Obesity is associated with prolonged activity of the quadriceps and gastrocnemii during gait

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Purpose

To examine the effect of obesity and its potential interaction with knee OA presence on the electromyography patterns of the major knee joint periarticular muscles during walking. Scope: One hundred and eighteen asymptomatic adults and 177 adults with moderate knee osteoarthritis were subdivided into categories of healthy weight (n = 77; 20 kg/m2 < BMI < 25 kg/m2), overweight (n = 117; 25 kg/m2 6 BMI < 30 kg/m2), and obese (n = 101; BMIP30 kg/m2 based on their body mass index (BMI). All individuals underwent a three-dimensional gait analysis. Surface electromyograms from the lateral and medial gastrocnemii, lateral and medial hamstrings, vastus lateralis, vastus medialis, and rectus femoris were recorded during self-selected speed walking. Principal component analysis was used to extract major features of amplitude and temporal pattern variability from the electromyograms of each muscle group (gastrocnemii, quadriceps, hamstrings separately). Analysis of variance models tested for main BMI category effects and interaction effects for these features (a = 0.05). Statistically significant BMI category (i.e. obesity) effects were found for features that described more prolonged activations of the gastrocnemii and quadriceps muscles during the stance phase of gait with obesity (P < 0.05). Conclusions: Obesity was associated with prolonged activation of quadriceps and gastrocnemii, which can result in prolonged knee joint contact loading, and thereby may contribute to the predisposition of knee OA development and progression in obese individuals.

Introduction

Osteoarthritis (OA) of the knee is common (Buckwalter et al.,2001) and frequently associated with disability and symptoms (Felson et al., 1987). Numerous risk factors are associated with the initiation and progression of knee OA, among which obesity is known as the most important modifiable risk factor (Felson et al., 1988; Davis et al., 1990; Hochberg et al., 1995; Grotle et al., 2008); however, its role in the initiation and progression of the disease is unclear (Zhang, 2010). Obesity affects the knee joint through mechanical (Hortobagyi et al., 2011; Messier et al., 2011) and systemic risk factors (Pottie et al., 2006; Lee and Kean, 2012). Mechanically, it may result in excessive joint loading (Miyazaki et al., 2002; Browning and Kram, 2007; Hortobagyi et al., 2011), usually inferred from observed differences in gait metrics, such as peak knee adduction moment (Gushue et al., 2005; Browning and Kram, 2007). However, in our previous work we have shown that the patterns of knee joint loading also differ in the presence of obesity, with less midstance unloading of the knee adduction moment during the stance phase of gait with increasing BMI (Harding et al., 2012).

Muscles are primary contributors to the joint loading (Herzog et al., 2003). Therefore, an altered mechanical environment of the knee in the presence of obesity and knee OA could be influenced by changed activation of the major muscles surrounding the joint. Several studies reported altered muscular activity of the knee periarticular muscles in the presence of knee OA. Increased co-contraction (Childs et al., 2004; Hortobagyi et al., 2005; Rudolph et al., 2007; Hubley-Kozey et al., 2009), increased EMG amplitude (Hubley-Kozey et al., 2006, 2009; Rutherford et al., 2011), and prolonged activation of the lateral muscles (Childs et al., 2004; Hubley-Kozey et al., 2006, 2009; Rutherford et al., 2011) are alterations that have been observed in the presence of knee OA, regardless of the disease severity, knee

alignment or joint laxity (Mills et al., 2013). These altered patterns of higher than typical activation of lateral muscles have been proposed to be an attempt to reduce medial joint loading and increase knee joint active stiffness during the stance phase (Childs et al., 2004, Hubley-Kozey et al., 2006, Hubley-Kozey et al., 2009, Rutherford et al., 2011, Mills et al., 2013). However, a recent simulation study of prolonged lateral muscle activation suggested that this compensatory mechanism increases lateral tibiofemoral contact forces, while preventing the medial forces from increasing, meaning that increased lateral co-contraction may improve joint stability, but may also subsequently increase total joint contact force (Brandon et al., 2014).

