Clinical Biomechanics 101 (2023) 105858

Contents lists available at ScienceDirect

Clinical Biomechanics

journal homepage: www.elsevier.com/locate/clinbiomech

The biomechanical fingerprint of hip and knee osteoarthritis patients during activities of daily living

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ARTICLE INFO

Keywords:
Hip osteoarthritis
Knee osteoarthritis
Joint contact forces - Opensim

ABSTRACT

Background: Osteoarthritis is a highly prevalent disease affecting the hip and knee joint and is characterized by load-mediated pain and decreased quality of life. Dependent on involved joint, patients present antalgic movement compensations, aiming to decrease loading on the involved joint. However, the associated alterations in mechanical loading of the ipsi- and contra-lateral lower limb joints, are less documented. Here, we documented the biomechanical fingerprint of end-stage hip and knee osteoarthritis patients in terms of ipsilateral and contralateral hip and knee loading during walking and stair ambulation.

Methods: Three-dimensional motion-analysis was performed in 20 hip, 18 knee osteoarthritis patients and 12 controls during level walking and stair ambulation. Joint contact forces were calculated using a standard musculoskeletal modelling workflow in Opensim. Involved and contralateral hip and knee joint loading was compared against healthy controls using independent *t*-tests ($p < 0.05$).

Findings: Both hip and knee cohorts significantly decreased loading of the involved joint during gait and stair ambulation. Hip osteoarthritis patients presented no signs of ipsilateral knee nor contralateral leg overloading, during walking and stair ascending. However, knee osteoarthritis patients significantly increased loading at the ipsilateral hip, and contralateral hip and knee joints during stair ambulation compared to controls.

Interpretation: The biomechanical fingerprint in knee and hip osteoarthritis patients confirmed antalgic movement strategies to unload the involved leg during gait. Only during stair ambulation in knee osteoarthritis patients, movement adaptations were confirmed that induced unbalanced intra- and inter-limb loading conditions, which are known risk factors for secondary osteoarthritis.

1. Introduction

Osteoarthritis (OA) is one of the most prevalent joint disorders that affect millions of people worldwide. OA is a chronic, multifactorial disease that mainly affects the knee and to a lesser extent the hip joint (Felson et al., 2000; Hunter and Felson, 2006; Lories and Luyten, 2011; Prieto-Alhambra et al., 2014). Age, gender and obesity are known to contribute to OA development, but biomechanical as well as biological changes within the joint are likely to play a significant role in the OA pathogenesis (Allen and Golightly, 2015; Johnson and Hunter, 2014; Palazzo et al., 2016). OA is characterized by loading pain, a reduction in muscle strength and increased joint stiffness, resulting in a decreased ability to perform activities of daily living (Cheng et al., 2010).

OA patients have an increased risk for developing secondary OA in

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https://doi.org/10.1016/j.clinbiomech.2022.105858
Received 4 November 2021; Accepted 7 December 2022
Available online 9 December 2022
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the adjacent joints of the ipsilateral and contralateral leg, which has been related to an altered mechanical loading environment in these joints (Kraus et al., 2013; Sayre et al., 2010; Shakoor et al., 2002; Shakoor et al., 2003; Shakoor et al., 2011). Patients with knee (KOA) and hip OA (HOA) adapt their movement pattern during level walking and these adaptations are not limited to the involved joint, but affect the whole kinematic chain (Meyer et al., 2015; Mills et al., 2013; Queen et al., 2011; Schmitt et al., 2015). It has been documented that the degree or direction of the adaptations depends on the primary involved joint (Schmitt et al., 2015). For instance, HOA patients decrease hip flexion, whereas KOA patients increase hip flexion during walking. Increased lateral trunk lean is found in both hip and knee OA patients (Mills et al., 2013). The effect of movement adaptations on joint moments – a parameter often used as an indirect proxy of joint loading – is less clear (Mills et al., 2013). Recently, using musculoskeletal modelling in combination with personalized gait kinematics, HOA patients were found to successfully decrease ipsi- and contralateral hip contact forces during level walking (Diamond et al., 2020; Meyer et al., 2018; Wesseling et al., 2018). In contrast, knee contact forces of the involved joint are not decreased in KOA patients, with medial compartment loading even being increased (Kumar et al., 2013; Meireles et al., 2016; Meireles et al., 2017a). Therefore, the reported kinematic strategies may be related to a conscious attempt to reduce loading on the involved joint during gait, potentially to avoid pain and discomfort.

Currently, the effect of the movement adaptations is merely studied in terms of the joint loading of the involved joint (either in terms joint moments or contact forces), thereby neglecting their effect on adjacent or contralateral joints. The high incidence of secondary OA might be related to loading transfers towards remote joints, thereby disturbing the local cartilage homeostasis (Kraus et al., 2013; Sayre et al., 2010; Shakoor et al., 2002; Shakoor et al., 2003; Shakoor et al., 2011). After long bouts of gait (45 mins), KOA patients presented increased knee joint loading compared to the initial level, however it remains unclear if these levels exceed control values (Gustafson et al., 2019).

