The impact of childhood obesity on musculoskeletal form

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Summary

Despite the greater prevalence of musculoskeletal disorders in obese adults, the consequences of childhood obesity on the development and function of the musculoskeletal system have received comparatively little attention within the literature. Of the limited number of studies performed to date, the majority have focused on the impact of childhood obesity on skeletal structure and alignment, and to a lesser extent its influence on clinical tests of motor performance including muscular strength, balance and locomotion. Although collectively these studies imply that the functional and structural limitations imposed by obesity may result in aberrant lower limb mechanics and the potential for musculoskeletal injury, empirical verification is currently lacking. The delineation of the effects of childhood obesity on musculoskeletal structure in terms of mass, adiposity, anthropometry, metabolic effects and physical inactivity, or their combination, has not been established. More specifically, there is a lack of research regarding the effect of childhood obesity on the properties of connective tissue structures, such as tendons and ligaments. Given the global increase in childhood obesity, there is a need to ascertain the consequences of persistent obesity on musculoskeletal structure and function. A better understanding of the implications of childhood obesity on the development and function of the musculoskeletal system would assist in the provision of more meaningful support in the prevention, treatment and management of the musculoskeletal consequences of the condition.

Keywords: Biomechanics, motor performance, obesity.

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Introduction

Obesity is recognized as a major global burden to health (1). Adult-onset obesity is associated with an increased risk of developing, what seems to be an ever increasing number of medical complications, including type 2 diabetes, hypertension, cardiovascular disease, respiratory maladies, gout and musculoskeletal disorders (2). Although these obesity-associated morbidities occur more frequently in adults, many of the cardiovascular consequences that typify adult-onset obesity are heralded by abnormalities that commence in childhood (3). Hypertension, insulin resistance, endothelial dysfunction and dyslipidaemia, once hallmarks of adult cardiovascular disease, are now common in obese children and adolescents (4). While it is currently unknown if early onset obesity in children carries a greater risk of developing associated morbidities in adulthood, the prevalence of childhood overweight and obesity is steadily increasing, and now affects as much as one in four Australian children (5,6).

Although adult-onset obesity has been associated with a greater prevalence of musculoskeletal disorders, primarily involving the lower limb (7–9), comparative data in children are lacking. There is, however, anecdotal evidence that

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persistent weight-bearing associated with childhood obesity may inhibit normal movement patterns and predispose children to musculoskeletal pain and injury (10–13). It is also known that overweight and obesity is a risk factor for the occurrence of childhood injury (14) and may also be associated with an adverse clinical prognosis following injury (15). Despite such findings, the consequences of childhood obesity on the development and function of the musculoskeletal system have received comparatively little attention within the literature. Of the limited number of studies performed to date, the majority have focused on the effect of childhood obesity on skeletal structure and alignment, and to a lesser extent its influence on clinical tests of motor performance.

With the rapid rise in childhood obesity (1,16) and the growing concern over the prevalence of musculoskeletal injury (17), there is a need to establish the long-term consequences of adiposity on the ontogenic development of the musculoskeletal system in children and its impact upon locomotor function and subsequent progression of musculoskeletal disorders.

The primary aim of this paper therefore is to review the effects of overweight and obesity on the musculoskeletal structure of the lower limb in children and, where possible, to highlight the impact of childhood obesity on musculoskeletal function. In particular, this review will focus on the influence of obesity on muscular strength and postural control in children and the performance of the most fundamental of motor tasks: walking.

Effects of obesity on musculoskeletal structure

Although numerous studies have identified the anthropometric changes associated with overweight and obesity, there is a paucity of research on the effects of adiposity on the musculoskeletal structure in children. Despite several studies indicating that the morphology of connective tissue may primarily be influenced during growth and development, the effects of childhood obesity on the morphology of soft tissue structures, such as muscle tendon and ligament remain unknown (18–20). In animal models, exercise has been shown to augment tendon development during growth (18), while in children, activity levels have been related to the development of knee joint cartilage, with low levels of physical activity accompanied by reduced cartilage accrual (19,20). Although childhood obesity is also commonly associated with low levels of physical activity (21), it remains unknown if obesity may also have a differential effect on the morphology of connective tissues with maturation. Of the limited studies investigating the effect of childhood obesity on musculoskeletal structure, the majority have primarily evaluated the effects of adiposity on skeletal structure and alignment, particularly of the joints of the lower extremity.

