## THESIS

# THE EFFECTS OF OBESITY AND DURATION ON THE ENERGETICS AND BIOMECHANICS OF WALKING IN CHILDREN 

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#### Abstract

THE EFFECTS OF OBESITY AND DURATION ON THE ENERGETICS AND BIOMECHANICS OF WALKING IN CHILDREN


INTRODUCTION: Children are encouraged to participate in at least 60 minutes of moderate to vigorous physical activity (MVPA) daily. Active transport, such as walking to school daily, may be a convenient way for children to accrue physical activity as long-duration (e.g. $>30$ minutes), moderate intensity activities have been cited as an effective way to reduce body fat. However, typical walking speeds do not elicit a moderate intensity in nonobese children and the slower self-selected walking speeds of obese children may not elicit a moderate intensity physiological response. In addition, obese children have a smaller aerobic capacity, are relatively weaker and walk with altered gait biomechanics, suggesting they may fatigue during longer bouts of walking and have a correspondingly greater risk of musculoskeletal injury when walking for physical activity. It is currently unknown how physiological intensity and gait characteristics change with walking duration in either obese or nonobese children. PURPOSE: The purpose of this study was to investigate the effect of a continuous bout of walking on the energetics and mechanics of nonobese and obese children. METHODS: Thirty-four children (21 nonobese, 13 obese) walked on a dual-belt force measuring treadmill at $1.00 \mathrm{~m} / \mathrm{s}$ for 20 minutes. Metabolic, kinematic, and kinetic data were collected at the $6^{\text {th }}, 10^{\text {th }}$, and $19^{\text {th }}$ minute of the trial. RESULTS: We found a significant effect of obesity, but not walking duration, on metabolic parameters. Obese children exhibited greater absolute and lean-mass-normalized metabolic rates, but similar mass-specific metabolic rates compared to the nonobese children. Conversely, we found an effect of walking duration on lower extremity joint angles and net muscle moments. Hip abduction angles in both
early and late stance increased, and both hip and knee extension and hip abduction net muscle moments were greater at the end versus the beginning of the trial. While the magnitude of the changes was similar between groups, we found greater absolute net muscle moments at all joints in the obese group throughout the course of the trial. CONCLUSION: This study suggests that obese children are able to accrue MVPA through engaging in walking at moderate speeds and that compared to nonobese children, obese children do not exhibit significantly greater changes in walking biomechanics during a 20-minute bout. Although our results suggest that obese children can walk at a moderate intensity for a relatively long duration, the effects of exposing the musculoskeletal system to this prolonged physical activity remain unknown.

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## CHAPTER I

## INTRODUCTION

Obesity rates have quadrupled in adults and doubled in children in the past 30 years (Ogden C. L., Carroll, Kit, \& Flegal, 2014). Obese children are more likely to have an increased risk for cardiovascular disease, prediabetes, and are likely to become obese adults, which is accompanied by a higher risk of heart disease, type 2 diabetes, cancer, stroke, and osteoarthritis (Centers for Disease Control, 2015). Children and adolescents who are obese are also at a greater risk for bone and joint problems, including lower back pain, decreased vertebral bone mineral content, slipped capital femoral epiphysis (SCFE), Blount's disease, and increased fracture risk (Wills, 2004).

Physical activity plays an important role in weight management and it is recommended that children participate in at least 60 minutes of moderate to vigorous physical activity (MVPA) per day. However, obese children are less likely to engage in vigorous intensity exercise (Dishman, 1991) and moderate intensity activities, such as walking, may be a more appropriate alternative. Low to moderate intensity, relatively long-duration (e.g. >30 minutes) activities have been cited as promoting the greatest decrease in body fat in an obese population (LeMura \& Maziekas, 2002). Activities such as walking to school daily or engaging in other forms of active transport may be an effective way for overweight and obese children to accrue MVPA. While walking at a self-selected speed ( $\sim 1.1-1.3 \mathrm{~m} / \mathrm{s}$ ) is considered light intensity ( $<3.0$ metabolic equivalents (METS)) for nonobese children, obese children walk more slowly ( $\sim 1.0-1.2 \mathrm{~m} / \mathrm{s}$ ) (Hills \& Parker, 1991; Shultz, D'Hondt, Lenoir, Fink, \& Hills, 2014; Huang, Chen, Zhuang, Zhang, \& Walt, 2013) and have a smaller aerobic capacity (Maffeis et al., 1994). While not measured directly, estimations based on previous work describing oxygen consumption in obese
children (Lazzer et al., 2003) suggests that obese children walking at $\sim 1.1 \mathrm{~m} / \mathrm{s}$ requires an intensity of 3.5-4 METs, which would be classified as moderate activity.

While walking may be a good form of exercise for obese children, the literature regarding the effect of obesity (adult and child) on gait biomechanics and relative risk of musculoskeletal injury/pathology is inconclusive. Some studies have reported smaller peak knee flexion angles during early stance (DeVita \& Hortobagyi, 2003; Gushue, Houck, \& Lerner, 2005), greater knee extension throughout stance (McMillan, Pulver, Collier, \& Williams, 2010; Gushue, Houck, \& Lerner, 2005) and increased hip (Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; Ko, Stenholm, \& Ferrucci, 2010) and knee abduction (McMillan, Pulver, Collier, \& Williams, 2010), while others report no difference in sagittal (Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; Browning \& Kram, 2007; Lai, Leung , Li, \& Zhang, 2008; Ko, Stenholm, \& Ferrucci, 2010) or frontal plane (Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009) joint angles between obese and nonobese individuals. Similarly, there is evidence to suggest that obese adults and children walk with similar sagittal plane joint moments as their nonobese counterparts (Ko, Stenholm, \& Ferrucci, 2010; Lai, Leung , Li, \& Zhang, 2008; Gushue, Houck, \& Lerner, 2005), while frontal plane joint moments are suggested to be greater (Gushue, Houck, \& Lerner, 2005; Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009) or reduced (McMillan, Pulver, Collier, \& Williams, 2010) in obese versus healthy weight children. Altered mechanics and increased stress on the joints as a result of excess mass may increase risk for musculoskeletal pain (Taylor et al., 2006) and pathologies (Wills, 2004). For example ,increased frontal plane knee angles and moments have been implicated in the development and progression of osteoarthritis (Shultz, D'Hondt, Fink, Lenoir, \& Hills, 2014), while increased hip abduction moments have a strong relationship with the development of SCFE (Wills, 2004). In order to reduce the risk of pain,
pathology, and perhaps chronic impairment, it is important to understand how obesity affects walking biomechanics in both adults and children.

The inconclusive biomechanical differences attributable to obesity reported in the literature may be due to the fact that many studies did not use methods that account for excess adipose tissue over bony landmarks (DeVita \& Hortobagyi, 2003; Hills \& Parker, 1991; Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991). Recently, methods have been described that attempt to account for the excess adipose tissue surrounding the pelvis, leading to more accurate kinematic and kinetic measures (Lerner, Board, \& Browning, 2014a). Use of these methods has revealed a significant effect of obesity on walking mechanics, specifically greater pelvic obliquity during late stance and a more extended knee during early stance in both obese adults (Lerner, Board, \& Browning, 2014b) and children (Lerner, Shultz, Board, Kung, \& Browning, 2014). Additionally, it has been shown that obese adults (Lerner, Board, \& Browning, 2014b; Haight, Lerner, Board, \& Browning , 2014) and children (Lerner, Shultz, Board, Kung, \& Browning, 2014) may walk with greater muscle forces and experience significantly greater peak tibiofemoral contact forces during level walking (Haight, Lerner, Board, \& Browning, 2014).

While walking is generally considered a low-risk activity, there is very little information how physiological intensity and gait mechanics change with walking duration in obese children. Obese children have higher relative muscular demands (Lerner, Shultz, Board, Kung, \& Browning, 2014) and while the increased muscular activation may lead to increased metabolic rate and physiological intensity, obese children also have decreased relative strength (Hulens et al., 2001), and increased muscle activation may lead to a more rapid onset of muscular fatigue in obese as compared to nonobese children. Fatigue of muscles that are important to control gait (e.g. gluteus medius (John, Seth, Schwartz, \& Delp , 2012) and quadriceps (Perry, 1992)), may
result in abnormal joint kinematics (e.g. more extended knee) and a disproportionate increase in joint loads during walking (Syed \& Davis, 2000).

The purpose of this study was to investigate the effect of a continuous ( 20 minute) bout of walking on the energetics and mechanics of nonobese and obese children. We hypothesize that a relatively slow walking speed will require a moderate intensity effort for obese children. Secondly, as obese children have greater relative muscular demands, reduced relative strength, and increased susceptibility to fatigue (Maffiuletti et al., 2007), we hypothesize that obese children will exhibit greater changes in mechanical gait parameters (e.g. peak joint angles and net muscle moments) as duration increases as compared to nonobese children.

## CHAPTER II

## LITERATURE REVIEW

## Adult and Childhood Obesity

In the wake of a rapid rise in obesity in the United States, it is estimated that one-third of children and adolescents are now considered overweight or obese (Ogden, Carrol, Kit, \& Flegal, 2014). Worldwide, over 155 million children also meet the criteria for overweight or obese (Aschemeier, Kordonouri, Danne, \& Lange, 2008), which is a BMI-z score (body mass index score adjusted for age and gender) greater than the $85^{\text {th }}$ percentile for age and gender. Obesity in children is defined as a BMI-z score above the 95th percentile (Ogden C. L., Carroll, Curtin, Lamb, \& Flegal, 2010). The increasing prevalence of childhood obesity worldwide is considered a major global public health problem.

While many factors contribute to the development of obesity, an imbalance between energy intake and energy expenditure is implicated as a major factor. A lack of physical activity is suggested to play a role in the gradual weight gain that leads to obesity, as obese children may not spend as much time being physically active as their nonobese peers (Lazzer et al., 2003; Maffeis, Zaffanello, Pinelli, \& Schutz, 1996) and may not accrue the recommended amount of daily moderate-to-vigorous-physical activity (MVPA) (Metzger et al., 2008). While this relationship is not causative per se, there is an inverse relationship between physical activity and fat mass in children (Goran, Hunter, Nagy, \& Johnson, 1997), which certainly suggests that physical activity plays an important role in maintaining an appropriate weight. The American College of Sports Medicine (ACSM) strongly advocates physical activity for the prevention and reduction of obesity. For long-term weight loss, it is recommended that adults accumulate 200300 minutes of MVPA per week (Donnelly et al., 2009). Children are advised to engage in at least 60 minutes of MVPA per day, including vigorous activity at least 3 days per week, as well
as muscle and bone strengthening activities, (i.e. jumping rope or running) on a regular basis (Physical Activity Guidelines Advisory Committee, 2008).

Being overweight or obese as a child may have psychological and social ramifications (e.g. low self-esteem) (Yanovski, 2015). As importantly, there are a number of health concerns associated with childhood obesity. Up to half of overweight and obese children meet the criteria for metabolic syndrome (Daniels et al., 2005) and have a higher risk for cardiovascular disease (CVD), type 2 diabetes, and respiratory conditions (Han, Lawlor, \& Kimm, 2010). Obese children report low health-related quality of life (Yanovski, 2015), and without intervention, obese children are likely to become obese adults, which is also associated with substantial health problems and significantly higher all-cause mortality (Flegal, Kit, Orpana, \& Graubard, 2013).

