Obesity is associated with higher absolute tibiofemoral contact and muscle forces during gait with and without knee osteoarthritis

Graeme T. Harding, Michael J. Dunbar, Cheryl L. Hubley-Kozey, William D. Stanish, Janie L. Astephen Wilson

Abstract

Background

Obesity is an important risk factor for knee osteoarthritis initiation and progression. However, it is unclear how obesity may directly affect the mechanical loading environment of the knee joint, initiating or progressing joint degeneration. The objective of this study was to investigate the interacting role of obesity and moderate knee osteoarthritis presence on tibiofemoral contact forces and muscle forces within the knee joint during walking gait.

Methods

Three-dimensional gait analysis was performed on 80 asymptomatic participants and 115 individuals diagnosed with moderate knee osteoarthritis. Each group was divided into three body mass index categories: healthy weight (body mass index < 25), overweight (25 ≤ body mass index ≤ 30), and obese (body mass index > 30). Tibiofemoral anterior–posterior shear and compressive forces, as well as quadriceps, hamstrings and gastrocnemius muscle forces, were estimated based on a sagittal plane contact force model. Peak contact and muscle forces during gait were compared between groups, as well as the interaction between disease presence and body mass index category, using a two-factor analysis of variance.

Findings

There were significant osteoarthritis effects in peak shear, gastrocnemius and quadriceps forces only when they were normalized to body mass, and there were significant BMI effects in peak shear, compression, gastrocnemius and hamstrings forces only in absolute, non-normalized forces. There was a significant interaction effect in peak quadriceps muscle forces, with higher forces in overweight and obese groups compared to asymptomatic healthy weight participants.

Interpretation

Body mass index was associated with higher absolute tibiofemoral compression and shear forces as well as posterior muscle forces during gait, regardless of moderate osteoarthritis presence or absence. The differences found may contribute to accelerated joint damage with obesity, but with the osteoarthritic knees less able to accommodate the high loads.

Keywords

Obesity; Knee osteoarthritis; Gait analysis; Tibiofemoral contact forces; Muscle

Introduction

The knee is the joint most commonly affected by osteoarthritis (OA) (Hendren and Beeson, 2009), and its initiation and progression are complex and not fully understood (Guilak, 2011). Biomechanical factors, particularly joint loading during gait, have been implicated in both initiation and progression (Radin et al., 1978, Miyazaki et al., 2002, Griffin and Guilak, 2005 and Bennell et al., 2011). Obesity is one of the most potent risk factors for both the development and progression of knee OA (Murphy and Helmick, 2012), with biomechanical factors theorized to be involved in its pathogenic role (Guilak, 2011).

The contribution of obesity to the OA process at the knee joint has traditionally been hypothesized to be due to the higher compressive forces on the joint during dynamic activity due to the increased bodyweight (Griffin and Guilak, 2005). Indeed, obesity has been associated with alterations of dynamic knee loading during gait (Browning et al., 2006), and with increased knee adduction moments in particular (Segal et al., 2009). A previous study by our research group, however, has shown that regardless of OA presence, a pattern of sustained net external knee adduction moment during the stance phase of gait, but not the overall magnitude of the moment, was observed with obesity, and obesity was additionally associated with less net external extension moment in late stance and a more sustained flexion moment during stance (Harding et al., 2012).

The loading alterations with obesity characterized by previous studies have most often been demonstrated using net resultant knee joint moments obtained by the method of inverse dynamics (Costigan et al., 1992). While net resultant joint moments are a reflection of the total loading exposure of the knee joint during dynamic activity, it is ultimately the tibiofemoral joint contact forces which are placed on the articulating joint surfaces during walking and presumed to be involved in the mechanistic breakdown of the joint tissues as a precursor to structural OA development and/or progression. In-vivo tibiofemoral joint contact forces are not directly measureable during dynamic tasks (unless by instrumented total knee replacement (Kim et al., 2009)), and therefore must be estimated mathematically. A few previous studies have investigated the effect of weight loss on tibiofemoral contact forces in obese subjects with knee OA (Messier et al., 2005, Aaboe et al., 2011 and Messier et al., 2011). These studies have shown that weight loss can be effective in reducing compressive joint loading, and that for each unit of weight lost, the joint compressive force was reduced by multiple units (Messier et al., 2005).

We know that there are alterations to the dynamic loading environment during walking occur with the presence of knee OA (Kaufman et al., 2001, Baliunas et al., 2002, Hurwitz et al., 2002, Astephen and Deluzio, 2004, Andriacchi and Mundermann, 2006, Astephen et al., 2008a and Astephen et al., 2008b). Our previous work has also shown an interaction between obesity and knee OA disease presence on joint mechanics alterations during gait (Harding et al., 2012). Specifically, this previous research has shown significant interactions between the two in the range of flexion/extension from late stance through to mid-swing phase, as well as in the peak to peak range of the flexion moment (early stance flexion moment to late stance extension moment) during stance phase captured using inverse dynamics (Harding et al., 2012). The range of the sagittal plane moment and angle were significantly reduced with increasing BMI category in the presence of moderate knee OA, however not in asymptomatic controls. While informative, a tibiofemoral joint contact force model that would account for these sagittal plane interactions would provide additional insight into the interacting role of obesity and knee OA presence on joint contact dynamics. The purpose of this study was to examine the separate and interacting roles

of moderate knee OA and overweight/obesity on tibiofemoral joint contact forces during gait. We hypothesized that tibiofemoral contact forces during gait would be significantly increased with obesity, and that there would be interactions between BMI category and presence of moderate knee OA, such that contact force increases with obesity would be lower in the OA group due to previously observed sagittal plane compensations (Harding et al., 2012).

Methods

Participants

We recruited 204 participants in total, including 115 asymptomatic adult participants (> 35 years) and 89 diagnosed with moderate, medial compartment knee OA. The asymptomatic participants were recruited through institutional postings and advertisements in public locations at the university and hospitals, and had no history of knee pain or surgery to their lower extremities. OA participants were recruited from the Orthopaedic Assessment Clinic at the Halifax Infirmary and the Orthopedic and Sports Medicine Clinic of Nova Scotia. A participating orthopaedic surgeon made a diagnosis of moderate knee OA based on radiographic evidence and the results of a clinical physical exam, according to the American College of Rheumatology criteria (Altman et al., 1986), and the stipulation that they were not yet candidates for total knee replacement surgery, similar to our previous studies (Landry et al., 2007 and Astephen et al., 2008a). Similar to our previous studies (Landry et al., 2007 and Astephen et al., 2008a), our goal was to capture any deviations in joint mechanics relatively early in the OA process, and so our osteoarthritis population was functionally capable of daily activities, being able to jog 5 m, walk stairs reciprocally, and walk a city block without a walking aid. Informed consent was obtained for each subject before laboratory testing began, in accordance with the ethics review board of the institution. According to definitions described by Health Canada, asymptomatic and moderate knee OA participant groups were each divided into three categories based on their BMI: healthy weight (BMI < 25), overweight ($25 \leq BMI \leq 30$), and obese (BMI > 30) (Health-Reports, 2006).