Previous studies have also examined the potential influence of obesity on activation patterns of lower limb muscles in healthy children (Hills and Parker, 1993, Blakemore et al., 2013) and in female adolescents in particular (de Carvalho et al., 2012), all reporting no difference between obese and non-obese individuals. Others have shown increased and prolonged activity of knee musculature during gait while carrying loads (Ghori and Luckwill, 1985, Simpson et al., 2011). This implies that the nature of obesity, as an undesirable multifaceted condition, is more complicated than solely increased mass, and requires more investigation to be appropriately understood.

The analysis of the electromyography data in the above studies focused on subjectively chosen discrete variables from time-varying EMG signals, such as integrated EMG (Simpson et al., 2011) or burst duration (Blakemore et al., 2013). Principal component analysis (PCA) is a multivariate analysis technique that retains the temporal and amplitude information of the electromyography signal during gait, and has shown utility in understanding temporal pattern differences (Hubley-Kozey et al., 2006, Hubley-Kozey et al., 2009, Rutherford et al., 2011), and has been used to understand the interacting influence of obesity and moderate knee OA presence on knee joint level kinematic and kinetic patterns during gait (Harding et al., 2012). The role of obesity and its interaction with knee OA on the activity of knee periarticular muscles will provide information to enhance these previously observed biomechanical alterations (Harding et al., 2012). The aim of this study was to examine the effect of obesity and its potential interaction with knee OA presence on the electromyography patterns of the major knee joint periarticular muscles during walking gait using a cross-sectional model. Based on our previous investigation of knee joint biomechanics during gait with obesity that showed a lack of mid-stance unloading with higher BMI (Harding et al., 2012), as well as the previous literature that has linked even early stages of knee osteoarthritis to co-contraction and higher mid-stance activity (Hubley-Kozey et al., 2006), we hypothesize that higher BMI category will be significantly associated with higher (i.e. more prolonged) mid-stance muscle activity in both the presence and absence of symptomatic knee osteoarthritis.

Methods

Subjects

One hundred and eighteen asymptomatic and 177 individuals over 35 years old with moderate knee OA were recruited to the Dynamics of Human Motion laboratory for gait testing. The asymptomatic and OA groups were divided into three body mass groups based on their BMI: healthy weight ($20 \text{ kg/m}^2 < \text{BMI} < 25 \text{ kg/m}^2$), overweight ($25 \text{ kg/m}^2 \leqslant \text{BMI} < 30 \text{ kg/m}^2$), and obese (BMI $\geqslant 30 \text{ kg/m}^2$) (Health-Reports, 2006) (see demographics, Table 1). There were 59 healthy weight, 42 overweight, and 17 obese individuals in the asymptomatic group, and 18 healthy weight, 75 overweight, and 84 obese individuals in the knee OA group. Asymptomatic subjects were recruited through university and hospital postings

and had no history of knee pain. Individuals with knee OA were recruited from the Orthopedic and Sports Medicine Clinic of Nova Scotia and the Orthopaedic Assessment Clinic at the QEII Health Sciences Center. In line with our previous work, subjects were diagnosed with knee OA based on a combination of their radiographs and a physical and clinical examination by an orthopaedic surgeon (WDS). Moderate knee OA was based on having Kellgren and Lawrence (KL) scores between 1 and 3 (Kellgren and Lawrence, 1957), showing mild to moderate joint changes, self-reported ability to walk a city block, jog five meters and walk up stairs in a reciprocal manner, and if they were not candidates for total knee replacement surgery (Hubley-Kozey et al., 2006, Harding et al., 2012). Exclusion criteria included history of cardiovascular disease, any neuromuscular disease, other forms of arthritis, gout, or history of surgery to the lower limb (Hubley-Kozey et al., 2006). All individuals signed a written consent form in accordance with the institutional ethics review.

Table 1 Demographics of healthy weight (HW), overweight (OV) and obese (OB) asymptomatic and moderate knee OA subject groups.

