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Self-reported walking difficulty and knee osteoarthritis influences limb dynamics and muscle co-contraction during gait

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ABSTRACT

Knee osteoarthritis (OA) gait is characterized by simultaneous flexor and extensor use, or co-contraction. Co-contraction can stabilize and redirect joint forces. However, co-contraction can push and pull on the femur and tibia that exacerbate OA symptoms and make walking difficult. Such movements are quantifiable by limb dynamics (i.e., linear acceleration and jerk); thus, this study examines limb dynamics and its relationship with co-contraction and OA related walking difficulty.

Three groups of age-and-sex-matched subjects with and without OA and walking difficulty (N = 13 per group) walked with electromyography (EMG) on the knee extensors and flexors and inertial measurement units (IMUs) at the femur and tibia. We calculated co-contraction from antagonistic EMG signals and linear acceleration and its derivative jerk from IMUs. We determined group differences using one-way ANOVAs, nonparametric equivalence, and effect sizes, and main and interaction effects of walking difficulty with regression modeling.

Medium effect sizes and differences for femoral acceleration (d = 0.64; P = .02) and jerk (d = 0.51; P = .01) were observed between with and without knee OA. Medium to large effect sizes (r = 0.33 to 0.51 and d = 0.81 to 0.97) and differences (P = .01 to 0.05) for tibial acceleration and jerk were obsevered between with and without walking difficulty. Walking difficulty moderated the relationship between tibial jerk and co-contraction ($p < .05$).

Tibial jerk differences were observed based on walking difficulty. The significant interaction effect suggested that walking difficulty explained the relationship between limb dynamics and co-contraction. Perhaps co-contraction levels used by those with knee OA and no walking difficulty are optimal as compared to those with walking difficulty.

1. Introduction

Knee osteoarthritis (OA) is the leading cause of walking difficulty and is predictive of detrimental outcomes, physical disability, and increased risk for developing or exacerbating comorbidities such as obesity, diabetes, and hypertension in older adults (Guccione et al., 1994; King, Kendzerska, Waugh, & Hawker, 2017). Knee OA symptoms, especially pain, can result in patients limiting or avoiding walking as a symptom management strategy (van Dijk et al., 2008). Although knee OA symptoms may temporarily improve, the risk of complications with subsequent health issues may increase (Ettinger, Davis, Neuhaus, & Mallon, 1994). In fact, knee OA-related walking difficulty is a leading risk factor for all-cause mortality among a general adult population (Nüesch et al., 2011).

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Therefore, it is essential to address the walking difficulty imposed by OA symptoms before patients completely avoid walking.

To develop intervention strategies that address walking difficulty among individuals with knee OA, actual gait-strategy differences between those with and without self-reported walking difficulty must be considered. Currently, only one study examines gait strategies based on self-reported walking difficulty (Na, Piva, & Buchanan, 2018). Group comparisons in most systematic reviews on knee OA gait strategies are typically between those with and without knee OA or between those of varying radiographic OA severity; fewer group comparisons are between those with varying self-reported symptoms or measures (Mills, Hunt, & Ferber, 2013; Mills, Hunt, Leigh, & Ferber, 2013). Additionally, the relationship between radiographic OA severity and gait mechanics continue to be debatable. Some studies report limited to no relationship between OA severity and gait mechanics (Mills, Hunt, & Ferber, 2013; Mills, Hunt, Leigh, & Ferber, 2013). In contrast, other studies report worsening OA severity exacerbating gait parameters such as elevated muscle activation when compared to those with less OA severity (Astephen, Deluzio, Caldwell, Dunbar, & Hubley-Kozey, 2008; Rutherford, Hubley-Kozey, Stanish, & Dunbar, 2011; Zeni, Rudolph, & Higginson, 2010). Perhaps sub-grouping knee OA gait characterization using self-reported symptoms or perceived function would provide insight into the varying OA gait mechanics.

Co-contraction, or the simultaneous activation of the knee extensors and flexors, appears to differ for both radiographic and self-reported knee OA severity sub-groups. In separate studies, greater co-contraction is found among those with more severe OA versus less severe or no radiographic OA (Hodges et al., 2016; Zeni et al., 2010), among those with greater versus lesser knee OA pain (Bouchouras, Patsika, Hatzitaki, & Kellis, 2015), and among those with greater versus lesser knee OA joint effusion (Rutherford, Hubley-Kozey, & Stanish, 2012). Co-contraction has both positive and negative attributions to the knee because of its ability to affect joint stability during differing demands that can impact movement efficiency and load distribution (Schmitt & Rudolph, 2007). Co-contraction is a naturally occurring weight-bearing phenomenon that syncs with joint demands by facilitating stability, absorbing distal forces, and redirecting loads proximally (Robertson, Winter, Gordon, Robertson, & Winter, 1980). The human system is known to respond to increasingly demanding tasks by adjusting muscle recruitment, which is illustrated in the increasingly levels of co-contraction during running, jumping, or sprinting when compared to walking (Lee et al., 2015). Previous studies found greater muscle activation during walking among older adults than for younger adults (Rudolph, Schmitt, & Lewek, 2007). In patients with knee OA, walking at faster gait speeds required elevated muscle co-contraction (Zeni et al., 2010). Therefore, perhaps for those with knee OA, the demanding task may be walking with OA symptoms and maintaining functional gait speeds, especially among those also experiencing aging (Ortega & Farley, 2014).

In contrast, co-contraction is expected to increase contact forces, which potentially progresses knee OA by increasing the overall joint load, or distributing forces to favorable areas for accepting repetitive weight-bearing (Heiden, Lloyd, & Ackland, 2009; Rudolph et al., 2007; Schmitt & Rudolph, 2007).

