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

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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.

1. 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; 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

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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; 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; 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).

2. Methods

2.1. 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; Astephen et al., 2008a). Similar to our previous studies (Landry et al., 2007; 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 ≤ BMI ≤ 30), and obese (BMI > 30) (Health-Reports, 2006).

2.2. 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 intersegmental 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 intersegmental 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)