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# Sagittal plane walking patterns are related to MRI changes over 18-months in people with and without mild-moderate hip osteoarthritis

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## Abstract

The purpose was to evaluate the association of sagittal plane gait mechanics with MRI changes in the hip joint over 18-months. Subjects with and without radiographic hip OA (n=57) underwent

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Conception and design – Deepak Kumar, Thomas Link, Richard Souza, Sharmila Majumdar
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Final approval of manuscript - Deepak Kumar, Cory Wyatt, Sonia Lee, Narihiro Okazaki, Ko Chiba, Thomas M. Link, Richard B Souza, Sharmila Majumdar
All authors have read and approved the final manuscript.

MRI at baseline and 18 months for grading of cartilage lesions, bone marrow lesions (BML), cysts, and labral tears. 3D gait analyses at baseline were used for sagittal plane hip kinematics and kinetics during the stance phase. Subjects were classified as progressors or non-progressors based on increase in any MRI OA parameter. Multivariate ANOVA were used for differences in sagittal gait parameters between progressors and non-progressors at baseline while adjusting for age. Logistic regression was used to estimate the probability of being classified as a progressor or non-progressor with increasing hip flexion while adjusting for age, BMI, sex, and presence of radiographic hip OA. Of the 57, 35 were classified as non-progressors and 22 were classified as progressors. At baseline, the progressors walked with 4.5° greater hip flexion during early stance (P = 0.021) and 3.5° lesser hip extension in late stance that was nearly significant (P = 0.059). Walking with greater hip flexion at baseline was associated with a greater risk of increase in MRI defined structural changes in the hip joint (Odds Ratio = 1.1, P = 0.038). Greater hip flexion during walking was associated with a risk of structural progression of hip OA. The results may guide future interventions to alter the walking patterns and slow structural hip OA progression.

**Keywords**

hip; osteoarthritis; gait; disease progression; MRI

## INTRODUCTION

Hip osteoarthritis (OA) affects 28% of the population over the age of 45 years in the US[1] and leads to significant disability.[2] However, factors related to progression of hip OA are not well understood. The use of high-resolution MRI has advanced the understanding of structural progression of knee OA.[3–5] High-resolution optimized MR imaging of the hip at 3.0 Tesla allows for a comprehensive evaluation of hip degeneration without the need for contrast.[6–10] However, studies using MR imaging to evaluate progression in people with hip OA are lacking. Understanding factors related to hip OA progression is important to identify individuals at high-risk who may be targeted for prevention strategies. It is especially important to identify factors related to progression in people with mild-moderate disease before end-stage structural changes occur.

Previous studies have reported differences in sagittal plane walking mechanics between individuals with and without hip OA.[11–17] These studies suggest that alterations in loading across the hip joint due to walking mechanics may be associated with progression of hip OA. However, previous studies have either been cross-sectional[14–16], or have focused on changes in walking mechanics post-total hip replacement (THR).[11; 18] In a cohort of 50 women with secondary hip OA, Tateuchi et al. recently observed reduced cumulative sagittal plane loading (product of sagittal moment impulse and number of steps/day) in people with lower radiographic joint space width.[19] Longitudinal analyses from the same cohort showed associations between greater cumulative frontal plane loading and greater radiographic progression of hip OA, with sagittal cumulative loading approaching significance.[20] These pair of studies only included women, mostly with established radiographic hip OA and significant pain. Identification of risk factors in individuals with early imaging signs of hip OA before onset of significant symptoms may offer an opportunity for early interventions.

Also, Tateuchi et al. utilized radiographic outcomes for determining progression that are known to have limited sensitivity.[21–23] Hence, longitudinal studies investigating the association of walking mechanics with MRI defined hip OA progression are needed. Identification of specific biomechanical parameters that may be associated with hip OA progression may allow for targeted interventions to prevent the progression of hip OA.

Hence, the goal of this longitudinal study was to evaluate the association of sagittal gait mechanics with structural changes in the hip joint using MRI over 18-months.

