Leg muscle activation patterns during walking and leg lean mass are different in children with and without developmental coordination disorder

Timothy T.T. Yam, Shirley S.M. Fong

School of Public Health, University of Hong Kong, Hong Kong

Correspondence to:

Shirley S.M. Fong

School of Public Health

University of Hong Kong

301F 3/F HKJCBIR, 5 Sassoon Road

Pokfulam, Hong Kong

Tel: (852)28315260

Fax: (852)28551712

E-mail: smfong@hku.hk

Abstract

Background: Previous studies have shown that children with developmental coordination disorder (DCD) have a higher body fat and greater gait variability. Little research has investigated the gait muscle activity and lean mass measures in children with DCD.

Aims: To compare the leg muscle activation patterns of the gait cycle and lean leg mass between children with and without DCD.

Methods: Fifty-one children were in the DCD group (38 males and 13 females; 7.95 ± 1.04 years) and fifty-two in the control group (34 males and 18 females; 8.02 ± 1.00 years). Peak muscle activation patterns of treadmill walking in the right leg for the eight-gait phases were measured by means of electromyography, an electrogoniometer, and foot contact switches. Leg lean mass measures were evaluated using a whole-body dual energy X-ray absorptiometry scan.

Results: Children with DCD had a lower leg lean mass and appendicular lean mass index compared to the control group. Furthermore, they exhibited a less-pronounced peak muscle activation during the heel strike (gastrocnemius medialis), early swing (biceps femoris) and late swing phases (gastrocnemius medialis) of gait.

Conclusions and Implications: Although lower limb total mass was similar between groups, the DCD group displayed lower lean mass measures than controls. Furthermore, children with DCD illustrated a lower leg peak muscle activation during the heel strike, early swing and late swing phases of gait when walking on a treadmill. Our results emphasize the need to incorporate lower limb phasic muscle strengthening components into gait rehabilitation programs for children with DCD.

Keywords: Developmental coordination disorder, kinetics, gait, muscle mass

What this paper adds

This is one of the few studies to investigate leg lean mass and gait muscle activation patterns in walking from children with DCD. We found that children with DCD had a lower gastrocnemius medialis muscle activation level during the heel strike and late swing phases of gait and lower biceps femoris muscle activation level during the early swing phase of gait compared to controls. These children also had lower leg lean mass and lower appendicular lean mass indices than their typically developing peers. This provides further insight on the different walking strategies they adopt and elements to incorporate into rehabilitation programs for children with DCD.

Highlights

- Children with DCD had a lower gastrocnemius medialis muscle activation level during the heel strike and late swing phases when walking on a treadmill.
- They also had lower biceps femoris muscle activation levels in the early swing phase of treadmill gait.
- Leg lean mass and appendicular lean mass indices were also lower in children with DCD than typically developing children.
- Phasic gastrocnemius strengthening should be incorporated into rehabilitation programs to improve treadmill gait propulsion and gait efficiency of children with DCD.

1. Introduction

Developmental coordination disorder (DCD) is a neurodevelopmental condition with a prevalence ranging from 1.8% to 8.6% worldwide (American Psychological Association, 2013; Kadesjö & Gillberg, 1999). Motor coordination deficits are diagnosed as early as age 5 years for children with DCD which may persist through adolescence and adulthood interfering significantly with daily activities (APA, 2013; Henderson, Sugden & Barnett, 2007). Motor deficits, including both physical and psychosocial aspects, exhibit a greater lower limb gait variability and lower self-worth in several physical and functional domains (Fong et al., 2011; Rosengren et al., 2009; Skinner & Piek, 2001). A lower self-efficacy level partly accounts for the lower participation in physical activities in children with DCD which may explain the greater likelihood of choosing sedentary activities (Cairney, Hay, Faught, & Hawes, 2005) and higher weight status in this population (Fong et al., 2011).

Most children acquire effective walking skills naturally as they mature. For typically developing (TD) children, temporal gait parameters continually mature starting as early as the first year in growth with a gradual decrease in cadence and an increase in stride length (Sutherland, 1997). Initial heel strike starts to develop after age 1 year and the knee flexion during loading response is not developed until the age of 4 (Sutherland, 1997). Stride-to-stride control is not fully developed even at the age of 7 years (Hausdorff, Zemany, Peng & Goldberger, 1999) suggesting that gait maturation goes through a complex process.

Muscle maturation (most notably tibialis anterior, gastrocnemius, soleus and vastus medialis) occurs between the ages 1 and 2 years (Sutherland, 1997) where muscles interact intricately to produce a metabolically efficient gait. For a typical gait cycle, rectus femoris extends the knee prior to the heel strike phase followed by tibialis anterior activity to oppose the plantarflexion ground reaction force. Simultaneously, the biceps femoris serves as a hip extensor to control forward rotation of the thigh. Muscles contract synergistically throughout the gait cycle to increase gait efficiency (Di Nardo, Mengarelli, Maranesi, Burattini, & Fioretti, 2015). Children with DCD often adopt an adaptive gait and exhibit a relatively higher cadence compared to their TD peers (Deconinck et al., 2006). This is accompanied by less precise control at the ankle joint with less pronounced ankle plantarflexion during the toe-off phase (Deconink et al., 2006). Furthermore, the shank (distal) section exhibits greater complexity than the thigh (proximal) segment which suggests that distal segments (ankle) produce greater variability (Rosengren et al., 2009). According to Deconinck et al. (2006), these gait differences are suggestive of an immature gait which may be a compensatory reaction to adopt a safer walking strategy.