Gait analysis

Each participant visited our laboratory once for gait testing, following a previously defined protocol (Landry et al., 2007). Demographic and anthropometric data was collected including age, height, weight, and thigh and calf circumference. Three-dimensional motion of either the OA affected or randomly chosen (for asymptomatic) lower limb during self-selected speed walking was recorded at 100 Hz using 2 Optotrak™ 3020 motion capture sensors (Northern Digital Inc., Waterloo, ON) during each walking trial, and a minimum of 5 walking trials were conducted for each participant. Ground reaction forces were collected at 2000 Hz using an AMTI force platform (Advanced Mechanical Technology Inc., Watertown, MA). Triads of infrared light emitting diodes were placed on the pelvis, thigh, shank and foot in order to calculate kinematics and kinetics at the ankle, knee, and hip joints, although this particular study focused on the knee joint. Individual diodes were placed on the shoulder and the greater trochanter, lateral epicondyle, and lateral malleolus. Virtual markers were identified during quiet standing on the right and left anterior superior iliac spines, medial epicondyle, fibular head, tibial tubercle, medial malleolus, second metatarsal, and heel (Landry et al., 2007). Positive anatomical axes in the tibia were defined medial to lateral through the malleoli (sagittal axis, y), the cross product of this axis (y) with one directed from the lateral malleoli to the fibular head (frontal axis, x), and the cross product of these two (x,y) axes (transverse, z). Positive anatomical axes in the femur were defined medial to lateral through the epicondyles (sagittal axis, y), the cross product of this axis (y) with one directed from the lateral

epicondyle to the greater trochanter (frontal axis, x), and the cross product of these two (x,y) axes (transverse, z). A least-squares optimization routine, minimizing the error between the position of each rigid body and the experimental marker data was used to determine the relative position and orientation of the 4 rigid bodies (Challis, 1995). Angles and moments at each joint were defined according to an anatomically based joint coordinate system (Grood and Suntay, 1983). An inverse dynamics method was implemented through custom Matlab software (The MathWorks, Natick, MA, USA). Three dimensional position data, ground reaction forces, and limb inertial properties were used to calculate net external inter-segmental joint reaction moments (Costigan et al., 1992). Waveforms were time normalized to a single gait cycle.

A tibiofemoral contact force model developed and described by DeVita and Hortobagyi was used to estimate compressive and shear tibiofemoral joint contact forces at the knee (DeVita and Hortobagyi, 2001). The contact force model is a two-dimensional sagittal plane torque-driven model, as it determines the tibiofemoral contact forces based on the resultant joint torques at the ankle, knee, and hip in the sagittal plane from inverse dynamics. Briefly, the net external inter-segmental joint reaction moments were calculated from the inverse dynamics procedure described above and averaged over the five walking trials to produce one set of net external moments for each participant. The forces in the three largest force producing muscle groups in the lower limb, the quadriceps, hamstrings, and gastrocnemii, were calculated. Gastrocnemius forces were estimated based on the net resultant plantar flexor moment at the ankle and the lever arm of the Achilles tendon, which varied depending on the flexion angle of the ankle (Klein et al., 1996). The plantar flexor moment was divided by the Achilles tendon moment arm to produce the triceps surae force (Klein et al., 1996). This force was then multiplied by a ratio of 0.319, which is an estimate of the gastrocnemius physiological cross-sectional area (PCSA) to the overall PCSA of the triceps surae muscles from the literature (Yamaguchi et al., 1990). The force in the hamstrings was calculated in a similar fashion based on the net extensor moment at the hip joint. The proportion of the net extensor moment generated by the hamstrings was determined based on the ratio of the hamstring PCSA to the combined PCSA of the hamstrings and gluteus maximus, as well as on the ratio of the hamstrings to gluteus maximus moment arms (DeVita and Hortobagyi, 2001). Given that the net flexion/extension moment at the knee is assumed to be a result of the hamstrings, gastrocnemius, and quadriceps muscles acting on their respective moment arms, the quadriceps force was then calculated with known hamstrings and gastrocnemius forces, and moment arms (Herzog and Read, 1993), and the net resultant flexion/extension moment at the knee from inverse dynamics. The three predicted muscle forces, and the net joint reaction forces from inverse dynamics were then applied to the tibia, with the predicted forces occurring along their respective lines of action. Compressive forces were positive when directed into the tibia, and shear forces were positive when applied anteriorly. Muscle forces (quadriceps, hamstrings and gastrocnemius) and knee joint compressive and anterior–posterior shear forces were calculated both in absolute units (in Newtons), and normalized units (expressed as multiples of the participant's body mass).

Statistical methods

Two peak values from early and late stance were extracted from the compressive and shear knee joint forces and a single peak value was extracted from the muscle force curves (Table 2). Differences in these forces were compared between groups using a two-factor ANOVA and post-hoc Tukey tests with moderate knee OA disease presence and BMI category as factors. Statistical significance was set at 0.05 for all comparisons.

Results

Mean group anthropometrics, demographics, and stride characteristics for the study population divided into asymptomatic and OA participant groups and into BMI categories are summarized in Table 1 and Table 2. The asymptomatic group contained 44 healthy weight participants, 32 overweight, and 13 obese. The moderate knee OA group contained 13 healthy weight participants, 45 overweight, and 57 obese (Table 3). OA participants were older ($P < 0.001$), heavier ($P = 0.006$), had higher BMI ($P = 0.008$), larger thigh ($P = 0.009$) and calf circumferences ($P = 0.003$), and the OA group had a higher percentage of male participants than the asymptomatic group. This is consistent with previous moderate OA groups for our laboratory (Landry et al., 2007 and Astephen et al., 2008a). OA participants walked significantly slower (1.24 m/s compared to 1.39 m/s), with longer stance time and percentage ($P < 0.001$, $P = 0.006$ respectively). The portion of the gait cycle spent in the stance phase increased progressively from healthy weight to obese. Walking speed, stride length, and stance time were not significantly different between BMI categories ($P > 0.05$).

