Altered hip muscle forces during gait in people with patellofemoral osteoarthritis

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ABSTRACT

Objectives

The study aimed to (i) assess whether higher vasti (VASTI), gluteus medius (GMED), gluteus maximus (GMAX) and gluteus minimus (GMIN) forces are associated with participant characteristics (lower age, male gender) and clinical characteristics (lower radiographic disease severity, lower symptom severity and higher walking speed); and (ii) determine whether hip and knee muscle forces are lower in people with patellofemoral (PFJ) osteoarthritis (OA) compared to those without PFJ OA.

Design

Sixty participants with PFJ OA and 18 (asymptomatic, no radiographic OA) controls ≥40 yrs were recruited from the community or via referrals. A three-dimensional musculoskeletal model was used in conjunction with optimization theory to calculate lower-limb muscle forces during walking. Associations of peak muscle forces with participant and clinical characteristics were conducted using Spearman's rho or independent t-tests and between-group comparisons of mean peak muscle forces performed with walking speed as a covariate.

Results

Peak muscle forces were not significantly associated with participant, symptomatic or radiographic-specific characteristics. Faster walking speed was associated with higher VASTI muscle force in the PFJ OA (r_s=0.499; p<0.001) and control groups (r_s=0.769; p<0.001) and higher GMAX muscle force (r_s=0.579; p=0.012) in the control group only. Individuals with PFJ OA (n=60) walked with lower GMED and GMIN muscle forces than controls (n=18): GMED, mean difference 0.15 [95% confidence interval: 0.01 to 0.29] body weight (BW); GMIN, 0.03 [0.01 to 0.06] BW. No between-group differences were observed in VASTI or GMAX muscle force: VASTI, 0.10 [-0.11 to 0.31] BW; GMAX, 0.01 [-0.11 to 0.09] BW.

Conclusion

Individuals with PFJ OA ambulate with lower peak hip abductor muscle forces than their healthy counterparts.

KEYWORDS

Patella, Knee Pain, Arthritis, Joint Biomechanics, Walking, Musculoskeletal model

RUNNING TITLE

Lower hip muscle forces in patellofemoral osteoarthritis

INTRODUCTION

Patellofemoral joint (PFJ) osteoarthritis (OA) is a common disease, affecting approximately two thirds of those with symptomatic knee OA [1, 2]. Importantly, the PFJ is also a major source of knee pain and reduced physical function [2], exceeding the contribution from the tibiofemoral joint [3, 4]. Despite its prevalence and associated morbidity, little is known about the features of people with PFJ OA. The biomechanics of the PFJ are distinct from the tibiofemoral joint and hence, interventions that have been designed to reduce pain and improve function in those with tibiofemoral disease may be inappropriate for those with predominant PFJ OA. Given the heterogeneity of aetiology, symptomatic presentation and natural history of knee OA, it appears as though optimal interventions should consider targeting the salient features associated with the compartmental involvement [5]. The local PFJ biomechanics, and in particular alignment of the patella within the femoral trochlea, is associated with PFJ OA [6, 7] and its progression [8]. Consequently, the few trials that evaluated targeted interventions for PFJ OA focused on addressing patellar alignment via passive techniques such as taping [6, 9] and bracing [10]. Such treatments resulted in positive immediate effects, but limited longer-term effects. It is possible that individuals exhibit more global impairments (e.g., thigh and hip muscle dysfunction) that should also be addressed in targeted interventions.

While there is a dearth of information on thigh and hip muscle dysfunction in PFJ OA, similarities in pain characteristics and the likely relationship between PFJ pain syndrome and incident PFJ OA imply that analogies may be drawn from the greater body of knowledge in PFJ pain syndrome. Impairments in hip muscle strength, specifically abduction, extension and external rotation, are features of individuals with PFJ pain syndrome [11]. Furthermore, quadriceps weakness, measured via dynamometry, has been identified as a feature of PFJ OA [12] and is associated with progression of OA in the PFJ [13]. The PFJ is intimately related to quadriceps function and consequently, individuals exhibiting pain arising from the PFJ may modify their walking behaviour in order to reduce quadriceps force [14]. However, it is not known whether individuals with PFJ OA ambulate with lower quadriceps and hip muscle forces than their healthy counterparts.

Biomechanical evaluations of people with PFJ pain syndrome are frequently performed to identify impairments in gait. While many studies have calculated net joint torques and powers to evaluate biomechanical load, such measures do not provide quantitative information about the function of individual muscles. Computational musculoskeletal modelling [15] may be used to estimate muscle forces during activities such as gait. Therefore, the aims of this study were to (i) assess whether higher vasti (VASTI), gluteus medius (GMED), gluteus maximus (GMAX) and gluteus minimus (GMIN) forces are associated with participant characteristics (lower age, male gender) and clinical characteristics (lower radiographic disease severity, lower symptom severity and higher walking speed); and (ii) determine whether hip and knee muscle forces are lower in people with PFJ OA compared to those without PFJ OA.

MATERIALS AND METHODS

Participants

Sixty people with symptomatic PFJ OA and 18 controls (no knee pain and no radiographic OA) participated in this study. People with predominant lateral PFJ OA were a subgroup of a larger cohort recruited for a randomised controlled trial [16] from advertisements in the community and via medical and health practitioners' referrals. Inclusion criteria included: (i) aged at least 40 years; (ii) anterior- or retro-patellar knee pain severity ≥ 4 on an 11 point numerical pain scale during at least two activities that load the PFJ (e.g. stair ambulation, squatting and/or rising from sitting); (iii) pain during these activities present on most days during the past month; and (iv) Kellgren and Lawrence (K/L) grading of the lateral PFJ ≥ 2 [17] from skyline views [18] and overall K/L grading (for the tibiofemoral joint) ≤ 2 from postero-anterior views. The control participants were also recruited from the community via advertisements placed in local newspapers and posters. They had no knee or other lower-limb complaints, were physically active and had no radiographic OA (K/L grade ≤ 1 in all compartments). Exclusion criteria included: (i) concomitant pain from other joints affecting lower-limb function; (ii) recent knee injections (prior 3 months); (iii) body mass index ≥ 35 kg.m^{-2}; (iv) knee or hip arthroplasty or osteotomy; (v) physical inability to undertake testing procedures; (vi) neurological or other medical conditions; and (vii) inability to understand written and spoken English. Participants underwent telephone and physical screening by a single researcher (JL) prior to radiographs. Approval was granted from the University of Melbourne Human Research Ethics Committee, and all participants provided written informed consent. Information on age and gender were collected from the participants and body mass index (BMI) calculated from weight and height measurements.

Radiographic disease severity

Radiographic severity of tibiofemoral joint OA was assessed from a semi-flexed, postero-anterior weight-bearing short film radiograph with the feet externally rotated by 10° using the K/L grading system [17]. Radiographic severity of PFJ OA was assessed from weightbearing skyline radiographs, with the knee positioned at 30-40° knee flexion [19], using the K/L grades applied to the PFJ joint[18]. All grading was performed by two investigators (KMC and RSH), with inter-rater reliability (κ) for grading tibiofemoral joint and PFJ radiographic OA on a subset of 39 participants ranging from 0.745-0.843.

Knee osteoarthritis symptoms

The Knee Injury and Osteoarthritis Outcome Score (KOOS) was used to assess patient reported outcomes [20]. The KOOS has five subscales: pain, symptoms, function in activities of daily living (ADL), function in sport and recreation (sport/rec), and knee-related quality of life (QoL). Each of the five subscales addresses symptoms over the previous week, and a normalised score (100 represents no symptoms and 0 represents maximum symptoms) is calculated for each subscale from the original Likert responses. The KOOS is reliable [20] and has face validity for people with PFJ OA symptoms. Thus, in the absence of any PFJ OA-specific outcome measures, the KOOS was deemed to be appropriate for this study.

Calculation of muscle forces

A musculoskeletal computer model, implemented in OpenSim [21], was used to calculate lower-limb muscle forces. Estimates of lower-limb muscle forces for walking obtained using this model have been evaluated previously [22, 23]. The skeleton was represented as an 8-segment, 21-degree-of-freedom linkage (Figure 1A). The head, arms, and torso were modelled as a single rigid body, which articulated with the pelvis via a ball-and-socket back joint. Each hip was modelled as a ball-and-socket joint, and each knee as a modified one-degree-of-freedom planar joint. Each talo-crural joint, subtalar joint and metatarsophalangeal joint was modelled as a hinge. The lower limbs and trunk were actuated by 92 muscle-tendon units, each represented as a line segment joining an origin point on the proximal segment to an insertion point on the distal segment. The paths of muscles that wrapped over underlying structures were modelled using via points [21]. Each muscle-tendon unit was modelled as a three-element Hill-type muscle in series with an elastic tendon [24] (Figure 1B). For each participant, body-segment inertial properties and muscle-tendon properties were scaled from a generic adult model [21] using body mass and segment dimensions as scaling factors, respectively.

