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I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis including any required final revisions, as accepted by my examiners.

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Abstract

Background: Knee osteoarthritis (OA) is one of the most debilitating and prevalent diseases associated with aging, and is estimated to affect 9% of men and 18% of women over 65 years of age (Davis et al., 1991). Knee OA affects the condylar surfaces of the joint and its associated structures (bone, cartilage, ligaments, and fat pad) and if left untreated generally leads to the slow and painful degeneration of the joint and surrounding structures. Knee OA remains one of the few chronic diseases of aging with few effective, non-invasive treatments. This study will be the first of its kind to investigate the effect of therapeutic taping techniques on knee pain, as well as the kinematics and kinetics of the knee and lower limb during walking gait.

Methodology: Fifteen participants (10 male, 5 female) with radiographic diagnosed knee osteoarthritis (OA) were recruited for this study (Types II and III). Participants had three taping conditions (no tape, sham tape, therapeutic tape) applied in a randomised, counterbalanced design. During each tape condition, each participant was required to complete several tasks of daily living. Spatiotemporal, knee pain, knee and lower limb kinetic and kinematic variables were measured during the straight line walking gait task. A one-way repeated measures ANOVA, with a Sidak post hoc comparison was used to compare differences in dependent variables between the three taping groups (α = 0.05).

Results: All participants were shown to have low function and quality of life from the KOOS questionnaire administered prior to the testing (57.0 ± 9.5. Therapeutic knee taping was shown to significantly reduce the self-reported levels of pain during activities of daily living (p = 0.017). No other differences were observed between the taping groups for the spatiotemporal, kinematic and kinetic variables.
Conclusion: Therapeutic knee taping was shown to reduce knee pain during activities of daily living which could improve knee related quality of life. There were no acute changes in knee spatiotemporal, knee kinetic or knee kinematic variables following therapeutic knee taping. Future research should be directed at the effect of prolonged periods of therapeutic knee taping and walking gait biomechanics; however, the results of this study are only relevant to the acute use of therapeutic knee taping.
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1.0 Research Problem

1.1 Introduction
Knee osteoarthritis (OA) is one of the most debilitating and prevalent diseases associated with aging, and is estimated to affect 9% of men and 18% of women over 65 years of age (Davis et al., 1991). Knee OA affects the tibiofemoral and patellofemoral compartments of the knee joint and its associated structures (bone, cartilage, ligaments, and fat pad). OA remains one of the few chronic diseases of aging with few effective, non-invasive treatments. Current treatment methods are all potentially invasive, expensive and/or do not treat the mechanical etiology of disease progression/initiation (drugs, exercise, surgery, walking aids). There are also many pharmaceutical interventions that treat the symptoms of OA related knee pain, but fail to influence the mechanical etiology of disease. Excessive joint loading has been shown to be related to elevated rates of knee OA progression. Current biomechanical models used to study OA progression rely on external knee joint moments, joint kinematics and/or muscle excitation measures in combination to estimate the internal forces applied to the condylar surfaces during functional activities such as walking, running and/or stair ascent/descent. Currently, no research has been conducted as to how therapeutic taping may affect the biomechanics of the knee joint, specifically knee joint kinematics and kinetics during the gait cycle, as well as how these changes are related to disease progression. Therapeutic knee joint taping has the potential to offer an inexpensive, simple and effective alternative to traditional conservative management options for knee pain among knee OA populations. Due to the large percentage of older populations (9% of all men, 18% of all women over 65) who suffer from knee OA, this research has the potential to greatly improve the quality of life of many people across Australia and worldwide by offering a non-invasive and inexpensive pain management option. Aside from the potential benefits to a participant’s health related quality of life, this
study will be the first of its kind to investigate the effect of therapeutic taping techniques on knee pain, knee kinematics and knee kinetics within a knee OA population.

1.2 Aims
The purpose of this research is to determine if therapeutic knee taping influences the knee mechanics and perceived knee pain and symptoms among knee OA populations (Types II and III) during walking gait. Specifically, we aim to investigate the effect of therapeutic knee taping on:

- Self-reported knee pain during walking gait
- Spatiotemporal variables during walking gait
- Knee kinetics during the stance phase of walking gait, with a particular interest in external knee flexion and adduction moments
- Knee flexion angle during the swing and stance phases of walking gait.

1.3 Hypotheses
It is hypothesised that therapeutic knee taping, relative to the no tape and sham tape conditions, will:

- Reduce self-reported levels of knee pain during walking gait
- Increase stride length and velocity during walking gait
- Reduce the magnitude of peak knee adduction and flexion moments during the first and second half of stance during walking gait
- Increase knee flexion range of motion during the stance and swing phases during walking gait.
2.0 Literature Review

2.1 Osteoarthritis
In Australia alone, the annual combined (direct and indirect) cost of all forms of arthritis is $9 billion (Access Economics, 2007). Osteoarthritis (OA) is the most prevalent form of arthritis worldwide (Felson et al., 2009). In individuals over 60 years of age, patients diagnosed with knee OA almost always present with knee pain, which is thought to be one reason these populations have a diminished health related quality of life and the primary reason these individuals actively request invasive knee replacement surgery as a viable treatment option (Losina et al., 2012; Nguyen et al., 2011). With a greater portion of our population living beyond 60 years of age, it is estimated that the prevalence of OA and the request for surgical intervention will double in the next two decades (Felson et al., 2007). In 2010, there were a total of 25,970 knee replacement surgeries in Australia, a 67% increase when compared to seven years prior. Though knee replacements are becoming common treatments for OA patients, up to 30% of patients report little to no functional improvement and/or are dissatisfied with various functional outcomes post-surgery, which are generally associated with increased knee pain and/or swelling (Levinger et al., 2012; Paulsen et al., 2011).

The knee is the largest and one of the most complex joints in the human body (Kulowski, 1932). In total, eight individual muscles cross the joint. The muscle groups crossing the knee are characterised by the movement they produce at the knee joint; the quadriceps (rectus femoris, vastus lateralis, vastus medialis, vastus intermedius) for knee extension, the hamstrings (semitendinosus, semimembranosus, biceps femoris) for knee flexion, the gastrocnemii (medial and lateral) for knee compression and flexion, and the popliteus muscle which unlocks the knee during stance by medially rotating the tibia (Williams,
All of these muscles are important contributors to the co-ordination of the knee and lower limb during the walking gait cycle. However, it has been theorised that the function of the muscles crossing knee more than any other joint generates the knee flexion/extension movement through dynamic coupling meaning there is a complex coordination/function between these muscles to carry out relatively simplistic tasks like walking and/or stair ascent (Kerrigan et al., 1991; Perry et al., 1987). Interestingly, using a computer simulation approach Yamaguchi and Zajac (1989) found that hip flexion moments generated during the swing phase of the walking gait cycle have the ability to generate a flexion moment about the knee. It is not well known how each individual muscle of the lower limb contributes to the human walking gait cycle; however, it is known that the functioning of the lower limb musculature to initiate dynamic human movement is incredibly complex and multifaceted, making the clinical interpretation of an individual’s walking gait pattern an incredibly detailed and time consuming process (Piazza and Delp, 1995).

