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Muscle force modification strategies are not consistent for gait retraining to reduce the knee adduction moment in individuals with knee osteoarthritis ☆

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ABSTRACT

While gait retraining paradigms that alter knee loads typically focus on modifying kinematics, the underlying muscle force modifications responsible for these kinematic changes remain largely unknown. As humans are generally thought to select uniform gait muscle patterns such as strategies based on fatigue cost functions or energy minimization, we hypothesized that a kinematic gait change known to reduce the knee adduction moment (i.e. toe-in gait) would be accompanied by a uniform muscle force modification strategy for individuals with symptomatic knee osteoarthritis. Ten subjects with self-reported knee pain and radiographic evidence of medial compartment knee osteoarthritis performed normal gait and toe-in gait modification walking trials. Two hundred muscle-actuated dynamic simulations (10 steps for normal gait and 10 steps from toe-in gait for each subject) were performed to determine muscle forces for each gait. Results showed that subjects internally rotated their feet during toe-in gait, which decreased the foot progression angle by 7° ($p < 0.01$) and reduced the first peak knee adduction moment by 20% ($p < 0.01$). While significant muscle force modifications were evidenced within individuals, there were no consistent muscle force modifications across all subjects. It may be that self-selected muscle pattern changes are not uniform for gait modification particularly for individuals with knee pain. Future studies focused on altering knee loads should not assume consistent muscle force modifications for a given kinematic gait change across subjects and should consider muscle forces in addition to kinematics in gait retraining paradigms.

1. Introduction

Knee osteoarthritis (OA) is a significant worldwide health concern characterized by joint pain and dysfunction and can lead to joint stiffness, muscle atrophy, and limb deformity (Buckwalter et al., 2004). In the United States, symptomatic knee OA affects 11% of women and 7% of men over age 60 (Felson et al., 1987) with similar incidence rates reported in China for men and even higher for Chinese women (Du et al., 2005; Zhang et al., 2001). Medications are often used to treat symptoms though disease progression generally leads to total knee replacement (Gabriel et al., 1997). Knee loading is believed to contribute to the degeneration of articular cartilage associated with OA progression (Andriacchi et al., 2004; Schipplein and Andriacchi, 1991). Thus conservative interventions often seek to reduce knee loading for early stage knee OA.

The knee adduction moment (KAM) is an important clinical measurement given the mechanical etiology of knee OA. In vivo instrumented knee replacement testing (D'Lima et al., 2006, 2005) has revealed a strong correlation between medial compartment loading and the KAM and shown that the KAM is a valid, reliable measure of the relative load distribution across the tibiofemoral knee joint (Zhao et al., 2007). It is thus often used as a surrogate measure of medial compartment loading though the estimate is not always guaranteed to be accurate (Walter et al., 2010). The first peak of the KAM has been linked with pain and the presence, severity, and progression of medial compartment knee OA (Hurwitz et al., 2002; Miyazaki et al., 2002; Sharma et al., 1998; Thorp et al., 2007) and the KAM impulse, i.e. the area under

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the KAM-time curve, has been shown to be predictive of cartilage loss over 12 months (Bennell et al., 2011).

Gait retraining is an effective method for reducing the KAM. Initial, proof-of-concept studies in healthy subjects showed that increased trunk sway, toe-in gait (internal foot rotation), reduced tibia angle, and medial thrust were all effective strategies for reducing the first peak KAM (Barrios et al., 2010; Hunt et al., 2011; Mündermann et al., 2008; Shull et al., 2011; Van den Noort et al., 2013), and gait retraining for individuals with knee osteoarthritis has confirmed these initial trends for toe-in gait and increased trunk sway (Shull et al., 2013a; Simic et al., 2012). Gait changes have also been shown to improve symptoms. Shull et al. (2013b) demonstrated that toe-in gait reduced the first peak KAM, reduced pain, and increased function for individuals with symptomatic knee OA after 6 weeks of gait retraining, and improvements in pain and function were approximately 75% larger than the expected placebo effect. Hunt and Takacs (2014) performed 10 weeks of gait retraining and showed that a toe-out gait modification reduced the second peak KAM, the KAM impulse, and knee pain, though it was unclear what portion of knee pain improvement was attributed solely to the placebo effect.

