The influence of pain on tibiofemoral joint contact force and muscle forces in knee osteoarthritis patients during stair ascent

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Abstract
This study investigated the tibiofemoral joint (TFJ) forces and supporting muscle forces displayed by knee osteoarthritis (OA) patients during stair ascent, and if these forces were influenced by the presence of pain. Fifteen knee OA patients partitioned into two groups based on pain experienced during stair ascent trails using a Visual Analogue Scale (VAS) (OA-pain = 10; OA-no pain = 5) and 14 healthy aged-matched controls took part in this study. Kinematic and kinetic data were collected during three stair ascent trials, which provided the inputs for the musculoskeletal model FreeBody. TFJ contact forces and muscles forces were predicted by the model at early, mid- and late stance. These variables were compared between groups using a one-way analysis of variance. The results show the OA-pain ($P < .05; d = 2.4$) and OA-no pain ($P < .05; d = 1.1$) groups displayed reduced medial TFJ contact forces and altered muscle forces in comparison to healthy controls during early stance. This suggests pain and the anticipation of pain results in knee OA patients deliberately offloading the front limb during stair ascent, which alters supporting muscle forces. This study provides valuable information on knee OA mechanics during stair ascent for clinicians developing rehabilitation programs.

KEYWORDS
muscle forces, musculoskeletal model, osteoarthritis, stair ascent, tibiofemoral joint forces

1 | INTRODUCTION

Stair ascent is a challenging everyday activity for those who suffer with knee osteoarthritis (OA). Many studies have reported stair ascent being one of the main activities which elicits knee pain for those with knee OA,\(^1,^2\) greatly affecting quality of life.\(^3\) The increased susceptibility of knee pain during stair ascent likely comes from weight-bearing at higher knee flexion angles.\(^4\) Because of this, knee OA patients have been reported to compensate while walking upstairs to offload the OA knee as a strategy to avoid pain.\(^5\) Asay et al\(^4\) reported that during stair ascent those with knee OA increased trunk flexion angle during the weight-acceptance phase of stance to reduce the quadriceps moment in comparison to healthy controls. Further, Meireles et al\(^6\) found that, in comparison to healthy controls, knee OA patients altered their stair ascent
strategy, either through increased trunk lean toward the leading leg or reducing speed. These compensation strategies are likely performed to reduce the load experienced by the knee, either as a response to pain felt when loading at higher knee angles, or a lack of support from the front limb.

Despite understanding the compensation strategy used by knee OA patients when performing stair ascent, our knowledge of tibiofemoral joint (TFJ) contact forces and the muscle forces during compensation is still limited. Recent advancements in musculoskeletal modeling has allowed for the prediction of TFJ contact forces and muscle forces; however, the majority of the research has been on knee OA patients performing gait. Recently, the study by Meireles et al. did use a musculoskeletal model with knee OA patients during stair ascent and reported the compensation strategy used by OA patients offloads both the medial and lateral compartments of the TFJ during the weight-acceptance phase of stance in comparison to healthy controls. This supports the theory that knee OA patients deliberately move to avoid loading the knee during the early stance phase of stair ascent. Muscle forces, however, were not reported, so how muscle forces contribute to the compensatory strategy is still unknown.

Knee OA pain is highly variable and can often be affected by psychosocial conditions. If pain is a contributing factor to knee OA patient compensation when performing stair ascent, the presence of pain would be very influential in the loading experienced in the TFJ. Pain is often assessed through the use of questionnaires, such as the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) questionnaire and the Knee Injury and Osteoarthritis Outcome Score questionnaire, where knee OA patients subjectively document pain experienced over the last 24 hours. However, pain is highly variable, and may not be experienced during every task and vary day to day. For example, a knee OA patient may perform stair ascent without pain despite feeling pain in the same knee over the last 24 hours. Because of this, our understanding of how pain influences movement strategy and TFJ contact forces during stair ascent is still unclear.

