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Effects of Obesity on Lower Extremity Muscle Function During Walking at Two Speeds

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Abstract

Walking is a recommended form of physical activity for obese adults, yet the effects of obesity and walking speed on the biomechanics of walking are not well understood. The purpose of this study was to examine joint kinematics, muscle force requirements and individual muscle contributions to the walking ground reaction forces (GRFs) at two speeds (1.25 $m \cdot s^{-1}$ and 1.50 $m \cdot s^{-1}$) in obese and nonobese adults. Vasti (VAS), gluteus medius (GMED), gastrocnemius (GAST), and soleus (SOL) forces and their contributions to the GRFs were estimated using three-dimensional musculoskeletal models scaled to the anthropometrics of nine obese (35.0 (3.78 $kg \cdot m^{-2}$); body mass index mean (SD)) and 10 nonobese (22.1 (1.02 $kg \cdot m^{-2}$)) subjects. The obese individuals walked with a straighter knee in early stance at the faster speed and greater pelvic obliquity during single limb support at both speeds. Absolute force requirements were generally greater in obese vs. nonobese adults, the main exception being VAS, which was similar between groups. At both speeds, lean mass (LM) normalized force output for GMED was greater in the obese group. Obese individuals appear to adopt a gait pattern that reduces VAS force output, especially at speeds greater than their preferred walking velocity. Greater relative GMED force requirements in obese individuals may contribute to altered kinematics and increased risk of musculoskeletal injury/pathology. Our results suggest that obese individuals may have relative weakness of the VAS and hip abductor muscles, specifically GMED, which may act to increase their risk of musculoskeletal injury/pathology during walking, and therefore may benefit from targeted muscle strengthening.

Keywords

Biomechanics; obesity; muscle function; musculoskeletal modeling; gait

Introduction

Obesity is a worldwide public health concern and obese adults and children are advised to engage in daily physical activity. Walking is a recommended form of physical activity for

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The authors declare no conflict of interest.

obese adults because it is convenient and suitable to elicit a moderate-vigorous metabolic response [1]. However, obese individuals have lower relative muscle strength compared to nonobese individuals [2]. Weakness and susceptibility to fatigue of certain key muscles (e.g. vasti (VAS) and gluteus medius (GMED)) can result in an abnormal gait pattern due to their critical role in locomotor tasks [3], predisposing individuals to musculoskeletal injury or pathology (e.g. large joint osteoarthritis (OA) and low back pain) [4, 5]. In addition, muscle force requirements increase with walking speed [6], so at the faster walking speeds used during exercise, certain muscles, including those responsible for forward progression (e.g. the gastrocnemius (GAST) and soleus (SOL)), may be unable to effectively perform their respective functions, resulting in gait deviations that may increase the risk of musculoskeletal injury/pathology.

Surprisingly, the degree to which obesity affects gait kinematics and kinetics is not clear. Some studies report that kinematics are similar in obese and nonobese groups [7, 8], while others report that obese individuals walk with a more extended leg and similar knee extensor moments during stance and greater step width compared to their nonobese counterparts [9], particularly at faster walking speeds. Unfortunately, there is limited information regarding how investigators did or did not account for the peripheral adiposity that obscures the motion of the underlying skeleton. Thus, differences in methodology may explain these equivocal kinematic results. In addition, studies that have reported lower extremity gait biomechanics in obese individuals [8, 9] have not provided a quantitative assessment of individual muscle function, which may help explain the observed gait patterns.

Musculoskeletal simulations can provide us with an improved understanding of the force requirements and roles that individual muscles play during locomotor tasks [10]. Recent studies have estimated the contributions of individual muscles to the ground reaction force (GRF) during walking in nonobese adults [11, 12]. These studies have shown that during early stance, VAS and GMED muscles are significant contributors to the vertical GRF (GRF_V), and function to decelerate and support the body, while during mid-late stance, the gastrocnemius (GAST) and soleus (SOL) are the primary contributors to the GRF_V and the anterior-posterior GRF (GRF_{AP}). In the frontal plane, GMED acts to maintain mediolateral (ML) stability and balance, and has been shown to be the primary contributor to the ML GRF (GRF_{ML}) [13]. Unlike in the sagittal plane, where a more aligned skeleton would reduce knee extensor muscle requirements, support and stability of the body in the frontal plane is largely accomplished by the hip abductor muscles (e.g. GMED). The effect of GMED weakness may be altered frontal plane kinematics of the pelvis (e.g. increased pelvic obliquity, an increase in pelvic drop of the contralateral hip) resulting in pathological hip joint articulation [14]. For this study, we focused our investigation on the muscles that have large contributions to all three components of the GRF (VAS, GMED, GAST, and SOL) [12].