Gait differences are also present in running and fast walking which interfere with a broader aspect of daily activities in children with DCD. In running, they demonstrate a deficit in ankle power generation (Diamond, Downs, & Morris, 2014). When examining ankle power generation in fast walking, the differences between children with and without DCD are comparable to the elderly population (Diamond et al., 2014). Since ankle power generation is relatively lower in elderly fallers (Perry et al., 2007), the gait deficits seen in this population are of great clinical importance with possible interference to dynamic balance. Chia, Licari, Guelfi, and Reid (2013) also investigated the kinematics and kinetics of running in children with DCD and found that they had a longer stance duration and decreased knee joint moments. However, joint moments and muscle forces were measured indirectly using a force platform, which did not capture individual muscle activations. Thus, it is essential to explore the differential muscle activation patterns of walking and to further understand the complexity of locomotion in children with DCD.

Gross motor difficulties in children with DCD could also be affected by body weight

(Cattuzzo et al., 2016). Since body weight includes multiple components, it is unknown which aspect of body composition is more influential to physical performance. What is certain is that children with DCD are less likely to participate in physical activities. This may be related to their higher body fat, body mass index (BMI) (Cairney et al., 2005) and their poorer movement skills (Okely, Booth & Chey, 2003). Although children with DCD have a higher body fat (Cairney, Hay, Veldhuizen, & Faught, 2011), the pattern may not necessarily translate to lean mass. To the best of our knowledge, no studies have investigated lean mass or related measures in this population. The only study that examined an outcome considered fat free mass (FFM) and the authors found no significant difference between children with and without DCD (Cairney et al., 2011). Since FFM also includes bone mineral content (BMC), it does not fully reflect on lean mass which is related to a higher likelihood of weakness in older adults (Cawthon et al., 2014). Given that children with DCD have deficits in fine ankle control (greater variability in ankle control) (Rosengren et al., 2009), it would also be beneficial to investigate lean mass targeted for the peripheral extremities such as appendicular lean mass index (ALMI). Comparing lean mass and the associated outcome measures (i.e. total mass and ALMI) between children with and without DCD may help to inform intervention programs.

The aim of this cross-sectional study was to compare (i) the leg lean mass, leg total mass and ALMI; and (ii) the leg muscle activation patterns throughout the gait cycle performed on a treadmill between children with and without DCD. Due to the previous gait deficits seen during push off in children with DCD with decreased ankle control, we hypothesized that they would exhibit less pronounced muscle activation during the force generating phase especially at the ankle muscles. Given that children with DCD had a higher body fat, BMI and reported lower strength (Cairney et al., 2005; Raynor, 2001), we hypothesized that their overall lean mass would be relatively lower than TD children.

2. Methods

2.1. Participants

Between March and August 2016, 200 children were recruited from primary schools in Hong Kong and our database of DCD participants through invitation letters, posters and social media advertisements, and personal invitations. One hundred and three volunteer children were eligible to participate in the study. Fifty-one were allocated to the DCD group (38 males and 13 females; age ± standard deviation = 7.95 ± 1.04) and fifty-two to the control group (34 males and 18 females; age ± standard deviation = 8.02 ± 1.00). All children were screened by two physiotherapists with the Movement Assessment Battery for Children, 2nd edition (MABC-2; Henderson et al., 2007).

Children with DCD was determined by a two-step method which has been previously used (Ferguson, Jelsma & Smits-Engelsman, 2013). As a first step, children aged 6 to 9 years with fine and gross motor problems affecting daily lives (i.e. sports activities, buttoning a shirt, writing) were selected and referred by teachers. For the second step, the referred children were screened by the MABC-2 (Henderson et al., 2007) and assessed against the Diagnostic and Statistical Manual of Mental Disorders 5th Edition (DSM-V) (APA, 2013); a score ≤15th percentile on the MABC-2 Test to indicate motor skills below that expected for their age (Henderson et la., 2007) (criterion A). Teachers and/or parents identified the presence of motor skill difficulties interfering with daily life activities (criterion B) and indicated that onset of symptoms from early childhood (criterion C). DCD questionnaire 2007 version (DCDQ; Wilson, Kaplan, Crawford, & Roberts, 2007) was used to provide additional information on motor deficits. Furthermore,

parents/guardian were asked to complete a series of screening questions to rule out those motor deficits caused by neurological disorder or intellectual delay (criterion D). The exclusion criteria were (i) history of leg fractures, (ii) congenital, musculoskeletal, neurological, or cardiopulmonary disorders that might influence exercise ability or motor development, (iii) a metal implant, (iv) a body mass index (BMI) >25, (v) recent physiotherapy or other related treatments within the past 2 months, (vi) emotional problems or excessive disruptive behavior, and (vii) the inability to follow instructions. The control group participants shared the same inclusion and exclusion criteria except that they did not have a diagnosis of DCD and had a score of >15th percentile on the MABC-2.