Investigating knee OA TFJ contact forces and muscle forces during stair ascent, and the role pain may play altering movement strategies would provide a better understanding of knee OA mechanics during a task which, if painful and difficult to perform, can greatly influence quality of life. It is therefore the aim of this study to use a musculoskeletal model to predict the TFJ contact forces and muscle forces of knee OA patients while performing stair ascent and compare them to predictions from healthy controls. Further, the knee OA patients will be partitioned into two groups based on the pain they experienced when performing stair ascent. It is hypothesized that the knee OA patients who experience pain during stair ascent will display reduced TFJ contact forces and muscle forces as they use the compensatory movement strategy reported by Asay et al. and Meireles et al. in comparison to knee OA patients who experience no pain and healthy controls. Understanding the differences between these groups may help clinicians provide better rehabilitation strategies for knee OA patients and help improve stair ascent performance, and thus improve quality of life.

2 MATERIALS AND METHODS

Fifteen OA patients were recruited from the OA clinic at Kingston Hospital, (Kingston upon Thames, KT2 7QB, London, UK). Those recruited from the OA clinic at Kingston Hospital had been referred to the clinic by their consultant. These patients had reported OA-type symptoms but after assessment by the consultant the OA was not severe enough for surgical intervention. Thus, these patients were classified as having mild knee OA. The inclusion criteria for participation in this study were being over 40 years of age, attending the OA clinic at Kingston Hospital, being able to perform everyday activities such as walking up and down stairs and sitting and standing to a chair without the use of a cane. Due to the musculoskeletal model used in this study being modeled on the right leg, only those with OA of the right knee were included in this group. Further exclusion criteria were also imposed: (i) the participant had been diagnosed with OA in both knees and (ii) had previously received surgical treatment for knee OA. Fourteen participants for the healthy group were recruited from the local community and were not involved with the OA clinic at Kingston Hospital. Participant data for the healthy knee group were excluded from data analysis if they failed the following criteria: (i) had previously sustained a lower limb or lower back injury which required surgery, (ii) experience undiagnosed pain in the knee during activities of daily living, and (iii) had any other lower limb or lower back injury within 6 months of testing. All participants refrained from taking ibuprofen or painkillers the week prior to testing, and did not exercise, or ingest caffeine or alcohol 24 hours prior to testing.

Ethical approval for data collection was obtained from the NHS research ethics committee (Reference: 15/LO/0668), the Clinical Research Network (CRN) South London, and the St Mary’s University ethics committee. All data collection took place at St Mary’s University. Each participant completed a WOMAC questionnaire, which documented their knee pain and function over the previous 24 hours, prior to starting data collection. All participants also recorded pain scores
obtained using a 10-cm VAS, which were collected after performing stair ascent. These data were used to determine groups by using the pain scale categories suggested by Jensen et al., where no pain, mild, and moderate, and severe pain are classified by 0 to 4 mm, 5 to 44 mm, 45 to 74 mm, and 75 to 100 mm, respectively. Therefore, the groups were categorized as OA-pain (VAS score of >5 mm), OA-no pain (VAS score of 0-4 mm), and healthy (VAS score of 0-4 mm). The participant descriptive data is shown in Table 1.

Upon arrival, the participant was introduced to the laboratory environment and their height was measured. Before testing, each participant signed a consent form, and completed a health questionnaire and WOMAC questionnaire. Eighteen 25-mm reflective markers were placed on landmarks of the right lower limb and pelvis, which were attached to the predetermined landmarks by the principal researcher every session. These landmarks were palpated, and a reflective marker was attached on the most prominent part of the bony landmark with double-sided tape. All participants wore shorts which would not obstruct the reflective markers from motion capture. If any markers became loose, obstructed or unattached, the trial would be discarded and repeated. Before the start of each data collection session an anatomical calibration trial was obtained with the participant standing motionless in the anatomical position.