Overweight and obese children are also at risk for a number of musculoskeletal problems including knee pain, lower back pain, and are at a greater risk of developing orthopedic pathologies like slipped capital femoral epiphysis (SCFE) and Blount's Disease (Wills, 2004; Taylor et al., 2006) as well as knee osteoarthritis (OA) (Anandacoomarasamy, Caterson, Sambrook, Fransen, \& March, 2008). SCFE is a separation between the femoral head and its neck, usually due to a fracture at the epiphyseal plate. There are two mechanisms that can elicit this detachment: an acute increase in the shear force that causes the femoral head to separate or a chronically increased shear force that causes a gradual slip (Wills, 2004). The link between SCFE and obesity is suggested to be related to an increase in force on the femoral head due to increased body mass as well as altered hip mechanics (Wills, 2004). Blount's disease, also referred to as "tibia vara", is a skeletal condition in which the lower leg angles inward, similar to bowleggedness. Infantile Blount's disease is usually resolved with time, while the development of Blount's disease later in life is a response to increased stress on the medial tibial condyle
(Wills, 2004). Carrying excess mass has been linked to the development of Blount's disease in adolescents and it has been suggested that static and dynamic malalignments at the knee could lead to disproportionate increases in medial compartment joint loads and a subsequent stunting of physeal growth (Wills, 2004). These children also have a greater prevalence of skeletal fractures than their nonobese counterparts (Taylor et al., 2006) and lower relative vertebral bone mineral content (Wills, 2004). The musculoskeletal pain and pathologies associated with childhood obesity may lead to a vicious cycle of pain, inactivity, and further weight gain, which could lead to lifelong impairments.

As obesity has become a significant health problem, numerous interventions have been implemented to reduce the prevalence of obese adults and children. Unfortunately, there is little evidence to suggest that physical activity alone elicits reductions in BMI, body weight, and central obesity in overweight and obese children (Kelley \& Kelley, 2013). However, Wittmeier and colleagues found that daily physical activity consisting of at least 45 minutes of moderate intensity and 15 minutes of vigorous intensity effectively reduced both body fat and BMI (Wittmeier, Mollard, \& Kriellaars, 2008). Aerobic exercise prescriptions of 155-180 minutes per week of MVPA have been shown to be effective in reducing body fat in overweight children and adolescents (Atlantis, Barnes, \& Singh, 2006). It has also been shown that breaking daily MVPA into shorter, more frequent bouts is also an effective means to accrue sufficient daily physical activity (Seres et al., 2006) and this may be a better option for overweight and obese children, who may struggle to complete longer bouts of activity due to decreased cardiorespiratory capacity (Maffeis et al., 1994). Physical activity based interventions appear to have a doseresponse relationship, with greater physical activity goals leading to improved outcomes (Jeffrey, Wing, Sherwood, \& Tate, 2003), including greater reductions in body fat percentage in children
and adolescents (Kelley \& Kelley, 2013). Increasing energy expenditure by $6300-8400 \mathrm{~kJ} / \mathrm{week}$ (or 1500-2000 kcal/week) has been shown to effectively improve weight maintenance in adults (Fogelholm \& Kukkonen-Harjula, 2000). Together, these studies implicate physical activity as an important component in both weight reduction and weight management.

While the inclusion of vigorous activity is recommended for weight management, obese children have difficulty participating in high intensity activities (Dishman, 1991), struggle with body-weight-related tasks (Karason, Lindroos, Stenlöf, \& Sjöström, 2000; Seres et al., 2006), and may experience greater levels of perceived exertion than their nonobese peers for a given task (Bovet, Auguste, \& Burdette, 2007; Chen, Fox, Haase, \& Wang, 2006; Deforche et al., 2003). It has been suggested that overweight and obese children may reap the greatest health benefits from engaging in longer-duration (e.g. $>30$ minutes), low-intensity activities such as walking (LeMura \& Maziekas, 2002). Walking is an easily accessible form of physical activity that can elicit significant metabolic energy expenditure (Browning, McGowan , \& Kram, 2009). A number of health benefits have been associated with walking, including improvements in obesity-related conditions, such as markers of CVD, musculoskeletal problems, and metabolic health (Guinhoya, 2012; Pan \& Pratt, 2008; Janssen \& LeBlanc, 2010).

However, walking at a self-selected speed typically does not elicit a moderate intensity response of at least 3.0 METS for nonobese children and there is little information on the physiological response in obese children. It is known that walking at faster speeds (Browning, Baker, Herron, \& Kram, 2006) or uphill (Browning, Reynolds, Board, Walters, \& Reiser II, 2013) evokes a moderate to vigorous response of >3.0 METS in both nonobese and obese adults. Obese children have slower self-selected walking speeds than nonobese children (Delaxtrat, Matthew, Cohen, \& Brisswalter, 2011; Hills \& Parker, 1991; Huang, Chen, Zhuang, Zhang, \&

Walt, 2013) and while it has not been reported directly, estimations based on previous work describing oxygen consumption in obese children suggests that they may, indeed reach the $>3.0$ METS threshold at these slower walking speeds. Lazzer et al. (Lazzer et al., 2003) report that obese girls walking at $\sim 1.11 \mathrm{~m} / \mathrm{s}$ expend $24.5 \mathrm{~kJ} / \mathrm{min}$, while obese boys expend $26.4 \mathrm{~kJ} / \mathrm{min}$. Given the average weight of the two groups, these equate to approximately $12.42 \mathrm{ml} / \mathrm{kg} / \mathrm{min}$ and $14.02 \mathrm{ml} / \mathrm{kg} / \mathrm{min}$, respectively. Assuming a resting rate of oxygen consumption of $3.5 \mathrm{ml} / \mathrm{kg} / \mathrm{min}$, a relatively slow walking speed elicits a response of 3.5-4 METS, which would be classified as moderate activity.

Together, this information suggests that obese children may reap health benefits from engaging in longer duration, moderate intensity activities. Active transport, such as walking to school, may be a convenient way for overweight and obese children to accumulate daily MVPA, and has been associated with significant health benefits (Faulkner, Buliung, Flora, \& Fusco, 2009).

## Walking Metabolic Energetics

## Metabolic Rate/Energy Expenditure

Metabolic rate refers to the amount of metabolic energy expended per unit of time.
Resting and exercise metabolic rates can be obtained from measurements of oxygen consumption (VO2) and carbon dioxide production, with oxygen consumption expressed in absolute (L/min) or relative to body mass ( $\mathrm{mL} / \mathrm{kg} / \mathrm{min}$ ) terms. The metabolic rate required to walk at a given speed is typically expressed in Watts (W) and is commonly normalized to body mass ( $\mathrm{W} / \mathrm{kg}$ ). Metabolic cost, expressed as $\mathrm{J} / \mathrm{m}$, refers to the energy required to walk a given distance and can be computed by dividing metabolic rate by walking speed. As humans choose a walking speed and associated gait parameters that minimize metabolic cost, this measure can provide insight
into preferred gait biomechanics and economy. As depicted in Figure 1, there is a U-shaped relationship between metabolic cost and gait parameters, with the preferred parameters occurring in conjunction with the minimal metabolic cost and deviations from preferred characteristics in either direction inciting an increase in metabolic cost.


Figure 1 Effect of selected gait parameters on walking energetics. (A) Relationship between cost of transport and walking speed in healthy-weight adults. Adapted from Ortega \& Farley, 2005. (B) Relationship between metabolic rate and stride frequency in healthy-weight adults. Adapted from Umberger \& Martin, 2007.

It is also common to normalize metabolic cost to body mass $(\mathrm{J} / \mathrm{kg} / \mathrm{m})$, which allows for comparison between individuals of different sizes. However, normalization of metabolic rate and metabolic cost to body weight using a constant ratio method (i.e. W/kg) assumes that these increase in direct proportion to body mass. This assumption may lead to prediction errors, as the mathematical relationship between body mass and energy expenditure has a nonzero y intercept and use of the ratio method does not fully remove the effect of mass under these conditions (Poehlman \& Toth, 1995). Additionally, normalization to body mass does not take into account levels of fat-free mass (FFM), which considered is the largest contributor to metabolic rate (Nelson, Weinsier, Long, \& Schutz, 1992) and consideration of body composition may allow for more accurate insights into the effect of adiposity on metabolic cost.

## Effect of Obesity

It has been established that obese adults expend more gross metabolic energy (W) during walking than nonobese adults across a range of speeds and grades (Browning, Reynolds, Board, Walters, \& Reiser II, 2013), presumably due to their increased body mass. Obese children also have greater gross metabolic costs during walking than their nonobese peers (Butte et al., 2007). It is estimated that obese individuals expend $71-84 \%$ more gross energy $(\mathrm{W})$ than nonobese individuals, dependent on walking speed (Lazzer et al., 2003). It has also been reported that obese adults (Browning, Baker, Herron, \& Kram, 2006) and children (Lazzer et al., 2003; Peyrot, et al., 2009; Maffeis, Schutz, Schena, Zaffanello, \& Pinelli, 1993) have greater net (grossstanding) metabolic rate (W) during walking than their nonobese counterparts.

However, when normalized by body mass, there are conflicting results in the literature. It has been reported that mass-specific net metabolic rate $(\mathrm{W} / \mathrm{kg})$ is greater in obese adults at selfselected speeds when walking on a level treadmill (Browning, Baker, Herron, \& Kram, 2006) and obese adolescents have a $25 \%$ higher gross metabolic cost $(\mathrm{J} / \mathrm{kg} / \mathrm{min})$ than their nonobese counterparts walking at the same speed (Lazzer et al., 2003). Energy expenditure relative to fatfree mass is also reported to be higher in obese individuals across multiple stride frequencies (Delaxtrat, Matthew, Cohen, \& Brisswalter, 2011). In contrast, it has also been suggested that body-weight-normalized gross and net metabolic rates during walking across a range of speeds and grades are lower in obese adults (Browning, Reynolds, Board, Walters, \& Reiser II, 2013). Others have found comparable gross and net metabolic rates between obese and nonobese children walking at similar speeds after normalization to body mass (DeJaeger, Willems, \& Heglund, 2001; Lazzer et al., 2003; Maffeis, Schutz, Schena, Zaffanello, \& Pinelli, 1993) and fat-free mass (Maffeis, Schutz, Schena, Zaffanello, \& Pinelli, 1993).

Typical normalization of metabolic rate accounts only for mass; however, other anthropometric factors such as age (Morgan et al., 2002), height (Maffeis, Schutz, Schena, Zaffanello, \& Pinelli, 1993), and fat-free mass (Nelson, Weinsier, Long, \& Schutz, 1992) can influence metabolic rate. Further, while normalization to body mass allows for comparisons between individuals of varying sizes, it does not eliminate the effect of mass on metabolic rate. In an effort to more accurately evaluate walking energetics in children and account for the confounding effect of mass, Schwartz et al. developed a normalization technique that uses net metabolic rate in conjunction with normalization variables (mass, gravitational acceleration, and leg length) to create a non-dimensional metabolic measure (Schwartz, Koop, Bourke, \& Baker, 2006). Using this technique, they found that net metabolic rates during walking are $20 \%$ greater in obese versus nonobese children of the same age (Schwartz, Koop, Bourke, \& Baker, 2006).

The wide range of methodologies has led to difficulty in drawing conclusions regarding the effect of obesity on walking energetics. For example, walking speed strongly influences metabolic rate and many studies have compared obese and nonobese populations walking at selfselected speeds. Obese adults and children walk slower than their nonobese counterparts, perhaps as a mechanism to reduce metabolic cost; therefore, comparing at different speeds does not allow for a true comparison between these groups. It is also possible that obese individuals may not exhibit significantly different metabolic rates at slower speeds, but there may be an interaction between obesity and walking at faster speeds. Browning et al. found that net normalized metabolic rates are similar between obese and nonobese adults across a range of speeds and grades (Browning, Reynolds, Board, Walters, \& Reiser II, 2013), but no other studies to date have attempted to corroborate or dispute these findings. Further, there is inherent variability in metabolic measures (Rubenson et al., 2007) as a result of the physical characteristics of a
population, such as sex, age, and adiposity, the methods used for indirect calorimetry, and calculation of resting/standing metabolic rate. Future studies, using the same protocols and similar populations, are needed in order to determine how obesity affects walking energetics and properly inform exercise prescriptions for obese adults and children.