The purpose of this study was to quantify joint kinematics, estimate individual muscle forces (VAS, GMED, GAST, SOL), and the individual muscle contributions to the walking GRFs at two speeds (1.25 $m \cdot s^{-1}$ and 1.50 $m \cdot s^{-1}$) in obese and nonobese adults. It has been reported that obese adults walking with a more erect posture and similar knee extensor joint torques compared to nonobese adults [9], suggesting reduced knee extensor muscle forces. We hypothesized that (1) peak knee flexion during stance would be less, while pelvis obliquity would be greater in the obese vs. nonobese group, and the differences between the obese and nonobese groups would be greater at the faster walking speed; (2) absolute and lean mass normalized forces for all muscles, except VAS, which we predict to be similar, would be greater in the obese vs. nonobese adults at both speeds; and (3) VAS contribution to the GRF_V would be similar between the obese and nonobese individuals at a velocity of 1.25 $m \cdot s^{-1}$ but would be reduced at a velocity of 1.50 $m \cdot s^{-1}$ in the obese group.

Methods

Subjects

A convenience sample of nine obese (8 female) adults and 10 nonobese adults (5 female) participated in our study. Inclusion criteria included a BMI of < 25 kg•m^{-2} (nonobese) and 30–40 kg•m^{-2} (obese), age 18–45, and sedentary to moderately active (< 2–3 bouts of exercise/week or participation in any sporting activities < 3 hours/week), while exclusion criteria included orthopedic, metabolic, or neurologic impairments, other than obesity, that would hinder movement and prevent safe participation in the study. Subject characteristics and anthropometrics are presented in Table 1. All subjects gave written informed consent approved by Colorado State University's Human Research Institutional Review Board.

Experimental Protocol

We quantified body mass composition for each subject via dual x-ray absorptiometry (DEXA, Hologic Discover, Bedford, MA). As part of a larger study, participants walked at nine randomized speed grade combinations (speeds: 0.50 m•s^{-1} to 1.75 m•s^{-1}, grades: 0–9°). Trials lasted 6 minutes with 5 minutes of rest between trials. During an acclimatization period, before the first trial, subjects walked on the treadmill at a self-selected speed. The acclimation period ended when participants had walked for at least 5 minutes and were observed to have a normal gait pattern (all participants walked 10 minutes or less during the acclimation period). Here, we are reporting only the results from two level (0° grade) trials: 1.25 m•s^{-1} and 1.50 m•s^{-1}. The 1.25 m•s^{-1} walking speed was selected as it is very near the self-selected speed for obese adults reported by DeVita and Hortobagyi (1.29 m•s^{-1}) [9], while a walking speed of 1.5 m•s^{-1} was selected because it is considered an appropriate exercise walking speed for obese adults to meet physical activity guidelines and achieve proper physiological benefits [15].

Experimental Data

Whole body kinematics and kinetics were collected using a 10-camera motion capture system (Nexus, Vicon, Centennial, CO) recording at 100 Hz and a dual-belt, force measuring treadmill (Fully Instrumented Treadmill; Bertec Corp, Columbus, OH) recording at 1000 Hz. We used an obesity-specific marker set methodology, which was utilized to attenuate the effects of subcutaneous adiposity obscuring the motion of the underlying skeleton, particularly the anterior pelvis. Physical reflective markers were placed over the following anatomical landmarks: 7th cervical vertebrae, acromion processes, right scapular inferior angle, sterno-clavicular notch, xyphoid process, 10th thoracic vertebrae, and bilaterally over posterior-superior iliac spines, medial and lateral epicondyles of the femurs, medial and lateral malleoli, calcanei, first metatarsal heads, second metatarsal heads, and proximal and distal heads of the 5th metatarsals. Marker clusters (four non-collinear markers affixed to a rigid plate) were adhered to the sacrum, and bilaterally to the thighs and shanks to aid in three-dimensional tracking. We also digitally marked the anterior superior iliac spines (ASIS) and iliac crests using a digitizing pointer (C-Motion, Germantown, MD). Borhani et al. showed improved repeatability and good reliability in tracking the movement of the pelvis with a cluster placed on the sacrum (similar design and placement as in our study) as compared to the "traditional' method of tracking via anterior and posterior ASIS markers in nonobese, overweight, and obese individuals [16]. Electromyographic (EMG) data (Noraxon, Scottsdale, Arizona) from bipolar surface electrodes recording at 1000 Hz was collected for the soleus, lateral gastrocnemius, vastus lateralis, vastus medialis, biceps femoris long head, and semimembranosus muscles using International Society for Electrophysiology and Kinesiology standard procedures [17]. The EMG signal was band-pass filtered (16–380 Hz), fully rectified and finally low-pass filtered at 7 Hz. All biomechanics data was collected during the final 30 seconds of each trial. Marker trajectory

and GRF data were digitally low-pass filtered at 5 Hz and 12 Hz, respectively, using fourth-order zero-lag Butterworth filters.

