Author/s:
Sritharan, P;Lin, YC;Richardson, SE;Crossley, KM;Birmingham, TB;Pandy, MG

Title:
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Date:
2018-08-01

Citation:
Sritharan, P., Lin, Y. C., Richardson, S. E., Crossley, K. M., Birmingham, T. B. & Pandy, M. G. (2018). Lower-limb muscle function during gait in varus mal-aligned osteoarthritis patients. Journal of Orthopaedic Research, 36 (8), pp.2157-2166. https://doi.org/10.1002/jor.23883.

Persistent Link:
https://hdl.handle.net/11343/283628

Research Article (Member)

LOWER-LIMB MUSCLE FUNCTION DURING GAIT IN VARUS MAL-ALIGNED OSTEOARTHRITIS PATIENTS[1]

Prasanna Sritharan 0000-0001-9543-4108[1,2], Yi-Chung Lin[2], Sara E. Richardson[3], Kay M. Crossley[1], Trevor B. Birmingham[3], Marcus G. Pandy[2]

[1]School of Allied Health, La Trobe University, Victoria, AUSTRALIA

[2]Dept of Mechanical Engineering, University of Melbourne, Victoria, AUSTRALIA

[3]Faculty of Health Sciences, University of Western Ontario, Ontario, CANADA

REVISION

Submitted as an original article to *Journal of Orthopaedic Research*

17 January 2018

Word count: 3717

Address for correspondence:

Prasanna Sritharan

La Trobe Sports and Exercise Medicine Research Centre

La Trobe University, Bundoora, Victoria 3086, AUSTRALIA

Phone: +613 9479 5871

Email: P.Sritharan@latrobe.edu.au

Running title. Muscle function in OA gait

[1] This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi:10.1002/jor.23883

Author contributions statement. All authors contributed equally to this work. Subject recruitment, experimental design and undertaking of gait experiments were performed by SER and TBB. The computational modelling pipeline was developed and implemented by PS with design input and technical guidance from YCL and MGP. Statistical analyses were designed and supervised by KMC and TBB, and undertaken by PS. All authors discussed and agreed on results and potential implications. The manuscript was initially prepared by PS, YCL and MGP, but all authors contributed substantially to its revision and final form.

Conflict of interest statement. All authors declare no conflict of interest.

ABSTRACT

This study quantified the contributions by muscular, gravitational and inertial forces to the ground reaction force (GRF) and external knee adduction moment (EKAM) for knee osteoarthritis (OA) patients and controls walking at similar speeds. Gait data for 39 varus mal-aligned medial knee OA patients and 15 controls were input into musculoskeletal models to calculate the contributions of individual muscles and gravity to the fore-aft (progression), vertical (support), and mediolateral (balance) GRF, and the EKAM. The temporal patterns of contributions to GRF and EKAM were similar between the groups. Magnitude differences in GRF contributions were small but some reached significance. Peak GRF contributions were lower in patients except hamstrings in early-stance progression (P<0.001) and gastrocnemius in late-stance progression (P<0.001). Both EKAM peaks were higher in patients, due mainly to greater adduction contribution from gravity (P<0.001) at the first peak, and lower abduction contributions from soleus (P<0.001) and gastrocnemius (P<0.001) at the second peak. Gluteus medius contributed most to EKAM in both groups, but was higher in patients during mid-stance only (P<0.001). Differences in GRF contributions were attributed to altered quadriceps-hamstrings action as well as compensatory adaptation of the ankle plantarflexors to reduced gluteus medius action. The large effect of varus mal-alignment on the frontal-plane moment arms of the gravity, soleus and gastrocnemius GRF contributions about the knee explained greater patient EKAM. Our results shed further light on how the EKAM contributes to altered knee-joint loads in OA and why some interventions may affect different portions of the EKAM waveform.

Keywords: muscle contributions, knee adduction, medial compartment, musculoskeletal modelling, gait

INTRODUCTION

Non-pharmacologic interventions are the cornerstone in the management of medial knee osteoarthritis[1] (OA) with many focussed on offloading the medial tibiofemoral compartment during gait by reducing the external knee adduction moment[2] (EKAM). The observed associations between high EKAM and medial knee OA progression provide the rationale for this treatment approach[3].

The EKAM is produced by the ground reaction force (GRF) acting about the knee in the frontal-plane. The GRF arises from the superposition of contributions from all muscles, with smaller contributions from gravitational (i.e. the weight force directly borne by the skeleton[4]) and inertial forces[4,5], acting by means of dynamic coupling[6]. A muscle's contribution to the fore-aft, vertical and mediolateral components of the GRF represents its contribution to the progression, support and balance of the body's centre-of-mass (COM), and is one means of describing that muscle's function in gait[4,5,7-9]. By decomposing the GRF into its contributions, recent studies have identified five prime movers which progress, support and balance the body during healthy gait – the vasti, gluteus medius, gluteus maximus, soleus and gastrocnemius[4,5,9]. However, muscle function in OA gait has yet to be quantified in this way. In particular, the effects on lower-limb muscle function during gait of neuromuscular changes due to OA, such as elevated knee-spanning muscle co-activations[10,11] and impairment of some hip[12], knee[13] and ankle[14] muscles, are not well understood.

By virtue of their contributions to the GRF, the muscular, gravitational and inertial forces also contribute to EKAM[5,15]. Of the prime movers in healthy gait, gluteus medius and gluteus maximus provide the largest EKAM contributions, while vasti, soleus and gastrocnemius modulate this quantity by providing substantial external *abduction* moment contributions[15].

Many interventions rely on reducing EKAM by independently manipulating the GRF and its frontal-plane moment arm about the knee[2]. One principal factor influencing EKAM is varus mal-alignment, a common feature of medial knee OA[16] associated with greater frontal-plane moment arm[17] and hence higher EKAM[18,19]. How mal-alignment influences the individual contributors to EKAM is unknown, and may be essential to the development of novel interventions that reduce EKAM, including efforts to restore normal muscle function in gait.