## METHODS

### Subjects

The data for this prospective cohort study (Level of evidence = 2) are from participants (n=57) recruited for a longitudinal observational study. This cohort has been described previously.[24; 25] Subjects were recruited from the community using flyers and advertisements. The inclusion criteria for subjects with radiographic hip OA were Kellgren-Lawrence grade (KLG) of 2 or 3 at the hip on weight-bearing anterior-posterior radiographs[21]. The side with greater KLG was selected as the “index hip”. The subjects without radiographic hip OA had a radiographic KLG grade of 0 or 1 at both hips, and were without history of diagnosed OA or previous hip injuries. General exclusion criteria for recruitment into the study for all subjects were any contra-indications to MR imaging, KLG grade of 4, a total joint replacement of any lower extremity joint, previous hip trauma, pain at any other lower extremity joint, radiographic evidence of any knee or ankle joint OA, systemic inflammatory arthritis or any other spine or lower extremity condition that would affect their ability to complete the study procedures. All subjects signed a written informed consent approved by the University of California, San Francisco Committee on Human Research.

### MR imaging

All imaging was performed with a 3-Tesla MR scanner (GE MR750, GE Healthcare, Waukesha, WI, USA) and an 8-channel cardiac coil (GE Healthcare, Waukesha, WI, USA) at baseline and 18 months. Patient positioning aids were used to immobilize and support patients, and ensure a consistent, reproducible, and comfortable hip positioning during scanning. Patients were positioned supine with their feet taped together, their knees supported by cushions to prevent movement. The acquisition protocol, including the imaging parameters, has been published previously.[10; 25–27] Two experienced board-certified musculoskeletal radiologists graded cartilage defects, labral tears, bone marrow lesions (BML) and subchondral cysts on MR studies using the SHOMRI (Scoring hip osteoarthritis with MRI) semi-quantitative grading system.[10; 2610] A third radiologist was consulted in case of a disagreement. Intra and inter-reliability for these measures has been reported previously.[10] Intra-reader kappa values were between 0.70 – 0.79 and percent agreement was between 74%–98%.[10] Inter-rater kappa values were between 0.55 – 0.71 and percent agreement was between 66%–97%.[10]

Cartilage defects were graded as 0 (no defect), 1 (partial thickness) and 2 (full thickness). BMEL were graded as 0 (absent), 1 (< or = 0.5 cm), 2 (0.5–1.5 cm) and 3 (> or = 1.5 cm). Subchondral cysts were graded as 0 (absent), 1 (< or = 0.5 cm) and 2 (> 0.5 cm). Labral tears were graded as 0 (normal or normal variant), 1 (fraying or signal abnormality), 2 (simple tear), 3 (labrum-cartilage separation), 4 (complex tear) and 5 (maceration). Paired readings were performed for the baseline and 18-month studies. The radiologists were blinded to the subject's radiographic OA status. Subjects were stratified into progressors (increase in any of the 4 hip OA structural parameter), and non-progressors (no change in any hip OA structural parameter).

## Motion Analysis

All subjects walked at a fixed speed of 1.35 meters/second while 3-D kinematics (at 250 Hz) using a passive 10-camera system (VICON, Oxford Metrics, UK) and kinetics (at 1000 Hz) using 2 floor embedded force platforms (AMTI, Watertown, MA, USA) were collected. The speed of 1.35 m/sec was selected as the mean of the reported average walking speeds on smooth level surfaces for adult men (1.43 m/sec) and women (1.28 m/sec) by Perry et al.[28] None of the subjects reported or exhibited difficulty attaining this speed. Self-selected walking speed was also recorded for all subjects as they walked across the laboratory over multiple trials (at least 5 trials per subject) at their comfortable pace. A trial was acceptable when there was clean foot-strike on a force platform and the speed was within ± 5% of the first good trial. Five trials were acquired for each subject. Fourteen-millimeter spherical retro-reflective markers were placed on bony landmarks of bilateral lower extremities for identification of joint centers. Rigid marker clusters placed bilaterally on the lateral surface of the subject's thighs, legs and heel shoe counters were used to track segment motions. Kinematic and kinetic data were calculated using Visual3D (C-motion, Georgetown, MD, USA) bilaterally. In the right-hand coordinate system convention used, flexion, abduction and internal rotation were assigned as positive. Variables calculated included the peak hip flexion and extension, sagittal excursion, and peak flexion and extension moments. All variables were calculated during the stance phase of walking when the foot was in contact with the floor. The joint moments are reported as external moments and are normalized to the subject's body (BW) and height (Ht) (% BW*Ht). The average of 5 trials was calculated for each subject.

