THE IMPLICATIONS OF CHILDHOOD OBESITY ON THE MUSCULOSKELETAL AND LOCOMOTOR SYSTEMS: BIOMECHANICAL ANALYSES AND EXERCISE INTERVENTION

by

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Abstract

This dissertation used both cross-sectional and intervention studies to investigate the implication of childhood obesity on the musculoskeletal system during locomotion. Specifically, it compared the gait biomechanical and energetic differences between obese and normal children. Two different intervention studies (weight loss and muscle strength training) were carried out to investigate the cause-and-effect relationship between body mass, muscle strength and gait strategies. In addition, this study applied subject-specific musculoskeletal modelling and simulation to quantify compressive tibiofemoral force and individual muscle function. These results suggested that the excess body mass plays a dominant role in a larger joint load and greater energy cost in obese children. The obese children preferred to walk at a speed that has minimal energy cost and maximal mechanical efficiency. Weight loss reduced the differences in gait kinetics and energetics between obese and normal weight children, but the change of spatiotemporal gait parameters is not a necessary outcome for a short-term weight loss. By contrast, obese children walked with a faster self-selected speed and a larger hip flexor moment after an eight week strength training program. This finding suggested that lower extremity muscle strengthening improves obese children's ability to promote locomotion through greater propulsion. However, it may not reverse the impact of excess body weight on natural walking biomechanics and energetics. A further simulation study was carried out to investigate the knee joint reaction force and muscle function in obese children using subject-specific model. The simulation results showed that obese children and normal weight children use similar muscles to support and accelerate body COM, but normal weight children had significantly greater normalized compressive tibiofemoral force and individual muscle contribution to COM. The absolute compressive tibiofemoral force and muscle forces were still greater in obese children. Therefore, the obese child may adapt a compensation gait strategy to avoid increasing compressive tibiofemoral force and muscle requirements during walking.

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List of Abbreviations

BMI	Body mass index
COM	Centre of mass
WHO	World Health Organization
CDC	Centers for Disease Control and Prevention of the United States
IOFT	International Obesity Task Force
MRI	Magnetic resonance imaging
DEXA	Dual energy X-ray
ANOVA	Analysis of variance
SPSS	Statistical Package for the Social Sciences
OA	Osteoarthritis
MEE	Mechanical energy expenditure
ME	Mechanical efficiency
LED	Light-emitting diode
PWM	Pulse width modulation
3D	Three-dimensional

Chapter 1: Introduction

1.1 BACKGROUND

Childhood obesity has become a major public health problem (Karnik & Kanekar, 2012). While the cardiovascular and metabolic consequences of obesity have been studied extensively, less attention has been paid to the impact of obesity on locomotor functions and musculoskeletal health (Tsiros, Coates, Howe, Grimshaw, & Buckley, 2011). Studies have shown obesity is associated with more musculoskeletal discomfort, injuries, inefficient body mechanics and further reductions in mobility (Wearing, Hennig, Byrne, Steele, & Hills, 2006a). Even worse, persistent weight-bearing associated with skeletal structure changes may develop certain musculoskeletal disorders at a younger age (Ding, Cicuttini, Scott, Cooley, & Jones, 2005; Messier, 1994). The chronic pain and disability associated with musculoskeletal conditions not only significantly affects an individual's quality of life, but also results in high medical cost to the public health system (Anandacoomarasamy, Caterson, Sambrook, Fransen, & March, 2008).

Treating obesity through physical activities is an important strategy. However, multiple musculoskeletal discomfort and pain associated with physical activity may seriously decrease motivation to exercise and limit the performance of obese children during locomotion (Hootman et al., 2001; Shultz, Anner, & Hills, 2009). When obesity is combined with increased musculoskeletal pain or disorders, the cycle of obesity would be perpetuated by encouraging sedentary behaviour for prolonged periods. Therefore, it is particularly important to identify the associations among childhood obesity, physical inactivity, and musculoskeletal conditions.

Walking is one of the most common physical activities in daily life, and has been widely used in weight management programs. Gait kinematic and kinetic differences between obese and normal weight children have been well documented (Browning & Kram, 2007; Hills & Parker, 1991a; McGraw, McClenaghan, Williams, Dickerson, & Ward, 2000; Runhaar, Koes, Clockaerts, & Bierma-Zeinstra, 2011). Knowledge of mass-driven mechanics during locomotion is helpful for the planning of treatments to improve musculoskeletal health for obese individuals. Attention has been given to the metabolic cost of walking. Children consume a

considerable amount of metabolic energy for walking compared to other activities in daily life (Shultz, Browning, Schutz, Maffeis, & Hills, 2011). The investigations of walking energetics may provide insights into the gait strategy of obese children (Shultz et al., 2011), and further explain the greater difficulty obese populations have in performing locomotor tasks and the decreased motivation to physical activity (Wearing et al., 2006). However, the relationship between gait strategy and musculoskeletal problems has not been demonstrated indisputably in obese children. More comprehensive research integrating the analysis of gait biomechanics and energetics is needed to determine the short- and long-term musculoskeletal consequences on obese children. Although there have been a number of experimental studies devoted to uncovering the role of excess body mass on musculoskeletal health (Wearing et al., 2006), less is known about the cause-and-effect relationship between excess body mass, gait biomechanics and energetics, especially in children. It is also not clear how muscle strength contributes to gait characteristics in obese individuals.

1.2 PURPOSES

The general purpose of this project was to better understand the implication of childhood obesity on the musculoskeletal system during locomotion, by examining the natural walking biomechanics and energetics. Four studies were conducted to address the research questions. The specific aims and hypotheses of this dissertation are:

1.2.1 Walking biomechanics and energetics differences between obese and normal weight children

The purpose of this study was to investigate the effect of childhood obesity on the gait biomechanics and energetics. This study quantified gait characteristics, joint kinematics, kinetics and mechanical efficiency while self-selected speed walking.. It was hypothesized that obesity would significantly influence the kinematics and kinetics of normal gait. These gait alterations in obese children would minimize the mechanical and metabolic energy cost to accommodate their excess body weight during walking.

1.2.2 The effects of weight loss on walking biomechanics and energetics

The purpose of this study was to investigate the cause and effect between excess body mass, gait biomechanics and walking energetics. This study quantified gait kinematics, kinetics, mechanical and metabolic energy cost during walking after substantial weight loss in a short period of time. It is hypothesized that the reduction in metabolic cost and mechanical work after weight loss is associated with changes in the biomechanical parameters of walking.

1.2.3 The effects of strength training on walking biomechanics and energetics

The purpose of this study was to determine whether muscle strengthening in obese children could reverse the impact of excess body weight on natural gait biomechanics and energetics. This study also quantified gait kinematics, kinetics, mechanical and metabolic energy cost during walking after an 8-week lower extremity strength training program in obese children.

1.2.4 The application of musculoskeletal modelling and simulation

The purpose of this study was to utilize musculoskeletal modelling and forward simulation to investigate the gait strategy of obese children at the musculoskeletal level. The tibiofemoral force was calculated to investigate the relationship between obesity and knee joint loading. By analysing individual muscle function, this study compared the mechanisms that how individual muscles contribute support and progression accelerations of COM and joint kinematics between obese and normal weight children during normal walking.

1.3 DEFINITION OF TERMS.

Child: Individuals aged 2-18 years old. However, all the participants in this study aged 8-12 years old.

Body mass index (BMI): BMI equals body weight (kg) divided by the square of height (m^2). It is an indirect screening tool for measuring fat to assist in identifying weight categories.

Obese children: Children with a BMI above the international cutoff points for age and gender for overweight and obesity. World Health Organization (WHO) (de Onis, 2007), Centers for Disease Control and Prevention (CDC) of the United States (Ogden et al., 2002), and International Obesity Task Force (IOTF) (T J Cole, Bellizzi,

Flegal, & Dietz, 2000) each have definitions of overweight and obesity in children and adolescents . This study used the IOTF cutoff points for BMI for obese children to define obesity in all participants.

Table 1.1 International cutoff points for BMI for overweight and obesity by sex between 8 and 12 years in this study (Cole et al., 2000).

	Overweight		Obese	
Age (years)	Male	Female	Male	Female
8.00	18.44	18.35	21.60	21.57
8.50	18.76	18.69	22.17	22.18
9.00	19.10	19.07	22.77	22.81
9.50	19.46	19.45	23.39	23.46
10.00	19.84	19.86	24.00	24.11
10.50	20.20	20.29	24.57	24.77
11.00	20.55	20.74	25.10	25.42
11.50	20.89	21.20	25.58	26.05
12.00	21.22	21.68	26.02	26.67
12.50	21.56	22.14	26.43	27.24

Musculoskeletal and locomotor system: It is made up of bones, muscles, cartilage, tendons, ligaments, joints, and other connective structures of the human body, that supports and binds tissues and organs together. The primary functions of the musculoskeletal and locomotor system are supporting the body, allowing motion, and protecting vital organs.

Acanthosis nigricans: A skin condition in that results in which darker, thick, velvety skin in body folds, such as neck, armpits, groin, navel, and other area.

Hypertension: High blood pressure.

Slipped capital femoral epiphysis: A paediatric and adolescent hip disorder that relate to the posterior and inferior displacement of the femoral neck.

Tibia vara: A growth disorder that causes the lower leg bone to curve inward.also known as bow legs.

Genu valgum: A knee angle that is abducted, also known as knock-knee.

Planes of the body： Movements descriptions are based on three imaginary planes that pass through the body in the anatomical position: the sagittal plane, divides the body into right and left halves; the frontal plane, divides the body into anterior and posterior halves; the transverse plane, divides the body into superior and inferior halves.

Inverse dynamics: The solution of algebraic equations for the joint torques (forces) that result from the substitution of system kinematics (positions, velocities and accelerations estimated from observations) into the equations of motion.

Forward dynamics: Forward dynamics is the application of forces (or torques) to evaluate the instantaneous acceleration of the system, in which case the equations of motion describe a set of ordinary differential equations. Integrating the second order system forward in time yields the velocity and positions of system coordinates (angles, translations) throughout the performance period to generate a forward (in time) simulation of human performance.

Gait analysis: A systematic study of human walking, using the eye and brain of experienced observers, augmented by instrumentation for measuring body movements (Whittle, 2007).

Gait cycle: The time interval between one foot contacting the ground (heel strike) and the same foot contacting the ground again (another heel strike) in walking. A gait cycle is also referred to as a stride.

Stance phase: The phase of the normal gait cycle that begins with the heel strike and ends with the ipsilateral toe-off.

Swing phase: The phase of the normal gait cycle during which the foot is off the ground.

Toe off (push off): The toe off of the normal gait begins as the toes leave the ground at the start of the swing phase.

Kinematics: The data describe the position of key landmarks (such as bony processes, spines, estimates of joint centres and COM locations) as they move in time and space.

Kinetics: The amount of force (magnitude and in what direction) the body imparts on the ground and how the ground reacts to accelerate the body.

Centre of mass: The point on a body that moves in the same way that a particle subject to the same external forces would move (Rodgers & Cavanagh, 1984).

Joint moment: The turning effect produced by joint forces (the result of muscle forces, gravity, and inertial forces). The calculation is the product of the force and the perpendicular distance between the line of application of the force and the axis of rotation (Rodgers & Cavanagh, 1984).

Power: The energy consumption per unit time. The unit of power is Watt or J/s.

Mechanical efficiency: The ratio of the mechanical work done to the metabolic energy expended.

Metabolic rate: The amount of metabolic energy expended per unit body weight per unit distance (J/kg/m).

Mechanical energy expenditure: The amount of mechanical work expended during movement over a period of time (J).

Equations of Motion: A set of mathematical equations obtained by using Newton's second law to equate, which describe the forces and movements of a body.

1.4 THESIS OUTLINE

This chapter briefly introduces the research purposes of this thesis. The next chapter reviews related work and describes the background of this study. Chapter 3 compares the differences in the gait biomechanics and energetics between obese and normal weight children. Chapter 4 and Chapter 5 present the effects of weight loss and muscle strengthening on gait differences observed in Chapter 3. Chapter 6 applies the computational modelling and simulation to provide insight into the knee internal loading and muscle functions during normal walking. The final chapter (Chapter 7) summarizes the key findings and the contributions of this study, and discusses possible future directions of this work.

Chapter 2: Literature Review

2.1 PREVALENCE OF CHILDHOOD OBESITY

The prevalence of obesity among children has increased dramatically in both developed and developing countries for the past few decades (Ebbeling, Pawlak, & Ludwig, 2002). In the United States, childhood obesity has been more than tripled in the past 30 years. The number of obese children aged 6 to 11 years has increased over 13% between 1980 and 2008, and more than 17% of children and adolescents are obese (Ogden, Carroll, Kit, & Flegal, 2012). The prevalence of obesity among Australian children has accelerated since the early 1970s and currently about 13% of all 2 to 16 years old children and adolescents were classified as obese (Norton & Dollman, 2006). The number of European school-age children who are suffering overweight and obesity is also rising (Lobstein, Baur, & Uauy, 2004). The rapid economic growth within the past decades has led to a greatly increased prevalence of obesity in developing countries. In China, the prevalence of childhood obesity increased from 0.2% in 1985 to 8.1% in 2010 (Sun et al., 2013). In Brazil, the prevalence of overweight among children aged years more than tripled between 1974 and 1997, while the prevalence of underweight children significantly decreased (Wang & Lobstein, 2006).

Although recent reports have noted stabilisation in obesity rates internationally (Jouret et al., 2007; Cynthia L Ogden, Carroll, & Flegal, 2008; Olds, Tomkinson, Ferrar, & Maher, 2010), childhood obesity is still recognized as a serious public health concern due to its high prevalence (Karnik & Kanekar, 2012). A large number of cross-sectional studies have shown that childhood obesity is a chronic paediatric disease with potentially immediate and future consequences (psychological, metabolic, and orthopaedic conditions) involving different body systems (Batch & Baur, 2005). Obesity is associated with an increased risk of developing insulin resistance, which may further progress to type 2 diabetes (Kahn, Hull, & Utzschneider, 2006). Acanthosis nigricans is an important predictor of the insulin resistance in childhood obesity. Obese children with acanthosis nigricans have a higher insulin and the degree of insulin resistance (Guran, Turan, Akcay, & Bereket, 2008). Childhood obesity may also associate with liver disease, hypertension, and

increased lipid levels (Dietz, 1998; Ebbeling, Pawlak, & Ludwig, 2002). In addition, obese children report more frequent and severe musculoskeletal pain and injuries (Wearing, Hennig, Byrne, Steele, & Hills, 2006b). The lower extremity malalignment (e.g. tibia vara, genu valgum) is more prevalent in overweight and obese children, and less mobility and physical function are observed (Shultz, Anner, & Hills, 2009). As childhood obesity is an important factor in the persistence of obesity into adulthood, these consequences are more likely to cause many future diseases in their later life (Reilly et al., 2003). A 40 year follow-up study found the obese children had significantly higher risks of many diseases in different body systems (Mossberg, 1989).

2.2 IMPLICATIONS OF CHILDHOOD OBESITY ON LOWER LIMB MUSCULOSKELETAL FUNCTION

Obese children may be at risk for both short term health consequences and long term tracking of obesity in adulthood (Singh, Mulder, Twisk, van Mechelen, & Chinapaw, 2008). While the cardiovascular and metabolic consequences of obesity have been studied extensively, less attention has been paid to the impact of obesity on musculoskeletal functions, including the functions of joints, muscle, tendons, ligaments and other bodily tissue (Wearing et al., 2006b). Despite significant advances in the knowledge about the effect of childhood obesity on skeletal structure and alignment, the implications of persistent obesity on the musculoskeletal and locomotor systems during weight bearing tasks are still poorly understood (Shultz et al., 2009; Wearing et al., 2006).

Due to the excess mass in overweight and obese children, there are increases in the absolute amount of forces applied to the joints and the muscular forces needed to control the trunk and maintain the stability of lower limbs during locomotion (Gushue, Houck, & Lerner, 2006; Shultz, Hills, Sitler, & Hillstrom, 2010). Recent reviews have reported that the additional loads on the body could result in more musculoskeletal discomfort or pain, impairment of mobility, and increased prevalence of musculoskeletal malaignment at a young age (Shultz et al., 2009; Wearing et al., 2006). Moreover, obesity is associated with an increased risk of fractures during childhood (Goulding, Jones, Taylor, Williams, & Manning, 2001). Although obese children have more advanced bone age (Levin, Lowry, Brown, & Dietz, 2003), after adjusting for the mechanical loading effects of total body weight

on bone mass, increased fat mass is associated with decreased bone mass (Zhao et al., 2007). Thus, the musculoskeletal implication of childhood obesity, particularly in the lower extremity, has become a recent focus of research on the obese children. This information is the first necessary step before prevention and intervention strategies can be formulated.

Studies have been consistently reported the musculoskeletal effect of childhood obesity on the lower extremities. At the hip joint, pain is associated with increased BMI in obese children and adolescents (Stovitz, Pardee, Vazquez, Duval, & Schwimmer, 2008). There are increased compressive and shear forces to the cartilaginous growth plate of the femoral head, which increases the risk of slipped capital femoral epiphysis in obese children (Chung, 1981; Shultz et al., 2009). Because of a large hip stress during physical activity, obesity is considered as an important independent risk factor for incident hip osteoarthritis (OA) in adult (Cooper et al., 1998).

The knee has been reported to be one of the most common sites for the musculoskeletal pain in obese children (Taylor et al., 2006). The abnormal amount of force and force distribution within the knee joint may result in malalignment of the lower limbs, such as genu varum and genu valgum, and further cause musculoskeletal diseases (Taylor et al., 2006). Both genu varum and genu valgum would concentrate nearly all joint load on a focal area of the knee (lateral/medial compartment), further resulting in cartilage damage (Johnson, Leitl, & Waugh, 1980; Felson, Goggins, Niu, Zhang, & Hunter, 2004). Moreover, obesity has been clinically linked with reduced femoral anteversion (Galbraith et al., 1987) and tibia vara (Henderson, 1992) in children. The dynamic gait deviations characteristic found in obese adolescents resulted in pathologic compressive forces in the medial compartment of the knee (Davids, Huskamp, & Bagley, 1996). In support of these results, Gushue et al. (2006) reported that the abnormal knee load during walking in obese children could result in knee osteoarthritis in the long term. In addition, Pirpiris et al. (2006) reported an association between Blount's disease and increased BMI in children and adolescents (1-17 years old).

Obese children also regularly presented musculoskeletal pain at the ankle joints and foot (Stovitz et al., 2008). The foot performs the essential tasks of load bearing while providing propulsion to the body during locomotion. During children's

developmental years, foot and ankle problems were reported as the second most common musculoskeletal problem other than acute injuries (Stanish, 1995). Obese children typically display flatter feet relative to their leaner counterparts, which may also raise the risk for musculoskeletal injury (Dowling, 2001). Greater plantar pressures associated with obesity may also lead to more discomfort and injuries during activities (Dowling, Steele, & Baur, 2004).

In brief, persistent weight-bearing associated with childhood obesity increases load on the lower extremities, causes musculoskeletal discomfort and pain, and may develop potential musculoskeletal disorders at a younger age. These consequences undoubtedly decrease obese individuals' mobility and quality of life in the long term (Wearing et al., 2006b). Therefore, before providing physical activity to obese children for any particular purpose, such as weight loss, it is worth understanding the biomechanical function to avoid unexpected joint injuries and musculoskeletal problems.

2.3 WALKING BIOMECHANICS AND ENERGETICS IN OBESE CHILDREN AND ADOLENSONE

Walking consumes a significant amount of metabolic energy and is the most common form of physical activity performed by individuals (Shultz, Browning, Schutz, Maffeis, & Hills, 2011). While walking biomechanics data can help to evaluate gait kinematics and kinetics and understand the locomotion system through the application of mechanical movement, energetics parameters are imperative to explain the mechanisms and metabolic characters of obese children during gait (Shultz et al., 2011; Whittle, 2007). The combined study of gait biomechanics and energetics is necessary to provide important insights into the relationship between the excess body weight and the associated risk of musculoskeletal pathology. It is also the first necessary step to develop effective physical activity recommendations that promote physical activity for weight management expenditure while reducing the risk of musculoskeletal problems in obese children.

2.3.1 Walking biomechanics

There are three major aspects of gait parameters, including spatiotemporal, kinematics and kinetics parameters (Winter, 2005). Spatiotemporal data provide a basic description of time and distance parameters during walking. Gait kinematics

parameters include position, velocity and acceleration of body segments. Kinetics is the term to describe gait with the force acting on body or body segments .

The spatiotemporal differences between obese children and normal weight children have been investigated for a few decades (Hills & Parker, 1991a). Previous studies have shown that obese children adapt to walk with a lower velocity than normal weight children (Hills & Parker, 1991a; McGraw et al., 2000). Walking speed is a simple indicator of gait abnormalities associated with musculoskeletal problems (Levangie & Norkin, 2011). Reduced walking speed in obese children was considered as a compensation for an increased mediolateral instability during walking (Nantel, Brochu, & Prince, 2006; Shultz et al., 2011). A larger step width and increased base of support were also identified, which could be a result of instability during gait and excess adipose tissue between the thighs (Hills & Parker, 1991a; McGraw et al., 2000; Nantel et al., 2006). Childhood obesity has also been consistently associated with lower cadence, shorter step length, longer single support and double support phase durations when compared with normal weight children (Hills & Parker, 1991a; McGraw et al., 2000). These differences could be a strategy to increase dynamic balance to prevent falls or other disturbances during walking (Colne, Frelut, Peres, & Thoumie, 2008). However, some studies did not find significant differences in the spatiotemporal characteristics between these two groups (Hills & Parker, 1991b; Nantel et al., 2006). It suggested that these parameters may vary depending on the extent of excess body weight.

Kinematic differences between obese and normal weight individuals were reported. Gait involves motion in the sagittal, frontal, and transverse planes. In the sagittal plane, less flexion at the knee and hip have been found during normal walking in obese children (Gushue et al., 2006; Hills & Parker, 1991a; McMillan, Pulver, Collier, & Williams, 2010). The reductions in range of motion (ROM) at the knee and ankle were associated with obesity in adults (DeVita, Hortobágyi, & Hortobagyi, 2003; Messier, 1994). However, the sagittal plane ankle kinematics in obese children have been inconsistent within the literature (McMillan et al., 2010; Nantel et al., 2006). By contrast, Shultz et al. (2009) reported the kinematics were not significantly different between obese children and normal weight children aged from 8 to 12 years old, while the self-selected walking speed is also similar. In the frontal plane, greater hip adduction was reported, and obese children maintained the

rearfoot inversion and knee abduction throughout the stance phase (McMillan, Auman, Collier, & Blaise Williams, 2009). This may result from the excess adipose tissue between the thighs, which required more hip abduction to avoid friction of the inner thighs during walking (Nantel et al., 2006). Genu valgum is a common condition common among obese children (Taylor et al., 2006). An increased knee abduction distributes the force across the medial compartment of the knee, which may contribute to the development of knee osteoarthritis (Kobayashi, Yoshihara, Yamada, & Fujikawa, 2000). Only one study has assessed the components of transverse plane joint kinematics in obese adolescences (Shultz, Sitler, Tierney, Hillstrom, & Song, 2009). There was no difference found in this plane between obese and normal weight participants. In general, movements occurring in the frontal and transverse plane are much smaller than that in the sagittal plane, being reported within 10° in the literature (McMillan et al., 2010; Shultz et al., 2009). Thus, the consistency and accuracy of these data are highly dependent on the capabilities of measurement systems and the specific model used (Levangie & Norkin, 2011).

Joint kinetics based on the inverse dynamic calculations provides important information about the joint moment and power that cause motions of body segments. It has been widely used to explain why an obese individual is more likely to have joint problems (Nantel, Mathieu, & Prince, 2011). Obesity results in greater absolute peak joint moments and power at hip, knee and ankle due to the larger body mass (Browning & Kram, 2007; Shultz et al., 2009). Increased absolute joint moments indicate more muscle requirements and more forces applied to the lower extremities. These may increase the risk of joint discomfort, malalignment and injury in obese children (Shultz et al., 2009). However, normalized joint moments are more frequently used than the absolute value by researchers to remove confounding anthropometric factors (e.g. normalized by weight or weight × height).

After normalizing the joint moments by body weight, McMillan et al. (2010) reported obese adolescents had a greater hip flexor moment in the late stance phase and a lower hip extensor moment during the initial contact than normal adolescents, while the peak hip extensor moment was similar between the two groups. Less hip extensor moment reduces the requirement of the hip extensors. The authors suggested that obese individuals adopt this gait to compensate for relatively weak hip extensors (McMillan et al., 2010). However, muscle strength of the hip extensor was

not available to test this hypothesis. At the knee joint, Browning and Kram (2007) showed that the obese individuals had a peak knee extensor moment about 43%~50% higher than the normal weight individuals when walking at the same speeds. Interestingly, while walking at a self-selected speed, previous studies found similar or a decreased knee moment at the sagittal plane in overweight and obese children compared with normal weight children (Gushue et al., 2006; McMillan et al., 2010). Peak ankle plantarflexor moments also have been reported to be reduced in overweight individuals (McMillan et al., 2010). Ankle plantarflexors provide support and propulsion during walking (Kepple, Siegel, & Stanhope, 1997). Reduced ankle plantarflexor moments may result in decreased push-off force and therefore the requirement of muscle force generating by other joints would increase. Compared with the sagittal plane, only few studies reported the joint moment at the frontal and transverse plane. McMillan et al. (2010) reported decreased hip abduction and knee abductor moment, while Gushue et al. (2006) reported obese children exhibited greater knee abductor moments than the normal weight participants. A reduction in hip abductor moment has been considered as a contributing factor to the relationship between medial compartment knee osteoarthritis and obesity (Sheehan & Gormley, 2012). An increase in knee abductor moment also places more loads on the medial compartment of the knee. (Shultz, Sitler, et al., 2009). No differences were reported in the frontal or transverse plane ankle moments between obese and normal weight children by the literature.

2.3.2 Walking energetics

Although many kinematics and kinetics differences were found between obese and normal weight children, some researchers pointed out that the walking energetics, which merges both kinematics and kinetics may allow a better understanding of the gait strategies selected by obese children (Samson, Desroches, Cheze, & Dumas, 2009; Shultz et al., 2011). Much evidence was found that obese children use more metabolic and mechanical energy than their lean peers during walking (Abadi, Muhamad, & Salamuddin, 2010; Nantel et al., 2006; Peyrot et al., 2010; Runhaar et al., 2011). This may further explain the greater difficulty obese populations have in performing locomotor tasks and the decreased motivation to exercise in these populations (Wearing et al., 2006b).

Walking is accomplished by bursts of positive and negative muscle work, which maintains the stability and generates energy to rise and accelerate the body weight against gravity (Grabowski, Farley, & Kram, 2005; Griffin, Roberts, & Kram, 2003). Due to the excess body weight, there are increased demands on muscles to support a greater weight during stance phase, and to redirect and accelerate a larger body mass during step-to-step transitions. As the muscle work is difficult to measure during locomotion, a biomechanical model is employed to compute the mechanical energy by summing the power entering and leaving the segment by all forces and moments acting on the segment over time (Winter, 2005). The power generations and absorptions correspond to concentric and eccentric muscular activities, respectively (Levangie & Norkin, 2011). Nantel et al. (2006) reported that compared to normal weight children, children who were obese would use more mechanical energy when walking at the same speed. Their results also suggested that obese children were less efficient than normal weight children in transferring mechanical energy within the hip flexor muscles from the stance phase to the swing phase. By contrast, the metabolic cost of walking receives more attention than the mechanical energy cost in the literature (Nantel et al., 2006). Body mass appears to be the primary determinant of the energy cost of normal walking (Shultz et al., 2011). In order to move a larger body weight, the absolute metabolic energy expenditure is greater in obese children to preserve the ability to control body posture and overcome increased body inertia (Behringer, Vom Heede, Yue, & Mester, 2010; Butte et al., 2007; Plewa, Cieślinska-Swider, Bacik, Zahorska-Markiewicz, Markiewicz & Błaszczyk 2007; Lazzer et al., 2003; Maffeis, Schutz, Schena, Zaffanello, & Pinelli, 1993; Peyrot et al., 2009). For example, Butte et al. (2007) reported that the energy expenditure in obese children walking at 1.25m/s, measured by a room respiration calorimeter, was significantly greater than in normal children, by about 5.25 kJ/min.

Many researchers have proposed that humans prefer to walk with a strategy that involves a minimum energy cost per distance (Gordon, Ferris, & Kuo, 2009; McNeill Alexander, 2002; Zarrugh, Todd, & Ralston, 1974). From a physiological perspective, studies also have suggested that the speed at which the metabolic cost is at a minimum is similar to the self-selected walking speed (Browning, Baker, Herron, & Kram, 2006; Ralston, 1958). There is a U-shaped relationship between the energy cost and speed (Holt, Hamill, & Andres, 1991; Zarrugh et al., 1974). Hence,

there is one speed for individuals that minimizes the amount of energy needed to walk a given distance. Obese children also have this ability to sense the energy cost per distance and walk so as to minimize it (Browning & Kram, 2005). Thus, the biomechanical changes in gait patterns may be strategies used by obese children, to reduce the mechanical and metabolic requirement to move their excess body weight. However, the biomechanical basis for the walking energetics is not well understood in obese children. It is still not clear whether the gait changes in obese children represent a change in strategy from selecting an energetically optimal gait.

Since the implication of excess body weight on the gait strategy has not been demonstrated indisputably in obese children, it is important to investigate the gait biomechanics and energetics to provide more detail information. Surprisely only one study group was found that has compared the mechanical energy and metabolic energy simultaneously during walking between obese and normal weight children (Peyrot et al., 2009). The authors speculated that the greater net metabolic rate in obese children might be partially explained by the greater internal work associated with a wide gait during walking. However, the metabolic and mechanical cost at self-selected speed was not determined in their studies. Therefore, the energetics of a natural walking strategy for obese children were still unclear.

Based on the above findings in walking biomechanics and energetics, there are three possible reasons why obese children alter their gait pattern: to decrease joint loading, to compensate potential muscle weakness, and to minimize energy expenditure. While this mechanism may maintain musculoskeletal health in the short term, chronic adjustment to the musculoskeletal system to accommodate the excess body weight is a concern (Forhan & Gill, 2013). More intervention studies are needed to demonstrate the cause and effect between childhood obesity and gait.

2.4 INTERVENTION STUDIES

2.4.1 Weight loss

Weight loss may be the most direct way to avoid or mitigate obesity-related health conditions. While extensive weight loss studies focus on the cardiometabolic consequences, a number of studies have been devoted to uncovering the beneficial effect of weight loss on musculoskeletal health (Wearing et al., 2006b). Surgical obesity treatment reduces the risk of developing musculoskeletal pain in adults at

most sites of the body (Hooper, Stellato, Hallowell, Seitz, & Moskowitz, 2007; Peltonen, Lindroos, & Torgerson, 2003). Significant improvements in physical function and reduced pain were found after diet and exercise-induced weight loss in obese adult with osteoarthritis (Messier, Gutekunst, Davis, & DeVita, 2005). Weight loss also led to an improvement in balance control (Handrigan et al., 2010). Comparing the walking biomechanics and energetics before and after weight loss provides valuable insight into the mass-driven mechanics of the musculoskeletal systems, which is particularly useful for the planning of treatments to improve musculoskeletal health for obese individuals.