Hip structure

During normal growth and development the head and neck of the femur undergo relative internal rotation with respect to the femoral condyles, such that the femoral head moves from a position of approximately 30° of anteversion to about 13° anteversion by adulthood (22,23). This reduction in femoral anteversion with maturation has been attributed to several factors including bone growth, tension within soft tissue structures about the hip and biomechanical and weight-bearing forces (24,25). Galbraith *et al.* (26) investigated the relationship between body weight and femoral anteversion in 12 obese (>93 percentile weight for age) and 13 non-obese (<93 percentile weight for age) children using computerized axial tomography. The mean femoral anteversion angle of obese children ($0.4 \pm 13.0°$) was significantly lower than that of normal-weight children ($10.6 \pm 8.6°$) indicating that, in the absence of other rotational deformities, obese children displayed an externally rotated lower limb. The authors proposed that increased weight-bearing forces associated with obesity resulted in local mechanical loads that promoted remodelling of the femoral neck. In support of this notion, body weight has been shown to influence loading of the femoral head, with greater loads typically noted in obese children (24,27). In particular, Pritchett and Perdue (27) proposed that the combination of reduced femoral anteversion and the increased weight associated with obesity induced sufficient shear force to exceed failure loads of the proximal femoral physis in adolescents. Indeed, clinical studies have reported an association between obesity, femoral anteversion and slipped capital femoral epiphysis (28–30).

Although biomechanical factors undoubtedly play an important role in the development of slipped capital epiphysis, it is unclear if the reduced femoral anteversion observed in obese children represents a delay in normal ontogenic development or a potentially pathological change in alignment induced by obesity. Although there is some evidence that obese adults have a greater out-toe angle during gait (31), there is little published data indicating that obese adults have reduced femoral anteversion. Consequently, additional research incorporating a longitudinal study design is required to further elucidate the effect of body composition on the ontogenic development and alignment of the femur in both children and adults.

Knee structure

The effect of obesity on the alignment of the knee has been sparsely reported within the literature. Severe obesity has been clinically linked with Blount's disease or idiopathic tibial vara in infants, juveniles and adolescents (32–35). Although little is known regarding the pathogenesis of the condition, biomechanical overload of the proximal tibial

physis secondary to static varus alignment and excessive body weight has been touted to suppress varus stress growth and disrupt endochondral ossification of the physis in infants (32,36). In the late-onset form, however, an underlying static varus malalignment of the knee might not be a prerequisite for its development. Rather, Davids *et al.* (37), using three-dimensional motion analysis and a simple anthropometric model, demonstrated that the dynamic gait deviations characteristic of obese adolescents resulted in pathologic compressive forces in the medial compartment of the knee. Thus, at least in adolescents, gait deviations, predominantly arising from the increased thigh girth associated with obesity, have been implicated in the development of Blount's disease. However, it is questionable that increased adiposity, alone, is sufficient to disrupt endochondral ossification, especially given the low incidence of tibial vara in the general populace (32,34) and the finding that Blount's disease, although more prevalent, is not exclusively restricted to the obese (33).

Foot structure

Despite the potential negative consequences of obesity on lower limb structure, only limited research has considered the effects of obesity on the anthropometry of the foot. Dowling and Steele (38) characterized the external shape of the feet and legs of 10 obese [age = 8.8 ± 2.0 years; body mass index (BMI) = 25.8 ± 3.8 kg m^{-2}] and 10 non-obese children (age = 8.9 ± 2.1 years; BMI = 16.8 ± 2.0 kg m^{-2}) matched on age, height and gender. Significant main effects for obesity were noted on 17 of the 26 anthropometric variables, which were primarily related to broader, higher and thicker structural features of the obese children's calves, ankles, feet and toes compared with their non-obese counterparts. Interestingly, there were no significant differences in the length of the feet of the obese (left = 22.4 ± 1.8 cm; right = 22.4 ± 2.1 cm) and non-obese (left = 21.6 ± 1.9 cm; right = 21.4 ± 2.0 cm) children. It is unclear therefore if the greater foot dimensions reported during stance in the obese are representative of an osseous splaying of the foot, secondary to a larger body mass, or merely an increase in soft tissue dimensions because of a greater fat mass, or a combination of both.