Therefore, the goals of this study were to investigate the individual muscular, gravitational and inertial force contributions to the GRF and the EKAM in varus mal-aligned OA patients, and to compare the results against those of healthy individuals walking at similar speeds.

METHODS

Design: Case-control study

Level of evidence: III

Recruitment and gait experiments. Previously-reported gait data for 39 medial knee OA patients and 15 healthy controls were used for this study[20] (Table 1). All gait experiments were performed at the Wolf Orthopaedic Biomechanics Laboratory, Fowler Kennedy Sport Medicine Clinic, University of Western Ontario. The study was approved by the Research Ethics Board for Health Sciences Research Involving Human Subjects of the University of Western Ontario (HSREB No. 09812E). Recruitment methodology, inclusion and exclusion criteria, radiographic assessment, patient-reported measures and experimental protocols are described in detail in Birmingham et al.[21] and are only briefly reproduced here.

Patients were recruited from a tertiary care centre specialising in orthopaedics and were referred to the centre for treatment of symptomatic radiographic knee OA. Inclusion criteria for patients were based on the American College of Rheumatology criteria[22] and included varus mal-alignment of the lower-limb and diagnosis of medial knee OA. The Knee Injury and Osteoarthritis Outcome Score (KOOS) was used to assess symptoms[23]. Full-limb standing anteroposterior radiographs were used to quantify static frontal-plane lower-limb alignment (mechanical axis angle[24]) and the severity of OA (Kellgren-Lawrence grade, K-L grade[25]). Healthy controls were recruited from the community, had no history of knee pain and were matched for sex, age, body mass index (BMI) and walking speed.

Each participant walked barefoot at his or her preferred speed over level ground while joint motion and GRFs were simultaneously recorded. An eight-camera motion capture system (Eagle EvaRT, Motion Analysis Corp., Santa Rosa, CA) sampling at 60 Hz was used to measure the three-dimensional spatial trajectories of 22 retro-reflective markers attached to the participant, with the locations of markers on the body determined using a modified Helen Hayes protocol[26]. A single strain-gauged force plate (Advanced Mechanical Technology Inc., Watertown, MA) sampling at 1200 Hz was used to measure the three components of the GRF as well as the centre-of-pressure. Five successful gait trials were recorded for the stance phase of gait.

Musculoskeletal modelling. A modelling pipeline[20] for calculating joint angles, joint torques and muscle forces was implemented using Matlab (The Mathworks Inc., Natick, MA) and OpenSim[27], an open-source musculoskeletal modelling package. For each subject, a scaled-generic whole-body three-dimensional musculoskeletal model was created, with segmental inertial properties, muscle-tendon attachment sites, and muscle-tendon paths scaled based on segmental dimensions calculated from each subject's static trial. Each model consisted of 10 rigid segments, 27 degrees of freedom and 92 Hill-type muscle-tendon units. A ball-and-socket was used to model each hip and knee, while a hinge was used to model each ankle and subtalar joint. The head, arms and torso were combined into a single rigid body that articulated with the pelvis by means of a ball-and-socket. Cartilage, ligaments and other soft tissues were not included in the model.

For each recorded trial, joint kinematics and muscle forces were calculated for the stance phase of gait. An inverse kinematics analysis was used to calculate joint angles from the experimental marker trajectories[28]. At each time step, the joint kinematics and the measured GRF were input into the model and inverse dynamics used to calculate the net internal joint torques. Muscle forces were subsequently calculated from joint torques using a static optimisation approach in which the sum-of-squares of muscle activations was minimised subject to constraints on the muscle forces imposed by each muscle's force-length-velocity property[29]. The internal knee adduction and internal-external rotation torques were not included in the static optimisation analysis. Time histories of joint angles and muscle forces calculated for these participant groups using the aforementioned procedures were previously reported[20], with peak values provided in the present study as Supplementary Material.

Contributions to GRF and EKAM. The three components of the GRF were defined as progression (fore-aft), support (vertical) and balance (mediolateral)[8,9]. At each time step, the GRF vector was decomposed into contributions by individual muscles, gravity and inertia using a pseudoinverse-based approach[9]. Briefly, five foot-ground interaction points were defined on the foot segment of the model to represent the distributed force experienced on the foot when in contact with the ground. Each point could be fully-constrained, partially-constrained or unconstrained in space by applying a weight between 0 (unconstrained) and 1 (fully-constrained) depending on the phase of foot-ground contact and the location of the foot-ground centre-of-pressure. At each time step during stance, the calculated joint angles were applied to the model. Each individual muscle force was then applied in isolation, and a weighted least-squares optimisation problem solved to calculate the induced three-dimensional force vector at each of the fully- or partially-constrained foot-ground interaction points. The force at each of the unconstrained points was zero by default. Individual muscle contributions to EKAM were calculated by applying each muscle's contribution to the GRF[15] and solving for the torques at the knee using inverse dynamics. This procedure was repeated for gravity and inertial forces.

Data analyses. Independent-samples *t*-tests were performed to compare participant characteristics and to determine group differences between the right leg of controls and the symptomatic limb of patients at the peak values of the contributions by muscles, gravity and inertia to the GRF components and also to the EKAM. The significance level was set *a priori* at $\alpha = 0.05$. All statistical analyses were performed using SPSS Statistics 22.0 (IBM Corp., Armonk, NY).

RESULTS

The temporal patterns of the mean measured GRFs were similar for both groups (Figure 1). Although the magnitudes of peak GRF components were only slightly lower in patients compared to controls, some differences reached significance (Figures 1 and 2); specifically, at both peaks of the vertical GRF (first: $P<0.001$, mean difference (MD): -0.038 BW; second: $P<0.001$, MD: -0.060 BW) (Figure 2), the first peak of the fore-aft GRF ($P<0.001$, MD: 0.015 BW) and the second peak of the mediolateral GRF ($P<0.001$, MD: 0.011 BW).