Only a few studies have reported the effect of weight loss on spatiotemporal gait parameters. Aaboe et al. (2011) found a 13.6% weight reduction resulted in a 4% increase in the self-selected walking speed in obese adults with knee osteoarthritis. Hortobágyi et al. (2011) reported that 0.15m/s increase of self-selected speed and 0.11m increase of stride length after 33.6% of the total weight loss in adults. Peyrot et al. (2010) found a significantly increased stride length after 12 weeks weight loss in obese children. The gait kinematics and kinetics also significantly changed following weight loss in obese adults. Hortobágyi et al. (2011) reported an increase in hip range of motion at a self-selected speed. Vartiainen et al. (2012) found the hip flexion angle significantly reduced at initial contact at a pre-determined gait speed (1.2m/s) after reducing 21.5% body weight. Studies similarly reported that the absolute value of the hip, knee and ankle moments reduced after weight loss, but the results of normalized joint moments varied among these studies (Aaboe et al., 2011; Hortobágyi et al., 2011; Messier et al., 2005; Vartiainen et al., 2012). Hortobágyi et al. (2011) reported normalized peak knee extensor moment increased after weight loss in obese adult without joint problems, while Vartiainen et al. (2012) reported normalized joint moment remained unchanged. The differences observed between these two studies may be caused by the walking speed. Hortobágyi et al. (2011) reported the joint biomechanics at the self-selected walking speed, and this speed significantly increased after weight loss. By contrast, Vartiainen et al. (2012) fixed gait speeds to prevent speed affecting the gait parameters. In obese adults with knee osteoarthritis, studies have found a significant reduction in the peak knee abductor moment, without changes in the sagittal plane knee moment after weight loss during normal walking (Aaboe et al., 2011; Messier et al., 2005). The reduction of the

absolute knee abductor moment suggests that less compressive loads were transmitted to the medial compartment of the knee associated with weight loss. These studies confirmed body mass exerted great influence on the differences in gait biomechanics between obese and normal weight individuals, and weight loss was able to reduce these differences. Nevertheless, whether or not the observations in obese adults are similar to those in obese children remains to be answered.

In order to gain the knowledge about the mass-driven changes in walking energetics, previous studies have applied weight loading or unloading on normal participants to simulate weight change. Grabowski et al. (2005) simulated both reduced weight and added load conditions. The study found that reduced weight decreased the demands on muscles, then further resulted in decreased energy cost (Grabowski et al., 2005). Griffin et al. (2003) found that the net metabolic rate and external mechanical work increased as participants carried heavier loads across four different walking speeds. The walking efficiency followed an inverse U-shaped trend that the value was lower at faster speeds and slower speed (Griffin et al., 2003). There are only two studies evaluating the effects of weight loss on the walking energetics in obese children (Peyrot et al., 2010, 2012). When walking at 1.25m/s, obese children had a significant reduction in net metabolic cost and net metabolic rate associated with increased stride length after a 6% weight loss (Peyrot et al., 2010). The gross metabolic rate and the net metabolic rate at self-selected walking speed did not change significantly after weight loss (Peyrot et al., 2012). An improved walking economy (rate of oxygen consumption per distance during walking) was also reported, which might relate to reduced muscle requirements to support body weight and maintain balance during walking (Peyrot et al., 2012). However, significant height growth over three month intervention may also induce changes in the measurements. A specially designed intervention program is needed to achieve substantial weight loss while the natural development is minimal. It may provide valuable information to evaluate more accurately the true mass-driven effects of weight loss on walking biomechanics and energetics.

2.4.2 Muscle strengthening

Muscle strength refers to the ability of a muscle group to produce force against a resistance (Taaffe & Marcus, 2000). Muscles of the lower extremities play an extremely important role in maintaining joint mobility, stability and function (Roos,

Herzog, Block, & Bennell, 2011). Insufficient muscular strength and power can impair motor function, limiting an individual's ability to perform activities of daily living (Visser, Deeg, Lips, Harris, & Bouter, 2000). In walking tasks, lower extremity muscles are recruited to provide forward progression and support body weight (Liu, Anderson, Pandy, & Delp, 2006). Due to the excess body weight, obese children need larger joint moments and powers to control the trunk and maintain the stability of the lower limbs (Gushue et al., 2006; Shultz et al., 2010). The metabolic cost of walking is determined primarily by the cost of generating muscle force throughout the gait cycle (Umberger & Robinson, 2011). Weak muscle strength may increase the metabolic cost to support body weight, because more fast glycolytic fibres would be recruited, and these fibres are less economical than slow oxidative fibres (Peyrot et al., 2010). Previous cross-sectional studies suggested that children may adapt to a gait strategy to compensate potential muscle weakness (Dufek et al., 2012; Hills & Parker, 1991a; McMillan et al., 2010). However, none of these biomechanical studies provided data to examine whether a relative weakness is present.

Intervention studies are needed to better understand the role of muscle strength in gait biomechanics and energetics. Muscle strength is a modifiable physical function. Substantial improvement can be achieved in a relatively short period of time by muscle strength training (Kraemer, Ratamess, & French, 2002). This makes it possible to study the gait changes associated with muscle strengthening directly. If there exists muscle weakness in obese children, gait differences observed in cross-sectional studies should reduce following muscle strengthening. For example, obese children usually walk with a slower speed than normal weight children to compensate for increased instability, perhaps due to lack of strength (Granacher, Gollhofer, & Kriemler, 2010; Nantel et al., 2006; Shultz et al., 2011). Increasing muscle strength may increase the self-selected walking speed associated with improved walking stability. Obese individuals generating more hip flexion forces rather than ankle plantarflexors to propel the body forward may suggest that the ankle plantarflexion muscles are weak during walking (McMillan et al., 2010). An increase in ankle plantarflexor strength may reduce the hip flexor moment during walking. Reduced knee flexor during walking were related to potential knee extensor weakness (McMillan et al., 2010). The knee flexion angle may increase after

increasing knee extensor strength. However, there have been no experimental investigations to test these hypotheses in obese children. Most exercise intervention programs for obese children were more targeted to weight loss and cardiometabolic health rather than the potential musculoskeletal health benefit (Ho, Garnett, & Baur, 2012).

2.5 THE APPLICATION OF SIMULATION

2.5.1 Theoretical framework

Traditional gait analysis using inverse dynamics have been widely used for the determinations of joint kinematics and kinetics during walking. However, this method is not able to reveal the cause-effect relationships between neuromuscular excitation patterns, muscle forces and body movement (Zajac, Neptune, & Kautz, 2003). Since it is far more difficult to obtain tissue stresses and muscle forces invasively *in vivo*, a theoretical framework is needed to provide an integrated understanding of muscle activities and joint movements (Delp et al., 2007). Musculoskeletal models based on computer simulation of walking is such a theoretical framework that describes how the forces in the human body cause the segment movements during gait. Rapid development of computer and medical imaging technology have enabled more detailed estimation of multiple variables of interest under dynamic conditions of human body. More efficient algorithms for modelling and simulation of movement provide more quantitative data about how the neuromuscular and musculoskeletal systems interact with each other to produce movement (Pandy & Andriacchi, 2010).

2.5.2 Procedures of the modelling and simulation

The simulation procedures usually consist of three parts (Figure 1.1). The first part is the collection of experimental data. 3D gait analysis using a motion capture system and forceplates is needed to provide measurements of body-segmental motion and ground reaction force data (Pandy & Andriacchi, 2010). The second part is the combination of experimental data with computational models in which the skeleton and muscles are both represented. The human body is modelled as a link-segment system actuated by muscle actuators, which include several mechanical, physiological and geometry properties required by the musculoskeletal system and dynamic human motion (Zajac, Neptune, & Kautz, 2002). The final part is the

application of an appropriate simulation method to solve for the solutions (actuator forces or torques in the model) based on the dynamic equations of motion. The solution can be calculated by using inverse dynamics or forward dynamics methods, and it should be able to reproduce the movement when applied to the model.

The differences between inverse and dynamic method have been reported extensively (Buchanan, Lloyd, Manal, & Besier, 2005; Erdemir, McLean, Herzog, & van den Bogert, 2007). Inverse dynamic-based static optimization calculates muscle forces by decomposing the net joint moment into individual muscle moments using experimental kinematics and ground reaction forces as inputs (Pandy & Andriacchi, 2010). This method has been widely used in the estimation of muscle forces during walking for almost three decades (Erdemir et al., 2007). Forward dynamic-based dynamic optimization calculates the muscle activations by tracking the experimental kinematics according to the input joint moments or muscle forces (Pandy & Andriacchi, 2010). Direct comparisons of muscle forces obtained from static and dynamic optimization have shown that the two approaches were similar in their determination of muscle forces in normal walking (Anderson & Pandy, 2001). However, the static optimization method cannot provide data of individual muscle contributions to the joint or body movement (e.g., COM acceleration, joint kinematics), because it uses an incomplete set of dynamical equations, which only include the segments and joints that the muscle is attached to (Zajac et al., 2003). By contrast, forward dynamic simulation models take the segments of the entire body into account (Winter, 2009). They are able to reveal cause-effect relationships between gait alterations and musculoskeletal function, which is especially useful for predicting the outcome of treatments. Thus, it is necessary to use forward dynamic optimization to extend the modelling analysis for investigating the gait adaptations.

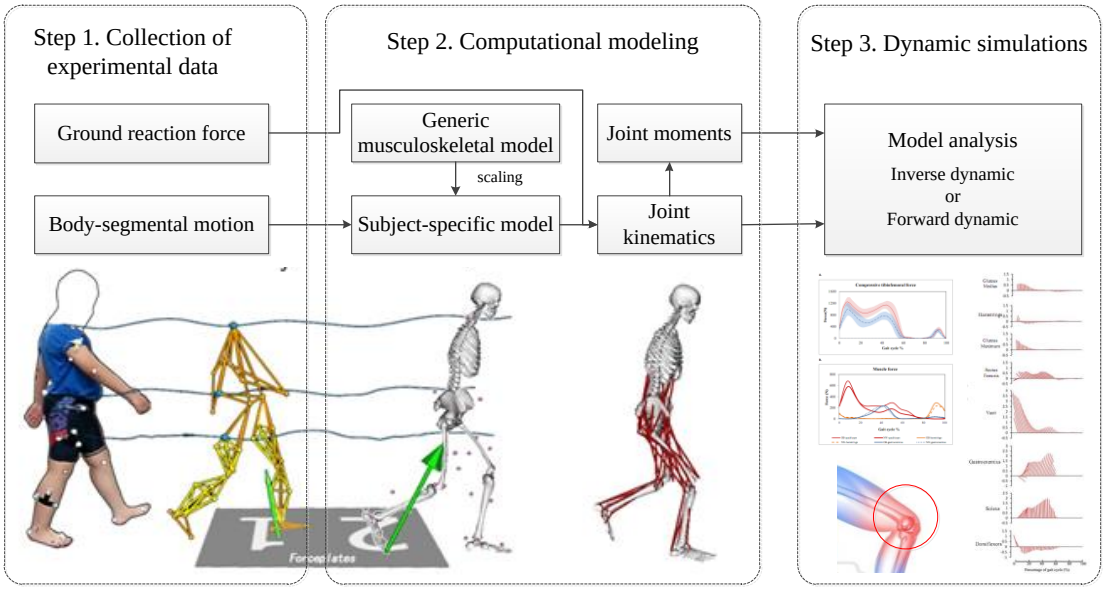


Figure 2.1 Procedures of the modelling and simulation

In recent decades, a large number of simulation studies have been developed to investigate the causal relation between muscle force and joint movement during walking (Erdemir et al., 2007). Based on the musculoskeletal model and accurate knowledge of muscle forces, researchers can further calculate the corresponding forces and stresses acting on the bones, individual muscle and joint functions, and the muscle energetics. Steele et al. (2012) examined how muscle forces and compressive tibiofemoral force change with the increasing knee flexion associated with crouch gait in cerebral palsy children. Liu et al. (2008) reported individual muscle contributions to support and accelerate body COM over different walking speeds in children. Furthermore, dynamic simulations also provide insight into muscle mechanical work and the resulting implications for metabolic cost and efficiency (Neptune, Zajac, & Kautz, 2004). Applications of muscle energetic models in conjunction with the forward dynamic can help to find the timing and distribution of energy consumption among muscle, as well as to evaluate alternative gait strategies and predict overall walking mechanics (Umberger, Gerritsen, & Martin, 2003). Due to the availability of experimental data following a standard gait analysis, it is feasible to apply this method in the obese population to study the gait biomechanics and energetics at the musculoskeletal level.

2.5.3 Challenge of application in obese children

Although musculoskeletal modelling and simulation have many advantages, the application for obese individuals is still faced with challenges. First of all, simulation analysis requires accurate experimental measurements of body motion. The kinematics data are usually collected by a marker-based motion capture system. For obese individuals, it is difficult to locate the landmarks of the segments because of extensive soft tissue covering the skeletal system. Previous study suggested that the reliance on hip markers, such as Anterior Superior Iliac Spine (ASIS) and greater trochanter markers, causes particular concerns (Browning, 2012). The inaccurate placements of ASIS affect the estimate of hip joint centre and the kinematics of hip and knee joints. There are also more skin movement artefacts compared to normal individuals. It is recommended to use medical imaging (e.g., MRI, DEXA etc.) to provide more accurate information for the calculation of joint centres. Secondly, the accuracy of model-based simulations is influenced by the musculoskeletal properties of the model, such as the number of segments and degree of freedom, and muscle model properties. Most of the generic models are created based on the data obtained from normal adults (Delp et al., 2007). There is no musculoskeletal model available for obese children. Although each segment and muscle can be scaled of a child according to subject-specific geometry, the muscle properties remain unchanged. Thus, the simulation results need to be verified by comparing with experimental kinematic and EMG. *In vivo* measurements using MRI or ultrasonic examination may also help improve the model accuracy while applying a generic model in obese children (Arnold, Salinas, Asakawa, & Delp, 2000; Pandy & Andriacchi, 2010).

2.6 SUMMARY

Given the prevalence of childhood obesity and the lack of knowledge about the effect of the excess body weight on musculoskeletal functions, the combined study of gait biomechanics and energetics is necessary to provide important insights into the relationship between obesity and the associated risk of musculoskeletal pathology. Obese children accommodate their greater body mass by altering their walking biomechanics and energetics. This compensation mechanism may be used to decrease joint loading, compensate potential muscle weakness, and minimize energy expenditure. In addition, using experimental data alone to estimate the muscle function and internal loading of the musculoskeletal system is still a big challenge.

The application of computational modelling and simulation technology is able to reveal the cause-effect relationships between obesity and walking strategy, and allow us to predict the outcome of the intervention studies.

Chapter 3: The Effects of Obesity on Gait Biomechanics and Energetics in Children

3.1 INTRODUCTION

Identification of the gait strategies of obese children can provide insight into the implications of obesity on their musculoskeletal system (Nantel et al., 2011; Wearing et al., 2006b). Compared with normal weight children, obese children show a few alterations in gait parameters when walking at self-selected speed. These include shorter step length, lower cadence and velocity, longer single support and double support phase, higher joint moments and smaller joint range of motion (Browning & Kram, 2007; Hills & Parker, 1991a; McGraw et al., 2000; Runhaar et al., 2011). Although many kinematic and kinetic differences were found between obese and normal weight children, some researchers pointed out that the mechanical power and work, which merges both kinematics and kinetics may allow a better understanding of children's gait strategies. (Samson et al., 2009).

Walking is accomplished by bursts of positive and negative muscle work, which maintains the stability and generates energy to rise and accelerate the body weight against gravity (Grabowski et al., 2005; Griffin et al., 2003). Power generations and absorptions correspond to concentric and eccentric muscular activities, respectively (Levangie & Norkin, 2011). Due to excess body weight, there are increased demands on muscles to support a greater weight during stance phase, and to redirect and accelerate a larger body mass during step-to-step transitions. In addition, obese children require more mechanical and metabolic energy expenditure to preserve the ability to control body posture and overcome increased body inertia (Behringer et al., 2010; Plewa et al., 2007). Thus, the investigation of biomechanical energetics may explain the increased metabolic cost of obese individuals (Grabowski et al., 2005). This may further explain the greater difficulty obese populations have in performing locomotor tasks and the decreased motivation to exercise in these populations (Wearing et al., 2006b). More attention has been given to the metabolic cost of walking rather than to the biomechanical consequences (Nantel et al., 2006).

The relationship between gait biomechanics and energetics has not been demonstrated indisputably in obese children.

From a physiological perspective, studies have suggested that the speed at which metabolic cost is minimized is similar to self-selected walking speed in both obese and normal weight individuals (Browning et al., 2006; Ralston, 1958). The possibility of an energy saving gait was suggested by Heglund Willems, Penta, and Cavagna (1995). In this case, the biomechanical changes in gait pattern could be a compensation strategy of obese children, to reduce the energy cost required to lift, lower, accelerate, and decelerate their excess body mass during walking. For example, a slow walking speed may reduce the muscle requirement for propulsion,then further reduce the mechanical and metabolic cost. Although a mechanical change in gait style can result in a different metabolic cost associated with walking (Peyrot et al., 2009), it is still not clear whether the differences represent a change in strategy from selecting an energetically optimal gait in obese children.

Investigating both gait biomechanics and energetics could explain that how gait modifications contribute to the energy cost of walking. Surprisingly only one study, a study by Peyrot et al. (2009), has compared mechanical parameters and metabolic energy simultaneously during walking between obese and normal weight children. Their findings suggested that the greater net metabolic rate in obese children might be partially explained by the greater internal work associated with a gait with wider step width during walking. However, these biomechanical and energetics parameters at self-selected speed were not determined in their study. Therefore, the energetics of a natural walking strategy for obese children was still unclear.

Therefore, the purpose of this study was to investigate the effect of childhood obesity on the gait biomechanics and energetics. Gait characteristics, joint kinematics, kinetics, metabolic cost and mechanical efficiency were quantified while walking at self-selected speed. It was hypothesized that obesity would significantly influence the kinematics and kinetics of normal gait. These gait alterations in obese children would minimize the mechanical and metabolic energy cost to accommodate their excess body weight during walking.

3.2 METHODS

3.2.1 Participants

This study included 16 obese children (OB group, 8 boys and 8 girls) and 16 normal weight children (NW group, 8 boys and 8 girls), aged between 8 and 12 years, with no orthopaedic or neurological disorders that could interfere with the gait pattern or influence their energy metabolism. All participants were recruited from three primary schools in the local community by advertisements. Obesity and normal weight were categorized according to participants' BMI level, using the age and gender specific cutoff points defined by Cole et al. (2000). All participants and their parents or guardians read and signed an informed consent form approved by the University of Auckland's Human Research Ethics Committee (see Appendix A). The demographic information about the participants are presented in Table 3.1.

Table 3.1 Demographics of participants in two groups *(means ± SD)*

Variable	OB group	NW group	*t*	*d*
Age (years)	10.97 ± .78	10.84 ± .57	.52	.190
Height (cm)	154.75 ± 7.02	145.54 ± 7.15	3.68	1.343**
Weight (kg)	69.92 ± 10.78	38.43 ± 8.16	9.32	3.403**
BMI (kg/m^2)	29.08 ± 3.22	18.00 ± 2.75	10.45	3.817**

Note: *t* = t value; *d* = effect size (Cohen's *d*); ***p< .01*

3.2.2 Experimental Protocol

All participants in the OB group and the NW groups were tested with the same measurements. Spherical reflective markers were attached to each participant based on the Cleveland Clinic marker set (OrthoTrak 4.2 Reference Manual, Motion Analysis Corporation, Santa Rosa, CA, USA; the marker placements are described in Appendix B). The test session started with a 5 minute standing to measure the resting metabolic rate. Then all participants were asked to walk along a nearly circular track 30 m in length and 0.6 m in width at self-selected speed without shoes (Figure 3.1). A digital LED speed control system (Huang, Zhuang, & Zhang, 2013) was attached along the middle of the track, which was able to provide a visual target moving along

the track at participant's self-selected walking speed (see Appendix C). The self-selected speed was pre-determined by 1 minute walking along the track, then input into the speed control system. The cadence was input into a digital metronome. According to real-time signals of the LED system and the metronome, a researcher was stationed in the test field to remind participants if they walked slower or faster than the targeted speed and cadence (i.e. 'Please walking a little slower/faster'), or to encourage them to maintain their speed and cadence (i.e. 'You are doing a good job, just keep going.'). Each walking trail lasted at least 3 minutes to achieve a metabolic steady state, and then the average of remaining 2 minutes of sampling was used for analysis. The metabolic rates were measured by a portable gas analyser ($K4b^2$, COSMED, Italy) throughout the test session. Two Kistler forceplates (9287C, Kistler Instruments Ltd., Winterhur, Switzerland) were used to measure the ground reaction forces at a sampling rate of 1000Hz while the participants stepping on them. The marker positions were recorded by a 16-camera VICON digital video-based motion analysis system with the sampling rate of 100Hz (Vicon, Oxford Metrics Group, Oxford, UK).

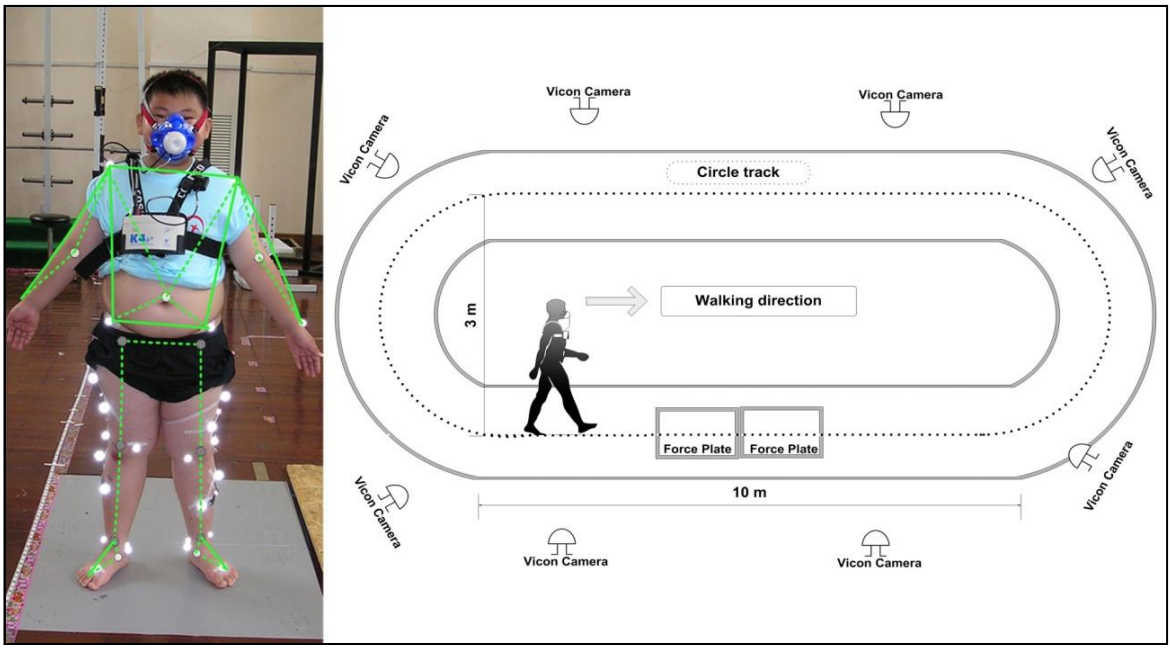


Figure 3.1 The participant and experimental set-up.

Maximal flexion/extension isokinetic muscle strength was measured at the knee and ankle by an isokinetic dynamometer (Con-Trex Multi-joint Module, CMV AG, Switzerland). After a low-intensity warm-up, leg and foot flexion and extension exercises were carried out using an angular velocity of 60 degree/s, the velocity at which children could generate the greatest torque compared with faster concentric velocities (Wiggin, Wilkinson, Habetz, Chorley, & Watson, 2006). Participants were instructed to try their best to push and pull through a standard range of motion using the manufacturer's recommendations. Peak torque (PT) was measured in one set of five maximal repetitions. The highest PT value was used for analysis.

3.2.3 Data analysis

All kinematic and kinetic data were processed by VICON Nexus software (Version 1.7, Oxford Metrics, Oxford, UK). 3D segment trajectories and ground reaction force data were filtered with a low pass 4th order zero-lag Butterworth filter with a cutoff frequency of 6Hz. The procedure of calculating joint kinematics, kinetics and mechanical energy was represented by a flow chart (Figure 3.2). In the first step, the marker's position data, obtained from 3D motion analysis system, were imported into the Cleveland Clinic biomechanical model to determine the joint angles, joint velocity and acceleration. An inverse dynamic analysis procedure (Winter, 2005) was performed to calculate the reaction forces and moment at all joints (ankle, knee, hip and trunk). The inertial parameters of the segments were calculated based on the participant's height and mass using standard biomechanical procedures (Winter, 2005). All gait parameters were averaged from the participants with three successful walking trials each for the OB and NW group, respectively.

To calculate the mechanical energy expenditure (MEE), Aleshinsky's equation was utilized (Aleshinsky, 1986). MEE (J) was represented as the total mechanical work performed, which is the time integral of the total power output:

$$MEE = \int_{T1}^{T2} |P_{total}| dt \quad (3.1)$$

P_{total} represents the power produced by body segments, and equals the sum of the absolute values of the powers developed by segments. The joint power was calculated from the dot product of the joint angular velocity and the joint moment. T1 and T2 stand for the initial and final time point of the gait cycle, respectively.

Metabolic rate was determined from oxygen consumption measured by indirect calorimetry. Oxygen uptake (ml/min) and carbon dioxide production (ml/min) were obtained from the gas analyser. Gross metabolic power (Watt) and resting metabolic power (Watt) were calculated using Brockway's standard equation (Brockway, 1987). Net metabolic power (Watt) was equal to gross metabolic power minus resting metabolic power. The net metabolic cost (J) for a gait cycle was then calculated by multiplying to the stride time. The net metabolic cost and MEE was divided by walking speed and body mass, respectively, to obtain the net metabolic rate and normalized MEE, which represented the amount of metabolic and mechanical energy expended per unit body weight walking through one unit of distance (J/kg/m).

Mechanical efficiency was then calculated as the ratio between MEE and the metabolic energy expenditure via the following formula:

$$Mechanical\ efficiency = \frac{MEE}{Net\ metabolic\ cost} \times 100\% \qquad (3.2)$$

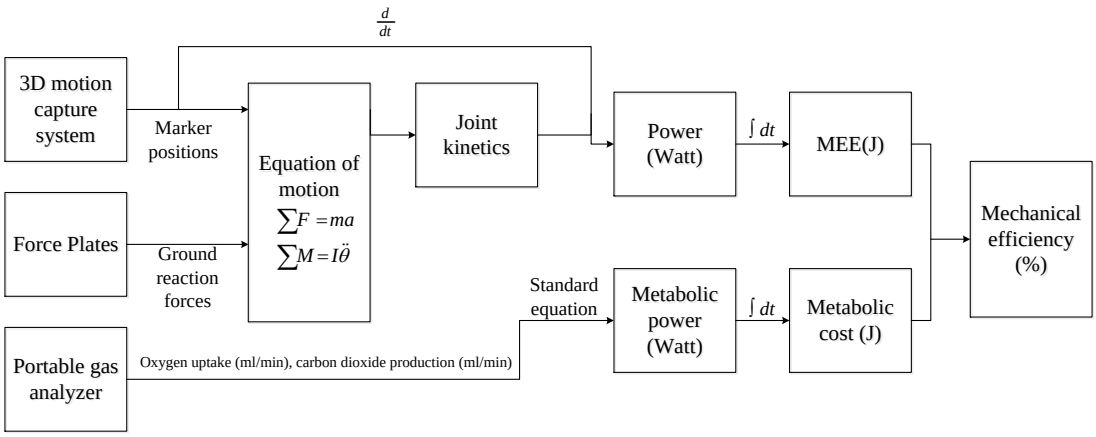


Figure 3.2 The flow diagram of the process of biomechanical and energetic data.

3.2.4 Statistical Analysis

Means and standard deviations of spatiotemporal gait parameters, kinematics, kinetics, and energetic parameters were calculated for each group, and *t*-tests were used to determine the statistical significance of differences between groups. $p < .05$ indicated statistical significance. The *t* value and the effect sizes (*d*) were reported. All data were analysed using the Statistical Package for the Social Sciences (SPSS) software (version 18.0, SPSS Inc, Chicago, IL).

3.3 RESULTS

The average body mass of the OB group was 69.92 kg, almost doubled than normal weight children (38.43 kg). The BMI of the OB group was 61.6% greater than the NW group. The height of the OB group was 6.0% taller than the NW group. The absolute PTs of ankle extensor and flexor in the OB group were significantly greater than that in the NW group. After normalized to body weight, the OB group showed significantly smaller PT of knee extensor, knee flexor, ankle extensor and ankle flexor than the NW group (Table 3.2).

Table 3.2 Isokinetic strength between groups

Peak torque at 60°/s	OB group	NW group	*t*	*d*
Absolute Knee Extensor	67.9 ± 16.7	59.7 ± 16.8	1.38	0.506
Normalized Knee Extensor	1.0 ± .2	1.6 ± .5	- 4.47	-1.633**
Absolute Knee Flexor	34.2 ± 10.2	30.0 ± 6.4	1.40	0.512
Normalized Knee Flexor	.5 ± .1	1.6 ± .5	- 4.48	-1.637**
Absolute Ankle Extensor	59.0 ± 7.5	47.2 ± 11.2	3.51	1.28**
Normalized Ankle Extensor	.9 ± .2	1.3 ± .4	- 3.86	1.41**
Absolute Ankle Flexor	23.6 ± 4.0	18.9 ± 4.4	3.18	1.16**
Normalized Ankle Flexor	.3 ± .1	.5 ± .2	- 4.405	1.478**

Note: *t* = t value; *d* = effect size (Cohen's *d*); ***p*< .01*

Significant changes were observed in spatiotemporal gait parameters except for stride length (Table 3.3). Compared with normal children, obese children had significantly lower walking speed, cadence and a smaller single support phase, while the step width, double support phase and stride time were significantly higher. Given the height of obese children (154.75 ± 7.02 cm) was significantly higher than normal weight children (145.54 ± 7.15 cm), the stride length was divided by the height to get a normalized stride length. Obese children had significantly greater normalized stride length than the normal weight children.

Table 3.3 Spatiotemporal gait parameters *(means ± SD)*

Variable	OB group	NW group	*t*	*d*
Speed (m/s)	1.10 ± .08	1.25 ± .11	-4.32	-1.577**
Cadence (step/min)	111.30 ± 5.10	122.45 ± 6.59	-5.36	-1.957**
Step width (cm)	.18 ± .05	.14 ± .03	3.11	1.136**
Stride length (m)	1.19 ± .09	1.22 ± .09	-1.12	- .409
Stride length/height	.77± .05	.84 ± .08	-3.93	-1.435**
Stride time (s)	1.08 ± .05	.99± .06	5.06	1.848**
Double support (%)	20.97 ± 2.52	16.03 ± 3.65	4.45	1.625**
Single support (%)	39.67 ± 1.26	41.83 ± 2.10	-3.52	-1.285**
Stance Phase (%)	60.65 ± 1.29	58.07 ± 1.83	4.61	1.683**

Note: *t* = t value; *d* = effect size (Cohen's *d*); ***p< .01*

The kinematics results were presented in Figure 3.3 and Table 3.4. In the sagittal plane, the OB group showed less flexed knee angles throughout the stance phase, and the knee ROM was significantly smaller than the NW group. The ankle ROM of OB group was also significantly less than the NW group during stance phase. In the frontal plane, more hip adduction was found in the OB group during stance phase. The hip adduction/abduction ROM for the OB group was significantly larger than the NW group at both the stance phase and swing phase. Compared with the OB group, the NW group had a significantly larger knee valgus/varus ROM. The knee was valgus throughout the stance phase in the OB group. In the transverse plane, no significant differences in the ROMs of hip, knee and ankle were found between groups. As presented in Figure 3.4, obese children had a slightly larger vertical COM displacement and smaller mediolateral COM displacement, but did not reach the significance level.