Gait retraining paradigms have thus far focused primarily on the relationship between altered gait kinematics and KAM while neglecting the potentially crucial role that muscle forces might play in intervention. For example, internal muscle forces may lead to higher knee joint compartment loading that is not captured by the KAM (Walter et al., 2010). In addition, uniform kinematic gait modifications shown to reduce knee loads for a population on average can actually be ineffective for individuals within that population (Erhart et al., 2008; Hunt and Takacs, 2014), which has led some to propose subject-specific modifications (Fregly et al., 2007; Gerbrands et al., 2014; Shull et al., 2011). Muscle force modification strategies may thus be crucial to the efficacy of gait retraining.

Although there are many potential muscle force combinations that produce stable gait, humans are generally thought to select uniform muscle patterns while walking such as strategies based on fatigue cost functions or energy minimization (Ackermann and van den Bogert, 2010; Bianchi et al., 1998; Sparrow and Newell, 1998). Thus, we performed this study to test the hypothesis that a kinematic gait change known to reduce the KAM (i.e. toe-in gait) would be accompanied by a uniform muscle force modification strategy for individuals with symptomatic medial compartment knee OA. We further sought to determine the relative degree of force change across individual muscles for the gait modification. Identifying the combinations of muscle force modifications adopted by individuals with symptomatic knee OA provides an objective tool to study and potentially improve gait retraining.

2. Methods

2.1. Subjects

Ten subjects with symptomatic, medial-compartment knee OA participated in this study (Table 1). To be included, subjects were required to have radiographic evidence of medial compartment knee OA defined as Kellgren and Lawrence (K/L) Grade >1. The K/L scale is comprised of four levels of increasing severity (Kellgren and Lawrence, 1957), Grade 1: doubtful narrowing of joint space and possible osteophytic lipping, Grade 2: definite osteophytes and possible narrowing of joint space, Grade 3: moderate multiple osteophytes, definite narrowing of joint space and some sclerosis and possible deformity of bone ends, and Grade 4: large osteophytes, marked narrowing of joint space, severe sclerosis and definite deformity of bone ends. Subjects were also required to have self-reported medial compartment knee pain at least one day per week during the six weeks prior to participation, to be between 18 and 80 years, and to be able to walk unaided for at least 25 consecutive minutes. Exclusion criteria included: body mass index greater than 35; inability to adopt a new gait due to previous injury or surgery on back or lower extremities; use of a shoe insert or hinged knee brace; or corticosteroid injection within the previous six weeks. Gait retraining was focused on the limb with greatest self-reported knee pain (4 right legs, 6 left legs). All subjects gave informed, written consent prior to participation.

2.2. Experimental data collection

Subjects performed weekly gait retraining sessions over six weeks to adopt a toe-in gait pattern (Fig. 1) and each session was experimentally recorded in a motion analysis laboratory. At the beginning of each testing session, a static standing calibration trial was performed with markers placed at the following locations: calcaneous, head of second metatarsal, head of the fifth metatarsal, lateral and medial malleoli, lateral and medial femoral epicondyles, lateral mid-shaft shank (2 markers), greater trochanter, lateral mid-shaft femur (2 markers), left and right anterior

Table 1
Subject characteristics.

	Mean (SD)
Age (yr)	60(13)
BMI (kg/m^2)	26.6(4.7)
Gender	F:4, M:6
Kellgren and lawrence grade	II:3, III:6, IV:1
Foot progression angle (deg)	
Normal gait	2.1(4.0)
Toe-in gait	−5.1(5.1)*
Knee adduction moment (%BW*HT)	
Normal gait	3.11(1.40)
Toe-in gait	2.61(1.47)*
Visual analog pain score	
Normal gait	3.20(2.30)
Toe-in gait	1.35(0.88)*

* Represents a significant difference compared with normal gait at the $p<0.01$ significance level.