Table 1.

Mean (SD) demographics, anthropometrics, and stride characteristics of asymptomatic and moderate knee OA subject groups (top) and BMI category groups (bottom). Two-factor ANOVA *P*-values for OA and BMI main effects are included.

	Asymptomatic	Moderate OA	OA effect *P*-value
N	89	115	–
Sex (F/M)	56/33	41/74	–
Age (years)	48.1 (9.0)	57.5 (9.0)	<0.001*
Height (m)	1.7 (0.10)	1.7 (0.1)	0.14
Weight (kg)	74.6 (14.4)	91.3 (17.9)	0.006*
BMI (kg/m2)	25.8 (4.0)	30.7 (5.0)	0.008*
Thigh circ. (m)	0.53 (0.05)	0.54 (0.05)	0.009*
Calf circ. (m)	0.37 (0.04)	0.39 (0.04)	0.006*
Stride characteristics			
Speed (m/sec)	1.39 (0.17)	1.24 (0.20)	<0.001*
Stride length (m)	1.46 (0.13)	1.38 (0.16)	0.019
Stance time (sec)	0.66 (0.06)	0.73 (0.08)	<0.001*

	Asymptomatic	Moderate OA	OA effect *P*-value
Stance (%)	62.6 (1.5)	64.0 (1.9)	0.006*

	HW	OV	OB	BMI effect *P*-value	Multiple comparisons *P*-values		
					HW vs. OV	HW vs. OB	OV vs. OB
N	57	77	70	–			
Sex (F/M)	36/21	31/46	31/39	0.23	–	–	–
Age (years)	50.8 (9.2)	53.8 (10.2)	55.2 (10.6)	0.81	–	–	–
Height (m)	1.7 (0.1)	1.7 (0.1)	1.7 (0.1)	0.54	–	–	–
Weight (kg)	65.5 (8.10)	82.0 (9.01)	101.2 (16.3)	<0.001*	<0.001*	<0.001*	<0.001*
BMI (kg/m2)	22.8 (1.4)	27.6 (1.4)	34.4 (3.8)	<0.001*	<0.001*	<0.001*	<0.001*
Thigh circ. (m)	0.50 (0.04)	0.53 (0.04)	0.57 (0.05)	<0.001*	<0.001*	<0.001*	0.004
Calf circ. (m)	0.35 (0.02)	0.38 (0.03)	0.40 (0.04)	<0.001*	<0.001*	<0.001*	<0.001*
Stride characteristics							
Speed (m/sec)	1.37 (0.19)	1.33 (0.19)	1.22 (0.21)	0.13	–	–	–
Stride length (m)	1.45 (0.13)	1.45 (0.14)	1.35 (0.16)	0.07	–	–	–
Stance time (sec)	0.66 (0.06)	0.70 (0.07)	0.73 (0.08)	0.085	–	–	–

	HW	OV	OB	BMI effect *P*-value	Multiple comparisons *P*-values		
					HW vs. OV	HW vs. OB	OV vs. OB
Stance (%)	62.3 (1.6)	63.2 (1.4)	64.6 (1.8)	<0.001*	0.021	<0.001	0.003

*Refers to statistically significant difference between asymptomatic and moderate OA group at $\alpha = 0.05$ level of significance.

Table 2.

Group mean (SD) demographics, anthropometrics, and stride characteristics of asymptomatic and moderate OA subjects divided by BMI into healthy weight (HW), overweight (OW), and obese (OB) categories.

	Asymptomatic			Moderate OA		
	HW	OV	OB	HW	OV	OB
N	44	32	13	13	45	57
Sex (F/M)	30/14	18/14	8/5	6/7	12/33	23/34
Age (years)	49.0 (9.0)	47.6 (8.7)	46.5 (10.3)	56.9 (7.2)	58.2 (8.8)	57.2 (9.7)
Height (m)	1.7 (0.1)	1.7 (0.1)	1.7 (0.1)	1.7 (0.1)	1.7 (0.1)	1.7 (0.1)
Weight (kg)	64.2 (7.5)	80.9 (9.5)	94.6 (12.0)	70.1 (8.5)	83.0 (8.6)	102.7 (16.9)
BMI (kg/m2)	22.6 (1.5)	27.3 (1.5)	32.7 (3.2)	23.5 (0.80)	27.7 (1.2)	34.8 (3.8)
Thigh circ. (m)	0.50 (0.04)	0.72 (0.97)	0.57 (0.04)	0.48 (0.04)	0.52 (0.04)	0.56 (0.05)
Calf circ. (m)	0.35 (0.02)	0.38 (0.03)	0.40 (0.06)	0.35 (0.02)	0.37 (0.02)	0.40 (0.03)
Stride characteristics						

	Asymptomatic			Moderate OA		
	HW	OV	OB	HW	OV	OB
Speed (m/sec)	1.38 (0.18)	1.40 (0.16)	1.35 (0.16)	1.32 (0.19)	1.27 (0.19)	1.19 (0.21)
Stride length (m)	1.44 (0.12)	1.49 (0.13)	1.45 (0.13)	1.46 (0.17)	1.42 (0.15)	1.34 (0.16)
Stance time (sec)	0.65 (0.06)	0.67 (0.07)	0.68 (0.05)	0.70 (0.05)	0.71 (0.08)	0.74 (0.08)
Stance (%)	62.2 (1.6)	62.8 (1.1)	63.5 (1.1)	62.4 (1.7)	63.5 (1.6)	64.8 (1.9)

Table 3.

ANOVA *P*-values and OA group means for normalized (x body mass, top panel) and non-normalized (N, bottom panel) tibiofemoral contact forces and muscle forces. Only statistically significant OA effects exist for normalized forces, and so group mean (SD) and absolute and percent difference between the OA and asymptomatic groups are provided.