The temporal patterns of the individual contributions by muscles and gravity to support, progression and balance were similar between the groups (Figure 1). Inertial contributions were negligible and are not considered further. Differences in the magnitudes of peak contributions to each component of the GRF differed between patients and controls and were generally small (Figure 2), but some differences reached significance as described below.

During early-stance progression, the magnitudes of peak contributions were greater for patients' hamstrings ($P<0.001$, MD: 0.009 BW) but smaller for vasti ($P<0.001$, MD: 0.026 BW). In late-stance progression, peak contributions were smaller for patients' gluteus medius ($P<0.001$, MD: -0.024 BW), but larger for patients' gastrocnemius ($P<0.001$, MD:

0.011 BW).

The magnitudes of peak contributions to early-stance support were smaller for patients’ gluteus medius (*P*<0.001, MD: -0.055 BW), gluteus maximus (*P*=0.003, MD: -0.021 BW) and vasti (*P*<0.001, MD: -0.049 BW). In late-stance support, peak contributions were smaller for gluteus medius (*P*<0.001, MD: -0.024 BW) and soleus (*P*<0.001, MD: -0.060 BW). Although patients’ gastrocnemius contributed more than controls in late-stance support, this difference did not reach significance (*P*=0.253, MD: 0.019 BW).

Peak contributions from gravity to early-stance balance were smaller for patients (*P*=0.009, MD: -0.002 BW). In late-stance balance, the peak magnitudes of patients’ gluteus medius (*P*<0.001, MD: 0.025 BW) and soleus (*P*<0.001, MD: -0.012 BW) contributions were lower while gravity contributions (*P*<0.001, MD: -0.003 BW) were higher.

Both peaks of the EKAM were higher in patients than controls (first: *P*<0.001, MD: 0.55 %BW*HT; second: *P*<0.001, MD: 1.63 %BW*HT). The difference in magnitude between controls and patients at the second peak was almost 3 times that at the first peak (Figure 3). The temporal patterns of muscle and gravity contributions to EKAM were qualitatively similar for patients and controls (Figure 3); however, the magnitudes of contributions to EKAM differed between the groups (Figure 3), with significant differences described below.

Gluteus medius was the largest contributor to EKAM throughout stance in both groups, and lower in patients at the second peak (*P*<0.001, MD: -0.43 %BW*HT). Patients’ soleus (*P*<0.001, MD: 0.80 %BW*HT) and gastrocnemius (*P*<0.001, MD: 0.65 %BW*HT) contributed less *abduction* moment in late stance. Patients’ vasti contributed less *abduction* moment in early-stance (*P*<0.001, MD: 0.23 %BW*HT) but more adduction moment in late-stance (*P*<0.001, MD: 0.06 %BW*HT). Patients’ gravity contributions were higher throughout stance (first peak: *P*<0.001, MD: 0.37 %BW*HT; second peak: *P*<0.001, MD:

0.15 %BW*HT).

Mid-stance EKAM was higher in patients (*P*<0.001, MD: 1.40 %BW*HT), due mainly to a greater contribution by gluteus medius (*P*<0.001, MD: 0.23 %BW*HT) and gravity (*P*<0.001, MD: 0.23 % BW*HT) and smaller *abduction* contributions by soleus (*P*<0.001, MD: 0.27 %BW*HT) and gastrocnemius (*P*<0.001, MD: 0.49 %BW*HT).

Significant differences were found between the groups for the moment arms of muscle and gravity contributions to EKAM (Table 2). Moment arms of contributors which provided an adduction moment were typically larger in patients, while those that provided an *abduction* moment were typically smaller. The largest percentage differences were found for gravity at the first peak, and soleus and gastrocnemius at the second peak.

DISCUSSION

This study quantified and compared individual contributions by muscles, gravity and inertia to the GRF and EKAM in healthy individuals and medial knee OA patients walking at similar speeds. We found that: (1) overall, differences in the magnitudes and patterns of GRF contributions between the groups were small; (2) peak GRF contributions by muscles were typically lower in patients, except in early-stance and late-stance progression, where the magnitudes of patients' hamstrings and gastrocnemius contributions were respectively higher; and (3) a higher first peak of EKAM in patients was due mainly to an increase in gravity's contribution, while a higher second peak was due mainly to a decrease in contributions from the soleus and gastrocnemius.

Although patients exhibited only slightly lower peak contributions to GRF compared to controls, two exceptions were evident in progression where the magnitudes of patients' early-stance hamstrings contribution and late-stance gastrocnemius contribution exceeded those of controls. The latter finding of greater gastrocnemius contribution in late-stance progression in patients, together with a soleus contribution that is comparable to controls

(Figure 2, top right), may indicate a potential compensatory mechanism for impaired action of the gluteus medius in accelerating the body COM forward. Correa et al.[30] found that children with cerebral palsy who walked with crouch or jump gait demonstrated an improved potential for the ankle plantarflexor muscles to accelerate the body COM forward. These children also exhibited impaired gluteus medius function during gait. Thus a similar, albeit much more subtle, compensatory mechanism may occur in our OA patient cohort to facilitate functional improvements during gait, enabling them to walk at speeds comparable to controls despite advanced disease and symptoms of OA. In fact soleus and gastrocnemius muscle forces were lower in patients (Supplementary Material and Sritharan et al.[20]) suggesting that kinematic adaptations in OA gait, currently understood to mitigate pain and improve knee-joint stability, may also confer small but important advantages for lower-limb muscle function. Further longitudinal research would help to determine if such functional adaptations do indeed develop with OA progression, and continue to change as these patients age and become less mobile.