Table 3.4 Joint ROM during walking

ROM	OB group	NW group	*t*	*d*
Stance Phase				
Hip adduction/abduction	8.9 ± 2.9	5.4 ± 2.5	2.93	1.070**
Knee valgus/varus	4.6 ± 1.6	8.7 ± 6.0	-2.80	-1.021**
Knee flexor/extension	32.6 ± 5.2	40.6 ± 9.7	-2.94	-1.073**
Ankle Plantar/Dorsiflexion	22.8 ± 4.1	27.3 ± 6.6	-2.64	-0.962*
Swing Phase				
Hip adduction/abduction	11.4 ± 2.9	6.3 ± 2.1	5.76	2.104**

Note: t = t value; d = effect size (Cohen's d); *p < .05, **p< .01. There were no significant differences in other ROMs between groups, so these results are omitted from the table.

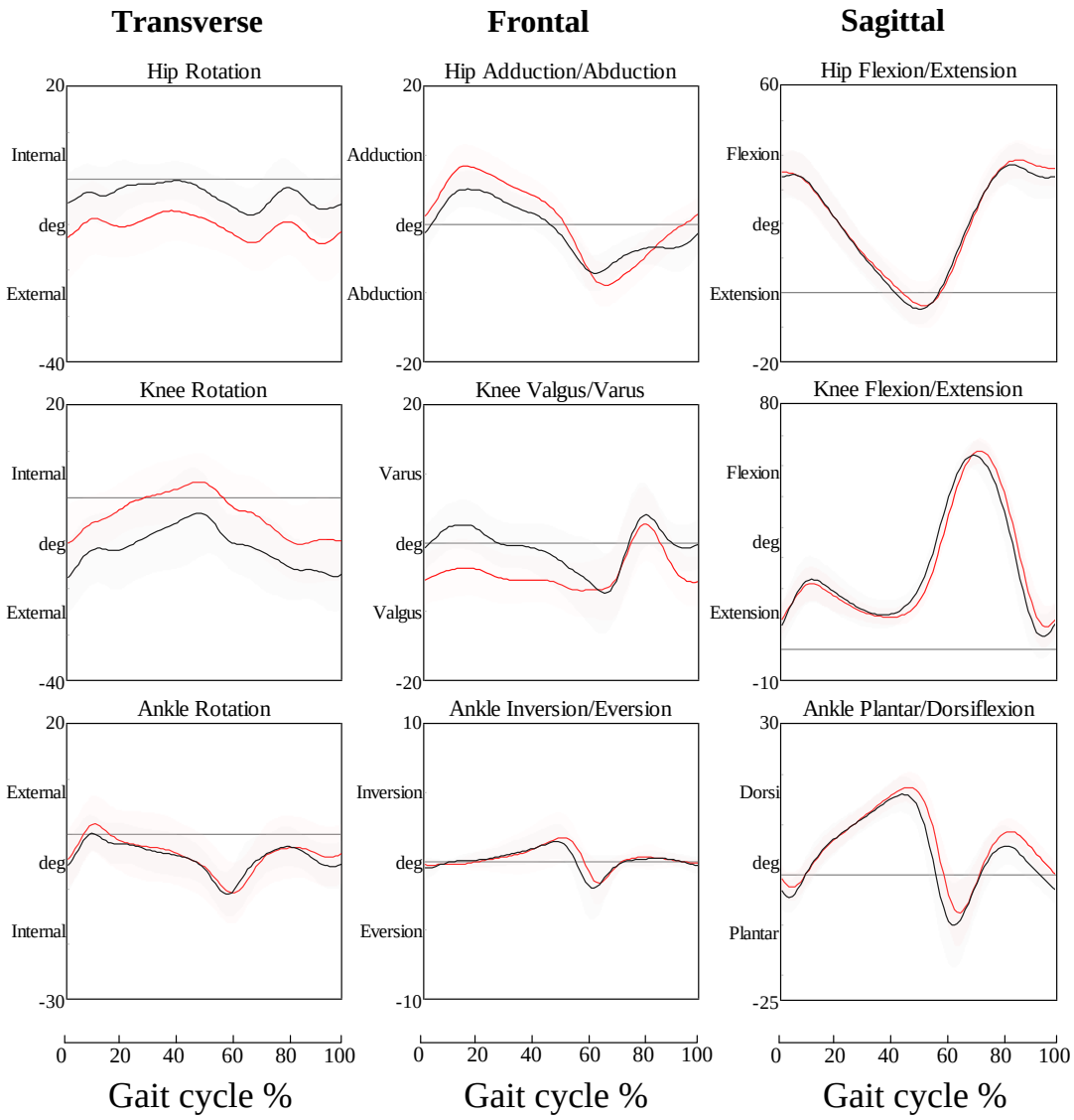


Figure 3.3 Mean joint moment (OB group: red line, NW group: black line) and standard deviation (OB group: red shaded area, NW group: grey shaded area) in the transverse, frontal, and sagittal plane, respectively.

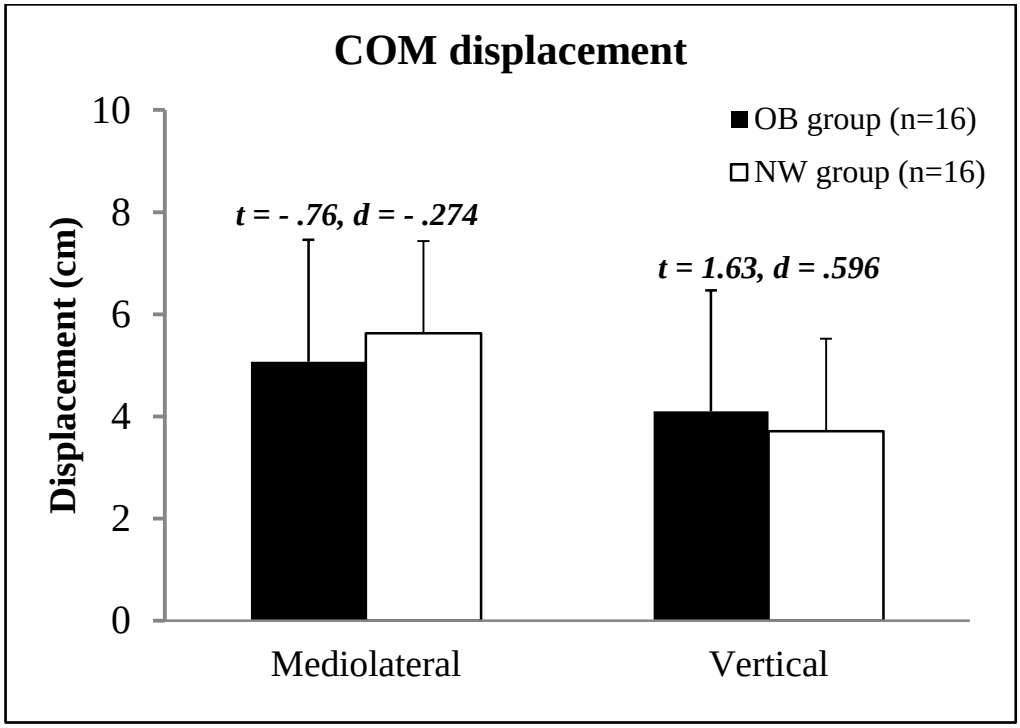


Figure 3.4 The mediolateral and vertical COM displacements during walking. Error bars represent one standard deviation. *t* = t value; *d* = effect size (Cohen's d).

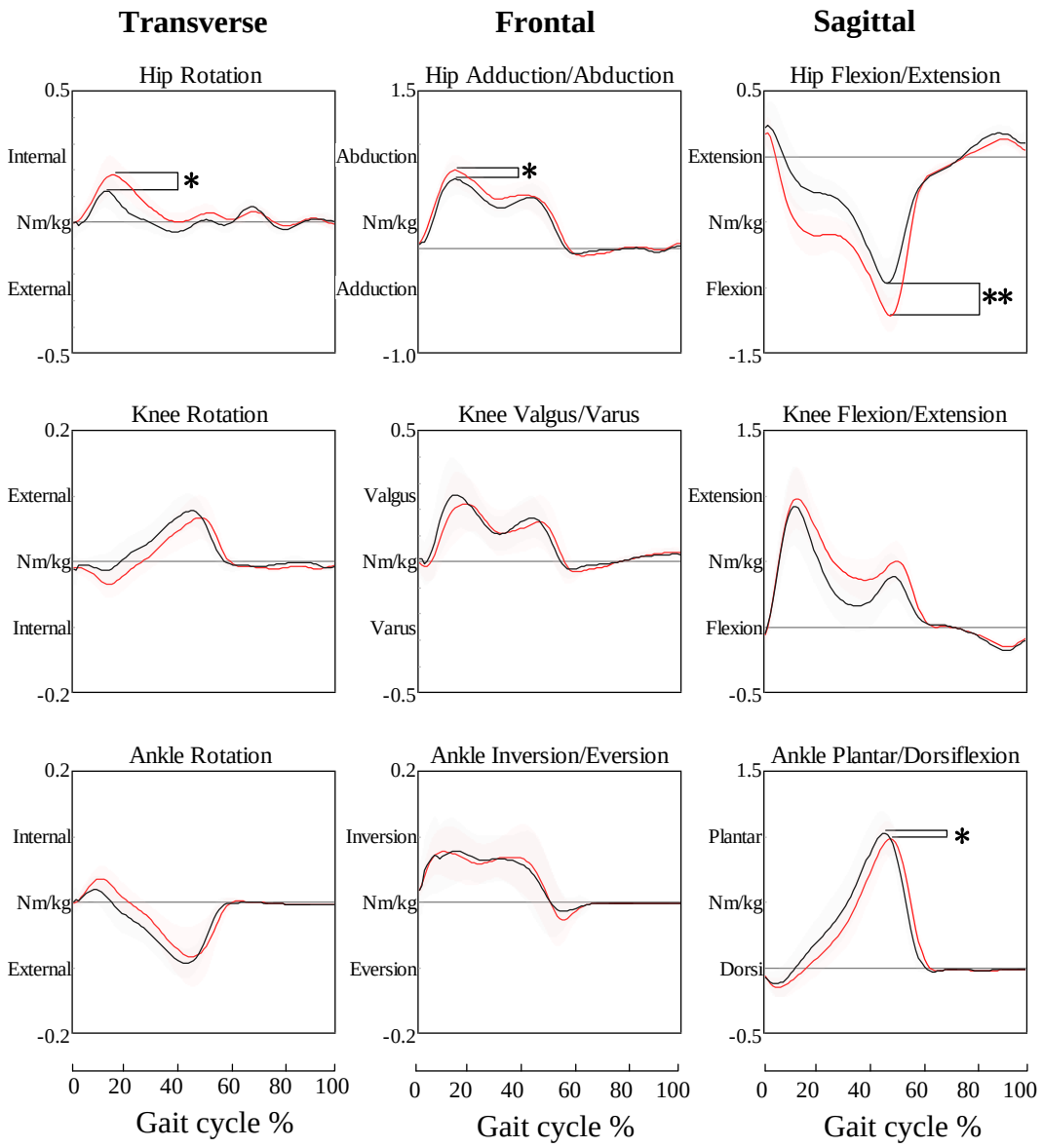


Figure 3.5 Mean joint moment (the OB group: red line, the NW group: black line) and standard deviation (the OB group: red shaded area, the NW group: grey shaded area) over a gait cycle in the transverse, frontal, and sagittal plane, respectively. *$p < .05$, **$p < .01$.

Table 3.5 Absolute peak joint moment during walking (means ± SD)

Joint	Moment	OB group	NW group	*t*	*d*
Hip	Internal	13.2 ± 5.1	5.3 ± 1.9	*5.871*	*2.144***
	Abduction	54.2 ± 9.6	25.3 ± 5.1	*10.647*	*3.888***
	Flexion	85.0 ± 9.2	36.7 ± 6.3	*17.415*	*6.359***
Knee	External	5.1 ± 2.4	2.9 ± 1.2	*3.12*	*1.139***
	Valgus	16.8 ± 7.6	10.0 ± 4.5	*3.088*	*1.128***
	Extension	70.4 ± 15.3	34.3 ± 12.7	*7.219*	*2.636***
Ankle	Internal	2.8 ± 1.3	1.0 ± .8	*4.764*	*1.740***
	Inversion	7.0 ± 2.9	4.1 ± 2.1	*3.219*	*1.745***
	Plantarflexion	70.5 ± 8.8	42.8 ± 5.9	*10.632*	*3.882***

Note: *t* = t value; *d* = effect size (Cohen's *d*); ***p*< *.01*. Non-significant results are omitted from the table.

Figure 3.5 represents the joint moment in the sagittal, frontal and transverse plane over a gait cycle. The magnitudes were normalized by body weight. The hip internal rotator moment and abductor moment in OB group were significantly greater than the NW group at early stance phase. OB group had a larger hip flexor moment and smaller ankle plantarflexor moment in late stance, which initiated swing. The peak value of the absolute joint moment was shown in Table 3.5. OB group had significantly greater joint moments at all joints.

Table 3.6 Walking energetic for one gait cycle (means ± SD)

Variable	OB group	NW group	*t*	*d*
Gross metabolic power (Watt)	316.81±52.09	212.52±37.80	6.48	2.366**
Resting metabolic power (Watt)	102.90±19.46	70.75±9.15	5.98	2.183**
Net metabolic cost (J)	230.64±38.74	139.17±31.20	7.36	2.687**
Net metabolic rate (J/kg/m)	3.026±0.33	2.94±0.46	0.55	0.201
MEE (J)	69.18±17,75	40.05±8.29	5.96	2.176**
Normalized MEE (J/kg/m)	0.89±0.12	0.85±0.13	0.95	0.347
Mechanical efficiency (%)	29.85±4.63	29.22±4.79	0.37	0.135

Note: *t* = t value; *d* = effect size (Cohen's *d*); ***p*< *.01*.

Table 3.6 shows the differences in energetic parameters between obese and normal weight children. The gross metabolic rate and pre-exercise standing oxygen consumption rates were 49% and 45% more in the OB group compared with NW group, respectively. The MEE and net metabolic cost were also found significantly greater in obese children. No differences were found between obese and normal weight groups in net metabolic rate, normalized MEE and mechanical efficiency.

3.4 DISCUSSION

3.4.1 Effect of childhood obesity on the gait biomechanics.

All participants were walking at self-preferred walking pace, which allowed us to determine the effect of obesity on the nature gait characteristics of children. Existing cross-sectional studies reported that obese children prefer to walk with a slower self-selected walking speed and cadence; a larger stride width and shortened stride length during walking were also characterized (Browning & Kram, 2007; Hills & Parker, 1991a; McGraw et al., 2000). Excess body weight also resulted in more time spent in stance and less time in swing, and a longer period of double support (Hills & Parker, 1991a). These adaptations are considered to arise from the need to maintain stability during gait (Browning, 2012). Our results confirmed the previous findings about the impact of childhood obesity on gait spatiotemporal parameters at a self-selected speed.

Consistent with a previous study (Shultz, Sitler, et al., 2009), we found that obese children presented similar joint angle and moment patterns with normal weight children. In the sagittal plane, the NW group had greater knee and ankle ROM during stance phase due to a faster walking speed. In the frontal plane, the significantly larger hip ROM throughout the gait cycle in the OB group was thought to avoid friction of the inner thighs during walking (Nantel et al., 2006). Although obese children walked at a slower speed, the absolute joint moments of hip, knee and ankle were greater than normal weight children. This finding emphasizes the impact of excess mass on the amount of force applied to the joints. The hip moments, even normalized to body weight, were still significantly larger in the OB group. Ankle plantarflexors provide majority of propulsion during walking (Kepple et al., 1997). Because of a faster self-selected walking speed, the ankle plantarflexor moment was significantly larger in the NW group after normalization. Thus, our results indicated that the excess body weight was the main factor causing higher joint moments. Previous studies have demonstrated this mass-driven negative impact can further cause injuries, pain and the development of certain disease in weight-bearing joints (Taylor et al., 2006; Tsiros et al., 2011; Wearing et al., 2006b).

The normalized isokinetic results showed that obese children had a significantly lower relative muscle strength at the knee and ankle than normal weight children. This finding may be explained by a previous point of view that obese

children alter their gait to compensate for potential muscle weakness (McMillan et al., 2010). There is a significant increase in the hip flexor moment and a decrease in the plantarflexor moment at the late stance before leg swing, which was consistent with similar findings reported by McMillan et al. (2010). McMillan et al. (2010)considered the change of hip flexor and ankle plantarflexor moment as a compensation mechanism that obese individuals pull the limb into swing by generating more hip flexion forces, rather than push it through with potential weak ankle plantarflexion muscles. Less knee flexion through stance may also compensate for unstable knees caused by knee extensor weakness (McMillan et al., 2010).

It is acknowledged that muscle strengthening has a beneficial effect on physical performance, injury prevention and metabolic consequences (Behringer et al., 2010; Faigenbaum, Westcott, Loud, & Long, 1999). However, according to the kinetics findings in this study, the negative impact of greater absolute joint loads in obese children is more likely to be driven by body weight. In addition, it is still not clear if the muscle weakness presents during walking in obese children. Therefore, for obese individuals, reducing body weight may be the most effective way to improve musculoskeletal health rather than muscle strengthening.

3.4.2 Effect of childhood obesity on the gait energetics

This study compared the differences in gait biomechanics and energetics between obese and normal weight children. Results showed that obese children and normal weight children have similar metabolic rate (J/kg/m), normalized MEE (J/kg/m) and mechanical efficiency during walking at self-selected speed. Body mass plays a dominant role in the gait characteristics and walk energetics during normal walking.

Many studies have been performed to compare only the metabolic or the mechanical cost between obese and normal weight individuals during walking. Nantel et al. (2006) reported that compared to normal weight children, obese children used more mechanical energy when walking at the same speed. Their results also suggested that obese children were less efficient than normal weight children in transferring mechanical energy within the hip flexors from the stance phase to the swing phase. Butte et al. (2007) showed that the energy expenditure in obese children walking at 1.25 m/s was significantly greater than in normal children walking at 1.25 m/s, by about 5.25 kJ/min. These studies suggested that walking for obese

individuals is more energy expensive than it is in their normal weight counterparts at given speeds. Browning and Kram (2005) found both obese and normal weight individuals preferred to walk at speeds where the gross energy cost per distance was minimized. Malatesta et al. (2009) did not find any significant differences in the external work per unit mass between obese and lean adults while walking at self-selected speeds.

Only one research group quantified the mechanical energy and metabolic energy simultaneously for obese children during walking (Peyrot et al., 2009). However, their study did not use a forceplate-based method to determine the mechanical work. The mechanical cost was calculated by the velocity and displacement of COM, which was measured by a tri-axial accelerometer and gyroscope device. The reported mechanical work should be smaller than actual mechanical energy expenditure, because each individual segment power could not be measured by this method. By contrast, the present study used the inverse dynamics method to compute the mechanical work by summing power entering and leaving the segment a gait cycle (Winter, 2005). Compared to COM movement, the joint moments have been reported to be more closely related to the actual muscular sources, as well as the transformation of mechanical energy in locomotion (Umberger & Martin, 2007). Furthermore, participants in their study were required to perform four given speed walking (0.75, 1, 1.25, and 1.5 m/s), the metabolic and mechanical cost at self-selected speed was not determined. The energetics of natural walking strategy for obese children were still unclear in their study. No direct studies have compared the mechanical efficiency during self-selected speed walking between obese and normal weight children so far.

3.4.3 Walking strategy of obese children

Many biomechanists have proposed that normal-weight adults will innately adopt energy-efficient gait strategies and velocities (Gordon et al., 2009; McNeill Alexander, 2002; Zarrugh et al., 1974). A U-shaped relationship has been reported between the energy cost and walking speed; each individual has a preferred walking speed which minimizes the energy costs in normal gait (Holt et al., 1991; Zarrugh et al., 1974). Based on these findings, Browning and Kram (2005) suggested that the body can adjust and minimize the energy cost per distance. They also reported the walking speed that corresponded to the minimum metabolic rate was 8% slower for

the obese adults. The present study found similar results in that obese children walked at a speed 12% slower speed than normal weight controls. Therefore, the ability to minimize the energy cost of self-selected speed may also exist in obese children.

In this study, most of the gait differences found in obese children could be considered to reduce the energetic cost except for the step width. Donelan et al. (2001) have reported that the normalized mechanical work and the net metabolic rate increased with the square of step width values above the self-selected step width. Peyrot et al. (2009) also suggested the greater net metabolic cost of walking by obese individuals may be a result of their greater step widths. The participants in the OB group had a wider step and wider legs because of the excess adipose tissue between thighs. Interestingly, although the obese participants walked with significantly larger step width, they still maintain the normalized MEE and metabolic rate. The slower walking speed and longer stride time reduced the COM velocity and acceleration, and further reduced the mechanical work required to redirect and raise the COM. In addition, slower walking speed with less cadence and a longer stance phase could limit COM displacement in the mediolateral direction. Other studies have reported that greater mediolateral COM displacement was accompanied by higher muscle activations (Donelan, Shipman, Kram, & Kuo, 2004), and a higher metabolic cost associated with raising the COM (Neptune, Zajac, & Kautz, 2004). Therefore, the findings of this study suggested that the preferred gait variables were selected by obese children to minimize metabolic cost and the mechanical work required to move their excess body mass.

3.4.4 Limitation

Several limitations should be highlighted in this study. First, skin movement artefacts in 3D gait analysis may affect the accuracy on joint angles and moments, especially in transverse planes. To minimize this effect, we used an elastic band to secure participants' thick abdominal adipose tissue in order to reduce the movement artefacts of the markers. The findings of this study just pertain to self-selected walking speed, not assessing the energetic cost at various speeds presents another limitation of our study. We cannot provide direct evidence that walking at self-selected speed is the most efficient way. Walking speed plays an important role in walking energetics. A slower walking speed in the OB group may be the main

contributor to the similar normalized energy cost and mechanical efficiency with the NW group. More comprehensive studies involving all participants walking at same speeds (including speeds that are faster and slower than their self-selected speed) are also needed to provide insight into the gait strategy selected by obese individuals in different conditions. The difference in body height between groups may also slightly affect the walking speed. In addition, we did not test body composition, which is usually measured by dual energy X-ray (DEXA), bioelectrical impedance analysis (BIA) or other methods. It may also be worthwhile to investigate the effect of body fat mass or fat free mass on the energy cost and efficiency in the future. Finally, given the cross-sectional nature of this study, it is impossible to determine the cause and effect among excess body mass, gait biomechanics, and energetics in children. Further intervention studies are needed to investigate the beneficial effect of weight loss and muscle strengthening on the musculoskeletal system.

3.5 CONCLUSION

In conclusion, excess body mass plays a dominant role in gait biomechanics and energetics. Obese children may choose an adaptive walking strategy that can minimize the increase of metabolic cost and the mechanical work required to lift, lower, accelerate, and decelerate their excess body weight.

Chapter 4: The effect of weight loss on gait biomechanics and energetics

4.1 INTRODUCTION

Previous studies have found differences in gait biomechanics and energetics between obese and normal weight children (Hills & Parker, 1991a; McGraw et al., 2000; McMillan et al., 2010). Walking with a lower speed and more erect posture was considered an adaptive strategy selected by obese children to minimize the energy cost and muscle activation (Browning & Kram, 2007). However, excess body weight increases the loading on the weight-bearing joints and causes continuous overload on musculoskeletal structures during movement (Wearing et al., 2006b). If obesity continutes from childhood into adulthood, it is more likely that the obese individuals may have structural changes in the locomotion system and develop potential joint diseases at a younger age (Taylor et al., 2006). Gushue et al. (2006) suggested that abnormal knee load during walking in obese children could result in knee osteoarthritis (OA) in the long term. In obese adults with OA, Messier et al. (2005) found increased joint loading and abductor moment at the knee.

Results of previous studies have formed the foundation of the current knowledge on mass-driven changes in walking mechanics and energetics, by using simulations of weight changes (weight loading or unloading). Grabowski, Farley, & Kram (2005) simulated both reduced weight and added load condition. The results showed that the reduced demands on muscles to support a greater weight during stance and to accelerate a greater mass during step-to-step transitions resulted in decreased energy cost (Grabowski et al., 2005). Knee flexion and hip ROM were also reported as being greater when an extra load was added on the body (Smith, Roan, & Lee, 2010). Griffin, Roberts, and Kram (2003) found that net metabolic cost and external mechanical work increased as participants carried heavier loads across four given walking speeds. Walking efficiency followed an inverse U-shaped trend, and it is both lower at the fastest speed and lowest speeds (Griffin et al., 2003). Although those studies suggested that the body mass exerted great influence on gait mechanics and energetics, whether or not this knowledge based on simulated conditions can be applied to actual weight changes remains to be answered.

There have been a number of experimental studies devoted to uncovering the beneficial effect of weight loss on musculoskeletal health (Wearing et al., 2006b). Surgical obesity treatment reduces the risk of developing musculoskeletal pain in adults at most sites of the body (Hooper et al., 2007; Peltonen et al., 2003). Significant improvements in physical function and pain were found after diet and exercise-induced weight loss in obese adults with OA (Messier, Gutekunst, Davis, & DeVita, 2005). However, less is known about the cause-and-effect relationship between excess body mass, gait biomechanics and energetics, especially in children. A metabolic surgery-induced weight loss of 27% in obese adults increased joint range of motion (ROM) in lower extremity, swing time, stride length and gait speed, and decreased the ankle and frontal plane knee torques during walking (Hortobágyi et al., 2011); however, walking energetic was not studied. Only one research group has reported the changes in both walking biomechanics and energetics (Peyrot et al., 2010, 2012). They found obese children who lost 5% of initial body weight had a significant reduction in energy cost associated with biomechanical changes during walking (Peyrot et al., 2010). An increased walking economy was also reported in a similar study (Peyrot et al., 2012). However, the participants grew taller during the 12 week period. It is not clear whether the changes in gait were simply mass-driven, since the natural growth in height and weight of children could affect the results. In addition, none of these studies tested a non-weight-loss control group that would have allowed to assess the reliability of the dependent variables.

Therefore, the purpose of the present study was to investigate the cause-and-effect relationship between excess body mass and gait modification in obese children. This study quantified gait kinematics, kinetics, mechanical and metabolic energy cost during walking after substantial weight loss in a short period. It was hypothesized that the reduction in metabolic cost and mechanical work after weight loss is associated with changes in the biomechanical parameters of walking.

4.2 METHODS

4.2.1 Participants

This study recruited 19 obese children (9 boys and 10 girls) from a residential weight loss intervention program provided by a weight management centre in Shanghai, China. A further 18 age-matched obese children (8 boys and 10 girls) were

recruited as a control group by advertisements placed in the local communities and primary schools. The inclusion criteria were children aged between 8 and 12 years, no orthopaedic or neurological disorders that could interfere with the gait pattern, and a BMI level above age and gender specific cut-off points for obesity as defined by Cole et al. (2000). Children in the intervention group who had lost less than 5% of body mass would be excluded from further analyses, because 5% weight loss is a common clinical endpoint (Blackburn, 1995). The control group received no treatment and engaged in unmonitored summer vacation activities during the time the weight loss intervention was taking place. Children in the control group who had lost or gained more than 5% of body mass would be also excluded from further analyses. Permission to collect data on participants was obtained from the intervention provider and school principals, respectively. All children and their guardians read and signed an informed consent form approved by the Human Research Ethics Committee of the University of Auckland. Table 4.1 shows the demographics of the participants in the intervention group and the control group at the baseline.

Table 4.1 Demographics of participants at the baseline (means ± SD)

	Intervention group n=19	Control group n=17	*t*	*d*	*p*
Age (years)	10.7 ± 1.1	10.7 ± .9	.12	0.041	.930
Height (cm)	154.1 ± 7.3	151.5 ± 6.6	1.12	.383	.280
Weight (kg)	71.9 ± 11.1	65.9 ± 12.1	1.55	.532	.130
BMI	30.2 ± 3.3	28.5 ± 3.5	1.50	.514	.153
Body circumferences					
Waist (cm)	95.6 ± 9.0	91.7 ± 8.7	1.32	.452	.204
Hip (cm)	99.9 ± 7.5	97.2 ± 8.0	1.05	.358	.305
Thigh (cm)	63.0 ± 5.4	62.6 ± 4.6	.24	.082	.837

4.2.2 Diet and exercise induced weight loss intervention

The weight loss intervention program is a 4-week short term residential summer camp for overweight and obese children and adolescents. The program was established at Shanghai Univeristy of Sport in 2005 to undertake a moderate dietary restriction, fun-based exercise interventions, nutrition and behaviour education, and research to successfully treat childhood obesity. All overweight and obese individuals aged 8-18 years are eligible to attend the program after a full medical examination and an interview to determine their suitability and willingness to adhere to the intervention. All enrolled campers are divided into different groups according to age and gender for all program activities. Housing assignments include three campers per room and onsite staff accommodation. The daily exercise program included one non-impact water-based exercise session and one aerobics session per day. Each session lasted for 2-3 hours and was led by certified instructors. Daily calorie intake ranged from 1600 to 2000 kcal/day dependent on the participants' own basal metabolic rate (Wang, Chen, & Chen, 2011). Energy was provided as 3 meals and 1 snack each day by registered dietitians and no other food is allowed. Parents paid for their children's attendance. In addition, family members and were encouraged to visit their children to help them adhere to the program.

4.2.3 Experimental Procedures

All participants were tested twice with the same measurements by the same investigators. The baseline test was done one day before the weight loss intervention program. The second test started one day after the intervention was completed. Body weight was measured to the nearest 0.1 kg with an electronic weight scale, and height was measured with a wall-mounted stadiometer to the nearest 0.5 cm. The body circumferences were measured by the same person using a flexible tape with the participants standing. The waist circumference was measured between the lowest rib margin and the iliac crest. The hip circumference was measured around the maximum protrusion of the buttocks. Thigh circumference was measured below the gluteal fold. The isokinetic muscle strength, gait kinematics, kinetics and metabolic cost were measured using the same protocol described in Chapter 3.

4.2.4 Statistical Analysis

Means and standard deviations of measured parameters were calculated for each group. The sagittal, frontal joint ROMs were calculated by the number of degrees that a joint moved in the three planes respectively. Both the absolute value and weight normalized value of sagittal and frontal joint moment were calculated. The Shapiro–Wilk normality test was used to check if all data were normally distributed. Homogeneity of variances were also checked for all variables. A two way analysis of variance (ANOVA) with repeated measures was conducted to determine the effect of the intervention on measures of spatiotemporal parameters, joint kinematics and kinetics. Group (intervention vs. control) was used as between-subjects factor and time of measurement (baseline vs. 4 weeks) as a within-subjects factor. Alpha level was adjusted to < .025 to control for the Type I error using the Bonferroni method. Correlations (Pearson's r) were used to examine the relationships between weight loss and changes in energetics parameters. All data were analysed using SPSS 18.0 (SPSS Inc, Chicago, IL).

