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A comparison of muscle activation and knee mechanics during gait between patients with non-traumatic and post-traumatic knee osteoarthritis

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SUMMARY

Objective: The objective was to compare muscle activation and knee mechanics during gait between participants with non-traumatic knee osteoarthritis (OA), post-traumatic knee OA, and healthy adults.
Design: Participants with non-traumatic knee OA ($n = 22$), post-traumatic knee OA ($n = 19$), and healthy adults ($n = 22$) completed gait trials for this observational, cross-sectional study. Post-traumatic OA group had a history of traumatic anterior cruciate ligament (ACL) rupture. Surface electromyography (EMG) measured activation of seven lower extremity muscles. Motion capture cameras and force plates measured motion and force data. Principal component analysis (PCA) determined waveform characteristics (principal components) from EMG, knee angle, and knee external moment waveforms. Analysis of variance (ANOVA) examined group differences in principal component scores (*PC-scores*). Regression analyses examined if a variable that coded for OA group could predict *PC-scores* after accounting for disease severity, alignment, and lateral OA.
Results: There was lower gastrocnemius EMG amplitudes ($P < 0.01$; ANOVA) in the post-traumatic OA group compared to healthy group. Non-traumatic OA group had higher vastus lateralis, vastus medialis, and rectus femoris EMG compared to post-traumatic OA group ($P = 0.01$ to 0.04) in regression analyses. Also, non-traumatic OA group had higher and prolonged lateral hamstring EMG compared to healthy ($P = 0.03$; ANOVA) and post-traumatic OA ($P = 0.04$; regression) groups respectively. The non-traumatic OA group had lower knee extension ($P < 0.05$) and medial rotation ($P < 0.05$) moments than post-traumatic and healthy groups.
Conclusions: Muscle activation and knee mechanics differed between participants with non-traumatic and post-traumatic knee OA and healthy adults. These OA subtypes had differences in disease characteristics that may impact disease progression.

Introduction

People that sustain a traumatic knee injury are 3.8 times more likely to develop knee osteoarthritis (OA)[1]. Further evidence that trauma leads to OA is supported by the fact that 51% of women developed radiographic knee OA 12 years after sustaining an anterior cruciate ligament (ACL) injury[2]. Knee OA can be classified as non-traumatic or post-traumatic. OA that develops in patients

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without a history of trauma is classified as non-traumatic, while OA that develops following trauma is classified as post-traumatic.

Disease characteristics differ between non-traumatic and post-traumatic knee OA subtypes[3]. Radiographic OA-related structural changes (e.g., osteophytes) are more equally distributed between medial and lateral knee compartments in patients with post-traumatic knee OA[3]. Medial compartment changes are more prominent in patients with non-traumatic knee OA[3]. In contrast, magnetic resonance imaging (MRI) demonstrated that cartilage volume was similar between medial and lateral compartments in patients with knee OA or with combined knee OA and ACL rupture[4,5]. However, this latter group had more frequent lateral compartment bone marrow lesions and meniscal derangements[4]. Thus, structural changes vary between OA subtypes but findings depend on the tissue type.

Gait metrics have been studied extensively in knee OA[6,7]. However, only one study has compared gait between non-traumatic and post-traumatic OA[8]. Patients with non-traumatic knee OA had greater knee adduction angles and moments during gait than patients with post-traumatic knee OA that had a previous ACL injury[8]. Other gait variables have not been compared. Specifically, muscle activation during gait differs in both patients with non-traumatic knee OA and patients that recently sustained an ACL rupture compared to healthy adults[9,10]. It is not clear if changes in muscle activation remain in patients that sustained an ACL rupture and then develop knee OA. Muscular co-contraction during gait is related to OA progression and changes in muscle activation can modify knee contact forces[11,12]. If we are to increase our understanding of how OA develops and progresses differently between non-traumatic and post-traumatic knee OA, a comparison of muscle function between these subtypes is required. This would help predict OA progression and potentially aid in developing interventions based on OA subtyping.

The objective was to compare muscle activation, knee angles, and external knee moments during gait between participants with symptomatic non-traumatic knee OA, symptomatic post-traumatic knee OA, and healthy controls. Consistent with previous non-traumatic knee OA research[9], it was hypothesized that participants with non-traumatic and post-traumatic knee OA would have higher vastus lateralis and lateral hamstring activation, but decreased medial gastrocnemius activation compared to healthy controls. The non-traumatic OA group would have higher knee adduction angles and moments compared to post-traumatic OA and healthy groups.

Method

Participants

This observational, cross-sectional study recruited participants diagnosed with symptomatic knee OA, according to clinical criteria from the American College of Rheumatology, using convenience sampling[13]. Participants were recruited from three tertiary hospitals in Montreal, Canada and the local community from January 2015 to March 2017. Participants were between 35 and 75 years of age. The lower age range was included because teenagers and young adults frequently injury their ACL and approximately 50% develop radiographic knee OA within 15 years[2]. Potential participants were excluded due to previous lower extremity joint arthroplasty, trauma or surgery within 1 year, inflammatory arthritis, or neurological conditions (e.g., Parkinson's Disease). A healthy group was also recruited from the local community. They had the same exclusion criteria. In addition, they had not been diagnosed with lower extremity OA and reported no knee pain on screening. Participants were part of an ongoing longitudinal study (unpublished), and all available participants were analysed for the current study. Participants provided written, informed consent prior to enrollment. Procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (Jewish General Hospital) and with the Helsinki Declaration of 1975, as revised in 2000.

