Gait, foot structure, and muscle strength in obese children: further steps towards evidence informed care.

Megan Le Warne

MRes Sport, Health and Applied Science

2017

Contents

Abstract

Childhood obesity is an increasing problem in the UK and co-morbidities of obesity are increasingly evident. Previous research has suggested altered musculoskeletal function including; lower limb and foot biomechanics, muscular strength and plantar pressures in obese children compared to healthy weight peers. However, there has been limited research to quantify the associations between weight status and excessive body fat mass with musculoskeletal function. The primary aim of this research was to explore lower limb and foot biomechanics, plantar pressures and lower limb strength in 7-to-11 year old children.

Sixty-nine children were recruited to participate in protocols for; body composition, three-dimensional gait analysis of the lower limb and foot, plantar pressures and strength assessment of the hip, knee and ankle. Principal component analysis was undertaken to determine regression scores representing elements of the gait cycle to be further analysed. Strength variables and regression scores were entered into MANCOVAs to assess the difference between Z score derived overweight/obese and healthy groups. Multiple regression was undertaken to assess the relationships between lower limb and foot variables, plantar pressures, and strength with body fat.

There was no difference in lower limb kinematics or foot segment angles between groups. Overweight/obese group had increased sagittal knee moment and frontal power. The overweight/obese group were stronger and more powerful in absolute terms in ankle dorsiflexion and knee extension and flexion. When strength was normalised to body mass, fat mass and fat free mass, overweight/obese were weaker and less powerful.

Body fat had no effect on lower limb kinematics but significantly effected metatarsals, midfoot and calcaneus angular motion. Body fat significantly increased ankle, knee and hip moment and powers. Midfoot and forefoot plantar pressure was significantly predicted by body fat. As body fat increased absolute strength in the knee extensors, flexors and ankle dorsiflexors increased. Strength normalised to body mass decreased with increasing body fat. Body fat significantly predicted (as body fat increased

strength decreased) strength normalised to fat free mass in all muscle groups except isokinetic ankle dorsiflexors.

This work demonstrates the need to use appropriate parameters for investigations into the effects of obesity. BMI-derived weight categorisation makes the findings easily relatable back to a clinical population, but it produces less significant findings compared to a direct measure body fat. The overall findings are that overweight/obese children are relatively weaker and have increased loading of lower limb and foot joints during gait. This work demonstrates the need to understand the effects of obesity on musculoskeletal function to aid clinical rehabilitation and reduce the health burden in later life.

1. Introduction

Over one third of children in the UK are reported to be overweight or obese (NCMP 2016; Lobstein & Jackson-Leach 2016). Children that are overweight at the start of primary school (4-5 years old) remain overweight (30%), become obese (30%) or severely obese (13%) by the time they leave primary education (10-11 years old) (Public Health England, 2017). Childhood obesity is associated with significant comorbidity and disability such as diabetes, cardiovascular disease, musculoskeletal pain, deviations and inefficiencies in gait, and restriction in general daily activities such as walking and moving from sitting to standing (Chan & Chen, 2009; Taylor et al., 2006; Tsiros, Coates, Howe, Grimshaw, & Buckley, 2011). As an increasing population of obese children age into obese adults the physical and physiological health implications, and the accompanying cost of treating obesity related comorbidities rise (Lobstein & Jackson-Leach, 2016). Child obesity is a complex issue with a number of suggested risk factors such as family environment and parental fatness, nevertheless, excess fat mass results principally from chronic positive energy imbalance (Gillman, 2008; Yu et al., 2011). However, increased fat mass has been linked to reduced physical activity and vice versa, due to the effects on the musculoskeletal system (Metcalf et al., 2011; Shultz, Anner, & Hills, 2009). The adverse impact of excess mass on the immature musculoskeletal system warrants investigation to tackle the negative cycle of increasing mass and decreasing physical activity.

Normal development is dependent on adequate magnitude and loading patterns on the musculoskeletal system (Levine & Drennan, 1982). However, carrying excess mass on an immature skeletal system can create structural damage, malalignment and lead to orthopaedic complications and pain (Shultz et al., 2009; Wearing, Hennig, Byrne, Steele, & Hills, 2006). Orthopaedic complications in obese children are generally in the weight bearing structures of the lower limb, including the hip, knee, and foot which can promote biomechanical changes and reduce physical activity (De Sá Pinto, De Barros Holanda, Radu, Villares, & Lima, 2006). Obese and overweight children have high incidence of reported disorders such as slipped capital femoral

epiphys (SCFE), Blounts disease and pes planus (Kelsey, Acheson, Keggi, Haven, & Haven, 2015; Pirpiris, Jackson, Farng, Bowen, & Otsuka, 2006; Stolzman, Irby, Callahan, & Skelton, 2015). A combination of excess mass and altered hip alignment increases forces during weight bearing activities such as walking, and increases shear and compressive forces across the growth plate increasing the risk of SCFE (Benson, Miller, Bosch, & Szalay, 2008; Manoff, Banffy, & Winell, 2005; Murray & Wilson, 2008; Galbraith et al., 1987; Wills, 2004). Chronic knee varus deformity and associated gait deviations in obese children lead to increased loading in the medial compartment of the knee which, with sufficient magnitude, can alter physeal growth and lead to Blounts disease (Cook, Lavernia, Burke, Skinner, & Haddad, 1983; Lerner, Board, & Browning, 2016). Obese children are also at risk for altered foot function, pes planus and foot pain as a result of excess mass. Current literature suggests increasing pes planus with increasing excess weight and BMI as well as a significant correlation of BMI and those reporting pain in the feet during activity (Sadeghi-Demneh et al., 2016; Stolzman et al., 2015). Previous studies have reported significant musculoskeletal pain in obese children, common areas for complaint are the general lower limb, knees, lower back, and feet (De Sá Pintoet al., 2006; Stovitz, Pardee, Vazquez, Duval, & Schwimmer, 2008; Taylor et al., 2006). Malalignment, joint disorder and pain significantly reduce physical function and quality of life of children who are overweight or obese. This contributes to a cycle of weight gain and reduced physical activity and function (Bout-Tabaku, Briggs, & Schmitt, 2013; Smith, Sumar, & Dixon, 2014).

Obese children and adolescents generally exhibit slower walking velocity, shorter stride, step lengths and greater step width compared to healthy weight counterparts (Dufek et al., 2012; Freedman Silvernail, Milner, Thompson, Zhang, & Zhao, 2013; Hills & Parker, 1992; McGraw, McClenaghan, Williams, Dickerson, & Ward, 2000). Obese children demonstrate a more cautious gait to control for larger inertial forces from excess mass and increased frontal plane movement due to greater medial/lateral sway (Dufek et al., 2012; Huang, Chen, Zhuang, Zhang, & Walt, 2013). As adiposity increases, range of motion of the pelvis in the frontal plane during gait also increases (Lerner, Shultz, Board, Kung, & Browning, 2014). This gain in pelvic obliquity during single limb stance causes a drop in the centre of the hip joint centre (Lerner et al.,

2014) . The pelvis is controlled by the gluteus medius, the strength of which (relative to lean weight during gait), is positively correlated with body fat percentage in children (Lerner et al., 2014). Carrying excess weight increases the demand of stabilising muscles to prevent collapse of the lower limb during single limb weight bearing (Shultz, D'Hondt, Lenoir, Fink, & Hills, 2014). Obese children have significantly higher peak hip flexion, extension moments (McMillan, Pulver, Collier, & Williams, 2010; Shultz, Sitler, Tierney, Hillstrom, & Song, 2009), as well as timing differences of peaks that require less hip extensor strength but also results in increased hip rotation moments (McMillan et al., 2010). Greater peak power generation and absorption seen in obese children in all three planes, and across gait phases, are due an increased mass to control and aid in the propelling of the heavier limb (Shultz et al., 2014; Shultz, Hills, Sitler, & Hillstrom, 2010).

Kinetic analysis shows obese children to have significantly larger peak knee abduction, adduction, extension and flexion joint moments (Gushue, Houck, & Lerner, 2005; Lerner & Browning, 2016; McMillan et al., 2010; & Shultz et al., 2009; McMillan, Auman, Collier & Williams, 2009). Obese children also exhibit increased power absorption in the knee extensors at weight acceptance, and push off to offset the increased power generation of the hip and ankle at these times (Shultz et al., 2010). Obese children have significantly increased plantarflexion moments and joint powers during gait (Shultz, D'Hondt, Fink, Lenoir, & Hills, 2014; Shultz et al., 2009) and significantly lower inversion moments during stance, with peak moments occurring earlier than in healthy weight boys (McMillan et al., 2009). Research has shown that increases in body size are not proportionate to increases in the articulating surface area of joints in adults (Ding, Cicuttini, Scott, Cooley, & Jones, 2005). Thus higher absolute peak moments in obese children may indicate increased forces on the lower limb joints (Shultz et al., 2009).

Excessive weight bearing, lower limb malalignment, altered biomechanics, flexibility and late ossification make the foot vulnerable to deformity such as pes planus. Overweight and obese groups have altered growth patterns and it is suggested that morphological changes are associated with structural changes within the foot (Mauch,

Grau, Krauss, Maiwald, & Horstmann, 2008). Particularly, obese children have been reported to have lower medial longitudinal arch (Adoración Villarroya, Manuel Esquivel, Tomás, Buenafé, & Moreno, 2008; Mueller, Carlsohn, Mueller, Baur, & Mayer, 2016; Yan, Zhang, Tan, Yang, & Liu, 2013).

During stance, obese children exhibit higher peak plantar pressures over all regions of the foot (Cousins, Morrison, & Drechsler, 2013; da Rocha, Bratz, Gubert, de David, & Carpes, 2014). This indicates increased loading in the developing foot, increasing the risk of injury, discomfort, and deformity (Yan et al., 2013). Relationships between body fat and midfoot, rearfoot sagittal and frontal planes have been found, suggesting a more pronated foot with higher body fat (Mahaffey, Morrison, Bassett, Drechsler, & Cramp, 2016). This has suggested to be a result of carrying excess mass, increasing load on weaker muscles or more compliant support structures of the arch (Van Boerum & Sangeorzan, 2003).

Authors have suggested muscular weakness, in relation to the increased demand of carrying excess mass, to be responsible for altered biomechanics (Lerner, Board, Browning, 2014; McMillan et al., 2010). Model simulations based on healthy adolescents, show that gait is sensitive to muscular weakness in the hip abductors, ankle plantar flexors and hip flexors, and robust to weaknesses in the hip and knee extensors (van der Krogt, Delp, & Schwartz, 2012). Muscular weakness results in increased activity of weaker and neighbouring muscles to maintain gait, causing unbalanced joint moments (van der Krogt et al., 2012).

Studies reporting strength in obese children measured using dynamometry, have generally shown equal or higher absolute muscular torque in the knee compared to healthy weight children (Abdelmoula et al., 2012; Blimkie, Ebbesen, MacDougall, Bar-Or, & Sale, 1989; Garcia-Vicencio et al., 2016; Tsiros et al., 2013). This is thought to be due to a larger muscle mass resulting from a training effect of carrying a heavier mass (Garcia-Vicencio et al., 2016). Strength relative to body weight, fat mas and fat free mass have found varying results possibly due to different methodologies in strength normalisation (Abdelmoula et al., 2012; Blimkie et al., 1989; Garcia-Vicencio

et al., 2016; Tsiros et al., 2013). Little data currently exists on direct measures of strength in the obese paediatric population in muscle groups other than the knee extensors. When referring to a functional tasks such as walking it would be valuable to examine isometric and isokinetic muscle strength and power in a range of lower limb muscle groups. In addition to the most effective normalisation methods to investigate functional differences in obese and non-obese children.

To date, no study has combined the use of 3D gait analysis of the lower limb and foot, plantar pressures and strength in the same cohort of obese children. Furthermore, given the role of hip extensors, flexors, abductors and adductors as well as ankle plantarflexors and dorsiflexors to daily function such walking, its important to examine strength in these muscles in obese children. Due to the varied use of both ratio scaling and allometric scaling in the strength literature, examining normalisation techniques will determine the most appropriate technique to compare muscular strength in obese children. Moreover, exploring different means of investigating gait and strength in obese children, regarding the use of weight status classification (which thresholds are easily applied back to clinical settings), and the effect of body fat (which provides a direct relationship between adiposity and dependent variables) on gait, plantar pressures and strength. Its important to view all this information together to build a complete picture of the effect of obesity on gait and begin to inform evidenced based care in obese children.

2. Literature review

2.1 Childhood Obesity.

Prevalence of obesity.

Over one third of children in the UK are overweight or obese and this number is currently set to rise (NCMP 2016, Lobstein & Jackson-Leach 2016). The National Child Measurement Programme 2015/16 revealed levels of overweight and obese in 4 to 5 year old children increased from 2014/15 and the number of 10 to 11 year old overweight and obese has increased to higher than any previous year. Similarly, a longitudinal study from 1946 to 2002 of 56,632 participants found a trend for younger children to have increasing BMI (BMI=mass/height^2) whilst predicted BMI trajectories have become steeper in later years (Johnson, Li, Kuh, & Hardy, 2015). However, these conclusions are based upon assumptions that increases in BMI across age groups over time, is a result of increased fat mass.

Definition and measurement of obesity.

Obesity is defined as excess body fat mass, to an extent that health may be adversely affected (Kopelman, Caterson, & Dietz, 2009). There are several ways obesity can be measured. Anthropometry is most common in epidemiology and clinical settings due to its speed and cost effectiveness. Whereas, measurements of adiposity through densitometry are more appropriate to smaller scale studies.

Anthropometric measurements such as height and weight can be used to calculate body mass index (BMI) equation 2.1.1.

2.1.1) $$BMI = \frac{mass\ (kg)}{height(m)^2}$$

During adulthood, anthropometric measurements remain relatively constant except when there is a loss or gain of weight, and therefore the classifications remain relevant despite age. However, boys and girls go through different patterns of growth and maturity at different ages, therefore at any given height, variation in mass will not be attributed to differences in fat mass alone (Flegal, 1993). Therefore, when estimating if a child's BMI is too high, sex and age needs be taken into consideration.

The United Kingdom growth reference data 1990 (UK90) (Cole et al., 1995) includes height and weight measures of 37,000 children from the UK. The data provides a reference population for tracking growth patterns as well as producing distributions of BMI in girls and boys through childhood and adolescence. The population distribution provides an average for a given sex and age whereby a child can be compared to. Current comparisons are defined in terms of centile or Z score (the number of standard deviations above or below the average BMI) where a specific centile or Z score for a specific growth reference will determine overweight and obese (table 2.1.1).

Table 2.1.1. UK90 centile and Z score thresholds and corresponding Department of Health population tracking weight status classification (Wang and Chen (2012).

Centile	Z score	Weight Status
0.2^{nd} – 5^{th}	-3.00 - -1.64	Underweight
15^{th} – 84^{th}	-1.04 – 1.00	Healthy weight
85^{th} – 94^{th}	1.04 -1.64	Overweight
95^{th} – 99.8^{th}	1.64 - 3	Obese

Thresholds were originally pragmatically selected for the purpose of categorising at either a clinical level or population tracking (SACN and RCPCH, 2012). Whilst cut offs coincide with adult cut offs, they have no direct correspondence to adverse health effect of excess mass in children, as limited data exists on long-term and short-term health effects of specific BMI values in children. Given that excess fat mass is linked to health complications, more representative measures of adiposity rather than BMI are required through valid and reliable body composition measures.

The current 'gold standard' for estimates of *in vivo* body composition is the 4-compartment (4-C) model comprising of more than one method to estimate fat, water, mineral and protein (Lohman, 1986). However, this process is a burden for children whilst also being time consuming and expensive. Therefore, there is need for a balance between accuracy and participant comfort when determining body fat from

body composition methods in research settings (Gately et al., 2003; Weber, Leonard, & Zemel, 2012)

Within an obese paediatric population the 4-C model has been compared to 2 compartment models such as DEXA scan, airdisplathemography (ADP) and total body water (Gately et al., 2003). DEXA and ADP with Siri model (body fat mass estimation equation based on adult 2 compartment densities) (Siri, 1961), significantly overestimated body fat percentage. Whilst total body water significantly underestimated body fat percentage (Gately et al., 2003). ADP with Lohman model (child specific density equation) and total body water was not significantly different from 4-C model, and ADP Lohman model had the lowest total error (Gately et al., 2003).

Causes of childhood obesity.

Child obesity is a complex issue and a number of possible risk factors have been identified, including birth weight, rate of growth in infancy, family environment and parental fatness (Gillman, 2008; Yu et al., 2011). Furthermore, children in deprived areas and certain ethnic backgrounds are at a higher risk of obesity (NCMP, 2016). However, despite these predisposing factors, chronic positive energy imbalance ultimately results in excess fat mass. Physical activity can account for 40% of total energy expenditure in children (Ball et al., 2001). However, physical activity levels have only been significantly correlated with percentage body fat in boys, additional factors are thought to influence body fat mass and distribution in girls (Ball et al., 2001). Additionally, greater time spent doing moderate to vigorous physical activity was associated with lower BMI, whilst more time spent in sedentary behaviours such as watching television were significantly linked to higher BMI in boys and girls (Mitchell et al., 2016). Whilst authors have concluded that lower activity leads to greater BMI, Metcalf et al. (2011) demonstrated that higher body fat percentage at age 7 predicted a relative decrease in daily moderate to vigorous intensity at age 10. Suggesting that children with a larger fat mass did less physical activity due to the excess mass. Shultz et al. (2009) suggest obese children may do less physical activity due to musculoskeletal pain and difficulty in carrying excess mass. This causes obese

children to fatigue faster than healthy peers, and suggests a negative cycle of reduced physical activity, increasing fat mass and worsening impact of obesity.

Impact of childhood obesity.

The impact of child obesity can be psychological, social and physical (Pulgarón, 2014). As the prevalence of child obesity has increased, so has obesity related comorbidities such as diabetes and cardiovascular disease. Hundreds of thousands of obese children in the EU are suggested to have type 2 diabetes, impaired glucose tolerance, show indicators of cardiovascular disease and metabolic syndrome, and show signs of early stage liver disorder (Lobstein & Jackson-Leach, 2006). Furthermore, childhood obesity has shown to impact musculoskeletal health and motor function of children (Chan & Chen, 2009; Wearing et al., 2006). Obese children have reduced relative strength, muscular endurance, cardiorespiratory fitness and perform less well in motor tasks (Tsiros et al., 2011). Moreover, child obesity is associated with increased musculoskeletal pain, deviations and inefficiencies in gait, and restriction in general daily activities such as walking and moving from sitting to standing, due to excess mass (Tsiros et al., 2011).

With obesity rates estimated to rise and the increased risk of remaining obese in to adulthood, the physical and health implications as well as the associated cost of treating obesity related comorbidities will also rise (Lobstein & Jackson-Leach, 2016). The cost of treating obesity related illness to the NHS (2006/07) was estimated to be £6.1 billion (Scarborough et al., 2011). Whilst severely obese adults were three times more likely to need social care estimated to cost £352 million (Morgan & Dent, 2010). Currently there are no financial estimates specific to child obesity and whilst no defined terms for costs exist to make year on year comparisons, it seems clear that as obesity rates rise so too will the cost.

Recommendations for child obesity.

Recommendations to tackle obesity focus on the need to stop children becoming obese in the future and to help those who are already obese to work towards a healthy weight (WHO, 2016). WHO (2016) and RCPCH (2015) have made recommendations

on sugar tax, restrictive advertising, promotion of healthy eating, healthy school environment, school programmes and physical activity. Current guidelines for 5 to 18 year olds are a minimum of 60 minutes moderate to vigorous activity every day and vigorous intensity exercise three times a week to promote muscle and bone growth, with emphasis on active transport such as walking to school (RCPCH, 2015 & WHO, 2016). HSCIC (2015) report, only 21% of boys and 16% of girls aged 5 to 15 years are meeting the guidelines and rates fall as children get older (14% boys and 8% girls 13-15yrs).

Despite the critical role physical activity plays in maintaining a healthy body mass or reducing excess mass, it has been suggested that inactivity does not simply lead to adiposity but that adiposity leads to inactivity (Metcalf et al., 2011). Less physical activity due to excess mass, may be a result of greater muscular and cardiovascular demand to move increased mass as well as significant musculoskeletal pain and lower limb dysfunction. These factors feed into a negative cycle of obesity, adverse biomechanical effects can reduce physical activity, and increasing fat mass (figure 2.1.1).

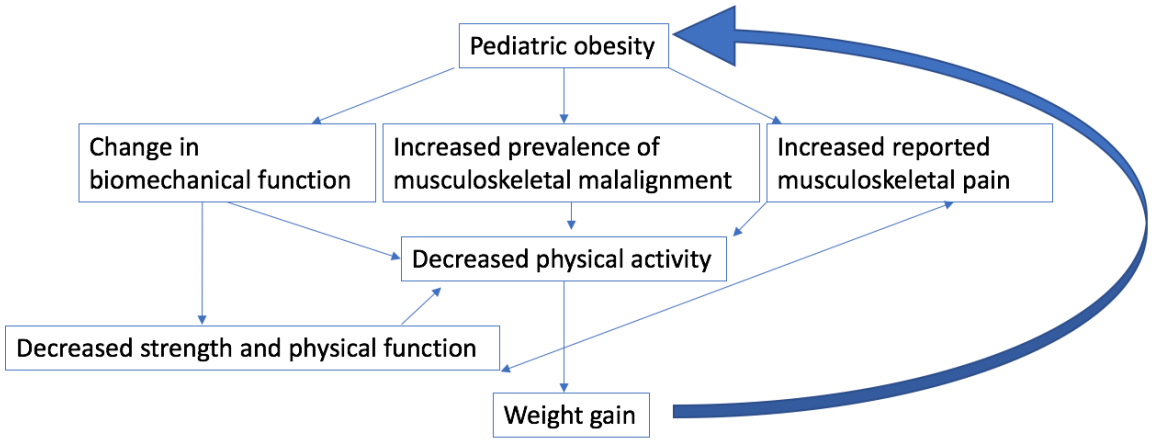

Figure 2.1.1. Schematic of the implications of child obesity on musculoskeletal health and physically activity (Shultz et al., 2009).

2.2 Musculoskeletal effects of obesity

Physical activity and nutrition are essential to healthy musculoskeletal development (Levangie & Norkin, 2005). The formation of bone and joints are dependent on adequate magnitude and direction of loading to promote normal growth (Bechard, Wroe, & Ellis, 2008). Normal development is dependent on loading patterns, however carrying excess mass on an immature skeletal system can create structural damage, malalignment and lead to orthopaedic complications and pain (Shultz et al., 2009; Wearing et al., 2006).