Experimental gait data were collected in the Biomotion Laboratory, Department of Mechanical Engineering, University of Melbourne, Australia. Three force plates embedded in the floor of the laboratory were used to record ground reaction forces under both legs at a sampling frequency of 1080 Hz (Advanced Mechanical Technology Inc., Watertown, MA, USA). All ground reaction force data were low-pass filtered using a fourth-order Butterworth filter with a cut-off frequency of 60 Hz. Kinematic data were recorded using a video-based, motion capture system (Vicon, Oxford Metrics, Oxford, UK) with nine cameras sampling at a frequency of 120 Hz. Reflective markers were attached at specific locations on the patient’s trunk, pelvis, both upper limbs and both lower limbs; specifically at the C7 spinous process, acromioclavicular joint, lateral elbow epicondyle, dorsal aspect of the wrist, anterior superior iliac spine, mid-point between posterior superior iliac spines, anterior mid and distal thigh, lateral mid and distal thigh, lateral femoral epicondyle, proximal and distal anteromedial shank, mid lateral shank, heel, lateral malleolus, lateral and medial midfoot, medial aspect of first metatarsal-phalangeal joint, lateral aspect of fifth metatarsal-phalangeal joint, and dorsal aspect of first toe. Muscle electromyographic (EMG) data were collected to enable evaluation of the temporal consistency between muscle force estimates and muscle activations during walking. The EMG data were recorded using pairs of Ag/AgCl surface electrodes (Motion Laboratory Systems, Baton Rouge, LA, USA) mounted on the skin over the gluteus maximus, gluteus medius, medial and lateral vasti, hamstrings, rectus femoris, gastrocnemius and soleus. EMG data were sampled at 1080 Hz. The raw EMG signal was full-wave rectified and a Teager-Kaiser Energy (TKE) filter was then applied to the rectified EMG signal to improve the onset and offset detection [25]. Cross-talk was minimised by following published recommendations regarding the placement of surface electrodes [26].

An initial static trial was performed with the participant standing in a neutral pose and additional markers placed on the left and right medial femoral epicondyles and medial malleoli. Following the static trial, participants performed three gait trials at a self-selected speed on a 10 m level walkway. Each participant's walking speed was calculated from the kinematic data by measuring the average horizontal velocity of a marker mounted on the posterior aspect of the pelvis.

A single representative gait trial for each participant was chosen for analysis, and all analyses were performed in OpenSim [21]. An inverse kinematics problem was solved to determine the model joint angles that best matched the marker data obtained from the gait analysis experiment [27]. The net joint torques were calculated using a traditional inverse dynamics approach [28]. A static optimization problem was then solved to decompose the joint torques into individual muscle forces by minimizing the sum of the squares of the muscle activations [29, 30]. The optimization solution was constrained to the force-length-velocity surface of each muscle [30] (Figure 1C).

The lower-limb muscle forces of interest were: (1) GMAX; (2) GMED; (3) GMIN; and VASTI (vastus lateralis, intermedius and medialis combined). For each muscle group, peak force during the stance phase was identified and then normalised to the participant's body weight (BW).

Statistical analysis

All analyses were performed using the Statistical Package for the Social Sciences (PASW Statistics 18, SPSS Inc., Chicago, IL) with an alpha level of 0.05. Between-group differences in participant and clinical characteristics were assessed using Student's t-tests or chi square tests, as appropriate. The associations between mean peak muscle forces with participant and clinical characteristics were mostly conducted using Pearson's r correlation coefficient. For the radiographic disease severity (ordinal data), the associations were calculated with the Spearman's rho correlation coefficients, while independent t-test were used for gender. Between-group differences in mean peak muscle forces were analysed with walking-speed as a covariate using an Analysis of Covariance (ANCOVA). The sample size (60 PFJ OA patients and 18 controls) provides >90% power to detect a between-group difference in muscle force of 10%, with a standard deviation of 10%.

RESULTS

There were no statistically significant differences for age, height or gender between the PFJ OA group (N=60) and the control group (N=18) (Table 1). Those with PFJ OA were heavier than the control individuals, with a greater BMI. In line with our eligibility criteria, the most prevalent radiographic grade (Table 2) was K/L grade 2 in the lateral PFJ and in the tibiofemoral joint.

Self-selected walking speed was not different between the PFJ OA group and the control group (mean difference [95% confidence interval]: 0.03 [-0.04 to 0.11]) (Table 1). However in the control group, walking speed was significantly correlated with VASTI (r=0.727; p=0.001) and GMAX (r=0.593; p=0.009) peak forces, but age was not statistically significantly correlated with peak muscle forces (Table 3). In the PFJ OA group, walking speed was significantly correlated with VASTI peak force (r=0.495; p<0.001), but age did not statistically significantly correlate with peak muscle forces (Table 3). There was no significant effect of gender on VASTI (0.12: [-0.11 to 0.34], GMAX (-0.03: [-0.12 to 0.06]), GMED (-0.05: [-0.18 to 0.08]) or GMIN

(-0.01: [-0.03 to 0.02]) peak muscle force. Radiographic disease severity in the tibiofemoral joint and lateral PFJ were not statistically significantly correlated with peak muscle forces in the PFJ OA group (Table 3). Additionally, no statistically significant correlations were observed between any subscale of the KOOS and peak muscle forces in the PFJ OA group (Table 3).

There were differences in the peak muscle forces for GMED and GMIN between the PFJ OA group and control group (Table 4 and Figure 2). Individuals with PFJ OA walked with lower GMED (0.15 [95% confidence interval: 0.01 to 0.29] BW); and GMIN (0.03 [0.01 to 0.06] BW) muscle forces than controls. No between-group differences were observed in VASTI or GMAX muscle force: VASTI, 0.10 [-0.11 to 0.31] BW; GMAX, 0.01 [-0.11 to 0.09] BW. Ensemble averages across the stance phase of gait for normalised muscle forces are presented in Figure 2. Model predictions of muscle forces were in temporal agreement with measured EMG activity (Supplementary Figure 1), providing a qualitative evaluation of the modelling approach used in this study.

DISCUSSION

Awareness of the importance of the PFJ in the clinical picture of knee OA is increasing due to its prevalence and contribution to knee OA symptoms. Knowledge of impairments associated with this subgroup of people with knee OA will advance our understanding of this chronic disease. We found that people with PFJ OA walk with reduced hip abductor muscle forces, compared with pain-free, aged-matched controls. Specifically, peak GMED and GMIN muscle forces were approximately 11% lower than pain-free individuals. The variability in GMAX, GMED, GMIN and VASTI peak muscle forces was not related to radiographic disease severity, knee osteoarthritis symptom severity or other participant characteristics.

To our knowledge, this is the first study to estimate lower-limb muscle forces in a cohort of people with PFJ OA. Our calculated peak VASTI muscle force was higher (1.16 [95% confidence interval 1.06 to 1.26] BW) than those reported for a single TKA patient walking with an instrumented knee (0.73 BW) [22]. The temporal patterns of lower-limb muscle forces were similar between the two studies. Since walking speed is known to influence the magnitude of lower-limb muscle forces [15], it is possible that differences in walking speed between the current study (1.37 [1.30 to 1.44] m.s^{-1} , Table 1) and that of Kim et al. [22] (1.24 (standard deviation 0.33) m.s^{-1}) partially explain some of the differences observed in the calculated values of muscle forces. Furthermore, in the study by Kim and colleagues [22], the patient had end stage OA warranting a total joint replacement, whereas our study contained individuals with no greater than K/L grade 2 radiographic OA. Thus, differences in peak VASTI force may be partially accounted for by the disparity in the patient population. It is difficult to directly compare our data with that from a previous study [31], where lower-limb muscle forces were computed for a cohort of younger individuals with PFJ pain syndrome, because an EMG-driven modelling approach was used and all muscle force data were normalised by the peak isometric force of each muscle.

