Rutherford, Moreside, Wong (2015). Knee joint motion and muscle activation patterns are altered during gait in individuals with moderate hip OA compared to asymptomatic cohort. Clinical Biomechanics 30, 578-584

Introduction:

Lower extremity osteoarthritis (OA) contributes to morbidity in adult populations around the world and significantly impacts mobility and activity (White et al., 2013; Song et al., 2013). As there is currently no cure, many management approaches focus on symptom relief (Lane et al., 2011; Escobar et al., 2012; Zhang et al., 2008; Fernandes et al., 2013; McAlindon et al., 2014), yet little impact has been shown in reducing disease progression (Lane et al., 2011). Surgical interventions are increasingly required to reduce the burden of OA, with rates projected to increase over 400% in coming decades (Kurtz et al., 2007). Many individuals receiving a knee or hip replacement will go on to require subsequent replacement of another lower extremity joint (Gillam et al., 2013), contributing to the escalating long-term burden of OA that is seldom captured by current projections.

In a recent study, Gillam et al., 2013 found significant hazard ratios of 1.83- 2.97 for a contralateral knee replacement when participants’ first joint replacement was a total hip. Hazard ratios were lower if the subsequent knee replacement was ipsilateral (0.51-0.52) (Gillam et al., 2013). This supports previous work indicating increased likelihood of contralateral knee OA progression, rather than ipsilateral, following a hip replacement (Shakoor et al., 2002). Given the influence of mechanics in lower limb OA (Felson, 2013) the question arises as to whether the progression of knee OA in this population is a result of abnormal knee mechanics secondary to hip OA.
Gait analysis has been used as a method to study joint mechanics in OA, because of the mechanical demands (Wilson et al., 2009; Andriacchi et al., 2004; Andriacchi and Mundermann, 2006; Felson, 2013) and importance as a daily functional activity. Biomechanics and muscle activation measures provide a non-invasive approach to understanding joint function during walking. While sagittal plane hip movement is typically reduced in individuals with hip OA (Ornetti et al., 2011; Kubota et al., 2007; Hurwitz et al., 1997) during stance, understanding of knee mechanics in this population is lacking. Moreover, studies that have looked at stance phase knee mechanics tend to focus on late stage disease or post total hip replacement populations (Shakoor et al., 2003; Foucher and Wimmer, 2012; Ornetti et al., 2011). Knee mechanics, including a muscular system assessment are poorly understood in individuals with hip OA, who are not candidates for hip replacement.

The passive osteoligamentous, muscular, and the neurological subsystems are thought to be fundamental to supporting joint function (Solomonow and Krogsgaard, 2001; Panjabi, 1992; Sims, 1999) yet how lower extremity muscles activate during walking and the relationships with joint mechanics in individuals with hip OA is not clear. A greater understanding of knee joint muscle activation patterns in individuals with knee OA is emerging, linked to OA severity (Rutherford et al., 2013), progression (Hubley-Kozey et al., 2013) and joint mechanics (Heiden et al., 2009). Quadriceps activation is greater and more prolonged and greater levels of lateral hamstring activation and prolonged stance phase activity is found in individuals with medial compartment OA (Rutherford et al., 2013; Lynn and Costigan, 2008). Compensations by the neuromuscular system may occur to preserve knee joint function as previously described in the context of knee OA, but whether such changes to the knee exist with hip OA is unclear. Understanding alterations to knee mechanics and muscle activation patterns in individuals with
hip OA may provide insight into the evolution of lower extremity OA and relationships between knee and hip OA pathomechanics.

The purpose of this investigation was to determine whether three-dimensional (3D) knee joint movements and quadriceps and hamstrings activation patterns in individuals with unilateral symptomatic moderate hip OA are different from an asymptomatic cohort and whether these mechanics and muscle activation patterns differ between contra and ipsilateral knee joints.