Participants with knee OA were classified as non-traumatic or post-traumatic. Participants that reported no previous knee trauma resulting in an ACL rupture were classified as non-traumatic OA. Participants that reported a previous traumatic ACL rupture were classified as post-traumatic OA. Other traumatic injuries (e.g., posterior cruciate ligament tear) were excluded from the study in order to ensure homogeneity within the post-traumatic OA group. ACL status for all participants (injured, normal, and/or reconstructed) was confirmed on MRI by a fellowship trained, musculoskeletal radiologist with 8 years of experience (MB). Participants provided an estimate of when the ACL tear occurred. In patients with bilateral knee OA, the side with the most severe symptoms was selected based on participants' reporting of pain intensity. The study limb was randomly selected for healthy participants.

Demographic variables (e.g., age, sex) were self-reported from participants. Height was measured with a measuring tape and mass using a force plate. Participants completed the Intermittent and Constant Osteoarthritis Pain Scale (ICOAP)[14]. This pain scale (11-items, 5 point scale) includes subscales for intermittent and

Table I
Means (standard deviations) for group demographics. Frequencies are provided for the KL-scores

Variables		Non-traumatic OA Group (n = 22, 16 women)		Post-traumatic OA Group (n = 19, 8 women)		Healthy Group (n = 22, 16 women)	P value†
Age (y)		60 (7)		56 (9)		59 (7)	0.35
Height (m)		1.66 (0.07)		1.70 (0.10)		1.65 (0.09)	0.13
Mass (kg)		80.9 (17.6)		75.8 (15.9)		72.7 (12.1)	0.21
Body mass index (kg/m^2)		29.63 (7.5)		26.0 (3.2)		27.0 (4.6)	0.09
ICOAP-Constant pain (/100)		18 (24)		17 (19)		0 (0)	<0.01
ICOAP- Intermittent pain (/100)		31 (21)		28 (20)		3 (8)	<0.01
MAA (°)*		−0.93 (6.59)		−2.48 (4.68)		–	0.40
Gait speed (m/s)		1.14 (0.14)		1.23 (0.15)		1.25 (0.17)	0.37
KL-scores*	Score	Medial	Lateral	Medial	Lateral		
	0	0	9	0	7	–	≥0.79
	1	2	2	1	3	–	
	2	12	4	11	7	–	
	3	4	4	5	1	–	
	4	3	2	2	1	–	

* One participant from the non-traumatic OA group was missing MAA and KL-scores.
† Statistical determination performed by ANOVA (P values form the omnibus F-tests are provided), independent t-test (MAA) or Mann–Whitney U test (KL-scores).

Table II
Regression coefficients (B) with 95% confidence intervals and associated p values for the osteoarthritis (OA) group variable for the regression analyses

Dependent Variable*	*PC*	OA group coefficient	95% Confidence interval	*P* value†
Lateral gastrocs ($n = 40$)	1	−35.61	−81.80, 10.59	0.13
	2	−5.74	−36.54, 25.06	0.71
	3	14.88	−9.59, 39.36	0.23
Medial gastrocs ($n = 38$)	1	−24.00	−76.81, 28.82	0.36
	2	−13.28	−48.55, 22.00	0.45
	3	8.90	−17.25, 35.06	0.49
Vastus lateralis ($n = 40$)	**1**	**−75.21**	**−131.50, -18.92**	**0.01**
	2	−4.30	−27.03, 18.44	0.70
	3	12.01	−3.51, 27.53	0.13
Vastus medialis ($n = 38$)	**1**	**−81.80**	**−159.71, -3.89**	**0.04**
	2	−1.30	−23.18, 20.58	0.91
	3	13.11	−8.60, 34.82	0.23
Rectus femoris ($n = 40$)	**1**	**−49.89**	**−91.79, -7.99**	**0.02**
	2	−5.69	−16.57, 5.19	0.30
	3	−1.55	−10.32, 7.22	0.72
Lateral hamstring ($n = 39$)	1	−50.54	−104.39, 3.30	0.06
	2	**−36.74**	**−71.81, -1.67**	**0.04**
	3	19.97	−2.54, 42.48	0.08
Medial hamstring ($n = 40$)	1	−18.37	−63.41, 26.67	0.41
	2	−14.10	−39.34, 11.15	0.26
	3	**16.56**	**0.17, 32.95**	**0.05**
Knee flexion angle ($n = 40$)	1	−15.74	−43.45, 11.97	0.26
	2	4.14	−15.67, 23.95	0.67
	3	−6.70	−21.59, 8.18	0.37
Knee adduction angle ($n = 40$)	1	3.76	−18.04, 25.55	0.73
	2	−8.33	−23.11, 6.44	0.26
	3	4.31	−2.30, 10.93	0.20
Knee medial rotation angle ($n = 40$)	1	−44.40	−119.92, 31.13	0.24
	2	−5.06	−19.71, 9.58	0.49
	3	−6.38	−17.83, 5.08	0.27
Knee flexion moment ($n = 40$)	**1**	**−0.65**	**−1.31, 0.01**	**0.05**
	2	0.26	−0.11, 0.63	0.16
	3	0.19	−0.05, 0.42	0.12
Knee adduction moment ($n = 40$)	1	−0.02	−0.45, 0.42	0.94
	2	0.05	−0.22, 0.31	0.71
	3	0.07	−0.09, 0.23	0.36
Knee lateral rotation moment ($n = 40$)	**1**	**−0.12**	**−0.25, 0.001**	**0.05**
	2	0.001	−0.06, 0.06	0.98
	3	−0.01	−0.08, 0.05	0.70

Note: The dependent variables were principal component (*PC*) scores from EMG, knee angles, and knee external moment waveforms. OA group was coded as: non-traumatic OA = 0 and post-traumatic OA = 1. Statistically significant differences are bolded.