4.3 RESULTS

The results of two-way ANOVA found a significant interaction in body weight, BMI and body circumferences. The effect of intervention (time factor) was also significant and was the main effect. The children in the intervention group lost 5.8 ± 1.2 kg, 8.0% of initial weight after 4 weeks and no participant dropped out of the intervention program. BMI was reduced by 2.4 ± .5 kg/m^2. The circumferences of waist, hip, and thigh were significantly reduced by 8.4 ± 1.3 cm, 7.0 ± 1.3 cm, and 3.3 ± 2.0 cm, respectively ($p < .01$). In the control group, one girl failed to complete data collection at the end of 4 weeks, so 17 of 18 participants entered the final analyses. No significant changes of anthropometric measures and isokinetic strength were found for the other participants. There was no significant change in isokinetic muscle strength for both intervention and control group (Table 4.2).

Neither statistically significant interactions nor effect of intervention for spatiotemporal gait parameters were found (Table 4.3). The effect of group was significant for cadence ($p = .017$) and stride time ($p = .021$).

Summaries of joint ROM during the stance phase were given in Table 4.4 and Table 4.5, respectively. Figure 4.1 and Figure 4.2 shows the joint angles and

normalized joint moment over 100% gait cycle. There were significant time-by-group interactions for hip abduction/adduction ROM during the stance phase and the swing phase, and knee varus/valgus ROM during the stance phase. The effect of group was significant for hip flexion/extension ROM, but there were no interactions. Changes of ankle ROMs were slight and not significant after 4 weeks. Table 4.5 shows the joint ROM during the swing phase. Absolute values of peak joint moment at baseline and 4 weeks are presented in Table 4.6. Time-by-group interactions were found significantly at the peak moment of hip flexor and knee abductor. A 17.8% decrease was found in the peak moment of hip abductor. The main effect of intervention was significant ($p = .016$), but there were no interactions. The effect of group was significant for the knee flexor moment ($p = .007$). The effect of intervention was significant for the peak moment of hip abductor ($p = .016$) and the ankle plantar flexor ($p = .023$), without any interaction. After normalized to body weight, only hip flexor moment was found significantly reduced after intervention. There was a 32.5% increase in the peak hip extensor moment for the intervention group, however, the p-value was slightly larger than .*025* and indicated nonsignificance (Figure 4.2).

Summaries of joint powers of the two groups were presented in Figure 4.3. For the intervention group, the hip power absorption during the stance phase (H2) was significantly reduced after weight loss. The ankle power generation (A2) was significantly higher after intervention.

Table 4.2 Changes of anthropometry parameters and isokinetic strength after 4 weeks *(means ± SD)*

	Intervention group		Control group		Two-way ANOVAs	
Parameters	Change	%	Change	%	F(1,34)	η^2
Height (cm)	.3 ± .4	.2 ± .3	.6 ± .9	.4 ± .6	2.321	.064
Weight (kg)	-5.8 ± 1.2**	-8.0 ± 1.4	.4 ± .9	.6 ± 1.6	264.808	.886
BMI (kg/m^2)	-2.4 ± .5**	-8.1 ± 1.4	- .2 ± .3	- .6 ± 1.2	296.515	.897
Body circumferences						
Waist (cm)	-8.4 ± 1.3**	-7.0 ± 4.3	.1 ± 1.7	.2 ± 1.8	296.515	.897
Hip (cm)	-7.0 ± 4.3**	-6.9 ± 4.1	.1 ± 1.6	.1 ± 1.7	39.573	.538
Thigh (cm)	-3.3 ± 2.0**	-5.2 ± 3.3	.1 ± .7	.2 ± 1.2	47.278	.582
Isokinetic strength (Nm)						
Knee flexor	1.0 ± 2.4	6.0 ± 12.5	- .4 ± 2.5	-1.8 ± 12.2	2.811	.076
Knee extensor	.1 ± 3.4	0.3 ± 7.7	-2.3 ± 5.4	-4.5 ± 11.2	.467	.014
Ankle dorsiflexor	-1.1 ± 7.4	-1.9 ± 10.6	1.7 ± 9.1	2.8 ± 14.5	.092	.003
Ankle plantarflexor	.7 ± 3.2	3.1 ± 11.8	.5 ± 6.9	1.7 ± 22.5	.985	.028

Note: Two-way ANOVAs: alpha level <0.025; η^2 =effect size (eta-squared); p_{t*g}, time by group interaction; **$p_{t*g} < .01$.

Table 4.3 Spatiotemporal gait parameters *(mean±SD)*

Variable	Intervention group		Control group		Two-way ANOVAs		
	Before	After	Before	After	p_t	p_g	p_{t*g}
Speed (m/s)	1.11 ± .06	1.10 ± .06	1.15 ± .07	1.16 ± .09	.950	.031	.488
Cadence (step/min)	112.6 ± 6.0	111.7 ± 5.0	117.4 ± 7.6	116.9 ± 6.9	.294	**.017**	.790
Step width (cm)	17.5 ± 4.6	16.7 ± 4.2	16.4 ± 3.5	17.0 ± 3.0	.873	.757	.171
Stride length (cm)	118.5 ± 7.4	118.7 ± 8.4	117.9 ± 6.3	119.2 ± 5.2	.486	.974	.640
Stride time (s)	1.07 ± .06	1.08 ± .05	1.03 ± 0.07	1.03 ± .06	.316	**.021**	.787
Double support (%)	20.3 ± 2.3	19.1 ± 2.4	18.9 ± 2.7	18.4 ± 2.7	**< .001**	.745	.728
Single support (%)	39.9 ± 1.2	40.3 ± 1.3	40.6 ± 1.4	40.5 ± 1.3	.527	.210	.279
Stance Phase (%)	60.3 ± 1.3	59.9 ± 1.2	59.6 ± 1.4	59.6 ± 1.2	.266	.199	.328

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 4.4 Joint kinematics (ROM) during the stance phase *(means ± SD)*

Joint	ROM	Intervention group		Control group		Two-way ANOVAs		
		Before	After	Before	After	p_t	p_g	p_{t*g}
Hip	Flexion/ Extension	39.6 ± 5.3	38.8 ± 5.6	35.8 ± 4.4	34.3 ± 3.8	.197	**.010**	.678
	Abduction/ Adduction	16.0 ± 3.5	11.9 ± 3.8	11.8 ± 4.5	11.2 ± 4.3	**.001**	.068	**.016**
Knee	Flexion/ Extenion	31.0 ± 6.1	34.8 ± 6.0	35.7 ± 7.0	37.2 ± 6.1	.072	.052	.422
	Varus/ Valgus	6.2 ± 3.3	9.5 ± 5.4	9.0 ± 14.7	8.5 ± 4.9	.079	.559	**.015**
Ankle	Dorsiflexion/ Plantarflexion	25.0 ± 5.8	25.0 ± 4.1	24.8 ± 5.4	25.7 ± 4.1	.620	.862	.665
	Inversion/ Eversion	3.3 ± 2.2	3.3 ± 2.2	4.2 ± 3.2	4.2 ± 3.1	.914	.314	.925

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 4.5 Joint kinematics (ROM) during the swing phase *(means ± SD)*

Joint	ROM	Intervention group		Control group		Two-way ANOVAs		
		Before	After	Before	After	p_t	p_g	p_{t*g}
Hip	Flexion/ Extension	36.8 ± 5.9	34.5 ± 6.4	32.4 ± 5.7	32.3 ± 4.7	.096	.006	.974
	Abduction/ Adduction	12.1 ± 4.2	9.3 ± 3.0	8.6 ± 3.0	8.5 ± 2.4	.013	.046	**.018**
Knee	Flexion/ Extenion	62.3 ± 9.0	60.4 ± 7.8	64.9 ± 6.0	66.2 ± 6.4	.836	.076	.257
	Varus/ Valgus	10.15 ± 5.12	11.71 ± 6.94	14.7 ± 4.7	15.1 ± 5.08	.195	.042	.395
Ankle	Dorsiflexion/ Plantarflexion	20.0 ± 6.5	18.1 ± 5.7	19.8 ± 4.2	19.2 ± 3.7	.137	.780	.453
	Inversion/ Eversion	2.9 ± 3.5	2.9 ± 2.3	4.1 ± 5.0	4.2 ± 4.4	.973	.329	.905

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 4.6 Peak joint moment (Nm) during the stance phase *(means ± SD)*.

Joint	Moment	Intervention group		Control group		Two-way ANOVAs		
		Before	After	Before	After	p_t	p_g	p_{t*g}
Hip	Flexor	87.27 ± 23.99	70.29 ± 18.96	82.54± 28.10	86.10± 28.91	.030	.406	**.002**
	Extensor	16.37 ± 6.81	21.69 ± 9.57	20.78 ± 9.41	19.62± 9.38	.140	.693	.025
	Abductor	66.82 ± 13.74	54.90 ± 15.79	68.77 ± 13.12	65.07± 16.08	**.016**	.183	.190
	Adductor	8.30± 3.56	8.93 ± 3.45	6.52± 6.30	6.22± 5.24	.826	.158	.548
Knee	Flexor	12.16 ± 3.54	11.20 ± 2.38	8.10 ± 4.23	8.45± 4.54	.660	**.007**	.349
	Extensor	80.03 ± 16.43	73.91± 19.55	59.9 ± 33.68	60.69± 32.68	.323	.081	.204
	Abductor	24.54± 8.90	16.43± 9.55	20.79 ± 12.04	18.90± 8.92	**.007**	.596	**.015**
	Adductor	4.58± 2.55	6.78± 8.03	6.36 ± 3.97	5.79± 3.47	.502	.778	.259
Ankle	Dorsiflexor	9.19± 4.97	8.09± 3.08	6.35 ± 3.88	5.90± 4.73	.349	.066	.693
	Plantarflexor	80.67± 17.43	73.07± 13.10	83.78 ± 15.58	82.78± 14.12	**.023**	.222	.076
	Everter	3.17± 2.25	3.28 ± 2.73	2.48 ± 3.40	3.81± 3.67	.157	.941	.224
	Inverter	6.04± 3.32	4.28± 1.96	8.25± 6.12	7.73± 5.79	.054	.079	.284

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

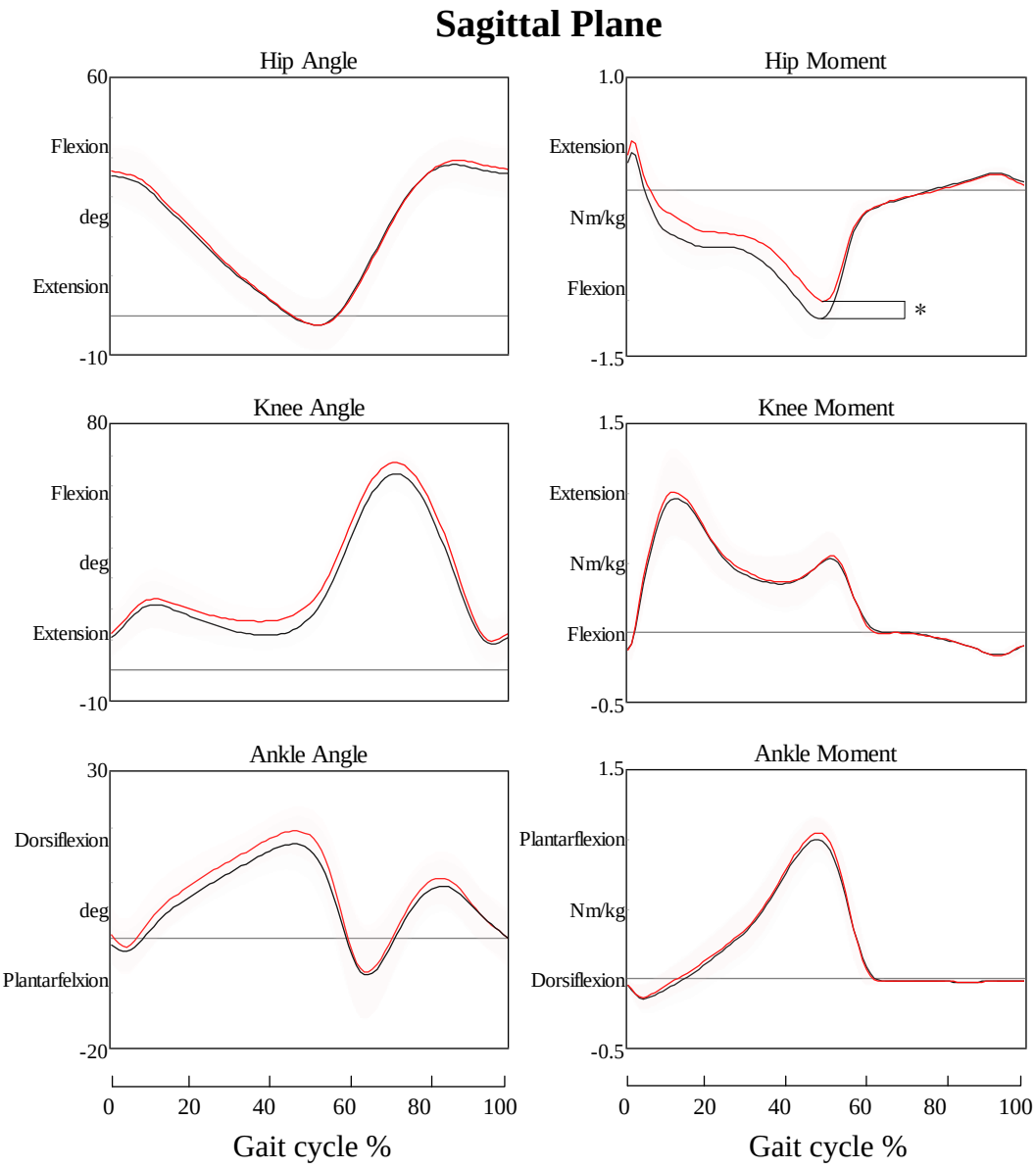


Figure 4.1 Mean joint angles and joint moments (before: black line, after: red line), and standard deviation (before: grey shaded area, after: red shaded area) over a gait cycle in the sagittal plane. * $p < .025$. There were no statistically significant changes in the control group (Appendix D, Figure D.1).

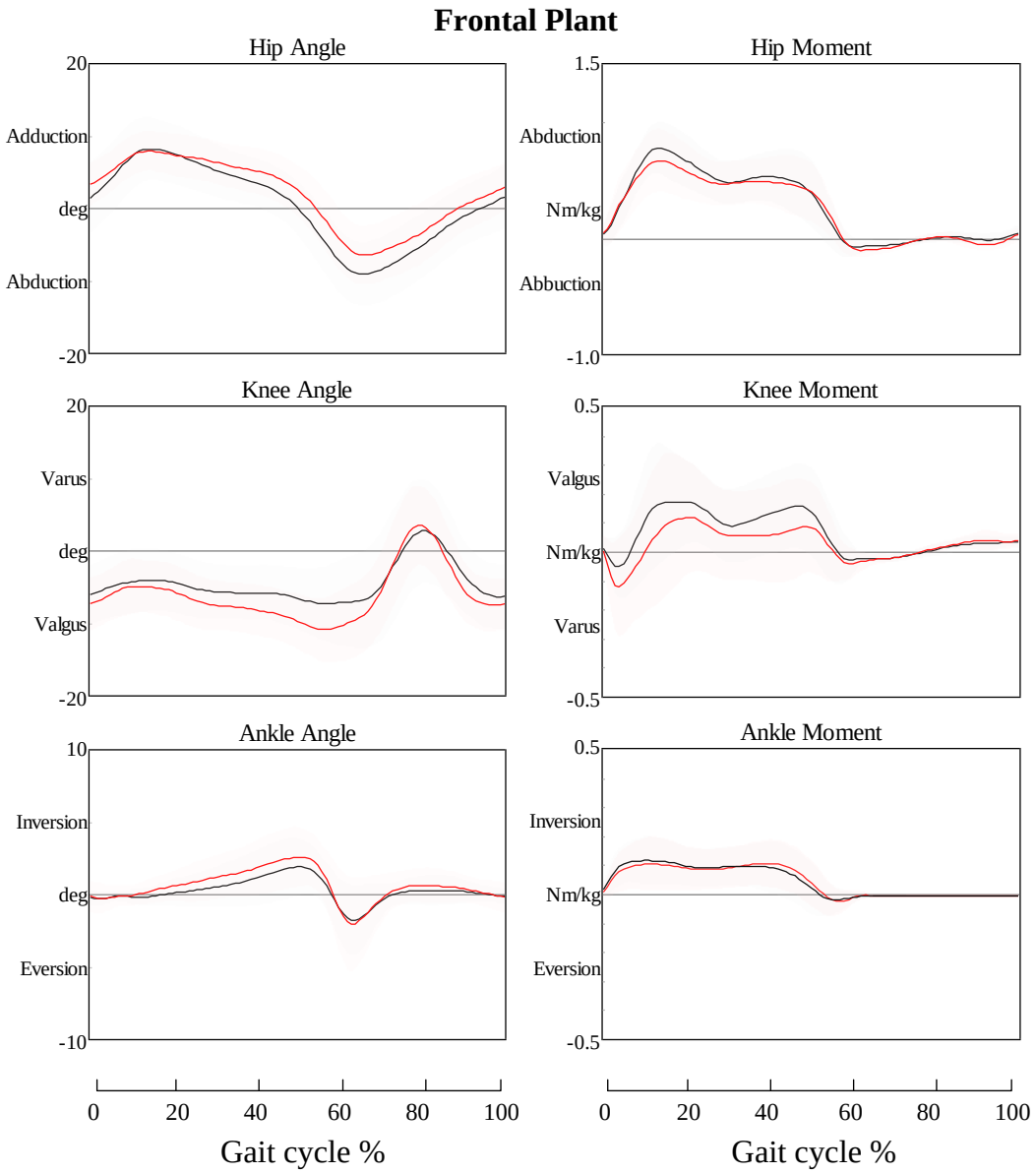


Figure 4.2 Mean joint angles and joint moments (before: black line, after: red line), and standard deviation (before: grey shaded area, after: red shaded area) over a gait cycle in the frontal plane. *$p < .025$. There were no statistically significant changes in the control groups (Appendix D, Figure D.2).

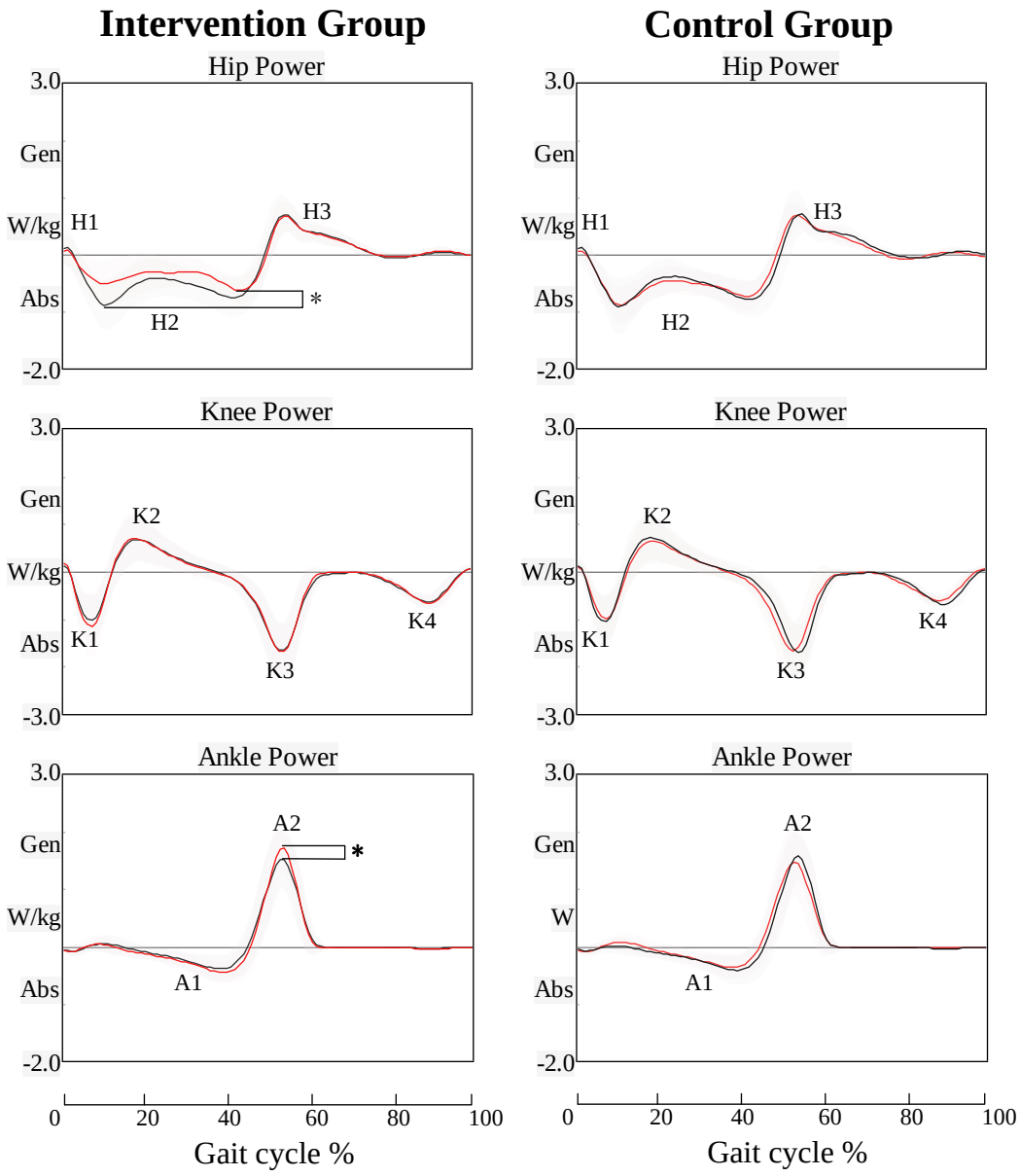


Figure 4.3 Mean joint powers (before: black line, after: red line), and standard deviation (before: grey shaded area, after: red shaded area) over a gait cycle. *$p < .025$

Table 4.7 The effects of weight loss on walking energetics *(means ± SD).*

Parameters	Intervention group		Control group		Two-way ANOVAs		
	Baseline	4 weeks	Baseline	4 weeks	p_t	p_g	p_{t*g}
Energetic parameters							
P_{gross} (W)	318.0± 52.3	293.9± 48.8	330.5± 52.7	332.4± 55.2	**0.004**	0.125	**0.001**
P_{rest} (W)	106.2± 15.2	106.8± 13.0	110.0± 15.9	112.3± 16.1	0.322	0.345	0.563
E_{net} (J)	222.5± 45.6	198.7± 46.1	224.1± 45.6	225.2± 43.4	**<0.001**	0.353	**<0.001**
MEE (J)	66.6± 15.0	58.7± 13.1	62.8± 13.8	63.1± 13.2	**<0.001**	0.957	**<0.001**
ME (%)	28.1± 1.7	27.9± 1.9	27.0± 2.0	26.5± 1.7	0.032	0.337	0.337
C_{net} (J/kg/m)	2.8± 0.3	2.7± 0.3	3.0± 0.2	2.9± 0.2	0.095	0.044	0.514
C_{mech} (J/kg/m)	0.8± 0.1	0.8± 0.1	0.8± 0.1	0.8± 0.1	0.144	0.953	0.332
Joint mechanical work (J/kg)							
H2	0.27± 0.06	0.19± 0.05	0.24± 0.05	0.26± 0.09	0.039	0.149	**0.002**
A2	0.15± 0.05	0.15± 0.03	0.17± 0.03	0.17± 0.04	0.918	0.095	0.508

Note: P_{gross}, gross metabolic power; P_{rest}, rest metabolic power; E_{net}, net metabolic cost; MEE, mechanical energy expenditure; ME, mechanical efficiency; C_{net} net metabolic rate; C_{mech}, normalzied MEE. Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 4.8 Correlation coefficients and p-value between weight loss and change in walking energetic parameters

	Weight	P_{gross}	P_{rest}	E_{net}	MEE	ME%	C_{gross}	C_{net}	C_{mech}
Weight	1								
P_{gross}	.511 **.001**	1							
P_{rest}	.117 .496	.394 **.017**	1						
E_{net}	.722 **.000**	.776 **.000**	.066 .704	1					
MEE	.717 **.000**	.507 **.002**	.082 .635	.849 **.000**	1				
ME	-.068 .691	-.262 .122	.112 .516	-.196 .252	.090 .601	1			
C_{gross}	-.224 .189	.207 .226	.392 **.018**	.175 .308	.205 .230	.256 .132	1		
C_{net}	.027 .877	.351 **.036**	-.148 .388	.553 **.000**	.476 **.003**	-.217 .204	.557 **.000**	1	
C_{mech}	.149 .386	-.082 .635	.028 .873	.314 .062	.639 **.000**	.360 **.031**	.520 **.001**	.609 **.000**	1

Despite 8% weight reduction, the net metabolic cost, mechanical efficiency, gross metabolic rate, net metabolic rate and the normalized MEE remained unchanged, whereas there were significant reductions in gross and net metabolic cost. The MEE also significantly reduced after weight loss (Table 4.7).

Table 4.8 shows the correlation coefficients and p-value between weight loss and the changes of walking energetics parameters. Weight loss was significantly correlated with P_{gross}, E_{net}, and MEE ($p < .05$).

4.4 DISCUSSION

This study evaluated the short-term outcomes of a multidisciplinary residential weight loss program for obese children in terms of gait biomechanical parameters and walking energetics. To our knowledge, this is the first study to investigate the effect of weight loss in these variables. The data provide critical information in the assessment and treatment for the development of musculoskeletal problem associated with obesity.

4.4.1 Effectiveness of the weight loss intervention program

All participants who underwent an intervention lost more than 5% of their body weight compared to baseline (minimum: 5.5%, maximum: 11.6%). BMI and body circumferences were also significantly reduced in the intervention group after a treatment period of 4 weeks. These data strengthen the evidence from previous reports regarding the effectiveness of diet and exercise induced weight loss treatment in reducing the body weight and obesity-related measures in a relatively short time period (Huelsing, Kanafani, Mao, & White, 2010; Wang et al., 2011). In the absence of any strength training in the intervention program, weight loss did not induce a change in absolute muscular strength for the intervention group.

Although a few studies have been found to investigate the effect of weight loss on gait pattern, the duration of the reported weight loss intervention ranged from 3 months to 12 months. Natural growth and development in children (i.e. height growth) could induce changes in walking mechanics and the metabolic cost (Peyrot et al., 2010; Weyand, Smith, Puyau, & Butte, 2010), and further influence the outcomes of these studies (Peyrot et al., 2012). Therefore, the major strength of this study is that substantial weight loss was achieved in a short time period. It is not my intent to argue whether a long term or a short term weight loss would be more beneficial for obese individuals. However, a short period treatment appears suitable to minimize the potential interfering factors when investigating changes to gait. The results provide valuable information to evaluate more accurately the true mass-driven effects on walking biomechanics and energetics in obese children. In addition, inclusion of a non-weight-loss control group has allowed us to assess the reliability of the dependent variables.

4.4.2 Effect of weight loss on gait biomechanics

Due to the considerable amount of weight loss in the intervention group, an increase in walking speed or cadence was expected compared with the control group. However, there was no difference in any of the spatiotemporal gait parameters after weight loss. One possible reason may be the percentage of weight reduction was not large enough to bring changes to these parameters. Aaboe et al. (2011) found a 13.6% weight reduction resulted in a 4% increase in self-selected walking speed. Peyrot et al. (2010) found a significantly increased stride length after 12 weeks weight loss. Hortobágyi et al. (2011) reported that 0.15 m/s increase of self-selected speed and 0.11 m increase of stride length after 33.6% of the total weight loss occurred in 7 months. They also suggested that weight loss may produce adaptive changes in gait behaviour, and there is a level of weight loss needed for gait velocity to increase. A more substantial loss in body weight may be an essential condition to produce changes in spatiotemporal parameters. However, using the spatiotemporal parameters did not allow to test this hypothesis.

Sagittal and frontal plane kinematics and kinetics have been reasonably well described (McMillan et al., 2010; Shultz et al., 2009), but the transverse plane data are inconsistent and dependent on variations in joint positions and the specific methodologies used (Levangie & Norkin, 2011). This chapter will discuss the sagittal and frontal plane joint kinematics and kinetics.

In the sagittal plane, although the hip joint angle did not change after weight loss, a 6.9% reduction in waist circumference may reduce obstruction at the pelvis and allow participants to flex the hip easier after weight loss. The significant decrease in hip flexor moments and hip power absorption (H2, corresponding to eccentric hip flexor activity) attests that less muscular effort was needed to prevent an abundance of hip flexion during stance. Much evidence has shown that the reduction of ROM in the knee and ankle were associated with obesity (DeVita et al., 2003; Messier, 1994). Although greater knee flexion angles were seen during the stance phase, neither joint ROM nor joint moment changed significantly after weight at knee and ankle. Increases in the swing time and stride length, can result in a larger joint ROM in the sagittal plane. However, the spatiotemporal gait parameters did not change after weight loss. This might explain similar sagittal plane joint ROM at knee and ankle. Browning and Kram (2005) reported that obesity associated with a higher

peak knee extensor moment when walking at the same speeds. This relationship between body fatness and knee moment was not statistically significant in our study. Ankle plantarflexors provide support and propulsion during walking (Kepple et al., 1997). A 9.4% decrease in absolute amount of the plantarflexor moment suggested that less muscle forces were needed to support the body at the stance phase, but the differences were not significant ($p = .076$). By contrast, Hortobágyi et al. (2011) found a reduction in the plantarflexor moment during walking was a hallmark of weight change in morbidly obese individuals. However, studies also suggested that the importance of ankle kinetics would reduce with weight loss and also became less in individuals who are less obese (Browning & Kram, 2007; Hortobágyi et al., 2011).

In the frontal plane, hip adduction/abduction ROM reduced in the stance phase and the swing phase after weight loss, and a smaller hip abduction angle was found throughout the gait cycle. Excess adipose tissue between the thighs required more hip abduction to avoid friction of the inner thighs during walking (Nantel et al., 2006). Thus, the decrease of hip abduction seems more likely to result from a 5.3% reduction in thigh circumference after weight loss. Reduced hip abduction then further resulted in a 17.8% decrease in the absolute hip abductor moment. However, only the effect of intervention was significant. Knee varus/valgus ROM became more dynamic and increased by 3 degrees. Improved knee ROM has been independently associated with reduced loss of cartilage thickness (Anandacoomarasamy et al., 2012). The reduction of absolute knee abductor moment suggested that less compressive loads were transmitted to the medial compartment of the knee associated with weight loss. Aaboe et al. (2011) found similar results in obese adults with knee osteoarthritis. They observed that a significant reduction of up to 13% in peak knee abductor moment after a 13.5% weight loss, but no significant changes in sagittal plane knee moment during walking at the self-selected speed (Aaboe et al., 2011). Our finding is also in agreement with Messier's study, which reported weight reduction was associated with a reduction in the knee abductor moment (Messier et al., 2005). Much evidence has shown that the obese individuals have greater knee moment in the frontal plane during walking, and this abnormal knee load could have long-term orthopaedic implications (Gushue et al., 2006; Shultz et al., 2009; Wearing et al., 2006b). Weight normalized results suggested that weight loss is the direct way to decrease the abductor moment.