Given these contrasting findings, perhaps co-contractions may be necessary to meet the high-demand task of maintaining walking ability. Meanwhile, co-contractions that accompany walking difficulty may be a poor gait adaptation warranting intervention. The expectation is that high and low co-contraction levels that parallel large or small dynamic stabilization, respectively, are meeting the joint and task demands of walking, whereas, asynchronous co-contraction may result in conflicting femoral and tibial movements. Although co-contraction is commonly quantified in knee OA gait via electromyography (EMG) and associated valid and reliable calculation equations (Hubley-Kozey, Robbins, Rutherford, & Stanish, 2013), its relationship to limb movement is yet to be studied.

Recent studies use limb dynamics, defined as linear acceleration and jerk, measured via inertial measurement units (IMUs), as a valid (Roberts, Khan, Kim, Slover, & Walker, 2013) and reliable (Liikavainio et al., 2007; Turcot, Aissaoui, Boivin, Hagemeister, et al., 2008) means to quantify limb movements. Linear acceleration—the change of speed as generated by the forces acting on the limb—can quantify three-dimensional forces traveling through the leg (Lafortune, 1991; Radin, Yang, Riegger, Kish, & O'Connor, 1991). For example, linear acceleration measured at the proximal tibia during gait would involve the predominate transverse plane force generated distally, sagittal and frontal plane tendon forces from gross muscle activation, and any force attenuation by adjacent structures (Lafortune, 1991). Linear acceleration measured at the distal femur would characterize the aforementioned forces identified at the proximal tibia, but after traveling via the knee joint (Radin et al., 1991). It is accepted that a healthy biological system focuses on reducing itself to the simplest control strategies when faced with repetitive tasks (Hogan, 1984); thus, small linear acceleration values are expected to be optimal for walking. Turcot et al. (2008) found higher femoral and tibial linear acceleration during gait among those with symptomatic knee OA when compared to a control group. Similarly, Roberts et al. reported more linear acceleration at the femur and tibia during gait among those with self-reported instability after a total knee replacement (TKA) when compared to their TKA counterparts that denied instability (Roberts et al., 2013). Patients with ACL deficiency also walk with higher tibial linear acceleration when compared to those with an intact ACL (Yoshimura, Naito, & Zhang, 2002). However, linear acceleration has never been quantified among patients with knee OA and walking difficulty.

The time-derivative of acceleration, jerk, is also gaining some traction in movement sciences and orthopaedic literature (Hogan & Sternad, 2009; Sakata et al., 2010; Yashiro, Miyawaki, Tome, Yasuda, & Takada, 2004; Roberts, et al., 2013). Similar to the time-derivative relationship between acceleration and velocity, jerk does not directly correlate with acceleration, and therefore needs to be examined independently. Jerk is a measurement of movement smoothness, and larger values can indicate less smoothness and greater cost-demand on the motor and movement systems (Hogan & Sternad, 2009; Sakata et al., 2010; Yashiro et al., 2004). A recent study found more jerk during walking among patients with TKAs who reported instability than those without instability (Roberts, et al., 2013). Among patients with Parkinson's disease, jerk is used to quantify wrist and finger coordination during various handwriting patterns to determine muscle coordination health (Teulings, Contreras-Vidal, Stelmach, & Adler, 1997). While these studies all propose that limb dynamics, greater linear acceleration, and/or jerk suggest increased task difficulty and worsening muscle strategies, the actual relationship between limb dynamics and muscle activation has never been examined. Also, it is unknown how limb dynamics may vary based on knee OA and walking difficulty presence.

Therefore, the purpose of this study is two-fold: (1) to compare limb dynamics (i.e., linear acceleration and jerk) among those with knee OA and self-reported walking difficulty (Diff), those with knee OA and no self-reported walking difficulty (NoDiff), and those with no knee OA (control group); and (2) to examine how self-reported walking difficulty and limb dynamics may influence neuromuscular strategies (i.e., co-contraction) observed among patients with knee OA. Since clinical knee OA severity signs and symptoms, including joint degradation, pain, and instability, are precursors for developing walking difficulty and are found to be related to large limb dynamics (i.e., linear acceleration and/or jerk) during walking, we hypothesize that when walking at a controlled gait speed, the Diff group will walk with the largest limb dynamics, followed by the NoDiff group, and the smallest values will be observed in the control group. Further, we hypothesize that self-reported walking difficulty will moderate the relationship between limb dynamics and muscle-*co*-contraction.

2. Methods

2.1. Participants

Adults who were English-speaking, between ages 50 and 80 years, with and without knee OA were recruited from the community and physician and physical therapy offices as described in Na et al. (2018). Exclusion criteria included: traumatic injuries; signs or symptoms of injury to the trunk, lower back, hip, leg, foot, or ankle within the past three months; symptomatic arthritis in the lower back, hip, leg, foot, or ankle in either leg that affects movement or function; interventions pertaining to the lower back, hip, foot, or ankle in either leg; history of knee replacement or skeletal realignment surgery in either leg; history of respiratory, cardiovascular, systemic, or neurological diseases; inability to walk without an assistive device (e.g., walker, cane); and walking at a self-selected gait speed of 1.0 m per second or slower as measured using the 10-meter walk test. Additional exclusion criteria for the OA group included a Kellgren-Lawrence (KL) OA score of < 2 and knee pain (i.e., an average of best, worst, and current) of ≤ 2 on a scale of 0 = no pain, to 10 = worst pain imaginable. Additional exclusion criteria for the control group included any single positive findings of the Altman's Criteria for knee OA (i.e., morning stiffness < 30 min, crepitus, bony tenderness, bony enlargement, palpable warmth) (Altman et al., 1986).