The height of the medial longitudinal arch has been one of the primary criteria for classifying foot structure and is considered paramount to efficient functioning of the foot and lower limb (39). To date, research into the effect of overweight and obesity on the medial longitudinal arch has largely employed indirect measures of arch height, such as those based on inked or electronic footprints (13,38,40–42). Of these studies, four have reported changes in footprint indices that purportedly reflect a lowered longitudinal arch or 'flatter foot' type in the obese (13,38,41,42). Characterized by increased midfoot contact of the obese footprint, 'flat' and 'pronated' feet have been speculated to consign greater strain to soft tissue structures of the lower limb, and as such raise the potential for musculoskeletal injury (31,43). However, the validity of employing footprint parameters as indirect estimates of arch height is controversial. Although some studies have observed moderate correlations between footprint parameters and either radiographic (44) or clinical measures of arch height (45,46), others have not (47,48). In the largest study to date, Hawes *et al.* (47) concluded that inconsistencies in the thickness of soft tissues beneath the feet nullified the use of footprints as indirect measures of arch height. Adiposity is known to influence the thickness of the subcalcaneal fat pad in adults (49). Moreover, Gilmour and Burns (40) noted that, while footprint-based estimates of arch height were significantly altered in childhood obesity, direct clinical measures of arch height were not.

While increased midfoot pressures are also characteristic of immature gait (50), Dowling *et al.* (13,41) speculated that the higher dynamic peak pressures and pressure-time integrals observed under the midfoot and forefoot of obese children placed them at greater risk of future foot pathology. In the absence of longitudinal cohorts incorporating radiographic measures of arch structure, it is unknown if the increased midfoot contact noted in obese children represents an increased risk of injury, a relative distortion of the footprint because of increased adiposity, or a 'true' collapse of the medial longitudinal arch. It is recommended therefore that future research employ direct radiographic measures of arch height to discern the effects of obesity on foot structure in children. Moreover, there is a need to establish the long-term consequences of altered foot anthropometry in the obese. It is likely that greater foot dimensions may present difficulties for obese individuals in obtaining comfortable footwear. Anthropometric foot variables, including arch configuration, have been shown to significantly influence perceived footwear comfort levels in healthy, normal-weight adults; with greater comfort levels, in turn, associated with a lower incidence of foot pain (51). Whether the anthropometric changes noted with childhood obesity may progress to become symptomatic with continued excessive weight-bearing and, in turn, hinder participation in physical activity is speculative and requires further investigation. Indeed, other factors, including plantar pressures and the cutaneous vibration sensation of the plantar surface of the foot, have also been shown to influence footwear comfort levels (51–53) and may provide an interesting direction for future biomechanical research in the obese.

Bone density

Reduced bone mineral density has been associated with a greater risk of fracture in both adults and children (54–56).

In adults, the majority of studies have identified that body weight is protective of bone mineral density (57–60), with most studies noting a positive association between bone mineral density and lean body mass (61–64). Others, however, have attributed the protective effect mainly to fat mass (65–68), suggesting that biochemical factors may also play an important role in bone metabolism (69–72). Although genetic, hormonal, and other non-mechanical factors likely affect bone mass (73), the strength of adult bone predominantly reflects factors that regulate bone quality (architecture) and density (bone mass or quantity of calcium deposited per unit of bone) acquired during childhood and adolescence (74).

In contrast to adults, the effect of body weight on bone strength and density in children is less clear. Although there is evidence that higher weight is associated with greater bone development in children and adolescents (75–78), childhood obesity has been linked with a lowered relative bone mass and reduced bone area when corrected for the maturational effects of age and size (75,79–83). Thus, at least in children, high adiposity is associated with low bone mass and reduced bone strength, when expressed relative to body weight. Goulding *et al.* (82) proposed that the disparity between weight gain and bone mineral accrual observed in overweight children induced greater stress within bone rendering it more vulnerable to fracture. Several studies have demonstrated a greater prevalence of forearm fractures in overweight and obese children (54,79,80). However, these studies have generally failed to consider the potentially mitigating effects of physical activity on indicators of bone mass. Physical activity has been shown to be a strong predictor of bone mass accrual in children (84–86), by presumably ameliorating weight-bearing and muscular forces (87,88). Thus, the relatively lower bone mass associated with childhood obesity may merely represent a reduced physical activity level within this population (75,85,89). Further research accounting for the potentially confounding effect of physical activity is required to ascertain the effect of heightened adiposity on bone mineral density in children. Moreover, given that body weight exerts a protective effect on bone in adults, there is a need to establish the differential effect of fat mass and fat free mass on indicators of bone strength during growth and maturation and into senescence.

