



## Full Length Article

# Walking challenges in moderate knee osteoarthritis: A biomechanical and neuromuscular response to medial walkway surface translations



Matthew Baker<sup>a</sup>, William Stanish<sup>c</sup>, Derek Rutherford<sup>a,b,\*</sup>

<sup>a</sup> School of Physiotherapy, Faculty of Health, Dalhousie University, Halifax, NS, Canada

<sup>b</sup> School of Biomedical Engineering, Faculty of Engineering, Dalhousie University, Halifax, NS, Canada

<sup>c</sup> Department of Surgery, Division of Orthopaedics, Dalhousie University, Halifax, NS, Canada

## ARTICLE INFO

## Keywords:

Knee osteoarthritis  
 Perturbations  
 Joint instability  
 Gait biomechanics  
 Electromyography  
 Principal component analysis

## ABSTRACT

Sensations of knee instability are self-reported in 60–80% of individuals with knee osteoarthritis. These sensations are most often reported during walking; however, it remains unclear how they affect knee joint biomechanics and muscle activation patterns as indicators of joint function. Perturbation paradigms may provide insight into how the knee joint responds to walking challenges. Thus, the purpose of this study was to determine how individuals with moderate medial compartment knee osteoarthritis respond to unexpected, 3 cm medial walkway surface translations during gait compared to an asymptomatic control group. It is hypothesized that individuals with knee osteoarthritis will demonstrate altered biomechanics, and elevated and prolonged muscle activation compared to the asymptomatic group. Twenty asymptomatic individuals and 20 individuals with knee osteoarthritis walked on a dual-belt instrumented treadmill. Participants experienced 24 unexpected medial/lateral, 1 cm/3 cm walkway translations during mid-stance on each leg. Joint motions, moments and maximal voluntary isometric contraction amplitude normalized muscle activations were analyzed for the 3 cm walkway translations. Discrete measures were extracted from each biomechanical waveform and Principal Component Analysis (PCA) was used to determine knee joint muscle activation patterns. PCA is a factorization method to reduce dimensionality of EMG envelopes into linearly uncorrelated principal patterns (PP1, PP2, PP3) that explain the largest possible variance in the dataset. PP1 is often interpreted as a feature that explains the overall amplitude, while PP2 and PP3 are features that explain the variance in temporal activation patterns (i.e. how activation patterns change over the gait cycle). Statistical significance was determined using Analysis of Covariance models ( $\alpha = 0.05$ ). In response to the medial 3 cm walkway translation, increased activation amplitudes in the hamstring and gastrocnemius, captured by PP1 were found in both groups, as well as alterations in temporal activation patterns (captured by combinations of PP2 and PP3 patterns) across all muscle sites ( $p < 0.05$ ). No group differences were demonstrated in joint motion and moment discrete metrics ( $p > 0.05$ ) in response to the 3 cm translation. These findings suggest that the medial 3 cm walkway translation posed a challenge to knee function, however the biomechanical and neuromuscular response was similar between individuals with moderate knee osteoarthritis and asymptomatic individuals.

\* Corresponding author at: School of Physiotherapy, 3rd Floor Forrest Building, Dalhousie University, 5869 University Ave, PO Box 15000, Halifax, NS, Canada.

E-mail addresses: [mdb@dal.ca](mailto:mdb@dal.ca) (M. Baker), [wstanish@stanishortho.com](mailto:wstanish@stanishortho.com) (W. Stanish), [djrr@dal.ca](mailto:djr@dal.ca) (D. Rutherford).

<https://doi.org/10.1016/j.humov.2019.102542>

Received 7 December 2018; Received in revised form 18 July 2019; Accepted 21 October 2019

Available online 08 November 2019

0167-9457/ © 2019 Elsevier B.V. All rights reserved.

## 1. Introduction

Sixty to 80% of individuals with knee osteoarthritis (OA) report joint instability, defined by a sensation of buckling, shifting, or giving way of the knee (Felson et al., 2007; Knoop et al., 2012). These sensations have been associated with reduced knee joint function confidence (Skou, Wrigley, Metcalf, Hinman, & Bennell, 2014) and higher rates of falling (Felson et al., 2007), which may ultimately limit physical function.

Most often, sensations of instability are felt during walking compared to other dynamic tasks (Felson et al., 2007). Previous studies have investigated if gait alterations occur in individuals who self-reported instability compared to those who do not (Farrokhi, O'Connell, Gil, & Kelley Fitzgerald, 2013; Farrokhi, O'Connell, Gil, Sparto, & Fitzgerald, 2015; Gustafson, Robinson, Fitzgerald, Tashman, & Farrokhi, 2015). Individuals with OA who self-reported knee instability walked with greater sagittal plane excursion (Farrokhi et al., 2015), larger external knee flexion moments (Farrokhi, O'Connell, et al., 2013), and an overall increased sagittal plane motion variability (Gustafson et al., 2015). Unfortunately, dichotomy exists in explaining these findings. First, strategies may be used to counteract instability, altering the mechanical environment to ensure continued walking (Lewek, Rudolph, & Snyder-Mackler, 2004). Divergent, alterations found may create an environment where instability is more likely, and therefore people report this sensation (Farrokhi et al., 2015). Regardless, improving knee stability is considered a fundamental component of knee OA management (Farrokhi, Voycheck, Tashman, & Fitzgerald, 2013). Currently, predictable gait environments used to investigate knee OA pathomechanics and muscle activation patterns have limited our understanding of instability in the context walking.

Stability is often defined as the way a system behaves following a perturbation, and if the state of that system remains within specific boundaries of control (Grenier & McGill, 2007). The interrelationship between osteoligamentous, muscular and neural subsystems is fundamental to joint function, as muscle forces create moments that govern motions to preserve joint stability (Panjabi, 1992). Research is limited in knee OA gait to understand biomechanical and muscular responses that occur as a result of walking perturbations (Kumar, Swanik, Reisman, & Rudolph, 2014; Schmitt & Rudolph, 2008).