In addition, it is less clear how joint loading distribution is affected in more demanding tasks, such as stair ambulation, where higher knee extensor muscle force generation and consequent higher compressive knee forces can be expected (Riener et al., 2002; Van Rossom et al., 2018). This might explain why KOA patients experience knee pain earlier during stair ambulation than during level walking (Hensor et al., 2015). Consequently, compensatory movement strategies have been described. Indeed, persons with KOA successfully limit the knee extensor muscle force demands during stair ambulation as obvious from the decreased sagittal plane knee moments, by increasing trunk and hip flexion but decreasing knee flexion and ankle dorsiflexion during stair ascent, as well as by decreasing hip and knee flexion during stair descent (Iijima et al., 2018; Riener et al., 2002). In patients with HOA, similar compensatory mechanisms such as decreased hip adduction and increased trunk lateroflexion have been described to decrease the demand on the weakened hip abductor muscles (Meyer et al., 2016). During stair descending, HOA patients increase hip flexion to reduce the demand on the hip extensors (Meyer et al., 2016). These movement adaptations might be associated with altered loading of various joints. Therefore, it is relevant to study the movement adaptations during stair ambulation and the associated joint loading of the involved and non-involved joint.

Therefore, this study aims to evaluate the biomechanical fingerprint of end-stage HOA and KOA patients during level walking and stair ambulation combining personalized movement characteristics with musculoskeletal modelling. We hypothesized that end-stage HOA and KOA patients will compensate by reducing loading of the involved joint compared to control subjects, thereby altering the loading of adjacent joints and joints of the contralateral leg and offsetting their mechanical environment, which is a recognized factor underlying the pathomechanics of secondary OA.

2. Methods

This is a controlled laboratory study (Level of Evidence: III). The local ethics committee approved the study protocol (S59857) and the participants provided written informed consent before the start of the study.

2.1. Study sample

Patients with end-stage HOA ($N = 20$) and KOA ($N = 18$) were compared to 12 healthy control subjects. Inclusion criteria were: age between 50 and 75 years; no pain or OA (healthy cohort) or painful unilateral hip or knee OA (patient cohort); BMI < 30 kg/m^2 (to avoid soft tissue artefacts of the pelvis markers); walking perimeter of >10 m including stair ambulation; no intra-articular steroid injection within 3 months pre-inclusion; no neurological or musculoskeletal disorders that could affect the movement pattern including joint replacement surgery.

2.2. Motion analysis

During a standard motion analysis, three-dimensional marker trajectories were recorded using a 13-camera VICON system (Vicon, Oxford Metrics, UK, 100 Hz) along with ground reaction forces using force plates embedded in the ground (AMTI, Watertown, USA, 1000 Hz). Markers were placed according to an adapted Plug-In-Gait marker set, in which the single marker on the thigh and shank was replaced by a rigid three-marker cluster and with additional anatomical markers on the sacrum, medial femur epicondyles and medial malleoli (Davis et al., 1991). For calibration purposes, a standing trial and five full-range knee flexion-extension cycles were recorded. Subsequently, participants were instructed to walk from a stand-still position at self-selected speed across the motion lab along a 10 m flat walkway. Thereafter, in separate trials, they were instructed to ascent and descent a four-step staircase at self-selected speed (step height = 0.16 m and tread length = 0.31 m). For both legs, three trials were retained for further processing.

2.3. Patient reported outcome measures

Patient reported outcome measures (PROMS) were assessed with the Knee Osteoarthritis Outcome Score (KOOS) and Hip Osteoarthritis Outcome Score (HOOS) after the motion capture session (Nilsdotter et al., 2003; Roos et al., 1998). All subitems (pain, symptoms, ADL function, Sport and recreation function and quality of life) of the KOOS and HOOS were individually included in the analysis.

2.4. Data analysis

A standard musculoskeletal modelling workflow was used in Opensim 3.3 (Delp et al., 2007a) to calculate joint contact forces. The generic gait2392 musculoskeletal model (Delp et al., 1990) with a 3 degrees of freedom knee joint was scaled to match the subject's anthropometrics and then modified to account for a more complex knee joint description with personalized knee joint axis orientation and position (Delp et al., 1990): To this end, the common axis of rotation was calculated based on the marker trajectories measured during the full range flexion-extension task using the SARA algorithm (Ehrig et al., 2007; Meireles et al., 2017b). Then, joint angles were calculated using the Kalman smoother algorithm (De Groote et al., 2008) and joint moments were calculated using an inverse dynamics approach (Delp et al., 2007b). Next, muscle forces that are required to balance the external joint moments, were calculated while minimizing the sum of the muscle activations squared, using static optimization (Anderson and Pandy, 2001). Lastly, joint contact forces that account for the forces associated with the ground contact as well as the muscle forces were calculated using the joint reaction analysis tool implemented in Opensim (Steele et al., 2012).

Stance phase was determined based on the contact times determined

based on a thresholding method (ground reaction force >20 N). Then, the magnitude of the first (FP) and second peak (SP) of the resultant hip and knee contact force was determined during the first and second half of the stance phase, coinciding with weight-acceptance (WA) and push-off (PO), respectively as well as the minimum hip and knee contact force during single leg support (midstance; MS). The contact forces were normalized to body weight to account for differences in body mass between subjects. At the determined timing of WP, MS and PO, the associated kinematics and kinetics were also determined. All variables were determined for individual trials and then averaged over the three trials, for the involved and the contralateral leg separately. For the healthy controls, an average value over both legs was calculated after statistical verification of joint loading symmetry.

2.5. Statistical analysis

To identify altered loading observed in HOA and KOA as either overloading compared to controls, joint loading at each timepoint (weight-acceptance, midstance and push-off) and for each joint (hip and knee of the involved and uninvolved joint) was compared between the healthy controls and each patient cohort separately using unpaired *t*-tests, after confirming normal distribution of the data ($p < 0.05$). Likewise, the kinematics and kinetics of these joints were compared between each patient group and the healthy control subjects. All tests were conducted in MATLAB (MATLAB 2018b, The Math Works, Inc. Natick, Massachusetts, USA).