Our finding of lower peak GMED and GMIN forces in those with PFJ OA is consistent with emerging evidence that hip muscle dysfunction is a dominant feature of individuals with PFJ pain syndrome [11]. The GMED and GMIN primarily contribute to hip abduction moments during walking [32]. Since the cross-sectional nature of the study design precludes knowledge of the temporal relationship between lower GMED and GMIN muscle forces and PFJ OA development or progression, further studies are required to confirm the clinical implications of our findings. Our results indicate that individuals with PFJ OA exhibit altered function that is isolated to the more proximal segments, providing further evidence for a potential link to PFJ pain syndrome.

We found no difference in peak VASTI and GMAX muscle force in those with and without PFJ OA. Our results contrast with previous studies that have measured peak isometric knee-extensor torque using a dynamometer [12, 13]; however, there is an imprecise relationship between knee-extensor torque measured in an open-kinetic-chain task and peak muscle force utilised during a functional activity such as walking. Our results may reflect variability in gait adaptations during walking in our population of people with symptomatic PFJ OA. Notably, there was a non-significant lower peak VASTI muscle force (~8%) in our PFJ OA patients, which may reflect that some individuals are likely to walk with lowered VASTI force, potentially as a pain-relieving strategy. It is also possible that deficits in the coordination (e.g. onset timing) of the medial and lateral components of the vasti may be more important than the total peak VASTI force in individuals with PFJ OA, in a similar manner to PFJ syndrome [33]. Future studies might evaluate VASTI and GMAX muscle forces in functional tasks, such as stair ambulation, which subject the PFJ to greater load, or evaluate the relative coordination of the medial and lateral vasti.

Peak muscle forces were mostly not correlated with participant, symptomatic or radiographic-specific characteristics, implying that muscle forces alone do not reflect the severity of radiographic or symptomatic disease. Although previous investigations of individuals with predominantly tibiofemoral joint OA have observed associations between radiographic OA severity and kinematics at the hip [34], these observations were only significant for those with severe radiographic OA (K/L grade 4). Similarly, many authors have noted a difference in the knee adduction moment only in those with more severe radiographic tibiofemoral disease [35]. It appears likely that changes in gait mechanics at the knee may be associated with the structural changes that accompany the OA disease process, such as altered frontal plane alignment. Since our cohort was restricted to those with a K/L grade ≤ 2, it is not surprising that radiographic disease severity was not associated with peak muscle loading during gait. Although faster walking speed was associated with a higher peak VASTI muscle force in the PFJ OA and control groups and a higher peak GMAX muscle force in the control group, walking speed was controlled for statistically and therefore the between-group differences in muscle force noted in the current study were not attributable to differences in walking speed.

It is not possible to discern the function of individual muscles from net joint torques alone, simply because a given joint torque can be satisfied by an infinite combination of muscle forces. Musculoskeletal modelling represented the only practicable method for determining lower-limb muscle forces in the current study.

However, there are several limitations and assumptions inherent in this modelling approach; for example, the physiological properties prescribed for the muscle-tendon actuators included in the model (e.g., peak isometric muscle force and the corresponding muscle-fiber length and tendon rest length; see Figure 1B). Importantly, the present study assumed the same unscaled muscle-tendon parameters across both the PFJ OA and control population and hence, any relative differences in muscle force predictions are attributable mostly to differences in the experimental gait data and not the parameters assumed in the model. We also elected to analyse synergistic groups of muscles (i.e., GMAX, GMED, GMIN, VASTI) and did not attempt to partition calculated forces onto the various components of these muscle groups (e.g., vastus medialis vs intermedius vs lateralis within VASTI). Several studies have shown that our approach of obtaining muscle force estimates for synergistic groups of muscles is relatively insensitive to changes in the values assumed for peak isometric muscle force (or physiological cross-sectional area) [36-38]. Despite the aforementioned limitations, the inverse-dynamics-based optimisation approach employed in the current study is robust, computationally efficient, and has been used extensively to estimate lower-limb muscle forces in walking [15, 39, 40]. Furthermore, indirect evidence is available to support the validity of predicting lower-limb muscle forces during walking using the approach taken in the present paper [22]. Lastly, previous studies have shown temporal agreement between predicted lower-limb muscle forces and recorded EMG [41], and this relationship was also demonstrated in the present study for a representative subject (Supplementary Figure 1).

A final limitation relates to the participant characteristics of the control group. Although we attempted to recruit participants who were matched on variables likely to influence muscle forces, the control group was lighter and trending towards being younger. We controlled for body weight by normalising all muscle force data, and age was not associated with the muscle force data. In order to be included in the control group, participants had to exhibit a K/L grade ≤ 1. While this is usually accepted as a criterion for no OA, it is possible that participants in the control group had some early/mild OA that may have affected their gait pattern. It is also possible that some participants in either group may have had coexisting hip OA. However, the control group were required to have no knee pain and all participants were required to report no hip/groin or lower-back symptoms. The sample size for the control group was chosen to be as large as could be practically achieved within the time and resource constraints, and consequently the control group included much fewer participants (N=18) than our PFJ OA cohort (N=60). This difference reflected the difficulties in recruiting an older population from the general community with no knee or other lower-limb complaints, who were physically active with no radiographic knee OA, and who had the time and inclination to attend for both radiographic and biomechanical evaluation. Nevertheless, our sample size calculations revealed that we had sufficient power, despite the discrepant sample sizes.

This study is the first to investigate the walking mechanics of individuals with predominant PFJ OA. Our findings indicate that individuals with PFJ OA ambulate with lower peak hip abductor muscle force than their healthy counterparts. It is not known whether a lower hip abductor muscle force contributes to, or is a consequence of, the PFJ OA disease process.

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Author contributions

Kay M Crossley obtained funding, designed and managed the project, assisted with statistical analyses and writing of the paper

Tim W Dorn and Josien van den Noort performed the muscle force calculations

Hannah Ozturk assisted with participant recruitment and collected the gait data

Anthony G Schache obtained funding and assisted with the data collection, analyses and writing of the paper

Marcus G Pandy obtained funding and assisted with the calculations of muscle force and writing of the paper

All authors contributed to the editing and approval of this work

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Conflict of interest

KMC, TWD, HO, JVDN, AGS MGP and do not have any conflicts of interest to declare

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Table 1: Participant and clinical characteristics: patellofemoral osteoarthritis and control groups

	Pain-free Control † N = 18	Patellofemoral osteoarthritis † N = 60	Mean difference [95% CI]	p value
Age (yr)	53 (7)	58 (10)	4 [-0.8 to 10]	0.096
Height (m)	1.65 (0.08)	1.69 (0.09)	0.03 [-0.06 to 0.08]	0.186
Weight (kg)	66 (12)	78 (13)	12 [5 to 19]	0.001*
BMI ($kg.m^{-2}$)	24.1 (3.4)	27.5 (3.7)	3.3 [1.4 to 5.3]	0.001*
Gender (n(%))	14 female (78%)	39 female (65%)	-	‡0.236
KOOS-pain	-	63 (15)	-	-
KOOS-symptoms	-	61 (16)	-	-
KOOS-ADL	-	70 (16)	-	-
KOOS-Sport/Rec	-	41 (22)	-	-
KOOS-QoL	-	12 (16)	-	-
Walking speed ($m.s^{-1}$)	1.34 (0.13)	1.37 (0.17)	0.03 [-0.04 to 0.11]	0.369

† all values are mean (SD) unless indicated; CI = confidence interval; ‡ $\chi 2$; * Denotes statistically significant, $p < 0.05$

BMI = body mass index

KOOS = Knee Injury and Osteoarthritis Outcome Score [20] (100 =no symptoms - 0 = maximum symptoms)

KOOS-pain = pain subscale of the KOOS

KOOS-symptoms =symptoms subscale of the KOOS

KOOS-ADL = activities of daily living subscale of the KOOS

KOOS-Sport/Rec =Sport and recreation subscale of the KOOS

KOOS-QoL =Knee-related quality of life subscale of the KOOS

Table 2: Radiographic disease severity for the patellofemoral osteoarthritis group (n = 60)

	Grade 0 n (%)	Grade 1 n (%)	Grade 2 n (%)	Grade 3 n (%)	Grade 4 n (%)
Tibiofemoral (K/L)	14 (23%)	18 (30%)	28 (47%)	0	0
Lateral patellofemoral (K/L)†	0	0	39 (65%)	11 (18%)	10 (17%)

K/L Kellgren and Lawrence scale [17]: 0 = no osteoarthritis ; 4 = severe osteoarthritis

K/L† Kellgren and Lawrence scale adapted for patellofemoral joint [18]

Table 3: Univariate associations between normalised peak muscle forces, participant and clinical characteristics