## Patient-reported Outcomes

The Hip disability and Osteoarthritis Outcome Score (HOOS) was completed by all subjects.[29] The HOOS Pain, Symptoms, and Activities of Daily Living (ADL) sub-scales were used in this study. The HOOS has been shown to be a valid, reliable, and responsive measure of overall hip joint function in people with OA.[29]

## Statistics

The analyses were conducted to investigate the association of gait mechanics with MRI progression of hip OA. Multivariate ANOVA were used to evaluate the differences in sagittal gait parameters between progressors and non-progressors at baseline while adjusting for age. Logistic regression models were used to estimate the probability of being classified as a progressor (increase in cartilage, labrum, BML, or cyst scores) vs. non-progressor with

increasing hip flexion. Both univariate and multivariate analyses were undertaken. For the multivariate logistic regression models, the covariates included age, BMI, sex, and presence of radiographic hip OA at baseline. Alpha was set at $P < 0.05$.

## RESULTS

### Subject characteristics

Of the 57 subjects, 35 were classified as non-progressors and 22 were classified as progressors. Demographics for the progressors and non-progressors are provided in Table 1. The progressors were older and had a greater proportion of subjects with KLG2 and KLG3, and a lower proportion of subjects with KLG = 0. Of the 22 subjects in the progressor group, 11 had a progression of cartilage lesions, 5 had a progression of BML, 5 had a progression of subchondral cysts, and 10 had a progression of labral tears. There was a nearly significant difference in self-selected walking speed with the progressors walking approximately 0.12 m/s faster than non-progressors after adjusting for age.

### Baseline differences in sagittal gait mechanics during a 1.35 m/s walking task

The results for baseline differences in sagittal gait mechanics are shown in Figure 1. The progressors walked with approximately 4.5° greater flexion during early stance ($P = 0.021$). During late stance, the progressors walked with approximately 3.5° lower hip extension but the difference was not significant ($P = 0.059$). The differences in sagittal ROM and moments were not significant.

### Association of baseline sagittal mechanics and structural progression of hip OA

Univariate logistic regression showed that each 1° increase in hip flexion was associated with 1.09 times higher risk of MRI progression of hip OA (OR=1.09 [1.00–1.18]; $P = 0.035$). Multivariate analyses (adjusting for age, sex, BMI, presence of ROA at baseline) showed that each 1° increase in hip flexion was associated with a 1.11 times higher risk of MRI progression of hip OA (OR=1.11 [1.00–1.22]; $p = 0.038$). Age (OR=1.04 [0.98–1.09]; p=0.188), BMI (OR=1.25 [0.96–1.62]; p=0.105), sex (OR=0.22 [0.0.05–1.02]; $p = 0.054$), and presence of radiographic OA (OR=3.34 [0.73–15.21]; $p = 0.119$) were not found to be significant predictors in the model. The overall model $\chi^2$ was 17.1 ($P = 0.004$), with model $R^2$ of 0.26 for the Cox & Snell method and $R^2 = 0.35$ for Nagelkerke method.

## DISCUSSION

The objectives of this 18-month longitudinal study were to evaluate the association of walking patterns with structural progression of hip OA parameters assesses using MR imaging over 18 months. The results show that individuals, who have structural progression of hip OA over 18-months walk with greater peak hip flexion and had a trend for less peak hip extension during the stance phase of walking at 1.35 m/s at baseline. Over 18-months, walking with greater peak hip flexion were associated with a greater risk of structural OA progression. These novel findings advance the understanding of the role of walking patterns and hip degeneration. The results advance the understanding of hip OA disease process and

also provide initial data for future research on interventions to alter the walking patterns and slow structural hip OA progression.