OA gait studies focussing on perturbations during gait have targeted medial and lateral walkway surface translations of different magnitudes, comparing different populations of individuals with knee OA and asymptomatic individuals as well as with varying levels of preparedness from experiencing or visualizing the perturbations prior to testing to completely random and unexpected movements (Baker, Rutherford, & Stanish, 2016; Kumar et al., 2014; Rutherford, Baker, & Stanish, 2016; Schmitt & Rudolph, 2008). Commonly, knee muscle activation is elevated, either specifically (i.e. increased medial co-contraction) (Schmitt & Rudolph, 2008) or generally (elevated gastrocnemius, hamstrings and quadriceps) (Kumar et al., 2014; Rutherford et al., 2016) in response to a perturbation, with knee biomechanical differences, such as knee motion and moments, less consistent (Baker et al., 2016; Kumar et al., 2014; Schmitt & Rudolph, 2008). Elevated muscle activation in individuals with knee OA has been found in addition to altered knee biomechanics, specifically a less dynamic sagittal plane moment in those with osteoarthritis (Rutherford, Baker, Wong, & Stanish, 2017b). This biomechanical feature has been interpreted as a knee stiffening strategy (Hatfield, Stanish, & Hubley-Kozey, 2015) thought to aid in stabilizing the knee joint. With multiple studies reporting elevated muscle activation and increased co-activity as a result of perturbations (Kumar et al., 2014; Rutherford et al., 2016; Schmitt & Rudolph, 2008), it is possible that reduced range of motion and less dynamic moments could be present as well. To date, understanding whether individuals with knee OA respond differently when compared to an asymptomatic group to unexpected walking surface translations in the medial/lateral direction is lacking.

In the perturbation protocols utilized previously (Kumar et al., 2014; Schmitt & Rudolph, 2008), participants were able to either observe (Kumar et al., 2014) or experience the perturbation (Schmitt & Rudolph, 2008) before testing and the surface translation was in one direction (lateral) at the same magnitude (5.8 cm). This methodology does not address the unexpected nature of instability reported by individuals with knee OA. Furthermore, studies (Baker et al., 2016; Rutherford et al., 2016; Schmitt & Rudolph, 2008) often lack a control group and thus it is unknown whether, like Kumar et al. found, individuals with knee OA respond to unexpected walking surface translations in a similar manner to those without (Kumar et al., 2014). An unclear understanding remains of how individuals with moderate knee OA respond to unexpected frontal plane walking challenges. With every step, individuals with medial compartment knee OA are responding to adduction moments that alter medial compartment load (Mundermann, Dyrby, D'Lima, Colwell, & Andriacchi, 2008) and may even induce lateral compartment lift-off (Kumar, Manal, & Rudolph, 2013) with instability often reported during walking. A response to unexpected medial translations may involve mechanical and neuromuscular factors that have implications for understanding how stability may be maintained in this group.

The purpose of this study was to determine how individuals with moderate medial compartment knee OA respond, biomechanically and through altered muscle activation patterns, to unexpected medial walkway surface translations during gait compared to an asymptomatic control group. As a response to walkway surface translations, compared to the asymptomatic group, it is hypothesized that individuals with knee OA will walk with less sagittal plane knee motion, reduced sagittal plane external knee moment dynamics and elevated and prolonged gastrocnemius, hamstring and quadriceps activation.

## 2. Methods

### 2.1. Participants

Individuals with moderate medial compartment knee OA (MOA) were recruited from local orthopaedic clinics, diagnosed using American College of Rheumatology guidelines (Altman et al., 1986). Individuals were classified as MOA using self-reported functional capabilities (Hubley-Kozey, Deluzio, Landry, McNutt, & Stanish, 2006). Individuals were excluded if they were candidates for

total knee arthroplasty. An age-matched, asymptomatic group (ASYM) was recruited locally, through advertisements and considered a sample of convenience. Participants were included if they were over the age of 50 years, reported no cardiovascular/respiratory disease or neurological disorders and no fractures/injury other than a sprain or strain within the last year. The protocol was approved by local ethics review committee (Romeo#:1020825).

## 2.2. Data collection

Participants completed the Knee Outcome Survey - Activities of Daily Living Scale (KOS-ADLS), Knee Injury and Osteoarthritis Outcome Score (KOOS) prior to perturbation testing. Participants changed into fitted shorts, T-shirt and removed footwear. Height, weight, and mid-thigh and mid-shank circumferences were measured. Participants then walked back-and-forth across the GAITRite™ (CIR Systems Inc., USA) pressure sensitive walkway using previously reported methods (Rutherford, Baker, Wong, & Stanish, 2017a) to acquire self-selected gait speed.

Passive, retro-reflective skin surface markers were affixed to each participant. Rigid clusters (foot, shank, thigh, pelvis, thorax) and individual markers fixed to the lateral aspect of the shoulders below the acromion, spinous process of the 7th cervical vertebra, greater trochanters, lateral/medial femoral and tibial epicondyles, lateral/medial malleoli, head of the 1st and 5th metatarsal, atop the 2nd metatarsal, and posterior heel were placed bilaterally (Rutherford et al., 2017a). Virtual points were collected to define sternal notch, and the left and right anterior superior iliac spines. Retro-reflective marker motions were sampled at 100 Hz using eight Qualisys® OQUS 500 (Qualisys®, Sweden) motion analysis cameras. Ground reaction forces and moments were sampled at 2000 Hz from the R-Mill (Motekforce Link, Netherlands) dual-belt instrumented treadmill.

Surface electromyography (EMG) was completed using standard procedures (Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). Skin was shaved and cleaned with alcohol wipes (70% alcohol) and 10 mm diameter electrodes (Ag/AgCl, 30 mm inter-electrode distance, Red Dot, 3M Health Care, USA) were affixed bilaterally, in a bipolar configuration, over vastus medialis (VM), vastus lateralis (VL), rectus femoris (RF), medial (MH) and lateral hamstrings (LH), and medial (MG) and lateral gastrocnemius (LG). A reference electrode was affixed to the tibia. Surface EMG was recorded at 2000 Hz using two AMT-8™ 8-channel EMG systems (input impedance: ~10 Gohm, Common Mode Rejection Ratio (CMRR): 115 dB at 60 Hz, band-pass (10–1000 Hz)) (Bortec Inc., Canada) and synchronized with kinematic and kinetic data using Qualisys Track Manager V2.10 (Qualisys®, Sweden).

