



## Knee joint kinematics, kinetics and muscle co-contraction in knee osteoarthritis patient gait

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### ABSTRACT

**Background:** Compared to matched controls, knee osteoarthritis patients walk with altered, kinematics, kinetics and muscle activity. Studies of osteoarthritis patient gait have focused on individual measures, and findings from these studies differ due to differences in patient levels of disability and age. Therefore, aims of this study were to examine kinematic, kinetic and muscle co-contraction gait variables within a single osteoarthritis patient group, and to determine if alterations in these variables are related to pain, symptom and function measures.

**Methods:** Thirty asymptomatic controls and 54 patients with radiographic evidence of knee osteoarthritis participated. Self-perceived measures of pain and symptoms, and gait (knee joint angles, moments and muscle co-contraction) were analysed and compared.

**Findings:** Osteoarthritis patients had greater self-perceived pain and symptoms on the questionnaires. Gait differences in the knee osteoarthritis patients were greater knee flexion at heel strike and during early stance along with reductions in the peak external knee extension moment in late stance. Co-contraction ratios highlighted greater lateral muscle activation in osteoarthritis patients, which were correlated with the magnitude of their adduction moments. Larger adduction moments were related to lower self-perceived pain and symptoms.

**Interpretation:** Osteoarthritis patients use predominantly lateral muscle activation during stance which may aid in stabilising the external knee adduction moment. Kinematic alterations in knee osteoarthritis patient gait occur without alterations in knee joint moments. Our results also suggest that adduction moments are lowered to reduce the patients' pain and symptoms.

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### 1. Introduction

Osteoarthritis (OA) is the most common rheumatic disease and the knee is the most often affected weight bearing joint (Martin, 1994). Knee OA causes low quality of life and functioning in activities of daily living, with increased pain, decreased muscle mass, proprioception deficits, and altered gait mechanics. In regard to gait, compared to matched controls, knee OA patients have reductions in walking speed (Astefhen and Deluzio, 2005; Brinkmann and Perry, 1985) and cadence (Chen et al., 2003; Stauffer et al., 1977), longer double support time (Chen et al., 2003; Smith et al., 2004), a smaller stride length (Baliunas et al., 2002), increased knee flexion at heel strike (Childs et al., 2004; Mundermann et al., 2005), and reduced knee flexion during the stance

phase of gait (Astefhen and Deluzio, 2005; Baliunas et al., 2002). They also walk with reduced heel strike forces on the affected limb (Stauffer et al., 1977), lower external knee flexion moments during early stance (Kaufman et al., 2001), lower external knee extension moments in terminal stance (Smith et al., 2004), and increased external knee adduction moments in stance (Astefhen and Deluzio, 2005). This is accompanied by increased hamstring activation (Hortobagyi et al., 2005), prolonged muscle activation in stance (Childs et al., 2004), and increased co-contraction (Schmitt and Rudolph, 2007).

Wide variability exists in the magnitude of impairment in walking speed, knee kinematics, and knee kinetics between OA and control groups in the aforementioned studies. This variability may be attributed to differences between the studies in age, knee alignment, disease severity, or walking speed. With increasing age comes altered neuromuscular function and reductions in walking speed (Rudolph et al., 2007). Reductions in gait speed are a function of both age and the disease. Healthy subjects walk at faster preferred gait speeds than those with knee OA (Hanlon and Anderson, 2006) and many gait variables change with walking speed (Andriacchi et al., 1977). Specifically, ground reaction forces

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(Andriacchi et al., 1977), peak knee flexion and sagittal plane knee joint moments (Lelas et al., 2003) have been shown to rise with increasing gait speed, and gait speed is a reflection of a patient's clinical state (Andriacchi et al., 1977; Brinkmann and Perry, 1985). With increasing OA disease severity there are alterations in the external knee adduction moment (Mundermann et al., 2004), knee flexion angle, and gastrocnemius muscle activity (Astefan et al., 2007) during gait. Given these differences, the selection of similarly aged OA patients and controls, and the control of confounding factors should be considered when examining gait alterations in knee OA patients.

Inclusion of patient self-perceived pain, symptoms and function are important due to their direct links to functioning in activities of daily living (Maly et al., 2006). The relationship between structural changes and symptoms in knee OA patients is small (Dieppe, 2004). However, the development of increased pain is associated with increased external adduction moments in the gait of knee OA patients (Miyazaki et al., 2002) and these adduction moments in turn are associated with increased radiographic severity (Sharma et al., 1998). Pain and symptoms may also be related to muscle activation patterns although this is yet to be tested.

There are only a few studies of knee OA patients compared with asymptomatic, similarly aged controls, that have comprehensively examined knee joint kinematics, kinetics and muscle activation, whilst controlling walking speed and age of comparison group (Lewek et al., 2004, 2006; Messier et al., 2005; Schmitt and Rudolph, 2007). Importantly, the findings of these studies varied, most likely due to differences in the OA patient populations examined. For example, Lewek et al. (2004, 2006) studied ten OA patients, 49 years old, with medial compartment OA and genu varum, while Messier et al. (2005) examined ten OA patients, 74 years old, with varying OA severities in all three knee compartments. Schmitt and Rudolph (2007) studied a larger cohort (28 patients), 60 years old, and OA confined to the medial compartment. So one must control for confounding factors such as walking speed and assess the level of pain and function if we are to examine OA gait and the relationships between gait kinematics, kinetics and muscle activation patterns.