		GMAX	GMED	GMIN	VASTI
Control group (n=18)					
Age (yr)	r	-0.195	-0.96	-0.84	106
	p	0.439	0.705	0.739	0.676
Walking speed ($m.s^{-1}$)	r	0.593	0.076	0.047	0.727
	p	0.009*	0.764	0.853	0.001*
PFJ OA group (n=60)					
Age (yr)	R	-0.86	-0.138	-0.125	-0.174
	P	0.514	0.295	0.340	0.185
KOOS-pain	R	0.081	0.050	0.023	0.103
	p	0.539	0.704	0.859	0.435
KOOS-symptoms	r	-0.026	-0.029	-0.041	0.069
	p	0.843	0.826	0.754	0.598
KOOS-ADL	r	-0.045	-0.053	-0.062	0.133
	p	0.734	0.685	0.637	0.311
KOOS-Sport/Rec	r	-0.018	-0.050	-0.056	0.261
	p	0.892	0.702	0.669	0.097
KOOS-QoL	r	-0.059	-0.036	-0.029	0.111
	p	0.652	0.784	0.824	0.396
Walking speed ($m.s^{-1}$)	r	0.145	0.090	0.040	0.495
	p	0.267	0.494	0.763	<0.001*
Tibiofemoral (K/L)‡	rho	-0.040	0.114	0.108	0.058
	p	0.763	0.385	0.413	0.660
Lateral patellofemoral (K/L) ‡†	rho	-0.084	0.104	0.060	-0.072
	p	0.524	0.430	0.649	0.586

Correlations using Pearson's r correlation co-efficient unless indicated

‡Correlations using Spearman's rho correlation co-efficient * Denotes statistically significant, $p < 0.05$

GMAX = gluteus maximus

GMED = gluteus medius

GMIN= gluteus minimus

VASTI = vastus medialis, vastus lateralis and vastus intermedialis

KOOS = Knee Injury and Osteoarthritis Outcome Score [20] (100=no symptoms – 0=maximum symptoms)

KOOS-pain = pain subscale of the KOOS

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KOOS-ADL = activities of daily living subscale of the KOOS

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KOOS-QoL =Knee-related quality of life subscale of the KOOS

K/L Kellgren and Lawrence scale [17]: 0 = no osteoarthritis ; 4 = severe osteoarthritis

K/L† Kellgren and Lawrence scale adapted for patellofemoral joint [18]

Table 4: Between group comparisons of normalised peak muscle forces between symptomatic PFJ OA and control groups

	Pain-free Control † N = 18	Patellofemoral osteoarthritis † N = 60	Mean difference †	P
GMAX (BW)	0.69 [0.61 to 0.78]	0.70 [0.66 to 0.75]	0.01 [-0.11 to 0.09]	0.796
GMED (BW)	1.41 [0.28 to 1.53]	1.26 [1.19 to 1.33]	0.15 [0.01 to 0.29]	0.041*
GMIN (BW)	0.24 [0.22 to 0.26]	0.21 [0.20 to 0.22]	0.03 [0.01 to 0.06]	0.013*
VASTI (BW)	1.26 [1.08 to 1.44]	1.16 [1.06 to 1.26]	0.10 [-0.11 to 0.31]	0.355

† all values are mean *95% confidence interval+ and adjusted for walking speed; * Denotes statistically significant, $p < 0.05$

BW = body weight

GMAX = gluteus maximus

GMED = gluteus medius

GMIN= gluteus minimus

VASTI = vastus medialis, vastus lateralis and vastus intermedialis

FIGURE CAPTIONS

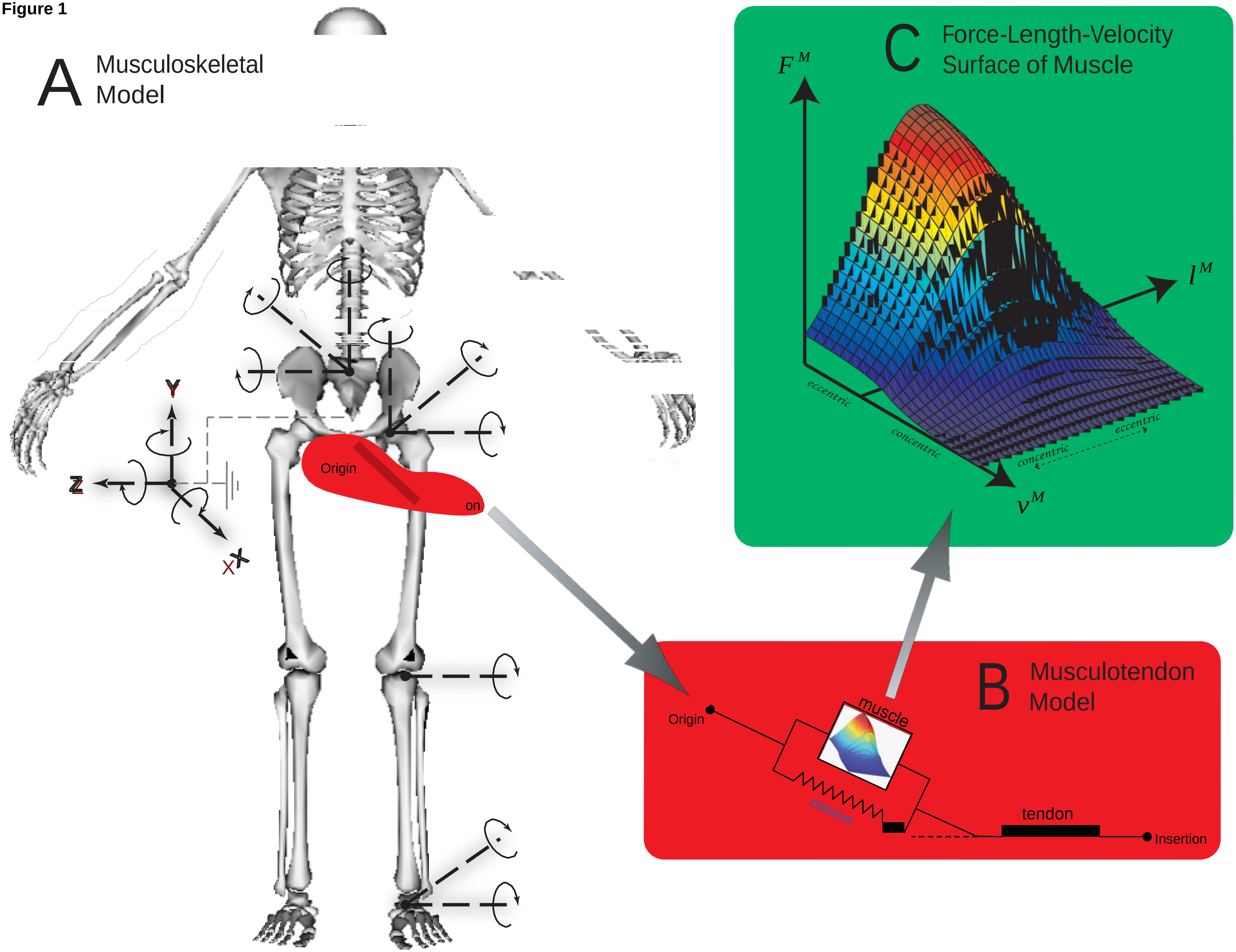
Fig. 1: Three-dimensional musculoskeletal model used in the present study. (A) The skeleton was modeled as a multi-body linkage comprised of 21 degrees of freedom, and was actuated by 92 muscle-tendon units. (B) Each muscle-tendon actuator was represented as a Hill-type muscle (active and passive) in series with an elastic tendon. (C) The active force, F^M, developed by muscle was governed by its force-length-velocity surface, defined by the muscle's length, L^M, and velocity of contraction, V^M.

Fig. 2: Muscle forces during the stance phase of walking. Mean (±1SD) data are presented for the control (solid line (mean) with dark grey shading (SD); N=18) and PF JOA (dashed line (mean) with light grey shading (SD); N=60) populations. Muscle symbols appearing in the graphs are: GMAX (gluteus maximus), GMED (gluteus medius), GMIN (gluteus minimus) and VASTI (vastus lateralis, vastus medialis and vastus intermedius heads). IFS, IFO, CFS and CFO signify ipsilateral foot-strike, ipsilateral foot-off, contralateral foot-strike and contralateral foot-off, respectively.