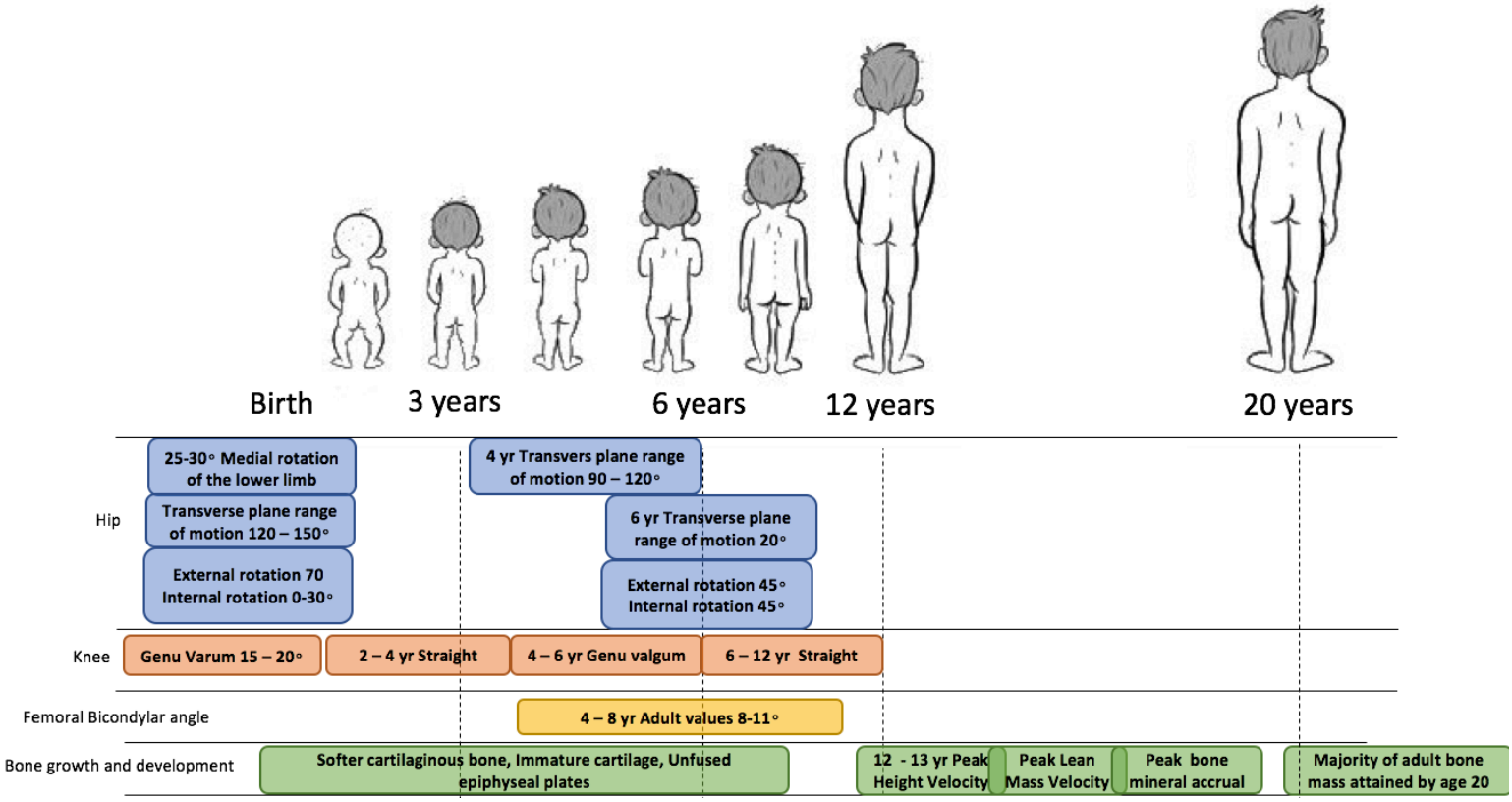

Figure 2.2.1. Key stages of alignment, growth and range of motion of the hip and knee in normal skeletal and joint development from birth to adulthood (Carriero, Zavatsky, Stebbins, Theologis, & Shefelbine, 2009; Levangie & Norkin, 2011; Yates, 2009)

During normal growth from birth to maturity the musculoskeletal system goes through ranges of mobility, alignment and growth (figure 2.2.1). Skeletal tissue adapts to mechanical loads placed upon bones through muscular contraction and gravitational loading (Kohrt, Barry, & Schwartz, 2011). Excessive forces across growth plates in the skeletal system can cause growth inhibition and lead to asymmetries and deformities within the growth physis (Sabharwal & Root, 2012). For obese children, orthopaedic complications generally surround the weight bearing structures of the lower limb including hip, knee, and foot which can promote biomechanical changes and reduce physical activity (De Sá Pinto et al., 2006). Orthopaedic complications effect normal movement patters and lead to greater impairment (Shultz et al, 2009). This is supported by relationships in lower limb malalignment and sedentary behaviour in obese children and lower mobility scale quality of life scores (Shultz, Kagawa, Fink, & Hills, 2014; Taylor et al., 2006).

Hip joint disorders.

Childhood overweight and obesity has been strongly linked to incidence of slipped capital femoral epiphys (SCFE) (Kelsey et al., 2015). SCFE is a change in the anatomic position of the femoral head in relation to the neck and shaft due to disruption of the epiphyseal plate (Wills, 2004). SCFE is most commonly seen during the adolescent growth spurt between the ages of 12 and 15 years (Chan & Chen, 2009). Whilst SCFE is a multifactorial disorder, presence of obesity in children with SCFE is high (81.1%), suggesting carrying excess mass may affect the epiphyseal plate and increase SCFE risk (Benson, Miller, Bosch, & Szalay, 2008; Manoff, Banffy, & Winell, 2005; Murray & Wilson, 2008). Additionally, obese (95^{th} and 93^{rd} percentile for age and sex) adolescents (12-16 years) exhibited significantly less hip anterversion (0.40 ± 13 vs 10.6 ± 8.6) and greater hip abduction (Galbraith et al., 1987; Wills, 2004). These factors combined with increased forces during weight bearing activities such as walking may increase shear and compressive forces across the growth plate. Therefore, child obesity may increase the risk of the SCFE through increased forces and altered alignment.

Knee joint disorder.

Obese children (7 -14 years) have significantly higher rates of genu valgum (55.1% vs 2%,), and genu recurvatum (24.2% vs 2%,)(De Sá Pinto et al., 2006). In addition Taylor et al. (2006) reported significantly greater degrees of valgus alignment in metaphyseal-disphyseal angle (-6.5 ± 4.6 vs -5.2 ± 3.9°). Chronic varus deformity and associated gait deviations in obese children have been shown to increase loading in the medial compartment of the knee, which with sufficient magnitude may alter physeal growth and lead to tibia vara/ blounts disease (Cook et al., 1983; Lerner et al., 2016; Pirpiris et al., 2006). In a study of 102 children being assessed for surgery for blounts disease, 62% had a BMI over the 95th centile, and in those requiring surgery, BMI centile that was significantly greater than those who did not require surgery (90.3 ± 19.5 vs 77.5 ± 22.7) (Pirpiris et al., 2006). Furthermore, Sabharwal et al. (2007) demonstrated a significant relationship between the level of obesity and magnitude of deformity particularly in BMI > 40. Child obesity effects knee alignment, joint loading and puts children at risk of significant lower limb disorder effecting function and that may require surgery.

Foot disorders.

Obese children have altered foot growth patterns, foot morphology and a trend for altered foot structure, particularly in the medial longitudinal arch (Jiménez-Ormeño, Aguado, Delgado-Abellán, Mecerreyes, & Alegre, 2013; Mauch et al., 2008; Villarroya et al., 2009). Pes planus is a term for a lowered or absent medial longitudinal arch, often further characterised as with or without valgus heel, pathological or non-pathological, rigid or flexible and symptomatic or asymptomatic. Prevalence of paediatric pes planus has been strongly linked to child obesity (Bordin, De Giorgi, Mazzocco, & Rigon, 2001; Chan & Chen, 2011; Riddiford-Harland, Steele, & Baur, 2011; Villarroya et al., 2009). Current literature suggests increasing pes planus with increasing excess weight (Stolzman et al., 2015). Pes planus is often measured through static measures such as the arch index, providing little information on the function of foot. However, Sadeghi-Demneh et al. (2016) did demonstrated a significant correlation between flat foot as measured by arch index and BMI in children as well as a significant correlation of BMI and those reporting pain in the feet during

activity. Obese children seem to be at risk for altered foot function, pes planus and associated pain as a result of excess mass.

Joint pain.

Pain in the lower back, hip and knee joints is associated with greater body mass and BMI. Obese children have repeatedly reported musculoskeletal pain. Frequent areas for complaint are the general lower limb, lower back, knees and feet (De Sá Pinto et al., 2006; Stovitz et al., 2008; Taylor et al., 2006). Despite the presence of increased pain with increasing BMI, assumed to be due to the addition of excess fat mass, BMI has shown to be a greater predictor for pain than adiposity (Tsiros et al., 2014). However, these studies did not include any imaging of joints so it is not possible to conclude that pain is solely due to changes in joint structure.

Summary.

Whilst links between overweight and obesity in children and musculoskeletal disorders are clear, the precise causes are yet to be determined. Malalignment and pain significantly reduces physical function particularly in the weight bearing structures of the lower limb, reducing the quality of life of children who are overweight or obese. This could contribute to a continuing cycle of weight gain and reduced physical activity and function (Bout-Tabaku et al., 2013; Smith, Sumar and Dixon, 2014).

2.4 Gait analysis

Normal gait is characterised by phases and events in a full gait cycle (one stride from initial contact of one foot to the following initial contact of the same foot) (figure 2.4.1).

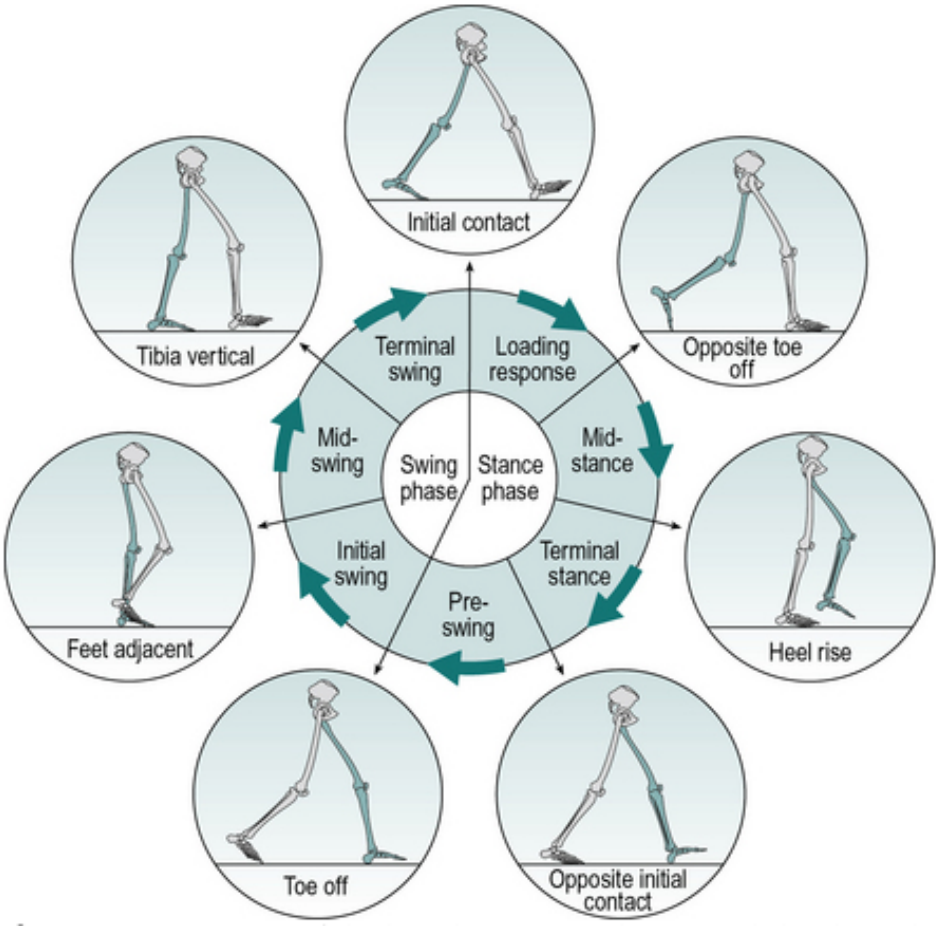

Figure 2.4.1. Full gait cycle phases, gait events and leg position. (Levine, Richards, & Whittle, 2012)

Gait can be described in kinematic and kinetic variables. Kinematic joint movements of one segment relative to another, defined by three orthogonal dimensions. These are usually reported as joint angles and can visually indicate signs of gait pathology. Kinetics describe the forces acting on joints (Levine et al., 2012). The Newton-Euler inverse dynamics method is commonly applied in gait analysis to calculate internal joint moments and powers (Winter, 2009). Moments are reported as Newton meters (Nm) unless normalised to body weight, and are a product of force and moment arm of the joint (Winter, 2009). Within gait analysis joint moments are described as external moments (ground reaction force acting on joints) and internal moments (muscle force

acting on joints). Power is a measure of doing work (Nm·s) and within gait analysis, is calculated as the product of joint moment and joint angular velocity (Winter, 2009). These are described in three orthogonal axes and defined as concentric muscle action of power generation and eccentric muscle action of power absorption (Winter, 2009). Joint powers during gait are often described as phases out lined by Eng & Winter (1995), corresponding to the main power bursts during gait. Joint moments and powers provide information on joint loading, and the ability to produce and control force contributions to the acceleration and control of the centre of mass.

Spatial-Temporal differences.

Obese children and adolescents generally exhibit slower walking velocity, shorter stride (distance between two successive placements of the same foot), step lengths (distance between the placement of one foot with the forward placement of the other foot), and greater step width (medio-lateral distance between the mid-point of each ankle)(table2.4.1) (Dufek et al., 2012; Freedman Silvernail et al., 2013; Hills & Parker, 1992; McGraw, McClenaghan et al., 2000). In addition, obese children spend a greater time in double support (Dufek et al., 2012; Huang et al., 2013), therefore reducing the propulsion period of the gait cycle (Yan et al., 2013). These differences indicate a slower more cautious gait that may reduce time in phases where instability is increased (i.e. single limb support) (Yan et al., 2013). Adaptive mechanisms such as these have been suggested to help control inertial characteristics of excess mass, and may increase frontal plane movement due to greater medial/lateral sway (Dufek et al., 2012; Huang et al., 2013).

Table 2.4.1. Mean ± SD of significant spatiotemporal differences between obese and healthy weight children during gait.

		Walking Velocity	Stance time % of Gait	Double support time % of gait	Swing phase % of cycle	Stance width
Pamukoff et al., 2016	OB	1.09 (1.07,1.17) m·s				
	HW	1.34 (1.27,1.39) m·s				
Shultz et al., 2014	OB	3.6 ± 0.43 km·h				
	HW	4.01 ± 0.46 km·h				
Shultz et al., 2014	OB	3.6 ± 0.4 km·h				
	HW	4.0 ± 0.5 km·h				
Maclean et al., 2016	OB	1.25 ± 0.15m·s				
	HW	1.55 ± 0.18m·s				
Cimolin et al., 2015	OB		62.74 ± 1.71 %			
	HW		58.60 ± 2.66 %			
Freedman Silvernail et al., 2013	OB	1.35 ± 0.13 m·s				
	HW	1.44 ± 0.16 m·s				
Dufek et al., 2012	OB	1.17 ± 0.16 m·s		28.03 ± 3.29	35.96 ± 1.74	11.09 ± 2.99 cm
	HW	1.25 ± 0.17 m·s		23.80 ± 3.13	38.16 ± 1.71	8.59 ± 3.35 cm
Nantel et al., 2006	OB				36.6 ± 3.6	
	HW				39.5 ± 2	

OB- Obese or overweight children, HW- healthy weight children

Gait kinetics and kinematics at the hip, knee and ankle are significantly affected by different walking speeds in sagittal, frontal and transverse planes (Dufek et al., 2012; Lerner et al., 2014; Shultz et al., 2009). Whilst walking velocity should be controlled for, changing the natural walking speed may result in altered walking patterns and

create data that is less applicable to every day function. Therefore, statistical analyses should control for walking velocity.

Effects of obesity on the pelvis during gait.

Pelvic obliquity during walking has been shown to correlate with levels of body fat in children. Thus, as body fat percentage increases range of motion of the pelvis in the frontal plane during gait also increases (Lerner, Shultz, Board, Kung, & Browning, 2014). Pelvic obliquity during single limb stance causes a drop in the centre of the hip joint centre (Lerner et al., 2014). One of the major muscles supporting the torso and controlling the pelvis and hip is the gluteus medius. Gluteus medius force production, relative to lean weight during gait, is positively correlated with body fat percentage in children (Lerner et al., 2014). This suggests obese children are having to generate greater muscular force during gait. It is estimated the gluteus medius works at ~70% of maximal voluntary isometric contraction in normal gait (John, Seth, Schwartz, & Delp, 2012; Rutherford & Hubley-Kozey, 2009). Whilst data does not currently exist on the percent of maximal muscle activation during gait in obese children, they do have an increased mechanical cost of walking per kg of mass, suggesting increased work and activation during gait (Peyrot et al., 2009).

Effects of obesity on the hip during gait.

Carrying excess weight increases the demand of stabilising muscles to prevent collapse of the lower limb during weight bearing (Shultz, D'Hondt, Lenoir, Fink, & Hills, 2014). This is evident from the greater power absorption seen in the hip abductors and external rotators (table 2.4.2) (Shultz et al., 2014). However, despite the reported increased work of hip abductors, evidence suggests this is still inadequate to stabilise the pelvis. This is also apparent in the increased hip adduction angles seen in obese boys during stance (McMillan et al., 2009). As previously seen in healthy adolescent population gait mechanics are sensitive to weakness in the hip abductors (van der Krogt et al., 2012). Working closer to maximum, and with increasing excess mass, to which the muscle needs time to adapt to controlling, the gluteus medius may be very susceptible to weakness or fatigue (Lerner et al., 2014). Whilst some adult studies have not found differences in sagittal hip joint kinematics (DeVita & Hortobagyi, 2003),

both McMillan et al. (2010); and Shultz et al. (2014) found obese children to have significantly decreased hip flexion during initial contact and throughout the stance phase (table 2.4.2). This is coupled with significantly lower hip extension moment at initial contact (McMillan et al., 2010). However, in late stance obese children were shown to have significantly higher hip flexion external moment normalised to body mass (McMillan et al., 2010). Shultz et al. (2009), reported greater hip flexion and extension internal moments throughout stance in obese children (table 2.4.3). During stance obese children moved into hip extension earlier than healthy weight children which brings the force line closer to under the hip joint earlier, which may require less hip extensor strength but also results in hip rotation moment (McMillan et al., 2010). The greater power generation and absorption (table 2.4.4) seen in obese children in the frontal, sagittal planes in all power phases and transverse plane at weight acceptance is considered to be due an increased mass to control and aid in the propelling of the heavier limb (Shultz, et al., 2014; Shultz et al,, 2010).

Table 2.4.2. Significant results of differences in obese and overweight, and healthy weight children joint angles during self-selected speed gait.

		Hip				Knee						Ankle	
		Abduction	Adduction	Extension	Flexion	External rotation	Adduction	Extension	Flexion	Internal rotation	External Rotation	Toe out	Toe in
Gushue et al., 2005	OB								SP 14.5 ± 5.5				
	HW								SP 21.1 ± 5				
Freedman Silvernail et al., 2013	OB						SP 5.3 ± 1.9						
	HW						SP 6.2 ± 1.7						
McMillan et al., 2009	OB						ES -11.96 ± 5.74						
	HW						ES 1.81 ± 5.01						
	OB						LS -6.23 ± 4.33						
	HW						LS 5.16 ± 5.60						
McMillian et al., 2010	OB	IC 4.12 ± 4.37			IC 18.01 ± 10.50				IC 1.38 ± 7.35				
	HW	IC 0.35 ± 2.46			IC 30.37 ± 9.62				IC 7.10 ± 3.41				
	OB	LS 9.55 ± 7.62											
	HW	LS 0.80 ± 3.94											
Shultz et al., 2014	OB		SP 10.80 ± 3.73	SP 4.80 ± 7		SP -2.33 ± 8.27		SP 2.91 ± 3.80		SP 9.06 ± 5.70	SP 10.67 ± 6.14	SP 27.74 ± 8.58	12.63 + 10.36
	HW		SP 5.81 ± 3.49	SP 7.60 ± 5.72		SP 5.22 ± 8.54		SP 6.24 ± 4		SP 13.70 ± 6.36	SP 6.71 ± 4.78	SP 12.41 ± 18.24	4.68 + 18.54

Values depicted are mean peak angle ° ± SD for each group.
OB, obese or overweight children. HW, healthy weight children. SP, Peak throughout stance phase. IC, Peak at initial contact. LS, Peak at late stance.

Table 2.4.3. Joint moment significant differences between obese, overweight and healthy weight children during self-selected speed gait

		Hip						Knee								Ankle					
		Abduction		Extension		Flexion		Abduction		Adduction		Extension		Flexion		Inversion		Dorsiflexion		Plantarflexion	
Gushue et al.,2005	OB							SP	22.5 ± 10.5			SP	25.8 ± 25.6								95 ± 27
	HW							SP	10.8 ± 5.5			SP	16.5 ± 8.4								67.6 ± 17
McMillan et al., 2009	OB	ES	0.50 ± 0.10																		
	HW	ES	0.24 ± 0.07																		
	OB	LS	0.55 ± 0.14																		
	HW	LS	0.27 ± 0.10																		
Shultz et al., 2009	OB	SP	66.3 ± 20.22	SP	65.36 ± 27.80	SP	54.66 ± 27.08	SP	26.24 ± 14.15	SP	9.81 ± 7.22	SP	51.67 ± 26.75	SP	23.65 ± 13.67	SP	7.22 ± 4.41	SP	6.44 ± 6.76*	SP	96.97 ± 33.81
	HW	SP	30.35 ± 10.69	SP	32.58 ± 5	SP	25.12± 7.88	SP	14.11 ± 4.35	SP	2.23 ± 1.41	SP	18.28 ± 8.48	SP	10.34 ± 3.66	SP	2.69 ± 1.83	SP	4.59 ± 3.43*	SP	50.93 ± 16.24
McMillan et al., 2010	OB	ES	0.42 ± 0.12	IC	0.43 ± 0.12	LS	0.37 ± 0.16	MS	0.16 ± 0.06	ES	0.03 ± 0.03			IC	0.19 ± 0.06	LS	0.07 ± 0.03	LS	0.67 ± 0.13		
	HW	ES	0.55 ± 0.13	IC	0.72 ± 0.23	LS	0.24 ± 0.08	MS	0.30 ± 0.09	ES	0.07 ± 0.06			IC	0.28 ± 0.11	LS	0.11 ± 0.02	LS	0.88 ± 0.07		
								LS	0.14 ± 0.06					LS	0.37 ± 0.16						
								LS	0.27 ± 0.09					LS	0.24 ± 0.08						
Lerner at al., 2016	OB									ES	11.9 ± 4.6										
	HW									ES	3.7 ± 1.8										
Lerner at al., 2016	OB					SP	0.33 ± 0.04														
	HW					SP	0.41 ± 0.12														

Gushue et al. (2005); Shultz et al. (2009), & Lerner et al. (2016) reported Internal joint moments (Nm).
McMillan et al. (2009); McMillian et al. (2016); Lerner et al. (2016b), reported external joint moments normalised to body weight (N/kg/m).
*Shultz et al. (2009), Dorsiflexion moment was only significant after body weight correction.
OB, Overweight and obese children. HW, Healthy weight children. SP, Peak throughout stance phase. ES, Peak in early stance. MS, Peak in mid stance
LS, Peak in late stance.

Table 2.4.4. Significant differences in joint powers between obese, overweight and healthy weight children during self-selected speed gait

		Hip				Knee				Ankle
		Abduction	Extension	Flexion	External rotation	Abduction	Extension	Flexion	Internal Rotation	Plantar flexion
Shultz et al., 2014	OB	WA -38.4 ± 16.8	WA 51.9 ± 37.9		WA -21.2 ± 17.3	WA -17.9 ± 14.2	WA -56.5 ± 44.4			
	HW	WA -26.2 ± 92	WA 33.4 ± 26.0		WA -9.5 ±6.3	WA -9.4 ± 6.1	WA -27.2 ± 18.3			P 166.5 ± 73.0
	OB	P 26.1 ± 15.1	P -42.8 ± 28.7	P 59.4 ± 38.6						P 110.1± 29.9
	HW	P 15.2 ± 4.8	P -28.4 ± 16.3	P 37.1 ± 14.3						
Shultz et al., 2010	OB	H1 -38.56 ± 20.54	H1 45.61 ± 19.36			K1 -7.60 ± 6.32	K1 -27.65 ± 24.99		K1 -1.63 ± 1.12	A1 -30.40 ± 13.83
	HW	H1 -12.92 ± 7.72	H1 24.87 ± 10.16			K1 -3.39 ± 5.28	K1 -14.21 ± 5.39		K1 -0.82 ± 1.12	A1 -14.85 ± 7.35
	OB	H2 36.38 ± 10.94		H2 -37.86 ± 18.75			K2 27.57 ± 25.94			A2 141.42 ± 64.83
	HW	H2 19.57 ± 6.77		H2 -20.64 ±9.26			K2 8.47 ± 21.30			A2 80.37 ± 21.63
	OB			H3 53.52 ± 25.19			K3 -100.31 ± 46.39			
	HW			H3 26.85 ± 8.05			K3 -43.64 ± 44.00			
	OB							K4 -63.86 ± 34.09		
	HW							K4 -29.83 ± 31.27		

Values are mean power ± SD for each group.
OB, obese or overweight children. HW, healthy weight children. WA, Peak during weight acceptance. P, propulsion phase. H1-3 Hip power phases 1-3, K1-3 Knee power phases 1-3, A1-2 Ankle power phases 1-2.

Effects of obesity on the knee during gait.

Significant differences in frontal knee kinematics have been found throughout the stance phase (table 2.4.2). Obese children have been found to remain in knee valgus (knee abduction) throughout stance compared to healthy weight controls (McMillan, et al,, 2010; McMillan et al., 2009). In addition, obese children exhibit ~ 2.5° more total frontal plane knee excursion during stance (McMillan et al., 2010; McMillan et al., 2009). Similarly, in a study of obese adolescents, knee adduction was significantly less during self-selected speed (Freedman Silvernail et al., 2013). Frontal knee kinetics show obese children to have significantly larger abduction and adduction internal joint moments (table 2.4.3) (Gushue, et al., 2005; Lerner & Browning, 2016; McMillan et al., 2010; Shultz et al., 2009). Gushue et al. (2005), Lerner & Browning (2016) and Shultz et al. (2009) all reported internal joint moments (Nm) and found obese to have greater adduction and abduction moments (table 2.4.3). Additionally, McMillan et al. (2010) reported external joint moments normalised to body weight and found obese children to exhibit lower external abduction and adduction moment (Nm·kg). However, Freedman Silvernail et al. (2013), reported external moment normalised to fat free mass and height and found no significant difference between healthy weight and obese children knee adduction moment. Additonally, obese children exhibit increased power absorption in the knee extensors at weight acceptance and push off (table 2.4.4) this is thought to offset the increased power generation of the hip and ankle at these times.

