterations are due to differences in the passive or active components of the related musculature.

It would be useful, initially, to establish any relationship between LBP and passive and active properties, and any pain- and function-based outcomes. It would then be useful to determine if any relationship is cause or consequence of pain. Future investigations may then explore what might be achieved by any intervention based upon physical therapy. In the absence of a better understanding of the effects of LBP on passive and active properties, at both the back and hip, it is problematic to recommend an appropriate treatment strategy.

2.2.1 The Functional Anatomy of the Hip Extensors

The muscles primarily responsible for extending the hip are the gluteus maximus, the hamstrings (i.e., the long head of the biceps femoris, semitendinosus and the semimembranosus)(figure 2.1), and the posterior head of the adductor magnus. The posterior fibres of the gluteus medius and anterior fibres of the adductor magnus are regarded as secondary extensors of the hip. With the hip flexed to at least approximately 70 degrees and beyond, most abductor muscles (with the possible exception of the pectineus) are capable of assisting with hip extension (Whittle *et al.*, 2012, Enoka, 2008, Neumann, 2010, Gray *et al.*, 2005, Gottschalk *et al.*, 1989).

The gluteus maximus has numerous proximal attachments from the posterior side of the ilium, sacrum, coccyx, sacrotuberous and posterior sacroiliac ligaments, and adjacent fascia. The muscle attaches into the iliotibial band of the fascia lata (along with the tensor fasciae latae)(figure 2.2) and the gluteal tuberosity on the femur. The hamstring muscles have their proximal attachment on the posterior side of the ischial tuberosity and attach distally to the tibia and fibula (Whittle *et al.*, 2012, Enoka, 2008, Neumann, 2010, Gray *et al.*, 2005, Gottschalk *et al.*, 1989).

Due to the hamstrings' attachments on the pelvis and below the knee, the hamstrings are responsible for both hip extension and knee flexion. Because of the locations of gluteus maximus attachment, this muscle helps stabilise the pelvis, externally rotates the hip, and extends the hip. During walking, the hamstrings are the primary hip extensors, with gluteus maximus activity increasing with greater knee flexion angles. The gluteus maximus is the

primary hip extensor during step climbing. In addition to the gluteus maximus, the gluteus medius and minimus are also responsible for stabilising the hip and pelvis during gait, with the gluteus medius mostly involved during initial contact and early stance phase, and gluteus minimus during the mid- and late-phases of the gait cycle (Whittle *et al.*, 2012, Enoka, 2008, Neumann, 2010, Gray *et al.*, 2005, Gottschalk *et al.*, 1989).

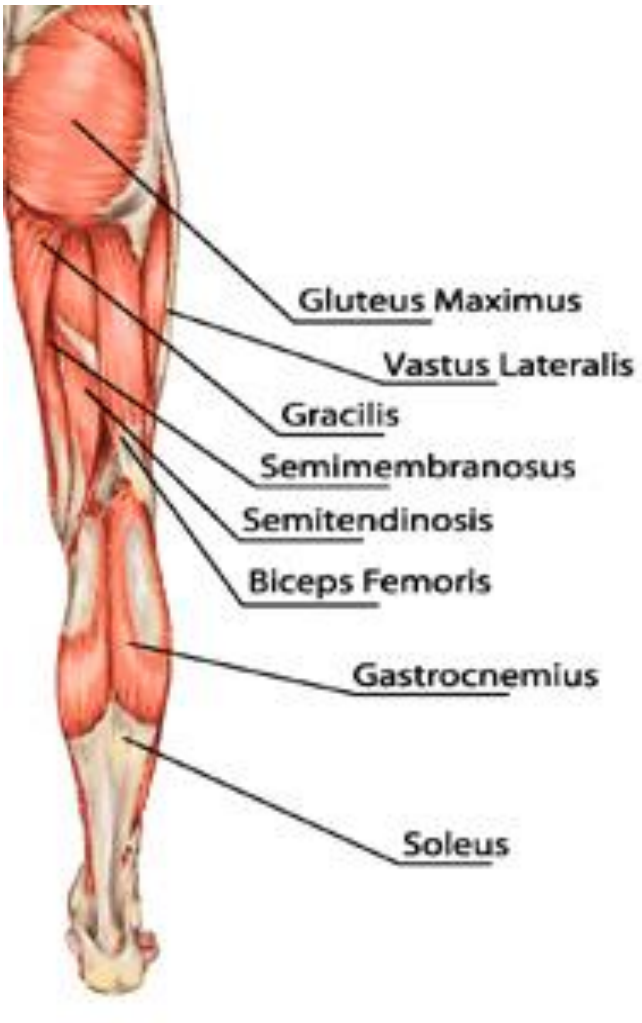

Figure 2.1. Anatomy of posterior hip and knee musculature

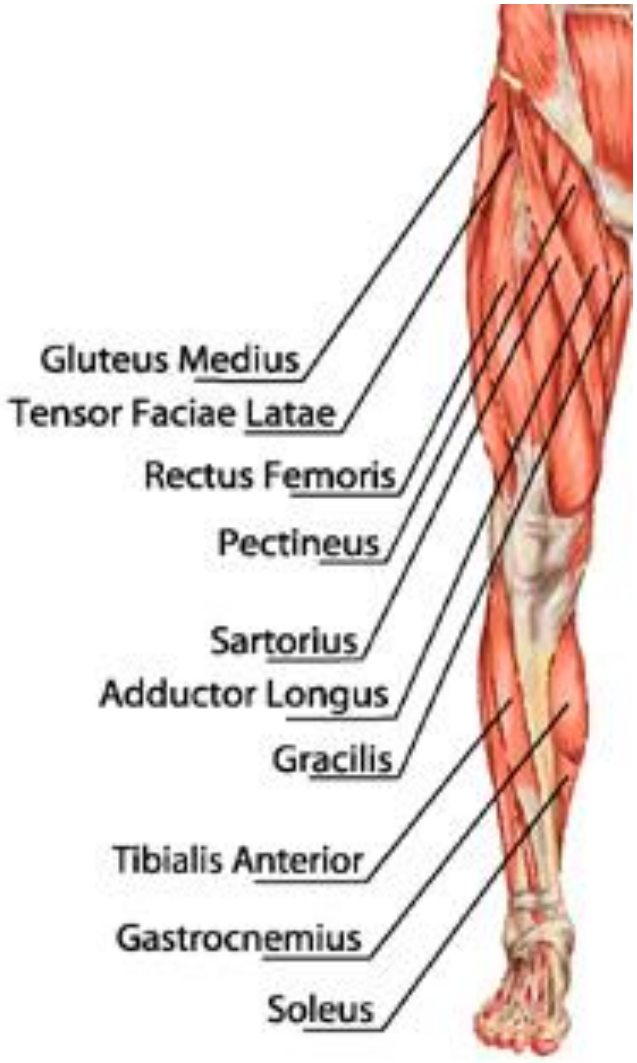

Figure 2.2. Anatomy of anterior hip and knee musculature

Bi-articular muscles are involved in the absorption and transfer of energy across neighbouring joints (Kaya *et al.*, 2005, Silder *et al.*, 2007), whereas uni-articular muscles act only at a single joint. Uni-articular structures also include non-contractile tissues, such as ligaments, the joint capsule and skin (Silder *et al.*, 2007). During gait, the bi-articular hamstrings transfer energy from the shank across the knee to the thigh and hip joint, whilst the gluteus maximus transfers energy from the thigh across the hip to the pelvis and sacro-iliac joint.

Thus, bi-articular coupling is an important component of movement, with active contractions pulling across two joints, and the passive resistance a product of the two joint angles (Silder *et al.*, 2007). A bi-articular muscle is able to absorb energy during a stretch from one joint, which is conserved and transferred for release at the neighbouring joint (Kaya *et al.*, 2005, Silder *et al.*, 2007). Such an absorption, storage and transfer of energy across two-joints is an important component of bi-articular muscles, which cannot occur with two uni-articular muscles (Silder *et al.*, 2007).

A complication in the biomechanical modelling of specific muscles involved in force production is that many muscles and other tissues act upon each joint (Yamaguchi *et al.*, 1995). Models require optimisation techniques to account for recruitment from multiple muscles with fibres within each muscle not involved, and a lack of involvement of specific fibre types (Dul *et al.*, 1984a, Dul *et al.*, 1984b, Kuo, 1994). This problem is known as the redundant problem in biomechanics (Yamaguchi *et al.*, 1995). Because of the redundant problem, it is more appropriate to refer to the actions of multiple muscles crossing a joint, rather than of individual muscles.

The central nervous system activates specific fibres across the available muscles to achieve a required movement (Dul *et al.*, 1984a, Dul *et al.*, 1984b, Ren *et al.*, 2007, Kuo, 1994) and maintain postural stability (Bunderson *et al.*, 2008). It is likely that the pattern of movement achieves an optimal bioenergetic efficiency for the speed and force required (Dul *et al.*, 1984a, Dul *et al.*, 1984b, Ren *et al.*, 2007, Kuo, 1994), whilst maintaining joint and postural stability (Bunderson *et al.*, 2008). Energy demands of movement will increase beyond forces required to move the weight of the limb, due to the effects of resisting

passive tissues and co-contractions (Dul *et al.*, 1984a, Dul *et al.*, 1984b, Kuo, 1994). The variety of hip muscles and muscle fibres available provide opportunities for the central nervous system to adjust recruitment patterns for the purpose of increasing hip and pelvic stability (Hungerford *et al.*, 2003, Pirouzi *et al.*, 2006, Souza and Powers, 2009), avoiding particular lines of stress on a joint (Hungerford *et al.*, 2003), or limiting actions that cause pain (Pierrynowski *et al.*, 2005, Park *et al.*, 2013, Dieterich *et al.*, 2016). Any such deviations may incur reductions in bioenergetic efficiency (Pirouzi *et al.*, 2006), and may transfer stress to other joint areas or other tissues (Hungerford *et al.*, 2003, Dieterich *et al.*, 2016).

During hip flexion, resistance is generated by the gluteus maximus and hamstrings muscle group, collectively regarded as the hip extensors, together with the resistive properties of the inferior and posterior hip capsule. When the knee is extended, the greatest resistance comes from the hamstrings, whereas as the knee becomes flexed it is the gluteus maximus that offers the most resistance (Whittle *et al.*, 2012, Enoka, 2008, Neumann, 2010, Gray *et al.*, 2005, Gottschalk *et al.*, 1989).

The hip flexors offer resistance to hip extension, and the muscles involved are the psoas major and rectus femoris, with additional resistance from the iliofemoral ligament and some fibres of the pubofemoral and ischiofemoral ligaments. It is the psoas major that provides the greatest resistance to hip extension when the knee is extended, and the rectus femoris when the knee is flexed (Whittle *et al.*, 2012, Enoka, 2008, Neumann, 2010, Gray *et al.*, 2005, Gottschalk *et al.*, 1989).

The pubofemoral ligament also provides resistance to hip abduction, together with the adductor muscles. Hip adduction is resisted by the superior fibres of the ischiofemoral ligament and iliotibial band, together with the tensor fasciae latae and gluteus medius, which comprise the abductor muscles. The ischiofemoral ligament also resists hip internal rotation, together with the piriformis and gluteus maximus, which comprise the hip external rotators. Hip external rotation is resisted by the iliofemoral and pubofemoral ligaments and the internal rotator muscles (tensor fasciae latae and gluteus minimus). During movement, the various hip muscles may work synergistically and antagonistically, whether to generate movement, oppose movement, or to stabilise the trunk, hip and pelvis (Whittle *et al.*, 2012, Enoka, 2008, Neumann, 2010, Gray *et al.*, 2005, Gottschalk *et al.*, 1989).

2.2.2 Hip-Spine Interaction during Simple Movements

In a study by Kadaba *et al.* (1989), the investigators assessed the relationship between the lumbar spine and hip during various trunk bending and twisting movements. It was reported that movements in sagittal and horizontal planes occurred in-phase, with frontal plane movements being initiated by the spine. The authors, therefore, concluded that clinical assessment of the back should include kinematic evaluation of both the lumbar spine and hips (Kadaba *et al.*, 1989).

A later study (Wong and Lee, 2004), reported approximately equal contributions of the lumbar spine and hips to forward bending of the trunk, and a greater contribution of the lumbar spine than hips in backward bending. Hip

flexion was lower in subjects with a lower straight leg raise (SLR) test, indicating a functional association between hip flexion during bending and limited flexion during the SLR test. Time to complete a cycle of forward and backward bending was more than twice that in LBP and limited SLR subjects, compared with asymptomatic subjects (Wong and Lee, 2004).

Other investigators have reported reduced hip range of motion (ROM) during passive straight leg raising (PSLR) in subjects with chronic, but not recurring LBP (Hultman *et al.*, 1992). Radwan *et al.* (2014) reported significantly lower hamstring extensibility in subjects with LBP than healthy controls, and considerable differences between left and right legs. Those investigators (Radwan *et al.*, 2014) further reported that severity of LBP was inversely related to hamstring extensibility.

Reduced flexibility of the hip flexors and hip extensors during adolescence are both predictors of LBP in adulthood (Feldman *et al.*, 2000). Assessments of thoracic ROM, and therefore any relationship between thoracic, lumbar and hip mobility, were not included in these investigations. The thoracic spine is considered to contribute only minimally to sagittal-plane movement (Horton *et al.*, 2005, Morita *et al.*, 2014). However, it would be interesting to understand if any reduced thoracic mobility required greater compensatory mobility at the hips and/or lumbar spine.

Although PSLR is often reported to be reduced in subjects with LBP when compared with healthy controls, this may not be due to lengthening differences in the hip extensors. Kellis *et al.* (2015) assessed PSLR and hamstring extensibility using ultrasound, and reported significantly reduced PSLR but no difference in MTU elongation. It was hypothesised (Kellis *et al.*,

2015) that the reduced PSLR may therefore be the result of reduced stretch tolerance due to sciatic nerve-root or ligamentous damage, or a stretch-reflex contraction of the hamstrings due to pain avoidance. However, Kellis *et al.* (2015) only measured a portion of the semitendinosus MTU, and did not assess the whole muscle, nor did they assess the biceps femoris or semimembranosus. The long head of the biceps femoris is the bi-articular hamstring muscle, with proximal attachments to the lower back and sacro-iliac (S.I.) joint, via the sacrotuberus ligament, so is potentially more relevant to the assessment of PSLR and LBP (Kellis *et al.*, 2015).

During extension of the spine from a forward bending position, the movement is initiated at the hips, with the lumbar spine contributing from the midrange of the movement (McClure *et al.*, 1997). It was noted in a study by McClure *et al.* (1997) that some investigators neglect to measure pelvic position and movement during assessment of the hip and lumbar spine, leading to underestimates of the amount of hip joint motion. In that study (McClure *et al.*, 1997), asymptomatic subjects with a history of LBP were found to move more from the lumbar spine than control subjects without a history of LBP (McClure *et al.*, 1997). This was attributed to earlier muscle activation in the lumbar spine, perhaps to compensate for deficiencies in the lumbar spine or hip extensors. PSLR tests demonstrated reduced hip flexion ROM in the group with previous LBP (McClure *et al.*, 1997).

Similar findings have been reported by other investigators (Paquet *et al.*, 1994). In a study by Paquet *et al.* (1994) it was found that the erector spinae muscles in a subgroup of LBP subjects remained active during trunk flexion. The subgroup had experienced LBP for a longer duration than the other LBP

subjects, so activation and movement anomalies may be associated with duration of chronic pain. The increased muscle activity in the lumbar spine is in agreement with the findings of McClure *et al.* (1997), for asymptomatic subjects with a history of chronic LBP. The results of these studies (McClure *et al.*, 1997, Paquet *et al.*, 1994), show that increased activation of lumbar spine muscles is associated with altered hip-spine interaction during trunk flexion and extension.

In the study by Wong and Lee (2004), it was reported that side bending of the lumbar spine was associated with ipsilateral hip abduction and contralateral hip adduction. This finding is in agreement with a study by Lamoth *et al.* (2002). However, it was found that frontal plane hip mobility was not associated with either LBP or SLR, whereas lumbar ROM was. Hence the ratio of lumbar spine to hip mobility was considerably reduced in LBP, and in LBP with reduced SLR groups (Wong and Lee, 2004). During twisting movements of the spine, the ipsilateral hip rotated medially and the contralateral hip rotated laterally, in agreement with the study by Lamoth *et al.* (2002). The hips contributed to back rotation to a greater extent than the lumbar spine, and there was greater variability in the relative contributions in those with LBP (Wong and Lee, 2004). This may be indicative of individual movement restrictions and compensations in the LBP group.

During a passive assessment of trunk ROM and passive resistance (Gombatto *et al.*, 2008b), greater asymmetry in lateral bending was reported for passive strain energy in LBP compared with pain-free subjects. There was no difference between groups in lumbar ROM (Gombatto *et al.*, 2008b). In this study, subjects were positioned in a prone lying position, ensuring the pelvis

was secured so as to not contribute to the overall movement. However, this increase in movement control by the investigators makes their observations less transferable to function-based tasks than the standing assessment of Wong and Lee (2004).

An investigation by Leinonen *et al.* (2000) found altered muscle activation timings between LBP subjects and healthy controls. The investigators assessed muscle activity during trunk flexion and extension, and reported that, at the beginning of flexion, the erector spinae were activated significantly prior to the gluteus maximus, whereas no such difference occurred in control subjects. At the end of the flexion movement, the erector spinae relaxed before the gluteus maximus and biceps femoris in controls, whereas in LBP subjects the erector spinae relaxed at the same time as gluteus maximus, and before biceps femoris (Leinonen *et al.*, 2000).

The same investigators (Leinonen *et al.*, 2000), reported that, at the beginning of lumbar extension, control subjects activated their biceps femoris prior to gluteus maximus and erector spinae, whereas in LBP subjects the biceps femoris and gluteus maximus were activated together. At the end of extension, the biceps femoris relaxed before the gluteus maximus and erector spinae in control subjects, whereas in LBP subjects the biceps femoris and gluteus maximus relaxed together, both ahead of the erector spinae (Leinonen *et al.*, 2000). Overall, the muscle activation occurred sequentially in healthy subjects, beginning with the biceps femoris and proceeding to gluteus maximus and erector spinae, for both trunk flexion and extension. Muscle relaxation followed the reverse order in control subjects (Leinonen *et al.*, 2000).

The altered activity of the gluteus maximus in LBP subjects was reported during both trunk flexion and extension (Leinonen *et al.*, 2000). The biceps femoris, gluteus maximus and erector spinae are activated synergistically to transfer loading during trunk flexion and extension, and hip extension (biceps femoris and gluteus maximus). LBP appears to influence the timing of activation of the hip extensors during trunk flexion and extension (Leinonen *et al.*, 2000) and they might also be influenced during hip extension.

The current research demonstrates a functional link between the hips and lumbar spine, and how limitations in hip passive and active mobility can influence mobility of the spine. LBP influences activation of the hip extensors, and the relative contributions of the biceps femoris and gluteus maximus. To what extent this occurs in different activities of daily living (ADLs) is poorly understood. Whilst the individual muscle forces may be different as a result of these alterations, whether passive, active and net joint moments are affected remains to be determined. Hence, it would be interesting to better understand the effect of LBP on other functional and occupational tasks, including gait and other ADLs.

2.2.3 Functional Tasks in Low Back Pain

In an assessment of sit-to-stand and stand-to-sit, in participants with or without LBP (Shum *et al.*, 2005a), a reduction in joint mobility and velocities was observed in LBP subjects, at both the lumbar spine and hip. Further, the contribution of the lumbar spine to the movement, as a ratio to that of the hip, was reduced in subjects with LBP, and this altered hip-spine coordination was

greatest in LBP subjects with positive SLR test. The authors (Shum *et al.*, 2005a), concluded this was a compensatory response to limit pain during movement. In a later study by the same investigators (Shum *et al.*, 2007b), the same tests were used to show that moments and powers at the lumbar spine and hips were altered in subjects with LBP. The differences were greatest in those with a positive SLR test. Again, the authors concluded that the alterations were a means to limit pain in susceptible tissues (Shum *et al.*, 2007b).

In a third investigation of sit-to-stand and stand-to-sit in LBP subjects (Shum *et al.*, 2009), transfer of energy was demonstrated from the pelvis to the thigh, then to the leg segments. It was noted that this energy transfer was accomplished by predominantly passive mechanisms. The investigators (Shum *et al.*, 2009) also reported that, although the power of the lower limb segments was decreased, the total mechanical work done at each of these segments increased. It was also found that the task was more energy-demanding in individuals with LBP, with or without a positive SLR test. As a consequence, the investigators (Shum *et al.*, 2009), recommended that treatments should be developed to restore a more normal, energy-efficient movement pattern.

Shum *et al.* (2005b) also investigated kinematics and joint coordination of the lumbar spine and hips, in LBP subjects putting on a sock. As with a previous study of sit-to-stand and stand-to-sit (Shum *et al.*, 2005a), it was observed that LBP subjects exhibited reduced joint range and velocity of the lumbar spine and hip, with a reduced contribution of the lumbar spine, relative to that of the hip (Shum *et al.*, 2005a, Shum *et al.*, 2005b). Lumbar spine-to-hip coordination was altered in LBP subjects, and this effect was exacerbated in individuals with a positive SLR test. It was reported that LBP subjects utilised

individual-specific strategies to compensate for limitations in lumbar spine and hip motions (Shum *et al.*, 2005b). Similarly, a study of lumbar and hip angular displacement during squatting (Sung, 2013), reported decreased lumbar flexion and increased hip flexion in subjects with LBP compared with controls. Thus, these findings suggest compensatory interactions between the lumbar spine and hips in individuals with LBP.

A study by Johnson and Thomas (2010), found no relationship between hamstring extensibility and the amount of hip and lumbar spine joint excursions during standardised reaching and forward bending tasks, in individuals with or without LBP. Further, there was no relationship between LBP and hamstring extensibility, leading the investigators to question the efficacy of hip flexibility training in individuals with LBP (Johnson and Thomas, 2010). As the assessment was performed passively, it would be inappropriate to rule-out the possibility that active determinants of hamstring stiffness could be involved. As any relationship between active stiffness and passive extensibility remains to be determined, the conclusion that flexibility training needs to be re-evaluated remains consistent with the available research.

In an assessment of a bending task to pick up an object, it was found that individuals with LBP exhibited limited range and velocity of segments of the lumbar spine and hips (Shum *et al.*, 2007a). Coordination of the lumbar-spine to hips was also affected by LBP, with restrictions in sagittal motion being compensated for by increased movement of the lumbar spine and hips in frontal and horizontal planes. In individuals with a positive SLR, additional limitations and compensatory movement patterns were observed (Shum *et al.*, 2007a). This interaction of the lumbar spine and hips, including specific compensation

patterns in different planes of motion, indicates a need for an individualised approach to any physical therapy and rehabilitation. Although this investigation assessed active functional movements, no measurements were made of passive structures, making it impossible to determine whether adaptations to pain were in passive or active components, or both.

Differences in muscle activation of the hip extensors and erector spinae have also been reported in subjects who develop LBP when standing, but who are not otherwise symptomatic (Nelson-Wong *et al.*, 2012). In these subjects, increased activation of erector spinae occurred prior to activation of gluteus maximus, during trunk extension from a flexed position (Nelson-Wong *et al.*, 2012). It was hypothesised that alterations in muscle activation patterns may be a useful tool for predicting the development of LBP. However, what might cause such alterations in muscle activity has not been explored. Whether the alterations might be secondary to structural anomalies, previous injury, or some other influence, ought to be investigated.

A study by Pirouzi *et al.* (2006), reported increased hamstring activation during standing trunk rotation exercises, in LBP subjects compared with healthy controls. Altered timing and increased muscle activity were reported for all muscle tests (internal and external obliques, external latissimus dorsi, erector spinae, gluteus maximus and hamstrings), and were most noticeable in the hamstrings and gluteus maximus, and in antagonist muscles during rotation (Pirouzi *et al.*, 2006).

Although there is interest in how work, lifestyle and postural factors might be involved in LBP (Arab and Nourbakhsh, 2014), these have not been found to be related to hip ROM (Arab and Nourbakhsh, 2014). It may be that structural

components of muscles are not related to LBP caused by work, lifestyle or postural factors. Various ADLs produce symptoms in individuals with LBP. These include activities such as forward reaching (Shum *et al.*, 2007a), forward bending (McClure *et al.*, 1997, Shum *et al.*, 2010), and returning to an upright position from a flexed position (McClure *et al.*, 1997).

2.2.4 Hip-Spine Interaction during Walking

The main role of the hip flexors and extensors is to support the body against gravity, whilst the hip flexors are secondarily recruited for propulsion (Sadeghi *et al.*, 2001). During gait in healthy individuals, peak hip extensor and flexor moments average a mean (SD) value of approximately 0.77 (0.26) Nm/kg (at 5% of gait cycle) and 0.62 (0.2) Nm/kg (at 48% of gait cycle), respectively (Sadeghi *et al.*, 2001). Peak angular hip displacement reaches approximately 35 degrees in extension and 28 degrees of flexion (Sadeghi *et al.*, 2001).

In a study by Bedard *et al.* (2013), LBP subjects and healthy controls engaged in 20 minutes of treadmill walking. There were no significant differences in active hamstring stiffness between groups before or immediately after exercise. However, between 48-72 hours post-exercise, LBP subjects exhibited significantly greater active hamstring stiffness than control subjects (Bedard *et al.*, 2013).

The biceps femoris is active during mid-swing to initial foot contact, and then begins to relax as gluteus maximus activity increases to stabilise the pelvis and sacro-iliac S.I. joint (Hossain and Nokes, 2005). It has been hypothesised that a contraction anomaly in gluteus maximus is responsible for LBP through

an increased need to stabilise the S.I. joint (Hossain and Nokes, 2005). However, it is unreasonable to conclude such a cause-effect relationship in all individuals with LBP.

There is a clear relationship between the hip extensors, S.I. joint and lumbar spine musculature during walking. The biomechanics of hip-spine interaction may be altered in LBP, although underlying mechanisms remain to be elucidated. Identifying and rectifying anomalies may be an essential component of the clinical assessment and treatment of LBP.

2.2.5 Hip-Spine Alterations in Muscular Endurance

Muscular endurance refers to a muscle's ability to use aerobic energy to produce a high volume of work with low fatigability. Walking, stair climbing and other repetitious movements rely upon muscular endurance. McKeon *et al.* (2006) reported differences in biceps femoris, gluteus maximus and erector spinae recruitment in LBP subjects compared with healthy controls, associated with the performance of an erector spinae endurance test. McKeon *et al.* (2006) reported that LBP subjects with the lowest erector spinae endurance had the earliest onset on LBP. However, there were no differences in localised blood volume or oxygen saturation. This suggests that any differences are likely to be muscular or neuromuscular in origin, rather than due to alterations in cardiovascular efficiency.

In a study of erector spinae muscle fibre type distribution in subjects with and without LBP (Mannion *et al.*, 1997), it was found that LBP subjects had a greater proportion of type IIC and IIB fibres, and fewer type I fibres, than healthy

controls. Samples were obtained during surgery in LBP subjects, and via a muscle biopsy in control subjects. Only the lumbar paraspinal muscles were assessed, although it would have been of interest to assess fibre type distribution of the biceps femoris, gluteus maximus and other hip extensor muscles.

The study by Mannion *et al.* (1997), demonstrates that erector spinae fibres of LBP subjects are more glycolytic, and therefore less resistant to fatigue, than the erector spinae of healthy controls. This is in agreement with the studies that have reported increased fatigability of erector spinae, and increased activation of biceps femoris and gluteus maximus, in LBP subjects compared to controls (McKeon *et al.*, 2006).

It is possible that the higher distribution of fast-twitch, type II fibres, is beneficial for controlling the lumbar spine and resisting short-term stress, such as might occur during unexpected perturbations, lifting, or uncommon movements. This leads to the increased activation of biceps femoris and gluteus maximus to compensate during common ADLs such as gait and muscle endurance tests. Thus, there is a potential for LBP to lead to a change in erector spinae fibre type distribution, which leads to increased activation of biceps femoris and gluteus maximus as a compensation during ADLs and endurance tests. It would therefore be useful to perform similar assessments of biceps femoris and gluteus maximus fibre distribution, and to more fully assess muscle activation during short-term stresses and specific ADLs.

Another study (Bernard *et al.*, 2008) has reported reduced endurance strength of the trunk muscles and hip extensors in those with chronic LBP. However, a study by Kankaanpaa *et al.* (1998), found similar endurance of

lumbar spine muscles in LBP subjects and healthy controls, but reduced endurance (increased fatigability) in the gluteus maximus in those with LBP. However, it is not possible to determine whether muscular imbalances and weakness are causes or effects of LBP, or whether any relationship is direct or the result of reduced physical activity habits.

An assessment of collegiate athletes (Nadler *et al.*, 2001) reported that differences in strength between left and right hip extensors predicted LBP treatment within a year, in previously pain-free females. However, those investigators (Nadler *et al.*, 2001), found no relationship between left and right hip abductors in males or females, and no effect of hip extensors in males. Thus, there may be gender differences in the mechanisms responsible for LBP.

It seems likely that this reduced muscle endurance is therefore an effect of LBP, with duration of LBP being inversely related to erector spinae muscle performance. Considering the compensatory muscle activation already reported in LBP subjects (Leinonen *et al.*, 2000), and the findings of the study by McKeon *et al.* (2006), it may be that the biceps femoris and gluteus maximus increase activity to reduce activation of the erector spinae, and thereby limit stress on the lower back.

2.2.6 Summary

In the absence of a better understanding of the effects of LBP on passive and active properties, at both the back and hip, it is problematic to recommend an appropriate treatment strategy. It would be useful, initially, to establish any relationship between LBP and passive and active properties, and any pain- and

function-based outcomes. It would then be useful to determine if any relationship is cause or consequence of pain, and what might be achieved by any intervention based upon physical therapy.

2.3 Clinical Assessments

Despite evidence demonstrating potential benefits of assessing passive hip extensor stiffness and joint moment-angle characteristics (Johnson and Thomas, 2010, Shum *et al.*, 2005a, Shum *et al.*, 2007a), such assessments have not yet been widely incorporated into clinical practice. Muscle passive stiffness can be calculated from measurements of passive joint moments, from which an estimation of other resistive materials (ligaments, skin, capsule, other connective tissue), can be taken into account (Han *et al.*, 2012).

Clinical tests may commonly be incorporated into assessments of LBP patients, with the intention of gaining an improved understanding of any individual factors contributing to the condition. However, there is a lack of consensus regarding the most appropriate and efficient tests to conduct, and how the results of such tests should be interpreted (Aberger *et al.*, 1987, Nelson-Wong *et al.*, 2009, Hu *et al.*, 2014, Truter *et al.*, 2014). This is particularly relevant where it is not possible to distinguish between positive test results for cause and effect.

An investigation of lumbar spine ROM and functional ability (Parks *et al.*, 2003), in individuals with LBP, found no significant correlation between ROM and functional test scores. The authors of that study concluded that clinicians should be sceptical regarding the use of flexibility assessments, and instead

focus on functional, occupation-based limitations and their correction (Parks *et al.*, 2003). However, their finding is not in agreement with that of Tafazzoli and Lamontagne (1996) who reported reduced trunk forward flexion in individuals with LBP, compared with healthy participants. Sjolie (2004) reported reduced hip flexibility of adolescents with LBP compared with healthy controls. Sjolie (2004) also reported an association between increased BMI and LBP. Whether the correlations might be a contributor to, or consequence of, LBP was not investigated. Different methodologies may be responsible for different findings in these studies. If ROM and the ability to perform activities of daily living are not directly related, this questions the relevance of ROM testing amongst researchers and clinicians. Further, this illustrates a need for researchers and clinicians to use alternative assessments of muscle function and functional capacity, for which direct passive and active assessments may be useful.

2.4 Joint Mechanical Properties

It has been suggested that associations between joint flexibility and injury are best interpreted in terms of resistance to stretch (Wilson *et al.*, 1991), with injury risk increasing with too little or too much muscle stiffness (Butler *et al.*, 2003). Further, it has been found that muscle elastic properties are affected by neuromuscular disorders (Cooney *et al.*, 2006), back pain (Hamill *et al.*, 2009, Marshall *et al.*, 2009, Gombatto *et al.*, 2008b), gender (Marshall *et al.*, 2009, Blackburn *et al.*, 2004a, Blackburn *et al.*, 2009a), physical activity (McNair and Stanley, 1996), strength (Marshall *et al.*, 2009) and the length of the limb(s) comprising the moment arm (Nagano and Komura, 2003).

Elastic properties can be either passive or active, with some investigators reporting musculotendinous extensibility being moderately related to passive stiffness, and weakly related to active stiffness (Blackburn *et al.*, 2004a). Furthermore, increased extensibility is not necessarily associated with reduced muscular stiffness (Blackburn *et al.*, 2004a), although findings are conflicting (Marshall *et al.*, 2009). An investigation by Halbertsma *et al.* (2001), reported an association between hamstring extensibility and LBP, but no differences in passive stiffness between patients and controls. Overall, there is a lack of agreement in the literature as to whether or not passive muscle stiffness is related to joint ROM and muscle extensibility (Blackburn *et al.*, 2004a, Tafazzoli and Lamontagne, 1996).

2.4.1 Nomenclature

Extensibility can be defined as the lengthening capacity of the musculotendinous units (MTUs) comprising a muscle or muscle group, and does not take into account resistive forces (Gleim and McHugh, 1997). Extensibility is commonly assessed using ultrasound assessment of muscle fascicles, or cadaveric specimens. Flexibility refers to a joint's ROM, typically measured using passive or active ROM assessments.

A muscle's stiffness is influenced by the forces required to lengthen the MTU, and is calculated by the following equation (Blackburn *et al.*, 2004a):

$$Stiffness = \frac{\Delta\, force}{\Delta\, length}$$

This indicates that stiffness is a product of both the elastic properties of the MTU, and its lengthening (extensibility) characteristics (Blackburn *et al.*, 2004a). About a joint, stiffness is measured in newtonmetres per degree (Nm/degree). The reciprocal of this equation yields the muscle's compliance (Magnusson *et al.*, 2003):

$$Compliance = \frac{\Delta\ length}{\Delta\ force}$$

2.4.2 Passive Stiffness

At rest, resistance to muscle lengthening is due to passive components, whereas in the active state it is a combination of both passive and active stiffness. Passive muscle stiffness is a product of the parallel elastic tissues, such as the sarcolemma, endomysium, perimysium and epimysium, and of the series elastic tissues, from the tendon, myofibrillar cross-bridges (Marshall *et al.*, 2009, Blackburn *et al.*, 2004a, McNair and Stanley, 1996) and passive structural elements such as titin (Rassier, 2012). The functions of the parallel and series elastic components are to absorb, store and transmit energy (Marshall *et al.*, 2009).

Because the total passive resistance of a joint to rotation is partially determined by the elongation characteristics of soft tissues other than the MTU, such as ligaments and skin, as well as capsular properties, it is not currently possible to measure *in vivo* muscle stiffness directly (Blackburn *et al.*, 2011). Instead, elastic forces of the MTU can be estimated following measurement of

passive joint moments, by incorporating estimations of the combined forces of other associated elastic materials.

The contributions of tendon and muscle fascicle to total MTU passive resistance is dependent upon architectural factors, including relative muscle and tendon lengths (Abellaneda *et al.*, 2009, Hoang *et al.*, 2007), overall distribution of series and parallel fibres (Hoang *et al.*, 2007, Epstein *et al.*, 2006) and fascicle pennation angle (Abellaneda *et al.*, 2009). In a study by Hoang *et al.* (2007), it was found that, because the Achilles tendon is longer than the gastrocnemius muscle fascicles, the tendon contributes the majority of the total passive compliance of the MTU. Further, tension is not uniformly distributed between the aponeurosis, extramuscular tendon and muscle fascicles, making simplistic models or interpretation of the MTU potentially inaccurate and misleading (Epstein *et al.*, 2006).

Morgan (1977) reported that both muscle tension and tendon stiffness are proportional to the number of MTUs. At maximum muscle tension, the tendon compliance is approximately equal to the compliance of the cross-bridge array for all cat soleus muscles, even over a wide range of tension. Further, the maximum exerted muscle tension was directly related to tendon stiffness (and so inversely related to tendon compliance). Hence, a stronger muscle can be expected to have a stiffer tendon. This is because a stronger muscle has more parallel muscle fibres, and each fibre bundle has the same length of tendon in series with it (relative to the overall length of the MTU) (Morgan, 1977).

2.4.3 Contribution of Joint Capsule and other Factors to Joint Resistance

In some studies (Foure *et al.*, 2011, Herda *et al.*, 2011, Halbertsma and Goeken, 1994, Halbertsma *et al.*, 1996, Halbertsma *et al.*, 1999), total joint passive stiffness is assumed to be the same as total musculotendinous stiffness, and does not take into account other contributing materials, such as the joint capsule, ligaments and skin. Clothing is not referred to in these studies, and it may be possible that this contributes to total stiffness measurements. Where measurements are not recorded through a full ROM, investigators must then fit their available data to nonlinear models to develop reasonable estimates of stiffness throughout range.

In addition to the MTU, ligaments, skin and other connective tissue, and the joint capsule itself, will all contribute to passive joint stiffness. Following injury to a joint, there are a number of elements that may increase joint capsule stiffness, such as adhesions, contractions and scar tissue formation within the capsule and ligaments, or an increase in myofibroblasts (Mattyasovszky *et al.*, 2010).

An increase in myofibroblasts can be due to mechanical stress, and is associated with fibroconnective disorders including Dupuytren contracture, carpal tunnel syndrome and frozen shoulder. Activation and differentiation of myofibroblasts are controlled by tissue-specific growth factors and cytokines (Mattyasovszky *et al.*, 2010).

An investigation by Mattyasovszky *et al.* (2010), reported that the function and proliferation of myofibroblasts could be affected by immunomodulatory treatments. This indicates modulation of the joints' healing

processes, and is of clear significance to our understanding of joint health. Further, by quantifying the effects of injuries, disease and dysfunction on joint health, and the contribution of joint capsule stiffness to total stiffness, it may be possible to improve physical rehabilitation and nutritional recommendations.

2.4.4 Passive Force Enhancement

The giant protein titin (also known as connectin), spans the half sarcomeres, connecting the thick filament and z-lines of striated muscle fibres (Rassier and Herzog, 2005, Tskhovrebova *et al.*, 2005). Titin is responsible for the resting length and passive elasticity of sarcomeres (Rassier and Herzog, 2005, Tskhovrebova *et al.*, 2005). Titin comprises proline-glutamate-valine-lysine (PEVK)-rich domains, and immunoglobulin-like (Ig) domains (Rassier and Herzog, 2005). When a muscle is stretched, the titin molecule unfolds (Rassier and Herzog, 2005). Different muscle types exhibit differences in resting length and passive elasticity of their sarcomeres, which is related to different titin isoforms (shorter and stiffer isoforms in cardiac muscle, and longer and more compliant isoforms in longer and more compliant sarcomeres) (Tskhovrebova *et al.*, 2005).

Rassier and Herzog (2005), have demonstrated an increase in muscle passive force when activated muscles are stretched, which differs to the forces found during passive stretches or isometric contractions. This passive force enhancement is thought to be related to an increase in titin stiffness, following increased intramuscular calcium binding with titin (due to muscle activation or stretch). The effect requires active stretch, rather than activation or passive

stretch alone (Joumaa *et al.*, 2008b, Rassier and Herzog, 2005) The passive force enhancement was found to be length dependent, as it increased with increasing sarcomere lengths (Rassier and Herzog, 2005). However, a more recent study (Joumaa *et al.*, 2008b) did find a relationship between increased titin binding with calcium and increased stiffness during passive stretching. Uptake of calcium was found to be responsible for 25% of the increased stiffness, with the remaining 75% thought to be related to the cross-bridges, during passive, rather than active, stretch (Joumaa *et al.*, 2008b).

The conclusion that the titin protein is responsible for this passive force enhancement, rather than cross-bridges, has been supported by various investigators (Bagni *et al.*, 1994, Bagni *et al.*, 2002, Campbell and Moss, 2002, Joumaa *et al.*, 2008a). Such an effect would occur in synergy with tendon and aponeurosis, to promote biomechanical efficiency during cyclic movements such as gait. Joumaa *et al.* (2008b), also reported that the passive force enhancement must occur with either cross-bridge formation or active force production for full manifestation. This supports the hypothesis that the passive force enhancement occurs in synergy with other processes to promote efficiency of movement.

Force enhancement is associated with an increased passive stiffness as activated muscle stretch continues, with passive force increasing proportionally to the amount of stretch (Rassier and Herzog, 2005, Joumaa *et al.*, 2008b). Passive joint moments increase with joint angle in an exponential manner (Lee and Munn, 2000). At neutral joint angles, and at angles approaching close to neutral, the passive stiffness is close to zero, and the short range over which this occurs is referred to as the MTUs' slack length (Hoang *et al.*, 2007).

With joint kinematics during ADLs not necessarily causing stretch of activated muscles far beyond their slack length, the relationship between force enhancement and net passive stiffness is poorly understood. Further research is required to determine any relationships between sarcomere lengths, titin lengths and joint angles, during functional tasks at which passive force enhancement makes a significant contribution to the passive moments and overall movement efficiency. The relationship between titin molecules and whole muscle passive and active properties remain to be elucidated (Tskhovrebova *et al.*, 2005).

2.4.5 Active Stiffness

During activity, total resistance to movement results from both passive and active tissues. The active component is derived from the actin and myosin cross-bridge myofilaments, within the active myofibrils during muscle contraction (Marshall *et al.*, 2009). Thus, active musculotendinous stiffness is an important component of the total resistance at a joint. The contribution of the active component will be governed by the number of parallel cross-bridges and the magnitude of activation (Blackburn *et al.*, 2004a). Active resistance to movement may also be increased due to the spinal stretch reflex (Blackburn *et al.*, 2004a).

Hamstring musculotendinous stiffness has been positively correlated with tendon stiffness, muscle cross sectional area, fascicle length and strength (Blackburn and Pamukoff, 2014). In a study by Blackburn *et al.* (2004b), females were shown to have lower active knee flexor stiffness compared with

males. Due to the relationship between active stiffness and cross-bridge formation, and similar loading conditions in male and female subjects, it was considered that this finding demonstrated that the males may have a greater capacity to resist muscle lengthening due to increased muscle mass (Blackburn *et al.*, 2004b). Of the variables influencing stiffness, only strength and tendon stiffness were found to differ with gender following normalisation to body mass (Blackburn and Pamukoff, 2014).

Due to active stiffness being a property of muscle activity and the number of myofilament cross-bridges, active stiffness should be considered a modifiable property of the neuromuscular system (Blackburn *et al.*, 2009a). This may have practical relevance in designing physical therapy interventions to reduce joint-stability-related injury risk (Blackburn *et al.*, 2009a, Blackburn *et al.*, 2008). For example, greater active hamstring stiffness has been associated with reduced anterior tibial translation, suggesting that this increased stiffness may protect against anterior cruciate ligament (ACL) injuries (Blackburn *et al.*, 2009a). Conversely, lower hamstring stiffness may predispose to ACL injuries, indicating a need to increase active stiffness through interventions designed to increase hamstring activation and muscle hypertrophy (Blackburn *et al.*, 2009a).

2.4.6 Interaction of Passive and Active Components

Total joint moments are a product of both active and passive components (Marshall *et al.*, 2009, Yoon and Mansour, 1982). Although total moments can be calculated at any joint during any activity, effective modelling of the relative

contributions of active and passive components requires further investigation (Yoon and Mansour, 1982).

In walking, passive stiffness of the hip flexors can reduce the need for active hip flexor power generation (Whittington *et al.*, 2008). It is reasonable to conclude that active and passive components interact to promote bioenergetic efficiency during normal activities. An unusual increase in a passive component could require an alteration in the active component to achieve a normal total amount of stiffness. Alternatively, a change in kinematics, such as reduced hip extension, might preserve the same passive contribution in an unusually stiff hip flexor, as would otherwise be achieved with a normal hip flexor and normal degree of hip extension (Whittington *et al.*, 2008).

Due to the paucity of research into the modelling of passive and active contributions to total joint moments and stiffness, it is unclear how these components interact. Some investigators assume that passive and active components are additive (Whittington *et al.*, 2008). Although this assumption may be reasonable during static tests, the interaction of passive and active components during dynamic tasks is more complicated.

Regarding joint stability and injury risk, reduced stiffness about a joint may predispose to ligamentous injury. Passive stiffness acts as a baseline, above which active muscle contraction is required to meet the total stiffness needs of the joint, to ensure sufficient stability and to prevent injury (Blackburn *et al.*, 2004a). Thus, deficiencies in passive stiffness should be compensated for with active muscle contraction, which increases bioenergetic demands and thereby reduces efficiency.

Because stiffness is a property of force over length, increasing muscle extensibility may lead to deficiencies in passive stiffness that need to be compensated for through active components (Blackburn *et al.*, 2004a). Equivalently, a less stiff muscle would permit increased lengthening to an applied load, potentially permitting an increased joint translation and increased risk of injury (Blackburn *et al.*, 2004b). However, there is limited research exploring the interactions of passive and active elements experimentally (Blackburn *et al.*, 2004a).

A lower passive stiffness will also mean a lower active stiffness for a given level of muscle activation to perform a task. Thus, active muscle contraction must increase to compensate to ensure an adequate level of total joint stiffness, and therefore sufficient joint stability (Blackburn *et al.*, 2004b). Through this mechanism, a suboptimal level of passive stiffness necessitates increased active stiffness to maintain dynamic joint stability and help prevent musculoskeletal injury (Blackburn *et al.*, 2004b).

2.4.7 Summary

To improve understanding of any interactions between soft tissues and LBP, or other dysfunction, it is necessary to assess individual aspects of biomechanics, so that contributing factors, and interactions that influence them, can be identified. In terms of assessment, it would be useful to understand whether LBP influences passive or active structures, and any interaction between them. By establishing how LBP affects other tissues, whether through referred pain or

by influencing movement, specific components of structure or function can be targeted in rehabilitative programmes.

If the joint capsule can be considered healthy, any unusual variations in joint stiffness in patients with LBP can most likely be attributed to the MTU. An increase in passive stiffness might indicate reduced MTU elasticity, whereas an increased elasticity with maintained stiffness could indicate changes in stretch tolerance. Alterations in active or total stiffness, whilst maintaining passive stiffness, would indicate changes in motoneuron activation. Thus, by establishing which tissues are affected by LBP and how, it ought to be possible to develop interventions that target these specifically.

2.5 Measuring Passive Stiffness

Measurements of joint moments have been conducted in both cadaver models (Makhsous *et al.*, 2008), and *in vivo* (Foure *et al.*, 2010, Nordez *et al.*, 2010a, Nordez *et al.*, 2010b, Hoang *et al.*, 2005, Lee and Munn, 2000, Vrahas *et al.*, 1990, Yoon and Mansour, 1982). For *in vivo* studies, calculations were based on data collected from multiple sensors, including load cells (Lee and Munn, 2000) or force plates (Hoang *et al.*, 2005, Nordez *et al.*, 2010a), inclinometers, goniometers (Makhsous *et al.*, 2008, Lee and Munn, 2000) and electromyography (Vrahas *et al.*, 1990).

Although more basic assessments utilising single force sensors have shown good intra-rater reliability (Andersen *et al.*, 2003, Moseley, 1991), it is unrealistic to suppose that such an approach yields accurate information on passive joint moments and muscle stiffness. These devices measure force

applied in a single direction, often without measurement of the transducer's position and angle in space, relative to the joint centre of rotation. The values referred to in such studies are therefore an indirect approximation of total resistive forces through a joint at the end ROM, rather than a direct measure of passive resistance based upon joint moment-angle data recorded during joint rotation.

Other techniques for measuring passive resistance include the Wartenberg pendulum test (Torres *et al.*, 2007), which does not assess passive properties through a full ROM, and which depends upon subjects to not interfere with the pendulum by contracting muscles or altering their posture. Isokinetic dynamometers have been used to measure passive resistance to movement (Foure *et al.*, 2011, Morse *et al.*, 2008), but these tests are confined to the positions available on the machine, and rely on an accurate assessment of joint centre of rotation.

As with the Wartenberg pendulum test, isokinetic dynamometers often do not assess passive resistance through a full ROM, such as via the short-range stiffness experiment (Foure *et al.*, 2011). Other investigators (Janecki *et al.*, 2011, Ylinen *et al.*, 2009), have used a computerised muscle tonometer to impose a force over a small area of muscle and then used an acceleration-transducer to record muscle deformation characteristics and the damped natural oscillations. This method only measures a specific area of superficial muscle tissue, making extrapolation of findings to whole muscle behaviour during elongation difficult.

More recently, magnetic resonance elastography (MRE) has been utilised to approximate muscle passive properties (Hatakenaka *et al.*, 2008,

Bensamoun *et al.*, 2006, Jenkyn *et al.*, 2003, Basford *et al.*, 2002). However, as this method requires use of a magnetic resonance imaging (MRI) scanner, it is less accessible to the majority of manual therapists. Further, this method attempts to approximate muscle stiffness directly, whereas the biomechanical modelling techniques developed by Lee and Munn (2000) and others (Yoon and Mansour, 1982, Halbertsma *et al.*, 1999), produce more complete data for calculation of moment-angles curves, which can be used in conjunction with range of motion assessments for a more complete clinical application and assessment. It may then be possible to measure the contribution of muscles and tendons via adaptation of the alpha method (Foure *et al.*, 2010), although *in vivo* experimental data has not yet supported the underlying assumptions of this approach.

One of the earliest assessments of *in vivo* hip passive stiffness was conducted by Yoon and Mansour (1982). In this investigation, subjects were positioned side-lying with the upper body and non-test leg on a raised surface and pelvis secured with straps. The test leg was secured to a low-friction cart, which was moved through the full range of hip joint motion in the sagittal plane, with load cells recording the force required to do so. Calculations of passive moments were derived from the force and hip-angle data, with the test being conducted at a predetermined velocity (Yoon and Mansour, 1982).

Halbertsma *et al.* (1999), used an instrumental straight leg raise (ISLR) test for assessing passive hip moments. This approach is similar to that used by Yoon and Mansour (1982), except that in the former the subject lies supine, whereas in the latter the participant is side-lying. Whilst this method produces continual moment-angle data throughout the test, it is essential that the subject

be positioned so that the hip axis of rotation is perfectly in line with the pivot of the ISLR frame. Also, due to the scale and expense of the ISLR equipment, it may not be appropriate for use in a clinical environment. Electromyograph electrodes were used to test for muscle activity during straight leg raising (Halbertsma *et al.*, 1999).

2.5.1 Measuring Passive Stiffness of Bi-articular Muscles

The ISLR is commonly used to measure passive hip moments (Raftry and Marshall, 2012), and has been found to have good reliability (Raftry and Marshall, 2012). However, the ISLR is designed to assess the hip only when the leg is straight. Hence, this apparatus is not appropriate for measuring hip moments with different knee angles. Measuring passive stiffness whilst varying the positions of two joints (for example hip and knee, or knee and ankle), can be more useful for integrating into functional movements, such as walking.

Other investigators have measured passive hip moments using standard dynamometers (Tafazzoli and Lamontagne, 1996), force plates (Lee and Munn, 2000), and other force transducers (Lee and Munn, 2000, Yoon and Mansour, 1982, Vrahas *et al.*, 1990, Riener and Edrich, 1999), with good reliability (Tafazzoli and Lamontagne, 1996, Lee and Munn, 2000, Yoon and Mansour, 1982, Vrahas *et al.*, 1990). This approach can be used to measure joint moments at one joint whilst varying the angle of an adjacent joint.

Whilst some researchers have attempted to assess passive resistance to stretch using handheld force transducers and other improvised techniques (Gajdosik, 1991, Gajdosik *et al.*, 1990, Gajdosik *et al.*, 1992, Vrahas *et al.*,

1990, Riener and Edrich, 1999, Yoon and Mansour, 1982, Palmer *et al.*, 2013), the measurements need to be taken relative to the distance from the joint centre of rotation, taking into account gravity, limb length, segment centre of mass, segment mass, and angular velocity and acceleration (Lee and Munn, 2000).

The benefit of a handheld force transducer is that it can be used for assessing various joints with a high degree of reliability, and therefore has the potential to be used in a clinical setting. Some handheld devices have shown limited efficacy (Palmer *et al.*, 2013, Bohannon and Andrews, 1987), either due to the experimental equipment or procedures, or the mathematical approach used. The biomechanical model developed by Lee and Munn (2000), permitted good coefficient of multiple correlation (CMC) scores and results that are generally in agreement with the published data.

A study by (Palmer *et al.*, 2013) compared the use of manual testing with a handheld load cell and an automated assessment with ISLR. The investigators (Palmer *et al.*, 2013) reported good reliability of the two assessment techniques, with intraclass correlation coefficients (ICC) of 0.81-0.86 for the manual method and 0.72-0.92 for the automated technique. It is important to note, however, that when comparing curve-data such as moment-angle curves, the CMC is a more sensitive assessment than ICC (Lee and Munn, 2000). Further, whilst there was good agreement within each measurement method, the handheld device was found to overestimate hip moments compared to the ISLR (Palmer *et al.*, 2013). This is likely due to the experimental equipment and procedures used for the manual tests, although more information is required regarding the biomechanical model used to calculate passive hip moments. The load cell used in the study by Palmer *et al.*

(2013) was of a low-profile, ‘pancake-style’ design, only measuring force in a single direction. Bi-axial or tri-axial load cells may be more appropriate, particularly when using a biomechanical model that calculates hip moments based upon vertical and horizontal force application. Further, electrogoniometers were attached to clothing, which may have caused incorrect measurement of hip angle.

2.5.2 Measuring Active Stiffness

Active MTU or joint stiffness cannot be measured directly, due to the contribution of all passive tissues. Instead, total MTU stiffness can be calculated, and an estimate of passive MTU stiffness subtracted from the total value(s), to estimate the active component. This approach assumes that passive and active components of total stiffness are additive (Whittington *et al.*, 2008), which remains to be confirmed. If this approach is correct, the active component can then be quantified directly, potentially utilising muscle EMG activity to assess correlation. However, this is further complicated where measures of total stiffness assume joints act as frictionless pins or hinges, with no passive component. How best to determine accurate measures of passive, active and total stiffness requires further investigation.

The active stiffness can only be estimated, rather than precisely determined, due to dynamic interactions between passive and active contributions during movement. Shifts in the lengths of fascicles and proximal and distal tendons, altering contributions from passive force enhancement, and viscoelastic influences, would all influence the active-passive interaction for

given joints through different combinations of angles, forces and accelerations. Such contributions would need to be determined via dynamic and complex biomechanical models.

In the current absence of such models, investigators either report the total MTU or joint stiffness, or else derive values for the active component by subtracting an estimate of the passive contribution from the total. More research on the passive-active interactions is required to improve the reliability and validity of dynamic biomechanical models.

A common method for measuring total MTU stiffness employs an oscillatory technique (Granata *et al.*, 2002a, Blackburn *et al.*, 2004a, Bell *et al.*, 2009, Bell *et al.*, 2011, Bell *et al.*, 2012, Blackburn *et al.*, 2009a), in which stiffness is estimated from the damped frequency of oscillation following perturbation. For an example of this method, to measure hamstring stiffness a subject might be positioned prone lying with their hip flexed and thigh secured to a test surface. The shank is maintained in a horizontal position, with a 30 degree angle between shank and the thigh maintained via isometric hamstring contraction. A weight (i.e. 10% body mass), would be placed over the ankle, an accelerometer on the shank, and a perturbating force is then applied to the back of the foot. The accelerometer and time information are used to calculate total MTU stiffness from the damped frequency of oscillation (Granata *et al.*, 2002b, Blackburn *et al.*, 2004a, Bell *et al.*, 2009, Bell *et al.*, 2011, Bell *et al.*, 2012). The same approach can be used to target other muscle groups, such as the quadriceps, by altering the subject's posture and joint position (Granata *et al.*, 2002b).

Assessments of active tendon forces and elongation characteristics have been used to calculate tendon stiffness at different %MVC (Burgess *et al.*, 2009b, Burgess *et al.*, 2009a, Kubo *et al.*, 2009a). In these, force is calculated using a dynamometer, and tendon elongation from measurements using ultrasound (Burgess *et al.*, 2009a, Burgess *et al.*, 2009b, Kubo *et al.*, 2009a). These measurements are highly specific to the tendon being assessed, and calculations based upon ultrasound measurements can contain errors where the full fibre length cannot be seen on a single image (so require estimations based upon fibre pennation angle).

Some investigators have used a stiffness-measuring protocol based upon either hopping (Eiling *et al.*, 2007) or jumping (Ford *et al.*, 2010). In the hopping-based assessment, following a thorough warm-up, subjects were required to hop unilaterally on a force plate and in time with a metronome. Peak ground reaction force is divided by the maximum displacement of the leg spring (from the vertical acceleration of the centre of mass) (Eiling *et al.*, 2007). A similar approach was used in the jump test, and calculations based upon a rotational spring plot model, where joint moment is calculated as a function of joint angle (Ford *et al.*, 2010).

Total MTU stiffness can also be calculated as a product of joint moment-angle data during walking assessments (Silder *et al.*, 2008), involving motion capture and force plates. In these, dynamic biomechanical models are used to determine moment data. The models incorporate body segment estimates, utilising limb lengths and joint centre of rotation data from anthropometric measurements, modelled on video systems using skin-based markers. Body

mass, limb lengths and joint excursion data can then be used with the video system and force plates to determine joint moments (Silder *et al.*, 2008).

2.5.3 Summary

Passive stiffness is measured as the derivative of the joint moment-angle curve. Stiffness is a product of the MTUs change in force and change in length, as opposed to extensibility, which simply refers to a length change. Passive stiffness is typically measured using some combination of dynamometers, load cells, accelerometers and/or inclinometers. However, muscle tonometry is sometimes used, although this gives information on a localised area of muscle tissue, rather than for the entire MTUs affecting joint stiffness. Some investigators measure tendon stiffness by assessing joint force production and tendon length changes, the latter most commonly measured via ultrasonography.

Active MTU stiffness cannot be measured directly, but rather total stiffness across a joint can be measured and active stiffness subsequently estimated. Some researchers refer to total stiffness and active stiffness interchangeably, but as passive properties can contribute approximately one-third or more of the value of the measured total stiffness, the terms should be regarded as distinct (Whittington *et al.*, 2008). Improved measuring techniques and dynamic biomechanical models are required, to enable investigators to more accurately assess passive and active properties and their interactions during movement. To measure bi-articular muscle properties at one joint whilst manipulating the angle of an adjacent joint, forces should be measured using a

handheld device comprising multiple force transducers. Such a device may need to be adapted from commercially available load cells and equivalent.