Figure Legends

Supp Figure: Model predicted muscle forces and experimental EMG signals for a representative PFJ OA subject (n=1; Body weight = 59kg) for walking at a self-selected speed of 1.34 m/s. Data are shown from foot strike to foot strike; shaded region represents the stance phase. Muscle symbols appearing in the graphs are: GMAX (gluteus maximus), GMED (gluteus medius), HAMS (hamstrings; semimembranous, semitendinosus, biceps femoris long head and biceps femoris short head combined), RF (rectus femoris), VASTI (vastus medialis, vastus intermedius and vastus lateralis combined); GAS (gastrocnemius) and SOL (soleus). Muscle electromyographic (EMG) activity was recorded using pairs of Ag/AgCl surface electrodes (Motion Laboratory Systems, Baton Rouge, LA, USA) mounted on the skin over the gluteus maximus, gluteus medius, vastus lateralis, medial hamstrings (semitendinosus), rectus femoris, medial gastrocnemius and soleus.

Figure 1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Musculoskeletal Model
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Origin
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Force-Length-Velocity Surface of Muscle
F^M
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Musculotendon Model
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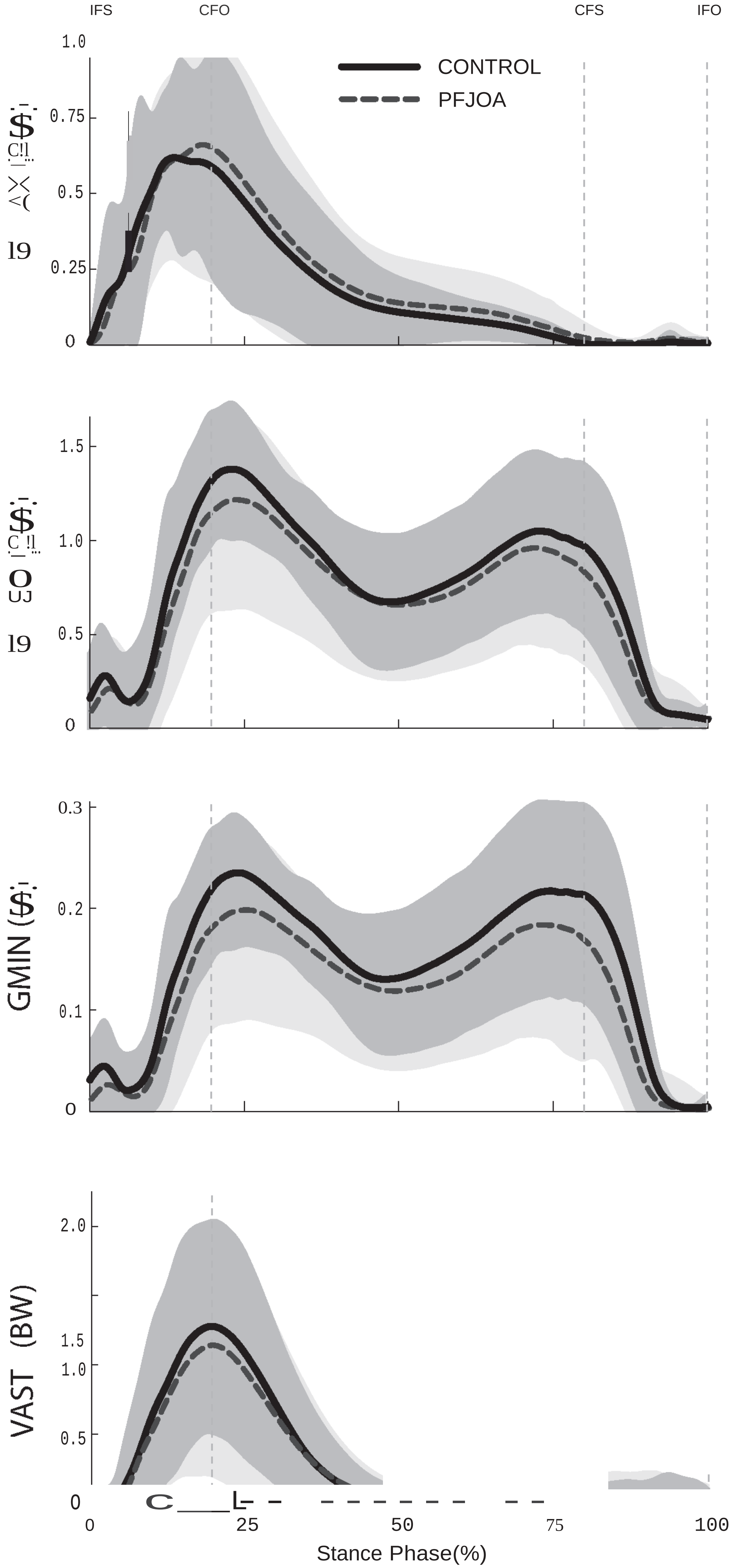
Figure 2



IFS
CFO
CFS
IFO
CONTROL
PFJOA
1.0
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0.5
0.25
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1.0
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GMIN
0.3
0.2
0.1
0
VAST (BW)
2.0
1.5
1.0
0.5
0
0
25
50
75
100
Stance Phase(%)

Altered hip muscle forces during gait in people with patellofemoral osteoarthritis

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ABSTRACT

Objectives

The study aimed to (i) assess whether higher vasti (VASTI), gluteus medius (GMED), gluteus maximus (GMAX) and gluteus minimus (GMIN) forces are associated with participant characteristics (lower age, male gender) and clinical characteristics (lower radiographic disease severity, lower symptom severity and higher walking speed); and (ii) determine whether hip and knee muscle forces are lower in people with patellofemoral (PFJ) osteoarthritis (OA) compared to those without PFJ OA.

Design

Sixty participants with PFJ OA and 18 (asymptomatic, no radiographic OA) controls ≥40 yrs were recruited from the community or via referrals. A three-dimensional musculoskeletal model was used in conjunction with optimization theory to calculate lower-limb muscle forces during walking. Associations of peak muscle forces with participant and clinical characteristics were conducted using Spearman's rho or independent t-tests and between-group comparisons of mean peak muscle forces performed with walking speed as a covariate.

Results

Peak muscle forces were not significantly associated with participant, symptomatic or radiographic-specific characteristics. Faster walking speed was associated with higher VASTI muscle force in the PFJ OA (r_s=0.499; p<0.001) and control groups (r_s=0.769; p<0.001) and higher GMAX muscle force (r_s=0.579; p=0.012) in the control group only. Individuals with PFJ OA (n=60) walked with lower GMED and GMIN muscle forces than controls (n=18): GMED, mean difference 0.15 [95% confidence interval: 0.01 to 0.29] body weight (BW); GMIN, 0.03 [0.01 to 0.06] BW. No between-group differences were observed in VASTI or GMAX muscle force: VASTI, 0.10 [-0.11 to 0.31] BW; GMAX, 0.01 [-0.11 to 0.09] BW.

Conclusion

Individuals with PFJ OA ambulate with lower peak hip abductor muscle forces than their healthy counterparts.

KEYWORDS

Patella, Knee Pain, Arthritis, Joint Biomechanics, Walking, Musculoskeletal model

RUNNING TITLE

Lower hip muscle forces in patellofemoral osteoarthritis

INTRODUCTION

Patellofemoral joint (PFJ) osteoarthritis (OA) is a common disease, affecting approximately two thirds of those with symptomatic knee OA [1, 2]. Importantly, the PFJ is also a major source of knee pain and reduced physical function [2], exceeding the contribution from the tibiofemoral joint [3, 4]. Despite its prevalence and associated morbidity, little is known about the features of people with PFJ OA. The biomechanics of the PFJ are distinct from the tibiofemoral joint and hence, interventions that have been designed to reduce pain and improve function in those with tibiofemoral disease may be inappropriate for those with predominant PFJ OA. Given the heterogeneity of aetiology, symptomatic presentation and natural history of knee OA, it appears as though optimal interventions should consider targeting the salient features associated with the compartmental involvement [5]. The local PFJ biomechanics, and in particular alignment of the patella within the femoral trochlea, is associated with PFJ OA [6, 7] and its progression [8]. Consequently, the few trials that evaluated targeted interventions for PFJ OA focused on addressing patellar alignment via passive techniques such as taping [6, 9] and bracing [10]. Such treatments resulted in positive immediate effects, but limited longer-term effects. It is possible that individuals exhibit more global impairments (e.g., thigh and hip muscle dysfunction) that should also be addressed in targeted interventions.