## Walking Biomechanics

Walking biomechanics are evaluated in the context of a gait cycle, which is defined as the stance-swing interval from initial contact of one foot to the subsequent contact of that same foot. Each gait cycle is divided into 2 periods, known as stance and swing. Stance begins with initial foot contact and denotes the entire period in which the foot is on the ground. This period is subdivided into three intervals - two periods of double support at the beginning and end of stance, with a period of single support in the middle of the stance period. Swing begins with the foot being lifted off the ground (toe-off) and refers to the time that the foot is in the air to be advanced forwards. Stance represents approximately $60 \%$ of the gait cycle and swing represents approximately $40 \%$, although these vary with walking velocity (Perry, 1992). As speed increases, less of the gait cycle is spent in stance but a greater percentage of stance is spent in single support; as speed decreases, the percentage of the gait devoted to stance increases and more time is spent in double support (Perry, 1992). Figure 2 provides a graphical representation of the gait cycle.


Figure 2. Divisions of the gait cycle. The dark limb represents the right leg and the light limb represents the left leg. Adapted from Perry, 1992.

The mechanics of human walking are often modeled as an inverted pendulum, with a continuous exchange of potential and kinetic energy (Margaria, 1976). In a perfect pendulum all of the energy is transferred. In human locomotion, this exchange is not perfect and mechanical work must be performed by the muscles to raise and accelerate the center of mass (COM)
(Cavagna, Saibene, \& Margaria, 1963), particularly during the double support phase (Donelan, Kram, \& Kuo, 2002b; Kuo, Donelan, \& Ruina, 2005) to transition the COM from one pendular arc to another. Energy recovery refers to the efficiency of the exchange between kinetic and potential energy; the less efficient this transfer, the more mechanical work is required to continue forward progression. In order to understand how obesity influences the metabolic cost of walking, it is important to understand the biomechanical factors that contribute to metabolic costs.

Biomechanical Contributions to the Metabolic Cost of Walking
The primary contributors to the metabolic cost of walking include the external work associated with accelerating and redirecting the center of mass, the cost of supporting weight,
and the cost associated with swinging the legs. Gait parameters, such as walking speed, stride frequency, stride length and step width can influence metabolic costs, as can deviation from an individual's preferred parameters.

## External Work

Mechanical work during walking is best evaluated as two components: external work ( $\mathrm{W}_{\text {ext }}$ ), which is the work required to raise and accelerate the COM, and internal work, or the cost of moving the limbs relative to the trunk (Willems, Cavagna, \& Heglund, 1995). During locomotion, $\mathrm{W}_{\text {ext }}$ can be calculated from the displacements and velocities of the COM and the forces applied to it, the ground reaction forces (GRFs) (Cavagna, Saibene, \& Margaria, 1963). There are two primary methods to calculate $\mathrm{W}_{\text {ext }}$ during walking: the combined limbs method (CLM) and the individual limbs method (ILM). The first approach treats the body as a point mass located at the COM and regards the actions of the legs as a single force applied to that point mass (Cavagna, Saibene, \& Margaria, 1963). This method leads to an underestimation of $\mathrm{W}_{\text {ext }}$, as it allows for the mathematical cancellation of the simultaneous positive and negative work performed by the leading and trailing legs during double support (Donelan, Rodger, \& Kuo, 2001). The ILM, on the other hand, accounts for the forces generated by each leg. Briefly, the external mechanical power for each limb is calculated as the dot product of the limb's GRF and the velocity of the center of mass (Donelan, Rodger, \& Kuo, 2001). The integration of power with respect to time results in an estimate of $\mathrm{W}_{\text {ext }}$. Restricting this to the intervals for which the integrand is positive gives an estimate of positive mechanical work; conversely, restriction to the intervals for which the integrand is negative provides an estimate of negative mechanical work (Donelan, Rodger, \& Kuo, 2001).

Approximately $50 \%$ of the metabolic cost of walking is attributable to $\mathrm{W}_{\text {ext }}$ (Grabowski, Farley, \& Kram, 2005). The amount of $\mathrm{W}_{\text {ext }}$ required is heavily influenced by changes in the position and velocity of the COM in all three planes of motion, walking speed (Willems, Cavagna, \& Heglund, 1995), vertical and horizontal work, lateral work, and obesity (Browning, McGowan, \& Kram, 2009). However, it is not possible to truly estimate metabolic costs solely from estimates of $\mathrm{W}_{\text {ext }}$, as this is a resultant measure that does not account for the effect of elastic energy in tendons, co-contractions of agonist muscles, or isometric force production, all of which can incur a metabolic cost (Umberger, 2010). For example, the transition from one step to another during the double support period has been suggested to contribute significantly to the metabolic cost of walking, due to external work requirements to redirect the center of mass (Donelan, Kram, \& Kuo, 2002a). However, there is a substantial amount of muscular work being performed during the single support phase to stabilize the limb, which incurs a significant metabolic cost, likely due to increased co-contraction of muscles (Neptune, McGowan, \& Kautz, 2009). Co-contractions and isometric force production are not accounted for in $\mathrm{W}_{\text {ext }}$ estimates, and recent musculoskeletal modeling studies have suggested that the majority ( $\sim 44 \%$ ) of total muscular energy consumption actually occurs during single limb support, while periods of double support account for a combined $27 \%$ (Umberger, 2010).

## Weight Support

During single limb support, the stance limb muscles must generate enough force to support body weight and prevent the limb from collapsing. Grabowski et al. suggest that the cost of supporting body weight accounts for approximately $28 \%$ of the metabolic cost of walking (W/kg) in nonobese individuals (Grabowski, Farley, \& Kram, 2005). It is intuitive to suggest that obese individuals incur greater metabolic costs during this period as a result of having greater
mass to support and this may explain differences in metabolic costs in obese individuals that are not explained by differences in external work (Browning, Reynolds, Board, Walters, \& Reiser II, 2013). However, Grabowski et al. only examined the addition of added weight and mass to the center of mass and it remains unclear how the addition of excess mass distributed throughout the body, as in the case of obese individuals, might influence the cost of supporting weight. Further, as obese individuals spent more time in stance than their nonobese counterparts (Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; DeVita \& Hortobagyi, 2003), it is possible that they have altered relative costs of weight support.

## Leg Swing

While $\mathrm{W}_{\text {ext }}$ represents the mechanical work required to move the COM, it does not account for the work required to accelerate the body segments with respect to the COM, known as internal work (Willems, Cavagna, \& Heglund, 1995). The cost of swinging the limbs has been suggested to be minimal in nonobese adults, approximately $10 \%$ (Gottschall \& Kram, 2005), as the motions are primarily passive and require minimal muscular activity (Griffin, Roberts, \& Kram, 2003). However, musculoskeletal models suggest that the swing phase may incur up to $30 \%$ of the total muscular cost of walking, dependent on walking speed (Umberger, 2010). The addition of excess mass near an individual's COM does not elicit an increase in the metabolic cost of swinging the limbs (Griffin, Roberts, \& Kram, 2003), but the addition of mass to the legs increases metabolic cost significantly (Browning, Modica, Kram, \& Goswami, 2007). It is likely that the cost of swinging the leg is greater in obese individuals, due to greater segment masses and increased lateral circumduction of the swing limb, which also increases metabolic cost (Donelan, Shipman, Kram, \& Kuo, 2004).

## Biomechanical Characteristics of Gait

Humans walk with a combination of gait parameters, including speed, stride length, stride frequency, and step width that minimizes the metabolic cost of transport (Zarrugh, Todd, \& Ralston, 1974). Stride length is the distance that a given foot travels between the same instance in two consecutive gait cycles, typically from initial contact to the next initial contact. Stride length is affected by physical characteristics, such as leg length, but can also be affected by pathologies. Step width is the distance between the midlines of the two feet (Bauby \& Kuo, 2000). During walking, humans prefer a step width of 0.12 L , where L is equal to leg length (Donelan, Rodger, \& Kuo, 2001). This appears to simultaneously minimize metabolic costs and maximize stability; however, wider steps are a mechanism for frontal plane stabilization (Bauby \& Kuo, 2000) and greater step widths are reported in populations that may have impaired balance, such as young children, the elderly (Dean, Alexander, \& Kuo, 2007), Parkinson 's disease patients, and the obese (McGraw, McClenaghan, Williams, Dickerson, \& Ward, 2000).

## Walking Speed

Walking speed plays a significant role in the amount of energy expended during walking. It has been shown that sagittal and frontal plane joint moments increase with walking speed, as does the rate of loading (Lelas, Merriman, Riley, \& Kerrigan, 2003; Landry, McKean, HubleyKozey, Stanish, \& Deluzio, 2007). These are suggestive of greater mechanical work by the muscles and this may contribute to increased energy expenditure during walking in both obese and nonobese individuals. In nonobese adults, there is a curvilinear relationship between walking speed and oxygen consumption. At slower speeds ( $0.8-1.4 \mathrm{~m} / \mathrm{s}$ ), this relationship is linear; as walking speed increases, the slope of the curve becomes increasingly steep, suggesting that walking becomes less economical (Katch, Becque, Marks, Moorehead, \& Rocchini, 1988).

Obese individuals exhibit a similar relationship between walking speed and oxygen consumption as do nonobese adults (Katch, Becque, Marks, Moorehead, \& Rocchini, 1988).

The amount of energy required to walk a given distance (metabolic cost) has a U-shaped relationship with walking speed, with the minimum metabolic cost coinciding with preferred walking speed. Both nonobese and obese individuals tend to select a walking speed that minimizes metabolic cost (Browning \& Kram, 2005), as do adults who have lost weight (Peyrot et al., 2012), suggesting that the minimization of energy cost is not impacted by obesity. These data suggest that minimization of metabolic costs is a primary determinant of the specific gait characteristics adopted by an individual.

## Stride Frequency

Stride frequency also has a strong influence on metabolic cost (Saibene \& Minetti, 2003), and similar to walking speed, there is a U-shaped relationship between metabolic cost and stride frequency, with the lowest cost occurring at the preferred stride frequency (Holt, Hamill, \& Andres, 1991). Varying stride frequencies elicits increases in gross normalized metabolic costs in both obese and nonobese adolescents, with no report of differences between groups (Delaxtrat, Matthew, Cohen, \& Brisswalter, 2011). Taking longer steps at a lower step frequency may elicit a greater vertical oscillation of the COM, which would lead to increased $\mathrm{W}_{\text {ext }}$ demands (Donelan, Rodger, \& Kuo, 2001; Holt, Hamill, \& Andres, 1991) or it may decrease the potential elastic energy return due to a longer stance time (Holt, Hamill, \& Andres, 1991). Lower step frequencies may also require greater use of postural muscles to maintain stability and this increase in muscular activity may contribute to increased metabolic costs (Holt, Hamill, \& Andres, 1991; Kram \& Taylor, 1990). Conversely, walking at higher step frequencies may
reduce the time available during stance to generate forces to support body weight (Saibene \& Minetti, 2003).

## Step Width

Step width has been shown to influence metabolic cost of walking, with both wider and extremely narrow steps eliciting higher metabolic costs (Donelan, Shipman, Kram, \& Kuo, 2004). Walking with wider steps is suggested to increase mediolateral GRFs and the mediolateral COM velocity, which would require greater $\mathrm{W}_{\text {ext }}$ to properly redirect the COM during the step-to-step transitions (Donelan, Rodger, \& Kuo, 2001; Peyrot et al., 2009). The mechanical work required to redirect the COM increases with the square of step width (Donelan, Rodger, \& Kuo, 2001) and implies that even slight increases in foot placement variability may increase metabolic cost (Donelan, Shipman, Kram, \& Kuo, 2004). The step width selected by humans during walking is suggested to allow for a minimization of $\mathrm{W}_{\text {ext }}$ required to redirect the COM, while still providing adequate stability (Donelan, Shipman, Kram, \& Kuo, 2004). The wider steps observed in obese populations suggests that maintaining stability may be of greater importance than minimizing metabolic cost.