Subject responses to the question, "How does your knee affect your ability to walk?" were adopted from the Knee Outcome Survey (KOS) (Irrgang, Snyder-Mackler, Wainner, Fu, & Harner, 1998) and used to define walking difficulty. Responses were scored on a 6-point scale (5 = Not Difficult, 4 = Minimally Difficult, 3 = Somewhat Difficult, 2 = Fairly Difficult, 1 = Very Difficult, and 0 = Unable to do) (Irrgang et al., 1998). Subjects with responses of *not difficult* (5) or *minimally difficult* (4) were sub-grouped into the no walking difficulty group (NoDiff). Subjects with responses from *somewhat difficult* (3) to *unable to do* (0) were sub-grouped into the walking difficulty group (Diff). Subjects were age- ($\pm$ 2.5 years) and sex-matched across the Diff, NoDiff, and control groups. The examiner was blinded to the subject's group assignment during data collection and processing. As per the Institutional Review Board protocol of the University of Delaware, informed consent was obtained and rights were protected for each subject.

Knee OA severity was measured using a standing, posterior-anterior radiograph with 20 degrees of knee flexion (Kellgren & Lawrence, 1957). A radiologist, blinded to the subject's group, scored OA severity based on the 4-point Kellgren-Lawrence (KL) scale, including 1: doubtful narrowing of joint space and possible osteophytic lipping; 2: osteophytes, definite narrowing of joint space; 3: moderate multiple osteophytes, definite narrowing of joint space, some sclerosis and possible deformity of bone contour; 4: large osteophytes, marked narrowing of joint space, severe sclerosis, and definite deformity of bone contour (Kellgren & Lawrence, 1957).

2.2. Testing protocol

Subjects wearing IMUs and EMG electrodes walked at a controlled gait speed of 1.0 $\pm$ 0.05 m/s across a 10-meter walkway with 2 force plates for a minimum of 5 trials. We used tape to mark a start line, followed by 10 m, and a line to mark stop. The time it took subjects to walk the 10 m allowed us to determine real-time gait speed for both screening and testing. The gait speed cutoff of 1.0 m/s was chosen because previous literature reports it to be a reliable indicator for functional independence and reduced risks of falls and hospitalization (Brach, Simonsick, Kritchevsky, Yaffe, & Newman, 2004; Fritz & Lusardi, 2009; Studenski et al., 2003).

Subjects practiced walking at controlled gait speed and were given verbal feedback as needed. Practice trials stopped when subjects consistently achieved the speed for three or more consecutive trials. Subjects were given the instructions to start behind the start line, walk past the stop line, and walk normally (i.e., ignore and not aim for the force plates). If subjects reported that they deviated their gait pattern (e.g., aimed for the force plates), we repeated the trial. Two examiners visually inspected each trial for quality control. Accepted trials required at least 2 strides of clear heel strike on the force plates and no altering gait patterns, defined as no visual observations by the examiner that the subject's gait mechanics varied from "normal walking" or when walking at self-selected gait speeds (e.g., reducing step length just before the force plates). Subjects were provided rest breaks of at least one minute between each trial to limit potential fatigue effects.

2.3. Instrumentation

We recorded raw limb dynamics data as linear acceleration (g) at 100 Hz using IMUs (Noraxon, 3D Myomotion, Scottsdale, USA). IMUs were strapped to bilateral tibial tuberosities, bilateral superior patellas, and the pelvis at S2. Each IMU (37.6 mm $\times$ 52 mm $\times$ 18.1 mm) weighed 34 g and included an onboard gyroscope and accelerometer that corrects for gravity and measures 3D angular velocity and linear acceleration, respectively. Femoral IMUs were strapped to the anterior femur superior to the

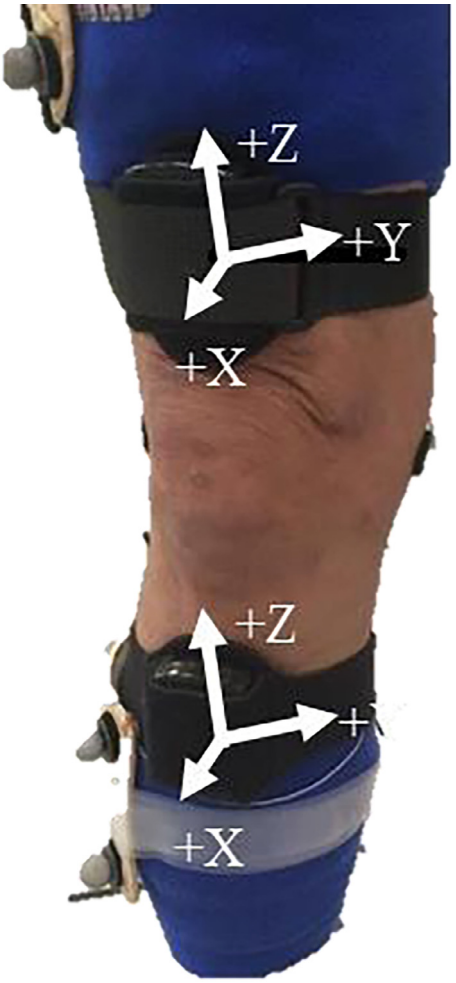

Fig. 1. Inertial measurement unit (IMU) at proximal tibia, which output vector data in the x-direction, or anterior-posterior, y-direction, or medial–lateral, and z-direction superior-inferior.

patella. Tibial IMUs were strapped to the anterior tibia inferior to the tibial tuberosity (Fig. 1). The pelvic IMU was strapped to the sacrum between the PSISs and at the sacral ridge of S2. IMU locations were selected to minimize movement and soft-tissue artifacts by maximizing bone contact closest to the joint (Lafortune, 1991), and have been previously reported to have excellent reliability (Liikavainio et al., 2007).

We recorded force plate data at 1000 Hz (AMTI Force Plate, Watertown, MA.) and movement from 8 motion-analysis cameras (Qualisys, Inc.) that tracked reflective markers at 100 Hz. Each patient wore markers secured to the head, bilateral acromion, C7 spinous process, pelvis, bilateral greater trochanter of the femur, bilateral medial and lateral epicondyles of the knee, fifth and first metatarsals of the foot, medial and lateral malleoli, and distal tubercle of the calcaneus for subject calibration and identifying joint centers ("Marker Set Guidelines – Visual3D Wiki Documentation," 2017; van Sint Jan, 2007). Rigid thermoplastic shells, with 4 markers firmly affixed, were attached to the posterior pelvis, lateral femur, lateral tibia, and dorsal surface of the foot (Cappozzo, Cappello, Della Croce, & Pensalfini, 1997). The combination of force-plate and motion-capture data were used to define gait intervals, real-time gait speed calculations, and synchronize limb dynamics and EMG data.