Timing differences in peak moment have also been reported. Healthy weight children exhibited two abduction moment peaks in the first half and second half of stance coinciding with frontal knee position, obese children showed adduction moment peaks at initial contact and toe off (McMillan et al., 2009). Greater frontal knee motion and moments increase loading on the medial comprtment of the knee (Gushue et al., 2005; McMillan et al., 2009). Body fat percentage in children has been negatively correlated with average knee flexion angle during stance (Lerner at al, 2014) and repeatedly been found to be reduced in studies comparing obese to non-obese (Gushue et al., 2005; McMillan et al., 2010). Some have suggested this is to allow adequate toe clearance when the contralateral hip joint centre drops (Lerner et al., 2014), whilst

others suggest it is a mechanism to compensate for increased knee loads or joint instability (McMillan et al., 2010). Through mechanical models it has been suggested that greater knee extension allows body weight to be more passively supported, therefore requiring less knee torque (Kuo & Donelan, 2010). This is somewhat supported by findings from Lerner et al. (2014), who reported body fat percentage to be negatively correlated with vasti contributions to acceleration of the centre of mass during walking in children. Some of these differences have been suggested to be due to the extensors unable to control for the excess mass because of relative muscular weakness (McMillan et al., 2010) . Hubley-Kozey & Earl (2000) reported the vastus lateralis and vastus medialis to work at less than 30% and 13% of maximal voluntary isometric contraction (MVIC), respectively, during the stance phase. Therefore, the altered sagittal knee biomechanics may not be due to reported weakness in the knee extensors, but knee extensor weakness may be due to walking with a straighter leg (Lerner et al., 2014).

Effects of obesity on the ankle during gait.

Obesity has shown to have little effect of sagittal ankle kinematics (McMillan et al., 2010; Shultz et al., 2009). However, significantly increased plantarflexion moments and joint powers have been found in obese children during propulsion (table 2.4.3 - 2.4.4) (Shultz et al,, 2014; Shultz et al., 2009). Shultz et al. (2009) found obese children to have significantly greater power generation and absorption in ankle sagittal power phases 1 and 2 respectively but significance did not remain after body weight normalisation. Differences in frontal plane and transverse plane kinematics have also been noted (table 2.4.2). Shultz et al. (2014) reported a greater toe out position in obese children and McMillan et al. (2009) noted significantly less rearfoot eversion in obese boys, which came later in the stance phase (McMillan et al., 2009). When normalised to body weight obese boys had significantly lower inversion moments during stance, with peak moments occurring earlier than in healthy weight boys (McMillan et al., 2009).

Research has shown that increases in body size are not proportionate to increases in the articulating surface area of joints in adults (Ding et al., 2005). This means the

higher absolute peaks moments in obese children may indicate increased stress on the lower limb joints (Shultz et al., 2009). Others authors have suggested that whilst obese children are able to adapt or compensate for altered mechanics in the sagittal plane, they are still unable to compensate or adapt to changes in the frontal plane (Gushue et al., 2005). Furthermore Shultz, et al. (2014) suggested the comparable joint powers between groups after normalising to body mass suggests that body mass is the main factor affecting gait. However, differences in normalisation methods between studies (i.e. body mass, fat mass and height), and mixed methodology, such as differences in mean BMI Z scores in obese groups, walking speeds as well as modelling methods of joint centres, may also contribute mixed results.

Previous studies reviewed above have typically used average peak values to determine differences in obese and non-obese children. However, waveform analysis such as principal component analysis (PCA) provides information on the pattern of movement and not just a comparion of peak data. PCA has been used in gait analysis in children to determine age-related differences and the relationship of body fat to 3D foot motion in gait. Thus far, PCA has not been applied to determine lower limb kinetic and kinematic gait differneces in obese and non-obese children (Chester & Wrigley, 2008; Mahaffey, Morrison, Bassett, et al., 2016). Analysis of the waveform would provide information about altered gait patterns over the entire gait cycle. Addtionally, authors have suggested muscular weakness in relation to the increased demand of carrying excess mass to be responsible for altered biomechanics (Lerner, et al., 2014; McMillan et al., 2010). However, none of the studies reviewed above reported a direct measure of muscular strength to be able to draw direct conclusions on this relationship.

2.3 Foot structure

During gait the foot provides support, balance, shock absorption and force transduction during gait. During normal growth and development of the musculoskeletal system, foot structure adapts to magnitude and direction of loading (Kohrt et al., 2011). However, excessive weight bearing, lower limb malalignment,

altered biomechanics, flexibility and late ossification make the foot vulnerable to deformity such as pes planus.

Foot structure and function can be assessed via anthropometry (foot length width, and arch height), arch index (ratio of the area of midfoot in relation to the whole foot excluding toes), plantar pressures (static or dynamic pressure under arears of the foot), whole foot or foot region 3D motion analysis. Obese children have been shown to have longer (214 ± 15 vs 197 ± 15 mm) and wider feet (87 ± 7 vs 76 ± 6 mm) than non-obese (Riddiford-Harland et al., 2011). Jiménez-Ormeño et al. (2013) studied 1,032 6-12 year old school children and found morphological differences between healthy weight, overweight, and obese groups. Overweight and obese groups showed larger foot dimensions and an altered growth pattern. It is suggested that morphological changes come with structural changes within the foot (Mauch et al., 2008). Additionally, altered loading has been reported in overweight children as young as 1- 2 years old, and it is suggested that adverse effects continue to deteriorate with age and excess mass (Mueller et al., 2016).

The arch index is a common methodology to determine arch height, and therefore presence of flat foot. Obese children have repeatedly been reported to have higher arch index (meaning more of the midfoot was in contact with the ground) than non-obese children (Villarroya, Esquivel, Concepción, Beunafe, Moreno., 2008; Mueller et al., 2016; Yan et al., 2013). Additionally, obese children have exhibited greater foot print angles and chippaux-smirak index (midfoot width divided by forefoot width percentage) (Dowling, Steele, & Baur, 2004). However, little can be concluded about the function of the foot from static foot prints, as the foot is not one rigid segment (Taisa Filippin, de Almeida Bacarin, & Lobo da Costa, 2008). To understand foot function during gait we need to look at the foot dynamically and as individual sections, rather than from static measures and whole foot motion analysis.

Dynamic plantar pressures taken from walking gives information on foot loading during gait. During single support, obese children exhibit higher peak absolute pressure over the whole foot (Da Rocha et al., 2014). However, when regions are compared, obese

children have significantly higher absolute pressure at the forefoot and midfoot (Da Rocha, Bratz, Gubert, De David, & Carpes, 2014). This supports Cousins et al. (2013) who reported greater peak pressures at the lateral heel, midfoot and metatarsophalangeal joints in toes 2-5. After normalisation for body weight, significant differences remained in the midfoot and metatarsophalangeal joints in toes 2-5. Additionally, pressure rate (speed of pressure changes) is increased in obese children in the medial and lateral heel (Yan et al., 2013), which indicates increased loading in the developing foot, increasing the risk of injury, discomfort, and deformity.

Relatively few studies have performed 3D motion capture on the paediatric obese foot, however the relationship of body fat to foot segment motion has been investigated. Mahaffey et al. (2016) found greater midfoot (talus to metatarsals) dorsiflexion in relation to the rearfoot, suggesting a more pronated foot during gait. This is supported by Yan et al. (2013), who found obese children to pronate more during gait, as well have altered pronation between left and right feet suggesting imbalance and asymmetry. Furthermore, increasing body fat mass in children is related to greater rearfoot plantarflexion (Mahaffey, et al., 2016). Similarly Mcmillan, Auman, Collier, and Williams, (2009), reported obese children to have greater peak rearfoot plantarflexion (-7.77 $\pm$ 5.32 vs -4.69 $\pm$ 2.21) in the early stance phase. Increased plantarflexion suggests horizontal positioning of the calcaneus as previously seen in pes planus (Kim & Weinstein, 2000). Further evidence of a lowered medial longitudinal arch in obese children during gait is the increased mid foot eversion seen with increasing body fat (Mahaffey et al., 2016). These factors combined with plantar pressure evidence and anthropometric measures suggest morphological flat foot in obese children. This has suggested to be a result of carrying excess mass, increasing load on weaker muscles or more compliant support structures of the arch (Van Boerum & Sangeorzan, 2003).

To date, no study has combined the use of 3D foot motion analysis, planter pressures and strength in obese children. The combination of these measures would better help explain the function of the foot during gait in obese children by linking the effects of altered foot segment motion, foot loading and relationships to strength. Altered

function and loading of the foot could affect the position of forces further up the kinetic chain increasing the risk of injury, pain and disorder (Villarroya et al., 2008).

2.5 Muscle function.

Obese children perform less well at functional tasks that require movement of body mass against gravity, whilst generally exhibiting equal or greater absolute muscular strength in the upper body (Thivel, Ring-Dimitriou, Weghuber, Frelut, & O'Malley, 2016). Model simulations based on healthy adolescents, show that gait is sensitive to muscular weakness in the hip abductors, ankle plantar flexors and hip flexors, and robust to detecting weaknesses in the hip and knee extensors (van der Krogt et al., 2012). Muscular weakness results in increased activity of weaker and neighbouring muscles to maintain gait causing unbalanced joint moments (van der Krogt et al., 2012).

A recent review of muscle strength and fitness in obese children concluded that in field tests requiring movement of body weight against gravity, such as broad jumps or vertical jumps, obese children performed less well (Thivel et al., 2016). Rose, Burns, and North, (2009) demonstrated a significant correlation between inversion, eversion, plantarflexion and dorsiflexion (as measured by hand held dynamometer) with performance in a 10 m run, long jump and vertical jump. It is expected that the poorer performance in weight bearing functional tasks could translate to poor gait function. However, there is little evidence for the use of functional tasks to asses strength in obese children (Mahaffey, Morrison, Stephensen, & Drechsler, 2016). Tests of isometric and isokinetic strength has however proven to be reliable in healthy weight, and an overweight and obese paediatric population (Old, Drechsler, & Stephensen, 2007).

Studies reporting strength measured using dynamometry in obese children, have generally shown equal or higher absolute muscular torque (table 2.5.1) compared to healthy weight children (Abdelmoula et al., 2012; Blimkie et al.,1989; Garcia-Vicencio et al., 2016; Tsiros et al., 2013). This is thought to be due to a larger muscle mass resulting from a training effect of carrying more mass (Garcia-Vicencio et al., 2016).

When strength data is expressed relative to body weight ($Nm \cdot Kg^{-1}$) there is either no significant difference between obese and non-obese, or obese children are reported to be weaker. Additionally, when knee extensor strength was expressed relative to total lean mass ($Nm \cdot Kg^{-1}$) Abdelmoula et al. (2012) found no significant difference between obese and non-obese children.

Table 2.5.1. Mean ± SD significant differences in strength as measured by isokinetic dynamometer on obese and healthy weight children.

		Isometric Knee extensors	Isokinetic Knee extensors
Abdelmoula et al., 2012	OB	232.1 ± 65.2 Nm	
	HW	176.0 ± 55.1 Nm	
	OB	2.46 ± 0.59 Nm·BM (kg)	
	HW	3.27 ± 0.78 Nm·BM (kg)	
	OB	40.2 ± 9.3 Nm·Thigh lean mass	
	HW	33.0 ± 5.9 Nm·Thigh lean mass	
	OB	57.3 ± 12.8 Nm·Thigh muscle mass	
	HW	46.9 ± 5.9 Nm·Thigh muscle mass	
Tsiros et al., 2013	OB	124.6 (4.6) Nm	101.0 (3.6) Nm
	HW	106.0 (2.9) Nm	88.5 (2.2) Nm
	OB	13.3 (0.4) $Nm \cdot kg^{-0.522}$	8.5 (0.3) $Nm \cdot kg^{-0.578}$
	HW	14.8 (0.4) $Nm \cdot kg^{-0.522}$	10.0 (0.2) $Nm \cdot kg^{-0.578}$
Maffiuletti et al., 2008	OB	193.8 ± 26 Nm	135.4 ± 25.1 Nm
	HW	155.5 ± 31.4 Nm	116.5 ± 23.9 Nm
Blimkie et al., 1989	OB	0.7 ± 0.2 Nm·kg	0.3 ± 0.3 Nm·kg
	HW	0.9 ± 0.3 Nm·kg	1.9 ± 0.4 Nm·kg

Body size is a factor that effects muscle strength and it is generally accepted that bigger is stronger (Garcia-Vicencio et al., 2016). Therefore, to obtain an index of

strength independent to body size, strength values should be divided by body length^2 or area (Jaric, 2002). However, there is data to suggest that muscle strength increases at a disproportionally slower rate than body mass (Jaric, 2002). Therefore, differences in mass, or particularly increased mass due to obesity, may not be directly proportionate to increases in strength. Consequently, the use of allometric scaling is commonly employed. Using allometric scaling Tsiros et al. (2013) found obese children to have significantly weaker knee extensors in isometric and isokinetic tests when scaled to body weight (table 2.5.1). However, when scaled to fat free mass (kg) no differences were observed between groups (4.7 $\pm$ 0.1 vs 46 $\pm$ 0.1 and 2.6 $\pm$ 0.2 vs 2.6 $\pm$ 0.1) (Tsiros et al., 2013). However, When ratio methods are used to normalise to fat free mass (Nm·FFM(kg)$^{-1}$), no significant differences have been found between obese and non-obese children (Abdelmoula et al., 2012; Maffiuletti, Jubeau, Agosti, Col, & Sartorio, 2008). This suggests that when strength is independent of the increased fat free mass, obese children may exhibit lower efficiency to produce force (Thivel et al., 2016). Abdelmoula et al. (2012) measured thigh lean mass and thigh muscle mass via DEXA scan in obese children. It was reported that when isometric knee extensor torque was normalised to thigh lean mas and thigh muscle mass (Nm· thigh lean mass (kg)$^{-1}$ and Nm· thigh muscle mass (kg) $^{-1}$) obese children were significantly stronger (table 2.5.1). This may be due to favourable muscle characteristics as evidenced by Garcia-Vicencio et al. (2016), who reported significantly greater knee extensor pennation angle and anatomical cross sectional area in obese female adolescents, as well as increased voluntary activation levels. However, when comparing two groups who are categorised by significant differences in body fat mass, comparing strength indices that are independent to fat mass negates these differences. Furthermore, when relating strength back to muscle function during weight bearing tasks such as walking, removing confounding factors to gait may make these comparisons less applicable.

Little data currently exists on direct measures of strength in the obese paediatric population in muscle groups other than the knee extensors. Given the contribution of hip and ankle musculature to the acceleration of the centre of mass and support in normal gait it is valuable to examine isometric and isokinetic muscle strength and

power in a range of lower limb muscle groups. In addition, the most effective normalisation methods should be explored to investigate functional differences in obese and non-obese children.

2.6 Summary

Previous literature has shown obese children to be at high risk of developing musculoskeletal disorders, pain, and impaired motor function perpetuating a negative cycle of reduced physical activity, excess weight gain, and poor motor function and quality of life. Additionally, the adverse effects of child obesity on the lower limb and foot during gait such as higher peak joint moments, malalignment and altered kinematics have been demonstrated. Given that walking is an integral daily motor function and an advised modality of exercise in children, the unfavourable changes in strength and joint loading currently highlighted in the literature warrants further investigation. The literature review has highlighted several areas that require further investigation.

Reported joint angle, moment and power peaks describe gait parameters and highlight peak differences between obese and non-obese children at singular points of the cycle. Factor reduction techniques will allow a high dimensional dataset to be reduced to low dimensional features that explain variations in the waveforms of lower limb biomechanics in obese children across the gait cycle. Furthermore, whilst group comparisons based upon BMI Z scores provide clinically relevant data, investigating the relationship of gait parameters and body fat would provide information on the direct impact of increasing fat mass on gait. There is limited data on the dynamic plantar pressures under different regions of the foot. Furthermore, plantar pressures have not been examined concurrently with 3D foot and lower limb gait analysis.

Additionally, there is limited information on lower limb strength in obese children. Current data mainly surrounds the knee extensors; given the role of hip extensors, flexors, abductors and adductors as well as ankle plantarflexors and dorsiflexers to motor tasks such walking, these areas warrant investigation. Furthermore, the use of normalisation methods to draw conclusions on strength differences between obese

and non-obese children are varied. Ratio scaling, and allometric scaling to body mass, fat mass and fat free mass have been reported in the literature. However, further discussion is required to determine appropriate techniques when comparing muscular strength to determine muscle function in obese children population.

Whilst there is information on foot loading, foot kinematics, lower limb kinematics and some strength data these variables have not been measured together in the same cohort to begin to investigate the interaction between these variables in obese children. Additionally, whilst these factors require comparisons between classified groups of obese and non-obese children to be able to refer to a specific population and highlight risks, the role of body fat on these parameters should also be examined. Thereby highlighting the effect of increased body fat on gait and strength parameters.

Two experimental studies aim to investigate the areas highlighted above. First, compare differences of lower limb and foot segment kinematics and kinetics, foot loading and strength between obese and non-obese children. This will provide information from over the gait cycle and in lower strength that can be related back to clinically relevant population. Additionally, study one aims to investigate normalisation techniques of isometric, isokinetic strength and power, of the hip, knee, and ankle to determine appropriate techniques to inform future research. The second study aims to investigates the relationship of body fat to gait, foot loading, and lower limb strength in children. This will provide information on the direct impact of increased body fat on motor function in children.

3. General methodology

3.1 Participants.

Sixty-eight children 7 to 11 years (36 male, 32 female) were recruited from local schools and sports clubs in the London borough of Richmond. Participants were excluded if they had any medical condition or injury affecting neuromuscular or orthopaedic integrity resulting in altered foot posture or gait disturbance. Participants attended the lab on one occasion, parental or guardian consent was obtained for each participant. Ethical approval was provided from St Mary's University Ethics Committee.

Participants were categorised into healthy weight, overweight and obese (overweight and obese participants were then grouped together to make the OWB group) by age and sex specific BMI Z score based on UK90 reference curves Department of Health thresholds (Cole et al., 1995) using a Microsoft Excel macro developed for use with this growth reference (Child Growth Foundation, Chiswick, UK). Body mass index (BMI) calculation:

$$BMI = mass\ (kg) \cdot height\ (m)^2$$

Body mass was measured to the nearest 0.1 kg (Model 700, Hamburg, Germany) and height measured barefoot to the nearest 0.1 cm. Body density estimated from age and body volume used to determine fat mass and fat free mass. Body volume was measured using ADP (BOD POD, Life Measurement, Inc, Concord, CA, USA). System calibration according to manufacturer guidelines was completed before each trial. All children wore tight swimwear and a swimming cap. Once seated in the chamber, children were asked to remain still and continue normal tidal breathing. Two body volume measurements were taken. If these first and second measurements were inconsistent (over 5% difference) a third trial was taken and the two closest volume values were taken for further analysis.

Raw body volume was corrected for isothermal air in lungs and skin surface (equation 3.1) (Haycock, Schwartz, 1978).

3.1) $$Surface\ area = mass(kg)^{0.5378} \times height\ (m)^{0.3964} \times 0.024265$$

Thoracic gas volumes (TGV) were estimated from gender and child specific equations (Fields, Cole, Chinn, Jones, White & Preece, 2004) (equation 3.2)

3.2) $$Thoracic\ gas\ volume = \ 0.00056H^2 \ - 0.02442H \pm 8.15194$$

Where H is height in cm. Corrected body volumes were converted to body percentages using age- and gender- specific equations (equation 3.3) (Lohman, 1989).

3.3) $$Body\ fat\ \% = 100\left[\left(\frac{Constant1}{Body\ Density}\right)\right] - Constant\ 2$$

3.2 General procedures

Gait analysis.

A fourteen camera Vicon Nexus system (Vicon Motion Systems Ltd, Oxford, UK) captured the motion of reflective markers attached to each subject's lower limbs at 200 Hz. Two floor mounted force plates (Kistler 9287CA Force Platform, Kistler Instruments Ltd. Hampshire, UK) recorded ground reaction forces during gait trials at 1000 Hz. Both the cameras and force plates were calibrated before each testing session. Eleven reflective markers (9 mm) on a rigid base were placed on the skin of the foot to assess movement in rearfoot, midfoot, and forefoot (figure 3.2.1). Rigid cluster markers to track the pelvis, thigh and shank were strapped to posterior pelvis, mid lateral thigh and mid lateral shank. Nine virtual markers (anterior and posterior superior illiac spine left and right, greater trochanter, lateral knee, medial knee, head of the femur, tibial tuberosity, medial and lateral malleolus) created by spring-activated instrumented pointer device (C-motion. Inc., ON, Canada) in static trials by the same assessor for each participant. The reflective markers and virtual markers were used to create lower limb and foot models based on modified Istituto Ortopedico Rizzoli (IOR) models in Visual 3D (figure 3.2.1) (Leardini et al., 2007). Foot pressure loading was measured concurrently using a pedobaragraph (RSScan 0.5 USB2 Plate (150Hz)

Rs Scan. Ipswich, UK) mounted on top of the force plate, with surrounding flooring built up to create a level surface. Children were asked to walk barefoot at a self-selected walking speed from end to end over a 7.5 m walkway, allowing 4 steps before contacting the force plate. The average of three clean trials were taken for further analysis. Clean trials consisted of having a full gait cycle (heel strike to heel strike on the dominant foot), where the participant did not aim for the force plate, the foot was not partially off the force plate and participants walked in a straight line.

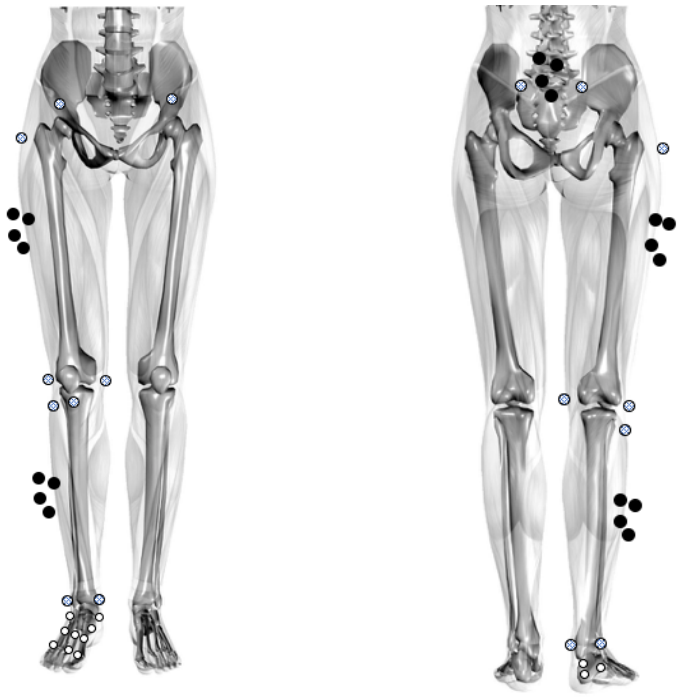

Figure 3.2.1. Cluster and marker placement for lower limb plug in gait and 3D foot model.

● Cluster marker (pelvis, thigh and shank), ○ Foot markers (metatarsal head 1,2 and 5, metatarsal base 1, 2 and 5, navicular tuberosity, peroneal tubercle, sustentaculum tali, calcaneus x2), ◎ Virtual markers (anterior superior iliac spice, posterior superior iliac spine, greater trochanter, medial knee, lateral knee, medial malleolus and lateral malleolus)

The modified Istituto Ortopedico Rizzoli (IOR) and 3D foot model (Leardini et al., 2007) were applied in Visual 3D professional V6 (C-motion, Inc., ON, Canada) to movement trials using static and pointer trials. Pelvic angle was calculated relative to a virtual lab that changes direction, dependent on the walking direction of participants. Joint angles, internal moments and powers for the hip, knee and ankle joints in all three-orthogonal axis were calculated. Segment angles were calculated for the calcaneus relative to the shank, the mid-foot relative to the calcaneus and the metatarsals relative

to the midfoot in all the orthogonal axis. All data were filtered (low pass 5 Hz), normalised to 100 data points and averaged over three trials for each participant.

Plantar pressures.

Foot axis angle, medio-lateral centre of pressure excursion (from the midline), medio-lateral centre of pressure velocity, forefoot, mid-foot and rear foot contact area and plantar pressures were extracted from pedabaragraph data from three clean trails. Masking of the foot regions was automatically completed by RS Foot Scan version 7. Total force of toe 1, toe 2-5 and met 1,2,3,4 and 5 were divided by the total area of the same regions to calculate forefoot pressure.