Given previous findings of reduced sagittal plane hip joint motion, it is hypothesized that sagittal plane knee motion in the ipsilateral knee of individuals with hip OA will be reduced compared to the contralateral knee and healthy group, and that no differences in transverse or frontal plane motions will exist within or between groups. Given the hypothesized alterations in knee motion, it is hypothesized that both ipsilateral and contralateral quadriceps and hamstrings will activate for longer durations during stance compared to the asymptomatic cohort, and that no activation differences between the medial and lateral sites of these muscles will be found, contrary to that previously described in individuals with knee OA.

**Methods**

**Participants**

Participants with unilateral symptomatic hip OA were recruited over 1 year (2013-14) from local orthopaedic clinics after consultation with an orthopaedic surgeon regarding hip arthroscopy for early OA management. Participants were excluded if they were candidates for total hip replacement. A functional classification was also used to determine moderate severity as has been used in knee OA gait studies whereby individuals with hip OA self-reported no limitations with walking a city block, climbing stairs and jogging approximately 5 meters (Hubley-Kozey et
al., 2006). Hip OA was determined using the American College of Rheumatology criteria (Altman, 1991). Asymptomatic participants were recruited from the general community using website and email based advertisements and considered a sample of convenience. These individuals had reported no pain or known functional limitations in the ankles, knees, hips or low back. All participants were required to be ≥50 years of age, have no fracture or injury other than a sprain or strain (within one year) or no previous knee/hip joint surgery. All participants had to be able to walk independently with no neurological or cardiovascular disorder that would impair walking ability. The protocol was approved by the local institutional ethics review committee and participants provided written informed consent.

Standard A/P pelvis and lateral radiographs were available in the hip OA group. Radiographs were graded using the Kellgren-Lawrence (KL) ordinal radiographic scale (Kellgren and Lawrence, 1957) by a single, experienced reader (IW), who was blinded to participant identification and gait analysis outcomes (Vignon et al., 1999) at the time of scoring.

Procedures

Once informed consent was obtained, participants changed into a T-shirt and fitted shorts, removed their footwear and completed at least five self-paced walking trials across the GaitRITE™ portable pressure sensitive walkway (CIR Systems, Clifton, NJ, USA) to determine average self-selected gait speed. This 5.4m x 0.6m walkway is a valid tool for measuring spatial and temporal gait characteristics (McDonough et al., 2001; Bilney et al., 2003). All participants
completed the Hip Outcome Osteoarthritis Score (HOOS), the International Hip Outcome Tool (iHOT-33) and Oxford 12 self-report questionnaires.

Following these trials, participants were prepared for surface electromyography (EMG); skin was lightly shaved and cleaned with 70% alcohol wipes. Consistent with guidelines (Hermens et al., 2000) and standard procedures, Ag/AgCl surface electrodes (10 mm diameter, 30 mm inter-electrode distance, Red Dot, 3M Health Care, St. Paul MN, USA) were placed bilaterally in a bipolar configuration over vastus lateralis (VL) and medialis (VM) and both medial (MH) and lateral (LH) hamstrings. Muscle palpation and a series of isometric contractions for specific muscle groups were used for signal validation and gain adjustment. Surface EMG was recorded with two AMT-8™ 8-channel Bortec systems (Bortec Inc. Calgary) (Input Impedance: ~10GΩ, CMRR: 115dB at 60 Hz, Band-pass (10-1000 Hz)) using a custom LabVIEW™ 2013 program (National Instruments Corporation, Austin, TX) at 2000Hz.

Using a standard lower extremity motion capture marker set, rigid sets of four retro-reflective markers were affixed to the trunk (at the level of the inferior scapular angles), over the sacrum, and bilateral posterior femur and tibia using Velcro straps and secured with adhesive tape. Single retro-reflective markers were placed over the lateral aspect of the shoulders (below acromion), 7th cervical vertebra, greater trochanters, medial and lateral femoral and tibial epicondyles, medial and lateral malleoli, 5th metatarsal head, and posterior heels.