We observed that walking with greater hip flexion was associated with a small but significant risk of hip OA progression over 18-months. Previous studies have reported that in healthy individuals, anterior and superior regions of the hip joint experience greater contact stress compared to the posterior regions throughout the stance phase of walking.[30] Speculatively, the differences in loading across the hip joint between the progressors and non-progressors due to the differences in hip kinematics, may lead to overload and underload of hip regions that are not physiologically adapted to the new loading patterns, leading to further hip degeneration. We had earlier reported that greater hip flexion in early stance and reduced hip extension in late stance during walking were associated with femoral cartilage lesions.[31] The results from the present study further corroborate our findings from the cross-sectional analyses, with peak hip flexion being predictive of hip OA structural progression. Greater hip flexion could alter the loading across the hip joint leading to higher risk of cartilage damage in certain regions of the hip joint. At the knee, greater loading during walking has been related to greater cartilage degeneration over time.[32; 33] Hence, multiple studies in people with knee OA have reported a reduction in knee loading and changes in gait mechanics with gait retraining interventions.[34–36] Similar to the research in people with knee OA, our results could guide future movement retraining interventions to slow the structural progression of hip OA. However, our findings need to be confirmed in larger samples with a longer follow-up. Although a difference in kinematics may be related to difference in magnitude and distribution of contact forces at the hip joint, musculoskeletal modeling studies should be undertaken to investigate these differences.

We quantified the gait mechanics while the subjects all walked at a fixed speed of 1.35 m/s. We decided to standardize the speed to minimize the effects of speed on the gait variables. The subjects did not have difficult achieving the 1.35 m/s walking speed as their preferred speed was on average greater than the prescribed speed (Table 1). However, it is possible that having the subjects walk at a speed that was not their preferred walking speed may have influenced their gait mechanics. Also, the progressor group had slightly greater preferred walking speed compared to the non-progressor group, although this difference was not significant. Therefore, we assessed if hip flexion during walking at their preferred speed was associated with risk of progression of MR parameters of hip OA (results not shown). Peak hip flexion angle was on average 2° greater during walking at preferred speed compared to walking at 1.35 m/s in both progressor and non-progressor groups. Greater hip flexion was still significantly associated with a risk of progression (OR = 1.10 [1.00–1.19], p = 0.041) in the multivariate analyses with similar OR as walking at 1.35 m/s. However, once we included walking speed as an additional covariate in the regression model, hip flexion was no longer significantly associated with progression (p = 0.309). These results suggest that standardized activities (e.g. walking at 1.35 m/s) should be used to minimize confounding due to differences in preferred speed between individuals in studies investigating the associations of biomechanics with clinical outcomes.

A limitation of some of the earlier studies has been the use of radiographic criteria to define hip OA. High-resolution optimized non-contrast imaging of the hip at 3.0 Tesla offers

significant advantages over radiographs for investigation of degeneration of various articular tissues seen in hip OA. MR-identified parameters of hip OA including cartilage defects, bone marrow lesions, and subchondral cysts are more strongly correlated with patient-reported symptoms, pain, and disability compared to radiographic KLG in people with mild-moderate hip OA.[10] In our study, female sex (P=0.054) tended to be associated with a risk of progression of MR parameters of hip OA in the multivariate logistic regression model, These results are similar to those observed at the knee and may reflect some common risk factors for generalized OA progression.[4; 37] However, since the result for female sex was not significant, studies in larger samples are needed to further investigate this association.

Interestingly, we did not find presence of radiographic hip OA to be associated with risk of progression of MR parameters, even though the groups were significantly different in the distribution of KLG (Table 1). The progressor group had greater proportion of subjects with KLG2 and 3 compared to the non-progressor group. However, there were very few subjects (n=2) in KLG3 category for non-progressor and KLG0 category for progressors. Because of the small sample size and few subjects in these categories, we combined KLG0 and 1 into one category (no radiographic hip OA), and KLG2 and 3 into another category (radiographic hip OA). To further explore the association of radiographic hip OA with progression, we conducted additional sensitivity analyses by stratifying the subjects into those with no radiographic hip OA (KLG0 or 1), KLG2, and KLG3 (results not shown). The results from the logistic regression showed that radiographic hip OA was still not significantly associated with progression ($p = 0.255$ for KLG2 vs. no OA, and $p = 0.161$ for KLG 3 vs no OA). Hip flexion was only borderline significant with OR of 1.1 ($p = 0.050$). Therefore, a larger cohort with more subjects per KLG would be needed to confirm these preliminary findings.