Participants were harnessed to the ceiling using an upper body system, while walking barefoot on the treadmill, at the speed determined by the GAITRite™. After a 6-minute acclimatization period (Rutherford et al., 2017a), participants were informed the perturbation protocol would begin. The participants had not witnessed treadmill translation capabilities prior to testing and no practice trials were permitted. The perturbation protocol contained three blocks of eight unexpected, random 1 cm and 3 cm medial and lateral treadmill surface translations initiated during mid-stance on each leg. This block of eight perturbations was repeated three times. If participants, used side supports or stepped onto the other treadmill belt, the trial was excluded from analysis. Participants were blinded to perturbation occurrence, direction, and magnitude.

After gait testing, a supine resting muscle bias was recorded. A Humac Norm Isokinetic Dynamometer (Computer Sports Medicine Inc., USA) was used to elicit maximal voluntary isometric contractions (MVIC). Gravity corrected knee flexor and extensor torque and muscle activation were tested at 45° of knee flexion (Rutherford et al., 2017a). Standing unilateral plantarflexion was also completed (Hubley-Kozey et al., 2006). Following one practice contraction, two, 3-second MVIC trials were completed, separated by 40-s of rest. Standard verbal encouragement was given.

## 2.3. Data processing

Custom programs were written in MatLab™ 2015b (Mathworks Inc., USA). All kinematic data were low-pass filtered (Butterworth 4th order, 6 Hz - recursive) and three-dimensional ground reaction forces and moments were low-pass filtered (Butterworth 4th order, 30 Hz - recursive) prior to processing. Joint angles were calculated using Cardan/Euler rotations and described as the distal segment moving about a fixed proximal segment (Rutherford et al., 2017a). External joint moments were calculated using inverse dynamics (Vaughan, Davis, & O'Connor, 1999) and projected onto the joint coordinate system (Grood & Suntay, 1983). Moments were low-pass filtered (Butterworth 4th order, 10 Hz - recursive) and normalized to body mass (Nm/kg). Raw EMG signals were band-pass filtered (Butterworth 4th order, 10–500 Hz - recursive), corrected for resting bias, full-wave rectified, and low-pass filtered (Butterworth 4th order, 6 Hz - recursive). EMG gait waveforms were amplitude normalized using the highest 100 ms moving average window (99 ms overlap) from MVIC trials (Hubley-Kozey et al., 2006). Angle and EMG waveforms were time normalized to the gait cycle. Moments were time normalized to stance phase.

## 2.4. Data analysis

The most symptomatic leg was chosen for the MOA group, while a random leg was determined for the ASYM group. Three strides preceding each perturbation were ensemble averaged to represent baseline (T0). The first stride after the perturbation was obtained to represent the response (T1). The range from initial contact to peak stance flexion, the range from peak stance flexion to minimum late stance flexion were calculated from joint motions and the difference from peak knee moment flexion to extension and peak knee adduction moment were calculated from joint moments, as previously reported (Rutherford et al., 2017a). These motion and moment planes have also been investigated in the context of knee stability (Farrokhi et al., 2015; Gustafson et al., 2015). These discrete gait metrics were calculated for statistical analysis.

Principal component analysis (PCA) was used to capture mutually uncorrelated amplitude and temporal EMG waveform features (Principal Patterns [PPs]) that together described at least 90% of data set variability (Hubley-Kozey et al., 2006). To analyze the data using PCA, original waveform data were organized into matrix [X] for each muscle group containing T0 and T1 waveforms from participants. A cross-product matrix was computed  $[S] = [X]^*[X]$ . An eigenvector decomposition of [S] was completed to yield the eigenvectors (PPs) and eigenvalues (Rutherford et al., 2017a). PPs that hierarchically explained the waveform variance (PP1, PP2, etc.) were retained. PP-scores were computed for each participant to provide a weighting coefficient relating the PP to each measured waveform. The use of this multivariate statistical technique has been used to understand muscle activation patterns in knee OA gait (Hubley-Kozey et al., 2006).

### 2.5. Statistical analysis

Student's *t*-test was used to determine between groups differences for subject demographics, KOOS scores, strength and walking speed. Assumptions of equal variance and normality were examined using Kolmogorov-Smirnov and Levene's test for continuous variables. If assumptions were violated, data were transformed using a Johnson transformation. A two-factor, mixed model Analysis of Covariance (ANCOVA), adjusting for the effects of walking velocity, was used for biomechanical variables to determine between and within group (time) main effects and interactions. For muscle data, a three-factor, mixed model ANCOVA, adjusting for walking velocity, was used to determine between and within group (muscle, time) main effects and interactions. Bonferonni post hoc testing was completed for all significant effects. *P*-values were adjusted to  $\alpha = 0.05$  depending on the number of comparisons. Statistical testing was completed using Minitab™ Ver.17 (Minitab Inc., USA).

### 3. Results

Table 1 provides group demographics, anthropometrics, walking velocity and self-report survey scores. MOA group Kellgren Lawrence grade was reported. No significant differences were found in age ( $p = 0.946$ ) and height ( $p = 0.864$ ) between groups. The MOA group was heavier ( $p < 0.001$ ) with a larger BMI ( $p < 0.001$ ) and walked with slower velocity ( $p = 0.004$ ) compared to the ASYM group. The KOS instability question suggests 60% of MOA participants reported this sensation, with 40% reporting it impacted their activity. The MOA group demonstrated worse KOOS ( $p < 0.001$ ) scores compared to the ASYM group. No significant between group strength differences were found for knee extensors ( $p = 0.195$ ) and flexors ( $p = 0.067$ ).

Only medial perturbations were analyzed. On average, the translation occurred at 40% ( $\pm 6\%$ ) of stance (i.e. 24% ( $\pm 4\%$ ) of the gait cycle). The average translation distance was 31.8 mm ( $\pm 0.007$  mm) at a mean rate of 0.11 m/s ( $\pm 0.0029$  m/s). For all medial 3 cm translations experienced, the ASYM group completed 47/60 and the MOA group completed 49/60 without stepping onto the other treadmill belt or using side supports (Table 1).

Given the study objective, only outcomes based on perturbation responses are reported (time or time interactions). In both groups, no significant differences in knee biomechanics were found in response to the medial 3 cm translation (Table 2). This included; i) knee range of motion between initial contact and peak stance knee flexion ( $p > 0.895$ ), ii) range of motion between peak stance

**Table 1**

Means and standard deviations for subject group demographics, gait characteristics, self-report survey outcomes, radiographic grade distribution (Moderate OA only), and strength measures. \*Indicate significant between-group differences ( $p < 0.05$ ).