In brief, these findings about the biomechanical effect of weight loss suggested that weight loss with reduced body circumferences caused mass-driven changes in joint kinematics and kinetics. These changes in obese children may have beneficial effects on their musculoskeletal health. It is not surprising that few differences were found in the joint angles, because gait kinematics highly correspond to the spatiotemporal parameters in obese children (Hills & Parker, 1991a; Nantel et al., 2006). Changes in joint moments would be more likely a simple mass-driven adaptation, the absolute joint moments decreased in proportion to weight loss. Hip kinematics and kinetic signified a key gait adaptation to weight loss.

4.4.3 Effect of weight loss on walking energetics

Without changes in walking speed and cadences, weight loss significantly reduced the metabolic and mechanical energy cost during walking, while the metabolic rate and the normalized MEE did not change. These findings are supported by previous physiological studies showing that obese individuals use more metabolic energy than normal-weight participants at a given walking speed (Lazzer et al., 2003; Peyrot et al., 2009). These findings are also in agreement with the findings presented by previous weight loss studies, in particular those of Peyrot et al. (2012). They found both the gross metabolic rate and the net metabolic rate at self-selected walking speed did not change significantly after weight loss. However, mechanical work and mechanical energy were not available in their studies.

Both metabolic and biomechanical factors may account for the greater energy cost of walking in obese individuals compared to normal weight counterparts (Nantel et al., 2011). However, studies showed that the metabolic factors just play a minor role for these differences (Delextrat, Matthew, Cohen, & Brisswalter, 2011; Treuth et al., 1998). Hence, the main factor to explain the reduced energy costs of walking associated with weight loss is the change in biomechanical factors,. This hypothesis was supported by the correlation between the reduction of MEE and the reduced net metabolic cost ($r = .849$, $p < .001$). The reduction of hip mechanical work at stance was significant, corresponding to less eccentric muscle activity to support body weight. Although there was an 8.5% increase in ankle power impulse at “push-off”, knee and ankle mechanical work still decreased in proportion to weight loss. Browning et al. (2009) proposed that the combination of body weight support, swinging heavier legs and wider steps would be the most probable factors for the

greater net metabolic cost of the obese individuals (Browning, McGowan, & Kram, 2009). Consistent with this notion, our results showed that less joint mechanical work was needed to support body mass, and less work was required for lateral leg swing circumduction because of significantly smaller hip abduction (step width did not change after weight loss). Hangrigan et al. (2010) found weight loss could lead to an improvement in balance control. The reduced mechanical work may relate to fewer muscular isometric contractions and cocontraction of antagonist muscles in the mediolateral direction for maintaining balance during walking (Peyrot et al., 2012). However, balance control was not available in this study. In addition, Browning et al. (2006) found that the body fat percentage explained ~ 45% of the net metabolic rate of walking. Although the body composition was not directly measured, the reduction in body circumduction and unchanged muscle strength suggested that the loss of adipose tissue accounted for most of the reduced weight. Reduced body fat could also account for the decreased energy cost.

The hypothesis of this study was supported by the results that weight loss did not increase the mechanical efficiency during walking at self-selected speed, whether this speed had changed or not. C_{net} and MEE decreased in proportion, so that the mechanical efficiency was unaffected by weight loss. Our previous study showed obese children had significantly greater net metabolic cost and MEE than normal weight children, but the differences disappeared after normalizing to both body weight and walking speed (Chapter 3). Peyrot et al (2012) reported weight loss could significantly improve walking economy (decrease in net metabolic rate) at four given speeds but not at the self-selected speed. At a slower walking speed, weight loss could reduce the energy consumption to support body weight during a longer stance phase and propel the body in the direction of progression (Sparrow, 2000). At a faster walking speed, weight loss could cause the metabolic cost to reduce more rapidly than mechanical work by reducing the energy wasted in accelerating and decelerating body segments (Griffin et al., 2003; Sparrow, 2000). Only while walking at self-selected speed, the metabolic cost and MEE would change in proportion to body weight, so the walking economy could not be further improved. Consistent with these findings, we found no significant changes in the net metabolic rate and the normalized MEE after weight loss. Therefore, this study confirms the conclusion of Chapter 3 that obese children are able to select an adaptive walking

strategy that minimized the metabolic cost and the mechanical work required to lift, lower, accelerate, and decelerate their body.

4.4.4 Strategies for selecting preferred walking speed

No association was identified between weight loss and change in self-selected walking speed. This could be explained by our gross metabolic rate data. It is well accepted that the gross metabolic rate vs. walking speed relationship follows a U-shaped trend, both obese and normal weight children prefer walking near the speed at which the gross metabolic rate is minimized (the bottom of the U-shaped curve) (Browning et al., 2006; Peyrot et al., 2010). The data showed that the gross energy cost significantly decreased by 7.6% associated with an 8.0% reduction in weight loss. This may explain why obese children did not increase their self-selected walking speed after intervention, as the gross metabolic rate simply is the gross energy cost divided by both body mass and walking speed. Since the gross metabolic rate remained unchanged and still fell into the bottom of the U-shaped curve where the magnitude was minimized, it was not necessary for obese children to select a significant different walking speed. This finding could be supported by a previous study about the effect of load and speeds on the energetic cost of walking, showing that the gross energy cost was directly proportional to the total mass at a given speed (Bastien, Willems, Schepens, & Heglund, 2005). For instance, carrying a load of 60% body mass increased 60% of gross energy cost while walking at 1.1 m/s.

Furthermore, the bottom of the gross metabolic rate vs. walking speed U-shaped curve showed a relatively flat region covered a range of walking speed. Walking at the speed within this region would show similar gross metabolic rate (Bastien et al., 2005; Peyrot et al., 2009, 2012). Bastien et al. (2005) also reported that the optimal speed for minimal net cost is always between 0.9 m/s and 1.2 m/s for carrying loads ranging from 0%~75% of their body weight. All these evidences suggested that energetics factor might not be the only determinant of the strategies for selecting preferred walking speed. As discussed earlier, obese children did not increase their walking speed after weight loss may also raise from the need to avoid an increase in joint moments. An increase in walking speed would increase the joint moments, and counteract the beneficial effects of weight loss on musculoskeletal loading. Thus, it is worth to qualify joint loads in future studies to clarify the musculoskeletal effects on selecting optimal walking speed.

4.5 CONCLUSION

This is the first study to examine the 3D lower extremity joint kinematics and kinetics in obese children after short term weight loss, accompanying with a control group. It is concluded that weight loss with reduced body circumferences causes mass-driven changes in joint kinematics and kinetics. Increased self-selected walking speed is not a necessary outcome of a short-term weight loss. It seems that obese individuals prefer to walk at a speed that not only has minimal energy cost and maximal mechanical efficiency, but also can avoid an increase in joint load. This study also confirmed previous findings in the influence of excess mass on the absolute amount of force applied to the weight-bearing joints during walking.

Chapter 5: The Effect of Muscle Strengthening on Walking Biomechanics and Energetics

5.1 INTRODUCTION

Muscle strength refers to the ability of a muscle group to produce force against a resistance (Taaffe & Marcus, 2000). Insufficient muscular strength and power can impair motor function, limiting an individual's ability to perform daily tasks successfully (Visser et al., 2000). Obese children have greater difficulty performing locomotor tasks than normal weight children, because of the larger mass against gravity they are required to move during the movement (Shultz et al., 2011). Studies have consistently reported similarity between obese and normal weight children in flexibility and absolute muscle strength tasks (Deforche et al., 2003). In walking tasks, muscle provides forward progression and supports body weight (Liu et al., 2006). Due to the excess body weight, obese children need larger joint moments and powers to control the trunk and maintain the stability of lower limb (Gushue et al., 2006; Shultz et al., 2010). It is reasonable to hypothesize that obese children have a potential muscle weakness during physical activity. However, no direct evidence has shown the relationship between the muscle weakness and the gait strategy in obese children.

Chapter 3 found that obese children have significantly smaller relative muscle strength (in Newton per kg body mass) than normal weight children due to the larger body weight and similar absolute muscle strength (in Newton). Although the results suggested body mass played a dominant role in gait differences between obese and normal weight children, some researchers have pointed out that relative muscle weakness in relation to the excess body weight might be a cause of these biomechanical differences associated with obesity (McMillan et al., 2010). In addition, the reduced relative strength may increase the metabolic cost to support body weight, because more fast glycolytic fibres would be recruited and these fibres are less economical than slow oxidative fibres (Peyrot et al., 2010). Chapter 4 concluded that weight loss was an effective way to reverse the impact of obesity on

gait biomechanics and energetics, but the relative muscle strength of the intervention group was increased associated with reduced body mass. Thus, based on previous findings, the possibility that muscle strength may influence the gait strategy used by obese children cannot be excluded.

The purpose of this study was to determine whether muscle strengthening could reverse the impact of excess body weight on natural gait biomechanics and energetics in obese children. This study quantified gait kinematics, kinetics, mechanical and metabolic energy cost during walking before and after an 8-week lower extremity strength training in obese children.

5.2 METHODS

5.2.1 Participants

All participants were recruited from two primary schools in Shanghai and selected from a larger pool of 353 pupils in order to match the inclusion criteria. The inclusion criteria were: aged between 10 and 12 years; no orthopaedic or neurological disorders that could interfere with the gait pattern; and a BMI level above age and gender specific cut-off points for obesity as defined by Cole et al. (2000). Prior to participation, each pupil completed a measurement of body height and weight, and was interviewed by a medical doctor to assess the eligibility. Permission for these tests and interviews on potential participants was obtained from the school principals. A total of 37 participants enrolled in the baseline tests. All participants and their guardians read and signed an informed consent form approved by the Human Research Ethics Committee of the University of Auckland. After the baseline measurements, the participants were randomized to either the intervention group or the control group using envelope-based randomization by a person independent of the investigators. The randomization process was stratified by gender and age to ensure equal numbers in the two groups. A flowchart of recruitment and follow-up of participants is shown in Figure 5.1.

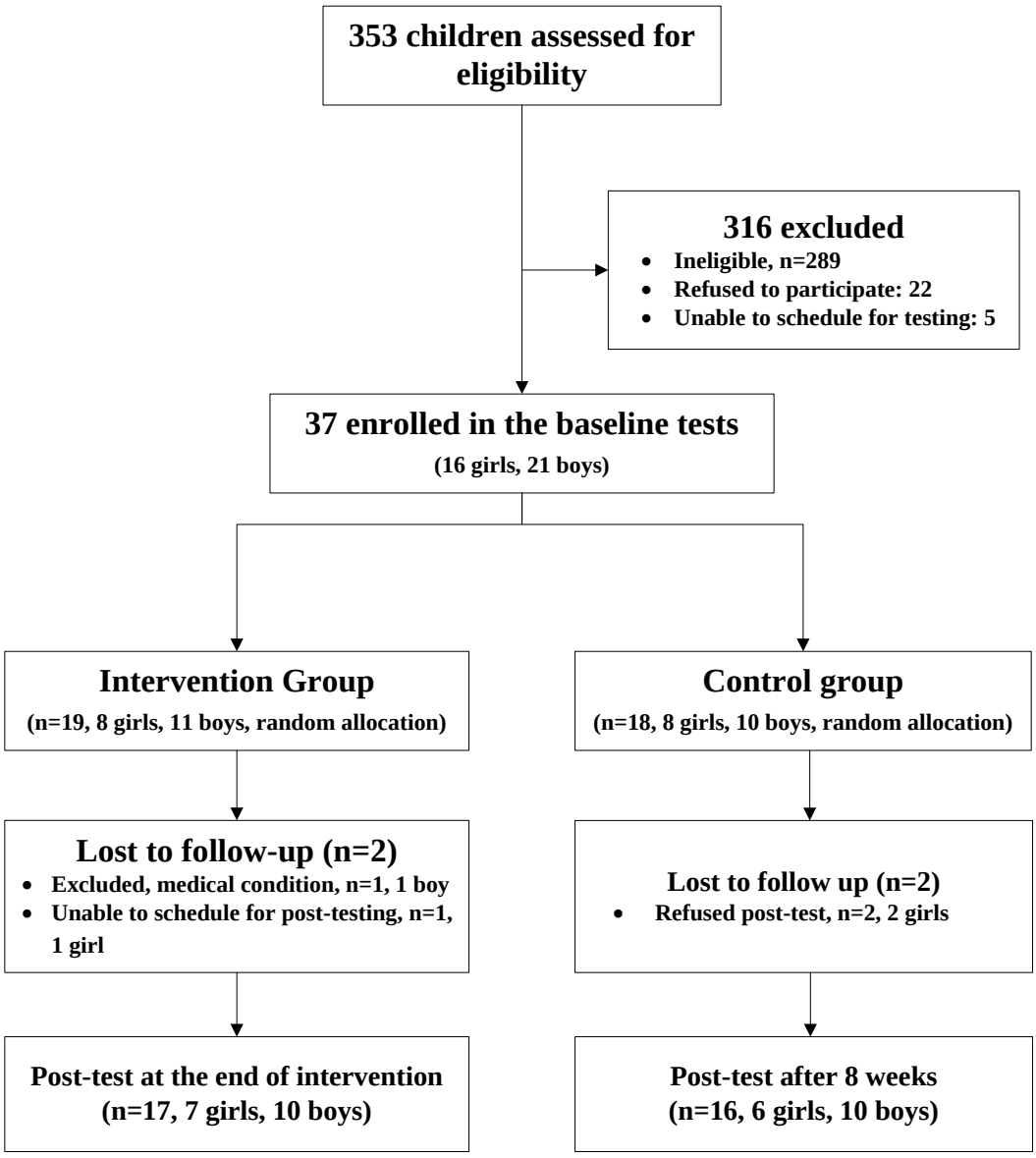


Figure 5.1 Flowchart of participants through the study.

5.2.2 Experimental Procedures

All participants were tested twice with the same measurements by the same investigators. The baseline test was done one day before the intervention program. The second test started one day after the intervention. Body weight was measured to the nearest 0.1 kg with an electronic weight scale, and height was measured with a wall-mounted stadiometer to the nearest 0.5 cm. Effectiveness of the strength training program was determined by measuring pre-post differences in isokinetic strength in flexion and extension of the knee and ankle joints using the angular velocity of 60 degree/s. The isokinetic muscle strength, gait kinematics, kinetics and metabolic cost were measured using the same protocol described in Chapter 3.

5.2.3 Strength training

This intervention program conditions the body through dynamic, resistance exercises. The elastic bands were used to create resistance. The exercise sessions consisted of 45 minutes on non-consecutive days (3 sessions per week) for at least 8 weeks. Each session began with a period of warm-up and stretching, followed by one set of several upper and lower body exercises that focus on the major muscle groups, 3 to 5 sets of exercises for targeted muscle groups. Each set includes 6 to 15 repetitions (Reps). The type of training typically includes internal/external rotation, abduction/abduction and flexion/extension movements of the hip, knee, ankle joints. The detailed exercises and target muscle groups were presented in Table 5.1.

The children's OMNI-Resistance Exercise Scale of Perceived Exertion (OMNI-RES) was used to determine the training intensity and progression for the intervention group (Robertson et al., 2005). OMNI-RES is a ratings of perceived exertion (RPE) scale specifically developed for use during upper and lower resistance exercise in children, and is a valid instrument for monitoring exertion in children (Robertson et al., 2005). The scale represents a range of feelings from "extremely easy", rated as 0, to "extremely hard", rated as 10. The scaling instructions were clearly explained to all participants before intervention. Participants were reminded that their feeling from the isokinetic tests could be considered as "extremely hard", and were asked to give a number after each set of exercise. All participants were encouraged to achieve a number of 8-10 for all exercises completed, which should be considered "hard" to "extremely hard" (see Appendix E).

Table 5.1 Detailed exercises and target muscle groups

Muscle Group	Elastic Band Exercise
Hip flexor: *iliopsoas*	Seated hip flexor: Sit on a chair with the band wrapped around one thigh, secure the two ends with the other foot and lift the wrapped thigh off the ground
Hip extensor: *gluteus maximus, hamstrings*	Hip extension: Stand on one leg with the band wrapped around the ankle of the free foot, take the free foot behind the body
Hip abductor: *gluteus medius, gluteus minimus*	Supine Hip Abduction: Wrap the band round ankles, move ankles apart against the resistance
Hip adductor: *adductors longus and brevis, adductor magnus*	Leg adduction: Attach one end of the band to a stationary object and the wrap the other end around the ankle closest to it
Knee flexor: *hamstrings, gastrocnemius*	Prone knee flexion: Lie on stomach with the band wrapped around the ankle, attach the other end to a fixed point.
Knee extensor: *vasti*	Seated knee extension: Sit on a chair with the band wrapped around ankles, extend one knee against the resistance
Ankle dorsiflexor: *tibialis anterior, ext digitorum and halluces*	Seated ankle dorsiflexion: Sit on the floor with one end of the band wrapped around one foot , attach the other end to a fixed point, pull the toes toward the body
Ankle plantar flexor: *gastrocnemius, tibialis posterior*	Seated ankle plantar flexion: Sit on the floor and wrap the elastic band around one foot, hold the ends in hands, point toes away from the body
Ankle inverter: *tibialis anterior, tibialis posterior*	Seated ankle inversion: Sit on the floor with one end of the band wrapped around one foot, attach the other end to a fixed point, invert foot to against the resistance
Ankle everter: *peronei longus, brevis and tertius*	Seated ankle eversion: Sit on the floor with one end of the band wrapped around one foot, attach the other end to a fixed point, evert foot to against the resistance

5.2.4 Statistical Analysis

Means and standard deviations of measured parameters were calculated for each group. A two way ANOVA with repeated measures was conducted to determine the effect of the intervention on measures of biomechanical and energetic parameters. Group (intervention vs. control) was used as between-subjects factor and time of measurement (baseline vs. 8 weeks) as a within-subjects factor. Alpha level was adjusted to <0.025 to control for a Type I error using the Bonferroni method. All data were analysed using SPSS (SPSS, version 18.0, SPSS Inc, Chicago, IL).

5.3 RESULTS

Table 5.2 shows that body height and mass slightly increased in both groups, there was no significant interaction in body height, weight and BMI. The group effect was significant in body height and weight.

The results of isokinetic muscle strength are presented in Table 5.3. The results of two-way ANOVAs found significant interactions in both absolute and relative isokinetic muscle strength. The absolute peak torque of knee flexor and extensor increased 16.8% and 11.8%, respectively. The relative peak torque of knee flexor and extensor increased 11.3% and 8.0%. The absolute peak torque of ankle plantarflexor and dorsiflexor increased 18.5% and 13.5%, respectively. The relative peak torque of ankle plantarflexor and dorsiflexor increased 17.9% and 13.3%, respectively.

Table 5.2 Anthropometry parameters *(means ± SD).*

	Intervention group		Control group		Two-way ANOVAs		
Variable	Before	After	Before	After	p_t	p_g	p_{t*g}
Height (cm)	152.5 ± 4.6	152.7 ± 4.9	151.8 ± 8.0	153.6 ± 7.5	.853	**.002**	.066
Weight (kg)	61.7 ± 4.9	62.2 ± 5.0	63.9 ± 12.5	64.3 ± 12.4	.521	**.023**	.776
BMI (kg/m^2)	26.5 ± 1.5	26.6 ± 1.3	27.5 ± 3.1	27.3 ± 3.0	.311	.692	.140

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 5.3 Absolute and relative peak isokinetic strength (*means* ± *SD*).

Index	Intervention group		Control group		Two-way ANOVAs		
	Before	After	Before	After	p_t	p_g	p_{t*g}
Absolute value (Nm)							
Knee flexor	49.00 ± 11.15	54.78 ± 10.91	49.56 ± 21.41	49.51 ± 19.20	.020	.673	**.018**
Knee extensor	77.61 ± 15.29	84.31 ± 15.34	78.08 ± 21.80	76.93 ± 15.29	.044	.586	**.006**
Ankle dorsiflexor	18.63 ± 3.65	21.14 ± 3.90	19.81 ± 4.23	19.78 ± 4.82	**.001**	.948	**< .001**
Ankle plantarflexor	40.70 ± 12.27	48.21 ± 14.50	41.23 ± 12.87	38.87 ± 11.7	**.014**	0.300	**< .001**
Relative value to body mass (Nm/kg)							
Knee flexor	.79 ± .17	.88 ± .17	.77 ± .30	.77 ± .25	.050	.372	**.025**
Knee extensor	1.25 ± .21	1.35 ± .21	1.24 ± .34	1.21 ± .26	.171	.360	**.007**
Ankle dorsiflexor	.30 ± .07	.34 ± .07	.32 ± .08	.31 ± .06	**.005**	.673	**< .001**
Ankle plantarflexor	.67 ± .22	.79 ± .26	.65± .18	.61 ± .17	.020	.176	**< .001**

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 5.4 Spatiotemporal gait parameters (*means* ± *SD*).

	Intervention group		Control group		Two-way ANOVAs		
Variable	Before	After	Before	After	p_t	p_g	p_{t*g}
Speed (m/s)	1.22 ± .09	1.28 ± .08	1.19 ± .09	1.17 ± .12	.199	.026	**.006**
Cadence (step/min)	121.3 ± 6.7	122.3 ± 6.5	119.5 ± 7.6	117.0 ± 7.5	.078	.131	.487
Step width (cm)	17.2 ± 3.0	16.4 ± 2.7	16.5 ± 2.5	17.2 ± 2.8	.935	.941	.043
Stride length (cm)	122.9 ± 8.6	123.7 ± 7.5	119.3 ± 6.4	119.6 ± 5.9	.654	.096	.812
Stride time (s)	1.00 ± .05	0.99 ± .06	1.01 ± .06	1.03 ± .07	.127	.092	.110
Double support (%)	16.1 ± 2.6	16.8 ± 2.9	19.0 ± 2.1	18.1 ± 2.1	.755	**.010**	.056
Single support (%)	41.8 ± 1.3	41.2 ± 3.1	40.6 ± 1.1	40.7 ± 1.7	.612	.097	.428
Stance Phase (%)	58.1 ± 1.3	58.5 ± 1.3	59.7 ± 1.1	59.2 ± 1.1	.997	.004	.030

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

The self-selected walking speed in the intervention group increased by 0.06m/s after intervention, and the interaction was significant (Table 5.4). Neither statistically significant interactions nor effect of intervention for the other spatiotemporal gait parameters were found. The effect of group was significant for the percentage of double support in a gait cycle ($p = .010$).

Table 5.5 Joint ROM (degree) during the stance phase (*means* ± *SD*).

Joint	ROM	Intervention group		Control group		Two-way ANOVAs		
		Before	After	Before	After	p_t	p_g	p_{t*g}
Hip	Flexion/ Extension	38.6 ± 5.4	41.1 ± 3.7	38.0 ± 3.7	37.7 ± 5.60	.166	.172	.089
	Abduction/ Adduction	14.6 ± 4.5	15.7 ± 4.4	13.9 ± 3.4	12.5 ± 3.3	.758	.134	.027
Knee	Flexion/ Extension	47.8 ± 4.6	46.4 ± 4.0	42.1 ± 5.7	40.4 ± 6.1	**.001**	.026	.845
	Varus/ Valgus	10.6 ± 4.6	9.3 ± 3.9	9.8 ± 4.0	9.2 ± 3.2	.261	.687	.701
Ankle	Dorsiflexion/ Plantarflexion	27.4 ± 4.1	26.9 ± 3.7	26.3 ± 3.5	25.9 ± 2.6	.496	.342	.931
	Inversion/ Eversion	5.6 ± 2.1	5.2 ± 2.2	4.0 ± 1.5	4.1 ± 1.9	.638	.030	.467

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Table 5.6 Joint ROM (degree) during the swing phase (*means* ± *SD*).

Joint	ROM	Intervention group		Control group		Two-way ANOVAs		
		Before	After	Before	After	p_t	p_g	p_{t*g}
Hip	Flexion/ Extension	26.2 ± 3.9	27.2 ± 3.3	30.9 ± 3.4	31.4 ± 4.9	.204	**.001**	.666
	Abduction/ Adduction	8.3 ± 2.5	9.5 ± 3.1	9.2 ± 2.6	8.3 ± 2.5	.626	.870	**.018**
Knee	Flexion/ Extension	61.5 ± 5.0	61.5 ± 4.3	61.7 ± 6.1	61.9 ± 7.4	.913	.902	.838
	Varus/ Valgus	14.8 ± 3.5	14.4 ± 2.9	15.2 ± 4.3	14.1 ± 2.5	.319	.991	.649
Ankle	Dorsiflexion/ Plantarflexion	19.8 ± 3.8	20.3 ± 4.1	19.4 ± 4.1	18.9 ± 4.9	.137	.780	.453
	Inversion/ Eversion	3.7 ± 1.9	3.8 ± 2.0	3.1 ± 1.4	3.2 ± 2.2	.950	.495	.461

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Summaries of joint ROM during the stance phase and swing phase were given in Table 5.5 and Table 5.6, respectively. There was a significant time-by-group interaction for hip abduction/adduction ROM during the swing phase, which increased 14.5% in the intervention group. The time effect was significant for knee flexion/extension ROM during the stance phase, but there were no interactions. The effect of group was significant for hip flexion/extension ROM during swing phase.

Table 5.7 The peak joint moment (Nm/kg) during the stance phase (*means* ± *SD*).

		Intervention group		Control group		Two-way ANOVAs		
Joint	Moment	Before	After	Before	After	p_t	p_g	p_{t*g}
Hip	Flexor	.94 ± .19	1.19 ± .15	1.15 ± .18	1.09 ± .16	**.003**	.266	**.000**
	Extensor	.39 ± .07	.37 ± .09	.41 ± .15	.42 ± .15	.377	.400	.274
	Abductor	.92 ± .15	.91 ± .10	.89 ± .13	.92 ± .11	.658	.814	.233
	Adductor	.09 ± .04	.08 ± .04	.11 ± .06	.08 ± .08	.169	.543	.362
Knee	Flexor	.13 ± .04	.12 ± .05	.16 ± .09	.15 ± .08	.410	.061	.649
	Extensor	.80 ± .20	.83 ± .22	.77 ± .24	.85 ± .23	.230	.996	.631
	Abductor	.28 ± .09	.30 ± .09	.34 ± .10	.35 ± .10	.317	.069	.818
	Adductor	.07 ± .04	.07 ± .03	.06 ± .03	.07 ± .05	.900	.622	.275
Ankle	Dorsiflexor	.09 ± .08	.10 ± .09	.10 ± .06	.11 ± .05	.218	.662	.516
	Plantarflexor	1.19 ± .09	1.16 ± .14	1.13 ± .10	1.15 ± .10	.650	.349	.183
	Everter	.06 ± .04	.04 ± .03	.05 ± .04	.05 ± .05	.209	.740	.076
	Inverter	.15 ± .04	.14 ± .04	.14 ± .04	.12 ± .05	.117	.235	.813

Note: Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; p_{t*g}, time by group interaction.

Normalized peak joint moment (Nm/kg) at baseline and 8 weeks are presented in Table 5.7. Time-by-group interactions were significant at the peak moment of hip flexor, showing a 26.6% increase in the intervention group. There were no significant changes in the knee and ankle joint moment for the intervention and the control group.

All energetic parameters and joint mechanical work increased in the intervention group, significant time-by-group interactions were found in in gross metabolic power, mechanical energy expenditure, net metabolic rate, the normalized MEE, and the hip mechanical work at H2 and H3 phase (Table 5.8). Figure 5.2 shows the joint powers over a gait cycle.

Table 5.8 The effects of muscle strengthening on walking energetics (*means* ± *SD*).

	Intervention group		Control group		Two-way ANOVAs		
Variable	Before	After	Before	After	p_t	p_g	p_{t*g}
Energetic parameters							
P_{gross} (W)	333.7 ± 41.5	357.1 ± 54.4	334.1 ± 64.1	328.8 ± 63.0	.145	.480	**.024**
P_{rest} (W)	114.0 ± 18.6	116.0 ± 19.5	109.7 ± 27.1	110.6 ± 22.3	.501	.519	.777
E_{net} (J)	216.0 ± 33.7	237.0 ± 45.4	225.4 ± 44.0	223.5 ± 49.3	.100	.885	.051
MEE (J)	60.0 ± 9.8	66.5 ± 11.5	63.5 ± 13.3	6.5 ± 14.6	.080	.958	**.019**
ME%	27.9 ± 2.5	28.3 ± 2.5	28.3 ± 2.8	28.1 ± 2.8	.085	.855	.501
C_{net} (J/kg/m)	2.9 ± .4	3.0 ± .4	3.0 ± .4	2.9 ± .5	.214	.941	**.025**
C_{mech} (J/kg/m)	.8 ± .1	.9 ± .1	.8 ± .1	.8 ± .1	.149	.984	**.004**
Joint mechanical work							
H2 (J/kg)	.19± .06	.24± .05	.21± .04	.20± .04	.001	.523	**.001**
H3 (J/kg)	.11± .02	.13± .04	.11± .02	.11± .03	.015	.606	**.011**
H2/H3	1.82± .60	2.01± .07	1.88± .30	1.86± .35	.105	.706	.103

Note: P_{gross}, gross metabolic power; P_{rest}, rest metabolic power; E_{net}, net metabolic cost; MEE, mechanical energy expenditure; ME, mechanical efficiency; C_{net} net metabolic rate; C_{mech}, normalzied MEE. Two-way ANOVAs: alpha level $p < .025$, p_t, time effect; p_g, group effect; $p_{t*g;}$ time by group interaction.