2.6 Passive and Active Contributions to Movement

During walking, the muscles of the triceps surae undergo minimal change in length, whilst experiencing a predominantly isometric contraction. This occurs due to dynamic length changes of the Achilles tendon (Magnusson *et al.*, 2003). This isometric contraction of the muscles with dynamic length changes of the tendon allows the passive-elastic transfer energy for walking, limiting the metabolic energy demand of the muscles (Magnusson *et al.*, 2003). Further, the metabolic energy that is required to sustain an isometric muscle contraction is less than that required for dynamic contractions. During a task such as walking, an optimal passive contribution allows for a minimised and efficient active input. Total muscle resistive force and total joint moments, which are a product of both active and passive components, may be site-specific, and variable according to load and velocity of the measured task (Lichtwark and Wilson, 2008, Arampatzis *et al.*, 2001, Arampatzis, 2006).

The main determinant of active MTU stiffness is the number of parallel cross-bridges, and so increases with electromyographic (EMG) activity 2004 (Blackburn *et al.*, 2004a, Marshall *et al.*, 2009, Morgan, 1977). Stretch reflex afferent signalling leads to an increase in parallel cross-bridge formation, and is thus a contributor to active musculotendinous stiffness (Blackburn *et al.*, 2004a, Nichols, 2004) and joint moment (Simonsen, 2002, Carpenter *et al.*, 1999). For this reason, it may be necessary to assess for EMG activity during

measurements of passive muscle stiffness and joint moments, although stretch reflex has been reported to be only 1-3% of the amplitude of maximal voluntary contractions (Hoang *et al.*, 2007). However, active contraction of muscles during stretch also increases passive force enhancement (Joumaa *et al.*, 2008b, Rassier and Herzog, 2005).

In an investigation of lower extremity joint stiffness in adolescent athletes (Ford *et al.*, 2010), it was reported that males experienced increases in active hip, knee and ankle stiffness, whereas females increased active stiffness at the knee only. However, once stiffness was normalised to body mass, no significant differences were reported (Ford *et al.*, 2010). Thus, active stiffness, a product of the number of muscle cross-bridges, can be expected to increase during growth and following training, as lean mass increases.

Muscle activation is unaffected by time of day (Onambele-Pearson and Pearson, 2007), which implies that active stiffness should also remain unchanged. Any differences in active stiffness may occur secondarily to changes in passive stiffness. The stiffness of the patella tendon was reported to decline between a morning and late-afternoon test (Onambele-Pearson and Pearson, 2007), possibly related to a reduction in fascicle length of the vastus lateralis muscle. It may be that by the afternoon the participants had spent many more hours being active than they had in the morning, and this, in turn, had some effect on muscle sarcomere length, accounting for altered tendon stiffness at the knee.

2.6.1 Effects of Gender and Hormones

Although gender has been found to affect both active (Blackburn *et al.*, 2004a, Blackburn *et al.*, 2009a) and passive lengthening characteristics (Marshall *et al.*, 2009), differences have been shown to be insignificant when anthropometric differences are taken into account (Blackburn *et al.*, 2009a, Blackburn *et al.*, 2004b). A study in which lower active muscle stiffness was reported in female subjects (56-73% of values for males) (Granata *et al.*, 2002a), standardised for phase of menstrual cycle (to account for any effects of hormonal fluctuations), but did not normalise stiffness to muscle cross-sectional area (CSA).

Cammarata and Dhaher (2008), compared frontal plane total knee joint stiffness between men and women, and between women using or not using hormonal contraceptives. Female subjects were divided into non-user, monophasic contraceptive and triphasic contraceptive users. The results of the study showed that males had greater MTU stiffness than females. Although stiffness was standardized to the product of mass and height, stiffness has been shown to be directly related to muscle CSA (Blackburn *et al.*, 2009a), which was not assessed. Further, it was found that frontal plane stiffness correlated positively with knee diameter and negatively with Q-angle, although the magnitude of the correlation was small.

The findings of that study (Cammarata and Dhaher, 2008) support a hypothesis that active and passive muscle properties interact to ensure joint stability during functional tasks. Developing an understanding of passive MTU characteristics throughout the ovarian cycle may be of some use, although the paper by Cammarata and Dhaher (2008), went some way to showing that

hormonal influences on total stiffness may be minimal or negligible. Another study of the effects of oral contraceptive use on total muscle stiffness (Bell *et al.*, 2011) reported no differences in stiffness over time (estimated menses and ovulation) for contraceptive users or non-users. Oestradiol, free testosterone and progesterone increased at ovulation, compared with menses for the non-users, and remained constant for contraception-users (Bell *et al.*, 2011).

This indicates that sex hormone fluctuations during the menstrual cycle do not influence either total muscle stiffness or laxity. However, oestradiol and free testosterone have both been found to be negatively associated with total muscle stiffness and rate of force production in females (but not males) (Bell *et al.*, 2011). In that study (Bell *et al.*, 2011), all female participants were tested during the follicular phase of their menstrual cycle, due to an association between this phase and increased anterior cruciate ligament (ACL) injury rates (Hewett *et al.*, 2007).

There has been some interest in the potential role of relaxin on muscle stiffness and ligamentous laxity (Dragoo *et al.*, 2011, Pearson *et al.*, 2011). Relaxin is understood to affect collagen synthesis, and relaxin concentrations may be influenced by other sex hormones, such as oestrogen (Pearson *et al.*, 2011) or progesterone (Dragoo *et al.*, 2011). A study by Pearson *et al.* (2011), reported an influence of relaxin on patellar, but not gastrocnemius tendon stiffness, with no changes in stiffness related to the menstrual cycle. Increased serum relaxin was found to reduce patellar tendon stiffness, without affecting tendon cross-sectional area. Another study (Arnold *et al.*, 2002) reported no relationship between relaxin and anterior tibial translation, a test of ACL laxity. How relaxin might influence tendon stiffness, and whether some tendons are

more sensitive to circulating relaxin concentrations due to increased receptors, remains to be elucidated.

2.6.2 Effects of Menstrual Cycle on MTU Stiffness and Joint Laxity

Total muscle stiffness has been found to be lowest at week 3 (ovulatory phase) of the menstrual cycle, by comparison to weeks 1 and 2 (follicular phase), with no changes in knee laxity throughout the cycle (Eiling *et al.*, 2007). However, a trend towards increased laxity during the ovulatory phase may not have reached significance due to low subject numbers (n = 11). A similar study (Park *et al.*, 2009b) did report that knee laxity was highest during the ovulatory phase, with no significant difference to follicular phase, but a significant difference compared with the luteal phase (P = 0.015). However, the values were small (ovulatory phase 5.2 (1.7) mm vs luteal phase 4.62 (1.53) mm), and the anterior draw test utilised an 89 N strain for all participants, regardless of body mass or other individual factors.

In another study by the same authors (Park *et al.*, 2009a) differences in knee laxity (greatest during ovulatory phase) and reduced total muscle stiffness during the ovulatory phase were reported. However, in 8 out of 9 comparisons there was no significant difference in stiffness, and an ANOVA showed no overall difference (P > 0.05). The same is true of joint laxity measurements, with the ANOVA showing no significant differences across the cycle.

An assessment of total muscle stiffness and hamstring extensibility (Bell *et al.*, 2009) found no change in stiffness, but increased extensibility, post-ovulation compared with post-menses. However, the subject number was small

(n = 8) and the extensibility measurements were performed by two, unblinded investigators, using a handheld goniometer. There is potential for the difference from 89.3 (9) degrees post-menses to 97.3 (9) degrees post-ovulation to have resulted from tester or participant bias.

Other investigators (Burgess *et al.*, 2009c, Kubo *et al.*, 2009b) have reported no significant fluctuations in the stiffness of the medial gastrocnemius tendon during the course of the menstrual cycle. There were also no differences in tendon length or CSA. Tendon stiffness was not correlated to serum levels of oestradiol or progesterone (Burgess *et al.*, 2009c, Kubo *et al.*, 2009b). Other biomechanical and neuromuscular characteristics, such as fine motor coordination, postural stability, hamstring-quadriceps strength ratio, knee flexion excursion, knee valgus excursion and peak proximal shear force, have been shown to not change during the menstrual cycle, despite fluctuations in oestradiol and progesterone (Abt *et al.*, 2007).

2.6.3 Effects of Age on Passive and Active Stiffness

As individuals mature from childhood to adulthood strength increases, and, on average, adult males are stronger than females (O'Brien *et al.*, 2010). An understanding of whether this reflects any structural or neuromuscular differences, due to age and/or gender, would be useful for understanding their influence on passive and active contributions to movement. For example, are any differences in the active contribution to walking related to differences in muscle CSA, the muscle specific tension or activation properties?

Muscle specific tension refers to the maximal force produced per unit of the physiological CSA of a muscle (Erskine *et al.*, 2011). It has been demonstrated that muscle specific tension is not influenced by age. In an investigation of the quadriceps muscle group (O'Brien *et al.*, 2010), it was found that tensile stress was similar in any of the quadriceps heads for male and female adults and children (55 (11) N/cm^{-2} for men, 57.3 (13) N/cm^{-2} for women, 54 (14) N/cm^{-2} for boys and 59.8 (15) N/cm^{-2} for girls). The increased strength associated with maturation, and of males compared with females, is not a product of structural differences within the muscle, but rather muscle size, moment arm length and the level of voluntary activation (O'Brien *et al.*, 2010).

An investigation of MTU properties (Faria *et al.*, 2011), comparing young (20.3 (2.8) years) and postmenopausal women (58.4 (5.6) years), reported increased MTU stiffness ($P < 0.001$), including MTU stiffness normalised by mass ($P < 0.05$), in older women. This finding is in agreement with a study of prepubescent children (Grosset *et al.*, 2007), in which both passive and active stiffness of the articular and musculotendinous structures were found to increase significantly ($P < 0.01$) with age (age range 7-11 years), but remained significantly lower than adult values ($P < 0.01$) (Grosset *et al.*, 2007). It has been found that the age-related increase in passive stiffness is associated with the replacement of degenerated muscle fibres with non-elastic connective tissue (Wolfarth *et al.*, 1997). Whether this is also the reason for the difference between children and young adults has not yet been investigated, although this seems unlikely.

Caution is recommended where investigators have neglected to consider other age-associated factors that could influence passive stiffness. For

example, the study by Faria *et al.* (2011), only excluded volunteers with diabetes or signs of neuropathy, yet various other conditions could potentially impact on passive properties. Parkinson's disease, for example, has been associated with an increase in muscle passive stiffness, independent of neurological changes (Marusiak *et al.*, 2010). The mechanism is thought to be due to structural changes (Marusiak *et al.*, 2010), as might occur following soft-tissue injury. This is in agreement with the finding that the age-associated increase in passive stiffness is associated with the replacement of elastic muscle fibres with non-elastic connective tissue (Wolfarth *et al.*, 1997).

At best, the effects of many age-associated conditions on passive properties are not known. It would seem prudent, however, to exercise caution and exclude those who have had serious injury (i.e. fractures, muscle or tendon tears), surgery, and any physical disorder that could potentially have led to long-term changes in stiffness.

2.6.4 Summary

It may be useful to assess passive and active stiffness independently, to better determine any influence of circulating hormone concentrations on biomechanical characteristics. Many current investigations refer to total stiffness only, whereas an understanding of any influences of age, gender and hormones on passive structures and active MTU behaviour would be useful.

It is clear that there is an age-associated increase in passive muscle stiffness, and this change appears to be due to replacement of elastic muscle fibres with non-elastic connective tissue. Due to the potential for non-elastic

connective tissue to replace damaged tissues in younger individuals, and at a higher rate in those with certain disease states, it is appropriate to ensure study inclusion/exclusion criteria reflect this.

2.7 Mechanical Properties of Joints and LBP

A comparison of the passive-elastic component and passive stiffness of the hip joint found significant differences between LBP participants and healthy controls (Tafazzoli and Lamontagne, 1996). In this study, it was noted that the variability of the passive elastic moment of the hip increased with hip angle, the moment range being 17.90 Nm to 43.87 Nm. The investigators (Tafazzoli and Lamontagne, 1996) reported a linear relationship between hip stiffness and hip angle in flexion, with stiffness being greater in the LBP group.

In difference to the investigation by Tafazzoli and Lamontagne (1996), a study by Gombatto *et al.* (2008b), found that, although lumbar spine muscle passive properties were influenced by LBP, this was not evident at end ROM. This suggests that common clinical assessments of LBP may be improved upon by incorporating tests of muscle stiffness, and that more information may be obtained by assessment of passive stiffness throughout range rather than maximum joint ROM alone. A potential reason for the difference in findings at end ROM, may be that the effects of LBP are site-specific, with an effect at the hip but not at the lumbar spine. This may be related to functional adaptations at the hip, due to leg swing contributing considerably to total body angular momentum, but little contribution of the lumbar spine of individuals with or without LBP (Bruijn *et al.*, 2008).

Halbertsma *et al.* (2001), measured extensibility and stiffness of the hip joint in individuals with LBP. Healthy subjects were assigned to a flexible group (finger-to-ground distance < 0 cm, n=8), or stiff group (finger-to-ground distance > 0 cm, n=12). Halbertsma *et al.* (2001), found that ROM and extensibility of the LBP group was significantly smaller than in the stiff group. However, there was no significant difference between the LBP group, stiff group and flexible group for total, active or passive joint moments, or passive muscle stiffness.

The onset of muscle activity (measured using EMG)was significantly earlier in the LBP than for either stiff or flexible groups, and there was no significant difference between EMG onset between the stiff and flexible groups (Halbertsma *et al.*, 2001). In 14 subjects in the LBP group, and 2 in the stiff group, there was also electromyographic activity in the back muscles during the SLR test. Pain was detected significantly earlier in the stiff group compared with the flexible group, and there were no significant differences between the stiff group and patient group. Lumbar lordosis during the SLR tests was not significantly different between any groups, reaching 0 (flat back) in 8 of the 20 subjects in the patient group, 8 out of 12 in the stiff group, and all subjects in the flexible group.

In a study comparing isometric MVCs of the lumbar paraspinal muscles in extension (Oddsson and De Luca, 2003), and 30 second maintenance of 40% and 80% MVC, it was reported that LBP subjects produced 55% of the MVC of healthy controls. In addition, during the 40% and 80% comparisons, LBP subjects demonstrated less fatigue, which the investigators attributed to those subjects failing to produce a ‘true’ MVC. It was also found that LBP subjects displayed lumbar spine muscle activation imbalances, reflecting

physiological impairments due to pain and/or injury (Oddsson and De Luca, 2003). Further, in healthy subjects, it was found that local segmental activation imbalances can be offset by altering segment activity on a more global scale, and this occurs to a greater extent in healthy versus LBP subjects (Oddsson and De Luca, 2003). The lack of variability in movement may indicate adaptations to limit pain, but could potentially promote fatigue or else increase over-use stress on some populations of muscle fibres.

2.7.1 Active Properties and LBP

The active component of movement is a product of the amount of motor units that are activated by the somatic nervous system (Blackburn *et al.*, 2004a, Marshall *et al.*, 2009). Inefficiencies in the active component may be due to early or late muscle activation, prolonged activation, altered patterns of activation (unusual activation sequences), or increased co-contraction of antagonist or stabilising muscles (Farmer, 2003, Wakeling *et al.*, 2011, Blake and Wakeling, 2015). There is also potential for active components to be increased to compensate for alterations in the passive component, such as increased agonist contractions due to increased antagonist passive stiffness.

Active hip abduction and active SLR tests are sometimes used in clinical assessments of LBP patients (Nelson-Wong *et al.*, 2009, Waddell *et al.*, 1992, Roussel *et al.*, 2007, Davis *et al.*, 2011, Vanti *et al.*, 2016). In a study by Nelson-Wong *et al.* (2013), trunk and hip muscle coordination were compared using EMG in LBP and control subjects, during active hip abduction and active SLR tests. During right active hip abduction, LBP subjects utilised a distal to

proximal activation pattern with the hip abductor (gluteus medius) activated before the ipsilateral trunk flexors (internal and external oblique muscles), whereas control subjects utilised a proximal to distal activation pattern. Similarly, during left hip abduction, LBP subjects utilised a distal to proximal pattern with the gluteus medius activated prior to the contralateral trunk extensors (right erector spinae) and ipsilateral internal oblique muscles, whereas control subjects demonstrated a proximal to distal activation pattern (Nelson-Wong *et al.*, 2013).

During active straight leg raising (Nelson-Wong *et al.*, 2013), both LBP and control subjects utilised a distal to proximal activation sequence, with rectus femoris activated prior to ipsilateral external oblique, although LBP subjects had a significantly shorter phase lag than controls ($P < 0.05$). LBP subjects also demonstrated a co-activation of rectus femoris and contralateral gluteus maximus, whereas control subjects utilised a proximal to distal activation strategy of these muscles. Overall, the results of the study by Nelson-Wong *et al.* (2013) demonstrate altered activation strategies to maintain lumbopelvic stability, between subjects with LBP and healthy controls, during frontal and sagittal plane tests.

During active prone hip extension exercise, activation patterns of various hip extensor and lumbar muscles were compared between LBP and control subjects (Guimaraes *et al.*, 2010). There were no significant differences between groups for muscle latencies, amount of activation or duration of activation. In LBP subjects the muscle activation pattern began at the semitendinosus, followed by the contralateral erector spinae, ipsilateral erector spinae and gluteus maximus. In control subjects it was the ipsilateral erector

spinae that was activated prior to the contralateral erector spinae (Guimaraes *et al.*, 2010). The investigators concluded that EMG activity was not able to distinguish between LBP and control subjects during prone hip extension (Guimaraes *et al.*, 2010).

An assessment of trunk forward flexion found no differences in total amounts of lumbar spine or hip motion or velocity between subjects with a history of LBP and subjects without (Esola *et al.*, 1996). However, differences were observed during early forward flexion, with LBP subjects bending more from their lumbar spines. During middle forward bending subjects with a history of LBP exhibited a lower lumbar-to-hip flexion ratio than controls (Esola *et al.*, 1996).

An assessment of lumbar extension (McClure *et al.*, 1997) demonstrated that subjects with a history of LBP moved from the lumbar spine earlier than controls without a history of LBP, and the difference was most notable during the initial 25% of extension. Hamstring length was not correlated with any kinematic characteristics during extension. Asymptomatic subjects with a history of LBP moved similarly to subjects without a history of LBP, except during the first 25% of extension when greater lumbar motion and velocity occurred. The authors (McClure *et al.*, 1997) suggested this may have been due to previous LBP or a factor in recurring LBP, although prospective study is required to confirm this hypothesis. There were no differences between subjects in total lumbar or hip motion or average lumbar or hip velocity.

Kim *et al.* (2014) assessed lumbopelvic hip motion during seated hip flexion, and compared subjects with and without LBP, where both groups had limited hip flexion. Overall, the hip flexion angle was significantly lower (P =

0.014), whilst lumbar flexion angle was significantly greater in LBP subjects (P = 0.006). In addition, the angle of posterior pelvic tilting in LBP subjects was significantly greater than control subjects (P = 0.019). There were no differences between groups for lumbar lateral bending or lumbar rotation (P > 0.05) (Kim *et al.*, 2014).

A study involving an assessment of hip lateral rotation (Gombatto *et al.*, 2006) reported no differences in movement pattern between LBP subjects who reported increased pain during the test, and LBP subjects who did not. Male and female subjects utilised different movement patterns, and male subjects were more likely to experience pain during the hip rotation (Gombatto *et al.*, 2006). Similarly, another study of hip lateral rotation (Van Dillen *et al.*, 2007) reported that LBP subjects adopted either a lumbar rotation, or lumbar rotation with extension movement, during the test. Further, subjects in the lumbar rotation subgroup moved the hip and lumbopelvic region symmetrically, whereas those in the lumbar rotation and extension subgroup moved the hip and lumbopelvic region asymmetrically. The authors concluded that symmetry of movement between the hip and lumbopelvic region, and between right and left sides, could have important clinical implications for intervention strategies (Van Dillen *et al.*, 2007).

During an assessment of muscle activity during spinal flexion and extension movements (Leinonen *et al.*, 2000), altered muscle activation was observed in LBP subjects compared with healthy controls. At the initiation of sagittal flexion, lumbar paraspinal and biceps femoris muscles were activated simultaneously in control subjects, with the biceps femoris being activated significantly before the gluteus maximus. However, in LBP subjects the lumbar

paraspinal muscles were activated significantly before gluteus maximus. Overall, gluteus maximus activity was reduced in LBP subjects during the flexion-extension cycle (Leinonen *et al.*, 2000). At the end of sagittal flexion, the lumbar paraspinal muscles relaxed before the gluteus maximus and biceps femoris in control subjects, whereas the lumbar paraspinal muscles and gluteus maximus relaxed simultaneously in LBP subjects (Leinonen *et al.*, 2000).

At the beginning of spinal extension, the biceps femoris was activated significantly before the lumbar paraspinal muscles and gluteus maximus in controls, whereas in LBP subjects the gluteus maximus was activated prior to the lumbar paraspinal muscles (Leinonen *et al.*, 2000). At the end of extension, relaxation of the lumbar paraspinal muscles and gluteus maximus occurred simultaneously, and followed relaxation of biceps femoris in control subjects. In LBP subjects the gluteus maximus and biceps femoris relaxed prior to the lumbar paraspinal muscles (Leinonen *et al.*, 2000). Due to the reduced gluteus maximus activation in LBP subjects compared with controls, it was considered that these muscles should be taken into consideration in the treatment of LBP (Leinonen *et al.*, 2000).

In a similar study (Nelson-Wong *et al.*, 2012), subjects without a history of LBP but who developed pain during prolonged standing, were found to exhibit altered recruitment strategies to those who did not develop pain, during an extension test. Nelson-Wong *et al.* (2012) assessed trunk flexion and extension EMG and kinematic variables prior to a 2-hour standing exposure. In pain developers, lumbar erector spinae were activated prior to gluteus maximus, whereas the reverse activation pattern occurred in non-pain-developers, during extension from full flexion. This is a different finding to the

study by Leinonen *et al.* (2000), which is likely due to the measurements occurring at different parts of the movement (extension from neutral in Leinonen *et al.* (2000) and extension from full flexion in Nelson-Wong *et al.* (2012). Although other muscles pairs (thoracic erector spinae and gluteus maximus, and thoracic erector spinae and lumbar erector spinae) showed a similar trend in activation differences in flexion and extension, these did not reach statistical significance (Nelson-Wong *et al.*, 2012). Whilst there were no gender differences in total range of motion at terminal flexion, there were differences in lumbar:hip ratio, hip ROM and lumbar ROM, independent of pain group (Nelson-Wong *et al.*, 2012).

The same authors (Nelson-Wong *et al.*, 2008), had previously performed a similar study of subjects without a history of LBP during a 2-hour standing test. 65% of subjects developed LBP during the protocol, and exhibited an increased activation of the bilateral gluteus medius muscles than subjects who did not develop LBP ($P = 0.002$). The authors (Nelson-Wong *et al.*, 2008) were able to correctly identify LBP-developers and non-developers based upon gluteus medius co-activation (75% accuracy, sensitivity 0.87). The authors (Nelson-Wong *et al.*, 2008) speculated upon whether the co-activation was an adaptive or causal factor in the development of LBP in previously asymptomatic subjects. A later study by the same authors (Nelson-Wong and Callaghan, 2010), confirmed elevated co-contraction of bilateral gluteus medius in pain developers. An additional finding was elevated trunk flexor and extensor muscle activation, most notably during the first fifteen minutes of standing, and a decreased rest time for gluteus medius and gluteus maximus muscles (Nelson-Wong and Callaghan, 2010).

A similar study (Marshall *et al.*, 2011b) assessed maximal hip abduction strength and side-bridge endurance before and after a 2-hour standing protocol, in subjects without a history of LBP. Side-bridge endurance was found to be lower prior to the standing protocol in subjects who developed LBP. There was no relationship between hip abduction strength and LBP. Both exercise tests were significantly associated with gluteus medius co-activation during standing, and the investigators (Marshall *et al.*, 2011b) suggested that side-bridge endurance and gluteus medius co-activation could potentially be used to identify pain-developers during prolonged standing.

In a comparison of female subjects with a history of recurrent LBP and those without, a 20-minute treadmill walking protocol demonstrated significantly greater active hamstring stiffness during the 48-72 hours post-exercise, despite no significant differences immediately after exercise (Bedard *et al.*, 2013). Isometric strength of the hamstring and quadriceps muscles was tested before and immediately after the walking protocol, with no differences observed between groups. The authors (Bedard *et al.*, 2013), concluded that the increased active hamstring stiffness may have been a compensation for weakness in the muscles that contribute to trunk, pelvis and hip stability during exercise. However, no such measurements or muscle activity was assessed, and other mechanisms may have been responsible.

It is plausible that the increased active stiffness observed in the Bedard *et al.* (2013), study may have been to reduce forces acting upon the lumbar spine, such as by reducing hip flexion during walking, or increasing knee flexion angle. Similar assessments of active stiffness of gluteus maximus, gluteus medius and erector spinae muscles may be of use. In an assessment of lumbar

flexion moments during lifting a negligible load, the external moment was significantly related to lifting posture, with two distinct lifting strategies observed in subjects with LBP (Wilson *et al.*, 1997). It was suggested that understanding how LBP subjects alter their movement kinetics to limit forces on their lumbar spine may be useful in treating LBP (Wilson *et al.*, 1997).

For an assessment of lumbar spine stiffness (Freddolini *et al.*, 2014b), LBP subjects and healthy controls were required to recover balance in a custom-built, freely-swinging chair. The model used demonstrated that stiffness was significantly increased ($P < 0.001$) in LBP subjects. A second investigation by the same authors (Freddolini *et al.*, 2014a), reported that there were no significant differences in muscle moments and power between LBP and control subjects ($P > 0.05$). However, the duration of contraction and co-contraction of various trunk muscles was found to be significantly longer in LBP subjects ($P < 0.05$). Muscle reaction times were reported to be lower in LBP subjects than control subjects ($P < 0.05$). The authors concluded that LBP subjects adopted an alternative muscle strategy to regain balance than control subjects, and this strategy preserved similar balance control and internal moment patterns between groups (Freddolini *et al.*, 2014a).

It is possible that trunk stiffness is associated with fear of pain during movement, rather than an inherent adaptation within the muscle. This would support a hypothesis that it is active stiffness rather than passive stiffness that is altered during pain. A study by Karayannis *et al.* (2013), reported a positive linear correlation between kinesiophobia and trunk stiffness in response to a sudden forward perturbation in LBP subjects ($P < 0.03$).

In a cross-over study of individuals without LBP, subjects received equal volume injections of either hypertonic or isotonic saline, in random order, to the L3-L5 interspinous ligaments (Wong *et al.*, 2016). Subjects receiving hypertonic saline injections reported the greatest pain and demonstrated greater spinal stiffness and muscle activity (erector spinae, external oblique, internal oblique and transversus abdominis) than when receiving isotonic injections. There was a direct relationship between pain intensity and spinal stiffness ($P < 0.05$) and between pain and muscle activity of all trunk muscles ($P < 0.05$). The authors (Wong *et al.*, 2016) reported that experimentally-induced LBP caused temporary increases in spinal stiffness and concurrent trunk muscle co-contraction, leading to the conclusion that spinal muscle stiffness increases due to pain, and that measuring stiffness may be a useful means of assessing severity and progression of LBP.

To summarise, muscle activation patterns have been found to differ between LBP and control subjects, and between asymptomatic subjects who develop pain during activity. Increases in active muscle stiffness may follow pain or fear of pain, and have been shown to increase during the 48-72 hours following aerobic activity, in asymptomatic subjects with a history of LBP. It is not possible to ascertain whether differences are causal or adaptive, but it is plausible that in some cases it is an adaptive response to limit forces on the lower back and reduce pain. Overall, further research is needed through prospective study to help identify factors relating to pain development, with interventions assessed that might contribute to pain alleviation.

2.7.2 Passive-Active Contributions to Walking

By being stretched, passive tissues contribute to hip power generated from the mid-stance through to the initial swing phase of the gait cycle, and have been found to produce approximately 35% (approximately 0.88 Nm/kg) of the peak total moment (Whittington *et al.*, 2008). During hip extensor power bursts, 38% of the negative hip extensor work done is absorbed then released, which contributes an average of 58% of the positive work during the subsequent hip flexor power burst (Whittington (Whittington *et al.*, 2008). Investigators (Whittington *et al.*, 2008) have reported a direct exchange of passive energy between the hip and knee, via the rectus femoris, and between the knee and ankle via the gastrocnemius.

Kang and Dingwell (2008), assessed walking characteristics of younger (mean 23 (3) years) and older (mean 72 (6) years) adults. Although older adults exhibited lower strength and ROM, preferred walking speeds remained similar. Greater variability in trunk roll was reported in the older adults. Step length and stride times were not influenced by age, when anthropometric variables and walking velocity were taken into account. Increasing speed affected variability in stride time, hip abduction/adduction angle, knee varus/valgus angle, knee internal/external rotation and all motions of the trunk (Kang and Dingwell, 2008).

The findings of the study by Kang and Dingwell (2008), are not in agreement with those of an earlier study by McGibbon and Krebs (2004). In the McGibbon and Krebs (2004) investigation, healthy participants were stratified into two groups of those younger than 50 years or older than 50 years (mean

29.7 (6.9) and mean 71.1 (8.2) years, respectively). A third group consisted of older individuals with functional impairments (mean 75.1 (6.1) years). The younger group was therefore older, on average, than those recruited into the Kang and Dingwell (2008) study, and there was a greater spread of ages within the groups. In this study (McGibbon and Krebs, 2004), younger subjects exhibited significantly greater step length and stride velocity than healthy older subjects ($P < 0.001$ and $P = 0.001$, respectively), and the healthy older group had greater step length and velocity than the disabled group ($P = 0.01$ and $P = 0.021$, respectively).

An investigation by Silder *et al.* (2008), compared passive and active contributions to walking in healthy young (18-35 years) and older (65-85 years) subjects. Passive stiffness was assessed in a side-lying position, using three-dimensional load cells. Passive stiffness, power and work were calculated during walking. There were significant differences between total work in the two groups during walking, but no difference in passive contributions. The difference in work at the hip was therefore attributed to increased active contributions (Silder *et al.*, 2008).

In that study (Silder *et al.*, 2008), a reduction in negative hip flexor power was attributed to a prolonged power output from the hamstrings. It is also possible that muscle activity at the hip was influenced by activity of the plantarflexors, as the power from these muscles was reduced during gait in the older subjects (Silder *et al.*, 2008). This is in agreement with a review article, in which it was asserted that neuromuscular adaptations at the hip compensate for a reduced capacity of the plantarflexors (McGibbon, 2003).

The investigation by McGibbon and Krebs (2004), reported greater ankle plantar flexion and lower knee maximum flexion in young, compared to older healthy subjects, and no difference between older subjects with or without disability. This demonstrates functional adaptations at the ankle, knee and hip that are associated with age but not disability. There were no differences between young and older healthy subjects and ankle dorsiflexor or plantarflexor moments, knee flexor or second knee extensor moments, or hip flexor moments. There were, however, significant differences for each of these between the disabled older and healthy young and older subjects (McGibbon and Krebs, 2004).

2.7.3 LBP and Walking

Normal walking in healthy individuals can be expected to promote locomotor efficiency. When a healthy individual walks at their preferred velocity, there may be a maximised efficiency of mechanical energy output, as a product of both kinetic and potential energy (Yoon and Mansour, 1982), thus reducing metabolic energy requirements across a given distance. This can be demonstrated by reduced oxygen consumption when an individual walks at their preferred velocity (Yoon and Mansour, 1982, Bereket, 2005). Increased agonist and/or antagonist muscle activity, or unusual compensation patterns, reduce mechanical efficiency (Yoon and Mansour, 1982), increasing energy expenditure and promoting fatigue

Although individuals with LBP may be encouraged to remain active, little is understood regarding the effects of pain on walking (Simmonds *et al.*, 2012).

Individuals with LBP tend to have a lower preferred walking speed than healthy controls, and a reduced stride length (Henchoz *et al.*, 2015, Lamoth *et al.*, 2006a, Lee *et al.*, 2007, Muller *et al.*, 2015, Selles *et al.*, 2001, Vogt *et al.*, 2003). LBP subjects have also been found to exhibit greater individual variability in walking pattern than control subjects (Selles *et al.*, 2001, Simmonds *et al.*, 2012). This variability in walking attenuates forces in some individuals but not in others (Simmonds *et al.*, 2012). The normal variability and lack of certainty regarding what constitutes 'normal' movement complicates the study of kinematic variability in LBP subjects, particularly when individual measures before pain first occurred are not available (Simmonds *et al.*, 2012). Despite variability in walking patterns, there is a lack of overall difference in mechanical work and bioenergetics of walking between LBP subjects and healthy controls (Henchoz *et al.*, 2015).

2.7.3.1 Walking Speed

Muller *et al.* (2015), reported that individuals with LBP self-select a walking speed 6.5% slower than healthy subjects over even ground, and 6% slower over uneven ground ($P < 0.01$). Lee *et al.* (2007) reported that, although the desired walking speed of individuals with LBP was significantly slower than in healthy subjects, fastest walking velocity was similar. This is in agreement with some researchers (Vogt *et al.*, 2003, Lamoth *et al.*, 2006a), whereas Selles *et al.* (2001) reported reductions in both preferred and fastest walking speeds. Individuals with both LBP and referred leg pain have been found to walk more

slowly at both preferred walking speed and fastest speed (Lamoth *et al.*, 2006a), indicating fastest speed may be related to pain intensity.

Reduced hip flexion angle and stride time have been associated with prolonged gluteus maximus and lumbar erector spinae activity (Vogt *et al.*, 2003). Overall, faster walking speeds are associated with increased muscle activation, increased ground reaction force (GRF) and joint forces, and greater hip and knee angles (Chung and Wang, 2010). Conversely, the GRF and amplitude of muscle activity are reduced with decreasing walking speed (den Otter *et al.*, 2004).

At preferred walking speeds, the swinging leg follows a ballistic trajectory, led by accelerations from muscles during the stance phase and early swing phase of gait (Fox and Delp, 2010). At much slower walking speeds a greater active muscle component may be required, to permit sufficient time for the swing leg to be adequately rotated forward (den Otter *et al.*, 2004). In addition, LBP subjects have been found to exhibit greater mediolateral centre of mass displacements than healthy subjects (Henchoz *et al.*, 2015).

It has been suggested that reduced walking speeds in LBP subjects may be due to an inability to counter-rotate the pelvis and thorax (Selles *et al.*, 2001). This counter-rotation should occur to preserve locomotor efficiency, as increased arm swinging contributes to total body angular momentum during faster walking (Bruijn *et al.*, 2008, Huang *et al.*, 2010, Selles *et al.*, 2001).

Although preferred walking speed is lower in LBP subjects, alterations in mechanical work and the energy cost of walking have not been found (Henchoz *et al.*, 2015). It may be that the observed alterations in walking pattern are not sufficient to reduce efficiency (Henchoz *et al.*, 2015). It is plausible that a

reduced walking speed is related to fear of pain at higher walking speeds, although such a relationship has not been identified in LBP subjects (Henchoz *et al.*, 2015, Lamoth *et al.*, 2006b), despite observed differences in walking during experimentally-induced pain (Lamoth *et al.*, 2004).

2.7.3.2 Swing Phase

During walking, subjects with LBP reduce their swing phase whilst increasing their stance phase time (Ertelt, 2014). It is thought that such alterations help to improve control of walking, distributing the total active muscle forces and GRF over a longer time, whilst being less likely to experience a perturbation during initial ground contact (Zehr and Stein, 1999, Ertelt, 2014). However, such alterations also lead to increased mediolateral centre of mass displacement (Henchoz *et al.*, 2015).

Activation of biceps femoris is increased in LBP subjects during the late swing phase to cause a more rapid retraction of the lower leg (Ertelt, 2014). When the foot is in contact with the ground the biceps femoris contributes to knee extension. This paradoxical function of biceps femoris is due to dynamic coupling, demonstrating a Lombard's effect (Ertelt, 2014). Overall, the hip extensors decelerate the swing-limb shank during late swing phase, with the muscle forces about the hip promoting knee extension, whilst the forces about the knee promote knee flexion (Arnold *et al.*, 2007).

The altered walking strategy in LBP subjects reduces the overall GRF, whilst concentrating the peak of GRF earlier in the stance phase. This increased GRF early in stance phase reduces the damping coefficient of

passive tissues, leading to less stress absorption and greater force transfer across joints, requiring increased activity of the biceps femoris to stabilise the hip and sacroiliac joint (Ertelt, 2014).

2.7.3.3 Muscle Activation

In an assessment of lumbar spine and hip extensor activation during walking in individuals with and without LBP (Vogt *et al.*, 2003), subjects with LBP demonstrated a reduced hip ROM (38.3 (9.1)° vs 25.2 (7.9)°) and stride time (1.06 (0.05)s° vs 1.03 (0.09)s°), for healthy controls and LBP groups, respectively ($P < 0.01$), which is in agreement with other studies (Lee *et al.*, 2007, Lamoth *et al.*, 2006a). Prolonged activity was recorded in gluteus maximus and lumbar erector spinae (L3) muscles in those with LBP ($P < 0.01$), and no differences were detected at T12 (Vogt *et al.*, 2003). Unfortunately, only a single side was selected for EMG analysis, so any potential asymmetries could not be assessed. The relationship between stride length and stride time is in agreement with studies of healthy, pain-free individuals (Bruijn *et al.*, 2008).

2.7.3.4 Pelvis-Trunk Coordination

In an investigation of trunk muscle coordination by Lamoth *et al.* (2006a), it was demonstrated that individuals with LBP exhibit limitations in motor control compared with asymptomatic subjects. An apparent inability to adjust variant and invariant walking patterns with altering velocity may be indicative of reduced walking stability. This may account for a preferred walking velocity, in

individuals with LBP, which is slower than that of healthy controls (Lamoth *et al.*, 2006a). This adaptation is thought to be a functional adaptation to help cope with internal and external perturbations. The investigators recommended management of LBP that targets improvements in both functional capacity and flexibility (Lamoth *et al.*, 2006a).

In another study by Lamoth *et al.* (2006b), the investigators reported greater rigidity and less variability between both thoraco-pelvic segment rotations and lumbar-pelvic segment rotations, when compared to healthy controls. However, frontal plane segment coordination showed increased variability, particularly at walking velocities above the individual's preferred velocity. This may be related to the stability required for permitting the in-phase to anti-phase shift that occurs with increasing velocity, as reported in an earlier study by the same authors (Lamoth *et al.*, 2002). An increased activity of the lumbar erector spinae (Lamoth *et al.*, 2006b) is in agreement with previous studies (Arendt-Nielsen *et al.*, 1996, Lamoth *et al.*, 2004).

During walking, frontal plane motion occurs as one side of the pelvis must be raised to permit the hip to rotate during swing phase. Despite a relatively low level of disability, LBP subjects in a study by Seay *et al.* (2011), exhibited different pelvis-trunk coordination than controls in lateral flexion. Frontal plane coordination was found to be less anti-phase in LBP subjects compared with controls and those with resolved LBP (Seay *et al.*, 2011). The authors (Seay *et al.*, 2011), concluded that frontal plane coordination was more affected by LBP than transverse plane during walking, and that the alterations were indicative of a more 'guarded' (in-phase) walking pattern. Differences due to LBP did not become more pronounced by increasing walking speed (Seay *et*

al., 2011). These findings are in agreement with Selles *et al.* (2001), who reported that reduced preferred and maximum walking speeds may be due to an inability for LBP subjects to counter-rotate the thorax and pelvis, and thus the inability to switch from in-phase to out-phase coordination.

Huang *et al.* (2010) reported that out-phase pelvic-thorax transverse rotation occurred in relation to stride length and not walking speed. Overall, walking speed did not influence trunk coordination, or rotational amplitudes of the pelvis, thorax or spine. Rotation of the pelvis increased in the direction of the swing leg, as stride length increased (Huang *et al.*, 2010). However, rotations became less out-phase with slow walking and large strides, which the authors were unable to explain (Huang *et al.*, 2010). Rotation of the spine also increased with increased stride length. In a study by Gombatto *et al.* (2015), LBP subjects were found to rotate their lumbar spines in the transverse plane less than controls, with no differences in sagittal and frontal planes. Slower walking in LBP subjects may be a functional adaptation to altered motor control of the pelvis and thorax, and not directly related to actual pain, fear of pain or re-injury, or level of disability (Lamoth *et al.*, 2006b).

2.7.4 Summary

Assessments of hip biomechanics have revealed differences in individuals with LBP compared with healthy controls. Differences have been observed in ROM, timings of movement and the nature of muscle activation, rather than in changes in MTU stiffness. The differences between LBP and pain-free subjects have been observed in functional assessments of individuals performing

activities of daily living. It is thought that compensations occur to increase stability and/or reduce stress on painful regions of the lumbar spine.

In individuals with chronic LBP, compensations occur to promote stability of the lumbar spine. Further, some biomechanical factors may be similar in individuals with or without LBP, but different in those with LBP and referred leg pain, such as a reduced maximum walking velocity. Increased rigidity, coupled with less walking pattern variability, have been reported in individuals with LBP.

2.8 Interventions to Influence Passive and Active Properties

Although the nature of how LBP might influence lower limb and trunk passive and/or active MTU characteristics remains to be determined, it would be of benefit to understand whether specific interventions can be used to influence passive and active MTU properties. This could be to gain a better understanding of what interventions might be useful in treating any LBP-associated biomechanical factors, or else to ensure activities that might contribute to, or exacerbate, LBP are avoided. The purpose of this section is to summarise the results of studies that aimed to influence passive or active MTU properties.

In addition to the studies summarised here, there are currently various movement-therapy-based courses available to physical therapists that attempt to target the active component of biomechanics, but these lack any objective scientific measure of their effectiveness and efficacy (Gray, 2016c, Gray, 2016b, Gray, 2016a, Dalcourt, 2016, Cormack, 2016, Hardy, 2016), and warrant further investigation.

Table 2.1. Findings of studies investigating influence of interventions on components of muscle stiffness and related biomechanical properties

Authors	Area	Intervention	Findings
Stretching			
Herda *et al* ., (2011)	Knee flexors	Constant angle vs constant torque, 16 x 30 secs	↑ ROM, NC in passive stiffness
Kay and Blazevich (2009b)	Ankle plantarflexors	3 x 60 reps (of 60 secs) passive stretching	↓ tendon stiffness at 50%, 70%, 90%, NC at 10%, 30%. Values returned to baseline within 30 minutes
Ylinen *et al* ., (2009)	Hips	4 weeks stretching	↑ ROM, NC in passive stiffness
Morse *et al* ., (2008)	Gastrocnemius	5 stretches, one minute each	↑ compliance of proximal MTU
Halbertsma *et al* ., (1996)	Hamstrings	10 x 30 seconds stretch	↑ ROM ↑ extensibility ↑ maximum moment ↑ stretch tolerance NC passive stiffness
Halbertsma and Goeken (1994)	Hamstrings	Contract-relax stretch 4 x 2/day 10 mins	↑ ROM (5 degrees) ↑ extensibility ↑ maximum moment ↑ stretch tolerance NC in passive stiffness
Resistance exercise			
Blackburn and Norcross (2014)	Hamstrings	6 weeks isometric vs isotonic training	↑ passive knee stiffness in isometric training, NC in isotonic training
Terada *et al* ., (2013)	Knee joint	MVIC 3 x 5 secs	↑ passive knee flexion and extension
Foure *et al* ., (2011)	Ankle plantarflexors	14 weeks plyometrics training	↑ total stiffness ↓ active stiffness
Janecki *et al* ., (2011)	Elbow	2 bouts of eccentric contractions	↑ passive muscle stiffness, including at 5 days post-intervention, NC immediately or 24-hours post
Kay and Blazevich (2009a)	Ankle joint	Isometric contractions of ankle joint with stretching	NC in muscle or tendon length, ↓ force ↓ tendon stiffness. Values returned to baseline within 30 minutes
Torres *et al* ., (2007)	Knee joint	Eccentric contractions with stretching	↑ passive muscle stiffness
Kubo *et al* ., (2002)	Ankle joint	Resistance vs resistance plus stretching	↑ MVC ↑ tendon stiffness NC resting tendon length or CSA. ↓total stiffness during contractions with NC at 30 minutes
Kubo *et al* ., (2001)	Knee joint	12 weeks static and dynamic resistance training	↑ tendon stiffness in static training, NC in dynamic training

Abbreviations: No change (NC), increase (↑), decrease (↓), range of motion (ROM), reps (repetitions), secs (seconds), cross sectional area (CSA)
Maximum voluntary contraction (MVC), maximum isometric voluntary contraction (MVIC), muscle-tendon unit (MTU)

2.8.1 Summary

Because passive stiffness is determined by the number of parallel myofibrils and the length of the MTU, there is potential for passive stiffness to be increased through hypertrophy, and reduced through stretching exercise. During movement, the total passive resistance at a joint may be augmented through altered angles of neighbouring joints, where reduced joint angles reduce muscle length and therefore passive resistance. Such interplay of joint angles would be through active muscle contraction. Active joint resistance is determined by the total number of activated motor units, and will be lower due to reduced activation, and higher if more motor units are activated.

Stretching has been shown to decrease strength and power, whilst having transient or no significant effect on passive joint resistance. Furthermore, the increased joint ROM is associated only with increased stretch tolerance, rather than any structural or mechanical changes within the MTUs. Decreased total and active joint moments following stretching are associated with decreased force production, and not changes in MTU stiffness. However, too much or too little net stiffness is associated with an increased risk of injury and dysfunction.

It is plausible that some exercise interventions lead to physiological adaptations in stiffness (whether passive or active), as demonstrated by the changes following some resistance training protocols. If these are physiological adaptations to improve efficiency, rather than short-term changes during the recovery period, it may be supposed that MTU stiffness adapts specifically to activities of daily living, and such adaptations are augmented by certain physical

activity types. How this information can be utilised in the development of LBP-specific rehabilitation programmes, will depend upon whether there are passive or active elements of MTU stiffness that are influenced by pain.

2.9 Exercise Interventions and LBP

The effectiveness of LBP interventions is often based upon improvements in pain reporting over time, or performance in clinical tests, but lack objective analyses of the specific passive or active mechanical components that were targeted. This makes it difficult to ascertain which specific components of the intervention were most useful, and how these could be improved upon for further study and clinical effectiveness. There is therefore a broad range of studies that utilise a wide variety of techniques to reduce LBP severity and disability, rather than simple studies assessing hip extensors in isolation. The purpose of this section is to summarise findings from a range of studies that aimed to use interventions to modify LBP severity, disability and other related outcomes.

Table 2.2. Findings of studies investigating influence of exercise interventions on LBP severity, disability and related factors

Exercise interventions in LBP

Authors	Intervention	Findings
Irandoust and Taheri (2015)	Aquatic exercise 12 weeks, 3 days/week in men > 65 years	↑ trunk mass ↓ pain severity
Limke *et al* ., (2008)	Addition of 1 vs 2 sets of resistance exercise to rehabilitation	No differences for pain, disability or strength
Jones *et al* ., (2007)	30-minute strength, flexibility and aerobic exercise	↑ ROM ↓ pain severity ↑ muscular endurance
Winter (2015)	A variety of hip stretching and strengthening exercises	↓ pain severity ↓ disability. Strengthening most effective
Nelson-Wong and Callaghan (2010a)	4 weeks, various strengthening exercises	↓ pain severity ↓ co-contraction Values returned to baseline ~15 mins
Shamsi *et al* ., (2015)	16 sessions trunk segmental stabilisation vs traditional trunk exercise	↓ pain severity ↓ disability ↑ lumbopelvic stability No between group differences
Kendall *et al* ., (2015)	6 weeks trunk stabilisation vs stabilisation plus hip strengthening	↓ pain severity ↓ disability ↑ hip strength No between group differences
Lee and Kim (2015)	Hip exercise and lumbar stabilisation exercise, in lumbar stability/instability	↓ pain severity ↓ disability Greatest benefits in lumbar instability group
Franca *et al.* , (2012)	6 weeks, 2 days/week stretching vs trunk segmental stabilisation	↓ pain severity ↓ disability Greatest benefits in stabilisation group
Mostagi *et al* ., (2015)	Pilates vs general exercise, 3 months after 8 weeks, 2 days/week	↑ function in general exercise, NC in pain
Chan *et al* ., (2011)	8 weeks addition of aerobic exercise to physical therapy	↓ pain severity ↓ disability No between group differences
Hill *et al* ., (2011)	Home-based training video with some group physical therapy	↓ disability at 12 months

Abbreviations: No change (NC), increase (↑), decrease (↓), range of motion (ROM)

2.9.1 Summary

Many different treatment approaches have shown some efficacy in reducing pain and disability in LBP subjects. However, interventions often comprise multiple components and are compared against sedentary control subjects, making improvements likely in the intervention groups. Future studies should compare multiple treatment approaches across more subjects, to help elucidate which treatments are the most effective. Overall, interventions focussing on strengthening the hip are more effective than stretching exercises or Pilates. Various treatment approaches include trunk lumbar segmental stabilisation, or 'core stability' exercises, but the benefit of these compared with general hip strengthening remains to be investigated. Whilst there are interventions that attempt to target the active, somatic nervous system, the results of such studies are conflicting, with some studies reporting a worsening of outcome measures.

2.10 General Summary

LBP is one of the leading causes of disability globally, responsible for approximately 83 million disability-adjusted life years. From the surveys examined, between one-fifth and one-third of individuals reported at least one day of LBP during the month prior to completing the survey. Despite being so common, between 85-95% of LBP is classified as non-specific. The traditional view of LBP as a single occurrence, or else multiple independent episodes, is being replaced by the view it can be a long-lasting, chronic condition, punctuated with acute, bouts of recurrence or relapse.

Alterations in motor activation and compensatory patterns have been identified in individuals suffering long-term pain, and research has shown that any such compensations are not short-term pain responses only. Pain itself is associated with a reduction in preferred walking velocity, increased motor activation, increased rigidity and reduced variability in movement of the lumbar spine during walking. It is thought that these compensations are to promote stability and reduce stress and pain in areas of the spine already susceptible to pain. Importantly, research conducted over the past decade has identified passive and active contributions to movement as interacting components that may both be altered by the presence of pain. Due to the different structures responsible for augmenting passive and active components, it is important to understand how these interact in LBP, in order to develop effective assessment and intervention strategies. Further, the manifestation of LBP on an individual level may be compounded by anthropometric, psychosocial and behavioural factors.

2.10.1 Limitations of Previous Studies

Complexities in the study of LBP occur, in part, through the lack of specificity of the condition. With only 5-15% of chronic LBP being of a specific cause (fracture, neoplasm or infection), there are 85-95% of cases that are non-specific. With such a high prevalence of LBP in the population, and such a lack of understanding regarding its cause, any study of LBP will include subjects with potentially very different underlying causes of their condition. This can make

study findings difficult to interpret without over-generalising, limiting the potential usefulness of the research to the individual or medical professionals.

Whilst effective and appropriate clinical assessments should be useful in improving our understanding of the pathogenesis of LBP, there remains a lack of consensus regarding the most appropriate and useful tests, with test results between studies lacking reproducibility, whether through limitations in methodological clarity, or differences in subject populations (such as age, gender, physical activity habits, severity of condition, disability level, level of pain, duration of pain). Conflicting results regarding joint ROM, standard tests and passive stiffness are common, despite very few studies that have attempted to assess these factors together. Further, the variety of clinical and experimental tests has permitted many studies to be published, with very few comparable findings. This is particularly the case for passive measuring of hip extensor stiffness, and muscle activation during trunk and hip movements, with multiple studies adopting varied methodologies and reporting conflicting results.

That there is a functional relationship between the hip extensors and lumbar spine is clear. In various movements there is an alteration in muscle activation of the lumbar spine and hip extensors in LBP subjects, but the paucity of research limits the scope for prediction and determination of passive-active interactions. Further, there is a lack of research specifically on hip extensor passive and active properties in subjects with LBP. Specifically, there is a lack of data regarding hip extensor passive moments, stiffness and strain energy during passive movements, and passive and active moments, power and mechanical work during walking.

Due to limitations in reporting on passive and active components of movement in LBP subjects, it has not been possible to devise treatment protocols that are specific to a particular biomechanical component. There is therefore a broad range of interventions that show some efficacy compared with non-intervention control subjects, but a lack of rationale for a particular approach, coinciding with few studies that compare different approaches to treatment.

2.10.2 Aims of Proposed Research

The aims of the proposed research are as follows:

1. Adapt a force transducer for use as a handheld device for measuring hip extensor passive properties
2. Use a previously validated dynamic biomechanical model to compare passive moments, stiffness and strain energy of LBP and pain-free subjects during supine leg raising
3. Develop a predictive equation to calculate passive hip extensor contributions to total hip moments at various hip and knee angles
4. Perform a gait analysis to compare passive hip extensor moments, total moments, power and work done in LBP and pain-free subjects

3. Development and Application of Handheld Force Measuring Device

3.1 Introduction

Total limb passive resistive properties across a joint can be measured using load cells (Yoon and Mansour, 1982, Lee and Munn, 2000, Halbertsma *et al.*, 2001, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008), or force plates (Freddolini *et al.*, 2014b), in conjunction with electrogoniometers (Lee and Munn, 2000, Halbertsma *et al.*, 2001). Load cells measure resistive forces acting upon the rotating joint, whilst electrogoniometers measure angle, angular velocities and accelerations. Motion tracking systems utilising skin markers can be used in place of electrogoniometers (Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008, Gombatto *et al.*, 2008a). Instrumental straight leg raise equipment and multi-functional dynamometers can also be used for passive joint resistance measurements, but these are not appropriate for measurements of bi-articular muscles, where angles at two joints need to be manipulated.

A custom-built force measuring device has many potential advantages over other commonly-used devices. These include accuracy and validity of measurement for understanding whole joint passive resistance, relatively low-cost, multiple joint applications, and minimal space needed for equipment storage (Yoon and Mansour, 1982, Lee and Munn, 2000, Halbertsma *et al.*, 2001, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008). Dynamic

biomechanical models can subsequently be used to calculate passive moments, stiffness and strain energy during passive testing.

Active MTU properties must be calculated as a component of total biomechanical properties. For assessments of total joint biomechanics during walking, motion capture and force plates can be used (Silder *et al.*, 2008, Whittington *et al.*, 2008). Dynamic biomechanical models can subsequently be used to calculate joint moments, power and mechanical work done (Silder *et al.*, 2008). The models require calculations of body segment parameters, measurement of limb lengths and joint centre of rotation data.

The purpose of this chapter is to describe how a force transducer was adapted for use in measuring passive joint moments. Ethical approval was granted for pilot work and subsequent studies by the ethics committees of both the University of Roehampton and the British College of Osteopathic Medicine.

3.2 Adapted Force Measuring Device

A biaxial, cantilever load cell (QLA263, Futek, US), was adapted for use as a force transducer for the measurement of passive joint moments (figure 3.1). Extension pieces were designed and built to be secured to either end of the load cell. At one end, the extension piece secured the load cell to a handle. A rod was positioned horizontally into the handle so the tester could limit any frontal-plane and transverse-plane rotations of the device when in use. A second rod was placed vertically above the transducer, to give the tester control to limit sagittal plane rotations. To the other end of the load cell a metal cylinder

was added that could be housed within a variety of limb supports or joint braces.

An inclinometer (PTAM27, ASM, Germany) was mounted on the frame of the force transducer, to measure pitch and roll of the device. Two spirit levels were placed on top of the unit to help the tester ensure the transducer was being held level. The inclinometer was necessary to resolve the measured horizontal and vertical vectors to true directions of force in the *x* and *y* planes, respectively. The inclinometer was mounted onto a wooden block, built onto the handle attachment to the load cell (Figure 3.2). Specifications for the construction of the transducer extension pieces can be found in Appendix B.

