



Does adiposity affect muscle function during walking in children?



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ABSTRACT

The biomechanical mechanisms responsible for the altered gait in obese children are not well understood, particularly as they relate to increases in adipose tissue. The purpose of this study was to test the hypotheses that as body-fat percentage (BF%) increased: (1) knee flexion during stance would decrease while pelvic obliquity would increase; (2) peak muscle forces normalized to lean-weight would increase for gluteus medius, gastrocnemius, and soleus, but decrease for the vasti; and (3) the individual muscle contributions to center of mass (COM) acceleration in the direction of their primary function (s) would not change for gluteus medius, gastrocnemius, and soleus, but decrease for the vasti. We scaled a musculoskeletal model to the anthropometrics of each participant ($n = 14$, 8–12 years old, BF%: 16–41%) and estimated individual muscle forces and their contributions to COM acceleration. BF% was correlated with average knee flexion angle during stance ($r = -0.54$, $p = 0.024$) and pelvic obliquity range of motion ($r = 0.78$, $p < 0.001$), as well as with relative vasti ($r = -0.60$, $p = 0.023$), gluteus medius ($r = 0.65$, $p = 0.012$) and soleus ($r = 0.59$, $p = 0.026$) force production. Contributions to COM acceleration from the vasti were negatively correlated to BF% (vertical— $r = -0.75$, $p = 0.002$, posterior— $r = -0.68$, $p = 0.008$), but there were no correlation between BF% and COM accelerations produced by the gastrocnemius, soleus and gluteus medius. Therefore, we accept our first, partially accept our second, and accept our third hypotheses. The functional demands and relative force requirements of the hip abductors during walking in pediatric obesity may contribute to altered gait kinematics.

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1. Introduction

Walking is the most common form of daily physical activity, yet obese children walk differently than nonobese children (Shultz et al., 2011). The altered gait exhibited by obese children have been associated with decreased gait stability (McGraw et al., 2000) and reduced walking performance (Maffels et al., 1993), as well as an increased prevalence of musculoskeletal pain (Taylor et al., 2006) and pathology (Wearing et al., 2006), which, collectively, may pose both short and long-term barriers to physical activity (Zabinski et al., 2003).

Gait analysis studies have shown that compared to nonobese children, obese children walk with wider step widths, increased medial–lateral motion, greater hip abduction, and reduced knee flexion during stance (Gushue et al., 2005; McGraw et al., 2000;

McMillan et al., 2009; Shultz et al., 2011). Prior studies have also reported similar absolute knee extensor moments (Nm), greater absolute ankle plantarflexor moments (Nm), and greater normalized frontal plane moments ($\text{Nm kg}^{-1} \text{m}^{-1}$) of the hip/pelvis in obese vs. nonobese children (Gushue et al., 2005; McMillan et al., 2009). Therefore, compared to nonobese children, obese children may walk with reduced force requirements for the knee extensor muscles, but greater force requirements for both the plantarflexor and hip abductor muscles.

While it is well established that obese children walk differently than nonobese children, it is not clear why or how gait mechanics change as adiposity increases. Previous studies have used body mass index (BMI) to categorize participants into obese and non-obese groups. Since BMI can be a poor predictor of pediatric adiposity (Daniels et al., 1997), the altered gait strategy could be a consequence of either excess mass or a body composition that impairs locomotor ability. Furthermore, it is not yet known if the relationship between relative adiposity (i.e. body fat percentage, BF%) and gait mechanics is continuous in children or whether there is an adiposity threshold above which gait mechanics

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change, as has been proposed in obese adults (DeVita and Hortobagyi, 2003).

Greater levels of adiposity appear to result in reduced muscle strength relative to total body mass (Blimkie et al., 1990; Shultz et al., 2010; Tsiros et al., 2013) and gait mechanics appear to be sensitive to weakness of certain muscle groups (van der Krogt et al., 2012). Therefore, understanding the muscle force requirements of walking across a range of adiposity in children should provide insight into the possible imbalance between muscle and fat mass that may be responsible for the altered gait kinematics and kinetics. Additionally, certain muscles likely have functional requirements to accelerate and reposition the body during walking that are independent of adiposity and therefore may result in greater relative muscle force requirements with increasing BF% in children.

The purpose of this study was to investigate the relationship between adiposity and lower extremity kinematics, muscle force requirements, and individual muscle contributions to the acceleration of center of mass (COM) determined from musculoskeletal simulations of walking in children. We focused our investigation on the muscles implicated in the altered mechanics reported in previous studies (Gushue et al., 2005; McGraw et al., 2000; McMillan et al., 2010; Shultz et al., 2010; Shultz et al., 2009) and which have primary roles acting to support (vasti (VAS), gastrocnemius (GAS), and soleus (SOL)), stabilize (gluteus medius (GMED)), and propel (GAS, and SOL) the whole body COM during the stance phase of walking (Anderson and Pandey, 2003; Lin et al., 2011). We hypothesized that as BF% increased: (1) knee flexion during stance would decrease while pelvic obliquity would increase; (2) peak muscle forces normalized to lean mass would increase for GMED, GAS, and SOL, but decrease for VAS; and (3) the individual muscle contributions to the acceleration of the COM in the direction of their primary function(s) would not change for GMED, GAS, and SOL, but decrease for the VAS.