	Asymptomatic	Moderate OA
Variable		
N	20	20
Sex (M:F)	11:9 (55% female)	10:10 (50% female)
Age (years)	62 (7)	62 (7)
Height (m)	1.69 (0.08)	1.69 (0.09)
Mass (kg)	67.4 (11.7)*	85.3 (13.9)*
BMI (kg/m <sup>2</sup> )	23.7 (3.2)*	30.3 (4.5)*
Walking velocity (m/s)	1.17 (0.12)*	1.05 (0.14)*
KOS stability score	[20] 5	[8]5 - [4]4 - [1]3 - [6]2 - [1]1
Successful perturbations (n/60)	47	49
KOOS	-	-
Symptoms (n/100)	97.9 (4.7)*	62.3 (13.9)*
Pain (n/100)	98.6 (3.7)*	64.6 (16.1)*
Activities of daily living (n/100)	99.5 (1.6)*	67.2 (22.3)*
Quality of life (n/100)	97.5 (6.4)*	46.9 (17.7)*
Radiographic grade (n)	-	-
KL I	-	6
KL II	-	12
KL III	-	2
KL IV	-	0
Strength	-	-
Knee extension - 45° (Nm)	116.8 (31.8)	129.0 (53.4)
Knee flexion - 45° (Nm)	72.3 (16.9)	75.6 (38.1)

**Table 2**

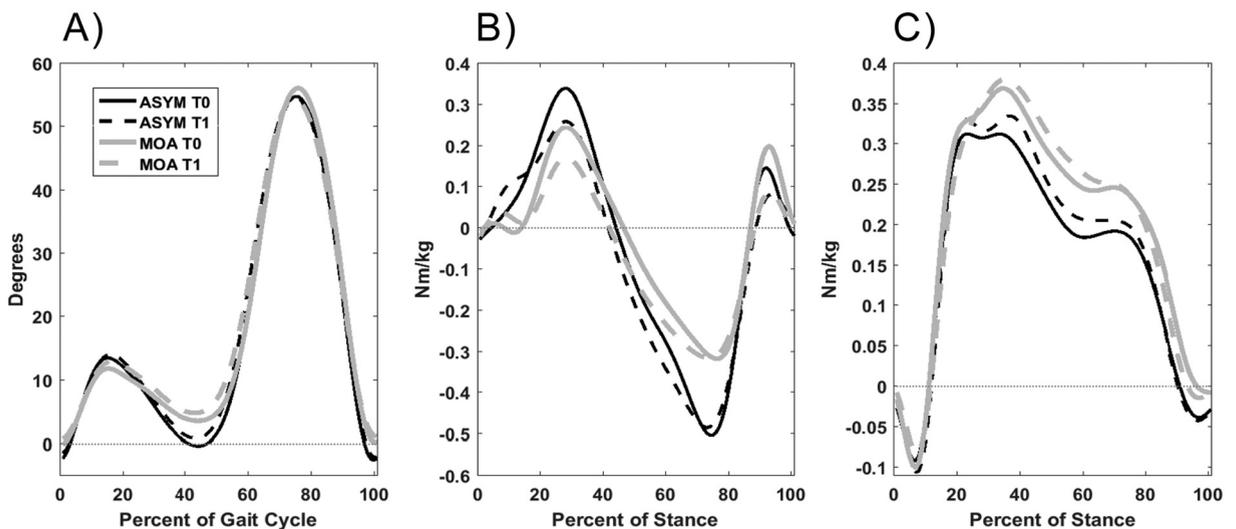
P-values for biomechanics and electromyography ANCOVA models. \*Indicates significant outcomes ( $p < 0.05$ ) that were post-hoc tested.

Biomechanics							
Variable	Group		Time		Group*Time		
Initial contact to peak flexion range of motion	0.064		0.895		0.759		
Peak stance flexion to minimum late stance flexion range of motion	0.010*		0.854		0.647		
Sagittal plane knee moment flexion to extension difference	0.014*		0.325		0.622		
Peak knee adduction moment	0.007*		0.338		0.734		
EMG							
Variable	Group	Time	Muscle	Group* Time	Group* Muscle	Muscle* Time	Group* Time* Muscle
Quadriceps	-	-	-	-	-	-	-
PP1	0.168	0.061	< 0.001*	0.690	0.965	0.910	0.974
PP2	< 0.001*	< 0.001*	< 0.001*	0.704	0.825	0.919	0.979
PP3	0.546	0.003*	0.187	0.309	0.956	0.618	0.884
Hamstring	-	-	-	-	-	-	-
PP1	0.922	0.001*	0.275	0.531	< 0.001*	0.570	0.634
PP2	0.132	< 0.001*	0.041	0.374	0.183	0.977	0.268
PP3	0.914	0.509	0.626	0.645	0.067	0.416	0.951
Gastrocnemius	-	-	-	-	-	-	-
PP1	0.580	0.005*	0.002*	0.942	0.118	0.336	0.741
PP2	0.711	0.691	< 0.001*	0.229	0.082	0.016*	0.328
PP3	0.479	< 0.001*	0.727	0.226	0.243	0.014*	0.939

flexion and late stance extension ( $p > 0.854$ ), iii) sagittal plane moment range, ( $p > 0.325$ ) and iv) peak knee adduction moment ( $p > 0.338$ ). Sagittal plane motion, moments and frontal plane moments in response to the perturbations are shown on Fig. 1.

Gastrocnemius electromyograms, PPs and PP interpretations are found in Fig. 2. Time effects were evident in gastrocnemius PP1-scores ( $p = 0.005$ ). PP1-scores were higher at T1 compared to T0. A time\*muscle interaction was found for PP3-scores ( $p = 0.014$ ) where MG PP3-scores were higher at T1 compared to T0, while the LG PP3-scores were the same at T0 and T1. Between MG and LG, PP3-scores were similar at T0 and T1. Higher PP1-scores suggest overall gastrocnemius activation amplitudes were increased at T1, whereas PP3 results suggest MG activation increased earlier (i.e. greater early stance activation) with respect later stance in response to the translation.