The knee joint is often modelled as possessing medial and lateral compartments, defined by the articulating surfaces of the medial and lateral femoral and tibial condyles. Contained within the knee are two distinct joints; the tibiofemoral and patellofemoral, which when combined, create a modified hinge to allow flexion and extension as well as internal/external rotation and varus/valgus movements (Williams, 1995). Bone, ligaments, cartilage, tendons, fat pads and menisci are all located within a joint capsule, which is filled with viscous synovial fluid. These structures, in part, function to allow cartilage to absorb oxygen and nutrients via diffusion during dynamic movements (Saladin, 2004). Knee cartilage is entirely hyaline cartilage, composed of chondrocytes and extracellular matrix molecules (Temenoff and Mikos, 2000). While chondrocytes only contribute about 1% by volume of hyaline cartilage, they are essential for cartilage nutrition as these cells are used
to recycle degraded or damaged matrix molecules. Proteoglycans are heavily glycosylated proteins found in connective tissue. Due to their ability to attract water, proteoglycans are thought to be responsible for much of the resilience and distribution of stress along the articular cartilage. Cartilage repair can occur naturally; however, cartilage loses its ability to self-replenish over time, making articular surface repair difficult (Temenoff and Mikos, 2000). Natural cartilage repair is further slowed by degradation of the articular surface and loss of hyaline cartilage, thought to be initiated by excess mechanical stress (Temenoff and Mikos, 2000).

Pathologically, OA affects all structures of the joint, and an appreciation for how changes in one area of the knee may affect others is necessary to understand the process and progression of this disease. Knee OA is characterised by hyaline articular cartilage loss; however, it is increasingly recognised that OA is a disease of the entire knee joint and associated structures (Felson et al., 2007). Therefore, it is the joint in its entirety, not merely the articular cartilage surfaces, that needs to be assessed when attempting to understand the cause, progression and severity of the disease in pathological populations if we are to effectively manage the symptoms of knee pain during the activities of daily living and mitigate disease progression. Excessive forces through the knee can cause degradation of the hyaline cartilage. Over time, bone surfaces become sclerotic and harden, resulting in osteophyte formation and changes in the synovium (Saladin, 2004).

There are three common fat pads of the knee which include the infrapatellar fat pad (IPFP), posterior suprapatellar fat pad and anterior suprapatellar fat pad (Figure 1.). The IPFP is located within the knee capsule and is a large collection of extra-synovial adipose tissue which is highly concentrated with nociceptors (Clockaerts et al., 2010). Degeneration of hyaline cartilage and the associated malalignment of condylar surfaces during active knee
flexion and extension can put excessive pressure on the IPFP, which is of interest to most clinicians for the management of OA related knee pain and is thought to be the number one contributor to knee pain among OA populations (Bastiaansen-Jenniskens et al., 2013; Clements et al., 2009; Ushiyama et al., 2003).

![Figure 1. Normal knee fat pads of a healthy adult population. Yellow = Infrapatellar fat pad (Hoffa’s), Blue = posterior suprapatellar fat pad, Red = anterior suprapatellar fat pad. (www.radiopaedia.org)](image)

Abnormal joint loading is a major factor for the increased progression of knee OA, with excessive loading to specific areas on the condylar surface of the knee joint thought to perpetuate cartilage damage and/or tibiofemoral malalignment during the walking gait cycle (Temenoff and Mikos, 2000). As articular cartilage is completely aneural, this mechanical process is unlikely to be the actuator of OA related knee pain. The true cause of OA related knee pain can be difficult to determine as there are many pathological features
that co-exist by the time OA is diagnosed. Guermazi et al. (2012) reported a prevalence of osteophytes (which form on the bony surface), cartilage damage and bone marrow lesions (BMLs) in almost 90% of people older than 50 years, who had no evidence of knee OA. Studies using MRI to understand the mechanical risk factors associated with OA related knee pain have shown there are three strong and consistent factors related to cartilage loss: malalignment of the tibiofemoral joint, BMLs and meniscal disease (manifested either as a tear or extrusion), all of which can cause knee pain or contribute to excessive stress placed on the fat pads of the knee (Felson et al., 2007).

2.2 Measuring knee contact forces
The progression of OA is thought to be accelerated by elevated joint contact forces concentrated on a specific area of the articular surface that is diseased (Guilak 2011; Roemhildt et al., 2012). Though possible in an advanced, computationally expensive biomechanics laboratory, the measurement and/or change of joint contact forces in clinical settings is not currently feasible for most laboratories interested in collecting large samples of data (n > 10) (Fregly et al., 2012). Current best-practice and clinically orientated research uses external knee loading measures, joint kinematics and, when possible, in combination with muscle activation to estimate the internal contact forces applied to the condylar surface of the knee during dynamic walking gait tasks (Heiden et al., 2009). Knee flexion angle during the stance phase is also of interest as it indicates the area over which the force is distributed over the condylar surface during a dynamic task like walking.

Inverse dynamics is the computational approach generally used to calculate estimates of external joint forces and surface electromyography (EMG) muscle activation. An understanding of what is being measured as well as these measurement limitations can provide researchers with clinically relevant information associated with condylar loading.
and the likely progression of this disease (Gerus et al., 2013). The condylar surface of the knee is unique to each individual and will be dependent on many factors including age, sex, height and the presence of bony deformities. It is currently not feasible to conduct subject specific modelling of the condylar surface in most clinical settings as expensive medical imaging techniques such as MRI and computer tomography (CT) scans need to be incorporated within the subject-specific scaling and kinematic/kinetic modelling procedures.

The rigid link assumption is described by Robertson et al. (2004) and is the assumption that all skeletal bones within the human body can be represented as a perfectly rigid segment. It is limited by our knowledge that bones are capable of deformation and that joint surfaces translate, resulting in a loss of energy through the joint to passive structures like cartilage and meniscus, and the environment. Biomechanical models also assume that segment lengths are fixed and rigidly connected to adjacent segments such that when a force is applied to a distal segment, it is conserved as it travels proximally through the limb. The foot is one such body part that is often considered a single segment in kinematic models; however, it can clearly bend at the metatarsal-phalangeal joints. Modelling assumptions simplify the complicated musculoskeletal system, eliminating the need to quantify changes in mass distribution brought about by tissue deformation and movement of bodily fluids. These assumptions are necessary for efficient measurement of forces through joints; however, it is the skill of biomechanists to understand where limitations and assumptions in the modelling process can influence clinical interpretation of their data.

Interpretation of muscle activation from EMG is limited to total muscle activation (TMA) and co-contraction ratios (CCR) across a joint; however, these are not estimates of individual muscle forces (Winter et al., 2001). A limitation of these measures is that they do
not account for the individual force-length and force-velocity properties of the muscle’s contractile element nor the dynamics of a muscle’s series and parallel elastic components. The gold standard measure for joint contact forces is finite element modelling; however, it is only achievable with subject specific kinematics, individual muscle force estimates (EMG-driven or static optimisation musculoskeletal models) and high resolution static and weight-bearing MRI (Halloran et al., 2005). This process is subject specific and the cost of multiple MRI scans as well as their time consuming nature result in it not yet being viable for use in clinical settings. TMA provides an estimate of the compressive forces placed through the joint, while directed CCR provides information on distribution of forces on the condylar surface. This gives critical insight into the neuromuscular adaptations that people with knee OA make to avoid pain during walking. CCR shows the relative hamstring to quadriceps force production across the walking gait cycle. Medial-lateral CCR gives insight into how the condylar surface load is distributed. CCR data for a healthy population has been widely reported making it is a useful comparison tool (Ebert et al., 2009).

Though current clinical measurement tools are limited in their ability to estimate condylar loading, we do have the ability to use external joint loading, joint kinematics and muscle activation together to estimate the magnitude and location of articular joint loading during the walking gait cycle. External knee adduction moments are useful measures to determine the medial-lateral distribution of external joint forces applied to the knee. Similarly, the knee flexion moment can be used to determine the anterior-posterior distribution. These measures indicate which knee compartment receives the majority of the external forces applied to the knee during the walking gait cycle.