Co-contraction of the knee muscles provides a means to alter the stability and articular loading of the joint (Hubley-Kozey et al., 2008). Additionally and as previously mentioned, knee OA patients may walk with increased co-contraction of the knee muscles. In general, there are two forms of knee muscle co-contraction: generalised co-contraction and directed co-contraction (Lloyd and Buchanan, 2001). In generalised co-contraction all agonists and antagonists of the knee co-activate equally, whereas in directed co-contraction medial agonists and antagonists are activated to support abduction moments and lateral muscles for adduction moments. Directed co-contraction is believed to directly support the external moment to prevent condylar lift-off and reduce the concentration of articular loading in the medial knee compartment (Schipplein and Andriacchi, 1991). Generalised co-contraction can also have this effect but because of the non-directionality it is less effective in preventing condylar lift-off, and may unduly increase all articular loading (Andriacchi et al., 1984; Lloyd and Buchanan, 2001; Zhang et al., 2001). Generalised co-contraction has been identified when people were supporting the isometric adduction/abductions knee moments (Lloyd and Buchanan, 2001; Zhang et al., 2001), and during sidestepping and cross-over cutting (Besier et al., 2003a,b). Directed co-contraction has been demonstrated in ligamento-muscular reflexes to resist adduction/abduction perturbations at the knee (Buchanan et al., 1996), and voluntary directed co-contraction has been shown to the support static knee abduction-adduction moments (Andriacchi et al., 1984; Zhang et al., 2001), and abduction moments at the knee during side stepping (Besier et al., 2003a,b). Studies examining muscle co-contraction in knee OA patients have found increased levels of medial muscle co-

contraction (Lewek et al., 2004, 2006) and lateral muscle co-contraction (Schmitt and Rudolph, 2007) in the OA patients compared with control subjects. However, these studies did not compare the medial versus lateral muscle co-activations, i.e. directed co-contraction. Recently, greater lateral relative to medial hamstring activation during gait has been shown in both severe (Hubley-Kozey et al., 2008) and moderate OA patients (Hubley-Kozey et al., 2006), possibly in an attempt to control medial joint articular loading. However, these investigations did not control for walking speed or age of the comparison group (Hubley-Kozey et al., 2006), or did not use a comparison group (Hubley-Kozey et al., 2008). Therefore it remains unclear if this pattern is distinctive to knee OA patient gait. It is also possible that knee OA patients may increase their directed co-contraction of the lateral muscles to support the increased external knee adduction moment commonly reported in knee OA patients. Finally, directed co-contraction may be related to the self-perceived pain and symptoms experienced by knee OA patients.

Consequently, the aims of this study were; (1) to determine if the knee joint kinematic and kinetic variables, previously determined to be altered in knee OA patient gait, all occur within the same OA patients, (2) determine levels of net muscle activation in knee OA patient gait and examine two directed co-contraction ratios; (a) medial and lateral quadriceps, hamstrings and gastrocnemius muscles and (b) medial and lateral hamstring muscles, and (3) examine the relationships between self-perceived pain, symptoms and physical function with gait kinematics, kinetics, and muscle directed co-contraction.

## 2. Methods

Thirty asymptomatic control subjects (19 females) and 54 physician-diagnosed knee OA patients (30 females) were recruited. Public advertisements were used for the recruitment of controls whilst OA patients were recruited through both local orthopedic outpatient clinics and advertisements. All OA patients had radiographic signs of knee OA, BMI <35, experienced morning knee stiffness <30 min, knee varus/valgus alignment  $\leq 5^\circ$ , could walk unassisted, and had not received steroid injections in the past 6 months, or regular physiotherapy in the last 12 months. Inclusion in the program for both OA patients and controls was restricted to those aged between 50 and 80. Exclusion criteria were neurological and/or cardiovascular disorders, surgery or injury to the back or lower limbs in the past 2 years, and any form of inflammatory arthritis (e.g., rheumatoid or psoriatic). All procedures were approved by the University of Western Australia Human Research Ethics Committee and all participants gave their informed, written consent prior to being enlisted in the study.

The Knee Osteoarthritis Outcome Survey (KOOS) (Roos et al., 1998) and the Medical Outcomes Study 36-item short form health survey (SF-36) (Ware and Sherbourne, 1992) questionnaires were administered to all participants prior to testing. The KOOS is a 42 item disease-specific instrument with subscales of pain, other symptoms, function in daily living (ADL), sport and recreation (Sport/Rec) and knee related quality of life (QOL) (Roos and Lohmander, 2003). Use of the KOOS in a knee OA population has been found to be a valid and reliable tool (Roos and Toksvig-Larsen, 2003). Due to the age and current activity status of the OA patients, however, the subscale Sport/Rec was given the additional selection option of "not applicable" (N/A) (Roos and Lohmander, 2003; Roos and Toksvig-Larsen, 2003). KOOS subscales are scored from 0 to 100 with 0 indicating extreme problems and 100 indicating no problems.

The SF-36 assesses general health and comprises a physical component score (PCS) and a mental component score (MCS). PCS and MCS scores were developed using norm based methods

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