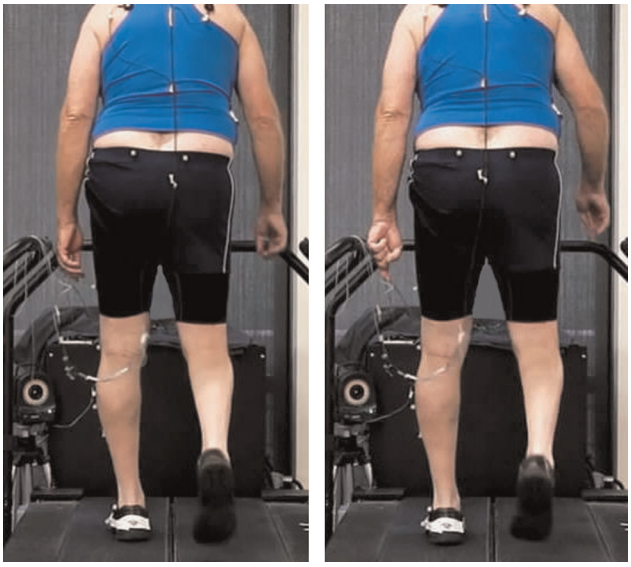

Fig. 1. A representative subject walking with (left) normal gait and (right) toe-in gait. The subject internally rotated the foot by 6° which reduced the first peak knee adduction moment by 20%.

superior iliac spines, left and right posterior superior iliac spines, left and right acromion, and seventh cervical vertebrae. Medial malleolus and medial epicondyle markers were removed for subsequent walking trials. Marker trajectories were recorded with an eight-camera motion capture system (Vicon, Oxford Metrics Group, Oxford, UK) at 60 Hz, and treadmill forces and moments from a split belt instrumented treadmill (Bertec Corporation; Columbus, OH, USA) were recorded at 960 Hz.

Each gait retraining session used real-time streaming motion capture data and real-time feedback to achieve internal foot rotation for toe-in gait. During the first session, the subject walked for a two-minute warm up period establishing a preferred treadmill walking speed (average 1.22 ± 0.21 m/s), which was used for all subsequent trials, and then walked another two minutes during the normal gait trial. Afterward, gait retraining was performed for the remainder of the first session and all subsequent sessions to train a toe-in gait modification with a target 5° of internal foot rotation. A vibration motor (Engineering Acoustics, Inc, FL, USA) was hypoallergenically adhered to the lateral–proximal aspect of the fibula and provided real-time haptic (touch) feedback (Shull et al., 2011) on each step during stance to inform the subject of the desired foot progression angle. A single vibration pulse indicated a required decrease in foot progression angle (toe-in more) and two vibration pulses indicated a required increase in foot progression angle (toe-out more). Data were analyzed from the normal gait trial, the initial walking trial performed at the beginning of the first session, and from the toe-in gait trial, the post-training walking trial performed following the six weeks of gait retraining. Subjects reported knee pain on a visual-analog pain scale before walking at the beginning of the normal gait trial session and the toe-in trial session. The visual-analog pain scale ranged from 0 'no hurt' to 10 'hurts worst' (Wong et al., 2001).

2.3. Muscle force estimation

A three-dimensional, lower-limb musculoskeletal model with 12 degrees of freedom and 43 muscle–tendon actuators was created by modifying the Gait 2392 model in OpenSim (Delp et al., 2007). The contralateral lower extremity, head, upper extremities, and torso were removed and represented as external, or residual, forces and torques acting on the pelvis. The position and orientation of the pelvis relative to ground was defined with 6 degrees of freedom. The remaining lower extremity joints were modeled as follows: the hip as a ball-and-socket joint, the knee as a planar joint with tibiofemoral and patellofemoral translational constraints as a function of knee flexion (Yamaguchi and Zajac, 1989), and the ankle and subtalar joints as revolute joints (Inman, 1976). All inertial parameters for the body segments of the model are derived from Anderson and Pandy (1999). Each muscle–tendon actuator was modeled as a Hill-type muscle in series with a tendon based on musculotendon parameters from Thelen (2003).

Twenty muscle-actuated dynamic simulations (10 consecutive steps from the end of the normal gait trial and 10 consecutive steps from the end of the toe-in gait trial) for each subject during stance phase of gait were created using a three-step process. First, the musculoskeletal model was scaled to represent the experimentally measured size of the subject. Second, inverse kinematics analysis was utilized to obtain values of generalized coordinates for the model that closely matched (RMS marker error < 2 cm) the experimentally measured kinematics of each subject. Third, static optimization (Seth et al., 2011) was implemented as an extension of inverse dynamics that solves the "distribution problem" (i.e. more muscles than joints) to determine individual muscle activations and forces producing the net joint moments at each instant in time that generate the experimentally measured kinematics of the subject.