We recorded muscle activation via a 16-channel EMG system (Motion Lab Systems, Baton Rouge, USA) at 1000 Hz. Disposable self-adhered surface electrodes were placed at the vastus medialis (i.e., medial quadriceps, MQ), the vastus lateralis (i.e., lateral quadriceps, LQ), the biceps femoris (i.e., lateral hamstring, LH), the semitendinosus and semimembranosus (i.e., medial hamstrings, MH), gastrocnemius medialis (i.e., medial gastrocnemius, MG) and gastrocnemius lateralis (i.e., LG) according to the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) guidelines (Merletti & Hermens, 2000; Merletti, Rau, Stegeman, & Hagg, n.d.). EMG signals from gait trials were normalized to muscle EMG signals from maximal voluntary contractions (MVC). MVCs were measured via isometric contraction on the Biodex System III (Biodex Medical Systems, Inc., Shirley, USA) dynamometer at 60 degrees of knee flexion and 80 degrees of hip flexion, which have demonstrated excellent reliability (de Araujo Ribeiro Alvares et al., 2014). This position also minimizes the potential effects of anterior knee pain (Thomeé, Renström, Karlsson, & Grimby, 1995). To ensure ankle MVCs during walking did not exceed EMG values, plantar flexion MVCs were measured with standing

bilateral heel raises and manual resistance through the shoulders. This testing procedure is an adapted version of the standardized manual muscle testing position for plantar flexion (Kendall, McCreary, Provance, Rodgers, & Romani, 2005). The adaptation was chosen because many patients with knee OA were unable to maintain a single-leg balance safely. Recordings for motion capture, force plates, EMG, and IMU were synced using an external trigger. Data were visually inspected for quality after each trial. Gait testing procedures were of similar methods to those previously reported by our lab (Na et al., 2018).

2.4. Data processing

Raw data from IMUs, EMG, force plate, and motion capture were exported to Visual 3D™ (C-Motion, Rockville, MD, USA) for processing. Force platform data and motion capture markers were low-pass filtered with a second-order Butterworth filter at 40 Hz and 4 Hz, respectively. Frontal and sagittal plane knee angles were calculated via Visual 3D using Euler angles and used to determine gait intervals (weight acceptance and mid-stance) common to knee-OA gait literature and our lab (Schmitt & Rudolph, 2007). Weight acceptance was the period defined from heel strike to the first peak knee flexion angle, and mid-stance was the period defined from the first peak knee flexion angle to the first peak knee extension angle during stance phase (Schmitt & Rudolph, 2007). Each gait interval was normalized to 100 data points to account for time and point differences. Final gait speed was calculated using temporal distance properties within Visual 3D. Temporal distance calculations required a minimum of two complete bilateral strides, with one heel strike of each foot landing on a force plate.

All EMG signals were high- and low-pass filtered to remove offsets and noise artifacts using a fourth order Butterworth filter at 20 Hz and 350 Hz, respectively. Filtered signals were rectified and low-pass filtered to eliminate high-frequency filters of the muscles with a fourth-order Butterworth filter at 10 Hz. Processed gait EMG was normalized to similarly processed MVC EMG to calculate percentage maximum of muscle activation. EMG signals from each gait interval were used to calculate co-contraction per Rudolph, Axe, and Snyder-Mackler (2000) (Eq. (1)). The co-contraction index accounts for the simultaneous muscle activations of opposing muscle groups, and were calculated for MQ and MH (i.e., MQMH), LQ and LH (i.e., LGLH), MQ and MG (i.e., MQMG), and LQ and LG (i.e., LQLG) (Rudolph, et al., 2000):

$$Co - contraction\ Index = \left(\sum_{i=IC}^{TASK} \left(\frac{EMG_S}{EMG_L} (EMG_S + EMG_L) \right) / 100 \right) \quad (1)$$

The co-contraction index equation divided the smaller EMG signal (EMG_s) by the larger EMG signal (EMG_L), multiplied by the sum of the small and large EMG signals, and integrated over the gait interval. A larger co-contraction index suggests a higher concurrent use of opposing muscles.

Linear acceleration and jerk data were processed for all gait phases in 3-physiological planes (anterior-posterior x-, medial–lateral y-, and superior-inferior z-) for the entire gait phase. Raw linear acceleration data was low-pass Butterworth filtered at 30 Hz. Jerk (g/s) was calculated from the first time derivative of linear acceleration. Time-synced gait intervals were created as events for linear acceleration and jerk. The magnitude of limb dynamics was computed using the line equation (Eq. (2)):

$$m = \sqrt{x^2 + y^2 + z^2} \quad (2)$$

The equation provided an instantaneous magnitude of vectors by calculating the square root of the sum of each axis when squared, which condensed the 3 physiological planes to a single value. The reduced data was exported for weight acceptance and midstance. Within each gait interval, data was then normalized to 100 points. The peak limb dynamic values of each gait interval were averaged across trials for each subject. The average peak limb dynamic values per gait interval were compared between groups. Prior studies found that peak values of limb dynamics demonstrated excellent reliability (Liikavainio et al., 2007) and validity (Roberts et al., 2013). Therefore, we used the peak values with the assumption that such psychometric properties previously reported would carry forward through the equation.