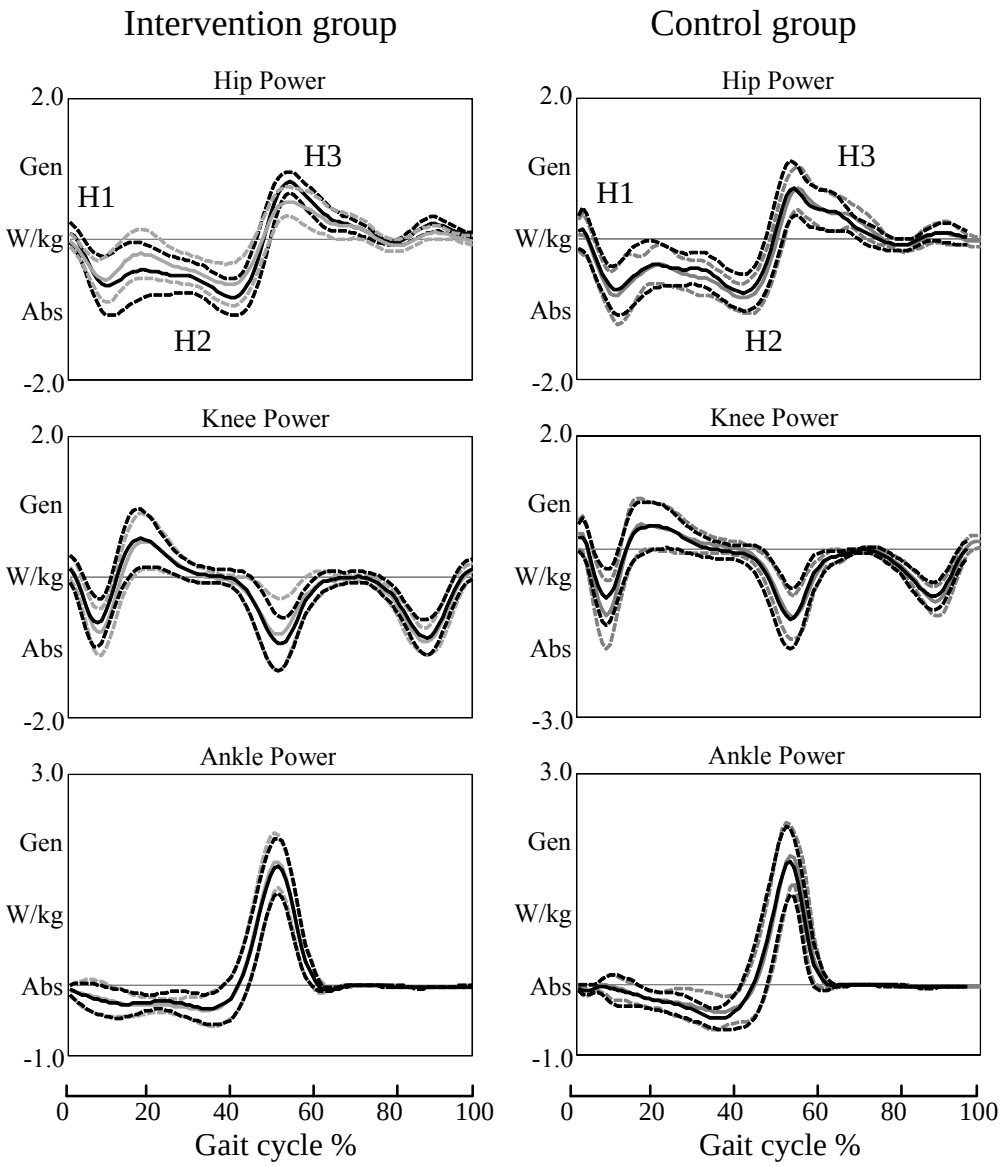


Figure 5.2 Joint powers over 100% gait cycle before (grey) and after 8 weeks (black). Solid lines = mean, dotted lines = SD.

5.4 DISCUSSION

Muscles of lower extremities play an extremely important role in maintaining joint mobility, stability and function (Roos et al., 2011). Low muscle strength combined with obesity has shown to be associated with more physical limitations in adults (Nantel et al., 2011). In addition, research interest has increased in determining if muscle weakness and obesity are risk factors for the development and progression of musculoskeletal problems (i.e. knee osteoarthritis) (Roos et al., 2011). Muscle strength is modifiable, and substantial improvement can be easily achieved in a relatively short period of time (Kraemer et al., 2002). Thus, muscle strength may be a therapeutic target in patients with musculoskeletal disorders. In addition, it has been well documented that excess body weight leads to gait modification in obese children, which may compensate for potential muscle weakness (Hills & Parker, 1991a; McGraw et al., 2000; McMillan et al., 2010; Shultz et al., 2009). Most of these changes have also been associated with an increased joint load and development of joint problems (McMillan et al., 2010; Shultz et al., 2009). To better understand whether muscle weakness could affect gait and a further increased risk of musculoskeletal problems with obesity, it is first necessary to examine whether relative weakness is present. Surprisingly no study has determined the relationship between muscle strength and walking biomechanics in obese children.

Chapter 4 has found that a reduction in body weight could directly reduce the differences in gait biomechanics and energetics between obese and normal weight children. However, the results have failed to exclude the possibility of changing muscle strength in these reverse effects. This study was carried out to find out how the gait biomechanical and energetic changes were associated with increases in muscle strength in response to resistance training.

The results indicated that there were significant increases in absolute (average: 15.2%) and relative (average: 12.6%) muscle strength of the lower extremities after 8 weeks of resistance training and that this increase was not observed in the control group. In addition, there was no significant difference in body mass after intervention, which emphasized the muscle strength-driven effects on gait. The results are in agreement with reported strength gains in obese and normal weight children or adolescents who trained using various resistance exercise protocols. Benson et al. (2008) reported improvements in lower body strength of 39.3% measured with one-

repetition maximum for leg press after 8 week progressive resistance training in overweight adolescents. Lubans et al. (2010) reported a 25.5% strength gain in the elastic tubing resistance training group after 12 week intervention. These increases are higher than the increase in this study. However, there is difficulty in comparing studies that vary in participants, intervention duration, exercise intensity, and types of training, and this study is specifically designed to improve muscular strength without gains or loss in body weight.

Walking speed is a simple indicator of gait abnormalities associated with musculoskeletal problems (Levangie & Norkin, 2011). Previous studies have shown that obese children walked with shorter step length and lower velocity (Hills & Parker, 1991a; McGraw et al., 2000). A slower walking speed suggested that obese children alter their gait strategy to compensate for increased instability (Nantel et al., 2006; Shultz et al., 2011). It has also been shown that strength of the flexor and extensor muscles is highly correlated with the capacity to execute mobility tasks in the obese individuals (Capodaglio et al., 2009). In this study, the self-selected walking speed in the intervention group significantly increased after muscle strengthening. The stride length also increased slightly but was not statistically significant. Thus, muscle strengthening reduced the gaps between obese children and normal weight children in terms of spatiotemporal gait parameters. Gains in lower extremity muscle strengthen may also improve gait stability. This finding is supported by Jadelis et al, who reported stronger knee extensor and flexor strength was associated with better balance (Jadelis, Miller, Ettinger & Messier, 2001). However, balance during walking was not directly measured in this study.

Concentric activity in the hip flexors is one of the primary sources for generating propulsion into the swing phase during walking (Olney, MacPhail, Hedden, & Boyce, 1990). While the walking speed increased, greater hip flexor moment and power generation during late stance and pre-swing phase suggested the resistance training improved participants' ability to promote locomotion through greater propulsion at the pre-swing phase. Compared to normal weight children, obese children showed a smaller normalized ankle plantarflexor moment during normal walking (Chapter 3). Ankle plantarflexors provide support and propulsion during walking (Kepple et al., 1997). Due to a faster walking speed after intervention, it is expected that the intervention group has a larger ankle plantarflexor moment

during the late stance and early swing phase. However, no difference was found. This finding confirmed that obese individuals prefer to pull their limb into swing by generating more hip flexion forces during walking, rather than use ankle plantarflexor muscles to propel the body forward. McMillan et al (2010) proposed that weak ankle plantarflexion muscles may result in an increased requirement for "push-off" at the hip. This possibility is not supported by my results. If there exists potential muscle weakness, the ankle plantarflexor moment should have increased with the increased muscle strength. Previous chapters have demonstrated that obese children had a greater hip flexor moment and a lower plantarflexor moment, and weight loss could significantly narrow these joint kinetic gaps between the obese and normal weight children. In fact, humans can reorganize their neuromuscular function to increase ankle muscle function, plantarflexor torque, and ankle power with increased body weight during walking (DeVita et al., 2003).

The metabolic cost of walking is determined primarily by the cost of generating muscle force thought the gait cycle (Umberger & Rubenson, 2011). Without changes in body weight, faster walking speed require more muscle forces to promote locomotion. This explained a 9.7% increase in net metabolic cost and a 10.8% increase in MEE. The metabolic cost and MEE increased in proportion, so that the mechanical efficiency was unaffected by strength training.

Although the primary focus of this study was the role of muscle strength on gait alterations in obese children, another interesting finding was that while the net metabolic rate and mechanical work per distance significantly increased after strength training, the increased value was similar (0.1 J/kg/m). Humans prefer to walk at a certain speed with a minimum energy cost per distance; results from previous chapters and other researchers found that metabolic rates are similar between obese and normal weight children during self-selected walking speed (Maffeis et al., 1993). It was expected that these values would not significantly change after the intervention in this study, because all participants were still walking at their self-selected speed. However, the results showed greater mechanical work at the hip (H2 and H3) throughout the gait cycle, which obviously resulted in an increase in energy cost.. The value of H2/H3 represents the ratio of energy transferred from the hip absorption (H2) to the hip generation (H3) phases. There was a 10.4% increase in H2/H3 value. Although the increase was not statistically

significant, it may suggest a reducing trend for the efficiency of energy transfer at the hips. This finding reinforced the suggestions that obese children were less efficient in transferring mechanical energy within the hip flexor muscles from the stance phase to the swing phase than normal weight children (Nantel et al., 2006). Muscle strengthening allowed obese children to adapt a faster walking speed, but it is unable to improve the efficiency of energy transfer at the hips. By contrast, Chapter 4 found that weight loss significantly reduced the H2/H3 value, but the self-selected walking speed did not increase. Taken these findings together, it suggested that the excess body weight may be the main factor causing the energy waste within the hips in obese children. Furthermore, this may also explain why the ankle kinetics did not change associate with increased walking speed. It could be a compensation for the efficiency loss at hip to maintain the mechanical efficiency of the locomotor system as a whole. Consistent with previous studies, this study confirmed that obese children expend more energy than lean children when walking at faster speeds, likely to be independent of body strength (Lazzer et al., 2003; Maffeis et al., 1993).

Body composition was not measured in this study. Thus, normalization per body mass instead of normalization per fat-free mass was used to represent in muscle strength and energetic parameters. Reporting relative weakness by dividing strength by body mass may result in classification of heavier persons as weak (Segal, Zimmerman, Brubaker, & Torner, 2011). Due to a larger body mass in obese individuals, the relative muscle strength will necessarily decrease due to an increased denominator. In this study, although the body mass did not significantly change after intervention, the fat-free mass may increase due to the training effect. Compared with normalizing strength by fat-free mass, the relative muscle strength may be overestimated. In this case, body composition data are helpful to understand a more accurate effect of resistance training on potential muscle weakness, and the relationship between muscle strength and obesity. However, some researchers believed that normalization per body mass may provide a realistic picture of the functional capacity of obese individuals with regard to their excess body mass (Capodaglio et al., 2009). Jaric (2002) also recommended body mass should be used in the normalization of isokinetic muscle torque. Similarly, mass normalization for energetics data in children was also questioned (Shultz et al., 2011). Some researchers proposed allometric scaling methods or non-dimensional normalization

methods to normalize metabolic data in children (Peyrot et al., 2009; Schwartz, Koop, Bourke, & Baker, 2006; Zakeri, Puyau, Adolph, Vohra, & Butte, 2006). It is recognized that normalization for body mass may cause potential errors while comparing the muscle strength and energetic data. However, this study did not have statistical power and sample size to examine which is the most appropriate method for obese children. Body composition should be included in future studies to investigate the relationship among body fat, muscle mass and walking biomechanics.

5.5 CONCLUSION

The obese individuals prefer to pull their limb into swing by generating more hip flexion forces during walking, rather than use ankle plantarflexion muscles to propel the body forward. Muscle strengthening allowed obese children to adapt a faster walking speed and consume more energy, but it is unable to improve the efficiency of energy transfer at the hips.

Chapter 6: Compressive tibiofemoral force and muscle functions in obese children: the application of musculoskeletal modelling and simulation

6.1 INTRODUCTION

The prevalence of obesity among children has increased dramatically in the past few decades and excess body weight during childhood was found to be indicative of skeletal problems in later life (Ding et al., 2005; Karnik & Kanekar, 2012; Messier, 1994; Wearing et al., 2006a). Activities of daily living such as walking and stair climbing impose relatively large loads and movements on weight bearing joints in obese children (Nantel et al., 2006; Strutzenberger et al., 2011). Abnormal loads can have adverse effects on joint health, resulting in more discomfort or pain of the musculoskeletal system (Dietz, Gross, Kirkpatrick, Dietz Jr., & Kirkpatrick Jr., 1982; Shultz, Anner, & Hills, 2009). Previous studies have also shown that excessive compressive forces may damage articular cartilage and lead to joint osteoarthritic changes (Clements, Bee, Crossingham, Adams, & Sharif, 2001; McCormack & Mansour, 1998). Since obesity is a known risk factor for musculoskeletal pain and disorders (Wearing, Hennig, Byrne, Steele, & Hills, 2006c), determining the differences in knee joint loads between obese and normal weight children may contribute to clarifying the pathophysiologic role of obesity in the development and progression of knee problems (e.g. knee osteoarthritis). Knowledge of individual muscle activity during movement could improve the diagnosis of the obese individual with potential gait abnormalities in terms of joint loading and muscle function.

Chapter 3 found there was an increase in the absolute amount of force applied to the joint and the muscular force needed to move the excess body weight during walking. Chapter 4 further suggested that the joint loads could be an important contributor to the gait strategy selected by obese individuals. However, these studies have failed to quantify the joint reaction forces and muscle forces. Traditional gait

analysis using inverse dynamics is limited by its ability to create an integrated understanding of muscle activities and joint movements (Pandy & Andriacchi, 2010).The results represent the force of all muscles crossing a joint, but the musculoskeletal system is mechanically redundant. Information about the co-contraction of muscles and the biarticular muscle activities is not available. Electromyography (EMG) data provide important information to support the inverse dynamic analysis for the estimation of joint moments. However, an EMG signal just represents the summed effect of the activity of a group of muscles; there are still no estimates of individual muscle forces (Zajac et al., 2003). Since it is far more difficult to invasively obtain tissue stresses and muscle forces *in vivo*, computational modelling and simulation are recognized as a vital complementary tool to estimating multiple variables of interest under dynamic conditions (Pandy & Andriacchi, 2010).

In recent decades, a large number of simulation studies have been developed to investigate the causal relation between muscle force and joint movement during walking (Erdemir et al., 2007). The information was integrated with joint kinematics to determine the corresponding forces and stresses acting on the bones. Liu et al. (2008) reported muscle contributions to support and accelerate body COM over different walking speeds in eight children. Steele et al. (2012) examined how muscle forces and compressive tibiofemoral force change with the increasing knee flexion associated with crouch gait in cerebral palsy children. Unfortunately, no study has investigated the effects of obesity on joint reaction force and individual muscle activity using musculoskeletal simulation.

Therefore, the purpose of this study was to utilize musculoskeletal modelling and forward simulation to investigate the gait strategy of obese children at the musculoskeletal level. The tibiofemoral force was simulated to investigate the relationship between obesity and knee joint loading. By analysing individual muscle function, this study compared the mechanisms that how individual muscles contribute support and progression accelerations of COMbetween obese and normal weight children during normal walking.

6.2 METHODS

6.2.1 Experimental data

The three-dimensional kinematic and kinetic data were collected from eight obese (the OB group) and eight normal weight (the NW group) boys aged 8-12 years old, walking at their self-selected speed over ground. All participants were recruited by advertisements placed in the local communities. BMI was used to classify all participants according to the age and gender specific cutoff points for obesity and normal as defined by Cole et al. (2000). The demographics of all participants are represented in Table 6.1. Protocols for measuring kinematics and ground reaction force have been described in Chapter 3. All children and their guardians read and signed an informed consent form approved by Human Research Ethics Committee of the University of Auckland.

Table 6.1 Demographics of participants (*means* ± *SD*).

Variable	OB	NW	*t*	*d*	*p*
Weight (kg)	75.1 ± 11.0	43.1 ± 10.5	5.95	2.041	**< .001**
Height (cm)	155.4 ± 4.9	150.6 ± 6.0	1.75	.601	.103
BMI (kg/m^2)	31.0 ± 3.4	18.8 ± 3.8	6.77	2.321	**< .001**
Self-selected speed (m/s)	1.10 ± .08	1.22 ± .08	3.00	1.029	**.014**

6.2.2 Musculoskeletal model

This study used a 3D generic musculoskeletal model built in OpenSim (http://simtk.org) v3.0 software (Delp et al., 2007), with 23 degrees of freedom (DOF) and 92 Hill-type muscle-tendon actuators (Delp et al., 1990). A ball-and-socket joint was used to represent the hip and pelvis to trunk joints (3 DOF). The knee joint (1 DOF) was modelled as a planar joint in the flexion/extension axis (Yamaguchi & Zajac, 1989). Each ankle was modelled as a revolute joint (1 DOF). This musculoskeletal model has been previously used for studies involving healthy children and children with cerebral palsy (Liu et al., 2008; Steele et al., 2012).

6.2.3 Simulation

The generic model was scaled to each participant according to the position of anatomical reference points. Inverse kinematics were applied to calculate the joint kinematics (joint angles and translations) over a gait cycle. According to the kinematics and measured ground reaction force (GRF), the equation of motion in this dynamic system was applied to calculate the forces and moments at each joint. A static optimization algorithm decomposed the net joint moments into individual muscle forces by solving an optimization problem that minimized the sum of the squares of the muscle activations. Then, the residual reduction algorithm was used to make the data of the joint kinematics more dynamically consistent with the experimental ground reaction force data. The next step involved using Computed Muscle Control, which found the muscle excitations that drove the models to track the desired kinematics (Delp et al., 2007). Simulated joint kinematics was compared to the measured kinematics data to make sure the simulations were able to track the experimental data. In order to evaluate the accuracy and validity of the simulation results, the simulated COM accelerations were compared to the measured COM acceleration, the simulated muscle activations were compared to the EMG data from 85 normal children aged 10.5±3.5 years old as reported by Schwartz et al. (2008).

6.2.4 Compressive tibiofemoral force

The compressive tibiofemoral force represented the sum of contact forces between the tibial and femoral cartilage and all ligament forces crossing the tibiofemoral joint. In this study, the compressive tibiofemoral force was calculated by the joint reaction analysis algorithm in OpenSim software, which incorporates a post-processing procedure that uses the muscle forces and joint kinematics to calculate resultant joint loads. The Newton–Euler equation is:

$$\underline{R}_{knee} = M_T(\underline{q})\ddot{\underline{q}} - [\underline{F}_M + G_T(\underline{q}) + \underline{R}_{ankle}] \tag{6.1}$$

where $M_T(\underline{q})$ is the mass matrix of a tibia. $\underline{q}$ represents the vector of angular and linear displacement, and $\ddot{\underline{q}}$ represents the vectors of angular and linear acceleration of tibia. $\underline{F}_M$ are the muscle forces required to reproduce the knee joint moments of each participant throughout the gait cycle, which were obtained from static optimization results. $G_T(\underline{q})$ represents the gravitational loading. $\underline{R}_{ankle}$ represents the ankle reaction force. The calculation details have been previously described by Steele et al.

(2012). The muscle forces were obtained from static optimization algorithm in OpenSim. Three major muscle groups across the knee (quadriceps, hamstrings and gastrocnemius) were analysed to investigate the relationship between muscle forces and the compressive tibiofemoral force.

6.2.5 Muscle function analysis

An induced acceleration analysis (IAA) in Opensim was used to compute the contributions of individual muscles to vertical and fore-aft COM accelerations over a complete gait cycle. Based on the muscle excitation level from CMC results, the motion was simulated forward over a short time interval (0.01s) to calculate the resulting change in the model's COM (Liu et al., 2006). For each muscle, its induced acceleration was integrated over the gait cycle and defined as its contribution to COM (Liu et al., 2006). The individual muscle contribution $\underline{\ddot{q}}_m$ was formulated as follows:

$$\underline{\ddot{q}}_m = [M(\underline{q})]^{-1} R(\underline{q}) \underline{F}_m \tag{6.2}$$

where $M(\underline{q})$ is the mass matrix of COM. $R(\underline{q})$ is a matrix of muscle moment arms. $\underline{F}_m$ is a vector of muscle force. The muscle forces obtained from CMC was used in this equation to estimate the individual muscle contribution to COM, while the muscle forces obtained from static optimization were used to calculate joint loads.

To simplify data analysis, the forces and contributions of the actuators performing similar functions were summed. The quadriceps forces and contributions were the sum of the rectus femoris, the vastus medialis, the vastus intermedius, and the vastus lateralis. The hamstring muscle group included the semimembranosus, semitendinosus, biceps femoris long head, and biceps femoris short head. The gastrocnemius was the sum of medial and lateral gastrocnemius forces. Contributions from the gluteus maximus superior, middle, and inferior muscles, and gluteus medius anterior, middle, and posterior muscles were summed into one gluteus maximus and one gluteus medius contribution, respectively. Vasti contribution was computed by the sum of the vastus medialis, vastus intermedius and vastus lateralis. A dorsiflexor contribution comprised of contributions from tibialis anterior, extensor hallucis longus, extensor digitorum longus and peroneus tertius.

6.2.6 Data statistics

Means and standard deviations of spatiotemporal gait parameters, peak compressive tibiofemoral force and muscle forces were calculated for each group. An independent t-test was performed to compare the differences between the OB group and the NW group. A linear regression analysis was performed to identify the relationship between walking speed and peak compressive tibiofemoral force for each participant and the regression coefficient value (r^2) was calculated. $p < .05$ indicated statistical significance.

6.3 RESULTS AND DISCUSSION

The purpose of this study was to use musculoskeletal modelling and forward simulation to investigate the gait strategy of obese children at musculoskeletal level. Simulated joint kinematics were able to track the measured kinematics data. Use of subject-specific modelling allowed this study obtain the agreement between the experimental and simulated data.

6.3.1 Validity of the simulated results

The joint angles were averaged from the simulation over eight participants with three walking trials each for the OB and NW group respectively. Simulated joint kinematics were able to track the measured kinematics data, the vertical COM acceleration also matched well with experimental data, as shown in Figure 6.1. In addition, the joint angles were consistent with previous experimental based studies (McMillan, Pulver, Collier, & Williams, 2010; Shultz, Sitler, Tierney, Hillstrom, & Song, 2009). The simulated muscle activation data were qualitatively compared to the EMG data reported by Schwartz et al. (2008). Simulated rectus femoris, vastus medialis, semitendinosus, biceps femoris long head and medial gastrocnemius were highly consistent with the experimentally measured EMG data (Figure 6.2). Unlike what Schwartz et al. (2008) reported, the anterior tibialis in this study did not exhibit a burst of activation at push-off and the end of swing phase. However, there was still a slight activation during this time. Therefore, use of subject-specific modelling allowed this study to obtain the agreement between the experimental and simulated data.

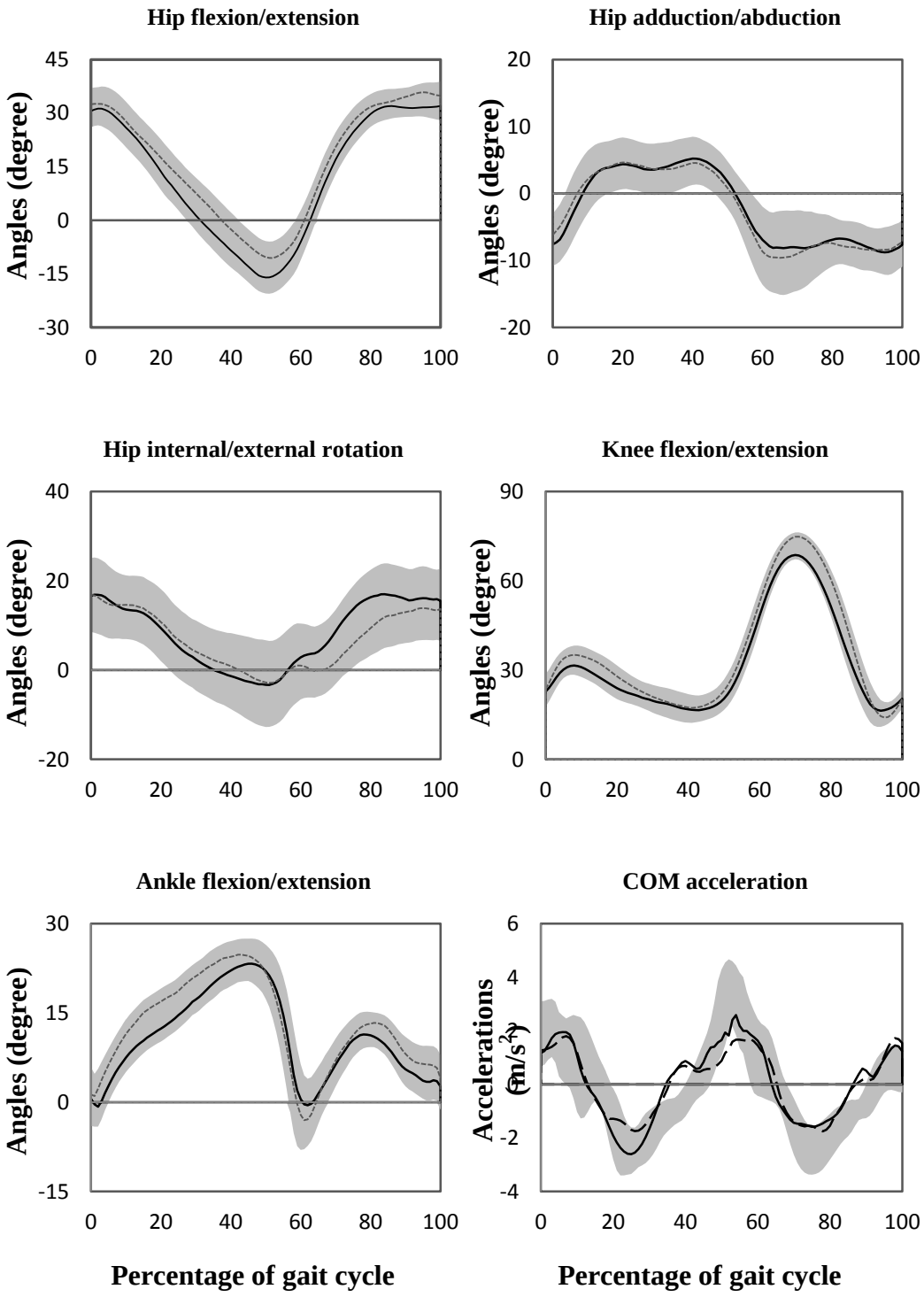


Figure 6.1 Kinematics tracking results comparison between simulated and measured results. The shaded areas represent the mean±one standard deviation of measured kinematics data. The solid lines represent the mean simulated kinematics for the OB group, while the dotted lines represent the mean simulated kinematics for the NW group.

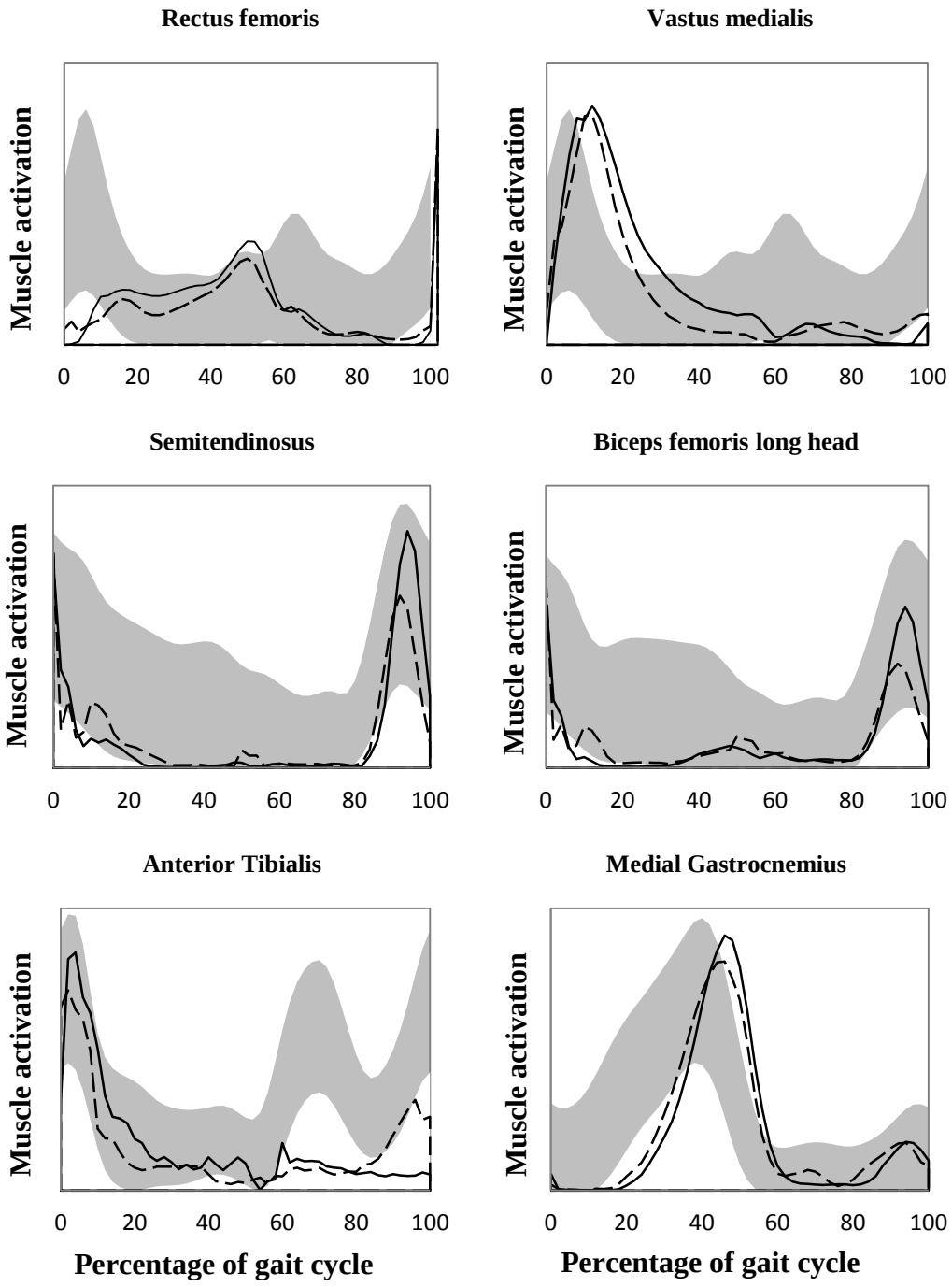


Figure 6.2 Qualitative comparisons of simulated muscle activity and experimentally measured EMG. The shaded areas represent the mean±one standard deviation of EMG data reported by Schwartz et al. (2008) The solid lines represent the mean simulated muscle activation level.