Our results suggest that relative differences in quadriceps-hamstrings function may also impact the function of other major muscles in early-stance progression. The elevated contribution by OA patients' hamstrings in early-stance progression would amplify forward acceleration of the body in early stance, which is undesirable as this is the braking phase of walking gait (Figure 1, left). Decelerating the body COM is an essential function of the vasti during this time period[5,31]. Yet because the vasti contribution in early-stance progression was diminished in patients (Figure 1, left), a subtle rebalancing of coordination was required across the other muscles of the lower-limb as well as slightly increased loading through the skeletal linkage to produce the necessary overall braking force (Figure 2, top left). Our present study shows that, although the hamstrings is not considered a key muscle involved in fore-aft acceleration of the body COM during stance[5,31], changes in hamstrings function can impact the coordination of the other prime movers. The combination of reduced vasti contribution and increased hamstrings contributions may be related to elevated knee-spanning muscle co-activity, which is a frequently-reported feature of medial knee OA patients[11], including the present cohort[20].

Overall however, our finding of only modest differences in muscle function between patients and controls at similar walking speeds is not surprising, and can be inferred to some extent from the similar temporal patterns and only slightly lower peaks of the measured GRF (Figure 1). Large qualitative and quantitative differences in the measured GRF between the groups would have hinted at substantial differences in muscle function, but this was not the case. Muscle function in our relatively young patient group (mean age: 49 years) demonstrated similarities with gait in healthy elderly individuals, where only modest differences in muscle function were found despite differences in joint kinematics and some muscle forces[7]. Yet sizable alterations in muscular coordination strategy do occur in severe cases of neuromuscular dysfunction, such as cerebral palsy[32]. Thus, we suggest two possible reasons for our findings: (1) that level walking is not a sufficiently focussed or demanding task to reveal all differences in muscle function in OA patients compared to controls; and/or (2) that OA does not produce large-scale changes in muscle function at this point in the disease process, rather it results in more subtle changes that may have deleterious effects over repeated cycles. If so, these rather subtle changes may be amenable to non-surgical intervention.

The temporal patterns of contributions to EKAM in controls and patients were similar to that reported previously for healthy individuals[15]. Similar to a study by Kutzner et al.[33] using subjects with instrumented total knee replacements, the second peak of EKAM showed a larger variation than the first peak (Figure 4). This was also reflected in greater variation of the major muscle contributions to the second peak of EKAM. The gluteus medius, a key muscle for frontal-plane balance during walking[5,9], was the muscle that contributed most to EKAM in both groups. However, it was higher in patients during mid-stance only, demonstrating no difference at the first peak of EKAM and a lower contribution in patients at the second peak. The higher first peak of EKAM in patients was mostly due to a sustained contribution by gravity throughout the first half of stance, whereas gravity contributed almost nothing during this period in controls (Figure 3). In contrast, the higher second peak of EKAM in OA patients relative to controls was largely due to a marked reduction in peak external *abduction* moment contribution from the soleus and gastrocnemius during the second half of stance. These observations can be explained to a large extent by examination of the moment arm of the GRF about the knee.

A muscle's/gravity's contribution to EKAM can be described as the product of that muscle's/gravity's GRF contribution vector and its frontal-plane moment arm about the knee[15]. In patients, the moment arms for gravity at the first peak, and for soleus and gastrocnemius at the second peak, showed the largest differences relative to controls (Table 2). Such dramatic differences in these moment arms were unlikely to be solely produced by the small differences in the vertical and mediolateral GRF vector components found for these contributors (Figure 2). In this way, structural differences in the lower-limb, specifically, greater varus mal-alignment in patients (Table 1), were more instrumental in elevating patients' EKAM than differences in muscle function. As *gravity* refers to the loads transmitted directly through the skeletal linkage[4], only the frontal-plane configuration of the lower-limb could have impacted gravity's contribution to EKAM. This implicates knee varus as a direct cause of the greater first peak of EKAM by shifting the knee away from gravity's GRF contribution vector (Figure 5). At the second peak of EKAM in patients, greater knee varus likely shifted the knee closer to the ankle plantarflexors' GRF contribution vectors (Figure 5), decreasing their moment arms about the knee (Table 2). This strongly impaired the ability of the soleus and gastrocnemius to resist the large adduction moment provided by the gluteus medius. In contrast, the moment arm of the gluteus medius contribution showed small differences between patients and controls compared to other muscles and gravity (Table 2), suggesting it was less impacted by structural differences of the lower-limb in the frontal-plane. Thus, while the association between mal-alignment and EKAM in patients has been well-studied[34], our present study demonstrates that the underlying effects of mal-alignment may be complex, affecting some contributors more than others.

Our findings may explain why some non-surgical interventions for medial knee OA may more successfully reduce the second peak of EKAM than the first[2]. In our present study, if patients' EKAM was "corrected" to the level of our healthy controls (Figure 3), patients would undergo a reduction of 0.5 %BW*HT at the first peak of EKAM due mainly to a reduction in the contribution by gravity; but at the second peak, a three-fold reduction of 1.5 %BW*HT would occur due mainly to a sizable increase in the *abduction* contributions by the soleus and gastrocnemius (Figure 4). Whether the difference between patients and controls at the second peak of the medial compartment force is also three-times that of the difference at the first peak is less clear. Modelling studies suggest that the difference in the first peak of medial compartment force between OA patients and controls may in fact be of comparable magnitude to the difference at the second peak[20,35].