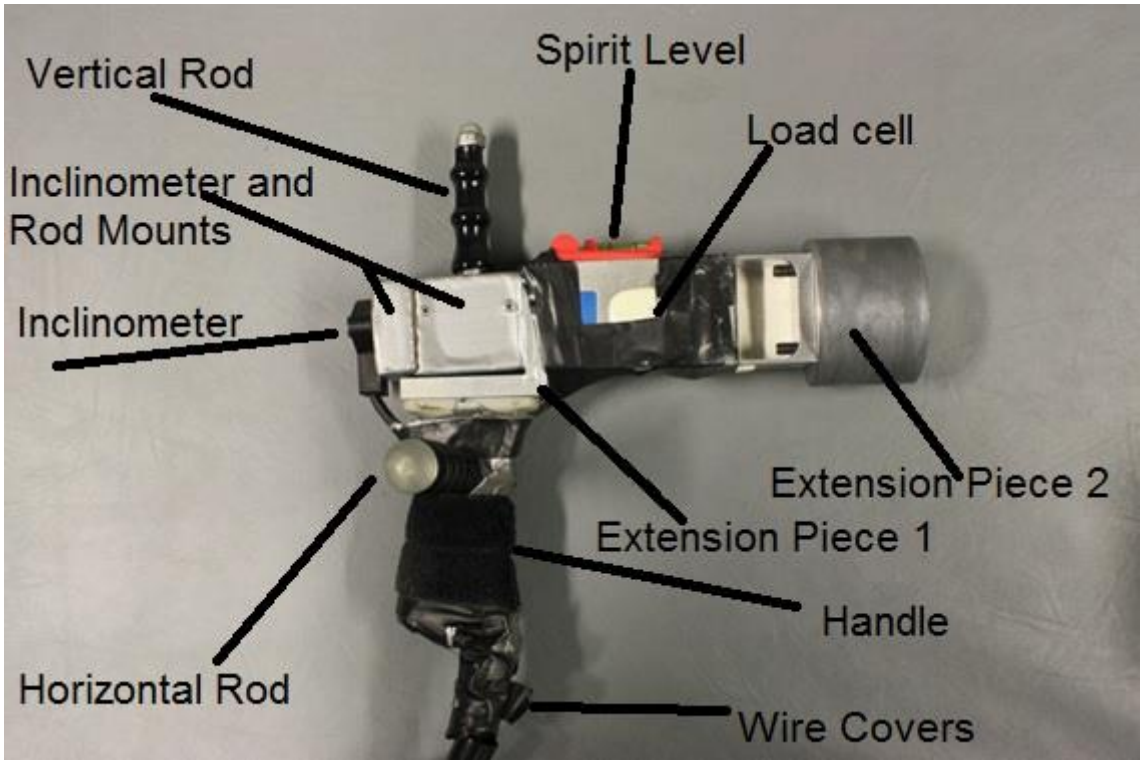

Figure 3.1. Side view of custom-built force transducer

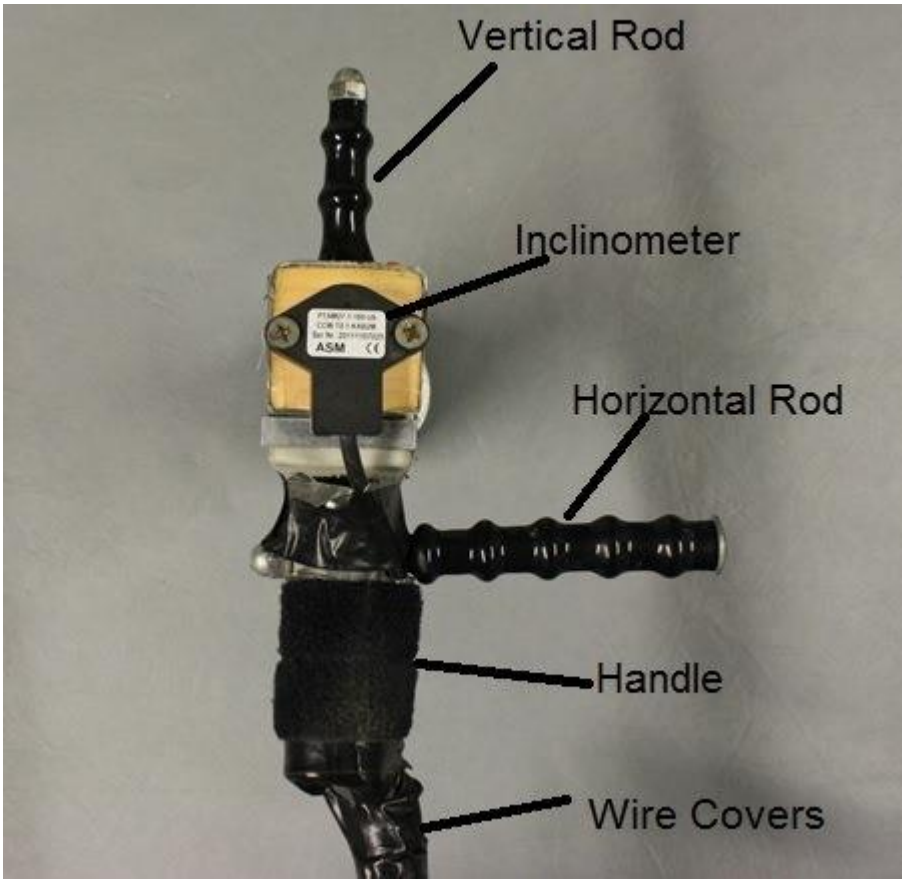

Figure 3.2 Rear view of force transducer, showing position of electro-inclinometer

3.2.1 Ankle Brace

For the purposes of the current investigation, the force transducer was used in conjunction with a custom-built ankle brace, designed to house the transducer with minimal friction, whilst maintaining the ankle in neutral (Figures 3.3 and 3.4). The housing for the force transducer was made from aluminium, smoothed and routinely oiled to ensure minimal friction to transducer rotations. The aluminium housing was attached to aluminium rods that formed the scaffold of the ankle brace, with polystyrene pads to support the lower leg and foot. Straps were added to the brace to secure the lower leg and foot in position. Pilot investigations revealed that some subjects with lower than average leg

length could not be effectively fitted with the ankle brace, and an additional polystyrene pad was available as an effective solution to this issue.

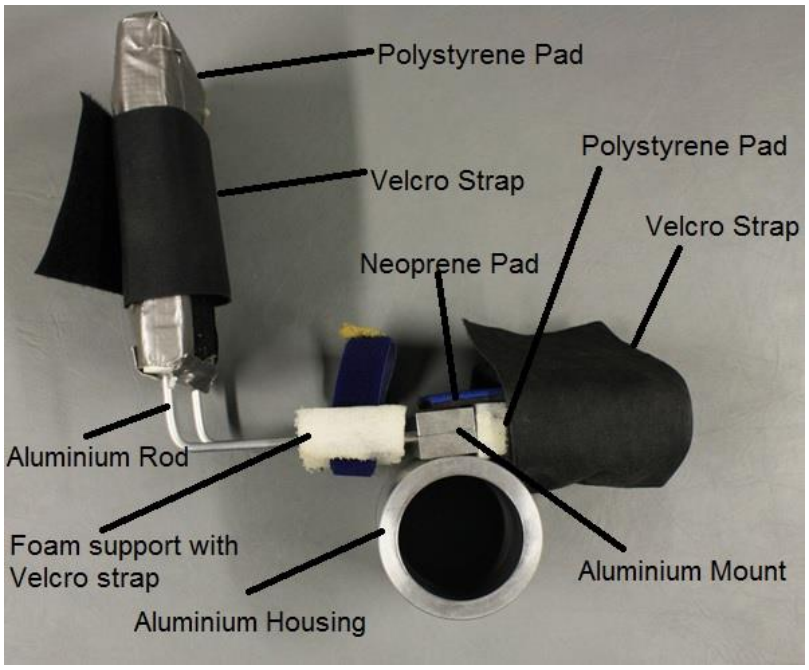

Figure 3.3 Side view of ankle brace

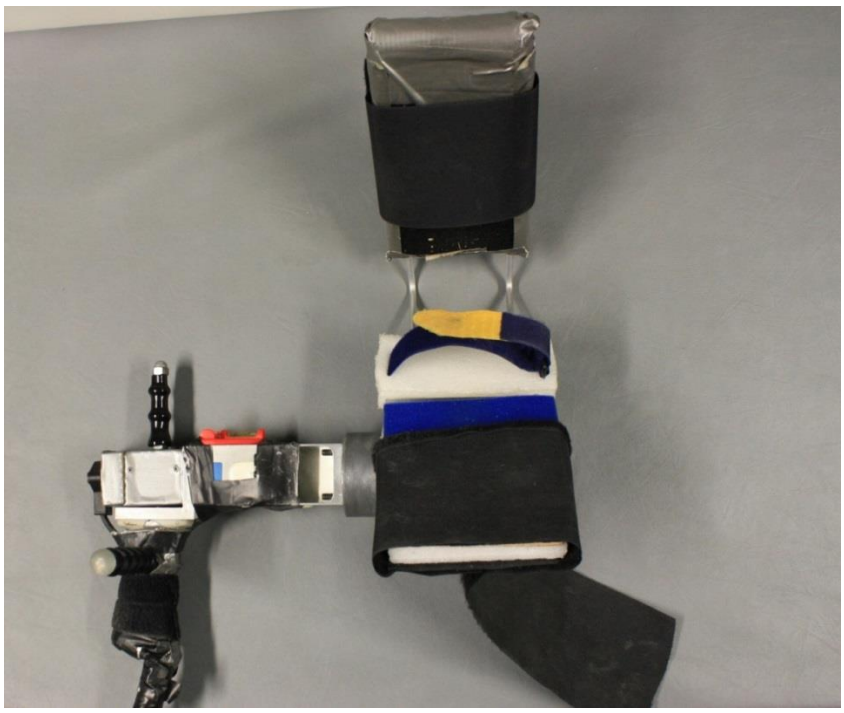

Figure 3.4 Force transducer housed within ankle brace

3.2.2 Knee Braces

A series of braces were used to maintain the knee at a given angle during testing. Four knee braces were pre-formed to make braces at 30 degree increments from 180 degrees (full extension, figures 3.5 and 3.6) to 90 degrees. Pilot investigation showed that these braces were effective in maintaining the knee at angles of 180, 170, 160 and 140 degrees. Lower angles were not possible due to excessive movement of the hip in transverse plane during leg raises. All braces were made from polystyrene pads, reinforced with aluminium rods and secured around the thigh and lower leg using straps. Each brace weighed 200 grams.

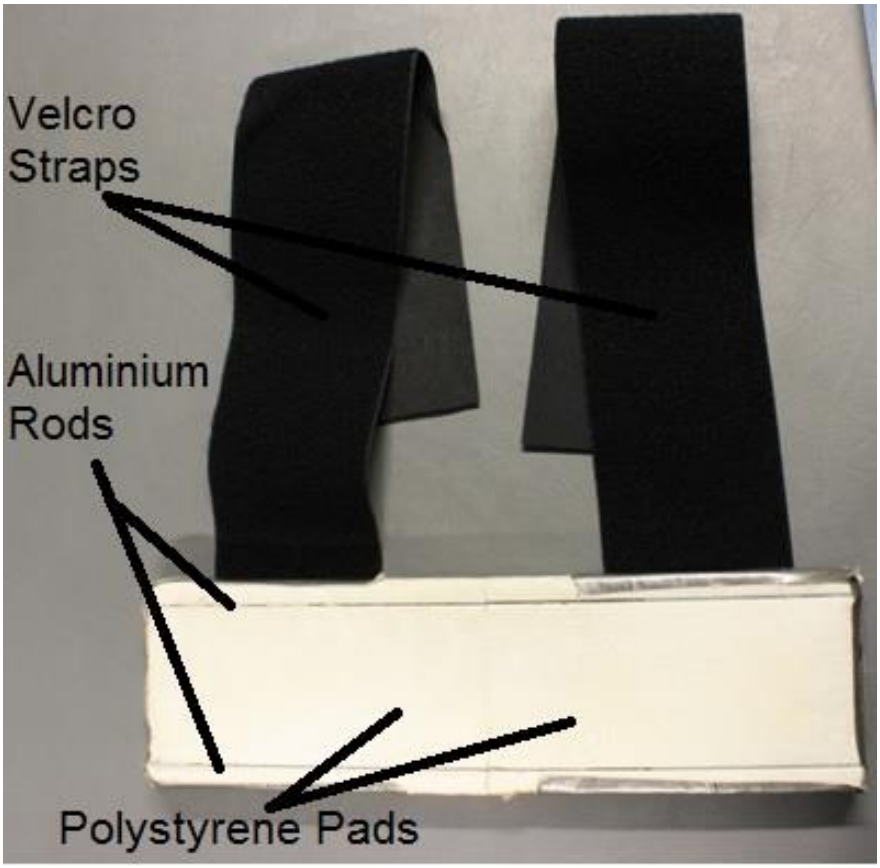

Figure 3.5 180-degree (full extension) knee brace

Figure 3.6 Complete set of four knee braces, which allow the knee to be supported at approximately 180, 170, 160 and 140 degrees, from bottom to top, respectively.

3.2.3 Electro-Inclinometers

During pilot testing it was found that the suitability of electro-goniometers was unacceptably poor, due to the differing movements of the skin on which they were placed, and the actual joint rotations, as measured with a standard goniometer. For this reason, two electro-inclinometers (PTAM27, ASM, Germany) were adapted for this use, housed within Velcro straps that were fixed about the thigh and lower leg, and set to zero degrees with the hip supported in a neutral position and knee straight. These inclinometers were used to measure hip angle and hip angular acceleration, and to monitor knee angle, respectively (Figure 3.7).

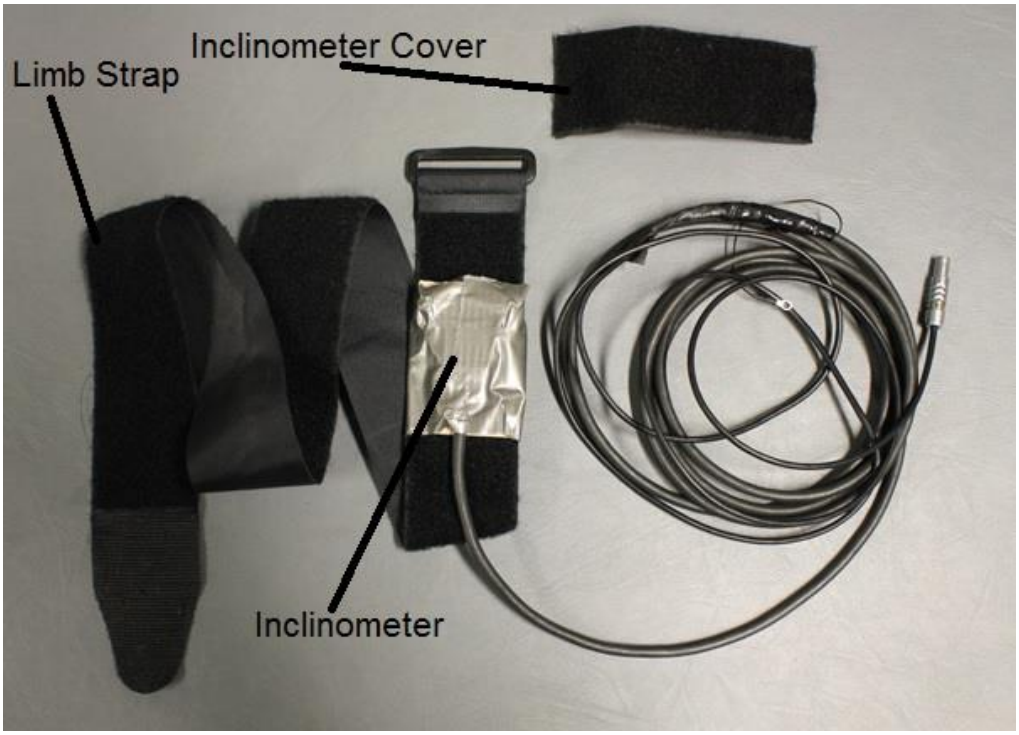

Figure 3.7 Inclinometer secured to limb strap for thigh or shank. A cover was used to limit movement of the inclinometer upon the strap.

3.2.4 Surface Electromyography

Electromyography (EMG) recording during testing was included to provide real-time feedback and to alert the tester to any unusual muscle activity. This aided the tester in ensuring muscles remained inactive and free of stretch reflexes during passive testing, in agreement with the approach used by other investigators (Halbertsma *et al.*, 2001, Silder *et al.*, 2008, Silder *et al.*, 2007, Blackburn *et al.*, 2004a).

3.3 Data Acquisition

The input voltage for each load cell was 15 V and each output was pre-amplified (CSG110, Futek, US) up to 4 V and analogue-digital converted. The input voltage for the inclinometers was 5 V with up to 4 V output. All analogue signals from the load cells and inclinometers were acquired at 50 Hz, and from the EMG electrodes at 1000 Hz, using a data acquisition unit (Datalink, DLK900, Biometrics, UK) with 3 V sensitivity. Load cell and inclinometer data was saved to a personal laptop computer (Dell Precision, M4500, Dell, US) for processing with Matlab programming software (Version 7.3, Mathworks, US). A foot pedal (IS2, Biometrics, UK) was used by the tester to start and stop data collection for each leg raise test.

Due to the manual lifting technique employed for the measurement of passive moments, it was necessary to remove movement artefacts. Passive leg raising generates a stretch of posterior hip tissues, with corresponding exponential moment-angle curves (Lee and Munn, 2000). The inclinometer and load cell outputs were expected to increase in a linear or curvilinear manner. However, the raw output data included additional rotations and forces due to manual corrections of the force transducer and position changes of the operator. As a consequence, movement artefacts were clearly noticeable on the raw outputs, and a 2-Hz low-pass Butterworth filter was determined to be most suitable, in consideration of the time the movement artefacts were produced over, their magnitude, and experimental appraisal of low-pass filtering. The 2-Hz filter was found to be appropriate to retain the overall trend

of inclinometer and force output data whilst minimising operator-induced artefacts.

3.4 Equipment Validity and Precision

3.4.1 Load Cell

The load cell was initially tested by clamping the device to a solid support structure and adding calibrated weights to a solid block extension piece (figure 3.8). The tests were repeated using the cylindrical extension piece that would form part of the force transducer. The position of the load cell was altered to ensure that both cells were effectively tested. The output in millivolts from the load cells was logged and recorded on an Excel spreadsheet (Microsoft Office Excel 2010). Calculations were also made of the load cell in different angles of tilt, from 0 to 10 degrees (the ankle brace would not remain on the force transducer at angles greater than 10 degrees, so these were not tested. Mean, standard deviation (SD), and range data were calculated (examples of calibration and validity tests are included in Appendix C.1). Linear relationships were recorded between load cell load and output (figure 3.9). Overall, with a range from 500 grams to 10 kilograms, total error was less than 0.5 %, including for tests across multiple tilt angles.

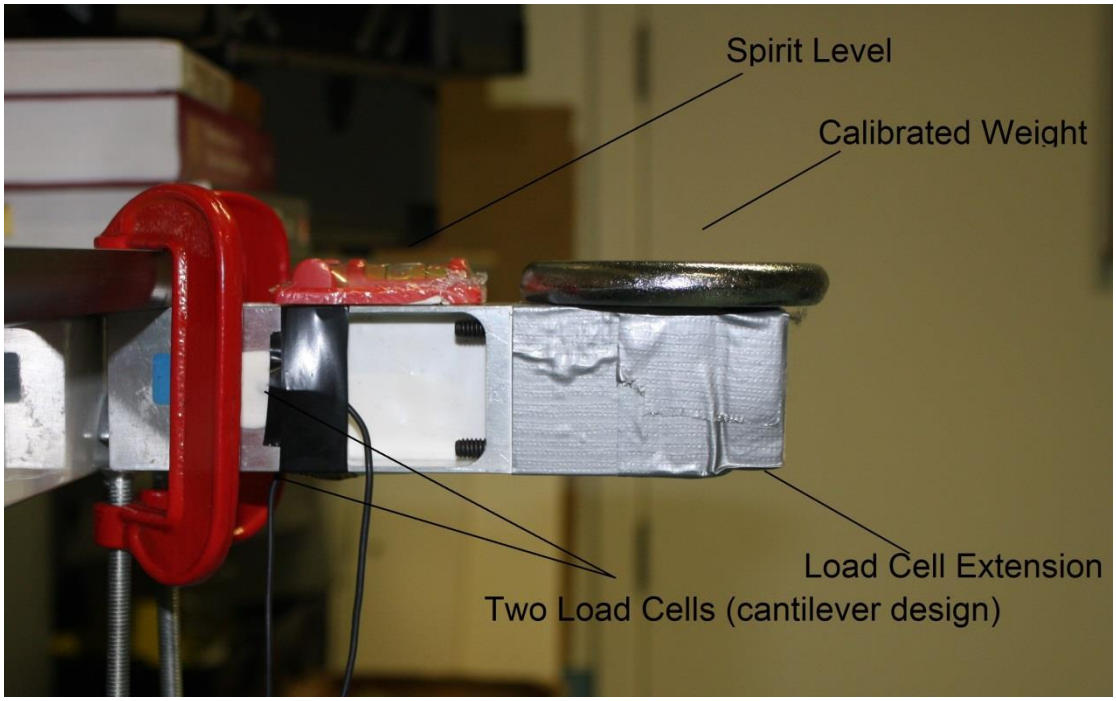

Figure 3.8 Load cell clamped to workstation for calibration, accuracy and precision tests

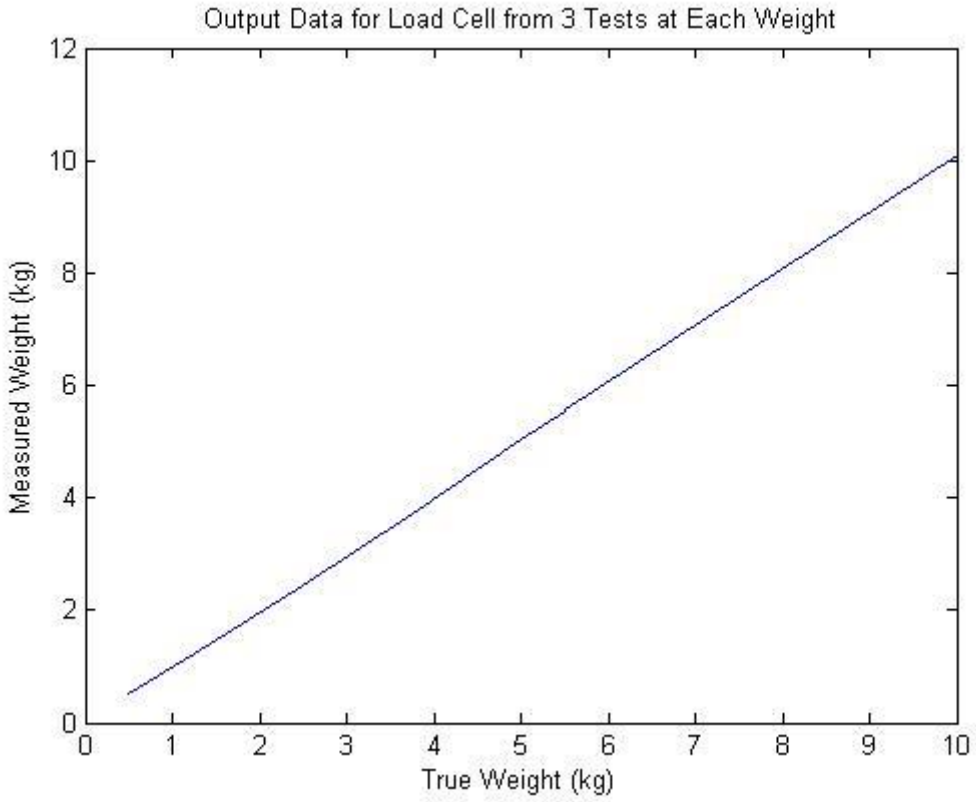

Figure 3.9 Linear relationship between weight measured from load cell output and calibrated weights used for testing.

Load cell calibration and accuracy was tested for each load cell prior to each subject testing procedure. The calibration and accuracy tests were performed using calibrated weights.

3.4.2 Inclinometers

All inclinometers used in the research were mounted onto pivot on a 360-degree plastic protractor. The protractor was fixed to a wooden board, at the base of which was another board and a lockable hinge that would be used to alter the angle of the inclinometer in the sagittal plane (figure 3.10). The millivolt outputs from the inclinometers were measured at different angles on the protractor, and with the wooden board positioned to provide different angles of tilt. The output in millivolts from the inclinometers was logged and recorded on an Excel spreadsheet (Microsoft Office Excel 2010). Mean, standard deviation (SD), and range data were recorded (examples of calibration and validity tests are included in Appendix C.2). A linear relationship was recorded between inclinometer angle and output (figure 3.11). Overall, the error of inclinometers was found to be less than 1 degree for any angle measured up to 140 degrees of rotation.

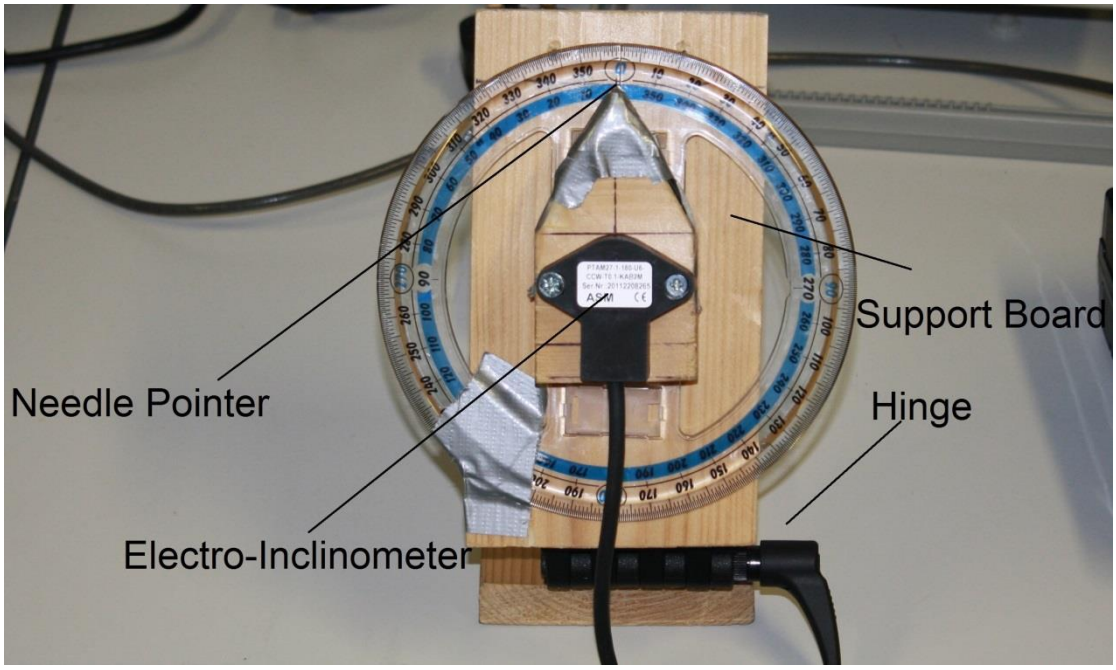

Figure 3.10 Inclinometer mounted upon protractor and hinged board for calibration, accuracy and precision testing

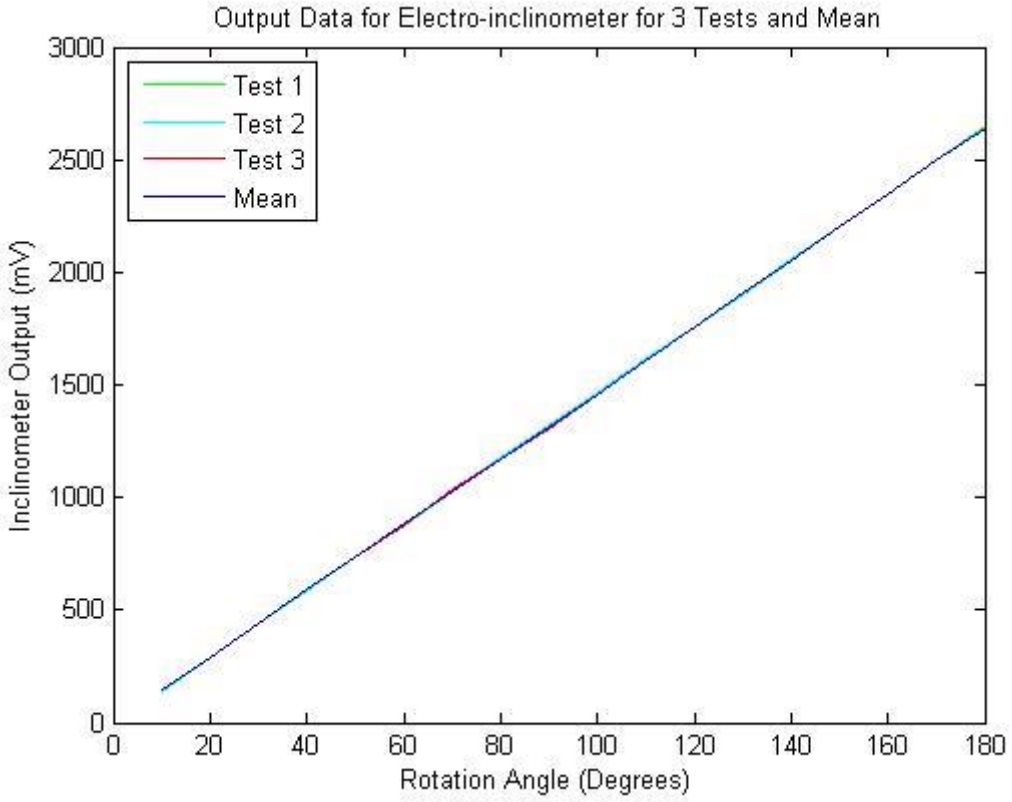

Figure 3.11 Linear relationship between inclinometer output and angle of inclinometer rotation.

The inclinometers were zeroed prior to subject testing and checked for accuracy between 0 and 90 degrees.

3.5 Experimental Procedures

3.5.1 Anthropometry

Anthropometric measurements were taken 3 times at each location and the mean value used in subsequent analysis. If initial values varied by a centimetre or more the measurements were repeated to ensure 3 values were within a centimetre of each other. Measurements included height and the following lower limb lengths: total leg length, hip joint centre to knee joint centre, knee joint centre to ankle joint centre, ankle height above ground, horizontal distance from ankle to longest toe, and foot length. Width of the elbow, wrist, knee and ankle joints were measured using callipers, in addition to hand thickness. Limb segment data was measured in accordance with the parameters set out by Dempster (1955).

3.5.2 Passive Hip Moments

Subjects were required to lie supine on a standard massage table with their hips positioned at one end of the couch and legs supported on a table of the same height. A towel was placed beneath the lumbar spine to support the back and help limit movement of the pelvis and lumbar spine during testing. At the start

of testing the test leg was repositioned off the table and supported by the tester, so as to allow the hip to move freely from extension into flexion.

Two single differential surface EMG electrodes were placed over the biceps femoris and rectus femoris, in accordance with the SENIAM guidelines for electrode placement. The EMG signals were used for real-time feedback to ensure no activity greater than resting levels, and not for subsequent analysis. The initial testing was conducted using a knee brace that maintained the knee close to full extension (180 degrees). Inclinometers were strapped to the thigh and shank, and the foot was positioned and strapped into the ankle brace. The force transducer was inserted into the ankle brace, ensuring the support table was correctly positioned with the non-test leg adequately and comfortably supported, and the test leg able to be moved freely without contacting the table (figure 3.12). This involved positioning the table and test leg off-centre of each subject's midline, with the test leg moved into a neutral position and supported by the tester throughout testing.

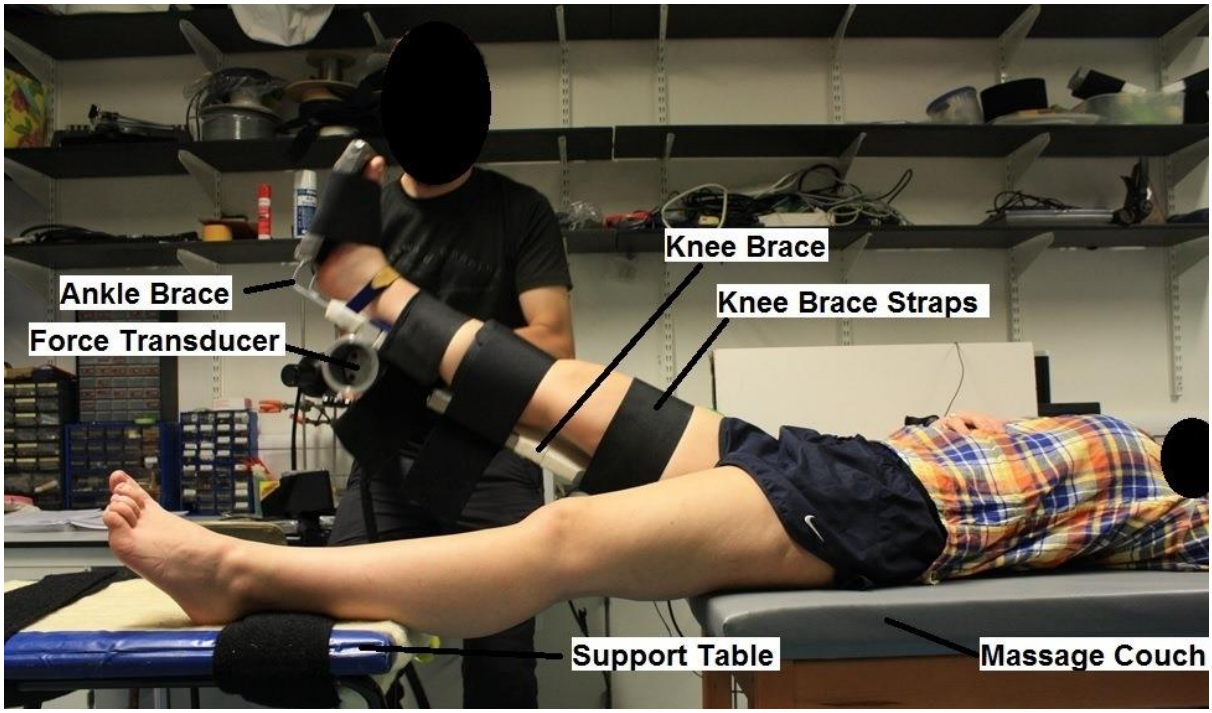

Figure 3.12 Subject set-up showing supine lying position on massage couch with non-test leg supported on table.

In accordance with the procedures of Lee and Munn (2000), the test leg was raised 10 times to precondition the tissues, and to account for any influence of variability in activity levels between subjects immediately prior to testing. The tester supported the test leg using the force transducer and lowered the leg until the hip was in extension (approximately 10 degrees of extension). The tester then performed three leg raises with a minimum of one minute rest between each. Data acquisition began from approximately 10 degrees of hip extension, to limit any influence of inertia from 0 degrees (hip neutral). The procedure was then repeated with each of the remaining knee braces in random order, and with a minimum of two minutes rest between testing at different knee angles. This was to limit the effects of any stretching on the posterior hip tissues. Each leg was fully tested at each knee angle before proceeding to test the other leg.

The velocity of the leg raise was subjectively controlled by the tester. Previous research has shown that velocity of hip flexion does not influence the hip moment at velocities between 1.2 and 92.9 s^{-1} (Yoon and Mansour, 1982). Velocity and angular acceleration of the hip were measured using the hip inclinometer and included within the dynamic biomechanical model used in the analysis (Lee and Munn, 2000). Pilot testing was sufficient to establish the velocities of manual leg raising used in the present study fell within the range that does not influence hip moments.

During testing, the subject was requested to verbally indicate if and when they felt an onset of stretch-related pain or discomfort. This would be used to inform the tester to cease the test at that hip angle, and to ensure the subject was not harmed during testing. The tester monitored the pelvis and torso of the subject during testing to detect any extraneous movement, whilst simultaneously viewing the EMG data being acquired. Any muscle activity above baseline levels would signal the end of that test. Whenever EMG activity was observed to be noticeably above resting levels during testing prior to the end range of motion, the test would be repeated.

3.6 Data Processing

3.6.1 Passive Moment Data

Data from the load cell and inclinometers was used in combination with anthropometric limb segment and mass data to calculate passive hip moments

(*M*). The calculation was based upon the dynamic biomechanical model established by Lee and Munn (2000):

$$M = F_x y_f + F_y x_f + m_{leg}\ddot{x}_{cg} y_{cg} - (m_{leg} g + m_{leg}\ddot{y}_{cg}) x_{cg} - m_{leg} k^2 \ddot{\theta}$$

Equation (1)

Where *Fx*, *Fy*, are the forces applied to the leg to flex the hip joint, x_f, y_f, are the locations of force application to the leg, m_{leg} is the mass of the leg, *g*, is acceleration due to gravity, x_{cg}, y_{cg}, refer to the location of the centre of mass of the leg and *k* is the radius of gyration. $\ddot{x}_{cg}, \ddot{y}_{cg}$ refer to the acceleration of the leg centre of mass, and $\ddot{\theta}$ is the angular acceleration of the leg (figure 3.13).

x_f, y_f, and x_{cg}, y_{cg} are calculated for the left and right legs using the directional cosine method, taking into account the segment positions at different hip and knee angles. x_{cg}, y_{cg} were based upon segment mass parameter data in the literature (Dempster, 1955). *k* of the whole leg is calculated by determining the mass moment of inertia for the individual lower limb segments. A complete description of how each component is calculated is included in Appendix F.

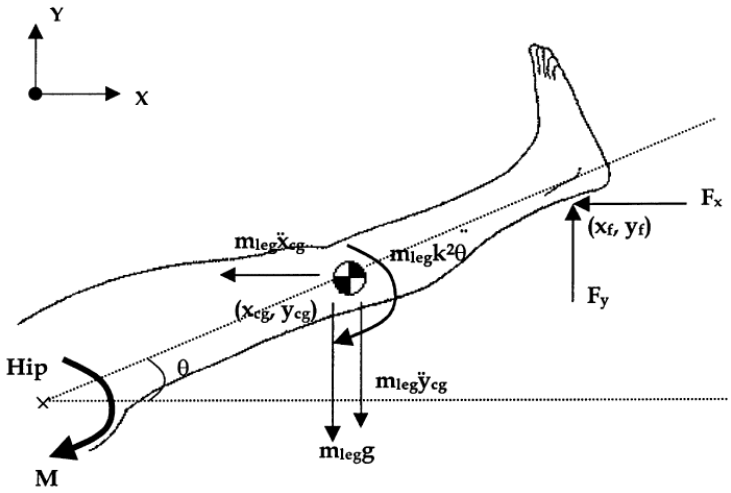

Figure 3.13 Representation of biomechanical model components

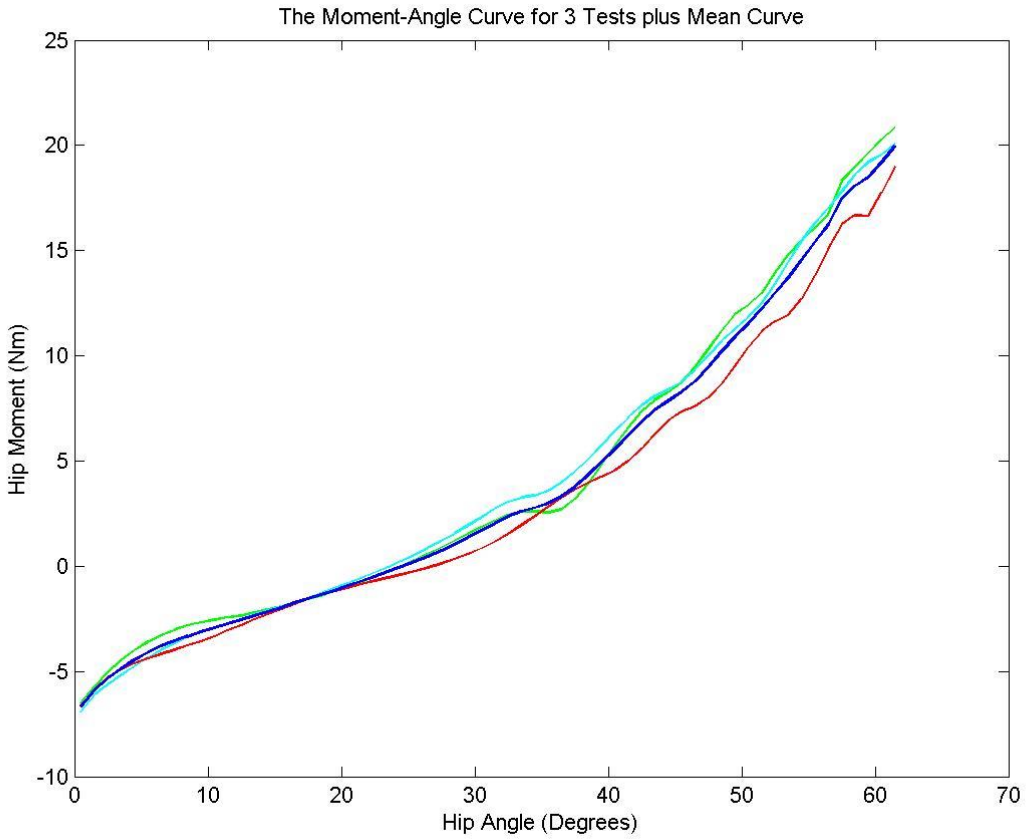

Figure 3.14 Three raw moment curves (green, red and pale blue) and their overall mean curve (dark blue) for a single subject.

From the biomechanical model a mean curve was subsequently determined from the three raw moment-angle curves (figure 3.14). The coefficient of multiple correlation (CMC) and coefficient of variation (CV) were calculated for the 3 lifts for each leg and each knee angle, and the moment-angle graphs visually analysed before continuing. CMC values below 0.8, CV values above 0.3, and any unusual features in the graph (such as those indicating extraneous movements not noticed by the tester during testing), would be reviewed. Where appropriate a single lift would be excluded from further analysis and mean moment-angle data re-calculated. This was to help ensure validity of the data used in the final analysis.

The CMC was calculated from the following formula:

$$CMC = \sqrt{1 - \frac{\sum_{i=1}^{3}\sum_{j=1}^{n}(M_{ij} - \bar{M}_j)^2 / n(3-1)}{\sum_{i=1}^{3}\sum_{j=1}^{n}(M_{ij} - \bar{M})^2 / (3n-1)}}$$

Equation (2)

Where M_{ij} is the *j*th sample point of the *i*th set of the moment data at a given angle. $\bar{M}_j$ is the mean of the three data sets of the *j*th sample. $\bar{M}_j$ is the grand mean of all *n* sample points across the three data sets.

A mean curve of each of the three test measurements was smoothed using cubic spline interpolation (figure 3.15). The pilot data and preliminary test data was analysed and found to have either exponential or linear plus exponential properties. Therefore, the data was fitted with an exponential equation, where *a* and *b* are curve-fitting coefficients, and θ is the angle at the hip (figure 3.16):

$$M = aexp^{b\theta}$$

Equation (3)

Moment-angle data was compared with published data (Yoon and Mansour, 1982, Vrahas *et al.*, 1990, Silder *et al.*, 2007), to ensure values were within published values.

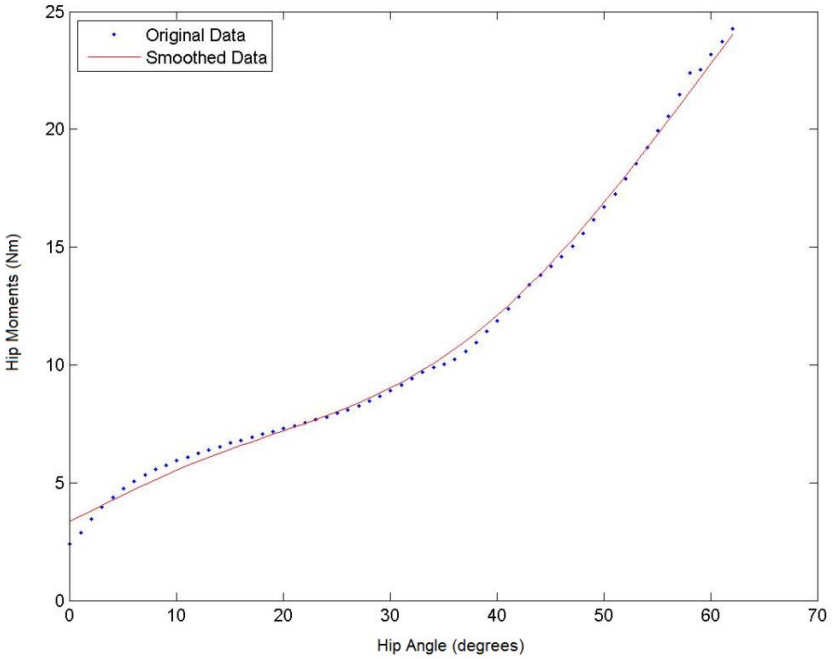

Figure 3.15 Subject example of mean of original data points from 3 raw moment-angle datasets plotted with smoothed mean

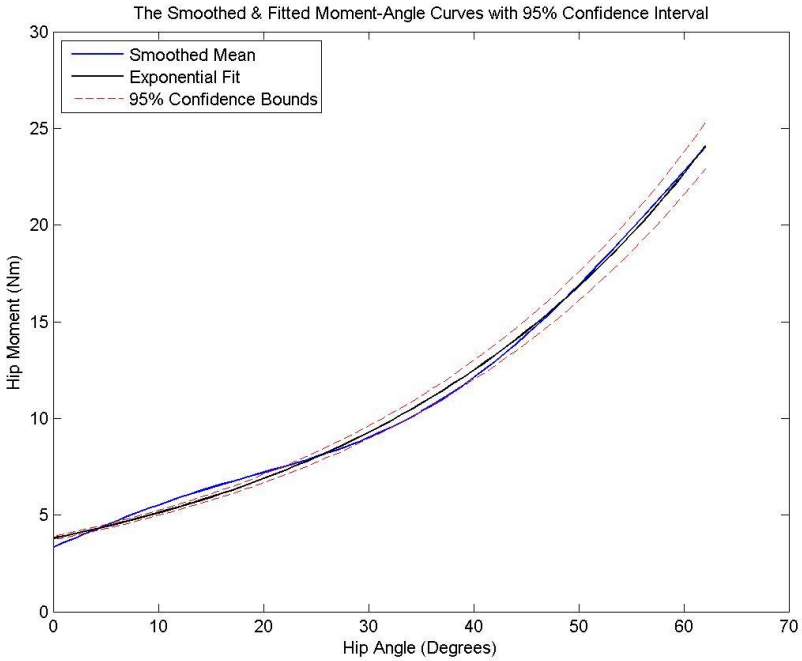

Figure 3.16 Subject example of smoothed mean of 3 raw moment-angle curves with exponential fit and 95% confidence bounds

4. Passive Properties of Hip Extensors in People with Low Back Pain

The following chapter describes the first study of the thesis and was submitted for publication. Hence, the study includes an introduction and methods that have already been described elsewhere in this thesis.

4.1 Introduction

Low back pain (LBP) is one of the leading causes of disability across the world (Buchbinder *et al.*, 2013). Biomechanics of the lumbar spine and hips have previously been investigated in subjects with LBP during gait (Lamoth *et al.*, 2006b), sporting activity (Stuelcken *et al.*, 2010), sit-to-stand and stand-to-sit (Shum *et al.*, 2005a), and other activities of daily living (ADLs) (Shum *et al.*, 2005b). Individuals with LBP have been reported to have modifications in gait velocity, and movements of the hips and spine, when compared with healthy controls (Lamoth *et al.*, 2006b). It has been hypothesised these modifications may be long-term pain-avoidance, or pain-limiting adaptations, which are an effect of chronic, rather than acute, LBP (Lamoth *et al.*, 2004). However, the mechanisms responsible for pain-avoidance strategies remain to be elucidated (Tucker *et al.*, 2009).

In dynamic, biomechanical assessments of the hip, net moments, stiffness and strain energy are comprised of both active and passive components. Active components are derived from active muscle contraction, whether concentric, eccentric or isometric, and so are a property of the amount of muscle fibres and their activation from the nervous system (Marshall *et al.*, 2009, Blackburn *et al.*, 2004a). The passive component, by contrast, is dependent upon the non-contractile tissues, such as the tendon, sarcolemma, endomysium, perimysium and epimysium (Marshall *et al.*, 2009, Blackburn *et al.*, 2004a, Blackburn *et al.*, 2004b, McNair and Stanley, 1996), structural proteins such as titin (Rassier, 2012), and inactive muscle fibres.

It has been proposed that an assessment of hip extensor passive properties may be a more appropriate indication of mechanical limitations than ROM (Gajdosik *et al.*, 1992, Hamill *et al.*, 2009, Marshall *et al.*, 2009, Gombatto *et al.*, 2008b). Such assessments can offer potentially useful information, such as passive stiffness (resistance to change in length), and strain energy (total energy required to move a limb about a joint) (Lee and Munn, 2000). This data can be determined throughout a functional range, or the full ROM about a joint, rather than only at the end ROM. Any changes to passive moments, stiffness and strain energy will reflect structural changes within the tissues. Changes in active properties, by contrast, will relate to changes in the active, contractile elements of muscle, or alterations in their activation. Establishing whether hip passive or active properties are associated with chronic LBP will be helpful in directing physical therapy-based treatments, and for measuring the effectiveness of any such interventions.

The purpose of the present study was to compare the passive properties of the hip extensors in individuals with and without chronic, non-specific LBP. A dynamic biomechanical model was used to calculate passive properties based upon data from an adapted handheld force transducer. Hip extensor passive properties were assessed during a leg raising test, incorporating a variety of pre-determined knee angles, so that the data can be later integrated into a dynamic model of net hip biomechanical properties during ADLs.

4.2 Methods

4.2.1 Subjects

61 participants aged 18 to 50 (LBP n = 31 (male (BPM) n = 15, female (BPF) n = 16), controls n = 30 (male (NPM) n = 15, female (NPF) n = 15)) from the community volunteered for this study. Following consent to participate, subjects were required to complete a medical screening form and International Physical Activity Questionnaire (short form) (IPAQ-SF). LBP subjects were required to complete a Roland Morris Disability Questionnaire (RMDQ) and to rate their level of pain on a visual analogue scale (VAS). During the recruitment process potential subjects were matched for age, body mass, gender and physical activity habits (type and experience). This study was approved by the ethics committees of the University of Roehampton and the British College of Osteopathic Medicine.

Subjects were excluded from the study if they were pregnant or had any tumours, rheumatological or musculoskeletal disorders, tuberculosis, or an injury or infection of the spine, hips or knees during the three months prior to their participation. Subjects were also excluded if they had a history of any dislocation or surgery of the spine or lower limbs, if they were allergic to adhesive tape, or if they had any orthopaedic or electrically-powered medical implant. Female subjects were only eligible for testing during the seven days following the first day of menstruation, to control for any potential effects of the ovarian cycle.

6.6% (n=4) of volunteers failed to meet the inclusion criteria. 57 subjects were tested, with 5% (n=3) excluded due to an inability to relax during the passive testing. Results from 54 subjects were initially examined, with 3.7% (n=2) identified as outliers and excluded. Outliers were determined to be those with data outside of published physiological data for this population. Subgroup data from 52 subjects was included in the final analysis (BPF (n=12), BPM (n=13), NPF (n=12), NPM (n=15)).

4.2.2 Experimental Procedures

Subjects were required to lie supine on a standard massage couch with their hips positioned at one end and legs supported on a table of the same height. Following an initial rest period of 5 minutes, the test leg was raised 10 times to precondition the tissues. The initial testing was conducted using a knee brace that maintained the knee close to full extension (180 degrees). The tester performed 3 leg raises with a minimum of 1 minute rest between each. The procedure was then repeated with 3 other knee braces (170, 160 and 140 degrees) in random order, and with a minimum of 2 minutes rest between testing at different knee angles. The first leg was fully tested at each knee angle before proceeding to test the other leg, and the order of which leg was tested first was randomised.

Passive moments about the hip were measured using an adapted force transducer, comprising a bi-axial cantilever load cell (QLA263, Futek, US), and two analogue inclinometers (PTAM27, ASM, Germany). The force transducer was inserted into a custom-built ankle brace, designed to house the transducer

with minimal friction, whilst maintaining the ankle in neutral. A series of braces were used to maintain the knee in a given angle during testing. Four separate knee braces were pre-formed to secure the knee at each measured angle (180, 170, 160 and 140 degrees). An additional two inclinometers were secured using straps to the thigh and shank, to measure hip angle and hip angular acceleration, and to monitor knee angle, respectively. Following skin preparation, two single-differential surface electromyography (EMG) electrodes (SX230, Biometrics, UK), were placed over the biceps femoris and rectus femoris, in accordance with the SENIAM guidelines for electrode placement.

The analogue signals from the load cell were pre-amplified (CSG110, Futek, US) for each output. All analogue signals from the load cell and inclinometers were acquired at 50 Hz, and from the electrodes at 1000 Hz, using a data acquisition unit (Datalink, DLK900, Biometrics, UK). The outputs were analogue-digital converted, digitally filtered at 2 Hz using a low-pass Butterworth filter, and saved to a personal laptop computer (Dell Precision, M4500, Dell, US) for processing with Matlab programming software (Version 7.3, Mathworks, US). EMG signals were observed during passive testing to ensure no muscular activity, and data was not used for subsequent assessment.

4.2.3 Data Processing

Data from the load cell and inclinometers was used in combination with anthropometric limb segment and mass data to calculate passive hip moments (M).

The calculation was based upon the dynamic biomechanical model established by Lee and Munn (2000) (Lee and Munn, 2000):

$$M = F_x y_f + F_y x_f + m_{leg}\ddot{x}_{cg}y_{cg} - (m_{leg}g + m_{leg}\ddot{y}_{cg})x_{cg} - m_{leg}k^2\ddot{\theta}$$

Equation 1

Where *Fx*, *Fy*, are the forces applied to the leg to flex the hip joint, *Xf*, *Yf*, are the locations of force application to the leg, m_{leg} is the mass of the leg, *g*, is acceleration due to gravity, $x_{cg,}$, y_{cg}, refer to the location of the centre of mass of the leg and *k* is the radius of gyration. $\ddot{x}_{cg}, \ddot{y}_{cg}$ refers to the acceleration of the leg centre of mass, and $\ddot{\theta}$ is the angular acceleration of the leg.

The coefficient of multiple correlation (CMC) and coefficient of variation (CV) were calculated for each of the 3 lifts for each leg and each knee angle, and the moment-angle graphs visually analysed before continuing. The CMC was calculated from the following formula:

$$CMC = \sqrt{1 - \frac{\sum_{i=1}^{3}\sum_{j=1}^{n}(M_{ij} - \bar{M}_j)^2 / n(3-1)}{\sum_{i=1}^{3}\sum_{j=1}^{n}(M_{ij} - \bar{M})^2 / (3n-1)}}$$

Equation 2

Where M_{ij} is the *j*th sample point of the *i*th set of the moment data at a given angle. $\bar{M}_j$ is the mean of the 3 data sets of the *j*th sample. $\bar{M}_j$ is the grand mean of all *n* sample points across the 3 data sets.

Pilot data and preliminary test data was analysed and found to have either exponential or linear plus exponential properties. Therefore, the data was

fitted with an exponential equation, where *a* and *b* are curve-fitting coefficients, and θ is the angle at the hip:

$$M = aexp^{b\theta}$$

Equation 3

The root mean squares error, r-squared and adjusted r-squared values were calculated for the fitted moment-angle curve.

Stiffness (*K*) of the posterior hip tissues was calculated as the derivative of the moment-angle curve (equation (3)):

$$K = \frac{dM}{d\theta}$$

Equation 4

Strain energy (*E*) of the posterior hip tissues was calculated as the integral of the moment-angle curve (equation (3)):

$$E = \int M d\theta$$

Equation 5

4.2.4 Statistical Analysis

Because moments are a property of segment mass and length about a pivot, moments were normalised to body mass and height. For each knee angle assessed the dependent variables (moment, stiffness and strain energy) were recorded at 15 degree increments from 0-60 degrees hip flexion, and at

maximum hip flexion, and averaged across the 3 passive trials performed at that knee angle for each leg, before generating group averages. A multivariate analysis of variance (MANOVA) with an alpha level of 0.05 was used to assess the main effects of group (LBP and control), gender and knee angle. A Pearson Product Moment was calculated to assess correlation between hip flexion ROM and hip moments. Questionnaire data was compared using t-tests and one-way analysis of variance (ANOVA), as appropriate.

4.3 Results

Overall, there was good reliability and repeatability of leg raising tests, within and between groups. There were no significant differences between lift velocities between groups and across knee angles ($P > 0.05$). The CMC had a mean range of 0.89 (SD = 0.12) to 0.97 (SD = 0.03), with no significant differences between groups and across knee angles ($p > 0.05$). The CV had a mean range of 13.82 (SD = 7.37) to 18.64 (SD = 13.5) with no significant differences between groups and across knee angles ($P > 0.05$). RMSE varied between groups and knee angles from 1.09 (SD = 0.69) to 2.49 (SD = 0.77), with no significant differences between groups ($P > 0.05$). Mean r-squared varied between 0.86 (SD = 0.12) and 0.98 (SD = 0.03), with no significant differences between groups ($P > 0.05$). Details of anthropometric and physical activity habits of subjects are included in Table 4.1.

Table 4.1. Anthropometry and Physical Activity Comparisons of all subgroups with results of ANOVA tests (P-value)

	BPF (12)	BPM (13)	NPF (12)	NPM (15)	P
Anthropometry					
Age (years)	30 (7.96)	33 (8.53)	33 (8.78)	29 (7.78)	0.420
Height (m)	1.65 (0.07)	1.78 (0.09)	1.68 (0.04)	1.8 (0.10)	0.000
Mass (kg)	63.5 (6.91)	81.1 (14.41)	68.2 (9.11)	76 (10.27)	0.010
BMI	23.2 (1.80)	25.3 (3.12)	24.2 (3.28)	23.4 (2.32)	0.166
Left ROM (degrees)	81.8 (8.16)	62.8 (15.15)	82.3 (5.58)	67 (9.91)	0.000
Right ROM (degrees)	81 (5.62)	63.5 (10.9)	78.7 (11.23)	68.3 (10.43)	0.000
Physical Activity					
Sitting	30.6 (17.91)	30 (12.43)	33.2 (11.88)	24.1 (15.71)	0.286
Walking	18.4 (21.06)	9.4 (15.53)	6.5 (4.5)	13.4 (16.99)	0.289
Moderate	6.3 (12.17)	7.6 (10.75)	4 (5.57)	4.2 (5.91)	0.696
Vigorous	7.8 (11.72)	5.6 (5.08)	4.9 (1.87)	5.2 (4.28)	0.698
Aerobic	10	8	10	10	0.662
Resistance	7	6	8	7	0.698
Flexibility	3	0	3	2	0.262

Table 4.1 shows anthropometry and physical characteristics of sub-groups. Values shown are the mean (standard deviation) except for aerobic, resistance and flexibility scores, which refer to the number of subjects in each group who participated in these activities. Significant differences ($P < 0.05$) were observed for height, mass and ROM as a product of gender, with no differences when effects of gender removed ($P > 0.05$). Physical activity scores for sitting, walking, moderate and vigorous activity are hours per week. Table 4.2 shows the duration of back pain, VAS and RMDQ scores for LBP subjects. There were no significant differences for males and females in any of these parameters ($P > 0.05$).

Table 4.2. LBP Duration, RMDQ scores and VAS scores of male and female LBP subjects with results of t-tests (P-value)

	Years LBP		RMDQ		VAS	
	Mean (SD)	P	Mean (SD)	P	Mean (SD)	P
BPF	4.652 (3.692)	0.31	4.40 (3.95)	0.509	4.722 (2.252)	0.821
BPM	8.808 (8.488)		4.167 (2.48)		5.00 (2.55)	

Tables 4.3, 4.4 and 4.5 show the moment, stiffness and strain energy values for normalised data. Figures 4.1(a-d) show sub-group moment-angle curves at knee angles of 180, 170, 160 and 140 degrees for normalised data, respectively. All curves displayed an exponential increase in hip moments with increasing hip flexion angle. The Pearson product moment demonstrated no linear correlation between maximum hip flexion and maximum hip moment at any knee angle (range r= -0.23 to r= 0.2). A MANOVA demonstrated no main effects of group, gender, leg, knee angle or hip angle for normalised moments, stiffness or strain energy (P > 0.05).