Prior to gait analysis, a kinematic model calibration was completed, including a standing calibration trial, a virtual sternum, 2 virtual anterior superior iliac spine location trials and 2 standing hip joint center calculation trials that required the subject to move each leg through hip
flexion, extension and abduction (Camomilla et al., 2006). Retro-reflective skin marker motion was captured at 50 Hz using four Qualisys® Pro-reflex motion analysis sensors (Gothenburg, Sweden).

Participants began walking on the treadmill with at least four minutes of accommodation/warm-up. Speed was set to that self-selected on the GaitRITE™ walkway. Following this, three 20-second data collections were completed, with approximately one minute between collections where participants continued to walk on the treadmill. Participants were blinded to collection intervals. After completion, all retro-reflective skin surface markers were removed and a resting muscle activity trial (EMG subject bias) was recorded with the participant lying supine. Electrodes were subsequently removed.

**Data Analysis**

Raw EMG signals were band pass filtered (Butterworth, 4th order, 20Hz – 500Hz) and digitally filtered using an inverse Fast Fournier Transformation algorithm to remove 60Hz and harmonics. All EMG signals were then corrected for resting bias, converted to micro-volts, full-wave rectified and filtered using a Butterworth, 6Hz recursive, 4th order, low-pass filter to create a linear enveloped signal (Hubley-Kozey et al., 2006). All EMG waveforms were amplitude normalized to the maximum EMG amplitude obtained for each muscle during the gait cycle (Burden et al., 2003).

Technical and local anatomical bone embedded coordinate systems for the pelvis, thigh, and shank were derived from virtual points and skin markers. Joint angles were calculated using a 6-degree of freedom model through Cardan/Euler rotations (z-y-x sequence), where knee joint
flexion occurred about the z-axis (Landry et al., 2007). A flexion/extension –
adduction/abduction – internal/external rotation sequence was utilized, where flexion, adduction,
and internal rotations were derived as a positive angle. The maximum forward trajectory of the
heel marker relative to the pelvis was used to indicate heel strike (Zeni et al., 2008). Knee joint
angles and EMG were time normalized to 100% of the gait cycle (i.e. 101 data points
representing heel strike to heel strike) using a cubic spline interpolation technique. All signal
processing and analyses were completed in MatLab™ Ver. 2014 (The Mathworks Inc., Natick,
Massachusetts, USA) using custom script.

Analysis

Motion data was ensemble averaged across the three 20-second walking trials (≥ 40
strides/side). In the sagittal plane, the stance phase ranges of motion from initial contact to peak
flexion and from peak flexion to peak extension were identified as previously investigated in
individuals with knee OA (Lewek et al., 2004; Rutherford et al., 2012). In the frontal and
transverse planes, the range of motion was obtained across the entire stance phase (0-60%).

Principal component analysis (PCA) was used to capture amplitude and temporal EMG
waveform features using custom MatLab™ 2014 script. PCA provides an analysis technique to
evaluate patterns of activation rather than amplitude specific metrics, identifying salient features
in the original data not possible with discrete gait waveform analysis techniques (Chau, 2001;
Wootten et al., 1990; Deluzio and Astephen, 2007). This technique has been previously
described in detail (Hubley-Kozey et al., 2006) and has been used to identify quadriceps and
hamstrings activation patterns in knee OA during gait (Hubley-Kozey et al., 2013; Rutherford et
al., 2013), but has yet to be implemented in hip OA gait literature.
For each muscle group (Quadriceps (VM/VL) and Hamstrings (MH/LH)), a matrix was formed from the linear enveloped EMG signal of each individual stride (≥40 strides/side/participant) \( [X] = [X \text{ strides } x 101] \). An eigenvector decomposition of the cross product matrix \( ([S] = [X^t] \times [X]) \) was performed, using standard notation \( U'SU = L \), yielding predominant orthonormal components called eigenvectors. These eigenvectors \( (U) \) are hereafter referred to as principal patterns (PP). A percent trace was calculated to determine how much variability was contained in each PP; those explaining the greatest percent of variation (>90% total variation) in the waveforms were retained and referred to as PP1, PP2 etc. Following PCA, EMG data were ensemble averaged across the 3 walking trials for each muscle, side and group. Principal pattern scores (\( PP\text{-scores} \)) were computed for individual gait waveforms by multiplying the ensemble-averaged waveform by the principal pattern generated by the data set that included each individual trial. \( PP\text{-scores} \) were utilized for testing waveform differences between groups, sides and muscles (e.g. OA vs. asymptomatic, affected vs. unaffected, MH vs. LH).