While there is a dearth of information on thigh and hip muscle dysfunction in PFJ OA, similarities in pain characteristics and the likely relationship between PFJ pain syndrome and incident PFJ OA imply that analogies may be drawn from the greater body of knowledge in PFJ pain syndrome. Impairments in hip muscle strength, specifically abduction, extension and external rotation, are features of individuals with PFJ pain syndrome [11]. Furthermore, quadriceps weakness, measured via dynamometry, has been identified as a feature of PFJ OA [12] and is associated with progression of OA in the PFJ [13]. The PFJ is intimately related to quadriceps function and consequently, individuals exhibiting pain arising from the PFJ may modify their walking behaviour in order to reduce quadriceps force [14]. However, it is not known whether individuals with PFJ OA ambulate with lower quadriceps and hip muscle forces than their healthy counterparts.

Biomechanical evaluations of people with PFJ pain syndrome are frequently performed to identify impairments in gait. While many studies have calculated net joint torques and powers to evaluate biomechanical load, such measures do not provide quantitative information about the function of individual muscles. Computational musculoskeletal modelling [15] may be used to estimate muscle forces during activities such as gait. Therefore, the aims of this study were to (i) assess whether higher vasti (VASTI), gluteus medius (GMED), gluteus maximus (GMAX) and gluteus minimus (GMIN) forces are associated with participant characteristics (lower age, male gender) and clinical characteristics (lower radiographic disease severity, lower symptom severity and higher walking speed); and (ii) determine whether hip and knee muscle forces are lower in people with PFJ OA compared to those without PFJ OA.

MATERIALS AND METHODS

Participants

Sixty people with symptomatic PFJ OA and 18 controls (no knee pain and no radiographic OA) participated in this study. People with predominant lateral PFJ OA were a subgroup of a larger cohort recruited for a randomised controlled trial [16] from advertisements in the community and via medical and health practitioners' referrals. Inclusion criteria included: (i) aged at least 40 years; (ii) anterior- or retro-patellar knee pain severity ≥ 4 on an 11 point numerical pain scale during at least two activities that load the PFJ (e.g. stair ambulation, squatting and/or rising from sitting); (iii) pain during these activities present on most days during the past month; and (iv) Kellgren and Lawrence (K/L) grading of the lateral PFJ ≥ 2 [17] from skyline views [18] and overall K/L grading (for the tibiofemoral joint) ≤ 2 from postero-anterior views. The control participants were also recruited from the community via advertisements placed in local newspapers and posters. They had no knee or other lower-limb complaints, were physically active and had no radiographic OA (K/L grade ≤ 1 in all compartments). Exclusion criteria included: (i) concomitant pain from other joints affecting lower-limb function; (ii) recent knee injections (prior 3 months); (iii) body mass index ≥ 35 kg.m^{-2}; (iv) knee or hip arthroplasty or osteotomy; (v) physical inability to undertake testing procedures; (vi) neurological or other medical conditions; and (vii) inability to understand written and spoken English. Participants underwent telephone and physical screening by a single researcher (JL) prior to radiographs. Approval was granted from the University of Melbourne Human Research Ethics Committee, and all participants provided written informed consent. Information on age and gender were collected from the participants and body mass index (BMI) calculated from weight and height measurements.

Radiographic disease severity

Radiographic severity of tibiofemoral joint OA was assessed from a semi-flexed, postero-anterior weight-bearing short film radiograph with the feet externally rotated by 10° using the K/L grading system [17]. Radiographic severity of PFJ OA was assessed from weightbearing skyline radiographs, with the knee positioned at 30-40° knee flexion [19], using the K/L grades applied to the PFJ joint[18]. All grading was performed by two investigators (KMC and RSH), with inter-rater reliability (κ) for grading tibiofemoral joint and PFJ radiographic OA on a subset of 39 participants ranging from 0.745-0.843.

Knee osteoarthritis symptoms

The Knee Injury and Osteoarthritis Outcome Score (KOOS) was used to assess patient reported outcomes [20]. The KOOS has five subscales: pain, symptoms, function in activities of daily living (ADL), function in sport and recreation (sport/rec), and knee-related quality of life (QoL). Each of the five subscales addresses symptoms over the previous week, and a normalised score (100 represents no symptoms and 0 represents maximum symptoms) is calculated for each subscale from the original Likert responses. The KOOS is reliable [20] and has face validity for people with PFJ OA symptoms. Thus, in the absence of any PFJ OA-specific outcome measures, the KOOS was deemed to be appropriate for this study.

Calculation of muscle forces

A musculoskeletal computer model, implemented in OpenSim [21], was used to calculate lower-limb muscle forces. Estimates of lower-limb muscle forces for walking obtained using this model have been evaluated previously [22, 23]. The skeleton was represented as an 8-segment, 21-degree-of-freedom linkage (Figure 1A). The head, arms, and torso were modelled as a single rigid body, which articulated with the pelvis via a ball-and-socket back joint. Each hip was modelled as a ball-and-socket joint, and each knee as a modified one-degree-of-freedom planar joint. Each talo-crural joint, subtalar joint and metatarsophalangeal joint was modelled as a hinge. The lower limbs and trunk were actuated by 92 muscle-tendon units, each represented as a line segment joining an origin point on the proximal segment to an insertion point on the distal segment. The paths of muscles that wrapped over underlying structures were modelled using via points [21]. Each muscle-tendon unit was modelled as a three-element Hill-type muscle in series with an elastic tendon [24] (Figure 1B). For each participant, body-segment inertial properties and muscle-tendon properties were scaled from a generic adult model [21] using body mass and segment dimensions as scaling factors, respectively.

Experimental gait data were collected in the Biomotion Laboratory, Department of Mechanical Engineering, University of Melbourne, Australia. Three force plates embedded in the floor of the laboratory were used to record ground reaction forces under both legs at a sampling frequency of 1080 Hz (Advanced Mechanical Technology Inc., Watertown, MA, USA). All ground reaction force data were low-pass filtered using a fourth-order Butterworth filter with a cut-off frequency of 60 Hz. Kinematic data were recorded using a video-based, motion capture system (Vicon, Oxford Metrics, Oxford, UK) with nine cameras sampling at a frequency of 120 Hz. Reflective markers were attached at specific locations on the patient’s trunk, pelvis, both upper limbs and both lower limbs; specifically at the C7 spinous process, acromioclavicular joint, lateral elbow epicondyle, dorsal aspect of the wrist, anterior superior iliac spine, mid-point between posterior superior iliac spines, anterior mid and distal thigh, lateral mid and distal thigh, lateral femoral epicondyle, proximal and distal anteromedial shank, mid lateral shank, heel, lateral malleolus, lateral and medial midfoot, medial aspect of first metatarsal-phalangeal joint, lateral aspect of fifth metatarsal-phalangeal joint, and dorsal aspect of first toe. Muscle electromyographic (EMG) data were collected to enable evaluation of the temporal consistency between muscle force estimates and muscle activations during walking. The EMG data were recorded using pairs of Ag/AgCl surface electrodes (Motion Laboratory Systems, Baton Rouge, LA, USA) mounted on the skin over the gluteus maximus, gluteus medius, medial and lateral vasti, hamstrings, rectus femoris, gastrocnemius and soleus. EMG data were sampled at 1080 Hz. The raw EMG signal was full-wave rectified and a Teager-Kaiser Energy (TKE) filter was then applied to the rectified EMG signal to improve the onset and offset detection [25]. Cross-talk was minimised by following published recommendations regarding the placement of surface electrodes [26].

An initial static trial was performed with the participant standing in a neutral pose and additional markers placed on the left and right medial femoral epicondyles and medial malleoli. Following the static trial, participants performed three gait trials at a self-selected speed on a 10 m level walkway. Each participant's walking speed was calculated from the kinematic data by measuring the average horizontal velocity of a marker mounted on the posterior aspect of the pelvis.

A single representative gait trial for each participant was chosen for analysis, and all analyses were performed in OpenSim [21]. An inverse kinematics problem was solved to determine the model joint angles that best matched the marker data obtained from the gait analysis experiment [27]. The net joint torques were calculated using a traditional inverse dynamics approach [28]. A static optimization problem was then solved to decompose the joint torques into individual muscle forces by minimizing the sum of the squares of the muscle activations [29, 30]. The optimization solution was constrained to the force-length-velocity surface of each muscle [30] (Figure 1C).

The lower-limb muscle forces of interest were: (1) GMAX; (2) GMED; (3) GMIN; and VASTI (vastus lateralis, intermedius and medialis combined). For each muscle group, peak force during the stance phase was identified and then normalised to the participant's body weight (BW).