Musculoskeletal Modeling

We scaled a generic OpenSim musculoskeletal model for each subject to account for individual anthropometrics. The mass and inertial properties of each body segment were scaled as a function of segment length, determine by anatomical landmarks, and total body mass. The model was comprised of 12 body segments with 19 degrees of freedom, and 92 muscle-tendon actuators [18, 19]. The knee joint included a planar patellofemoral joint based on kinematics from Yamaguchi et al. [20] and Delp, et al. [18], that articulates with the femur. The joint angles during each gait trial were calculated using OpenSim's inverse kinematics analysis, which minimized the errors between markers on the scaled model and experimental marker trajectories. We used standard marker weighting factors and accuracy criteria (total root mean square and maximum marker errors for the primary anatomical landmarks less than 2cm, and 4cm, respectively) that follow OpenSim guidelines [21, 22]. If needed, we iteratively improved the marker placement on the generic model prior to rerunning scaling and inverse kinematics until these qualifications were met. To calculate the pelvic obliquity angle, we used the method described by Michaud, et al. [23], implemented in Matlab (The MathWorks, Inc., Natick, Massachusetts, United States), using the right and left ASIS marker trajectories. A negative pelvic obliquity angle signified a lower position of the contralateral ASIS marker (i.e. pelvic drop). We reduced residual forces and moments by implementing the Residual Reduction Algorithm (RRA) analysis in OpenSim [22]. To resolve the net muscular moments into individual muscle forces, a weighted static optimization approach was chosen. The objective function minimized the sum of squared muscle activations, while incorporating individual muscle weighting constants of seven for the gastrocnemius, three for the hamstrings and one for all other muscles in the model. These weighting constants were indirectly validated by comparing model estimated tibiofemoral forces to those measured experimentally from an instrumented knee joint replacement [19, 24]. Individual muscle contributions to the GRFs for each gait cycle were determined using a method described previously by Lin et al., implemented in a validated OpenSim plugin [12, 25].

We assessed the quality and accuracy of our simulations by analyzing the final residual forces from RRA and qualitatively comparing the simulated muscle activations to the on/off timing of the experimentally collected EMG. We found the residuals forces applied to the center of mass of the pelvis were less than 4.1% BW in each coordinate direction for all participants, suggesting our simulations were adequately dynamically consistent. Additionally, we found good agreement between the EMG data of the collected muscles and model estimates for both activation timing and changes in magnitude across speeds (Supplemental Fig. 1).

We present the joint kinematics, muscle forces, and muscle contributions from the right leg, normalized to each gait cycle, averaged across five representative gait cycles for each subject, and then averaged across subjects to obtain group means at each speed. Muscle forces are reported in absolute, total body weight (BW) normalized, and lean body weight (LW, lean weight = total body mass – fat mass – skeletal mass) normalized quantities. Muscle contributions to the GRFs are normalized to BW. All muscle force normalizations occurred post simulation.

Statistical Analysis

No a-priori power analysis was performed. Two-factor repeated measures ANOVA tests determined how obesity and walking speed affected joint kinematics, muscle forces, and

muscle contributions to the GRF. When a significant main effect was observed, post hoc comparisons were made using the Holm-Sidak method, where $P<0.05$ defined significance. SigmaPlot version 11.0 (Systat Software, Inc., San Jose, CA) was used to perform statistical analyses.

Results

The kinematics of the lower extremity, specifically, pelvic obliquity and knee joint angle, were affected by obesity but not speed (Fig. 1). At the slower speed, there were no statistical differences in peak sagittal plane hip and knee joint angles between groups. At the faster speed, the obese group walked with a more extended knee during early stance compared to the nonobese group ($p=0.012$). The peak knee flexion angle during early stance at the faster vs. slower speed in the nonobese group was not significantly different ($p=0.076$). Pelvic obliquity was significantly greater during late stance in the obese vs. nonobese participants at both the slower ($p=0.024$) and the faster ($p=0.016$) speeds.