Strength assessment.

Isometric and isokinetic strength were measured using isokinetic dynamometry (Cybex II, CSMI, Saughton, USA). Warm up and familiarisation was completed in each test, consisting of ramped up effort concentric contraction until consistent, maximal effort results were obtained. Standardised positional set up was used and then adjusted to each participant to assure alignment of joint axis (table 3.2.1). Stabilisation straps were placed over the limbs to reduce unwanted movement. Two isometric contractions were performed and held for 5 seconds. Participants were instructed to push as hard as they could and were given verbal encouragement through out the contraction. If trials differed by >10% an additional trial was performed.

Table 3.2.1. Summary of isometric testing muscle group, joint position angle (º) and isokinetic dynamometer set up position.

Muscle group	Joint Position (º)	Position
Ankle Dorsiflexion	90° foot-tibia	Supine
Ankle plantarflexion	90° foot-tibia	Supine
Knee Extension	60° (0° being full extension)	Seated
Knee Flexion	30° (0° being full extension)	Seated
Hip Flexion	30°	Supine
Hip Extension	60°	Supine
Hip Abduction	neutral	Side lying
Hip adduction	20°	Side lying

Isokinetic trials were completed in the same position as isometric trials. Isokinetic movements were performed within each participant's own range of motion. Each extension and flexion contraction was performed three times starting from an extended joint position. Participants were instructed to push and pull against the lever arm as hard and fast as they could, and verbal encouragement was given throughout. Isokinetic velocity for plantarflexion and dorsiflexion was set at 30°/s, and extension and flexion of the knee and hip was set at 60°/s. An average peak from 3 trials was taken for each for each isometric and isokinetic joint torque and filtered using a 5 Hz zero-lag low-pass butterworth filter. Power was calculated for isokinetic trials using the calculated torque divided by angular velocity (Iossifidou & Baltzopoulos, 2000).

3.3 Data analysis

Principal component analysis was used as a form of dimension reduction to minimise the kinematic, kinetic and plantar pressure waveform outputs into component scores based on areas of similar variation over the gait cycle. Principal component analysis of gait variables and foot pressures through the gait cycle was completed using SPSS version 22 for each joint angle, moment and power for each axis. Each variable (n=57) waveform was entered as a 68 x 100 matrix (subjects x time points). Varimax

orthogonal rotation was used to produce components that maximally explain variability in the original waveform. Component regression scores were only taken for further analysis if they explained >10% variance and areas of the waveform were selected for each component if they had a rotated component matrix value >0.722 or <-0.722. Summary of principal components for lower limb gait, 3D foot segments and plantar pressures are in tables 3.3.1 – 3.3.3.

Strength.

Each torque and power variable was normalised to body mass (kg), body fat %, fat mass (kg) and fat free mass (kg), using ratio standard equation 3.3.1. Each variable was allometrically scaled to body mass (kg), body fat %, fat mass (kg) and fat free mass (kg) using the equation 3.3.2.

3.3.1) $$ratio\ scaled\ strength = \frac{measured\ torque\ or\ power}{mass^1}$$

3.3.2) $$Allomtric\ scaled\ strength = \frac{measured\ torque\ or\ power}{mass^b}$$

Where mass represents either body mass, body fat percentage, fat mass or fat free mass and *b* is the lower limb joint specific power from Wren & Engsberg (2007).

Table 3.3.1. Summary of principle component analysis showing principle component for each lower limb variable, the percent variance explained by the component and percent of the gait cycle the component refers to.

	PC1		PC2		PC3		PC4		PC5	
	% variance explained	% gait cycle	% variance explained	% gait cycle	% variance explained	% gait cycle	% variance explained	% gait cycle	% variance explained	% gait cycle
Pelvis Frontal	99.99									
Pelvis Transverse	99.99									
Pelvis Sagittal	99.98									
Ankle Frontal	40.11	12-50	23.68	63-81	16.21	92 -100	11.14	54 -61		
Ankle Transverse	42.65	4-26	30.357	47-56						
Ankle Sagittal	22.821	82-100	21.257	7-26	18.190	29-44	17.303	62-77	13.698	49-59
Ankle Moment Frontal	29.369	29-54	24.927	1-23	24.546	61-86	7.770	89-95	4.700	98-100
Ankle Moment Transverse	31.246	25-55	27.602	62-88	20.846	2-20	9.143	89-98		
Ankle Moment Sagittal	28.344	56-87	18.907	9-26	15.870	28-42	11.728	44-54		
Ankle Power Frontal	17.816	63-78	11.426	30-39						
Ankle Power Transverse	39.789	20-28	13.697	39-52						
Ankle Power Sagittal	19.137	60-82	15.354	24-36	11.056	6-16	10.764	40-48		
Knee Angle Frontal	35.309	23-52	23.504	1-5	22.438	58-77	12.194	79-86		
Knee Angle Transverse	36.294	1-11	34.344	37-55	27.047	67-77				
Knee Angle Sagittal	32.497	1-29	20.208	35-52	17.371	73-87	16.783	55-69		
Knee Moment Frontal	44.723	6-55	22.185	62-84	11.849	88-97				
Knee Moment Transverse	31.221	28-56	23.727	1-25	17.115	75-87				
Knee Moment Sagittal	18.990	39-57	18.917	5-23	14.453	73-86	13.081	25-36		
Knee Power Frontal	14.584	70-84	10.475	42-49						
Knee Power Transverse	19.577	77-99								
Knee Power Sagittal	15.285	6-23	13.685	47-58	10.469	60-68				
Hip Angle Frontal	43.865	7-51	38.476	64-100	12.282	55-61				
Hip Angle Transverse	35.362	29-58	35.219	80-6	14.022	63-70				
Hip Angle Sagittal	41.56	1-27	32.7	34-59	24.98	63-79				
Hip Moment Frontal	42.464	6-50	20.421	63-82						
Hip Moment Transverse	31.863	5-38	22.369	43-60	19.968	66-81	10.666	85-92		
Hip Moment Sagittal	26.573	4-30	26.248	32-55	14.637	77-90	11.176	61-70		
Hip Power Frontal	10.351	52-59								
Hip Power Transverse	15.900	35-63	14.67	13-21	11.602	79-87				
Hip Power Sagittal	20.561	9-28	16.647	33-46						

Table 3.3.2. Summary of principle component analysis showing principle component for foot segment, the percent variance explained by the component and percent of the gait cycle the component refers to

		PC1		PC2		PC3		PC4	
		% of variance	% gait cycle	% of Variance	% gait cycle	% of Variance	% gait cycle	% of Variance	% gait cycle
Calcaneus to Mid foot	Frontal	49.23	51-100	48.786	5-47				
	Transverse	54.52	1-50	44.663	53-100				
	Sagittal	39.91	11-36	31.092	48-60				
Midfoot to Metatarsals	Frontal	98.41							
	Transverse	98.69							
	Sagittal	98.76							
Shank to Calcaneus	Frontal	35.38	19-49	25.303	56-69	21.137	91-99	15.952	7-11
	Transverse	98.53							
	Sagittal	33.78	5-30	27.48	42-60	20.877	87-97	14.207	65-70

Table 3.3.3. Summary of principle component analysis showing principle component for foot segment plantar pressure, the percent variance explained by the component and percent of the gait cycle the component refers to

	PC1		PC2		PC3	
	% of Variance	% of gait cycle	% of Variance	% of gait cycle	% of Variance	% of gait cycle
Forefoot	37.37	12-48	34.38	55-88	11.84	91-100
Midfoot	41.39	11-51	39.88	58-97		
Rearfoot	34.30	7-49	25.68	77-100	33.88	53-71

4. Comparison of gait kinetic and kinematics, plantar pressure, strength and strength normalisation methods in obese and healthy weight children aged 7 to 11 years.

4.1 Introduction

Childhood obesity is associated with significant metabolic and physiological comorbidities on an increasing global scale (Lobstein & Jackson-Leach, 2016; Wills, 2004). Childhood obesity is associated with musculoskeletal problems such as lower back and lower limb pain, slipped capital femoral epiphysis, Blounts disease and, pes planus (Tsiros et al., 2011; Wearing et al., 2006; Wills, 2004). Reduced physical activity has also been linked to child obesity due to these associated musculoskeletal disorders (Shultz et al., 2009). This can then lead to a cycle of weight increase, further reductions in physical activity and altered biomechanical function.

Physical activity is advised to reduce and prevent child obesity, and in particular walking is recommended (Shultz, Browning, Schutz, Maffeis, & Hills, 2011). However, the gait of obese children is significantly effected by carrying excess weight. Data comparing mean peak kinetic and kinematic values in obese and non-obese children has shown a straighter more upright position and greater peak joint moments and powers from the hip, knee, and ankle (Freedman Silvernail et al., 2013; Gushue et al., 2005; Lerner et al., 2016; McMillan et al., 2010; Shultz, et al., 2014; Shultz et al., 2010, 2009). Whilst kinetics and kinematics are also significantly effected by selected walking velocity, whereby obese children have shown to have reduce walking speeds (Dufek et al., 2012; Lerner et al., 2014; Shultz, et al., 2009). Furthermore, peak plantar pressures in obese children during standing and gait have been shown to be increased (Da Rocha et al., 2014).

The increased joint loads and plantar pressure indicates obese children are at greater risk of pain discomfort and joint dysfunction during walking. However, previously reported differences between obese and non-obese represent amplitudes of singular points of the gait cycle, and not the pattern throughout the whole cycle. Waveform reduction techniques such as principal component analysis, previously used to analyse gait in children (Chester & Wrigley, 2008; Mahaffey, Morrison, Bassett, Drechsler, & Cramp, 2016), would allow analysis of the entire gait waveform (Chau, 2001). Using principal component analysis, Mahaffey, et al. (2016), found body fat percentage to be significantly related to sagittal and transverse motion between the shank and calcaneus and the calcaneus and the mid foot. Whilst these results show a significant effect of fat mass on the function of the foot, these results could be more clinically relevant by applying BMI cut-offs and comparing between groups.

It has been suggested that gait differences in the lower limb and foot may be due to relative muscle weakness. Currently, there is little data on muscle groups of the lower limb other than knee extensors that have major roles in gait such as plantar flexors, hip flexors, extensors and abductors. Whilst is it has been demonstrated that obese children are generally stronger in absolute values (Abdelmoula et al., 2012; Maffiuletti et al., 2008; Tsiros et al., 2013), they are weaker when strength is expressed relative to body weight. Scaling removes the effect of a covariate such as muscle mass on strength measures and can identify the extent to which differences can be attributed to size or mass (Payton & Bartlett, 2008). However, the relationship between body size and strength may not be directly proportionate so the application of allometric scaling has been utilised (Wren & Engsberg, 2007). Wren and Engsberg (2007) reported a systematic approach to allometric scaling in non-disabled children and derived lower limb specific equations for normalising to mass. Standardised scaling techniques would make findings more comparable across studies.

Lower limb and foot gait analysis, plantar pressures, and strength have not been investigated together in the same cohort of obese children. These measures combined will provide a multifaceted picture of the effects of child obesity on gait function and

inform current approaches to rehabilitation and implementation of physical activity to manage obesity in children.

Aims

1) To compare the kinematics and kinetics of the lower limb, kinematics of the foot and plantar pressures throughout the gait cycle in healthy weight and obese children aged 7 to 11 years.
2) To assess normalisation techniques of torque data for comparing isometric and isokinetic strength in the ankle, knee and hip musculature between obese and healthy weight children aged 7 to 11 years.

4.2 Methods

Twenty-eight overweight and obese participants were matched by gender, age and height to twenty-six healthy weight children. Each participant completed anthropometric measures, including body composition analysis and weight status classified by BMI Z score, using Department of Health population tracking thresholds. Gait analysis of the pelvis, lower limb and foot segments, and plantar pressures were collected simultaneously. Participants completed isometric and isokinetic strength assessment of the hip, knee, and ankle on an isokinetic dynamometer. See general methods for details.

4.3 Data Analysis

All statistical analysis was completed using SPSS version 22. All data was tested for normality using Shapiro-Wilks. Independent sample t-tests (mann-whitney U test for non-parametric data) were performed to compare obese and healthy weight group characteristics.

Strength.

To examine the effectiveness of normalisation methods a correlation between absolute torque and power and scaled torque values was performed to determine if the covariate (body mass, body fat percentage, fat mass and fat free mass) had been

effectively removed by the scaling method (ratio or allometric) (Payton & Bartlett, 2008). An ANOVA (kruskal-wallis one way analysis of variance for non-parametric data) was then used to determine differences between obese and healthy weight groups in all absolute, ratio scaled and allometrically scaled isometric and isokinetic torque and power.

Gait analysis.

Regressions scores for each principal component derived from the PCA were compared between OWB and HW groups using a MANCOVA with walking velocity set as the covariate to control for the effect of walking speed on gait biomechanics (Dufek et al., 2012; Lerner et al., 2014). Where there was a significant effect of weight status with or without walking velocity, the interaction on the regression score for individual principal component was reported. When the MANCOVA result determined no significant effect of weight status on the gait variable no further analysis was performed.

4.4 Results

There were no statistically significant differences in age ($t(54)= -.030$, $p=0.977$) or height ($t(54)=1.639$, $p=0.107$) between OWB and HW group. The OWB had significantly higher BMI Z score ($t(55)= -12.121$, $p=0.000$), body fat % ($t(54)=-9.621$, $p=0.000$), fat mass (kg) ($t(54)=-8.829$, $p=0.00$), and fat free mass (kg) ($t(54)=-2.813$, $p=0.007$) compared to the HW group (table 4.4.1).

Table 4.4.1. Age, gender, height, body mass (kg), BMI Z score, body fat percentage, fat mass (kg) and fat free mass(kg) (mean ± SD) of obese and healthy participants grouped by gender, age and height. Significance value of comparisons between obese and healthy weight groups.

	OWB	HW	p value
n	28	28	
Gender	50% Female	50% Female	
Age (years)	9.17 ± 0.91	9.16 ± 1.07	0.977
Height (m)	1.34 ± 0.07	1.37 ± 0.06	0.107
Weight (kg)	41.13 ± 6.5	29.46 ± 4.4	0.000
BMI Z score	2.19 ± 0.8	-0.62 ± 0.9	0.000
BF%	34.50 ± 8.23	16.27 ± 5.62	0.000
FM (kg)	14.67 ± 5.38	4.9 ± 2.22	0.000
FFM (kg)	26.87 ± 3.12	24.53 ± 3.05	0.007
Walking Velocity (m/s)	1.32 ± 0.22	1.26 ± 0.15	0.340

Gait.

There was no significant effect of weight status on lower limb kinematics. Table 4.14 shows the significant lower limb kinetic findings. There was a significant effect of weight status on sagittal ankle moment as well as a significant effect on walking velocity, however when the weight status was corrected for walking velocity sagittal ankle moment was no longer significant between groups. There was a significant difference between OWB and HW groups in frontal knee moment and power, furthermore walking velocity had a significant effect on frontal knee power, significance of weight status remained when corrected for walking velocity.

Table 4.4.2. Summary of MANCOVA results of lower limb kinetics, showing p value of model weight status as fixed factor and walking velocity as covariate.

		Model					
		Status		Walking velocity		Status*Walking velocity	
Ankle Moment	Frontal	0.226		0.193		0.189	
	Transverse	0.253		0.312		0.337	
	Sagittal	0.03	*	0.023	*	0.77	
Ankle Power	Frontal	0.282		0.187		0.292	
	Transverse	0.072		0.213		0.088	
	Sagittal	0.286		0.002	*	0.383	
Knee Moment	Frontal	0.189		0.134		0.126	
	Transverse	0.429		0.804		0.434	
	Sagittal	0.008	*	0.028	*	0.008	*
Knee Power	Frontal	0.033	*	0.234		0.048	*
	Transverse	0.947		0.260		0.913	
	Sagittal	0.579		0.021	*	0.578	
Hip Moment	Frontal	0.621		0.142		0.661	
	Transverse	0.265		0.732		0.333	
	Sagittal	0.573		0.000	*	0.506	
Hip Power	Frontal	0.252		0.139		0.262	
	Transverse	0.726		0.085		0.816	
	Sagittal	0.837		0.001	*	0.836	

Table 4.4.3. Post hoc analysis of significant MANCOVA results for lower limb kinetics, mean ± SD regression score from principal component analysis for OBW and HW groups, significance value of status, walking velocity or status corrected for walking velocity.

		Regression score		Model					
				Status		Walking velocity		Status*Velocity	
		OBW	HW	p		p		p	
Knee Moment Sagittal	PC1	-0.22 ± 1.13	0.02 ± 0.63	0.238		0.168		0.258	
	PC2	-0.01 ± 0.87	-0.03 ± 1.00	0.012		0.024	*	0.011	*
	PC3	0.01 ± 1.41	-0.03 ± 0.73	0.015	*	0.236		0.017	*
	PC4	0.03 ± 1.13	0.06 ± 0.92	0.549	*	0.137		0.497	
Knee Power Frontal	PC1	-0.25 ± 1.56	0.15 ± 0.23	0.033	*	-		-	
	PC2	0.29 ±0.93	-0.15 ± 0.60	0.137		-		-	

A significant effect of walking velocity was found on sagittal ankle moment at 56-87 %, sagittal ankle power at 40-48%, sagittal knee power at 47-58%, sagittal hip moment 32-55%, sagittal hip power at 33-46 % of the gait cycle. As walking velocity increased, ankle and hip moments and powers increased, whilst knee moment decreased. There was a significant difference in sagittal ankle moment between OBW and HW groups. Walking velocity was also significantly affected sagittal ankle moment and, when this was account for, significant differences between groups was lost. Weight status had a significant effect on sagittal knee moment at 5-23% and 73-86 % of the gait cycle even after correction for the significant effect of walking velocity, respectively (figure 4.1). Additionally, there was a significant difference of groups in frontal knee power at 70-84% of the gait cycle (figure 4.4.1).

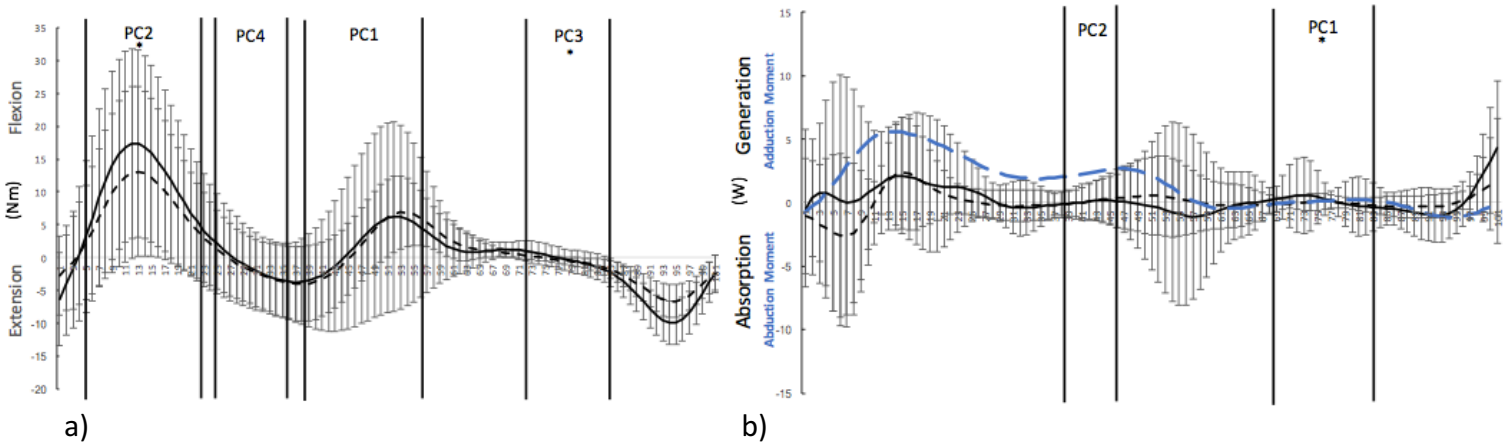

Figure 4.4.1. Mean ± SD sagittal knee moment (a) and frontal knee power (b) for OWB (solid black line) and HW (black dashed line), mean moment (blue dashed line) over the gait cycle. * denotes significant ($p<0.05$) difference between group regression scores for principal component.

OWB had significantly greater ($F(1,54)=7.001$, $p=0.011$) knee flexion moments in PC2 and moved to a significantly greater ($F(1,54)=6.071$, $p=0.017$) knee extensor moment at PC3. OWB generated significantly more ($F(1,54)=4.025$, $p=0.033$) knee adduction power absorption at PC1 whilst HW generally exhibited low power absorption of the adductors at PC1. No significant difference between OWB and HW groups were found in foot segment motion.

Plantar pressures.

There was no significant effect of weight status on forefoot ($p=0.233$), midfoot ($p=0.245$), or rearfoot ($p=0.172$) plantar pressures. Medio-lateral centre of pressure excursion was significantly greater in OWB ($F(1,54)=5.681$, $p=0.021$) (table 4.4.4).

Table 4.4.4. Foot axis angle, centre of pressure total excursion (mm), centre of pressure velocity (m/s), centre of pressure root mean sq, forefoot, midfoot and rearfoot total contact area (cm^2) and significance value of the compared means of healthy weight and obese groups.

	HW	OWB	*p*
Axis Angle	5.62 ± 6.59	4.59 ± 11.68	0.831
CoP M/L Total Excursion (mm)	45.83 ± 9.27	52.51 ± 11.59	0.033*
CoP M/L velocity (m/s)	0.08 ± 0.02	0.09 ± 0.02	0.137
CoP M/L root mean sq	0.15 ± 0.22	0.09 ± 0.02	0.589
Forefoot contact area (cm^2)	56.42 ± 53.26	73.59 ± 68.16	0.235
Midfoot contact area (cm^2)	70.5 ± 28.27	87.43 ± 36.91	0.059
Rearfoot contact area (cm^2)	68.33 ± 21.87	74.69 ± 28.23	0.171

Effectiveness of strength normalisation.

When ratio scaled to body weight, all torque and power values showed a significant and positive correlation with absolute torque and strength. When allometric scaling was applied for body weight all values except isometric ankle and knee torque remained significantly correlated with absolute strength. Strong and significant correlations remained in ratio scaled remove body fat percentage, fat mass or fat free mass. Allometric scaling to body fat percentage, fat mass and fat free mass generated non-significant and weak correlations with absolute values (table 4.4.5)

Table 4.4.5. R^2 values of correlation between absolute isometric and isokinetic torque, and power with ratio scaled and allometric scaled strength values.

			Ratio scaled to BM	Allometric scalling to BM	Ratio scaled to BF%	Allometric scalling to BF%	Ratio scaled to FM	Allometric scalling to FM	Ratio scaled to FFM	Allometric scalling to FFM
Isometric	Ankle	Dorsiflexion	.856**	.079	159	-.017	.060	-.021	0.128	.119
		Planterflexion	.710**	-.101	-.164	.044	-.255	.082	0.113	.073
	Knee	Extention	.523**	-.046	.127	.082	-.054	.023	.867**	.124
		Flexion	.631**	.053	.133	.091	-.037	.069	.886**	.173
	Hip	Flexion	.843**	.819**	.561**	-.086	.377**	-.077	.963**	-.077
		Extention	.828**	.779**	.505**	-.181	.267*	-.185	.955**	-.003
		Abduction	.877**	.786**	.528**	-.002	.381**	-.005	.947**	-.007
		Adduction	.886**	.784**	.495**	.13	.310*	.105	.958**	.034
Isokinetic	Ankle	Dorsiflexion	.694**	.313*	.18	-.002	-.086	.027	.906**	-.231
		Planterflexion	.838**	.663**	.436**	.072	.285*	.067	.934**	.193
	Knee	Flexion	.708**	.500**	.187	.102	-.251	.073	.720	.182
		Extention	.749**	.554**	.256	-.04	.061	-.065	.918**	.217
	Hip	Extention	.836**	.705**	.508**	-.127	.302*	-.143	.932**	-.067
		Flexion	.873**	.761**	.569**	-.025	.347*	-.023	.956**	.030
Power	Ankle	Dorsiflexion	.694**	.313*	.18	-.002	-.086	.027	.906**	-.231
		Planterflexion	.838**	.663**	.436**	.072	.285*	.067	.934**	.193
	Knee	Flexion	.696**	.500**	.187	.102	.009	.073	.905**	.182
		Extention	.734**	.554**	.256	-.04	.061	-.065	.918**	.217
	Hip	Extention	.837**	.704**	.508**	-.127	.302*	-.143	.932**	-.067
		Flexion	.870**	.761**	.569**	-.025	.347*	-.023	.956**	.030

BM Body Mass, BF% Body fat percentage, FM Fat mass, FFM Fat free mass

* Correlation is significant $p < 0.05$

** Correlation is significant $p < 0.01$

Group differences in strength.