Effects of childhood obesity on motor performance

Research into the effect of adiposity on musculoskeletal function in children has largely focused on the effect of obesity on muscular strength and postural balance and, to a lesser extent, on the locomotor characteristics of obese gait. Although the disadvantages of childhood obesity have been readily inferred, much of the evidence is without scientific validation and, as such, has resulted in considerable subjective reporting of the movement capacity of the obese child. While it has long been recognized that motor performance in children is age-dependant, there is evidence that the development of motor skills may also be influenced by environmental factors, including reduced fitness and adiposity (90).

Postural balance

The development of static balance is a basic characteristic of normal motor development, with improved balance control typically observed with maturation (91). Although motion analysis techniques and force platforms are frequently employed to evaluate postural control in adults, research investigating the effect of childhood obesity on postural balance has primarily focused on clinical measures of stability during unipedal or bipedal stance.

Similar to findings in children with developmental coordination disorder (92), obesity appears to have negligible impact on static balance control during normal conditions, but under difficult or novel situations, may result in impaired performance and greater postural sway by presumably altering the joint torque required to stabilize the body. For instance, Goulding *et al.* (93) found no significant difference between obese (>85 percentile in BMI) and non-obese boys (<85 percentile in BMI) during bipedal measures of static balance; however, clinical measures of balance (Bruininks-Oseretsky sub-test of balance) involving single limb stance on a reduced base of support (balance beam) were significantly impaired in obese subjects. Habib *et al.* (94) reported a similar adverse effect of weight on the same balance sub-set in non-obese children. The authors hypothesized that inadequate muscular function in obese children leads to impaired balance (93). In support of this concept, muscle torque regulatory abilities have been clearly associated with improved balance in children (95). Although insightful, clinical tests of balance provide minimal information as to whether childhood obesity is associated with impaired regulation of anteroposterior or mediolateral balance.

In contrast to anteroposterior stability, perturbations in the mediolateral direction are primarily stabilized by muscular adjustments about the hip and to a lesser extent the foot (96,97). Thus, in theory, mediolateral balance represents a more difficult task, particularly during single limb stance, when the centre of mass trajectory must be maintained within the relatively narrow limits imposed by the base of support offered by the foot (97). McGraw *et al.* (98) demonstrated that, although anteroposterior stability was compromised in pre-pubertal boys who were obese (>95 percentile in BMI), the most marked difference was found in centre of pressure measures of mediolateral stability. While mediolateral stability is of particular impor-

tance to dynamic activities such as gait (97,99,100), modifications of the visual system (darkness and confused vision) and narrowing of the base of support (tandem foot position) yielded even greater displacement and variability of the centre of pressure measured within the mediolateral direction. The authors proposed that because the frequency characteristics of the force vectors remained relatively unchanged with adiposity, the decreased stability observed in obese children was likely the result of the greater inertial properties of adipose tissue, rather than an impaired postural control system itself. Other studies have indicated that once adjustments for body weight are made, the contribution of body adiposity to the prediction of balance performance is negligible (101,102). Thus, while it is impossible to attribute the impaired postural control associated with childhood obesity to greater adiposity or a larger body mass, the concomitant finding by McGraw *et al*. (98) that obese children were dependent on visual input to maintain mediolateral stability raises the possibility that, in the absence of vestibular dysfunction, the obese child may also have impaired sensory feedback. The influence of adiposity on the state and development of the sensory-motor system, however, has not been established within the literature.