The obese group had greater absolute GMED and SOL peak force output at both speeds compared to the nonobese group (GMED: $p<0.001$, SOL: $p=0.006$) (Fig. 2, Table 2). Peak absolute and LW normalized VAS forces were not different between the obese and nonobese groups at both 1.25 $m \cdot s^{-1}$ and 1.50 $m \cdot s^{-1}$. There was a main effect of speed for absolute VAS forces ($p=0.03$). Peak absolute and LW normalized GMED forces were significantly greater in the obese vs. nonobese at both the slower (absolute: $p=0.003$, LW: $p=0.01$) and faster (absolute: $p=0.001$, LW: $p=0.02$) speeds, while the peak BW normalized force was not significantly different (Fig. 2, Table 2). The difference in LW normalized GMED peak force output between the obese and nonobese groups increased from 26% at the slower speed to 35% at the faster speed.

All muscles were significant contributors to the GRF_V (Fig. 3A). The peak VAS and SOL contributions to the GRF_V were significantly greater in the nonobese compared to the obese group at the slower (VAS: 0.44 vs. 0.28 BW, $p=0.007$; SOL: 1.08 vs. 0.86 BW, $p<0.001$) and faster speeds (VAS: 0.59 vs. 0.38 BW, $p<0.001$; SOL: 1.14 vs. 0.99 BW, $p=0.013$). GMED was the primary contributor to the GRF_{ML}, and was similar between groups at both speeds (Fig. 3B). During late-stance, the GAST and SOL induced a lateral contribution to GRF_{ML} causing the medially acting contribution from GMED to exceed the GRF_{ML}. VAS was the main contributor to the posterior GRF_{AP} during early-stance, while the SOL was the main contributor to the anterior GRF_{AP} during late-stance in both groups at both speeds (Fig. 3C). Peak VAS contributions to the posterior GRF_{AP} were not significantly different between the groups at both speeds. The SOL contribution to the anterior GRF_{AP} was significantly greater in the nonobese vs. obese participants at the slower (0.23 vs. 0.17 BW, $p=0.024$) and faster (0.27 vs. 0.22 BW, $p=0.041$) speeds. During late stance, the sum of the contributions to the anterior GRF_{AP} for the reported muscles, particularly in the nonobese group, appear to exceed the anterior GRF_{AP}. These muscles must overcome the net contribution of the muscles acting posteriorly (not reported) to the GRF_{AP} at this instant in the gait cycle.

Discussion

Obese individuals walk with similar sagittal plane (hip and knee), but altered frontal plane (pelvic obliquity) kinematics compared to non-obese individuals at 1.25 $m \cdot s^{-1}$, which is close to the reported preferred walking speed for obese adults (1.29 $m \cdot s^{-1}$) [9]. At a speed of 1.50 $m \cdot s^{-1}$, obese individuals walk with both sagittal and frontal plane kinematic alterations (more extended knee, greater pelvic obliquity) vs. their nonobese counterparts. As hypothesized, compared to nonobese individuals, obese individuals walked with greater

sagittal plane knee kinematic differences at the faster speed, and greater pelvic obliquity during mid-late stance at both speeds. Not all muscles had greater absolute force production at the faster speed. At both speeds, only absolute GMED, and SOL forces and LW normalized GMED forces were greater in the obese vs. nonobese subjects; so, we partially accept our second hypotheses. Because the VAS contribution to the GRF_V was less in the obese vs. nonobese group at both the slower and faster velocities, we partially accept our third hypothesis.

Our finding of similar early stance knee flexion angles between obese and nonobese participants at the 1.25 $m \cdot s^{-1}$ walking speed is in agreement with those previously reported at the same speed [8], and at obese specific self-selected speeds [7]. Devita and Hortobágyi found that obese participants walked with a more extended knee at 1.50 $m \cdot s^{-1}$, which is in agreement with our results at that speed [9].

The estimated muscle forces (magnitudes and temporal characteristics), and their contributions to the GRFs from our nonobese group are in good agreement with those previously reported [11, 12]. Absolute GMED and SOL muscle forces were greater in obese vs. nonobese individuals due to the increase in body mass. When these muscle forces are normalized to BW, however, they are similar between groups, the lone exception being VAS, which is lower in the obese group. In order to improve our understanding of the relative muscular requirements per skeletal muscle tissue to perform the same task, we normalized the muscle forces to LW. In doing so, we found that the obese subjects had significantly greater GMED requirements compared to the nonobese group during walking at the same speeds. This result suggests that because the relative force requirement for this muscle is greater in obese individuals, it may be unable to function normally and may be more susceptible to fatigue in this population.