A custom built staircase was created to the specification of Aminaka et al. The second step of the staircase was replaced by a separate box, which was placed directly on the force platform. An opening was cut into the staircase to receive the box, with a 3-cm border of space in order to avoid any contact between the staircase and the box, thus minimizing noise artifact when the participant was in contact with the other stairs. Participants were instructed not to focus on the second step to avoid any deliberate maneuver to contact it. A trial was deemed successful when the participant made clear contact with their right foot on the box representing the second step and no accidental contact was made between the stairs and any of the reflective markers. Handrails were attached to the staircase as it is common for the knee OA population to use handrails regularly when performing stair ascent. The bannisters were made of steel and painted black, so they would not reflect the infra-red light from the cameras. The handrails were 900 mm tall to comply with current U.K. building regulations (The Department for Communities and Local Government, 2010). During the stair ascent trials all participants were instructed to “have their hands on the rails, but to use them as little as possible”. This aimed to minimize any movement compensations from the use of handrails between healthy and pathological populations. Force applied to the handrails by each participant was not measured. Before testing the participant practiced stair ascent at a self-selected pace until they felt comfortable performing the activity. Data were then collected from three stair ascent trials, after which a rating of perceived pain was also recorded using a 10-cm VAS. The participant used a pen to mark on the line the level of perceived pain in their right knee based on a scale of “no pain” to “worst pain imaginable.”

Three-dimensional motion capture data were collected using an 11 camera Vicon motion capture system (Vicon MX System, Vicon Motion Systems Ltd, Oxford, UK) at a sampling rate of 200 Hz. Each marker of the model was manually identified for each trial by the principal researcher. For frames where marker data was absent a Woltring filter function within the Vicon pipeline was used to estimate the path and location of that marker. These data were then exported to Microsoft Excel for further analysis. GRF data were collected using a recessed 600 mm × 900 mm Kistler 9287BA force plate (Kistler Instruments Ltd, Hook, UK), which was under the box representing the second step of the staircase, at a sampling rate of 1000 Hz. The center of pressure data for these trials was changed accordingly. In short, the center of pressure of the foot when in contact with the box assumed that the sum of the forces and moments acting on the box was 0. The forces applied to the box were based on the GRF vector and weight of the box. The center of pressure was then recalculated based on the GRF vector acting through it, with the sum of the moments acting on the box around its center of mass equaling 0. Kinetic data and kinematic data were filtered using a fourth order low-pass Butterworth filter with a cut-off frequency of 15 and 6 Hz, respectively, which was performed in Matlab (Mathworks, MA). Kinetic data were then down sampled using a five-point average in Microsoft Excel to synchronize the data with the kinematic data at 200 Hz. These kinetic and kinematic data provided the inputs for the musculoskeletal model used in this study.

The FreeBody model is a musculoskeletal model which is freely available in the public domain (https://www.msksoftware.org.uk/software/freebody/). FreeBody is a linked five segment (foot, shank, thigh, pelvis, and patella) musculoskeletal model, which places no kinematic constraints on the segments that is, the ankle, knee and hip have six degrees of freedom. The patella, however, has zero degrees of freedom as its location and orientation is calculated based on the knee angle and the position of the thigh segment. The musculoskeletal geometry, taken from the Klein Horsmann cadaver dataset is added to the model once the location and orientation for each segment for each frame has been established. This includes the origin and insertions for 163 muscle elements, 14 ligament elements, and the patellar tendon. The anthropometric data for each segment is provided by the work of De Leva.

The musculoskeletal model and the collected kinetic and kinematic data are used to determine the equations of motion which govern the motion of the lower limb. This creates an indeterminacy problem of 193 unknown forces and many
possible solutions. An optimization approach is used to find the most physiologically likely solution by imposing physiological constraints developed by the work of Crowinshield and Brand and Raikova. To achieve this, a cost function based on maximizing muscular endurance is minimized. The model optimization and postprocessing were also done in Matlab. The paper by Cleather and Bull provides a full description of the FreeBody model and the reliability and validity of the TFJ and muscle force estimations from the model has been previously investigated.

Model predictions were interpolated using a spline filter to create a waveform of 100 data points, which represented 100% of stance. Each participant was considered to be in stance when the GRF data exceeded 15 N to account for noise error. The mean for each activity was calculated by averaging the waveforms from all three trials. Waveforms representing the group mean for each group for stair ascent were subsequently calculated from the mean waveforms from each participant. Upper and lower confidence intervals of 95% were also calculated for each group.