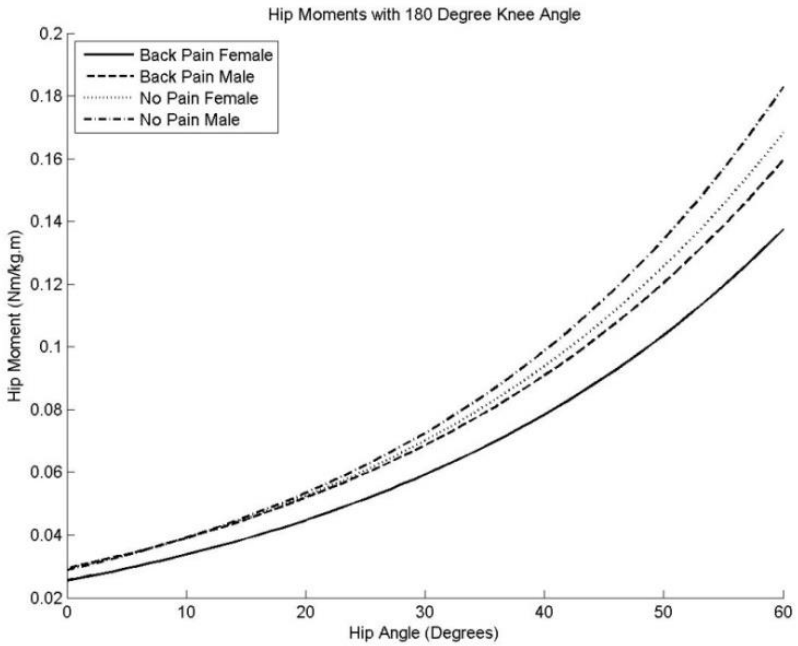

Figure 4.1a. Normalised moment-angle data of sub-groups (180-degree knee angle)

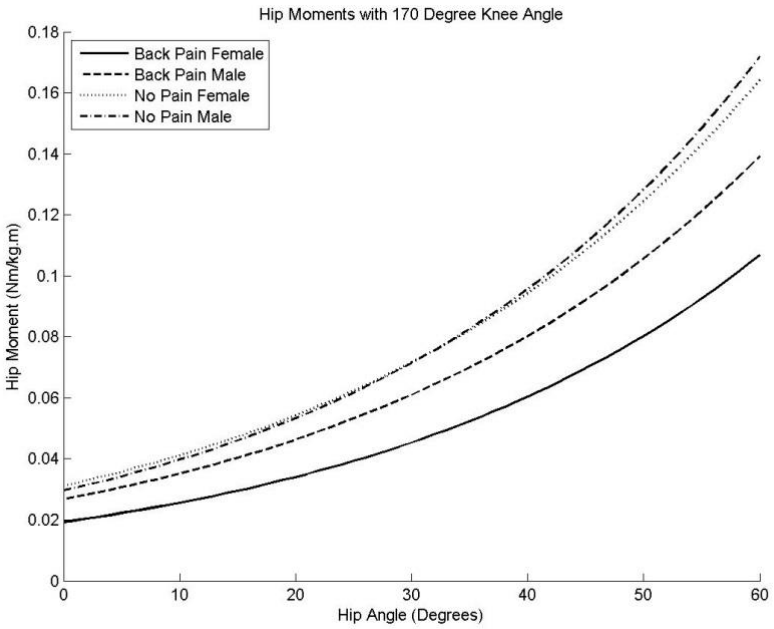

Figure 4.1b. Normalised moment-angle data of sub-groups (170-degree knee angle)

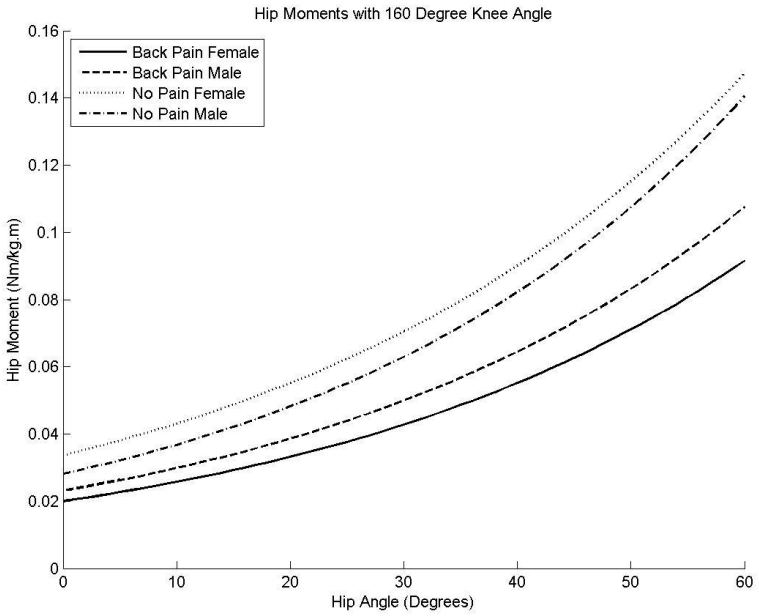

Figure 4.1c. Normalised moment-angle data of sub-groups (160-degree knee angle)

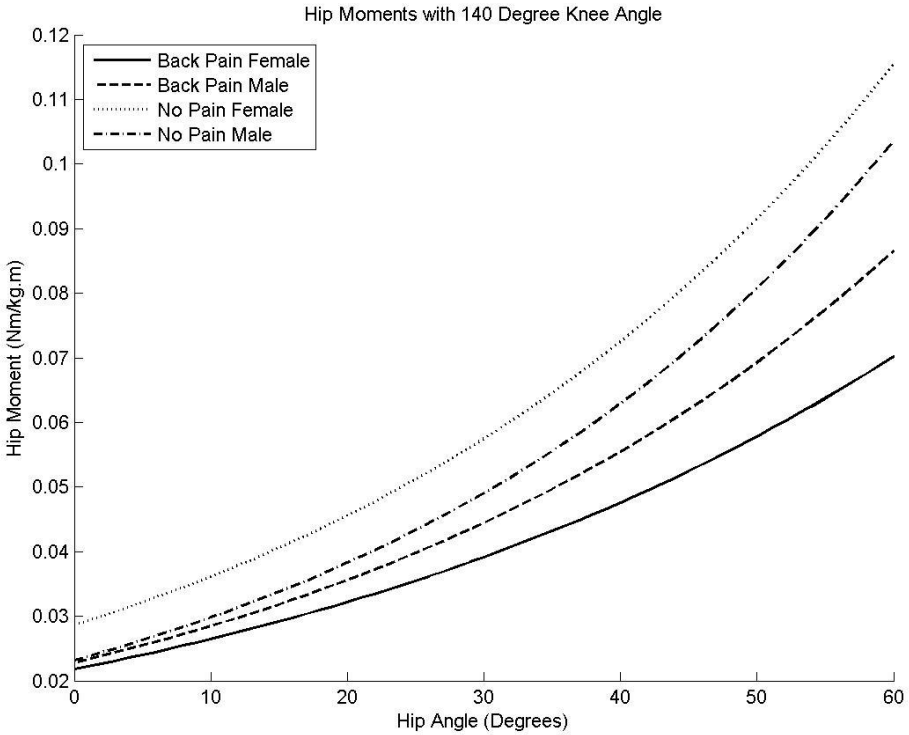

Figure 4.1d. Normalised moment-angle data of sub-groups (140-degree knee angle)

Table 4.3. Normalised hip moments at each knee angle for all subgroups. MANOVA results show no main effects of group, gender or knee angle at any hip angle assessed (P > 0.05)

Hip Angle (Degrees)	Knee Angle (Degrees)	Hip Moments (Nm/(kg.m))				P-value
		BPF	BPM	NPF	NPM	
0	180	0.027 (0.016)	0.029 (0.021)	0.029 (0.013)	0.028 (0.015)	0.890
	170	0.020 (0.012)	0.027 (0.023)	0.031 (0.021)	0.031 (0.019)	
	160	0.021 (0.014)	0.025 (0.021)	0.033 (0.021)	0.029 (0.017)	
	140	0.024 (0.019)	0.024 (0.021)	0.029 (0.015)	0.024 (0.018)	
15	180	0.039 (0.020)	0.043 (0.025)	0.043 (0.017)	0.045 (0.022)	0.860
	170	0.030 (0.016)	0.040 (0.029)	0.045 (0.026)	0.046 (0.023)	
	160	0.030 (0.018)	0.034 (0.025)	0.046 (0.025)	0.042 (0.021)	
	140	0.029 (0.022)	0.033 (0.024)	0.038 (0.018)	0.034 (0.021)	
30	180	0.059 (0.024)	0.068 (0.029)	0.077 (0.047)	0.072 (0.031)	0.812
	170	0.045 (0.020)	0.060 (0.036)	0.079 (0.058)	0.071 (0.029)	
	160	0.042 (0.022)	0.049 (0.031)	0.080 (0.061)	0.062 (0.026)	
	140	0.038 (0.025)	0.043 (0.028)	0.065 (0.047)	0.048 (0.025)	
45	180	0.090 (0.029)	0.107 (0.039)	0.111 (0.055)	0.117 (0.047)	0.753
	170	0.069 (0.025)	0.095 (0.048)	0.111 (0.066)	0.111 (0.039)	
	160	0.062 (0.027)	0.072 (0.038)	0.105 (0.063)	0.093 (0.034)	
	140	0.052 (0.028)	0.061 (0.031)	0.084 (0.050)	0.071 (0.030)	
60	180	0.139 (0.041)	0.144 (0.038)	0.163 (0.065)	0.175 (0.060)	0.733
	170	0.109 (0.032)	0.134 (0.050)	0.159 (0.077)	0.173 (0.058)	
	160	0.094 (0.031)	0.110 (0.045)	0.141 (0.066)	0.142 (0.048)	
	140	0.072 (0.031)	0.090 (0.033)	0.110 (0.053)	0.105 (0.037)	
Max	180	0.249 (0.058)	0.213 (0.067)	0.283 (0.092)	0.252 (0.068)	0.455
	170	0.220 (0.052)	0.190 (0.066)	0.274 (0.096)	0.250 (0.060)	
	160	0.191 (0.047)	0.204 (0.054)	0.241 (0.080)	0.241 (0.057)	
	140	0.142 (0.049)	0.174 (0.044)	0.188 (0.064)	0.212 (0.060)	

Table 4.4. Normalised hip stiffness at each knee angle. MANOVA results show no main effects of group, gender or knee angle at any hip angle assessed (P > 0.05)

Hip Angle (Degrees)	Knee Angle (Degrees)	Stiffness (Nm/(kg.m)) x10^3				P-value
		BPF	BPM	NPF	NPM	
0	180	0.688 (0.264)	0.765 (0.334)	0.768 (0.265)	0.878 (0.386)	0.829
	170	0.524 (0.236)	0.674 (0.405)	0.751 (0.358)	0.837 (0.330)	
	160	0.468 (0.245)	0.548 (0.317)	0.707 (0.280)	0.719 (0.293)	
	140	0.382 (0.224)	0.455 (0.247)	0.562 (0.206)	0.531 (0.262)	
15	180	1.052 (0.337)	1.266 (0.476)	1.165 (0.371)	1.438 (0.581)	0.798
	170	0.802 (0.310)	1.065 (0.570)	1.115 (0.483)	1.316 (0.465)	
	160	0.687 (0.324)	0.812 (0.417)	0.998 (0.356)	1.087 (0.417)	
	140	0.515 (0.279)	0.655 (0.294)	0.774 (0.261)	0.788 (0.337)	
30	180	1.639 (0.494)	2.234 (1.103)	1.879 (0.645)	2.382 (0.929)	0.806
	170	1.260 (0.428)	1.803 (0.904)	1.759 (0.756)	2.104 (0.739)	
	160	1.041 (0.433)	1.235 (0.565)	1.434 (0.519)	1.666 (0.633)	
	140	0.733 (0.358)	0.949 (0.386)	1.108 (0.365)	1.194 (0.458)	
45	180	2.607 (0.898)	3.470 (1.798)	2.831 (0.956)	3.883 (1.521)	0.877
	170	2.052 (0.700)	2.854 (1.642)	2.607 (1.063)	3.424 (1.319)	
	160	1.653 (0.605)	1.949 (0.786)	2.019 (0.711)	2.593 (1.025)	
	140	1.098 (0.505)	1.510 (0.582)	1.515 (0.492)	1.850 (0.688)	
60	180	4.233 (1.847)	5.250 (2.872)	4.333 (1.680)	5.888 (2.087)	0.947
	170	3.505 (1.534)	4.097 (1.829)	3.932 (1.653)	5.320 (2.290)	
	160	2.852 (1.230)	3.293 (1.236)	2.879 (1.067)	4.102 (1.743)	
	140	1.786 (0.969)	2.624 (1.566)	2.092 (0.733)	2.934 (1.174)	
Max	180	7.533 (3.466)	7.759 (4.418)	7.427 (2.557)	8.535 (2.950)	0.977
	170	7.636 (4.621)	6.915 (3.561)	6.974 (2.570)	8.057 (3.075)	
	160	6.672 (4.503)	6.822 (3.489)	5.224 (2.334)	7.259 (3.049)	
	140	4.346 (3.410)	5.588 (3.726)	3.760 (1.681)	6.310 (3.275)	

Table 4.5. Normalised strain energy at each knee angle, taken at 15-degree hip angle increments from 0 degrees to maximum hip angle (Max). MANOVA results show no main effects of group, gender or knee angle at any hip angle assessed (P > 0.05)

Hip Angle (Degrees)	Knee Angle (Degrees)	Strain Energy (J/(kg.m)) BPF	BPM	NPF	NPM	P-value
15	180	0.102 (0.034)	0.119 (0.045)	0.113 (0.037)	0.136 (0.056)	0.806
	170	0.077 (0.032)	0.101 (0.056)	0.109 (0.049)	0.126 (0.046)	
	160	0.067 (0.033)	0.079 (0.042)	0.099 (0.037)	0.105 (0.041)	
	140	0.052 (0.029)	0.065 (0.031)	0.078 (0.027)	0.077 (0.034)	
30	180	0.552 (0.168)	0.697 (0.278)	0.607 (0.190)	0.773 (0.305)	0.793
	170	0.422 (0.153)	0.575 (0.290)	0.577 (0.243)	0.697 (0.242)	
	160	0.356 (0.159)	0.422 (0.206)	0.506 (0.175)	0.567 (0.214)	
	140	0.265 (0.136)	0.338 (0.139)	0.389 (0.128)	0.409 (0.166)	
45	180	1.723 (0.522)	2.147 (0.881)	1.865 (0.585)	2.479 (0.969)	0.813
	170	1.332 (0.438)	1.822 (0.951)	1.748 (0.704)	2.219 (0.783)	
	160	1.097 (0.432)	1.298 (0.571)	1.468 (0.483)	1.744 (0.660)	
	140	0.776 (0.366)	1.032 (0.362)	1.105 (0.357)	1.250 (0.470)	
60	180	4.347 (1.490)	4.837 (1.800)	4.596 (1.543)	5.859 (1.993)	0.881
	170	3.448 (1.152)	4.214 (1.614)	4.250 (1.692)	5.515 (2.059)	
	160	2.803 (0.967)	3.290 (1.246)	3.404 (1.102)	4.319 (1.682)	
	140	1.884 (0.841)	2.608 (0.994)	2.508 (0.819)	3.089 (1.133)	
Max	180	12.314 (4.034)	8.602 (5.236)	13.068 (3.733)	10.723 (4.392)	0.131
	170	11.562 (3.795)	7.867 (4.664)	12.604 (3.799)	10.895 (3.967)	
	160	9.934 (3.598)	10.111 (3.405)	11.083 (3.872)	11.428 (4.015)	
	140	7.124 (3.507)	8.718 (3.416)	8.429 (3.127)	11.020 (4.278)	

4.4 Discussion

Passive hip moments, stiffness and strain energy were measured during supine leg raising tests at 4 predetermined knee angles. The results of the present study demonstrate that there were no significant main effects of group (back pain versus no back pain), gender or knee angle (P > 0.05). The values obtained in the present study for control subjects are in agreement with other published values (Tafazzoli and Lamontagne, 1996, Gajdosik *et al.*, 1992, Lee

and Munn, 2000, Halbertsma *et al.*, 2001, Halbertsma and Goeken, 1994, Halbertsma *et al.*, 1996, Halbertsma *et al.*, 1999). The values are lower than those published by Yoon and Mansour (Yoon and Mansour, 1982) and differences are attributed to the different convention for describing joint angles in that study. These differences have similarly been noted by other investigators (Brand, 1989, Vrahas *et al.*, 1990).

Tafazzoli and Lamontagne (1996) reported the passive elastic moment in terms of percentages of ROM, rather than at defined angles of hip flexion. This approach requires a consistent measure of ROM between individuals and across groups. In the present study, hip flexion ceased during testing when the subject reported uncomfortable tension or pain, but this was considered too subjective to be very closely related to actual leg extension capacity. Hence, it was considered most appropriate to assess at defined increments, and to assess any relationship between maximum ROM and passive resistance independently.

The finding of no significant differences in passive hip moments between LBP and control subjects is in agreement with the majority of published data (Raftry and Marshall, 2012, Marshall *et al.*, 2011a, Halbertsma *et al.*, 2001). The findings differ from those of Tafazzoli and Lamontagne (1996), who reported significant differences between LBP subjects and controls. However, because Tafazzoli and Lamontagne (1996) compared based upon percentages of ROM, rather than increments of hip flexion in degrees, it is difficult to draw comparisons. Further, it may be that their differences between groups were more closely related to the reduced ROM in the LBP group, rather than a predictable relationship between moments and ROM.

In the present study, stiffness was calculated as the derivative of the moment-angle curve, in agreement with the approach by Lee and Munn (2000). The present study found no significant effects of muscle stiffness between groups ($P > 0.05$). This finding is in agreement with that of Raftry and Marshall (2012) but differs from that of Marshall *et al.* (2009) and Tafazzoli and Lamontagne (1996). The limitations in comparing to the Tafazzoli and Lamontagne (1996) study have already been described. The findings of increased stiffness in LBP in that study (Tafazzoli and Lamontagne, 1996) can be expected to reflect their convention for comparisons, and are not directly comparable to the findings of the present study, or that of others (Raftry and Marshall, 2012, Marshall *et al.*, 2009).

Strain energy, in the context of the present study, relates to the total amount of energy required by the operator to flex the subject's hip, against the resistance offered by the hip extensors and other tissues. In active hip flexion, whether performed supine in a test, or else during a functional activity such as walking, strain energy represents the total energy required to flex the hip. During active hip flexion, the total energy required will be influenced by passive-elastic muscle and tendon characteristics, active muscle contraction of all agonistic, antagonistic and synergistic fibres, passive force enhancement, and inertial properties. It is reasonable to suppose that any increase in stiffness of the posterior hip extensors will require an increase in hip flexor muscle activity, to meet the energy requirements for a given hip flexion task (Lee and Munn, 2000, Riener and Edrich, 1999, Mansour and Audu, 1986, Yoon and Mansour, 1982, Vrahas *et al.*, 1990). Thus, any effects of LBP on stiffness and strain energy may lead to alterations in movement efficiency. The present study

found no significant effects of strain energy between groups ($P > 0.05$), suggesting that any mechanical inefficiencies in LBP patients may be due to active, rather than passive components of movement. However, direct measurement of active or total mechanical energy during movement was beyond the scope of the present investigation.

There was no relationship between maximum ROM and passive joint moments, stiffness or strain energy in the present study ($P > 0.05$). This is in agreement with Gajdosik (1991), who reported that different lengthening characteristics of those with short or long hamstrings is most likely a result of extensibility rather than maximum resistance to stretch. Hip ROM was significantly different ($P < 0.01$) between males and females but not between left and right legs, or between LBP subjects and controls (BPF = 81.8 degrees (8.16), NPF = 82.3 (5.58), BPM = 62.8 (15.15), NPM = 67 (9.91) for left leg and BPF = 81 (5.62), NPF = 78.7 (11.23), BPM = 63.5 (10.9), NPM = 68.3 (10.43) for right leg).

Some studies (Marshall *et al.*, 2009, Raftry and Marshall, 2012) use only a limited ROM for assessment of passive properties (20-50 degrees). The rationale for this approach is that this 'common range' enables comparisons to all subjects, independent of total hamstring extensibility (Raftry and Marshall, 2012). However, the range from 0 – 30 degrees is most useful for understanding hip passive properties during gait (Lee and Munn, 2000, Yoon and Mansour, 1982, Vrahas *et al.*, 1990), so the common range may not be transferable to evaluation of function-based activities.

In the present study, there were no significant differences in SLR between LBP and control subjects. This finding is in agreement with that of

Tafazzoli and Lamontagne (1996) and Halbertsma *et al.* (2001). Other investigators (Gajdosik *et al.*, 1992, Gajdosik, 1991, Gajdosik *et al.*, 1990), have reported a good relationship between the SLR and hamstring extensibility, and that the SLR may have potential to indicate passive stiffness (Gajdosik, 1991). Blackburn *et al.* (2004a) reported that, although stiffness and extensibility are related entities, they are not synonymous. Overall, there is a lack of consistency in the literature regarding any relationship between passive joint moments, stiffness and hamstring extensibility (Blackburn *et al.*, 2004a).

It is plausible that the subjects in the present study experienced lower pain levels and less disability than subjects in other studies, and this may be related to their lifestyle habits. Whether this is indicative of a more physically active population in the present study cannot be confirmed, as physical activity and lifestyle habits were not reported in the comparative studies (Tafazzoli and Lamontagne, 1996, Gajdosik *et al.*, 1992, Blackburn *et al.*, 2004a, Halbertsma *et al.*, 2001, Halbertsma and Goeken, 1994, Halbertsma *et al.*, 1996, Halbertsma *et al.*, 1999, Yoon and Mansour, 1982, Silder *et al.*, 2007). Further, whether physical activity habits interact with LBP severity is beyond the scope of the present study to evaluate.

In agreement with other investigators (Lee and Munn, 2000, Vrahas *et al.*, 1990), passive moments were relatively small for hip and knee angles similar to those of normal gait (approximately 30 degrees of hip flexion with the knee straight). In the present study, passive hip moments varied between a lowest of 6 Nm (2.5) and a highest 10 Nm (5.6) as means (SD) of the BPF and BPM groups, respectively, at 30 degrees of hip flexion with the knee fully extended. Lee and Munn (2000) reported mean hip moments at 30 degrees of

3.2 Nm, and Vrahas *et al.* (1990) reported mean hip moments of 2.2 Nm. The differences in values between those in the present study and those of other investigators (Lee and Munn, 2000, Vrahas *et al.*, 1990) may be due to population differences, or any minor errors due to different methodologies. During gait, net moments have been reported to reach approximately 60 Nm (Whittington *et al.*, 2008). Thus, it may be that passive structures contribute between 5% and 20% of the net moments during gait, and thereby contribute to overall movement efficiency, even if to a relatively small extent. Such an influence should be included when modelling joint moments, as this study demonstrates the hip cannot behave like a frictionless 'pin' or hinge during movement, as assumed in inverse dynamics models.

The potential for the influence of passive joint moments to be altered in individuals with musculoskeletal and/or neuromuscular pathologies is worthy of investigation (Yoon and Mansour, 1982). Further, increases in muscle stiffness will increase the passive component of gait (Lee and Munn, 2000), and whether this increases the percentage passive contribution to net moments, or interacts differently with the active component, requires further investigation (Silder *et al.*, 2007). Failing to account for the passive contribution to net moments during ADLs may lead to an overestimation of muscle force and incorrect predictions of muscle activation and timings (Mansour and Audu, 1986).

The finding that passive structures are not altered in LBP has important implications for manual therapists. Any differences in clinical assessments of hip extensor characteristics between LBP and control subjects may be related to active, rather than passive, components. Manual therapy that targets structural adaptations, such as increased muscle length from stretching to

improve extensibility, is not supported by the findings of the present study. Rather, it may be that the active components of hip extensors should be targeted, although further investigation is required to assess active contributions to hip flexion during ADLs. Should active differences between LBP and control subjects be confirmed, further research will be required to determine the most appropriate interventions to effectively target neuromuscular characteristics, or other aspects of active muscle behaviour.

4.5 Conclusions

LBP did not significantly affect passive joint moments, stiffness or strain energy of the hip during passive leg raising. The magnitude of the passive hip moment is not insignificant, and should be taken into account in biomechanical modelling of forces acting on the hip. More research is required to further our understanding of the interaction between active and passive moments during everyday activities, and how such interaction may be altered in people with back pain.

5. A Predictive Equation for the Contribution of Passive-Elastic Structures to Net Moment Calculations using a Bi-Articular Model

5.1 Introduction

Hip joint moments during dynamic tasks are commonly modelled using the inverse dynamics method, based upon the Newton-Euler equations. In the inverse dynamics approach, the hip joint is considered to behave as a frictionless line-hinge (Koopman *et al.*, 1995), without appreciable interaction of passive structures. However, investigators have reported that passive structures about the hip joint will contribute to the net moments measured during dynamic tasks, such as gait (Silder *et al.*, 2007, Silder *et al.*, 2008, Whittington *et al.*, 2008).

Although various optimisation techniques have been developed to improve accuracy of inverse dynamics models (Koopman *et al.*, 1995, Ren *et al.*, 2007, De Groote *et al.*, 2010), these do not take into account the passive-elastic influences of tissues crossing the joint. Of the optimisation techniques reviewed, only De Groote *et al.* (2010) attempted to incorporate a muscle-tendon unit (MTU), physiology-based optimisation technique, although this was based on MTU activation rather than passive influences.

Passive joint resistance is a property of the non-contractile tissues, such as the tendon, sarcolemma, endomysium, perimysium and epimysium (McNair

and Stanley, 1996, Blackburn *et al.*, 2004a, Marshall *et al.*, 2009), structural proteins such as titin (Rassier, 2012), and inactive muscle fibres. The stretch of these tissues during movement generates a passive joint moment (Silder *et al.*, 2007). Passive joint resistance can also be increased due to local pathologies, such as rheumatoid arthritis and osteoarthritis, and any other disease affecting the joint capsule surface or synovial fluid.

Passive joint resistance has also been shown to increase with age (Wolfarth *et al.*, 1997, Silder *et al.*, 2008), due to a loss of elasticity in soft tissues, and the replacement of damaged tissues with less elastic scar tissue (Wolfarth *et al.*, 1997). Should changes in passive characteristics follow injury (Mattyasovszky *et al.*, 2010) or pain (Halbertsma *et al.*, 2001), it would be of use to understand how these might influence net moments. Such information could be useful in directing rehabilitation techniques, as establishing whether interventions should target passive or active moments is directly relevant, as is the development of approaches to measure the efficacy of any such interventions. Such an approach could be expanded to enhance our understanding of training adaptations, and may have use in sports performance programme optimisation, and sports injury prevention.

Importantly, the passive structures involved in movement contribute to the absorption, storage and transmission of mechanical energy (Yoon and Mansour, 1982, Marshall *et al.*, 2009). Thus, the passive structures have a direct influence on the bioenergetics of dynamic tasks, particularly across bi-articular joints, and contribute to the energy efficiency of movement (Yoon and Mansour, 1982, Umberger, 2010, Zelik *et al.*, 2014). Passive-elastic influences at all joints of the lower limb may reduce the energy costs of locomotion (Zelik

et al., 2014, Takahashi and Stanhope, 2013), although the specific contributions to individual joints is poorly understood and requires improved modelling.

In shod walking and running, the stiffness of the sole has the potential to interact with the wearer's forefoot stiffness (Oleson *et al.*, 2005), so an understanding of dynamic changes in forefoot stiffness during gait is of direct relevance to performance shoe design. An improved understanding of the passive influences on mechanical energy during gait is also important for the development of efficient lower-limb prosthetics. Experiments to improve understanding of prosthetic design are aiding in our understanding of passive-elastic contributions to the bioenergetics of normal human gait (Takahashi and Stanhope, 2013). An improved understanding of the contributions of passive structures is improving robot design in passive-dynamic walking robots (Collins *et al.*, 2005). Thus, understanding passive-elastic influences on movement is beneficial for a wide range of medical and scientific fields, including injury prevention and rehabilitation, understanding the effects of pathology, optimising training protocols, and the development of footwear, prosthetics and robots.

Despite the potential for passive structures to contribute to joint moments and energetics of dynamic tasks, few investigators have attempted to measure and integrate passive and active contributions to net moments (Yoon and Mansour, 1982, Vrahas *et al.*, 1990, Silder *et al.*, 2007, Silder *et al.*, 2008, Whittington *et al.*, 2008). Of these, only one concluded that the passive contribution is negligible (Vrahas *et al.*, 1990), whereas others reported the contrary (Silder *et al.*, 2007, Silder *et al.*, 2008, Whittington *et al.*, 2008). Further, only one research group (Silder *et al.*, 2007, Silder *et al.*, 2008, Whittington *et al.*, 2008) has modelled bi-articular muscles. An understanding

of passive and active contributions of bi-articular muscles is essential for modelling force and energy absorption, storage and release across neighbouring joints.

In the present investigation, passive hip moments were calculated at four fixed knee angles, so as to determine the interaction of the hip and knee joint angles with passive hip moments. This information can be used in the design of investigations for understanding the passive and active contributions to net joint moments, power and biomechanical efficiency. The purpose of the present paper was to develop a predictive equation for the calculation of passive hip moments using a bi-articular model.

5.2 Methods

5.2.1 Overview

Hip moment-angle data from 52 subjects, measured during supine passive leg raising tests, was reassessed for development of the predictive equation. The original study compared physically healthy and active subjects, with or without LBP, and found no difference in the passive joint resistance between LBP and control groups.

Full details of the original investigation are included in the previous chapter. Data was pooled to allow for up to 104 measurements at each hip angle. Due to an inability of some subjects to reach the greater hip angles, or movement anomalies, some data was excluded from further analysis. Table 1

shows the number of measurements obtained at each knee and hip angle. A 180-degree knee angle refers to the knee in full extension (neutral).

Table 5.1. Total number of measurements from all test subjects at each hip and knee angle for analysis.

Number of Measurements	Knee Angle (degrees)	Hip Angle (degrees)					
		0	15	30	45	60	75
	180	88	93	96	93	80	46
	170	85	95	98	97	92	64
	160	86	97	99	99	99	87
	140	87	95	99	99	99	98

5.2.2 Data Processing

Moment-angle data for both absolute and normalised moments were included in this investigation. Normalisation was based upon body mass and height. Hip moment-angle data was originally determined from 0 degrees (hip neutral) to maximum hip range of motion. Because the number of samples reduced as hip angle increased, due to individual limitations in ROM, the range 0 to 75 degrees was used for analysis.

Passive hip moments were calculated based upon the dynamic biomechanical model developed by Lee and Munn (2000):

$$M = F_x y_f + F_y x_f + m_{leg}\ddot{x}_{cg} y_{cg} - (m_{leg} g + m_{leg}\ddot{y}_{cg}) x_{cg} - m_{leg} k^2 \ddot{\theta}$$

Equation (1)

where *Fx*, *Fy*, are the forces applied to the leg to flex the hip joint, *Xf*, *Yf*, are the locations of force application to the leg, m_{leg} is the mass of the leg, *g*, is acceleration due to gravity, $x_{cg,}$, y_{cg}, refer to the location of the centre of mass of the leg and *k* is the radius of gyration. $\ddot{x}_{cg}, \ddot{y}_{cg}$ refer to the acceleration of the leg centre of mass, and $\ddot{\theta}$ is the angular acceleration of the leg. The ensemble mean curve of three lifts, smoothed using cubic spline interpolation and fitted with an exponential function, was used for further analysis.

The dynamic biomechanical model was used to calculate passive hip moments during leg raising at four knee angles. From the four mean curves for each subject, a 3-D surface plot was fitted in Matlab to best represent the relationship between hip moments, hip angle, and all knee angles between 140 and 180 degrees. In agreement with other researchers (Lee and Munn, 2000, Silder *et al.*, 2007), passive hip moments were found to increase exponentially as a function of hip angle. By introducing a variable knee angle component, it was found that this contributed a linear component to the equation. A predictive equation was created to determine hip moments based upon a linear knee angle component, an exponential hip angle component, and a constant.Visual and residual analyses were used to adjust equation coefficients for the individual datasets, derived from the surface-fitting programme.

Passive hip moments with 95% confidence intervals were recalculated using the derived predictive equation. Root mean squared error (RMSE) and adjusted r-squared were calculated to assess goodness of fit of each surface-plot. The processing and analysis assessed both absolute and normalised passive hip moments.

5.3 Results

Tables 5.2(a) and 5.2(b) show the ensemble mean moment-angle data, standard deviation (S.D.), and upper and lower 95% confidence bounds (C.I. High, C.I. Low, respectively), for absolute and normalised moments, respectively.

Table 5.2(a) Absolute passive hip moments (Nm) at each hip and knee angle

Knee Angle (degrees)	Hip Moments (Nm)	Hip Angle (degrees) 0	15	30	45	60	75
180	Mean	3.627 (2.530)	5.506 (3.259)	8.742 (4.444)	13.489 (5.995)	18.949 (6.412)	24.932 (7.512)
	C.I. Low	3.107	4.836	7.839	12.25	17.516	22.686
	C.I. High	4.147	6.175	9.645	14.727	20.381	27.177
170	Mean	3.531 (2.855)	5.175 (3.593)	7.996 (4.865)	12.123 (6.319)	19.916 (8.016)	23.833 (9.428)
	C.I. Low	2.96	4.456	7.032	10.859	16.278	21.505
	C.I. High	4.102	5.893	8.959	13.387	19.554	26.161
160	Mean	3.458 (2.679)	4.850 (3.271)	7.210 (4.521)	10.317 (5.439)	15.176 (6.808)	21.656 (7.704)
	C.I. Low	2.922	4.195	6.314	9.241	13.828	20.018
	C.I. High	3.994	5.504	8.105	11.394	16.524	23.294
140	Mean	3.225 (2.591)	4.291 (3.044)	6.041 (3.940)	8.384 (4.615)	12.031 (5.659)	17.707 (7.655)
	C.I. Low	2.712	3.691	5.276	7.488	10.932	16.214
	C.I. High	3.738	4.891	6.805	9.279	13.129	19.199

Table 5.2(b) Normalised passive hip moments (Nm/(kg.m)) at each hip and knee angle

Knee Angle (degrees)	Hip Moments (Nm/(kg.m))	Hip Angle (degrees) 0	15	30	45	60	75
180	Mean	0.028 (0.016)	0.043 (0.021)	0.070 (0.034)	0.108 (0.045)	0.158 (0.056)	0.216 (0.070)
	C.I. Low	0.025	0.038	0.063	0.098	0.145	0.195
	C.I. High	0.031	0.047	0.077	0.117	0.170	0.237
170	Mean	0.027 (0.019)	0.040 (0.025)	0.064 (0.039)	0.097 (0.049)	0.144 (0.061)	0.204 (0.079)
	C.I. Low	0.023	0.035	0.056	0.087	0.132	0.184
	C.I. High	0.031	0.045	0.072	0.107	0.157	0.223
160	Mean	0.027 (0.019)	0.038 (0.023)	0.058 (0.039)	0.083 (0.045)	0.122 (0.053)	0.176 (0.058)
	C.I. Low	0.023	0.033	0.051	0.074	0.112	0.164
	C.I. High	0.031	0.043	0.066	0.092	0.132	0.189
140	Mean	0.025 (0.018)	0.033 (0.021)	0.048 (0.033)	0.067 (0.037)	0.095 (0.041)	0.139 (0.050)
	C.I. Low	0.021	0.029	0.042	0.059	0.087	0.129
	C.I. High	0.029	0.038	0.055	0.074	0.103	0.149

From the present investigation, the following equation was developed to predict passive hip moments ($M_{passive}$) during flexion:

$$M_{passive} = a.\theta_{knee} + b.exp^{c.\theta_{hip}} + d$$

Equation (6)

where θ_{knee} is the angle at the knee, θ_{hip} the angle at the hip, and *a*, *b*, *c*, and *d* are the equation coefficients. This equation shows that the knee angle contributes a linear component, whilst the hip angle contributes an exponential component.

The coefficients were calculated for the 104 data sets, for both absolute and normalised passive hip moments. The predictive equation coefficients are shown in Table 5.3(a) for absolute moments, and table 5.3(b) for normalised moments. The wide spread of values for each coefficient is demonstrative of interactions between each coefficient, and a lack of any relationship between coefficients and different subjects. Hence, it is appropriate that the coefficients be calculated on an individual basis, rather than estimated based upon averaged population norms.

The exponential component is well-established in the literature on passive hip moments during straight leg raising (Silder *et al.*, 2007, Lee and Munn, 2000), and supported through the findings of the present investigation (chapter 4). A plot of the pooled, normalised hip moment data illustrates the linear component contributed by varying knee angle during passive leg raising (figure 5.1).

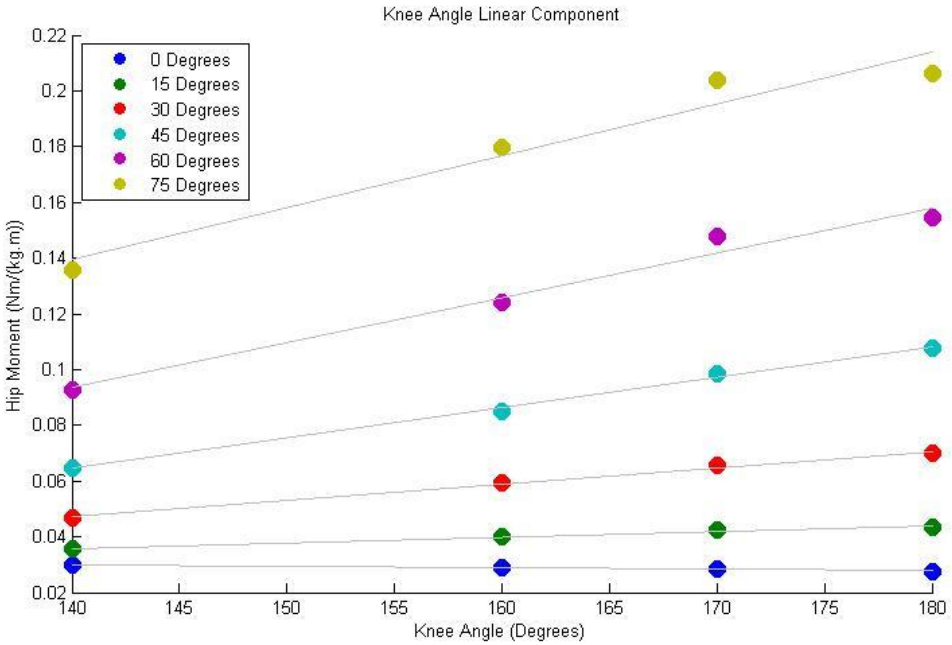

Figure 5.1 Linear contribution of varying knee angle to hip moments during passive leg raising, determined by line of best fit through mean hip moment data at each knee angle. Values were determined by averaging pooled hip moment data from all subjects.

To calculate individual subject data, the mean moment-angle curves from each set of thee leg lifts, and at each of the four knee angles, was plotted using a surface plot function in Matlab. To determine the predictive equation coefficients a Matlab programme was written, with hip angle (x) and knee angle (y) as independent variables, and hip moment (z) was the dependent variable. The programme determined the equation coefficients (a,b,c,d) for the predictive equation based upon the individual surface plot (equation 6.1), in addition to calculating goodness of fit of each coefficient. The coefficients were determined separately for both absolute and normalised hip moments:

$$z = (a.y) + (b.exp^{c.x}) + d$$

Equation (6.1)

The Figure 5.2 surface plot shows the four ensemble mean exponential curves for hip moments during passive leg raising, at each of four fixed knee angles. The surface plot is constructed as the best fit across all the data, from which a predictive equation and its coefficients can be derived. An individual subject example is included in Appendix F.

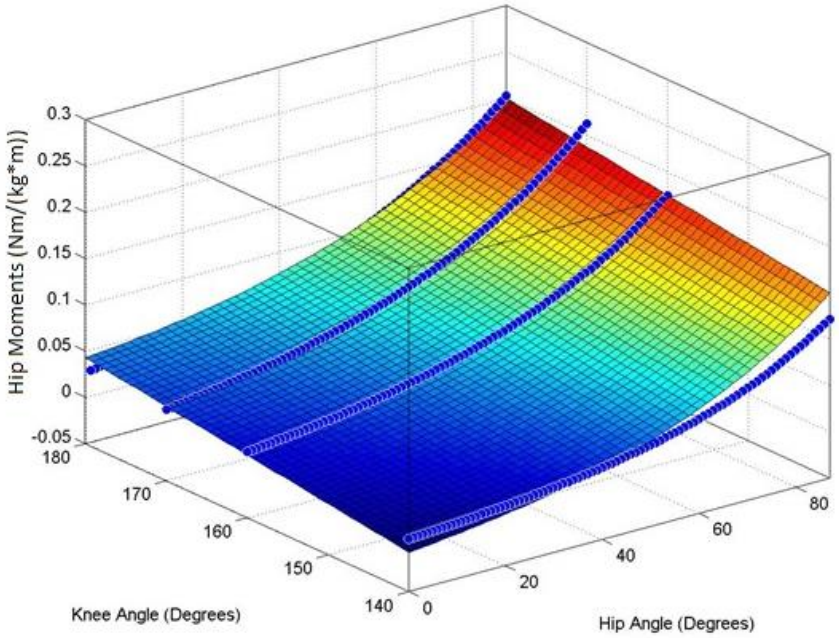

Figure 5.2 An example of a typical surface fit for a single subject, containing four mean curves and surface plot, used to derive the coefficients for the predictive equation

Table 5.3(a) Predictive equation coefficients for calculation of absolute moments (coefficients are without units)

	Equation Coefficients			
	a	*b*	*c*	*D*
Mean (SD)	0.125 (0.085)	9.518 (13.947)	0.023 (0.013)	-26.436 (20.1)
Median	0.114	4.907	0.021	-23.161
Min	-0.047	0.055	0.002	-105.624
Max	0.549	85.157	0.085	7.356

Table 5.3(b) Predictive equation coefficients for calculation of normalised moments (coefficients are without units)

	Equation Coefficients			
	a	b	c	d
Mean	0.001 (0.001)	0.014 (0.785)	0.022 (0.014)	-0.152 (0.771)
Median	0.001	0.038	0.021	-0.166
Min	0	-7.629	0	-1.592
Max	0.003	1.446	0.085	7.274

A residual analysis of individual subject data was used to assess model validity. A whiteness test revealed whether predicted data points from the model were within the 95% confidence limits of the original, smoothed mean curves (table 5.4). Figures 5.3(a-d) show a typical residual analysis for a subject.

Table 5.4 Residual analysis of predictive model for single subject

Knee Angle (degrees)	Hip Angle (degrees)	Hip Moments (Nm/(kg.m))				
		Mean	C.I.Low	C.I.High	Model	Pass/Fail
180	0	0.043	0.04	0.045	0.046	Fail
	15	0.058	0.054	0.062	0.064	Fail
	30	0.08	0.074	0.087	0.085	Pass
	45	0.112	0.101	0.123	0.111	Pass
	60	0.155	0.138	0.173	0.142	Pass
170	0	0.04	0.036	0.044	0.043	Pass
	15	0.054	0.048	0.06	0.061	Fail
	30	0.075	0.064	0.086	0.082	Pass
	45	0.103	0.086	0.122	0.108	Pass
	60	0.142	0.116	0.173	0.139	Pass
160	0	0.047	0.045	0.05	0.04	Fail
	15	0.061	0.057	0.064	0.058	Pass
	30	0.079	0.073	0.085	0.079	Pass
	45	0.102	0.093	0.112	0.105	Pass
	60	0.133	0.12	0.147	0.135	Pass
140	0	0.044	0.038	0.05	0.034	Fail
	15	0.056	0.048	0.065	0.052	Pass
	30	0.073	0.06	0.087	0.073	Pass
	45	0.094	0.076	0.116	0.099	Pass
	60	0.122	0.095	0.154	0.129	Pass
	75	0.158	0.12	0.206	0.166	Pass

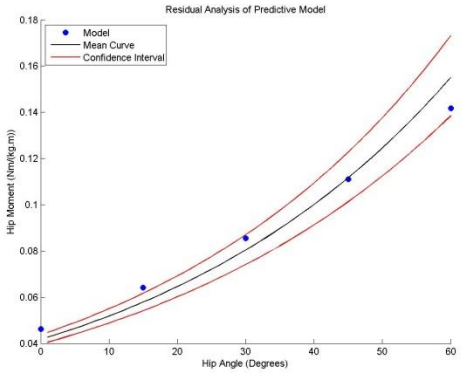

Figure 5.3a

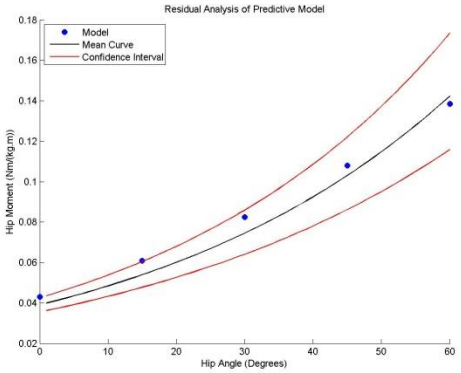

Figure 5.3b

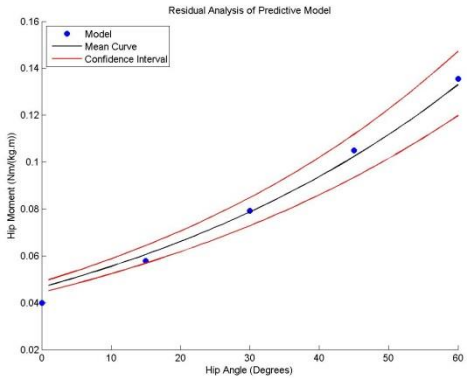

Figure 5.3c

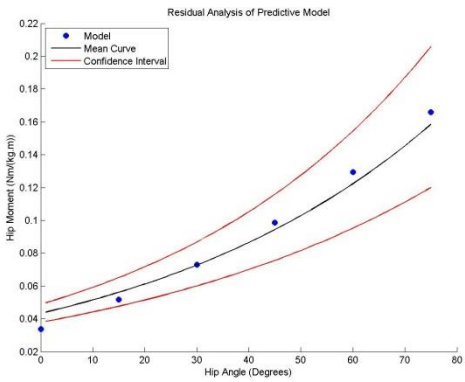

Figure 5.3d

Figures 5.3(a-d) Residual analysis of an individual subject, with four figures to represent each knee angle (180, 170, 160 and 140 degrees, for figures 5.2a, 5.2b, 5.2c and 5.2d, respectively) showing predictive model data points and 95% confidence limits of original smoothed mean curve.

If the predictive model failed the test, the coefficients would be manipulated to ensure the greatest number of passes for the subject. Due to the nature of the leg lifts it would not be possible for a surface fit to pass within the 95% confidence limits of all data, particularly where the confidence interval was small; typically at the lowest values of hip angle and moments.

For both absolute and normalised moments, the mean adjusted r-squared was 0.894 (0.077), with a range of 0.603 to 0.995. For absolute moments, the mean RMSE was 2.059 (0.891), with a range of 0.588 to 4.8. For

normalised moments, the RMSE was 0.017 (0.007), with a range of 0.004 to 0.035.

5.4 Discussion

To improve our understanding of passive-elastic effects on joint mechanics, direct measurements were experimentally-derived from passive testing. The results of the model developed in the present study can be used to quantify passive contributions to net joint moments, stiffness and work done, during gait and other ADLs. This approach can be used to improve the accuracy of biomechanical models, help in the analysis of movement dysfunctions, injury prevention and treatment, and any short- or long-term effects of pain or injury (Adouni and Shirazi-Adl, 2013, Hoy *et al.*, 1990).

The following equation (equation 6) can be used to derive the contribution of passive moments ($M_{passive}$) about the hip during flexion, in dynamic movements utilising a variety of hip and knee angles:

$$M_{passive} = a.\theta_{knee} + b.exp^{c.\theta_{hip}} + d$$

Equation (6)

where, *a*, *b*, *c* and *d* are equation coefficients, and θ_{knee} and θ_{hip}, are the angles at the knee and hip, respectively. This biomechanical model should be determined for individual subjects, and can be integrated into calculations for net moments, so as to establish passive contributions.

Passive hip moments were found to increase with hip flexion angle in an exponential manner. This finding is in agreement with other investigators (Silder *et al.*, 2007, Lee and Munn, 2000). Altering the knee angle during passive leg raising led to an increase in hip moments as the knee became more extended, also in agreement with the findings of other investigators (Riener and Edrich, 1999, Silder *et al.*, 2007). The relationship between knee angle and hip moments was found to be linear for any given angle of hip flexion. Thus, the predictive equation contains both linear and exponential functions, in addition to a constant.

The coefficients obtained in the predictive equation (tables 3a and 3b) interact to produce the closest fit for each subject. A visual assessment of figures and a residual analysis were used to correct coefficient bounds and improve goodness of fit. As a consequence, the adjusted r-squared and RMSE showed excellent goodness of fit.

It is noteworthy that the coefficients themselves are individual-specific, and should not be extrapolated as mean values for general application. Rather, this method of predicting passive contributions should be determined on an individual basis and applied to biomechanical models for the calculation of total joint moments. This approach is similar to that of Silder *et al.* (2007), who determined a subject-specific model of passive contributions to total joint moments during gait.

Although other investigators have assessed passive contributions to joint moments (Lee and Munn, 2000, Blackburn *et al.*, 2009a, Blackburn *et al.*, 2004a, Yoon and Mansour, 1982, Mansour and Audu, 1986, Hatze, 1997), few have assessed influences of neighbouring joints (Yoon and Mansour, 1982,

Mansour *et al.*, 1982, Vrahas *et al.*, 1990, Riener and Edrich, 1999, Silder *et al.*, 2007). Including values for passive-elastic joint coupling across bi-articular muscles is important for deriving accurate biomechanical information. Further, information can subsequently be determined regarding active contributions to net moments and active muscle timings and force production (Riener and Edrich, 1999, Mansour and Audu, 1986).

The involvement of passive multi-articular muscles in joint mechanics is often overlooked (Adouni and Shirazi-Adl, 2013, Hoy *et al.*, 1990). An understanding of these is important for multi-segment modelling, such as for interactions between the ankle, knee and hip joints during gait (Adouni and Shirazi-Adl, 2013). Adouni and Shirazi-Adl (2013) has modelled muscle and ligament forces, and joint surface compression pressures during gait. Hoy *et al.* (1990) modelled muscle and tendon contributions to moment-angle relationships at the hip, knee and ankle joints.

Because the lower limbs are crossed by bi-articular muscles, such as the gastrocnemius at the ankle and knee, and the biceps femoris and rectus femoris at the knee and hip, the angle at one of these joints can influence passive movement characteristics at a neighbouring joint (Riener and Edrich, 1999, Silder *et al.*, 2007). Hence, in developing models for understanding passive contributions to gait and ADLs, it is important to assess moments at one joint whilst manipulating the angle at a neighbouring joint (Riener and Edrich, 1999).

There have been few attempts to model any such relationships experimentally (Riener and Edrich, 1999, Mansour and Audu, 1986, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008). Further, any such

assessments should include the physiological ranges used in gait or other ADLs, so that the model can be of direct use in assessing passive contributions to net joint moments.

It is noteworthy that some investigators refer to the hamstrings as the primary hip extensors during gait (Silder *et al.*, 2007, Whittington *et al.*, 2008), and develop their passive moment models accordingly. However, the stretch of the hamstrings is reduced for any given angle of hip flexion as knee flexion angle increases. As this occurs, stretch of the gluteus maximus increases. Thus, stretching of the hamstrings is conducted during hip flexion whilst the knee is extended, whilst stretching of gluteus maximus is achieved by flexing the hip whilst the knee is also flexed. Further, due to dynamic coupling across the body, joints and limb segments are accelerated by muscles that do not span the accelerating joints. For example, the soleus crosses the ankle joint but also accelerates the hip and knee (Zajac *et al.*, 2002). Further, during gait the hip abductors perform actions commonly associated with the bi-articular hamstring muscles, such as extending the hip and decelerating the extending knee (Arnold *et al.*, 2007, Fox and Delp, 2010). Importantly, muscles on the stance limb contribute more to joint accelerations and decelerations on the swing limb than swing limb muscles (Arnold *et al.*, 2007, Fox and Delp, 2010). Hence, modelling for specific muscle actions during gait is complicated due to the role of muscles on the contralateral limb, and of muscles influencing joint accelerations at joints they do not cross.

Yoon and Mansour (1982) reported on the passive-elastic moments acting on the hip, and their experiment was followed by a similar investigation (Vrahas *et al.*, 1990). However, neither group reported moment-angle data

throughout their tested ranges, and subject numbers were low in both studies (4 and 15, for the studies by Yoon and Mansour (1982) and Vrahas *et al.* (1990), respectively). Both groups reported different passive moments at the highest hip angles measured, which may be due to the small sample sizes used. In agreement with the findings of the present paper, other researchers support a need to accurately measure individual passive joint moments for integration into modelling of more complex, dynamic activities (Silder *et al.*, 2007, Riener and Edrich, 1999, Yoon and Mansour, 1982).

A similar approach to that used in the present study has been developed by Silder *et al.* (2007) and used in subsequent investigations by the same research team (Silder *et al.*, 2008, Whittington *et al.*, 2008). In that study (Silder *et al.*, 2007), 20 healthy adults were recruited (males, n = 9, age = 26.1 (4.1); females, n = 11, age = 25.5 (3.1)). Individual results were not reported, but a model for determining passive contributions was developed for integration into a gait model of active and net moments (equation 8). In their model (Silder *et al.*, 2007), uni-articular components account for what is described as single-joint dependent stretch of ligaments, skin, inactive MTUs and the joint capsule. The models for bi-articular muscles were designed to account for energy transfer across the respective joints.

The model developed by Silder *et al.* (2007) for calculating passive hip moments ($\widehat{M}_h$), is as follows:

$$\widehat{M}_h = exp^{-\beta_h^{RF}\left(\theta_h - \left(\frac{\beta_k^{RF}}{\beta_h^{RF}}\right)\theta_k - \alpha^{RF}\right)} + exp^{-\beta_h^{HF}(\theta_h - \alpha^{HF})}$$

$$-exp^{\beta_h^{HAM}\left(\theta_h - \left(\frac{\beta_k^{HAM}}{\theta_h^{HAM}}\right)\theta_k - \alpha^{HAM}\right)} - exp^{\beta_h^{HE}(\theta_h - \alpha^{HE})},$$

Equation (7)

where, gain (β_{joint}^{muscle}) and offset angle (α^{muscle}) constants are used to determine passive moments, based upon hip (*h*) and knee (*k*) joints, and the contributions from the bi-articular rectus femoris (*RF*) and hamstrings (*HAM*), and the uni-articular hip flexors (*HF*) and hip extensors (*HE*) (Silder *et al.*, 2007). The constants used in the Silder *et al.* (2007) model are shown in table 5.5.

Table 5.5 shows the mean (S.D.) constants from Silder *et al.* (2007) determined from the 20 subjects tested in their study

Muscle	Gain, β		Offset (degrees), α
	Hip	Knee	
Rectus femoris (RF)	3.1 (1.4)	1.9 (0.7)	24.4 (9.0)
Hamstrings (HAM)	5.1 (2.0)	3.9 (2.5)	30.8 (14.9)
Hip flexors (HF)	5.1 (0.9)		19.5 (10.0)
Hip extensors (HE)	2.0 (0.9)		27.3 (18.0)

A potential limitation in the Silder *et al.* (2007) model is their averaging of moments derived from flexing and extending the hip. For example, at 45 degrees of hip flexion, the moment data obtained is the average of moments determined by flexing the hip to 45 degrees, and from extending the hip to 45 degrees from a more flexed position. This is important because during flexion the hip extensors are being stretched between neutral and full flexion, whereas during extension the hip extensors are being relaxed from a more stretched position. Thus, there is less passive resistance when extending the hip to 45 degrees of flexion from full flexion.

Silder *et al.* (2007) comment that by averaging the moment values the error is reduced. However, it seems more parsimonious to have a moment

calculation for flexion and another for extension movements. The purpose of the present study was to determine a predictive equation for the calculation of hip moments during flexion, as this will be of greatest relevance to understanding the role of the hip extensors during gait and other ADLs. Consequently, the present equation attempts only to establish a means of predicting the passive contribution of the hip extensors to hip flexion.

A strength of the approach used by Silder *et al.* (2007) is that it models for both hip flexion and extension moments. However, by assigning values to both uni-articular and bi-articular muscles there is a likelihood that errors occur due to dynamic coupling and the influences of muscles on the contralateral limb. By contrast, a strength of the present study is that it promotes a simple model for predicting passive hip moments during flexion, and does not require assigning values to muscles that may not be accurate. It may be more appropriate to assess total passive contributions to the net joint moments, rather than attempt to distribute moments between muscles. This is due to the inherent complexity of models that attempt to distribute muscle forces, as these may involve many parameters which cannot be directly measured. This consideration is in agreement with a similar finding by Riener and Edrich (1999), who suggest that distributing moments between muscles may only be appropriate if passive tissue loading or contact loading is of interest.

Should there be an interest in assessing muscles individually, it is important to consider the redundant problem in biomechanics, regarding the interactions of various muscles that act upon a joint. Further complexities are therefore encountered when attempting to model alterations in passive and active contributions to specific movements, and there may be limitations in the

accuracy of such models (Riener and Edrich, 1999, Yamaguchi *et al.*, 1995). The majority of investigators agree that it is important to account for the effects of bi-articular muscles on neighbouring joints (Riener and Edrich, 1999, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008), by calculating moments whilst controlling the angle of those associated joints. The present study and those of few others (Riener and Edrich, 1999, Silder *et al.*, 2007), have developed models based upon experimental study of passive bi-articular muscles.

A further strength of the present study is the large dataset used, when compared with the studies by Riener and Edrich (1999) (n = 10), and Silder *et al.* (2007) (n = 20). However, because the models in the present study and that of Silder *et al.* (2007) are subject-specific and mean values are not derived, the larger samples do not directly influence the model itself, in difference to the models developed by Riener and Edrich (1999) (equation 9).

The model for calculating passive hip moments (M_H), developed by Riener and Edrich (1999) is as follows:

$$M_H = exp^{(1.4655 - 0.0034_{\theta K} - 0.0750_{\theta H})} - exp^{(1.3403 - 0.0226_{\theta K} + 0.0305_{\theta H})} + 8.072$$

Equation (8)

where, θK and θH are knee and hip angles, respectively, in degrees. It was noted by Riener and Edrich (1999) that model error increased with increasing joint angles, and that there was a high deviation between experimental- and model-derived hip moments (> 10 Nm) when the knee was flexed to 90 degrees. The high inter-subject deviations reported in the present study and

others (Riener and Edrich, 1999), combined with the high deviations from the mean values obtained from published predictive equations (Riener and Edrich, 1999, Silder *et al.*, 2007), supports the need to determine passive-elastic joint contributions on a subject-specific basis.