* Some data were not available due to data collection errors (i.e., poor contact between skin and electrode). The sample size (n) for each analysis is provided.

† *P* values were from the t-statistic for the associated regression coefficient.

constant pain with each subscale converted to a 0–100 score (higher scores = greater pain).

Radiographs

Participants in OA groups only underwent hip to ankle, anterior-posterior radiographs. They stood barefoot, with feet and toes facing forward, and patellae centered on the femoral condyles[15]. Radiographic disease severity was determined using Kellgren–Lawrence disease severity scores (KL-scores) (0 = no OA to 4 = severe)[16]. Scores were completed for medial and lateral knee compartments separately. Participants were further classified as having predominantly lateral compartment knee OA if KL-scores were greater in the lateral than medial compartment. Mechanical axis angle (MAA) was calculated as a measure of knee alignment as previously described[15]. Valgus and varus alignment were represented by positive and negative MAA values respectively. Measurements were performed on ImageJ software (National Institutes of Health).

Gait data collection

A16-channel electromyography (EMG) system (Trigno, Delsys) sampled at 2000 Hz measured muscle activation (band-width 20–450 Hz, signal amplification 1000). Bipolar surface electrodes were placed bilaterally over the following muscles according to guidelines[17,18]: medial and lateral gastrocnemius, vastus lateralis and medialis, rectus femoris, and medial and lateral hamstrings. The skin was shaved and cleaned with alcohol prior to electrode placement. Muscle palpation and isometric contractions validated electrode placement.

Motion capture data were collected with an eight camera, three-dimensional optical motion capture system (OQUS 300+, Qualisys) sampled at 100 Hz. Ground reaction forces were collected with two force plates (BP400600, AMTI) sampled at 2000 Hz. Reflective markers (12.7 mm diameter) were attached to participants over boney landmarks bilaterally according to guidelines including: lateral malleolus, first and fifth metatarsal heads, calcaneus, lateral femoral epicondyle, greater trochanter, anterior superior iliac spines, posterior superior iliac spines, and acromial processes[19]. Marker clusters (four markers attached to a base) were secured to mid-shank and mid-thigh bilaterally on lateral aspects of those segments using Velcro straps. Reflective markers were also attached to the third metatarsal head, medial malleolus, and medial femoral epicondyle during a static trial to identify joint centers but were removed during gait trials. Data were collected with Qualisys Track Manager (version 2.16).

Participants then completed a static standing trial on a force plate that identified knee and ankle joint centres and measured body mass. Next, they completed trials to identify functional hip

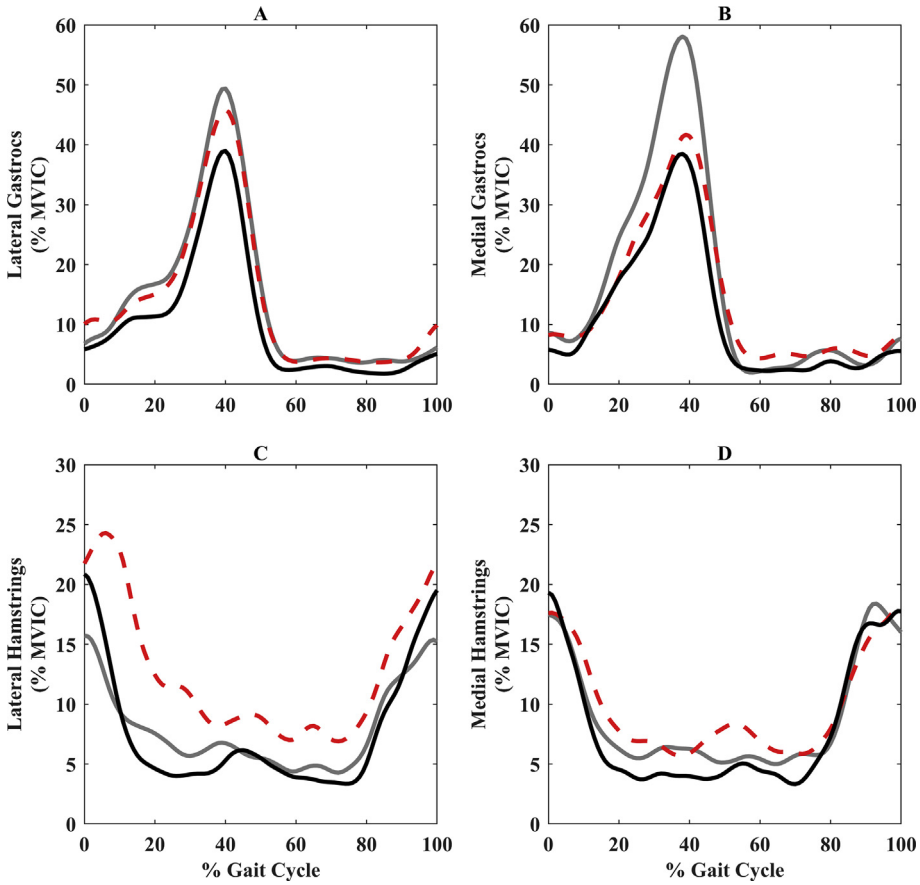

Fig. 1. Lateral gastrocnemius (A), medial gastrocnemius (B), lateral hamstring (C), and medial hamstring (D) EMG during gait normalized to MVIC for the non-traumatic OA (red, dashed lines), post-traumatic OA (black, solid lines), and healthy (grey, solid lines) groups.

joint centers. Participants were required to flex/extend and abduct/adduct their hip, and one trial was performed for each side. They then performed at least four practice gait trials to acclimatize to the environment. Testing required them to ambulate barefoot, at self-selected speeds, over eight metres. Seven successful gait trials, based on adequate force place contact, were acquired. Only five trials were processed and additional trials were collected to account for potential errors.