## Balance

Active stabilization and balance have been suggested to incur a metabolic cost (Donelan, Shipman, Kram, \& Kuo, 2004). As the COM must also move laterally during the step-to-step transition, $W_{\text {ext }}$ must be performed to redirect it. Further, lateral movement of the swing leg to place the foot appropriately does incur a metabolic cost (Donelan, Rodger, \& Kuo, 2001). Donelan et al. found that providing external stabilization elicits a decrease in both step width and metabolic cost, perhaps due to decreases in $\mathrm{W}_{\text {ext }}$ during the step-to-step transition and decreased foot placement variability (Donelan, Shipman, Kram, \& Kuo, 2004). Obesity has been associated
with decreases in stability (McGraw, McClenaghan, Williams, Dickerson, \& Ward, 2000) and it is likely that excess adipose tissue increases the amount of muscular activity required to maintain stability (Hue et al., 2007). Following weight loss, obese adolescents exhibit improvements in balance and a reduction in metabolic cost (Peyrot et al., 2012), further corroborating the contribution of stabilization to metabolic cost during walking.

## The Effect of Obesity on Gait Parameters

Obesity-related gait adaptations have been suggested to contribute to the observed differences in walking energetics and have also been implicated in the relationship between obesity and musculoskeletal conditions, such as OA (Powell, Teichtahl, Wluka, \& Cicuttini, 2005). However, there is not yet a clear pattern of obesity-related gait adaptations, likely the result of methodological differences that make it challenging to draw conclusions. Walking speed can affect both gait kinematics and kinetics (Lelas, Merriman, Riley, \& Kerrigan, 2003), in addition to muscle activity and spatiotemporal parameters (Schwartz, Rozumalski, \& Trost, 2008). For example, obese adults walking at $1.25 \mathrm{~m} / \mathrm{s}$ have sagittal plane hip and knee angles that are similar to nonobese adults (Lerner, Shultz, Board, Kung, \& Browning, 2014). When the speed is increased to $1.5 \mathrm{~m} / \mathrm{s}$, obese adults exhibit greater knee extension angles and moments during early stance (Lerner, Shultz, Board, Kung, \& Browning, 2014). As previously noted, many studies do not make use of kinematic methodology that accounts for excess adiposity, especially at the pelvis, and this may also lend to the variability in results.

## Quantifying Joint Kinematics and Kinetics

## Joint Kinematics

Kinematic data (i.e. joint angles) are commonly determined from data collected using a motion capture system. These systems are comprised of cameras that capture the trajectories of
reflective markers placed on key anatomical landmarks. A "marker set" refers to an arrangement of markers on the body. A version of the Helen Hayes marker set (Kadaba, Ramakrishnan, \& Wootten, 1990) is commonly used in a number of research studies and for clinical purposes. This marker set was designed to have a minimal number of markers to simplify the identification of marker trajectories, allowing for easy implementation (Kadaba, Ramakrishnan, \& Wootten, 1990).

Accurate kinematic data is important not only for proper evaluation of joint angles during locomotion, but also to properly inform estimates of kinetic parameters, as discussed below. Errors in marker placement and the effect of soft tissue artifact can lead to inaccurate kinematic and kinetic data calculations (Della Croce, Leardini, Chiari, \& Cappozzo, 2005; Leardini et al., 1999; Taylor et al., 2005). Errors in marker placement at the anterior-superior iliac spine (ASIS), in particular, can lead to inaccurate estimates of the hip joint center and subsequent errors in hip and knee kinematic and kinetic calculations (Stagni, Leardini, Cappozzo, Benedetti, \& Cappello, 2000). In obese populations, markers placed on the torso may not accurately track the movement of the underlying skeleton, as these are placed on the surface of the skin and likely move with soft tissue instead of the intended landmark. Standard marker sets do not adequately account for excess adiposity, but these are still commonly used to evaluate gait biomechanics in overweight and obese populations (Lerner, Board, \& Browning, 2014a).

Lerner et al. proposed a marker set that results in more accurate estimates of the kinematics and kinetics of walking in obese individuals (Lerner, Board, \& Browning, 2014a). Briefly, this method involves placement of reflective markers on the common marker locations on both sides of the body and marker clusters placed on the thighs, shanks, and sacrum. The ASIS and iliac crest (IC) are digitally marked using a digitizing pointer and these virtual markers
defined relative to the sacral cluster in post-processing (Lerner, Board, \& Browning, 2014a). This new marker set allowed for improved tracking of pelvic motion in obese adults and more accurate estimates of muscle and joint forces, suggesting the need for researchers to account for adiposity during gait analysis, as this can lead to confounds in both kinematic and kinetic results.

## Joint Kinetics

## Joint Moments/Net Muscle Moments

Inverse dynamics is a process used to estimate the joint moments, or net muscle moments (NMMs), using kinematic data and ground reaction forces (GRFs) as inputs. This process involves modeling each segment as a "rigid body", which is assumed to have fixed inertial properties and cannot be deformed. Free-body diagrams can be drawn for each segment and all of the forces acting on the segment are represented in this diagram. To avoid indeterminacy, individual muscle forces and moments are combined as a single force and moment of force. Calculation of forces begins at a terminal segment, such as the foot, where the external forces are known or zero. Three equations of motion can be written for each segment and the forces and moments can be solved. Once the forces and moments at the distal segment are calculated, the joint reactions of the distal segment can be used to solve the equations of motions for a proximal adjacent segment, in accordance with Newton's third law (Robertson, Caldwell, Hamill , Kamen, \& Whittlesey, 2013).

While this method is not capable of quantifying the forces in individual anatomical structures, it provides an estimate of the net effect of all the internal forces and moments of force acting across a given joint or several joints (Robertson, Caldwell, Hamill, Kamen, \& Whittlesey, 2013). This approach is an easy and non-invasive way to estimate joint forces as there is a good correlation between the NMMs and tibiofemoral contact forces (Lerner, Haight, DeMers, Board,
\& Browning, 2014). Estimating these moments allows insight into neuromuscular control, joint loading, and evaluation of mechanical work and power (Browning, 2012), which have been connected to metabolic cost (Umberger \& Martin, 2007). It is common practice to normalize these moments to body mass or body mass times height, as these anthropometric factors can influence the forces produced at a joint. This normalization allows for comparisons of individuals of different sizes and statures without confounding anthropometric characteristics. However, as joints experience the absolute forces, non-normalized data may provide a more accurate representation of total stress on a joint and may be a more clinically relevant measure for evaluating conditions such as OA (Robbins, Birmingham, Maly, Chesworth, \& Giffin, 2011).

Due to its strong dependence on kinematics, inverse dynamics is highly sensitive to any errors in collecting data, such as those that result from inaccurate estimates of bony landmarks or the hip joint center (Della Croce, Leardini, Chiari, \& Cappozzo, 2005; Stagni, Leardini, Cappozzo, Benedetti, \& Cappello, 2000). As previously discussed, errors in identifying anatomical landmarks and the presence of excess soft tissue can lead to gross errors in kinetic calculations (Lerner, Board, \& Browning, 2014a). It is also important to bear in mind that the calculated moments are a resultant (net) moment at the joint based on all the musculature crossing a given joint and do not provide any insight regarding the contribution of individual muscles.

## Spatiotemporal Adaptations Associated with Obesity

Similar to obese adults (Browning, McGowan, \& Kram, 2009; Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; Browning \& Kram, 2007; DeVita \& Hortobagyi, 2003; Lai, Leung, Li, \& Zhang, 2008), obese children walk slower than their nonobese peers (Hills \& Parker, 1991; Huang, Chen, Zhuang, Zhang, \& Walt, 2013; Dufek et al., 2012; Delaxtrat,

Matthew, Cohen, \& Brisswalter, 2011). While walking speed can be affected by age (Morgan et al., 2002), typical walking speeds are $1.0-1.3 \mathrm{~m} / \mathrm{s}$ for obese children and $1.2-1.4 \mathrm{~m} / \mathrm{s}$ for nonobese children (Hills \& Parker, 1991; Huang, Chen, Zhuang, Zhang, \& Walt, 2013; Silvernail, Milner, Thompson, Zhang, \& Zhao, 2013; Shultz, D'Hondt, Lenoir, Fink, \& Hills, 2014). This slower speed is generally accompanied by shorter steps (Lai, Leung, Li, \& Zhang, 2008), greater amount of time spent in stance and a reduced time in swing (Dufek et al., 2012), longer periods of double support (Hills \& Parker, 1991; Huang, Chen, Zhuang, Zhang, \& Walt, 2013; Dufek et al., 2012), and wider steps (Hills \& Parker, 1991; Huang, Chen, Zhuang, Zhang, \& Walt, 2013; Dufek et al., 2012).

A greater step width may be the result of excess adipose tissue between the thighs or malalignment at the knee joint (Shultz, D'Hondt, Fink, Lenoir, \& Hills, 2014; de Sa Pinto, de Barros Holanda, Radu, Villares, \& Lima, 2006) forcing obese individuals to take wider steps. However, wider steps are associated with increases in stability, which is impaired in obese adults (Hue et al., 2007) and children (McGraw, McClenaghan, Williams, Dickerson, \& Ward, 2000). Similarly, increased double support time is associated with increased stability during walking (Hills \& Parker, 1991), as are slower walking speeds (England \& Granata, 2007). Slow speeds may be the product of a reduced aerobic capacity (Maffeis et al., 1994), but they have also been associated with reduced compressive and shear loading of the tibiofemoral joint and reduced rates of loading (Lerner, Haight, DeMers, Board, \& Browning, 2014). These data suggest that obese adults and children may adopt spatial and temporal gait parameters to maximize stability and reduce joint loads during walking.

## Kinematic Adaptations Associated with Obesity

Ankle and Foot
Obese adults walk with greater foot eversion (Lai, Leung, Li, \& Zhang, 2008; Messier et al., 1994), but this has not been observed in obese children (McMillan, Pulver, Collier, \& Williams, 2010; Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009). Obese children are reported to walk with flatter feet (Hills \& Parker, 1991) and a greater toe-out/ foot progression angle (Messier et al., 1994; de Souza et al., 2005) than their nonobese peers. Obese children enter plantarflexion sooner during the swing phase, which leads to a reduction in toe clearance and is likely to contribute to the flatter foot observed at ground contact (Hills \& Parker, 1991). At selfselected speeds, obese adults walk with significantly decreased plantarflexion angles during stance and swing (Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; Lai, Leung , Li, \& Zhang, 2008). At matched speeds, obese individuals exhibit greater amounts of plantarflexion than nonobese adults at toe-off and throughout stance (DeVita \& Hortobagyi, 2003). In the latter study, the obese individuals were walking faster than their preferred speed and the increase in plantarflexion at toe-off could be a mechanism to facilitate an increase in velocity and allow for greater propulsion into the swing phase. During stance, the increased plantarflexion may reflect a compensatory action to maintain balance, as the plantarflexors act to restrain the forward fall of body weight (Perry, 1992).

## Knee, Hip, Pelvis

Obese children, like obese adults (DeVita \& Hortobagyi, 2003), tend to walk with a straighter leg than their nonobese peers. They exhibit a more extended hip and knee at initial contact (McMillan, Pulver, Collier, \& Williams, 2010) and tend to maintain greater extension throughout stance (McMillan, Pulver, Collier, \& Williams, 2010; Gushue, Houck, \& Lerner,
2005). However, at self-selected speeds, there have also been no reported differences in sagittal plane hip and knee angles between obese and nonobese adults (Browning \& Kram, 2007; Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; Ko, Stenholm, \& Ferrucci, 2010; Lai, Leung, Li, \& Zhang, 2008).

Similarly, there are inconsistent results reported for frontal plane hip and knee angles. It has been reported that obese individuals have increased hip (Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009; Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991; Ko, Stenholm, \& Ferrucci, 2010) and knee abduction (McMillan, Pulver, Collier, \& Williams, 2010) versus their nonobese peers and tend to maintain a more valgus knee alignment throughout stance (McMillan, Pulver, Collier, \& Williams, 2010). Conversely, Lai et al. (2008) report greater hip and knee adduction angles, while Shultz et al. (2009) report no difference in frontal plane knee angles.