2. Methods

2.1. Subjects

We used normalized frontal-plane hip joint moments between obese and non-obese children reported from prior literature (McMillan et al., 2009) and power analysis to determine that a sample size of $n=14$ would allow us to detect strong (Taylor, 1990), and meaningful correlation coefficients (e.g. $r=0.80$) with a power level of $\beta=0.95$ (SigmaPlot version 11.0, Systat Software, Inc., San Jose, CA). For this study, we defined a correlation coefficient as meaningful if greater than half of the variability in a gait measure could be attributed to adiposity. Gait analysis data from 14 children ages 8–12 years were selected from a larger study on the basis of creating a nearly continuous and even distribution of BF% from lean to obese (BF% 16–41%) (Table 1). We also analyzed subsets of 5 non-obese (BF% < 25%) and 5 obese children (BF% > 35%) children to allow comparisons between our results and previously published data. Participants were selected who did not report lower-extremity malalignment and were relatively tall, so as to minimize musculoskeletal model scaling (see below). Exclusion criteria included any neuromuscular, musculoskeletal, or cardiovascular disorder, other than obesity, impacting safe participation in the study. Prior to data collection, the study was approved by the Massey University Human Ethics Committee and informed written assent and consent was obtained from the participants and their parents, respectively.

Table 1
Subject characteristics and analyzed walking speeds (values are mean (SD)).

	N	Age (years)	Height (cm)	Mass (kg)	Lean mass (kg)	BF%	LEF%	Walking speed (m s)	Dim. less speed	Gender (# male)
All	14	10.1 (1.5)	151 (10.8)	54.9 (22.5)	36.3 (11.6)	29.6 (8.7)	45.2 (3.3)	0.96 (0.08)	0.34 (0.02)	6
Obese	5	10.6 (1.1)	157 (8.0)	77.3 (7.9)	46.7 (12.1)	37.6 (4.0)	45.2 (2.7)	0.98 (0.08)	0.34 (0.02)	2
Overweight	4	10.5 (1.9)	151 (8.5)	50.8 (8.3)	33.6 (6.1)	32.2 (3.1)	45.6 (2.3)	0.93 (0.1)	0.33 (0.03)	0
Nonobese	5	9.4 (1.6)	145 (13.5)	35.9 (21.1)	28.1 (6.2)	19.4 (2.2)	44.8 (4.9)	0.98 (0.08)	0.35 (0.02)	4

Bold denotes a significant difference between the obese and nonobese groups. BF%—body fat percentage. LEF%—percent of total adiposity present on the lower-extremities.

2.2. Experimental protocol

We quantified body composition, specifically BF% and lean tissue mass, for each subject using dual x-ray absorptiometry (DEXA, Hologic Discover, Bedford, MA). Participants walked barefoot on an instrumented treadmill at self-selected speeds. Self-selected speed was identified as the average walking speed of 5 overground trials (Blakemore et al., 2013). Participants had similar self-selected walking speeds and dynamically similar dimensionless walking speeds (Hof, 1996) (Table 1). Participants were given a familiarization period on the treadmill that lasted several minutes and was terminated upon verbal and visual confirmation of comfortable gait.

2.3. Experimental data

Three-dimensional kinematic data were collected using a 9-camera motion capture system (VICON MX System, Vicon, Oxford, UK), while kinetic data were collected using a dual-belt, force measuring treadmill (Fully Instrumented Treadmill; Bertec Corp, Columbus, OH). We used a previously reported obesity-specific marker set that was specifically developed to reduce the effects of subcutaneous adiposity obscuring the motion of the underlying skeleton (Lerner et al., 2013a) (Supplemental material).

2.4. Musculoskeletal modeling

We used OpenSim (Delp et al., 2007) to estimate the muscle activations that reproduced the walking dynamics of two representative strides for each participant. Anatomical landmarks were used to scale a generic, 12 segment musculoskeletal model with 21 degrees of freedom (DOF) and 92 muscle-tendon actuators (Delp et al., 1990) to the individual anthropometrics (i.e. total body mass and segment length) of each participant. Model DOF included a ball-and-socket joint at the third lumbar vertebra, 3 translations and 3 rotations at the pelvis, a ball-and-socket joint at each hip, single DOF tibiofemoral joints with anterior/posterior and superior/inferior translations prescribed as a function of knee flexion (Delp et al., 1990; Yamaguchi and Zajac, 1989), and revolute ankle and subtalar joints.

Inertial properties of each segment were scaled as a function of segment length and total body mass, regardless of BF%. Lower extremity joint angles were calculated using OpenSim's inverse kinematics analysis, which minimized the errors between markers on the scaled model and experimental marker trajectories. Segment masses of the pelvis, thigh and shank were adjusted during a residual reduction algorithm that minimizes the residual forces and moments acting on the model arising from dynamic inconsistency (Delp et al., 2007).

We resolved individual muscle forces from net joint moments using a weighted static optimization approach implemented in a custom OpenSim plugin (Lerner et al., 2013c; Steele et al., 2012). The objective function minimized the sum of squared muscle activations, while incorporating individual muscle weighting constants (3 for the hamstrings, 7 for the gastrocnemius, and 1 for all remaining muscles) that were previously determined by minimizing the difference between model estimated tibiofemoral forces and those measured experimentally from an instrumented knee joint replacement (Steele et al., 2012). The muscle forces predicted using static optimization are sensitive to the relative maximum isometric forces of each muscle. Therefore, because we lacked the information to scale the maximum isometric force of individual muscles (see limitations), the maximum isometric forces of all muscles in a model were scaled uniformly only if one or more muscles reached maximal activation for a given participant.

Individual muscle contributions to the acceleration of the center of mass for each simulated gait cycle were quantified using an induced acceleration analysis method described previously by Lin et al., implemented in a validated OpenSim plugin (Dorn et al., 2011; Lin et al., 2011). This method was selected because it allowed us to conveniently specify the interaction between the feet and the treadmill as 5 contact points were located geometrically around each foot. Contact conditions (i.e. constraint type) for each point are modulated based on the phase of the gait cycle determined from the ground reaction forces. While described previously in extensive detail, this methodology resolves individual muscle contributions to the acceleration of the COM by solving the equations of motion

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