Participants then completed a series of maximum voluntary isometric contractions (MVIC) that were used to amplitude normalize gait EMG waveforms[18]. This series included two contractions for each muscle group to ensure maximum contractions were obtained[18]. A similar protocol has demonstrated good test-retest reliability when used to normalize gait EMG waveforms in patients with knee OA[17]. Exercises (with targeted muscle) included: 1) knee extension in sitting with the knee in 45° of flexion (quadriceps); 2) knee flexion in sitting with the knee in 55° of flexion (hamstrings); 3) knee extension in supine with the knee in 15° of flexion (quadriceps); 4) knee flexion in prone with the knee in 55° of flexion (hamstrings); 5) ankle plantarflexion in long sitting with the ankle in neutral (gastrocnemius); 6) unilateral heel raise (gastrocnemius). Exercises 1–5 were performed on an isokinetic dynamometer (Cybex Norm). Participants performed one submaximal and one maximal practice trial. Two collection trials for each exercise were performed, with each trial lasting 5 s. They rested 60 s between trials.

Gait data processing

EMG processing of gait trials included band-pass (20–500 Hz) filtering with a fourth order recursive Butterworth filter, wave rectification, and applying a low-pass, fourth order recursive Butterworth filter at 6 Hz to create a linear envelope. From MVIC trials, the maximum EMG signal was determined from rectified data using a moving-average window (100 ms) and the peak value from these windows was extracted. These values were used to amplitude normalize gait EMG.

Recursive, low-pass, fourth order Butterworth filters with frequency cuts offs of 8 Hz and 20 Hz were used to filter reflective marker and force plate data respectively. Ankle joint centers were identified as the midpoint between medial and lateral malleolus markers. Knee joint centers were the midpoint between medial and lateral femoral epicondyle markers. Functional hip joint centers were identified using published methods[20]. Three-dimensional knee angles were calculated using previously described joint coordinate systems[21]. Three-dimensional, net external knee moments were calculated using inverse dynamics, published inertial properties, described in joint coordinate systems, and amplitude normalized to body mass[22]. Gait speed was determined by tracking the speed of posterior superior iliac spine makers. EMG, angle, and external moment waveforms were time normalized to 100% of the gait cycle (i.e., heel strike to subsequent heel strike of the same side) and ensemble averages created from five trials for each participant. Processing of gait data were performed with Visual3D (version 5.02, C-motion). Similar procedures for collecting and processing gait data have demonstrated reliability[17,23].

Statistical analysis

Descriptive statistics were determined for demographic variables, ICOAP and gait speed. One-way analysis of variance (ANOVA)

compared these variables between groups. MAA and KL-scores were compared between OA groups with a Student's *t*-test or Mann–Whitney *U* test respectively.

Principal Component Analysis (PCA) was used to identify important waveform characteristics from EMG, joint angles, and external moments. PCA reduces waveform dimensionality but still considers temporal information and retains pattern structure[24]. In comparison, selecting discrete parameters (e.g., peak value) ignores temporal information and subjective decisions must be made about which discrete parameters to analyse[24]. For PCA, ensemble waveforms from EMG for each muscle group (gastrocnemius, quadriceps, hamstrings), angle, and external moment were entered into separate PCAs. From this data matrix (**X**), cross-product matrices were determined for EMG waveforms while covariance matrices were determined for angle and moment waveforms. An eigenvector decomposition of cross-product and covariance matrices produced eigenvectors (**U**) also called principal components (*PC*). The *PCs* represent unique waveform characteristics (e.g., shape, timing) and they vary for each analysis. The first three *PCs* were maintained for each variable because they often account for greater than 80% of the variance in gait waveforms[17,23]. Participant ensemble waveforms were scored against *PC*s producing principal component scores (*PC-score;* EMG *PC-scores* = $\mathbf{X}^{*}\mathbf{U}$; angle/moment *PC-scores*=$(\mathbf{X}\text{-}\overline{\mathbf{X}}\cdot)^{*}\mathbf{U}$). *PC-scores* are indicative of how closely a participant's waveform matches the specific waveform characteristic (i.e., *PC*). *PC-scores* were used in further analyses.