	Asymptomatic			Moderate OA		
	HW	OV	OB	HW	OV	OB
Age (years)	49.4 (8.9)	48.0 (8.7)	48.8 (10.5)	56.7 (7.0)	58.1 (8.3)	57.6 (8.7)
Height (m)	1.67 (0.1)	1.70 (0.1)	1.69 (0.1)	1.71 (0.11)	1.74 (0.08)	1.72 (0.1)
Mass (kg)	64.5 (7.8)	79.8 (9.0)	95.2 (15.9)	68.5 (9.2)	84.0 (9.1)	103.7 (16.9)
BMI (kg/m^2)	22.6 (1.6)	27.5 (1.5)	33.2 (3.5)	23.4 (0.7)	27.8 (1.3)	34.8 (4.0)

Gait analysis

Three-dimensional biomechanics and surface electromyography (EMG) testing were performed on all individuals. The aim of this study was to examine the muscle activation patterns; therefore, only details pertaining to EMG collection and processing are explained her, based on a standard protocol that has between day reliability for those with knee OA (Hubley-Kozey et al., 2006, Hubley-Kozey et al., 2013). Biomechanics methods and results can be found in a previous publication (Harding et al., 2012). Standard preparation of skin was performed (shaving and cleaning with alcohol + water) and silver/silver chloride pellet surface electrodes (10 mm diameter, 20 mm inter-electrode distance) were attached in a bipolar configuration over the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), lateral and medial hamstrings (LH and MH), lateral and medial gastrocnemius (LG and MG) using standard anatomical landmarks (Hubley-Kozey et al., 2006). Isolated movements aimed at activating the different muscles were performed to validate locations and electrode attachment (Winter et al., 1994). A reference electrode was mounted on the shaft of tibia. Raw EMG signals were pre-amplified (500x) and

further amplified (band-pass 10–1000 Hz), using an eight channel surface EMG system (AMT-8 EMG, Bortec Inc., Calgary, Alberta) with a common mode rejection ration of 115 dB (at 60 Hz), an input impedance of about 10 GOhms, and sampled at 2000 Hz. Subjects then performed a second set of isolated movements and the gains of the amplifier were adjusted to ensure the collection of a good quality signal and to asses crosstalk (Winter et al., 1994). To determine baseline activity, a bias trial was performed while subjects lying relaxed and supine.

Synchronized EMG, three-dimensional marker data and ground reaction force data were collected while the subjects walked across a 6-meter walkway at their self-selected speed for a minimum of 5 trials. Three dimensional motion data were collected using an Optotrak 3020 motion capture system at 100 Hz (Northern Digital Inc., Waterloo, ON), and ground reaction forces were sampled at 2000 Hz using an AMTI force platform (AMTI, Watertown, MA). These data were used to determine foot contact and toe-off (Astephen et al., 2008).

To elicit maximal voluntary EMG activity, a series of 8 different maximum voluntary isometric contraction (MVIC) exercises were performed, with contractions held for 3-s, with a rest interval between exercises to minimize fatigue (7 exercises performed on a Cybex dynamometer, Lumex, NY (Hubley-Kozey et al., 2006)). Exercises included: (1) seated knee extension in supine, with knee at 45° flexion; (2) simultaneous knee extension and hip flexion in the same position as 1; (3) seated knee flexion in supine, with knee at 55° flexion; (4) knee extension in supine, at 15° knee flexion; (5) knee flexion in supine, at 15° knee flexion; (6) plantarflexion in supine, with the ankle in neutral and knee close to full extension; (7) heel rise while standing on only one foot; and (8) knee flexion in prone position, at 55° knee flexion. Following a practice trial for each, two MVIC trials were performed with verbal encouragement and visual feedback of the torque production provided for all exercises except 7. If torques differed by more than 10% between trials one additional trial was performed. A relaxed, gravity correction trial was performed for each.