To date, most studies investigating postural control strategies in obese children have employed cross-sectional study designs and have failed to consider the potentially confounding relationship between postural control and physical activity. Performance on clinical tests of co-ordination and balance have long been associated with leisure-time activity levels in normal-weight and obese children (103). Thus, given the association between obesity and physical inactivity (104,105), it is unclear if the additional mass associated with obesity results in reduced postural stability, or alternately, if the greater adiposity of the obese is the consequence of postural instability and reduced activity. Moreover, it is unclear as to what extent, if any, the girth of the lower limbs in obese children influence their performance on balance tests incorporating dramatic reductions in the base of support. Further research employing a longitudinal study design and measures of physical activity may provide greater insight into the effects of obesity on postural balance in children.

Muscular strength and power

Although research has generally indicated that obese children and adolescents present with either similar (106–109) or greater absolute strength of the trunk and muscles of the upper extremity (91,110), absolute measures of strength do not account for the increase in muscular strength noted to occur with maturation (111,112) and are confounded by the concomitant increase in fat free mass that is often, but not always (113), associated with childhood obesity (106,109,114). Field tests in which children are required to transfer body weight against gravity, however, provide a convenient, although indirect, method that may partially account for these confounding influences (115). For instance, Riddiford *et al*. (116) investigated the effects of obesity on strength and power in 43 obese (BMI: 24.1 ± 2.3 kg m^{-2}) and 43 non-obese (BMI: 16.9 ± 0.4 kg m^{-2}) pre-pubescent children (8.4 ± 0.5 years) using age-appropriate field tests of a basketball throw for distance, arm push-pull ability, and vertical and standing long jump performance. Although the execution of absolute strength tests of the upper limb did not differ between groups, obese children were found to have impaired performance in both the standing long jump and vertical jumping tasks. Deforche *et al*. (117) reported similar findings in a battery of field tests (Eurofit) that assessed the physical fitness of obese ($n = 230$) and non-obese ($n = 2976$) Flemish youth aged between 12 and 18 years. Whereas obese children were found to have greater absolute grip strength, they performed poorly on field tests in which they were required to move their larger mass against gravity. When adjustments are made for body weight obese adolescents have been shown to have reduced knee extensor and elbow flexor strength (106) and lower power output during cycling (118), jumping and stair climbing activities (119). It would appear therefore that despite the purported training effect and subsequent hypertrophy incurred by lower extremity muscles in supporting and moving additional fat mass (120,121), obesity is characterized by reduced muscular strength.

Whether the relative strength reduction associated with childhood obesity is indicative of impaired neuromuscular function secondary to an altered metabolic state is unknown. However, when strength characteristics are electrically evoked, the observed difference in intrinsic strength between obese and normal-weight adolescents seems to disappear (106,109), suggesting that the lowered strength during maximum voluntary contraction represents a reduced activation of motor units (109). Motor unit activity has been shown to be dependent on several factors, including motivation and physical activity levels (106,120). Thus, the impaired strength associated with childhood obesity may reflect a lower level of physical activity and muscle de-conditioning in association with the additional mass associated with adiposity. Whether this, in turn, affects the ability of children to learn and successfully perform activities of daily living, as proposed within the literature (116), is speculative. Although there is anecdotal evidence that inadequate muscular strength, particularly of the lower limbs, can limit individuals from successfully performing everyday tasks, such as rising to a standing position (10), and may predispose the obese to a greater risk of fatigue and musculoskeletal injury, further research into the long-term effect of a relative reduction

muscular strength, particularly in obese children, seems warranted.

Effects of childhood obesity on walking gait

While quantitative temporal and spatial aspects of gait have been reported for normal-weight children (122,123), there is still minimal data regarding the basic gait characteristics of the obese child. Within the limited literature, childhood obesity has been consistently associated with slower habitual or self-selected walking speeds when compared with normal-weight children (98,124). Accompanied by concomitant reductions in step length and step frequency (98,124–127), the reduced walking speed observed in obese children is typified by longer single support and double support phase durations along with a shorter swing phase in comparison with normal-weight children (98,125). Collectively, the changes have been interpreted as an underlying instability in the obese, with a slower walking speed and longer period of double support thought to assist with the maintenance of dynamic balance (98). In support of this concept, static tests of postural control have revealed reduced mediolateral stability in obese children (93,98) and similar changes in gait parameters have also been reported in children with neurological deficits (128,129). However, the exact relationship between such changes in gait parameters and the risk of falling has not been established within the literature. Thus, in the absence of clear evidence that obese children present with a greater risk of falling, the suggestion that the gait characteristics of obese children is a manifestation of impaired postural stability is speculative. For instance, the reduced swing phase observed in obese children may also represent an effective shortening of the inverted pendulum model because of a relatively greater accumulation of mass within the thigh segment. Irrespective of cause, however, Hills *et al.* (124) demonstrated that conservative gait changes were accentuated in obese children when walking slower or faster than their preferred speed, highlighting that adiposity may be an important factor governing the ability of obese children to accommodate to changes in walking speed.