2.5. Statistical analysis

Statistical analyses were performed using SPSS (version 13, Chicago, IL, USA). All variables of interest were examined for homogeneity of variance using the Levene's test ($P < .05$) and for normal distribution using the Shapiro-Wilk test. Limb dynamic differences among the 3 groups were tested using one-way ANOVA for parametric testing and Wilxcon Sign Rank Test for non-parametric testing. An a priori power analysis was conducted for limb dynamics using an effect size of 0.45, alpha of 0.05, and power of 0.80; we needed a total sample size of 38 or 13 in each group. Given the experimental nature of the study, we determined group, comparisons, and direction a priori; therefore, *P*-values were not adjusted for repeated comparisons. Additionally, standardized effect sizes (ES) were considered, which included *d* values for parametric tests, with 0.20 defined as small, 0.50 as medium, and 0.80 as large, and *r* values for non-parametric tests, with 0.10 as small, 0.30 as medium, and 0.50 as large (Cohen, 1988).

Stepwise regression analyses were used to examine the interaction effects of self-reported walking difficulty on the relationship between limb dynamics and neuromuscular strategies. Regression diagnostics were performed to account for potential outliers and collinearity for non-interaction main effects. To eliminate redundant testing and risk for errors, we determined a priori that only the limb dynamics variable with the largest group difference will be entered into the regression model to examine its relationship with co-contraction.

Table 1
Group characteristics, including mean and standard deviation (SD) for age, body mass index (BMI), and self-selected gait speed for the 3 groups. Subjects for the walking difficulty (Diff), no walking difficulty (NoDiff), and control groups were age and sex matched.

	Diff	NoDiff	Control	*P*-value
N	13	13	13	n/a
Age, mean (SD), years	66.1 (SD 6.3)	65.8 (SD 5.8)	66.1 (SD 6.2)	n/a
Sex, N, females	8	8	8	n/a
BMI mean (SD), Kg/m^2	31.4 (SD 5.6)	29.7 (SD 5.6)	27.6 (SD 3.7)	$P = .18$
OA Severity, N				$P = .61$
2	4	4	N/A	
3	5	6	N/A	
4	4	3	N/A	
Pain, mean (SD), 0 to 10	6.5 (SD 1.5)	5.6 (SD 2.0)	N/A	$P = .92$
Gait speed (SD), m/sec	1.2 (SD 0.19)	1.34 (SD 0.16)	1.31 (SD 0.10)	$P = .23$

Pain reported on a scale from 0, defined as no pain, to 10, defined as worst pain imaginable.
OA severity defined based on the Kellgren-Lawrence (KL) scale, including 2: osteophytes, definite narrowing of joint space; 3: moderate multiple osteophytes, definite narrowing of joint space, some sclerosis and possible deformity of bone contour; 4: large osteophytes, marked narrowing of joint space, severe sclerosis, and definite deformity of bone contour.

3. Results

A total of 39 age- and sex-matched subjects participated in the study, with 13 per group. OA severity, pain severity, and body mass index (BMI) were not statistically different between the groups (Table 1). Of those in the NoDiff group, walking was *not difficult* for 7 and *minimally difficult* for 6 subjects. Of those in the Diff group, walking was *somewhat difficult* for 11 subjects, *fairly difficult* for 1, and *very difficult* for 1 subject.

3.1. Hypothesis 1: Limb dynamics and walking difficulties

During weight acceptance, limb dynamic magnitudes between the groups yielded small effect sizes and no significant differences (ES, d = 0.05 to 0.44 and r = 0.09 to 0.24; $P = 0.22$ to 0.84) (Table 2).

During mid-stance, tibial acceleration yielded medium effect sizes and was significantly different among all three groups (r = 0.33 to 0.51 and $P = .01$ to 0.05). All other limb dynamics during mid-stance yielded medium to large effect sizes and were significantly different between those with and without knee OA (i.e. Diff vs. control and NoDiff vs. control) (ES, r = 0.33 to 0.60 and d = 0.60 to 1.08; $P < .01$ to 0.05), except tibial jerk (Table 2). Tibial jerk in the NoDiff group yielded a small effect size, d = 0.29, and was not significantly larger than the control group $P = .29$, but yielded a large effect size, d = 0.97, and was significantly smaller than the Diff group, $P = .01$ (Fig. 2).

Of all limb dynamic comparisons between the Diff and NoDiff group, tibial jerk during mid-stance yielded the largest effect sizes and the smallest *P*-values; therefore, it represented limb dynamics in the regression model.

3.2. Hypothesis 2: Limb dynamics and neuromuscular strategies

Individually, the main effects of group and the main effects of limb dynamics did not significantly explain co-contraction levels. Significant interactions between group and limb dynamics were found for the LQLH and LQLG co-contraction indices (Table 3,

Table 2
Mean and 95% confidence interval (CI) for femoral and tibial limb dynamics magnitudes during weight acceptance and mid-stance gait intervals for the walking difficulty group (Diff), no walking difficulty group (NoDiff), and the control group (Left). Group comparisons with results represented by effect size (ES) values for parametric tests are presented as a value of *d* and non-parametric tests results are presented in *r* and *P*-values (Right).

							NoDiff v. Diff		NoDiff v. Control		Diff v. Control	
	Diff	95% CI	NoDiff	95% CI	Control	95% CI	*P*-value	ES	*P*-value	ES	*P*-value	ES
Weight Acceptance												
Femoral acceleration (g)	1.5	1.3, 1.8	1.4	1.2, 1.6	1.4	1.2, 1.6	0.39	d = 0.27	0.89	d = 0.05	0.27	d = 0.44
Tibial acceleration (g)	2.1	1.7, 2.5	1.9	1.6, 2.2	1.8	1.5, 2.1	0.65	r = 0.09	0.51	r = 0.13	0.22	r = 0.24
Femoral jerk (g/s)	79.6	64.4, 94.6	76.6	58.4, 94.7	81.6	63.4, 99.8	0.77	d = 0.09	0.59	d = 0.20	0.84	d = 0.06
Tibial jerk (g/s)	124.4	98.0, 150.8	113.8	89.9, 137.7	107.8	86.5, 129.2	0.65	r = 0.09	0.60	r = 0.11	0.22	r = 0.22
Mid-Stance												
Femoral acceleration (g)	0.9	0.7, 1.1	0.7	0.6, 0.9	0.5	0.4, 0.6	0.11	d = 0.36	0.02*	d = 0.64	0.01*	d = 1.08
Tibial acceleration (g)	0.9	0.7, 1.1	0.7	0.5, 0.9	0.5	0.4, 0.6	0.03*	r = 0.36	0.05*	r = 0.33	0.01*	r = 0.51
Femoral jerk (g/s)	35.1	23.0, 47.1	25.8	18.7, 33.1	14.9	10.6, 19.2	0.25	r = 0.26	0.01*	r = 0.51	< 0.01*	r = 0.60
Tibial jerk (g/s)	39.2	28.1, 50.4	22.6	15.4, 29.8	19.2	14.6, 23.8	0.01*	d = 0.87	0.29	d = 0.40	< 0.01*	d = 0.97