Both uncontrolled and controlled analyses that adjusted for potential confounders were completed to compare EMG, knee angles, and external knee moments between groups. Firstly, uncontrolled analyses included mixed model, two-way ANOVAs to compare EMG *PC-scores* between groups (non-traumatic OA, post-traumatic OA, healthy) and between muscles within a muscle group (e.g., medial and lateral gastrocnemius). Likewise, one-way ANOVAs compared knee angle and external moment *PC-scores* between groups[25]. Effect sizes (Cohen' d) were calculated for statistically significant pairwise comparisons to present the magnitude of the difference and were interpreted as small ($d = 0.20$), moderate ($d = 0.50$), and large ($d = 0.80$)[26]. Secondly, forward linear regression analyses examined if OA subtype classification (non-traumatic vs post-traumatic OA) was related to gait *PC-scores* (dependent variable) after accounting for confounders including KL-scores (highest value from medial or lateral compartment), MAA, and lateral OA classification. These confounders were selected a priori and assumed to impact gait *PC-scores* since previous studies have found KL-scores, MAA, and lateral OA classification relate to either EMG or joint mechanics in patients with knee OA[27–30]. These confounders were not assumed to be colliders and thus unlikely to be impacted by unconsidered factors. Thus, to determine if differences between non-traumatic and post-traumatic OA groups truly existed and were not due other factors, these confounders needed to be included in analyses. Confounder variables did not exist in the healthy group or were unavailable, and the healthy group was not considered in regression analyses. For the regression analyses, confounder variables were entered in the first step. OA group was entered in the final step and knee OA groups were coded as 0 = non-traumatic OA and 1 = post-traumatic OA. The regression coefficients with 95% confidence intervals and associated significance from the t-statistic were reported for the OA group variable. Separate regression analyses were performed for each *PC-score*. Statistical assumptions were examined to ensure analyses were appropriate including normality, sphericity, linearity, homoscedasticity, and collinearity. A p value of 0.05 was used to determine statistical significance. Analyses were completed with SPSS version 20.0 (IBM).

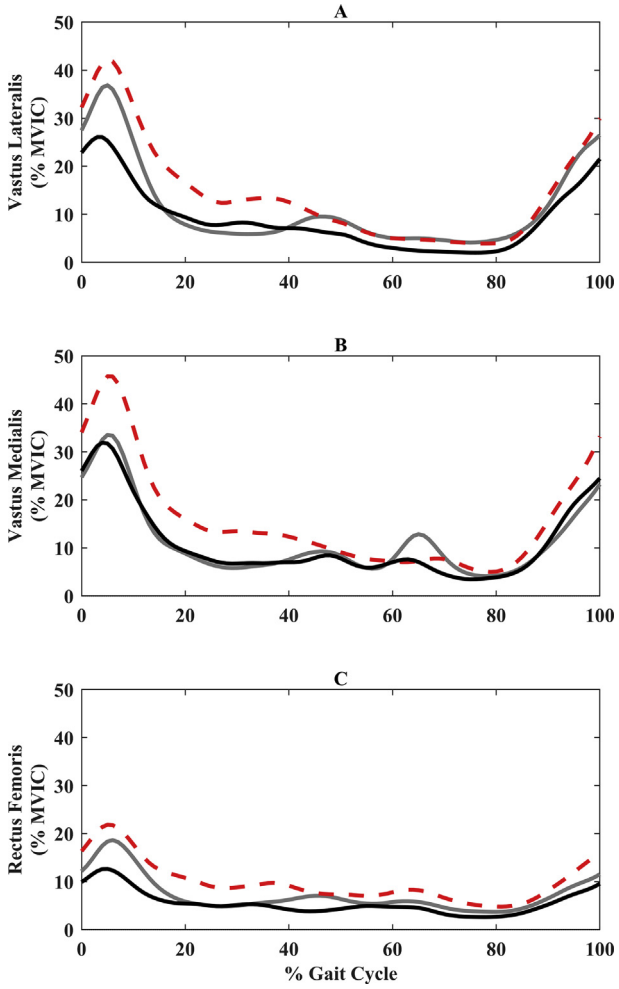

Fig. 2. Vastus lateralis (A), vastus medialis (B), and rectus femoris (C) EMG during gait normalized to MVIC for the non-traumatic OA (red, dashed lines), post-traumatic OA (black, solid lines), and healthy (grey, solid lines) groups.

Results

A flow diagram demonstrating exclusions at each stage is provided in the Supplemental. The final sample included 22 participants in both the non-traumatic OA (16 women) and healthy (16 women) groups. Nineteen participants (8 women) in the post-traumatic OA group were analyzed and ten of these participants had a previous ACL reconstruction. The mean time from initial ACL injury was 24 years (standard deviation 12 years). Six participants in the non-traumatic OA group and no participants in the post-traumatic OA group had lateral knee OA (i.e., greater KL-scores in lateral vs medial compartment). There were no statistically significant differences in group demographics, gait speed, MAA or KL-scores (Table I). There were statistically significant differences in ICOAP scores ($P < 0.01$), and pairwise comparisons revealed both OA groups had statistically significant ($P < 0.01$) higher scores than the healthy group (Table I).

For ANOVA EMG comparisons, statistically significant group effects and muscle–group interactions are described since they address study objectives. Muscle effects were not described in detail, but are similar to previous studies[9]. Statistically significant findings are reported in the text, while additional details are provided in Table II (regression coefficients with 95% confidence intervals) and in the Supplemental (ANOVA results) for significant and nonsignificant findings. Interpretations of the *PCs* are available

in the Supplemental. One participant from the non-traumatic OA group refused to undergo radiographs and was excluded from regression analyses since confounder variables were not available. Some participants had missing EMG data for a muscle due to collection errors (i.e., poor electrode contact) and their data were excluded from the relevant analysis. The number of participants in ANOVA (Supplemental) and regression analyses (Table II) are provided. All statistical assumptions were met and analyses deemed appropriate.

Gastrocnemius EMG

For the ANOVA, there were statistically significant group ($P < 0.01$) and muscle ($P = 0.03$) effects for gastrocnemius *PC1-scores*. The interaction ($P = 0.13$) effect was not statistically significant. *PC1* captured overall magnitude and shape. Pairwise comparisons revealed post-traumatic OA group ($P < 0.01$) had lower *PC1-scores* than the healthy group indicating lower lateral ($d = 0.74$) and medial ($d = 1.05$) gastrocnemius EMG (Fig. 1), which represented moderate to large effect sizes. There were no statistically significant findings for regression analyses.