A recent study by Meyer et al. (2013) investigated how well external knee loads and EMG measures predicted internal knee contact forces during walking gait. A participant with an
An instrumented knee implant capable of measuring medial and lateral joint contact forces was recruited for this study. Four walking gait patterns were performed while internal knee contact and moment data were collected simultaneously with 3D motion capture, ground reaction force and muscle activation parameters. The study showed that, while different walking gait patterns produced variable internal and external estimates of joint forces/moments, the estimated external force values were moderate predictors ($R^2 < 0.48$) of the magnitude of internal forces. Additionally, Meyer et al. (2013) demonstrated that external knee adduction moments were moderate to strong predictors ($R^2 = 0.59$) of joint articular loading. Knee joint kinetics, kinematics and muscle activation can provide a reasonable estimate of joint contact forces at the knee during walking gait. Though we are unable to measure the exact magnitude of condylar loads in-vivo, the changes observed following a treatment have been proven reliable.

2.2.1 Walking gait patterns in healthy adults
Musculoskeletal models of the knee have shown that the medial compartment bears the majority of load during the stance phase of walking gait and in some individuals can be large enough to completely unload the lateral compartment (Schipplein and Andriacchi, 1991). It has been suggested that medial compartment loading results from high knee adduction moments during walking gait is the mechanical etiology characteristic of knee OA (Miyazaki et al., 2002).

Recent EMG-driven models, which take into consideration the moments generated by individual muscles, have been used to estimate joint contact forces during healthy walking gait (Winby et al., 2009). These models show that while the medial compartment of the knee does support the majority of the load during the stance phase of walking gait in healthy adults, forces produced by muscles on the lateral aspect of the knee are sufficient to
stabilise the joint and counter the tendency to unload the lateral compartment. Joint compression was largely provided by the quadriceps, hamstrings and gastrocnemius muscles as well as the tensor fascia latae (TFL). This suggests that it may be important to look at the muscle contribution at the hip via the TFL as well as the gluteal muscles when making estimates of medial-lateral compartment loading during the walking gait cycle. Recording the muscle activation and calculating the TMA and CCR of muscles crossing the hip and knee can provide invaluable information when assessing the joint contact forces among OA populations (Ebert et al., 2010).

2.2.2 Walking gait in OA patients
Substantial research has been conducted investigating the differences in walking gait between a knee OA population and healthy adults. Consistently reported findings include differences in spatio-temporal, kinetic, kinematic and muscle activation measures. Additionally, patients with knee OA exhibit greater subjective assessments of knee pain during dynamic tasks when compared to healthy controls (Heiden et al., 2009). It was also shown that OA patients demonstrate a significantly reduced self-selected walking speed, shorter stride length and a greater time in stance when compared to healthy controls. Kinematic differences include a lower range of motion at the hip, knee and ankle joints (Al-Zahrani et al., 2002) as well as greater knee flexion at heel strike (Heiden at al., 2009). These adaptations have implications on the mechanical etiology of knee OA progression; greater stance time results in a greater time of contact between articular surfaces of the knee, and lower knee flexion range of motion results in the contact forces being concentrated through a smaller area on the articular cartilage.

Muscle activation patterns and kinetic differences have been closely examined, and while it has been shown that different strategies can be adopted to complete the same task
(Liikavainio et al., 2010), OA patients exhibit significantly greater net or total muscle activation in both loading and early stance, with lateral muscles showing increased activation with increasing knee adduction moments (Heiden et al., 2009). Greater TMA of the muscles crossing and supporting the knee is a characteristic of knee OA patients, which is thought to be associated with elevated joint stiffness and compressive forces, both of which would be considered a negative adaptation in the context of knee OA disease progression.

Some discrepancies have been observed between studies with some reporting greater knee extension moments (e.g. Mundermann et al., 2005), some showing greater knee flexion moments (Baliunas et al., 2002, Childs et al., 2004, Joss et al., 2006), while others reporting no difference between OA populations and health age-matched controls (Rudolph et al., 2007, Smith et al., 2004). However, most studies have shown that increased adduction moments are consistent among OA populations and can lead to a bow-legged posture and increased loading through the medial compartment of the knee. These findings reflect the complex nature of OA disease progression and should be considered together when attempting to make clinical assessment of these patient populations.

2.3 Treatment of knee osteoarthritis
Orthopedic surgery is a method frequently used to treat knee OA when other, more conservative modalities, have failed to reduce pain or improve a patient’s ability to ambulate and/or complete normal activities of daily living. Three surgeries are commonly used to treat knee OA; high tibial osteotomy, partial knee arthroplasty (PKA) and total knee arthroplasty (TKA). High tibial osteotomy can be used to achieve more even distribution of force across the condylar surface, or an increased application of force to a non-diseased portion of the knee. This surgery is becoming less common due to the increasing success of
knee arthroplasty; however, it remains useful in appropriate patients with unicompartmental knee OA, particularly for younger patients. A controlled fracture in the tibia, or uncommonly the femur (low femoral osteotomy), allows the alignment of the tibia with respect to the knee to be altered so that force during walking gait tasks are redirected through the healthy compartment of the knee. The goal of high tibial osteotomy is to unload articular surfaces and to correct angular deformity at the tibiofemoral articulation (Wright et al., 2005).

PKA surgery is subject specific and dependent on the progression of knee OA within the joint. This procedure is only recommended when OA is confined to a single knee compartment, and will be one of two types; patellofemoral arthroplasty or unicondylar arthroplasty. PKA surgery consists of a metal component that is implanted into the knee, to replace the diseased articular surface (Schweigel et al., 2010). Advantages of PKA over a TKA include preservation of bone stock and cruciate ligaments, less invasive surgery, faster surgical recovery, minimal blood loss, greater range of knee flexion and lower costs of surgery (Rajasekhar et al., 2004).

TKA surgery is a viable treatment when the disease is not contained to a single compartment. Alignment is critical to the function and survival of TKA. The reason alignment is so important is because it is the number one predictor of poor outcomes following a TKA (Insall et al., 1985). While surgery is a popular and often successful treatment, knee implants tend to have a limited lifespan of no more than 15 years before requiring replacement. Surgery also has many associated risks and side effects including pain, infection and failure to correct malalignment as well as a long period of recovery that tends to increase with age (Insall et al., 1985). Subsequent to the risks of the surgery itself,
the average cost of a bilateral TKA is $15,045 making it by far the most expensive treatment option (Lavernia et al., 1997).

Many non-invasive treatments of knee OA fail to modify the mechanisms associated with the pathology and/or prevent the progression of the disease. These non-invasive treatments are generally focused on reducing knee pain and inflammation to allow patients to continue with their activities of daily living. Although they mask the symptoms of pain, these treatments fail to address the underlying mechanical etiology and the disease, meaning it will likely continue to progress until an invasive treatment is required. Treatment methods include, but are not limited to; oral and topical non-steroid anti-inflammatory drugs, cyclooxygenase-2 inhibitors, bracing or taping, orthotics and exercise therapy. Prescription pharmaceutical treatments have been shown to be the most effective non-invasive treatment for treating OA related knee pain when compared to the other treatment options (Felson et al., 2007). Though effective, side effects may include gastrointestinal risk for oral treatments and dryness, itching and redness for topical treatments in up to 40% of patients (Felson et al., 2007). Prescription pharmaceutical agents to treat OA related knee pain also have the potential to have confounding effects on chronic health conditions that generally present among OA populations over the age of 60 - including heart disease, Type II diabetes and cancer.