(*) Significant group differences, $P \leq 0.05$.

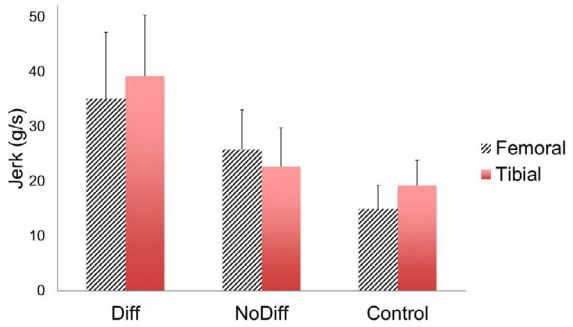

Fig. 2. Average femoral and tibial jerk for the three groups (OA subjects with walking difficulty, OA subjects without walking difficulty and controls) during mid-stance. The error bars represent 95% confidence limits. See Table 2 for statistical comparisons between groups.

Table 3
Regression analyses results when examining the relationship between tibial jerk and co-contraction using self-reported walking difficulty as the moderating variable.

	β	SE	p-value	95% CI		Partial R
				Lower	Upper	
Tibial Jerk						
LQLH	0.43	0.17	0.02*	0.08	0.78	0.50
MQMH	0.17	0.14	0.25	−0.13	0.46	0.26
LQLG	0.39	0.16	0.03*	0.05	0.72	0.47
MQMG	0.15	0.15	0.31	−0.15	0.46	0.23

(*) Significant group interaction, $P \leq 0.05$.

Fig. 3), with the relationships between lateral co-contraction indices and limb dynamics to be positive for the Diff group and negative for the NoDiff group. Although the interacting slopes were observed between medial co-contraction and limb dynamics for the respective groups, the interaction was not statistically significant (Fig. 3). Furthermore, statistically controlling for BMI and OA severity did not yield different results.

4. Discussion

The purpose of our study was to determine limb dynamics and muscle co-contraction strategy differences based on the presence of knee OA and walking difficulty. For this study, we quantified limb dynamics and muscle co-contraction in a homogenous population of age and sex-matched patients with and without knee OA, and with and without self-reported walking difficulty. The study design included subjects who walked at self-selected gait speeds at or faster than the controlled gait speed of 1.0 m per second. Consistent with our hypothesis, our study results identified limb dynamic group differences, and self-reported walking difficulty moderated the tibial jerk and co-contraction relationship.

Although the study results supported our hypothesis in that limb dynamics were largest among those with knee OA and walking difficulty and smallest among the control group, we did not expect greater limb dynamic differences between the Diff and NoDiff groups than between the NoDiff and Control group. This finding was especially interesting, because patients with knee OA recruited for this study, regardless of walking difficulty status, were required to have knee OA severe enough to seek medical attention—including moderate to severe radiographic OA evidence and notable pain scores. Despite the OA severity impairment, our results suggested that limb dynamics varied more with self-reported walking difficulty than disease severity alone. Perhaps those with knee OA and walking difficulty walked with inefficient movement strategies that were poor at attenuating dynamic gait forces, quantified via linear acceleration, and facilitating movement smoothness, quantified via jerk. Meanwhile, those with NoDiff appeared to have sustained pre-morbid gait strategies or minimally adapted gait strategies to limit walking difficulty. However, given the cross-sectional study design, such temporal relationships cannot be discerned and would require future research to determine how gait strategies are utilized for sustaining walking among patients with knee OA.

The differences in limb dynamics did not consistently achieve statistical significance, but they did demonstrate a trend that was consistent with our hypothesis and previous research. Specifically, our linear acceleration data were consistent with Turcot and colleagues who found that those with knee OA walked with greater limb dynamics when compared to those without OA (2008). Additionally, our linear acceleration peaks were slightly smaller than those reported by Lafortune (1991) and slightly larger than those reported by Turcot et al. (2008), and the variations may be best explained by gait speed differences (Table 4). Lafortune (1991) examined tibial acceleration in a healthy control subject who walked at a self-selected gait faster than this study, and Turcot et al. (2008) reported tibial acceleration data of those with and without knee OA while walking at average gait speeds slower than the controlled gait speed reported in this study (Table 4). Thus, the order of gait speed in these studies paralleled the limb dynamics