2.4. Data analysis

To analyze the muscle force modifications for toe-in gait, gait analysis data was filtered, anatomic conventions defined, simulated muscle forces estimated, and differences between normal and toe-in gait computed. Marker data were low-pass filtered at 6 Hz and force plate data at 50 Hz using a zero-lag fourth-order, Butterworth filter. Foot progression angle was defined in the laboratory horizontal plane as the angle between the line connecting the calcaneus and second metatarsal head and the line of forward progression, which was aligned with the long axis of the treadmill. Toe-out was considered positive. Muscle forces were estimated from the muscle-actuated dynamic simulations described in the section above. Mean muscle force was the muscle force estimate averaged over ten steps of stance. Repeated measures, one-way analysis of variance was used to detect a difference among muscle force estimates for normal and toe-in gait; Tukey's method was used for post-hoc pairwise comparison ($\alpha = 0.01$).

3. Results

While significant muscle force modifications were evidenced within individuals, there were no consistent muscle force modifications across all subjects (Table 2). Individuals altered muscle forces to achieve the toe-in gait modification by increasing force in some muscles and decreasing force in others (Table 2). For example, muscle force profiles during stance for a subject walking with the typical toe-in kinematic gait modification demonstrate these muscle force tradeoffs showing increases in solues and gluteus medius force and simultaneous decreases in vastus lateralis and rectus femoris force (Fig. 2). Toe-in gait, on average, decreased the foot progression angle by 7° ($p < 0.01$) and reduced the first peak KAM by 20% ($p < 0.01$) (Fig. 3, Table 1). Knee pain was reduced by 2 points on the visual-analog pain scale ($p < 0.01$) (Table 1).

4. Discussion

This study examined muscle force modifications due to a toe-in gait kinematic modification as compared with normal gait and tested the hypothesis that consistent muscle pattern changes would emerge. Contrary to expectations, muscle force modifications were not consistent across subjects. Muscle force modifications were significant within individuals as evidenced by tradeoffs in the amount of force required among muscles.

Pain might help explain the lack of a uniform muscle force modification strategy for subjects in the present study. While previous research has suggested humans adopt consistent muscle force strategies and by assumption would also modify muscle forces uniformly, these models have been based on pain-free walking in healthy individuals (Ackermann and van den Bogert, 2010; Bianchi et al., 1998; Sparrow and Newell, 1998). However, all individuals in the present study experienced knee pain symptomatic of knee OA, and thus these assumptions may no longer hold. Henriksen et al. (Henriksen et al., 2010) showed that adding experimental knee pain to healthy subjects caused changes in gait patterns in a way that reduced the KAM. Similarly, when knee pain was reduced, individuals with symptomatic knee OA changed their gait patterns in a way that increased the KAM (Shrader et al., 2004). Thus there seems to be a cause-and-effect relationship between knee pain and gait changes, and it may be that this relationship extends to muscle force modifications as well as kinematic changes. Subjects in the present study had varying levels of knee pain and changes in knee pain pre- and post-

Table 2
Percent change in mean muscle force between baseline and toe-in gait for all subjects. Though muscle forces changed within subjects, there were no muscle force modifications across all subjects.

