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# AMBULATORY KINEMATICS CORRELATES WITH FUTURE DISEASE PROGRESSION IN MEDIAL OSTEOARTHRITIC KNEES

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## INTRODUCTION

Knee osteoarthritis (OA) is a degenerative joint disease estimated to affect about 50% of the US population 85 and older [1]. Although this disease is recognized as the major source of physical disability in the elderly, its etiology is still incompletely understood. Early detection is particularly challenging as OA often remains symptomatically silent until an advanced stage. Yet information on early detection is needed to develop new prevention and treatment strategies.

When looking for early markers for this complex disease, it is beneficial to consider the framework of Andriacchi et al. [2] which proposes a multifactorial model for the initiation and progression of knee OA. In the context of this framework, kinematic changes during ambulation are thought to contribute to the degeneration by shifting tibiofemoral contact to regions of cartilage that cannot accommodate the new mechanical conditions. While this mechanical pathway has been well-described for post-traumatic knee OA [3], there is a paucity of information for idiopathic OA occurring in older individuals who did not sustain any major knee injury.

The results of a recent cross-sectional study reporting higher flexion angle and more anterior translation of the femur relative to the tibia at heel-strike of walking with increasing age and OA severity [4] strongly suggest that these two kinematic measures could be elements of a mechanical pathway to idiopathic knee OA. Taken together with other studies showing that cartilage morphology is conditioned to heel-strike kinematics in asymptomatic subjects [5, 6], there is substantial evidence to motivate a longitudinal study of the heel-strike flexion angle and anterior femoral translation relative to OA progression.

Thus the purpose of this study was to test the hypothesis that, in OA knees without history of serious injury, larger knee flexion angle and more anterior femoral translation at heel-strike of walking are correlated with greater loss of cartilage thickness over the following 5 year period.

## METHODS

This IRB-approved study analyzed a group of 16 patients with medial compartment knee OA and no history of major lower limbs injury. The experimental protocol consisted of a baseline gait test and MRI scan and of a follow-up MRI scan 5 ± 1 years after baseline evaluation. Only the index knee of each patient was analyzed in this study. At baseline, the demographics of this group including 10 females were 60 ± 9 years old, 1.6 ± 0.1 m, 79 ± 15 kg, and KL grades [7] of 2.1 ± 1.1.

For the baseline gait test, three trials at normal self-selected walking speed were collected for each knee using a marker-based system (Qualisys, SE) and a force plate (Bertec, OH). The point cluster technique was used to track the position and orientation of the thigh and shank anatomical frames during ambulation [8]. The knee flexion angle was calculated using the joint coordinate system [9] and the knee translation was calculated as the position of the center of the femoral epicondyles relative to the anterior-posterior axis of the tibial anatomical frame [10]. For each trial, the flexion angle and femoral translation were recorded at heel-strike event, and the results from the three trials were averaged to have only one baseline flexion angle and one baseline femoral translation per patient.

Since loss of cartilage is a primary hallmark of knee OA, decreases in cartilage thickness over the 5 year follow-up period were used to characterize disease progression after the baseline gait test. Each knee was scanned at both time points using a 1.5 T MRI device (GE Medical Systems, WI) and a sagittal 3D-SPGR sequence. MR images were segmented and 3D models were reconstructed for the femoral and tibial cartilages using custom software [11]. The mean cartilage thicknesses were then calculated over the total medial femoral load-bearing region and over the total medial tibial compartment [12]. Changes in cartilage thickness were defined as the difference in mean cartilage thickness between scans (follow-up minus baseline).

Changes in cartilage thickness were compared to baseline gait variables using scatter plot and linear regression analysis. Pearson correlation and its associated p-value were also used to test the relationship between knee kinematics and OA progression.

## RESULTS

The knee translation at baseline was correlated with the progression of the disease during the 5 year follow-up period: more anterior position of the femur relative to the tibia corresponded to greater loss of femoral (Figure 1a) and tibial (Figure 1b) cartilage thickness. Larger knee flexion angle at baseline was also correlated with greater decreases in tibial cartilage thickness (Figure 1d).