We propose that relative muscle weakness may be a possible explanation for the altered knee joint kinematics exhibited by obese people when walking at faster speeds. Walking with a straighter leg may be a compensatory mechanism to reduce VAS force requirement during early stance. Obese adults have relatively weaker quadriceps (both absolute and relative to BW) [2, 26] and lower quadriceps fatigue resistance [2] compared to nonobese adults. Obese individuals may preemptively adopt a gait strategy to minimize VAS force requirement while walking at faster speeds in an attempt to reduce fatigue in those muscles. Our analysis of the individual muscle contributions to the GRF_V suggest that by walking with a straighter knee, a greater proportion of body weight support is achieved by a more aligned skeleton rather than the knee extensor muscles. Additionally, obese individuals may walk with this kinematic adaptation in order to reduce the metabolic cost of locomotion by reducing VAS requirements. Still, while the results of our simulations indicated that there was not an increased contribution from other (not reported) hip/knee extensor muscles to the GRF_V, it cannot be completely ruled out that other muscles not reported in this study may compensate for reduced VAS contribution, ultimately resulting in similar metabolic cost. Obese individuals may have a heightened risk of musculoskeletal pathology at the knee during fast walking because there is increasing evidence that reduced shock absorption during repetitive loading (i.e. locomotion) resulting from muscle dysfunction can lead to radiographic features of OA, such as articular cartilage deterioration and sclerosis of subchondral bone [27].

Presumably due to the difficulties associated with accurately capturing the motion of the underlying pelvis, no prior studies have examined the effect of obesity on pelvic region motion during walking. Our results may suggest a hierarchical control strategy for certain muscles during gait because the GMED contribution to the GRF_{ML} is similar in obese vs. nonobese while frontal plane pelvic kinematics are not similar. In obese adults, GMED may

be unable to provide whole body support and stability in addition to maintaining normal pelvic girdle kinematics. It has been reported that GMED operates at ~70% of its maximum voluntary isometric contraction in nonobese individuals during gait [28]. This information, combined with our finding of significantly greater lean mass normalized GMED force requirement in the obese vs. nonobese groups, leads us to propose that this muscle may be at a particularly high risk for overload and fatigue during walking in this population, especially at faster speeds. Amaro et al., report that GMED weakness has been associated with radiographic osteoarthritis of the hip and suggest preventative GMED muscle strengthening [29]. Our results, coupled with the knowledge that obesity is a strong independent risk factor in hip OA [30], suggest GMED strengthening as a preventative measure may be applicable to obese individuals as well. Additionally, there is growing evidence that hip muscle dysfunction may be related to knee joint degeneration [31], which should be an important consideration in this population given the strong link between obesity and knee joint osteoarthritis [32].

Several limitations inherent to this study warrant discussion. Our sample size was small, and the obese group was predominantly female. Small differences in pelvic motion have been reported between males and female adults [33]. However, the differences between our obese and nonobese subjects are larger than those reported between genders [33]. Another limitation of this study was that the scaling of the inertial properties of each musculoskeletal model's segments did not directly account for the overlying mass of adipose tissue. However, body mass distributions are generally similar between obese and nonobese adults [34] and inertial properties of the body segments have minimal influence on model kinetics during the stance phase of gait [35]. Thus, scaling of the inertial properties based on segment size and body mass in obese adults should have limited impact on the results of this study. Other modeling limitations included a knee joint with no frontal plane degree of freedom and estimates of moment arm distances. We are confident that the relative differences in model estimates (i.e. muscle forces) between subjects are a result of differences in the kinematic and kinetic data used as inputs rather than a result of model limitations because all participants had normal lower extremity alignment and were generally of average height, minimizing the amount of model scaling required and subsequent influence on the muscle moment arms. Additionally, our EMG data suggests similar timing and magnitudes of the muscles used in this comparison and is consistent with the literature [36]. Greater amounts of subcutaneous adiposity may obscure the motion of the pelvis and result in greater marker errors associated soft tissue artifact in the obese vs. nonobese individuals. However, we attempted to minimize these inaccuracies by implementing an obesity specific marker set methodology that incorporated digitally marked locations on the pelvis with sacral, thigh, and shank marker clusters. Finally, the muscle weighting factors specified in the static optimization objective function were established from one walking speed; however, we found that the relative differences between groups are insensitive to the weighting factors themselves.

In conclusion, the altered knee kinematics exhibited at the faster speed and pelvic kinematics exhibited at both speeds in obese individuals may result from changes in the function of lower extremity muscles acting to support, stabilize, and propel the whole body center of mass during walking. Obese individuals appear to adopt a gait pattern that reduces the role of the VAS muscles in supporting and accelerating the whole body center of mass, but walking still requires relatively large muscle forces, particularly in the GMED. Greater lower extremity force requirement per skeletal muscle tissue in specific muscles in obese individuals may contribute to altered kinematics and increased risk of musculoskeletal injury/pathology.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

We used musculoskeletal models to quantify muscle function in obese adults.

Kinematic differences exist at certain speeds in obese vs. nonobese adults.