This variability can likely be attributed to methodological differences such as walking speed, but it is also highly likely that the use of marker sets that do not account for excess adipose tissue and different methods to estimate the hip joint center contribute to the inconsistent results. It is widely accepted that obese children and adults walk with wider steps and it seems intuitive that they would have subsequently have increased frontal plane knee motion. Further, genu valgum deformities are common in obese children (de Sa Pinto, de Barros Holanda, Radu, Villares, \& Lima, 2006) and this persists during gait (Shultz, D'Hondt, Fink, Lenoir, \& Hills, 2014), further suggesting that frontal plane knee and hip angles are altered with obesity.

Using the aforementioned obesity-specific marker set, Lerner et al. found increased pelvic obliquity, or pelvic tilt, in obese versus nonobese adults (Lerner, Board, \& Browning, 2014b). An increase in pelvic tilt leads to a drop of the contralateral hip joint center and in order to allow toe clearance of the swing limb, either the swing limb would need to become more
flexed or the stance limb would need to become more extended (Lerner, Shultz, Board, Kung, \& Browning, 2014). A straighter stance limb is suggested to be more metabolically economical and would likely allow for normal swing kinematics (Borghese, Bianchi, \& Lacqaunti, 1996). This increase in frontal plane pelvic motion can also negatively impact hip joint mechanics and alter the articulation of the femoral head and acetabulum, which has been implicated in SCFE (Taylor et al., 2006). Additionally, increased movement of the pelvis is likely to increase strain on the lower back; this may be one of the mechanisms behind the high prevalence of back pain in obese children (Stovitz, Pardee, Vazquez, Duval, \& Schwimmer, 2008).

## Kinetic Adaptations to Obesity

Ankle
Obese adults (DeVita \& Hortobagyi, 2003) and children (Gushue, Houck, \& Lerner, 2005; Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009) have greater absolute sagittal plane NMMs at the ankle. When body mass is accounted for, these are generally lower in obese adults (Browning \& Kram, 2007) and children (McMillan, Pulver, Collier, \& Williams, 2010; Gushue, Houck, \& Lerner, 2005), although it has conversely been reported that normalized NMMs are greater in obese adults (DeVita \& Hortobagyi, 2003).

Greater ankle inversion moments are reported in obese adults (Ko, Stenholm, \& Ferrucci, 2010; Lai, Leung , Li, \& Zhang, 2008) and in children (McMillan, Pulver, Collier, \& Williams, 2010), which are indicative of a control mechanism for medial acceleration of body mass during single support (MacKinnon \& Winter, 1993), and as obese adults tend to have a greater mediolateral COM displacement (Malatesta et al., 2009), it is not surprising that these are larger in magnitude.

The ankle plantarflexors play a significant role in forward progression (Neptune, Kautz, \& Zajac, 2001; Zajac, Neptune, \& Kautz, 2002) and ankle plantarflexion serves as a mechanism to propel the leg into swing. It has also been suggested that there is an inverse relationship between action of the hip flexors to pull the leg into swing and action of the plantarflexors to pull the leg forward (Lewis \& Ferris, 2008). Increased plantarflexor moments, such as those observed in obese adults (DeVita and Hortobaygi, 2003) may be a compensatory response to walking with a straighter leg, as a decrease in hip flexion, as observed by DeVita and Hortobagyi (2003), would necessitate an increase in plantarflexion (DeVvita and Hortobaygi, 2003) to successfully advance the swing limb (Lewis \& Ferris, 2008). Conversely, obese children are reported to walk with lower normalized plantarflexion NMMs but greater hip flexion moments at toe-off (McMillan, Pulver, Collier, \& Williams, 2010).

Observed kinematic changes at the knee, or lack thereof, may also help explain the differences in ankle angles and moments. DeVita and Hortobaygi (2003) report that obese adults walk with less knee flexion, while Browning and Kram (Browning \& Kram, 2007) report similar knee flexion between nonobese and obese adults. Levels of adiposity may also help explain the observed differences, as the plantarflexors act to restrain the forward fall of the body during walking (Perry, 1992), and greater body weight may necessitate greater muscular action to maintain balance. Many of the adults studied by DeVita and Hortobaygi (2003) had a BMI of $>40 \mathrm{~kg} / \mathrm{m}^{2}$ and it is possible that a high level of adiposity creates a greater stability challenge than that faced by nonobese or less obese adults.

It has also been suggested that reductions in hip flexion and shorter strides may allow obese individuals to accept their weight at initial contact with less plantar flexion, and this may also be a mechanism to bring the body weight over the stance limb quickly and increase stability
(Spyropoulos, Pisciotta, Pavlou, Cairns, \& Simon, 1991). Given the multiple mechanisms proposed to increase stability during walking, it is plausible that the variations observed are related to the unique challenges faced by the population being studied, whether that is a faster-than-preferred walking speed, reduced hip flexor muscle strength, or high levels of adiposity.

## Knee and Hip

Absolute knee extension moments are greater in obese adults (Browning, McGowan, \& Kram, 2009) and children (Gushue, Houck, \& Lerner, 2005); normalization of these to body mass suggests that obese individuals walk with similar knee extension moments as their nonobese peers (Gushue, Houck, \& Lerner, 2005; Ko, Stenholm, \& Ferrucci, 2010; Lai, Leung , Li, \& Zhang, 2008). The adoption of a straighter stance limb likely results in similar knee extension moments between groups and it is suggested that this is a compensatory mechanism to reduce knee extensor loads in the presence of excess mass (Gushue, Houck, \& Lerner, 2005). It has also been suggested to be the result of reduced knee extensor muscle forces (DeVita \& Hortobagyi, 2003).

While normalized sagittal plane knee moments are generally reported to be similar, there is no consistency in reports of frontal plane knee moments. It has been reported that normalized knee abduction moments are greater in obese children (Gushue, Houck, \& Lerner, 2005; Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009). Greater frontal plane moments are suggested to reflect increased stress on the medial compartment and have been implicated in the relationship between obesity and the development of OA (Powell, Teichtahl, Wluka, \& Cicuttini, 2005). However, it has also been reported that obese children have lower knee abduction moments (McMillan, Pulver, Collier, \& Williams, 2010) and that there is no difference between groups (Ko, Stenholm, \& Ferrucci, 2010).

Absolute sagittal plane hip moments are greater in obese adults (Browning \& Kram, 2007) and body-weight-normalized moments are reported to be similar in obese and nonobese adults (DeVita \& Hortobagyi, 2003; Browning \& Kram, 2007; Lai, Leung , Li, \& Zhang, 2008) and children (Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009). However, similar to the reports of frontal plane knee moments, normalized hip abduction moments have been reported to be similar between groups (Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009), lower in obese versus nonobese children (McMillan, Pulver, Collier, \& Williams, 2010), as well as greater in obese children (Gushue, Houck, \& Lerner, 2005).

There are several plausible explanations for the observed discrepancies, including walking speed, levels of adiposity, and inaccurate kinematic data. Joint kinetics are influenced by walking speed (Lelas, Merriman, Riley, \& Kerrigan, 2003) and the lack of a standard speed makes it challenging to draw comparisons between studies. Inaccurate kinematic data, inaccurate estimates of the hip joint center, and the failure to account for excess adiposity may also help explain the conflicting findings. While there is no clear consensus on relative joint loads in obese adults and children, there is agreement that absolute NMMs are greater in obese and children (Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009; Lai, Leung , Li, \& Zhang, 2008; Browning \& Kram, 2007; McMillan, Pulver, Collier, \& Williams, 2010) and increase in proportion to body mass (N/kg) (Browning \& Kram, 2007; Hortobagyi, Herring, Pories, Rider, \& DeVita, 2011). The larger absolute NMMs indicate increased stress on the joints and, as joint articulating surface area does not increase proportionately with body mass (Ding, Cicuttini, Scott, Cooley, \& Jones, 2005), absolute NMMs may be a more relevant clinical measure. In the presence of kinematic changes, this suggests that obese adults and children may be subject to abnormal
loading patterns at a higher magnitude and may be at increased risk of musculoskeletal damage, including the development of OA (Miyazaki et al., 2002; Baliunas et al., 2002).

In summary, obese individuals walk slower, take wider steps and spend more time in double support at self-selected speeds. They tend to walk with a straighter leg than nonobese individuals, exhibiting a more extended hip and knee throughout the gait cycle. Increased movement in the frontal plane has been observed at the pelvis, hip, and knee, with obese individuals exhibiting greater pelvic obliquity, hip abduction, knee abduction, and a more valgus alignment throughout stance. Absolute NMMs are reported to be greater at all joints in both the frontal and sagittal planes, due to increases in body mass. Sagittal plane knee and hip moments are generally suggested to be similar when normalized to body mass and frontal plane knee and hip abduction moments are generally reported to be greater in obese individuals, although there is no clear consensus in the literature at this time.

Muscular Adaptations Associated with Obesity
Obese adults (Hulens et al., 2001) and children (Deforche et al., 2003) are reported to have greater absolute muscular strength, but greater levels of adiposity appear to result in a reduction in strength relative to total body mass (Shultz, Hills, Sitler, \& Hillstrom, 2010; Lafortuna, Maffiuletti, Agosti, \& Sartorio, 2005; Hulens et al., 2001), particularly in the lower extremity (Maffiuletti et al., 2007; Wearing, Hennig, Byrne, Steele, \& Hills, 2006). It is also reported that obese adults (Lerner, Board, \& Browning, 2014b) and children (Lerner, Shultz, Board, Kung, \& Browning, 2014) have greater muscular force requirements during walking compared to their nonobese counterparts. Further, it has been suggested that obese individuals are subject to quicker muscle fatigue than their nonobese counterparts (Maffiuletti et al., 2007).

As gait mechanics are sensitive to the weakness of certain muscle groups (van der Krogt, Delp, \& Schwartz, 2012), this also may help explain why obesity elicits gait adaptations. The gluteus medius, in particular, is highly involved in controlling pelvic movement in the frontal plane and operates at approximately $70 \%$ of its maximal voluntary isometric contraction force in nonobese adults during gait (Rutherford \& Hubley-Kozey, 2009). As obese adults have significantly greater lean-mass-normalized force requirements, it is certainly possible that the gluteus medius could be more susceptible to overload and fatigue during walking in obese individuals, especially at higher speeds (Lerner, Board, \& Browning, 2014b). Increased adiposity has a strong positive relationship with pelvic obliquity (Lerner, Shultz, Board, Kung, \& Browning, 2014) and it has also been suggested that relative weakness and/or increased fatigue of this muscle may contribute to the altered knee joint kinematics exhibited by obese adults (Lerner, Board, \& Browning, 2014b).

Weakness of the quadriceps has been suggested to precede knee pain and disability, including OA (Slemenda et al., 1997; Lewek, Rudolph, \& Snyder-Mackler, 2004). As this muscle group aids in the control of knee motion and attenuation of impact at heel contact, reduced muscle strength of the quadriceps has been suggested to result in earlier muscular fatigue; this leads to a reduction in shock attenuation and increased loading rates at the knee (Syed \& Davis, 2000), which may contribute to accelerated damage to the articular cartilage in the knee (Slemenda et al., 1997; Slemenda et al., 1998; Lewek, Rudolph, \& Snyder-Mackler, 2004; Murdock \& Hubley-Kozey, 2012).

## Effect of Weight Loss on Gait Parameters

As obesity may lead to unfavorable gait adaptations, it is important to know if these effects can be reversed through weight loss. There is evidence to suggest that weight loss can
result in kinematic changes, with previously obese adults exhibiting gait kinematics similar to nonobese adults following weight loss (Hortobagyi, Herring, Pories, Rider, \& DeVita, 2011). Kinetic changes were also observed, including an expected decrease in absolute vertical ground reaction force but an increase in normalized vertical ground reaction force, suggesting that obese adults who lose weight might increase joint loads relative to their body mass (Hortobagyi, Herring, Pories, Rider, \& DeVita, 2011). Self-selected walking speeds, stride length, and percent of time spent in the swing phase increase following weight loss (Hortobagyi, Herring, Pories, Rider, \& DeVita, 2011). Decreased lateral leg swing has been observed in obese adolescents who lost weight ( $\sim 5 \%$ of their body mass), in addition to a decrease in stride frequency and increase in stride length (Peyrot et al., 2012). Interestingly, the net metabolic cost decreased more than was expected based on the amount of weight lost. It was proposed that this reduction could also be due to changes in lateral kinetic and vertical potential energy, single support duration, and adiposity (Peyrot et al., 2012).