Force (% body mass, N/kg)	Peak	OA effect	BMI effect	Interaction effect	Asymptomatic mean (SD)	OA mean (SD)	Difference
Shear	1	0.01*	0.43	0.22	0.46 (0.20)	0.37 (0.17)	−0.09 (19.6%)
	2	0.07	0.13	0.41	0.43 (0.11)	0.40 (0.11)	−0.03 (7.0%)
Compressive	1	0.20	0.37	0.23	1.99 (0.68)	1.81 (0.63)	−0.17 (9.0%)
	2	0.06	0.21	0.14	1.61 (0.35)	1.52(0.42)	−0.09 (5.6%)
Gastrocnemius	1	0.003*	0.18	0.52	1.23 (0.21)	1.12 (0.26)	−0.11 (8.9%)
Hamstrings	1	0.21	0.50	0.23	1.01 (0.37)	1.10 (0.40)	0.09 (−8.9%)
Quadriceps	1	0.001*	0.40	0.08	1.24 (0.43)	0.93 (0.44)	−0.31 (25%)

Force (N)	Peak	OA effect	BMI effect	Interaction effect
Shear	1	0.17	0.004*	0.16
	2	0.92	<0.001*	0.23
Compressive	1	0.95	0.001*	0.12
	2	0.99	<0.001*	0.12
Gastrocnemius	1	0.48	<0.001*	0.94
Hamstrings	1	0.29	<0.001*	0.08
Quadriceps	1	0.23	0.03	0.05*

*Refers to statistically significant difference between groups at $\alpha = 0.05$ level of significance.

Body mass normalized tibiofemoral and muscle forces

There were no significant BMI category or interaction effects of peak values of body mass normalized AP shear, compressive, and muscular forces (in N/kg) (Table 3). However, there were significant OA effects in some body mass normalized forces. The OA group had significantly lower first peak normalized shear forces (19.6% lower), lower peak normalized quadriceps forces (25%), and lower peak normalized gastrocnemius forces (8.9%) (Fig. 1, Table 3).

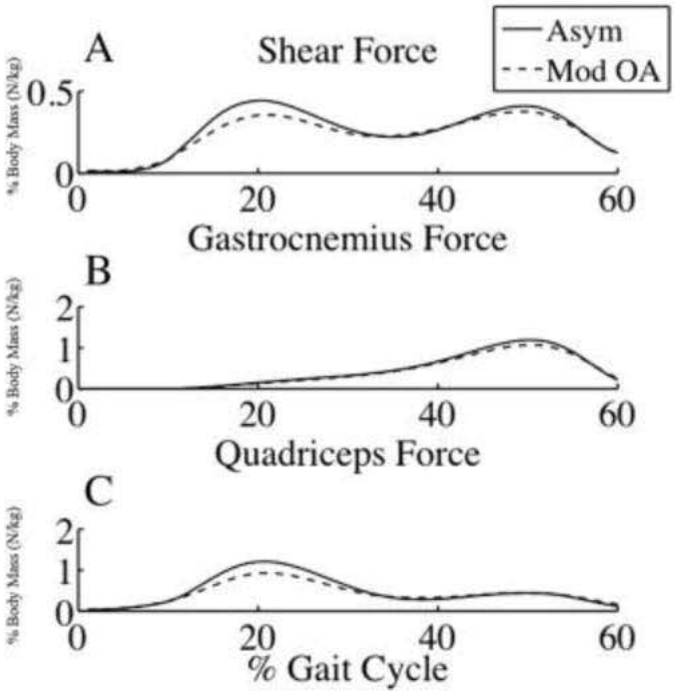

Fig. 1.

Mean anterior–posterior shear contact force (A), quadriceps force (B), and gastrocnemius force (C) waveforms normalized to body mass for OA and asymptomatic subject groups. The OA group demonstrated statistically significant lower body mass normalized first peak shear forces, as well as lower normalized peak gastrocnemius and lower normalized peak quadriceps forces than the asymptomatic control group ($P < 0.05$).

3.2. Absolute tibiofemoral and muscle forces

There were a number of statistically significant BMI category effects in absolute forces for both peaks of shear and compressive forces, and for all peaks of all muscle groups (in N) (Table 4). The second peak of the shear and compressive forces increased progressively through the BMI categories from healthy weight through obese (Fig. 2). Both overweight and obese groups had higher first peak shear and compressive contact forces than healthy weight, but with no statistically significant difference between overweight and obese groups in these first peak values (Table 4, Fig. 2). Peak hamstring and peak gastrocnemius forces also increased progressively through the categories (Fig. 3). In contrast to the body mass normalized results, there were no statistically significant OA main effects in absolute tibiofemoral contact forces and muscle forces (Table 3). A significant interaction between moderate knee OA disease presence and BMI category was found in the absolute peak quadriceps force (Fig. 4). The asymptomatic group showed higher absolute peak quadriceps force in the overweight and obese categories compared to healthy weight, whereas there were no differences in peak quadriceps forces between BMI categories within the OA group.

Table 4.

Peak values of absolute tibiofemoral contact forces and muscle forces for healthy weight (HW), overweight (OW) and obese (OB) subject groups with multiple comparison *P*-values and percent mean difference between BMI category groups.

Force (N)	Peak	HW	OW	OB	BMI *P*-value	Multiple comparison *P*-values			
						HW vs. OW	HW vs. OB	OV vs. OB	
Shear		1	273.5 (137.7)	355.4 (150.2)	382.1 (187.5)	0.004	0.02 (30%)	0.005 (39.7%)	>0.05 (7.5%)
		2	272.5 (82.3)	356.1 (89.1)	420.5 (135.8)	<0.001	0.002 (30.7%)	<0.0001 (54.3)	0.02 (18.1%)
Compressive		1	1227.3 (521.3)	1586.0 (543.7)	1812.9 (703.8)	0.001	0.009 (29.2%)	0.0005 (47.7%)	>0.05 (14.3%)
		2	980.6 (257.9)	1264.1 (321.5)	1528.2 (516.2)	<0.001	0.002 (28.9%)	<0.0001 (55.8%)	0.02 (20.9%)

Force (N)	Peak	HW	OW	OB	BMI *P*-value	Multiple comparison *P*-values		
						HW vs. OW	HW vs. OB	OV vs. OB
Gastrocnemius	1	769.4 (168.1)	978.6 (227.4)	1107.9 (324.2)	<0.001	<0.0001 (27.2%)	<0.0001 (44.0%)	0.02 (13.2%)
Hamstrings	1	645.4 (309.7)	876.4 (266.2)	1096.5 (506.2)	<0.001	0.01 (35.8%)	0.0003 (69.9%)	0.22 (25.1%)
Quadriceps	1	758.9 (341.8)	891.2 (403.9)	935.4 (409.3)	0.03	>0.05 (17.4%)	0.03 (23.2%)	>0.05 (5%)

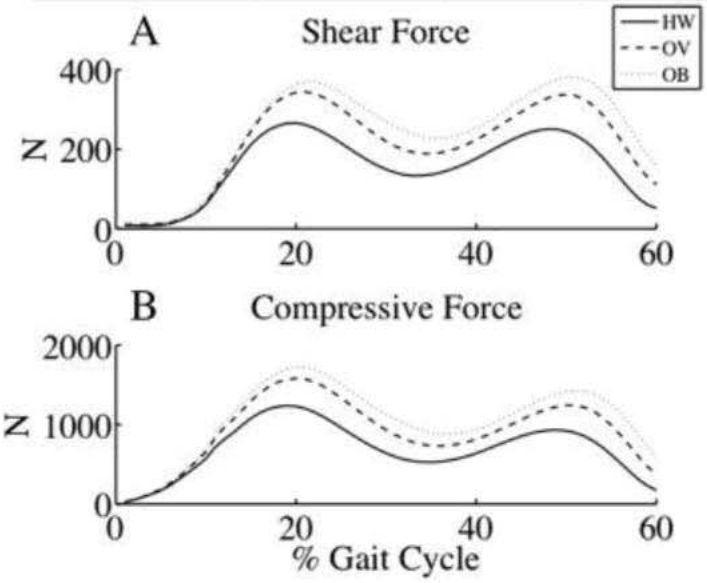

Fig. 2.