EMG Processing

Raw EMG signals were visually inspected and a Fast Fourier Transform (FFT) performed to identify noise based on the power spectrum. A band pass filter (20 and 500 Hz) was applied to minimize baseline, low frequency noise (primarily movement artefact observed in power spectra of some participants), and frequencies above 500 Hz as there was negligible EMG activity outside that range. This filter is consistent with empirical data showing a low frequency cut-off of 20 Hz removes the majority of artefact noise, while preserving the EMG signal information as minimal signal energy was found between 10 and 20 Hz (De Luca et al., 2010). EMG signals were then corrected for gain and bias, full wave rectified and low pass filtered using a second order non-recursive Butterworth low pass filter with a cut off frequency of 6 Hz (Winter, 1990). A moving average algorithm with a window of 0.1 s was used to find the maximum EMG activity for each muscle during MVIC trials and the maximal values among all different exercises were used to amplitude normalize the EMG waveforms. EMG waveforms of at least 5 trials then were time normalized to 100% of the gait cycle and ensemble averaged to yield the final waveforms for each of the 7 muscles, each person. Final waveforms for each muscle group were placed into the rows of 3 matrices, including a matrix for the 3 quadriceps muscles ($X_{885 \times 101}$), a matrix for the 2 gastrocnemius muscles ($X_{590 \times 101}$), and one for the two hamstrings muscles ($X_{590 \times 101}$). PCA was applied separately to each matrix (Jackson, 1991). Principal component scores (PCscores) were calculated by projecting the original waveforms onto the extracted and retained principal components (PCs). The first 3 PCs for all muscle

groups were chosen, consistent with our previous research, in order to capture the major modes of variability among the individual patterns and muscle groups (Hubley-Kozey et al., 2006, Rutherford et al., 2011). There are different methods to interpret obtained PCs (Brandon et al., 2013), including interpreting the pattern of the PC over the gait cycle, as well as examining extremes at either end of each PCscore distribution (5 highest, 5 lowest) (Hubley-Kozey et al., 2006, Hubley-Kozey et al., 2009, Kirkwood et al., 2011, Rutherford et al., 2011).

Statistical analysis

PCscores were examined for normality, and then a 3 factor analysis of variance was used to examine the main effects and 2 and 3 way interactions among the three factors (moderate OA disease, BMI, and muscle), separately for each muscle group (9 PCscores in total). Bonferroni post hoc procedures were performed to test for pairwise differences when significant interaction or main effects were present (α = 0.05).

Results

Table 1, Table 2 show the demographics, anthropometrics and stride characteristics of the subgroups. Similar to our previous studies, the OA group was older, with higher mass and BMI compared to the asymptomatic group ($P < 0.05$) (Hubley-Kozey et al., 2006). BMI had no significant effect on self-selected walking velocity, but was associated with longer stride and stance times. Mean processed and normalized electromyograms are presented in Fig. 1 for BMI categories.

Table 2 Stride characteristics of healthy weight (HW), overweight (OV) and obese (OB) asymptomatic and moderate knee OA subject groups. P-values for ANOVA main and interaction effects is provided (α = 0.05). Bold indicates statistically significant effects.

	Asymptomatic			Moderate OA			P-value (OA)	P-value (BMI)	P-value (interaction)
	HW	OV	OB	HW	OV	OB			
Speed (m/s)	1.37 (0.18)	1.39 (0.15)	1.37 (0.16)	1.31 (0.17)	1.26 (0.19)	1.20 (0.20)	**0.019**	0.14	0.23
Stride length (m)	1.43 (0.12)	1.47 (0.13)	1.44 (0.14)	1.43 (0.17)	1.42 (0.15)	1.35 (0.16)	**<0.001**	0.22	0.54
Stride time (sec)	1.06 (0.08)	1.07 (0.09)	1.06 (0.07)	1.10 (0.08)	1.14 (0.12)	1.13 (0.10)	**<0.001**	**0.014**	0.29