6.3.2 Compressive tibiofemoral force

The results showed that the absolute compressive tibiofemoral force was significantly higher in obese children over the gait cycle due to the excess body mass (Figure 6.3). The association between obesity and joint load during walking is intuitive. Even though the obese children walked at a significantly slower speed, the absolute compressive tibiofemoral force was still 25% higher than the normal weight children. However, after body weight normalization, the peak value of compressive tibiofemoral force was significantly higher in normal weight children than obese children (Table 6.2). There was a linear relationship between the average self-selected speed and the normalized peak compressive tibiofemoral force ($r^2 = .611$). The relationship is described by:

$$F_{\text{knee}} = 3.585 \times V_{walking} - 1.975 \quad (3)$$

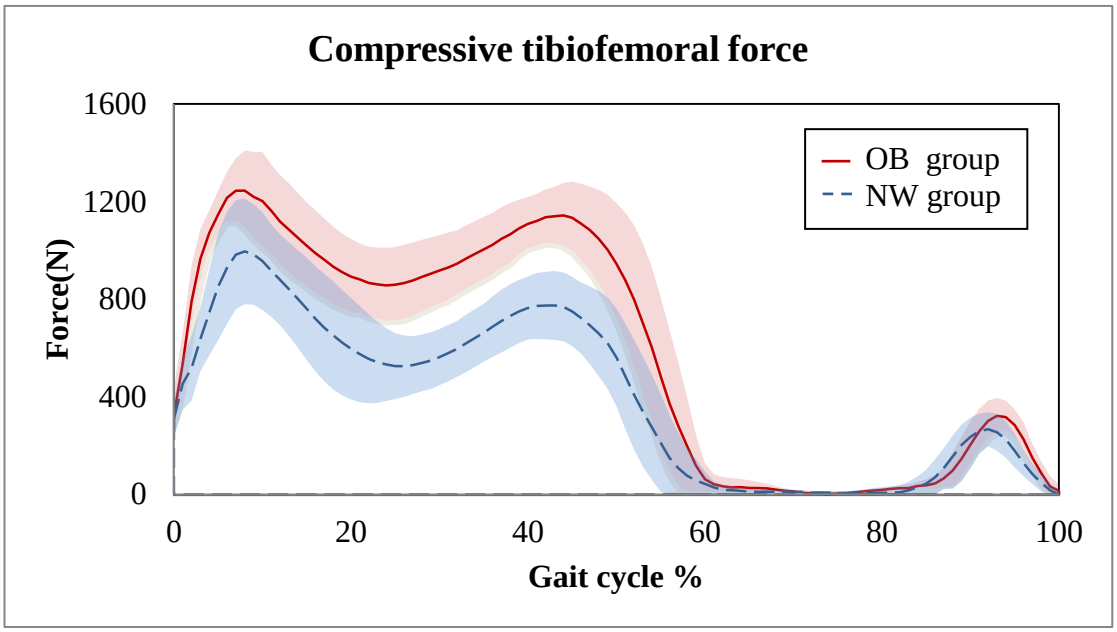
Where F_{knee} is the peak compressive tibiofemoral force normalized by body weight, and $V_{walking}$ is the most comfortable walking speed selected by participants (Figure 6.4).

Table 6.2 Body weight normalized joint loading and muscle forces

	OB	NW	*t*	*d*	*p*
CTF (×BW)	1.84 ± .24	2.49 ± .45	- 3.60	1.236	**.003**
Hamstring (×BW)	.47 ± .07	.60 ± .07	- 3.71	1.274	**.002**
Quadriceps (×BW)	.98 ± .26	1.53 ± .52	- 2.68	.918	**.019**
Gastrocnemius (×BW)	.38 ± .12	.57 ± .08	- 3.73	1.278	**.003**

Note: CTF, Compressive tibiofemoral force; BW, body weight.

A.



B.

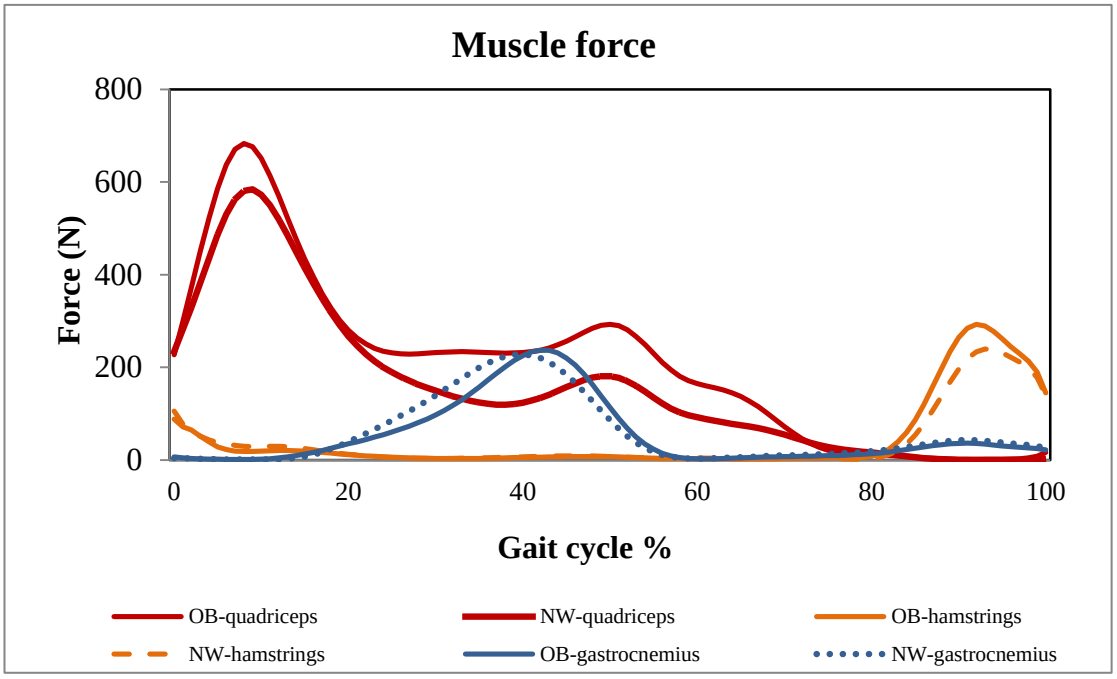


Figure 6.3 (A) represents the compressive tibiofemoral force over gait cycle during normal walking. The solid lines represent the mean compressive tibiofemoral force for the OB group, while the dotted lines represent the mean compressive tibiofemoral force for the knee in the NW group. (B) represents the forces of three major muscle groups acting at the knee.

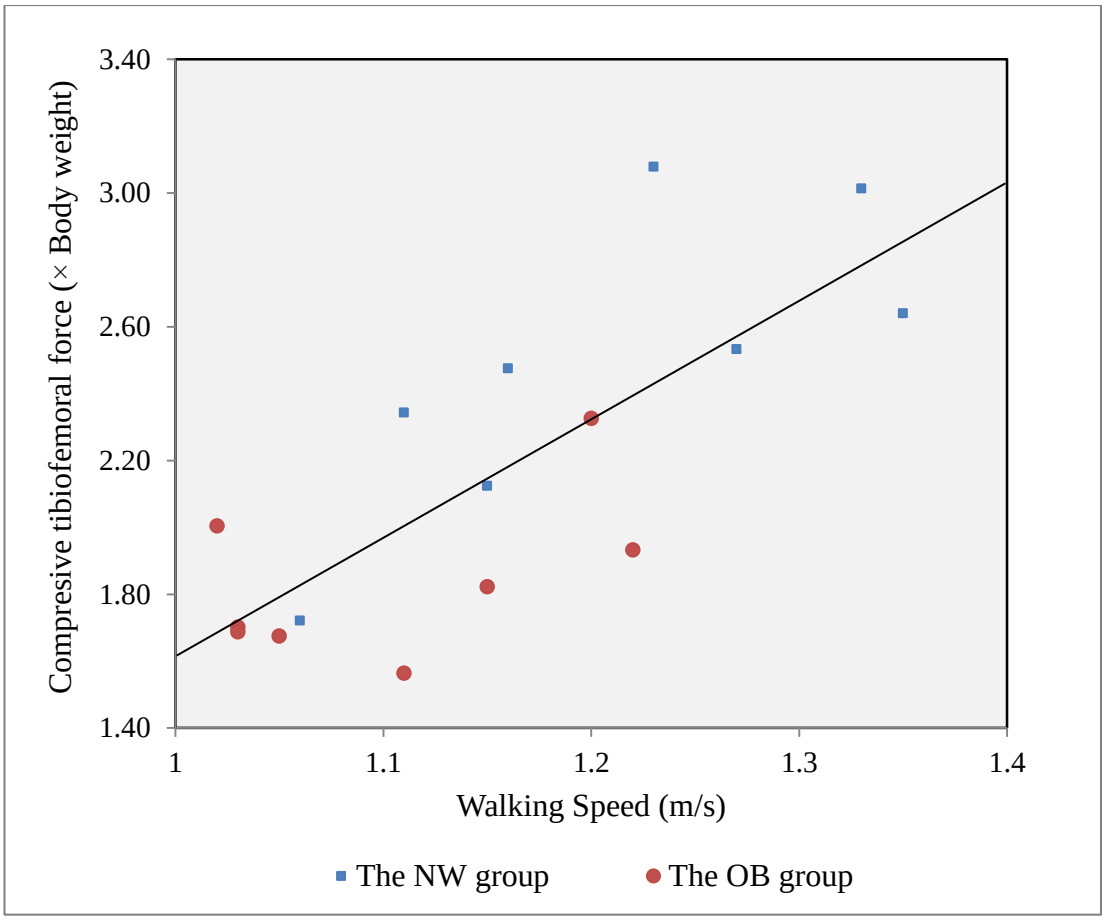


Figure 6.4 Regression of self-selected walking speed with peak compressive tibiofemoral force.

The peak value of compressive tibiofemoral force appeared at the contralateral toe-off following the first heel strike with a magnitude of approximately 2.5 times body weight (BW) in normal children, and 1.8 times BW in obese children (Table 6.2). These findings are within the range previously reported in *vivo* knee contact force measurements made by instrumented implants during overground walking, showing that the peak value of compressive tibiofemoral force ranged from 2.1 to 3.0 times BW (Fregly et al., 2012). Within the limited studies in the obese population, Messier et al. (2005) found 3.1 times BW of compressive tibiofemoral force in 142 obese elderly persons with knee osteoarthritis. Aaboe et al. (2011) reported 2.7 times BW of peak knee loadings during walking in a similar population. These results were considerably higher than this study. However, both of the studies focused on the obese adults with knee disorders. No *in vivo* experimental data or computational data were available for obese, but otherwise healthy children.

According to the muscle force results (Figure 6.3), this study was able to analyse how muscle forces act on the joints over a gait cycle. In agreement with previously studies (Steele et al., 2012; Taylor, Walker, Perry, Cannon, & Woledge, 1998; Winby, Lloyd, Besier, & Kirk, 2009), my results showed the total joint compressive force had two peaks. The first peak appeared at the contralateral toe-off following the initial heel strike, which was mainly caused by the activity of the quadriceps. The second peak was slightly lower than the first peak, presenting at contralateral heel-strike before toe-off. It resulted from the force developed by both the gastrocnemius and quadriceps. There was no significant difference in the magnitude of gastrocnemius force between obese and normal weight children at the stance phase. In the obese group, quadriceps contributed the most to joint loading throughout the stance phase, and the contribution of the quadriceps and gastrocnemius was almost the same with the second peak. In contrast, normal weight children used more gastrocnemius force than the quadriceps force in the late stance phase. The difference between quadriceps forces at this period partly explained a higher second force peak. The action of hamstrings also contributed to the knee joint reaction force, but only appeared in early stance before contralateral toe-off, and the end of swing phase. Therefore, more quadriceps forces resulted in more compressive tibiofemoral force acting on the knees.

6.3.3 Muscle function

Induced acceleration analysis was used to determine the relationship between an isolated change in a muscle force and the corresponding changes in the movement (Erdemir et al., 2007). Based on forward dynamics solutions, the contribution of individual muscles to the vertical (support the body) and forward (progression) COM acceleration was calculated.

The results showed that muscle coordination appeared to be invariant to the differences in body mass between groups. Specifically, hip extensors (gluteus maximus and hamstrings), gluteus medius, knee extensors (rectus femoris and vasti) and ankle dorsiflexors were active in early stance to serve the function of providing support in both groups. This activity explained the appearance of the first peak in the vertical COM acceleration in early stance. The results also confirmed that quadriceps, especially vasti, generated the majority of support and decelerations in the first half of stance, which caused the first peak of compressive tibiofemoral force. In late stance phase, gastrocnemius and soleus, which are the primary muscles for plantar flexion, contributed most of the vertical and forward COM acceleration. The gluteus maximus and gluteus medius, play only a minor role in generating forward acceleration during walking. Clearly, the ankle plantarflexors provided most of the 'fore-aft' force for continued progression in both obese and normal weight children. The hamstrings also contributed to accelerate the COM throughout the stance phase, but the magnitude was relatively small (Figure 6.5). It is not surprising that obese children use similar muscles to support and accelerate body COM, because there were no neuromuscular impairments or diagnosed malalignment syndrome in these obese children. These results were in broad agreement with similar studies for walking at the self-selected (free) speed (Frank C Anderson & Pandy, 2003; Liu et al., 2008).

Although the vertical acceleration of COM was almost the same in both groups (Figure 6.1), individual muscle contributions differed (Figure 6.6). Normal weight children obviously had greater contributions to COM for almost all muscle groups due to a faster walking speed, except the contribution of hamstrings to vertical accelerations ($p = .183$) and the contribution of soleus to forward accelerations ($p = .564$). The magnitude of muscle activity generally increases with walking speed (Liu et al., 2008; Murray, Mollinger, Gardner, & Sepic, 1984). Due to a slower gait

speed, smaller muscle accelerations were observed in the OB group than the NW group in this study. Liu et al. (2008) also reported a greater vasti and gluteus maximus forces in early stance, and greater soleus and gastrocnemius forces in late stance increased with walking speeds. The activity of gluteus medius in their results was considerably greater than this study. This suggested less hip abductor moment might be reproduced in my simulation results. The magnitude of the hip abductor moment was previously found slightly less than hip flexor and knee extensor (Shultz et al., 2009). Thus, the movement of hip abductor might be underestimated in my study, though data is not available at this time to confirm this.

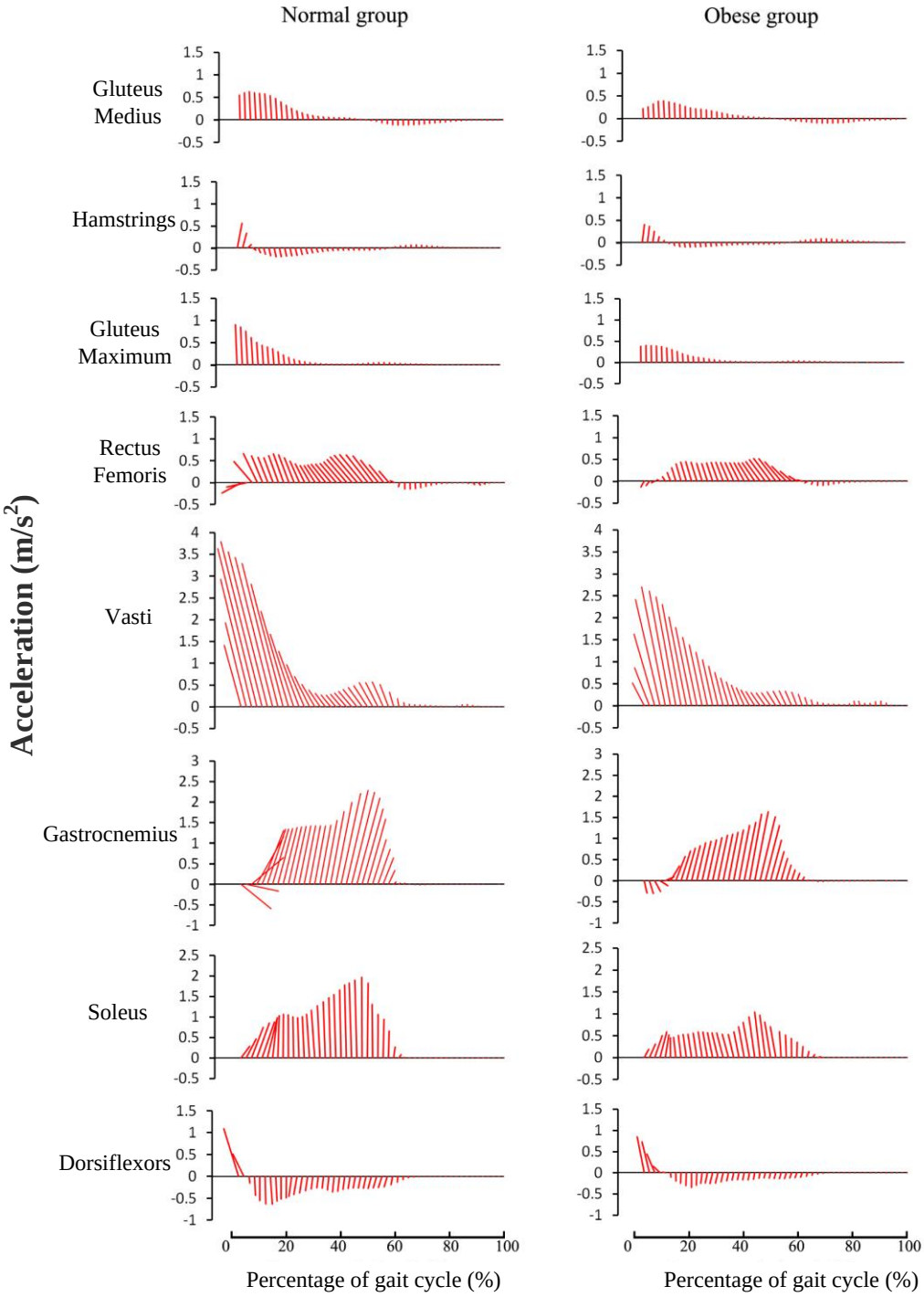
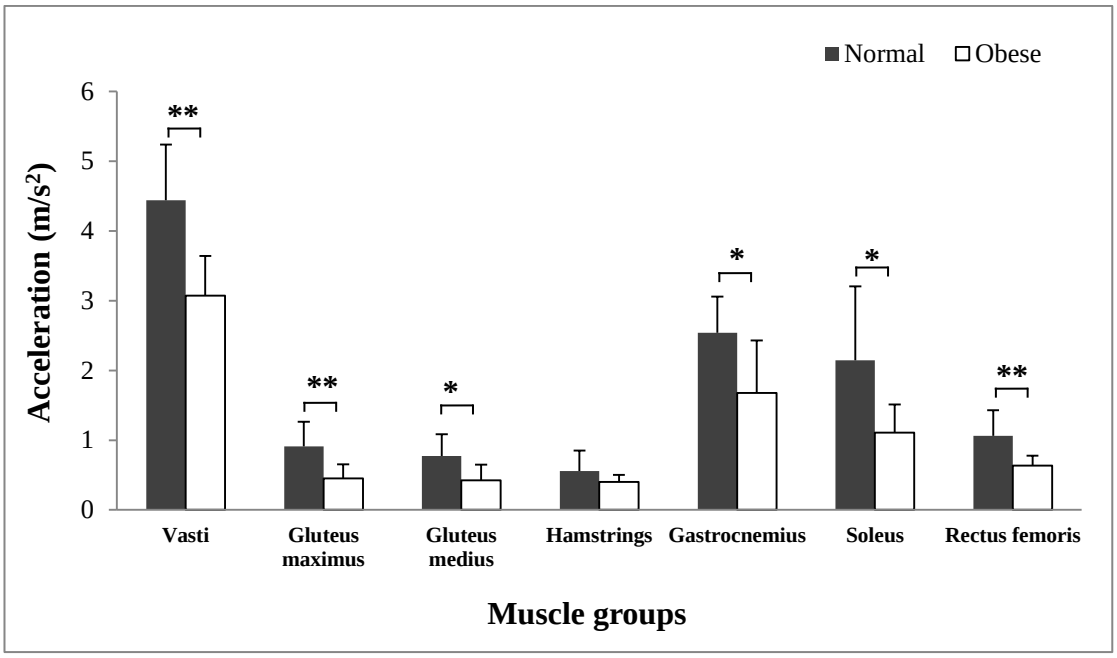


Figure 6.5 Muscle contributions to the acceleration of COM between obese and normal weight children during self-selected speed walking. Each ray is the resultant vector of the vertical and fore-aft accelerations, averaged across eight participants.

A.



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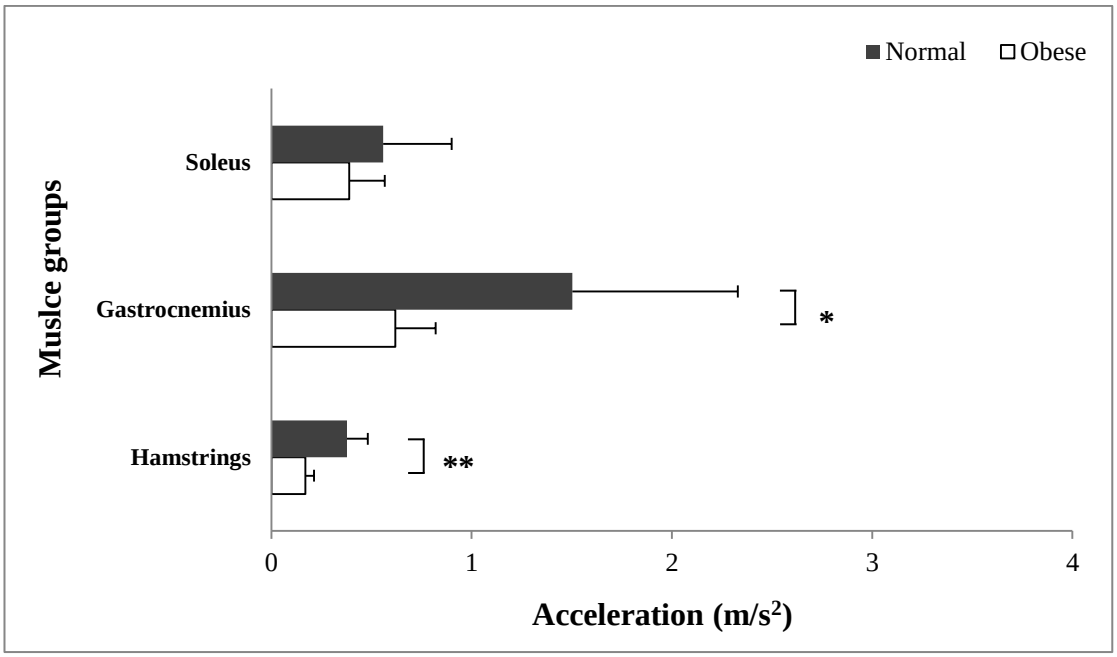

Figure 6.6 Comparison of peak vertical accelerations (A) and fore-aft accelerations (B) of the major muscles between the OB group and the NW group. *$p < .05$, **$p < .01$.

6.3.4 Recommendations for clinical applications

Taking the joint reaction force and muscle function together, it is suggested that reduced walking speed is one of the strategies used by obese children to decrease the joint load and muscle requirement. Walking has been widely used in weight loss interventions for obese youth as a primary aerobic exercise modality (Lubans, Morgan, & Tudor-Locke, 2009). According to my findings, self-selected speed walking should be more appropriate rather than fast walking as an exercise option for obese children. Although faster walking could consume more energy, it may increase the risk of musculoskeletal injury due to greater joint reaction forces and muscle requriements. Optimised mechanical efficiency and relatively lower joint loading during self-selected speed allows obese individuals to exercise for a longer time without fatigue and joint discomforts. Browning (2012) also suggested that moderate speeds should be recommended as an appropriate form of exercise for obese adults and children without osteoarthritis or varus knee alignment to promote or maintain weight loss. However, if the malalignment or joint disorders have occurred, non-weight bearing activities or weight supported exercises should be used.

6.3.5 Limitation

It is acknowledged that there are some limitations in this study. Firstly, musculoskeletal modelling requires accurate experimental marker placements. However, skin movement artefacts may affect the accuracy, especially on the hips. This may cause uncertainties in the determination of joint centres, resulting in potential over- or underestimated muscle activities. A second limitation is that the knee was modelled as a one-DOF planar joint. The varus or valgus movements cannot be investigated by using this model. The actual frontal knee alignment of the participants is impossible to match perfectly with this model. In fact, greater frontal plane moments during stance may cause damage to knee joint, limited motion and potential disability (McMillan et al., 2010). This model error may reduce the activities of those muscles attached to the medial and lateral knee. Although this study was not aimed to study the medial and lateral compartment of joint loads, this inaccuracy could result in potential over- or underestimated muscle activities, such as hip abductors. In addition, EMG was not measured in this study to verify the muscle excitations. This Hill-type muscle based generic model has shown its reliability in various gait studies, but it is indeed possible that obese participants stimulated

slightly different muscle groups but showing similar gait kinematics as normal weight participants. Thus, conclusions for obese children based on simulations with current generic musculoskeletal parameters should be interpreted with caution.

6.3.6 Future work

Given the prevalence of childhood obesity and the lack of specific musculoskeletal model for obese population, it is critical to continue to develop a more accurate model to investigate the biomechanical effect of obesity at musculoskeletal level. Use of medical imaging (e.g. MRI, DEXA etc.) in obese population will be extremely important to provide more accurate information on landmark positions, mass distribution of segments and the muscle properties, then to further reduce the inaccuracy of dynamic simulation results. For those obese individuals who already have an abnormal knee alignment (e.g., bow legs, knock-kneed legs), a more complex knee model is needed to determine gait mechanics with knee malalignment (varus/valgus). Researchers can further obtain the medial and lateral compartment joint load and the ligament forces by integrating with medical imaging data. In addition, forward dynamic simulations have great potential in obesity prevention and intervention. In a recent review, Browning (2012) pointed out that the utilization of complex, individualized musculoskeletal models would allow us to predict the outcome of interventions before implementation. Based on an improved and verified "obese" musculoskeletal model, a computational framework can be developed to predict post-treatment outcome from pre-treatment movement data for obese individuals. Dynamic simulations not only can assist specialists in designing targeted obesity treatments or exercise interventions, but also can theoretically test the effectiveness of an orthopaedic device (e.g. wedged insoles, knee varus/valgus brace) on musculoskeletal problems. The final challenge, similar to most model based simulation, is addressing muscle fatigue if using dynamic simulations to analyse exercise in obese children. Fatigue can limit the ability of a muscle to generate force and change the muscle activation characteristics (Gandevia, 2001; Maffiuletti et al., 2007). It will be necessary to develop a model that can be used in some situations where fatigue is likely to occur.

6.5 CONCLUSION

Obese children had a lower normalized compressive tibiofemoral force than normal weight children when walking at their self-selected speed. The vertical acceleration of COM was almost the same between two groups, while the obese children had lower contributions of individual muscles to support and progress the body during gait. These findings suggested that obese children may adapt to a compensation gait strategy (e.g. reduced walking speed) to avoid increasing joint loads and muscle requirements during walking.

Chapter 7: Conclusions

This dissertation includes both cross-sectional and intervention studies to investigate the implication of childhood obesity on the musculoskeletal system during locomotion, by examining the natural walking biomechanics and energetics. The findings of this work were summarised in four studies (Chapter 3 to Chapter 6).

7.1 MAJOR FINDINGS OF THIS THESIS

7.1.1 The differences in walking biomechanics and energetics between obese and normal weight children

The first study is a cross-sectional study which compared the differences in walking biomechanics and energetics between obese and normal weight children. Obese children walked with 0.15 m/s slower walking speed, 10.0% lower cadence and 30.9% longer double support phase than normal children, but no difference was found in mediolateral and vertical body COM displacement. The excess body weight caused higher absolute joint moments in the obese children. Mechanical energy expenditure was 72.7% higher in obese children than in normal-weight participants. The net metabolic cost was 65.7% higher in obese children. When normalized by body mass, no difference was found in metabolic rate, mechanical work or efficiency between obese and normal weight group.

These results suggested that the excess body mass plays a dominant role in gait biomechanics and energetics. Obese children may choose an adaptive walking strategy that can minimize the increase of energy expenditure and maintain the mechanical efficiency. It may be also a compensation strategy used by obese children to aviod increading knee joint loads.

7.1.2 The effect of weight loss

An 8.0% weight loss with reduced body circumferences caused mass-driven changes in joint kinematics and kinetics. Without significant changes in spatiotemporal parameters, few differences were found in the joint kinematics. Changes in joint moments are more likely a simple mass-driven adaptation, the absolute joint moments decreased in proportion to weight loss. Hip kinematics and kinetic signified a key gait adaptation to weight loss. In addition, weight loss

significantly reduced the metabolic and mechanical energy cost during walking, while the metabolic rate and the normalized MEE did not change.

This is the first study to examine the 3D lower extremity joint kinematics and kinetics in obese children after short term weight loss, compared to a control group. This study confirmed the dominant role of the body mass in the gait differences between obese and normal weight children. It also provided evidence that obese individuals prefer to walk at a speed that not only has minimal energy cost and maximal mechanical efficiency, but also can avoid an increase in joint load. An increase in self-selected walking speed is not a necessary outcome for a short-term weight loss.

7.1.3 The role of muscle strength

Previous studies have failed to exclude the influence of muscle strength in the gait differences between obese and normal weight children, and the biomechanical and energetic changes following weight loss. This study was carried out to find out how these parameters were associated with increases in muscle strength in response to resistance training. Without changes in body weight, muscle strengthening resulted in a faster walking speed and a larger hip flexor moment during the late stance phase and early swing phase. Increased joint moments required more muscle forces to promote locomotion, which explained significant increases in the metabolic and mechanical cost.

The results indicated that the obese individuals prefer to pull their limb into swing by generating more hip flexion forces during walking, rather than use ankle plantarflexion muscles to propel the body forward. Muscle strengthening allowed obese children to adapt a faster walking speed and consume more energy, but it is unable to improve the efficiency of energy transfer at the hips. Therefore, muscle strengthening in lower extremities improved obese children's ability to promote locomotion through greater propulsion, but it cannot reverse the impact of excess body weight on natural walking biomechanics and energetics.

7.1.4 Qualification of knee joint loads and musculoskeletal function

This study utilized musculoskeletal modelling and simulation to investigate the knee joint reaction force and muscle function in obese children. The compressive tibiofemoral force and individual muscle contribution to the support and progression

accelerations of centre of mass (COM) were computed for each participant based on the subject-specific model. The simulated results were evaluated by comparison with the experimental kinematics and EMG data. There was a linear relationship between the average self-selected speed and the normalized peak compressive tibiofemoral force. The activity of the quadriceps contributed the most to the peak compressive tibiofemoral force during the stance phase. Obese children and normal weight children use similar muscles to support and accelerate body COM, but normal weight children had significantly greater contributions of individual muscles. Therefore, this study confirmed that the obese children adapt a compensation gait strategy to avoid increasing joint loads and muscle requirements during normal walking.

7.2 CONTRIBUTION OF THIS THESIS

7.2.1 Intellectual contribution

Understanding and addressing the musculoskeletal implications associated with childhood obesity are the key to developing safe and effective interventions that may improve musculoskeletal health and increase participants' ability to engage in physical activities. To the best of our knowledge, this is the first study to examine 3D gait biomechanics and energetics in obese children after substantial weight loss and muscle strengthening, respectively. This study supplies scientific and reliable reference data into the relationship between gait adaptation and excess body weight. It also fills the knowledge gap about the effect of weight reduction and muscle strengthening on the biomechanics and energetics of walking. In addition, , this study extends the usage of a musculoskeletal simulation model to walking in obese children. This is the first study with the purpose to examine the dynamic joint reaction force and individual muscle activity in obese children during normal walking.