The GRF curve for each group was used to define early stance, mid-stance, and late stance. Early stance and late stance were defined as the time point as a percentage when the peak GRF occurred during the first and second half of stance, respectively. Mid-stance was defined as the time point where the lowest GRF occurred between early and late stance. The percentage of stance (with 0% being initial foot contact and 100% representing toe-off) when early stance, mid-stance, and late stance occurred for the healthy participants (obtained from the GRF waveform representing the mean of the group) were used as points during stance to assess the differences between groups for all selected variables. Thus, peak early stance, mid-stance, and late stance occurred at 32%, 54%, and 84% of stance, respectively. This was done to identify if TFJ contact forces and muscle forces in knee OA patients differed during the key phases of stance seen in healthy controls. At these intervals, values for total, medial and lateral compartment contact forces, medial load share, muscle forces for the gluteals, quadriceps, hamstrings, and triceps surae, were compared between groups. Gluteal muscle force was the sum of forces from the force actuators in FreeBody representing the gluteus maximus, gluteus medius and gluteus minimus. Quadriceps force was the sum of the vasti muscle group (vastus medius, vastus intermedius, and vastus lateralis). Hamstring force was the sum of forces predicted in the semimembranosus, semitendinosus and biceps femoris. The triceps surae muscle force comprised of the predicted forces of both the medial and lateral gastrocnemii, and soleus.

All data were assessed for normality using a Shapiro-Wilks test. For the comparison between the OA-pain group, OA-no pain group and healthy controls, normally distributed data were analyzed using a one-way analysis of variance, with differences between groups determined using a Hochberg GT2 post-hoc test to account for the different group sizes. Nonnormally distributed data were analyzed using a Kruskal-Wallis test. If a significant difference was identified, separate Mann-Whitney U tests using a Bonferroni correction were run to determine significant differences between groups. Effect sizes (Cohen’s d) were calculated to determine the magnitude of group differences, where small (0.2-0.6), moderate (0.6-1.2), large (1.2-2) and very large (2-4) effect sizes were interpreted in line with the previous studies. Significance values were set at $P < .05$. All statistical analyses were performed using SPSS (SPSS Inc. Chicago, IL).

### 3 | RESULTS

Table 1 displays the comparison between groups for stair ascent speed, VAS and WOMAC. The OA-pain group experienced significantly more pain in comparison to both the OA-no pain group ($P < .05$, $d = 1.6$) and healthy controls ($P < .05$, $d = 1.8$). Both OA-pain ($P < .05$, $d = 1.8$) and OA-no pain ($P < .05$, $d = 2.0$) reported significantly higher WOMAC scores than the healthy controls, but no significant difference in scores were reported between the OA-pain and OA-no pain groups ($P > .05$, $d = 0.9$). The healthy controls performed stair ascent at a significantly faster speed than both the OA-pain group ($P < .05$, $d = 1.1$) and OA-no pain ($P < .05$, $d = 1.3$). No differences in stair ascent speed were seen between the OA groups ($P > .05$, $d = 0.0$).

Figure 1 displays the GRF waveform comparisons between groups during the stance phase of stair ascent. Both OA-pain ($P < .05$; $d = 1$) and OA-no pain ($P < .05$; $d = 1$) groups reported significantly lower GRF during early stance of stair ascent in comparison to the healthy controls. The OA-no pain group reached early stance peak GRF more quickly (33% of stance) than the OA-pain group (37% of stance), reaching peak GRF at a similar point during early stance to the healthy control group (32% of stance). However, the magnitude of GRF for the OA-no pain group was lower than the healthy control group (0.94 × BW vs 1.03 × BW).