3. Results

3.1. Demographics

Cohort demographics are shown in Table 1. Thirty-eight OA patients and 12 healthy controls participated in the present study, all presenting KL 4 (Kellgren and Lawrence, 1957). Eighteen patients with unilateral end-stage KOA (mean age: 65 ± 5.3 years, mean weight: 79.6 ± 8.9 kg, mean length: 1.74 ± 0.08 m, 11 females) and 20 patients with unilateral end-stage HOA (mean age: 63 ± 6.2 years, mean weight: 75.4 ± 11.6 kg, mean length: 1.75 ± 0.08 m, 11 females) were included and were compared to 12 healthy controls (mean age: 60 ± 7 years; mean weight: 74.3 ± 14.9 kg, mean length: 1.71 ± 0.10 m, 12 females). KOA patients were significantly older than controls. Both OA groups scored significantly worse than the controls on all subsets of the PROMS (Table 1). Both OA groups walked significantly slower compared to healthy controls and KOA patients were significantly slower during stair ascent compared to healthy controls.

3.2. Joint loading description

During walking and stair ambulation, the hip and knee contact force shows a double-peaked pattern, coinciding with the weight-acceptance and push-off phase (Fig. 1 and Table 2). During walking, the highest hip contact force is observed in the push-off phase, whereas during stair ambulation the highest hip contact force is observed in the weight-acceptance phase. However, in OA patients the highest hip contact force of the involved limb is shifted to the weight-acceptance phase for all movements. During walking and stair ascending, the highest knee contact force is observed in the weight-acceptance phase, whereas during stair descending the highest knee contact force is observed in the push-off phase and is not affected by OA.

3.3. Hip osteoarthritis patients

3.3.1. Involved hip joint

During walking and stair ascending, hip joint loading was significantly lower compared to the healthy controls, whereas it was not different during stair descending (Fig. 2**)**.

3.3.2. Ipsilateral knee

Likewise, loading of the ipsilateral knee was decreased during all movements (Fig. 2).

3.3.3. Contralateral hip

During walking, hip joint loading was significantly lower compared to the healthy controls. During stair ascending and descending, hip joint loading was not significantly altered compared to healthy controls (Fig. 2).

3.3.4. Contralateral knee

During stair descending, knee joint loading was significantly higher at weight-acceptance and midstance. During gait or stair ascending, no other significant differences in knee loading compared to healthy controls were observed (Fig. 2).

A detailed overview with respective *p*-values is presented in Table 2.

3.3.5. Compensatory kinematic strategy

Hip OA patients walked with significantly decreased ipsilateral hip extension and compensatory increased trunk and ipsilateral knee flexion. Similarly, during stair ambulation (i.e. both stair ascent and descent) trunk and contralateral knee flexion was significantly increased (Fig. 3**)**.

A detailed overview with respective *p*-values is presented in Table 3.

Table 1
Patient characteristics mean (SD).

	Controls	HOA	KOA	Main effect	C vs HOA	C vs. KOA
Mass (kg)	74 (14,5)	75 (11,5)	79,5 (8,5)	0,40	–	–
Height (m)	1,70 (0,05)	1,75 (0,05)	1,70 (0,05)	0,46	–	–
Age (years)	59,5 (7)	63 (6)	65 (5)	0,06	–	–
Gender (M/F)	6/6	12/9	11/7			
Gait Speed (m/s)						
Level walking	1.30 (0.16)	1.15 (0.15)	1.16 (0.13)	0.019*	0.016*	0.016*
Stair ascent	0.46 (0.06)	0.43 (0.08)	0.39 (0.03)	0.014*	0,35	<0.001*
Stair descent	0.52 (0.07)	0.5 (0.13)	0.46 (0.07)	0,32	–	–
Patient reported outcome measures						
Pain	95,35 (6,1)	50,85 (11,85)	56,75 (17)	<0.001*	<0.001*	<0.001*
Symptoms	96,9 (5,5)	52,25 (17,9)	51,75 (19,1)	<0.001*	<0.001*	<0.001*
Activities of daily life (ADL) function	98,7 (2,55)	56,4 (15,45)	57,9 (19,65)	<0.001*	<0.001*	<0.001*
Sport and recreation function	93,3 (9,2)	24,05 (23,2)	35,75 (23,6)	<0.001*	<0.001*	<0.001*
Quality of life	92,05 (9,8)	27,95 (15,85)	41,65 (16,85)	<0.001*	<0.001*	<0.001*