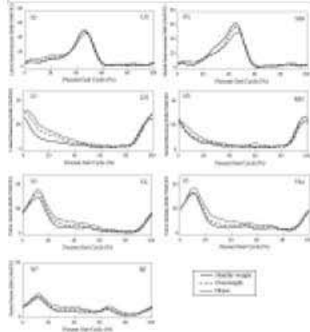|								
Stance time (sec)	0.66 (0.06)	0.67 (0.06)	0.68 (0.05)	0.69 (0.05)	0.73 (0.08)	0.74 (0.07)	**0.003**	**0.000**	0.13
Stance percent (%)	62.4 (1.7)	63.0 (1.1)	63.8 (1.4)	62.4 (1.6)	63.9 (1.7)	65.0 (1.8)	**<0.001**	0.27	0.30

Fig. 1

Mean EMG signals of the lateral and medial gastrocnemius (a: LG, b: MG), vastus lateralis and medialis (e: VL, f: VM), rectus femoris (g: RF), lateral and medial hamstrings (c: LH, d: MH) are shown for the healthy weight (HW, solid line), overweight (OV, dotted line), and the obese group (OB, dashed line). All EMG waveforms were time normalized to 100% of the gait cycle and amplitude normalized to a percentage of maximum voluntary isometric contraction (% MVIC) during standardized exercises described in the text.

Three PCs captured 85.2% of the amplitude and temporal pattern variation for each of the gastrocnemius and quadriceps muscle groups, and 84% for hamstrings muscles. Statistical results are presented in Table 3; however, as the OA main effects for EMG patterns have been described previously by our research group (Hubley-Kozey et al., 2006), only BMI main effects and/or interaction effects with disease presence are discussed here. PC1 for each muscle group captured the overall activation magnitude of the muscle group during stance. There were no statistically significant BMI main effects or interaction effects on PC1 scores for any of the 3 muscle groups.

Table 3Statistical results (*P*-values) for 3-factor ANOVA main effects (BMI category, OA presence, Muscle) and interaction effects for the first 3 PCs of each muscle group are provided (*P* < 0.05 shown in bold).

Muscle group	PC	BMI	OA	Muscle	BMI/OA	BMI/muscle	OA/muscle	BMI/OA/muscle

Muscle	PC							
Gastrocnemius	1	0.98	**<.0001**	**<.0001**	0.53	0.90	**0.03**	0.30
	2	**<.0001**	**0.01**	**<.0001**	**0.02**	0.72	**0.04**	0.63
	3	**<.0001**	**<.0001**	0.09	0.20	0.67	0.66	0.71
Hamstrings	1	0.71	**0.00**	**<.0001**	0.51	0.89	**0.05**	0.87
	2	0.07	**0.02**	0.26	0.40	0.70	0.38	0.40
	3	0.70	**0.00**	0.10	0.54	0.62	0.21	0.55
Quadriceps	1	0.30	**<0.0001**	**<.0001**	0.54	0.99	0.40	0.97
	2	**<0.0001**	0.08	**<.0001**	0.97	0.70	0.69	0.99
	3	0.91	**<.0001**	**<.0001**	0.60	0.70	0.24	0.98

Gastrocnemius muscles

PC2 captured a phase shift for higher gastrocnemius activity to later in stance, with higher PC2 scores associated with an earlier peak activity (Fig. 2a). A significant BMI by disease interaction was found for PC2 ($P < 0.05$, Table 3, Fig. 3). Pairwise post hoc analysis revealed that obese asymptomatic individuals had significantly lower PC2 scores than all 5 other subgroups ($P < 0.0026$), showing that obesity in the absence of OA was associated with a delay in peak gastrocnemius activity in late stance. PC3 captured the difference between the EMG activity of the gastrocnemius from early to late stance (Hubley-Kozey et al., 2006, Rutherford et al., 2011), with lower PC3 scores associated with a smaller differential in muscle activity between early mid-stance to late stance (i.e. sustained activity) (Fig. 2b). A BMI main effect was found for PC3 ($P < 0.05$, Table 3). Post hoc results revealed that obese individuals had significantly lower PC3 scores compared to overweight ($P = 0.007$) and healthy weight ($P = 0.0001$), meaning obesity was associated with more sustained gastrocnemius activity through mid to late stance.
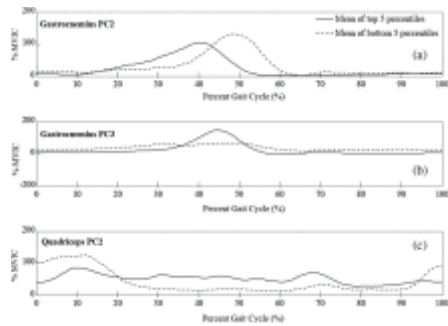
Fig. 2