Statistical analysis

All analyses were performed using the Statistical Package for the Social Sciences (PASW Statistics 18, SPSS Inc., Chicago, IL) with an alpha level of 0.05. Between-group differences in participant and clinical characteristics were assessed using Student’s t-tests or chi square tests, as appropriate. The associations between mean peak muscle forces with participant and clinical characteristics were mostly conducted using Pearson’s r correlation coefficient. For the radiographic disease severity (ordinal data), the associations were calculated with the Spearman’s rho correlation coefficients, while independent t-test were used for gender. Between-group differences in mean peak muscle forces were analysed with walking-speed as a covariate using an Analysis of Covariance (ANCOVA). The sample size (60 PFJ OA patients and 18 controls) provides >90% power to detect a between-group difference in muscle force of 10%, with a standard deviation of 10%.

RESULTS

There were no statistically significant differences for age, height or gender between the PFJ OA group (N=60) and the control group (N=18) (Table 1). Those with PFJ OA were heavier than the control individuals, with a greater BMI. In line with our eligibility criteria, the most prevalent radiographic grade (Table 2) was K/L grade 2 in the lateral PFJ and in the tibiofemoral joint.

Self-selected walking speed was not different between the PFJ OA group and the control group (mean difference [95% confidence interval]: 0.03 [-0.04 to 0.11]) (Table 1). However in the control group, walking speed was significantly correlated with VASTI (r=0.727; p=0.001) and GMAX (r=0.593; p=0.009) peak forces, but age was not statistically significantly correlated with peak muscle forces (Table 3). In the PFJ OA group, walking speed was significantly correlated with VASTI peak force (r=0.495; p<0.001), but age did not statistically significantly correlate with peak muscle forces (Table 3). There was no significant effect of gender on VASTI (0.12: [-0.11 to 0.34], GMAX (-0.03: [-0.12 to 0.06]), GMED (-0.05: [-0.18 to 0.08]) or GMIN

(-0.01: [-0.03 to 0.02]) peak muscle force. Radiographic disease severity in the tibiofemoral joint and lateral PFJ were not statistically significantly correlated with peak muscle forces in the PFJ OA group (Table 3). Additionally, no statistically significant correlations were observed between any subscale of the KOOS and peak muscle forces in the PFJ OA group (Table 3).

There were differences in the peak muscle forces for GMED and GMIN between the PFJ OA group and control group (Table 4 and Figure 2). Individuals with PFJ OA walked with lower GMED (0.15 [95% confidence interval: 0.01 to 0.29] BW); and GMIN (0.03 [0.01 to 0.06] BW) muscle forces than controls. No between-group differences were observed in VASTI or GMAX muscle force: VASTI, 0.10 [-0.11 to 0.31] BW; GMAX, 0.01 [-0.11 to 0.09] BW. Ensemble averages across the stance phase of gait for normalised muscle forces are presented in Figure 2. Model predictions of muscle forces were in temporal agreement with measured EMG activity (Supplementary Figure 1), providing a qualitative evaluation of the modelling approach used in this study.

DISCUSSION

Awareness of the importance of the PFJ in the clinical picture of knee OA is increasing due to its prevalence and contribution to knee OA symptoms. Knowledge of impairments associated with this subgroup of people with knee OA will advance our understanding of this chronic disease. We found that people with PFJ OA walk with reduced hip abductor muscle forces, compared with pain-free, aged-matched controls. Specifically, peak GMED and GMIN muscle forces were approximately 11% lower than pain-free individuals. The variability in GMAX, GMED, GMIN and VASTI peak muscle forces was not related to radiographic disease severity, knee osteoarthritis symptom severity or other participant characteristics.

To our knowledge, this is the first study to estimate lower-limb muscle forces in a cohort of people with PFJ OA. Our calculated peak VASTI muscle force was higher (1.16 [95% confidence interval 1.06 to 1.26] BW) than those reported for a single TKA patient walking with an instrumented knee (0.73 BW) [22]. The temporal patterns of lower-limb muscle forces were similar between the two studies. Since walking speed is known to influence the magnitude of lower-limb muscle forces [15], it is possible that differences in walking speed between the current study (1.37 [1.30 to 1.44] m.s^{-1} , Table 1) and that of Kim et al. [22] (1.24 (standard deviation 0.33) m.s^{-1}) partially explain some of the differences observed in the calculated values of muscle forces. Furthermore, in the study by Kim and colleagues [22], the patient had end stage OA warranting a total joint replacement, whereas our study contained individuals with no greater than K/L grade 2 radiographic OA. Thus, differences in peak VASTI force may be partially accounted for by the disparity in the patient population. It is difficult to directly compare our data with that from a previous study [31], where lower-limb muscle forces were computed for a cohort of younger individuals with PFJ pain syndrome, because an EMG-driven modelling approach was used and all muscle force data were normalised by the peak isometric force of each muscle.

Our finding of lower peak GMED and GMIN forces in those with PFJ OA is consistent with emerging evidence that hip muscle dysfunction is a dominant feature of individuals with PFJ pain syndrome [11]. The GMED and GMIN primarily contribute to hip abduction moments during walking [32]. Since the cross-sectional nature of the study design precludes knowledge of the temporal relationship between lower GMED and GMIN muscle forces and PFJ OA development or progression, further studies are required to confirm the clinical implications of our findings. Our results indicate that individuals with PFJ OA exhibit altered function that is isolated to the more proximal segments, providing further evidence for a potential link to PFJ pain syndrome.

We found no difference in peak VASTI and GMAX muscle force in those with and without PFJ OA. Our results contrast with previous studies that have measured peak isometric knee-extensor torque using a dynamometer [12, 13]; however, there is an imprecise relationship between knee-extensor torque measured in an open-kinetic-chain task and peak muscle force utilised during a functional activity such as walking. Our results may reflect variability in gait adaptations during walking in our population of people with symptomatic PFJ OA. Notably, there was a non-significant lower peak VASTI muscle force (~8%) in our PFJ OA patients, which may reflect that some individuals are likely to walk with lowered VASTI force, potentially as a pain-relieving strategy. It is also possible that deficits in the coordination (e.g. onset timing) of the medial and lateral components of the vasti may be more important than the total peak VASTI force in individuals with PFJ OA, in a similar manner to PFJ syndrome [33]. Future studies might evaluate VASTI and GMAX muscle forces in functional tasks, such as stair ambulation, which subject the PFJ to greater load, or evaluate the relative coordination of the medial and lateral vasti.

Peak muscle forces were mostly not correlated with participant, symptomatic or radiographic-specific characteristics, implying that muscle forces alone do not reflect the severity of radiographic or symptomatic disease. Although previous investigations of individuals with predominantly tibiofemoral joint OA have observed associations between radiographic OA severity and kinematics at the hip [34], these observations were only significant for those with severe radiographic OA (K/L grade 4). Similarly, many authors have noted a difference in the knee adduction moment only in those with more severe radiographic tibiofemoral disease [35]. It appears likely that changes in gait mechanics at the knee may be associated with the structural changes that accompany the OA disease process, such as altered frontal plane alignment. Since our cohort was restricted to those with a K/L grade ≤ 2, it is not surprising that radiographic disease severity was not associated with peak muscle loading during gait. Although faster walking speed was associated with a higher peak VASTI muscle force in the PFJ OA and control groups and a higher peak GMAX muscle force in the control group, walking speed was controlled for statistically and therefore the between-group differences in muscle force noted in the current study were not attributable to differences in walking speed.

It is not possible to discern the function of individual muscles from net joint torques alone, simply because a given joint torque can be satisfied by an infinite combination of muscle forces. Musculoskeletal modelling represented the only practicable method for determining lower-limb muscle forces in the current study.

However, there are several limitations and assumptions inherent in this modelling approach; for example, the physiological properties prescribed for the muscle-tendon actuators included in the model (e.g., peak isometric muscle force and the corresponding muscle-fiber length and tendon rest length; see Figure 1B). Importantly, the present study assumed the same unscaled muscle-tendon parameters across both the PFJ OA and control population and hence, any relative differences in muscle force predictions are attributable mostly to differences in the experimental gait data and not the parameters assumed in the model. We also elected to analyse synergistic groups of muscles (i.e., GMAX, GMED, GMIN, VASTI) and did not attempt to partition calculated forces onto the various components of these muscle groups (e.g., vastus medialis vs intermedius vs lateralis within VASTI). Several studies have shown that our approach of obtaining muscle force estimates for synergistic groups of muscles is relatively insensitive to changes in the values assumed for peak isometric muscle force (or physiological cross-sectional area) [36-38]. Despite the aforementioned limitations, the inverse-dynamics-based optimisation approach employed in the current study is robust, computationally efficient, and has been used extensively to estimate lower-limb muscle forces in walking [15, 39, 40]. Furthermore, indirect evidence is available to support the validity of predicting lower-limb muscle forces during walking using the approach taken in the present paper [22]. Lastly, previous studies have shown temporal agreement between predicted lower-limb muscle forces and recorded EMG [41], and this relationship was also demonstrated in the present study for a representative subject (Supplementary Figure 1).