Knee bracing and taping have both been shown to greatly reduce pain in patients with patellofemoral OA as well as populations with general knee pain (Cushnaghan et al., 1994, Hinman et, al., 2003, Warden et al., 2008). Many people who suffer from medial compartment OA have been shown to adopt walking gait postures to reduce pain through the medial compartment of the knee. This knowledge has been adapted into potential treatments. Specifically, valgus knee braces have been shown to reduce second peak knee
adduction moments in knee OA patients in recent studies (Pagani et al., 2011), which is a strong predictor of medial compartment loading (Felson et al., 2007). However clinical trials of foot orthotics used to reduce adduction knee moments have been unable to consistently reduce knee pain (Felson et al., 2007). It was hypothesised that this negative result was associated with patient to patient variability, discrepancies in individual walking gait patterns, and the magnitude of the adduction moment during the walking gait cycle. Exercise has been shown to reduce OA related knee pain; however, patient drop out has been a major limiting factor in most clinical trials of this nature (Felson et al., 2007). Some studies have also shown that the effect of exercise on knee pain is only moderate and that some participants have reported no benefit (Messier et al., 2005).

While multiple treatment options are available for OA patients for the reduction of knee pain, the majority have not been shown to alter the lower limb biomechanics nor muscle activation of these patients. Thus, it is unknown if these treatments influence the mechanical aetiology of OA disease progression. The ideal scenario is a non-invasive treatment that reduces knee pain and also alters an individual’s knee joint biomechanics and muscle activation in a way that limits disease progression. Many studies have looked at reduction of pain in non-invasive treatment options for knee OA. However, few, if any, have assessed the potential for changes in knee kinematics, kinetics and muscle activation. Such data could provide information of the mechanical etiology of disease progression. Therapeutic knee taping offers a viable conservative treatment that addresses the alteration of knee biomechanics, muscle activation and progression of knee OA. It is a non-drug, non-invasive treatment which is both cheap and easy to apply.
2.3.1 Knee taping methods
Knee taping offers the potential to reduce OA related knee pain during the activities of daily living as well as allowing for improved patellar tracking. A significant advantage of knee taping is its ability to reduce symptoms of pain among patella-femoral, tibio-femoral and tri-compartmental knee OA populations, which also has the added benefit of being customisable to an individual’s disease pathology with a knowledgeable clinician.

Therapeutic taping immobilises the joint and may also improve neurosensory input which has the potential to improve motor function (Felson et al., 2007). Therapeutic taping aims to alleviate pressure from the infra patellar fat pad, a major source of pain, by shortening the soft tissue and pulling the tibia forward to reduce stress on this structure (McConnell et al., 2013).

A direct benefit of reduced pain is the ability of patients to strengthen the quadriceps. A quadriceps strength deficit often leads to a fixed flexion deformity in knee OA patients (Levinger et al., 2012). The exact mechanism by which taping reduces pain is unclear and research is ongoing to investigate the plausible neuromusculoskeletal mechanisms (Ushiyama et al., 2003). Apart from pharmaceutical interventions, taping is an effective non-invasive method, with reported reductions of up to 25% in subjective knee pain scales (Cushnaghan et al., 1994). However, little research has investigated the mechanical and pain effects together. Therapeutic taping offers a cheap and effective alternative to many currently available treatments of knee related OA pain and it is important to investigate its effect on knee kinetics and kinematics among OA populations.
3.0 Methods

3.1 Participants
Fifteen participants (10 male, 5 female) with radiographic diagnosed knee osteoarthritis (OA) were recruited for this study. Participant information is displayed in Table 1. Participants were recruited through public advertisement and orthopaedic outpatient clinics. All participants were required to have had no prior surgery (with the exception of arthroscopy), be greater than 60 years of age, have a fixed flexion angle no greater than 15° and be able to complete basic activities of daily living without the use of an external aid such as a cane or walker. Testing was conducted at the University of Western Australia (UWA) Clinical Gait Laboratory located at the School of Sport Science, Exercise and Health (SSEH). Upon arrival, participants were given an information sheet outlining the procedures of data collection as well as the significance of the research. The research project was approved by the Human Ethics Office (RA/4/1/6088) (Appendix D). Following informed consent to the testing protocol, each participant’s height, age, weight and anthropometric data were recorded.

3.2 Data collection
The Knee Injury and Osteoarthritis Outcome Score (KOOS) (Roos et al., 1998) was administered prior to testing. The KOOS is a 42 item disease-specific instrument which includes ratings of pain, other symptoms, activities of daily living (ADL), sport and recreation participation (SR) and knee related quality of life (QOL) (Roos and Lohmander, 2003). The KOOS is considered a gold standard pain scale for knee OA populations and has been proven both valid and reliable as a measurement tool (Roos et al., 1998).

The lower-body and trunk UWA marker sets were attached to the skin of the participant over specific anatomical landmarks and segments. Kinematic marker triads (12 mm) were
attached bilaterally to the shank and thigh of the participant. Single markers were placed over the anterior and posterior iliac crests on the left and right sides, as well as the spinous processes of the C7 and T10 vertebrae, the suprasternal notch, xiphoid process and left and right acromion processes. Single markers to define each foot were placed over the metatarsal-phalangeal articulation of the first and fifth digits, as well as the calcaneal tuberosity (Besier et al., 2003). All functional tasks performed during biomechanical testing were completed barefoot.

An 11-camera VICON motion analysis system, consisting of 7 MX and 4 T40 cameras, (VICON MX, Oxford Metrics Limited, Oxford, UK) operating at 100Hz were used to collect kinematic marker data while simultaneously recording ground reaction forces from two AMTI force plates (MCA-6, Advanced Mechanics Technology Incorporated, Watertown, USA) at 2,000Hz. Data were synchronised with GRF force and kinematic data in the Vicon Giganet control box and within the Nexus software (Vicon Peak, Oxford Metrics Ltd., UK). Prior to the taping conditions, participants were required to complete a series of calibration trials specific to the models used in the data processing, in order to create subject specific lower limb and trunk kinematic models of each participant.

3.3 Data modelling
Depending on the capabilities of the participant, non-weight bearing knee swing or weight-bearing squat trials were completed to define the functional flexion/extension axis of the knee. Weight bearing trials were used where possible as the preferred method for defining knee axes. Hip-swing trials were also completed for each leg. Custom MatLab (MatLab 7.9, The Math Works Inc., Natick, Massachusetts, USA) software was applied to the hip-swing and knee trials to define joint axes (Besier et al., 2003; Donnelly et al., 2012). A custom foot alignment rig was used to measure calcaneus inversion/eversion and foot
abduction/adduction which were then used to define the anatomical coordinate system of the ankle joint (Besier et al., 2003). Ankle joint centres and axes were defined using kinematic markers placed over the medial and lateral malleoli of the tibia and fibula respectively. Femoral condyles were digitised using a pointer wand during the calibration to define knee joint centres. Marker trajectories and GRF data were filtered with a low-pass, zero-lag, fourth-order Butterworth filter at 10Hz following residual analysis and visual inspection of the kinematic data. Kinetic and kinematic variables were calculated using custom lower limb kinematic and inverse dynamic BodyBuilder models run through Nexus software.

Peak, minimum and mean knee flexion and adduction moments were calculated for the entire stance phase as well as the first and second half of stance. Peak hip flexion moments were calculated across stance as well as the first and second half of the stance phase. Peak knee flexion angles were calculated across stance, swing and the weight acceptance (WA) phases as well as at heel strike (HS), with knee flexion ROM and minimum flexion across stance also recorded. Peak knee adduction and peak hip flexion and adduction angles were calculated across stance and WA. Additionally, the support moment, which was defined as the percent contribution of the lower limb joints (i.e. ankle, knee, hip) was calculated during the first half of stance (Winter, 2001.).