This study is not without limitations. Firstly, to decompose the GRF, a simplified foot-ground interaction model with five discrete contact points was used[9] while in reality the pressure distribution is continuous and variable across the surface of the foot. However, by allowing the foot-ground contact points to be fully-constrained, partially-constrained or unconstrained depending on the phase of gait, our models provide a substantially more realistic representation of the foot-ground interaction[36] than single-point models[37,38], and produce results consistent with foot-ground models of up to 30 contact points[39]. Thus, we believe our approach yields suitable estimates of muscle function during gait. Secondly, while our static optimisation-based muscle forces used to calculate contributions to GRF did indeed previously demonstrate qualitative agreement with recorded EMG, as well as the muscle force deficits and elevated co-contraction expected for OA gait[20], the magnitude of these effects may be under-represented based on lower-limb muscle activations and co-contraction levels reported in EMG studies for various levels of OA severity[11]. In particular, elevated quadriceps-hamstrings co-activation levels may be important for stabilising the osteoarthritic knee-joint in the frontal-plane, however we did not include knee-spanning muscle EMG activity or frontal-plane knee-joint torques in the calculation of muscle forces. The knee adduction angle was, however, included. Whilst we are confident that our study has identified the key differences in muscle function and contributions to EKAM between OA patients and controls, our present study may understate the magnitude of these differences. Although static optimisation is a robust and reliable method for calculating muscle forces, future studies using more sophisticated dynamic optimisation methods such as direct collocation[40] may potentially identify larger differences than those reported in our present work. Other functional and morphological changes associated with advanced medial knee OA, such as muscle atrophy and activation failure[41], as well as pain[42], were not included in our analyses as they are difficult to quantify and implement. The inclusion of these characteristics in musculoskeletal models should be an important objective for future studies of OA gait, as they may have substantial impact on muscular coordination during dynamic tasks.

In conclusion, whilst muscle function in OA gait was only subtly different from healthy gait, small but important magnitude differences were observed. To compensate for deficits in specific lower-limb muscle contributions to GRF, a subtle rebalancing of GRF contributions across the other muscles and skeletal linkage was necessary. Varus mal-alignment played a substantial role in producing greater EKAM by changing the moment arms of contributors' GRF vectors about the knee, however only three contributors were markedly impacted – gravity, soleus and gastrocnemius. These findings provide a foundation for further study of gait changes associated with knee OA and the broader systemic impacts of interventions on gait mechanics, especially the effects of gait modifications.

ACKNOWLEDGEMENTS

This work was supported in part by grants from the Australian Research Council (DP120101973 and DP160104366) and an Innovation Fellowship from the Victorian Endowment for Science, Knowledge and Innovation to MGP, as well as grants from the Canadian Institutes of Health Research, The Arthritis Society of Canada, and the Canada Research Chairs Program to TBB.

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FIGURE LEGENDS

Figure 1. Mean contributions by the major muscles and gravity to progression (left column), support (centre column) and balance (right column) in controls (solid lines) and OA patients (dashed lines). Black lines represent the mean GRF components measured from the force plate. "Other" refers to the summed contributions from all remaining muscle forces and inertial forces. Positive values for progression, support and balance represent forces directed forwards, upwards and laterally, respectively.

Figure 2. Means and standard deviations of the contributions by major muscle groups and gravity at each of their peak values during early stance (left column) and late stance (right column) in controls and patients, for progression (top row), support (middle row) and balance (bottom row). Asterisks (*) indicate statistically significant differences (P<0.05). Abbreviations: GMAX, gluteus maximus; GMED, gluteus medius; HAMS, hamstrings; VAS, vasti; SOL, soleus; GAS, gastrocnemius; GRAV, gravity; OTHER, summed contributions from all remaining muscle forces and inertial forces; TOTAL, summed contributions of muscle, gravitational and inertial forces to the peak fore-aft (progression), vertical (support) and mediolateral (balance) components of the ground reaction force during early- and late-stance. Positive values for progression, support and balance represent forces directed forwards, upwards and laterally, respectively.

Figure 3. Contributions by major muscle groups and gravity to the external knee adduction moment in controls (solid lines) and OA patients (dashed lines). Positive values represent adduction, while negative values represent abduction. "Other" refers to the summed contributions from all remaining muscle forces and inertial forces. Black lines represent the sum of these major muscle groups, gravity and other contributors.

Figure 4. Means and standard deviations of the contributions by major muscle groups and gravity at the first peak (left), mid-stance (centre) and second peak (right) of the external knee adduction moment. Asterisks (*) indicate statistically significant differences (P<0.05). Abbreviations: GMAX, gluteus maximus; GMED, gluteus medius; HAMS, hamstrings; VAS, vasti; SOL, soleus; GAS, gastrocnemius; GRAV, gravity; OTHER, summed contributions from all remaining muscle forces and inertial forces; TOTAL, sum of the major muscle groups, gravity and other forces. Positive values represent adduction, while negative values represent abduction.

Figure 5. Schematic diagram illustrating how frontal-plane mal-alignment in the lower-limb may influence the contributions from gravity and the ankle plantarflexors (i.e. soleus and gastrocnemius) to the first and second peaks of the external knee adduction moment (EKAM), respectively. At the time instant corresponding to the first peak of EKAM (left panel), the frontal-plane vector of gravity's GRF contribution points almost vertically upwards (vector sum of gravity's support and balance contributions from Figure 1). Varus mal-alignment in the patient's leg shifts the knee-joint centre away from gravity's GRF contribution vector in the frontal plane, increasing its moment arm about the knee, thus increasing its contribution to EKAM (compare red and blue arrows). At the time instant corresponding to the second peak of EKAM (right panel), the ankle plantarflexors' GRF contribution vector points vertically and laterally (vector sum of the soleus and gastrocnemius contributions to support and balance from Figure 1). Varus mal-alignment in the patient's leg shifts the knee-joint centre towards the ankle plantarflexors' GRF contribution vector in the frontal plane, decreasing its *abduction* moment arm about the knee, thus decreasing its external knee *abduction* moment contribution (compare red and blue arrows).