Quadriceps EMG

For ANOVAs, there were no statistically significant group or interaction effects for any quadriceps *PC-scores* (Fig. 2). For the regression analyses that accounted for confounders, the relationship between OA group and *PC1-scores* for vastus lateralis, vastus medialis, and rectus femoris was statistically significant. For each muscle, non-traumatic OA group was associated with higher *PC1-scores*, indicating greater overall quadriceps EMG amplitudes, than the post-traumatic OA group (Fig. 2).

Hamstring EMG

For ANOVAs, there was a statistically significant interaction ($P = 0.03$) for hamstring *PC1-scores*. Pairwise comparisons revealed lateral hamstring *PC1-scores* from the non-traumatic OA group were statistically significantly higher compared to the healthy ($P = 0.03$; $d = 0.62$) and post-traumatic OA ($P = 0.04$; $d = 0.58$) groups, which represented moderate effect sizes. *PC1* captured overall magnitude and shape and the non-traumatic group had higher lateral hamstring EMG amplitudes (Fig. 1).

For regression analyses, the relationship between OA group and lateral hamstring *PC2-scores* was statistically significant. Hamstring *PC2* represented prolonged activity during mid-stance (Fig. 3). Higher *PC2-scores* were associated with non-traumatic OA indicating this group had prolonged and increased lateral hamstring EMG during mid-stance (Fig. 1). In addition, the relationship between OA group and medial hamstring *PC3-scores* was statistically significant. Hamstring *PC3* was a difference operator and represented the difference in hamstring EMG during early stance/late

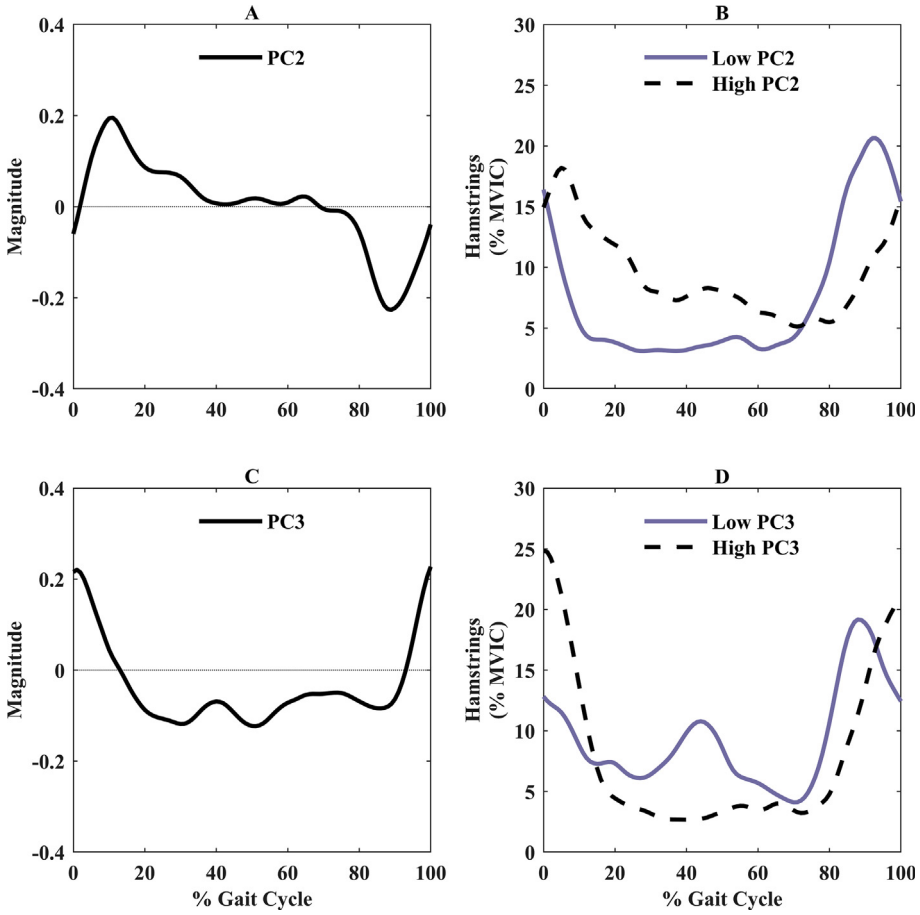

Fig. 3. Principal components (*PC*) for hamstring EMG. Hamstring *PC2* (A) represents activity during mid-stance. A subset of participants with high and low hamstring *PC2-scores* (B) demonstrates that higher scores represent prolonged and increased hamstring activation during mid-stance. Hamstring *PC3* (C) represents the difference in hamstring EMG during early stance/late swing with mid-/late stance. A subset of participants with high and low hamstring *PC3-scores* (D) demonstrates that higher scores represent a greater difference in hamstring activation between these times.