**Statistical Analysis**

Student’s t-tests were utilized to identify between-group differences in demographics, anthropometrics, HOOS, iHOT-33, Oxford-12, and WOMAC scores derived from the HOOS. Normality and equal variance of gait velocity collected on the GaitRITETM walkway, three-dimensional knee joint motion and EMG \( PP\text{-scores} \) were tested using Kolmogorov-Smirnov and Levene’s tests, respectively. A 2-factor repeated analysis of variance determined significant within-group differences between leg and within muscle group (e.g. LH/MH). Between-group differences were identified using Student’s t-tests. Significance level was set at \( \alpha=0.05 \), except when Bonferonni adjustments were used, as in the case of multiple comparisons, then
\( \alpha = 0.05 / \text{number of comparisons.} \) All statistical procedures were completed using Minitab™ Ver.17 (Minitab Inc. State College, PA, USA).

**Results**

Table 1 provides the group demographics, gait velocity and questionnaire outcomes. Individuals with hip OA had greater BMI, walked slower and reported worse outcomes on the HOOS, WOMAC, iHOT-33 and Oxford self-report questionnaires compared to the asymptomatic group \((p<0.05)\).

No significant between-leg differences were found in either group for sagittal plane knee angles \((p>0.05)\). There was a significant between-group difference in stance phase knee sagittal plane range of motion from peak flexion to peak extension \((5 \text{ degrees (95\% CI: 1.78, 8.77, Cohen's d effect size: 0.96 (large), p=0.004)})\). The ensemble averaged sagittal plane knee motion is illustrated in Figure 1. No significant between-leg or group differences were found for the frontal and transverse plane joint range of motion \((p>0.05)\). Table 2 provides the values corresponding to the knee motion analyses and Table 3 provides the PP-scores for the quadriceps and hamstring muscle analyses.

For the quadriceps, 3 principal patterns explained 95% of the waveform variance. Principal patterns can be found in Figure 2A. For PP1-scores, no within or between group differences or interactions were found \((p>0.05)\). A significant muscle main effect \((p=0.002)\) was found for PP2-scores, where VM had a pattern of elevated activity during late stance/early swing compared to early stance when compared to VL. No group/leg differences or interactions were found for PP2-scores \((p>0.05)\). A group main effect was found for the PP3-scores \((p=0.010)\). Individuals with hip OA had a pattern of elevated quadriceps activity during mid/late stance...
compared to early stance when compared to the asymptomatic group (Figure 3). No PP3-score muscle/side main effects or interactions were found (p>0.05).

Three principal patterns explained 91% of the hamstring waveform variance. Principal patterns can be found in Figure 2B. No between group/leg differences or interactions were found for PP1-scores (p>0.05). A muscle main effect was found where MH levels of activity across the stance phase were greater than LH (p=0.001). For the PP2-scores, no differences were found within the asymptomatic group for leg or muscle main effects or interactions (p>0.05). In those with hip OA, a borderline significant finding, when accounting for 3 comparisons, was shown for a leg x group interaction, whereby the contralateral leg had greater PP2-scores than the ipsilateral leg (p=0.018), suggesting hamstring activation during mid/late stance was elevated when compared to early stance in the contralateral leg (Figure 4). No differences existed between the affected leg and the asymptomatic group (p=0.453) or between the unaffected leg and asymptomatic group (p=0.045). For PP3-scores, no within or between group differences were found (p>0.05).