Table 1. Participant characteristics of the OA patient and healthy control groups.

	Control n = 15	OA n = 39	*P*
Age, yrs	49 (7)	49 (7)	0.869
BMI, kg/m^2	26.5 (4.8)	28.2 (3.6)	0.177
Mass, kg	77 (15)	89 (17)	***0.015***
Height, m	1.70 (0.10)	1.78 (0.09)	***0.006***
Females, no. (% of total)	4 (27%)	8 (21%)	0.634
Walking speed, m/s	1.21 (0.07)	1.20 (0.10)	0.575
KOOS subscale scores, 0–100			
Pain	-	55 (18)	-
Symptoms	-	54 (18)	-
Activities of daily living	-	65 (20)	-
Sport and recreation	-	31 (23)	-
Knee-related quality of life	-	29 (19)	-
K-L grade 0/1/2/3/4, no. of patients	-	0/1/10/15/13	
Mechanical axis angle, deg	-	9 (3)	
Static frontal-plane alignment, deg	6 (6)	13 (5)	***< 0.001***

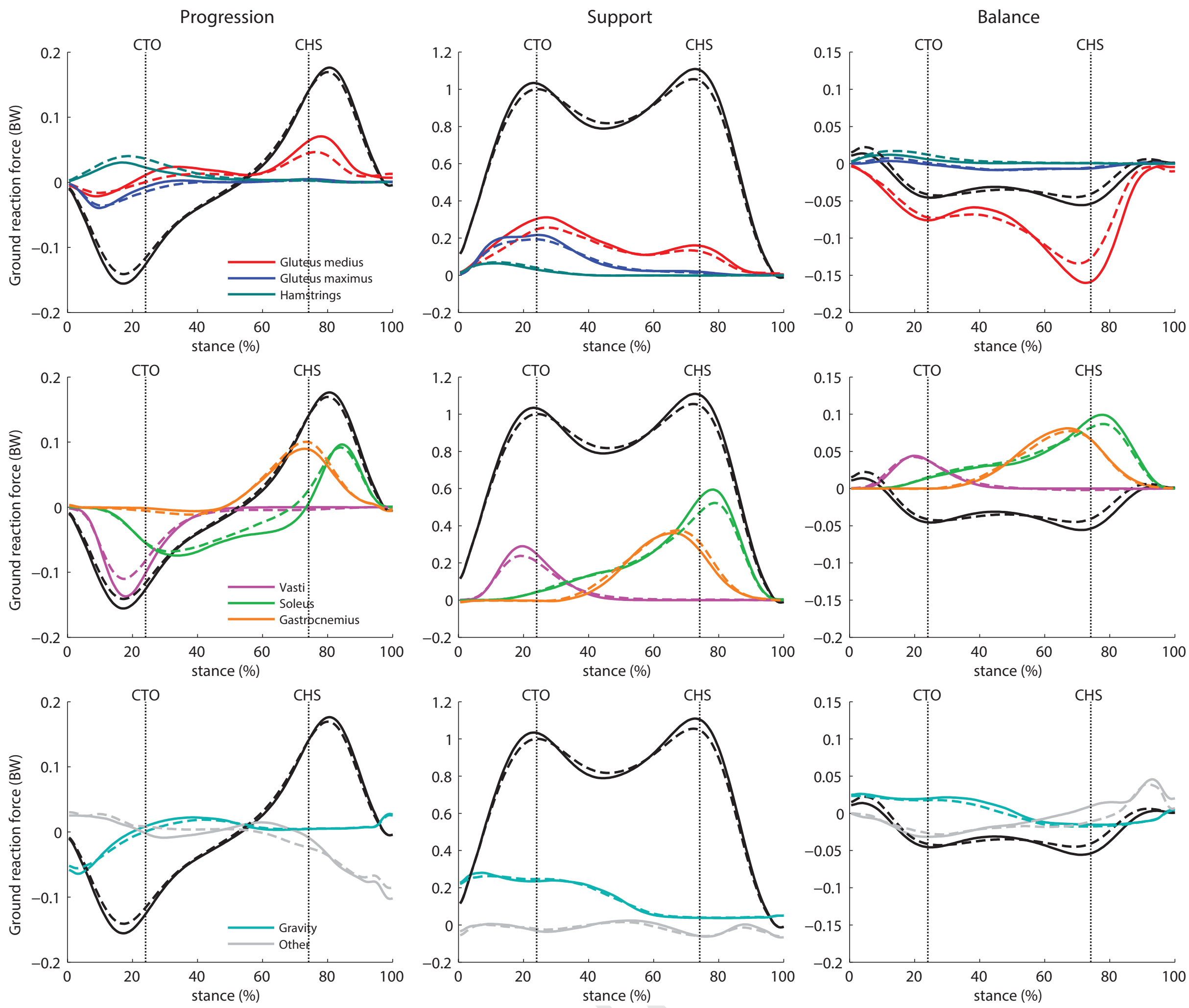
These participant data were previously published (Sritharan et al., 2016). Values are presented as mean (standard deviation) unless otherwise indicated. *P*-values were calculated using independent-samples *t*-tests. Significance level was defined as $\alpha = 0.05$. Significant differences are shown in ***bold italic***. Two measures of lower-limb alignment are presented: (1) mechanical axis angle measured from full-limb standing anteroposterior radiographs (positive values represent varus); and (2) static frontal-plane alignment measured using marker data from static gait trial (positive values represent varus).

Table 2. Mean frontal-plane moment arms of the GRF contribution vectors about the knee-joint centre at the first and second peaks of EKAM.