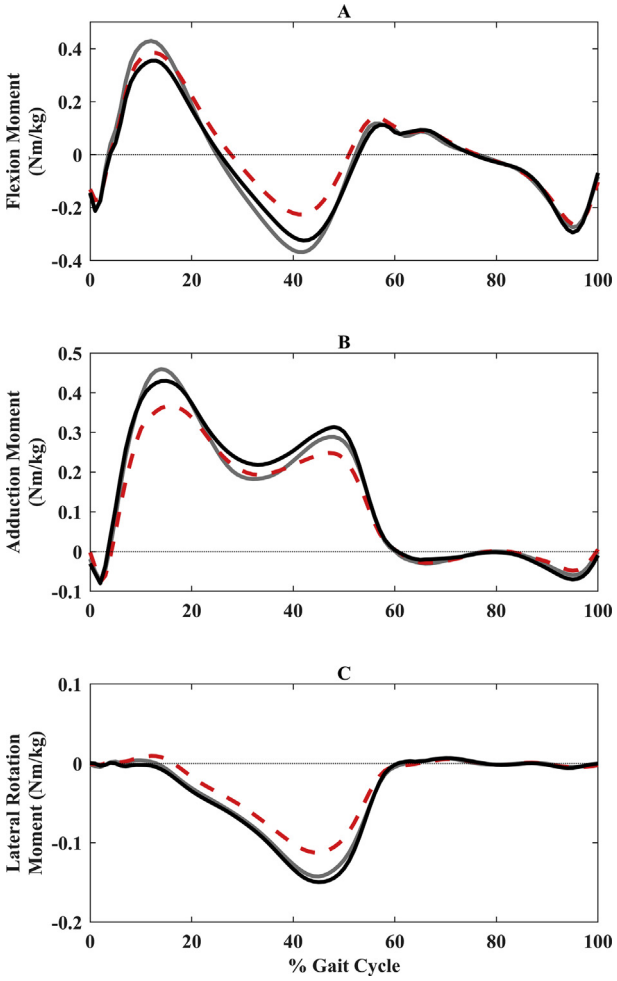

Fig. 4. Flexion/extension (A), adduction/abduction (B), and lateral/medial rotation (C) external knee moments during gait for the non-traumatic OA (red, dashed lines), post-traumatic OA (black, solid lines), and healthy (grey, solid lines) groups. Flexion, adduction, and lateral rotation represent positive values.

swing vs mid-/late stance (Fig. 3). Non-traumatic OA group was associated with lower *PC3-scores* indicating they had smaller differences in medial hamstring EMG between those times (Fig. 1).

Knee angles

ANOVAs and regression analyses were not statistically significant for knee angles (Supplemental and Table 2).

External knee moments

For ANOVAs, there was a statistically significant group effect ($P = 0.01$) for knee flexion moment *PC2-scores*. Pairwise comparisons revealed the non-traumatic OA group had statistically significant ($P < 0.01$) lower *PC2-scores*, indicating a smaller difference between early stance knee flexion moment and late stance knee extension moment, than the healthy group (Figs. 4 and 5). This resulted in a large effect size ($d = 0.87$). When only OA groups were considered in regression analyses that accounted for confounders, a statistically significant relationship existed between OA group and knee flexion moment *PC1-scores*. Non-traumatic OA was associated with higher *PC1-scores* which indicated a greater flexion moment compared to the post-traumatic OA group (Fig. 4). This occurred mainly during mid- and late stance when knee extension moments were lower for the non-traumatic OA group (Fig. 4).

There was a statistically significant ($P = 0.03$) group effect for knee lateral rotation moment *PC1-scores*. Pairwise comparisons revealed the non-traumatic OA group had statistically significant higher *PC1-scores* than post-traumatic OA ($P = 0.01$; $d = 0.78$) and healthy ($P = 0.04$; $d = 0.66$) groups, which were moderate to large effect sizes. This indicated that the non-traumatic OA group had lower medial rotation moments throughout stance (Fig. 4). Similarly, the relationship between OA group and knee lateral rotation moment *PC1-scores* was statistically significant in the regression analysis.

Discussion

This is one of the first studies to compare muscle activation during gait between knee OA subtypes, specifically non-traumatic

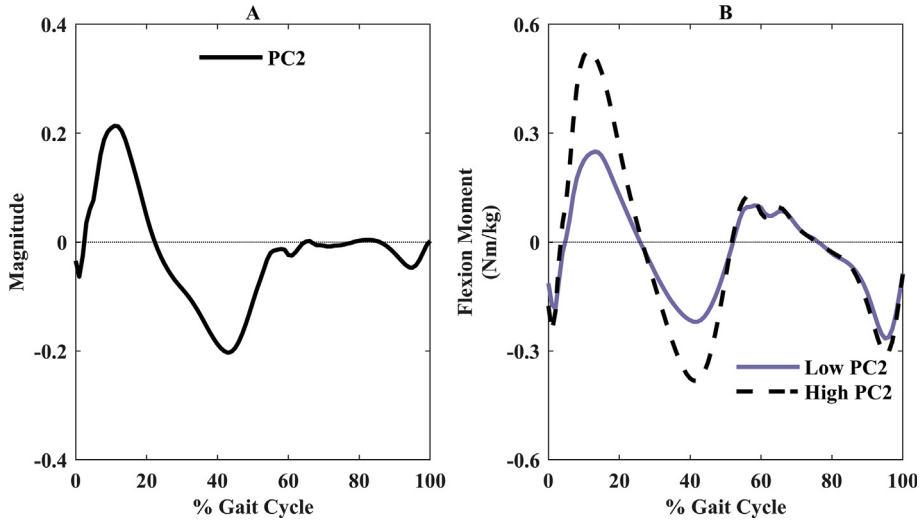

Fig. 5. Principal components (*PC*) for external knee flexion moment. Knee flexion moment *PC2* (A) represents the difference between early stance knee flexion moment and late stance knee extension moment. A subset of participants with high and low hamstring *PC2-scores* (B) demonstrates that higher scores represent a greater difference in the flexion and extension moment.

and post-traumatic OA. The non-traumatic OA group had higher quadriceps EMG than the post-traumatic OA group. Also, higher and prolonged lateral hamstring activation was found in the non-traumatic OA group. Differences remained between OA subtypes after accounting for disease characteristics (e.g., severity). Furthermore, the non-traumatic OA group had lower knee extension moments during mid- and late stance, and lower knee medial rotation moments during stance. Thus, gait differences exist between OA subtypes and history of ACL injury impacts joint and muscle function in patients with knee OA.