## Implications for Exercise Prescription

## Relationship between Obesity-Related Gait Adaptations and Injury

The observed obesity-related gait adaptations are of concern as there is a strong positive relationship between obesity and musculoskeletal injury (Wearing, Hennig, Byrne, Steele, \& Hills, 2006) and strong positive relationships between walking speed and joint loading (Lelas, Merriman, Riley, \& Kerrigan, 2003; Lerner, Haight, DeMers, Board, \& Browning, 2014) and between joint loading and injury (Slemenda et al., 1997; Slemenda et al., 1998; Lewek, Rudolph, \& Snyder-Mackler, 2004; Murdock \& Hubley-Kozey, 2012; Syed \& Davis, 2000). It has been suggested that even walking can elicit musculoskeletal pain (Wearing, Hennig, Byrne, Steele, \&

Hills, 2006) and the repetitive impacts experienced by the joints could potentiate the development of OA in an obese population (Powell, Teichtahl, Wluka, \& Cicuttini, 2005).

Knee forces are of particular interest, due to the strong link between medial knee joint loads and OA of the knee (Wearing, Hennig, Byrne, Steele, \& Hills, 2006; D'Lima, Fregly, Patil, Steklov, \& Colwell Jr, 2012). The knee experiences compressive forces up to 3 times body weight (D'Lima, Fregly, Patil, Steklov, \& Colwell Jr, 2012) and in-vivo measurements of joint loads show that the majority (up to $88 \%$ ) of this force passes through the medial compartment of the knee (Fregly et al., 2012). Even subtle malalignments, such as a 3-5 degree increase in tibial varus alignment, can lead to significant increases (up to $50 \%$ ) in medial compartment loads (Wong et al., 2011). The increased risk of lower extremity malalignment in overweight and obese children (Taylor et al., 2006) in conjunction with increased knee abduction moments may be a contributing mechanism to the high prevalence of knee pain reported in this population (Taylor et al., 2006) or the development of skeletal disorders such as Blount's disease (Wills, 2004).

## The Effect of Fatigue

Obese children exhibit lower cardiorespiratory capacity (Maffeis et al., 1994), greater relative muscle force requirements (Lerner, Shultz, Board, Kung, \& Browning, 2014), and Mattson et al. (1997) found that obese women use a greater percentage of their VO2max as compared to women of a nonobese during walking ( $56 \%$ and $36 \%$, respectively) (Mattson, Larsson, \& Rossner, 1997). The combination of these factors may expedite the onset of fatigue in this population. Therefore, prescribing long-duration activities, even at low intensities, may be sufficient to induce significant fatigue and subsequent gait alterations (Syed \& Davis, 2000).

Several studies have attempted to study changes in gait mechanics following a fatiguing protocol. It has been suggested that quadriceps fatigue reduces knee flexion angle and peak knee extensor moment (Parijat \& Lockhart, 2008) and increases the knee adduction moment in healthy, young participants (Walter, D'Lima, Colwell Jr, \& Fregly, 2010; Murdock \& HubleyKozey, 2012). It has also been reported that lower extremity muscular fatigue does not lead to negative alterations in knee joint mechanics in healthy individuals (Longpre, Potvin, \& Malt, 2013), suggesting that other factors (e.g. repeated tissue loading) may also contribute to gait alterations.

However, there is very little information on how a longer bout of walking might influence gait kinematics and kinetics in children, obese or not. Obese children exhibit gait alterations and may be subject to greater joint loads and abnormal loading patterns. Further, it seems intuitive to suggest that obese children may fatigue more quickly than nonobese children, as they typically have a reduced cardiorespiratory capacity (Maffeis et al., 1994), higher absolute costs of exercise (Maffeis, Schutz, Schena, Zaffanello, \& Pinelli, 1993; Peyrot et al., 2009), and greater relative muscular demands (Lerner, Shultz, Board, Kung, \& Browning, 2014). Combining abnormal loading conditions with musculoskeletal fatigue through longer duration activities may exacerbate the risk of musculoskeletal injuries or further alterations in joint loading (Syed \& Davis, 2000).

At this time, there is very limited information regarding simultaneous changes in metabolic and mechanical gait parameters over the course of time. Evaluating these together allows for a better understanding of how obesity affects both the energetics and mechanics of walking. To date, only two previous studies have simultaneously examined mechanical parameters and metabolic cost in obese children. Peyrot et al. found greater net metabolic rates in
children when walking at a standard speed and associated this with greater internal work demands as a result of walking with wider steps (Peyrot et al., 2009). However, they used inertial sensors to estimate kinetic parameters as opposed to measuring them with a force platform. Huang et al. (2013) compared metabolic and mechanical parameters while children walked at a self-selected speed around a track. They report no differences in net normalized metabolic rate or mechanical efficiency; however, the obese children walked slower than the nonobese children and this may explain why there were no differences between groups. Neither of these studies examined a bout of walking lasting more than 4 minutes.

Longer bouts of walking must be studied in order to ascertain if it is relatively safe for obese children to engage in moderate intensity activity for a longer period of time and if this provides them with sufficient stimulus to accumulate MVPA. If long duration activity is going to be prescribed as an intervention for weight loss and weight management for children, it is imperative to the effects of obesity.

## Conclusion

As childhood obesity continues to be a significant problem, appropriate interventions must be developed to prevent a vicious cycle of inactivity, musculoskeletal pain and pathologies, and perpetual weight gain throughout life. Long-duration, low intensity activities are recommended for obese adults; however, there is little information to suggest that slow walking speeds provide a sufficient aerobic stimulus for children to accrue MVPA. While walking is generally considered a low risk activity, obese children walk differently than their nonobese peers and may be at a greater risk of musculoskeletal injury. It is not known how these parameters change over time and if obesity elicits disproportionate changes in mechanical parameters or in energy expenditure during a longer bout of walking. The existing body of
literature is inconsistent, highlighting a need for further research into this area to assist in properly informing pediatric exercise prescription.

## CHAPTER III

## METHODS

Experimental Protocol
Thirty-four $(\mathrm{N}=34)$ children ages $8-12$ participated in this study. During their visit, participants completed a body composition assessment via dual X-ray absorptiometry (DEXA, Hologic Discovery, Bedford, MA). Standing oxygen consumption was determined for each participant during a period of quiet standing. Oxygen consumption and biomechanics measurements were collected while participants walked on a level treadmill at $1.0 \mathrm{~m} / \mathrm{s}$ for 20 minutes. While it has been suggested that 30+ minutes of activity is beneficial for body fat reduction (LeMura \& Maziekas, 2002), a shorter bout was selected given the relatively short attention span of children when walking on a treadmill as well as with the aim of applying the findings of this study to an activity like walking to or from school.

Subjects
BMI-Z score was used to classify participants, with the obese group defined as having a BMI-Z score at or above the 95th percentile. Thirteen obese children (7 males and 6 females) and twenty-one nonobese children (10 males and 11 females) participated in this study. All participants were free of any musculoskeletal pain, injury, or other condition that might affect their gait or energy expenditure. Participants and their parents gave written and informed assent/consent that followed the guidelines of, and was approved by, the Colorado State University Human Research Institutional Review Board.

## Energetic Measurements

Metabolic rate was determined through the measurement of oxygen consumption (VO2) and carbon dioxide production (VCO2) using a portable open-circuit respirometry system
(Oxycon Mobile, Jaeger, Hoechberg, Germany) with expired gas averaged every 30 seconds. Metabolic rate during standing and for the 6th, 10th, and 19th minute of the trial were calculated as the mean of the 2 minutes prior to the time point of interest. We calculated gross metabolic rate $\left(\mathrm{E}_{\text {gross }}, \mathrm{W}\right)$ and mass-specific gross metabolic rate $\left(\mathrm{E}_{\text {gross } / \mathrm{kg}}, \mathrm{W} / \mathrm{kg}\right)$ from V02 and VCO2 using a standard equation (Brockway, 1987) and subtracted standing metabolic rate from the walking values to derive mass-specific net metabolic rate ( $\mathrm{E}_{\text {net } / \mathrm{kg}}, \mathrm{W} / \mathrm{kg}$ ) and lean-mass-specific net metabolic rate ( $\mathrm{E}_{\text {net } / \mathrm{kgLm}}, \mathrm{W} / \mathrm{kg}$ ). Resting metabolic rates (RMR) were estimated for each participant using a standard equation (Schofield, 1985). Relative aerobic intensity was determined by dividing the average energy expenditure ( $\mathrm{kcal} / \mathrm{kg} / \mathrm{hr}$ ) by the estimated RMR to determine metabolic equivalent values (METs).

## Biomechanics Analysis

Three-dimensional kinematic data were collected using a 10-camera motion capture system (Nexus, Vicon, Centennial, CO) at 100 Hz . Ground reaction forces (GRF) were collected at $1,000 \mathrm{~Hz}$ with a dual-belt, force measuring treadmill (Fully Instrumented Treadmill; Bertec Corp., Columbus, OH ). We used an obesity-specific marker set to reduce the effects of motion artifact associated with excess adiposity (Lerner, Board, \& Browning, 2014a). We placed reflective markers on the following anatomical landmarks: 7th cervical vertebrae, acromion processes, right scapular inferior angle, sternoclavicular notch, xyphoid process, 10th thoracic vertebrae, 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. We placed marker clusters (four non-collinear markers attached to a plate) on the shanks, thighs, and the sacrum to track segment motion in the sagittal, frontal, and transverse planes.. Additionally, we digitally marked the anterior-superior iliac
spines (ASIS) and iliac crests (IC) using a spring-loaded digitizing pointer (C-Motion, Germantown, MD). Forty seconds of data were collected at the $6^{\text {th }}, 10^{\text {th }}$, and $19^{\text {th }}$ minute of each trial and a minimum of 7 consecutive steps per time point in each trial were used for analysis. An acceptable step was one in which the participant walked with one foot on each force platform and there was no crossover onto the other platform. Raw marker trajectory and ground reaction force (GRF) data were digitally low-pass filtered using a fourth-order zero-lag Butterworth filter at 6 and 12 Hz , respectively.

We used the vertical GRF and a 12-N threshold to determine heel-strike and toe-off; we then determined stride parameters (stride length/frequency, double support, and duty factor (percentage of stride spent in stance)) for each trial using a custom MatLab program (Mathworks, Natick, MA). Step width was calculated as the distance between the midstance center of pressure of the right and left leg during consecutive steps of each trial. Joint angles were defined as the angle formed between adjacent segments; net muscle moments (NMMs) were defined as the moment about a joint center created by all of the soft tissues (e.g. muscles and ligaments) spanning the joint. Angles and NMMs at the hip, knee, and ankle in all three planes were calculated using Visual3D software (C-Motion; Germantown, MD). Our Visual3D model allowed for 3 rotations and 3 translations at each joint (six degree of freedom) and was scaled to each participant's height and weight. Representative values for joint angles were calculated as the average angle at a given joint across the 40 seconds of data collected for each time point ( $\sim 40$ strides); NMMs were also calculated as an average across the data collection period. All variables were normalized to represent a percentage of a stride.

## Statistical Analysis

Energetics data, kinematic data, and kinetic data were compared at the 6th, 10th, and 19th minute of the trial. We used SPSS version 22 (IBM Corp., Armonk, NY) to perform statistical analysis to test the effect of both time and obesity on metabolic and mechanical gait parameters. A t-test was used to determine significant differences in group physical characteristics. A twoway ANOVA (duration x obesity) was used to determine differences in metabolic rates, spatiotemporal gait parameters, peak joint angles, and peak joint moments. If a significant main effect was noted, post hoc comparisons using the Bonferroni method were performed. $\mathrm{P}<0.05$ indicated significance.