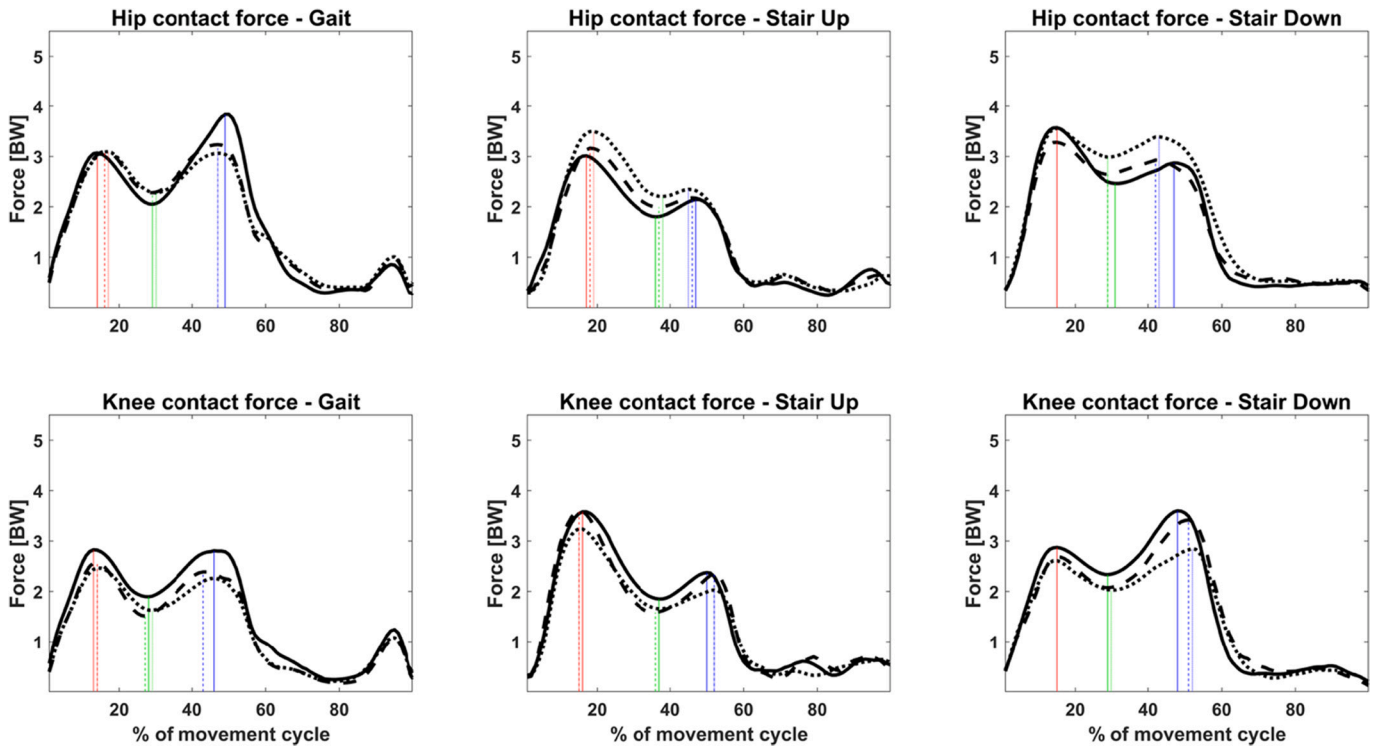
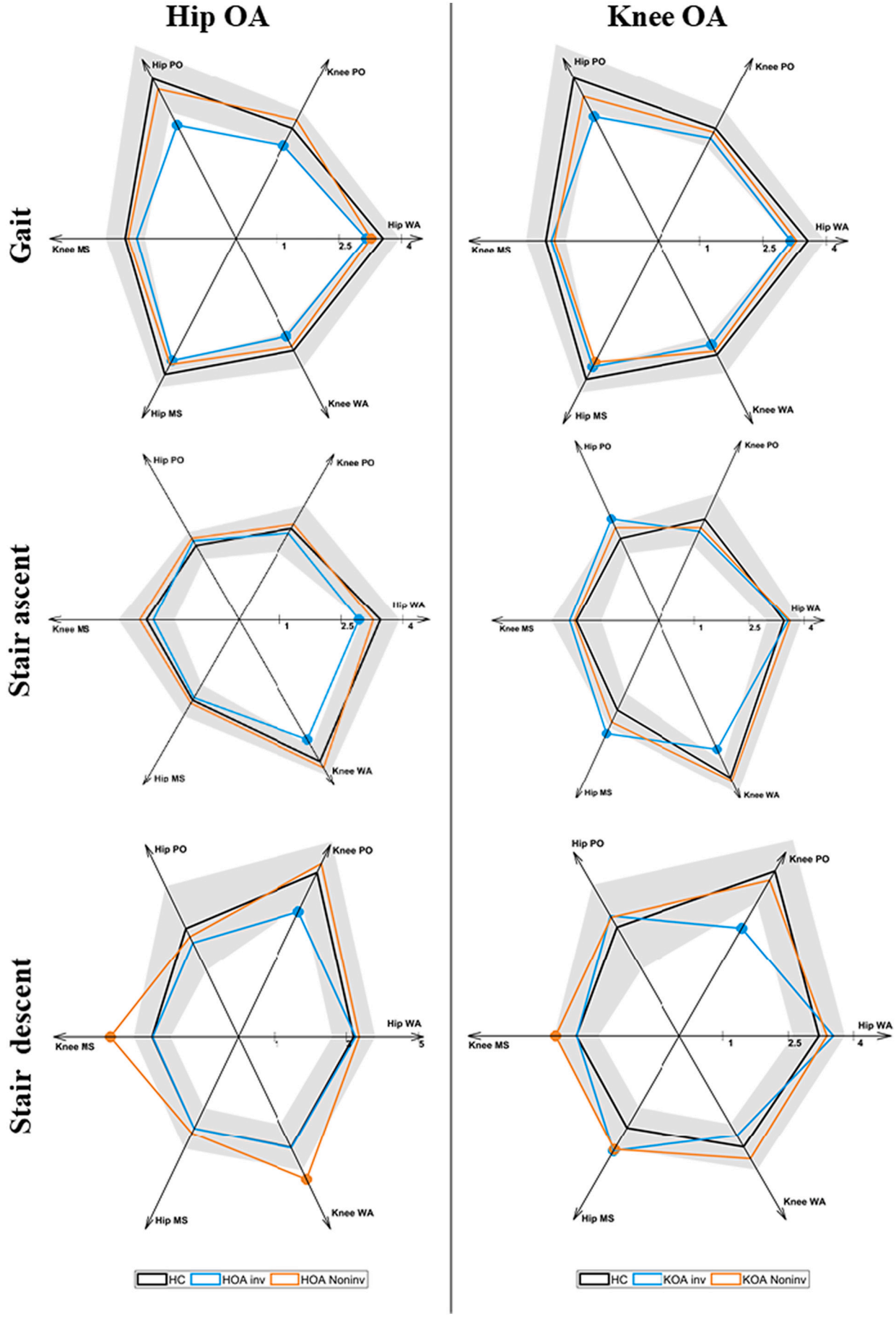


Fig. 1. Average hip and knee contact force patterns for the healthy controls (solid line), HOA (dashed line) and KOA (dotted line). The timing of the first (WA) and second peak (PO) of the resultant hip and knee contact force as well as the minimum hip and knee contact force during single leg support (MS) is indicated.