Modelling precisely how passive elements contribute to active movements will be complicated by changes in tendon lengths and fascicle pennation angles, in addition to passive force enhancement. Because the passive component contributes up to 50% of the joint moment during gait (the 50% value of 20 Nm was derived from the hip flexors at toe-off) (Silder *et al.*, 2007), these complicating variables may be considered minimal to the overall passive joint resistance. Thus, although the model developed in the present study can be adapted and optimised to predict forces in specific muscles, at joint surfaces, and in other soft tissues, the increasing complexity of such approaches may increase the error.

The present study produced a predictive equation for passive hip moments during sagittal-plane hip flexion. During testing, any movement in frontal or transverse planes was restricted. However, complete modelling of the hip during gait should take into account such movements. For example, the long-head of biceps femoris has been found to resist the knee adduction moment in the frontal plane, in addition to resisting the knee extension moment in the sagittal plane (Adouni and Shirazi-Adl, 2013).

The present study assessed hip moments at various combinations of hip and knee flexion, with the ankle supported in a neutral position. This may be considered a limitation if using the data to predict passive hip moments whenever the ankle is not in neutral (Riener and Edrich, 1999, Mansour and

Audu, 1986, Palmer *et al.*, 2014). However, Adouni and Shirazi-Adl (2013) modelled ankle, knee and hip biomechanics during gait, and reported that optimisation techniques that included the ankle had minimal effects on muscle forces and knee joint response. Thus, the influence of varying ankle positions on hip moments during gait can also be expected to be minimal.

There are various sources of error when directly measuring passive joint moments. As described in the previous chapter, these include errors in anthropometric measurements and joint centres of rotation, the use of generalised body segment parameter data, undetected muscle contractions, skin movement and tester non-tangential force application (Riener and Edrich, 1999, Mansour and Audu, 1986, Vrahas *et al.*, 1990). However, such errors are considered sufficiently small to be acceptable (Riener and Edrich, 1999).

It may be a benefit in future research to develop models that demonstrate interactions between passive and active muscle forces and joint moments, such as by combining models similar to those used by other investigators (Silder *et al.*, 2007, Hoy *et al.*, 1990, Yamaguchi *et al.*, 1995, De Groote *et al.*, 2010, Adouni and Shirazi-Adl, 2013) with that used in the present study. This will help to better quantify the interactions between the neuromuscular and skeletal systems, and specifically the interactions between muscles and joints. This will further improve biomechanical assessments, and may help determine cause and effect relating to acute and chronic pain, injuries and other sources of musculoskeletal and neuromuscular dysfunction.

Although the predictive equation was determined for the calculation of hip moments, the approach is appropriate for any joints where multi-articular muscles interact to influence moments. Future research should develop the

sagittal-plane hip flexion model developed here into a 3-D model to assess hip MTU contributions throughout gait and other ADLs. Such an approach can be expanded to describe agonist-antagonist influences on biomechanical and kinematic variables, muscle activation and force production, and bioenergetics. Further research can utilise such models within studies to assess sensorimotor control of gait and other ADLs, to help improve our understanding of central influences on movement (Herzog *et al.*, 1995). Regardless of the potential for further development and optimisation, the approach used in the present study is sufficient to quantify passive-elastic contributions to movement across joints with bi-articular muscles.

5.5 Conclusions

The following equation was developed to derive the contribution of passive moments about the hip during flexion, in dynamic movements utilising a variety of hip and knee angles:

$$M_{passive} = a.\theta_{knee} + b.exp^{c.\theta_{hip}} + d$$

Equation (6)

This predictive equation can be used to calculate passive moments generated from bi-articular hip extensor muscles during walking and other ADLs. The equation is based upon subject-specific measurements of passive hip and knee moments during leg raising tests, and can be adapted to assess the influence of bi-articular muscles acting at any joint.

6. Biomechanical contribution of passive hip extensor muscles during human walking

The following chapter describes the second study of the thesis and was submitted for publication. Hence, the study includes an introduction and methods that have already been described elsewhere in this thesis.

6.1 Introduction

Hip joint moments during dynamic tasks are commonly modelled using the inverse dynamics method, and the hip joint is considered to behave as a frictionless line-hinge (Koopman *et al.*, 1995). However, investigators have reported that passive structures about the hip joint will contribute to the net moments measured during dynamic tasks, such as gait (Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008). Importantly, the passive structures involved in movement contribute to the absorption, storage and transmission of mechanical energy (Marshall *et al.*, 2009, Yoon and Mansour, 1982). Thus, the passive structures have a direct influence on the bioenergetics of dynamic tasks, and contribute to the energy efficiency of movement (Yoon and Mansour, 1982, Umberger, 2010, Zelik *et al.*, 2014, Takahashi and Stanhope, 2013).

Passive joint resistance is a property of the non-contractile tissues, such as the tendon, sarcolemma, endomysium, perimysium and epimysium (Marshall *et al.*, 2009, McNair and Stanley, 1996, Blackburn *et al.*, 2004a, Blackburn *et al.*, 2004b), structural proteins such as titin (Rassier, 2012), and inactive muscle fibres. The stretch of these tissues generates a passive joint moment (Silder *et al.*, 2007). Passive joint resistance has been shown to increase with age (Whittington *et al.*, 2008, Wolfarth *et al.*, 1997), due to a loss of elasticity in soft tissues, and the replacement of damaged tissues with less elastic scar tissue (Wolfarth *et al.*, 1997). Should changes in passive characteristics follow injury (Mattyasovszky *et al.*, 2010) or pain (Halbertsma *et al.*, 2001), it would be of use to understand how these might influence joint mechanical properties. Such information could be useful in directing rehabilitation techniques, to target

passive or active contributions to total moments, and to measure the efficacy of any such interventions.

Despite the potential for passive structures to contribute to joint moments and energetics of dynamic tasks, few investigators have attempted to measure and integrate passive and active contributions to total moments (Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008, Yoon and Mansour, 1982, Vrahas *et al.*, 1990). Of these, only one concluded that the passive contribution is negligible (Vrahas *et al.*, 1990), whereas others reported the contrary (Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008).

The purpose of the present chapter was to examine passive hip extensor and total hip moments during the hip flexion component of walking, between hip neutral and maximum hip flexion. The study incorporates the dynamic biomechanical model utilised in chapter four, and the predictive equation developed in chapter five.

6.2 Methods

6.2.1 Subjects

27 subjects (male n = 15, mean age = 29 (SD = 7.78), female n = 12, mean age = 33 (SD = 8.78)) volunteered for this study. Subjects were excluded if they were pregnant or had any tumours, rheumatological or musculoskeletal disorders, tuberculosis, back pain or an injury or infection of the spine, hips or knees during the 3 months prior to their participation. Subjects were also excluded if they had a history of any dislocation or surgery of the spine or lower

limbs, if they were allergic to adhesive tape, or if they had any orthopaedic or electrically-powered medical implant. Female subjects were only eligible for testing during the 7 days following the first day of menstruation, to control for any potential effects of the ovarian cycle. The study was approved by the ethics committees of both the University of Roehampton and the British College of Osteopathic Medicine.

6.2.2 Experimental Setup

6.2.2.1 Passive Hip Moments

Passive hip extensor moments were measured using a custom-built force transducer, comprising a bi-axial cantilever load cell (QLA263, Futek, US), and two analogue electro-inclinometers (PTAM27, ASM, Germany). The force transducer was inserted into a custom-built ankle brace, designed to house the transducer with minimal friction, whilst maintaining the ankle in neutral.

A second series of braces was used to maintain the knee in a given angle during testing. Four knee braces were pre-formed to secure the knee at 180, 170, 160 and 140 degrees. Two electro-inclinometers were secured using straps to the thigh and shank, to measure hip angle and hip angular acceleration, and to monitor knee angle, respectively. Two surface electromyography (EMG) electrodes (SX230, Biometrics, UK), were placed over the biceps femoris and rectus femoris, in accordance with the SENIAM guidelines for electrode placement. EMG signals were observed during passive

testing to ensure no muscular activity, with EMG data not used for further analysis.

The analogue signals from the load cell were pre-amplified (CSG110, Futek, US) for each output. All analogue signals from the load cell and inclinometers were acquired at 50 Hz, and from the electrodes at 1000 Hz, using a data acquisition unit (Datalink, DLK900, Biometrics, UK). The outputs were analogue-digital converted, digitally filtered at 2 Hz using a low-pass Butterworth filter, and saved to a personal laptop computer (Dell Precision, M4500, Dell, US) for processing with Matlab programming software (Version 7.3, Mathworks, US). EMG data was used only for real-time feedback during the passive testing.

Passive leg raises were performed with the subject lying supine on a standard massage couch. Following 10 passive raises to pre-condition the leg, the initial testing was conducted using a knee brace that maintained the knee close to full extension (180 degrees). The tester performed 3 leg raises with a minimum of one minute rest between each. The procedure was repeated with each of the remaining knee braces in random order, and with a minimum of two minutes rest between testing at different knee angles.

Each leg raise would cease upon the onset of stretch-related pain or any muscle activity. Data was assessed at 15-degree increments from 0 to 75 degrees. Due to an inability of >50% of subjects to reach 75 degrees of hip flexion with the knee fully extended (180 degrees), only data up to 60 degrees was included.

6.2.2.2 Total Hip Moments

Total hip moments were measured during walking with the use of a 9 camera, 3-D motion-capture system (T-series, Vicon, UK) and two force plates (9281CA, Kistler, Switzerland). 35 retro-reflective markers were placed on each subject in accordance with the approach used by previous researchers (Kadaba *et al.*, 1989, Gorton *et al.*, 2009, Tsushima *et al.*, 2003). Motion capture data was sampled at 100-Hz and force plate data at 1000-Hz. The data was stored on a personal computer (Dell Precision, M4500, Dell, US). Data was initially assessed via the Nexus software programme (Vicon Nexus version 1.8, Vicon, UK), before being imported to Microsoft Excel (2010, Microsoft Excel, US) and Matlab for further processing.

Correct marker placement is essential for the correct determination of joint moments. The operator had accumulated more than one hundred hours of experimental practice of skeletal assessments, including anthropometric measurements of approximately one hundred complete skeletons, which was deemed useful in identification of the bony landmarks required for marker placements. In addition, the operator had more than one hundred hours of body composition and anthropometric measurements of more than one hundred healthy individuals, prior to pilot testing with the motion capture marker system. Following initial familiarisation with the testing protocols for motion capture, the operator performed a sensitivity analysis by varying the location of the hip marker by one centimetre above the normal hip marker location, and one centimetre posterior to the normal hip marker location. This analysis was useful in establishing that hip marker placement can significantly influence peak hip

moments during walking (see Appendix E for results of this assessments on a single test subject). Although a similar approach has been used to test reliability of different motion capture hardware and laboratories (Davis *et al*., 1991; Kadaba *et al*., 1990), variability in walking trials can be high, with the standard five complete walking trials being insufficient to detect sensitivity to altered marker placements.

Subjects were required to walk along a 10-metre walkway in view of the motion capture cameras and over the two force plates. Subjects were requested to look ahead and to move at their normal walking pace. The tester observed foot contact onto the force plates and ensured a minimum of five walks contained both left and right clear foot strikes. Once this was achieved the tester completed preliminary processing to ensure all markers were still visible in a minimum of 5 of the tests on each leg.

6.2.3 Data Processing

6.2.3.1 Passive Moment Data

Passive hip extensor moments were calculated based upon the dynamic biomechanical model developed by Lee and Munn (2000):

$$M = F_x y_f + F_y x_f + m_{leg}\ddot{x}_{cg}y_{cg} - (m_{leg}g + m_{leg}\ddot{y}_{cg})x_{cg} - m_{leg}k^2\ddot{\theta}$$

Equation (1)

where F_x, F_y, are the forces applied to the leg to flex the hip joint, X_f, Y_f, are the locations of force application to the leg, m_{leg} is the mass of the leg, g, is acceleration due to gravity, x_{cg}, y_{cg}, refer to the location of the centre of mass of the leg and k is the radius of gyration. $\ddot{x}_{cg}$, $\ddot{y}_{cg}$ refer to the acceleration of the leg centre of mass, and $\ddot{\theta}$ is the angular acceleration of the leg. An ensemble mean curve was created from each subject's 3 measurements for each leg and knee angle. The mean curve was subsequently smoothed using cubic spline interpolation. At all knee angles, passive moments about the hip increased exponentially with increasing hip angle, and an exponential function was fitted to each mean curve.

To establish a predictive equation for passive hip extensor moments based upon a combination of hip and knee angles, 3-D surface plots were generated for assessing the changes in hip moments with hip angle, and at different knee angles. Data was collected across the full range of motion, with hip flexion angles from 0 to 60 degrees used in the final analysis.

A surface fitting programme was written for Matlab, and visual and residual analyses used to determine each equation coefficient for individual datasets (chapter 5). From the present investigation, the following equation (equation 6) was developed to predict passive hip moments ($M_{passive}$) during flexion:

$$M_{passive} = a.\theta_{knee} + b.exp^{c.\theta_{hip}} + d$$

Equation (6)

where θ_{knee} is the angle at the knee, θ_{hip} the angle at the hip, and *a*, *b*, *c*, and *d* are equation coefficients. In agreement with other researchers (Lee and Munn, 2000), passive hip moments were found to increase exponentially as a function of hip angle. By introducing a variable knee angle, this contributed a linear component to the equation, determined through visual and residual analysis (chapter 5).

Passive hip extensor moments with 95% confidence intervals were recalculated using the derived predictive equation. Root mean squared error (RMSE) and adjusted r-squared were both calculated to assess goodness of fit of each subject's surface-plot. Each surface plot was constructed as the best fit across subject data, from which a predictive equation and its coefficients could be derived.

6.2.3.2 Total Moment Data

A data processing pipeline was created in Vicon Nexus to perform standard data modelling of the walking trials. The pipeline included Woltring filtering and gap filling. Each subject trial was subsequently checked for errors and marker gaps were filled as appropriate. A final pipeline was created for smoothing the data and producing model trajectories, calculating gait cycle parameters, and saving and exporting the output data required for further analysis.

Hip moments and joint angles and forces were calculated using the Vicon Nexus Plug-in Gait software, using a 3-degrees of freedom 'conventional gait model' based upon the Newington-Helen Hayes model. The conventional gait model has been previously validated (Davis R., 1991, Kadaba *et al.*, 1990).

The data was transferred to Microsoft Excel and Matlab for further processing. Data was smoothed using a low-pass Butterworth filter at 6 Hz. Mean moment data was calculated from the 5 smoothed moment curves.

6.2.4 Statistical Analysis

Passive hip extensor and total moment-angle data for the hip was initially established for the complete gait cycle (GC). Coefficients of multiple correlation (CMCs) of the moment-angle curves were calculated based upon the equation developed by Lee and Munn (2000). Where CMC values were less than 0.8 the moment-angle curves were visually analysed for outliers. Any outliers were removed and the mean calculated from the remaining curves. Outliers were those with a clear deviation from the remaining data curves. RMSE and the adjusted r-squared were used to assess goodness of fit of the predictive equation coefficients for calculating passive moment contributions. All data was normalised to body mass and height. Passive hip extensor and total hip moments were calculated for the GC data corresponding to hip flexion, between hip neutral and maximum hip flexion (figure 6.1). The percentage of passive hip extensor to total hip moments was calculated where these were acting in the same direction (hip extensor moments).

6.3 Results

Goodness of fit of the predictive equation to the original passive data curves was assessed using the adjusted r-squared and RMSE. For normalised

moments, the mean adjusted r-squared was 0.897 (0.075), with a range of 0.703 to 0.995. The RMSE was 0.017 Nm/kg.m (0.007), with a range of 0.005 Nm/kg.m to 0.034 Nm/kg.m.

CMCs and CVs were used to assess intra-subject gait cycle characteristics. The CMC mean was 0.937 (0.051). The CV mean was 26.3% (13.9). Although the CV is commonly used, the CMC is considered the most appropriate means of assessing intra-subject reproducibility of curve data, such as gait data, and the very low range of values (from 0.692 to 0.986, overall), demonstrates the high reproducibility of individual subject gait data.

Hip neutral occurred at approximately 64% of GC, coinciding with early swing phase shortly after toe-off (60% of GC). Maximum hip flexion angle was 29.05 (4.5) degrees, and occurred during late swing phase at approximately 91% GC (figure 6.1). Passive hip extensor contributions to total hip moments were evaluated for hip flexion, from hip neutral to maximum hip flexion , corresponding to most of the swing phase of walking (figure 6.2). Passive hip extensor moments without total moments are shown in figure 6.3. Values of passive hip extensor and total hip moments are shown in table 6.1.

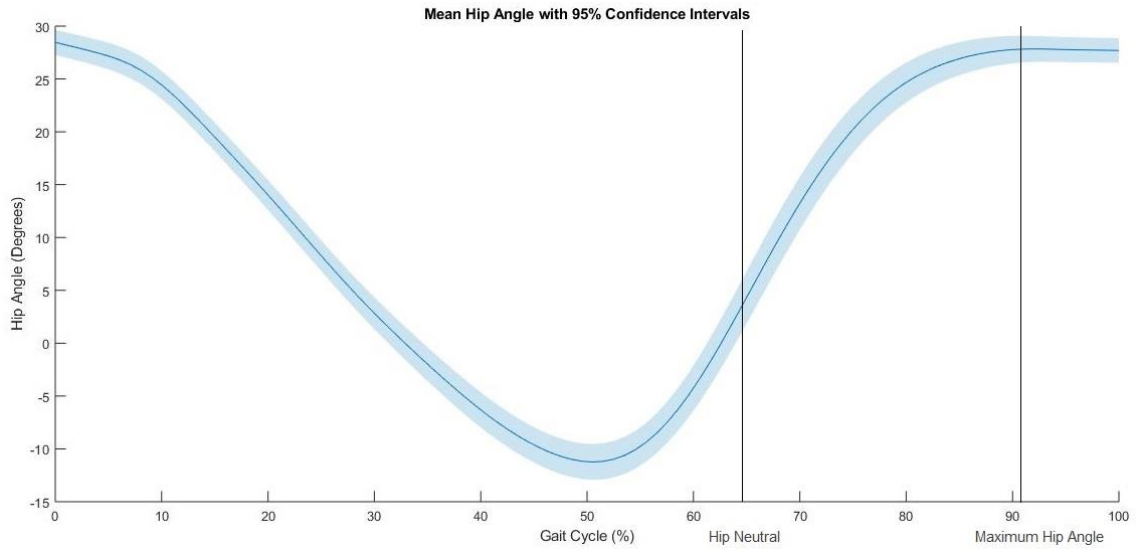

Figure 6.1 Mean hip angles with 95% confidence intervals for all subjects. Figure shows period of further analysis, from hip neutral to maximum hip flexion (vertical lines).

Total hip moments were positive (flexor moments) during the first half of hip flexion, corresponding with early and mid-swing phase, crossing zero at 58% of hip flexion (figure 6.2). Hip moments were then negative (extensor moments) throughout the remainder of hip flexion, corresponding with the late swing phase of gait before terminal swing and foot contact. Passive hip extensor moments increased (becoming more negative) throughout hip flexion, reaching a maximum value at maximum hip flexion angle. At maximum hip flexion the passive extensor moments were approximately 40% of total hip moments.

Table 6.1. Normalised passive hip extensor and total hip moments during the hip flexion component of walking, from hip neutral to maximum hip angle. Table includes mean (standard deviation (SD)) and 95% confidence intervals (C.I.)

		Mean (S.D.)	C.I. Low	C.I. High
Moments (Nm/(kg.m))				
Hip Flexion (%)				
0	**Passive**	-0.033 (0.016)	-0.038	-0.029
	Total	0.194 (0.080)	0.172	0.216
25	**Passive**	-0.046 (0.026)	-0.052	-0.039
	Total	0.074 (0.073)	0.054	0.094
50	**Passive**	-0.060 (0.037)	-0.070	-0.050
	Total	0.020 (0.044)	0.008	0.032
75	**Passive**	-0.077 (0.040)	-0.088	-0.066
	Total	-0.116 (0.167)	-0.162	-0.071
100	**Passive**	-0.089 (0.037)	-0.099	-0.079
	Total	-0.226 (0.257)	-0.296	-0.157

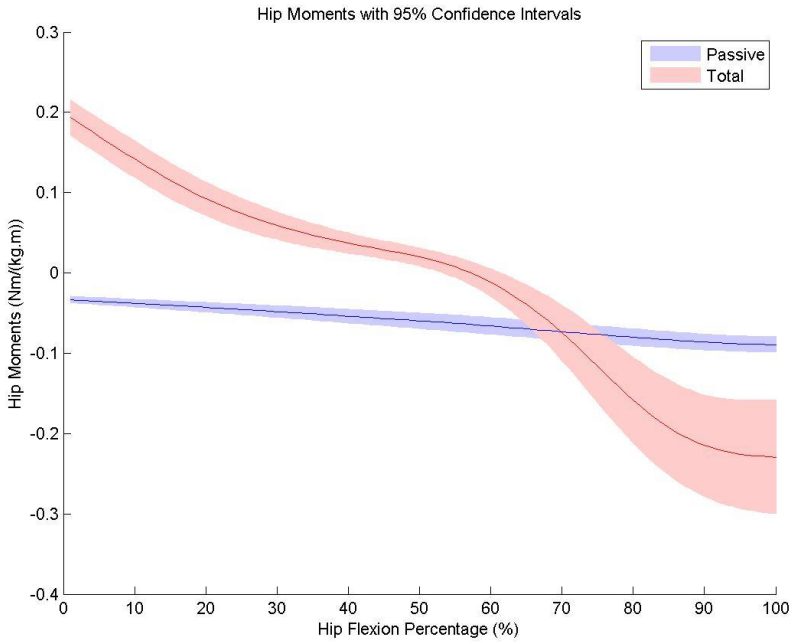

Figure 6.2. Passive hip extensor and total hip moments during hip flexion, from hip neutral to maximum hip angle, for all subjects.

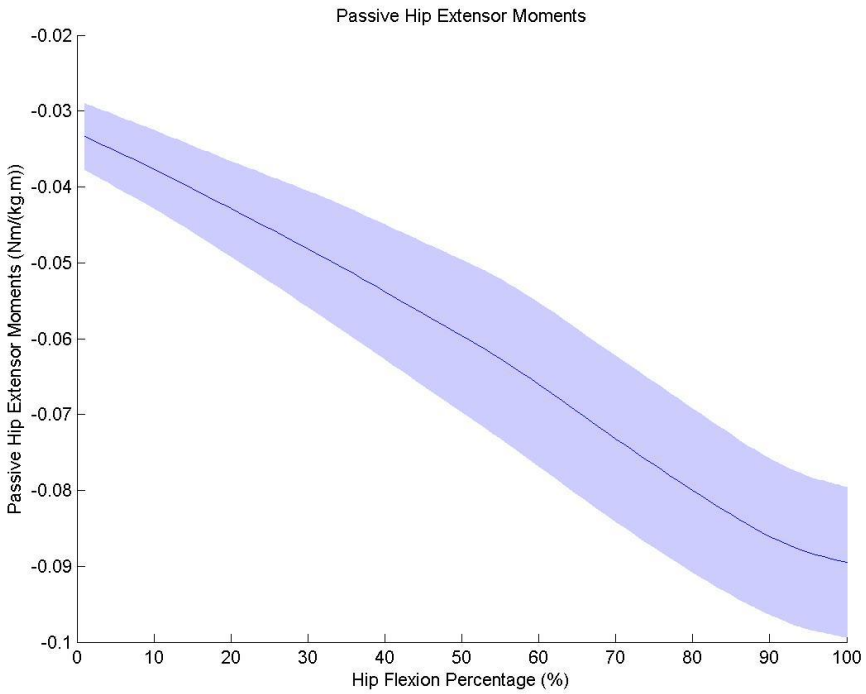

Figure 6.3. Passive hip extensor moments during hip flexion, from hip neutral to maximum hip angle for all subjects.

6.4 Discussion

The involvement of passive bi-articular muscles, such as the hamstrings, in joint mechanics is often overlooked (Adouni *et al.*, 2012, Hoy *et al.*, 1990). An understanding of these is important for multi-segment modelling, such as for interactions between the ankle, knee and hip joints during gait (Adouni *et al.*, 2012). Although other investigators have assessed passive contributions to joint moments (Yoon and Mansour, 1982, Blackburn *et al.*, 2009a, Blackburn *et al.*, 2004a, Lee and Munn, 2000, Mansour *et al.*, 1982, Hatze, 1997, Mansour and Audu, 1986), few have assessed influences of neighbouring joints (Silder *et al.*, 2007, Yoon and Mansour, 1982, Vrahas *et al.*, 1990, Mansour *et al.*, 1982,

Mansour and Audu, 1986, Riener and Edrich, 1999). The present study demonstrates that bi-articular muscles can be assessed passively to determine the passive contributions to total joint moments.

In the present study, passive hip extensor and total hip moments were assessed during normal walking in healthy subjects. The portion of gait measured was from hip neutral to maximum hip flexion, which was from approximately 64% to 91% of GC, within the swing phase of gait. In agreement with other researchers (Marshall *et al.*, 2011a, Whittington *et al.*, 2008, Lee and Munn, 2000, Vrahas *et al.*, 1990, Yoon and Mansour, 1982), passive extensor moments at hip neutral were close to zero (figure 6.2, 0.033 (0.016) Nm/(kg.m)), so values between minimum hip flexion angle and neutral were assumed to be zero and excluded.

During the late stance phase the hip flexors contract to rotate the pelvis anteriorly and shift the centre of mass backwards, permitting acceleration of the swing shank (Arnold *et al.*, 2007). Investigators (Arnold *et al.*, 2007, Fox and Delp, 2010), have reported that the hip flexors (iliacus and psoas) and the short head of biceps femoris are primarily responsible for accelerating the pre-swing knee, in addition to gastrocnemius and the ankle dorsiflexors (Fox and Delp, 2010). The knee reaches peak flexion velocity at close to toe-off (Arnold *et al.*, 2007), with toe-off occurring at approximately 60% of GC in the present study, shortly before the hip passed through neutral.

In the present study, during early swing phase the total hip forces generate flexor moments, whilst passive hip extensor moments were small but steadily increasing (becoming more negative). This finding for total hip moments is in agreement with other investigators (Silder *et al.*, 2008,

Whittington *et al.*, 2008, Prilutsky *et al.*, 1998). The hamstrings and rectus femoris have shown reciprocal force patterns, with rectus femoris generating the most force during the first half of swing with minimal force from the hamstrings (Prilutsky *et al.*, 1998). In the mid-swing phase the hip forces were close to zero. This finding is in agreement with other researchers (Prilutsky *et al.*, 1998), whilst the passive hip extensor moments continued to increase, reaching their greatest value at the end of hip flexion, at approximately 91% of GC.

The total hip forces generate extensor moments during the late swing phase, with passive extensor moments close to 40% of total hip moments at maximum hip flexion (91% GC). This finding is similar to that of other researchers (Whittington *et al.*, 2008), and coincides with increased hamstring activation from 80% GC, which has been found to peak at 90% GC (Lyons *et al.*, 1983). Peaks of hip extensor moments occur with some activation of the hamstrings and minimal or no activation of the rectus femoris (Prilutsky *et al.*, 1998, Arnold *et al.*, 2007). The absence of activity of the hamstrings during the majority of the swing phase is due to dynamic coupling, whereby the hamstrings are decelerating the shank without any considerable rotational force exerted upon the knee (Arnold *et al.*, 2007). This is due to the hamstrings' knee flexion moment accelerating the knee into flexion, whilst the hamstrings' hip extension moment accelerates the knee into extension (Arnold *et al.*, 2007). It has been reported that hip abductor muscle activity is initiated at 80% GC, potentially to ensure an optimal position of the hip prior to loading phase (Lyons *et al*., 1983), and to absorb energy and stabilise the hip (Goldberg and Neptune, 2007).

Other investigators (Silder *et al.*, 2008, Whittington *et al.*, 2008), have used passive moment calculations to determine passive power and mechanical work done. However, power is a product of joint moment and angular velocity, and as velocity during walking is a product of interactions between momentum, gravity and active muscle contractions, a truly passive power calculation may not be reliable. Further, previous investigators have found no influence of angular velocities on passive properties, at the velocities observed during gait (Vrahas *et al.*, 1990, Yoon and Mansour, 1982). Therefore, calculating passive power by multiplying passive moments by joint angular velocity cannot generate a realistic value of passive contributions. As mechanical work done is calculated from power curves, estimates of passive work done are similarly confounded by interactions of active muscle contractions.

It is important to note that the equation coefficients themselves are subject-specific, and should not be extrapolated as mean values for general application. Rather, this method of predicting passive contributions should be determined on an individual basis and applied to biomechanical models. This approach is similar to that of Silder *et al.* (2007), who determined a subject-specific model of passive contributions to total joint moments during gait, averaging hip flexion and extension data to incorporate into a gait model. The model developed in the present study has the advantage of direct passive measurements during hip flexion, without averaging of hip flexion and extension data. This approach can be used to assess passive contributions during dynamic movements, to determine whether pain or injury influences passive or active components, and to evaluate the efficacy of physical therapy interventions on these components.

There were some potential sources of error when directly measuring passive joint moments. These include errors in anthropometric measurements and joint centres of rotation, the use of generalised body segment parameter data, undetected muscle contractions, skin movement and tester non-tangential force application (Vrahas *et al.*, 1990, Mansour and Audu, 1986, Riener and Edrich, 1999). Anthropometric measurement errors were minimised by repeating measurements where values varied by more than a centimetre. EMG was used to help minimise the likelihood of muscle contraction anomalies. Further, the CMC and CV data reported in this study demonstrate that any such errors were negligible (Riener and Edrich, 1999).

The reliability of the Plug-in Gait software and conventional gait model could be questioned in terms of appropriateness. However, the purpose of the present investigation was to assess sagittal plane kinematics of the hip and knee during walking, for which the 3 degrees of freedom model was considered sufficient. This was to ensure the sagittal-plane biomechanical model of passive moments could be adequately assessed with the sagittal plane gait data. As a consequence, there was no requirement for measurements of the lower limb of greater than 3 degrees of freedom.

6.5 Conclusions

The contribution of passive-elastic tissues to joint biomechanics is significant and can be considerable during walking. Models of dynamic movements that do not include a measure of passive contributions are therefore at risk of overlooking an important biomechanical component. The approach used in the

present study is sufficient to quantify passive-elastic contributions to movement across joints with bi-articular muscles. Such information can be used to improve biomechanical assessments, and to assess the efficacy of injury treatment and prevention strategies, as well as short- and long-term effects of pain and injury. Future studies should assess passive contributions to joints in subjects suffering chronic pain, in relation to sports injury risk, or as a component of a broader, multi-plane biomechanical model of movement.

7. Passive elastic contribution of hip extensors to joint biomechanics during walking in people with low back pain

The following chapter describes the third study of the thesis and was submitted for publication. Hence, the study includes an introduction and methods that have already been described elsewhere in this thesis.

7.1 Introduction

LBP can only be attributed to a specific cause in 5-15% of cases, in which osteoporotic fracture, neoplasm or infection are regarded as responsible for the condition (Hoy *et al.*, 2010). Thus, LBP in 85-95% of patients is considered non-specific (Hoy *et al.*, 2010, Balague *et al.*, 2012). Clinical assessments of LBP patients often include tests of hip extensor extensibility (Ekedahl *et al.*, 2010). Tests can include assessment of knee extension angle and sacral angle, the sit and reach test (Davis *et al.*, 2008), and the straight leg raise test (Davis *et al.*, 2008, Ylinen *et al.*, 2010). However, the relevance of hip muscle extensibility to LBP and any relationship to movement remains unclear (Rebain *et al.*, 2002). There is a growing interest in including more comprehensive assessments of joint and muscle resistive properties, due to the lack of consensus with assessing extensibility alone (Marshall *et al.*, 2009, Halbertsma *et al.*, 2001).

Some investigators report musculotendinous extensibility being moderately related to passive stiffness, and weakly related to active stiffness (Blackburn *et al.*, 2004a). An investigation by Halbertsma *et al*.(2001), reported an association between hamstring extensibility and LBP, but no differences in passive stiffness between LBP patients and controls. Overall, there is a lack of agreement in the literature as to whether or not passive muscle resistance is related to extensibility (Blackburn *et al.*, 2004a, Tafazzoli and Lamontagne, 1996). Further, any relationship between extensibility, passive and active stiffness and activities of daily living (ADLs) in LBP patients remains to be determined.

There is a clear relationship between the activation of hip extensors and lumbar spine musculature during gait. It has been found that muscle resistive properties can be affected by LBP (Hamill *et al.*, 2009, Marshall *et al.*, 2009, Gombatto *et al.*, 2008b), and these may be responsible for the reduced leg swing, stride length and gait velocity often observed in LBP subjects when compared with healthy controls (Elbaz *et al.*, 2009, Henchoz *et al.*, 2015, Barzilay *et al.*, 2015, Ertelt, 2014). During the late swing phase of walking, the activation of biceps femoris is increased in LBP (Ertelt, 2014), indicating altered active factors. The biomechanics of hip-spine interaction may therefore be altered in LBP, although underlying mechanisms remain to be elucidated. Identifying alterations to passive or active components may be a useful tool for the clinical assessment of LBP, and the first stage in developing effective physical therapy-based treatment strategies.

Passive hip moments have previously been reported to be approximately 30% of total hip moments (Whittington *et al.*, 2008) during walking, with as much as 40% reported elsewhere (unpublished data, chapter 6). Interactions between passive and active properties in LBP therefore warrant further investigation. If LBP affects either the passive or active components of total hip biomechanics, this may negatively impact upon the biomechanical efficiency of movement, assessed through mechanical work done. Passive or active resistance that is increased in individuals with LBP, above that of healthy controls, may require antagonist muscles to increase their active contractions to compensate, reducing overall energy efficiency of movement and potentially increasing pain or injury risk. To improve the effectiveness of interventions for LBP patients, it would be useful to determine whether interventions should

target the passive or active components of movement during ADLs such as walking.

In a recent investigation in our laboratory, a biomechanical model was adopted to measure passive moments, stiffness and strain energy of the hip extensors, as a function of hip and knee angles during hip flexion. A predictive equation was developed to calculate passive hip extensor moments during hip flexion as a product of hip and knee angle. The purpose of the present study was to assess total hip and passive hip extensor moments in people with LBP during the hip flexion component of walking, and to compare them with pain-free controls. Comparisons were also made of total hip power and work done during hip flexion and the complete gait cycle.

7.2 Methods

7.2.1 Subjects

Fifty-two subjects volunteered for this study. Subjects were excluded if they were pregnant or had any tumours, rheumatological or musculoskeletal disorders, tuberculosis, or an injury or infection of the spine, hips or knees during the 3 months prior to their participation. Subjects were also excluded if they had a history of any dislocation or surgery of the spine or lower limbs, if they were allergic to adhesive tape, or if they had any orthopaedic or electrically-powered medical implant. Female subjects were only eligible for testing during the 7 days following the first day of menstruation, to control for any potential effects of the ovarian cycle.

Subjects meeting the inclusion criteria were divided into groups according to if they had suffered with chronic, non-specific low-back pain (LBP group, n = 25 (male n = 13, age = 34(8.53) female n = 12 age=30(7.96)) for at least 6 weeks, including at least one episode during the week of the study, or were back-pain-free (NBP group, n = 27 (male n = 15, age=29(7.78), female n = 12, age=33(8.78)). Subjects in the NBP group needed to have been without back pain during the 6 months prior to the study.

Following consent to participate, subjects were required to complete a medical screening form and International Physical Activity Questionnaire (short form) (IPAQ-SF). LBP subjects were required to complete a Roland Morris Disability Questionnaire (RMDQ) and to rate their level of pain on a visual analogue scale (VAS). During the recruitment process the groups were matched for age, body mass, gender and physical activity habits (type and experience). The study was approved by the ethics committees of both the University of Roehampton and the British College of Osteopathic Medicine.

7.2.2 Experimental Setup

Passive hip extensor moments during supine leg raising, and total hip moments during walking, were measured in accordance with the procedures described in chapter 6.

7.2.3 Data Processing

Passive contributions to total hip moments were calculated using the methods described in chapter 6. Hip moments were calculated within the Vicon Nexus Plug-in Gait software, using the conventional gait model based upon the Newington-Helen Hayes model. Total hip power was calculated based upon hip angular velocity and total hip moments. All data was subsequently normalised to body mass and height. Total hip moments were calculated during the peak of hip flexor moments (FL), and two hip extensor peaks (Ext1, Ext2) (figure 7.3). Total hip power was calculated at the two peaks of power generation (H1, H3) and peak of power absorption (H2). Mechanical work done at H1 and H3 was calculated by integrating the complete positive portions of the corresponding power generation curves. Work done was also calculated for the full negative power absorption curve (H2). Passive hip extensor moments and total hip moments, power and work done were additionally calculated for the portion of the gait cycle corresponding to hip flexion, between hip neutral and maximum hip flexion angle (figure 7.1). Scores for the RMDQ were calculated as the sum of ticked statements. Scores for RMDQ and VAS were entered into a Microsoft Excel spreadsheet for further analysis. Physical activity data was used to ensure matching of subjects in LBP and NBP groups.

7.2.4 Statistical Analysis

Independent t-tests were used to compare LBP and NBP group data using SPSS (version 24, IBM Statistics, U.S.). Comparisons included spatio-temporal

gait parameter data, maximum hip flexion angle, hip extension angle and knee angle. CMCs and CVs were used to assess intra-subject gait cycle characteristics. RMSE and the adjusted r-squared were used to assess goodness of fit of the predictive equation coefficients for calculating passive moment contributions. Passive hip extensor moments and total hip moments, power and work done were compared at 25% increments of hip flexion, between neutral and maximum hip flexion. Total hip moments were additionally compared at FL, Ext1 and Ext2, and total power and work done were compared at H1, H2 and H3.

7.3 Results

Subjects in the LBP group reported occurrence of back pain for 6.97 (5.98) years with a range of 6 weeks to 30 years. RMDQ scores were 3.92 (3.1) and VAS scores were 5.06 (2.27). Independent samples t-tests demonstrated no significant differences between LBP and NBP groups for any of the spatio-temporal gait parameters measured ($P > 0.05$, Supplementary Table D.31, Appendix D). There were no significant differences ($P > 0.05$) in any of the physical activity parameters measured, which included walking (13.6 (18.3) hours per week LBP, 9.9 (12.9) hour NBP), moderate intensity exercise (6.9 (11.0) hours per week LBP, 4.1 (5.6) hours NBP) and vigorous exercise (6.7 (8.7) hours per week LBP, 5.1 (3.4) hours NBP).

The gait cycle CMC means (SD) were 0.955 (0.037) for LBP and 0.937 (0.051) for NBP. The CV means (SD) were 21.4% (11.6) and 26.3% (13.9), for LBP and NBP, respectively. The RMSE was 0.016 (0.008) Nm/(kg.m) for LBP

and 0.017 (0.07) Nm/(kg.m) for NBP. The adjusted r-squared values were 0.889 (0.081) for LBP and 0.896 (0.075) for NBP.

Minimum hip angle was -12.42 (5.5) degrees and -12.02 (5.98) degrees ($P > 0.05$) for LBP and NBP, respectively, and occurred during the late stance phase, at approximately 50% of the gait cycle (GC) (figure 7.1). Maximum hip angle was 29.43 (5.31) degrees and 29.05 (4.5) degrees ($P > 0.05$), for LBP and NBP respectively, and occurred during late swing phase at approximately 90% GC. Stance phase terminated with the initiation of swing phase at approximately 60% GC for both groups ($P > 0.05$). There was a non-significant trend towards increased knee flexion during the first 65% GC, and greater knee extension from 70-90% GC in the LBP group ($P > 0.05$) (figure 7.2). Passive hip extensor moment contributions to total hip moments, power and work done were assessed from hip neutral to maximum hip flexion.

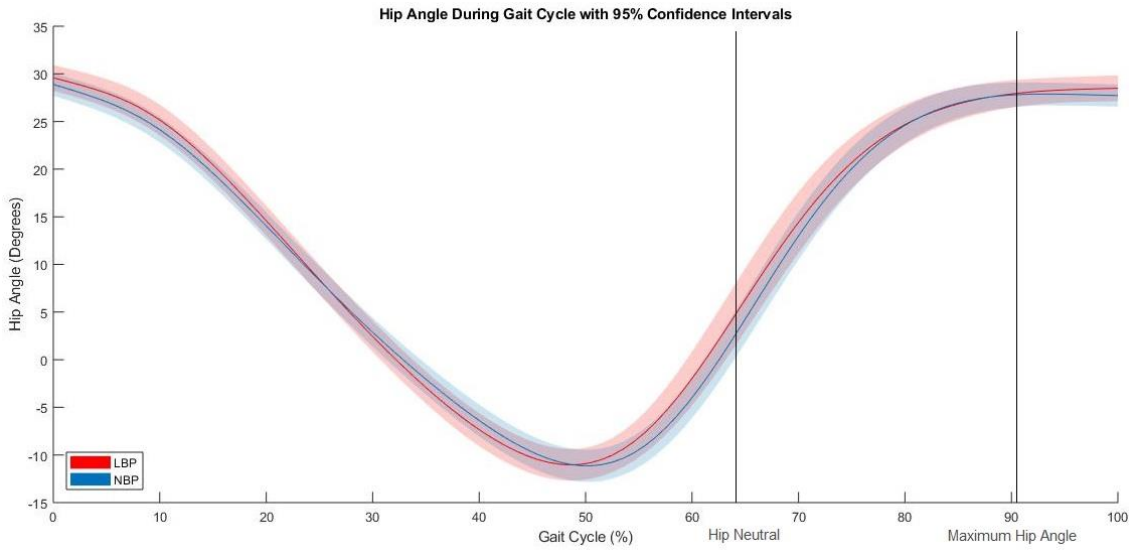

Figure 7.1 Mean hip angles with 95% confidence intervals for LBP and NBP subjects during complete gait cycle. Figure includes period of hip flexion further analysed between hip neutral and maximum hip flexion angle (vertical lines).

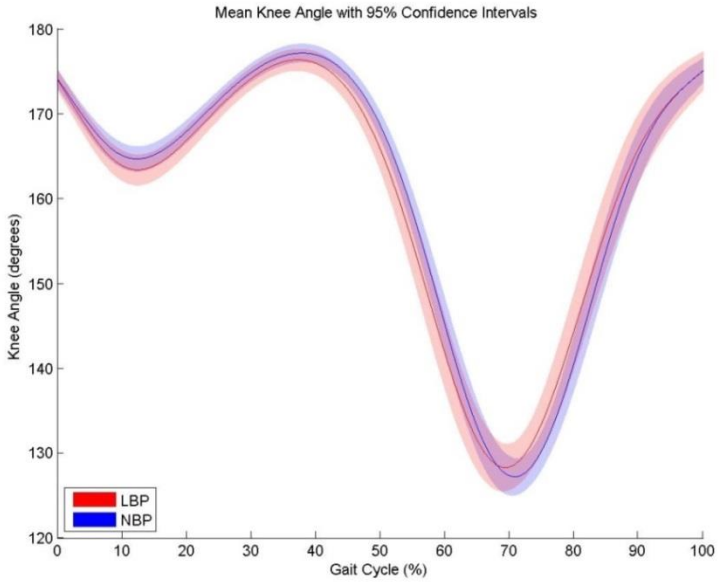

Figure 7.2. Mean knee angles with 95% confidence intervals of all subjects in LBP and NBP groups during gait cycle.

During early stance phase total hip moments (figure 7.3) were negative (hip extensor moments) and increasing to zero, becoming positive at 18% GC for LBP and 22% for NBP. Throughout mid-to-late stance total hip moments were positive (hip flexor moments), with a peak (FL) at approximately 50% GC for both groups. From late stance to mid-swing, total hip moments were positive but decreasing towards zero. Peak total hip flexor moments (FL) were greater in LBP (0.557(0.169) Nm/(kg.m)) than NBP (0.482(0.174) Nm/(kg.m)) ($P = 0.031$), with no difference in timings ($P > 0.05$). There were no differences in the total hip extensor peaks (Ext1, Ext2) or their timings ($P > 0.05$, table 7.1).

During the hip flexion component of the gait cycle, total hip moments were significantly ($P = 0.040$) greater (hip flexor moments) in LBP subjects (0.228 (0.083) Nm/(kg.m) vs 0.194 (0.08) Nm/(kg.m)) as the hip passed through zero degrees (hip neutral). There were no other significant differences between groups during the remainder of hip flexion ($P > 0.05$).

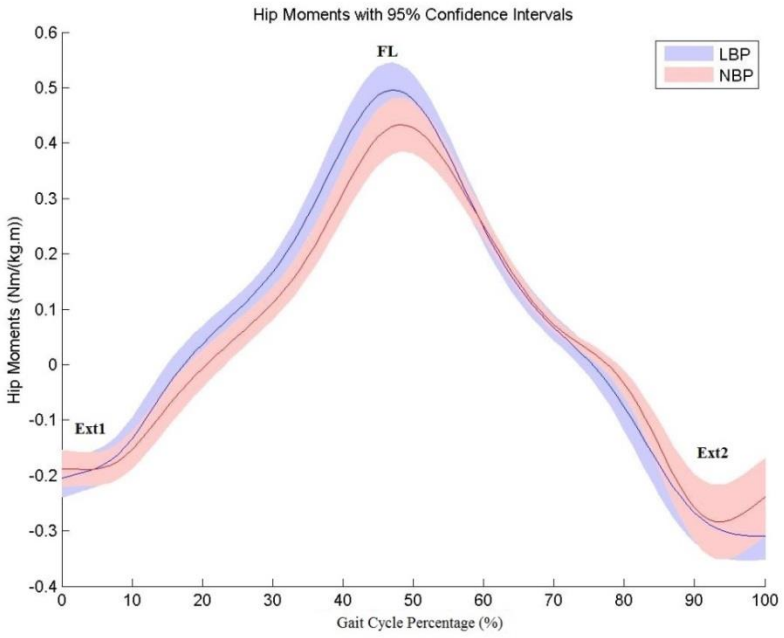

Figure 7.3. Mean total hip moments with 95% confidence intervals in LBP and NBP groups during gait cycle.

Passive hip extensor moments increased throughout hip flexion (figure 7.4), with no significant ($P > 0.05$) differences between groups at hip neutral or 25%. From 50-100% of hip flexion, passive moments were significantly ($P < 0.05$) more negative in LBP subjects, demonstrating greater extensor moments. At maximum hip flexion, the passive hip extensor moments were 46.6% and 39.4% of total hip moments, for LBP and NBP, respectively. Passive hip extensor and total hip moment mean, standard deviation and results of the independent t-tests are shown in table 7.2.

Table 7.1. Variable data for complete gait cycle for all subjects in LBP and NBP groups.

		Moments (Nm/(kg.m))				Gait Cycle (%)			
		Mean (SD)	CI Low	CI High	P-value	Mean (SD)	CI Low	CI High	P-value
FL	LBP	0.557 (0.169)	0.510	0.604	0.031	49.2 (4.4)	48.0	50.4	0.126
	NBP	0.482 (0.174)	0.435	0.529		50.6 (4.7)	49.3	51.9	
Ext1	LBP	-0.317 (0.122)	-0.351	-0.282	0.110	4.5 (4.0)	3.4	5.6	0.331
	NBP	-0.278 (0.119)	-0.310	-0.245		5.3 (4.0)	4.2	6.4	
Ext2	LBP	-0.434 (0.153)	-0.477	-0.391	0.732	92.9 (5.5)	91.4	94.5	0.515
	NBP	-0.421 (0.221)	-0.481	-0.361		93.6 (3.9)	92.5	94.6	
		Power (W/(kg.m))				Gait Cycle (%)			
		Mean (SD)	CI Low	CI High	P-value	Mean (SD)	CI Low	CI High	P-value
H1	LBP	0.280 (1.96)	0.225	0.335	0.990	8.2 (4.5)	7.0	9.5	0.049
	NBP	0.281 (0.159)	0.237	0.324		10.0 (4.4)	8.8	11.2	
H2	LBP	-0.520 (0.231)	-0.585	-0.456	0.041	42.2 (4.7)	40.9	43.6	0.137
	NBP	-0.429 (0.212)	-0.487	-0.372		43.6 (4.1)	42.4	44.7	
H3	LBP	0.844 (0.298)	0.761	0.927	0.045	61.8 (6.1)	60.1	63.5	0.616
	NBP	0.736 (0.235)	0.672	0.800		62.3 (3.9)	61.2	63.4	
		Work Done (J/(kg.m))							
		Mean (SD)	CI Low	CI High	P-value				
H1	LBP	0.028 (0.028)	0.020	0.036	0.329				
	NBP	0.034 (0.027)	0.026	0.041					
H2	LBP	-0.111 (0.059)	-0.128	-0.094	0.034				
	NBP	-0.087 (0.054)	-0.101	-0.072					
H3	LBP	0.115 (0.033)	0.106	0.124	0.017				
	NBP	0.101 (0.026)	0.094	0.108					

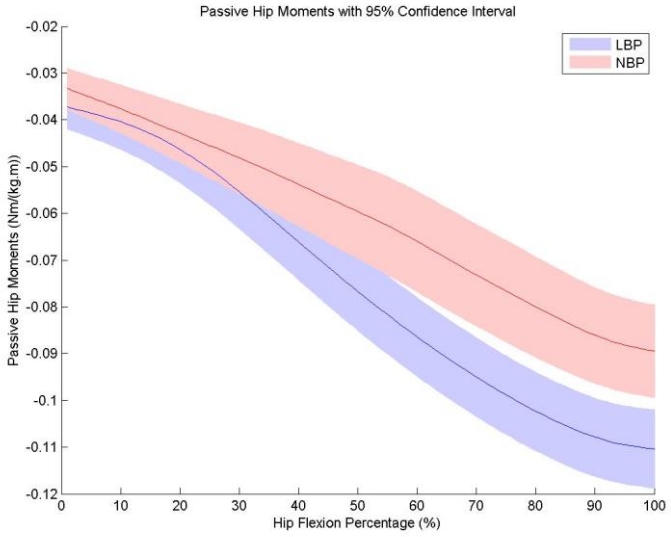

Figure 7.4. Mean passive hip extensor moments with 95% confidence intervals in LBP and NBP groups during hip flexion from neutral to maximum hip flexion

Table 7.2. Passive hip extensor and total hip moments (Nm/(kg.m)) during hip flexion, from neutral to full hip flexion

Hip Flexion Angle (%)			Hip Moments			
			Mean (S.D.)	C.I. Low	C.I. High	P-value
0	Passive	LBP	-0.037 (0.017)	-0.042	-0.033	0.225
		NBP	-0.033 (0.016)	-0.038	-0.029	
	Total	LBP	0.228 (0.083)	0.204	0.251	**0.040**
		NBP	0.194 (0.080)	0.172	0.216	
25	Passive	LBP	-0.050 (0.027)	-0.058	-0.043	0.345
		NBP	-0.046 (0.026)	-0.052	-0.039	
	Total	LBP	0.075 (0.069)	0.056	0.094	0.949
		NBP	0.074 (0.073)	0.054	0.094	
50	Passive	LBP	-0.077 (0.030)	-0.085	-0.068	**0.012**
		NBP	-0.060 (0.037)	-0.070	-0.050	
	Total	LBP	0.011 (0.043)	-0.002	0.023	0.283
		NBP	0.020 (0.044)	0.008	0.032	
75	Passive	LBP	-0.099 (0.03)	-0.107	-0.090	**0.002**
		NBP	-0.077 (0.040)	-0.088	-0.066	
	Total	LBP	-0.141 (0.168)	-0.188	-0.094	0.463
		NBP	-0.116 (0.167)	-0.162	-0.071	
100	Passive	LBP	-0.110 (0.030)	-0.118	-0.102	**0.002**
		NBP	-0.089 (0.037)	-0.099	-0.079	
	Total	LBP	-0.236 (0.192)	-0.290	-0.183	0.827
		NBP	-0.226 (0.257)	-0.296	-0.157	

Total hip power was generated during the first 20% of the gait cycle, with an initial peak of power generation (H1) during early stance phase in both LBP and NBP (figure 7.5). The first peak of power generation (H1) occurred significantly earlier in LBP than NBP (8.2 (4.5) %, 10.0 (4.4) %, for LBP and NBP, respectively, P = 0.049), with no difference in power (P > 0.05) (table 7.1). The hips then absorbed power, with a peak of power absorption (H2) during mid-stance. The peak of power absorption at H2 was significantly greater in LBP than NBP (-0.520 (0.231) W/(kg.m), -0.429 (0.212) W/(kg.m), for LBP and

NBP, respectively, P = 0.041), with no difference in timings between groups (P > 0.05). Total hip power was positive from approximately 50% to 85% GC in both groups, as the hip flexors generated power from the end of stance phase through to the end of swing phase. Power generation peaked at toe-off and the initiation of swing phase, at approximately 60% GC (H3). The second peak of power generation at H3 was significantly greater in LBP than NBP (0.844 (0.298) W/(kg.m), 0.736 (0.235) W/(kg.m) for LBP and NBP, respectively, P = 0.045), with no difference in timings between groups (P > 0.05). Total hip power became negative from 90-100% of the GC in LBP, as the hip muscles absorbed power at the end of the swing phase and initial foot contact, where in NBP they were positive. During the hip flexion component of gait (figure 7.6), total hip power was significantly greater (P = 0.012) in LBP subjects when the hip was in neutral (LBP = 0.717 (0.300) W/(kg.m), NBP = 0.583 (0.22) W/(kg.m). Mean, standard deviation and results of the independent t-tests for hip flexion are shown in table 7.3.

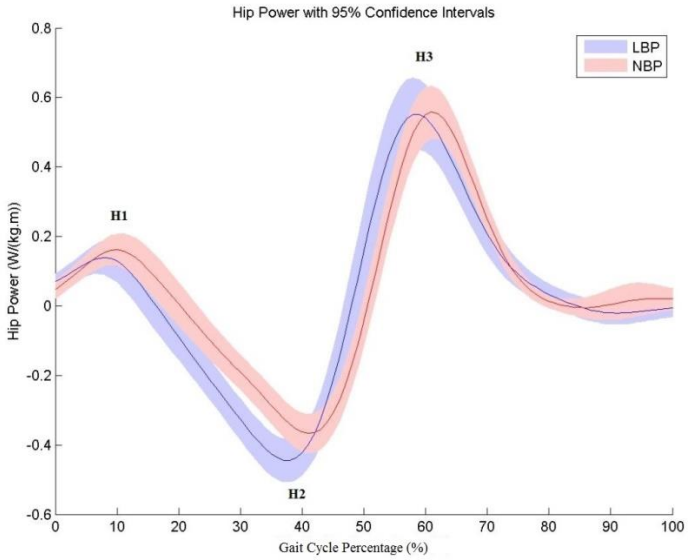

Figure 7.5. Mean total hip power with 95% confidence intervals in LBP and NBP groups during gait cycle.

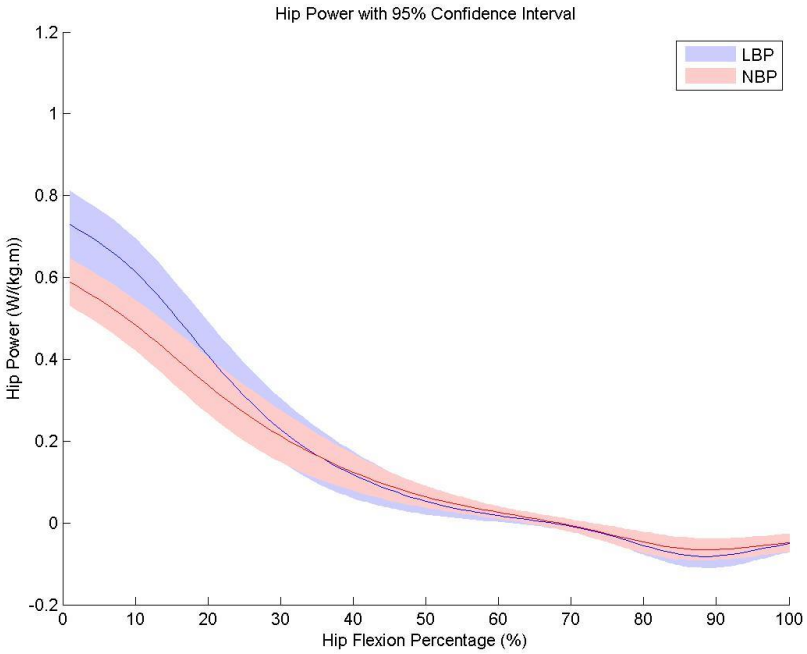

Figure 7.6. Mean hip power with 95% confidence intervals in LBP and NBP groups during hip flexion from neutral to maximum hip angle.

Table 7.3. Total hip power (W/(kg.m)) during hip flexion, from neutral to full hip flexion

			Hip Power			
			Mean (S.D.)	C.I. Low	C.I. High	P-value
Hip Flexion Angle (%)	0	LBP	0.717 (0.300)	0.633	0.801	0.012
		NBP	0.583 (0.220)	0.523	0.643	
	25	LBP	0.309 (0.287)	0.228	0.389	0.455
		NBP	0.269 (0.252)	0.200	0.337	
	50	LBP	0.053 (0.115)	0.021	0.085	0.608
		NBP	0.064 (0.098)	0.037	0.090	
	75	LBP	-0.027 (0.059)	-0.043	-0.01	0.928
		NBP	-0.026 (0.074)	-0.046	-0.006	
	100	LBP	-0.054 (0.076)	-0.075	-0.032	0.868
		NBP	-0.051 (0.085)	-0.074	-0.028	

Figure 7.7 shows the total hip mechanical work done during the H1, H2 and H3 gait cycle peaks. Negative mechanical work done during the H2 power

absorption curve was greater in LBP than NBP during H2 (-0.111(0.059) J/(kg.m), -0.087(0.054) J/(kg.m), for LBP and NBP, respectively, P = 0.034). Positive mechanical work was greater in LBP than NBP during the H3 power generation curve (0.115(0.033) J/(kg.m), 0.101(0.026) J/(kg.m), for LBP and NBP, respectively, P = 0.017). During the hip flexion component of the gait cycle (figure 7.8), total mechanical work done was significantly greater in LBP compared with NBP from hip neutral to 25% of hip flexion (LBP = 0.038 (0.017) J/(kg.m), NBP = 0.031 (0.015), P = 0.023, table 7.1). There were no other statistically significant differences in work done between groups (P > 0.05). Total work done mean, standard deviation and results of the independent t-tests for hip flexion are shown in table 7.4.