3.4 Trial conditions
Three taping conditions were applied to each participant in a randomised counter-balanced design. The three taping conditions included:

(1) No tape: no tape was attached to the skin of the participants;
(2) Sham tape: three strips of tape were loosely attached to the skin of the participant above and below the knee joint; and

(3) Therapeutic tape: A four-piece knee taping method for OA. Tape was used to medially align and tilt the patella, and alleviate pressure from the infra-patella fat pad (Figure 2).

![Figure 2. Therapeutic knee taping methods. Each piece serves a specific purpose. Tape 1 tilts the patellar, tape 2 glides the patellar medially, tape 3 and 4 unload the infra-patellar fat pad. An optional tape 5 was used to externally rotate the tibia. (Note: This image shows a left knee)](image)

A sham tape trial was included in the design to mitigate the possibility of a placebo effect. Participants with bilateral knee OA, where both knees met the inclusion criteria, were asked to select the limb which caused more pain and discomfort in day-to-day tasks.
Participants were required to complete three activities of daily living for each of the taping conditions; each was presented in a randomised order:

(1) Natural walking gait trial at a self-selected speed;

(2) Sit-to-stand task from a chair, initiated from a position whereby the thighs were parallel to the floor and knees flexed at 90°, with hands across the torso; and

(3) Ascent and descent of a flight of three stairs (rise = 200mm; going = 300mm).

Six trials were completed for familiarity, involving two repetitions of each activity of daily living. Using a randomised, counter balanced design, participants were then required to complete six successful trials for each activity. The three aforementioned activities were included in the test session that aimed to replicate the different tasks an average participant might encounter during the day. Only the walking gait trials were analysed for this study. A successful walking gait trial was defined as one where the affected limb struck the centre of the force plate, without targeting. Participants completed all tasks at a self-selected pace and were instructed to rest if they felt fatigued. A compulsory 10-minute rest period between each of the taping conditions was enforced to allow for any residual effect of the previous taping condition to disperse. Before the start of testing and following each taping condition a visual analogue scale (VAS), designed to measure participant perceptions of knee discomfort, was completed. The VAS was a measure from 0-100 mm on a horizontal scale. Scores for each section of the knee pain scale were summed to create a total knee pain score. A high score represented a large amount of pain in that specific area of the knee (Appendix B). The VAS was used since no specific knee-related pain scale exists to the best of our knowledge, and the VAS is consistent with assessments of self-reported pain in lower back literature (Donnelly et al., 2007).
3.5 Data analysis
The following events were identified within the walking gait cycle were identified using Vicon Nexus software:

- heel strike (HS), the first frame where the foot contacts the ground (first frame where vertical ground reaction force (GRF) > 10 N)
- toe off (TO), the first frame where no part of the foot is in contact with the ground (first frame where vertical GRF drops below 10 N)
- the first trough in the vertical GRF trace, which will be used to define the WA phase
- mid stance (MS), the mid-point between HS and TO

The phases of the gait cycle analysed included; swing (TO to HS), stance (HS to TO), weight acceptance (WA) (HS to first trough in vertical GRF) and stride (HS to HS of opposite leg), with the stance phase split into the first half of stance (HS to MS) and the second half of stance (MS to TO).

Spatiotemporal variables (walking gait velocity, time in stance, cadence, stride length, stride width) were calculated as per Ebert et al. (2012). Briefly, walking gait velocity (m.s$^{-1}$) was calculated using horizontal displacement of the centre of the pelvis (pelvic origin) over one complete stride divided by the time taken to complete that cycle. Cadence was calculated using the mean number of steps per minute. Stride length (m) was calculated as the difference in the x-coordinate of the left and right ankle joint centres at the HS event for each leg. Stride width was calculated using the mean perpendicular distance between the origins of each foot’s anatomical coordinate system across the time in stance. Time in stance was calculated as the average time from HS of one foot to TO of the same foot.
3.6 Statistical analyses
A one-way, repeated measures analysis of variance (ANOVA) was used to compare differences for the spatiotemporal and walking gait variables between the three taping conditions. For most variables, ANOVAs were a 3-level design with each of the taping conditions; however, the VAS included a fourth, baseline level. All statistical analyses were completed using SPSS for Windows (Version 21.0; SPSS, Chicago, IL, USA). A significance level of $p \leq 0.05$ was used for each test. Post-hoc Sidak tests were used to examine pairwise comparisons to show which specific means differed when significant main effects were observed.
4.0 Results

The KOOS showed a high average pain score and a low quality of life (QOL) among participants with most reporting an inability to complete many (or any) of the sport and recreation based activities (Table 1.). Each section of the KOOS is presented as a percentage of total possible score in that section. A low score suggests that the participant had difficulty completing the activities in that section.

Table 1. Subject characteristics (mean ± SD) and initial pain questionnaire responses for the five subscales of the Knee Injury and Osteoarthritis Outcome Score (KOOS).

<table>
<thead>
<tr>
<th>Subject characteristic</th>
<th>Current study</th>
<th>*Heiden et al. (2009) OA</th>
<th>*Heiden et al. (2009) healthy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>69.0 ± 5.7</td>
<td>65.0 ± 8.0</td>
<td>64.0 ± 6.0</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.6 ± 7.8</td>
<td>170.0 ± 9.0</td>
<td>170.0 ± 9.0</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.6 ± 12.0</td>
<td>81.4 ± 14.2</td>
<td>71.2 ± 13.2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>KOOS questionnaire</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>57.0 ± 9.5</td>
<td>57.6 ± 18.2</td>
<td>96.5 ± 6.0</td>
</tr>
<tr>
<td>Symptoms</td>
<td>62.5 ± 13.2</td>
<td>53.6 ± 17.6</td>
<td>94.3 ± 7.9</td>
</tr>
<tr>
<td>ADL</td>
<td>64.4 ± 10.6</td>
<td>60.0 ± 20.0</td>
<td>96.8 ± 5.5</td>
</tr>
<tr>
<td>SR</td>
<td>15.4 ± 9.1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>QOL</td>
<td>32.5 ± 10.0</td>
<td>32.4 ± 16.8</td>
<td>89.6 ± 14.9</td>
</tr>
</tbody>
</table>

*Data from Heiden et al. (2009) were chosen for comparison as they recruited a similar patient group, and employed similar methods in the same laboratory. ADL = activities of daily living, SR = functions in sport and recreation, QOL = knee-related quality of life. Possible scores are 0-100, with higher numerical scores indicating fewer symptoms, lower knee pain and greater quality of life.

VAS pain results across the three taping conditions were examined (Table 2.). Analysis of these results showed significant main effect differences between tape conditions. (F (1.887, 22.650) = 12.551, p<0.05). Post hoc analysis showed that the therapeutic tape condition
elicited significantly less pain than the baseline level (p = 0.017), as well as the no tape (p = 0.007) and sham tape (p = 0.026) conditions (Figure 3).