Mean absolute tibiofemoral anterior–posterior shear force (A) and compressive force (B) during the stance phase of gait for healthy weight (HW), overweight (OW), and obese (OB) subject groups. Second peak absolute shear and compression forces were progressively higher with increasing BMI category ($P < 0.05$). First peak shear and compression forces were significantly higher for both the overweight and obese groups compared to the healthy weight group ($P < 0.05$).

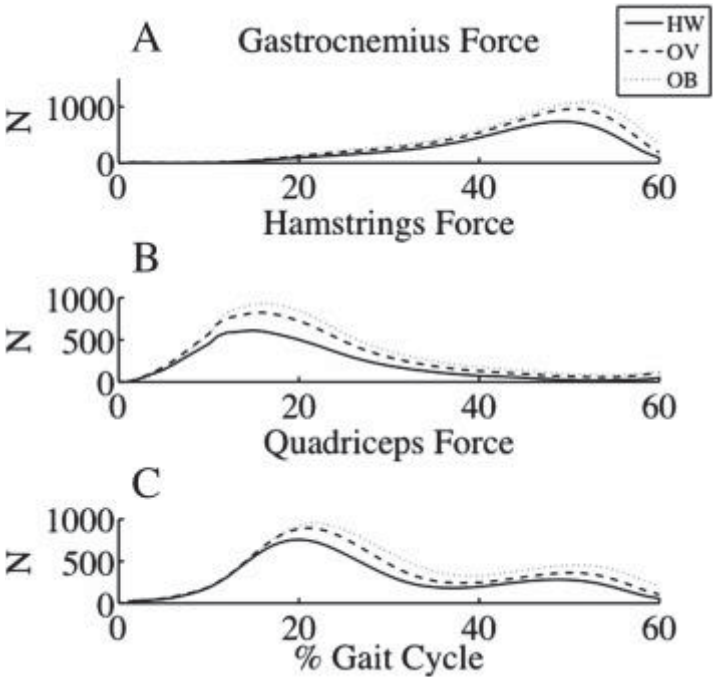

Fig. 3.

Mean absolute gastrocnemius (A), hamstrings (B), and quadriceps (C) force waveforms during the stance phase of gait for healthy weight (HW), overweight (OW), and obese (OB) subject groups. Peak absolute gastrocnemius and hamstring forces were progressively higher with increasing BMI category ($P < 0.05$). There was a statistically significant interaction between BMI category and OA presence in the absolute peak value of the quadriceps muscle force during stance, which is further interpreted in Fig. 4.

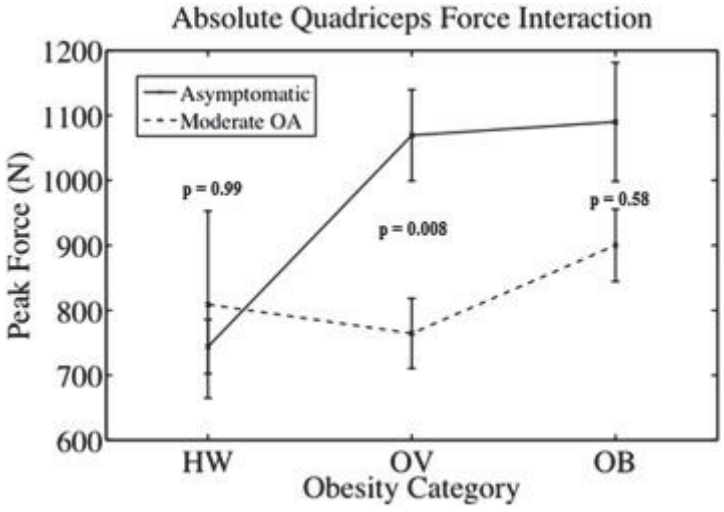

Fig. 4.

Interaction between moderate knee osteoarthritis disease presence and BMI category (healthy weight—HW, overweight—OW, and obese—OB) in peak absolute quadriceps force during the stance phase of gait. *P*-values represent the pairwise comparisons between OA and asymptomatic for each BMI category. Not shown on figure were significant pairwise differences between HW and OV in the

asymptomatic group ($P = 0.004$) and between HW and OB in the asymptomatic group ($P = 0.05$). There were no statistically significant pairwise differences between BMI categories within the moderate OA group, or between the OV and OB groups within the asymptomatic group ($P > 0.05$).

4. Discussion

While there is clear evidence that overweight and obese individuals are at a substantially higher risk of knee OA development and progression, the mechanistic pathway for accelerated OA development and progression is not well described. We know from previous research that overweight and obese individuals adopt characteristic changes in their gait kinematics and net resultant kinetics compared to those of healthy weight (Messier, 1994, DeVita and Hortobagyi, 2003, Browning et al., 2006, Browning et al., 2009 and Harding et al., 2012), however it is unclear how these changes in gait patterns (stride characteristics, joint angles and net resultant moments) consequently contribute to changes in the forces between articulating surfaces of the knee joint during walking. Understanding how these tibiofemoral contact forces change with increasing BMI category both in the presence and absence of symptomatic knee OA is important for examining the potential role of obesity in OA development and progression pathways.