Muscles	Subjects										Mean	STD	*p*-Value
	1	2	3	4	5	6	7	8	9	10			
Adductor brevis	−51.5	−24.4	9.7	102.3	164.2	171.0	52.9	−38.4	38.6	−4.8	42.0	80.4	0.13
Adductor longus	−52.6	37.8	250.2	82.6	212.8	−38.3	159.9	2.3	−24.8	−5.8	62.4	109.4	0.10
Adductor magnus 1	32.3	−32.7	2.0	6.1	−32.7	755.1	57.8	−47.9	18.8	−18.7	74.0	241.5	0.36
Adductor magnus 2	68.8	−21.1	−3.0	10.5	−29.6	199.1	30.7	−18.1	−0.4	−8.1	22.9	68.2	0.32
Adductor magnus 3	72.9	43.4	4.6	50.9	32.0	−38.8	8.8	−12.2	45.6	8.1	21.6	33.5	0.07
Biceps femoris long head	99.9	1.3	−0.8	37.3	−27.5	−19.0	−19.9	8.5	27.3	14.0	12.1	37.2	0.33
Biceps femoris short head	11.5	−41.4	13.8	−22.7	496.8	−47.9	−49.8	37.7	−60.4	54.6	39.2	165.6	0.47
Extensor digitorum longus	−22.9	−2.2	−8.9	−35.3	25.5	226.2	−43.8	−31.7	7.0	−18.0	9.6	78.9	0.71
Extensor hallucis longus	−8.8	5.0	−13.7	−21.5	8.1	127.1	−29.8	−14.3	−0.7	−10.2	4.1	44.7	0.78
Flexor digitorum longus	0.1	4.0	−6.8	3.7	−14.9	1.6	0.4	0.9	12.0	−13.2	−1.2	8.2	0.65
Flexor hallucis longus	0.8	1.7	−6.0	8.6	−27.1	−10.1	11.7	4.6	18.5	−15.3	−1.3	13.6	0.78
Gastrocnemius lateral head	13.8	−8.3	3.8	−4.5	105.2	−34.3	−15.0	25.1	−27.0	36.2	9.5	40.2	0.47
Gastrocnemius medial head	14.4	−9.1	3.7	−4.2	110.0	−35.8	−15.6	26.0	−28.0	40.6	10.2	42.2	0.46
Gemellus	146.8	−67.6	−64.2	−56.7	−94.7	2098.4	−48.8	31.7	9.9	−50.8	190.4	674.1	0.40
Gluteus maximus 1	42.9	−19.7	−32.2	−14.2	−70.1	71.5	−24.9	42.2	52.3	18.4	6.6	45.4	0.66
Gluteus maximus 2	66.5	−28.8	−37.6	−17.7	−84.6	98.2	−16.5	33.5	65.9	13.9	9.3	56.6	0.62
Gluteus maximus 3	104.6	16.0	3.9	11.8	−42.6	45.1	23.8	0.3	28.9	−1.2	19.1	38.0	0.15
Gluteus medius 1	−33.4	81.4	28.9	10.1	115.9	−48.5	−19.1	16.4	−3.1	27.6	17.6	50.3	0.30
Gluteus medius 2	−3.2	7.5	−7.2	−9.1	−2.7	−14.9	−21.5	19.3	1.1	12.5	−1.8	12.4	0.65
Gluteus medius 3	16.1	−14.6	−30.0	−26.4	−53.7	83.5	−29.8	22.4	13.3	2.6	−1.6	38.7	0.90
Gluteus minimus 1	−33.0	94.9	28.6	3.6	258.9	−59.0	−6.6	3.8	−12.2	27.7	30.7	90.0	0.31
Gluteus minimus 2	−17.0	29.7	13.4	−4.3	68.2	−39.7	−10.3	8.4	−3.3	19.0	6.4	29.3	0.51
Gluteus minimus 3	−6.0	6.4	1.7	−12.6	−8.2	−4.1	−18.9	17.5	2.2	10.3	−1.2	11.0	0.74
Gracilis	−6.6	−23.4	86.0	24.2	101.7	−8.0	3.9	−2.5	−45.3	59.9	19.0	48.3	0.25
Iliacus	−23.2	25.2	24.7	−2.9	342.9	−36.6	−20.2	−3.8	−44.0	0.0	26.2	113.6	0.48
Pectineus	−33.1	13.9	147.7	47.3	189.1	−23.3	74.2	−16.1	−23.2	6.0	38.3	77.0	0.15
Peroneus brevis	0.3	6.0	−8.2	−3.1	−2.1	25.3	−12.1	−5.2	1.3	−6.8	−0.5	10.4	0.89
Peroneus longus	−4.5	5.2	1.6	12.0	−16.0	−4.1	8.1	−2.2	22.8	−14.7	0.8	11.9	0.83
Peroneus tertius	−4.8	4.1	−13.0	−16.9	6.3	96.2	−26.1	−8.4	−1.4	−9.0	2.7	34.2	0.81
Piriformis	23.1	−28.0	−35.6	−49.5	−78.2	447.5	−37.4	31.7	3.9	−13.1	26.4	151.7	0.59
Psoas major	−20.1	22.6	21.2	−6.1	325.6	−28.8	−26.6	0.9	−45.8	0.1	24.3	108.1	0.50
Quadratus femoris	51.0	−64.1	−28.3	−53.2	−92.4	318.3	78.2	−4.3	20.5	−45.3	18.0	118.1	0.64
Rectus femoris	−46.9	−38.8	−38.5	−27.1	−59.2	223.1	98.3	−33.1	20.7	−67.2	3.1	91.2	0.92
Sartorius	2.6	−48.3	−21.1	−35.6	2.8	100.2	−22.0	6.3	−24.0	−15.5	−5.5	41.0	0.68
Semimembranosus	79.7	40.3	7.1	24.5	58.1	−17.3	−35.9	15.7	−16.8	29.8	18.5	35.9	0.14
Semitendinosus	60.1	74.7	13.8	52.8	451.4	−60.6	−19.1	−0.7	−43.4	37.0	56.6	145.8	0.25
Soleus	8.9	2.5	−5.1	12.7	−46.3	−17.5	31.4	5.6	38.2	−5.8	2.4	24.0	0.75
Tensor fasciae latae	−14.9	−12.0	−15.3	−39.5	1.5	5.9	−3.1	−3.4	−24.7	−9.4	−11.5	13.3	0.02
Tibialis anterior	−24.5	0.3	−7.4	−39.3	23.0	282.3	−45.5	−37.2	13.7	−19.3	14.6	96.8	0.64
Tibialis posterior	−2.6	3.7	2.8	12.5	−29.5	−10.4	20.3	1.0	27.7	−8.2	1.7	16.3	0.75
Vastus intermedius	0.7	3.0	−29.3	−6.6	−77.6	90.8	103.9	−18.6	32.6	−28.4	7.1	55.4	0.70
Vastus lateralis	0.2	2.8	−30.8	−6.6	−79.9	95.6	117.3	−18.7	34.4	−28.3	8.6	59.5	0.66
Vastus medialis	0.8	2.6	−31.2	−6.1	−79.5	95.7	119.5	−18.5	33.7	−28.4	8.9	59.9	0.65