## CHAPTER IV

## RESULTS

Overview
We found a significant effect of obesity, but not time, on metabolic parameters. Across all time points, obese children exhibited greater absolute gross and net metabolic rates, as well as metabolic rates relative to fat-free mass (FFM). We found a significant effect of obesity on spatiotemporal parameters, with obese children taking wider steps and spending more time in double support. Joint angles and body-weight normalized NMMs at the hip and knee were affected by time, but not obesity. Absolute NMMs were greater in obese children throughout the trial.

## Subjects

Table 1. Physical characteristics of participants.

|  | Nonobese (NO) | Obese (OB) |
| :--- | :--- | :--- |
| $N$ | $21(11$ females) | $13(6$ females) |
| Age (yr) | $9.09(1.54)$ | $9.30(1.1)$ |
| Height (m) | $1.36(0.09)$ | $1.44(0.96)$ |
| Body mass (kg) | $31.05(7.02)$ | $52.57(14.31)^{*}$ |
| BMI-Z\% | $42.04(27.5)$ | $95.30(6.14)^{*}$ |
| Body Fat \% | $26.87(4.26)$ | $40.17(5.88)^{*}$ |

Values are mean $\pm$ SD; $n$, number of subjects.

* indicates significant difference between groups ( $p<0.05$ )


## Energetics

Metabolic rates are presented in Table 2. One obese participant's metabolic data was not included in analysis due to equipment malfunction during the trial.

## Effect of Duration

We found no significant effect of time on gross oxygen consumption, net oxygen consumption, $\mathrm{E}_{\text {gross }}, \mathrm{E}_{\text {net }}, \mathrm{E}_{\text {gross } / k g}, \mathrm{E}_{\text {net } / k g}$, or $\mathrm{E}_{\text {net } / k g L M}(\mathrm{p}>0.05)$. We found no interaction between time and obesity on any measured metabolic parameter ( $\mathrm{p}>0.20$ ).

## Effect of Obesity

Obese children exhibited significantly greater gross absolute oxygen consumption than the nonobese children throughout the trial ( $\mathrm{p}<0.001$ ). However, when normalized to body weight, obese children had a smaller rate of oxygen consumption (gross VO2/kg) than the nonobese group ( $\mathrm{p}<0.001$ ) and we found no difference in net VO2/kg between groups ( $\mathrm{p}=0.385$ ).

Obese children exhibited greater metabolic rate ( $\mathrm{E}_{\text {gross }}$ and $\mathrm{E}_{\text {net }}, \mathrm{p}<0.001$ for both). When normalized to body weight, there was a small but significant difference in $\mathrm{E}_{\text {gross }} / \mathrm{kg}$ (W/kg) across the duration of the trial ( $\mathrm{p}<0.001$ ), with obese children requiring less metabolic power per kilogram of body weight. We found no difference in $\mathrm{E}_{\text {net } / \mathrm{kg}}(\mathrm{p}=0.338)$. When normalized to lean body mass, we found similar $\mathrm{E}_{\text {gross } / \mathrm{kgLm}}$ between groups ( $\mathrm{p}=0.58$ ) and greater $\mathrm{E}_{\text {net } / \mathrm{kgLM}}$ in obese children ( $\mathrm{p}<0.05$ ) throughout the trial.

## Relative Aerobic Intensity

Respiratory exchange ratio (RER) values were $<1.0$ for all participants over the course of the trial. Average metabolic equivalent values (METs) were 1.66 for nonobese participants and 3.04 for the obese participants, which was significantly different ( $p<0.00$ ).

Table 2. Effect of Weight Status and Walking Duration on Metabolic Rate.

| Parameter | Nonobese (NO) |  |  | Obese (OB) |  |  |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- |
|  | 6 min | 10 min | 19 min | 6 min | 10 min | 19 min |
| $\mathrm{E}_{\text {gross }}(\mathrm{W})$ | 183.9 <br> $(6.7)$ | $180.8(6.6)$ | $177.6(6.1)$ | 263.2 <br> $(18.1) \dagger$ | 258.0 <br> $(17.3) \dagger$ | 259.9 <br> $(17.8) \dagger$ |
| $\mathrm{E}_{\text {gross/kg }}$ <br> $(\mathrm{W} / \mathrm{kg})$ | $6.0(0.2)$ | $5.9(0.2)$ | $5.8(0.2)$ | $4.9(0.2) \dagger$ | $4.8(0.1) \dagger$ | $4.8(1.0) \dagger$ |
| $\mathrm{E}_{\text {net }}(\mathrm{W})$ | 92.8 <br> $(4.9)$ | $89.6(4.9)$ | $86.4(4.1)$ | 153.1 <br> $(13.7) \dagger$ | 147.9 <br> $(12.7) \dagger$ | 149.8 <br> $(13.2) \dagger$ |
| $\mathrm{E}_{\text {net } / \mathrm{kg}}(\mathrm{W} / \mathrm{kg})$ | $3.0(0.1)$ | $2.9(0.1)$ | $2.8(0.1)$ | $2.8(0.2)$ | $2.7(0.1)$ | $2.8(0.1)$ |
| $\mathrm{E}_{\text {net } / \mathrm{kgLM}}$ <br> $(\mathrm{W} / \mathrm{kg}$ LM $)$ | $4.1(0.2)$ | $3.9(0.2)$ | $3.8(0.1)$ | $4.8(0.3) \dagger$ | $4.7(0.3) \dagger$ | $4.7(0.3) \dagger$ |

Values are mean (SEM). $\dagger=$ different from nonobese (NO)

## Biomechanics

## Spatiotemporal

## Effect of Duration

Spatiotemporal parameters are presented in Table 3. As walking duration increased, we found significant increases in stance time, as well as stride length ( $\mathrm{p}=0.003$ and $\mathrm{p}<0.001$, respectively). We also found that children walked with significantly narrower steps at the $19^{\text {th }}$ minute vs the $6^{\text {th }}$ minute of the trial $(\mathrm{p}<0.001)$. Nonobese children spent more time in double support ( $\mathrm{p}=0.003$ ) at the end versus the beginning of the trial; however, the effect size was small (Cohen's $\mathrm{d}=0.03$ ). We found no interaction between time and obesity on spatiotemporal parameters.

## Effect of Obesity

Obese children spent more time in double support ( $\mathrm{p}<0.001$ ) throughout the trial and walked with wider steps at the end of the trial ( $\mathrm{p}=0.05$ ).

Table 3. Effect of Weight Status and Walking Duration on Spatiotemporal Parameters.

| Parameter | Nonobese 6 <br> min | Nonobese 19 <br> min | Obese 6 min | Obese 19 <br> min |
| :--- | :--- | :--- | :--- | :--- |
| Step Width <br> $(\mathrm{m})$ | $0.21(0.01)$ | $0.19(0.01)^{*}$ | $0.23(0.01)$ | 0.21 <br> $(0.01)^{*} \dagger$ |
| Stance <br> Time (sec) | $0.62(0.01)$ | $0.63(0.01)$ | $0.63(0.01)$ | $0.66(0.01)^{*}$ |
| Stride <br> Frequency <br> (Hz) | $1.00(0.02)$ | $0.96(0.02)^{*}$ | $1.00(0.02)$ | $0.96(0.02)$ |
| Stride <br> Length (m) | $1.02(0.02)$ | $1.05(0.02)^{*}$ | $1.01(0.02)$ | $1.04(0.02)^{*}$ |
| Double <br> Support <br> Time (sec) | $0.20(0.02)$ | $0.22(0.02)^{*}$ | $0.26(0.02) \dagger$ | $0.26(0.1) \dagger$ |

Values are mean (SEM). * $=$ different from $6 \mathrm{~min} . \dagger=$ different from NO

## Kinematics

## Effect of Duration

As shown in Figure 3, we found a significant effect of duration on the hip abduction angle in both early and late stance ( $\mathrm{p}=0.008$ and $\mathrm{p}=0.001$, respectively). Early stance hip abduction increased by $4.3 \%$, while late stance hip abduction was $25 \%$ greater at the end of the trial. There were no other significant changes in joint angles from the beginning to the end of the trial and no interactions between duration and obesity.

## Effect of Obesity

We did not find significant group differences, with the exception of peak stance phase knee flexion angle at the 19th minute. Obese children exhibited a 5\% more extended knee (24.78 (2.08) degrees (mean (SEM))) as compared to the nonobese group (26.09 (1.91) degrees; $\mathrm{p}=0.001$ ).

## Kinetics

## Net Muscle Moments

Normalized net muscle moments (NMMs) are shown in Figure 4. We found greater absolute NMMs in obese children at all joints throughout the trial. Additionally, we found a significant effect of duration ( $\mathrm{p}<0.05$ ), but not obesity ( $\mathrm{p}=0.123$ ), on frontal and sagittal plane normalized NMMs. We did not find any interaction between duration and obesity.

## Effect of Duration

Both groups exhibited a $23 \%$ decrease in the peak ankle inversion NMM from the beginning to the end of the trial. During early stance, the peak knee extension moment increased $13 \%(\mathrm{p}=0.018)$ and peak knee abduction NMM increased $14 \%(\mathrm{p}=0.038)$. Similarly, peak hip extension during early stance increased $10 \% ~(\mathrm{p}<0.001$ ) and peak hip abduction increased by $19 \%$ ( $\mathrm{p}=0.006$ ). During late stance, both groups exhibited a $15 \%$ increase in hip extension NMMs at the end of the trial versus the beginning $(\mathrm{p}=0.004)$ and a $21 \%$ increase in peak hip abduction NMMs ( $\mathrm{p}<0.001$ ).

## Effect of Obesity

Obese children exhibited greater absolute peak NMMs at the ankle, knee, and hip in both the frontal and sagittal planes ( $\mathrm{p}>0.00$ ). Body-weight normalized moments were similar between
groups, with the exception of the peak ankle inversion moment, which was lower in the obese group at 6 minutes $(p=0.026)$.


Figure 3. Mean sagittal and frontal plane hip (A), knee (B), and ankle (C) angles. Sagittal plane angles are depicted on the left; frontal plane angles are on the right. * indicates a significant effect of time, $\dagger$ indicates a significant effect of group; $\mathrm{p}<0.005$.


Figure 4. Mean sagittal and frontal plane hip (A), knee (B), and ankle (C) NMMs. Sagittal plane NMMs are depicted on the left; frontal plane NMMs are on the right. * indicates a significant effect of time, $\dagger$ indicates a significant effect of group; $\mathrm{p}<0.005$.

## CHAPTER V

## DISCUSSION

This is the first study to simultaneously examine metabolic and biomechanical parameters during a reasonably long bout of walking in children. Our results confirm that walking at $1.0 \mathrm{~m} / \mathrm{s}$ elicits a moderate aerobic intensity for obese children and while obese children exhibit some differences in gait characteristics, they do not exhibit greater biomechanical differences across time as compared to nonobese children.

We accept our first hypothesis that walking at a moderate speed would elicit a moderate intensity effort that would allow obese children to accumulate time spent in MVPA. Relative aerobic intensity (METs) was significantly greater in obese children and met the 3.0 MET threshold for MVPA. Our results are similar to estimates based on the work of Lazzer et al. that indicate that walking at $1.11 \mathrm{~m} / \mathrm{s}$ elicits a metabolic response of 3.5-4.0 METs in obese children (Lazzer et al., 2003). Given that self-selected walking speeds are greater than $1.0 \mathrm{~m} / \mathrm{s}$ for both normal weight and obese children (Hills \& Parker, 1991; Huang, Chen, Zhuang, Zhang, \& Walt, 2013) and that metabolic rate increases with walking speed, our results support the recommendations that engaging in regular walking physical activity, including active transport, could be beneficial for physiologic health outcomes and weight management in obese children.