Figure 2 displays knee joint angles during the stance phase of stair ascent. No significant differences between all groups were seen in all three planes, with all effect sizes being moderate to negligible ($P > .05$; $d < 1.1$). Medial knee contact force...
Table 1: Descriptive statistics for the osteoarthritis (OA)-pain group

| Group     | Participant | Gender | Age (years) | Height (cm) | Body mass (kg) | Speed (ms⁻¹) | VAS (mm) | WOMAC |
|-----------|-------------|--------|-------------|-------------|----------------|--------------|----------|-------|
| OA-pain   | Os1         | M      | 82.0        | 163.5       | 62.5           | 0.32         | 9.0      | 38.0  |
|           | Os2         | F      | 79.0        | 154.5       | 72.3           | 0.42         | 20.0     | 32.0  |
|           | Os3         | F      | 64.0        | 157.5       | 62.7           | 0.22         | 24.0     | 18.0  |
|           | Os4         | F      | 62.0        | 160.5       | 120.2          | 0.23         | 8.0      | 29.0  |
|           | Os5         | F      | 80.0        | 158.0       | 42.6           | 0.38         | 14.0     | 27.0  |
|           | Os6         | F      | 67.0        | 158.0       | 80.3           | 0.33         | 10.5     | 37.0  |
|           | Os11        | M      | 81.0        | 165.0       | 79.2           | 0.37         | 19.0     | 28.0  |
|           | Os13        | F      | 70.0        | 160.0       | 66.9           | 0.46         | 14.0     | 6.0   |
|           | Os14        | F      | 71.0        | 156.5       | 69.8           | 0.40         | 17.5     | 28.0  |
|           | Os15        | F      | 77.0        | 150.0       | 44.2           | 0.53         | 8.0      | 26.0  |
| Mean ± SD |             |        | 73.3 ± 7.4a | 158.4 ± 4.3a| 70.1 ± 21.7 | 0.37 ± 0.1a | 14.4 ± 5.6ab | 26.9 ± 9.3a |

| OA-no pain | Os7         | F      | 58.0        | 160.5       | 75.5           | 0.38         | 1.0      | 27.0  |
|           | Os8         | F      | 63.0        | 165.5       | 56.2           | 0.41         | 3.0      | 24.0  |
|           | Os9         | M      | 63.0        | 183.0       | 96.9           | 0.40         | 4.0      | 23.0  |
|           | Os10        | F      | 78.0        | 167.0       | 50.2           | 0.27         | 3.0      | 10.0  |
|           | Os12        | F      | 75.0        | 160.0       | 68.0           | 0.37         | 4.0      | 10.0  |
| Mean ± SD |             |        | 67.4 ± 8.6  | 167.2 ± 9.3 | 69.4 ± 18.3 | 0.37 ± 0.1c | 3.0 ± 1.2 | 18.8 ± 8.2c |

| Healthy   | Hs1         | F      | 65.0        | 157.0       | 45.2           | 0.37         | 0.0      | 2.0   |
|           | Hs2         | M      | 65.0        | 170.5       | 83.5           | 0.34         | 0.0      | 0.0   |
|           | Hs3         | M      | 66.0        | 180.0       | 82.7           | 0.36         | 0.0      | 4.0   |
|           | Hs4         | F      | 53.0        | 173.0       | 70.6           | 0.54         | 0.0      | 0.0   |
|           | Hs6         | F      | 68.0        | 157.5       | 67.4           | 0.47         | 2.0      | 1.0   |
|           | Hs7         | F      | 65.0        | 159.5       | 59.9           | 0.51         | 0.0      | 1.0   |
|           | Hs8         | F      | 67.0        | 160.0       | 76.5           | 0.55         | 3.0      | 1.0   |
|           | Hs9         | M      | 61.0        | 180.0       | 101.2          | 0.54         | 3.0      | 0.0   |
|           | Hs10        | M      | 65.0        | 185.0       | 93.8           | 0.50         | 2.0      | 1.0   |
|           | Hs11        | M      | 69.0        | 169.0       | 78.3           | 0.47         | 0.0      | 0.0   |
|           | Hs12        | F      | 67.0        | 172.5       | 51.6           | 0.58         | 0.0      | 0.0   |
|           | Hs13        | M      | 68.0        | 178.0       | 84.8           | 0.51         | 0.0      | 0.0   |
|           | Rs14        | M      | 72.0        | 175.0       | 68.6           | 0.47         | 0.0      | 0.0   |
|           | Rs16        | M      | 59.0        | 181.0       | 90.6           | 0.52         | 0.0      | 2.0   |
| Mean ± SD |             |        | 65 ± 4.7    | 171.3 ± 9.5 | 75.3 ± 15.9 | 0.48 ± 0.1  | 0.7 ± 1.2 | 0.9 ± 1.2 |

Abbreviations: WOMAC, Western Ontario and McMaster Universities Osteoarthritis Index.