Absolute knee extension (p=0.002, p=0.003, p=0.003), knee flexion (p=0.013, p=0.003, p=0.003) isometric torque, isokinetic torque and power were significantly greater on OWB (Table 4.4.6, 4.4.7, 4.4.8, 4.4.9). OWB also had significantly (p=0.01) greater absolute isokinetic torque and power.

Scaling to body mass, the OWB group were significantly weaker in isometric ankle dorsiflexion, hip abduction and hip adduction. Ankle plantar flexors were significantly (p=0.009, p=0.009) weaker and less powerful in OWB body weight scaled isokinetic torque and power. Isokinetic hip flexion scaled to bodyweight was significantly weaker in the OWB group (p=0.045). Hip extensors were significantly (p=0.014) less powerful when scaled to body weight. OWB were significantly weaker in all movements for isometric, isokinetic and power scaled to body fat percentage (table 4.4.9). When isometric and isokinetic was scaled to fat mass OWB were significantly weaker (table 4.4.6, 4.4.7, 4.4.8). OWB produced significantly less power scaled to fat mass in all movement except knee extension, which was non-significant (table 4.4.9).

When isometric, isokinetic and power was allometricaly scaled to body mass and fat mass OWB were significantly weaker in all muscle groups. Allometrically scaled to fat free mass, isokinetic torque and power for the OWB group was significantly less than HW. Isometric torque allometricaly scaled to fat free mass showed OWB to be weaker in al muscle groups except the hip flexors.

Table 4.4.6. Mean ± SD frontal plane isometric absolute torque, and ratio and allometrically scaled to, body weight, body fat percentage, fat mass and fat free mass comparison for healthy weight group, and overweight and obese group.

Joint	Direction		HW	OWB	*p*	
Hip	Adduction	Nm	21.2 ± 9.1	22.71 ± 11.92	0.645	
		Nm/Kg	0.7 ± 0.3	0.53 ± 0.3	0.029	*
		Nm/BF%	1.4 ± 0.8	0.68 ± 0.41	0.000	*
		Nm/FM(kg)	4.9 ± 3.2	1.65 ± 1.05	0.000	*
		Nm/FFM(Kg)	0.9 ± 0.3	0.82 ± 0.48	0.499	
		$Nm/Kg^{1.4}$	0.52 ± 0.4	0.17 ± 0.11	0.000	*
		$Nm/BF\%^{1.4}$	0.23 ± 0.09	0.23 ± 0.13	0.967	
		$Nm/FM(kg)^{1.4}$	3.04 ± 2.8	0.66 ± 0.4	0.000	*
		$Nm/FFM(Kg)^{1.4}$	0.18 ± 0.07	0.13 ± 0.07	0.004	*
	Abduction	Nm	17 ± 6.4	16.1 ± 7.14	0.544	
		Nm/Kg	0.6 ± 0.2	0.38 ± 0.2	0.001	*
		Nm/BF%	1.2 ± 0.7	0.5 ± 0.3	0.000	*
		Nm/FM(kg)	4.1 ± 2.6	1.24 ± 0.82	0.000	*
		Nm/FFM(Kg)	0.7 ± 0.2	0.58 ± 0.29	0.056	
		$Nm/Kg^{1.4}$	0.44 ± 0.32	0.13 ± 0.08	0.000	*
		$Nm/BF\%^{1.4}$	0.2 ± 0.07	0.16 ± 0.07	0.086	
		$Nm/FM(kg)^{1.4}$	2.6 ± 2.33	0.49 ± 0.33	0.000	*
		$Nm/FFM(Kg)^{1.4}$	0.15 ± 0.06	0.09 ± 0.05	0.000	*

* Denotes significance between HW and Own groups $p<0.05$

Table 4.4.7. Mean ± SD sagittal plane isometric absolute torque, ratio and allometrically scaled torque to body weight, body fat percentage, fat mass and fat free mass comparison for healthy weight group, and overweight and obese group.

	Joint	Direction		HW	OWB	*p*	
Isometric	Ankle	Dorsiflexion	Nm	6.5 ± 2.5	7.22 ± 2.79	0.321	
			Nm/Kg	0.2 ± 0.1	0.18 ± 0.08	0.001	*
			Nm/BF%	2.1 ± 0.9	1.2 ± 0.51	0.000	*
			Nm/FM(kg)	7.2 ± 3.2	2.96 ± 1.43	0.000	*
			Nm/FFM(Kg)	1.3 ± 0.4	1.44 ± 0.43	0.584	
			$Nm/Kg^{1.6}$	0.45 ± 0.28	0.15 ± 0.08	0.000	*
			$Nm/BF\%^{1.6}$	0.19 ± 0.06	0.2 ± 0.07	0.414	
			$Nm/FM(kg)^{1.6}$	3.43 ± 2.58	0.69 ± 0.45	0.000	*
			$Nm/FFM(Kg)^{1.6}$	0.14 ± 0.04	0.1 ± 0.04	0.001	*
		Planterflexion	Nm	32.2 ± 12.3	38.56 ± 11.23	0.067	
			Nm/Kg	1.1 ± 0.4	0.94 ± 0.3	0.057	
			Nm/BF%	0.5 ± 0.3	0.23 ± 0.16	0.000	*
			Nm/FM(kg)	1.6 ± 1.2	0.58 ± 0.5	0.000	*
			Nm/FFM(Kg)	0.3 ± 0.1	0.27 ± 0.1	0.594	
			$Nm/Kg^{1.6}$	0.1 ± 0.1	0.03 ± 0.03	0.001	*
			$Nm/BF\%^{1.6}$	0.04 ± 0.01	0.04 ± 0.01	0.673	
			$Nm/FM(kg)^{1.6}$	0.81 ± 0.89	0.14 ± 0.17	0.000	*
			$Nm/FFM(Kg)^{1.6}$	0.03 ± 0.01	0.02 ± 0.01	0.001	*
	Hip	Extention	Nm	17.7 ± 7.2	20.68 ± 10.72	0.417	
			Nm/Kg	0.6 ± 0.2	0.54 ± 0.28	0.161	
			Nm/BF%	1.2 ± 0.6	0.65 ± 0.41	0.001	*
			Nm/FM(kg)	4.1 ± 2.2	1.61 ± 1.04	0.000	*
			Nm/FFM(Kg)	0.7 ± 0.3	0.77 ± 0.37	0.941	
			$Nm/Kg^{1.4}$	0.43 ± 0.28	0.17 ± 0.11	0.000	*
			$Nm/BF\%^{1.4}$	0.2 ± 0.07	0.21 ± 0.09	0.609	
			$Nm/FM(kg)^{1.4}$	2.51 ± 1.94	0.62 ± 0.43	0.000	*
			$Nm/FFM(Kg)^{1.4}$	0.15 ± 0.06	0.12 ± 0.06	0.030	*
		Flexion	Nm	25.3 ± 13.1	30.57 ± 16.93	0.29	
			Nm/Kg	0.8 ± 0.4	0.7 ± 0.41	0.276	
			Nm/BF%	1.7 ± 1.2	0.98 ± 0.62	0.002	*
			Nm/FM(kg)	5.9 ± 4.8	2.4 ± 1.56	0.000	*
			Nm/FFM(Kg)	1 ± 0.5	1.15 ± 0.56	0.652	
			$Nm/Kg^{1.4}$	0.63 ± 0.6	0.25 ± 0.16	0.002	*
			$Nm/BF\%^{1.4}$	0.28 ± 0.14	0.31 ± 0.14	0.475	
			$Nm/FM(kg)^{1.4}$	3.7 ± 4.12	0.91 ± 0.64	0.001	*
			$Nm/FFM(Kg)^{1.4}$	0.22 ± 0.11	0.17 ± 0.09	0.080	
	Knee	Extention	Nm	30.3 ± 6.8	38.07 ± 10.4	0.002	*
			Nm/Kg	2 ± 0.4	1.69 ± 0.47	0.018	*
			Nm/BF%	3.9 ± 2.1	2.15 ± 0.88	0.000	*
			Nm/FM(kg)	13.9 ± 8.8	5.24 ± 2.5	0.000	*
			Nm/FFM(Kg)	2.4 ± 0.5	2.6 ± 0.59	0.487	
			$Nm/Kg^{1.4}$	0.79 ± 0.59	0.29 ± 0.13	0.000	*
			$Nm/BF\%^{1.4}$	0.35 ± 0.08	0.38 ± 0.1	0.184	
			$Nm/FM(kg)^{1.4}$	4.71 ± 4.07	1.08 ± 0.62	0.000	*
			$Nm/FFM(Kg)^{1.4}$	0.27 ± 0.07	0.21 ± 0.06	0.001	*
		Flexion	Nm	57.8 ± 12.9	69.41 ± 18.92	0.013	*
			Nm/Kg	1 ± 0.2	0.92 ± 0.25	0.081	
			Nm/BF%	2.1 ± 1.1	1.17 ± 0.43	0.000	*
			Nm/FM(kg)	7.4 ± 4.5	2.86 ± 1.25	0.000	*
			Nm/FFM(Kg)	1.3 ± 0.3	1.43 ± 0.37	0.232	
			$Nm/Kg^{1.4}$	1.47 ± 1.16	0.54 ± 0.27	0.000	*
			$Nm/BF\%^{1.4}$	0.66 ± 0.13	0.69 ± 0.16	0.374	
			$Nm/FM(kg)^{1.4}$	8.76 ± 8.27	1.97 ± 1.23	0.000	*
			$Nm/FFM(Kg)^{1.4}$	0.51 ± 0.11	0.38 ± 0.1	0.000	*

* Denotes significance between HW and Own groups $p<0.05$

Table 4.4.8. Mean ± SD isokinetic absolute torque, ratio and allometrically scaled torque to body weight, body fat percentage, fat mass and fat free mass comparison for healthy weight group, and overweight and obese group.

Joint	Direction		HW	OWB	*p*	
Isokinetic Ankle	Dorsiflexion	Nm	4.8 ± 1.5	5.86 ± 1.56	0.01	*
		Nm/Kg	0.2 ± 0	0.14 ± 0.03	0.057	
		Nm/BF%	0.3 ± 0.1	0.18 ± 0.07	0.000	*
		Nm/FM(kg)	1.1 ± 0.5	0.43 ± 0.18	0.000	*
		Nm/FFM(Kg)	0.2 ± 0	0.22 ± 0.05	0.069	
		$Nm/Kg^{1.6}$	0.07 ± 0	0.02 ± 0	0.000	*
		$Nm/BF\%^{1.6}$	0.03 ± 0.01	0.03 ± 0.01	0.304	
		$Nm/FM(kg)^{1.6}$	0.52 ± 0.41	0.1 ± 0.06	0.000	*
		$Nm/FFM(Kg)^{1.6}$	0.02 ± 0.05	0.02 ± 0.01	0.000	*
	Planterflexion	Nm	16.9 ± 6.3	18.44 ± 6.88	0.528	
		Nm/Kg	0.6 ± 0.2	0.45 ± 0.18	0.009	*
		Nm/BF%	1.2 ± 0.7	0.57 ± 0.3	0.000	*
		Nm/FM(kg)	4 ± 2.8	1.41 ± 0.81	0.000	*
		Nm/FFM(Kg)	0.7 ± 0.3	0.68 ± 0.24	0.673	
		$Nm/Kg^{1.6}$	0.26 ± 0.25	0.07 ± 0.04	0.000	*
		$Nm/BF\%^{1.6}$	0.1 ± 0.04	0.09 ± 0.03	0.469	
		$Nm/FM(kg)^{1.6}$	2.05 ± 2.28	0.33 ± 0.24	0.000	*
		$Nm/FFM(Kg)^{1.6}$	0.08 ± 0.03	0.05 ± 0.02	0.000	*
Hip	Extention	Nm	19.5 ± 6.1	19.58 ± 8.77	0.866	
		Nm/Kg	18.8 ± 7	19.58 ± 8.77	0.954	
		Nm/BF%	1.2 ± 0.6	0.62 ± 0.34	0.000	*
		Nm/FM(kg)	4.2 ± 2.1	1.52 ± 0.89	0.000	*
		Nm/FFM(Kg)	0.8 ± 0.3	0.72 ± 0.29	0.608	
		$Nm/Kg^{1.4}$	0.43 ± 0.26	0.16 ± 0.1	0.000	*
		$Nm/BF\%^{1.4}$	0.22 ± 0.06	0.2 ± 0.08	0.329	
		$Nm/FM(kg)^{1.4}$	2.56 ± 1.59	0.6 ± 0.41	0.000	*
		$Nm/FFM(Kg)^{1.4}$	0.16 ± 0.05	0.11 ± 0.05	0.001	*
	Flexion	Nm	15.6 ± 5.7	16.41 ± 9.29	0.96	
		Nm/Kg	0.51 ± 0.19	0.40 ± 0.21	0.045	*
		Nm/BF%	1 ± 0.5	0.51 ± 0.31	0.000	*
		Nm/FM(kg)	3.3 ± 1.6	1.25 ± 0.81	0.000	*
		Nm/FFM(Kg)	0.6 ± 0.2	0.62 ± 0.33	0.511	
		$Nm/Kg^{1.4}$	0.34 ± 0.19	0.13 ± 0.09	0.000	*
		$Nm/BF\%^{1.4}$	0.18 ± 0.06	0.17 ± 0.09	0.574	
		$Nm/FM(kg)^{1.4}$	2.01 ± 1.09	0.48 ± 0.37	0.000	*
		$Nm/FFM(Kg)^{1.4}$	0.13 ± 0.05	0.09 ± 0.05	0.003	*
Knee	Extention	Nm	30.6 ± 9.3	40 ± 13.15	0.003	*
		Nm/Kg	1.05 ± 0.33	0.98 ± 0.31	0.421	
		Nm/BF%	2.1 ± 1.1	1.24 ± 0.56	0.001	*
		Nm/FM(kg)	7.3 ± 4.7	3.05 ± 1.6	0.000	*
		Nm/FFM(Kg)	1.3 ± 0.4	1.5 ± 0.45	0.045	*
		$Nm/Kg^{1.4}$	0.75 ± 0.58	0.32 ± 0.17	0.000	*
		$Nm/BF\%^{1.4}$	0.35 ± 0.11	0.4 ± 0.12	0.098	
		$Nm/FM(kg)^{1.4}$	4.54 ± 4.23	1.17 ± 0.78	0.000	*
		$Nm/FFM(Kg)^{1.4}$	0.27 ± 0.09	0.22 ± 0.07	0.031	*
	Flexion	Nm	21.9 ± 5.7	28 ± 8.38	0.003	*
		Nm/Kg	0.8 ± 0.2	0.68 ± 0.21	0.214	
		Nm/BF%	1.5 ± 0.8	0.87 ± 0.37	0.000	*
		Nm/FM(kg)	7.08 ± 3.60	1.97 ± 1.45	0.000	*
		Nm/FFM(Kg)	1.26 ± 0.48	0.91 ± 0.48	0.009	*
		$Nm/Kg^{1.4}$	0.55 ± 0.4	0.22 ± 0.11	0.000	*
		$Nm/BF\%^{1.4}$	0.25 ± 0.07	0.28 ± 0.08	0.200	
		$Nm/FM(kg)^{1.4}$	3.34 ± 2.98	0.8 ± 0.51	0.000	*
		$Nm/FFM(Kg)^{1.4}$	0.2 ± 0.06		0.007	*

* Denotes significance between HW and Own groups $p<0.05$

Table 4.4.9. Mean ± SD absolute power, ratio and allometrically power scaled to, body weight, body fat percentage, fat mass and fat free mass comparison for healthy weight group, and overweight and obese group.

	Joint	Direction		HW	OWB	*p*	
Power	Ankle	Dorsiflexion	W	143.3 ± 43.7	175.92 ± 46.77	0.01	*
			W/Kg	4.8 ± 1.1	4.21 ± 1.03	0.057	
			W/BF%	9.5 ± 4.2	5.39 ± 2.02	0.000	*
			W/FM(kg)	32.7 ± 15	13.04 ± 5.39	0.000	*
			W/FFM(Kg)	5.8 ± 1.4	6.55 ± 1.5	0.069	
			$Nm/Kg^{1.6}$	2.08 ± 1.43	0.68 ± 0.34	0.000	*
			$Nm/BF\%^{1.6}$	0.85 ± 0.2	0.91 ± 0.21	0.304	
			$Nm/FM(kg)^{1.6}$	15.75 ± 12.28	3 ± 1.83	0.000	*
			$Nm/FFM(Kg)^{1.6}$	0.64 ± 0.14	0.46 ± 0.12	0.000	*
		Planterflexion	W	507.3 ± 188.4	553.06 ± 206.49	0.528	
			W/Kg	17.3 ± 6.2	13.52 ± 5.45	0.009	*
			W/BF%	34.5 ± 21.4	17.19 ± 8.91	0.000	*
			W/FM(kg)	121.5 ± 82.9	42.18 ± 24.33	0.000	*
			W/FFM(Kg)	21 ± 7.8	20.3 ± 7.13	0.673	
			$Nm/Kg^{1.6}$	7.84 ± 7.64	2.19 ± 1.34	0.000	*
			$Nm/BF\%^{1.6}$	3.07 ± 1.2	2.85 ± 1.02	0.469	
			$Nm/FM(kg)^{1.6}$	61.37 ± 68.47	9.76 ± 7.08	0.000	*
			$Nm/FFM(Kg)^{1.6}$	2.29 ± 0.86	1.5 ± 0.65	0.000	*
	Hip	Extention	W	1167.4 ± 364	1175.15 ± 526.05	0.866	
			W/Kg	37.8 ± 12.5	28.69 ± 13.03	0.014	*
			W/BF%	72.6 ± 35.5	37.07 ± 20.41	0.000	*
			W/FM(kg)	249.5 ± 126.4	90.91 ± 53.49	0.000	*
			W/FFM(Kg)	46 ± 15.5	43.21 ± 17.69	0.609	
			$Nm/Kg^{1.4}$	25.75 ± 15.62	9.67 ± 5.74	0.000	*
			$Nm/BF\%^{1.4}$	13.07 ± 3.5	11.96 ± 4.73	0.329	
			$Nm/FM(kg)^{1.4}$	153.51 ± 95.19	35.87 ± 24.76	0.000	*
			$Nm/FFM(Kg)^{1.4}$	9.76 ± 3.27	6.81 ± 3.04	0.001	*
		Flexion	W	933.9 ± 339.1	984.67 ± 557.45	0.96	
			W/Kg	30.3 ± 11.4	23.64 ± 13.15	0.069	
			W/BF%	57.5 ± 27.9	30.7 ± 18.88	0.000	*
			W/FM(kg)	197.3 ± 96.4	75 ± 48.85	0.000	*
			W/FFM(Kg)	36.7 ± 13.7	37.04 ± 19.83	0.522	
			$Nm/Kg^{1.4}$	20.32 ± 11.12	7.85 ± 5.19	0.000	*
			$Nm/BF\%^{1.4}$	10.67 ± 3.31	10 ± 5.17	0.575	
			$Nm/FM(kg)^{1.4}$	120.59 ± 65.54	28.92 ± 21.91	0.000	*
			$Nm/FFM(Kg)^{1.4}$	7.95 ± 2.93	5.49 ± 2.95	0.003	*
	Knee	Extention	W	1836 ± 557.4	2400.15 ± 788.8	0.003	*
			W/Kg	62.8 ± 19.4	57.63 ± 19.11	0.367	
			W/BF%	123.3 ± 67.6	74.64 ± 33.61	0.001	*
			W/FM(kg)	435.6 ± 279.2	182.84 ± 96.12	0.000	*
			W/FFM(Kg)	76.1 ± 23.3	89.81 ± 27.2	0.045	*
			$Nm/Kg^{1.4}$	45.24 ± 34.61	18.99 ± 10.19	0.000	*
			$Nm/BF\%^{1.4}$	21.1 ± 6.86	24.29 ± 7.18	0.098	
			$Nm/FM(kg)^{1.4}$	272.31 ± 253.51	70.01 ± 46.91	0.000	*
			$Nm/FFM(Kg)^{1.4}$	16.38 ± 5.48	13.43 ± 4.47	0.031	*
		Flexion	W	1312.6 ± 342.1	1680.12 ± 502.93	0.003	*
			W/Kg	45 ± 12.2	40.65 ± 13.21	0.202	
			W/BF%	89.5 ± 46.7	52.18 ± 21.91	0.000	*
			W/FM(kg)	317.3 ± 195.2	128.15 ± 63.77	0.000	*
			W/FFM(Kg)	54.4 ± 14.5	62.67 ± 18.04	0.074	
			$Nm/Kg^{1.4}$	33.08 ± 24.28	13.12 ± 6.59	0.000	*
			$Nm/BF\%^{1.4}$	15.18 ± 4.38	16.78 ± 4.79	0.200	
			$Nm/FM(kg)^{1.4}$	200.13 ± 178.94	48.29 ± 30.42	0.000	*
			$Nm/FFM(Kg)^{1.4}$	11.81 ± 3.5	9.36 ± 2.99	0.007	*

* Denotes significance between HW and Own groups $p<0.05$

4.5 Discussion

The aim of the study was to examine lower limb and foot gait kinetics and kinematics, plantar pressures, lower limb strength and the effect of torque and power normalisation methods between OWB and HW groups. The main findings of this study show OWB to have significantly greater knee flexion moment, knee adduction power generation and greater mediolateral centre of pressure excursion. OWB tended to be relative weaker. Allometric scaling was effective in scaling to body fat percentage, fat mass and fat free mass, but an effective scaling method to body mass is still needed.

Gait kinematics.

No significant differences between OWB and HW groups were found in any lower limb or foot segment angles. Differences in peak joint angles in obese children at the ankle hip and knee have previously been reported (Freedman Silvernail et al., 2013; Gushue et al., 2005; McMillan et al., 2010; McMillan et al., 2009; Shultz, et al., 2014). However, angular motion pattern was not statistically different between groups in this study. This may be due to the greater inter-subject variability seen in paediatric gait kinematics whilst movement patterns may remain relatively stable (Sutherland, 1997). Mahaffey et al. (2012) found a significant effect of body fat on foot segment angular motion during gait in obese children in calcaneus and midfoot. However, the current study suggests, when children are categorised by BMI Z score, there are no statistically significant differences in foot segment motion throughout gait in obese and healthy weight children.

Gait Kinetics.

The only significant effect of weight status was found in the knee extensor moment using 5 to 23% and 73-86% of the gait cycle and frontal knee power 70-84% estimated to be loading phase to midstance phase and initial swing to midswing. During loading to midstance the knee flexes during support, requiring an eccentric action of the quadriceps to limit speed and magnitude of knee flexion, then as the knee reaches peak knee flexion the extensors move from eccentric to concentric. The change from eccentric to concentric allows the knee to act as a spring preventing rapid build up of vertical force (Levine et al., 2012). OWB had an increased knee extensor moment

suggesting the knee extensors are having to produce more force to control joint angles and counter greater vertical force due to increased mass. During initial swing to midswing, the knee moves from flexion to extension, and is largely a passive pendulum motion (Levine et al., 2012). However, the shorter swing phase characteristically seen in obese children, may require greater moments of the knee extensors to achieve adequate knee extension in a shorter time period (Wearing et al,, 2006).

The greater knee adduction absorption during occurs during the same stage of the swing phase as increased extension moments, this suggests that power absorption is required to help control against forces with this peak in knee moment. The lack of kinematic differences between groups but increased joint moments and powers suggest these kinetic differences are due to carrying excess mass and not in producing greater or altered joint angles (Morrison, Mahaffey, Cousins, & Drechsler, 2012; Shultz et al., 2009). Body mass increases are not proportionate to increases in joint articulating surface area, this may put increased stress on joint and lead to discomfort and deformity of the knee such as Blounts disease (Morrison et al., 2012).

Plantar pressures.

The only significant finding from pedabaragraph data was the increased total medio-lateral centre of pressure excursion during stance in the OWB group. This is a similar finding to previous literature, where obese children have had significantly larger lateral centre of pressure displacement during standing trials, and obese adults during gait initiation (Cau et al., 2014; McGraw et al., 2000). In adult populations a larger centre of pressure displacement is a marker of instability and fall risk (Bizovska et al., 2014; Svoboda et al., 2015). Whilst these results may indicate greater instability on OWB, the values presented were absolute excursion from the midline and not relative to foot width. Given the trend for obese to have larger and wider feet (Morrison et al., 2012) relative to foot width mediolateral excursion may not be greater.

However, unlike previous literature plantar pressures under the three regions were not significant between groups in this study. This could be due to a limitation within the methodology, where the areas of the foot have been automatically masked, foot region force over the gait cycle was divided by a total area of the segment, and not the area in contact with the plate at each time point. However, total contact area in the three regions of the foot were also not significantly different between OWB and HW group.

Effectiveness of strength normalisation.