Fig. 3 illustrates the hamstring electromyograms, PPs and PP interpretations. Time main effects were found for hamstring PP1-scores ( $p = 0.001$ ) and PP2-scores ( $p < 0.001$ ). LH and MH PP1-scores and PP2-scores were greater at T1 compared to T0. Higher



**Fig. 1.** (A) Ensemble averaged knee sagittal plane motion time normalized to the gait cycle (B) Ensemble averaged net external sagittal plane knee moment time normalized to stance phase and amplitude normalized to body mass. Positive values indicate a net external flexion moment and negative values indicated external extension moments. (C) Ensemble averaged net external frontal plane knee moment time normalized to stance phase and amplitude normalized to body mass. Positive values indicate a net external adduction moment.

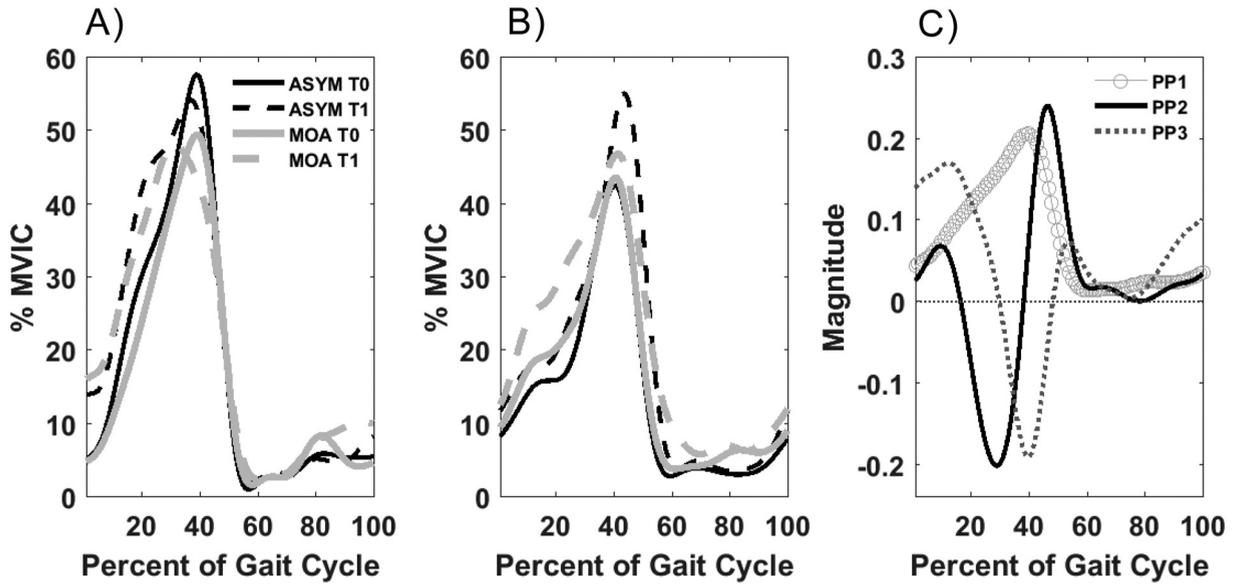


Fig. 2. Ensemble averaged (A) MG and (B) LG amplitude normalized to % MVIC. (C) Three PPs captured 96% of waveform variability. PP1 captured overall magnitude and shape, explaining 86% of waveform variability. PP2 captures a phase shift in activation where higher scores indicate a delayed activity and explained 6% of waveform variability. PP3 captured a difference operator between early and late stance phase and explained 4% of waveform variability, where higher scores indicate a lower difference.

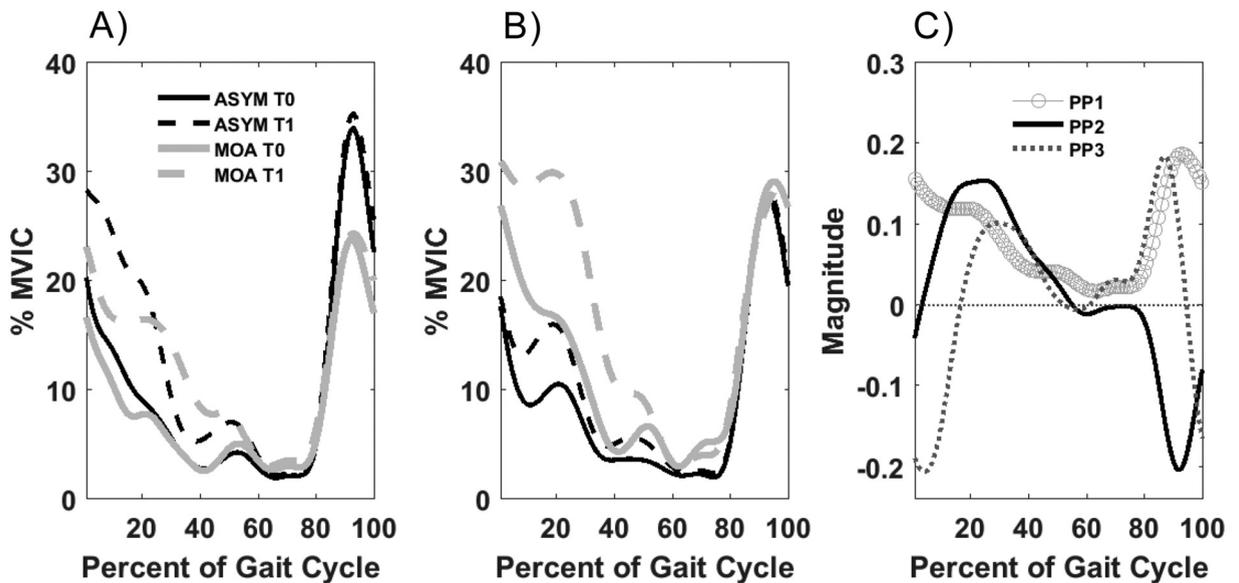


Fig. 3. Ensemble averaged (A) MH and (B) LH amplitude normalized to % MVIC. (C) Three PPs captured 93% of waveform variability. PP1 captured overall magnitude and shape, explaining 80% of waveform variability. PP2 captures prolonged elevated activation during early stance where higher scores indicate more prolonged activation and explained 9% of waveform variability. PP3 captured a difference between early and late stance activation, where higher scores indicate a greater late stance activity, explaining 4% of waveform variability.

hamstring *PP1*-scores at T1 suggest higher activation amplitudes over the gait cycle, where higher hamstring *PP2*-scores suggests this activation is prolonged during early stance in response to the translation.