The obese participants exhibited greater $\mathrm{E}_{\text {gross }}$ and $\mathrm{E}_{\text {net }}$ than the nonobese group, which is consistent with reports of greater absolute metabolic energy expenditure in obese versus nonobese individuals throughout the literature (Browning, Reynolds, Board, Walters, \& Reiser II, 2013; Browning, Baker, Herron, \& Kram, 2006; Butte, et al., 2007; Maffeis, Schutz, Schena, Zaffanello, \& Pinelli, 1993; Peyrot et al., 2009). Egross/kg was comparable between groups, similar to previous reports in obese and nonobese children (Lazzer et al., 2003; Maffeis, Schutz, Schena,

Zaffanello, \& Pinelli, 1993; DeJaeger, Willems, \& Heglund, 2001) and our findings of similar $\mathrm{E}_{\text {net } / \mathrm{kg}}$ are in agreement with previous findings in obese adults (Browning, Baker, Herron, \& Kram, 2006; Browning, Reynolds, Board, Walters, \& Reiser II, 2013) and adolescents (Delaxtrat, Matthew, Cohen, \& Brisswalter, 2011). The obese participants exhibited greater $\mathrm{E}_{\text {net } / \mathrm{kgLM}}$ than the nonobese group, which is not surprising as the skeletal muscle must generate sufficient force to move larger amounts of metabolically inert tissue.

However, our results are in contrast to those reporting increased mass-specific metabolic rates in obese versus nonobese children (Peyrot et al., 2009; Lazzer et al., 2003). Similar massspecific metabolic rates have been reported in obese versus nonobese adults and children during walking at self-selected speeds (Huang, Chen, Zhuang, Zhang, \& Walt, 2013; Delaxtrat, Matthew, Cohen, \& Brisswalter, 2011), while walking at faster matched speeds appears to elicit increased mass-specific metabolic rates in obese children (Peyrot et al., 2009; Lazzer et al., 2003). In the current study, obese and nonobese children walked at a speed that was slower for than self-selected speeds for either group and exhibited similar mass-specific metabolic rates. Together, these data are suggestive of an interaction between obesity, walking speed, and energy expenditure, although the mechanism is not currently well understood. External work required to raise and accelerate the center of mass has been shown to contribute significantly to the metabolic cost of walking (Grabowski, Farley, \& Kram, 2005) and increases with walking speed (Browning, McGowan, \& Kram, 2009); however, it has been shown that obesity does not result in increased external work requirements relative to body mass (Browning, McGowan, \& Kram, 2009), suggesting that other factors contribute to the differences in normalized metabolic rates. Increased internal work associated with swinging more massive limbs may be increased at faster speeds and result in increased energy expenditure. Similarly, less time spent in stance at faster
speeds (Perry, 1992) may present a significant balance challenge to obese adults, who are reported to be less stable during gait (McGraw, McClenaghan, Williams, Dickerson, \& Ward, 2000) and would need to increase muscle activation to maintain stability, leading to increased energy expenditure without an obligatory increase in external work requirements.

We did not find a significant effect of obesity on joint angles or mass-normalized NMMs, with a few exceptions; we therefore partially reject our second hypothesis. We found a significant effect of time on step width and time in double support: both groups walked with narrower steps and nonobese children spent more time in double support at the end versus the beginning of the trial. The reduction in step width may be the result of familiarization with the split-belt treadmill used in the current study and this reduction would lead to the observed decrease in ankle inversion moments over the course of the trial. Across all time points, obese children maintained a greater step width than nonobese children, which is suggested to be a compensatory mechanism to achieve increased frontal plane stability (Bauby \& Kuo, 2000). Wider steps have been reported in populations with balance impairments (Kao, Dingwell, Higginson, \& Binder-Macleod, 2014), including obese individuals (McGraw, McClenaghan, Williams, Dickerson, \& Ward, 2000), while external stabilization resulted in the spontaneous adoption of a narrower step width (Donelan, Shipman, Kram, \& Kuo, 2004). However, wider steps elicit increases in the metabolic cost of walking (Donelan, Shipman, Kram, \& Kuo, 2004), presumably due to greater mediolateral displacement of the COM and subsequent increases in muscular work to redirect the COM during step-to-step transitions (Donelan, Rodger, \& Kuo, 2001; Peyrot et al., 2009). The increase in muscular work to redirect the COM as a result of wider steps may help explain the greater relative force requirements of the gluteus medius (Lerner, Shultz, Board, Kung, \& Browning, 2014) and the greater energy expenditure observed
in obese children. Increased time in double support has been suggested to increase stability and reduce metabolic cost (Hills \& Parker, 1991); it is possible that increases in double support time and step width occur simultaneously in obese adults and children in order to maximize stability while minimizing metabolic cost.

We did not find a significant effect of either time or obesity on knee joint angles, with the exception of greater peak knee extension during early stance at the end versus the beginning of the trial and greater peak knee extension in obese versus nonobese children at the end of the trial. The adoption of a straighter stance limb has been reported in both obese adults (DeVita \& Hortobagyi, 2003) and children (Gushue, Houck, \& Lerner, 2005) and is suggested to be a mechanism to reduce knee joint moments in the sagittal plane (DeVita \& Hortobagyi, 2003). However, both the obese and nonobese groups exhibited greater knee extension NMMs at the end versus the beginning of the trial. As knee flexion serves to absorb the weight of the body during early stance (Perry, 1992), a more extended knee during this portion of the gait cycle elicits increases in vertical GRFs (Cook, Farrell, Carey, Gibbs, \& Wiger, 1997), which would lead to subsequently greater NMMs, indicating greater compressive joint forces.

Additionally, while it appears that frontal plane knee angles in late stance are different between groups, there was considerable variability and these differences did not reach statistical significance. Previous work suggests that obese children exhibit greater peak knee abduction angles (Gushue, Houck, \& Lerner, 2005) and tend to maintain a more valgus alignment during stance (McMillan, Pulver, Collier, \& Williams, 2010). Walking with wider steps may lead to increased frontal plane knee motion, and while not directly measured in the present study, the greater knee abduction angle in late stance may be the result of increased pelvic obliquity, which is greater in late stance in both obese adults (Lerner, Board, \& Browning, 2014b) and children
(Lerner, Shultz, Board, Kung, \& Browning, 2014). Regardless of the cause, increased knee motion in the frontal plane has been implicated in the development of osteoarthritis (Shultz, D'Hondt, Fink, Lenoir, \& Hills, 2014) and is suggestive of altered loading conditions.

We found small but statistically significant increases and sagittal plane hip angles and NMMs over the course of the trial. Increased sagittal plane NMMs are suggestive of increased compressive forces on the joints, which have been implicated in the development of musculoskeletal conditions, such as chondromalacia or OA (Slemenda et al., 1997; Slemenda et al., 1998; Murdock \& Hubley-Kozey, 2012; Lewek, Rudolph, \& Snyder-Mackler, 2004; Syed \& Davis, 2000; Powell, Teichtahl, Wluka, \& Cicuttini, 2005), especially in obese individuals (Powell, Teichtahl, Wluka, \& Cicuttini, 2005; Syed \& Davis, 2000). We did not find any group differences, in contrast to reports of lower hip extension moments at initial contact and greater hip flexion moments during late stance in obese children (McMillan, Pulver, Collier, \& Williams, 2010). However, we are confident in the accuracy of our results, as our methods for kinematic data collection are designed to reduce the effects of motion artifact associated with excess adipose tissue and our findings are similar to previous reports in both obese adults (Browning \& Kram, 2007) and children (Nantel, Brochu, \& Prince, 2006).

Greater hip abduction angles and normalized NMMs were observed at the end versus the beginning of the trial, suggesting that joint loads increase and become more medially distributed as walking duration increases. Increased hip and pelvic motion in the frontal plane in obese children may support previous research identifying greater gluteus medius force requirements in obese children (Lerner, Shultz, Board, Kung, \& Browning, 2014). As the gluteus medius operates at approximately $70 \%$ of its maximal voluntary isometric force (Rutherford \& HubleyKozey, 2009) during walking, it is plausible that this muscle is most susceptible to fatigue during
longer bouts of activity, especially for obese children. The increase in pelvic obliquity may induce strain on the lower back and may contribute to the increased prevalence of back pain in overweight and obese children (Stovitz, Pardee, Vazquez, Duval, \& Schwimmer, 2008). Further, increased hip abduction, especially in combination with excess mass, has been linked to increased shear forces at the femoral epiphysis and development of slipped capital femoral epiphysis (SCFE) (Wills, 2004).

Increased NMMs are indicative of increased muscular activation to produce greater muscle forces and it would logically follow that increased muscular activity would elicit an increase in metabolic cost. Interestingly, we did not find a significant increase in our energetic measures, suggesting that both nonobese and obese children adopt strategies to minimize metabolic cost during walking. Increased time in double support (Hills \& Parker, 1991) and the adoption of narrower steps (Donelan, Kram, \& Kuo, 2001) have been implicated in reducing metabolic costs during walking; it is possible that the observed changes in these parameters served to attenuate metabolic costs over the course of the trial.

The lack of group differences suggests that joint loads do not increase disproportionately in obese children as walking duration increases. Due to their increased body weight, obese children did exhibit greater absolute NMMs, consistent with previous findings in adults (Browning \& Kram, 2007; Lai, Leung , Li, \& Zhang, 2008) and children (McMillan, Pulver, Collier, \& Williams, 2010; Shultz, Sitler, Tierney, Hillstrom, \& Song, 2009). As absolute NMMs are reflective of the total load placed on a joint, it is still possible that overweight and obese children are at a higher risk of musculoskeletal injury. Overweight children have lower bone mineral content relative to area (Wills, 2004) and it has been suggested that articulating surface areas do not increase proportionally with body mass (Ding, Cicuttini, Scott, Cooley, \& Jones,
2005). Further, even mild malalignment or alterations in joint load distribution in combination with excess weight may be sufficient to contribute to the development of musculoskeletal problems in overweight and obese children (Taylor et al., 2006).

A limitation of this study was the use of a single walking speed for comparison, as gait kinematics and kinetics are sensitive to walking speed (Lelas, Merriman, Riley, \& Kerrigan, 2003). Obese adults walking at slower speeds do not exhibit the same alterations in gait as when they walk at faster speeds (Lerner, Board, \& Browning, 2014b); it is possible that we did not see more significant differences between groups due to the relatively slow walking speed. We were also limited by an inability to stratify for levels of adiposity. DeVita and Hortobagyi (2003) found that a BMI of greater than $30 \mathrm{~kg} / \mathrm{m}^{2}$ was associated with greater kinematic changes in obese adults and Lerner et al. (Lerner, Shultz, Board, Kung, \& Browning, 2014) found that a positive relationship between body fat percentage and pelvic obliquity in children. It is possible that higher levels of adiposity elicit greater changes in gait biomechanics, which we could not determine in the present study. Finally, we cannot disregard the fact that walking on a treadmill may have been a novel experience for these children; however, it has been suggested that young children are able to acclimate to treadmill walking quickly (Tseh et al., 2000) and gait mechanics are similar between treadmill and overground walking (Matsas, Taylor, \& McBurney, 2000; Riley, Paolini, Della Croce, Paylo, \& Kerrigan, 2007).

As the prevalence of childhood obesity remains high, it is imperative to better understand how obesity affects mechanical and metabolic aspects of locomotion in children. The current study utilizes methods that account for excess adiposity around the pelvis, which allows for more accurate kinematic data collection in an obese population, and the results suggest that, at moderate speeds, obese children do not exhibit gait alterations that are significantly different
than nonobese children. However, in order to properly inform pediatric exercise prescription, future studies are needed to fully understand how obesity affects metabolic and mechanical parameters across a range of walking and running speeds, as well as other common physical activities. Additionally, there is a need for longitudinal studies that examine the relationship between obesity-related gait adaptations during childhood and the development of musculoskeletal pain or pathologies that may impair physical activity throughout their lives.

## Conclusion

The results of this study suggest that obese children are able to accrue MVPA through engaging in walking at moderate speeds. Obese children exhibited significantly different spatiotemporal parameters and greater absolute net muscle moments, but changes in mechanical gait parameters (joint angles and mass-normalized NMMs) over a 20 minute bout of walking were similar between obese and nonobese children. Although our results suggest that obese children can walk at a moderate intensity for a relatively long duration, the effects of exposing the musculoskeletal system to this prolonged physical activity remain unknown.

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