Peak quadriceps forces (occurring in early to mid-stance), peak gastrocnemius forces (occurring in late stance) and peak early to mid-stance shear forces, all normalized to body mass, were lower in the OA group compared to the asymptomatic group. Because these are normalized values, they reflect differences in the force environment of the joint that are independent of body mass differences between individuals and groups. This normalized difference between groups may reflect a deliberate compensatory change in neuromuscular function to reduce joint forces. The lower shear force in early stance with OA is directly related to the lower quadriceps force, as the quadriceps would tend to contribute to anterior shear force generation during the early stance phase of the gait cycle. Force generation and muscular activity in the gastrocnemii occur later in stance phase, and contribute to posterior shear in the sagittal plane knee model. However, no significant difference was found in the peak value of anterior–posterior shear force occurring later in stance phase between participants with and without moderate knee OA, despite the lower normalized peak gastrocnemius force in the OA group. As can be seen in Fig. 1, the mean shear force was lower with OA in late stance, but not statistically significant, likely due to the smaller size of the difference in late stance compared to early stance. In late stance, the knee is approaching full extension, with the moment arm of the gastrocnemius, the primary force producing muscle, approaching its minimum value. It is therefore unsurprising that the differences in shear force due to the gastrocnemius force are less pronounced in later stance. The lower gastrocnemius force in late stance is reflective of less propulsion, and, similar to the lower peak quadriceps and shear forces, associated with an approximately 0.15 m/s lower self-selected walking speed in the OA group. Speed was not however included as a covariate in the analysis, as reduction in walking velocity was assumed to be intrinsic to the knee OA disease process (Astephen Wilson, 2012).

Individuals with knee OA have frequently been shown to have higher peak knee adduction moments, which has been associated with increased medial compartment loading relative to lateral (Vincent et al., 2012). It was therefore interesting that peak normalized compressive contact forces were not significantly different between the OA and asymptomatic groups in the current study in either early or late stance. It was also interesting that there were no statistically significant OA presence effects in any

absolute muscle or contact force peak values. This may suggest that the presence of symptomatic knee OA may lead to compensatory changes (such as reduced walking velocity) that may contribute to maintaining lower joint and muscle forces. Despite this, OA joints may be less able to accommodate the same level of joint contact forces as asymptomatic joints without further deterioration, and it has been theorized that physiological loads applied to already damaged OA joints may lead to OA progression (Andriacchi et al., 2004 and Andriacchi et al., 2009).

There were no significant differences observed between BMI categories in normalized tibiofemoral contact forces or muscle forces, and so there were no observed differences in tibiofemoral or muscle contact forces above those which could be attributed to the higher body masses of the overweight and obese cohorts in the current study. This finding supports the argument that obesity may be associated with behavioral gait alterations to maintain overall knee loading and musculoskeletal health (DeVita and Hortobagyi, 2003). There were, however, significant differences in all absolute forces (measured in Newtons) associated with BMI category, with late stance peak and compressive forces all increasing progressively through the BMI categories, and early stance peak shear and compressive forces higher with overweight and obesity compared to healthy weight A similar effect, a significant association between weight loss and decreased compressive force has been previously shown (Messier et al., 2005, Aaboe et al., 2011 and Messier et al., 2011). Aaboe et al. showed that when obese participants with knee OA reduced their BMI from a mean of 36.9 to a mean of 31.9, peak compressive tibiofemoral joint contact force was reduced by 7%, along with lower axial impulse and decreased peak values of the external knee adduction moment (Aaboe et al., 2011). For each Newton of weight loss, compressive loading was reduced by 2.2 N. Similarly, Messier et al. have demonstrated a direct relationship between reduced body weight and reduced compressive tibiofemoral joint contact forces, with each pound of weight loss corresponding to a reduction of approximately 4 pounds of joint load (Messier et al., 2005 and Messier et al., 2011). Results of the current study further emphasize this direct relationship in an observational, cross-sectional study, although our results show more of a one-to-one relationship between body mass and associated increase in compressive tibiofemoral contact forces. The percent differences in tibiofemoral contact forces between our overweight and obese groups compared to the healthy weight group were substantial (all ≥ 29% and up to 55% between healthy weight and obese in late stance peak compressive force). However, these percent differences in contact forces were incidentally very similar to the percent differences in body mass between the groups (overweight group mass was 25.2% higher than healthy weight, obese group was 54.5% higher), suggesting more of a 1:1 cross-sectional ratio of body mass to tibiofemoral contact force based on our results. The higher compressive *and* shear loading associated with higher BMI category may be contributing to the accelerated development and progression of knee OA with higher BMI. Previous animal models have shown OA-like induced joint changes with direct application of increased non-physiologic compressive forces in rats (Roemhildt et al., 2013). As well, excessive shear force has been associated with biochemical changes to joint tissues that can initiate or progress degenerative pathways (Sakai et al., 1999 and Heiner and Martin, 2004).

The current study also demonstrated that estimated absolute forces generated by the three major force generating muscle groups acting at the knee joint were all also significantly higher with increasing BMI category, with a significant interaction effect of the quadriceps such that peak quadriceps force was higher with overweight and obesity compared to healthy weight only in the asymptomatic group. The higher hamstrings and quadriceps muscle forces both occur during the early stance phase, and perhaps

increase in an effort to provide additional joint stability during loading response onto a single limb in response to excess body weight. Higher gastrocnemius forces in later stance may reflect the need for increased propulsive force due to additional body mass as well. In our applied contact force model, lower quadriceps force generation should reduce anterior–posterior shear force, and therefore the relatively lower quadriceps force with higher BMI in OA subjects compared to asymptomatic may represent a compensation strategy for participants with OA who are overweight or obese. Higher quadriceps forces with higher BMI within the asymptomatic group would create a higher net internal extension moment in early stance. Interestingly, inverse dynamics results did not show an increase in early stance flexion moment with obesity (Harding et al., 2012). However, the inverse dynamics results do not take into account any potential muscular co-contraction around the joint, which could explain the discrepancy. This is a limitation of the current model because overall quadriceps muscle activity in vastus lateralis and the duration of rectus femoris activity have both been demonstrated to be higher on average in individuals with moderate knee OA (Hubley-Kozey et al., 2006), as well as hamstring co-activity (Hubley-Kozey et al., 2009). It was interesting that this was the only evidence of an interaction between knee OA disease presence and BMI category because the mechanical pathways of OA initiation and progression are theorized to be relatively distinct events because non-physiological loading would be required to precipitate OA in a healthy joint, whereas physiological loading may be sufficient to further progress an already diseased joint (Andriacchi et al., 2004 and Andriacchi et al., 2009).