EMG Pattern Differences with Significant Obesity Effects. Statistically significant ($P < 0.0001$) BMI category effects were found in features of the gastrocnemius and quadriceps EMG. Mean waveforms of the five individuals with the highest (solid lines) and lowest (dashed lines) PCscores associated with the gastrocnemius PC2 (a), the gastrocnemius PC3 (b) and the quadriceps PC2 (c) are shown. (a) The gastrocnemius PC2 described an earlier peak gastrocnemius activity in stance; obese asymptomatic individuals had low scores indicating a delay in peak activity for the obese asymptomatic group. (b) PC3 of the gastrocnemius described a difference in activity from early to late stance. The obese group had significantly lower scores than other BMI categories, meaning more constant gastrocnemius activity through stance. (c) PC2 of the quadriceps described a difference in activity from early to mid-late stance. The obese group had higher PC2 scores, and therefore more constant quadriceps activity through stance.
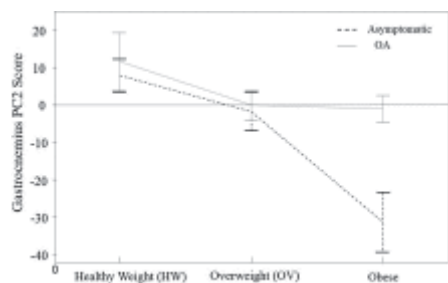

Fig. 3

Interaction Plot of Gastrocnemius PC2. There was a statistically significant interaction in the gastrocnemius PC2 scores, indicating significantly lower PC2 scores for the asymptomatic obese group than all other subgroups. This was interpreted as a relatively later peak activation (Fig. 2a) of the gastrocnemius muscles in the obese asymptomatic group.

Hamstrings

There were no statistically significant BMI main effects or interaction effects on the PCs of the hamstrings, meaning that obesity had little to no effect on the magnitude or pattern of activation of the hamstrings muscles during gait.

Quadriceps

PC2 of the quadriceps muscles captured the difference in activation from early (0–20%) to later stance phase, with higher PC2 scores associated with less difference in activity, or more sustained activation of

the quadriceps muscles from early through mid to late stance (Fig. 2c). A significant main effect of BMI was found for PC2 scores (Table 3). Post hoc comparisons showed higher PC2 scores, and therefore more sustained quadriceps activity for the obese group compared to the other groups.

Discussion

In contrast to previous studies examining lower limb muscle activity in children and adolescents that have reported no significant effect of obesity on EMG measures (Hills and Parker, 1993, de Carvalho et al., 2012, Blakemore et al., 2013), we identified alterations in the activation patterns of the quadriceps and gastrocnemius muscles during gait with obesity in adults. The demographic differences in study populations, and also the differences in analysis techniques, may explain our different findings as compared to the previous investigations. Several effects of moderate knee OA presence on EMG patterns during gait were found, and these were consistent with our previous studies (Childs et al., 2004, Hubley-Kozey et al., 2006, Rutherford et al., 2011), and so not discussed here.