The baseline differences in sagittal hip moments between the progressors and non-progressors were not significant in our cohort. This could be related to the fact that the individuals were only mildly affected by the hip OA and were high functioning as described above. It is possible that with early hip OA, there are kinematic deviations in walking patterns that precede the changes in hip moments that may occur with a loss of hip muscle strength seen in advanced disease. This is supported by our observation of a reduction in the sagittal excursion (P=0.039) and trends for reduction in hip extension moments (P=0.056) in the progressors over 18-months (results not shown). We also excluded subjects with KLG = 4 from the study. In our earlier cross-sectional study, we did not observe significant differences in sagittal hip moments between individuals with and without radiographic hip OA. Pain in people with hip OA may also affect movement patterns. Our cohort reported minimal pain (Table 1) and we did not observe a significant change in the patient-reported HOOS pain scores over 18-months (P=0.105) (results not shown). However, future studies should investigate the association of hip flexion during walking with progression of both structural and symptomatic hip OA over the longer term.

Limitations of this study include a small sample size and a relatively short follow-up period. Future studies in larger samples with a longer follow-up are needed to confirm these findings. We did not acquire radiographs at the 18-month timepoint. Since joint space width is the currently accepted measure of assessing hip OA progression, future studies should compare the sensitivity of MRI and radiographs. The sample size did not allow us to

separately evaluate the association of different gait parameters with progression of various structural OA parameters. Additionally, the results may not be generalizable to populations with more severe disease since we excluded those with KLG 4.

In conclusion, subjects who have a progression of hip OA assessed with MRI, walk with greater hip flexion during a 1.35 m/s walking task. Greater hip flexion during walking is associated with an increased risk of hip OA progression over 18-months. This is an initial study on a small cohort of subjects and the results need to be replicated in larger cohorts. The results can potentially guide future interventions to alter the walking patterns and slow structural hip OA progression.

