

## A comparison of gait patterns between the offspring of people with medial tibiofemoral osteoarthritis and normal controls

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### ABSTRACT

**Objectives.** *The aim of this study was to explore the contribution of biomechanical factors to the development and progression of knee osteoarthritis (OA) by investigating whether the offspring of subjects with medial tibiofemoral OA demonstrate gait abnormalities in the absence of OA.*

**Methods.** *Three-dimensional gait analyses were performed on 9 offspring of people with medial tibiofemoral OA and 9 age, gender and Body Mass Index (BMI) matched individuals with no parental history of knee OA. External knee adduction, extension and flexion moments, as well as the magnitude of foot rotation during early stance were compared between the groups.*

**Results.** *The offspring of people with medial tibiofemoral OA walked with less external rotation at the foot than control subjects during early stance (4.5° versus 13.5°,  $p < 0.01$ ). There were no significant differences between groups for the peak knee adduction moments (dominant leg,  $p = 0.49$ ; non-dominant leg,  $p = 0.70$ ) or peak knee extension moments (dominant leg,  $p = 0.46$ ; non-dominant leg,  $p = 0.48$ ). Moreover, there was no difference between groups for the knee flexion moment occurring when the force adducting the knee was greatest (dominant leg,  $p = 0.35$ ; non-dominant leg,  $p = 0.33$ ).*

**Conclusions.** *Although the offspring of people with medial tibiofemoral OA walked with less external foot rotation than the control subjects during early stance, whether this increases their risk of developing knee OA is yet to be determined.*

### Introduction

Although osteoarthritis (OA) is a common cause of disability in people over 65 years (1), the factors that predate the development and progression of OA are not clear. In addition to biological studies, the contribution of biomechanical variables toward this disease is of growing interest (2-8). Increased regional load across articular cartilage is argued to be an important factor involved in the pathogenesis of knee OA (2) and people with medial tibiofemoral OA

walk with larger than normal knee adduction moments (3, 4), resulting in increased medial tibiofemoral compartment pressure. A recent longitudinal study found that increased knee adduction moments were associated with the progression of medial tibiofemoral OA (8).

Studies investigating the contribution of biomechanical variables to the pathogenesis of knee OA have mainly examined subjects with well established OA. Despite the identification of several gait abnormalities (3, 4, 6-8), it is not clear whether these disturbances are a cause or a result of disease. Whereas some investigators argue that abnormal biomechanics are associated with the progression of OA (8), it is possible that biomechanical abnormalities may predate disease onset. The contribution of mechanical factors toward the development and progression of knee OA remains unclear.

A strong genetic predisposition toward knee OA is well established (9). However, the mechanism accounting for the expression of the genetic component remains unclear. While radiographic OA changes have been genetically linked (10), there may be inherited mechanisms such as biomechanical abnormalities during gait that contribute to the risk of OA. To explore this possibility, the gait patterns of healthy subjects who had at least one parent with medial tibiofemoral OA were compared with healthy age, body mass index, and gender matched subjects with no parental history of knee OA.

### Methods

#### Subjects

Nine healthy offspring of subjects with primary medial tibiofemoral OA (Kellgren-Lawrence (K-L) grades 2) who were partaking in other studies within our department were invited to participate in this study. These subjects were aged 20-50 years. The control group comprised nine healthy age ( $\pm 3$  years), gender and Body Mass Index ( $\pm 3$  kg/m<sup>2</sup>) matched subjects with no parental history of knee OA recruited from staff at our institution. Exclusion criteria in both groups were as follows: a history of knee OA or symptoms requiring

medical treatment; heavy work occupations (e.g. brick layer); current use of analgesic or anti-inflammatory medications and any neurological, cardiovascular or orthopaedic conditions affecting gait. The subject's preferred kicking leg was nominated as their dominant leg.

#### Apparatus and procedure

Testing was conducted in the gait laboratory in the Musculoskeletal Research Center, La Trobe University, Australia. A six-camera Vicon motion analysis system (Oxford Metrics Ltd., Oxford, UK) was used to capture three-dimensional kinematic data during four walking trials on each leg. Ground reaction forces were measured by a Kistler 9281 force-platform (Kistler Instruments, Winterthur, Switzerland). Inverse dynamic analyses were performed using "PlugInGait" modeler (Oxford Metrics, Oxford, UK) to obtain joint moments around the coronal, sagittal and longitudinal joint axes.

#### Statistical analysis

The data was initially examined for normality and found to conform to a normal distribution. The baseline characteristics and mean differences in the biomechanical parameters between the groups were assessed using independent t-tests. Comparisons between groups were made before and after adjustment for baseline differences. A Pearson correlation was used to exam-

ine the relationship between the peak knee adduction moment and the corresponding magnitude of foot rotation. Positive values at the foot represent internal rotation. The dominant leg and non-dominant leg were treated as separate entities since combining the right and left leg fails to acknowledge the independence between knees. Results where there were p-values of less than 0.05 (two-tailed) were considered to be statistically significant. All analyses were performed using SPSS (version 10.0.5, SPSS, Cary, NC).

#### Results

##### Subjects

There were 6 females and 3 males in each group. No significant differences between groups existed for the mean age (offspring  $39.4 \pm 9.0$  years, control group  $40.6 \pm 9.9$  years;  $p = 0.60$ ) or BMI (offspring  $24.7 \pm 4.0$  kg/m<sup>2</sup>, control group  $22.7 \pm 3.4$  kg/m<sup>2</sup>;  $p = 0.60$ ).