Table 2
Comparison Hip Loading and Knee Loading– Controls versus Involved and non-involved leg Knee OA and Hip OA patients at weight acceptance (WA), Mid Stance (MS) and Push Off (PO).

HIP LOADING*[BW]*	Control	KOA Inv		KOA non-inv		HOA Inv		HOA non-inv	
Gait									
WA	3.56 (0.41)	3.16 (0.25)	<0.001*	3.3 (0.52)	0.078	3.16 (0.45)	0.004*	3.27 (0.47)	0.039*
MS	3.42 (0.33)	3.11 (0.25)	0.002*	3 (0.45)	0.001*	3.07 (0.48)	0.008*	3.17 (0.51)	0.062
PO	4.01 (0.86)	3.05 (0.4)	<0.001*	3.55 (0.57)	0.059	2.84 (0.55)	<0.001*	3.74 (0.82)	0.308
Stair ascent									
WA	3.45 (0.48)	3.56 (0.64)	0.540	3.61 (0.61)	0.379	2.93 (0.41)	<0.001*	3.28 (0.46)	0.238
MS	2.26 (0.45)	2.85 (0.41)	<0.001*	2.56 (0.59)	0.086	2.19 (0.37)	0.593	2.34 (0.55)	0.582
PO	2.09 (0.38)	2.58 (0.35)	<0.001*	2.36 (0.4)	0.051	2.22 (0.39)	0.265	2.3 (0.49)	0.129
Stair descent									
WA	3.21 (0.61)	3.54 (0.73)	0.141	3.39 (0.68)	0.389	3.26 (0.52)	0.764	3.36 (0.65)	0.437
MS	2.44 (0.56)	3.06 (0.54)	0.002*	3.02 (0.93)	0.020*	2.44 (0.47)	0.997	2.57 (0.66)	0.490
PO	2.9 (1.15)	3.21 (0.79)	0.375	3.17 (0.88)	0.044	2.52 (0.57)	0.184	2.68 (0.83)	0.483
KNEE LOADING									
Gait									
WA	2.82 (0.45)	2.57 (0.31)	0.048*	2.73 (0.51)	0.547	2.47 (0.43)	0.012*	2.72 (0.56)	0.535
MS	2.66 (0.48)	2.53 (0.33)	0.338	2.46 (0.51)	0.201	2.38 (0.49)	0.067	2.59 (0.59)	0.665
PO	2.76 (0.45)	2.52 (0.45)	0.098	2.67 (0.44)	0.516	2.32 (0.31)	<0.001*	2.96 (0.82)	0.299
Stair ascent									
WA	3.97 (0.42)	3.25 (0.53)	<0.001*	4.05 (0.73)	0.682	3.36 (0.6)	<0.001*	4.15 (0.59)	0.256
MS	2.25 (0.7)	2.43 (0.6)	0.401	2.3 (0.73)	0.822	2.08 (0.74)	0.451	2.41 (0.93)	0.517
PO	2.58 (0.66)	2.27 (0.49)	0.126	2.37 (0.42)	0.310	2.44 (0.51)	0.463	2.7 (0.9)	0.624
Stair descent									
WA	2.96 (0.63)	2.65 (0.36)	0.096	3.27 (0.62)	0.134	2.97 (0.89)	0.956	3.81 (1.07)	0.002*
MS	2.38 (0.52)	2.37 (0.3)	0.956	2.86 (0.78)	0.027*	2.37 (0.86)	0.946	3.53 (1.59)	0.002*
PO	4.39 (0.84)	2.87 (0.63)	<0.001*	4.15 (0.74)	0.361	3.35 (0.85)	<0.001*	4.63 (1.08)	0.420

*Significantly different compared to healthy controls (HC), $p < 0.05$, WA = weight-acceptance, MS = Midstance, PO = push-off.



(caption on next page)

Fig. 2. Hip and Knee contact forces (BW) at weight-acceptance (WA), midstance (MS) and push-off (PO) for the healthy controls (black with shaded area indicating the SD in healthy controls), Involved leg (blue) and contralateral leg (orange) in both OA cohorts during walking, stair ascending and descending. A dot indicates a significantly altered contact force compared to the healthy controls ($p < 0.05$). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

3.4. Knee osteoarthritis patients

3.4.1. Involved knee joint

During all movements, knee joint loading was significantly lower compared to healthy controls (Fig. 2).

3.4.2. Ipsilateral hip

During walking, hip joint loading was significantly lower compared healthy controls, whereas during both stair ascending and descending hip joint loading was significantly higher compared to healthy controls (Fig. 2).

3.4.3. Contralateral knee

During stair ascending and descending, knee joint loading was significantly higher compared to healthy controls, whereas it was not different during walking (Fig. 2).

3.4.4. Contralateral hip

During walking hip contact force was significantly lower compared to healthy controls, whereas during stair ascending and descending hip contact force was significantly increased compared to healthy controls (Fig. 2**).**

A detailed overview with respective p-values is presented in Table 2.