Notes: OA-no pain group and the healthy control group for stair ascent. Statistical significance set at (P < 0.05).

- A significant difference between the OA-pain group and the healthy control group.
- A significant difference between the OA-pain group and the OA-no pain group.
- A significant difference between the OA-no pain group and the healthy control group.

During early stance was significantly greater in the healthy controls in comparison to both OA-pain (P < .05; d = 2.4) and OA-no pain groups (P < .05; d = 1.1) (Figure 3). Medial force share was also significantly greater in the healthy controls in comparison to both groups during the early phase of stance. However, it was only significantly greater than the OA-pain group (P < .05; d = 1.4). During late stance, the OA-pain group displayed less medial contact forces and greater lateral contact force than both the OA-no pain and healthy control groups, which also affected the medial load share waveform. However, these differences between groups were not significant.
The OA-pain group and OA-no pain group produced reduced muscle forces during early stance in all four muscle groups seen in Figure 4. Only significant differences were found between the OA-pain group and healthy controls for the gluteal muscles \((P < .05; d > 1.1)\) and both the OA-pain group \((P < .05; d > 0.9)\) and OA-no pain \((P < .05; d > 0.8)\) group for the triceps surae muscles. Muscle forces were similar for the remainder of stance for all muscle groups except the hamstring muscle group, where it remained elevated during mid-stance in the OA-pain group. Differences in hamstring muscle forces during mid-stance between groups were, however, not statistically significant.

4 | DISCUSSION

The aim of this study was to investigate the OA knee during stair ascent. To achieve this, TFJ contact force and muscle force model predictions using a musculoskeletal model from knee OA patients and healthy controls were compared during stair ascent. The knee OA patients were split into two groups (OA-pain and OA-no pain) based on the pain they experienced during performing the stair ascent trials. It was hypothesized that the OA-pain group would display reduced TFJ contact forces and muscle forces when compared to the OA-no pain and healthy controls. This study confirms the hypothesis as the knee OA-pain group displayed significantly reduced medial contact force and altered muscle force strategies during early stance in comparison to healthy controls. However, the OA-no pain also displayed reduced medial TFJ contact forces and muscle forces during early stance, disproving the hypothesis that, despite having knee OA, experiencing no pain during stair ascent would result in less compensation and therefore display TFJ contact forces similar to healthy controls.
All groups produced similar GRF during stair ascent during mid-stance and late stance, with the only difference between groups occurring during early stance. Both OA-pain groups produced lower GRF (Figure 1) and the OA-pain group reached peak early stance GRF later during stance (32% vs 39% of stance) in comparison to healthy controls. Interestingly, there was no difference between TFJ contact forces between the OA-pain and OA-no pain groups during all three phases of stance. It was hypothesized that, despite the presence of OA, not experiencing pain during stair ascent would result in the OA patient not compensating and having TFJ contact forces similar to healthy controls. The results of this study suggest this to be false as the OA-no pain group displayed similar TFJ contact force magnitudes and medial load share to the OA-pain group. It is likely the OA-no pain group still employed a movement strategy with the anticipation of pain during stair ascent, which therefore experienced the lower knee contact forces similar to the OA-pain group. It is possible that pain may be a poor marker for determining progress for knee OA patients undergoing a rehabilitation program. Though a reduction in pain during stair ascent would improve quality of life, a compensation strategy, and thus different TFJ contact forces, may still affect the progression of the disease.