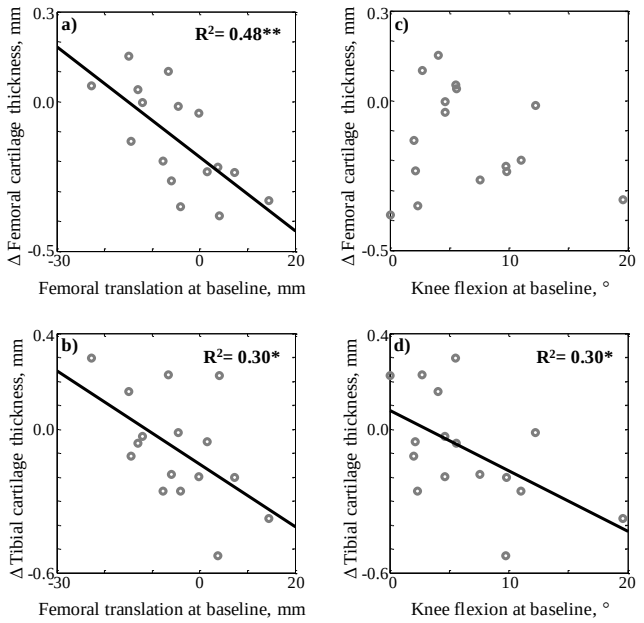


**Figure 1**: Correlation between changes in medial cartilage thickness over 5 years and baseline knee kinematics at heel-strike of walking. Stars indicate the strength of the correlation (*p< 0.05, **p<0.001).

## DISCUSSION

The finding that walking kinematics at baseline was correlated with cartilage loss at 5 year follow-up was fundamentally important because it provided new insights into a potential mechanical pathway to knee OA previously highlighted for post-traumatic knee OA [2, 3]. When considered in light of a recent cross-sectional study [4] reporting similar differences in knee flexion and translation with increasing age in asymptomatic subjects and with increasing OA severity in diseased knees, the results of the present study also provided novel support for the idea that age-related changes in knee kinematics might be an initiating factor in idiopathic OA [2].

The specific gait variables identified in this study enhanced our understanding of some of the unique characteristics of knee OA. For example, the knee translation at heel-strike was a better predictor of future cartilage changes than the knee flexion angle. The concave-convex geometry of the medial tibiofemoral compartment might be an explanation for this difference, since a change in translation could result in a larger shift in contact location than a change in flexion angle [13]. Thus the increased incidence of medial knee OA could have a basis in early anterior-posterior translational changes during heel-strike.

Identifying specific kinematic features of gait that indicate risks for future cartilage loss also provides a rational basis for developing new prevention strategies. Several effective gait interventions have been developed to modify the loading environment at the knee [14, 15], and there is a possibility that modifying femoral translation and flexion angle might also be beneficial to OA patients.

When interpreting these results, it is important to consider the limited number of patients in this study, and the fact that future stimulus-response research [16] is necessary to demonstrate that changes in knee kinematics are a cause and not a consequence of knee OA.

## CONCLUSIONS

The correlations between baseline gait kinematics and changes in cartilage thickness at 5 year follow-up provided new insights into the mechanism of a mechanical pathway to knee OA. These results have important implications for the early detection as well as the design of preventative strategies for patients at risk for developing more advanced disease.


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## REFERENCES


1. Murphy L, et al. Arthrit Care Res **59**:1207-13, 2008.
2. Andriacchi TP, et al. Ann Biomed Eng **32**:447-57, 2004.
3. Andriacchi TP, et al. J Bone Joint Surg Am **91**: suppl 1 95-101, 2009.
4. Favre J, et al. Proceedings of ORS, San Antonio, P189, 2013.
5. Koo S, et al., J Biomech **29**:1405-9, 2009.
6. Scanlan SF, et al. J Biomech: in press, 2013.
7. Kellgren JH, et al. Ann Rheum Dis **16**:494-502, 1957.
8. Andriacchi TP et al., J Biomech Eng **120**:743-9, 1998.
9. Grood ES , et al. J Biomech Eng 105:136-44, 1983.
10. Dyrby CO, et al. J Orthop Res **22**:794-800, 2004.
11. Koo S, et al. Osteoarthritis Cartilage **13**:782-9, 2005.
12. Wirth W, et al. IEEE Trans Med Imaging **27**:737-44, 2008.
13. Koo S, et al. J Biomech **40**:2961-6, 2007.
14. Erhart JC, et al. Journal of Orthopedic Research **28**:873-9, 2010.
15. Fregly BJ. Hospital for Special Surgery **8**:45-8, 2012.
16. Andracchi TP. Nat Rev Rheumatol **24**:371-2, 2012.