Ratio scaling was not effective in removing the effect of covariates: body mass, body fat percentage, fat mass and fat free mass, as evidenced by the high r^2 values and significant relationships between normalised strength values and absolute strength values (Payton & Bartlett, 2008). This shows that growth, whether in body mass, fat mass or fat free mass, is not proportionate to increases in isometric, isokinetic torque or muscle power. Empirically derived parameters from Wren and Engsberg, (2007) were more appropriate to scaling strength to body fat percentage, fat mass and fat free mass. This is similar to Karavelioglu, Harmanci, & Caliskan, (2017) who found allometric scaling to be far more effective in removing mass as a covariate in child athletes. However, significant relationships remained in allometrically scaled strength to body mass. These results indicate that the proposed normalisation equations were not appropriate for body mass normalisation in cohort containing obese children.

Differences in strength between OWB and HW.

Absolute torque and power were not significantly different between OWB and HW groups in all muscle groups except knee extensors and flexors for isometric, isokinetic and power variables where OWB were stronger than HW. This is in line with previous literature where obese children have been shown to be significantly stronger in the knee extensors (Abdelmoula et al., 2012; Maffiuletti et al., 2008; Tsiros et al., 2013). However, the absolute torque values in the current study were lower than previously reported in the literature for the knee extensors (obese values 232.1 $\pm$ 65.2 Nm, 193 $\pm$ 26 Nm and 124.6(4.6) Nm) (Abdelmoula et al., 2012; Maffiuletti et al., 2008; Tsiros et al., 2013). As well as at the hip and ankle in healthy weight children (hip adductors 54.3 ± 40.9 Nm, ankle plantarflexors 43 ± 30.5) (Wren & Engsberg, 2007). This may

be due to the relatively younger cohort in the current study (7-11 years) compared to the adolescent (10 – 17 years) participants in previous literature.

OWB and HW children showed no significant difference in allometrically scaled strength to body fat percentage torque or power. However, when torque and power ratio scaled and allometrically scaled to fat mass (kg), the OWB were significantly weaker in all muscle groups. These results suggest that any training effect of carrying excess weight is not adequate to manage the increased fat mass (Jaric, 2002). Furthermore, torque and power allometrically scaled to fat free mass (kg) showed the HW group to be significantly stronger in all muscle groups except hip flexors during isometric contraction. This suggests the OWB were less able to produce torque and power to the same extent of HW group, due to either poor co-ordination and/or reduced neuromuscular activity. This finding differs from Abdelmoula et al. (2012) who found obese children to be significantly stronger when strength was normalised to thigh lean mass and thigh muscle mass. However, in the current study torque and power normalised to whole body fat free mass as calculated from BOD POD estimates compared to DEXA scan which can calculate limb specific values as used by Abdelmoula et al. (2012). If there is a training effect of carrying excess mass, the increased muscle mass seen in obese children may be in the weight bearing structures of the lower limb. Therefore, obese children may have a larger lean mass in the lower limbs altering the effect of total body fat free mass normalisation in lower limb strength. Although, the relationship between fat free mass, muscle mass and neuromuscular factors effecting strength production in the lower limb need further investigation in obese children.

Limitations.

Whilst this study has highlighted differences in strength, stability during gait and knee joint moments, unlike previous literate no difference was found in plantar pressures or ankle and hip joint moments or powers. This may due to methodology in defining foot segments and the use of an overweight and obese group, whereby the removal of the overweight may have highlighted greater difference between obese and heathy weight children. Furthermore, the use of BODPOD derived estimated whole-body fat free

mass rather than a measure of muscle mass may affect the results and interpretation of strength normalised to fat free mas in this study.

4.6 Conclusion

The current study has demonstrated joint angle gait patterns to be similar between OWB and HW groups, meaning the increased knee joint moments and powers in OWB are due to increased mass and not altered kinematics. This could increase loading and lead to disorders such as Blounts disease. OWB children may be less stable during stance phase, however measures accounting for the overall larger foot width seen on OWB children should be investigated. Additionaly, the current study has shown that allometric scaling parameters for the lower limb (ankle torque/mass $^{1.6}$, hip and knee torque/mass $^{1.4}$) were not effective in normalising isometric torque, isokinetic torque and power to body mass in a cohort containing healthy weight, overweight and obese children. The parameters were effective in normalising to body fat percentage, fat mass and fat free mass. Whilst OWB were stronger in absolute terms at the knee, and ankle dorsiflexors. When scaled to body mass and fat mass obese tended to be significantly weaker than HW, suggesting inadequate strength per kg of mass compared to HW children. However, the use of an overweight and obese group may have limited further significant findings. Additionally, the use of BMI derived Z scores which has its own inherent limitations and therefore examining the roll of body fat in strength and gait parameters may reveal more about how adiposity effects function in obese children.

5. Relationship of body fat to gait kinetic and kinematics, plantar pressure, strength and strength normalisation methods in children aged 7 to 11 years.

5.1 Introduction.

Over one third of children in the UK are overweight or obese, and this number is currently set to rise (Lobstein & Jackson-Leach, 2016). Obese and overweight children have been shown to have altered lower limb and foot biomechanics during gait due to the carriage of excess mass on an immature musculoskeletal system (Freedman Silvernail et al., 2013; Gushue et al., 2005; Lerner et al., 2016; McMillan et al., 2010; Shultz et al., 2014; Shultz et al., 2010, 2009; Mahaffey et al., 2016). Poor biomechanical function can effect musculoskeletal health and have effects on physical activty and quality of life (Shultz et al., 2009). Relative lower limb strength has been implicated as a factor for altered gait in obese children, and strength differences have been noted in OWB and HW groups (Abdelmoula et al., 2012; Maffiuletti et al., 2008; Tsiros et al., 2013). Whilst it has been shown that obese children exhibit a training effect similar to restiance training through the carraige of excess mass, the rate at which strength is gained may not be adequate to the rate of increasing mass.

The majority of studies investigating gait, plantar pressure and strength in obese children use BMI Z scores or centiles based on relevant population distributions to determine obese, overweight and obese. This makes data easy to compare between groups and apply findings back to a clinically relevent population. However, BMI and thefore BMI derived Z scores rely on a set of assumptions. In adults, anthropometric measurements remain relatively constant except when there is a loss or gain of weight and therefore the classifications remain relevant despite age. However, children go through different patterns of growth and maturity at different ages. Therefore, at any given height, variation in mass will not be attributed to differences in fat mass alone (Flegal, 1993). Furthermore, where data is included from healthy weight (<2nd centile – >85th centile), overweight (<85th centile - >95th centile) and obese (>95th centile) participants the difference between the upper extreme of the healthy weight scale and the bottom extreme of overweight or obese is relatively small.

It is the impact of excess fat mass rather than overall body mass (which includes fat mass and contributory fat free mass) that is thought to be driving relative biomechanical changes and strength deficits (Shultz et al., 2010). Therefore, to begin to determine how strength and gait are significantly affected by increased body fat in children, the relationship between these must be investigated.

Aim. To investigate the relationship of body fat with plantar pressures, gait kinetic and kinematics in the lower limb and foot, and maximal strength of the lower limb.

5.2 Methods

Sixty-six children completed anthropometric measures, including body composition analysis. Gait analysis of the pelvis, lower limb and foot segments, and plantar pressures were collected simultaneously. Participants completed isometric and isokinetic strength assessment of the hip, knee, and ankle on an isokinetic dynamometer. See general methods for details.

5.3 Data analysis

All statistical analyses was completed using SPSS version 22. All data was tested for normality using shapiro-wilks.

Gait.

Principal component analysis was used to reduce angular, moment, and power time curves from the pelvis, hip, knee, ankle and angular motion of three foot segments (shank to calcaneus, calcaneus to midfoot and midfoot to metatarsals) as well as plantar pressures over the forefoot, midfoot and rearfoot over the stance phase to select principal components (see general methods). Regressions scores for each principal component derived from the PCA were entered into linear regression as dependent variables and body fat percentage and walking velocity as predictor variables using stepwise regression method. If body fat percentage and walking

velocity was significantly associated with strength, further analysis was completed in mixed model linear regression to account for confounding effects of predictor variables.

Plantar pressure.

Foot axis angle, medio-lateral centre of pressure displacement, centre of pressure medio-lateral centre of pressure velocity, centre of pressure root mean square and total contact area of the forefoot, midfoot and rearfoot were entered in linear regression with body fat as the predictor variable.

Strength.

Each torque and power variable was scaled to body mass (kg) (torque·BM(kg)$^{-1}$) and allometricaly scaled to fat free mass based on Wren & Engsberg (2007) derived parameters for hip, knee and ankle. To determine the association between body fat and strength, absolute torque and power, and scaled strength were entered into linear regression as the dependent variable and body fat as the predictor variable. Confounding effects of age and height on absolute strength were entered in multiple linear regression using a stepwise regression method for absolute strength. If body fat percentage and one or more other variables was significantly associated with strength, further analysis was completed in mixed model linear regression to account for confounding effects of predictor variables.

5.4 Results

Sixty-six children were recruited (53% male, mean ± SD, age 9.23 ± 0.99 yrs, height 1.35 ± 0.53 m, mass 34.23 ± 8 kg). Participants included 39 healthy weight, 9 overweight and 18 obese (mean ± SD Z score 0.57 ± 1.64, BF% 23.55 ± 11.25).

Gait.

Pelvis, hip, knee and ankle kinematics were not significantly predicted by body fat percentage. Figure 5.4. shows the significant effect of body fat percentage on ankle moment and power. As body fat percentage, increased ankle abduction moments increased at 29-54% ($F(1,65)=11.824$) and 61 – 86% ($F(2,62)=5.897$) of the gait cycle.

Ankle internal rotation moments increased 25-55% ($F(1,65)=5.239$) and 62-88%($F(2,65)=5.634$) of the gait cycle and plantar flexion moment increased 56-87% ($F=(2,62)=7.203$) and 44-54% ($F(1,65)=11.307$) of the gait cycle. Body fat percentage also significantly predicted frontal, transverse and sagittal plane ankle power. As body fat increased so did power generation in ankle abductors and internal rotators at 63 - 78% ($F(1,65)=6.508$) of the gait cycle 20-28% ($F(2,62)=5.681$) of the gait cycle respectively. Power absorption increased with body fat in the plantarflexions 60-82% ($F(1,65)=6.578$) at of the gait cycle after accounting for walking velocity. Figure 5.3 shows significant association of body fat percentage on knee moments and powers. Knee abduction moments at 88 -97% ($F(2,62)=5.286$) and knee internal rotation moment increased at 28 – 56% ($F(1,65)=4.897$) of the gait cycle increased as body fat percentage increased. Power absorption of the knee adductors at 70-84% ($F(2,62)=10.135$) increased as body fat percentage increased. Body fat percentage significantly predicted hip moments and transverse hip power. Figure 5.4 shows the significant effect of body fat percentage on hip moments and transverse power. As body fat increased hip abduction moment increased at 6-50% ($F(1, 65)=10.135$) of the gait cycle, hip flexion moment increases at 77 -90 % ($F(2,62)=11.061$) of the gait cycle and external hip rotation moment increased at 5-38% ($F(1,65)=11.608$) and 43-60% ($F(1,65)=9.229$). Power absorption of internal hip rotators increased as body fat percentage increased at 79-87% ($F(1,62)=5.669$) of the gait cycle. °

Table 5.4.1. Summary of significant linear regression relationship of lower limb kinetics and body fat percentage and walking velocity.

				BF%		BF%* Walking velocity		Predictor variable
				p	R2	p	R2	Beta, (std error)
Ankle	Moment	Frontal	PC1	0.001	0.158	-		-.398, (0.01)
			PC3	-		0.004	0.167	-.254, (.010)
		Tansverse	PC1	0.026	0.079	-		.281, (.011)
			PC2	-		0.006	0.157	.304, (.010)
		Sagittal	PC1	-		0.002	0.238	.296, (.010)
			PC4	0.001	0.159	-		398, (.010)
	Power	Frontal	PC1	0.013	0.104	-		.323, (0.011)
		Transverse	PC1	-		0.005	0.157	301, (0.010)
		Sagittal	PC1	0.013	0.107	-		-.327, (0.010)
Knee	Moment	Frontal	PC3	-		0.008	0.144	-2.281, (0.011)
		Transverse	PC1	0.03	0.071	-		2.213, (0.011)
	Power	Frontal	PC1	-		0.004	0.169	-2.714, (0.010)
Hip	Moment	Frontal	PC1	0.002	0.141	-		.375, (0.011)
		Transverse	PC1	0.001	0.180	-		-.424, (0.010)
			PC2	0.003	0.130	-		-.360, (0.011)
		Sagittal	PC3	-		0.045	0.266	.242, (0.010)
	Power	Transverse	PC3	0.021	0.09	-		.301, (0.011)

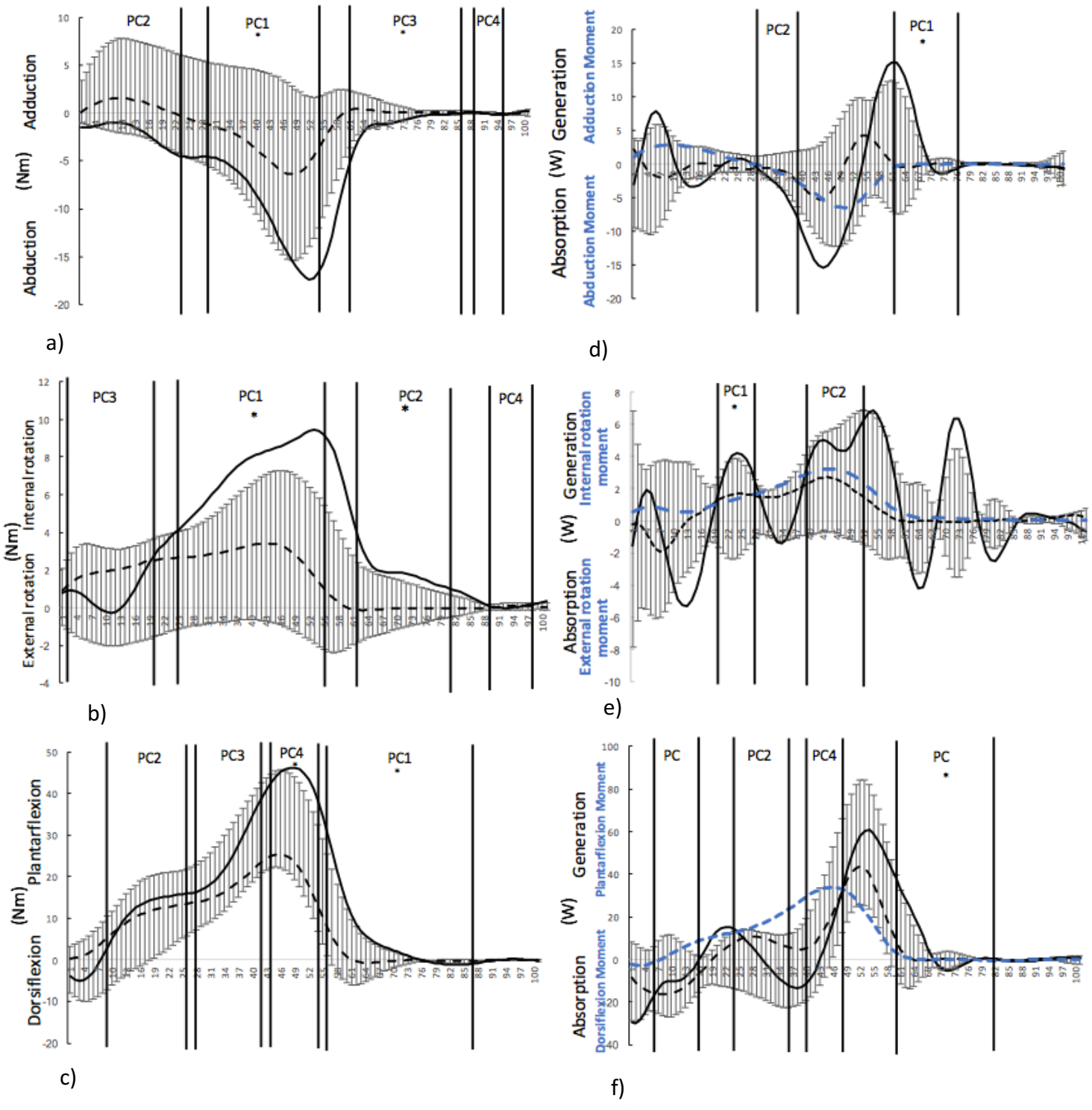

Figure 5.4.1. Ankle moments (Nm) (a-c), and ankle powers (W) (d-f), mean of five participants with highest body fat percentage (solid black line), mean of five participants with lowest body fat percentage (black dashed line) over the gait cycle standard deviation of the mean across all participants. Blue dashed lines represent mean joint moment for all participants. * denotes significant ($p<0.05$) prediction of body fat percentage on principal component scores.

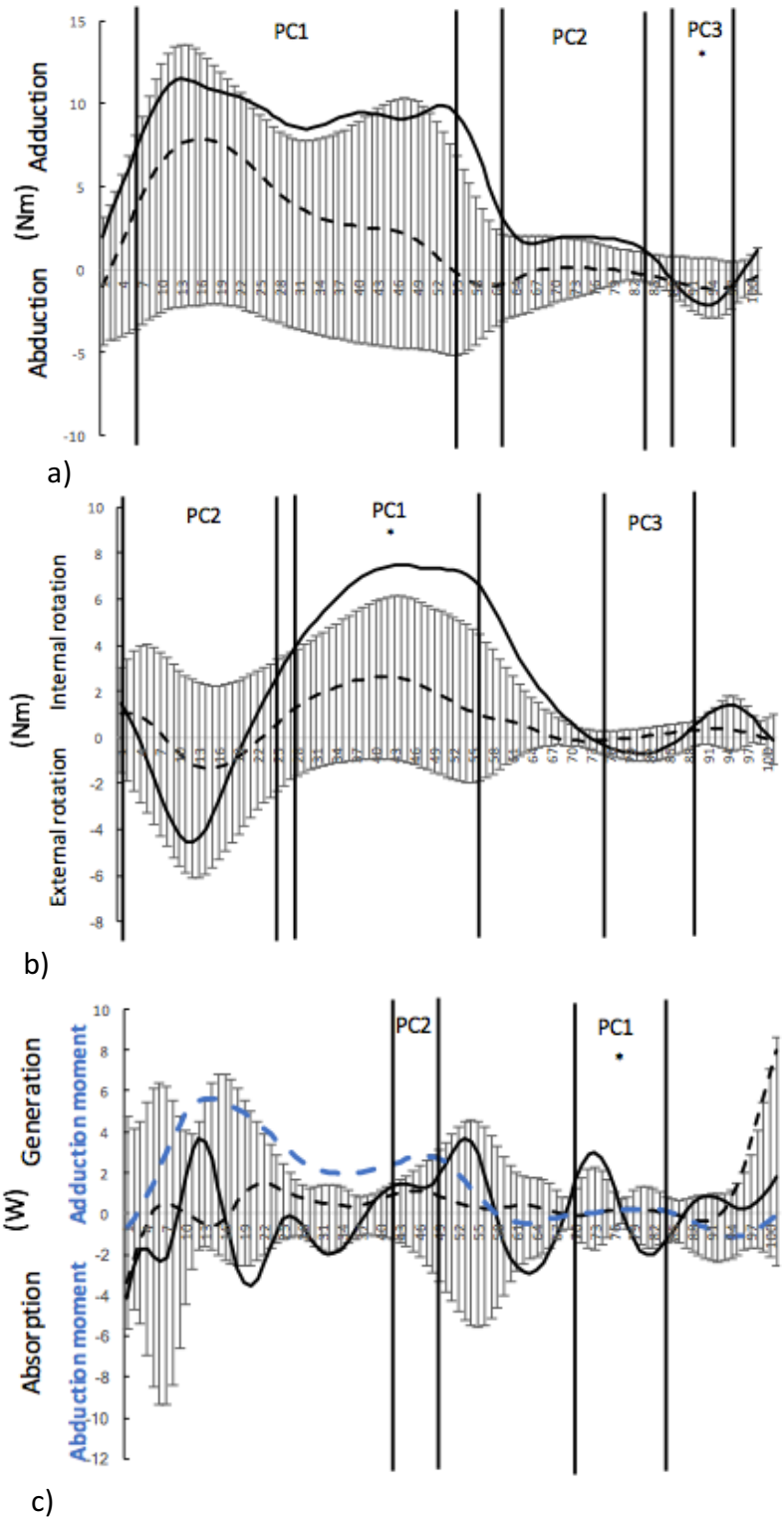

Figure 5.4.2. Knee moments (a-b), and frontal knee power (c), mean of five participants with highest body fat percentage (solid black line), mean of five participants with lowest body fat percentage (black dashed line) over the gait cycle standard deviation of the mean across all participants. Blue dashed lines represent mean joint moment for all participants. * denotes significant ($p<0.05$) prediction of body fat percentage on principal component scores.

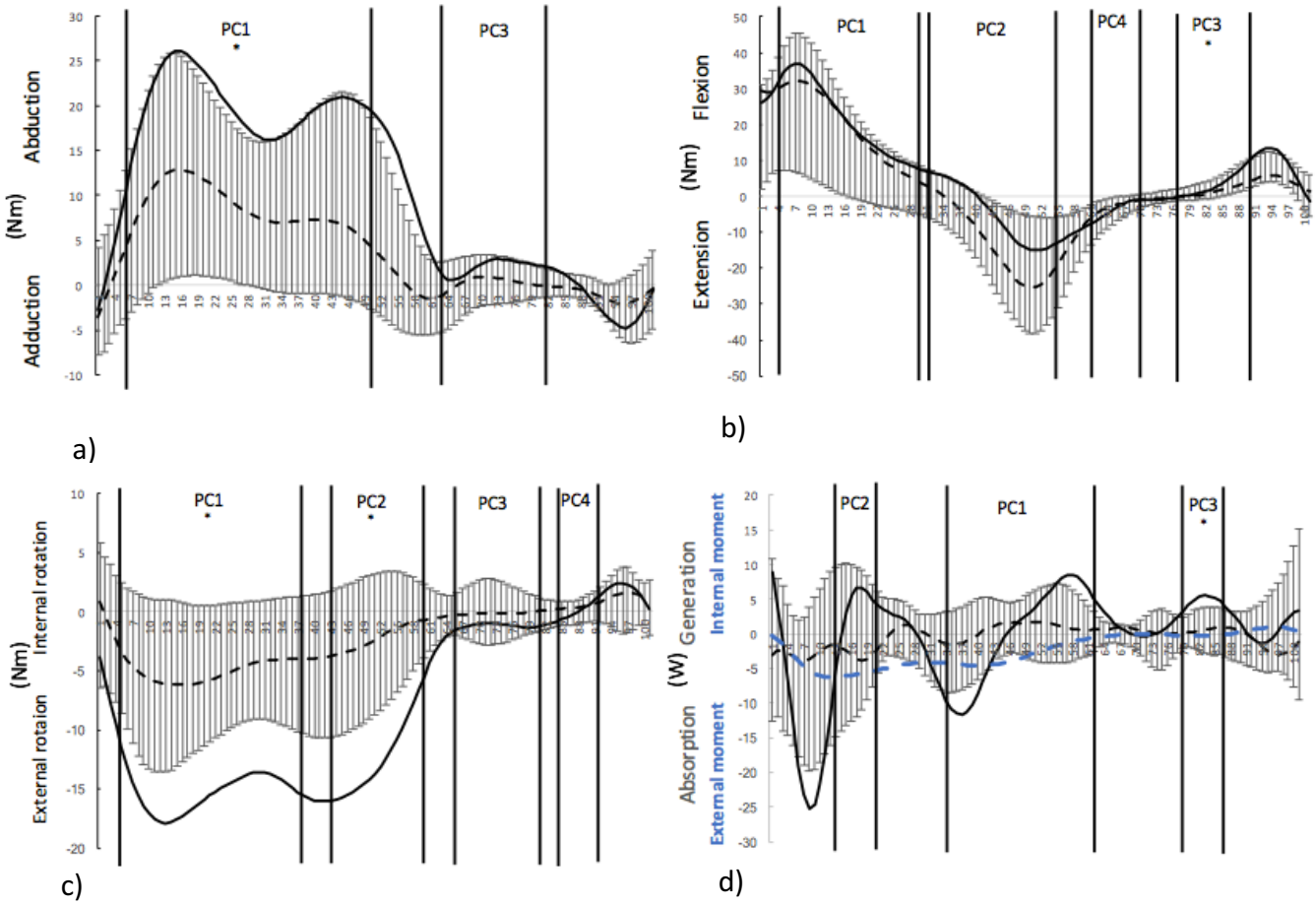

Figure 5.4.3. Hip moments (a-c), and transverse hip power (d), mean of five participants with highest body fat percentage (solid black line), mean of five participants with lowest body fat percentage (black dashed line) over the gait cycle standard deviation of the mean across all participants. Blue dashed lines represent mean joint moment for all participants. * denotes significant ($p<0.05$) prediction of body fat percentage on principal component scores.