Muscle activation varied between knee OA subtypes. For gastrocnemius, findings were: 1) medial and lateral gastrocnemius EMG were lower in the post-traumatic OA compared to the healthy group; 2) medial gastrocnemius EMG were lower in the non-traumatic OA compared to the healthy group, although results were not statistically significant; and 3) there were no differences between OA subtypes. For hamstring and quadriceps EMG, generally the post-traumatic OA group more closely resembled the healthy group than the non-traumatic OA group. There was increased quadriceps EMG in the non-traumatic compared to the post-traumatic OA group in regression analyses (Fig. 2). This is supported by previous studies that have found increased quadriceps EMG during gait in patients with non-traumatic knee OA compared to healthy controls[6]. Furthermore, the non-traumatic OA group had increased and prolonged lateral hamstring EMG compared to healthy and post-traumatic OA groups respectively (Fig. 1). Likewise, previous studies found increased and prolonged lateral hamstring EMG in patients with non-traumatic knee OA compared to healthy adults[6]. Therefore, results indicate altered muscle activation during gait between non-traumatic and post-traumatic OA subtypes, and both groups demonstrated differences with healthy adults.

Several hypotheses could account for the more divergent EMG waveforms in the non-traumatic OA group compared to healthy adults. Higher lateral muscle EMG in patients with non-traumatic knee OA, especially lateral hamstrings, could off-load the medial compartment[9]. However, this hypothesis has been challenged[11]. Alternatively, considering knee moment and EMG waveforms in the non-traumatic OA group deviated more from normal values, perhaps these abnormalities play a role in disease initiation or progression in this OA subtype. In other words, knee OA partially developed and progressed due these gait abnormalities. This is supported by a study that found that knee muscular co-contraction was related to OA progression[12]. In patients with post-traumatic knee OA, the initial trauma and subsequent tissue damage might have played a greater role in the disease process and gait abnormalities could have played a diminished role. Regardless, differences in gait exist between these OA subtypes, and these gait metrics relate to OA progression[12,31]. This might indicate that disease processes differ between non-traumatic and post-traumatic OA, and perhaps interventions for these knee OA subtypes should also differ. Additional experimental and longitudinal research is required to evaluate these hypotheses.

Differences in external knee moments existed. Lower extension moments during mid- and late stance, and lower medial rotation moments during stance were found in the non-traumatic OA group. A previous study comparing patients with moderate and severe knee OA to healthy adults supported these findings[28]. Alterations in transverse plane joint motion has been hypothesized to be involved in cartilage loss, thus potentially impacting patients with non-traumatic knee OA[31]. There were no differences in knee adduction angles and moments between OA subtypes, contrary to the hypothesis. Only one study has compared knee angles and moments during gait between OA subtypes, and found patients with non-traumatic knee OA had higher knee adduction angles and adduction moments with similar flexion moments compared to patients with post-traumatic knee OA[8]. Differences between study samples likely account for these discrepancies including the previous study only enrolled patients with medial compartment knee OA, patients were being assessed for knee osteotomy, and they had greater knee varus alignment[8].

This study had several limitations. The post-traumatic OA group included both participants that were ACL deficient or had a reconstructed ACL. The impact on the findings is not clear, although gait changes in patients with an ACL deficiency persist up to 3 years after ACL reconstruction and longer term data are not available[32]. Findings cannot be generalized to other injuries. Healthy adults did not undergo radiographs to rule out radiographic OA. The cost was prohibitive and they reported no pain at screening. Disease characteristics that impact knee OA gait were considered in regression analyses such as lateral compartment knee OA. However, additional research in larger samples is still required to determine how lateral compartment knee OA specifically impacts muscle activation during gait as there is limited research on this topic. Other disease impairments (e.g., knee range of motion, swelling) were not considered although they might affect gait. Although there were significant differences between groups that represented moderate to large effect sizes, the clinical impact of these findings requires further investigation including if these differences affect disease progression.

In conclusion, differences in muscle activation and knee moments existed between OA subtypes. The non-traumatic OA group had increased quadriceps and lateral hamstring EMG, and decreased knee extension and medial rotation moments. Most gait waveforms were similar between post-traumatic OA and healthy groups, except for gastrocnemius EMG. Considering increased muscle activation leads to higher joint loads, these neuromuscular adaptions might play a role in disease progression in patients with non-traumatic knee OA[11,12]. Longitudinal studies are needed to confirm this hypothesis and determine if gait difference between these OA subtypes impact disease progression.

Author contributions

All authors made substantial contributions. Shawn Robbins was responsible for conception and design, obtaining funding, collection and assembly of data, analysis and interpretation of the data, drafting of the article, and final approval of the article. Moreno Morelli, Paul Martineau, Ron Dimentberg, and John Antoniou were responsible for provision of patients, critical revision of the article for important intellectual content, and final approval of the article. Mathieu Boily was responsible analysis and interpretation of the data, critical revision of the article for important intellectual content, and final approval of the article. Nancy St-Onge was responsible for conception and design, critical revision of the article for important intellectual content, and final approval of the article. Shawn Robbins (shawn.robbins@mcgill) takes responsibility for the integrity of the work as a whole, from inception to finished article.

Competing interest statement

There are no conflicts of interest to report.

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Supplementary data

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