7.2.2 Clinical applications

Walking is one of the primary aerobic exercises in childhood obesity management programs (Lubans et al., 2009). The findings of this project suggest that walking at the self-selected speed should be recommended in the early stages of intervention programs for obese children to promote or maintain their weight loss. Optimised walking energetics allows obese individuals to exercise for a longer time without fatigue and expend a considerable amount of energy (Sahlin, Tonkonogi &

Söderlund, 1998). Walking at the self-selected speed would also allow obese children to compensate for potential muscle weakness, develop correct motor patterns, and reduce the risk of injuries. The latter would be enhanced if severe walking regimens are introduced, especially if introduction is rapid. Conversely, if the self-selected walking speed is low, increase in muscle force or power is unlikely, fatigue is more likely, bone and joint stresses increase, all leading to another pathway to higher risk of injury. Thus, monitoring and recording of the self-selected gait velocity is recommended, so that progress can be tracked. Progress in all features of the gait as it changes over time would be best determined by longitudinal measurements using data acquisition systems similar to described in Chapter 3. There is a temptation to emphasise energy expenditure when formulating exercise regimens in obesity management. But because of the known predisposition of young people who are obese to higher risk of many orthopaedic diseases, including traumatic injury due to falling, it is timely to emphasise exercise regimens that are broad-based and not confined to only an energetics approach to weight loss, which in adults (National Institutes of Health, 1998) and young people (Barlow & Dietz, 1998) often are less than successful. Thus the clinical approach should be aimed at also specific muscle force development programme as a strategy to correct gait deficits, and at brain development and training aimed at reversal of compromised proprioception and muscle power development. Such an approach may enhance chances that musculoskeletal pain and discomfort is not associated with exercise in the mind of a young person, and that resentment or refractoriness to exercise declines.

Thus, it is recommended that gait assessment and musculoskeletal simulation is performed together with physiological examination before the implementation of an exercise intervention, especially when risk or fast walking or jogging is to be used in the program. For obese individuals, a faster walking/running speed dramatically increases energy expenditure (Nantel et al., 2009). However, injuries may occur as a result of increased joint reaction forces and muscle activities when obese individuals modify their gait to move the body faster (Wearing et al., 2006). At this point, musculoskeletal simulations provide quantitative information about joint reaction forces and individual muscle function during movement, and thus is the opportunity to perform a serial critical reviews of the intervention program. It also allows determination of the gait speed would achieve a better balance between energy

expenditure and musculoskeletal health. Lastly, there is an important clinical opportunity here, namely that the clinical team can use serial, accurate data to carefully design and serially modify (on successive examinations) all of several elements of the exercise regimen, including warm-up, speed, slope, added weights, and speed/duration of all sectors of the daily walking/jogging regimen, as well as proprioception/power elements of the comprehensive exercise prescription.

As outlined above, another clinical application of the research described in this thesis, namely innovative muscle strength training which focuses on target muscles specific to the obese individual's needs, should be integrated into a weight management program. The simulation results, along with the experimental data, can be used to determine if and when strength training is required, which muscle group should be treated, and what the specific prescription for that muscle (group) should be. For example, obese children are more likely to advance the limb into the swing phase by generating higher hip flexor forces. The resulting overuse of the quadriceps group may cause discomfort, pain or patellofemoral joint dysfunction. Strengthening the quadriceps muscle is a key factor for preventing this overuse injury. The serial simulations will provide quantitative data to determine rehabilitation goals and evaluate the effectiveness of the strength training when training obese individuals. This method can be linked to the musculoskeletal model so that more functional clinical measures could easily completed in the clinical setting.

The implications of the research described in this thesis are perhaps representative of the next logical development of exercise prescription for individuals who do not have normal neuromusculoskeletal development because of their primary metabolic disease. Until now, exercise prescription has been general. The novelty which gives cause for optimism is that kinematic acquisition and processing capability has increased, and data on specific muscle action can be acquired during ambulation. This capability, together with rapid processing speeds and miniaturisation of hardware, heralds the possibility that such measures can become a central part of clinical assessment, and of supervision of exercise in obese individuals.

7.3 PUBLICATIONS ARISING FROM THIS THESIS

The following papers have been published or in press in peer-reviewed journals or conference proceedings:

- **Huang, L.**, Chen, P., Zhuang J., Zhang Y., & Walt S. (2013). Metabolic cost, mechanical work, and efficiency during normal walking in obese and normal weight children. *Research Quarterly for Exercise and Sport*, 84: S72-S79

- **Huang, L**, Zhang, Y, & Zhuang, J. (2013) The Application of Computer Modelling and Simulation to Investigate Compressive Tibiofemoral Force and Muscle Functions in Obese Children, Computational and Mathematical Methods in Medicine, 2013: 305434

- **Huang, L.**, Zhuang, J., & Zhang, Y. (2013) A method of speed control during over-ground walking: using a digital light-emitting diode light strip. *Advanced Materials Research*, 718-720, 1371-1376

- **Huang, L**., Zhang, Y., & Zhuang, J. (2012). The effect of weight loss on gait characteristics of obese children. *Obesity Research & Clinical Practice*, 6, 35.

- **Huang, L**., Zhang, Y., & Zhuang, J. (2013). Effects of lower extremity strength training on gait patterns in obese children. *ISBS - Conference Proceedings*, 4 pages

The following oral presentations have been given at international conferences:

- The 31th Conference of the International Society of Biomechanics in Sport, *Effects of lower extremity strength training on gait patterns in obese children,* Taipei 2013, Taiwan, 7 - 11 July, 2013

- The Australian and New Zealand Obesity Society Annual Conference, *The effect of weight loss on gait characteristics of obese children*, Auckland, New Zealand, 18 - 19 October, 2012

- SESNZ Annual Conference 2011, *The effect of body mass on the children's biomechanics and energetic cost during walking*, Auckland, New Zealand, 18 - 19 November, 2011.

7.4 FUTURE WORK

The findings of this study pertain to normal walking, more biomechanical studies involving walking over various speeds and a wider range of physical activities are also needed to establish the relationship between childhood obesity, musculoskeletal function and physical activities. Additional studies of obese children of varying ages and levels of adiposity are helpful to develop a comprehensive understanding of how obesity affects children's musculoskeletal system during the developmental age. Use of medical imaging (e.g. MRI, DEXA) in future studies will

be extremely important to provide more accurate information on landmark positions, mass distribution of segments, body composition and the muscle properties for motion analysis and dynamic simulations. In light of the musculoskeletal benefits of the diet and exercise-induced weight loss and strength training associated with a number of changes in biomechanical and energetic parameters, future randomized controlled trials are needed to investigate their combined effect. The data are necessary for prescribing an effective program that not only promotes physical activity to increase energy expenditure in obese individuals, but also minimizes the risk of joint problems to maintain their musculoskeletal health.

Appendix B. Marker placement and biomechanical model

The Cleveland Clinic marker set (Sutherland, 2002) was used in the biomechanical gait analysis. In static trial, 23 markers were placed on anatomical landmarks of the body, an additional 12 markers which are grouped in triads on rigid plates and wrapped around shanks and thighs. The local coordinate system was set up for each segment based on these markers. The hip, knee, ankle joint centres were defined relative to local coordinate systems. The hip joint centre was estimated using a regression equation (Davis, Ounpuu, Tyburski, & Gage, 1991). The knee joint centre was the midpoint between medial and lateral epicondyle, and the ankle joint centre was defined as the midpoint of medial and lateral malleolus. The triads along with the medial and lateral markers placed on knee and ankle were used to define the relative positions of the knee and ankle joint centre to the local coordinate system. Then the knee and ankle joint markers can be removed during the dynamic trials.

a. b.

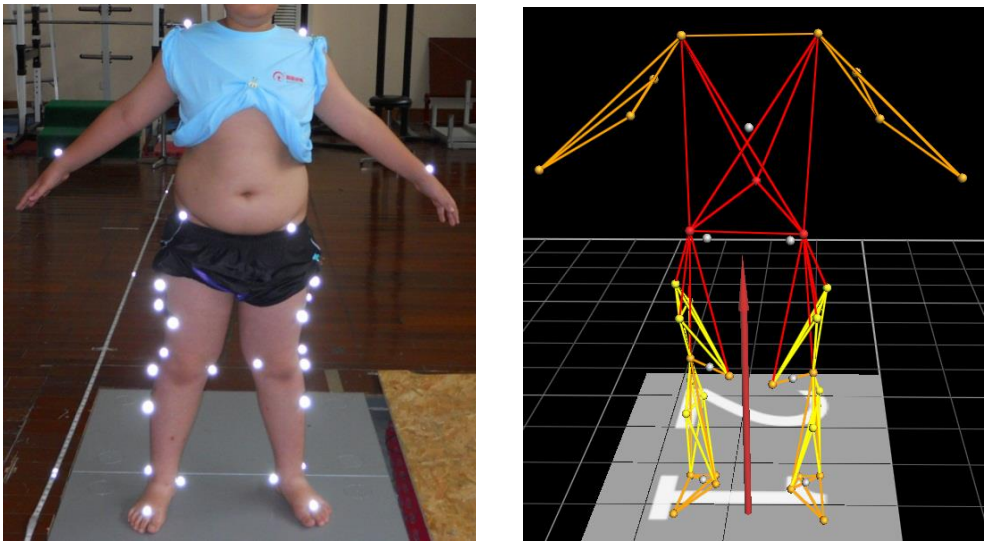

Figure B.1. An example of the marker placement on participant. b. Screenshot of the biomechanical model with virtual joint centres in Nexus.

Table B.1 The detailed positions of the markers placed.

Upper body	
Shoulder	Acromion process
Arms	
Triceps	Muscle belly
Elbow	Lateral epicondyle
Wrist	Posterior aspect (in anatomical position)
Pelvis	
ASIS	Anterior superior iliac spine
Sacrum	The midpoint of posterior superior iliac spine
Thigh	
Triads	Any position on lateral aspect
Shank	
Triads	Any position on lateral aspect
Knee	Medial and lateral epicondyle
Ankle	Medial and lateral malleolus
Foot	
Heel	Posterior aspect of heel
Forefoot	Head of the 2nd metatarsal

Appendix C. The speed control system for overground walking

Our study required the marker displacements, ground reaction forces and metabolic data measured simultaneously during self-selected walking speed. To collect accurate data, the participants must be walking at a relatively constant speed during measurement. Since the force-plate-integrated treadmills were not available, an alternative speed control method for overground walking was needed. A low cost LED system was developed to provide a visual cue for walking speed control (Huang et al., 2013).

The LED system includes a custom-made LED light strip and a digital micro-controller. The LED light strip consists of a number of LED units with built-in microchips, and each LED unit can be controlled individually (Figure C.1). The distance between each two LED unit is the same. The digital micro-controller is used to create a visual target moving along the light strip by controlling the timing of power supply for each LED unit (Figure C.2). The flexibility of the LED light strip enables it to be wired to form a digital track that has built in force-plates (Figure C.3).

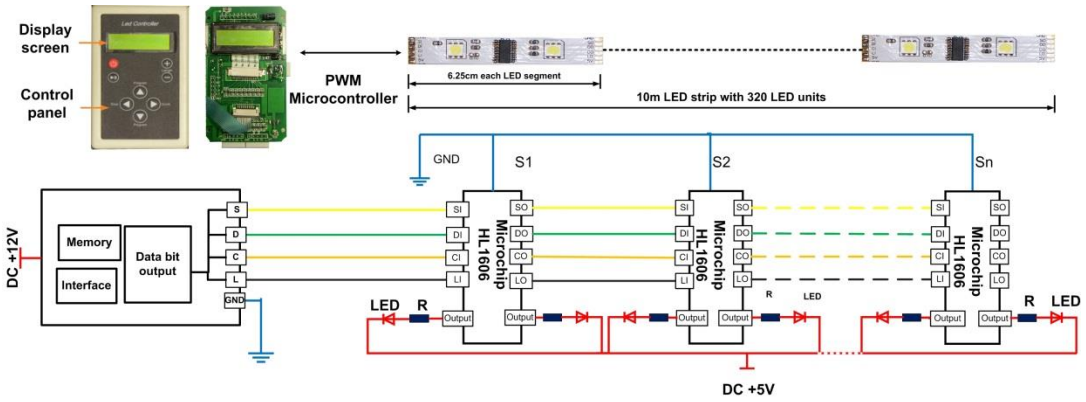

Figure C.1 Schematic of the LED system. The system includes a LED strip with HL1606 microchips and a Pulse Width Modulation (PWM) built-in microcontroller. The data is firstly sent and copied on the DI (data in), CI (clock in) and SI (speed clock input) lines. Under control of the LI (latch in), the data were pushed down the line to the next microchip through the DO (data out) pin, CO (clock out), SO (speed clock output) and LO (latch out) pins. In this way, two LED units were connected and each one can be controlled individually.

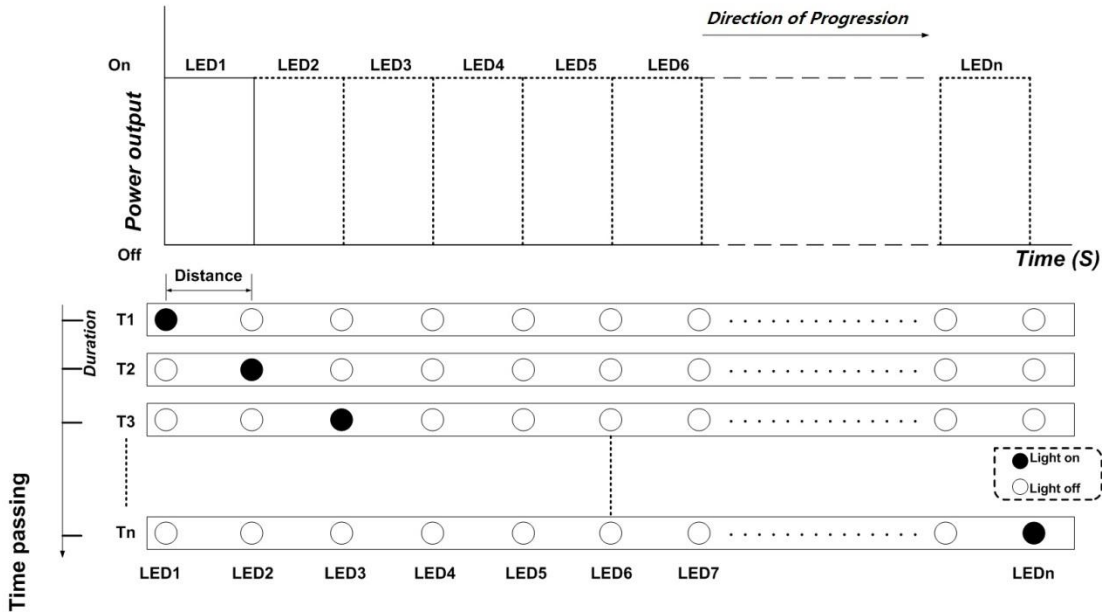

Figure C.2 Schematic of the "running" effect. The distance between each two adjacent LED unit is the same. The duration time of power supply for each LED is also the same. Duration=T_2-T_1=T_3-T_2=…=T_n-T_{n-1}.

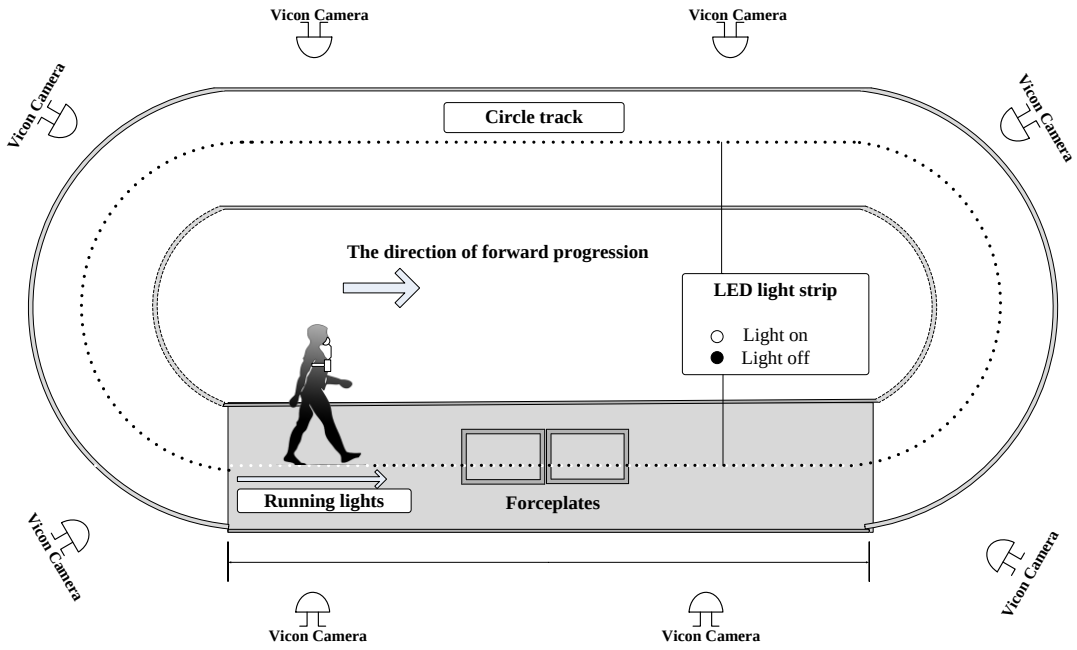

Figure C.3 Experimental set-up for the LED light strip.

Appendix D. Joint kinematic and kinematic data of the control group for Chapter 4

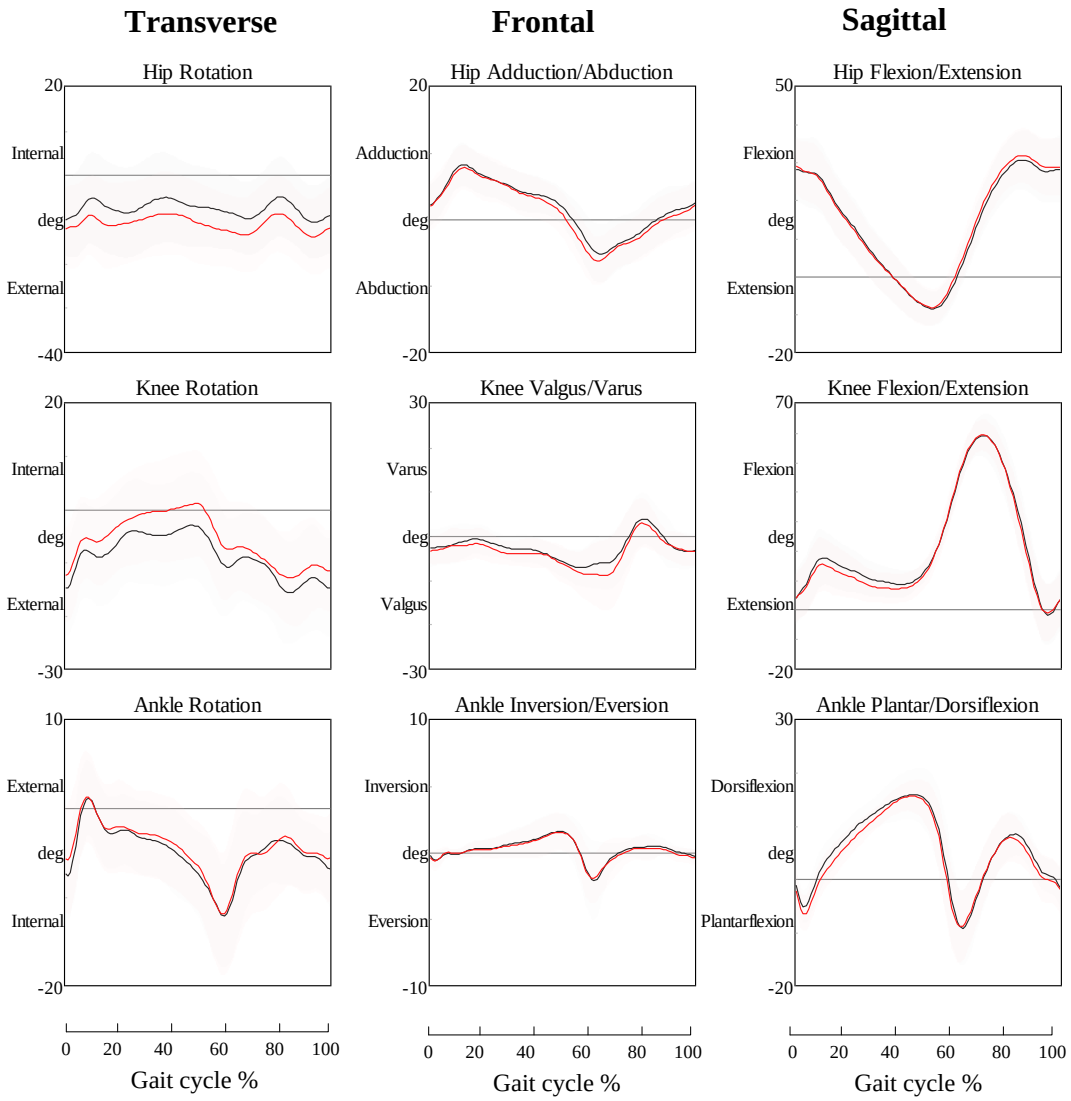

Figure D.1 Mean joint angles (before: black line, after: red line), and standard deviation (before: grey shaded area, after: red shaded area) over a gait cycle for the control group.

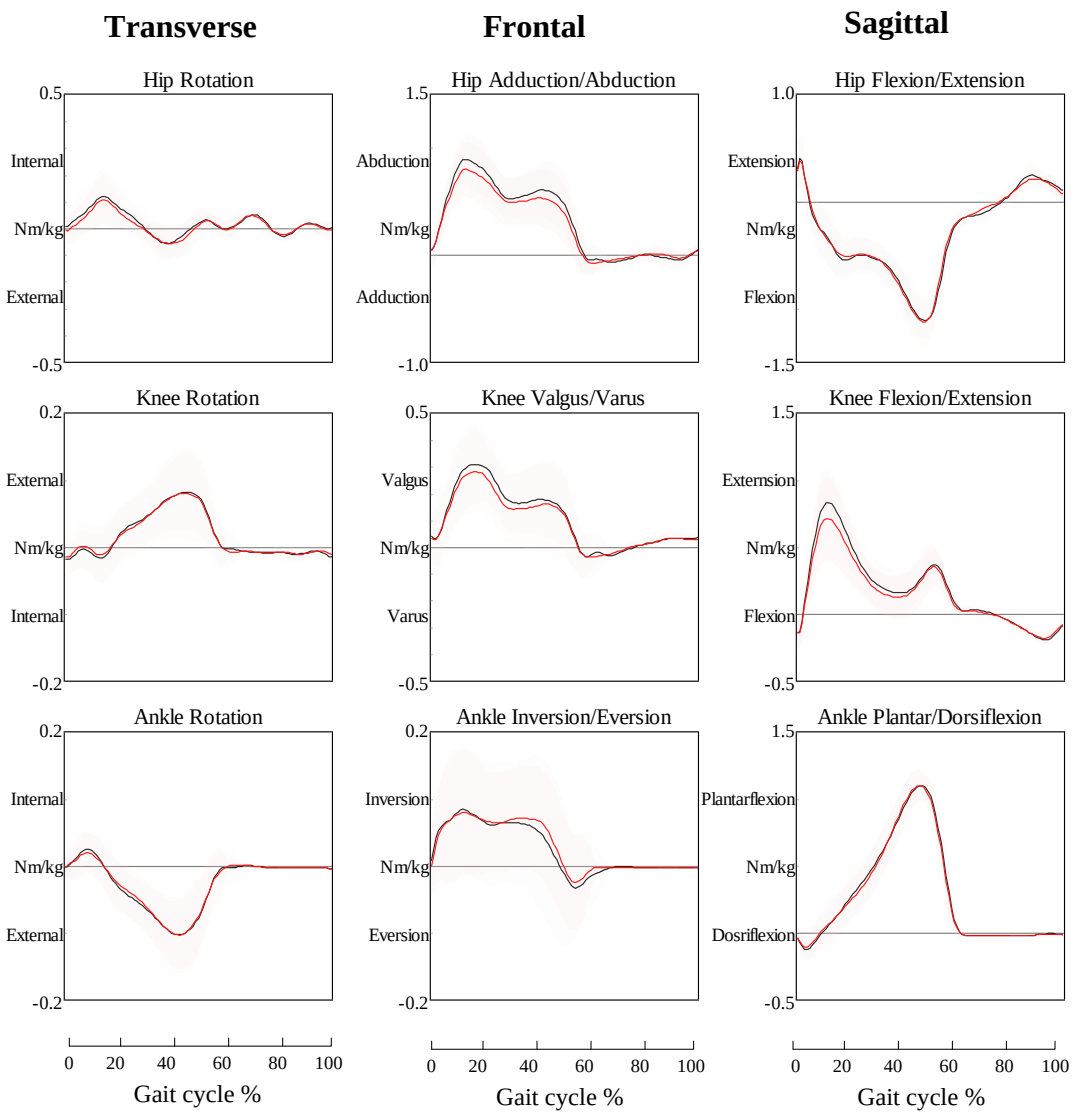

Figure D.2 Mean joint moments (before: black line, after: red line), and standard deviation (before: grey shaded area, after: red shaded area) over a gait cycle for the control group.

Appendix E. Children's OMNI Resistance Exercise Scale (OMNI-RES) of perceived exertion.

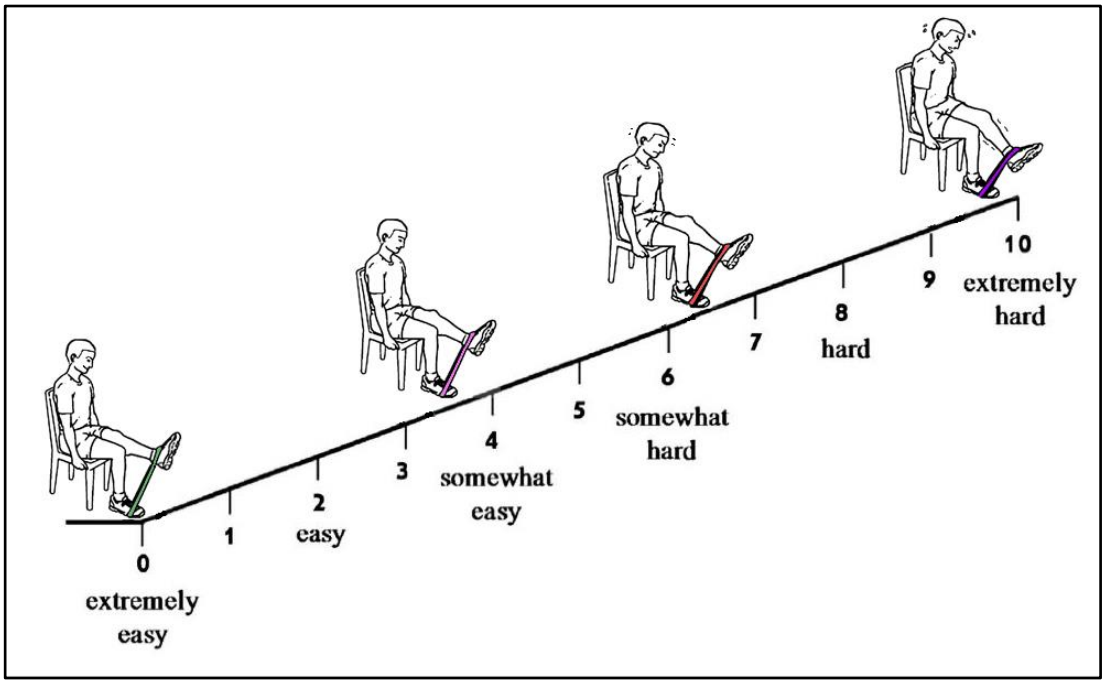

Figure E.1 Children's OMNI-RES for the strength training program.

Appendix F. Simulation of walking

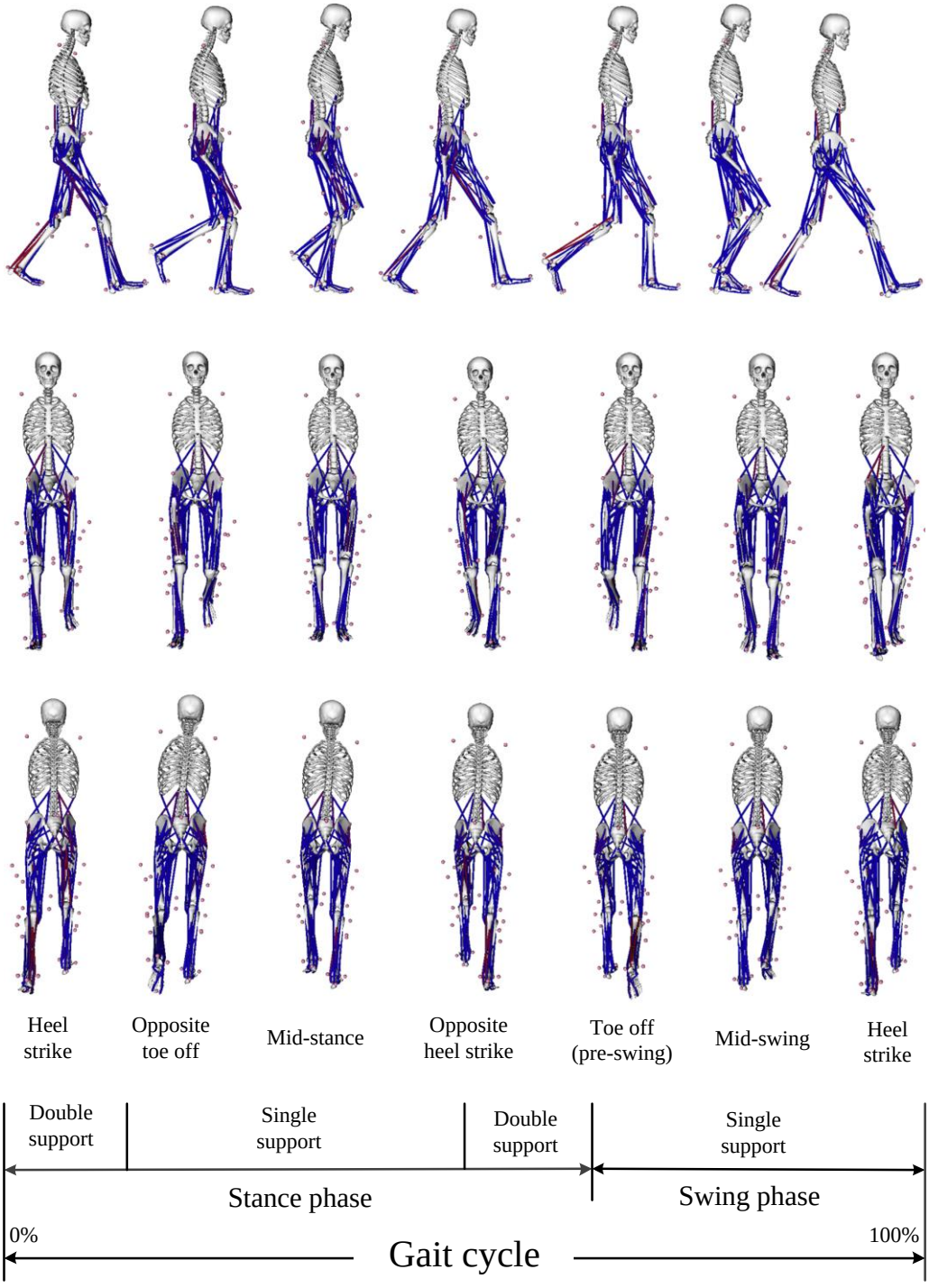

Figure F.1 3D walking simulation over a gait cycle from lateral, anterior, and posterior views. The virtual markers are shown as pink dots, and all muscles are represented by lines. The muscle colours represent the activation level changing from blue to red (Blue = no activation, bright red = full activation).

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