3.4.5. Compensatory strategy

Knee OA patients walked with decreased ipsilateral hip extension. During stair ascent, trunk flexion and ipsilateral hip flexion were significantly increased, but ipsilateral knee flexion was significantly decreased compared to healthy controls. During stair descent, ipsilateral knee flexion was significantly decreased. Contralateral hip flexion was significantly increased (Fig. 3 – Pane B and Table 3).

A detailed overview of all kinematic and kinetic changes in individual HOA and KOA patients is presented in supplementary figs. 1 to 12.

4. Discussion

The current study identified the biomechanical fingerprint of patients with end-stage knee and hip OA during level walking and stair ambulation. Joint loading was evaluated in terms of hip and knee contact forces calculated using musculoskeletal modelling and patient-specific movement patterns. This analysis indicated that patients with hip or knee OA decreased loading at the involved joint. In addition, load transfer towards the adjacent joint in the involved leg and towards the contralateral leg were found in knee OA patients during stair ambulation. This analysis provides new biomechanical insights explaining the increased incidence of secondary OA in other joints in patients with hip and knee OA and the potential role of altered mechanical loading therein (Kraus et al., 2013; Sayre et al., 2010; Shakoor et al., 2002; Shakoor et al., 2003; Shakoor et al., 2011).

In line with our hypothesis, hip OA patients were found to decrease loading on the involved hip and ipsilateral knee during walking and stair ascending. This indicates that hip OA patients adopt a walking strategy that decreases the overall loading of the involved leg without shifting the load to the contralateral leg. Similar to previous studies, hip OA patients walked with increased trunk and knee flexion but decreased hip extension. As a result, the hip extension and adduction moment decreased, as well as the knee adduction moment, contributing to the decreased hip and knee contact force, especially in combination with a decreased walking speed (Fig. 1 and supplementary fig. 2) (Meyer et al., 2015). These adaptations were previously identified as a potential compensatory strategy to overcome muscle weakness of the hip abductor muscles found in hip OA patients while at the same time unloading the involved hip (Diamond et al., 2020; Meyer et al., 2018). During stair ascending, loading was only reduced at the instant of peak hip loading (i.e. weight-acceptance). Similar to walking, HOA patients increased trunk flexion and ipsilateral lumbar bending towards the supporting leg in order to decrease the hip abduction and knee adduction moment at weight-acceptance (Supplementary Fig. 3 and 4) (Meyer et al., 2016). During stair descent hip loading was not altered. Ipsilateral knee loading decreased prior to push-off, which might suggest that HOA patients try to decrease ipsilateral leg loading. However, the observed kinematic and kinetic changes were not sufficient to decrease hip loading (Supplementary Fig. 5 and 6). This hypothesis is further supported by the fact that contralateral knee loading was increased during weight-acceptance and midstance. This coincides with the ipsilateral push-off suggesting that hip OA patients try to take up more contralateral loading in order to unload the involved leg.

Knee OA patients showed different compensatory strategies compared to control subjects. Ipsilateral knee and bilateral hip loading was decreased during walking. This general trend towards unloading might be explained by the reduced walking speed in combination with a decreased hip extension and resulting hip extension moment (Supplementary fig. 7 and 8). This finding contrasts with previous studies that found similar or even increased contact forces in earlier stage KOA patients (Meireles et al., 2016; Meireles et al., 2017a). This increased loading in earlier stage KOA may contribute to disease progression to end-stage knee OA (Meireles et al., 2016; Meireles et al., 2017a). During stair ascending, KOA patients were found to unload the involved knee with a load shift towards the ipsilateral hip. Knee OA patients had increased trunk and hip flexion and decreased knee flexion, resulting in a decreased knee flexion moment and increased hip flexion moment (Supplementary fig. 9 and 10). During stair descent, KOA patients successfully decreased loading on the involved knee at the moment of highest knee loading (i.e. push-off). This resulted in increased loading of both hips and the contralateral knee. Decreased loading was obtained by decreasing knee flexion and the knee flexion moment (Supplementary fig. 11 and 12). These findings are in line with our hypothesis that KOA patients compensate in favor of their involved knee and thus shift the loading towards the other joints. This shift results in an altered mechanical environment in the neighboring joints (i.e. the ipsilateral hip joint and contralateral hip and knee) potentially disturbing the cartilage homeostasis locally which might explain the increased risk for secondary OA development (Kraus et al., 2013; Sayre et al., 2010; Shakoor et al., 2002; Shakoor et al., 2003; Shakoor et al., 2011).

Some limitations need to be considered when interpreting the results of this study. First, sample size of the patient cohort ($n = 38$) was limited and likely does not represent the heterogeneity in locomotor function in KOA and HOA patients. Second, all patients had end-stage OA waiting for joint replacement within a month after study participation. Although we assumed a no OA involvement in the joints of healthy control subjects and the non-involved leg, it is likely that some cartilage degeneration, without clinical symptoms may be present as no medical imaging data was available. However, no sign of joint stiffness during clinical investigation was present. Third, our findings might not directly be translated to patients with early OA. Last, the static optimization algorithm that was used in the current study to calculate the muscle forces minimized muscle activity and therefore did not account for subject-specific muscle co-contraction or muscle weakness that might be altered in this patient population, potentially underestimating the