Fig. 4 illustrates PPs, and PP interpretations and electromyograms of VL and VM. No significant time effect or time interactions were found for *PP1*-scores. Time main effect was found for quadriceps *PP2*-scores ( $p < 0.001$ ) and *PP3*-scores ( $p = 0.003$ ). At T1, individuals demonstrate higher *PP2*-scores and lower *PP3*-scores, suggesting the quadriceps activation is less dynamic in response to the translation with greater activation during midstance (*PP2*-scores) less differential activation between early/mid stance and swing phases (*PP3*-scores).

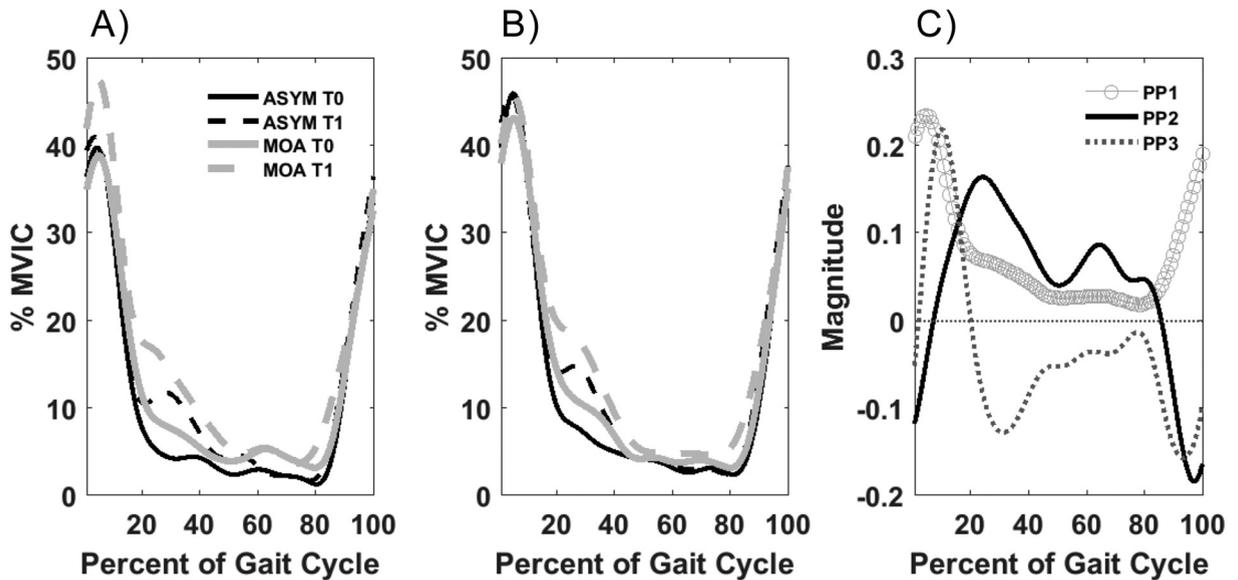


Fig. 4. Ensemble averaged (A) VM and (B) VL amplitude normalized to % MVIC. (C) Three PPs captured 97% of the waveform variability. PP1 captured overall magnitude and shape explaining 92% of waveform variability. PP2 captured prolonged activation during stance explaining 3% of waveform variability. High PP3-scores captured a greater difference between early-to-mid stance and swing phases, explaining 2% of waveform variability.

#### 4. Discussion

This study aimed to determine how individuals with moderate medial compartment knee OA respond, biomechanically and through altered muscle activation patterns, to unexpected medial walkway surface translations during gait compared to an asymptomatic control group. Based on previous work, the hypothesis was that individual with knee OA would demonstrate reduced sagittal plane knee motion, reduced sagittal plane external knee moment dynamics and increased activation amplitudes and prolonged activation patterns across all muscle sites. Study results do not fully support the hypothesis of reduced sagittal plane motion and moment ranges and elevated and prolonged muscle action across all muscles sites in the MOA group compared to the ASYM group after medial 3 cm walkway translations. This study demonstrated no statistically significant biomechanical alterations in both groups between T0 and T1. Altered muscle activations were evident, with elevated activation in hamstrings and gastrocnemius (*PP1-Scores*) and temporal alterations in all muscles (*PP2 and PP3-Scores*) at T1 compared to T0.

Detailed in Table 1, the MOA group was heavier, walked slower and self-reported worse KOOS outcomes compared to the ASYM group. The MOA group also demonstrated a median KL-II grade. Kumar et al. reported similar descriptive characteristics of their OA group, reporting more pain, knee related symptoms, and functional restrictions compared to controls, as well as similar radiographic severity (median KL-II grade) (Kumar et al., 2014). Both Kumar et al. and the present study reported similar neuromuscular findings, however, the results contrasted in terms of changes in knee biomechanics as a result of challenged walking. Kumar et al. reported less knee flexion excursion during the loading responses after a perturbation compared to baseline in both the OA and control groups. The current results suggest no changes in either sagittal plane knee motion metrics, as well as no alterations in the sagittal and frontal plane knee moments. This could be explained by contrasting methods as Kumar et al. used larger (58 mm) and faster (0.4 m/s) perturbations (Kumar et al., 2014). A perturbation larger in magnitude, and quicker in speed may provide a larger challenge to the groups, which could result in contrasting biomechanical alterations with the current work. Despite the smaller perturbations resulting in a lack of biomechanical change, significant changes in knee muscle activation were present.