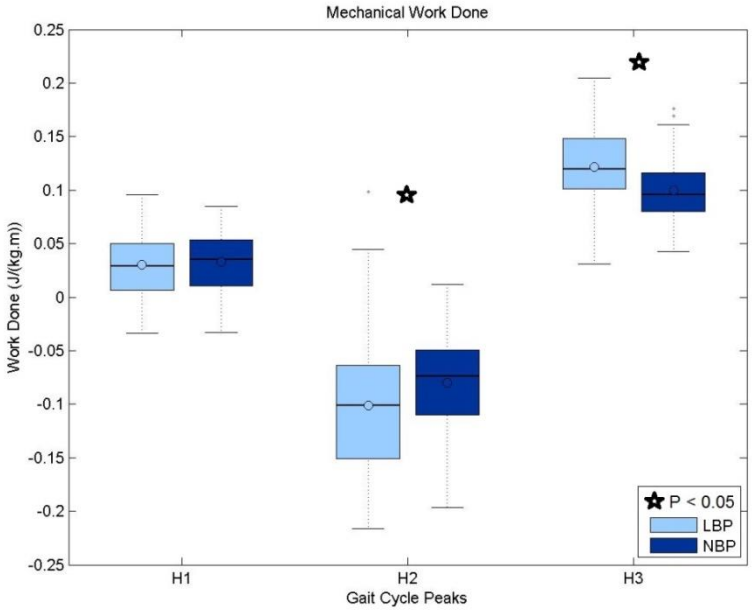

Figure 7.7. Total hip work done in LBP and NBP groups during H1, H2 and H3 gait cycle peaks

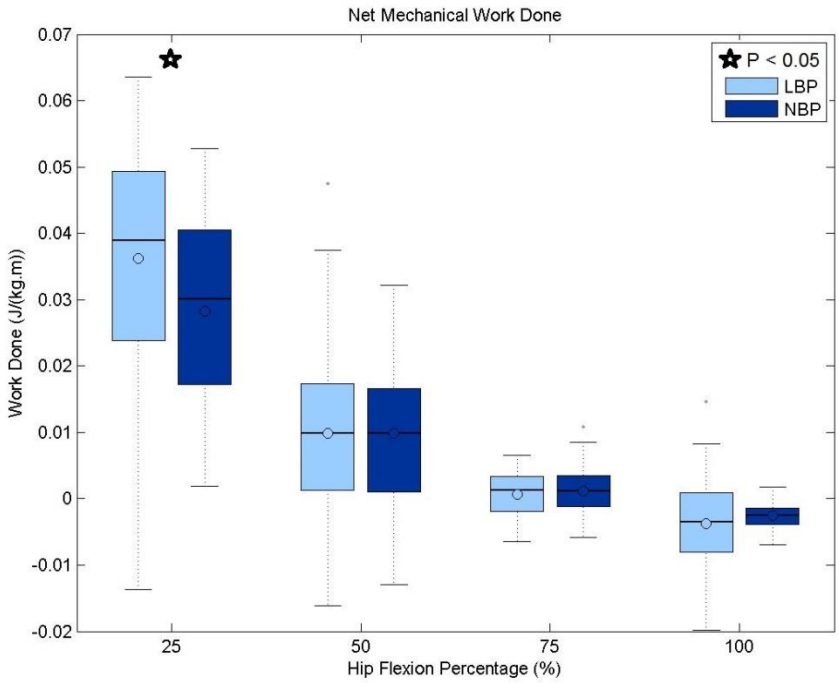

Figure 7.8. Total hip work done in LBP and NBP groups during hip flexion from hip neutral to maximum flexion

Table 7.4. Total hip mechanical work done (J/(kg.m)) during hip flexion, from neutral to full hip flexion in 25% intervals and for overall hip flexion

			Net Hip Work Done			
			Mean (S.D.)	C.I. Low	C.I. High	P-value
Hip Flexion Angle (%)	25	LBP	0.038 (0.017)	0.033	0.043	0.023
		NBP	0.031 (0.015)	0.027	0.035	
	50	LBP	0.009 (0.012)	0.006	0.013	0.790
		NBP	0.010 (0.011)	0.007	0.013	
	75	LBP	0.001 (0.003)	0.000	0.002	0.810
		NBP	0.001 (0.004)	0.000	0.002	
	100	LBP	-0.006 (0.007)	-0.007	-0.004	0.478
		NBP	-0.005 (0.007)	-0.006	-0.003	
	Flexion	LBP	0.043 (0.028)	0.036	0.051	0.321
		NBP	0.038 (0.028)	0.030	0.046	

7.4 Discussion

Passive hip extensor moments were 46.6% and 39.4% of total hip moments for LBP and NBP, respectively, at maximum hip flexion angle. This finding demonstrates that passive structures have a considerable influence on total hip moments during walking (Whittington *et al.*, 2008), and should be integrated into future biomechanical models.

The present study found no statistically significant differences in hip and knee angles between groups. Values of passive hip extensor moments, and total hip moments, power and work done for NBP subjects were similar to those reported elsewhere (Whittington *et al.*, 2008). Other investigators have reported alterations in hip and knee angles during walking in LBP subjects compared with healthy controls (McGregor and Hukins, 2009, Cimolin *et al.*, 2011, Vogt *et al.*, 2003). Reduced hip flexion and knee extension during walking would reduce stretch of the hip extensor and knee flexor muscles, potentially allowing active muscle contraction to absorb more impact stress, increase pelvic stability, and reduce pain or stress on the lower back (van Wingerden *et al.*, 1993, Jonkers *et al.*, 2002, Wakeling *et al.*, 2003, Vogt *et al.*, 2003, McKeon *et al.*, 2006), although this was not a finding of the present study. Muller *et al.* (2015), reported increased knee extension during walking in LBP at initial contact, hypothesised to modulate ground reaction force.

Passive moment calculations demonstrated significantly greater extensor moments in the LBP group compared with controls. Passive hip moments during walking are a product of the passive resistance to hip flexion and knee extension. The most likely cause of increased passive moments in LBP is an

increased passive resistance of the hip extensor and knee flexor muscles, such as the bi-articular hamstrings muscles. However, a previous study using the same subjects demonstrated no significant differences in passive moments, stiffness or strain energy between groups during supine leg raising (unpublished data, chapter 4). For this reason it is most likely that increased moments during walking in LBP results from small alterations in hip and knee angles. Because passive hip moments increase with hip angle in an exponential manner, it is predictable that small increases in hip angle have a considerable influence on passive moments during walking, as observed in the present study.

Other investigators (Ertelt, 2014, Vogt *et al.*, 2003) have reported earlier onset of biceps femoris activation in swing phase in LBP subjects, which could be required to control the hip and knee in anticipation for loading at terminal swing phase, and so may be activated earlier to decelerate the shank and to promote greater hip stability, particularly with increased hip angles during swing phase. However, Vogt *et al.* (2003) reported a reduced maximum hip flexion in LBP compared with healthy controls, in difference to the present investigation. It is plausible that individual alterations and interactions of joint angles and passive moments may be masked by averaging individual and group leg data. However, as no data is available on LBP and any influence on sidedness of joint angle differences (within and between LBP and control subjects and groups), it was not possible to investigate this within the present study. Because there were no group differences in passive moments during supine testing, differences in passive moments during walking are most likely due to active, neuromuscular factors controlling hip and knee angles.

Peak hip flexor moments (FL) were greater in LBP than NBP, during the late stance phase of gait. Hip flexor moments were also greater as the hip passed through neutral during the early swing phase, with no differences in hip moments during the remainder of hip flexion. Greater total hip flexor moments coincided with increased hip flexor power generation and mechanical work done in LBP compared with controls, as the hip passed through neutral in early swing phase. Greater moments, power generation and work done may be due to increased passive resistance earlier in swing phase or during the stance phase, but this was not measured in the present study. There were no differences between groups in passive hip extensor moments at hip neutral. Conversely, increased hip moments may coincide with increased muscle activation, with increased power generation either as a pre-emptive strategy to overcome the increased passive resistance later in the swing phase, or to increase hip-pelvic-lumbar stability.

Power absorption and work done at H2 were significantly greater in LBP than NBP. The H2 power curve occurs during the stance phase of gait, as the hip flexors eccentrically contract to stabilise the pelvis and support the trunk, preventing backwards movement of the trunk relative to the pelvis. Power generation and work done at H3 were also greater in LBP than NBP, and occurs during the late stance (pre-swing) phase of gait, as the hip flexors contract concentrically to pull the leg forward and facilitate toe-off. This coincided with greater power generation and work in LBP than NBP as the hip moved through neutral. Although there were no group differences in the H1 peak of power generation that follows initial contact in early stance, power generation occurred earlier in LBP than NBP.

The results of the present study demonstrate increased passive extensor moments in LBP, with no differences in total moments during the same portion of hip flexion. Whether the passive moments contribute to total moments, thereby reducing the need for active contraction and promoting efficiency, or if values of total moments should be altered due to the influence of greater passive moments, is not currently understood (Whittington *et al.*, 2008). Alternatively, altered joint angles, and therefore passive moments, later in the swing phase may follow the increased total power generation from the hip flexors during early swing, directly following toe-off. In this case, increased passive moments may be due to increased joint angles, resulting from greater momentum of the hip where active power generation is greater in LBP with no differences in passive moments following toe-off. Greater momentum of the swinging limb could cause greater hip flexion angles, contributing to greater passive resistance, and therefore higher passive moments. The lack of difference in total moments may reflect reduced active components late in swing, or be due to total moments being miscalculated due to lack of account of passive influences.

LBP subjects may adopt a variety of strategies to limit pain during walking, which may complicate attempts to identify generic walking adaptations (Simmonds *et al.*, 2012). However, investigators have reported altered muscle activation in LBP subjects during gait, including earlier onset and prolonged activation of paraspinal muscles (Arendt-Nielsen *et al.*, 1996, Vogt *et al.*, 2003) and gluteus maximus (Vogt *et al.*, 2003), and earlier onset of biceps femoris (Vogt *et al.*, 2003). It has been reported elsewhere that LBP subjects are unable to counter-rotate their thorax and pelvis during walking (Selles *et al.*,

2001), and demonstrate altered thorax and trunk rotation and trunk inclination (Muller *et al.*, 2015). LBP subjects have also been found to exhibit increased mediolateral centre of mass displacements compared with healthy controls (Henchoz *et al.*, 2015). Even low levels of LBP-related disability have been found to cause altered pelvic-trunk lateral flexion during walking than control subjects (Seay *et al.*, 2011). Frontal plane kinematics have been shown to be more influenced by LBP than transverse plane coordination during walking (Seay *et al.*, 2011), despite reduced lumbar rotation and no differences in frontal or sagittal lumbar kinematics reported elsewhere (Gombatto *et al.*, 2015). Differences may be due to severity of LBP, or in the regions assessed. Alterations in trunk and lower limb coordination, and altered biceps femoris activation may be linked in LBP (Muller *et al.*, 2015), although these were not assessed in the present study.

The results of the current study demonstrate alterations in passive and total moments, and total power and work done that may be due to alterations in muscle activation and neuromuscular control of walking. However, any cause and effect relationship between passive and total moments cannot be determined without prospective study. It would be useful to improve our understanding of such alterations during walking in LBP subjects. At present there is a lack of research relating to interactions between passive and total moments. Where the present study reported an increased passive moment with no change in total moments, it cannot be determined whether active components were reduced, or if total moments measurements were inaccurate due to models failing to incorporate the passive influence. Future studies should take into account passive contributions and an electromyographical

assessment of hip muscles, including muscle activation timing and magnitude, to develop our understanding of any interactions between passive and active components. Prospective studies of LBP, sports injuries and other musculoskeletal dysfunctions may help to determine cause and effect relationships between passive and active components.

Subjects in LBP research are often recruited from rehabilitation clinics and objective measures of LBP duration, severity (VAS) or disability (RMDQ) are not included (Tafazzoli and Lamontagne, 1996, Halbertsma *et al.*, 2001). In a study where pain and disability were included (Marshall *et al.*, 2009) the mean VAS score was 2.9 (2.4) and Oswestry score was 23.7 (11.5). Elsewhere, studies have reported mean RMDQ scores of between 10 and 13 (Wong and Lee, 2004, Shum *et al.*, 2007a, Shum *et al.*, 2007b) with mean VAS scores between 0.8 and 10 (Wong and Lee, 2004, Shum *et al.*, 2007a, Shum *et al.*, 2009, Shum *et al.*, 2007b, Lamoth *et al.*, 2006a, Muller *et al.*, 2015, Seay *et al.*, 2011, Song *et al.*, 2012). Thus, whilst the population in the present study reported RDMQ scores that were low (3.92 (3.1)) compared with other studies, VAS scores were considered moderate (5.06 (2.27)), and similar to those reported elsewhere. Although self-reported pain is a subjective measure, subjects in the studies where VAS and RMDQ scores were higher were recruited from physical therapy and rehabilitation clinics (Wong and Lee, 2004, Shum *et al.*, 2007a, Shum *et al.*, 2007b). Overall, the findings in the present study remain appropriate to the population assessed. Whilst LBP severity and disability may be lower than in subjects attending rehabilitation clinics, there were clear adaptations in gait to modify passive and active joint biomechanics compared with healthy controls.

A limitation of the current study was the measurement of hip passive moments during hip flexion only. In previous studies (Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008), researchers averaged hip moments during flexion and extension, and reported only a small underestimation of moments during flexion, and overestimation during extension. However, in the present study it was considered more appropriate to determine accurate values of passive hip extensor moments, and integrate these into the hip flexion component of the gait cycle, where hip flexor moments would be close to zero. Future studies would benefit from direct measurement of both passive hip extensor and hip flexor moments to model the complete gait cycle accurately in the sagittal plane.

Researchers (Muller *et al.*, 2015, Arnold *et al.*, 2007, Lyons *et al.*, 1983) have reported considerable contributions of the adductor muscles, hip flexors, glutei and ankle dorsiflexors during the hip flexion component of gait in addition to forces generated by the hip abductors and extensors during the stance phase (Arnold *et al.*, 2007). Because several muscles influence hip biomechanics in multiple planes, evaluation of frontal and transverse plane passive hip biomechanics may also be useful. Comparisons of LBP and healthy subjects may further integrate pelvic, lumbar and thoracic spine models and assessments. Such research may be useful to help elucidate any cause and effect interactions between passive and active components, and may be useful in determining more appropriate clinical assessments and interventions for LBP.

7.5 Conclusions

The present data demonstrates that subjects with LBP have altered passive hip extensor moments and total hip moments, power and work done during walking compared with healthy controls. Although it is not possible to extrapolate cause and effect relationships, rehabilitation programmes for LBP patients should differentiate between the active, neuromuscular components of movement, and the passive components. Biomechanical models should include individual measurements of passive joint moments. The approach used in the present study may be a useful measurement model for clinicians assessing low back pain.

8. General Discussion

8.1 Summary of Findings

Passive hip moments, stiffness and strain energy were measured in LBP and NBP subjects during supine leg raising tests at four predetermined knee angles. There were no main effects of group (LBP versus NBP), gender, leg, or knee angle at any hip angle ($P > 0.05$). The Figure 8.1 surface plot shows a subject example of the mean exponential curves for hip moments during passive leg raising, at each of the four fixed knee angles. The surface plot is constructed as the best fit across subject data, from which a predictive equation and its coefficients were derived.

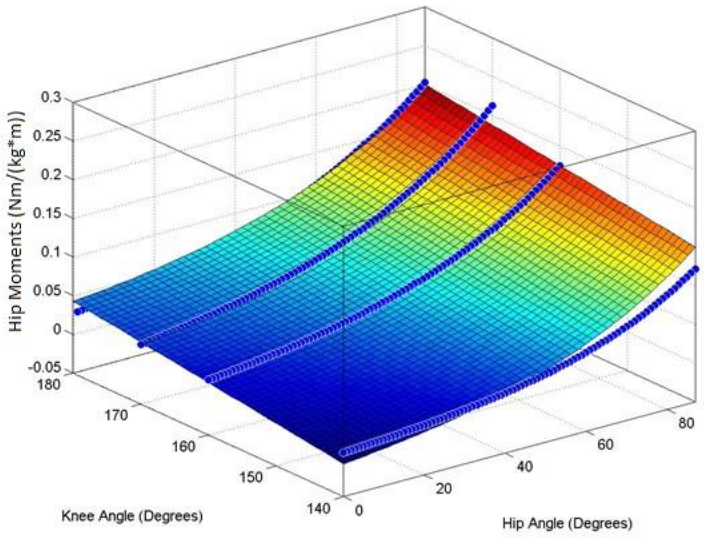

Figure 8.1. An example of a typical surface fit used to derive the coefficients for the predictive equation for one subject.

Passive hip moments were found to increase with hip flexion angle in an exponential manner. This finding is in agreement with other investigators (Silder *et al.*, 2007, Lee and Munn, 2000). The relationship between knee angle and hip extensor moments was found to be linear for any given angle of hip flexion. Thus, the predictive equation contains both linear and exponential functions, in addition to a constant. The following predictive equation (equation 6) can be used to determine the contribution of passive hip moments to dynamic movements at various hip and knee angles:

$$M_{passive} = a.\theta_{knee} + b.exp^{c.\theta_{hip}} + d$$

Equation (6)

where, *a*, *b*, *c* and *d* are equation coefficients, and θ_{knee} and θ_{hip}, are the angles at the knee and hip, respectively. Passive moments should be assessed on an individual basis during passive testing, and the predictive equation coefficients determined for individual subjects. The predictive equation can subsequently be integrated into calculations for total moments during dynamic movements, so as to establish passive contributions.

The coefficients obtained in the predictive equation (table 8.1) interact to produce the closest fit for each subject. A visual assessment of figures and a residual analysis were used to correct coefficient bounds and improve goodness of fit. As a consequence, the RMSE and adjusted r-squared showed an excellent goodness of fit. The RMSE was 0.016 (0.008) Nm/(kg.m) and 0.017 (0.07) Nm/(kg.m) for LBP and NBP, respectively. The adjusted r-squared values were 0.889 (0.081) and 0.896 (0.075) for LBP and NBP, respectively.

Table 8.1 Predictive equation coefficients for calculation of normalised passive hip extensor moments

	Equation Coefficients			
	a	***B***	***C***	***d***
Mean	0.001	-0.036	0.020	-0.094
SD	0.001	1.085	0.010	1.062
Min	0.000	-7.629	0.000	-1.592
Max	0.003	1.446	0.048	7.274

To improve our understanding of passive-elastic effects on joint mechanics during gait, in LBP and NBP subjects, passive hip extensor moments were calculated based upon the biomechanical model. Passive hip extensor moments were found to be significantly greater in LBP than NBP during the second half of hip flexion, mostly likely due to a trend towards increased hip angles. At the greatest hip flexion angles, passive hip extensor moments were 46.6% and 39.4% of total hip moments in LBP and NBP, respectively, demonstrating a considerable influence of passive tissues.

Peak hip flexor moments (FL) occurred during the late stance phase, and were significantly greater ($P < 0.05$) in LBP than NBP. This coincided with increased total hip power generation by the hip flexors and increased mechanical work done (H1, $P < 0.05$). Whether the increased moments and power generation led to the marginally increased hip flexion angles in LBP that caused greater passive moments cannot be known. Alternatively, the increased total moments, power and work done during late stance phase, and the increased passive moments during the second half of the swing phase were biomechanically independent, without cause and effect interactions. It is

plausible that increased passive moments will have reduced the amount of active muscle contraction, if total moments are assumed to be the product of passive and active contributions. However, it is unknown how passive components interact with the total moments, and joints are modelled with an assumption of no passive resistance. Due to the high contribution of passive moments to joint biomechanics, it is likely that a different modelling approach is required to accurately determine total moments, beyond the standard Newton and Euler-based equations, where the hip is assumed to behave as a frictionless line-hinge. It is likely that the precise interactions between passive, active and total joint moments require further investigation.

Differences between LBP and NBP subjects were also found during hip extensor power absorption and negative work done at H2 (chapter 7), where power absorption and negative work was greater in LBP ($P < 0.05$). Hip flexor power generation and positive mechanical work done at H3 were also greater in LBP than NBP ($P < 0.05$). Overall, it can be seen that the passive hip extensors of LBP and NBP subjects contribute considerably to the moments, power and work done of walking. There are clear differences in passive hip extensor moments and total hip moments, power and work done during walking in LBP and NBP subjects.

8.2 Comparative Studies

8.2.1 Passive Moments

From the initial investigation of passive hip extensor moments during supine leg raising, the values obtained for control subjects are in agreement with other published values (Gajdosik, 1991, Tafazzoli and Lamontagne, 1996, Lee and Munn, 2000, Halbertsma *et al.*, 2001, Halbertsma *et al.*, 1996, Halbertsma *et al.*, 1999). Differences between the data published in the present study and that of Yoon and Mansour (1982) are attributed to their different convention for describing joint angles. These differences have been similarly noted by other investigators (Brand, 1989, Vrahas *et al.*, 1990).

Tafazzoli and Lamontagne (1996) reported the passive elastic moment in terms of percentage of SLR, rather than at defined angles of hip flexion. This approach requires a consistent measure of SLR between individuals and across groups. In the present study, hip flexion ceased during testing when the subject reported pain, but this was considered too subjective to be very closely related to actual leg extension capacity. Hence, it was considered more appropriate to assess at defined increments, and to include maximum passive resistance within the statistical analysis.

There were no significant differences in SLR between LBP and control subjects. This finding is in agreement with that of Tafazzoli and Lamontagne (1996) and Halbertsma *et al.* (2001). Other investigators (Gajdosik, 1991, Gajdosik *et al.*, 1990, Gajdosik *et al.*, 1992), have reported a good relationship between the SLR and hamstring extensibility, and that the SLR may have

potential to indicate passive stiffness (Gajdosik, 1991). Blackburn *et al.* (2004a) reported that, although stiffness and extensibility are related entities, they are not synonymous. Overall, there is a lack of consistency in the literature regarding any relationship between passive joint moments, stiffness and hamstring extensibility (Blackburn *et al.*, 2004a).

8.2.2 Passive Stiffness

In agreement with the definitions by McNair *et al.* (1992), and others (Blackburn *et al.*, 2004b, Lee and Munn, 2000), stiffness is a specific term that refers to a resistance in change to length, and is the inverse of compliance. Stiffness therefore has a direct clinical relevance (Lee and Munn, 2000), and should not be considered as simply a synonym for extensibility or flexibility (Blackburn *et al.*, 2004a). Nor should stiffness be considered as equivalent to joint moment, as has also occurred in the literature (Halbertsma *et al.*, 2001, Gajdosik, 2001). In the present study, stiffness was calculated as the derivative of the moment-angle curve (Lee and Munn, 2000). The present study found no significant effects of muscle stiffness between groups ($P > 0.05$).

The results of the supine leg-raising investigation demonstrated that the passive moments and stiffness of the hip extensors were not influenced by chronic, non-specific low back pain, in agreement with Halbertsma *et al.* (2001). This finding contradicts that of Tafazzoli and Lamontagne (1996), who reported significant differences between LBP and NBP subjects. Testing in the Tafazzoli and Lamontagne (1996) study was performed using a dynamometer, with 9 LBP and 8 NBP subjects.

Marshall *et al.* (2009) reported no differences in peak hip moments or moments at 50 degrees of hip flexion. A difference in stiffness was reported between 20 and 50 degrees, with greater extensibility in NBP subjects. Stiffness in the Marshall *et al.* (2009) study was calculated based upon a linear line of best fit through the exponential curve between 20 and 50 degrees (the 'common range'). Muscle activity was permitted during the leg raising, which may have influenced stiffness during the common range, which was closer to maximum hip range in the LBP group. Thus, the approach of Marshall *et al.* (2009), does not take into account the steepness of the curve, only the moments at 20 degrees and 50, which could also influence their results.

8.2.3 Passive Strain Energy

Strain Energy during supine leg raising was calculated as the area under the moment-angle curve (Lee and Munn, 2000). Strain energy, in the context of this investigation, relates to the total amount of energy required by the operator to flex the subject's hip, against the resistance offered by the passive hip extensors and other tissues.

In active hip flexion, whether performed supine in an active test, or else during a functional activity, such as walking, strain energy represents the total energy required to rotate the hip. During active hip flexion, the total energy required will be influenced by passive-elastic muscle and tendon characteristics, active muscle contraction of the hip flexors, passive force enhancement, and inertial properties. It is reasonable to suppose that any increase in stiffness of the posterior hip extensors will require an increase in hip flexor muscle activity,

to meet the energy requirements for functional tasks (Vrahas *et al.*, 1990, Yoon and Mansour, 1982, Mansour and Audu, 1986, Riener and Edrich, 1999, Lee and Munn, 2000). The present study found no significant main effects of strain energy between groups ($P > 0.05$).

8.2.4 Hip Range of Motion

There was no relationship between maximum ROM and passive joint moments, stiffness or strain energy in the supine leg raising study. This is in agreement with Gajdosik (1991), who reported that different lengthening characteristics of those with short or long hamstrings is most likely a result of extensibility rather than maximum resistance to stretch. In the present investigation, hip ROM was significantly different ($P < 0.01$) between males and females but not between left and right legs, or between LBP and NBP (BPF = 81.8 (8.16), NPF = 82.3 (5.58), BPM = 62.8 (15.15), NPM = 67 (9.91) for left leg and BPF = 81 (5.62), NPF = 78.7 (11.23), BPM = 63.5 (10.9), NPM = 68.3 (10.43) for right leg).

Halbertsma *et al.* (2001) reported that there was no relationship between hamstring extensibility and maximum hip moments in those with or without LBP. Halbertsma *et al.* (2001) further reported that differences in extensibility in the control groups were similarly unrelated to hip extensor peak passive resistance. However, although maximum passive moments were similar across groups, the maximum hip flexion angle differed considerably.

In the study by Halbertsma *et al.* (2001), subjects were tested throughout the hip ROM, including after the onset of muscle activity. This makes it difficult to draw true comparisons, as the contribution of reflexive muscle activity is

difficult to determine. Indeed, the spread of data in that study raises questions regarding the population tested and the procedures used, with maximum hip moments in the LBP group recorded as 34.9 Nm with a standard deviation of 22.11 (Halbertsma *et al.*, 2001). Such a high standard deviation varies considerably from the data obtained in the present study and other published data (Tafazzoli and Lamontagne, 1996, Blackburn *et al.*, 2004a, Blackburn *et al.*, 2004b, Blackburn *et al.*, 2009b, Gajdosik, 1991, Gajdosik *et al.*, 1990, Gajdosik *et al.*, 1992).

Some studies (Marshall *et al.*, 2009, Raftry and Marshall, 2012) use only a limited ROM for assessment of passive properties (20-50 degrees). The rationale for such an approach has been stated as being that the common range enables comparisons to all subjects, independent of total hamstring extensibility (Raftry and Marshall, 2012). However, the range from 0 – 30 degrees is most useful for understanding hip passive properties during gait (Lee and Munn, 2000, Vrahas *et al.*, 1990, Yoon and Mansour, 1982). Passive properties when the hip is in neutral are representative of the resting values for the hip extensors, and so also has clinical relevance (Lee and Munn, 2000). Passive properties between 50 degrees and maximum ROM may have some relevance for understanding any relationship with joint extensibility, and may be of use in relating passive values to their contribution to functional activities at greater hip angles. Hence, some studies offer only a limited insight into passive hip moments and stiffness, and their conclusions may not have been drawn from the full range of data collected.

8.2.5 Modelling of Bi-Articular Muscles

Although other investigators have assessed passive contributions to joint moments (Lee and Munn, 2000, Blackburn *et al.*, 2009b, Blackburn *et al.*, 2004b, Yoon and Mansour, 1982, Mansour and Audu, 1986, Hatze, 1997), few have assessed influences of neighbouring joints (Yoon and Mansour, 1982, Mansour and Audu, 1986, Vrahas *et al.*, 1990, Riener and Edrich, 1999, Silder *et al.*, 2007). Including values for passive-elastic joint coupling across bi-articular joints is important for deriving accurate biomechanical information. Further, information can be determined regarding active contributions to total moments and active muscle timings and force production (Riener and Edrich, 1999, Mansour and Audu, 1986).

The involvement of passive multi-articular muscles in joint mechanics is often overlooked (Adouni and Shirazi-Adl, 2013, Hoy *et al.*, 1990). An understanding of these is important for multi-segment modelling, such as for interactions between the ankle, knee and hip joints during gait (Adouni and Shirazi-Adl, 2013). Adouni and Shirazi-Adl (2013) modelled muscle and ligament forces, and joint surface compression pressures during gait. Hoy *et al.* (1990) modelled muscle and tendon contributions to moment-angle relationships at the hip, knee and ankle joints.

Because the lower limbs are crossed by bi-articular muscles, such as the gastrocnemius at the ankle and knee, and the biceps femoris and rectus femoris at the knee and hip, the angle at one of these joints can influence passive biomechanical properties at a neighbouring joint (Riener and Edrich, 1999, Silder *et al.*, 2007). Hence, in developing models for understanding

passive contributions to gait and ADLs, it is important to assess moments at one joint whilst manipulating the angle at a neighbouring joint (Riener and Edrich, 1999). There have been few attempts to model any such relationships experimentally (Riener and Edrich, 1999, Mansour and Audu, 1986, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008). Further, any such assessments should include the physiological ranges used in gait or other ADLs, so that the model can be of direct use in assessing passive contributions to total joint moments.

Yoon and Mansour (1982) reported on the passive-elastic moments acting on the hip, and their experiment was followed by a similar investigation (Vrahas *et al.*, 1990). However, neither group reported moment-angle data throughout their tested ranges, and subject numbers were low in both studies (4 and 15, for the studies by Yoon and Mansour (1982) and Vrahas *et al.* (1990), respectively). Both groups reported different passive moments at the highest hip angles measured, which may be due to the small sample sizes used. In agreement with the findings of the present investigation, other investigators support a need to accurately measure individual passive joint moments for integration into modelling of more complex, dynamic activities (Yoon and Mansour, 1982, Riener and Edrich, 1999, Silder *et al.*, 2007).

8.2.6 Comparative Models

A similar approach to that used in the present study was developed by Silder *et al.* (2007) and used in subsequent investigations by the same researchers (Silder *et al.*, 2007, Whittington *et al.*, 2008). In their initial study (Silder *et al.*,

2007), 20 healthy adults were recruited (males, n = 9, age = 26.1 (4.1); females, n = 11, age = 25.5 (3.1)). Individual results were not reported, but a model for determining passive contributions was developed for integration into a gait model of active and total moments. In their model (Silder *et al.*, 2007), uni-articular components account for what is described as single-joint dependent stretch of ligaments, skin, inactive MTUs and the joint capsule. The models for bi-articular muscles were designed to account for energy transfer across the respective joints.

The model developed by Silder *et al.* (2007) for calculating passive hip moments ($\widehat{M}_h$), is as follows:

$$\widehat{M}_h = exp^{-\beta_h^{RF}\left(\theta_h - \left(\frac{\beta_k^{RF}}{\beta_h^{RF}}\right)\theta_k - \alpha^{RF}\right)} + exp^{-\beta_h^{HF}(\theta_h - \alpha^{HF})}$$
$$-exp^{\beta_h^{HAM}\left(\theta_h - \left(\frac{\beta_k^{HAM}}{\theta_h^{HAM}}\right)\theta_k - \alpha^{HAM}\right)} - exp^{\beta_h^{HE}(\theta_h - \alpha^{HE})},$$

Equation (7)

where, gain (β_{joint}^{muscle}) and offset angle (α^{muscle}) constants are used to determine passive moments, based upon hip (*h*) and knee (*k*) joints, and the contributions from the bi-articular rectus femoris (*RF*) and hamstrings (*HAM*), and the uni-articular hip flexors (*HF*) and hip extensors (*HE*) (Silder et al., 2007). Table 8.2 shows the mean (SD) constants from Silder *et al.* (2007) determined from the 20 subjects tested in their study.

Table 8.2 The mean (SD) constants determined from the 20 subjects tested in the Silder *et al*. (2007) study

	Gain, β		
Muscle	**Hip**	**Knee**	**Offset (degrees), α**
Rectus femoris (RF)	3.1 (1.4)	1.9 (0.7)	24.4 (9.0)
Hamstrings (HAM)	5.1 (2.0)	3.9 (2.5)	30.8 (14.9)
Hip flexors (HF)	5.1 (0.9)		19.5 (10.0)
Hip extensors (HE)	2.0 (0.9)		27.3 (18.0)

A potential limitation of the Silder *et al.* (2007) model is their averaging of moments derived from flexing and extending the hip. Silder *et al*., (2007) comment that by averaging the moment values the error is reduced. A strength of the approach used by Silder *et al*., (2007) is that it models for both hip flexion and extension moments, and assigns values to both uni-articular and bi-articular muscles. By contrast, a strength of the present investigation is that it promotes a simple model for predicting passive hip extensor moments during flexion, and does not require assigning values to individual muscles or muscle groups that may not be accurate. This is due to the inherent complexity of models that attempt to distribute muscle forces, as these may involve many parameters which cannot be directly measured. This consideration is in agreement with a similar statement by Riener and Edrich (1999), who suggest that distributing moments between muscles may only be appropriate if passive tissue loading or contact loading is of interest. It may be most parsimonious to have a moment calculation for flexion and another for extension movements.

A limitation of the present investigation was measuring only hip extensor moments during hip flexion. Other investigators (Silder *et al.*, 2007, Whittington *et al.*, 2008), measured both passive hip extension and hip flexion, and averaged the mean curves to establish an estimate of passive moments. This approach is open to errors, with hip moments during flexion being underestimated, and moments during extension being overestimated. A complete model of sagittal plane passive contributions to total moments during walking will require independent measures of both flexion and extension and for modelling to account for both hip position and hip and knee direction. The present investigation was concerned with hip flexion in particular, due to common clinical assessments of the hip extensors in subjects with LBP, and the potential value of assessing passive hip extensors during walking.

Should there be an interest in assessing muscles individually, it is important to consider the redundant problem in biomechanics, regarding the interactions of various muscles that act upon a joint. Further complexities are therefore encountered when attempting to model alterations in passive and active contributions to specific movements, and there may be limitations in the accuracy of such models (Riener and Edrich, 1999, Yamaguchi *et al.*, 1995).

The majority of investigators agree that it is important to account for the effects of bi-articular muscles on neighbouring joints (Riener and Edrich, 1999, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008), by calculating moments whilst controlling the angle of those associated joints. The present study and those of a few others (Riener and Edrich, 1999, Silder *et al.*, 2007, Whittington *et al.*, 2008), have developed models based upon appropriate experimental study.

A further strength of the present study is the large dataset used, when compared with the studies by Riener and Edrich (1999) (n = 10), and Silder *et al.* (2007) (n = 20). However, because the models in the present study and that of Silder *et al.* (2007) are subject-specific and group mean values are not derived, the larger samples do not directly influence the model itself, in difference to the models developed by Riener and Edrich (1999).

The model for calculating passive hip moments (M_H), developed by Riener and Edrich (1999) is as follows:

$$M_H = exp^{(1.4655-0.0034_{\theta K}-0.0750_{\theta H})} - exp^{(1.3403-0.0226_{\theta K}+0.0305_{\theta H})} + 8.072,$$

Equation (8)

where, *θK* and *θH* are knee and hip angles, respectively, in degrees. It was noted by Riener and Edrich (1999) that model error increased with increasing joint angles, and that there was a high deviation between experimental- and model-derived hip moments (> 10 Nm) when the knee was flexed to 90 degrees.

The high inter-subject differences in passive moments reported in the present study and others (Riener and Edrich, 1999), combined with the high deviations from the mean values obtained from published predictive equations (Riener and Edrich, 1999, Silder *et al.*, 2007), supports the need to determine passive-elastic joint contributions on a subject-specific basis.

Modelling precisely how passive elements contribute to active movements will be complicated by changes in tendon lengths and fascicle pennation angles, in addition to passive force enhancement. Because the

passive hip extensor component is up to approximately 40% of the total hip joint moment in NBP during gait, these complicating variables may be considered minimal to the overall passive joint resistance. Thus, although the model developed in the present study can be adapted and optimised to predict forces in specific muscles, at joint surfaces, and in other soft tissues, the increasing complexity of such approaches will likely increase error and not be of benefit to the overall model.

8.3 Strengths of Present Investigation

The present investigation utilised a custom-built, handheld force transducer that showed excellent accuracy and precision. Further, the production cost of the device was considerably lower than ISLR equipment, and has a greater versatility of application, making it accessible to clinicians and other investigators.

The values obtained for passive hip properties were similar to those reported elsewhere, but the predictive model and measuring approach was particularly robust. Direct measurements were taken without averaging of opposing movements, and values obtained were individual-specific. Measurements obtained were of total joint elastic properties, rather than of localised, superficial areas of the MTU. Values derived from the model represent the total MTU, and these were not subdivided into estimates of individual muscles, as such an approach ignores the redundant problem in biomechanics, and the contributions of contralateral limb and other muscles to joints they do not cross.

Other investigators (Riener and Edrich, 1999, Silder *et al.*, 2008, Silder *et al.*, 2007, Whittington *et al.*, 2008) have reported on the importance of measuring passive biomechanical properties at one joint, whilst manipulating the angle at a neighbouring joint, and the present investigation was one of the few to have done so. Further, the dataset was considerably greater than that of comparable studies (Riener and Edrich, 1999, Silder *et al.*, 2007).

Subject assessments were comprehensive, including measures of pain (VAS), disability (RMDQ) and lifestyle factors (physical activity habits). Reporting these values helped ensure lifestyle factors were matched between LBP and NBP subjects, and ensures such data is available to other researchers. Because physical activity may cause alterations in disability and passive biomechanical properties, it is appropriate to report such data. This may help improve our understanding of population differences when study findings are not in agreement.

The bi-articular biomechanical model developed in the present study is transferable to the calculation of passive properties at other joints and for other multi-articular MTUs. The model and approach are appropriate for the assessment of LBP, and can be utilised in evaluation of interventions to reduce LBP severity and related disability. Further, the model and approach can be integrated into assessments of pain elsewhere, and to improve our understanding of pain and injuries and their effective treatment.

8.4 Limitations of Present Investigation

The end ROM was determined subjectively as the point of onset of uncomfortable tension or pain, verbally communicated to the tester. Using the perceived onset of tension or pain is in agreement with other studies (Marshall *et al.*, 2009, Tafazzoli and Lamontagne, 1996, Halbertsma *et al.*, 2001, Lee and Munn, 2000, Vrahas *et al.*, 1990), with some communicating verbally (Tafazzoli and Lamontagne, 1996, Vrahas *et al.*, 1990, Lee and Munn, 2000) and others using a button during ISLR (Marshall *et al.*, 2009, Halbertsma *et al.*, 2001). Tests have also been ceased when the tester felt firm resistance to stretch (Tafazzoli and Lamontagne, 1996). Any extraneous movement of the pelvis was also determined to be indicative of end ROM, and the end of the test. This is in agreement with other studies (Vrahas *et al.*, 1990). In the present study, data was collected throughout the ROM, and analysed at hip angles between neutral and 60 degrees. Because the number of subjects reaching higher ranges of hip motion was considerably limited, particularly beyond 70 degrees, values above 60 degrees were excluded from analysis.

Some minor errors may have occurred due to incorrect measurements of limb lengths and hip joint centre of rotation, which were determined based upon the palpated tip of the greater trochanter. This has been the finding of other investigators (Vrahas *et al.*, 1990, Riener and Edrich, 1999, Silder *et al.*, 2007), and such errors may be responsible for day-to-day variations in passive moments (Vrahas *et al.*, 1990). In the present study, all measurements were made during a single visit, and any differences in anthropometric

measurements or joint centre positions between left and right legs were checked.

De Leva (1996b) indirectly assessed hip joint centre in 6 cadaveric specimens, and reported that the average location of the hip joint centre was 0.7% of the distance from the tibial plateau to the greater trochanter, superior to the greater trochanter. The values obtained in the de Leva (1996b) study were based upon reported values from direct anthropometric measurements, reported in an earlier study (Chandler, 1975). Although the estimation of hip joint centre using the method proposed by de Leva (1996b) was recommended as a means of improving accuracy, with such a limited sample and indirect assessment it is difficult to know how representative the findings might be to the population in the present study.

The hip joint centre can be estimated using predictive equations based upon regression and via a functional method (Camomilla *et al.*, 2006, Leardini *et al.*, 1999, Piazza *et al.*, 2004, Piazza *et al.*, 2001). However, these still rely on accurate determination of palpated bony landmarks and are therefore still susceptible to errors (Piazza *et al.*, 2004, Leardini *et al.*, 1999, Camomilla *et al.*, 2006). Due to the complexity of such measurements and limited efficacy for their use (Leardini *et al.*, 1999, Piazza *et al.*, 2001), these approaches were not included in the present study, although they may have been of benefit. It may be that biomechanics studies will benefit most from improved equations for calculating the location of the hip joint centre, based upon the static location of the greater trochanter and anterior and posterior superior iliac spine.

Hip joint moments were calculated based upon the measured leg segment lengths, leg mass and segment centre of mass. Segment mass and

centre of mass data is typically beyond the scope of biomechanics researchers to measure directly. Instead, estimates are based upon published data from limited samples (Dempster, 1955, Zatsiorsky, 1990), with subsequent refinements to reduce measurement error (de Leva, 1996a). These samples may include cadaveric specimens (Dempster, 1955) and the populations may not be closely representative of the populations used in biomechanics and kinematics research. Cadaveric specimens are generally of elderly subjects, so not representative of a younger population. By contrast, Zatsiorsky (1990) used a sample of 100 male and 15 female Caucasian subjects and calculated body segment parameters using gamma-ray scanning. In the study by Zatsiorsky (1990) the subjects were undergraduate students, with mean ages of 24 and 19, for males and females, respectively, and may not be representative of an older population. Investigators do not generally reference the source of body segment data used in their studies, which may lead to differences in moment calculations.

The Zatsiorsky (1990) data reported body segment data relative to bony landmarks that were sometimes distant from joint centres, meaning that movement would increase such distances and increase error (de Leva, 1996a). de Leva (1996a) revised the Zatsiorsky (1990) data to improve reliability, although the Dempster (1955) is most commonly used (Winter, 2005), including in the present study.

There is a potential for joint moment measurement errors due to the limited available body segment parameter (BSP) data. Altering body segment data for segment mass and centre of mass will have a small effect on hip moment data. Using one typical subject from the present investigation as an

example, instead of calculating based upon Dempster (1955) BSPs, if the data from Zatsiorsky (1990) is used, hip moments are altered at 30 degrees of hip flexion from 9.5 Nm to 9.9 Nm, and at maximum ROM from 29.5 Nm to 31 Nm, in the straight leg condition. Further research is necessary to improve BSP equations and make them more appropriate to individual subjects. This can be achieved by collecting data based upon radiographic or ultrasound-based measurements of different populations, thereby accounting for effects of age, gender, body composition and lifestyle factors.

The degree of lumbar lordosis may have influenced the position of the pelvis during testing. Lumbar lordosis was not measured in the present study. A similar study by Halbertsma *et al.* (2001) found no differences in lumbar lordosis between subjects with or without LBP, or between those with normal or reduced hip ROM.

There is potential for error from skin movement relative to the total limb (Riener and Edrich, 1999), such as where movement leads to measurement errors from electro-goniometers. Pilot investigation prior to the present study highlighted the effects of skin movement on distortion of electro-goniometer data. For this reason electro-inclinometers were used instead, with the devices secured to a single strap and mounted on the thigh (for measuring hip angle) and the shank (for measuring knee angle). This limited the effects of skin movement, and was found to be a more reliable method than the use of electro-goniometers.

It was not possible to keep the knee entirely rigid during the leg lifts when the knee was flexed. This was due to compression of soft tissues between the rigid brace and wide, Velcro straps. Although neither the brace nor the straps

moved, the tissues either side of the knee could be compressed as muscle positions shifted during hip flexion, permitting some movement at the knee. This movement was measured throughout the lift using the electro-inclinometers, and the biomechanical model accounted for this input of data. The mean deviation from the target knee angle was less than five degrees for each knee angle.

During leg raising tests, there was potential for subjects to rotate their hips in transverse or frontal planes, or to rotate the pelvis during tests, although if this was sufficient to be observed the test would have been repeated (however, this did not occur to any observable extent). Lee and Munn (2000) used an audible alarm to report a deviation from the intended direction of joint rotation (five degrees in the frontal plane) or out-of-plane force (10 Nm). Other investigators (Riener and Edrich, 1999) demonstrated that angular deviation from the tangential direction does not strongly influence joint moment measurements. Those investigators (Riener and Edrich, 1999) calculated that a 1.54% overestimation of joint moment followed a 10 degree angular deviation. The experimental set-up in the present study prevented frontal plane rotation of the hips.

A potential source of error was non-tangential force application through the force transducer and hip joint by the tester. This was reported as a potential source of error in previous studies (Riener and Edrich, 1999), and was found to be sufficiently small as to be considered acceptable. The tester attempted to consciously limit any such non-tangential force application, and the use of three lifts, with the curve fitted to the smoothed mean being an appropriate means to further reduce the effects of any such external force application.

The present study produced a predictive equation for passive hip moments during hip flexion. During testing, any movement in frontal or transverse planes was restricted. However, complete modelling of the hip during gait should take into account such movements. For example, the long-head of biceps femoris has been found to resist the knee adduction moment in the frontal plane, in addition to resisting the knee extension moment in the sagittal plane (Adouni and Shirazi-Adl, 2013).

Hip moments were assessed at various combinations of hip and knee flexion, with the ankle supported in a neutral position. This may be considered a limitation if using the data to predict passive hip moments whenever the ankle is not in neutral (Riener and Edrich, 1999, Mansour and Audu, 1986, Palmer *et al.*, 2014). However, Adouni and Shirazi-Adl (2013), modelled ankle, knee and hip biomechanics during gait, and reported that optimisation techniques that included the ankle had minimal effects on muscle forces and knee joint response. Thus, the influence of varying ankle positions on hip moments during gait can also be expected to be minimal. The use of 3 degrees of freedom modelling of the hip during walking was considered appropriate for the assessment total hip moments in the sagittal plane. These measurements would be required for the integration of the predictive equation derived from passive sagittal measurements.

8.5 Future Research

The present study assessed passive moments about the hip at different knee angles. It would have been useful to also assess passive properties of other hip

and lumbar spine musculature, including around the hip joint during hip extension from flexion, and movement in frontal and transverse planes. Such investigations would provide a more complete understanding of the relationship between the passive structures about the hips and spine in individuals with LBP. Integrating this additional level of information into the gait model used in the present study would help improve our understanding of total hip passive-active interaction in LBP. Further, the use of electromyography may help to determine the influence of passive characteristics on active contraction timing and magnitude, and any altered active behaviour of the hamstrings, gluteals and erector spinae, as has been reported elsewhere.

Although the predictive equation was determined for the calculation of passive hip extensor moments, the approach is appropriate for any joints where multi-articular muscles interact to influence moments. Future research should develop the sagittal-plane hip flexion model developed here into a 3-D model to assess passive hip contributions throughout gait and other ADLs. Such an approach can be expanded to describe agonist-antagonist influences on biomechanical variables, muscle activation and force production, and bioenergetics. Further research can utilise such models within studies to assess sensorimotor control of gait and other ADLs, to help improve our understanding of central influences on movement (Herzog *et al.*, 1995). Future research might also benefit from integrating an individual-focussed, principle component analysis, or similar, sufficiently sensitive to detect individual-specific alterations in biomechanics, rather than grouping for comparisons.

Improved reporting on physical activity habits would be useful for study comparisons, with a specific definition of what constitutes a sedentary individual

(if the study is to recruit from this population). Should future investigations demonstrate chronic alterations in passive-elastic properties due to specific types of physical activity, it will be seen that matching subjects for these is appropriate. With such a scarcity of literature on physical activity and passive-elastic muscle properties, it was considered appropriate to match subjects in the present study. Future studies should compare passive joint properties of sedentary individuals to those from a variety of physically active populations, including both recreational and professional athletes.

An interventional approach that targets the active components of movement should be developed and tested. This work should be extended to improve our understanding of pain-avoidance adaptations in other conditions, such as other chronic pain and sports injuries, and to help develop the most appropriate and effective rehabilitation techniques. Regardless of the potential for further development and optimisation, the approach used in the present study is sufficient to quantify passive-elastic contributions to movement across joints with bi-articular muscles. Such information can be used to improve biomechanical assessments, and to assess the efficacy of injury treatment and prevention strategies, as well as short- and long-term effects of pain and injury.

During the present investigation, passive differences were detected during gait but not during supine leg raising with a controlled knee angle. This suggests there may be subtle modifications of joint angles during gait that increase the passive component in LBP subjects. This should be explored further, and might benefit from subject-specific analyses. Without changes in total moments, it may be that passive and active components interact to maintain a normal total moment, or calculations of total moments need to be

modified to account for the influence of passive moments. Future studies should explore potential cause and effect relationships, and interactions between passive and active components. Overall, the findings indicate that future research is needed to develop our understanding of passive-active relationships, and physical therapy interventions should be developed to target these distinct components separately in people with LBP. The biomechanical model and predictive equation should be integrated into such experiments, combined with electromyography, to fully assess interactions and effects of both passive and active components. This approach can be expanded to explore other sources of chronic pain, musculoskeletal injuries, and related dysfunctions.

9. General Conclusions

1. A novel handheld device was developed in this study to assess passive joint characteristics during passive leg raising, and was found to have good reliability and versatility of application. The device was used to assess passive hip joint characteristics in LBP and control subjects. It was found that LBP did not significantly affect passive joint moments, stiffness or strain energy of the hip during passive straight leg raising.

2. A predictive equation was developed in this study to derive the contribution of passive hip extensor moments about the hip during flexion, in dynamic movements utilising a variety of hip and knee angles. By using this equation it was found that the contribution of passive-elastic tissues to joint biomechanics is significant and can be considerable. Models of dynamic movements that do not include a measure of passive contributions are therefore at risk of overlooking an important biomechanical component.

3. By using this equation, the present study demonstrates a range of specific biomechanical alterations during walking in subjects with LBP. These alterations influence passive hip extensor and total hip moments, power and mechanical work done. Although it is not possible to extrapolate cause and effect relationships, rehabilitation techniques that address individual biomechanical alterations in LBP patients should

target the active, neuromuscular components of movement, rather than passive, structural components.

4. This work demonstrates that clinical assessments and biomechanical studies that do not account for passive and active contributions are missing an important and commonly overlooked component. Passive and active components likely interact and identifying which component is influenced by low back pain will have direct clinical relevance to rehabilitation and therapy. The traditional biomechanical modelling of the hip as a frictionless line-hinge should be re-evaluated to account for passive elastic contributions to total joint moments.

5. Manual therapists, other clinicians and researchers interested in developing interventions to treat LBP could benefit from using the approach used in this thesis, in order to differentiate between passive and active components in clinical assessments. This approach could subsequently be used to evaluate the effectiveness of interventions that target passive or active components in LBP patients.

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Appendices

Appendix A

Forms and Questionnaires

A.1 Participant Information Sheet

Roehampton University London

INFORMATION SHEET FOR PARTICIPANTS

Research Title:

Hip-Spine Interaction in Individuals with Low Back Pain: The Role of the Hip Extensors

Invitation Paragraph

We would like to invite you to participate in this research project. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others as you wish. Please ask us if there is anything that is not clear or if you would like more information. Please take your time in deciding if you wish to take part.

If you do decide to take part, you will be given this information sheet to keep and asked to sign the attached consent form to say that you agree and understand what this study is about. You are free to withdraw at any time and without giving reason.

Thank you for reading this.

What is the purpose of the study?

The aim of this project is to investigate how the biomechanics of the hips and spine, and muscle stiffness of the hamstrings, vary between people with and without lower back pain. We hope our findings will be used to improve the effectiveness of treatment for people with back pain, and to give us more information about how different people are affected by back pain. We also hope to find out if there are changes over time in people with pain (i.e. if back pain for 6 months affects people differently to back pain over several years).

Who have we asked to participate?

Anyone aged 18-50, either with or without back pain. *Female subjects will be requested to attend during the follicular phase of their menstrual cycle (within the first 7 days of the start of menstruation).*

Who must we exclude?

Unfortunately, we ask you not to participate if you have tumours, rheumatological disorders, tuberculosis or an infection in your spine, hips or knees, or if you have had a dislocation or surgery of the spine and lower limbs at any time of your life. You will not be able to participate if you are pregnant or allergic to ultrasound gel or adhesive tape.

We will ask you not to participate if you have an orthopaedic implant (a medical device that replaces part or a whole joint) or an electrically powered medical implant (for example a pacemaker, an implantable defibrillator, a cochlear implant, neurostimulators or an insertable cardiac monitor).

When and where will the study take place?

The study will take place in the biomechanics laboratory inside Whitelands College of Roehampton University. It will take place at a time that is convenient to you.

How long will the study last?

You will be asked to come to the biomechanics laboratory for a single visit. A medical screening form, informed consent form, and Roland-Morris Disability Questionnaire will have to be completed before the testing can begin. The testing will last no more than one hour.

What will happen to me if I take part?

Leg Raise Test

What does it tell us?

The straight-leg raise test is commonly used by manual therapists to assess hip mobility. The results of the test are purported to indicate whether or not there are any functional limitations in the hip, such as might contribute to low back pain. We will perform two versions of the test. The first is simply to test movement of the hip, and the second is to measure the stiffness of the hamstring muscles. The reason for this is because range of motion (ROM) and muscle stiffness are two different – not necessarily related – aspects of muscle function, and we are interested in measuring both. We can then see if there is a relationship between the two, and whether or not there is a

relationship between the two *and* how an individual walks.

How is the test performed?

Before the test is performed the tester will measure your height and weight, and then use a tape measure to find the length of the leg (to be used in calculations later on). The test is performed whilst lying supine on a massage couch with the legs straight. A digital goniometer will be attached to the hip and upper thigh, as this will give an accurate measurement of hip angle during the test. A knee-brace (commonly used in rehabilitation to support the knee), will be used to fix the knee at various angles. Electromyograph (EMG) electrodes will be placed on the front and back of the legs, as this will tell us if any muscle activity contributes to the movement. A device containing a load cell will be held by the tester beneath the ankle to measure resistance from the leg. The tester will support the lower leg in his hands and raise the leg, until the natural end of hip range of motion is met. This test will be repeated three times on each leg.

What is required of the participant?

You should be wearing loose-fitting shorts, swimming trunks or underwear for the test. This is to ensure that the tester can access the thigh to attach sensors for the test. A screen will be provided to ensure privacy. During the test, you will be lying on your back on a massage couch, with your hips close to one end and your legs supported on a table. Sensors and a knee and ankle brace will be fitted, and the tester will raise your legs whilst measuring the resistance given by your relaxed muscles. This enables us to measure the stiffness of the joint and other tissues.

Kinematic Analysis

What does it tell us?

In our kinematic analysis, we will be measuring how the legs move relative to the lower back, hips and knees. We will be wanting to know what sort of angles and what sort of velocities the limbs move at. From this we will be able to measure forces through joints, and compare the gait of people with back pain and those without. In addition, we will use electromyography (EMG) to measure the electrical activity of the muscles.

How is the test performed?

Vicon sensors are small round devices that can be attached to the skin by adhesive tape. The movement of the sensors is then captured by a series of cameras in our biomechanics laboratory. We will attach those sensors to the front and back of the hips, the base of the spine, the spine where the lowest ribs attach, the thigh, lower leg and back of the foot. As you walk across the floor, a force platform recognises when your feet touch it, and it records the force down through it. This is synchronised with the Vicon system. We will ask you to walk along the walkway a number of times, until we have three clear images of each foot on the force platform. For measuring muscle electrical activity, we will attach self-adhesive electrodes to key areas of the body: the muscles at the front and back of the thigh, the front of the hip, the butt, and the lower back. Leads from the electrode pads will be attached to a portable device which we will ask you to carry in a small rucksack over both shoulders

What is required of the participant?

All participants will be required to wear short, loose-fitting shorts. Women will be required to wear a sports bra, to allow for placement of reflective markers at the base of the sternum (breastbone) and at various points on the back. Men will be required to remove their tops for this aspect of testing. There is sometimes interference from jewellery, which negatively affects the sensors, and so we would prefer participants not to wear necklaces, watches or bracelets (rings should be fine, or alternatively a plaster can be placed over them). Jewellery and such like can be safely stored out of range of the Vicon cameras.

Once the tester has affixed the markers it will be necessary to check that they can all be 'picked up' by the cameras. A brief calibration of the system will be required at this point, although it will only last a minute or so. Once the unit is ready you will be asked to walk, at your normal walking speed, across a plate that will measure foot pressures. We will request that you repeat this until we have sufficient clear images from the camera and force platform.

Are there any risks involved in participating?

The sensors and adhesive electrode pads do not have any health risks known to the researchers. The adhesives used should not cause any allergic reaction. If any concern or discomfort is felt either before or during testing, please tell us. The tester will be there to monitor you, to talk you through the tests, and will

expect you to tell him if you want to stop (the tester will stop the test in any case, if he feels it is the appropriate thing to do).

Are there any benefits involved in participating?

At the conclusion of the project, if you agreed to give us your personal contact information we will send you a description of the major findings as well as reference to research publications generated from this project.

Will we compensate you for your time?

You will not have any financial benefit from the research in any respect.

How will we maintain your privacy and confidentiality?

To preserve anonymity you will be given an identification number known only to the principal researcher and the project supervisors. Using the same number, data will be stored on a password secure computer within Roehampton University ensuring your individual information remains confidential. Once the results are reported it will not be possible to identify individual persons. After the study, data will be stored in the same way for a period of ten years after which it will be destroyed.

What will happen if I don't want to carry on with the study?

You have the right to withdraw from this study at any time, without fear of prejudice. If you decide to withdraw, please tell the principal investigator at any time. If you withdraw, it may be beneficial to use your data already collected up to the point of withdrawal, but all other data will be destroyed.

Who is organising and funding the research?

This research is organised by the investigators outlined below along with the Department of Life Sciences, Roehampton University, and with partial funding from the British College of Osteopathic Medicine and British Naturopathy and Osteopathy Association. This study has been subject to full ethical review, approved and funded by Roehampton University.