	Moment arm (cm)			Difference	
	Control	OA	*P*	cm	%
First peak					
Gluteus medius	15.6 (2.8)	20.2 (5.8)	< ***0.001***	4.6	30
Gluteus maximus	3.9 (1.6)	6.9 (5.7)	< ***0.001***	3.0	78
Vasti	-3.4 (1.8)	-4.3 (8.2)	0.147	-0.9	26
Gravity	0.2 (1.2)	2.9 (1.8)	< ***0.001***	2.7	1495
Total	5.2 (1.7)	7.2 (2.2)	< ***0.001***	2.0	39
Second peak					

Gluteus medius	33.5 (3.4)	37.2 (4.4)	< ***0.001***	3.7	11
Soleus	-4.9 (2.2)	-2.2 (2.4)	< ***0.001***	2.6	-54
Gastrocnemius	-7.7 (3.1)	-4.1 (3.2)	< ***0.001***	3.6	-47
Gravity	18.7 (4.6)	24.2 (6.1)	< ***0.001***	5.5	29
Total	3.5 (1.9)	7.2 (2.6)	< ***0.001***	3.7	103

Moment arms are presented as mean (standard deviation). *P*-values were calculated using independent-samples *t*-tests. Significance level was defined as $\alpha = 0.05$. Statistically significant differences are shown in ***bold italic***. Positive values represent moment arms for GRF vectors directed medially with respect to the knee-joint centre, while negative values represent moment arms for GRF vectors directed laterally with respect to the knee-joint centre.



Progression
Support
Balance
CTO
CHS
Ground reaction force (BW)
stance (%)
Gluteus medius
Gluteus maximus
Hamstrings
Vasti
Soleus
Gastrocnemius
Gravity
Other

Figure 1

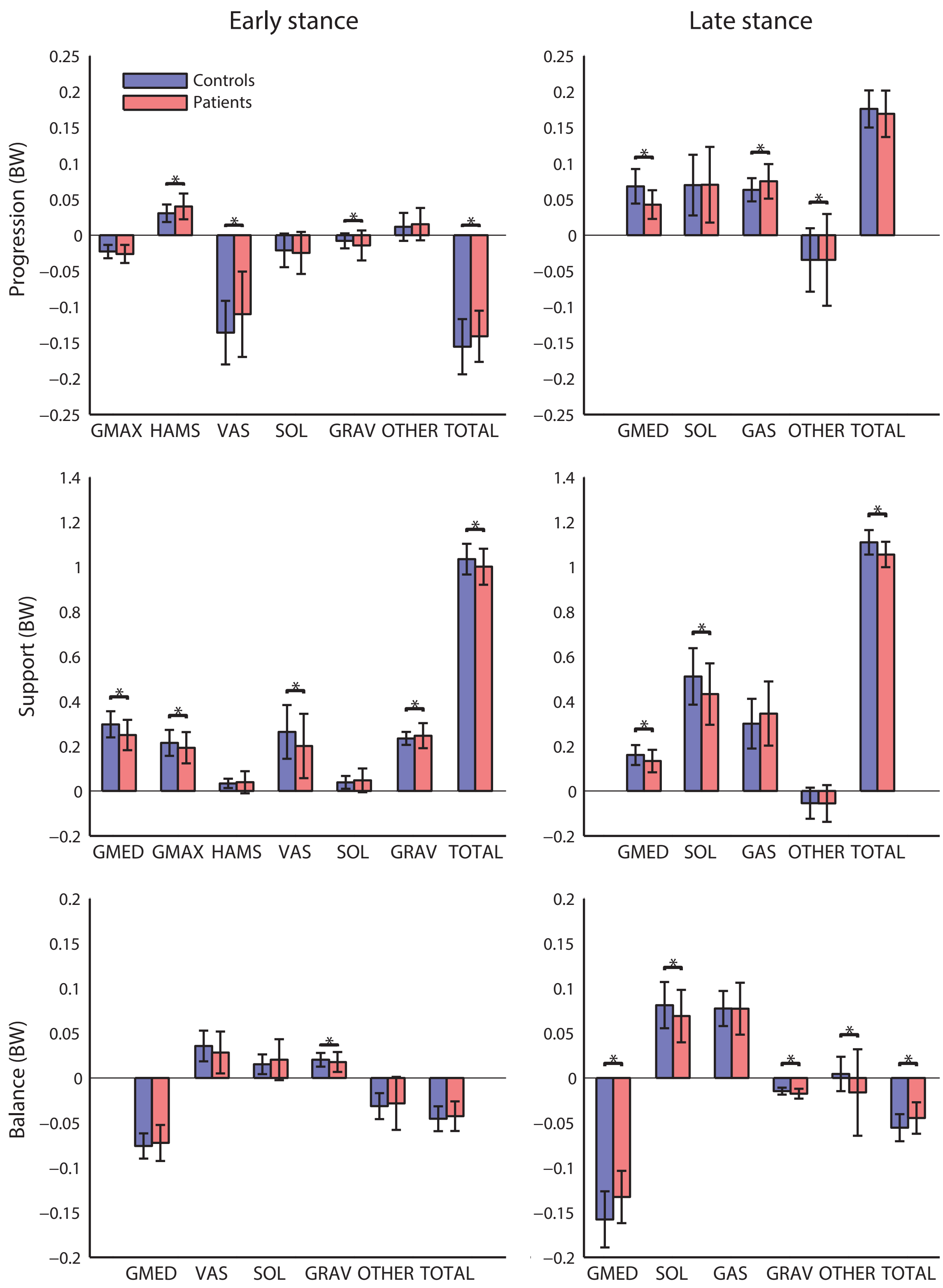

Early stance
Late stance
Controls
Patients
Progression (BW)
Support (BW)
Balance (BW)
0.25
0.2
0.15
0.1
0.05
0
−0.05
−0.1
−0.15
−0.2
−0.25
1.4
1.2
1
0.8
0.6
0.4
0.2
GMAX HAMS VAS SOL GRAV OTHER TOTAL
GMED SOL GAS OTHER TOTAL
GMED GMAX HAMS VAS SOL GRAV TOTAL
GMED SOL GAS OTHER TOTAL
GMED VAS SOL GRAV OTHER TOTAL
GMED SOL GAS GRAV OTHER TOTAL
*