A final limitation relates to the participant characteristics of the control group. Although we attempted to recruit participants who were matched on variables likely to influence muscle forces, the control group was lighter and trending towards being younger. We controlled for body weight by normalising all muscle force data, and age was not associated with the muscle force data. In order to be included in the control group, participants had to exhibit a K/L grade ≤ 1. While this is usually accepted as a criterion for no OA, it is possible that participants in the control group had some early/mild OA that may have affected their gait pattern. It is also possible that some participants in either group may have had coexisting hip OA. However, the control group were required to have no knee pain and all participants were required to report no hip/groin or lower-back symptoms. The sample size for the control group was chosen to be as large as could be practically achieved within the time and resource constraints, and consequently the control group included much fewer participants (N=18) than our PFJ OA cohort (N=60). This difference reflected the difficulties in recruiting an older population from the general community with no knee or other lower-limb complaints, who were physically active with no radiographic knee OA, and who had the time and inclination to attend for both radiographic and biomechanical evaluation. Nevertheless, our sample size calculations revealed that we had sufficient power, despite the discrepant sample sizes.

This study is the first to investigate the walking mechanics of individuals with predominant PFJ OA. Our findings indicate that individuals with PFJ OA ambulate with lower peak hip abductor muscle force than their healthy counterparts. It is not known whether a lower hip abductor muscle force contributes to, or is a consequence of, the PFJ OA disease process.

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Author contributions

Kay M Crossley obtained funding, designed and managed the project, assisted with statistical analyses and writing of the paper

Tim W Dorn and Josien van den Noort performed the muscle force calculations

Hannah Ozturk assisted with participant recruitment and collected the gait data

Anthony G Schache obtained funding and assisted with the data collection, analyses and writing of the paper

Marcus G Pandy obtained funding and assisted with the calculations of muscle force and writing of the paper

All authors contributed to the editing and approval of this work

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Conflict of interest

KMC, TWD, HO, JVDN, AGS MGP and do not have any conflicts of interest to declare

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Table 1: Participant and clinical characteristics: patellofemoral osteoarthritis and control groups

	Pain-free Control † N = 18	Patellofemoral osteoarthritis † N = 60	Mean difference [95% CI]	p value
Age (yr)	53 (7)	58 (10)	4 [-0.8 to 10]	0.096
Height (m)	1.65 (0.08)	1.69 (0.09)	0.03 [-0.06 to 0.08]	0.186
Weight (kg)	66 (12)	78 (13)	12 [5 to 19]	0.001*
BMI ($kg.m^{-2}$)	24.1 (3.4)	27.5 (3.7)	3.3 [1.4 to 5.3]	0.001*
Gender (n(%))	14 female (78%)	39 female (65%)	-	‡0.236
KOOS-pain	-	63 (15)	-	-
KOOS-symptoms	-	61 (16)	-	-
KOOS-ADL	-	70 (16)	-	-
KOOS-Sport/Rec	-	41 (22)	-	-
KOOS-QoL	-	12 (16)	-	-
Walking speed ($m.s^{-1}$)	1.34 (0.13)	1.37 (0.17)	0.03 [-0.04 to 0.11]	0.369

† all values are mean (SD) unless indicated; CI = confidence interval; ‡ $\chi2$; * Denotes statistically significant, $p < 0.05$

BMI = body mass index

KOOS = Knee Injury and Osteoarthritis Outcome Score [20] (100 =no symptoms - 0 = maximum symptoms)

KOOS-pain = pain subscale of the KOOS

KOOS-symptoms =symptoms subscale of the KOOS

KOOS-ADL = activities of daily living subscale of the KOOS

KOOS-Sport/Rec =Sport and recreation subscale of the KOOS

KOOS-QoL =Knee-related quality of life subscale of the KOOS

Table 2: Radiographic disease severity for the patellofemoral osteoarthritis group (n = 60)

	Grade 0 n (%)	Grade 1 n (%)	Grade 2 n (%)	Grade 3 n (%)	Grade 4 n (%)
Tibiofemoral (K/L)	14 (23%)	18 (30%)	28 (47%)	0	0
Lateral patellofemoral (K/L)†	0	0	39 (65%)	11 (18%)	10 (17%)

K/L Kellgren and Lawrence scale [17]: 0 = no osteoarthritis ; 4 = severe osteoarthritis

K/L† Kellgren and Lawrence scale adapted for patellofemoral joint [18]

Table 3: Univariate associations between normalised peak muscle forces, participant and clinical characteristics

		GMAX	GMED	GMIN	VASTI
Control group (n=18)					
Age (yr)	r	-0.195	-0.96	-0.84	106
	p	0.439	0.705	0.739	0.676
Walking speed ($m.s^{-1}$)	r	0.593	0.076	0.047	0.727
	p	0.009*	0.764	0.853	0.001*
PFJ OA group (n=60)					
Age (yr)	R	-0.86	-0.138	-0.125	-0.174
	P	0.514	0.295	0.340	0.185
KOOS-pain	R	0.081	0.050	0.023	0.103
	p	0.539	0.704	0.859	0.435
KOOS-symptoms	r	-0.026	-0.029	-0.041	0.069
	p	0.843	0.826	0.754	0.598
KOOS-ADL	r	-0.045	-0.053	-0.062	0.133
	p	0.734	0.685	0.637	0.311
KOOS-Sport/Rec	r	-0.018	-0.050	-0.056	0.261
	p	0.892	0.702	0.669	0.097
KOOS-QoL	r	-0.059	-0.036	-0.029	0.111
	p	0.652	0.784	0.824	0.396
Walking speed ($m.s^{-1}$)	r	0.145	0.090	0.040	0.495
	p	0.267	0.494	0.763	<0.001*
Tibiofemoral (K/L)‡	rho	-0.040	0.114	0.108	0.058
	p	0.763	0.385	0.413	0.660
Lateral patellofemoral (K/L) ‡†	rho	-0.084	0.104	0.060	-0.072
	p	0.524	0.430	0.649	0.586

Correlations using Pearson's r correlation co-efficient unless indicated

‡Correlations using Spearman’s rho correlation co-efficient * Denotes statistically significant, $p < 0.05$

GMAX = gluteus maximus

GMED = gluteus medius

GMIN= gluteus minimus

VASTI = vastus medialis, vastus lateralis and vastus intermedialis

KOOS = Knee Injury and Osteoarthritis Outcome Score [20] (100=no symptoms – 0=maximum symptoms)

KOOS-pain = pain subscale of the KOOS

KOOS-symptoms =symptoms subscale of the KOOS

KOOS-ADL = activities of daily living subscale of the KOOS

KOOS-Sport/Rec =Sport and recreation subscale of the KOOS

KOOS-QoL =Knee-related quality of life subscale of the KOOS

K/L Kellgren and Lawrence scale [17]: 0 = no osteoarthritis ; 4 = severe osteoarthritis

K/L† Kellgren and Lawrence scale adapted for patellofemoral joint [18]

Table 4: Between group comparisons of normalised peak muscle forces between symptomatic PFJ OA and control groups

	Pain-free Control † N = 18	Patellofemoral osteoarthritis † N = 60	Mean difference †	P
GMAX (BW)	0.69 [0.61 to 0.78]	0.70 [0.66 to 0.75]	0.01 [-0.11 to 0.09]	0.796
GMED (BW)	1.41 [0.28 to 1.53]	1.26 [1.19 to 1.33]	0.15 [0.01 to 0.29]	0.041*
GMIN (BW)	0.24 [0.22 to 0.26]	0.21 [0.20 to 0.22]	0.03 [0.01 to 0.06]	0.013*
VASTI (BW)	1.26 [1.08 to 1.44]	1.16 [1.06 to 1.26]	0.10 [-0.11 to 0.31]	0.355

† all values are mean *95% confidence interval+ and adjusted for walking speed; * Denotes statistically significant, $p < 0.05$

BW = body weight

GMAX = gluteus maximus

GMED = gluteus medius

GMIN= gluteus minimus

VASTI = vastus medialis, vastus lateralis and vastus intermedialis