Table 5.4.2. Summary of linear regression significant findings in 3D foot segment motion.

		Model						
		BF%		Walking velocity		BF%*Walking velocity		Predictor variable
		p	r2	p	r2	p	r2	Beta, (Std error)
Calcaneus - midfoot Sagittal	PC1	-	-	0.038	0.065	-	-	-
	PC2	-	-	0.000	0.292	0.000	0.402	0.324 (0.009)
Midfoot - metatarsal Transverse	PC1	0.032	0.07	-	-	-	-	-0.268 (0.010)
Shank - calcaneus Sagittal	PC1	-	-	0.002	0.139	0.008	0.154	-0.051 (0.011)
	PC2	-	-	0.000	0.238	-	-	-

Body fat percentage significantly predicted transverse midfoot to metatarsal angle throughout the gait cycle with the metatarsals being more abducted to the midfoot throughout with increasing body fat percentage. A mixed model of body fat percentage and walking velocity significantly predicted sagittal plane motion between the calcaneus and mid foot and shank and calcaneus. As body fat increased calcaneus to midfoot dorsiflexion increased at 48 to 60% ($F(2,62)=21.482$) of the gait cycle, and shank to calcaneus dorsiflexion decreased at 5 to 30% ($F(1,65)=4.812$ of the gait cycle.

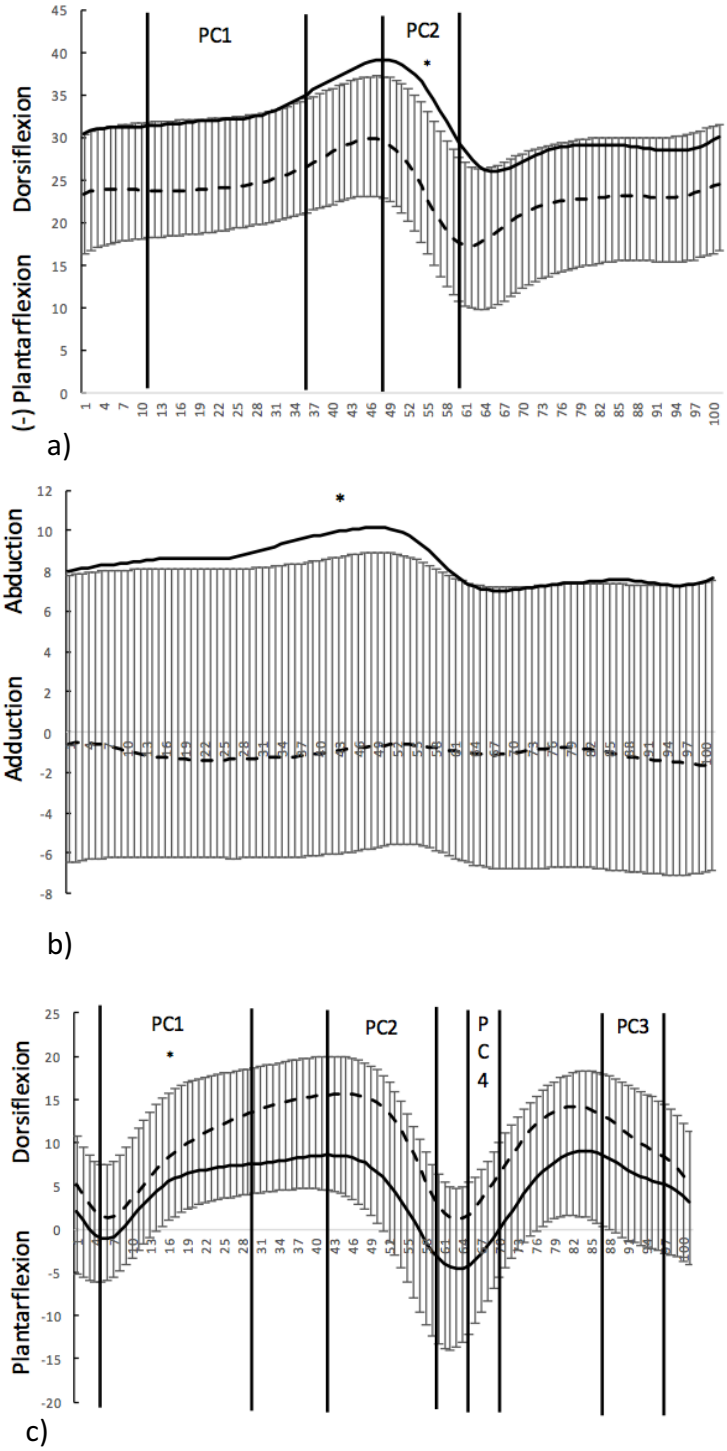

Figure 5.4.4. Foot segment angles (°) significantly predicted by body fat percentage over the gait cycle. a, Sagittal calcaneus to midfoot, b, Frontal midfoot to metatarsals and c, Sagittal shank to calcaneus. Solid black line is the mean angle of the 5 participants with the highest body fat percentage and dotted line is the mean of the 5 participants with the lowest body fat percentage. * denotes a significant ($p<0.05$) prediction of body fat percentage on segment angle.

Plantar Pressure.

Increasing body fat percentage increased plantar pressure at the midfoot at PC1 (11-51%) (R2=0.81, F(1,66)=5.702, p= 0.022), (β = .309, p= .011) and forefoot at PC3 (55-88%) (R2=.096, F(1,66)=5.702, p=0.020), (β =.284, p=.020). No significant effect of body fat percentage was found in axis angle, centre of pressure, or total area contact area.

Strength.

Absolute isometric knee extensor (F(3,66)=21.523) and flexor torque (F(2,66)=13.650) was significantly predicted by body fat percentage. Body fat percentage significant predicted absolute isokinetic ankle dorsiflexion (F(3,66)=13.170) and knee extension (F(2,66)=7.014). Power in the ankle dorsiflexors (F(3,66)=13.170) and knee extensors (F(2,66)=7.014).

Table 5.4.3. Summary of linear regression of body fat percentage, age and height with absolute torque and power.

						Model								
			BF%	Age	Height	Age*BF%*Height			BF%*Height			Age*BF%		
			p	p	p	p	r2	Beta	p	r2	Beta	p	r2	Beta
Isometric	Ankle	Dorsiflexion	0.861	0.352	0.299		-			-			-	
		Planterflexion	0.091	0.055	0.005		-			-			-	
	Knee	Flexion	0.001*	0.000	0.014	0.000	0.709	0.314		-			-	
		Extention	0.001*	0.083	0.000		-		0.000	0.544	0.376		-	
	Hip	Flexion	0.134	0.587	0.001		-			-			-	
		Extention	0.391	0.725	0.003		-			-			-	
		Abduction	0.162	0.705	0.000		-			-			-	
		Adduction	0.336	0.402	0.001		-			-			-	
Isokinetic	Ankle	Dorsiflexion	0.001*	0.028	0.002		-		0.000	0.618	0.376		-	
		Planterflexion	0.397	0.020	0.340		-			-			-	
	Knee	Flexion	0.069	0.002	0.152		-			-			-	
		Extention	0.025*	0.012	0.108		-			-		0.002	0.421	0.260
	Hip	Extention	0.875	0.513	0.000		-			-			-	
		Flexion	0.313	0.856	0.000		-			-			-	
Power	Ankle	Dorsiflexion	0.001*	0.028	0.002	0.000	0.618	0.345		-			-	
		Planterflexion	0.397	0.020	0.340		-			-			-	
	Knee	Flexion	0.069	0.002	0.152		-			-			-	
		Extention	0.025*	0.012	0.108		-			-		0.002	0.421	0.260
	Hip	Extention	0.875	0.513	0.000		-			-			-	
		Flexion	0.313	0.855	0.000		-			-			-	

Body fat percentage was significant in predicting isometric torque, isokinetic torque and power normalized to body weight in all muscle groups except isometric hip extension and flexion and isokinetic knee extension. For all significant variables as body fat increased, strength relative to body weight decreased.

Table 5.4.4. Linear regression of body fat percentage, with body weight normalised torque and power.

			p		R2	Beta
Isometric	Ankle	Dorsiflexion	0.002	*	0.138	-0.3246
		Planterflexion	0.048	*	0.058	-0.24
	Knee	Extention	0.014	*	0.087	-0.295
		Flexion	0.021	*	0.078	-0.28
	Hip	Flexion	0.106		-	-
		Extention	0.059		-	-
		Abduction	0.000	*	0.252	-0.502
		Adduction	0.041	*	0.062	-0.249
Isokinetic	Ankle	Dorsiflexion	0.044	*	0.046	-0.25
		Planterflexion	0.001	*	0.139	-0.39
	Knee	Flexion	0.014	*	0.089	-0.298
		Extention	0.079		-	-
	Hip	Extention	0.001	*	0.156	-0.395
		Flexion	0.023	*	0.076	-0.276
Power	Ankle	Dorsiflexion	0.044	*	0.06	-0.245
		Planterflexion	0.001	*	0.152	-0.39
	Knee	Flexion	0.016	*	0.085	-0.291
		Extention	0.080		0.046	-0.214
	Hip	Extention	0.001	*	0.149	-0.387
		Flexion	0.025	*	0.074	-0.272

Torque and power normalised to fat free mass was significantly predicted by body fat percentage in isokinetic ankle dorsiflexors (r^2=.061, F(1,67)=-4.261 ,p=0.0043), (β= -.246) and dorsiflexion power (r^2=.061, F(1,67)=-4.261 ,p=0.0043), (β= -.246).

5.5 Discussion

The aim of this study was to examine the relationships between gait analysis of lower limb and foot, plantar pressures and strength with body fat in children 7 to 11 years old. This study found, body fat percentage significantly predicted ankle moment and powers in all axis, frontal and transverse knee moment and frontal power frontal and sagittal hip moments and both transverse moments and powers in the hip. In the foot, body fat percentage significantly predicted sagittal plane calcaneus to midfoot and shank to calcaneus motion, and transverse midfoot to metatarsal movement. Plantar pressure under the midfoot and forefoot were significantly predicted by body fat percentage. Absolute isometric torque, isokinetic torque and power of the knee extensors, isometric knee flexors, isokinetic torque and power of the ankle were significantly predicted by body fat percentage. Body weight normalised strength significantly decreased with increasing body fat percentage in all variables, except isometric hip flexion and extension, and isokinetic knee extension.

Gait.

No significant effect of body fat percentage was found on pelvis or lower limb joint angles. This is similar to chapter 4, where no significant difference in weight status was found. As there was no effect of body fat percentage on kinematics, but effects on kinetics, this suggests significant effects of body fat are due to carrying excess mass and not in producing greater or altered joint angles (Morrison et al, 2012; Shultz et al., 2009)

Ankle abductor moment increased at 29-54% (estimated midstance to terminal swing) and 61-86% (estimated initial swing) as body fat increased. This is in contrast with Shultz et al. (2009) who found obese children to have greater peak inversion moment during stance phase. However, eversion (abduction) moments were not reported and only comparison of peaks between two groups were reported. During terminal stance the foot moves into its peak supination from pronation after contact with the ground, increased peak abductor moments here may be to control this move into an adducted position (Levine et al., 2012). During swing phase in normal gait there is relatively little torque generation, and ankle movements only involve position of the foot (Levine et

al., 2012). The increased abductor moments at 61 – 86% of the gait cycle may be due to a heavier and larger foot, or children with a larger body fat percentage progressing into phases later (Mcmillan et al., 2009). This may also require more work to be done during a shorter period of time as evident from the greater power generation during the same period (61-76% of gat cycle) with increasing body fat percentage. Similarly, greater body fat was associated with increased ankle internal rotation moments during midstance to terminal swing (25-55% of gait cycle) and initial to terminal swing (62-88% of gait cycle). The increased internal rotation moment and power may be to counter increased out toeing (external rotation) reported in obese children (Shultz, et al., 2014).

Body fat percentage significantly predicted sagittal ankle moment and power absorption. With increasing fat mass plantar flexion moment increased 56-87 % (estimated to be toe off to midswing) of the gait cycle. This is in agreement with Gushue et al. (2005) and Shultz et al. (2009) who reported obese children to have increased plantar flexion moments (95 ± 27 vs 67.6 ± 17 Nm and 97 ± 33.81cvs 93 ± 16.24 Nm respectively). This was suggested to be a mechanisms to help propel the larger mass and may impact upon the plantar pressures as seen in the results of this study (Shultz et al., 2009). Additionally, body fat percentage significantly predicted plantarflexion power absorption (eccentric action of plantarflexors) at 60-82% of the gait cycle. Participants with the highest body fat percentage had greater power absorption, whilst participants with the lowest body fat showed no plantarflexion absorption or power during the same period.

Sagittal plane knee moments and powers were not associated with body fat percentage. Knee abduction moments increased with body fat percentage at 58-77% of the gait cycle. Similar results have been reported on group comparisons of obese and non-obese children, with obese having higher peak abduction moments (Gushue et al., 2005; Shultz et al., 2009). This is coupled with a greater power absorption in the knee adductors at 70-84%, conceivably to help control the increased moment of abductors and maintain normal joint position. Shultz, et al. (2014) reported obese children to exhibit greater external knee rotation. The greater internal knee rotation

moment seen with increased fat mass in this study might also additionally be a mechanism to maintain normal joint motion.

Body fat percentage significantly predicted hip abduction, flexion and external rotation moments and internal rotation power generation. Abduction moment at 6 – 50% of the gait cycle increased with increasing body fat. This is in line with previously reported higher peak abduction moments in stance phase in obese children (Shultz et al., 2009). This is in response to the moving to single limb stance as the swing limb moves from heel off to toe off and initial swing, the stance limb must now support the pelvis on its own (Levine et al., 2012). These results suggest that those with increased body fat require greater abduction moment to maintain pelvic stability. Hip flexion moment increased at 77-90% of the gait cycle, to drive the heavier limb forward in swing phase (Shultz et al., 2014). External hip rotation moment increased with body fat at 5 - 38% and 43 - 60% of the gait cycle with a corresponding increased power absorption of the internal rotators 79 -87% of the gait cycle. Shultz et al. (2009) reported similar results with obese children having significantly larger external moments during stance (33.63 ± 13.68 vs 14.15 ± 4.50 Nm). Increased external rotation moments may be a compensatory mechanism for increased femoral anteversion, a toe in posture or increased tibial rotation often characterised in obese children (Riegger-Krugh & Keysor, 1996; Shultz et al., 2009).

Throughout the gait cycle the metatarsals were more abducted as body fat percentage increased indicating a more pronated foot type as body fat increases. Calcaneus to midfoot angle was more dorsiflexed at 48-60 % of the gait cycle, and the shank to calcaneus angle was more plantarflexed 5 to 30% of the gait cycle, with increasing body fat percentage. This is in line with Mahaffey et al. (2016) who found boys with increased fat mass to have more midfoot dorsiflexion and calcaneus plantarflexed throughout gait. This further suggests increased pronation with increased fat mass.

Plantar pressures.

Body fat percentage significantly predicted total midfoot contact area, as body fat percentage increased so did total contact area of the midfoot during gait. This suggest

a lowering of the medial longitudinal arch as seen on obese children in previous static trails (Riddiford-Harland et al., 2011). Furthermore, midfoot plantar pressures during 11-51% of stance phase increased with body fat percentage as well as forefoot plantar pressure 55 – 88% of stance phase. This is similar to previous findings of increased peak pressures in obese children under the midfoot and metatarsal heads (Dowling, Steele, Baur, 2004; Yan et al., 2013). However, the findings of this study have been able to highlight altered foot loading and increasing midfoot and forefoot pressures with increasing fat mass, which may increase the risk of foot pain and dysfunction.

Strength.

Absolute isometric knee flexion and extension, isokinetic dorsiflexion and knee extension, and dorsiflexion and knee extension power were all significantly predicted by body fat percentage and age or height. As body fat increased so did these absolute torque and power values. This suggests a training effect of carrying larger mass, similar to resistance training in these muscle groups (Garcia-Vicencio et al., 2016). Torque and power normalised to body mass was significantly predicted by body fat percentage in all variables except isometric hip extension, flexion and isokinetic knee extension. In significantly predicted strength/BM variables as body fat increased strength decreased. This suggests any training effect does not appear to be adequate for the increased body mass and excess non-contributory fat mass in these muscle groups (Cimolin et al., 2015). When strength was normalised to account for differences in fat free mass, body fat percentage only significantly predicted ankle dorsiflexion isokinetic torque and power. As adiposity increased ankle dorsiflexion decreased suggesting reduced ability to generate torque and power in those with greater body fat with same amount of fat free mass. In chapter four there was no significant difference between groups in absolute isokinetic ankle dorsiflexion and knee extension, however when the relationship of body fat percentage is examined there is a significant effect of body fat.

Limitations.

Although the results of this study suggest increased joint stress, risk of pain and associated musculoskeletal disorders in the foot and lower limb with increasing fat mass, the participants of this study had no reported pain or musculoskeletal disorders. Longitudinal work to examine the long-term effect of body fat on the musculoskeletal system in children would illustrate the links between increased joint stress and musculoskeletal disorders. Furthermore, the relationships between variables have not been explored here and may begin to provide causal relationship between increased fat mass, altered gait biomechanics, plantar pressures, and strength.

5.6 Conclusion

This study shows that children with higher body fat percentage generated greater joint moments, and powers in the hip, knee and ankle, whilst there was no effect on pelvis and lower limb joint angles. Meaning those with higher body fat percentage produce greater joint moments and powers due to the excess fat mass and not altered kinematics. Additionally, children with a higher body fat percentage have increased plantar pressures under the forefoot and midfoot. Increased joint moments and powers and plantar pressures put the children with a higher body fat percentage at risk of pain and joint dysfunction. Increased body fat produces a training effect on knee and ankle dorsiflexor muscle groups. However lower limb muscles are still weaker in those with higher body fat percentage relative to body mass except in the hip flexors and extensors and knee extensors.

6. Thesis Summary

6.1 Main aims and findings

The aims of this thesis were to investigate differences in obese and non-obese children's gait using waveform reduction to explain differences in kinetic, kinematic and simultaneously measured plantar pressure patterns over the gait cycle. Compare strength between the same paediatric cohort, at the hip, knee and ankle and investigate normalisation techniques for strength data. Secondly the thesis aimed to explore the relationship of adiposity on the same gait, plantar pressure and strength measures.

Gait.

Kinematic gait patterns were similar between OWB and HW groups, whilst the OWB group showed increased sagittal plane knee moments and frontal plane knee powers. This increases loading on joints and increase the risk of disorders such as Blounts disease on obese and overweight children.

The relationship of body fat percentage to gait revealed children with higher body fat percentage generated greater joint moments, and powers in the hip, knee and ankle. Similarly, to group comparisons no effect of adiposity was found on pelvis, hip, knee or ankle ankles. However, there was a significant effect of body fat on foot kinematics, which suggested a more pronated foot type throughout stance.

Similarly, between comparison of weight status groups and relationship of body fat, all lower limb joint angles were not effected. Meaning those with higher body fat percentage produce greater joint moments and powers due to the excess fat mass and not altered kinematics, increasing risk for joint pain and disorders. Weight status grouping did not highlight the effect of excess fat mass on moments and powers of the ankle, hip and transverse knee as seen in chapter 5. Additionally, weight status comparisons produced a significant effect of higher sagittal knee moments in OWB, that did not remain when the relationship fat body fat percentage on sagittal knee moment was examined. This suggests altered sagittal knee moments may be due to other factors increasing BMI such as higher fat free mass rather than excess fat mass.

Furthermore, comparisons of OWB and HW failed to show the significant effect of increased body fat on the foot during gait.

Plantar pressure.

OWB children show indicators of being less stable during stance phase, however further investigation including measures accounting for the overall larger foot width seen on OWB children is needed. Group comparisons revealed no difference in plantar pressure pattern under the forefoot, midfoot or rearfoot. However, when examining the relationship of body fat percentage to plantar pressure over stance phase children with a higher body fat percentage have increased plantar pressures under the forefoot and midfoot.

Weight status comparisons did not show the effect of increased fat mass on plantar pressures of the midfoot and forefoot. However, comparisons in weight status showed a greater mediolateral excursion of the centre of mass. Given the relationship of BMI to height and body mass, this may be an indication that instability is more associated with growth than obesity.

Strength.

Allometric scaling parameters for the lower limb was not effective in normalising strength to body mass in a cohort containing healthy weight, overweight and obese children. However, allometric scaling was effective in normalising to body fat percentage, fat mass and fat free mass. OWB were stronger in absolute terms at the knee, but no difference between groups at the hip or ankle were found. When scaled to body mass and fat mass OWB tended to be significantly weaker than HW suggesting inadequate strength per kg of mass.

The relationship of body fat to absolute strength showed a training effect on knee extensors and flexors and ankle dorsiflexors. However, lower limb muscles are still weaker in those with higher body fat percentage relative to body mass except in the hip flexors and extensors and knee extensors suggesting any training effect is not

adequate for increases in body mass except in the hip extensors and flexors and knee extension.

The use of weight status comparisons and relationship of body fat percentage to determine effect of obesity on absolute strength produced relatively similar results showing OWB or those with higher fat mass to be generally stronger in the knee extensors and flexors, ankle dorsiflexors whilst other joints movements were similar in HW or unaffected by body fat percentage. When normalised to body mass, weight status comparisons and body fat percentage relationship produced mixed results. Strength normalised to body weight was scientifically effected (reduced with increasing body fat) by body fat in all but isokinetic hip flexion and extension, weight status comparisons showed OBW to be weaker but not in all muscle groups. Additionally, comparisons based on weight status of strength scaled to fat free mass showed OWB to be significantly weaker all muscles groups but hip flexors. This suggested different muscle characteristics and reduced ability to produce strength in OWB. However, this was contradicted when the relationship of body fat percentage of fat free mass scaled strength was examined. There was no effect of body fat percentage on strength scaled for fat free mass except in ankle dorsiflexors. Therefore, the differences between groups in fat free mass scaled strength is possibly due to the inability of BMI and therefore BMI derived Z scores to distinguish between fat mass and fat free mass.

6.2 Clinical Implications

The findings of altered lower limb and foot biomechanics and increased plantar pressures in children with greater adiposity demonstrates a need for body fat to be reduced to prevent damage to the developing musculoskeletal system. A common recommendation for increasing physical activity to reduce adiposity is walking (Shultz et al., 2011). However, higher joint loading during walking may cause discomfort. Foot orthoses may aid in supporting the midfoot, and strengthening the relatively weaker muscles in the lower limb as demonstrated in this study, to improve strength to body weight ratio and balance joint moments.

6.3 Further research

Examining the relationship between lower limb strength, power, and gait biomechanics and plantar pressures in obese children would provide support for the previously expected relationship between gait and strength in obese children stated in previous literature. As well as provide more detailed information on which muscle groups may be most beneficial to be trained. Research using interventions such as strength training and orthortics would begin to provide information on effectiveness of these proposed methods on improving gait function in obese children.

References

Abdelmoula, A., Martin, V., Bouchant, A., Walrand, S., Lavet, C., Taillardat, M., … Ratel, S. (2012). Knee extension strength in obese and nonobese male adolescents. *Applied Physiology, Nutrition, and Metabolism*, *37*(2), 269–275.

Ball, E. J., O'Connor, J., Abbott, R., Steinbeck, K. S., Davies, P. S., Wishart, C., … Baur, L. A. (2001). Total energy expenditure, body fatness, and physical activity in children aged 6-9 y. *The American Journal of Clinical Nutrition*, *74*(4), 524–8.

Bechard, L., Wroe, E., & Ellis, K. (2008). *Body composition and growth.* (C. Duggan, J. Watkins, & A. Walker, Eds.).

Benson, E. C., Miller, M., Bosch, P., & Szalay, E. A. (2008). A New Look at the Incidence of Slipped Capital Femoral Epiphysis in New Mexico. *Journal of Pediatric Orthopaedics*, *28*(5), 529–533.

Bizovska, L., Svoboda, Z., Kutilek, P., Janura, M., Gaba, A., & Kovacikova, Z. (2014). Variability of centre of pressure movement during gait in young and middle-aged women. *Gait and Posture*, *40*(3), 399–402.

Blimkie, C. J., Ebbesen, B., MacDougall, D., Bar-Or, O., & Sale, D. (1989). Voluntary and electrically evoked strength characteristics of obese and nonobese preadolescent boys. *Human Biology*, *61*(4), 515–32.

Bordin, D., De Giorgi, G., Mazzocco, G., & Rigon, F. (2001). Flat and cavus foot, indexes of obesity and overweight in a population of primary-school children. *Minerva Pediatrica*, *53*(1), 7–13.

Bout-Tabaku, S., Briggs, M. S., & Schmitt, L. C. (2013). Lower extremity pain is associated with reduced function and psychosocial health in obese children pediatrics. *Clinical Orthopaedics and Related Research*, *471*(4), 1236–1244.