Table 2. VAS pain scores for each taping condition. Large numbers indicate high pain, small numbers indicate low pain.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Baseline</th>
<th>No Tape</th>
<th>Sham Tape</th>
<th>Therapeutic Tape</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score (mm)</td>
<td>16.94(^a)</td>
<td>19.68(^a)</td>
<td>14.57(^a)</td>
<td>4.47(^b)</td>
</tr>
</tbody>
</table>

\(^a,b\) indicate significant difference between conditions

Figure 3. VAS pain scores for each of the taping conditions and baseline measure (* denotes significance).
Table 3. Spatiotemporal variables for the three taping conditions (mean ± SD)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Current Study</th>
<th>*Ebert et al. (2010)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No Tape</td>
<td>Sham Tape</td>
</tr>
<tr>
<td>Stride length (cm)</td>
<td>63.9 ± 5.6</td>
<td>64.2 ± 3.7</td>
</tr>
<tr>
<td>Stride width (cm)</td>
<td>8.9 ± 3.3</td>
<td>8.7 ± 2.6</td>
</tr>
<tr>
<td>Walking velocity (m.s⁻¹)</td>
<td>1.1 ± 0.2</td>
<td>1.2 ± 0.1</td>
</tr>
<tr>
<td>Time in stance (s)</td>
<td>0.7 ± 0.1</td>
<td>0.7 ± 0.1</td>
</tr>
<tr>
<td>Cadence (steps/min)</td>
<td>89.0 ± 9.7</td>
<td>87.3 ± 9.9</td>
</tr>
</tbody>
</table>

*Ebert et al. (2010) was chosen for comparison as testing was done in the same laboratory using the same methods.

No spatiotemporal variables were significantly different between the three taping conditions. A comparison with a previous study showed similar values for measures of stride width and walking velocity.

Knee and hip flexion and adduction angles were analysed across the stance and weight acceptance phases. No significant differences were observed between the taping conditions. Knee flexion was also examined over swing as well as at heel strike, with no differences observed between the taping conditions (Table 4).
Table 4. Kinematic variables across various phases of walking gait (mean ± SD).

<table>
<thead>
<tr>
<th>Phase</th>
<th>Angle (°)</th>
<th>No Tape</th>
<th>Sham Tape</th>
<th>Therapeutic Tape</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stance</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Peak knee flexion</td>
<td>45.6 ± 6.1</td>
<td>47.3 ± 5.9</td>
<td>44.5 ± 9.2</td>
</tr>
<tr>
<td></td>
<td>Minimum knee flexion</td>
<td>10.3 ± 5.7</td>
<td>9.5 ± 6.1</td>
<td>7.9 ± 9.2</td>
</tr>
<tr>
<td></td>
<td>Knee flexion ROM*</td>
<td>35.3 ± 3.2</td>
<td>37.8 ± 5.7</td>
<td>36.6 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>Peak knee adduction</td>
<td>5.0 ± 6.4</td>
<td>5.7 ± 6.2</td>
<td>3.9 ± 7.4</td>
</tr>
<tr>
<td></td>
<td>Peak hip flexion</td>
<td>34.4 ± 4.9</td>
<td>34.6 ± 4.9</td>
<td>33.5 ± 5.6</td>
</tr>
<tr>
<td></td>
<td>Peak hip adduction</td>
<td>4.7 ± 6.6</td>
<td>2.8 ± 6.3</td>
<td>0.7 ± 7.6</td>
</tr>
<tr>
<td><strong>Swing</strong></td>
<td>Peak knee flexion</td>
<td>65.6 ± 5.3</td>
<td>66.2 ± 5.8</td>
<td>66.8 ± 7.1</td>
</tr>
<tr>
<td></td>
<td>Peak knee flexion</td>
<td>20.4 ± 6.0</td>
<td>20.1 ± 7.0</td>
<td>19.8 ± 6.8</td>
</tr>
<tr>
<td><strong>Weight Acceptance</strong></td>
<td>Peak knee adduction</td>
<td>3.7 ± 6.3</td>
<td>5.7 ± 5.9</td>
<td>4.1 ± 7.6</td>
</tr>
<tr>
<td></td>
<td>Peak hip flexion</td>
<td>34.8 ± 4.1</td>
<td>34.4 ± 4.8</td>
<td>33.6 ± 4.8</td>
</tr>
<tr>
<td></td>
<td>Peak hip adduction</td>
<td>4.1 ± 5.4</td>
<td>1.7 ± 4.8</td>
<td>1.4 ± 6.4</td>
</tr>
<tr>
<td><strong>Heel Strike</strong></td>
<td>Knee flexion</td>
<td>12.7 ± 2.5</td>
<td>13.5 ± 3.1</td>
<td>17.8 ± 2.9</td>
</tr>
</tbody>
</table>

*ROM = range of motion

There were no differences observed between any kinetic variables measured during stance, ES or LS. Between group differences for peak knee valgus angles were close to significance (p = 0.059) (Table 5.).
Table 5. Peak normalised knee and hip moments during three phases of walking gait (mean ± SD).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Condition</th>
<th>No Tape</th>
<th>Sham Tape</th>
<th>Therapeutic Tape</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Phase</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak knee flexion</td>
<td></td>
<td>0.52 ± 0.31</td>
<td>0.55 ± 0.28</td>
<td>0.51 ± 0.27</td>
</tr>
<tr>
<td>Min knee flexion</td>
<td></td>
<td>-0.11 ± 0.06</td>
<td>-0.10 ± 0.06</td>
<td>-0.10 ± 0.08</td>
</tr>
<tr>
<td>Mean knee flexion</td>
<td></td>
<td>0.27 ± 0.17</td>
<td>0.27 ± 0.14</td>
<td>0.23 ± 0.18</td>
</tr>
<tr>
<td>Peak knee adduction</td>
<td></td>
<td>0.47 ± 0.21</td>
<td>0.56 ± 0.43</td>
<td>0.53 ± 0.19</td>
</tr>
<tr>
<td>Min knee adduction</td>
<td></td>
<td>-0.07 ± 0.09</td>
<td>-0.04 ± 0.05</td>
<td>-0.02 ± 0.05</td>
</tr>
<tr>
<td>Mean knee adduction</td>
<td></td>
<td>0.34 ± 0.146</td>
<td>0.42 ± 0.3</td>
<td>0.34 ± 0.12</td>
</tr>
<tr>
<td>Peak hip flexion</td>
<td></td>
<td>0.88 ± 0.42</td>
<td>0.69 ± 0.19</td>
<td>0.71 ± 0.21</td>
</tr>
<tr>
<td>Early Stance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak knee flexion</td>
<td></td>
<td>0.49 ± 0.33</td>
<td>0.50 ± 0.41</td>
<td>0.49 ± 0.32</td>
</tr>
<tr>
<td>Min knee flexion</td>
<td></td>
<td>-0.11 ± 0.06</td>
<td>-0.21 ± 0.45</td>
<td>-0.09 ± 0.09</td>
</tr>
<tr>
<td>Mean knee flexion</td>
<td></td>
<td>0.27 ± 0.18</td>
<td>0.26 ± 0.17</td>
<td>0.24 ± 0.21</td>
</tr>
<tr>
<td>Peak knee adduction</td>
<td></td>
<td>0.49 ± 0.19</td>
<td>0.61 ± 0.36</td>
<td>0.53 ± 0.14</td>
</tr>
<tr>
<td>Min knee adduction</td>
<td></td>
<td>-0.07 ± 0.09</td>
<td>0.01 ± 0.07</td>
<td>0.01 ± 0.06</td>
</tr>
<tr>
<td>Mean knee adduction</td>
<td></td>
<td>0.37 ± 0.16</td>
<td>0.26 ± 0.02</td>
<td>0.34 ± 0.11</td>
</tr>
<tr>
<td>Peak hip flexion</td>
<td></td>
<td>0.60 ± 0.16</td>
<td>0.48 ± 0.23</td>
<td>0.51 ± 0.20</td>
</tr>
<tr>
<td>Late Stance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak knee flexion</td>
<td></td>
<td>0.28 ± 0.32</td>
<td>0.37 ± 0.16</td>
<td>0.35 ± 0.6</td>
</tr>
<tr>
<td>Min knee flexion</td>
<td></td>
<td>-0.07 ± 0.09</td>
<td>-0.15 ± 0.42</td>
<td>-0.06 ± 0.09</td>
</tr>
<tr>
<td>Mean knee flexion</td>
<td></td>
<td>0.14 ± 0.16</td>
<td>0.16 ± 0.12</td>
<td>0.15 ± 0.16</td>
</tr>
<tr>
<td>Peak knee adduction</td>
<td></td>
<td>0.43 ± 0.17</td>
<td>0.56 ± 0.22</td>
<td>0.41 ± 0.16</td>
</tr>
<tr>
<td>Min knee adduction</td>
<td></td>
<td>-0.03 ± 0.07</td>
<td>-0.03 ± 0.05</td>
<td>-0.03 ± 0.03</td>
</tr>
<tr>
<td>Mean knee adduction</td>
<td></td>
<td>0.34 ± 0.16</td>
<td>0.41 ± 0.31</td>
<td>0.34 ± 0.15</td>
</tr>
<tr>
<td>Peak hip flexion</td>
<td></td>
<td>-0.05 ± 0.13</td>
<td>-0.02 ± 0.18</td>
<td>-0.04 ± 0.10</td>
</tr>
</tbody>
</table>
Additionally, participants were divided into two groups – ‘pain’ and ‘no-pain’ as defined by a VAS score reduction of greater than 10 mm. Subsequent analysis of the same variable showed no significant differences. Interested readers can see these data and the analyses, which are presented in Appendix A.