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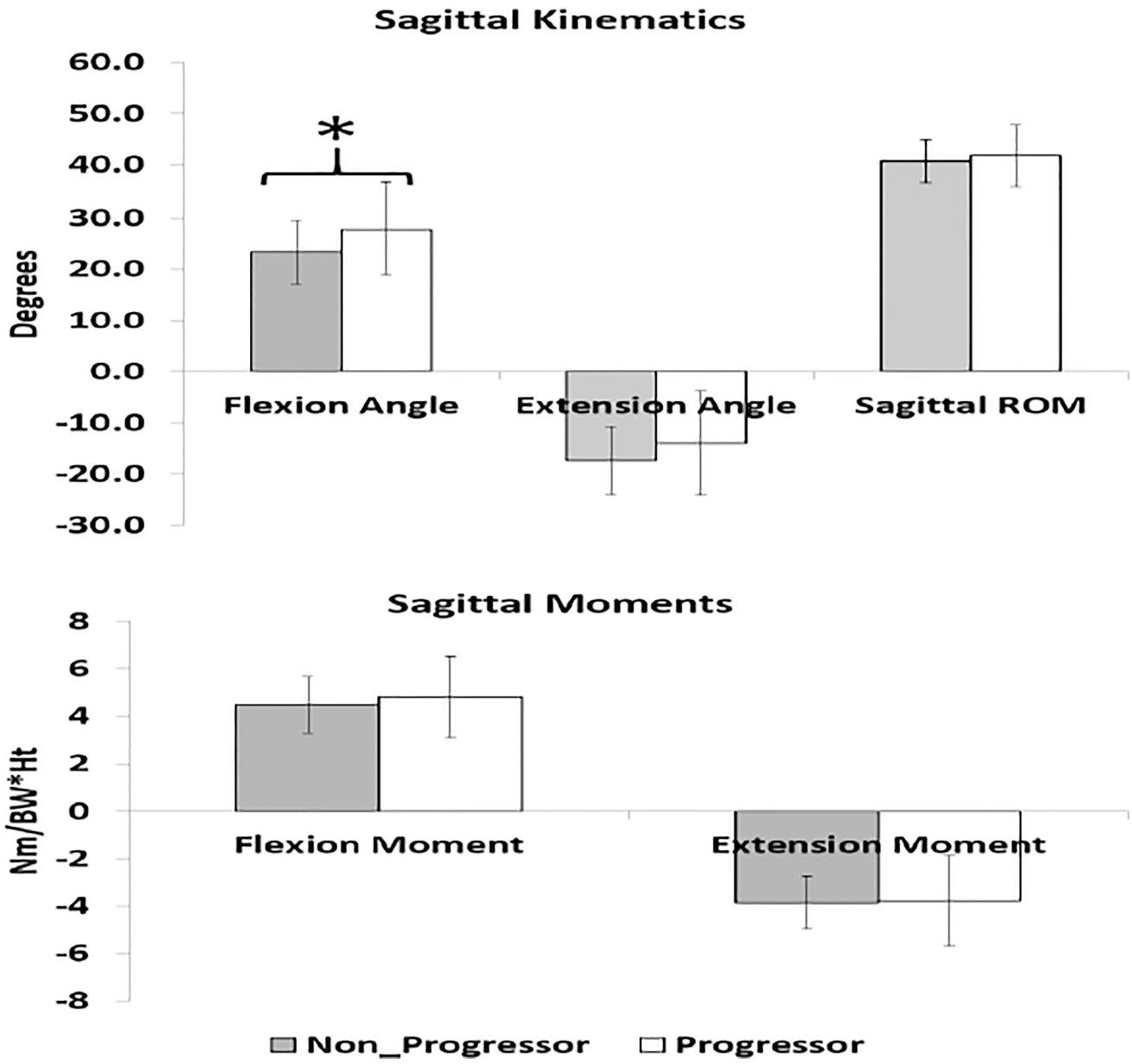


**Figure 1.**

Baseline sagittal kinematics and kinetics during the stance phase of walking at 1.35 m/s. The bars represent mean values and error bars represent 1 standard deviation. The non-progressors are shown in grey and the progressors are shown in white. The * indicates statistical significance at $P < 0.05$.

## Table 1

Baseline data on age, BMI, sex distribution, KLG, and HOOS for the prgoressor and non-progressor groups.

| | Non-progressor (n = 35) | Progressor (n = 22) | *P* |
|---|---|---|---|
| **Age (years)** | 44.1 (13.4) | 52.0 (11.8) | **0.028** |
| **BMI (kg/m$^2$)** | 23.6 (2.6) | 24.0 (3.6) | 0.636 |
| **Men:Women** | 22:13 | 9:13 | 0.105 * |
| **KLG** | KLG0=13 (37.1 %); KLG1=14 (40%); KLG2 = 6 (17.1 %); KLG3 = 2 (5.7%) | KLG0=2 (9.1%); KLG1=9 (40.9%); KLG2 = 5 (22.7%); KLG3 = 6 (27.3 %) | **0.033** * |
| **Walking speed (m/s)** | 1.46 (0.23) | 1.59 (0.19) | 0.053 ** |
| **HOOS Symptoms** | 90.7 (15.7) | 87.1 (15.5) | 0.414 ** |
| **HOOS Pain** | 89.6 (17.0) | 90.9 (13.3) | 0.860 ** |
| **HOOS ADL** | 92.7 (13.8) | 95.4 (10.2) | 0.497 ** |

Data for age, BMI, walking speed, and HOOS are mean (standard deviation). P values for Age and BMI are from independent samples t-tests.

* P value from Chi-square test

** P adjusted for age

KLG = Kellgren-Lawrence Grade
HOOS = Hip disability and Osteoarthritis Outcome Score
ADL= Activities of Daily Living