It was hypothesized that muscle activation alterations would be present in the MOA group compared to the ASYM participants in response to perturbations, as elevated and altered muscle activation has been discussed previously to assist with providing stability during walking in the OA population (Hubble-Kozey et al., 2006; Kumar et al., 2014). The results of this study did not fully support this hypothesis as muscle alterations were present, however, they were not different between the MOA and ASYM groups. Kumar et al. reported a general increase in MVIC normalized, mean loading response muscle activation in medial and lateral quadriceps, hamstring and gastrocnemius. Our response was similar, with elevated *PP1-scores* found in the hamstring and gastrocnemius, elevated *PP2-scores* found in the quadriceps and hamstrings and altered *PP3-scores* were found in the quadriceps and hamstrings. Elevated *PP1-scores* have been interpreted as increase overall activation, while elevated *PP2-scores* have been interpreted as prolonged activation during stance. Schmitt and Rudolph also demonstrated neuromuscular alterations in response of a perturbation, demonstrating elevated medial co-contraction in combination with no biomechanical alterations (Schmitt & Rudolph, 2008), supporting the results of our study. This however, must be taken in light of different muscular normalization strategies and studying a population of OA individuals classified as stable and unstable based on the presence of self-reported instability. The neuromuscular responses

demonstrated by both the MOA and ASYM in response to a perturbation are in the direction of the patterns reported in OA gait literature. Rutherford, Hubley-Kozey, & Stanish, 2013 reported that quadriceps and hamstrings develop a pattern of elevated and more prolonged activation as the OA disease becomes more radiographically severe (Rutherford et al., 2013), suggesting that a response to a perturbation may be similar to how individuals with OA walk day-to-day. With the majority of individuals with OA self-reporting sensations of instability (van der Esch et al., 2012), it may be that as individuals with OA consistently respond to gait challenges from sensations of buckling, shifting or giving way, that a habitual pattern of elevated and prolonged activation develops to counteract sensations of instability.

Knee instability is self-reported in 60–80% of individuals with OA (van der Esch et al., 2012), however it remains unclear how this symptom impacts gait mechanics in moderate and severe OA subpopulations. Given the implications of stability for rehabilitation (Farrokhi, Voycheck, et al., 2013); understanding how individuals in an early disease state respond to challenges is important. KOS scores specific to instability have not been reported for a MOA group specifically, however the current findings establish that approximately 40% report these symptoms impacting their activity in some manner, slightly less than the general findings of those with OA (van der Esch et al., 2012). During gait however, translating these subjective reports to biomechanical and muscle activation outcomes is less clear, while joint function is altered in the MOA state, these alterations may not have significant impacts on maintaining joint function during gait challenges.

Principal Component Analysis was employed to reduce the dimensionality of EMG envelopes (Hubley-Kozey et al., 2006), however other methods such as Non-Negative Matrix Factorization (NNMF) used for the same purpose, have been presented (Ting & Macpherson, 2005). Factorization methods identify data features that explain maximal amounts of variance (Deluzio, Wyss, Zee, Costigan, & Sorbie, 1997). While no method is ideally suited for the analysis of EMG, addressing the pros and cons of each method is appropriate (Naik, Selvan, Gobbo, Acharyya, & Nguyen, 2016). Features selected by NNMF identify possible combinations of muscle activity and is a method of understanding muscle synergies and co-activations, while features selected by PCA help to explain the major direction of activation and is a method of determining patterns of activation that occur in a dataset (Ting & Chvatal, 2010). Based on the studies objectives, PCA was suited to evaluate EMG activation features in response to the gait perturbations used in this study.

The results from this study need to be interpreted considering certain limitations. The treadmill was translated at its maximum rate and is limited to a distance of 5 cm from midline. Magnitudes of 1 and 3 cm were chosen for this study to ensure random perturbations (direction, leg) could be employed. Given the rate and magnitude and perturbations initiated during mid-stance (~40% of stance), there may have been some instances where the treadmill was still moving when the contralateral foot contacted the treadmill. While all perturbations were delivered in single-leg stance, the stimulus for response was standardized, yet we could not control for the response. Secondly, fitness level was not controlled, which may influence one's ability to respond to a perturbation. Less fit individuals may demonstrate altered neuromuscular and/or biomechanical response to perturbations compared to with a higher fitness level. KOOS scores suggest that function was limited in the MOA group, however the perturbation responses were generally the same between groups. Further work to understand how fitness level impacts perturbation responses is warranted. Lastly, not all individuals were able to successfully complete all 3 cm perturbations (Table 1). The block of perturbations took approximately 24-min to complete after a 6-minute warm-up. Each participant completed at least one perturbation successfully. It was not possible to extend testing time on an individual basis to ensure all perturbations were completed. This also impacts the random nature of perturbations.

## 5. Conclusion

Both ASYM and MOA groups responded to unexpected medial walkway surface translations, with no biomechanical alterations, and features of elevated and prolonged muscle activation, indicative of a stabilizing strategy. The findings do not suggest OA specific neuromuscular response, but future studies could focus on groups of individuals who report that instability impacts their functional abilities. Perturbation protocols that challenge stability may have utility in understanding mechanisms of knee OA gait pathomechanics.

## Acknowledgements

We would like to thank the participants for their time and efforts in helping with this study and would like to acknowledge Jereme Outerleys for his technical expertise and contributions. Furthermore, we would like to thank Dr. Ivan Wong and Dr. Nathan Urquhart for participant recruitment.

## Role of funding source

Support for this study was provided by the Nova Scotia Health Research Foundation Development and Innovation Grant (MED-DI-2014-8668). The sponsors had no role in the design and conduct of this study; collection analysis, and interpretation of data; preparation, review or approval of the manuscript; and decision to submit the manuscript for publication.

## Declaration of competing interest

Authors have no competing interests pertaining to this manuscript.