As the average BMI of our obese group was 34.4, lower than in previous obesity-related studies (Hortobagyi et al., 1985, DeVita and Hortobagyi, 2003 and Browning and Kram, 2007), it is possible that our results reflect only those within this BMI range. This may also explain discrepancies between our results and those of previous study. Similar to other models in the OA literature that have been used to examine knee joint contact forces during gait, such as electromyography-driven models (Lloyd and Besier, 2003) and those that aim to capture the distribution of forces throughout the joint (Winby et al., 2009), the model used in this study relies on a number of assumptions which are not easily validated. This model was two-dimensional and torque-driven, and therefore relied on the net resultant joint moments calculated at the ankle, knee, and hip joints. It also applies tibiofemoral contact forces at a single point, and our statistical model captured only peak contact and muscle force differences between groups. However, the current model provides an improved estimation of the mechanical environment of the knee joint than net resultant moments alone, and our results point to a slightly more complex relationship of the loading exposure of the knee joint with obesity, as a risk factor for knee OA development and progression. Our mean normalized compressive contact force of 1.99 × BW for asymptomatic subjects was within the range of calculated forces of 1.7 × BW and 2.4 × BW during level walking with a similar model applied in previous study (D'Lima et al., 2012). Finally, it should be noted that our moderate OA group consisted of more male participants than females, which is atypical for an osteoarthritis population. Given that there are established biomechanical differences during gait between men and women with knee osteoarthritis that may affect tibiofemoral contact forces (Mckean et al., 2007 and Astephen Wilson et al., 2015), this demographic difference in our study should be noted.

Conclusion

These results support a clear association between higher body mass index and tibiofemoral compressive and shear contact forces, as well as muscle forces, during the stance phase of gait. The magnitude of contact force increase with body mass index category was consistent with the change in body mass between the groups, and there were no observed differences in forces above those which could be

attributed to higher body masses. Despite this, the differences found may contribute to accelerated joint damage and the higher rate of development and progression of knee osteoarthritis with obesity. Knee osteoarthritis was significantly associated with lower shear, quadriceps and gastrocnemii forces during gait, which may be a reflection of a lower walking speed compensation in an effort to keep contact forces low. Compressive tibiofemoral contact forces during stance were similar between the osteoarthritis and asymptomatic control groups, however the osteoarthritis knee joints may be less able to accommodate the same level of forces without further damage.

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References

Aaboe, J., Bliddal, H., Messier, S.P., Alkjaer, T., Henriksen, M., 2011. Effects of an intensive weight loss program on knee joint loading in obese adults with knee osteoarthritis.

Osteoarthr. Cartil. 19, 822–828. Altman, R., Asch, D., Block, G., Bole, G., Borenstein, K., Brandt, K., al., e., 1986. The American

College of Rheumatology Criteria for the Classification and Reporting of Osteoarthritis of the Knee. Arthritis Rheum. 29, 1039–1049.

Andriacchi, T.P., Mundermann, A., 2006. The role of ambulatory mechanics in the initiation and progression of knee osteoarthritis. Curr. Opin. Rheumatol. 18, 514–518.

Andriacchi, T.P., Mundermann, A., Smith, R.L., Alexander, E.J., Dyrby, C.O., Koo, S., 2004. A framework for the in vivo pathomechanics of osteoarthritis at the knee. Ann. Biomed.

Eng. 32, 447–457.

Andriacchi, T.P., Koo, S., Scanlan, S.F., 2009. Gait mechanics influence healthy cartilage morphology and osteoarthritis of the knee. J. Bone Joint Surg. Am. 91A, 95–101.

Astephen Wilson, J.L., 2012. Challenges in dealing with walking speed in knee osteoarthritis gait analyses. Clin. Biomech. 27, 210–212.

Astephen Wilson, J.L., Dunbar, M.J., Hubley-Kozey, C.L., 2015. Knee joint biomechanics and neuromuscular control during gait before and after total knee arthroplasty are sex-specific. J. Arthroplasty 30, 118–125.

Astephen, J.L., Deluzio, K.J., 2004. A multivariate gait data analysis technique: application to knee osteoarthritis. Proc. Inst. Mech. Eng. H 218, 271–279.

Astephen, J.L., Deluzio, K.J., Caldwell, G.E., Dunbar, M.J., 2008a. Biomechanical changes at the hip, knee, and ankle joints during gait are associated with knee osteoarthritis severity. J. Orthop. Res. 26, 332–341.

Astephen, J.L., Deluzio, K.J., Caldwell, G.E., Dunbar, M.J., Hubley-Kozey, C.L., 2008b. Gait and neuromuscular pattern changes are associated with differences in knee osteoarthritis severity levels. J. Biomech. 41, 868–876.

Baliunas, A.J., Hurwitz, D.E., Ryals, A.B., Karrar, A., Case, J.P., Block, J.A., Andriacchi, T.P., 2002. Increased knee joint loads during walking are present in subjects with knee osteoarthritis. Osteoarthr. Cartil. 10, 573–579.

Bennell, K.L., Bowles, K.A., Wang, Y., Cicuttini, F., Davies-Tuck, M., Hinman, R.S., 2011.

Higher dynamic medial knee load predicts greater cartilage loss over 12 months in medial knee osteoarthritis. Ann. Rheum. Dis. 70, 1770–1774.

Browning, R.C., Kram, R., 2007. Effects of obesity on the biomechanics of walking at different speeds. Med. Sci. Sports Exerc. 39, 1632–1641.

Browning, R.C., Baker, E.A., Herron, J.A., Kram, R., 2006. Effects of obesity and sex on the energetic cost and preferred speed of walking. J. Appl. Physiol. 100, 390–398.

Browning, R.C., McGowan, C.P., Kram, R., 2009. Obesity does not increase external mechanical work per kilogram body mass during walking. J. Biomech. 42, 2273–2278.

Challis, J.H., 1995. A procedure for determining rigid-body transformation parameters. J. Biomech. 28, 733–737.

Costigan, P.A., Wyss, U.P., Deluzio, K.J., Li, J., 1992. A semi-automatic 3D knee motion assessment system. Med. Biol. Eng. Comput. 343–350 (May).

DeVita, P., Hortobagyi, T., 2001. Functional knee brace alters predicted knee muscle and joint forces in people with ACL reconstruction during walking. J. Appl. Biomech. 17, 297–311.

DeVita, P., Hortobagyi, T., 2003. Obesity is not associated with increased knee joint torque and power during level walking. J. Biomech. 36, 1355–1362.

D'Lima, D.D., Fregly, B.J., Patil, S., Steklov, N., Colwell Jr., C.W., 2012. Knee joint forces: prediction, measurement, and significance. Proc. Inst. Mech. Eng. H 226, 95–102.

Griffin, T.M., Guilak, F., 2005. The role of mechanical loading in the onset and progression of osteoarthritis. Exerc. Sport Sci. Rev. 33, 195–200.