training (Table 1), which could at least partially account for the inconsistent muscle modification strategies. While pain measures are notoriously difficult to quantify due to subjectivity, future work focused on discovering a link between pain and muscle force strategies could shed light on this issue.

This study provides further evidence for the need to perform subject-specific gait retraining. While training a population to make uniform kinematic changes may work on average, generalized treatments may be ineffective for individuals. Hunt and Takacs (2014) showed that on average 10 weeks of toe-out gait retraining reduced the 2nd peak KAM for 15 individuals with symptomatic knee OA. However, for five of the subjects, toe-out gait either did not change or increased the 2nd peak KAM. Similarly, Erhart et al. (2008) showed that variable stiffness shoes on average initiated gait changes to reduce the 1st peak KAM in a population of 79 individuals with symptomatic knee OA. However, for 18% of these individuals, the 1st peak KAM either did not change or increased. Thus, it has been suggested that gait retraining should be subject-specific to ensure benefits for each individual (Gerbrands et al., 2014). For example, subject-specific gait retraining based on lower limb alignment, baseline gait kinematics, or other factors could inform some subjects to perform toe-in gait (Shull et al., 2013a) to reduce the 1st peak KAM and others to perform toe-out gait (Simic et al., 2013) to achieve the same goal. Subject-specific training should account for subject-to-subject differences in muscle force strategies as they can affect internal forces potentially increasing knee loads, and it is important to identify which muscles necessitate higher forces to inform muscle fatigue and injury prevention. In particular, elevated muscle force is associated with increased muscle fatigue and soreness (Clarkson and Newham, 1995) and increased risk of muscle injury (McMahon, 1984).