##### Lower limb biomechanics

The biomechanical results for the offspring and control groups are presented in Table I. There were no significant differences in the mean magnitudes of the knee moments between the offspring of subjects with medial tibiofemoral OA and comparison subjects. This did not change when the moments were normalised for body weight (BW) and height (Ht) (% BW x Ht). There was, however, a statistically significant difference between the

groups for foot rotation occurring when the force adducting the knee was maximal for the dominant leg. The same comparison for the non-dominant leg indicated a similar trend, but was not statistically significant (see Table I).

To determine whether an inherent biomechanical relationship existed between the magnitude of the knee adduction moment and the corresponding degree of foot rotation, the data from the offspring and control subjects were combined. A moderate positive linear correlation was observed between the mean peak knee adduction moment and the degree of external foot rotation for the dominant leg during early stance ( $r = 0.44$ ,  $p = 0.07$ ). Little or no relationship was observed between the mean peak knee adduction moment and the degree of foot rotation on the non-dominant side during early stance ( $r = 0.064$ ,  $p = 0.80$ ).

#### Discussion

To our knowledge, this is the first study comparing the gait patterns between the healthy offspring of people with well-defined medial tibiofemoral OA and normal controls. Previous studies have predominantly examined subjects with established OA (3, 4, 6-8) and demonstrated that people with knee OA walk with larger than normal peak knee adduction moments (3, 4). Although increased knee adduction moments are associated with the radiological progression of established OA (8), there is no evidence to suggest whether this knee adduction moment contributes to the development of medial tibiofemoral OA. Provided that the genetic predisposition to knee OA is substantiated by the offspring of people with medial tibiofemoral OA developing disease, our results suggest that the larger than normal knee adduction moments in people with OA may be a consequence of the OA, rather than a precursor of disease. Furthermore, the degree of foot rotation adopted during walking may influence the risk of developing medial tibiofemoral OA.

The offspring of subjects with medial tibiofemoral OA walked with less external foot rotation during early stance than the control group. Previous studies

**Table I.** Comparison of biomechanical variables in the offspring and control subjects.

	Dominant leg			Non-dominant leg		
	Offspring group*	Control group*	P	Offspring group*	Control group*	P
Peak adduction moment	4.0 (1.0)	4.5 (1.6)	0.49	4.0 (1.3)	4.2 (1.2)	0.70
Flexion moment occurring at time of peak knee adduction moment	2.6 (1.5)	3.3 (1.6)	0.35	2.2 (1.5)	2.8 (1.1)	0.33
Peak extension moment	2.6 (0.9)	2.9 (1.0)	0.46	3.0 (1.3)	2.6 (0.9)	0.48
Foot rotation occurring at time of peak knee adduction moment (degrees)	4.5 (6.9)	13.5 (6.2)	0.01	3.0 (3.4)	9.2 (8.7)	0.06

\* Mean (standard deviation); moments are expressed as a percentage of body weight multiplied by height (% BW x Ht).

examining subjects with knee OA showed that symptomatic individuals walk with a larger degree of toe-out than non-diseased people (5). It has been suggested that people with knee OA may adopt a compensatory toe-out gait pattern to reduce the pain that arises from repeated medial tibiofemoral joint compression (5). Toe-out alignment of the lower limb directs the ground reaction force toward the center of the knee joint, thereby reducing the lever arm of the moment that adducts the knee and causes medial tibiofemoral compression and pain (5). However if toe-out accounts for some of the variability in the knee adduction moment, it is possible that walking with limited external rotation at the foot may also influence the peak knee adduction moment. The relationship between the joint kinematic variables that contribute to a toe-out gait pattern and therefore knee adduction moment variability requires further clarification.

While this study found that offspring walked with less external foot rotation than control subjects during early stance, a clear relationship between the magnitude of the knee adduction moment and the corresponding degree of foot rotation could not be confirmed. Foot rotation correlated with the knee adduction moment for the dominant leg during early stance, although this relationship was not apparent on the non-dominant side. No previous study has examined the relationship between the knee adduction moment and the degree of foot rotation. However the toe-out angle, which may be reliant upon foot rotation, has been shown to predict the magnitude of the knee adduction moment during late stance but not during early stance (7, 11). These observations infer the likelihood of a complex spatial and temporal interaction between lower limb joint kinematics and knee kinetics during walking. Whether foot rotation influences the toe-out phenomenon and therefore knee adduction moment variability during late stance is not clear.

A strong genetic predisposition toward knee OA is now well established, with 39-65% of the variance in OA at the

hand and knee being attributed to genetic factors (9). The mechanism accounting for the expression of the genetic contribution toward OA is however largely unknown. One study has identified genes that are associated with radiographic knee OA (10). However, the expression of the genetic contribution toward knee OA may be multifactorial and include biomechanical risk factors. The reduced foot rotation during early stance may explain some of the increased risk of disease in the offspring of people with knee OA.

The findings of this study are limited by a relatively small sample size. Future investigations will need to examine a larger number of people, randomly selected from a non-convenient sample. Moreover, because subjects were not radiographed, there is no certainty that people with OA were excluded from this study. Between-group differences for foot rotation may have been the result of early OA in the offspring, rather than an inherent biomechanical difference. However, this is unlikely given that the cohort was younger than 50 and experienced no knee pain, which does not comply with the ACR's criteria for diagnosing clinical OA (knee pain and at least three of the following: age > 50 years, stiffness, crepitus, bony tenderness, enlargement and warmth) (12). Furthermore, this study is limited by the uncertainty surrounding whether all offspring will develop OA. Our results will need to be validated by a follow-up study examining the same cohort to determine whether the genetic risk for developing knee OA was substantiated.

This study has described differences between the gait patterns in the healthy offspring of subjects with knee OA and normal controls. Whether these biomechanical differences reflect part of the genetic predisposition toward OA and therefore contribute to the risk of developing knee OA, will need to be determined by longitudinal studies.

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