	Name	University Address	Email	Telephone
Principal investigator	**Mark Hines** PhD Researcher	Department of Life Sciences, Roehampton University, Whitelands College, Holybourne Avenue, London, SW15 4JD	hinesm@roehampton.ac.uk	+44(0)7788 561898
Supervisor	**Raymond Lee** Professor of Biomechanics and Head of Department		r.lee@roehampton.ac.uk	+44(0)20 8392 3539

What if I have questions about the project?

For information about this research study, please speak with the principal investigator.

What if there is a problem or complaint?

Technical staff fully trained as first-aiders will be available throughout your participation.

If there is a problem at any time and you would like to contact someone independent of the study then your contact should be:

	Name	University Address	Email	Telephone
Department of Life Sciences	**Siobhan Strike**	Department of Life Sciences, Whitelands College, Roehampton University, Holybourne Avenue, SW15 4JD	s.strike@roehampton.ac.uk	+44 (0)20 8392 3546

If you are a student in Roehampton University and feel any physical or emotional discomfort about any aspect of the study, please contact your **Student Welfare Officer** who will be able to advise you on support groups that can deal with your particular concern:

College	*Officer*	*Telephone*
Frobel	Anne-Marie Joyes	+44 (0)20 8392 3304
Digby Stuart	Jo Granger	+44 (0)20 8392 3204
Southlands	Belinda Scott	+44 (0)20 8392 3402
Whitelands	Ejiro Ejoh	+44 (0)20 8392 3502

If you feel that your concerns are more serious or complex please contact the **Student Medical Centre** on +44 (0)20 8392 3679. If you are not a student at Roehampton University, please contact your nearest **General Practitioner**.

If you need this information sheet in large printing, please request it from the principal investigator.

A.2 Participant Consent Form

Roehampton University London

ETHICS BOARD

PARTICIPANT CONSENT FORM

"Hip-Spine Interactions in Individuals with Low Back Pain: The Role of the Hamstrings"

Brief Description of Research Project:

The purpose of this study is to investigate how the hamstrings, hips, knees and spine all interact when walking, and to compare those interactions between individuals with and without low-back pain. The study will involve the use of a force transducer to measure muscle stiffness, Vicon motion capture system, EMG electrodes to record muscle activity, and force platforms.

Investigator Contact Details:

Mark Hines
Department of Life Sciences
Whitelands College
Roehampton University, London
Hinesm@roehampton.ac.uk
07788 561898

Consent Statement:

I agree to take part in this research, have read and understood the appropriate participant information sheet, and am aware I am free to withdraw at any point. I understand that the information I provide will be treated in confidence by the investigator and that my identity will be protected in the publication of any findings.

Name .....

Signature .....

Date .....

Please note: if you have a concern about any aspect of your participation or any other queries please raise this with the investigator. However if you would like to contact an independent party please contact Dr. Siobhan Strike.

Director of Studies Contact Details:
Professor Raymond Lee
Department of Life Sciences
Whitelands College
Roehampton University
London
r.lee@roehampton.ac.uk
0208 392 3000

Independent Contact:
Doctor Siobhan Strike
Department of Life Sciences
Whitelands College
Roehampton University
London
s.strike@roehampton.ac.uk
+44 (0)20 8392 3546

A.3 Medical Screening Form

Name:_____ Date of Birth:_____

Age: _____

Please answer all sections and questions below:

Section One: Physical Activity

(Please circle answer)

Do you currently participate in any structured physical activity? Yes

No

If so, please include details below:

_____

Approximately how many hours a week are you physically active? (please tick appropriate box)

Less than one hour ☐ One to two hours ☐ More than two hours ☐

Section Two: Medical Conditions

Do you currently, or have you ever, suffered from: (Please circle answer)

Rheumatoid arthritis or other inflammatory joint disorder? Yes No

If yes, please give details: _____

Fracture or dislocation Yes No

If yes, please give details: _____

Any neurologic disorder Yes No

Any orthopaedic disorder Yes No

Any autonomic disorder Yes No

Have you ever had surgery? Yes No

If yes, please give details: _____

Do you have any bone abnormalities? Yes No

If yes, please give details: _____

Do you have any medical implants? Yes No

If yes, please give details: _____

Section Three: Back Pain

Do you currently suffer from back pain? Yes No

If yes, over what period has the pain persisted? _____

Does the pain radiate to other areas of the body? Yes No

If yes, please give details: _____

Have you been prescribed medication or recommended for treatment?

Yes No

If yes, please give details: _____

Have you ever suffered from back pain in the past? Yes No

If yes:

How many times has this occurred?

_____

Approximately how long did each bout last?

_____

Specifically, where on your back did/does the pain occur?

_____

Did you experience pain down one or both legs?

_____

Have you ever required hospital treatment for back pain? Yes No

Is there any other information, about any aspect of your health, lifestyle or physical condition, which we have not asked about above? If there is, please write out the details in the box below:

Please sign to state that all the above information is correct.

Full Name: _____

Signature: _____ Date: _____

A.4 Roland-Morris Disability Questionnaire

When your back hurts, you may find it difficult to do some of the things you normally do.

This list contains sentences that people have used to describe themselves when they have back pain. When you read them, you may find that some stand out because they describe you *today*.

As you read the list, think of yourself *today*. When you read a sentence that describes you today, put a tick against it. If the sentence does not describe you, then leave the space blank and go on to the next one. Remember, only tick the sentence if you are sure it describes you today.

1. I stay at home most of the time because of my back
2. I change position frequently to try and get my back comfortable
3. I walk more slowly than usual because of my back
4. Because of my back I am not doing any of the jobs that I usually do around the house
5. Because of my back, I use a handrail to get upstairs
6. Because of my back, I lie down to rest more often
7. Because of my back, I have to hold on to something to get out of an easy chair
8. Because of my back, I try to get other people to do things for me
9. I get dressed more slowly than usual because of my back
10. I only stand for short periods of time because of my back

11. Because of my back, I try not to bend or kneel down
12. I find it difficult to get out of a chair because of my back
13. My back is painful almost all the time
14. I find it difficult to turn over in bed because of my back.
15. My appetite is not very good because of my back pain
16. I have trouble putting on my socks (or stockings) because of the pain in my back
17. I only walk short distances because of my back
18. I sleep less well because of my back
19. Because of my back pain, I get dressed with help from someone else
20. I sit down for most of the day because of my back
21. I avoid heavy jobs around the house because of my back
22. Because of my back pain, I am more irritable and bad tempered with people than usual
23. Because of my back, I go upstairs more slowly than usual
24. I stay in bed most of the time because of my back

Note to users:

This questionnaire is taken from: Roland MO, Morris RW. A study of the natural history of back pain. Part 1: Development of a reliable and sensitive measure of disability in low back pain. Spine 1983; 8: 141-144.

The score of the RDQ is the total number of items checked – i.e. from a minimum of 0 to a maximum of 24. It is acceptable to add boxes to indicate where patients should tick each item. The questionnaire may be adapted for use on-line or by telephone.

Roehampton University London

Please Help us to Understand Low Back Pain!

We are looking for volunteers to help us understand the biomechanics of low back pain.

Our study will compare people with low back pain to those without. We want to find if there are differences in the way people with pain walk, and the way in which their muscles function to control movement. We also want to find if we can develop better ways of assessing and treating low back pain. Your assistance could help us learn how to recognise the causes and effects of back pain, and how best to treat people who suffer.

Initially, we are seeking sedentary male participants between the ages of 18 and 50, with or without back pain, to attend the biomechanics laboratory at Whitelands College for a single visit. We want to recruit people with current back pain, or those who are back-pain free and have not experienced back pain in the last twelve months. Unfortunately, we cannot include people with neurologic or orthopaedic conditions, a history of fractures of the spine, hips or legs, or anyone with restricted hip mobility.

The assessment will involve a simple straight-leg raise test followed by gait analysis. We will be incorporating ultrasound, Vicon Motion Capture and EMG, so as to really understand what affects joints and muscle function. The whole assessment should last no more than an hour. If you might be interested in helping us with this important research, then we would be very keen to hear from you.

For more information please contact Mark Hines: hinesm@roehampton.ac.uk, Department of Life Sciences, Whitelands College, Roehampton, Holybourne Avenue, SW15 4JD

Appendix B

Engineering Specifications and Data Sheets

B.1 Extension Piece 1: Transducer Handle Mount

Material: Aluminium

All measurements are in inches. Images are not to scale.

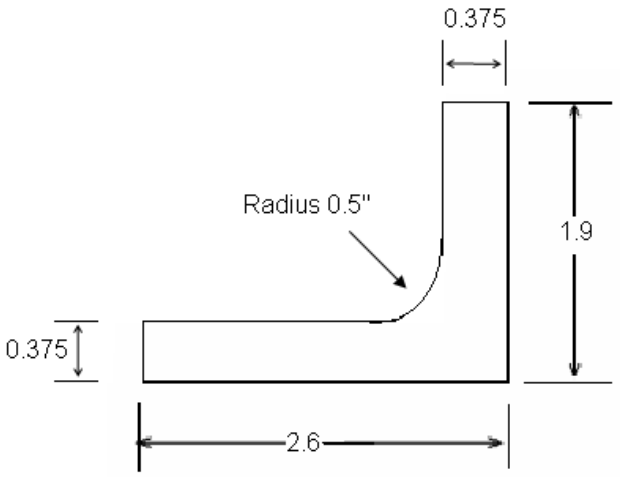


Figure B.1.1 Side view

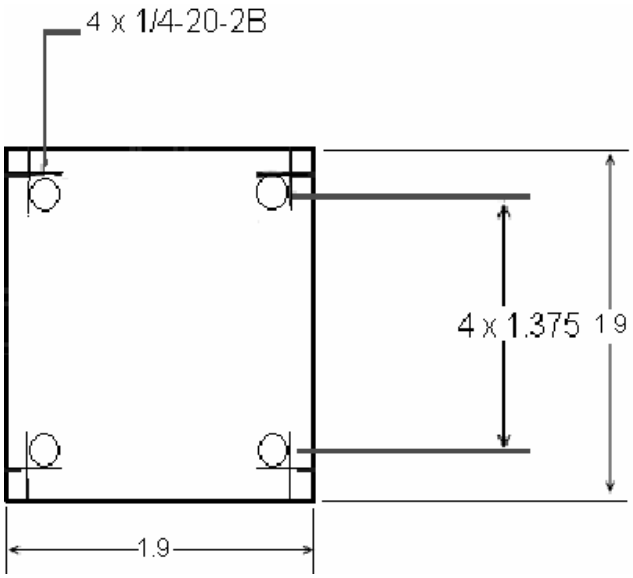


Figure B.1.2 Rear / Front view

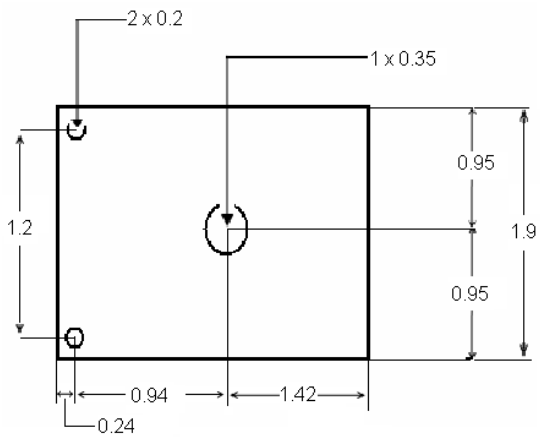


Figure B.1.3 Base View

B.2 Extension Piece 2: Transducer Cylindrical Measuring Surface

Material: Aluminium

All measurements are in inches. Images are not to scale.

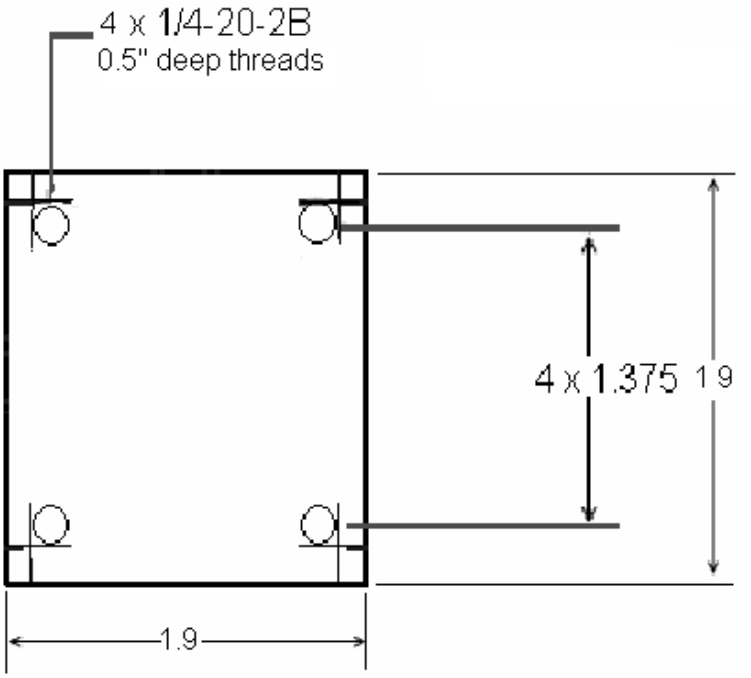


Figure B.2.1 Rear / Front view

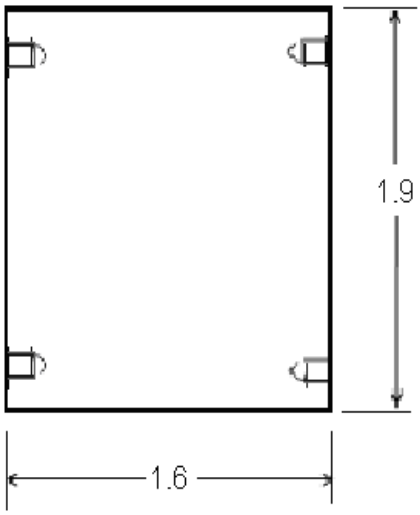


Figure B.2.2 Side View

B.3 Polystyrene Ankle Brace Pad (Lower Leg)

Polystyrene pad to have aluminium rods inserted

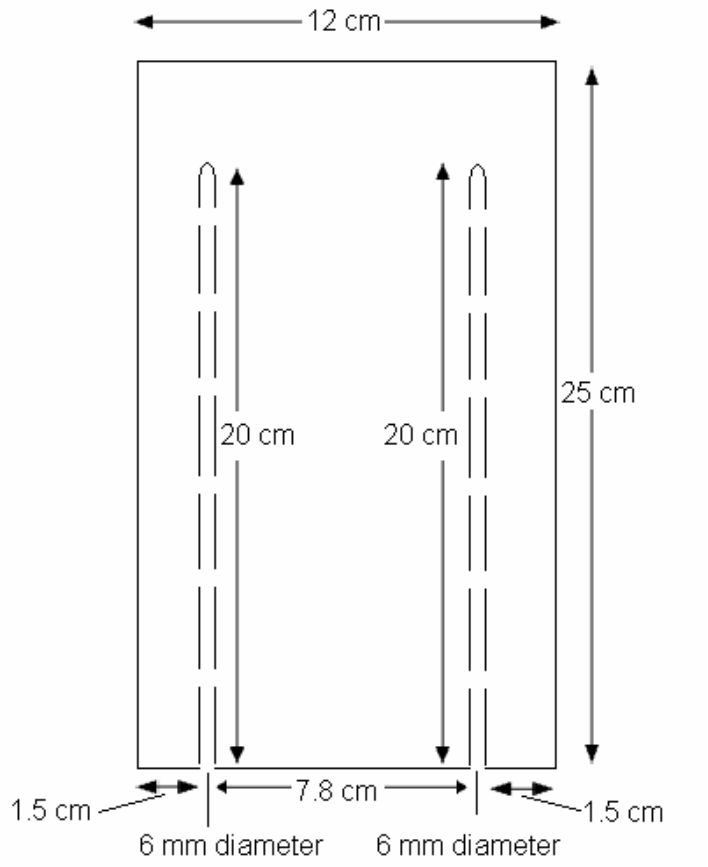


Figure B.3.1 Face view

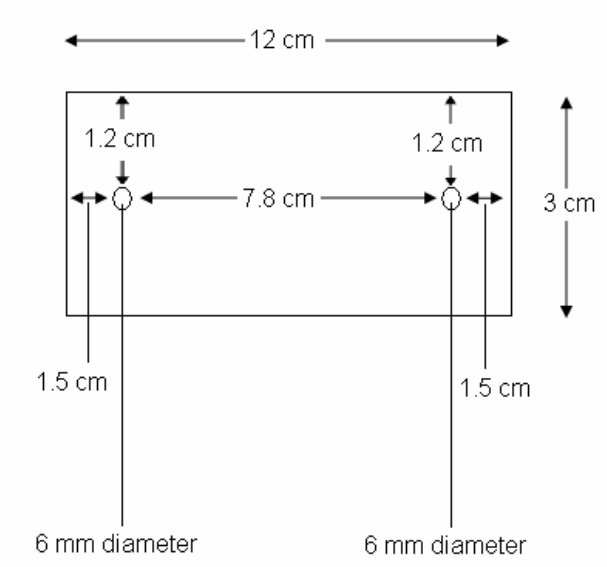
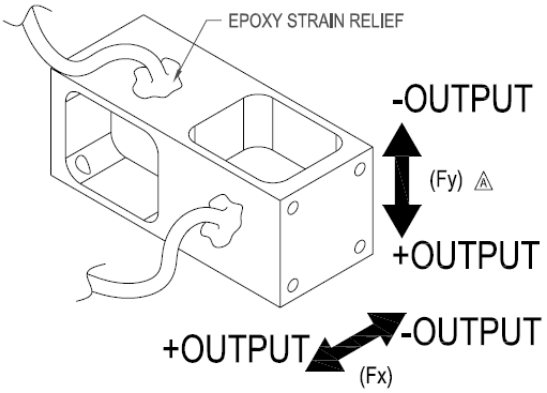


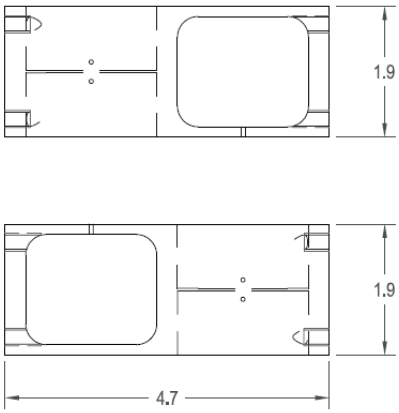
Figure B.3.2 Front view

B.4 Futek QLA263 Datasheet

FUTEK MODEL QLA263 *SUBMERSIBLE DUAL AXIS LOAD CELL*

INCH (mm)	R.O.= Rated Output		
WIRING CODE (WC1)			
+Excitation	-Excitation	+Signal	-Signal
RED	BLACK	GREEN	WHITE
Shield			
Floating			





SPECIFICATIONS:

CAPACITY	*See Chart*
EXCITATION (VDC OR VAC)	*18 MAX*
BRIDGE RESISTANCE	*350 Ω nom.*
OPERATING TEMP.	*0 to 160°F*
MATERIAL	*ALUMINUM*

CABLE: 2x #29 AWG, 4 Conductor, Spiral Shielded, Black Silicone Cable, 30 ft. Long.

	CAPACITIES	
	STOCK NO:	lb
	QSH00752	250
B	QSH00802	100

NOTE: FLEXURE SUPPLIED BY OFFSHORE MODEL BASIN

MATERIAL: —

HEAT TREATMENT - FINISH: —

REVISIONS: (Refer to dwg # revision sheet)
A 5/3/06
B 4-5-07

OUTLINE DRAWING

STANDARD NOTES: (Unless Otherwise Specified)
- ALL DIMENSIONS ARE IN INCHES
- DRAWING INTERPRETATION DIMS. PER ASME-Y14.5M
- REMOVE BURRS AND BREAK SHARP EDGES .005 - .015
- THREADS PER HANDBOOK H-28
- DIMENSIONS ARE SHOWN AFTER PLATING

3rd ANGLE PROJ.

ANGLE: ±1/2°
CHAMFER: ±5°
TOLERANCE: .X ±0.1 .XX ±0.01 .XXX ±0.005

CAGE: 1X8M6

FUTEK ADVANCED SENSOR TECHNOLOGY, INC.
10 THOMAS
IRVINE, CA 92618 USA
Phone: (949) 465-0900
Fax: (949) 465-0905

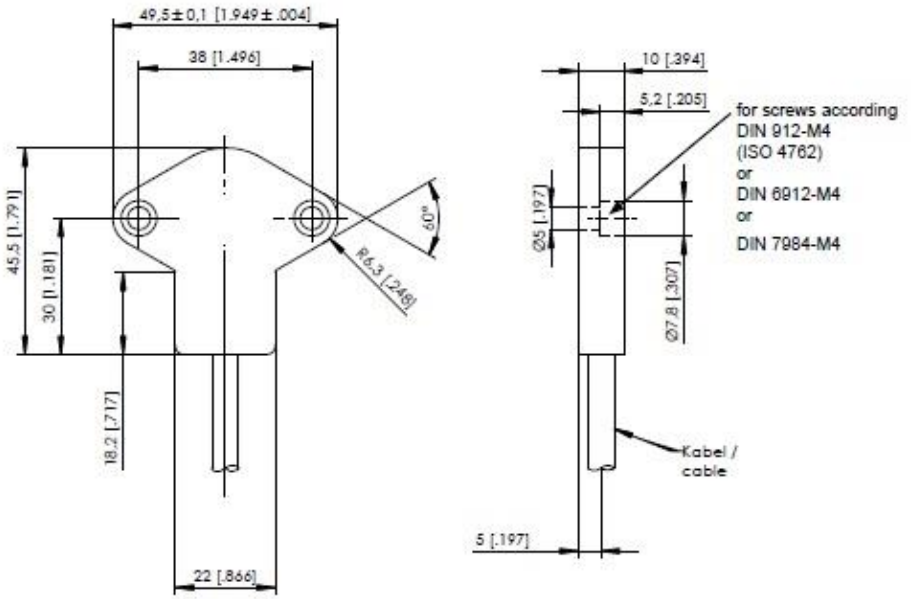
MODEL: QLA263 DWG No.: FO1131
DRAWN BY: H. AU CHECKED BY: -
APPROVED BY: R. WALKER DATE: APR. 25, 2006

B.5 ASM PTAM27 Inclinometer

POSITILT®
Outline drawings

ASM®

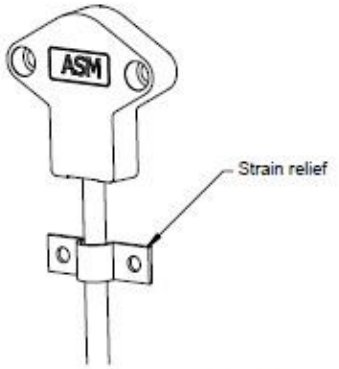
Outline drawing
PTAM27



Dimensions in mm [inch]

Dimensions informative only.
For guaranteed dimensions consult factory.

Reference position



U6 Voltage Output 0.5 ... 4.5 V DC	Excitation voltage	5V DC ±5 %
	Excitation current	13 mA typ., 16 mA max.
	Output voltage	0.5 ... 4.5 V DC
	Output current	2 mA max.
	Measuring rate	1 kHz standard
	Stability (temperature)	±100 x 10^{-6} / °C f.s. (typ.)
	Operating temperature	-40 ... +85 °C
	Protection	Reverse polarity, short circuit
	EMC	EN 61326-1:2013

Appendix C

Equipment Validation

C.1 Load Cells

Table C.1.1 Example of initial load cell testing

Test	True Weight (Kg)	Expected Output (mV)	Newtons	Measured Output (mV)	Difference
1	0.5	49	4.9	0.499490316	-0.0005097
2	0.5	49	4.9	0.499490316	-0.0005097
3	0.5	49	4.9	0.499490316	-0.0005097
Mean	0.5	49	4.9	0.499490316	-0.0005097
1	1.25	120	12	1.22324159	-0.0267584
2	1.25	119	11.9	1.21304791	-0.0369521
3	1.25	120	12	1.22324159	-0.0267584
Mean	1.25	119.6666667	11.96667	1.219843697	-0.0301563
1	2.5	240	24	2.44648318	-0.0535168
2	2.5	240	24	2.44648318	-0.0535168
3	2.5	241	24.1	2.45667686	-0.0433231
Mean	2.5	240.3333333	24.03333	2.449881074	-0.0501189
1	3.75	364	36.4	3.71049949	-0.0395005
2	3.75	364	36.4	3.71049949	-0.0395005
3	3.75	364	36.4	3.71049949	-0.0395005
Mean	3.75	364	36.4	3.71049949	-0.0395005
1	5	493	49.3	5.0254842	0.0254842
2	5	494	49.4	5.03567788	0.03567788
3	5	494	49.4	5.03567788	0.03567788
Mean	5	493.6666667	49.36667	5.032279986	0.03227999
1	5.5	544	54.4	5.545361876	0.04536188
2	5.5	545	54.5	5.555555556	0.05555556
3	5.5	546	54.6	5.565749235	0.06574924
Mean	5.5	545	54.5	5.555555556	0.05555556
1	10	990	99	10.09174312	0.09174312
2	10	990	99	10.09174312	0.09174312
3	10	989	98.9	10.08154944	0.08154944
Mean	10	989.6666667	98.96667	10.08834523	0.08834523

Table C.1.2 Load Cell Data with Mean and Confidence Interval Calculations

				Calculations for Mean and 95% Confidence Intervals					
True Weight	**Test1**	**Test2**	**Test3**	**Mean**	**SD**	**n**	**Confidence**	**CIL**	**CIH**
0.5	0.49949	0.49949	0.49949	0.49949	0	2	#NUM!	#NUM!	#NUM!
1.25	1.223242	1.213048	1.223242	1.219844	0.005885	3	0.006659749	1.213183948	1.226503
2.5	2.446483	2.446483	2.456677	2.449881	0.005885	4	0.005767511	2.444113562	2.455649
3.75	3.710499	3.710499	3.710499	3.710499	0	6	#NUM!	#NUM!	#NUM!
5	5.025484	5.035678	5.035678	5.03228	0.005885	3	0.006659749	5.025620238	5.03894
5.5	5.545362	5.555556	5.565749	5.555556	0.010194	3	0.011535023	5.544020533	5.567091
10	10.09174	10.09174	10.08155	10.08835	0.005885	3	0.006659749	10.08168548	10.095

Table C.1.3 Load Cell Tests at Different Tilt Angles

	0 Degrees				1 Degree				2 Degrees				3 Degrees				4 Degrees				5 Degrees			
Actual	0.5	0.5	0.5	Error (g)	0.5	0.5	0.5	Error (g)	0.5	0.5	0.5	Error (g)	0.5	0.5	0.5	Error (g)	0.5	0.5	0.5	Error (g)	0.5	0.5	0.5	Error (g)
mV	49	49	49		49	49	49		49	49	49		49	49	49		49	49	49		48	49	49	
Newtons	4.9	4.9	4.9		4.9	4.9	4.9		4.9	4.9	4.9		4.9	4.9	4.9		4.9	4.9	4.9		4.8	4.9	4.9	
Measured	0.49949	0.49949	0.49949		0.49949	0.49949	0.49949		0.49949	0.49949	0.49949		0.49949	0.49949	0.49949		0.49949	0.49949	0.49949		0.489297	0.49949	0.49949	
Diff	0.00051	0.00051	0.00051	0.509684	0.00051	0.00051	0.00051	0.509684	0.00051	0.00051	0.00051	0.509684	0.00051	0.00051	0.00051	0.509684	0.00051	0.00051	0.00051	0.509684	0.010703	0.00051	0.00051	0.003908
Actual	1.25	1.25	1.25		1.25	1.25	1.25		1.25	1.25	1.25		1.25	1.25	1.25		1.25	1.25	1.25		1.25	1.25	1.25	
mV	120	119	120	119.6667	120	120	119	119.6667	120	120	120		120	120	119	119.6667	120	120	119	119.6667	119	119	120	119.3333
Newtons	12	11.9	12	11.96667	12	12	11.9	11.96667	12	12	12		12	12	11.9	11.96667	12	12	11.9	11.96667	11.9	11.9	12	11.93333
Measured	1.223242	1.213048	1.223242	1.219844	1.223242	1.223242	1.213048	1.219844	1.223242	1.223242	1.223242		1.223242	1.223242	1.213048	1.219844	1.223242	1.223242	1.213048	1.219844	1.213048	1.213048	1.223242	1.216446
Diff	0.026758	0.036952	0.026758	0.030156	36.95209	0.026758	0.036952	0.030156	36.95209	0.026758	0.026758	0.026758	26.75841	0.026758	0.036952	0.030156	36.95209	0.026758	0.036952	0.030156	36.95209	0.036952	0.026758	0.033554
Actual	2.5	2.5	2.5		2.5	2.5	2.5		2.5	2.5	2.5		2.5	2.5	2.5		2.5	2.5	2.5		2.5	2.5	2.5	
mV	240	240	241	240.3333	241	241	241	241	241	241	240	240.6667	241	241	241	240.6667	240	241	240	240.3333	240	241	241	240.6667
Newtons	24	24	24.1	24.03333	24.1	24.1	24.1	24.1	24.1	24.1	24	24.06667	24.1	24.1	24.1	24.06667	24	24.1	24	24.03333	24	24.1	24.1	24.06667
Measured	2.446483	2.446483	2.456677	2.449881	2.456677	2.456677	2.456677	2.456677	-24.0732	-24.0732	-23.9732	-24.0399	26.53991	26.57324	26.57324	26.47324	2.446483	2.446483	2.456677	2.449881	2.446483	2.456677	2.452279	
Diff	0.053517	0.053517	0.043323	0.050119	53.51682	0.043323	0.043323	0.043323	43.32314	26.57324	26.57324	26.47324	26.53991	26573.24	0.053517	0.043323	0.046721	53.51682	0.053517	0.043323	0.050119	53.51682	0.053517	0.043323
Actual	3.75	3.75	3.75		3.75	3.75	3.75		3.75	3.75	3.75		3.75	3.75	3.75		3.75	3.75	3.75		3.75	3.75	3.75	
mV	364	364	364		364	365	364	364.3333	364	365	363		364	363	364	363.6667	364	364	364		363	364	363	363.3333
Newtons	36.4	36.4	36.4		36.4	36.5	36.4	36.43333	36.4	36.5	36.3		36.4	36.3	36.4	36.36667	36.4	36.4	36.4		36.3	36.4	36.3	36.33333
Measured	3.710499	3.710499	3.710499		3.710499	3.720693	3.710499	3.713897	3.710499	3.720693	3.700306	3.710499	3.700306	3.710499	3.710499	3.707102	3.710499	3.710499	3.710499		3.700306	3.710499	3.700306	3.703704
Diff	0.039501	0.039501	0.039501	39.50051	0.039501	0.029307	0.039501	39.50051	0.039501	0.029307	0.049694	0.039501	49.69419	0.049694	0.039501	49.69419	0.039501	0.039501	0.039501	39.50051	0.049694	0.039501	0.046296	49.69419
Actual	5	5	5		5	5	5		5	5	5		5	5	5		5	5	5		5	5	5	
mV	493	494	493.6667		493	493	494	493.3333	493	492	493	492.6667	493	493	492	492.6667	493	492	493	493.3333	492	493	493	492.6667
Newtons	49.3	49.4	49.36667		49.3	49.3	49.4	49.33333	49.3	49.2	49.3	49.26667	49.3	49.3	49.2	49.26667	49.3	49.2	49.3	49.33333	49.2	49.3	49.3	49.26667
Measured	5.025484	5.035678	5.03228		5.025484	5.025484	5.035678	5.028882	5.025484	5.015291	5.025484	5.022086	5.025484	5.025484	5.015291	5.022086	5.015291	5.025484	5.035678	5.025484	5.015291	5.025484	5.025484	5.022086
Diff	0.025484	0.035678	0.03228	35.67788	0.025484	0.025484	0.035678	0.028882	35.67788	0.025484	0.015291	0.025484	0.022086	25.4842	0.025484	0.015291	0.022086	25.4842	0.015291	0.025484	0.035678	35.67788	0.015291	0.025484
Actual	5.5	5.5	5.5		5.5	5.5	5.5		5.5	5.5	5.5		5.5	5.5	5.5		5.5	5.5	5.5		5.5	5.5	5.5	
mV	544	545	546	545	544	544	545	544.3333	543	542	544	543	544	542	543	543	544	543	542	543	544	542	543	543
Newtons	54.4	54.5	54.6	54.5	54.4	54.4	54.5	54.43333	54.3	54.2	54.4	54.3	54.4	54.2	54.3	54.3	54.4	54.3	54.2	54.3	54.4	54.2	54.3	54.3
Measured	5.545362	5.555556	5.565749	5.555556	5.545362	5.545362	5.555556	5.54876	5.535168	5.524975	5.545362	5.535168	5.545362	5.524975	5.535168	5.535168	5.545362	5.535168	5.524975	5.535168	5.545362	5.524975	5.535168	5.535168
Diff	0.045362	0.055556	0.065749	0.055556	65.74924	0.045362	0.045362	0.055556	0.04876	55.55556	0.035168	0.024975	0.045362	0.024975	0.035168	0.035168	45.36188	0.045362	0.035168	0.024975	0.035168	45.36188	0.045362	0.024975
Actual	10	10	10		10	10	10		10	10	10		10	10	10		10	10	10		10	10	10	
mV	990	990	989	989.6667	985	987	987	986.3333	987	988	987	987.3333	985	986	988	986.3333	985	986	987	986	985	985	985	985
Newtons	99	99	98.9	98.96667	98.5	98.7	98.7	98.63333	98.7	98.8	98.7	98.73333	98.5	98.6	98.8	98.63333	98.5	98.6	98.7	98.6	98.5	98.5	98.5	98.5
Measured	10.09174	10.09174	10.08355	10.08835	10.04077	10.06116	10.06116	10.05437	10.06116	10.07136	10.06116	10.06456	10.04077	10.05097	10.07136	10.05437	10.04077	10.05097	10.06116	10.05097	10.04077	10.04077	10.04077	10.04077
Diff	0.091743	0.091743	0.081549	0.088345	91.74312	0.040775	0.061162	0.061162	0.054866	61.16208	0.061162	0.071356	0.061162	0.06456	71.35576	0.040775	0.050968	0.061162	0.050968	61.16208	0.040775	0.040775	0.040775	0.040775

C.2 Inclinometers

Table C.2.1. Calculation of inclinometer degrees measurement and error from output

6 Degrees				7 Degrees				8 Degrees				9 Degrees				10 Degrees				Low	High	Mean	SD	Low (g)	High (g)	Mean (g)	SD (g)
0.5	0.5	0.5	Mean	Error (g)	0.5	0.5	0.5	Mean	Error (g)	0.5	0.5	0.5	Mean	Error (g)	0.5	0.5	0.5	Mean	Error (g)								
4.8	4.8	4.9	4.833333		4.9	4.9	4.9	4.833333		4.8	4.9	4.8	4.833333		4.8	4.8	4.8	4.833333			4.8	4.9	4.869697	0.466694		
4.8	4.8	4.9	4.833333		4.9	4.9	4.9	4.833333		4.8	4.9	4.8	4.833333		4.8	4.8	4.8	4.833333			4.8	4.9	4.869697	0.466694		
0.489297	0.489297	0.49949	0.492695		0.49949	0.49949	0.49949	0.492695		0.489297	0.49949	0.489297	0.492695		0.489297	0.489297	0.489297	0.492695			0.489297	0.49949	0.496401	0.004757		
0.010703	0.010703	0.00051	0.007305	10.70336	0.00051	0.00051	0.00051	0.007305	10.70336	0.010703	0.00051	0.010703	0.007305	10.70336	0.010703	0.010703	0.010703	0.007305	10.70336	0.00051	0.010703	0.003599	0.004757	0.508684	10.70336	3.598678	4.757326
1.25	1.25	1.25	1.25		1.25	1.25	1.25	1.25		1.25	1.25	1.25	1.25		1.25	1.25	1.25	1.25			1.25	1.25	1.25	0		
119	119	120	119.3333		119	119	119	119		118	119	118	118.3333		118	118	118	118			118	120	119.2121	0.73938		
11.9	11.9	12	11.93333		11.9	11.9	11.9	11.9		11.8	11.9	11.8	11.83333		11.8	11.8	11.8	11.8			11.8	12	11.92121	0.073938		
1.213048	1.213048	1.223242	1.216446		1.213048	1.213048	1.213048	1.213048		1.202854	1.213048	1.213048	1.209655		1.202854	1.213048	1.202854	1.209252			1.202854	1.223242	1.21521	0.007542		
0.036952	0.036952	0.026759	0.033554	36.95209	0.036952	0.036952	0.036952	0.033652	36.95209	0.047146	0.036952	0.036952	0.040335	47.14577	0.047146	0.047146	0.047146	0.047146	47.14577	0.026759	0.047146	0.03479	0.007542	26.75841	47.14577	34.78979	7.541591
2.5	2.5	2.5	2.5		2.5	2.5	2.5	2.5		2.5	2.5	2.5	2.5		2.5	2.5	2.5	2.5			2.5	2.5	2.5	0				
240	240	240	240		239	239	240	239.3333		239	239	238	238.6667		238	237	237	237.3333			237	241	239.697	1.310165				
24	24	24	24		23.9	23.9	24	23.93333		23.9	23.9	23.8	23.86667		23.8	23.7	23.8	23.73333			23.7	24.1	23.9697	0.131065				
2.446483	2.446483	2.446483	2.446483		2.43629	2.45629	2.439687			2.43629	2.45629	2.426096	2.432892		2.426096	2.415902	2.426096	2.42698			-24.0732	2.456677	0.034923	7.731194				
0.053517	0.053517	0.053517	0.053517	53.51682	0.06371	0.06371	0.063313	63.7165	0.06371	0.06371	0.063371	0.073904	0.067108	73.90418	0.073904	0.084098	0.073904	0.07302	84.09786	0.084098	0.084098	0.073904	0.10807	84.09786	0.043232	26.57324	2.465077	7.731194	43.33214	26.57324	2.465077	7.731194
3.75	3.75	3.75	3.75		3.75	3.75	3.75	3.75		3.75	3.75	3.75	3.75		3.75	3.75	3.75	3.75			3.75	3.75	3.75	0				
363	363	364	363.3333		362	363	362	362.3333		362	362	361	361.6667		361	361	361	361			359	365	362.8485	1.523179				
36.3	36.3	36.4	36.33333		36.2	36.3	36.2	36.23333		36.2	36.2	36.1	36.16667		36.1	36.1	36.1	36.1			35.9	36.5	36.28485	0.152318				
3.700306	3.700306	3.710469	3.703704		3.690112	3.700306	3.690112	3.69351		3.690112	3.690112	3.679918	3.686714		3.679918	3.679918	3.679918	3.679918			3.659331	3.726939	3.698761	0.015527				
0.049694	0.049694	0.039501	0.046296	49.69419	0.059888	0.049694	0.059888	0.05649	59.88787	0.059888	0.059888	0.070082	0.062896	70.08155	0.070082	0.070082	0.070082	0.070082	70.08155	0.080275	0.090469	0.080275	0.103673	90.48891	0.029307	0.090469	0.051239	0.015527	29.30683	90.48891	51.23869	15.5188
5	5	5	5		5	5	5	5		5	5	5	5		5	5	5	5			5	5	5	0				
492	491	493	492		490	492	492	491.3333		488	490	490	489.3333		487	488	488	487.3333			486	494	491.3636	2.408555				
49.2	49.1	49.3	49.2		49	49.2	49.2	49.13333		48.8	49	49	48.93333		48.7	48.8	48.8	48.73333			48.6	49.4	49.13636	0.240855				
5.015291	5.015291	5.025484	5.015291		4.994903	5.015291	5.015291	5.008495		4.974516	4.994903	4.994903	4.988107		4.964322	4.974516	4.954128	4.964322			4.954128	5.035678	5.008804	0.024652				
0.015291	0.015291	0.025484	0.015291	25.4842	-0.0051	0.015291	0.008495	15.29052	-0.02548	-0.0051	-0.0051	-0.01189	-5.09684	-0.03568	-0.02548	-0.04587	-0.03568	-0.02548	-25.4842	-0.03568	-0.02548	-0.03568	-0.03828	-25.4842	-0.04587	0.035678	0.008804	-0.02452	-45.8716	35.67788	8.803633	24.55204
5.5	5.5	5.5	5.5		5.5	5.5	5.5	5.5		5.5	5.5	5.5	5.5		5.5	5.5	5.5	5.5			5.5	5.5	5.5	0				
541	542	542	541.6667		540	540	541	540.3333		540	540	539	539.6667		537	538	537	537.3333			535	546	542.4848	2.851767				
54.1	54.2	54.2	54.16667		54	54	54.1	54.03333		54	54	53.9	53.96667		53.7	53.8	53.7	53.73333			53.5	54.6	54.14848	0.285177				
5.514781	5.524975	5.524975	5.521577		5.504587	5.504587	5.514781	5.507895		5.504587	5.504587	5.494339	5.501189		5.474006	5.464382	5.474006	5.447404			5.453819	5.565749	5.519723	0.02907				
0.014781	0.024975	0.024975	0.021577	24.97452	0.004587	0.004587	0.014781	0.007985	14.78784	0.004587	0.004587	-0.00356	0.001189	4.587156	-0.02399	-0.01038	-0.02399	-0.02026	-15.8802	-0.04638	-0.01038	-0.02399	-0.01819	-25.9939	-0.04638	0.035678	0.019723	0.02907	-46.3812	65.74924	19.72323	29.07
10	10	10	10		10	10	10	10		10	10	10	10		10	10	10	10			10	10	10	0				
985	986	985	985.3333		985	984	983	984		980	982	981	981		980	980	981	980.3333			978	990	984.5455	3.275586				
98.5	98.6	98.5	98.53333		98.5	98.4	98.3	98.4		98	98.2	98.1	98.1		98	98	98.1	98.03333			97.8	99	98.45455	0.327959				
10.04077	10.03097	10.04077	10.04417		10.04077	10.03658	10.02039	10.03658		9.989806	10.01019	10	10		9.989806	9.989806	9.969419	9.993204			9.969419	10.09174	10.03614	0.033431				
0.040775	0.050968	0.040775	0.044173	50.9684	0.040775	0.036581	0.020387	0.036581	40.77472	-0.01019	0.010194	-1.8E-15	-1.2E-15	10.19368	-0.01019	-0.01019	-0.01019	-0.01819	-10.1937	-0.03658	0.091743	0.036141	0.033431	-30.581	91.74312	36.14123	33.43105

Table C.2.2. Calculation of inclinometer degrees measurement based upon 1 degree equivalent to 14.65 mV

4950 mV input, 4000 mV full output range, 3 V sensitivity

Angle	Test 1	Test 2	Test 3	Mean	SD	mV/deg	14.65*deg	Minimum mV	Maximum mV	M-Min mV	Max-M mV
0	0	0	0	0	0	**0**					
10	150	141	145	145.3333333	4.50924975	**14.533333**	146.5	141	150	5.5	3.5
20	293	290	287	290	3	**14.5**	293	287	293	6	0
30	441	440	438	439.6666667	1.52752523	**14.655556**	439.5	438	441	1.5	1.5
40	589	586	588	587.6666667	1.52752523	**14.691667**	586	586	589	0	3
50	737	736	735	736	1	**14.72**	732.5	735	737	-2.5	4.5
60	880	884	880	881.3333333	2.30940108	**14.688889**	879	880	884	-1	5
70	1030	1032	1034	1032	2	**14.742857**	1025.5	1030	1034	-4.5	8.5
80	1171	1177	1173	1173.666667	3.05505046	**14.670833**	1172	1171	1177	1	5
90	1310	1309	1308	1309	1	**14.544444**	1318.5	1308	1310	10.5	-8.5
100	1460	1460	1455	1458.333333	2.88675135	**14.583333**	1465	1455	1460	10	-5
110	1614	1612	1606	1610.666667	4.163332	**14.642424**	1611.5	1606	1614	5.5	2.5
120	1758	1761	1760	1759.666667	1.52752523	**14.663889**	1758	1758	1761	0	3
130	1905	1905	1907	1905.666667	1.15470054	**14.658974**	1904.5	1905	1907	-0.5	2.5
140	2052	2058	2053	2054.333333	3.21455025	**14.67381**	2051	2052	2058	-1	7
150	2205	2201	2202	2202.666667	2.081666	**14.684444**	2197.5	2201	2205	-3.5	7.5
160	2349	2350	2346	2348.333333	2.081666	**14.677083**	2344	2346	2350	-2	6
170	2499	2500	2495	2498	2.64575131	**14.694118**	2490.5	2495	2500	-4.5	9.5
180	2646	2643	2642	2643.666667	2.081666	**14.687037**	2637	2642	2646	-5	9
				Mean	**SD**	**0.0614785**	739.7887367	741.5595103	741.7571249	4.770466742	4.783550244
				14.650705			1391.75	1390.888889	1395.333333	0.861111111	3.583333333

Table C.2.3 Retest check of output data with one degree equivalent to 14.65 mV

One degree equivalent to 14.65 mV. Maximum error is 10.5 mV, equivalent to 0.71 degrees.

Re-Test Check:

Angle	Test 1	14.65*deg	Difference mV	Deg Equivalent
10	146	146.5	0.5	0.034129693
20	293	293	0	0
30	441	439.5	-1.5	-0.102389078
40	595	586	-9	-0.614334471
50	742	732.5	-9.5	-0.648464164
60	887	879	-8	-0.546075085
70	1030	1025.5	-4.5	-0.307167235
80	1179	1172	-7	-0.4778157
90	1324	1318.5	-5.5	-0.375426621
100	1470	1465	-5	-0.341296928
110	1617	1611.5	-5.5	-0.375426621
120	1764	1758	-6	-0.409556314
130	1911	1904.5	-6.5	-0.443686007
140	2057	2051	-6	-0.409556314
150	2210	2197.5	-12.5	-0.853242321
160	2359	2344	-15	-1.023890785
170	2512	2490.5	-21.5	-1.467576792
180	2652	2637	-15	-1.023890785
		Max Value:	0.5	0.034129693
		Min Value:	-21.5	-1.467576792

Single test shows accurate to within 1.5 degrees at 180 degree rotation

Accurate to within 0.5 degrees to 140 degrees rotation, within 1 degree at 150, and between 1 and 1.5 from 160-180

Table C.2.4. Comparison of alternative calibration values for reference

Angle	Test 1	Test 2	Test 3	Mean	SD	mV/deg	14.651322*deg	Minimum	Maximum	M-Min	Max-M	14.6*deg	Minimum	Maximum	M-Min	Max-M	14.7*deg	Minimum	Maximum	M-Min	Max-M
10	150	141	145	145.333333	4.50924975	14.533333	146.513224	141	150	5.513224	3.486776	146	141	150	5	4	66.285971	141	150	-74.714	83.71403
20	293	290	287	290	3	14.5	293.026448	287	293	6.026448	-0.02645	292	287	293	5	1	44.1	287	293	-242.9	248.9
30	441	440	438	439.666667	1.52752523	14.655556	439.539672	438	441	1.539672	1.460328	438	438	441	0	3	22.454621	438	441	-415.545	418.5454
40	589	586	588	587.666667	1.52752523	14.691667	586.052896	586	589	0.052896	2.947104	584	586	589	-2	5	22.454621	586	589	-563.545	566.5454
50	737	736	735	736	1	14.72	732.56612	735	737	-2.43388	4.43388	730	735	737	-5	7	14.7	735	737	-720.3	722.3
60	880	884	880	881.333333	2.30940108	14.688889	879.079344	880	884	-0.92066	4.920656	876	880	884	-4	8	33.948196	880	884	-846.052	850.0518
70	1030	1032	1034	1032	2	14.742857	1025.592568	1030	1034	-4.40743	8.407432	1022	1030	1034	-8	12	29.4	1030	1034	-1000.6	1004.6
80	1171	1177	1173	1173.66667	3.05505046	14.670833	1172.105792	1171	1177	1.105792	4.894208	1168	1171	1177	-3	9	44.909242	1171	1177	-1126.09	1132.091
90	1300	1322	1308	1310	11.1355287	14.555556	1318.619016	1300	1322	18.61902	3.380984	1314	1300	1322	14	8	163.69227	1300	1322	-1136.31	1158.308
100	1460	1460	1455	1458.33333	2.88675135	14.583333	1465.13224	1455	1460	10.13224	-5.13224	1460	1455	1460	5	0	42.435245	1455	1460	-1412.56	1417.565
110	1614	1612	1606	1610.66667	4.163332	14.642424	1611.645464	1606	1614	5.645464	2.354536	1606	1606	1614	0	8	61.20098	1606	1614	-1544.8	1552.799
120	1758	1761	1760	1759.66667	1.52752523	14.663889	1758.158688	1758	1761	0.158688	2.841312	1752	1758	1761	-6	9	22.454621	1758	1761	-1735.55	1738.545
130	1905	1905	1907	1905.66667	1.15470054	14.658974	1904.671912	1905	1907	-0.32809	2.328088	1898	1905	1907	-7	9	16.974098	1905	1907	-1888.03	1890.026
140	2052	2058	2053	2054.33333	3.21455025	14.67381	2051.185136	2052	2058	-0.81486	6.814864	2044	2052	2058	-8	14	47.253889	2052	2058	-2004.75	2010.746
150	2205	2201	2202	2202.66667	2.08166	14.684444	2197.69836	2201	2205	-3.30164	7.30164	2190	2201	2205	-11	15	30.60049	2201	2205	-2170.4	2174.4
160	2349	2350	2346	2348.33333	2.08166	14.677083	2344.211584	2346	2350	-1.78842	5.788416	2336	2346	2350	-10	14	30.60049	2346	2350	-2315.4	2319.4
170	2499	2500	2495	2498	2.64575131	14.694118	2490.724808	2495	2500	-4.27519	9.275192	2482	2495	2500	-13	18	38.892544	2495	2500	-2456.11	2461.107
180	2646	2643	2642	2643.66667	2.08166	14.687037	2637.238032	2642	2646	-4.76197	8.761968	2628	2642	2646	-14	18	30.60049	2642	2646	-2611.4	2615.4
				Mean	SD	14.651322	1391.875628	1390.444444	1396	1.431184	4.124372	1387	1390.44444	1396	-3.444444	9	42.386543	1390.44444	1396	-1348.06	1353.613
					SD	0.06024469	739.8555147	741.6675022	741.602468	6.01584	3.603291	737.2638605	741.667502	741.602468	7.223837	5.359159	33.85764	741.667502	741.602468	742.6129	742.3398

Table C.2.5. Comparison of clockwise and anti-clockwise inclinometer rotations

Angle	Test 1	14.65*deg	Difference mV	Deg Equivalent
10	142	146.5	4.5	0.307167235
20	295	293	-2	-0.136518771
30	435	439.5	4.5	0.307167235
40	584	586	2	0.136518771
50	734	732.5	-1.5	-0.102389078
60	883	879	-4	-0.273037543
70	1030	1025.5	-4.5	-0.307167235
80	1170	1172	2	0.136518771
90	1315	1318.5	3.5	0.23890785
100	1462	1465	3	0.204778157
110	1606	1611.5	5.5	0.375426621
120	1744	1758	14	0.955631399
130	1895	1904.5	9.5	0.648464164
140	2046	2051	5	0.341296928
150	2187	2197.5	10.5	0.716723549
160	2333	2344	11	0.750853242
170	2491	2490.5	-0.5	-0.034129693
180	2641	2637	-4	-0.273037543
		Max Value:	14	0.955631399
		Min Value:	-4.5	-0.307167235

Angle	Test 1	14.65*deg	Difference mV	Deg Equivalent
10	145	146.5	1.5	0.102389078
20	290	293	3	0.204778157
30	448	439.5	-8.5	-0.580204778
40	601	586	-15	-1.023890785
50	791	732.5	-58.5	-3.993174061
		Max Value:	3	0.204778157
		Min Value:	-58.5	-3.993174061

Accurate anti-clockwise to within 1 degree up to 40 degrees

Accurate clockwise to within 1 degree throughout range

Table C.2.6. Raw output data with best degree conversion calculations

Angle	Test 1	Test 2	Test 3	Mean	SD	mV/deg	14.651322*deg	Minimum	Maximum	M-Min	Max-M
10	150	141	145	145.333333	4.50924975	**14.533333**	146.513224	141	150	**5.513224**	**3.486776**
20	293	290	287	290	3	**14.5**	293.026448	287	293	**6.026448**	**-0.02645**
30	441	440	438	439.666667	1.52752523	**14.655556**	439.539672	438	441	**1.539672**	**1.460328**
40	589	586	588	587.666667	1.52752523	**14.691667**	586.052896	586	589	**0.052896**	**2.947104**
50	737	736	735	736	1	**14.72**	732.56612	735	737	**-2.43388**	**4.43388**
60	880	884	880	881.333333	2.30940108	**14.688889**	879.079344	880	884	**-0.92066**	**4.920656**
70	1030	1032	1034	1032	2	**14.742857**	1025.592568	1030	1034	**-4.40743**	**8.407432**
80	1171	1177	1173	1173.66667	3.05505046	**14.670833**	1172.105792	1171	1177	**1.105792**	**4.894208**
90	1300	1322	1308	1310	11.1355287	**14.555556**	1318.619016	1300	1322	**18.61902**	**3.380984**
100	1460	1460	1455	1458.33333	2.88675135	**14.583333**	1465.13224	1455	1460	**10.13224**	**-5.13224**
110	1614	1612	1606	1610.66667	4.163332	**14.642424**	1611.645464	1606	1614	**5.645464**	**2.354536**
120	1758	1761	1760	1759.66667	1.52752523	**14.663889**	1758.158688	1758	1761	**0.158688**	**2.841312**
130	1905	1905	1907	1905.66667	1.15470054	**14.658974**	1904.671912	1905	1907	**-0.32809**	**2.328088**
140	2052	2058	2053	2054.33333	3.21455025	**14.67381**	2051.185136	2052	2058	**-0.81486**	**6.814864**
150	2205	2201	2202	2202.66667	2.081666	**14.684444**	2197.69836	2201	2205	**-3.30164**	**7.30164**
160	2349	2350	2346	2348.33333	2.081666	**14.677083**	2344.211584	2346	2350	**-1.78842**	**5.788416**
170	2499	2500	2495	2498	2.64575131	**14.694118**	2490.724808	2495	2500	**-4.27519**	**9.275192**
180	2646	2643	2642	2643.66667	2.081666	**14.687037**	2637.238032	2642	2646	**-4.76197**	**8.761968**
					Mean	**14.651322**	1391.875628	1390.444444	1396	**1.431184**	**4.124372**
					SD	**0.0602469**	739.8555147	741.6675022	741.602468	**6.01584**	**3.603291**

Values in red show high errors (greater than 5 degrees variation from the mean)

Table C.2.7. Raw, converted, mean and 95% confidence interval data

	Output (mV)				Degrees			Calculations for Mean and 95% Confidence Intervals					
Angle	**Test 1**	**Test 2**	**Test 3**	**Angle**	**Test 1**	**Test 2**	**Test 3**	**Mean**	**SD**	**n**	**Confidence**	**CIL**	**CIH**
10	150	141	145	10	10.23891	9.624573	9.897611	9.920364	0.307799	3	0.348300525	9.572064	10.26866
20	293	290	287	20	20	19.79522	19.59044	19.79522	0.204778	3	0.231724041	19.5635	20.02695
30	441	440	438	30	30.10239	30.03413	29.89761	30.01138	0.104268	3	0.117988107	29.89339	30.12936
40	589	586	588	40	40.20478	40	40.13652	40.11377	0.104268	3	0.117988107	39.99578	40.23175
50	737	736	735	50	50.30717	50.23891	50.17065	50.23891	0.068259	3	0.077241347	50.16167	50.31615
60	880	884	880	60	60.06826	60.3413	60.06826	60.15927	0.157638	3	0.17838125	59.98089	60.33765
70	1030	1032	1034	70	70.30717	70.44369	70.5802	70.44369	0.136519	3	0.154482694	70.2892	70.59817
80	1171	1177	1173	80	79.93174	80.3413	80.06826	80.11377	0.208536	3	0.235976213	79.87779	80.34974
90	1300	1322	1308	90	88.7372	90.23891	89.28328	89.4198	0.760104	3	0.860123239	88.55967	90.27992
100	1460	1460	1455	100	99.6587	99.6587	99.31741	99.54494	0.197048	3	0.222976563	99.32196	99.76791
110	1614	1612	1606	110	110.1706	110.0341	109.6246	109.9431	0.284186	3	0.321581372	109.6215	110.2647
120	1758	1761	1760	120	120	120.2048	120.1365	120.1138	0.104268	3	0.117988107	119.9958	120.2318
130	1905	1905	1907	130	130.0341	130.0341	130.1706	130.0796	0.078819	3	0.089190625	129.9904	130.1688
140	2052	2058	2053	140	140.0683	140.4778	140.1365	140.2275	0.219423	3	0.248296192	139.9792	140.4758
150	2205	2201	2202	150	150.5119	150.2389	150.3072	150.3527	0.142093	3	0.160790686	150.1919	150.5135
160	2349	2350	2346	160	160.3413	160.4096	160.1365	160.2958	0.142093	3	0.160790686	160.135	160.4566
170	2499	2500	2495	170	170.5802	170.6485	170.3072	170.5119	0.180597	3	0.204361395	170.3076	170.7163
180	2646	2643	2642	180	180.6143	180.4096	180.3413	180.4551	0.142093	3	0.160790686	180.2943	180.6159

Appendix D

Supplementary Tables and Figures

D.1 Study One

Table D.1.1. Absolute passive hip moments, stiffness and strain energy during passive leg raising at four knee angles

			Moments (Nm)					Stiffness					Strain Energy				
Hip Angle	Knee Angle		BPF	BPM	NPF	NPM	p	BPF	BPM	NPF	NPM	p	BPF	BPM	NPF	NPM	p
0	180	Mean	2.754	2.506	3.386	3.664	0.768	0.070	0.089	0.089	0.115	0.726					
		SD	1.607	1.623	1.508	2.031		0.029	0.041	0.030	0.047						
	170	Mean	2.342	2.868	3.686	3.711		0.060	0.078	0.088	0.110						
		SD	1.229	2.683	2.456	2.112		0.023	0.048	0.040	0.048						
	160	Mean	2.493	2.518	3.407	3.540		0.054	0.066	0.076	0.096						
		SD	1.592	2.349	1.534	2.148		0.024	0.045	0.029	0.043						
	140	Mean	2.729	3.737	3.406	3.444		0.045	0.069	0.065	0.074						
		SD	2.359	3.152	1.722	2.585		0.024	0.042	0.025	0.040						
15	180	Mean	4.036	5.957	5.020	5.862	0.767	0.106	0.169	0.135	0.189	0.348	50.268	73.497	62.218	70.099	0.940
		SD	2.036	4.031	1.963	2.790		0.037	0.062	0.041	0.069		27.210	55.842	25.862	35.810	
	170	Mean	3.168	5.774	5.278	6.382		0.085	0.144	0.130	0.180		39.325	72.709	66.451	78.478	
		SD	1.766	4.869	3.045	3.585		0.037	0.086	0.054	0.071		23.209	65.978	41.057	48.132	
	160	Mean	3.178	5.021	5.421	5.766		0.073	0.114	0.115	0.146		40.556	64.118	69.883	72.368	
		SD	2.070	4.103	2.831	3.205		0.036	0.062	0.044	0.058		27.967	56.076	39.008	43.946	
	140	Mean	3.022	5.009	4.532	4.778		0.055	0.094	0.273	0.109		39.610	65.444	59.091	61.022	
		SD	2.649	3.975	2.012	3.098		0.032	0.048	0.870	0.054		37.345	56.119	27.887	42.425	
30	180	Mean	5.992	9.992	8.384	9.490	0.761	0.163	0.308	0.209	0.314	0.676	124.436	201.976	179.255	182.959	0.935
		SD	2.546	5.647	3.883	3.882		0.052	0.136	0.061	0.113		61.258	141.446	100.521	84.843	
	170	Mean	4.741	8.593	8.675	9.773		0.132	0.242	0.198	0.288		97.774	178.578	191.168	197.675	
		SD	2.309	5.893	5.036	4.547		0.052	0.125	0.079	0.113		53.416	143.322	132.319	108.085	
	160	Mean	4.499	7.107	8.624	8.447		0.109	0.172	0.160	0.223		97.467	154.017	200.780	177.549	
		SD	2.558	4.949	5.135	3.912		0.048	0.080	0.062	0.088		62.346	123.488	146.202	96.422	
	140	Mean	3.993	6.229	7.006	6.765		0.078	0.132	0.123	0.164		91.790	140.941	165.041	146.590	
		SD	3.037	4.623	3.913	3.762		0.040	0.061	0.046	0.075		79.777	121.206	110.130	93.200	
45	180	Mean	9.032	15.738	12.192	15.267	0.753	0.256	0.492	0.314	0.505	0.595	235.425	400.735	331.640	364.639	0.934
		SD	3.199	7.190	4.528	5.560		0.089	0.245	0.097	0.166		102.968	239.593	162.114	155.224	
	170	Mean	7.237	13.260	12.266	15.242		0.213	0.419	0.295	0.469		186.138	341.021	346.452	381.990	
		SD	3.017	7.513	5.848	6.074		0.084	0.215	0.123	0.199		92.146	244.810	211.804	183.604	
	160	Mean	6.521	10.315	11.460	12.583		0.170	0.271	0.226	0.347		179.001	282.887	350.207	333.003	
		SD	3.140	5.929	5.457	4.998		0.070	0.106	0.090	0.141		104.065	203.761	223.945	160.069	
	140	Mean	5.410	8.682	9.156	9.786		0.117	0.209	0.169	0.252		161.604	251.367	285.412	269.115	
		SD	3.465	5.183	4.198	4.666		0.055	0.093	0.067	0.111		127.886	193.862	169.819	154.228	
60	180	Mean	13.848	19.951	17.966	22.325	0.727	0.410	0.739	0.479	0.750	0.428	404.224	528.414	554.800	594.035	0.949
		SD	4.323	5.066	5.558	5.712		0.173	0.492	0.177	0.207		153.670	190.297	232.100	203.205	
	170	Mean	11.338	19.720	17.668	23.294		0.358	0.640	0.447	0.721		322.826	565.816	568.164	661.251	
		SD	4.085	8.360	7.179	8.395		0.162	0.305	0.213	0.298		140.776	324.117	302.278	289.010	
	160	Mean	9.764	15.537	15.480	19.074		0.281	0.461	0.322	0.549		299.122	473.362	550.484	566.729	
		SD	3.834	6.862	6.010	6.901		0.127	0.179	0.138	0.240		152.976	296.219	304.649	240.087	
	140	Mean	7.714	12.781	12.125	14.479		0.191	0.369	0.235	0.395		261.226	409.471	443.812	448.476	
		SD	3.826	5.761	4.670	6.027		0.095	0.273	0.105	0.179		178.479	270.889	232.595	228.190	
Max	180	Mean	24.790	29.979	31.491	33.616	0.797	0.728	1.079	0.832	1.140	0.444	779.839	791.213	1064.999	877.935	0.860
		SD	5.914	9.547	8.773	8.387		0.315	0.702	0.307	0.382		257.359	337.822	414.321	271.027	
	170	Mean	23.053	26.985	30.361	33.941		0.795	0.992	0.780	1.100		671.880	728.179	1069.596	963.185	
		SD	5.782	8.311	9.353	8.152		0.475	0.545	0.309	0.448		212.233	355.243	486.641	370.505	
	160	Mean	19.962	28.625	26.632	32.270		0.688	0.961	0.584	0.978		618.740	889.712	1058.143	993.398	
		SD	5.049	7.780	8.976	7.169		0.455	0.576	0.295	0.440		243.094	447.647	472.063	283.622	
	140	Mean	15.287	24.528	20.817	28.688		0.477	0.797	0.426	0.836		532.485	817.686	864.813	935.006	
		SD	5.355	7.024	7.311	9.545		0.336	0.615	0.236	0.452		288.917	393.907	356.810	340.382	

Table D.1.2. Group comparisons of anthropometric data before outliers were removed, showing no significant differences between groups ($p > 0.05$)

		Age (years)			Height (m)			Weight (kg)			BMI		
	n	Mean	SD	p	Mean	SD	p	Mean	SD	p	Mean	SD	p
BPF	12	29.8	8.0	0.42	1.65	0.07	0.00	63.48	6.91	0.01	23.18	1.80	0.166
BPM	13	33.0	8.5		1.78	0.09		81.10	14.41		25.30	3.12	
NPF	12	32.9	8.8		1.68	0.04		68.22	9.11		24.23	3.28	
NPM	15	8.8	7.8		1.80	0.10		75.95	10.27		23.36	2.32	

Table D.1.3. Group comparisons of physical activity habits (hours per week), showing no significant differences between groups ($p > 0.05$).