Figure 2

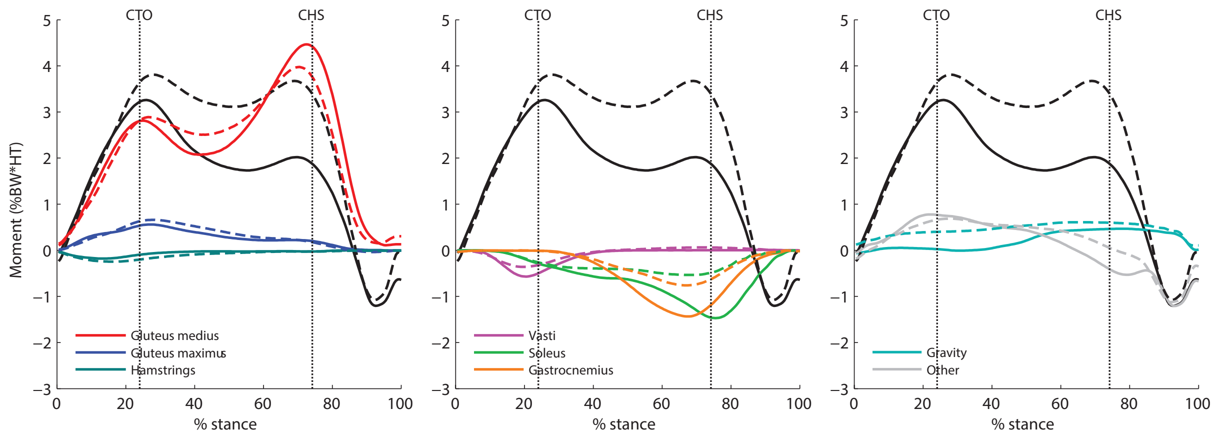


Figure 3

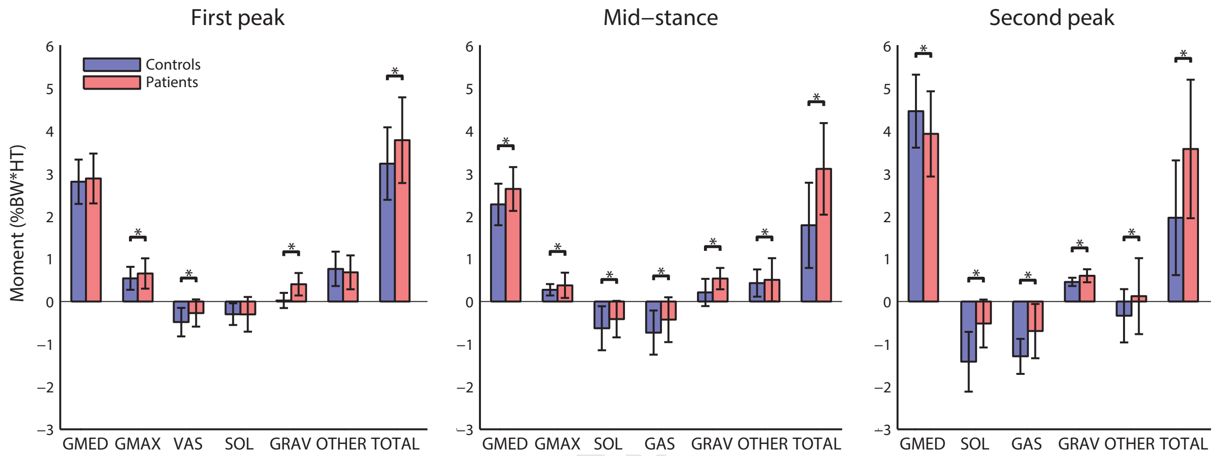


Figure 4

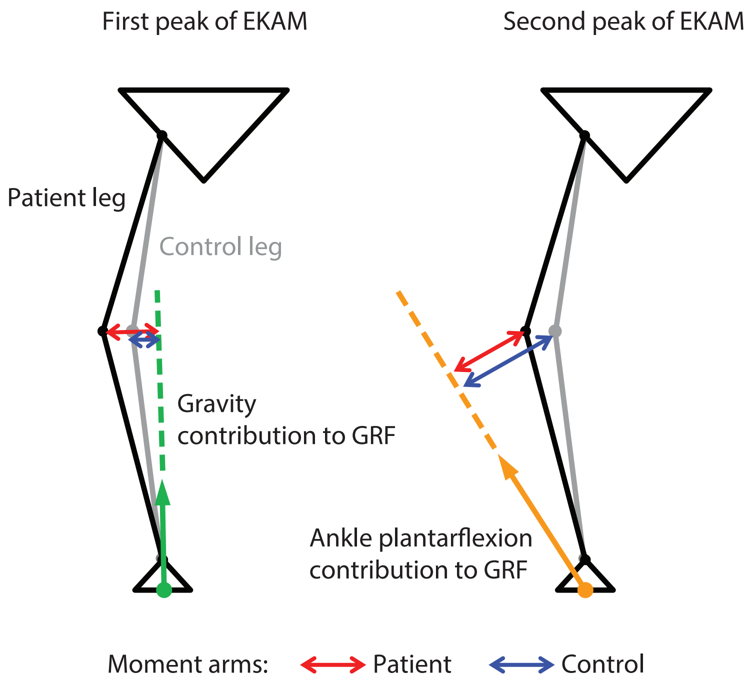

First peak of EKAM
Second peak of EKAM
Patient leg
Control leg
Gravity contribution to GRF
Ankle plantarflexion contribution to GRF
Moment arms:
Patient
Control

Figure 5