Faster than preferred walking speeds have also been shown to incur a greater energy cost in obese adolescents (+12%) when compared with lean adolescents matched on body weight (130). While body mass appears to be the primary determinant of the energy cost of walking at preferred speeds (131,132), the higher metabolic cost observed with faster walking speeds has been attributed, in part, to a higher cost of ventilation (130). Although receiving some support within the literature (133), the larger metabolic cost associated with walking in obese children most likely represents a mechanical difference in gait style, such as an anterior tilt of the upper body, an altered step frequency, a greater vertical displacement of the centre of mass, or extraneous movements arising from the greater dimensions and inertial properties of obese limbs (130,134). While there is evidence that thigh girth may alter gait parameters in children (37), a detailed kinematic and kinetic evaluation of the gait of obese children while walking over a range of speeds is lacking. However, similar changes in gait parameters have also been observed during sprint running in obese children, with lower horizontal velocities also associated with a shorter step length, lower step frequency and a longer foot contact time (119).

What is the long-term influence of obesity on musculoskeletal structure and function?

Obese adults who have been obese from childhood may face a compendium of medical problems (135). During normal walking the major joints of the lower extremity are exposed to considerable loads (136,137) with joint reaction forces of approximately three to five times body weight (138). Participating in movement tasks such as stair climbing, jogging and running involves joint reaction forces at the higher end of this range and beyond. Based on Newtonian Laws of Motion it would appear reasonable to hypothesize that obese individuals will experience greater loads on their joints than normal-weight individuals. Many of the orthopaedic conditions that are manifested in obese adults may be the consequence of an excessive and prolonged loading of tissues. Because of the progressive nature of such developments it may be reasonable to hypothesize that younger individuals, who have been obese for a relatively short period of time, would be less likely to display orthopaedic anomalies related to the locomotor apparatus than their older counterparts. However, this review has confirmed that even young pre-pubertal children display evidence of alterations to both their musculoskeletal structure and function as a consequence of obesity. With the rapid rise in childhood obesity evident throughout the developed world and the concern over the growing prevalence of musculoskeletal injury, there is a need to establish the long-term consequences of adiposity on the ontogenic development of musculoskeletal structures in children and its impact upon locomotor function and the subsequent development of musculoskeletal disorders.

Summary

The rise in childhood obesity represents a major health concern in developed countries. While associations between childhood overweight, adverse cardiovascular risk and adult obesity are beginning to emerge, the consequences of childhood obesity on development and function of the musculoskeletal system have received comparatively little attention. Although there is indirect evidence that the

alignment and structure of the hip, knee and foot may be influenced by overweight and obesity in children, the impact of such changes on musculoskeletal function and its progression into adulthood is poorly understood. While the effect of obesity on skeletal structure is controversial, there is a definitive lack of research into the effect of obesity on the morphology and properties of connective tissue structures. Moreover, the delineation of the effects of childhood obesity on musculoskeletal structure in terms of mass, adiposity, anthropometry, metabolic effects or physical inactivity, or their combination, has not been investigated. Although it is universally recognized that the maintenance of functional mobility should be one of the highest priorities in the management of obesity, high levels of body fat plus increased loading of the musculoskeletal system have the potential to lead to pain and discomfort, inefficient body mechanics and further reductions in mobility. An understanding of locomotor characteristics and biomechanical efficiency of obese children during the performance of daily living tasks would undoubtedly provide a greater understanding of movement-related difficulties of obese children and the potential impact on the development of musculoskeletal disorders.

Conflict of Interest Statement

No conflict of interest was declared.

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