Figure 4. Percentage contribution of total support moment at three lower limb joints during the first half of stance.

The percentage contribution of each lower limb joint was calculated across the stance phase for each of the taping conditions (Figure 4.). The sham tape condition showed greater contribution from the knee; however statistical analyses were not conducted for these measures.

Continuous data were examined and observations were made between the three conditions. Figure 5 shows a typical example of the ankle, knee and hip flexion moments as well as support moments. There were no noticeable differences between the three taping conditions; however statistical analyses were not conducted for this continuous data.
Figure 5. The support moment for a typical OA participant. \( M_S \) is the total support moment, \( M_A \) is ankle flexion moment, \( M_H \) is hip flexion moment and \( M_K \) is knee flexion moment. Dotted lines show the mean ± the standard deviation.
5.0 Discussion

This paper sought to examine the effect of therapeutic knee taping on knee pain, as well as spatiotemporal, kinetic and kinematic variables among knee OA populations (type II and III) during walking gait. Results showed that therapeutic knee taping significantly reduced knee pain during walking gait. No changes in spatiotemporal, kinetic or kinematic variables were observed immediately following therapeutic knee taping during walking gait.

Self-reported pain scores from the VAS were compared between the three taping conditions and it was shown that the therapeutic knee tape significantly lowered knee pain during walking gait ($\alpha = 0.05$). Post-hoc analysis demonstrated that the pain scores during the therapeutic taping condition were significantly lower than both the sham tape and the no tape conditions. These pain results support our hypothesis that therapeutic knee taping would reduce knee pain during walking gait in people who suffer from OA.

Furthermore, nine (88%) of the participants elected to leave the lab with the therapeutic taping, reporting reductions in knee pain for up to three days. Six participants continued to use this therapeutic knee taping as a treatment option to manage their OA related knee pain. These 6 participants were contacted by phone six months after testing and each one reported sustained reductions in knee pain, which was said to have improve their health related quality of life and allowed them to exercise more regularly. These anecdotal findings provide a rationale for future research to investigate the long term neuromusculoskeletal benefits of therapeutic knee taping among knee OA populations.

Returning to the results presented in this study, the significant reductions in OA related pain are extremely positive from a clinical perspective, and can have potentially broad applications. Specifically, reducing a patient’s knee pain will afford patients the
opportunity to become more active and exercise more regularly, which may increase the probability of reducing body mass (BMI), which is the single largest predictor of OA initiation and disease progression (Manninen, 1996).

These OA related knee pain findings align with current literature that suggests that therapeutic knee taping can reduce knee pain during daily activities among patients with knee OA (Hinman et al., 2003). Therapeutic taping has the potential to be used as an inexpensive, easy and effective treatment for OA patients who suffer from knee pain. As therapeutic taping can be self-applied, it has the added benefit of being a long term treatment option for OA patients managing their OA related knee pain. An added benefit to therapeutic taping as a knee pain treatment option is that patients can reduce their reliance on prescribed pain medications, which is particularly relevant to elderly populations with co-existing health problems e.g. heart disease, type II diabetes, cancer.

Further benefits associated with reduced knee pain when undertaking daily activities include the delaying of TKA surgery. This is an important consideration as it is often reported in the literature that most patients require a revision surgery within 10-15 years of their first surgery (Waldorff et al., 2012). If knee pain can be managed by therapeutic taping and the first TKA delayed, such that only one surgery is required in a patient’s lifetime, the annual healthcare cost savings would be substantial. In 2012, there were 48,502 TKA surgeries reported in Australia (National Joint Replacement Registry) with 4026 of these (8.3%) consisting of secondary replacements on the same joint. At an average cost of $24,347, the ability to delay primary TKA such that secondary surgery is unnecessary has the potential to save upwards of $98 million annually.
Results of the KOOS showed that participants had low health related quality of life and had many symptoms of OA and regular pain during daily activities. These results align with previous research that has shown that OA populations have limited abilities to perform functional tasks due to elevated levels of pain (Ebert et al., 2010). Examination of the spatiotemporal variables showed no differences in any measures across the three taping conditions.

None of the kinematic and kinetic variables that were analysed in this study changed significantly between the taping conditions. When the kinematic and kinetic data were analysed to see if the patients who presented with knee pain at the time of testing were affected differently by the therapeutic taping, results were inconclusive (Appendix A). These results suggest that reduction in knee pain is not a mediator for changes in an individual’s lower limb biomechanics following the acute application of therapeutic taping.

During testing sessions, it was evident that each participant who reported OA related knee pain when conducting the activities of daily living possessed unique, patient specific walking gait strategies to avoid pain during the testing protocol (walking, stair ascent/descent and sit to stand). Under each taping condition, irrespective of a patient’s self-reported knee pain score, they continued to use these subject-specific walking gait strategies. One plausible reason may be because these walking gait strategies have been learned following years of knee pain and/or the fear of knee pain when conducting the activities of daily living. Additionally, it is possible that a walking velocity of 1.2m.s$^{-1}$ may not have been sufficiently demanding to observe significant changes in their lower limb biomechanics following the acute application of therapeutic knee tape. There may be scope for future research to investigate the long term effects of therapeutic knee taping to see if, when allowed to walk without knee pain for long periods of time, patients would adopt a
more normative spatiotemporal, kinematic and kinetic walking gait pattern (Heiden al., 2009).

Support moments were calculated as a percentage contribution of each joint (ankle, knee, hip) during walking gait, with no differences observed between taping conditions. Time varying data were presented and these showed little variation between the taping conditions. However, following therapeutic knee taping, an interesting trend was observed with participants tending to use their knee to a greater extent when generating a support moment during the first half of stance. This suggests that future neuromusculoskeletal adaptations may occur if therapeutic taping was applied for longer periods of time. Again, this would need to be verified with future research.

One limitation of these findings is the use of external knee joint moments and knee kinematics as surrogate measures of knee joint condylar loading. It is also possible that mechanical changes would be observed following the application of therapeutic knee taping if the participants were asked to conduct a more demanding task during testing. A further limitation of this study was that neuromuscular changes were not reported following each of the taping conditions. EMG data were recorded during data collection, but is currently undergoing analysis. Recent strength training studies among OA populations (Heiden et al., 2009) have shown changes in co-contraction ratios and muscle activation strategies between OA patients and healthy individuals. Further investigation into the effect of therapeutic knee taping for knee OA and neuromuscular activation is recommended for future research.