Discussion

The evolution of lower extremity OA is complex. Some studies support a mechanical influence in the progression of this disease (Wilson et al., 2009; Felson, 2013; Shakoor et al., 2002; Andriacchi and Mundermann, 2006). While attention has been paid to the effect of knee OA on hip joint mechanics and muscle activation (Mundermann et al., 2004; Astephen et al., 2008; Rutherford et al., 2014), there is a lack of similar research regarding the effect of hip OA on other lower extremity joints. The current study sought to determine whether hip OA was associated with altered knee mechanics and muscle activation patterns, on either the ipsilateral or
contralateral side. The results indicate that, when compared to a cohort of individuals with no symptoms of OA, individuals with moderate hip OA present with less sagittal plane knee motion bilaterally during stance and alterations to mid-stance quadriceps and hamstring muscle activation patterns.

For this study individuals with unilateral symptomatic hip OA who were not deemed severe enough to warrant total joint replacement were recruited. To assist this “moderate” classification, a functional criterion was adopted that has been used in the knee OA literature (Hubley-Kozey et al., 2006). In the current study, individuals with hip OA walked faster than those previously classified as severe (Hodt-Billington et al., 2012; Kubota et al., 2007), a similar velocity to those with moderate knee OA (Rutherford et al., 2012) and slower than individuals with mild to moderate hip OA (Eitzen et al., 2012). Regarding self-report metrics, HOOS and iHOT scores were similar to a group of younger individuals who underwent hip arthroscopy for severe hip chondropathy (Kemp et al., 2014). HOOS scores were greater than those found prior to total hip arthroplasty and lower than scores reported 6 months after total hip arthroplasty (Nilsdotter et al., 2003). These findings suggest that the population in this study was in a relatively early stage of hip OA, yet demonstrated altered knee mechanics while walking.

The only knee joint kinematic difference that occurred in this study was reduced sagittal plane motion during stance. This partially confirms our hypothesis; no differences in frontal and transverse plane motions were expected or found, as these individuals did not present with known knee pathology. In knee OA studies, moments are typically used to understand mechanics in the frontal and transverse plane and differences have been found in this population (Astephen et al., 2008). While transverse plane motions have not been reported, varus thrust has been used as a frontal plane kinematic variable in knee OA gait studies and authors have shown that this
movement (based on visual inspection) is related to knee OA progression (Chang et al., 2004). Varus thrust calculations using skin surface markers have yet to be fully established however, given no differences in frontal plane motion during stance occurred between our groups, we are confident that varus thrust was not apparent.

Reduced ipsilateral hip range of motion during stance has been found in individuals with mild to moderate hip OA (Eitzen et al., 2012), thus we hypothesized that concomitant alterations would occur in the ipsilateral knee. Reduced hip extension during late stance is found in individuals with hip OA (Ornetti et al., 2011) and thus it was expected that during late stance knee extension motion would also be reduced. Reduced sagittal knee motion was found bilaterally, as the knee transitioned from peak flexion to peak extension during stance. While this difference has been found in individuals with knee OA (Kaufman et al., 2001; Astephen et al., 2008), it is not clear whether the current findings have implications for understanding whether knee motions in individuals with hip OA influence knee OA pathomechanics. Regardless, the position of knee extension provides inherent stiffness to the knee through passive osteoligamentous constraints, allowing muscle activity to reduce (Markolf et al., 1976). As illustrated in Figure 1, the knees of individuals with hip OA are not moving fully into extension during mid to late stance, ultimately requiring additional muscular support, given their flexed position.