## References

- Altman, R., Asch, E., Bloch, D., Bole, G., Borenstein, D., Brandt, K., ... Howell, D. (1986). D., K., Koopman, W., Longley, S., Mankin, H., McShane, D. J., Medsger, T., Meenan, R., Mikkelsen, W., Moskowitz, R., Murphy, W., Rothschild, B., Segal, M., Sokoloff, L., & Wolfe, F. *Development of criteria for the classification and reporting of osteoarthritis. Arthritis & Rheumatism*, 28, 1039–1049.
- Baker, M., Rutherford, D., & Stanish, B. (2016). Walking challenges in moderate knee osteoarthritis: A biomechanical response to medial/lateral walkway surface perturbations. *Osteoarthritis and Cartilage*, 24, S124–S125.
- Deluzio, K. J., Wyss, U. P., Zee, B., Costigan, P. A., & Sorbie, C. (1997). Principal component models of knee kinematics and kinetics: Normal vs. pathological gait patterns. *Human Movement Science*, 16, 201–217.
- Farrokhi, S., O'Connell, M., Gil, A., & Kelley Fitzgerald, G. (2013). Alterations in sagittal-plane knee joint kinematics and kinetics during gait in knee osteoarthritis patients with complaints of instability. *Osteoarthritis and Cartilage*, 21.
- Farrokhi, S., O'Connell, M., Gil, A. B., Sparto, P. J., & Fitzgerald, G. K. (2015). Altered gait characteristics in individuals with knee osteoarthritis and self-reported knee instability. *The Journal of Orthopaedic and Sports Physical Therapy*, 45, 351–359.
- Farrokhi, S., Voycheck, C. A., Tashman, S., & Fitzgerald, G. K. (2013). A biomechanical perspective on physical therapy management of knee osteoarthritis. *The Journal of Orthopaedic and Sports Physical Therapy*, 43, 600–619.
- Felson, D. T., Niu, J., McClellan, C., Sack, B., Aliabadi, P., Hunter, D. J., ... Englund, M. (2007). Knee buckling: Prevalence, risk factors, and associated limitations in function. *Annals of Internal Medicine*, 147, 534–540.
- Grenier, S. G., & McGill, S. M. (2007). Quantification of lumbar stability by using 2 different abdominal activation strategies. *Archives of Physical Medicine and Rehabilitation*, 88, 54–62.
- Grood, E. S., & Suntay, W. J. (1983). A joint coordinate system for the clinical description of three-dimensional motions: Applications to the knee. *Transactions of the ASME*, 105, 136–144.
- Gustafson, J. A., Robinson, M. E., Fitzgerald, G. K., Tashman, S., & Farrokhi, S. (2015). Knee motion variability in patients with knee osteoarthritis: The effect of self-reported instability. *Clinical Biomechanics (Bristol, Avon)*, 30, 475–480.
- Hatfield, G. L., Stanish, W. D., & Hubley-Kozey, C. L. (2015). Three-dimensional biomechanical gait characteristics at baseline are associated with progression to total knee arthroplasty. *Arthritis Care Res (Hoboken)*, 67, 1004–1014.
- Hermens, H. J., Freriks, B., Disselhorst-Klug, C., & Rau, G. (2000). Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology*, 10, 361–374.
- Hubley-Kozey, C. L., Deluzio, K. J., Landry, S. C., McNutt, J. S., & Stanish, W. D. (2006). Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. *Journal of Electromyography and Kinesiology*, 16, 365–378.
- Knoop, J., van der Leeden, M., van der Esch, M., Thorstenson, C. A., Gerritsen, M., Voorneman, R. E., ... Steultjens, M. P. (2012). Association of lower muscle strength with self-reported knee instability in osteoarthritis of the knee: Results from the Amsterdam osteoarthritis cohort. *Arthritis Care Res (Hoboken)*, 64, 38–45.
- Kumar, D., Manal, K. T., & Rudolph, K. S. (2013). Knee joint loading during gait in healthy controls and individuals with knee osteoarthritis. *Osteoarthritis and Cartilage*, 21, 298–305.
- Kumar, D., Swanik, C. B., Reisman, D. S., & Rudolph, K. S. (2014). Individuals with medial knee osteoarthritis show neuromuscular adaptation when perturbed during walking despite functional and structural impairments. *Journal of Applied Physiology*, 116, 13–23.
- Lewek, M. D., Rudolph, K. S., & Snyder-Mackler, L. (2004). Control of frontal plane knee laxity during gait in patients with medial compartment knee osteoarthritis. *Osteoarthritis and Cartilage*, 12, 745–751.
- Mundermann, A., Dyrby, C. O., D'Lima, D. D., Colwell, C. W., Jr., & Andriacchi, T. P. (2008). In vivo knee loading characteristics during activities of daily living as measured by an instrumented total knee replacement. *Journal of Orthopaedic Research*, 26, 1167–1172.
- Naik, G. R., Selvan, S. E., Gobbo, M., Acharyya, A., & Nguyen, H. T. (2016). Principal component analysis applied to surface electromyography: A comprehensive review. *IEEE Access*, 4, 4025–4037.
- Panjabi, M. M. (1992). The stabilizing system of the spine. Part I. function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders*, 5, 383–389.
- Rutherford, D., Baker, M., Wong, I., & Stanish, W. (2017a). Dual-belt treadmill familiarization: Implications for knee function in moderate knee osteoarthritis compared to asymptomatic controls. *Clinical Biomechanics (Bristol, Avon)*, 45, 25–31.
- Rutherford, D., Baker, M., Wong, I., & Stanish, W. (2017b). The effect of age and knee osteoarthritis on muscle activation patterns and knee joint biomechanics during dual belt treadmill gait. *Journal of Electromyography and Kinesiology*, 34, 58–64.
- Rutherford, D. J., Baker, M., & Stanish, B. (2016). Muscle activation responses to medial and lateral walkway perturbations during gait in individuals with moderate knee osteoarthritis. *Osteoarthritis and Cartilage*, 24, S115–S116.
- Rutherford, D. J., Hubley-Kozey, C. L., & Stanish, W. D. (2013). Changes in knee joint muscle activation patterns during walking associated with increased structural severity in knee osteoarthritis. *Journal of Electromyography and Kinesiology*, 23, 704–711.
- Schmitt, L. C., & Rudolph, K. S. (2008). Muscle stabilization strategies in people with medial knee osteoarthritis: The effect of instability. *Journal of Orthopaedic Research*, 26, 1180–1185.
- Skou, S. T., Wrigley, T. V., Metcalf, B. R., Hinman, R. S., & Bennell, K. L. (2014). Association of knee confidence with pain, knee instability, muscle strength, and dynamic varus-valgus joint motion in knee osteoarthritis. *Arthritis Care & Research*, 66, 695–701.
- Ting, L. H., & Chvatal, S. A. (2010). Decompositng muscle activity in motor tasks: Methods and interpretation. In F. Danion, & M. L. Latash (Eds.). *Motor control: Theories, experiments, and applications* (pp. 102–121). New York, NY: Oxford University Press.
- Ting, L. H., & Macpherson, J. M. (2005). A limited set of muscle synergies for force control during a postural task. *Journal of Neurophysiology*, 93, 609–613.
- van der Esch, M., Knoop, J., van der Leeden, M., Voorneman, R., Gerritsen, M., Reiding, D., ... Roorda, L. D. (2012). Self-reported knee instability and activity limitations in patients with knee osteoarthritis: Results of the Amsterdam osteoarthritis cohort. *Clinical Rheumatology*, 31, 1505–1510.
- Vaughan, C. L., Davis, B. L., & O'Connor, J. C. (1999). *Dynamics of human gait 2nd edition*. Cape Town, South Africa: Kiboho Publishers.