There are many clinical applications of therapeutic knee taping resulting from the positive findings of this study. A reduction in self-reported knee pain during activities of daily
living shows therapeutic knee taping may provide an effective, non-invasive and inexpensive treatment to manage OA related knee pain. Anecdotal evidence suggests that there is potential for therapeutic knee taping as a long term treatment option for patients to manage their OA related pain and increase the quality of life knee OA populations worldwide.
6.0 Conclusion

The acute application of therapeutic knee taping was effective in reducing OA related knee pain during walking gait. Therefore we can accept the first hypothesis that therapeutic knee taping can reduce self-reported knee pain during walking gait. Stride length and velocity did not change during walking gait under the therapeutic taping condition meaning the second hypothesis was not confirmed. Additionally, knee adduction and flexion moments did not change during the stance phase of walking gait meaning the third hypothesis must be rejected. Knee joint kinematics were not different between tape conditions meaning the fourth hypothesis was not confirmed. The acute application of therapeutic knee taping was not effective in altering an OA population’s lower limb kinematics and kinetics during walking gait. It is recommended that an intervention study investigate the effects of therapeutic knee taping over a prolonged period of time on knee kinetics and kinematics during walking gait.
7.0 References


Joss, B.K., Clinical and biomechanical outcomes following unicompartmental knee arthroplasty with preservation fixed and mobile bearing tibial components, thesis. 2006. University of Western Australia, Perth.


8.0 Appendix

8.1 Appendix A

Table 6. Kinetic and Kinematic data for the pain only group.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase</td>
</tr>
<tr>
<td></td>
<td>Stance</td>
</tr>
<tr>
<td></td>
<td>Early Stance</td>
</tr>
<tr>
<td></td>
<td>Late Stance</td>
</tr>
<tr>
<td></td>
<td>No Tape</td>
</tr>
<tr>
<td></td>
<td>Sham Tape</td>
</tr>
<tr>
<td></td>
<td>Therapeutic Tape</td>
</tr>
<tr>
<td>Peak knee flexion moment</td>
<td>0.48 ± 0.20</td>
</tr>
<tr>
<td>Peak adduction moment</td>
<td>0.39 ± 0.20</td>
</tr>
<tr>
<td>Peak abduction moment</td>
<td>-0.08 ± 0.06</td>
</tr>
<tr>
<td>Peak adduction angle</td>
<td>6.15 ± 5.15</td>
</tr>
<tr>
<td>Peak abduction angle</td>
<td>-5.17 ± 6.65</td>
</tr>
<tr>
<td>Peak knee flexion angle</td>
<td>43.28 ± 4.80</td>
</tr>
<tr>
<td>Peak ankle flexion moment</td>
<td>0.56 ± 0.16</td>
</tr>
<tr>
<td>Peak knee flexion moment</td>
<td>0.46 ± 0.24</td>
</tr>
<tr>
<td>Peak hip flexion moment</td>
<td>0.57 ± 0.16</td>
</tr>
<tr>
<td>Peak ankle flexion moment</td>
<td>1.09 ± 0.21</td>
</tr>
<tr>
<td>Peak knee flexion moment</td>
<td>0.31 ± 0.15</td>
</tr>
<tr>
<td>Peak hip flexion moment</td>
<td>-0.02 ± 0.13</td>
</tr>
</tbody>
</table>
Knee Pain Survey (Tape A, Trial A)

To answer each question place a vertical dash [ | ] through the corresponding line

<table>
<thead>
<tr>
<th>Front (Anterior)</th>
<th>No Discomfort</th>
<th>Extreme Discomfort</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Knee Cap (Top)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Knee Cap (Bottom)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Lateral Side of Knee</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Medial Side of Knee</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Middle of Knee</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Back (Posterior)

6. Left Side of Knee

7. Right Side of Knee

The number displayed in the regions in the diagram to the left correspond with the numbers in the survey above.
Knee Pain Survey (Tape A, Trial A)

To answer each question place a vertical dash [ | ] through the corresponding line.

The number displayed in the regions in the diagram above correspond with the numbers in the survey to the right.

<table>
<thead>
<tr>
<th>No Discomfort</th>
<th>Extreme Discomfort</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. Lateral inner knee</td>
<td></td>
</tr>
<tr>
<td>9. Posterior (Back) inner knee</td>
<td></td>
</tr>
<tr>
<td>10. Medial inner knee</td>
<td></td>
</tr>
<tr>
<td>11. Anterior (front) inner knee</td>
<td></td>
</tr>
</tbody>
</table>
8.3 Appendix C
Examination of the kinetic data for the pain group showed there were no significant differences between the taping conditions in the pain group for flexion moment during stance (p = 0.321). Analysis of peak adduction moment showed no difference between the taping conditions (p = 0.421). Abduction moment was also not found to be significantly different (p = 0.393). Kinetic data for the first 50% of the stance phase (early stance) was not significantly different between the taping conditions for ankle (p = 0.440), knee (p = 0.386) or hip (p = 0.417) moments. Similarly, during the last 50% of the stance phase (late stance), no significant differences were noted between the taping conditions for ankle (p = 0.176), knee (p = 0.333) and hip (p = 0.318) moments.

Kinematic data for the pain group showed no significant differences between the taping conditions for peak abduction (p = 0.701), peak adduction (p = 0.396) and peak knee flexion angle (p = 0.426) during the stance phase.

Figure 6. A comparison of knee flexion moment during stance between all participants and those in the ‘pain only’ group.
8.4 Appendix D

Our Ref: RA/4/1/6088

30 April 2013

Dr Cyril Donnelly
School of Sport Science, Exercise & Health
MBDP: M408

Dear Doctor Donnelly

HUMAN RESEARCH ETHICS APPROVAL - THE UNIVERSITY OF WESTERN AUSTRALIA

The effects of clinical taping techniques on the lower limb biomechanics and subjective assessment of pain in knee osteoarthritis populations

Student(s):

Ethics approval for the above project has been granted in accordance with the requirements of the National Statement on Ethical Conduct in Human Research (National Statement) and the policies and procedures of The University of Western Australia. Please note that the period of ethics approval for this project is five (5) years from the date of this notification. However, ethics approval is conditional upon the submission of satisfactory progress reports by the designated renewal date. Therefore initial approval has been granted from 20 April 2013 to 01 May 2018.

You are reminded of the following requirements:

1. The application and all supporting documentation form the basis of the ethics approval and you must not depart from the research protocol that has been approved.
2. The Human Research Ethics Office must be approached for approval in advance for any requested amendments to the approved research protocol.
3. The Chief Investigator is required to report immediately to the Human Research Ethics Office any adverse or unexpected event or any other event that may impact on the ethics approval for the project.
4. The Chief Investigator must inform the Human Research Ethics Office as soon as practicable if a research project is discontinued before the expected date of completion, providing reasons.

Any conditions of ethics approval that have been imposed are listed below:

Special Conditions

None specified

The University of Western Australia is bound by the National Statement to monitor the progress of all approved projects until completion to ensure continued compliance with ethical standards and requirements.

The Human Research Ethics Office will forward a request for a Progress Report approximately 60 days before the due date. A further reminder will be forwarded approximately 30 days before the due date.

If your progress report is not received by the due date for renewal of ethics approval, your ethics approval will expire, requiring that all research activities involving human participants cease immediately.

If you have any queries please contact the HREO at vbro-researchs@uwa.edu.au.

Please ensure that you quote the file reference — RA/4/1/6088 — and the associated project title in all future correspondence.

Yours sincerely

Peter Johnstone
Manager, Human Research Ethics Office