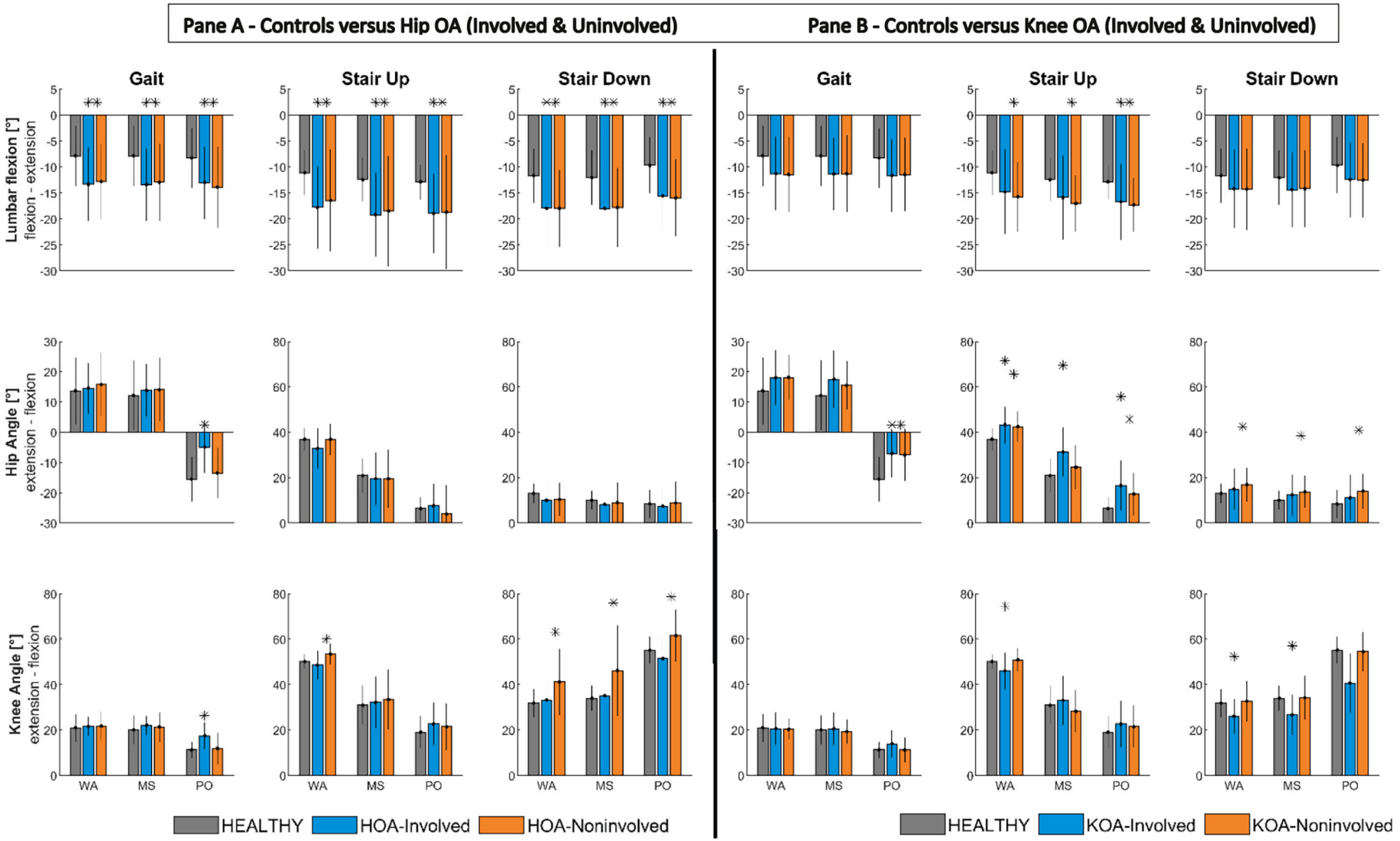


Fig. 3. Joint angles at weight-acceptance (WA), midstance (MS) and push-off (PO) between healthy controls (grey), involved leg (blue), non-involved leg (orange), for the Hip OA patients (Pane A) and Knee OA patients (Pane B) during walking, stair ascending and descending. * Indicates a significant difference compared to healthy controls ($p < 0.05$). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 3
Comparison Kinematics– Controls versus Involved and non-involved leg Knee OA and Hip OA patients at weight acceptance (WA), Mid Stance (MS) and Push Off (PO).

Lumbar Extension [°]	Control	KOA Inv		KOA non-inv		HOA Inv		HOA non-inv	
Gait									
WA	−7,84 (5,8)	−11,28 (7,02)	0,09	−11,47 (7,21)	0,081	−13,32 (7,01)	0,008*	−12,81 (7,25)	0,017*
MS]	−7,84 (5,78)	−11,32 (7)	0,089	−11,3 (7,38)	0,1	−13,38 (6,99)	0,007*	−12,95 (7,43)	0,015*
PO	−8,26 (5,7)	−11,65 (6,99)	0,10	−11,49 (7,05)	0,113	−13,02 (6,99)	0,018*	−13,87 (7,83)	0,01*
Stair ascent									
WA	−11,08 (4,25)	−14,78 (8,1)	0,071	−15,76 (6,67)	0,015*	−17,78 (7,9)	0,001*	−7,04 (42,09)	0,649
MS	−12,43 (4,23)	−15,9 (8,09)	0,088	−17,02 (5,42)	0,008*	−19,2 (8,14)	0,001*	−8,98 (42,72)	0,702
PO	−12,87 (3,34)	−16,69 (7,39)	0,035*	−17,34 (5,17)	0,003*	−18,94 (7,7)	0,001*	−9,15 (43,06)	0,682
Stair descent									
WA	−11,63 (5,23)	−14,21 (7,54)	0,22	−14,23 (7,85)	0,221	−17,95 (7,27)	0,002*	−17,96 (7,4)	0,002*
MS	−12,04 (5,3)	−14,38 (7,17)	0,26	−14,17 (7,45)	0,303	−18 (7,13)	0,003*	−17,81 (7,65)	0,006*
PO	−9,68 (5,41)	−12,4 (7,22)	0,193	−12,54 (7,17)	0,164	−15,58 (6,7)	0,003*	−15,95 (7,4)	0,003*