The findings in this study contribute to the growing body of literature on gait modification for treatment of early-stage knee OA. Barrios et al. (2010) showed that training subjects to adopt a gait with increased hip adduction and hip internal rotation significantly reduced the KAM. Subjects were instructed to maintain a constant foot progression angle, causing distal kinematics to change, i.e., increased knee flexion and foot eversion. A similar phenomenon occurs for medial thrust gait, which encourages medializing the knee while maintaining a constant foot progression angle (Fregly et al., 2007). However, this modification may be less than optimal as increased knee flexion may increase the overall knee contact force counteracting the benefits of a reduced

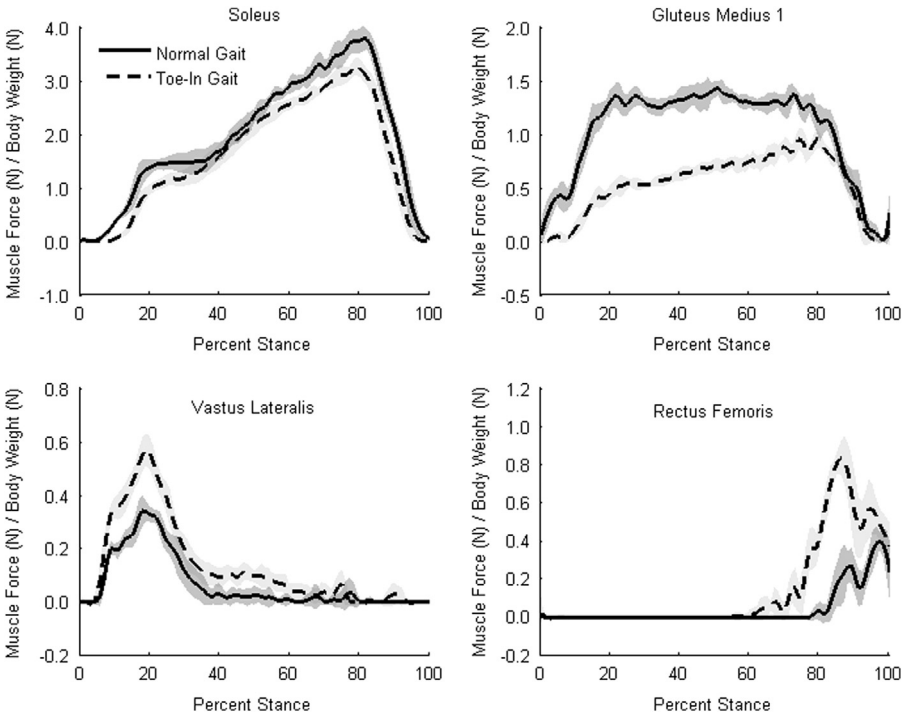

Fig. 2. Example muscle force profiles from the representative subject shown in Fig. 1 showing muscle force tradeoffs to perform toe-in gait. Solues and gluteus medius forces decreased, while vastus lateralis and rectus femoris forces increased. Muscle forces are averaged over ten steps of stance and shading represents one standard deviation. Significant muscle force modifications were evidenced in individuals like this representative subject, though no consistent muscle force modifications emerged for the gait modification across all subjects.