Differences in the function of certain muscles exist between groups.

Gait adaptation may be due to abnormal muscle requirements in obese adults.

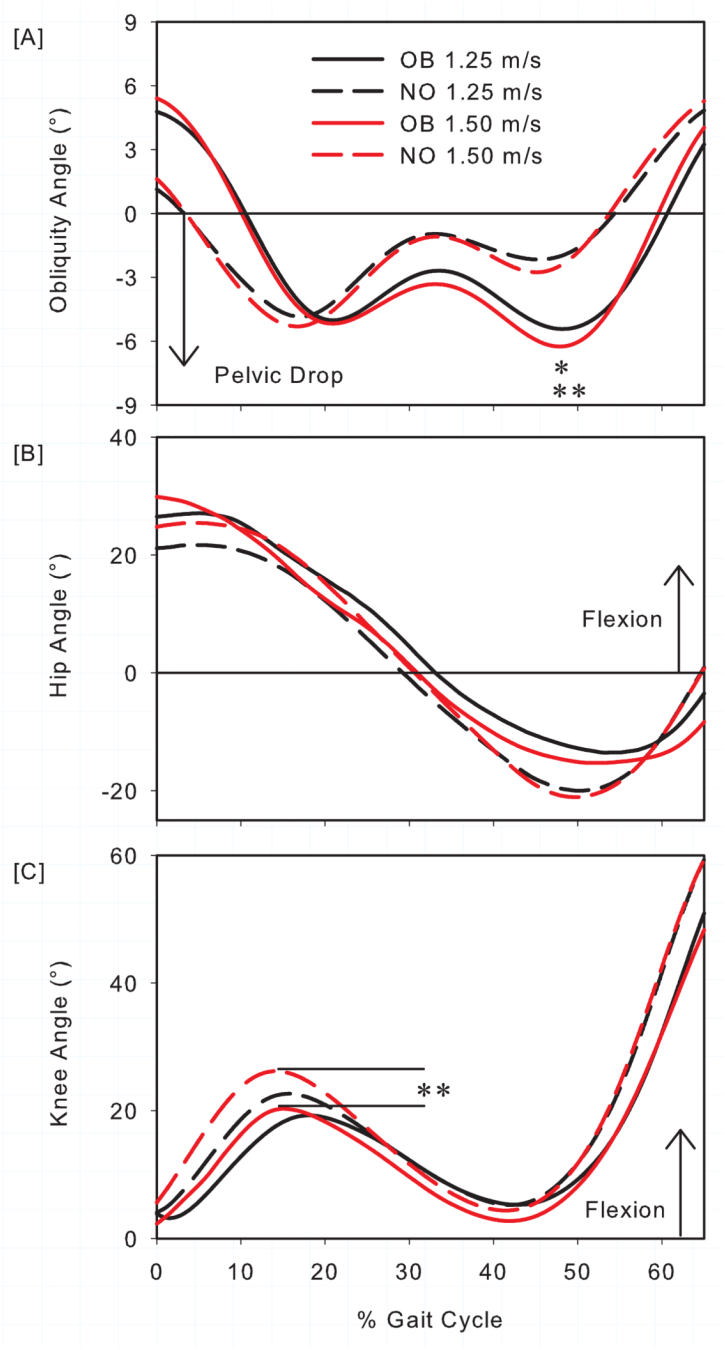

Figure 1.

Mean (A) pelvic obliquity, (B) hip, and (C) knee joint angles for obese and nonobese participants across walking conditions (obese (solid), nonobese (dashed), 1.25 $m \cdot s^{-1}$ (black) and 1.5 $m \cdot s^{-1}$ (red)). Compared to nonobese individuals, obese individuals exhibited a more extended knee during weight acceptance at the faster speed. * and ** denote a significant difference at the slower and faster speeds, respectively.