The quadriceps and hamstring muscles have been studied extensively in knee OA literature and are thought to provide stability to deteriorating knee joints (Lewek et al., 2004; Hubley-Kozey et al., 2006) or those with joint effusion (Rutherford et al., 2012). Given our hypothesis of altered sagittal plane kinematics, it was also hypothesized both ipsilateral and contralateral quadriceps and hamstrings will activate for longer durations during stance compared to the asymptomatic cohort. This hypothesis was partially supported. The amplitude normalization method utilized in
this study precluded the ability to make inferences about overall levels of activity; however, the use of PCA provided the ability to test for waveform pattern differences. As shown in Figure 3, the quadriceps were elevated during mid to late stance in both ipsilateral and contralateral knees of individuals with hip OA compared to the asymptomatic group. This prolonged activation has been shown in individuals with knee OA (Rutherford et al., 2013), and is thought to be a result of greater stability demands secondary to joint degeneration. The findings in this study suggest that given the knee remained in a more flexed position during mid to late stance (Figure 1), greater quadriceps activation was required. While this increase may be considered subtle, accumulated over the 10,000 steps/day that has been benchmarked to result in health improvements (White et al., 2013), these differences may result in fatigue and altered joint loading.

Contrary to the quadriceps and sagittal plane motion results, no group differences occurred in the hamstring activation patterns. A borderline significant difference ($p=0.018$) in $PP2$-scores was found between the ipsilateral and contralateral knees of individuals with hip OA. As shown in Figure 4, this corresponded to two particular increases in activation, occurring at approximately 15% and 45% of the gait cycle. Interestingly, these are transition periods to and from contralateral single leg stance. Using mathematical modelling, Schipplin and Andriacchi, 1991, predicted that increased hamstring co-activity was required to stabilize the knee, particularly lateral stability, during walking. In addition, Messier et al., 2011 found that compressive forces in the tibio-femoral joint can reduce as a result of reductions in hamstring co-contraction. Together these findings suggest greater stability in the contra-lateral knee may be required in these individuals with moderate hip OA. What remains to be determined, is why this might be the case. Previous work in individuals with late stage hip OA and post total hip arthroplasty find increased knee adduction moments in the contralateral knee (Shakoor et al., 2011; Foucher and
Wimmer, 2012). Knee moments were not calculated in this study, given our standard treadmill (i.e. no force plate). However, the time signatures of elevated contralateral hamstring activation occur at approximately the same point as the first and second peak knee adduction moments previously described (Astephen et al., 2008). In addition, Heiden et al., 2009 reported that lateral muscle activation was positively correlated to adduction moment magnitude during gait in individuals with knee OA. Further evidence is required to more fully understand these associations in this moderate hip OA population, particularly as it pertains to both ipsilateral and contralateral 3-dimensional knee moments and knee muscle electromyography. This study provides preliminary evidence that further work is needed to understand the biomechanical basis of lower extremity OA etiology and progression.

Limitations:

Several limitations exist in this study. Firstly, differences in BMI existed between groups. Previous work has shown knee moments to be affected by BMI (Harding et al., 2012), however these authors did not report differences in knee joint kinematics. In addition, it is not clear how BMI might affect muscle activity patterns during walking. There is the assumption that those with greater BMI may require higher levels of activation to support their mass. However if increased BMI is a result of greater lean muscle mass, individuals may in fact use less relative levels of activation to walk. Our normalization methods preclude overall amplitude based comparisons, in which BMI might have the greatest affect. Secondly, while all OA participants reported only one symptomatic hip, most also exhibited degenerative changes in the contralateral hip (Table 1), although all were under a KL score of II. While many studies use KL grade II as a benchmark for radiographic OA (Schiphof et al., 2008), the asymptomatic hip cannot be considered non-arthritic, and between-leg or between-group differences cannot be attributed
entirely to compensations due to unilateral hip OA. Thirdly, treadmill speed was set to the self-selected walking speed as determined from the GaitRITE™ walkway. The assumption was made that participants would adopt similar spatial/temporal gait characteristics on the treadmill after a period of accommodation (Lee and Hidler, 2008). While GaitRITE™ validity and reliability have been shown (McDonough et al., 2001), some participants commented that the treadmill seemed faster despite the same velocity although this sensation tended to reduce after 2 – 3 minutes of treadmill walking. Fourthly, the treadmill used for this investigation was not instrumented and therefore, we were unable to calculate joint moments. Previous works have reported frontal plane joint moments in individuals with severe hip OA, directly relating pathomechanics between hip and knee OA. These studies however have not included muscle activation patterns, which can also provide information on joint loading and stability demands of walking. Future work should consider a comprehensive biomechanical analysis of early to moderate hip OA gait. Finally, in that EMG amplitudes were normalized to the peak amplitude obtained, absolute amplitude comparisons between legs or groups could not be done (Burden, 2010). This method was chosen to avoid maximum muscle contractions, which may have elicited pain and reduced ability to obtain a true maximum. Instead, PCA was applied to quantify muscle activation temporal patterns, as previously performed in a knee OA population (Hubley-Kozey et al., 2006). Thus, only those patterns that were difference operators were interpreted (PP2 and PP3). To our knowledge, this is the first use of PCA to analyze activation patterns in the muscles surrounding the knee in a group of participants with unilateral symptomatic hip osteoarthritis.

Conclusions

Previous literature has found that hip and knee OA development occur non-randomly. This study provides a novel account of knee mechanics in individuals with moderate hip OA. Individuals
with unilateral hip OA walk with less sagittal knee motion bilaterally during the stance phase, compared to a non-arthritic cohort and with greater quadriceps activation during mid to late stance compared to early stance. The only difference found between the contra-lateral and ipsilateral knees of individuals with hip OA was greater mid-stance hamstring activity. From these data (motion and EMG) there is potential for increased loads and knee muscle fatigue bilaterally. However, there is no indication that one knee would be more susceptible to knee OA than the other in this population with moderate hip OA.
Figure Captions

Figure 1: Ensemble averaged sagittal plane knee range of motion collapsed across leg for the asymptomatic and hip OA groups. Shaded region indicates one standard deviation above and below the asymptomatic waveform. The vertical line demarcates toe off (i.e. end of stance). Degrees of flexion (+ve) and extension (-ve) on the y-axis.

Figure 2: Principal patterns of A) Quadriceps and B) Hamstrings. PP1-solid, PP2-dotted and PP3-dashed. For Quadriceps, PP1 captured the overall level of activation, explaining 89% of the variance. PP2 explained 3% of the variance, capturing a difference between early stance and the rest of the gait cycle. PP3 explained 3% of the variance, capturing an amplitude difference between early and mid/late stance, where a higher score indicates greater mid-stance activity. For the Hamstrings, 82% of variance was explained by PP1, capturing the overall level of activation. PP2 captured a difference between early stance, mid to late stance and late swing, where greater scores indicate greater mid to late stance activity. PP2 explained 6% of the waveform variance. PP3 explained 3% of the variance, capturing an amplitude difference between initial, mid and late stance.

Figure 3: Ensemble averaged quadriceps (VL/VM) electromyogram, collapsed across leg and muscle. Shaded region indicates one standard deviation above and below the asymptomatic waveform. The vertical line demarcates toe off (i.e. end of stance). EMG amplitude on the y-axis.
Figure 4: Ensemble averaged electromyogram of the contralateral and ipsilateral hamstrings (LH/MH), collapsed across muscle in individuals with hip OA. Shaded region indicates one standard deviation above and below the contralateral waveform. The vertical line demarcates toe off (i.e. end of stance). EMG amplitude on the y-axis.

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Competing Interest Statement

Authors have no competing interests pertaining to this manuscript.

Role of Funding Source

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References:


Burden, A., 2010. How should we normalize electromyograms obtained from healthy participants? What we have learned from over 25 years of research. J. Electromyogr. Kinesiol. 20, 1023-1035.