	Walking (hrs/week)			Sitting (hrs/week)			Moderate (hrs/week)			Vigorous (hrs/week)		
	Mean	SD	p	Mean	SD	p	Mean	SD	p	Mean	SD	p
BPF	18.35	21.06	0.289	30.63	17.91	0.286	6.29	12.17	0.696	7.79	11.72	0.698
BPM	9.42	15.53		29.96	12.43		7.55	10.75		5.59	5.08	
NPF	6.54	4.5		33.17	11.88		4.04	5.57		4.88	1.87	
NPM	13.4	16.99		24.11	15.71		4.17	5.91		5.2	4.28	

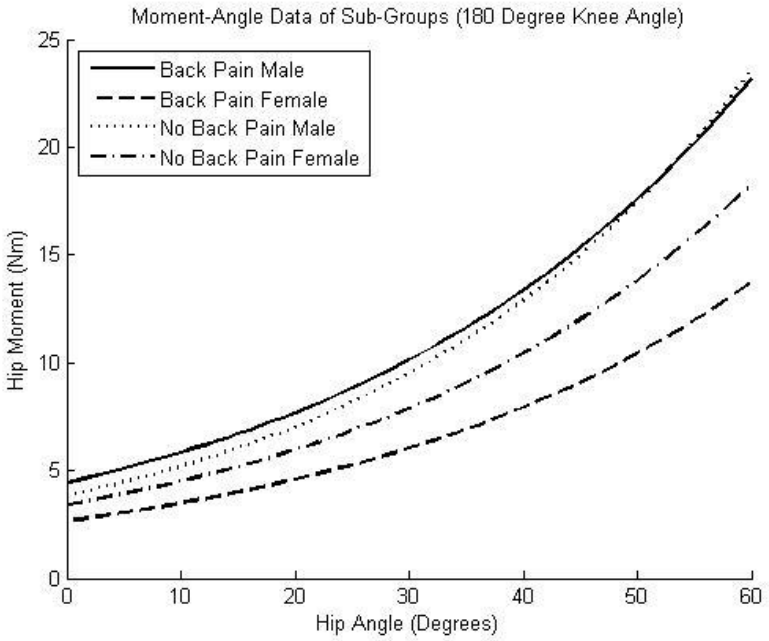


Figure D1.a

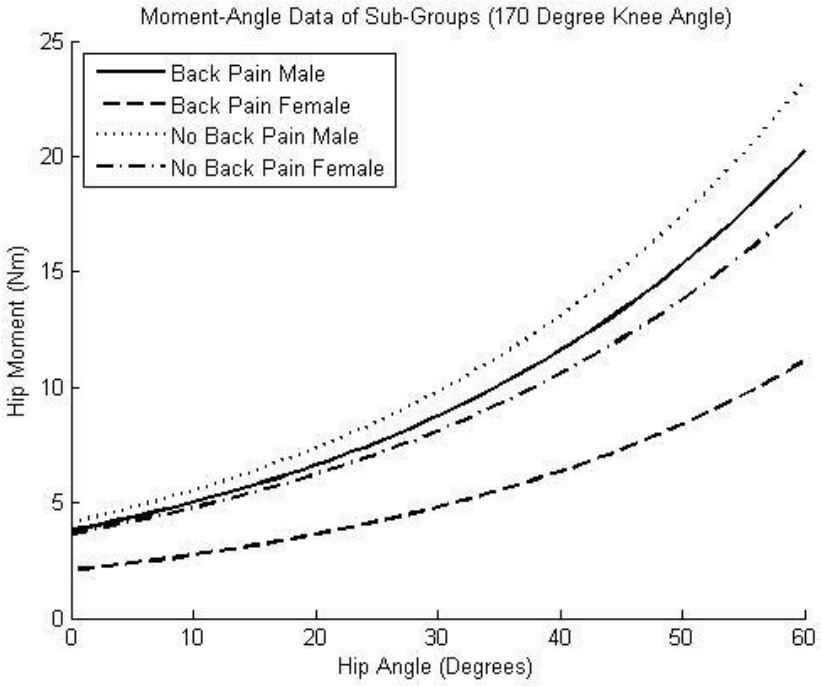


Figure D1.b

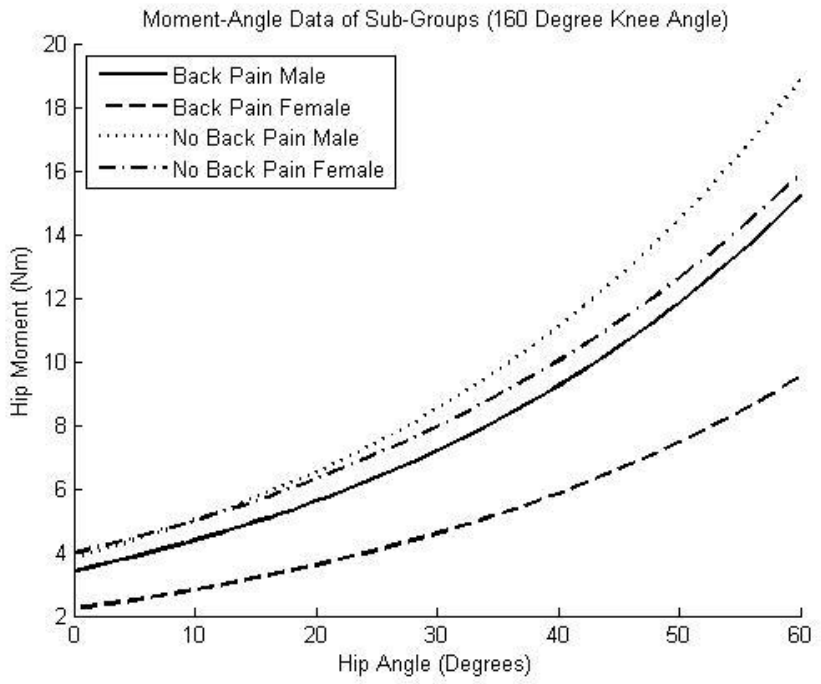


Figure D1.c

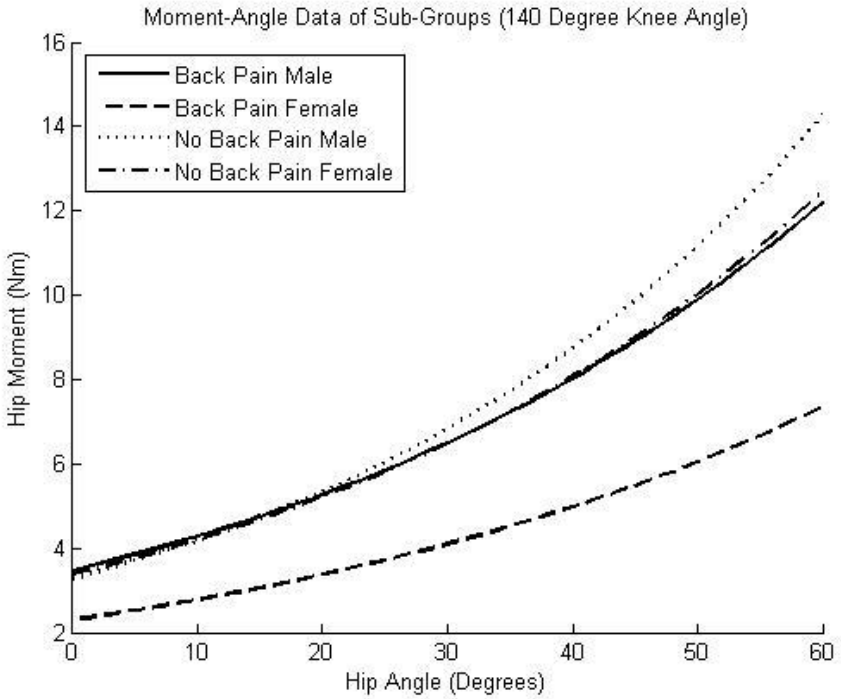


Figure D1.d

Figures D1a, D1b, D1c and D1d show absolute sub-group moment-angle curves at knee angles of 180, 170, 160 and 140 degrees, respectively. A MANOVA demonstrated no significant main effects of group and gender at any hip angle for moments, stiffness or strain energy, within any of the 4 knee angle conditions ($P > 0.05$).

D.2 Study Two

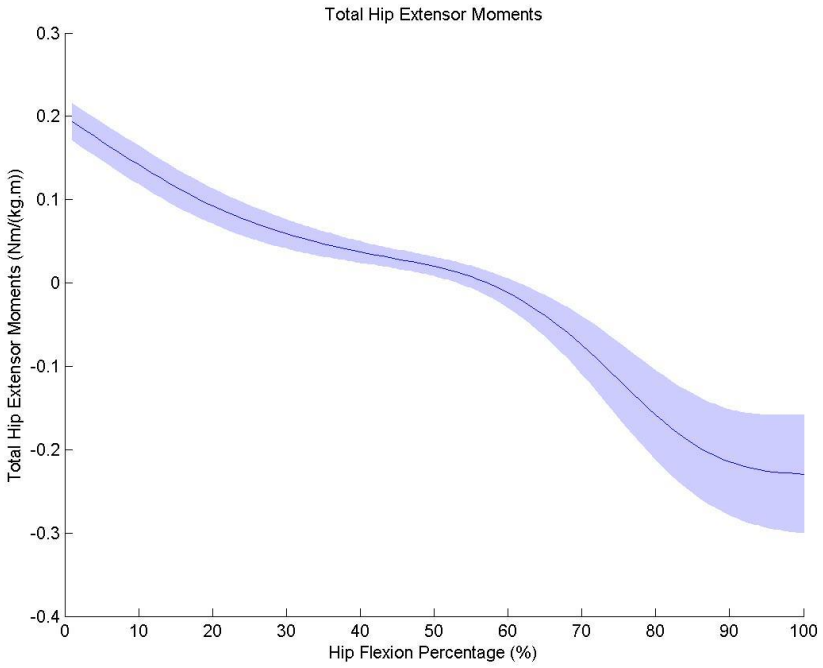


Figure D.2.1 Mean curve with 95% confidence intervals of normalised total hip moments during hip flexion component of gait cycle, between hip neutral and maximum hip angle. Curves show unsmoothed data.

D.3 Study Three

Table D.3.1. Gait parameter data for LBP and NBP groups, expressed as mean (SD)

Gait Parameter	LBP	NBP	P
Cadence (steps/min)	113.05 (-8.90)	112.31 (-5.97)	0.626
Double Support (s)	0.23 (-0.05)	0.25 (-0.07)	0.151
Foot Off (%)	60.52 (-1.91)	60.65 (-3.47)	0.818
Opposite Foot Contact (%)	50.01 (-2.17)	50.2 (-2.3)	0.663
Opposite Foot Off (%)	11.16 (-2.8)	11.88 (-3.27)	0.239
Single Leg Support (s)	0.72 (-2.1)	0.42 (-0.04)	0.315
Step Length (m)	0.66 (-0.06)	0.65 (-0.07)	0.881
Step Time (s)	0.53 (-0.05)	0.54 (-0.04)	0.844
Stride Length (m)	1.3 (-0.1)	1.26 (-0.14)	0.105
Stride Time (s)	1.07 (-0.09)	1.07 (-0.08)	0.755
Walking Speed (m/s)	1.86 (-4.4)	1.2 (-0.17)	0.274

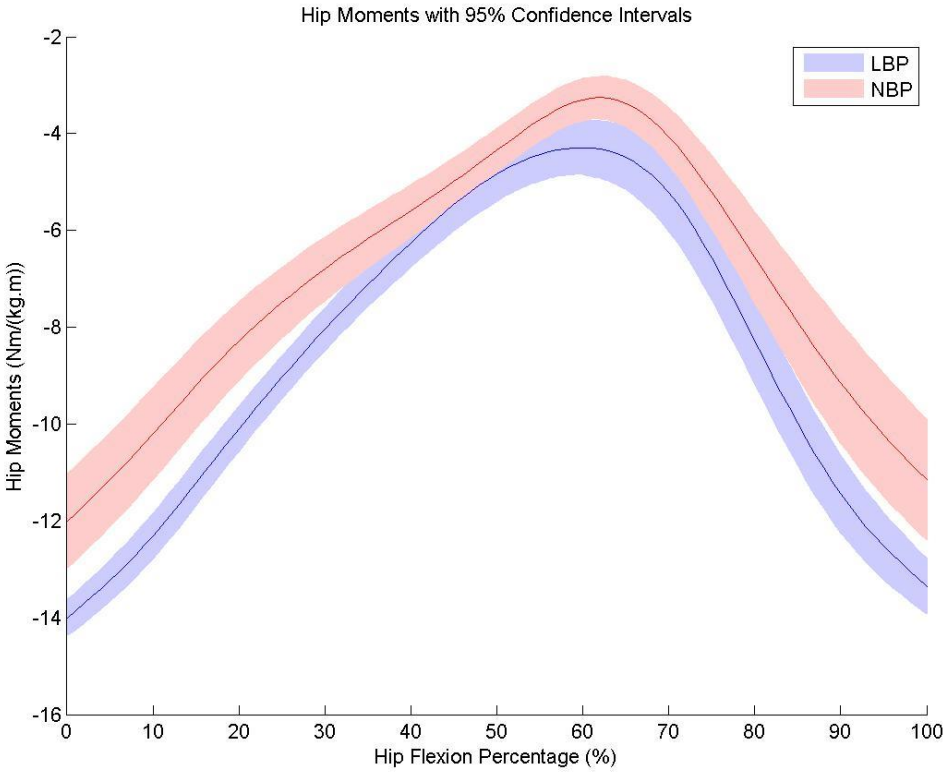


Figure D.3.1. Absolute values of total hip moments during hip flexion from hip neutral to maximum hip angle

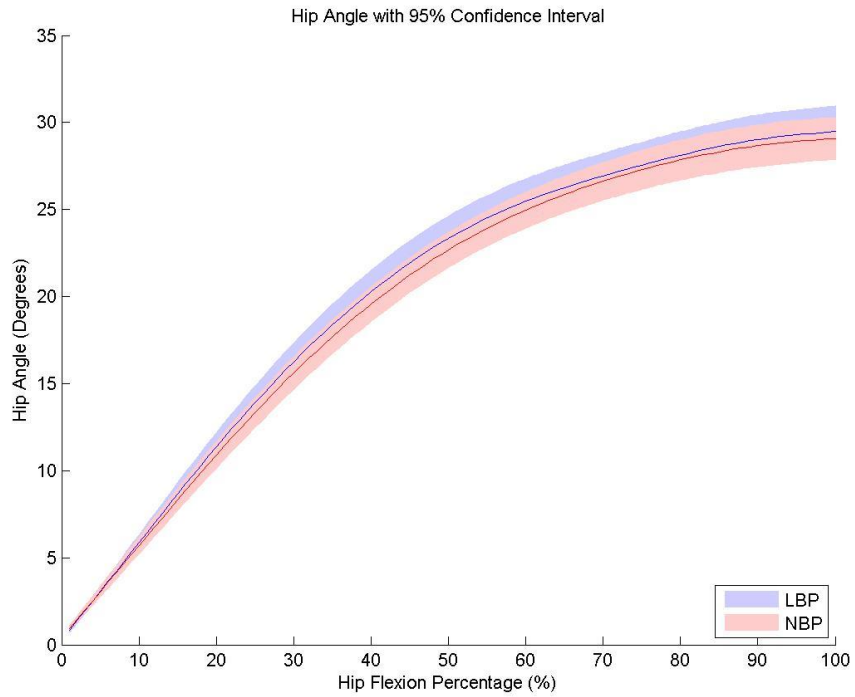


Figure D.3.2 Hip angle during hip flexion component of gait cycle, from hip neutral to maximum hip angle

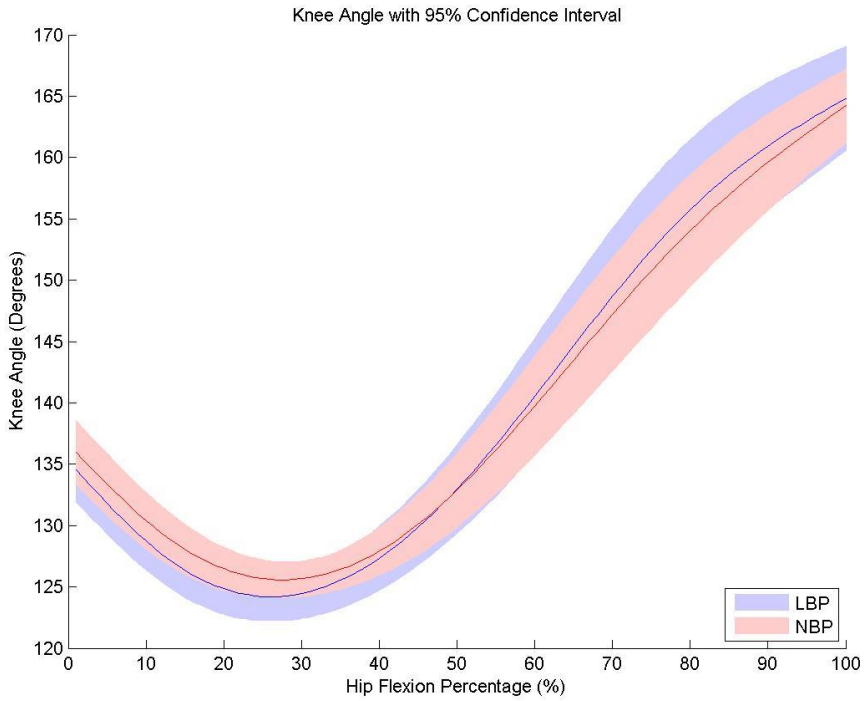


Figure D.3.3. Knee angle during hip flexion component of gait cycle, from hip neutral to maximum hip angle

Appendix E

E. Motion Capture and Force Plate Data Collection and Processing

E.1 Validity of Motion Capture and Force Plates

Various studies have independently assessed the accuracy and precision of Kistler force plates (Bobbert and Schamhardt, 1990, Nayak, 1987, Rogind *et al.*, 2003, Schmiedmayer and Kastner, 1999, Middleton *et al.*, 1999) and Vicon camera systems (Kuxhaus *et al.*, 2009, Tsushima *et al.*, 2003, Kidder *et al.*, 1996, Windolf *et al.*, 2008, Eichelberger *et al.*, 2016), including test-retest and interrater reliability (Tsushima *et al.*, 2003). Further, both systems were independently assessed via a service company for the equipment in the laboratory. Assessments of force plate accuracy and precision were performed prior to each subject's test, and the force plates were zeroed prior to subject measurements. Vicon cameras were checked via calibration tests before each subject was tested, in accordance with the manufacturer's guidelines.

In addition, sensitivity tests were performed to assess variability in hip moments due to placement errors of the markers. Initially, the standard walking trials were completed to ensure 5 full gait cycles on each leg. The tests were then repeated with the two hip joint markers moved posteriorly by 1 cm. A final series of tests were completed with the hip joint markers moved distally 1 cm, and the knee joint markers moved 1 cm proximally. Hip moments during gait

were assessed during peaks of hip flexor moments (FL), and two peaks of hip extensor moments (Ext1, Ext2, figures E.1.1-E.1.3).

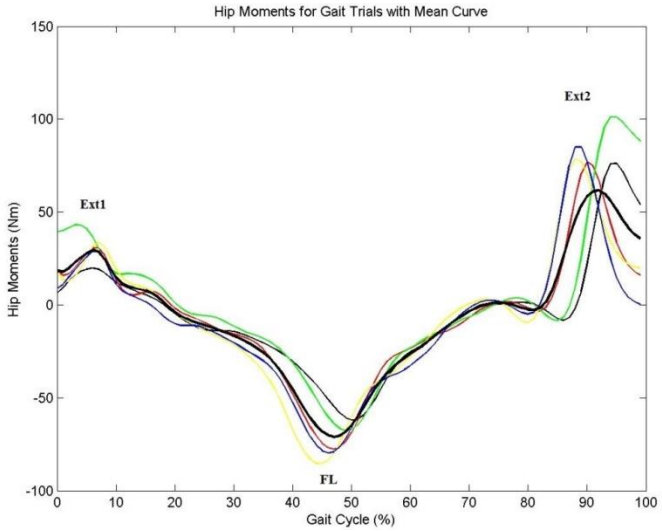


Figure E.1.1 Hip moments during gait cycle showing curves of 5 walking trials and mean curve (black line), with peaks of hip flexor moments (FL) and hip extensor moments (Ext1, Ext2).

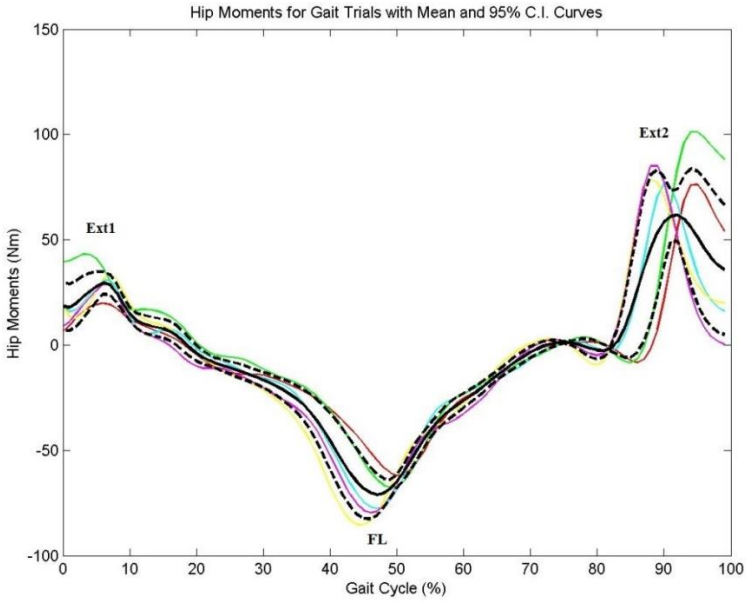


Figure E.1.2 Hip moments during gait cycle showing curves of 5 walking trials and mean curve (solid black line), and 95% confidence intervals (dashed black line). Includes peaks of hip flexor moments (FL) and hip extensor moments (Ext1, Ext2).

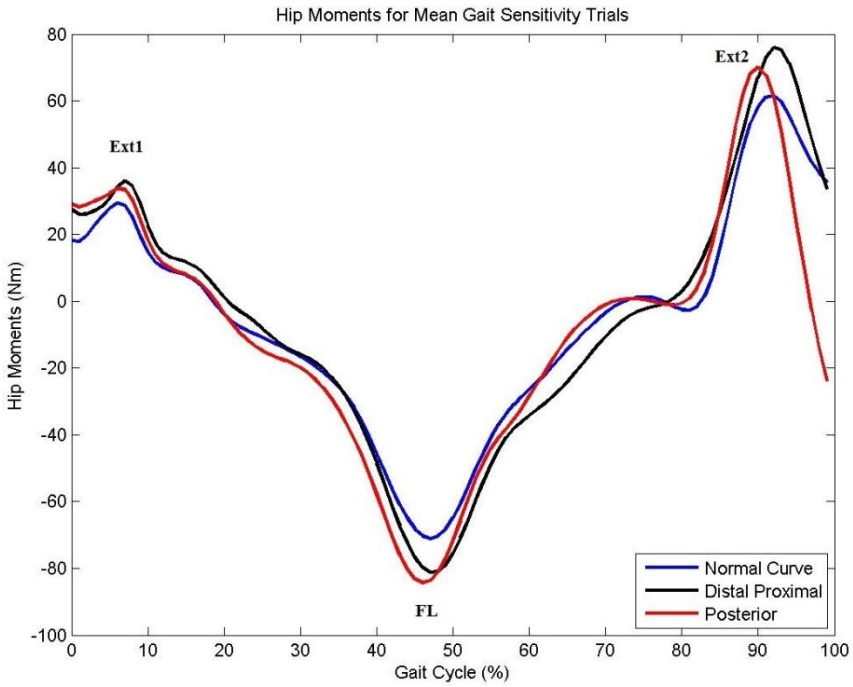


Figure E.1.3 Mean curves for alternate marker locations, with peaks of hip flexor moments (FL), and hip extensor moments (Ext1, Ext2) used in comparisons. Marker locations were in the recommended locations ('Normal Curve'), with the hip marker moved distally 1 cm and knee marker moved 1 cm proximally ('Distal Proximal'), or the hip marker positioned 1 cm posteriorly ('Posterior').

The importance of correct marker placement for gait analysis has been reported in the literature. Comparisons were made of absolute hip moments during the peak of hip flexor moments (table E.1) and the first and second peaks of hip extensor moments (tables E.2 and E.3, respectively). Future tests should involve a greater number of walking trials to maximise consistency and reduce trial variability, to better assess any effect of altered marker placement. Assessments of multiple subjects of markedly different anthropometry would also be of use.

Table E.1. Comparisons of peak hip flexor moments during gait cycle with altered positions of lower limb markers

		Peak Hip Flexor Moments (Nm)			
Marker Placement	**Leg**	**Mean (SD)**	**C.I. Lower**	**C.I. Upper**	**P-Value**
Normal	Left	-74.466 (9.531)	-82.820	-66.112	
	Right	-78.308 (3.832)	-81.667	-74.949	
Distal-Proximal	Left	-84.914 (2.598)	-87.191	-82.637	0.02
	Right	-87.569 (8.151)	-94.714	-80.424	
Posterior	Left	-86.034 (7.336)	-92.464	-79.604	
	Right	-89.059 (7.308)	-95.465	-82.654	

Table E.2 Comparisons of first peak hip extensor moments during gait cycle with altered positions of lower limb markers

		First Peak Hip Extensor Moments (Nm)			
Marker Placement	**Leg**	**Mean (SD)**	**C.I. Lower**	**C.I. Upper**	**P-Value**
Normal	Left	31.235 (8.393)	23.879	38.591	
	Right	56.814 (28.472)	31.858	81.771	
Distal-Proximal	Left	41.693 (11.961)	31.210	52.178	0.961
	Right	50.746 (23.087)	30.509	70.982	
Posterior	Left	43.545 (17.698)	28.031	59.058	
	Right	45.291 (13.594)	33.375	57.206	

Table E.3 Comparisons of second peak hip extensor moments during gait cycle with altered positions of lower limb markers.

		Second Peak Hip Extensor Moments (Nm)			
Marker Placement	**Leg**	**Mean (SD)**	**C.I. Lower**	**C.I. Upper**	**P-Value**
Normal	Left	83.632 (10.416)	74.502	92.762	
	Right	73.838 (6.151)	68.446	79.230	
Distal-Proximal	Left	81.518 (7.426)	75.009	88.028	0.546
	Right	62.072 (26.334)	38.990	85.155	
Posterior	Left	90.607 (3.596)	87.454	93.759	
	Right	68.739 (29.672)	42.731	94.748	

Overall, movement of the markers away from the recommended location of joint centres led to an overestimation of hip joint moments during the peak hip flexor and extensor moments, with significant differences between normal location and posterior and distal-proximal positions ($P < 0.05$), with a trend but no significant differences between posterior and distal-proximal positions ($P >$

0.05), and no significant differences at the first or second peak extensor moments for any marker positions (P > 0.05). Non-significant differences during Ext1 and Ext2 were attributed to normal variability in each of the 5 gait cycles on each leg (approximately 10% variation between normal mean and means of the alternative marker locations). However, variation between normal mean and alternative marker means during FL were greater than 30% of normal mean values in one set of comparisons (left leg) (see appendix C.3 for data tables and graphs of comparisons). The findings of this sensitivity assessment show the importance of correct marker placement for ensuring validity of collected data. Although there was potential for errors in marker placement, errors were reduced through having a single, experienced tester placing all markers on all subjects.

E.2 Total Moment Data Processing

Data was initially assessed via the Nexus software programme (Vicon Nexus version 1.8, Vicon, UK). Each subject walk was visually checked to ensure 5 clear foot strikes of each foot onto a force plate, and that reflective markers were visible for a minimum of one complete gait cycle for each of the 5 walks. Each subject's reflective markers were labelled following the Vicon Nexus guidelines. A data processing pipeline was created in Vicon Nexus to perform standard data modelling of the walking trials. The pipeline included Woltring filtering and gap filling. Each subject trial was subsequently checked for errors and marker gaps were filled as appropriate. A final pipeline was created for smoothing the data and producing model trajectories, calculating gait cycle

parameters, and saving and exporting the output data required for further analysis.

Hip moments and joint angles and forces were calculated using the Vicon Nexus Plug-in Gait software, using the 'conventional gait model' based upon the Newington-Helen Hayes model. The conventional gait model has been previously validated (Davis R., 1991, Kadaba *et al.*, 1990). The data was transferred to Microsoft Excel and Matlab for further processing. Data was smoothed using a low-pass Butterworth filter at 6 Hz. Mean moment data was calculated from the 5 smoothed moment curves (figure E.2).

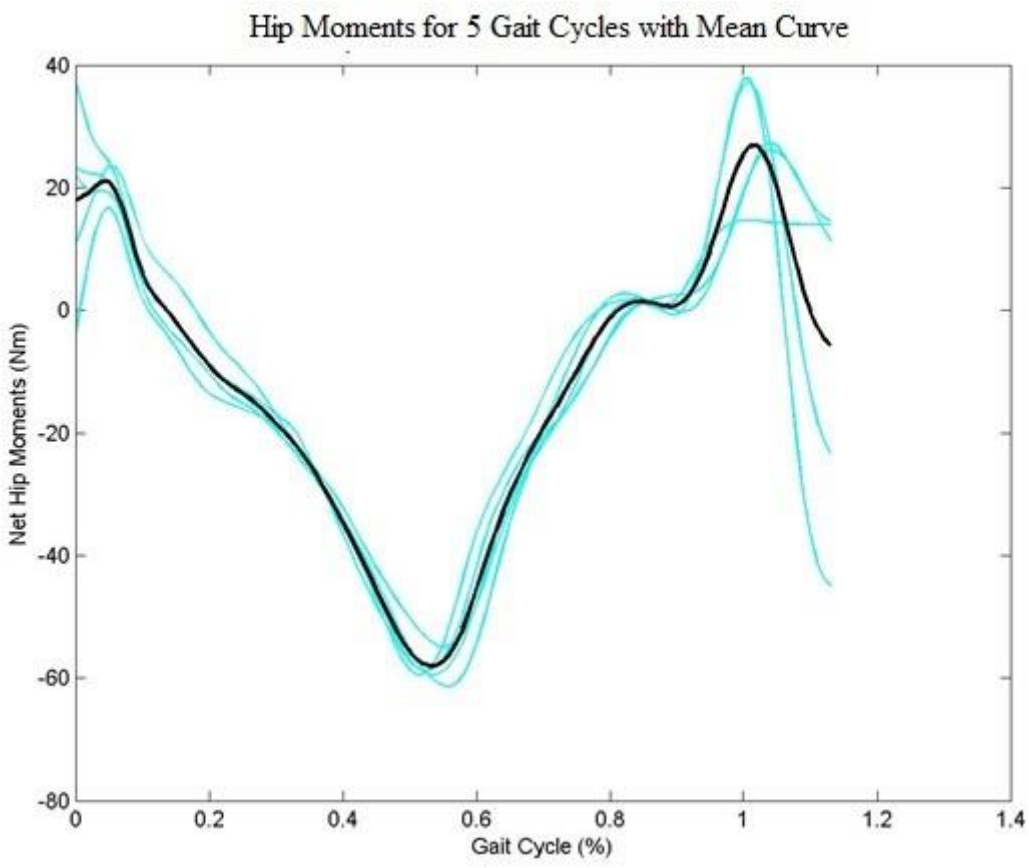


Figure E.2 5 smoothed moment datasets (light blue) and mean moment data (black line) for one leg of a subject.

Appendix F

Description of Models and Equations

F.1 Dynamic Biomechanical Model

Summary of Passive Moment Data Processing

The calculation of passive hip moments was initially described in Chapter 3. The information is repeated here for reference before describing in greater detail how each component was calculated.

The following is from Chapter 3:

The dynamic biomechanical model was established by Lee and Munn (2000):

$$M = F_x y_f + F_y x_f + m_{leg}\ddot{x}_{cg}y_{cg} - (m_{leg}g + m_{leg}\ddot{y}_{cg})x_{cg} - m_{leg}k^2\ddot{\theta}$$

Equation (F.1)

Where *Fx*, *Fy*, are the forces applied to the leg to flex the hip joint, x_f, y_f, are the locations of force application to the leg, m_{leg} is the mass of the leg, *g*, is acceleration due to gravity, $x_{cg,}$, y_{cg}, refer to the location of the centre of mass of the leg and *k* is the radius of gyration. $\ddot{x}_{cg}, \ddot{y}_{cg}$ refer to the acceleration of the leg centre of mass, and $\ddot{\theta}$ is the angular acceleration of the leg (figure F.1).

x_f, y_f, and $x_{cg,}$, y_{cg} are calculated for the left and right legs using the directional cosine method, taking into account the segment positions at different

hip and knee angles. $x_{cg,}$, y_{cg} were based upon segment mass parameter data in the literature (Dempster, 1955). k of the whole leg is calculated by determining the mass moment of inertia for the individual lower limb segments.

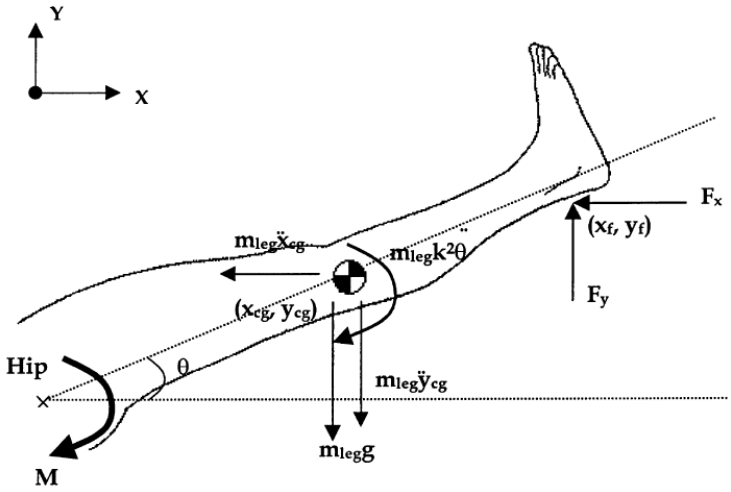


Figure F.1 Representation of biomechanical model components

Data Processing in Matlab

The following refers to the stages involved in calculating passive hip moments from the raw input data. The data acquired is from two load cells (to represent horizontal and vertical forces applied to raise the leg) and three inclinometers (for the measurement of hip angle and knee angle, the determination of hip angular acceleration, and tilt of the force transducer). Data from three passive leg raising tests was processed concurrently.

- Data is analogue-digitally converted at 50 Hz
- A 2 Hz low-pass Butterworth filter is applied to the raw data
- Inclinometer outputs are converted from mV to degrees and radians
- Load cell outputs are converted from mV to Newtons

Calculation of Fx and Fy

F_x, F_y, are the horizontal and vertical forces applied to the leg, respectively, in order to rotate the hip joint through sagittal plane flexion. The *x* and *y* load cell outputs are resolved to account for any rotation of the force transducer within the housing on the ankle brace where the transducer is located. This approach requires the directional cosine method. Values calculated are in Newtons:

For the left leg:

$$F_x = L_x \cos\theta_{lcp} + L_y \sin\theta_{lcp}$$

$$F_y = L_x \sin\theta_{lca} + L_y cos\theta_{lcp}$$

For the right leg:

$$F_x = L_x \cos\theta_{lcp} + L_y \sin\theta_{lcp}$$

$$F_y = L_x \sin(-1\theta_{lca}) + L_y cos\theta_{lcp}$$

Equation (F.2)

Where L_x, L_y are the load cell outputs for horizontal and vertical forces, respectively. θ_{lc} is the roll of the force transducer, and therefore the two load cells, in radians. The load cell roll is either as positive (absolute, θ_{lcp}), or actual (positive or negative, θ_{lca}), to ensure the directional cosine method can resolve the forces correctly for both legs. For F_y, the sine of the angle is sensitive to being either positive or negative (from θ_{lca}), whereas in the rest of the calculation it is always positive (from θ_{lcp}). This accounts for whether the rotation of the transducer is towards or away from the hip. When the right leg is

measured the transducer faces the opposite direction to the left leg, and the inclinometer direction must also be reversed, as shown by a negative sine angle for the calculation of Fy for the right leg in equation F.2.

Calculation of X_f and Y_f

X_f, Y_f, are the locations of force application to the leg, in horizontal and vertical directions, respectively. In real terms, this is the location relative to the hip joint from where the force transducer is pushed up and towards the hip via the ankle brace into which it is inserted.

$$X_f = l_{thigh}.cos(\theta_{hip}) + l_{res}.cos(\theta_{hip} - (180 - \theta_{knee} + \theta_{tran}))$$

$$Y_f = l_{thigh}.sin(\theta_{hip}) + l_{res}.sin(\theta_{hip} - (180 - \theta_{knee} + \theta_{tran}))$$

Equation (F.3.1)

Where l_{thigh} is the length of the thigh segment and l_{res} is the resultant length (hypotenuse) of the shank segment that includes the force transducer (equation F.3.2). The force transducer was 13.15 centimetres from the base of the foot towards the knee in the x-plane, and 3.85 centimetres below the shank in the y-plane, when the leg was in neutral (knee extended with subject lying supine). θ_{hip} and θ_{knee} are the hip and knee angles, respectively. θ_{tran} is the angle of the line from the knee joint to the force transducer location when the knee is in neutral (equation F.3.3). $\theta_{hip} - (180 - \theta_{knee} + \theta_{tran})$, gives the resultant angle

between the knee joint to the force transducer. The force transducer is positioned below the ankle when the hip is in neutral. θ_{knee} is for angles of the knee where 180 degrees is the knee in neutral (extended), hence $(180 - \theta_{knee})$ converts this to the knee in extension being equal to 0.

$$l_{res} = \sqrt{(l_{shank} + l_{foot(x)} - 0.01315)^2 + 0.0385^2}$$

Equation (F.3.2)

Where l_{shank} is the length of the shank and $l_{foot(x)}$ is the length of the foot (x-plane).

$$\theta_{tran} = tan^{-1}\ (-0.0385 / l_{sh} + l_{ft(x)} - 0.01315)$$

Equation (F.3.3)

Calculation of m_{leg}

The mass of the leg, m_{leg}, was calculated based upon body mass (m_{body}) and body segment data from Dempster (1955) (table F.1).

$$m_{leg} = m_{body}\ x\ 0.161$$

Equation (F.4)

Calculation of Leg Centre of Mass (X_{cg}, Y_{cg})

$x_{cg,}$, y_{cg}, refer to the location of the centre of mass of the leg. Because the passive leg raise tests included four different knee angles, a single value of leg

centre of mass could not be used, and was calculated based upon the individual segment centre of mass and knee angle. Body segment parameters are different for males and females, so calculations were gender-specific, based upon published values (Dempster, 1955, table F.1). The directional cosine method was used to resolve leg centre of mass into horizontal (*x)* and vertical (*y)* components (equation F.5.1).

Table F.1. Body segment parameters from Dempster (1955) showing segment mass (SM) proportion of body mass (BM) and centre of gravity (CG) relative to segment length.

	Location (Proximal to Distal)	**SM / BM**	**CG / Length**
Total Leg	Greater trochanter to medial malleolus	0.161	0.447
Thigh	Greater trochanter to femoral condyles	0.1	0.433
Shank	Femoral condyles to medial malleolus	0.0465	0.433
Shank and Foot	Femoral condyles to medial malleolus	0.061	0.606
Foot	Lateral malleolus to head of 2nd metatarsal	0.0145	0.5

$$x_{cg} = \frac{(m_{body}.0.1)(l_{th}.0.433).\cos(\theta_{hip}) + (m_{body}.0.061)(l_{th}.\cos(\theta_{hip}) + sf_{cg}.\cos(\theta_{hip} - (180 - \theta_{knee} + \theta_{sfcg})}{m_{leg}}$$

$$y_{cg} = \frac{(m_{body}.0.1)(l_{th}.0.433).\sin(\theta_{hip}) + (m_{body}.0.061)(l_{th}.\sin(\theta_{hip}) + sf_{cg}.\sin(\theta_{hip} - (180 - \theta_{knee} + \theta_{sfcg})}{m_{leg}}$$

Equation (F.5.1)

Where m_{body} is body mass, m_{leg} is leg mass, l_{th} is the length of the thigh segment, sf_{cg} is the centre of gravity of the combined shank and foot segment, and θ_{sfcg} is the angle between the midline of the shank in neutral and sf_{cg}. These were calculated by initially determining the centre of gravity of the shank and foot in both x and y planes $x(sf_{cg})$, $y(sf_{cg})$ (equation F.5.2), respectively, from

which the resultant length (equation F.5.3) and angle to centre of gravity (equation F.5.4) could be calculated. Values are of segment mass relative to body mass, and segment centre of gravity location, relative to proximal segment joint.

$$x(sf_{cg}) = \frac{(m_{body}.0.061)(l_{sf}.0.606)}{m_{body}.0.061}$$

$$y(sf_{cg}) = \frac{(m_{body}.0.0145)(l_{ft(y)}.0.5)}{m_{body}.0.061}$$

Equation (F.5.2)

Where l_{sf} is the sum of the lengths of the shank (femoral condyle to medial malleolus) and foot in the x-plane (medial malleolus to base of foot), and $l_{ft(y)}$ is the length of the foot in the y-plane (heel to end of longest toe). There is no y-plane value for the shank, as this midline is zero relative to the knee joint, so only the foot causes the centre of gravity to be raised above the midline of the shank itself.

$$sf_{cg} = \sqrt{x(sf_{cg})^2 + y(sf_{cg})^2}$$

Equation (F.5.3)

$$\theta_{sfcg} = tan^{-1}\,(y(sf_{cg})/x(sf_{cg}))$$

Equation (F.5.4)

Calculation of *k*

The radius of gyration of the leg, *k*, can be calculated based upon leg length, l_{leg}, and body segment data from Dempster (1955).

$$k = l_{leg} \cdot 0.326$$

Equation (F.6.1)

l_{leg} is measured as the distance from the greater trochanter to the medial malleolus, in agreement with the measurements by Dempster (1955). However, this equation is only correct when the knee is in neutral. When the knee is bent, the radius of gyration must be calculated based upon the moment mass of inertia and segment mass of each segment (thigh, shank and foot).

$$I = mk^2$$

Equation (F.6.2)

Where *I* is the mass moment of inertia, *m* is the segment mass, and *k* is the radius of gyration with respect to the segment centre of gravity. The mass moment of inertia at the hip is equal to the sum of the moment mass of inertia of the hip (I_{th}), shank (I_{sh}) and foot (I_{ft}):

$$I_{hip} = I_{th} + I_{sh} + I_{ft}$$

Equation (F.6.3)

Each segment mass moment of inertia can be calculated from the segment centre of gravity, segment mass and distance of segment centre of gravity to the hip:

$$I_{th} = th_{cg} + (m_{th} \cdot d_{th}^2)$$

$$I_{sh} = sh_{cg} + (m_{sh} \cdot d_{sh}^2)$$

$$I_{ft} = ft_{cg} + (m_{ft} \cdot d_{ft}^2)$$

Equation (F.6.4)

Where *d* is the distance from segment centre of gravity to hip for thigh (d_{th}), shank (d_{sh}) and foot (d_{ft}) segments. *cg* is the location of the segment centre of gravity for the thigh (th_{cg}), shank (sh_{cg}) and foot (ft_{cg}).

The moment of inertia for the thigh segment is derived from equation F.6.4, incorporating segment parameter data from Dempster (1955), shown in table F.2:

$$I_{th} = (m_{body} \cdot 0.1) \cdot (l_{th} \cdot 0.323)^2 + (m_{body} \cdot 0.1) \cdot (l_{th} \cdot 0.433)^2$$

Equation (F.6.5)

Calculation of moment of inertia for combined shank and foot section is:

$$I_{sfc} = (m_{body} \cdot 0.061).(l_{sf} \ 0.416)^2$$

Equation (F.6.6)

The hypotenuse of the moment mass of inertia (d_{sf}) of the combined shank and foot segment establishes a single value from x and y planes (I_{sfx}, I_{sfy}, respectively):

$$I_{sfx} = L_{th} + (L_{sh} \cdot 0.606) \cos (180 - \theta_{knee})$$

$$I_{sfy} = (Lsh \cdot 0.606) \sin (180 - \theta_{knee})$$

$$d_{sf} = \sqrt{I_{sfx}{}^2 + I_{sfy}{}^2}$$

Equation (F.6.7)

Where L_{th} and L_{sh} are the segment lengths of the thigh and shank, respectively. The resultant moment mass of inertia of the shank is calculated as:

$$I_{sh} = I_{sfc} + (m_{body} \cdot 0.061) \cdot d_{sf}^2$$

Equation (F.6.8)

Calculation of whole leg mass moment of inertia (I_{hip}):

$$I_{hip} = I_{th} + I_{sh}$$

Equation (F.6.9)

Hip radius of gyration (k_{hip}):

$$k_{hip} = \sqrt{\frac{I_{hip}}{(m_{body} \times 0.161)}}$$

Equation (F.6.10)

Table F.2. Segment parameters. Segment mass (SM) as proportion of body mass (BM), centre of gravity (CG) location relative to segment length from proximal joint centre, and radius of gyration relative to segment length centre of gravity (Dempster, 1955).

		SM / BM	CG / Length	k / Length
Total Leg	Greater trochanter / medial malleolus	0.161	0.447	0.326
Thigh	Greater trochanter / femoral condyles	0.1	0.433	0.323
Leg	Femoral condyles / medial malleolus	0.0465	0.433	0.302
Foot and leg	Femoral condyles / medial malleolus	0.061	0.606	0.416
Foot	Lateral malleolus / head of 2nd metatarsal	0.0145	0.5	0.475

Calculation of $\ddot{x}_{cg}$, $\ddot{y}_{cg}$ and $\ddot{\theta}$

Acceleration of the leg centre of gravity, $\ddot{x}_{cg}$, $\ddot{y}_{cg}$ and angular acceleration of the hip, $\ddot{\theta}$, were determined from the calculated values of leg centre of gravity, *xcg*, *ycg* and hip angle, *θ*, during leg lifting. Numerical differentiation was used to calculate the acceleration of *xcg*, *ycg* and *θ*, based upon the change in values (*n*) over time of each during leg lifting (Stroud, 2013). Equation F.7 was established from the standard formula for centred five-point second derivative numerical differentiation from Taylor theorem. Centred numerical differentiation was preferred to forward or backwards methods due to its being considered more accurate. Second derivative differentiation was considered most

appropriate for determining accelerations. Five-points were used to give an adequate spread of *n*.

$$\ddot{x}_{cg} = \frac{-x_{cg}(n+2) + 16x_{cg}(n+1) - 30x_{cg}(n) + 16x_{cg}(n-1) - x_{cg}(n-2)}{12(0.02^2)}$$

$$\ddot{y}_{cg} = \frac{-y_{cg}(n+2) + 16y_{cg}(n+1) - 30y_{cg}(n) + 16y_{cg}(n-1) - y_{cg}(n-2)}{12(0.02^2)}$$

$$\ddot{\theta} = \frac{\theta_{hip}(n+2) + 16\theta_{hip}(n+1) - 30\theta_{hip}(n) + 16\theta_{hip}(n-1) - \theta_{hip}(n-2)}{12(0.02^2)}$$

Equation (F.7)

The denominator, 12, is a constant to agree with the values in the nominator. The value of 0.02 is the difference in values along the x-axis that the differentiation applies to. The first two and last two values in the datasets are assigned a zero value to ensure there are sufficient data points for differentiation to occur across the full dataset.

F.2 Predictive Equation

To estimate hip joint passive moments at any combination of hip and knee angle during gait, a predictive equation was developed, based upon actual data collected during passive leg raise tests at four pre-determined knee angles. Three lifts were performed at each fixed knee angle, and the mean of each three lifts was fitted with an exponential function. The four mean curves were

subsequently fitted with a best-fit, 3-D surface plot of hip angle (x), knee angle (y), and passive hip moments (z) (figure F.2).

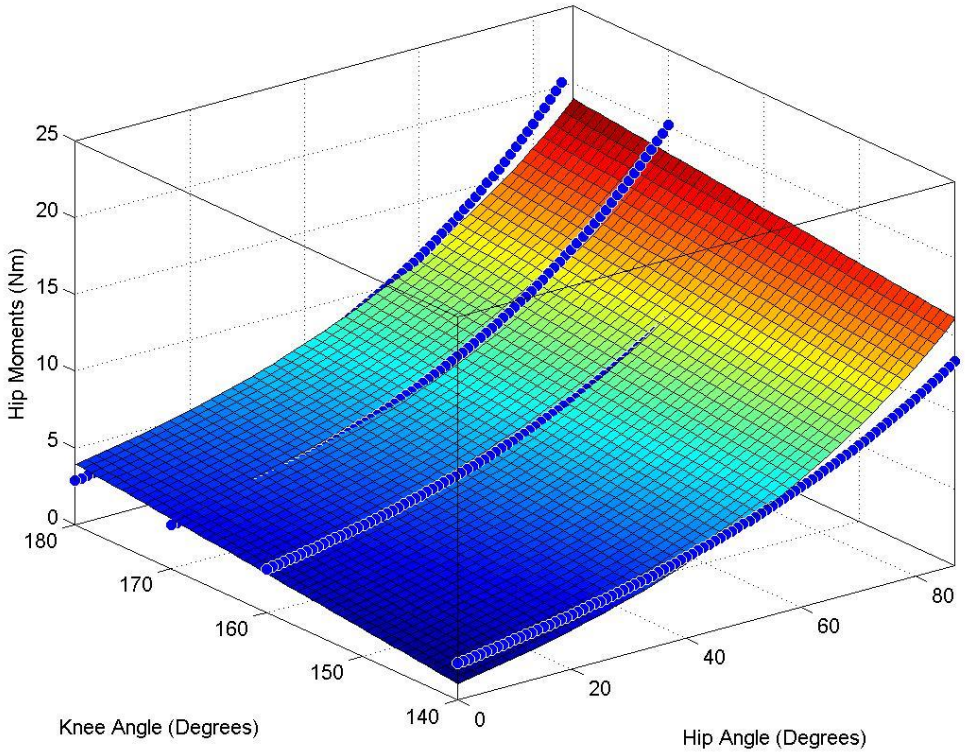


Figure F.2. Single subject surface plot showing four mean curves, each with an exponential fit, determined at knee angles of 140, 160, 170 and 180 degrees. This surface plot shows absolute passive hip moments (Nm).

A programme was written into Matlab to calculate the four coefficients for the predictive equation. The first code used was to calculate goodness of fit (gof) of the coefficients (sum of squares due to error (SSE), r-squared, error degrees of freedom (DFE), adjusted r-squared, and root mean squared error (RMSE):

```
fitresult = cell( 2, 1 );
gof = struct( 'sse', cell( 2, 1 ), ...
    'rsquare', [], 'dfe', [], 'adjrsquare', [], 'rmse', [] );
```

Further code was written with predetermined fit-type and the predictive equation, from which the programme would calculate the coefficients:

```
[xInput, yInput, zOutput] = prepareSurfaceData( x, y, z );
ft = fittype( 'a*y+b*exp(c*x)+d', 'indep', {'x', 'y'}, 'depend', 'z' );
opts = fitoptions( ft );
opts.Lower = [-10 -10 -10 -200]; opts.Upper = [10 100 10 100];
```

The coefficients with 95% confidence bounds are shown in table F.2. Goodness of fit for this dataset was calculated with values for r-squared (0.965), adjusted r-squared (0.965) and RMSE (0.832), DFE (346), SSE(239.512).

Coefficient	Value	CI Lower	CI Upper
a	0.071682	0.06576	0.077604
b	2.862015	2.28842	3.435611
c	0.021058	0.01909	0.02303
d	-11.81216	-13.0246	-10.5998

Table F.2. Coefficients with 95% confidence bounds for single subject leg data determined from surface fitting programme for predictive equation

The example above shows single leg data (left leg) of one subject and includes absolute values only. Coefficients for the right leg were subsequently calculated, and the process was repeated to determine the equation coefficients for predicting passive hip moments normalised to body mass and height.