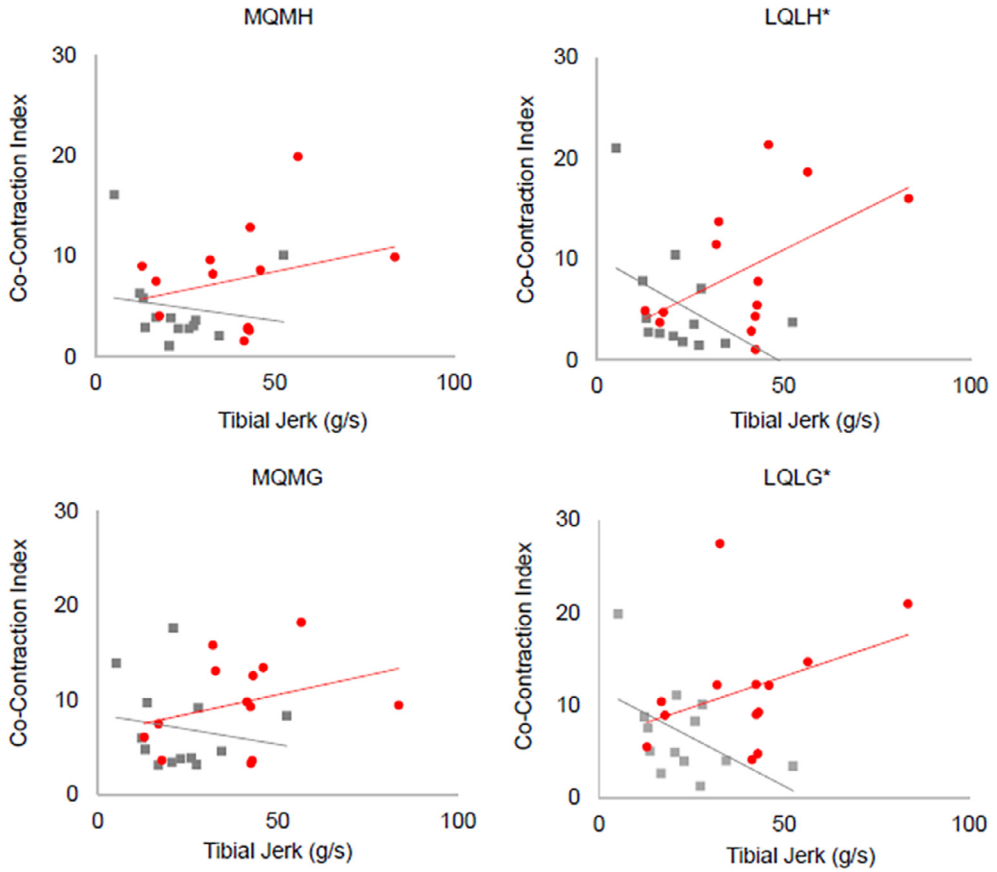

Fig. 3. Group interaction effects between medial quadriceps-medial hamstring (MQMH), lateral quadriceps-lateral hamstring (LQLH), medial quadriceps-medial gastrocnemius (MQMG), and lateral quadriceps-lateral gastrocnemius (LQLG) co-contraction and tibial jerk for the (■) Walking Difficulty group (Diff), the (■) No Walking Difficulty Group (NoDiff), during mid-stance gait interval. (*) Indicates significant interaction effects, $P \leq 0.05$.

Table 4
Average gait speed and average (SD) peak linear acceleration in the x-direction, or sagittal plane, and in the y-direction, or frontal plane, as reported in the literature and found in this study. *Lafortune et al reported data for tibia x and tibia y in non-pathological subjects; therefore additional limb dynamics was not available (NA).

	Lafortune et al.*	Current	Turcot et al.
Average gait speed (m/s)	1.5	1.0	0.85–0.89
Osteoarthritis Group			
Tibia x	NA	1.90 (SD 0.49) g	0.55 (SD 0.26) g
Tibia y	NA	0.67 (SD 0.53) g	0.48(SD 0.37) g
Femur x	NA	1.20 (SD 0.41) g	1.00 (SD 0.27) g
Femur y	NA	0.48 (SD 0.32) g	0.20 (SD 0.17) g
Control Group			
Tibia x	2.33 (SD 0.37) g	1.70 (SD 0.45) g	0.46 (SD 0.19) g
Tibia y	1.28 (SD 0.46) g	0.55 (SD 0.17) g	0.17 (SD 0.06) g
Femur x	NA	1.20 (SD 0.38) g	0.38 (SD 0.21) g
Femur y	NA	0.53 (SD 0.21) g	0.18 (SD 0.11) g

findings respectively (Table 4), which further supported the accuracy of our results.

Consistent with the hypotheses, the relationships between limb dynamics and co-contraction were negative for the NoDiff group but positive for the Diff group. This suggests that self-reported walking difficulty moderated the relationship between limb dynamics and co-contraction among those with knee OA. Statistical significance for the interaction effect was only observed in the lateral co-contraction muscle groups. However, despite the lack of statistical significance, the relationship between medial co-contraction and limb dynamics similarly demonstrated a negative relationship for the NoDiff group and a positive relationship for the Diff group. These findings suggest that greater co-contractions were associated with smoother movements among patients with knee OA but no

walking difficulty, while greater co-contractions were associated with more erratic movements among patients with knee OA and walking difficulty. However, it is unclear if co-contraction produced stabilizing or erratic forces that changed the tibial jerk, or if co-contraction was responding to tibial jerk given the cross-sectional study design. Although further research is needed, perhaps the inverse roles of co-contraction and limb dynamics were beneficial—while increasing co-contraction with greater limb dynamics was a poor movement strategy that requires interventions.

We believe our study findings, especially the moderating effects of walking difficulty on the relationship between limb dynamics and co-contraction, may be explained by sensorimotor adaptations. Knee OA and associated symptom severity, especially pain, can alter sensory input (Kavchak et al., 2012; Skou et al., 2014). Knee OA symptoms that manipulate sensory input are commonly facilitated by joint degradation and disrupted mechanoreceptors, and, in turn, dictate altered movement patterns and strategies that prioritize symptom avoidance (Shanahan, Wrigley, Farrell, Bennell, & Hodges, 2014). Specifically, pain and psychosocial factors can facilitate central sensitization, which, in turn, affects how sensory inputs are processed, motor plans are dictated, and negative behavioral change strategies for symptom management. Although cognitive processing and psychosocial factors were beyond the scope of this study, self-perceived walking difficulty was a consequence of the patient's perception and, thus, can be vulnerable to such influences. For example, kinesophobia, or fear of movement, a construct common among patients with prolonged musculoskeletal symptoms, is known to facilitate catastrophizing thoughts and physical activity avoidance (Larsson, Ekvall Hansson, Sundquist, & Jakobsson, 2016), which could include walking difficulty among patients with knee OA. Hence, future studies should also explore various psychosocial factors that pertain to knee OA and walking difficulty to understand the intersecting relationship between walking ability, afferent sensory signals, and efferent motor commands.