Grood, E.S., Suntay, W.J., 1983. A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. J. Biomech. Eng. 105, 136–144.

Guilak, F., 2011. Biomechanical factors in osteoarthritis. Best Pract. Res. Clin. Rheumatol. 25, 815–823.

Harding, G.T., Hubley-Kozey, C.L., Dunbar, M.J., Stanish, W.D., Astephen Wilson, J.L., 2012.

Body mass index affects knee joint mechanics during gait differently with and without moderate knee osteoarthritis. Osteoarthr. Cartil. 20, 1234–1242. Health-Reports, 2006. Statistics Canada, Volume 17, Number 3.

Heiner, A.D., Martin, J.A., 2004. Cartilage responses to a novel triaxial mechanostimulatory culture system. J. Biomech. 37, 689–695.

Hendren, L., Beeson, P., 2009. A review of the differences between normal and osteoarthritis articular cartilage in human knee and ankle joints. Foot (Edinb.) 19, 171–176.

Herzog, W., Read, L.J., 1993. Lines of action and moment arms of the major force-carrying structures crossing the human knee joint. J. Anat. 182 (Pt 2), 213–230.

Hortobagyi, T., Herring, C., Pories, W.J., Rider, P., DeVita, P., 1985. Massive weight loss induced mechanical plasticity in obese gait. J. Appl. Physiol. 111, 1391–1399.

Hubley-Kozey, C.L., Deluzio, K.J., Landry, S.C., McNutt, J.S., Stanish, W.D., 2006. Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. J. Electromyogr. Kinesiol. 16, 365–378.

Hubley-Kozey, C.L., Hill, N.A., Rutherford, D.J., Dunbar, M.J., Stanish,W.D., 2009. Co-activation differences in lower limb muscles between asymptomatic controls and those with varying degrees of knee osteoarthritis during walking. Clin. Biochem. 24, 407–414.

Hurwitz, D.E., Ryals, A.B., Case, J.P., Block, J.A., Andriacchi, T.P., 2002. The knee adduction moment during gait in subjects with knee osteoarthritis is more closely correlated with static alignment than radiographic disease severity, toe out angle and pain. J. Orthop. Res. 20, 101–107.

Kaufman, K.R., Hughes, C., Morrey, B.F., Morrey, M., An, K., 2001. Gait characteristics of patients with knee osteoarthritis. J. Biomech. 34, 907–915.

Kim, H.J., Fernandez, J.W., Akbarshahi, M., Walter, J.P., Fregly, B.J., Pandy, M.G., 2009.

Evaluation of predicted knee-joint muscle forces during gait using an instrumented knee implant. J. Orthop. Res. 27, 1326–1331.

Klein, P., Mattys, S., Rooze, M., 1996. Moment arm length variations of selected muscles acting on talocrural and subtalar joints during movement: an in vitro study. J. Biomech. 29, 21–30.

Landry, S.C., Mckean, K.A., Hubley-Kozey, C.L., Stanish, W.D., Deluzio, K.J., 2007. Knee biomechanics of moderate OA patients measured during gait at a self-selected and fast walking speed. J. Biomech. 40, 1754–1761.

Lloyd, D.G., Besier, T.F., 2003. An EMG-driven musculoskeletal model to estimate muscle forces and knee joint moments in vivo. J. Biomech. 36, 765–776.

Mckean, K.A., Landry, S.C., Hubley-Kozey, C.L., Dunbar, M.J., Stanish, W.D., Deluzio, K.J., 2007. Gender differences exist in osteoarthritic gait. Clin. Biomech. 22, 400–409.

Messier, S.P., 1994. Osteoarthritis of the knee and associated factors of age and obesity:effects on gait. Med. Sci. Sports Exerc. 26, 1446–1452.

Messier, S.P., Gutekunst, D.J., Davis, C., DeVita, P., 2005. Weight loss reduces knee-joint loads in overweight and obese older adults with knee osteoarthritis. Arthritis Rheum. 52, 2026–2032.

Messier, S.P., Legault, C., Loeser, R.F., Van Arsdale, S.J., Davis, C., Ettinger, W.H., DeVita, P., 2011. Does high weight loss in older adults with knee osteoarthritis affect bone-onbone joint loads and muscle forces during walking? Osteoarthr. Cartil. 19, 272–280.

Miyazaki, T., Wada, M., Kawahara, H., Sato, M., Baba, H., Shimada, S., 2002. Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. Ann. Rheum. Dis. 61, 617–622.

Murphy, L., Helmick, C.G., 2012. The impact of osteoarthritis in the United States: a population-health perspective. Am. J. Nurs. 112, S13–S19.

Radin, E.L., Ehrlich, M.G., Chernack, R., Abernathy, P., Paul, I.L., Rose, R.M., 1978. Effect of repetitive impulsive loading on the knee joints of rabbits. Clin. Orthop. Relat. Res. 131, 288–293.

Roemhildt, M.L., Beynnon, B.D., Gauthier, A.E., Gardner-Morse, M., Ertem, F., Badger, G.J., 2013. Chronic in vivo load alteration induces degenerative changes in the rat tibiofemoral joint. Osteoarthr. Cartil. 21, 346–357.

Sakai, K., Mohtai, M., Shida, J., Harimaya, K., Benvenuti, S., Brandi, M.L., Kukita, T., Iwamoto,

Y., 1999. Fluid shear stress increases interleukin-11 expression in human osteo blast like cells: its role in osteoclast induction. J. Bone Miner. Res. Off. J. Am. Soc. Bone Miner. Res. 14, 2089–2098.

Segal, N.A., Yack, H.J., Khole, P., 2009. Weight, rather than obesity distribution, explains peak external knee adduction moment during level gait. Am. J. Phys. Med. Rehabil. 88, 180–188.

Vincent, K.R., Conrad, B.P., Fregly, B.J., Vincent, H.K., 2012. The pathophysiology of osteoarthritis: a mechanical perspective on the knee joint. PM.R. 4, S3–S9.

Winby, C.R., Lloyd, D.G., Besier, T.F., Kirk, T.B., 2009. Muscle and external load contribution to knee joint contact loads during normal gait. J. Biomech. 42, 2294–2300.

Yamaguchi, G.T., sawa, A., moran, G., Fessler, M., Winters, J.M., Winters, J.M., 1990. A survery of human musculotendon actuator parameters. In: Woo, S.L.-Y. (Ed.), Multiple Muscle Systems: Biomechanics and Movement Organization. Springer-Verlag, New York, pp. 717–774.