Consistently for all 3 muscle groups, the first principal component, PC1, which explained the most variability among participants, described the overall amplitude of muscle activities during stance phase. There were no statistically significant effects of BMI category on PC1 scores for any of the 3 muscle groups, meaning no statistically significant differences in EMG amplitudes during gait through the 3 BMI categories. This is consistent with the results of previous studies, which reported no difference in the EMG activity of the gastrocnemii muscles of obese and non-obese female adolescents (de Carvalho et al., 2012) and children (Hills and Parker, 1993, Blakemore et al., 2013). This is interesting because peak gastrocnemius activity happens in late stance to propel the body (Winby et al., 2009, Lin et al., 2010) and is expected to increase with higher body mass; indeed previous studies that interrogated the effect of load carriage on the lower limb joint reported an increase in the activity of gastrocnemius muscles with loads up to 40% of BW and no change afterwards (Han et al., 1992). In addition, we found no effect of BMI category on hamstring muscle activity during gait, consistent with previous studies on children and adolescents (Hills and Parker, 1993, de Carvalho et al., 2012, Blakemore et al., 2013). Thus these results suggest that there is not a simple systematic overall activation increase in response to increased mass.

The most significant findings of this study were the sustained activations of the gastrocnemii and quadriceps muscles throughout stance phase with higher BMI category (pattern differences described by gastrocnemius PC3 and quadriceps PC2). Obese individuals walked with more sustained gastrocnemius activity from early mid to late stance, and more sustained quadriceps activity throughout mid to late stance phase than the overweight and healthy weight participants. It was interesting to find these prolonged muscle activation patterns for the obese group and not overweight group, supporting the concept of a threshold for BMI after which individuals may start to adapt altered neuromuscular patterns (DeVita and Hortobagyi, 2003). Ghori and Luckwill (1985)) reported backpack carriage equal to 20% of the body weight increased the duration of the activity of VL, MH, and gastrocnemius muscles in the gait of healthy subjects, and when the backpack weight increased to 50% of the body weight, only VL burst duration was furthered. They concluded these altered neuromuscular patterns in the presence of increased weight can be an attempt to maintain the same kinematics of normal gait (Ghori and Luckwill, 1985), however specific knee joint biomechanics were found to alter with obesity (DeVita and Hortobagyi, 2003, Harding et al., 2012). Thus, prolonged activation of the gastrocnemius and quadriceps muscles may be an attempt to enhance dynamic joint stability during the late stance phase, possibly as a

response to increased weight (McGraw et al., 2000, Menegoni et al., 2009). Comparing obesity to load carriage has its limitations, keeping in mind that obesity is a complicated multifaceted condition, with interacting physiological, mechanical and psychological factors that can affect gait.

The current finding of prolonged muscle activity during the stance phase of gait with obesity is potentially a compensation strategy adopted by obese individuals, which suggests a lack of confidence in joint stability and use, as compared to healthy and even overweight individuals. This sustained muscle activity may expose the joint to higher shear and cumulative contact loading per step, which is undesirable for the cartilage (Maly, 2008). Although some previous studies have shown reduced magnitudes of sagittal plane motion of the knee joint during gait with obesity (DeVita and Hortobagyi, 2003), our previous study on this same population found no significant differences in knee flexion angles or walking speeds with obesity (Harding et al., 2012), and so this prolonged muscle activity is not reflected in the joint kinematics. However, the prolonged activity is somewhat consistent with our previous study that showed significant associations between obesity and less unloading, or more constant knee adduction moment during gait (Harding et al., 2012), also highlighting a lack of weight acceptance during stance. This previous work also showed significantly less range or transverse plane moment during stance with obesity, suggesting a rotationally 'stiffer' joint than healthy weight, which may also reflect some of the prolonged muscle activity captured in the current results. It may also result in muscle fatigue, which disrupts the active mechanism for shock absorption in the knee joint (Bennell et al., 2013), all of which may predispose the joint to an unfavorable mechanical environment more conducive to accelerated osteoarthritis damage. In another previous study, we found significant associations between similar prolonged quadriceps and gastrocnemius activation patterns during stance with increased anterior–posterior motion of a total knee arthroplasty tibial implant post-operatively (Wilson et al., 2012), where movement has been linked to early implant failure revision surgery (Ryd et al., 1995). This previous result supports the link between prolonged activation of these muscles with increased mechanical shearing within the joint, which can have negative consequences to joint health. We also independently found a high BMI pre-operatively linked to this same increased implant migration (Astephen Wilson et al., 2010).