Regardless, perhaps our co-contraction and limb dynamics findings among those with knee OA and no walking difficulty suggested that the absence of central processing factors that related to walking difficulty was ideal for dictating efficient movement patterns for maintaining functional mobility. In turn, patients with knee OA and no walking difficulty appropriately match co-contraction levels to corresponding movement smoothness. In contrast, those with self-reported walking difficulty were unable to optimize motor recruitment, thus potentially resulting in the over- or under-firing of co-contraction. This proposed mechanism is consistent with previous literature that demonstrates the prolonged effects of musculoskeletal conditions on the feedback loop via altered sensory inputs, cortical processing, and subsequent neuromuscular output. Specifically, patients with low back pain (Kiesel et al., 2012; Mehta, Cannella, Smith, & Silfies, 2010; Tsao, Tucker, & Hodges, 2011), shoulder pain (Bandholm, Rasmussen, Aagaard, Diederichsen, & Jensen, 2008), postoperative total knee arthroplasty (Mizner, Stevens, & Snyder-Mackler, 2003), and knee OA (Bennett, Hanratty, Thompson, & Beverland, 2009) have demonstrated that acute and chronic pain can dictate muscle activity. Paralleling these prior study findings, perhaps the group differences based on walking difficulty observed during the mid-stance gait interval of this study can be attributed to distorted neural inputs, processing, and outputs; however, additional studies are needed to confirm these relationships.

This was the first study to test the relationships between co-contraction and limb dynamics, as many previous studies using limb dynamics have only suggested theoretical and mathematical relationships (Flash & Hogan, 1985; Hogan, 1984; Hogan & Sternad, 2009; Roberts et al., 2013; Schneider & Zernicke, 1989; Teulings et al., 1997; Yashiro et al., 2004). Despite the findings, this study had limitations. For example, the study design required subjects to walk at a controlled gait speed of 1.0 m/s, which was at or slower than the regular pace of the subject's self-selected or normal walking speed. This design may cause some subjects to adopt less natural gait patterns due to walking slower, which may influence movement strategies and results. However, we attempted to address this issue by allowing subjects to practice until the controlled gait speed felt natural. Given the findings, it would be beneficial for future studies to consider comparing limb dynamic and muscle co-contraction differences based on OA and walking difficulty presence in variable gait speeds. We also recognize that our subjects in the Diff group primarily included subjects reporting walking to be *somewhat difficult*, and only single subjects reporting walking to be *fairly* or *very difficult*. Consequentially, the results of this study may be limited, given the limited variability among our subjects in walking difficulty levels. Regardless, the results of our study found several differences between the two knee OA subgroups, which makes it likely that more severe walking difficulty should accentuate our findings. Another potential study weakness is the risk of a *type I error* due to repeated testing when using a non-compensated alpha level of 0.05 (Portney & Watkins, 2000). A post-hoc alpha adjustment for a 3 group repeated testing would require a *P*-value < 0.017 for statistical significance (Portney & Watkins, 2000). According to Table 2, limb dynamic group differences based on OA presence will no longer be significant for femoral ($P = .02$) or tibial acceleration ($P = .05$) during mid-stance, and when based on walking difficulty, differences will no longer be significant for tibial acceleration during mid-stance ($P = .03$). However, these variables also yielded medium to large effect sizes, so non-significant findings from *P*-value adjustments may increase the risks of a type 2 error (Table 2). We believe reporting and interpreting effect sizes instead of adjusting the *P*-value would be the best approach to limit risks of *type 1* and *2 errors* for several reasons. First, effect sizes, more so than *P*-values alone, accounted for the experimental nature of this study and the large variances associated with limb dynamics measured during gait (Lafortune, 1991; Roberts et al., 2013). Also, the limb dynamic hypothesis dictated pre-determined comparisons and direction. Therefore, not only were post-hoc alpha adjustments unnecessary, but also the directional hypothesis allowed for a more liberal one-tail test to indicate significance than the two-tailed test currently used in the study. Regardless, significant findings from the first aim were used for the second aim, and, irrespective of *P*-value adjustments, tibial jerk during midstance continued to be significant and indicated for the latter aim. We also recognized the constraints of a cross-sectional study design, as temporal relationships were difficult to discern. However, the findings were promising in providing biomechanical data based on self-reported walking difficulty, a common question already asked in the clinic by clinicians and via self-reported questionnaires (Irrgang et al., 1998). As a result, this study assists in bridging the gap between gait and self-reported constructs.

5. Conclusion

The findings of this study support the importance of looking into knee OA-related walking difficulty, as it can provide insight into biomechanical movement patterns that are not observable via clinical examination. However, the role of co-contraction and optimal movement patterns for those with knee OA continue to remain unclear and would benefit from future studies. Future studies should focus on strategies to reduce knee OA-related walking difficulty. Motor learning related to repetitive training are known to be beneficial in facilitating ideal limb dynamics (Teulings et al., 1997; Turcot et al., 2009) and addressing co-contraction (Chmielewski, Hurd, Rudolph, Axe, & Snyder-Mackler, 2005; Gutierrez, Kaminski, & Douex, 2009; Hall, Tsao, MacDonald, Coppieters, & Hodges, 2009). Although further research is needed, perhaps targeted repetitive neuromuscular training for gait may be useful for those with knee OA-related walking difficulty. Future studies can focus on rehabilitation techniques that may retrain the neuromuscular system and restore the closed-loop system among those with knee OA-related walking difficulty.

6. Disclosure statement

No potential conflicts of interest were reported by the authors.

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