Further support that the OA groups compensated to offload the knee during the early stance phase of stair ascent is shown by the muscle forces reported in this study. All muscle forces investigated in this study were lower in both knee OA groups in comparison to healthy controls, but only significantly reduced in the gluteal muscles in the OA-pain group, and the triceps surae in both OA groups (Figure 4). These reduced muscle forces coincide with the reduced TFJ contact forces during the early stance phase. As the compensation strategy deliberately avoids loading the front limb during stair ascent, musculature whose function it is to provide stability during the weight-acceptance phase of stance, such as the gluteal muscles, contribute significantly less as they are not required to provide greater support forces. Though this strategy may acutely reduce pain while performing stair ascent, it may be detrimental to the progression of knee OA. For example, a compensation strategy which reduces the loads experienced at the knee may increase muscle atrophy due to lack of use.
A recent review by Shorter et al.\textsuperscript{20} highlighted that increased muscle wastage can lead to decreased stability and mobility of the knee resulting in progressing knee OA. However, further research is needed to determine if this movement strategy is a result of weakness in these muscles or this movement strategy, used because of the disease, leads to muscle inhibition and subsequent muscle weakness.

One of the strategies which may have helped reduced TFJ loading during early stance is reducing the speed of performing stair ascent. Those with both mild\textsuperscript{6,21,22} and severe\textsuperscript{23} OA have reportedly performed stair ascent slower than their aged-matched controls. Indeed, slowing down stair ambulation would allow more time during the stance phase, where the initial loading during early stance can be done more gradually. Both OA groups in this study reduced stair ascent speed (Table 1) in comparison to healthy controls. This suggests both knee OA groups were tentative in loading the front limb during early stance, and therefore slowed down stair ascent speed as a compensatory strategy to load the stance limb more gradually.

The presence of the handrails also could have been used by both knee OA groups as a strategy to offload the knee during the early stance phase. All three groups were instructed to “have their hands on the rails throughout stair ascent, but to use them as little as possible”. This was done so the knee OA groups performed stair ascent how they would during everyday life, with the healthy control group instructed to do the same for comparison. It is possible the OA groups used the handrails more than the healthy control group in order to provide stability and slow down the loading rate of the front limb, and thus the TFJ. This type of strategy has been reported previously by Reid et al.\textsuperscript{24} where those with fear of falling utilized the handrails more than aged-matched healthy controls during stair ascent, which slowed down the velocity of the center of pressure in the front stance foot. This would be indicative of someone using the rails for additional stability rather than gaining full stability by loading the front limb. Indeed, the process of using the rails for stability during the early stance phase may be the cause of the longer stance phases in both knee OA groups, that is, this strategy takes longer and therefore slows down the OA patient during stair ascent. This suggests the presence of the handrails may have played a role in the differences in TFJ and muscle forces between the OA groups and the controls seen in this study.

**FIGURE 4** Muscle forces for the gluteals, quadriceps, hamstrings, and triceps surae during the stance phase of stair ascent. The thick black line represents the osteoarthritis (OA)-pain group, the thick, dotted line represents the OA-no pain group, and the thick grey line represents the healthy control group. 95% confidence intervals for both groups are represented by the thin black line, dotted black lines, and grey lines, respectively. * denotes a significant difference between group the OA-pain group and healthy controls. Statistical significance set at \((P < .05)\). # denotes a significant difference between group the OA-no pain group and healthy controls. Statistical significance set at \((P < .05)\).
Unfortunately, force through the handrails was not recorded in the current study so the contribution of the handrails to the findings of this study is unknown. Further research should consider measuring the force applied to handrails to provide understanding to the role they play in the magnitude and distribution of loading the knee in knee OA patients.

The compensation strategy to reduce loading during early stance may have also had an effect on TFJ load distribution as both OA groups displayed significantly reduced medial TFJ contact forces, and thus total TFJ contact forces. Some of the reduction in medial TFJ contact force could be attributed to changes to the distribution of contact force. In the healthy knee, as seen in Figure 3, medial load share is approximately between 70% and 80% during the weight acceptance phase, which has also been reported previously. However, in the OA groups, medial load share is considerably lower at approximately 50%. This suggests that the OA groups in this study reduced loading of the TFJ during stair ascent by reducing Ground Reaction Force (GRF) which distributed TFJ contact force more evenly through the medial and lateral compartments during early stance. These findings differ to those reported by Meireles et al. Despite both studies reporting reduced TFJ contact forces in those with an OA knee, the strategy of how this was achieved appears to be different. Meireles et al report that contact force was reduced in both the medial and lateral TFJ compartments of the OA knee while negotiating stair ascent at a self-selected pace, whereas the current study shows only medial TFJ contact force was reduced as lateral TFJ contact force was similar to healthy controls (Figure 3).