Hip Flexion [°]	Control	KOA Inv		KOA non-inv		HOA Inv		HOA non-inv	
Gait									
WA	13,65 (10,98)	18,08 (9,12)	0,18	18,13 (7,34)	0,144	14,56 (8,44)	0,763	15,83 (10,5)	0,511
MS	12,19 (11,46)	17,52 (9,46)	0,119	15,53 (7,95)	0,298	13,83 (8,63)	0,602	14,05 (10,47)	0,582
PO	−15,53 (7,32)	−6,99 (7,91)	0,001*	−7,5 (8,57)	0,002*	−4,92 (8,55)	<0.001*	−13,5 (8,33)	0,401
Stair ascent									
WA	36,93 (4,83)	43,3 (8,05)	0,004*	42,5 (6,63)	0,006*	32,84 (8,82)	0,06	33,94 (14,71)	0,364
MS	20,91 (7,36)	31,24 (10,92)	0,001*	24,6 (9,57)	0,204	19,55 (11,67)	0,645	17,47 (15,44)	0,349
PO	6,39 (5,17)	16,57 (11,17)	<0.001*	12,8 (9,42)	0,012*	7,62 (9,79)	0,603	2,5 (14,06)	0,224
Stair descent									
WA	13,02 (4,41)	14,92 (9,03)	0,392	16,91 (7,33)	0,045*	10,08 (7,59)	0,124	10,42 (7,32)	0,16
MS	10,03 (4,19)	12,39 (9,05)	0,28	13,72 (7,07)	0,048*	8,17 (8,57)	0,36	9,01 (8,9)	0,624
PO	8,43 (6,27)	11,17 (9,96)	0,304	14,03 (7,71)	0,017*	7,4 (8,42)	0,648	8,9 (9,46)	0,849

Knee Flexion [°]	HC	KOA Inv	KOA non-inv			HOA Inv		HOA non-inv	
Gait									
WA	20,91 (6,09)	20,64 (6,96)	0,90	20,39 (4,61)	0,76	21,58 (4,17)	0,68	21,73 (6,43)	0,67
MS	20,08 (6,28)	20,49 (7,09)	0,84	19,31 (5,41)	0,68	22,11 (4,14)	0,23	21,33 (6,45)	0,52
PO	11,3 (3,5)	13,9 (5,84)	0,09	11,21 (5,33)	0,95	17,47 (5,98)	<0.001*	11,81 (6,84)	0,76
Stair ascent									
WA	50,18 (3,11)	45,93 (8,18)	0,03*	50,84 (5,08)	0,63	48,61 (6,2)	0,29	54,29 (5,73)	0,01*
MS	30,95 (8,47)	33,04 (10,84)	0,5	28,28 (9,36)	0,39	32,21 (11,41)	0,68	35,32 (15,28)	0,25
PO	18,99 (7,14)	22,61 (10,31)	0,20	21,45 (9,28)	0,38	22,68 (9,33)	0,15	23,67 (13,71)	0,16
Stair descent									
WA	31,82 (6,28)	26,01 (7,52)	0,01*	32,76 (8,82)	0,7	33,23 (8,58)	0,54	41,21 (14,63)	0,01*
MS	34,02 (5,42)	26,72 (8,7)	<0.001*	34,24 (9,75)	0,93	35,09 (10,05)	0,66	46,19 (19,92)	0,01*
PO	55,13 (5,91)	40,59 (13,1)	<0.001*	54,57 (8,62)	0,81	51,5 (9,61)	0,14	61,55 (11,38)	0,02*

contact forces. Therefore, deviations in joint kinematics and external forces explain primarily the observed deviations in joint loading. However, it was recently reported that hip contact forces were still decreased in hip OA patients during walking (Diamond et al., 2020), even while accounting for the increased co-contraction.

5. Conclusion

This study identified the biomechanical fingerprint of two cohorts of OA patients during walking and stair ambulation. Knee OA and Hip OA patients had different compensatory strategies compared to controls both during level walking and during stair ambulation. Movement adaptations that resulted in decreased ipsilateral joint loading were confirmed in both OA cohorts: Hip OA patients decreased ipsilateral leg loading without largely affecting the loading on the contralateral leg. Similarly, knee OA patients decreased loading on the involved leg during walking. During more demanding task such as stair ambulation a load shift towards the ipsilateral hip and the contralateral limb was confirmed. The identified movement patterns should be monitored and if present, corrected as part of a physical retraining program. This may prevent secondary changes in the mechanical environment of the adjacent joints (Kraus et al., 2013; Sayre et al., 2010; Shakoor et al., 2002; Shakoor et al., 2003; Shakoor et al., 2011) and impact the increased risk for secondary OA development.

Funding

This research was funded by Research Foundation Flanders (FWO) grant number T004716N.

Author contributions

SVR, IJ, AT, BV, KC contributed to conception and design. SVR, JE, MW, RVDS, JT, JM, JB, KC contributed to collection and processing of data. SVR, JE, MW, KC, AT, BV, IJ contributed to analysis and interpretation. SVR, IJ and MW contributed to drafting of the article. RVDS, JT, JM, JB, JE, KC contributed to provision of patients. AT, BV, IJ contributed to obtaining funding. All the authors contributed to the final approval of the article.

Declaration of Competing Interest

All authors hereby declare that there are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clinbiomech.2022.105858.

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