Figure 1:

Figure 2:
Figure 3:

Figure 4:
Table 1: Mean (SD) group demographics, Kellgren-Lawrence grades, gait velocity and questionnaire outcomes.

<table>
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<td></td>
</tr>
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<td>20 (5 female)</td>
</tr>
<tr>
<td>Age (years)</td>
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<td>59 (8)</td>
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<tr>
<td>Mass (kg)</td>
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Shading indicates significant between group difference (p<0.05)

* three individuals obtained magnetic resonance imaging to investigate Hip OA.
Table 2: Mean (Standard Deviation) knee joint ranges of motion in degrees

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<th>Asymptomatic Participants</th>
<th>Hip OA Participants</th>
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<tr>
<td>Initial Contact to Peak Flexion</td>
<td>15 (6)</td>
<td>16 (6)</td>
</tr>
<tr>
<td>during stance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak flexion to Peak extension</td>
<td>20 (6)</td>
<td>20 (5)</td>
</tr>
<tr>
<td>during stance</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Frontal Plane</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range of motion during stance</td>
<td>7 (2)</td>
<td>6 (2)</td>
</tr>
<tr>
<td><strong>Transverse Plane</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range of motion during stance</td>
<td>16 (4)</td>
<td>14 (5)</td>
</tr>
</tbody>
</table>

*Please refer to text for significant group and/or leg main effects and/or group x leg interactions*
Table 3: Mean (Standard Deviation) Principal Pattern scores for each Group, leg and muscle

<table>
<thead>
<tr>
<th></th>
<th>Asymptomatic Participants</th>
<th></th>
<th>Hip OA Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Ipsilateral</td>
</tr>
<tr>
<td>Vastus Lateralis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP1</td>
<td>366.7 (28.3)</td>
<td>372.5 (28.3)</td>
<td>369.4 (37.5)</td>
</tr>
<tr>
<td>PP2</td>
<td>-18.4 (46.5)</td>
<td>-2.9 (47.4)</td>
<td>-29.0 (61.4)</td>
</tr>
<tr>
<td>PP3</td>
<td>-25.6 (36.6)</td>
<td>-16.0 (51.6)</td>
<td>4.0 (54.6)</td>
</tr>
<tr>
<td>Vastus Medialis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP1</td>
<td>370.1 (26.3)</td>
<td>370.6 (37.0)</td>
<td>383.4 (39.2)</td>
</tr>
<tr>
<td>PP2</td>
<td>-3.5 (60.8)</td>
<td>12.6 (50.5)</td>
<td>7.9 (78.7)</td>
</tr>
<tr>
<td>PP3</td>
<td>-12.9 (39.0)</td>
<td>-26.1 (38.0)</td>
<td>26.2 (81.3)</td>
</tr>
<tr>
<td>Lateral Hamstrings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP1</td>
<td>377.2 (44.2)</td>
<td>386.4 (41.3)</td>
<td>363.3 (52.3)</td>
</tr>
<tr>
<td>PP2</td>
<td>-15.9 (101.4)</td>
<td>-14.8 (88.4)</td>
<td>1.1 (101.3)</td>
</tr>
<tr>
<td>PP3</td>
<td>-5.5 (76.5)</td>
<td>-3.8 (53.1)</td>
<td>-27.9 (68.0)</td>
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<tr>
<td>Medial Hamstrings</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>PP1</td>
<td>405.7 (43.2)</td>
<td>404.2 (44.6)</td>
<td>390.5 (43.9)</td>
</tr>
<tr>
<td>PP2</td>
<td>-21.0 (80.8)</td>
<td>-24.2 (73.6)</td>
<td>1.6 (111.4)</td>
</tr>
<tr>
<td>PP3</td>
<td>8.7 (60.5)</td>
<td>-3.8 (53.1)</td>
<td>-6.9 (65.7)</td>
</tr>
</tbody>
</table>

*Please refer to text for significant group, leg, muscle main effects and/or interactions*