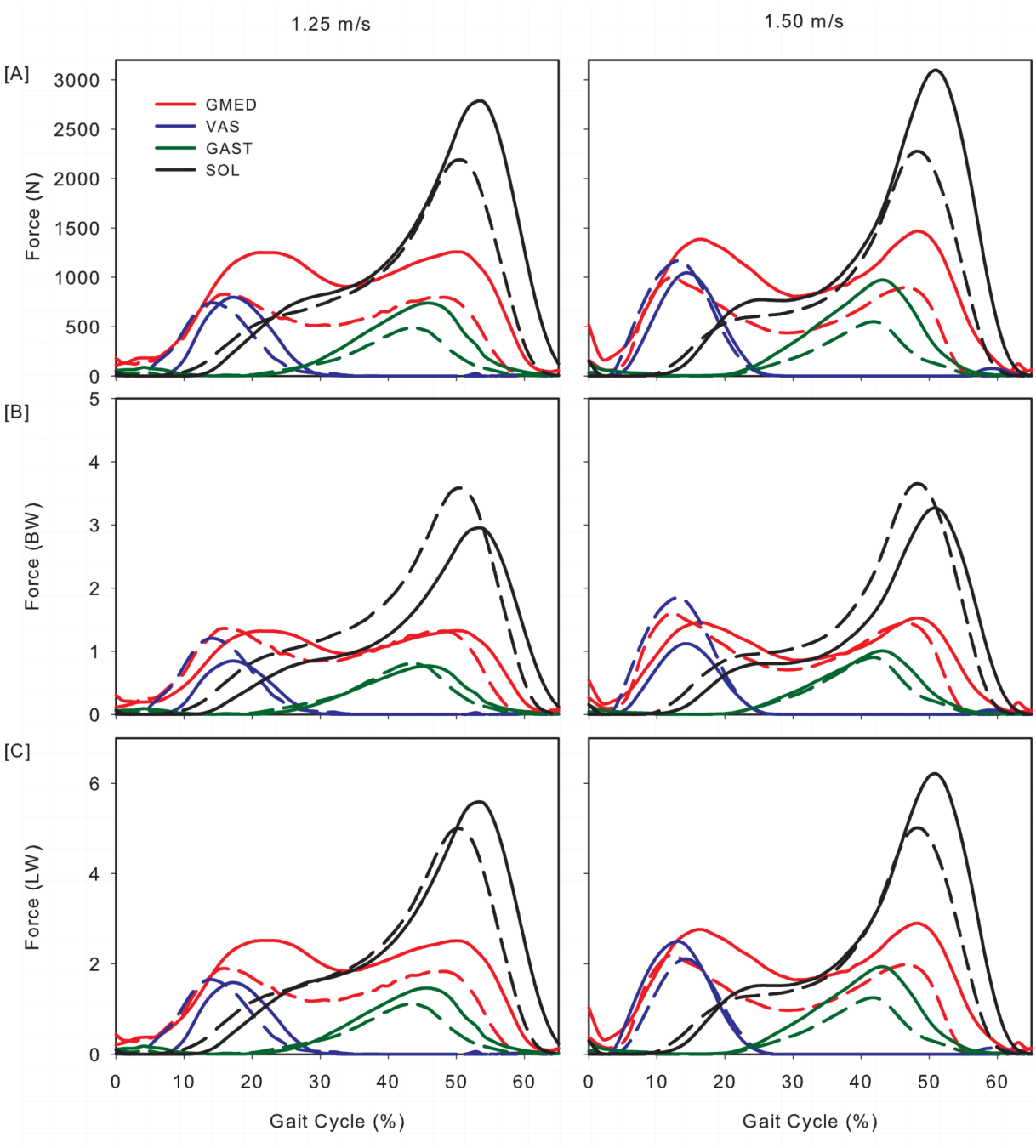

Figure 2.
Mean (A) absolute, (B) BW normalized, and (C) LW normalized muscle forces for obese (solid) and nonobese (dashed) participants across walking conditions (1.25 m•s^{-1} (left column) and 1.5 m•s^{-1} (right column)). Absolute and BW normalized VAS forces were lower, while LW normalized GMED forces were greater in obese vs. nonobese at both speeds.