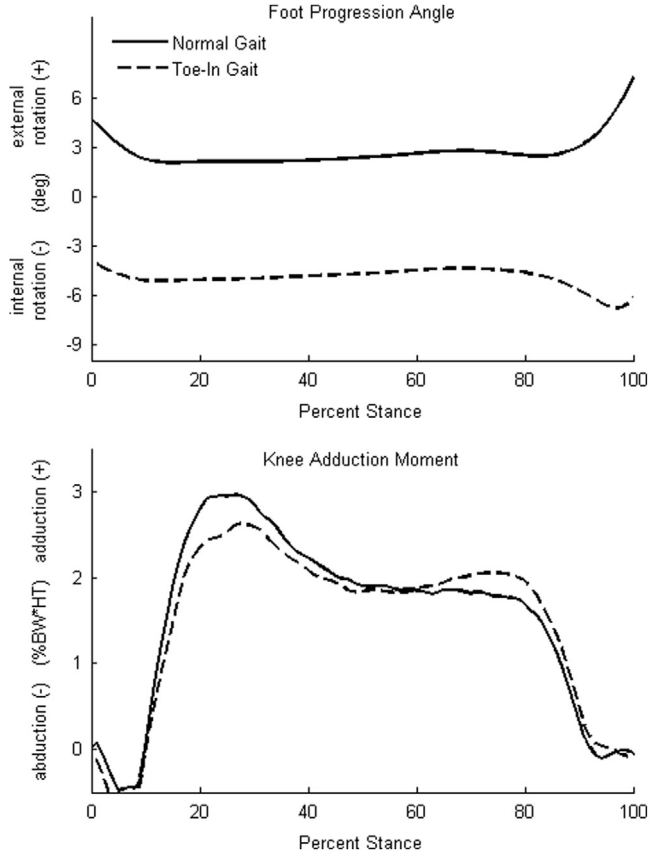

Fig. 3. Averaged (top) foot progression angle and (bottom) knee adduction moment for all subjects for normal and toe-in gait.

KAM in the medial compartment (Walter et al., 2010). The toe-in gait modification performed in the present study doesn't constrain the foot progression angle or hip angles, and thus may be a more natural gait modification (Shull et al., 2013a). Toe-out gait allows similar freedom of movement for all lower limb kinematics, and several studies have shown that toe-out gait reduces the 2nd peak KAM while toe-in gait reduces the 1st peak (Guo et al., 2007; Shull et al., 2013a, 2013b; Simic et al., 2013; Van den Noort et al., 2013). Given the recent interest in wearable, portable systems for gait analysis and intervention (Shull et al., 2014), future real-time feedback gait retraining studies for knee OA may want to incorporate portable electromyographic sensing of muscle forces as complementary input to the feedback control loop. In addition to unassisted gait modifications, several devices have been shown to cause kinematic changes to reduce the KAM, including: center-of-pressure modifying shoes (Haim et al., 2012), lateral wedge insoles (Hinman et al., 2012), variable stiffness shoes (Erhart et al., 2008), and valgus knee braces (Pollo et al., 2002). Current gait retraining paradigms generally target only kinematic changes, however muscle force modifications can also contribute significantly to knee loads and focusing on gait modification strategies incorporating both kinematics and muscle forces could more effectively reduce medial compartment loads.

Though there are different approaches to estimate muscle forces from experimental data, we chose static optimization because it is a well-established, computationally efficient method based on inverse dynamics for estimating in-vivo muscle forces during movement (Seth et al., 2011). Another common modeling approach is dynamic optimization, based on forward dynamics. Dynamic optimization has been shown to produce nearly equivalent solutions to static optimization during gait for predicted muscle forces and joint contact forces (Anderson and Pandy, 2001), though it tends to be computationally expensive typically requiring 1000 times more computation time than static optimization. Erdemir et al. (2007) provide an extensive review of

various modeling approaches including inverse dynamics-based static optimization, optimal control strategies, and alternative methodologies for model-based estimation of muscle forces. Although different approaches may result in different muscle force estimates, the relationship of muscle force modifications observed between normal and toe-in gait would not be likely to change because static optimization was performed consistently across all subjects and the muscle force estimates are constrained by the experimental data and net joint torques generating the movement.

A potential limitation in this study is that subject-to-subject differences in bony geometry and muscle size were not considered. It is possible these parameters play a significant role in solving the muscle force optimization problem and that failing to account for them could lead to inaccurate force estimations. Though our approach of static optimization through scaled musculoskeletal models has been widely used for muscle force estimation, future work incorporating bony geometry and muscle size could improve, clarify, or modify our findings that muscle force strategies are not necessarily consistent across subjects for a given uniform kinematic gait change.

In conclusion, this study showed that muscle force modifications were not consistent for toe-in gait retraining to reduce the knee adduction moment in individuals with knee osteoarthritis. It may be that self-selected muscle pattern changes are not uniform for gait modification particularly for individuals with knee pain. Thus, there is a need for subject-specific gait retraining which accounts for variations in muscle force modification strategies. Furthermore, improved subject-specific musculoskeletal models involving additional patient parameters such as bony geometry and muscle size could further clarify these findings. Future studies focused on altering knee loads should not assume consistent muscle force modifications for a given kinematic gait change across subjects and should consider muscle forces in addition to kinematics in gait retraining paradigms.

Conflict of interest

None of the authors had any conflict of interest regarding this manuscript.

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