Carriero, A., Zavatsky, A., Stebbins, J., Theologis, T., & Shefelbine, S. J. (2009). Correlation between lower limb bone morphology and gait characteristics in children with spastic diplegic cerebral palsy. *Journal of Pediatric Orthopaedics*, *29(1)*, 73–79.

Cau, N., Cimolin, V., Galli, M., Precilios, H., Tacchini, E., Santovito, C., & Capodaglio, P. (2014). Center of pressure displacements during gait initiation in individuals with obesity. *Journal of Neuroengineering and Rehabilitation*, *11*(1), 82.

Chan, G., & Chen, C. T. (2009). Musculoskeletal effects of obesity. *Current Opinion in Pediatrics*, *21*, 65–70.

Chau, T. (2001). A review of analytical techniques for gait data. Part 2: neural network and wavelet methods. *Gait & Posture*, *13*(2), 102–120.

Chester, V. L., & Wrigley, A. T. (2008). The identification of age-related differences in kinetic gait parameters using principal component analysis. *Clinical Biomechanics*, *23*(2), 212–220.

Cimolin, V., Galli, M., Vismara, L., Albertini, G., Sartorio, A., & Capodaglio, P. (2015). Gait pattern in lean and obese adolescents. *International Journal of Rehabilitation Research. Internationale Zeitschrift Für Rehabilitationsforschung. Revue Internationale de Recherches de Réadaptation*, *38*(1), 40–8.

Cole, T. J., Freeman, J. V., Preece, M. A. (1995). Body mass index reference curves for the UK, 1990. *Archives of Disease in Childhood, 73(1)*, 25-29.

Cook, S. D., Lavernia, C. J., Burke, S. W., Skinner, H. B., & Haddad, R. J. (1983). A biomechanical analysis of the etiology of tibia vara. *Journal of Pediatric Orthopedics*, *3*(4), 449–454.

Cousins, S. D., Morrison, S. C., & Drechsler, W. I. (2013). Foot loading patterns in normal weight, overweight and obese children aged 7 to 11 years. *Journal of Foot and Ankle Research*, *6*(1), 36.

Da Rocha, E. S., Bratz, D. T. K., Gubert, L. C., De David, A., & Carpes, F. P. (2014). Obese children experience higher plantar pressure and lower foot sensitivity than non-obese. *Clinical Biomechanics*, *29*(7), 822–827.

De Sá Pinto, A. L., De Barros Holanda, P. M., Radu, A. S., Villares, S. M. F., & Lima, F. R. (2006). Musculoskeletal findings in obese children. *Journal of Paediatrics and Child Health, 42(6), 341-344*.

DeVita, P., & Hortobagyi, T. (2003). Obesity is not associated with increased knee joint torque and power during level walking. *Journal of Biomechanics*, *36*, 1355–1362.

Ding, C., Cicuttini, F., Scott, F., Cooley, H., & Jones, G. (2005). Knee structural alteration and BMI: a cross-sectional study. *Obesity Research*, *13*(2), 350–61.

Dowling, M., Steele, J. R., & Baur, L. (2004). What are the effects of obesity in children on plantar pressure distributions? *International Journal of Obesity and Related Metabolic Disorders : Journal of the International Association for the Study of Obesity*, *28*(11), 1514–1519.

Dufek, J. S., Currie, R. L., Gouws, P. L., Candela, L., Gutierrez, A. P., Mercer, J. A., & Putney, L. G. (2012). Effects of overweight and obesity on walking characteristics in adolescents. *Human Movement Science*, *31*(4), 897–906.

Eng, J. J., & Winter, D. A. (1995). Kinetic analysis of the lower limbs during walking: What information can be gained from a three-dimensional model? *Journal of Biomechanics*, *28*(6), 753–758.

Freedman Silvernail, J., Milner, C. E., Thompson, D., Zhang, S., & Zhao, X. (2013). The influence of body mass index and velocity on knee biomechanics during walking. *Gait and Posture*, *37*(4), 575–579.

Fields, D. A., Hull, H. R., Cheline, A. J., Yao, M. and Higgins, P. B. (2004). 'Child-specific thoracic gas volume prediction equations for air-displacement plethysmography'. Obesity Research, 12(11), 1797-1804.

Galbraith, R. T., Gelberman, R. H., Hajek, P. C., Baker, L. a, Sartoris, D. J., Rab, G. T., … Griffin, P. P. (1987). Obesity and decreased femoral anteversion in adolescence. *Journal of Orthopaedic Research*, *5*(4), 523–528.

Garcia-Vicencio, S., Coudeyre, E., Kluka, V., Cardenoux, C., Jegu, A.-G., Fourot, A.-V., … Martin, V. (2016). The bigger, the stronger? Insights from muscle architecture and nervous characteristics in obese adolescent girls. *International Journal of Obesity (2005)*, *40*(2), 245–251.

Gately, P. J., Radley, D., Cooke, C. B., Carroll, S., Oldroyd, B., Truscott, J. G., … Wright, A. (2003). Comparison of body composition methods in overweight and obese children. *Journal of Applied Physiology*, *95*(5), 2039–46.

Gillman, M. W. M. W. (2008). The first months of life: a critical period for development of obesity. *American Journal of Clinical Nutrition*, *87*(6), 1587–1589.

Gushue, D. L., Houck, J., & Lerner, A. L. (2005). Effects of childhood obesity on three-dimensional knee joint biomechanics during walking. *Journal of Pediatric Orthopedics*, *25*(6), 763–768.

Haycock GB, Schwartz GJ, W. D. (1978). Geometric method for measuring body surface area: A height weight formula validated in infants, children and adults. *The Journal of Pediatrics*, *93*(1), 62–66.

Hills, P., & Parker, W. (1992). Locomotor characteristics of obese children. *Child: Care, Health and Development*, *18*(1), 29–34.

HSCIC. (2015). *Statistics on Obesity, Physical Activity and Diet*. Leeds. Retrieved from http://www.hscic.gov.uk/catalogue/PUB16988/obes-phys-acti-diet-eng-2015.pdf

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*(sup2), S72–S79.

Hubley-Kozey, C., & Earl, E. M. (2000). Coactivation of the ankle musculature during maximal isokinetic dorsiflexion at different angular velocities. *European Journal of Applied Physiology*, *82*(4), 289–296.

Iossifidou, A. N., & Baltzopoulos, V. (2000). Peak power assessment in isokinetic dynamometry. *European Journal of Applied Physiology*, *82*(1–2), 158–160.

Jaric, S. (2002). Muscle Strength Testing: Use of Normalisation for Body Size. *Sports Medicine*, *32*(10), 615–631.

Jiménez-Ormeño, E., Aguado, X., Delgado-Abellán, L., Mecerreyes, L., & Alegre, L. M. (2013). Foot morphology in normal-weight, overweight, and obese schoolchildren. *European Journal of Pediatrics*, *172*(5), 645–652.

John, T. J., Seth, A., Schwartz, M. H., & Delp, S. L. (2012). Contributions of muscle to mediolatereal ground reaction force over a range of walking speeds. *Journal of Biomechanics*, *45*(14), 2438–2443.

Johnson, W., Li, L., Kuh, D., & Hardy, R. (2015). How Has the Age-Related Process of Overweight or Obesity Development Changed over Time? Co-ordinated Analyses of Individual Participant Data from Five United Kingdom Birth Cohorts. *PLoS Medicine*, *12*(5), 1–20.

Karavelioglu, M. B., Harmanci, H., & Caliskan, G. (2017). Gender differences in hand grip strength of the child athletes by using absolute, ratio and allometric scaling methods. *Biomedical Research (India)*, *28*(4), 1533–1537.

Kelsey, J. L., Acheson, R. M., Keggi, K. J., Haven, N., & Haven, N. (2015). The body build of patients with slipped capital femoral epiphysis. *American Journal of Deseases of Children, 124(2),* 276-281.

Kim, H. W., & Weinstein, S. L. (2000). Flatfoot in children: Differential diagnosis and management. *Current Orthopaedics*, *14*(6), 441–447.

Kohrt, W. M., Barry, D. W., & Schwartz, R. S. (2011). Muscle forces or gravity: What predominates mechanical loading on bone? *Medicine & Science in Sports & Exercise*, *41*(11), 2050–2055.

Kopelman, P., Caterson, A., & Dietz, W. (2009). *Clinical obesity in adults and children* (3rd ed.). Oxford: Wiley-Blackwell.

Kuo, A. D., & Donelan, J. M. (2010). Dynamic Principles of Gait and Their Clinical Implications. *Physical Therapy*, *90*(2), 157–174.

Leardini, A., Benedetti, M. G., Berti, L., Bettinelli, D., Nativo, R., & Giannini, S. (2007). Rear-foot, mid-foot and fore-foot motion during the stance phase of gait. *Gait & Posture*, *25*(3), 453–462.

Lerner, Z. F., Board, W. J., & Browning, R. C. (2014). Effects of obesity on lower extremity muscle function during walking at two speeds. *Gait & Posture*, *39*(3), 978–984.

Lerner, Z. F., Board, W. J., & Browning, R. C. (2016). Pediatric obesity and walking duration increase medial tibiofemoral compartment contact forces. *Journal of Orthopaedic Research : Official Publication of the Orthopaedic Research Society*, *34*(1), 97–105.

Lerner, Z. F., & Browning, R. C. (2016). Compressive and shear hip joint contact forces are affected by pediatric obesity during walking. *Journal of Biomechanics, 49,* 1547-1553..

Lerner, Z. F., Shultz, S. P., Board, W. J., Kung, S., & Browning, R. C. (2014). Does adiposity affect muscle function during walking in children? *Journal of Biomechanics*, *47*(12), 2975–2982.

Lerner, Z. F., Shultz, S. P., Board, W. J., Kung, S., & Browning, R. C. (2014). Does adiposity affect muscle function during walking in children? *Journal of Biomechanics*, *47*(12), 2975–2982.

Levangie, K., & Norkin, C. (2005). *Joint Structure and Function: A Comprehensive Analysis* (5th ed.). Philidelphia: F. A. Davis Company.

Levine, A. M., & Drennan, J. C. (1982). Physiological bowing and tibia vara. The metaphyseal-diaphyseal angle in the measurement of bowleg deformities. *The Journal of Bone and Joint Surgery. American Volume*, *64*(8), 1158–63.

Levine, D., Richards, J., & Whittle, M. W. (2012). *Whittle's Gait Analysis* (5th ed.). Elsevier Health Sciences.

Lobstein, T., & Jackson-Leach, R. (2016). Planning for the worst: estimates of obesity and comorbidities in school-age children in 2025. *Pediatric Obesity*, *11*(5), 321–325.

Lohman, T. G. (1986). Applicability of body composition techniques and constants for children and youths. *Exercise and Sport Sciences Reviews, 14,* 325-367.

Maffiuletti, N. A., Jubeau, M., Agosti, F., Col, A., & Sartorio, A. (2008). Quadriceps muscle function characteristics in severely obese and nonobese adolescents. *European Journal of Applied Physiology*, *103*(4), 481–484.

Mahaffey, R., Morrison, S. C., Bassett, P., Drechsler, W. I., & Cramp, M. C. (2016). Gait & Posture The impact of body fat on three dimensional motion of the paediatric foot during walking. *Gait & Posture*, *44*, 155–160.

Mahaffey, R., Morrison, S. C., Stephensen, D., & Drechsler, W. I. (2016). Clinical outcome measures for monitoring physical function in pediatric obesity: An integrative review. *Obesity*, *24*(5), 993–1017.

Mahaffey, R., Morrison, S., Drechsler, W., & Cramp, M. (2012). Reliability of three foot models to examine paediatric gait. *Journal of Foot and Ankle Research*, *5*(Suppl 1), O18.

Manoff, E. M., Banffy, M. B., & Winell, J. J. (2005). Relationship between Body Mass Index and slipped capital femoral epiphysis. *Journal of Pediatric Orthopedics*, *25*(6), 744–6.

Mauch, M., Grau, S., Krauss, I., Maiwald, C., & Horstmann, T. (2008). Foot morphology of normal, underweight and overweight children. *International Journal of Obesity (2005)*, *32*(7), 1068–1075.

McGraw, B., McClenaghan, B. A., Williams, H. G., Dickerson, J., & Ward, D. S. (2000). Gait and postural stability in obese and nonobese prepubertal boys. *Archives of Physical Medicine and Rehabilitation*, *81*(4), 484–489.

McMillan, A. G., Auman, N. L., Collier, D. N., & Blaise Williams, D. S. (2009). Frontal Plane Lower Extremity Biomechanics During Walking in Boys Who Are Overweight Versus Healthy Weight. *Pediatric Physical Therapy*, *21*(2), 187–193.

Mcmillan, A. G., Auman, N. L., Collier, D. N., & Williams, D. S. B. (2009). Frontal plane lower extremity biomechanics during walking in boys who are overweight versus helathy weight. *Pediatric Physical Therapy, 21,* 187-193.

McMillan, A. G., Pulver, A. M. E., Collier, D. N., & Williams, D. S. B. (2010). Sagittal and frontal plane joint mechanics throughout the stance phase of walking in adolescents who are obese. *Gait and Posture*, *32*(2), 263–268.

Metcalf, B. S., Hosking, J., Jeffery, A. N., Voss, L. D., Henley, W., & Wilkin, T. J. (2011). “Fatness leads to inactivity, but inactivity does not lead to fatness- A longitudinal study in children (earlybird 45).” *Archives of Disease in Childhood*, *96*, 942–947.

Mitchell, J. A., Dowda, M., Pate, R. R., Kordas, K., Froberg, K., Sardinha, L. B., … Page, A. (2016). Physical Activity and Pediatric Obesity: A Quantile Regression Analysis. *Medicine and Science in Sports and Exercise*, 466–473.

Morgan, L., Monica, D. (2010). *The economic burden of obesity*. Oxford:National Obesity Observatory.

Morrison, S. C., Mahaffey, R. D., Cousins, S. D., & Drechsler, W. I. (2012). Current Understanding of the Impact of Childhood Obesity on the Foot and Lower Limb. *The Association of Peadriatric Chartered Physiotherapists*, *3*(2), 5–11.

Mueller, S., Carlsohn, A., Mueller, J., Baur, H., & Mayer, F. (2016). Influence of obesity on foot loading characteristics in gait for children aged 1 to 12 years. *PLoS ONE*, *11*(2), 1–12.

Murray, A. W., & Wilson, N. I. L. (2008). Changing incidence of slipped capital femoral epiphysis: A relationship with obesity? *Journal of Bone and Joint Surgery*, *90–B*(1), 92–94.

Nantel, J., Brochu, M., & Prince, F. (2006). Locomotor strategies in obese and non-obese children. *Obesity, 14(10)*, 1789-94.

NCMP, (2016). National Child Measurement Programme: England, 2015/16 school year. *Health and Social Care and Information Centre.*

Old, S., Drechsler, W., & Stephensen, D. (2007). Feasability and reliability fo knee muscle strength testing and ultrasound imaging of vastus lateralis in 6-12 year old children. *Physiotherapy*, *93*, S104.

Payton, C., & Bartlett, R. (2008a). *Biomechanical Evaluation of Movement in Sport and Exercise*. London: Routledge.

Peyrot, N., Thivel, D., Isacco, L., Morin, J.-B., Duche, P., & Belli, A. (2009). Do mechanical gait parameters explain the higher metabolic cost of walking in obese adolescents? *Journal of Applied Physiology*, *106*(6), 1763–70.

Pirpiris, M., Jackson, K. R., Farng, E., Bowen, R. E., & Otsuka, N. Y. (2006). Body mass index and Blount disease. *Journal of Pediatric Orthopedics*, *26*(5), 659–63.

Pulgarón, E. R. (2014). Childhood Obesity: A Review of Increased Risk for Physical and Psychological Co-morbidities. *Clinical Therapeutics*, *35*(1), 1–21.

Public Health England. (2017). *Changes in the weight status of children between the first and final years of primary schoo*l. Crown Copywrite.

RCPCH. (2015). *Tackling England ' s childhood obesity crisis.* London.

Riddiford-Harland, D. L., Steele, J. R., & Baur, L. a. (2011). Are the feet of obese children fat or flat? Revisiting the debate. *International Journal of Obesity*, *35*(1), 115–120.

Riegger-Krugh, C., & Keysor, J. J. (1996). Skeletal malalignments of the lower quarter: correlated and compensatory motions and postures. *The Journal of Orthopaedic and Sports Physical Therapy*, *23*(2), 164–170.

Rose, K. J., Burns, J., & North, K. N. (2009). Relationship between foot strength and motor function in preschool-age children. *Neuromuscular Disorders, 19,* 104-107.

Rutherford, D. J., & Hubley-Kozey, C. (2009). Explaining the hip adduction moment variability during gait: Implications for hip abductor strengthening. *Clinical Biomechanics*, *24*(3), 267–273.

Sabharwal, S., & Root, M. Z. (2012). Impact of Obesity on Orthopaedics. *The Journal of Bone and Joint Surgery-American Volume*, *94*(11), 1045–1052.

Sabharwal, S., Zhao, C., & McClemens, E. (2007). Correlation of Body Mass Index and Radiographic Deformities in Children with Blount Disease. *The Journal of Bone and Joint Surgery (American)*, *89*(6), 1275–1283.

SACN and RCPCH expert group. (2012). *Position Statement: Consideration of issues around the use of BMI centile thresholds for defining underweight , overweight and obesity in children aged 2-18 years in the UK*.

Sadeghi-Demneh, E., Azadinia, F., Jafarian, F., Shamsi, F., Melvin, J. M. A., Jafarpishe, M., & Rezaeian, Z. (2016). Flatfoot and obesity in school-age children: a cross-sectional study. *Clinical Obesity*, *6*(1), 42–50.

Scarborough, P., Bhatnagar, P., Wickramasinghe, K. K., Allender, S., Foster, C., & Rayner, M. (2011). The economic burden of ill health due to diet, physical inactivity, smoking, alcohol and obesity in the UK: An update to 2006-07 NHS costs. *Journal of Public Health*, *33*(4), 527–535.

Shultz, S., Kagawa, M., Fink, P. W., & Hills, A. P. (2014). Knee Alignment can help predict sedentry behaviour in children. *The Journal of Sports Medicine and Physical Fitness*, *54*, 631–635.

Shultz, S. P., Anner, J., & Hills, A. P. (2009). Paediatric obesity, physical activity and the musculoskeletal system. *Obesity Reviews*, *10*(5), 576–582.

Shultz, S. P., Browning, R. C., Schutz, Y., Maffeis, C., & Hills, A. P. (2011). Childhood obesity and walking: guidelines and challenges. *International Journal of Pediatric Obesity*, *6*(5–6), 332–341.

Shultz, S. P., D'Hondt, E., Fink, P. W., Lenoir, M., & Hills, A. P. (2014). The effects of pediatric obesity on dynamic joint malalignment during gait. *Clinical Biomechanics*, *29*(7), 835–838.

Shultz, S. P., D'Hondt, E., Lenoir, M., Fink, P. W., & Hills, A. P. (2014). The role of excess mass in the adaptation of children's gait. *Human Movement Science*, *36*, 12–19.

Shultz, S. P., Hills, A. P., Sitler, M. R., & Hillstrom, H. J. (2010). Body size and walking cadence affect lower extremity joint power in children's gait. *Gait and Posture*, *32*(2), 248–252.

Shultz, S. P., Sitler, M. R., Tierney, R. T., Hillstrom, H. J., & Song, J. (2009). Effects of Pediatric Obesity on Joint Kinematics and Kinetics During 2 Walking Cadences. *Archives of Physical Medicine and Rehabilitation*, *90*(12), 2146–2154.

Siri, W. (1961). Body composition frm fluid spaces and density: analysis of methods. In *Techniques for measuring body composition,* (pp. 223–244).

Smith, S. M., Sumar, B., & Dixon, K. a. (2014). Musculoskeletal pain in overweight and obese children. *International Journal of Obesity (2005)*, *38*(1), 11–5.

Stolzman, S., Irby, M. B., Callahan, A. B., & Skelton, J. A. (2015). Pes planus and paediatric obesity: a systematic review of the literature. *Clinical Obesity*, *5*(2), 52–59.

Stovitz, S. D., Pardee, P. E., Vazquez, G., Duval, S., & Schwimmer, J. B. (2008). Musculoskeletal pain in obese children and adolescents. *Acta Paediatrica*, *97*(4), 489–493.

Sutherland, D. (1997). The development of mature gait. *Gait and Posture*, *6*(2), 163–170.

Svoboda, Z., Bizovská, L., Kováčiková, Z., Kutílek, P., Cuberek, R., & Janura, M. (2015). Effect of lower limb preference on variability of centre of pressure movement during gait. *Acta Gymnica*, *45*(4), 203–207.

Taisa Filippin, N., de Almeida Bacarin, T., & Lobo da Costa, P. H. (2008). Comparison of static footprints and pedobarography in obese and non-obese children. *Foot & Ankle International*, *29*(11), 1141–4.

Taylor, E. D., Theim, K. R., Mirch, M. C., Ghorbani, S., Tanofsky-Kraff, M., ADler-Wailes, D. C., … Yanovski, J. A. (2006). Orthopedic Complications of Overweight in Children and Adolescents. *Pediatrics*, *117*(6), 2167–2174.

Thivel, D., Ring-Dimitriou, S., Weghuber, D., Frelut, M.-L., & O'Malley, G. (2016). Muscle Strength and Fitness in Pediatric Obesity: a Systematic Review from the European Childhood Obesity Group. *Obesity Facts*, *9*(1), 52–63.

Tsiros, M. D., Buckley, J. D., Howe, P. R. C., Walkley, J., Hills, A. P., & Coates, A. M. (2014). Musculoskeletal Pain in Obese Compared with Healthy-weight Children. *The Clinical Journal of Pain*, *30*(7), 1–16.

Tsiros, M. D., Coates, A. M., Howe, P. R. C., Grimshaw, P. N., & Buckley, J. D. (2011). Obesity: The new childhood disability? *Obesity Reviews*.

Tsiros, M. D., Coates, A. M., Howe, P. R. C., Grimshaw, P. N., Walkley, J., Shield, A., … Buckley, J. D. (2013). Knee extensor strength differences in obese and healthy-weight 10-to 13-year-olds. *European Journal of Applied Physiology*, *113*(6), 1415–1422.

Van Boerum, D. H., & Sangeorzan, B. J. (2003). Biomechanics and pathophysiology of flat foot. *Foot and Ankle Clinics*, *8*(3), 419–30.

van der Krogt, M. M., Delp, S. L., & Schwartz, M. H. (2012). How robust is human gait to muscle weakness? *Gait & Posture*, *36*(1), 113–119.

Villarroya, M. A., Esquivel, J. M., Tomás, C., Moreno, L. A., Buenafé, A., & Bueno, G. (2009). Assessment of the medial longitudinal arch in children and adolescents with obesity: Footprints and radiographic study. *European Journal of Pediatrics*, *168*(5), 559–567.

Villarroya, M., Esquivel, J., Tomás, C., Buenafé, A., & Moreno, L. (2008). Foot structure in overweight and obese children. *International Journal of Pediatric Obesity : IJPO : An Official Journal of the International Association for the Study of Obesity*, *3*(1), 39–45.

Wang, Y., & Chen, H.-J. (2012). *Use of Percentiles and Z-Scores in Anthropometry. In Handbook of Anthropometry* (pp. 29–48). New York, NY: Springer New York.

Wearing, S. C., Hennig, E. M., Byrne, N. M., Steele, J. R., & Hills, A. P. (2006). Musculoskeletal disorders associated with obesity : a biomechanical perspective. *Obesity Reviews*, *7*, 239–250.

Weber, D. R., Leonard, M. B., Zemel, B. S. (2012) Body composition analysis in the pediatric population. *Pediatric endocrinology reviews, 10(1),* 130-139.

WHO. (2016). *Report of the commision on ending childhood obesity*. Geneva: World Health Organization.

Wills, M. (2004). Orthopedic complications of childhood obesity. *Pediatr Phys Ther*, *16*(4), 230–235.

Winter, D. (2009). *Biomechanics and Motor Control of Human Movement*. New Jersey: John Wiley & Sons.

Wren, T. A., & Engsberg, J. R. (2007). Normalizing Lower-Extremity Strength Data for Children Without Disability Using Allometric Scaling. *Archives of Physical Medicine and Rehabilitation*, *88*(11), 1446–1451.

Yan, S. H., Zhang, K., Tan, G. Q., Yang, J., & Liu, Z. C. (2013). Effects of obesity on dynamic plantar pressure distribution in Chinese prepubescent children during walking. *Gait and Posture*, *37*(1), 37–42.

Yates, B. (2009). *Merriman's Assessment of the Lower Limb* (3rd ed.). Churchill Livingstone.

Yu, Z. B., Han, S. P., Zhu, G. Z., Zhu, C., Wang, X. J., Cao, X. G., & Guo, X. R. (2011). Birth weight and subsequent risk of obesity: A systematic review and meta-analysis. *Obesity Reviews*, *12*(7), 525–542.