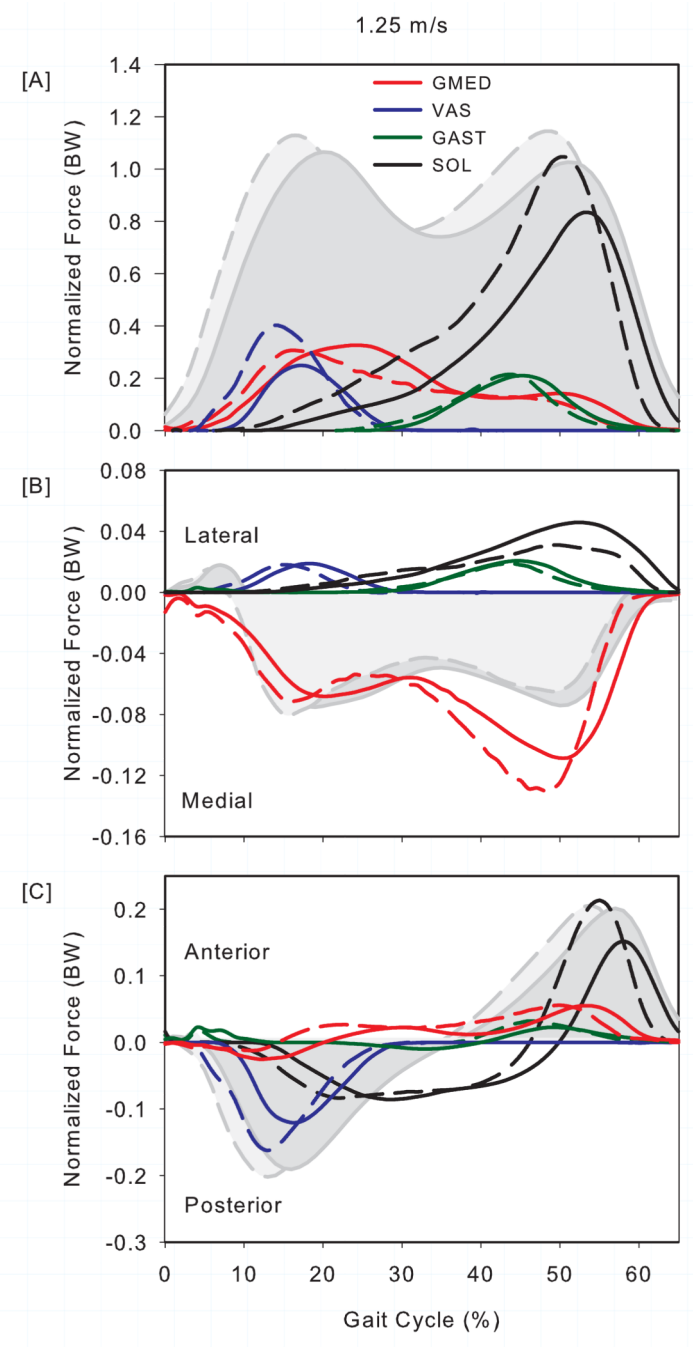

Figure 3.

Mean muscle contributions to the (A) GRF_V, (B) GRF_{ML}, and (C) GRF_{AP} for obese (solid) and nonobese (dashed) participants walking at 1.25 m•s^{-1}. The grey shaded areas are the mean GRF for the obese (solid boarder, darker grey) and nonobese (dashed boarder, lighter grey) groups in each direction.

Table 1

Physical characteristics of obese and nonobese participants

Participant Characteristics	Obese	Nonobese
Body Mass (kg)	96.8 (11.5)	63.7 (4.47)
Lean Mass (kg)	51.4 (8.55)	46.9 (6.83)
Height (m)	1.66 (0.069)	1.69 (0.051)
BMI (kg/m^2)	35.0 (3.78)	22.1 (1.02)
Age (years)	35 (7.6)	26 (6.0)

Values are mean (SD)

Table 2

Absolute, BW normalized, and LW normalized peak muscle forces from obese and nonobese participants walking at 1.25 $m \cdot s^{-1}$ and 1.50 $m \cdot s^{-1}$.

		OB 1.25 $m \cdot s^{-1}$	NO 1.25 $m \cdot s^{-1}$	OB 1.50 $m \cdot s^{-1}$	NO 1.50 $m \cdot s^{-1}$
Absolute (N)	GMED	1468 (133)*	970 (66)	1962 (316)*	1063 (51)
	VAS	880 (95)	818 (83)	1109 (119)†	1202 (124)†
	GAST	778 (131)	537 (43)	999 (147)*†	562 (65)
	SOL	2867 (203)*	2271 (82)	3153 (258)*	2301 (96)
BW Normalized ($N \cdot BW^{-1}$)	GMED	1.54 (0.12)	1.61 (0.13)	2.06 (0.32)	1.70 (0.07)
	VAS	0.93 (0.10)*	1.33 (0.12)	1.18 (0.13)*	1.91 (0.16)†
	GAST	0.81 (0.10)	0.89 (0.08)	1.03 (0.13)†	0.92 (0.12)
	SOL	3.05 (0.21)*	3.72 (0.10)	3.32 (0.20)	3.69 (0.14)
LW Normalized ($N \cdot LW^{-1}$)	GMED	2.92 (0.24)*	2.09 (0.14)	3.31 (0.29)*	2.33 (0.12)
	VAS	1.75 (0.17)	1.83 (0.15)	2.23 (0.24)	2.58 (0.19)†
	GAST	1.55 (0.21)	1.235 (0.12)	1.98 (0.28)*†	1.28 (0.19)
	SOL	5.78 (0.42)	5.19 (0.28)	6.32 (0.45)	5.07 (0.29)

Values are mean (SE).

* indicates significant difference of obese (OB) from nonobese (NO) individuals;

† indicates significant difference from 1.25 $m \cdot s^{-1}$.