A significant BMI by OA interaction for PC2 of the gastrocnemii demonstrated that obese asymptomatic individuals had a delayed peak in gastrocnemius muscle activity in late stance compared to the healthy weight and overweight groups, as well as the obese subjects with OA. Gastrocnemius muscle forces are significant contributors to the knee joint contact forces during late stance (Winby et al., 2009, Lin et al., 2010). Therefore, this delayed activity and the potentially resultant imbalance between flexors and extensors (Slemenda et al., 1997) may disrupt normal loading of the joint and expose the cartilage to high contact loads at inappropriate time intervals.

Concerns have been expressed with EMG measurement in the presence of adiposity, including reduced amplitude of the measured signal (Kuiken et al., 2003, Bartuzi et al., 2010) and increased crosstalk (Winter et al., 1994, Kuiken et al., 2003). Precautions were taken, consistent with published guidelines to minimize the effects on both including standardized electrode placement protocols (Soderberg and Knutson, 2000), using electrodes with small surface areas and minimal bipolar spacing differences (Winter et al., 1994), palpating electrode placements by an experienced tester while subject performed isolated tasks (Winter, 1990), and assessing signal quality and antagonist crosstalk during a set of isolated movements (Winter et al., 1994). Furthermore, the first PC of each muscle group reflects the overall amplitude of activation during gait, and would be most influenced by the amount of the adipose

due to signal attenuation. Secondly concerns have been expressed with generating MVIC in knee OA participants, but similar motor unit recruitment percentages for maximal voluntary effort activation has been shown between healthy asymptomatic (96%) and moderate OA (93%) participants using a superposition protocol (Lewek et al., 2004). Furthermore we have shown excellent between day reliability for those with moderate knee OA for EMG amplitude and temporal measures using a series of different MVIC exercises and the protocol described above (Hubley-Kozey et al., 2013). The statistically significant findings of the current work reflect differences in temporal patterns of activation and not magnitude features, which should be less affected by adiposity and normalization. A second limitation is that although a frequently used metric for body mass (Felson et al., 2004), BMI may not adequately reflect body mass composition and obesity in all individuals. Despite this, BMI has been the obesity-related variable used in previous longitudinal epidemiological investigations of the role of obesity in later OA development and progression (Felson et al., 1988).

Altered activation patterns due to obesity and knee OA were found in this cross sectional study. Higher BMI category was significantly associated with more prolonged gastrocnemius and quadriceps EMG activity during the stance phase of gait. Because this was a cross-sectional observational study, it is unclear how these muscle activity patterns may relate to knee OA disease development and progression. However, these results do support an altered neuromuscular control strategy with obesity that is consistent with some of the EMG alterations found with knee osteoarthritis (Hubley-Kozey et al., 2006), and support the need to further explore how these patterns may be contributing to a negative mechanical joint environment that could precipitate and accelerate the osteoarthritis process. These results also support the role of neuromuscular training (Farrokhi et al., 2013) to potentially improve the mechanical environment of the joint to avoid accelerated osteoarthritis with obesity. Future work should include longitudinal investigations to further investigate the effect of these mechanisms over time, as well as experimental work to understand if weight loss alone can alter these muscular activation patterns.

Conclusions

This study identified alterations in the temporal activity patterns of the lower limb gastrocnemii and quadriceps muscles due to obesity, knee OA presence, and their interactions during gait. Obesity was significantly associated with prolonged activation of quadriceps and gastrocnemii, which can result in prolonged knee joint contact loading, and thereby may contribute to the predisposition of knee OA development and progression in obese individuals.

Acknowledgements

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