There are two possible factors which have resulted in the differences in findings between the study by Meireles et al and the current study. Firstly, the model used by Meireles et al consisted of a deformable element of constant 2 mm thickness, which represented the articular cartilage, where the FreeBody model used in this study did not include cartilage. The inclusion of cartilage may have influenced the estimation of how TFJ is distributed between the medial and lateral compartments. Secondly, the inclusion of letting the participants have their hands on the handrails may also have influenced TFJ load distribution. Meireles et al discussed how an increased forward trunk lean reduced the quadriceps moments arm, reducing total TFJ contact force and decreasing contact force in both the medial and lateral compartments. The inclusion of the handrails may have played a similar role in this study but by altering the moment arm in the frontal plane. In Figure 2, both OA groups are in knee abduction during the point of early stance, whereas the healthy controls were in knee adduction. This would have reduced the medial moment arm and explain why medial TFJ contact forces and medial load share is lower in the knee OA groups when compared to the healthy controls. Utilizing and gaining more stability from the handrails may alter frontal plane knee kinematics by keeping the knee in abduction during early stance. Similar findings have been reported previously by Doslikova who found using a handrail on the contralateral side of the leading leg while performing stair ascent reduced knee abduction moment, and by proxy medial TFJ contact force, during the early stance phase of stair ascent. Despite all groups in the current study having their hands on the rails during stair ascent, it is possible that both knee OA groups actively using them to gain stability during early stance is what can cause changes in the magnitude and distribution of TFJ contact forces, and why the results of the current study differ from those of Meireles et al.

There are further limitations of this study that should be discussed. Firstly, the sample size for the OA-no pain group was small (n = 5). Variance within a sample of this size can increase the chance of a type 2 error and therefore these results must be interpreted with caution. Secondly, the information about the OA participants was limited in this study. Imaging data for each OA patient was not available, and therefore key information such as disease severity (often measured by the Kellgren-Lawrence score), and disease location (medial, lateral, or both) was not known. As all OA participants were active and not awaiting surgery it was deemed that the OA severity of these patients was mild. In support of this, the reduced TFJ contact forces reported in this study match the findings from other studies where those with early or mild knee OA display reduced TFJ contact forces in comparison to healthy controls. Missing disease location provides the bigger limitation for this study. Differences in TFJ loading and muscle forces between those with medial and lateral TFJ OA have been reported and therefore the differences in disease location for the OA groups may have influenced the findings of this study. Future studies investigating TFJ contact forces during stair ascent should have extensive knee OA information on each participant and use strict inclusion criteria to further our understanding of knee OA mechanics during stair ascent.

5 | CONCLUSION

The aim of this study was to provide insight into the differences in TFJ contact forces and muscle forces between knee OA patients and healthy controls during stair ascent. Further, this study investigated if pain influences TFJ contact forces and muscle forces in knee OA patients during stair ascent. The findings presented here show both knee OA groups displayed
significantly reduced medial contact force and altered muscle force strategies in comparison to healthy controls during the early stance phase of stair ascent, whether pain was experienced during stair ascent or not. Pain and the anticipation of pain result in knee OA patients deliberately offloading the front limb during early stance, possibly by slowing down stair ascent or becoming more reliant on the use of handrails. As a result, muscle forces are reduced as less force is required to support this movement strategy. This study provides valuable information on knee OA mechanics during stair ascent and the influence of pain, which is essential for clinicians when developing rehabilitation programs for knee OA patients with the aim of improving stair ascent performance and quality of life. Further research is needed using knee OA patients performing stair ascent with the location and severity of the OA confirmed by radiographs to progress the findings of this study.

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CONFLICT OF INTEREST
There is no conflict of interest for this study.

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