Ethical approval was obtained from the Human Research Ethics Committee at the University of Hong Kong. Written informed consent was obtained from the participants and their parents. All experimental procedures were conducted by two physiotherapists in accordance with the Declaration of Helsinki (2013).

2.2. Outcome measurements

2.2.1. Procedure

The participants attended a single session at the Physical Activity Laboratory and Dual Energy X-ray Absorptiometry (DXA) Laboratory at the University of Hong Kong. Children and their parents provided demographic data, medical history, and exercise habits to calculate the habitual physical activity level (in metabolic equivalent (MET) hours per week) by using the Compendium of Energy Expenditures for Youth (Ridley, Ainsworth, & Olds, 2008). Exercise habits were reported by parents/guardian on the sport or activity performed on a regular basis and specifics to each exercise (i.e. intensity (light, moderate, vigorous), duration (hours/session) and frequency (times/week)). This method was used in our previous studies (Fong et al., 2015; Fong et al., 2016). Body weight and height were measured using an electronic scale (A and D, UC-321, Tokyo, Japan) and a height stadiometer (seca 213, Seca, CA, USA). The children completed the MABC-2 according to standardized procedures stated in the user manual (Henderson et al., 2007) to evaluate their motor proficiency. Although no reliability information is available in Hong Kong, the MABC-2 has been reported to be both a reliable and valid assessment of motor competence in Taiwanese children (Wuang, Su & Su, 2012). UK age- related percentile norms were used in this study as used by Wuang et al. (2012).

2.2.2. DXA-derived lean mass and total mass

Each child underwent a whole-body scan using a DXA scanner (Horizon A, Hologic Inc., Bedford, MA). They were instructed to wear loose clothing with no metal or plastic material attached. When positioning each participant in a supine position, hands were placed vertically at the sides with the fifth finger on the table pad with palm facing down. Hip joints were internally rotated until both big toes came into contact. The children were instructed to remain still and breathe normally during the scan. All scans were administered by two licensed operators, and the body was positioned in accordance with the Hologic user manual (Hologic, 2015). After the scan, the axial and appendicular lean mass and total mass (i.e., fat mass + lean mass + bone mineral content) of the participants were determined by the DXA scanner's region of interest program. The ALMI in kg/m^2 was also calculated automatically based on the formula appendicular lean mass/height^2. The bilateral leg lean mass, total mass and ALMI were used in the outcome analyses. The precision of the DXA scanner in vivo is

very good, with the coefficient of variation for whole-body assessment of soft tissue ranging from 0.7% to 2% (Toombs, Ducher, Shepherd, & De Souza, 2012).

2.2.3. Leg muscle peak activation during gait

Before walking on a motorized treadmill, the following apparatuses were applied to the children to measure kinetic and kinematic gait outcomes. Based on our assumption of bilateral gait symmetry, only the right leg was measured which was partly because of device channel limitations. Furthermore, unilateral EMG measurements have been previously used to investigate gait kinematics (Kagawa, Ohta, & Uni, 2011). Circular Ag/AgCl bipolar surface electromyographic (EMG) electrodes (EMG sensor SX230-1000, Biometrics, Newport, UK), with an interelectrode distance of 2 cm, were applied on the belly of the right leg muscles (rectus femoris, biceps femoris, tibialis anterior and gastrocnemius medialis) to detect muscle activity. The participant's skin was prepped with alcohol swabs and shaved as necessary to reduce the skin impedance before applying the electrodes to the designated location as specified by Barbero, Merletti, and Rainoldi (2012). EMG signals were sampled at a rate of 1000 Hz with amplification (×1000) with a band-pass filtered between 20 and 450 Hz. Input impedance was set at $>10^{15}$ Ω, and the common mode reject ratio was >96dB (Biometrics, 2012). A reference electrode (R506, Biometrics, Newport, UK) was located at the ipsilateral tibial tuberosity. An electrogoniometer (twin-axis goniometer SG150B, Biometrics, Newport, UK) was applied at the lateral aspect of the right knee as recommended by the Biometrics manual (Biometrics, 2002) with a sampling rate of 1000 Hz (Biometrics, 2012) to monitor knee flexion and extension movements during walking. Finally, two foot contact switches (FS4 contact switch assembly, Biometrics, Newport, UK) were situated at the ipsilateral mid heel and first metatarsal as suggested by Blanc, Balmer, Landis, & Vingerhoets (1999) to register the heel strike and toe-off phases of gait. The EMG, electrogoniometer, and foot contact switches were secured with adhesive tape and connected to a separate DataLOG device (Biometrics, Newport, UK), which was attached to the participant's waist to minimize artifacts during data collection.

During the gait analysis trial, the children walked in socks on a motorized treadmill (KLS-008 2B2, X2Fit treadmill, PT. Maharupa Gatra, Indonesia). Given that kinematic measures were not significantly different between treadmill and overground walking (Murray et al., 1985), a treadmill was used for its speed consistency which was chosen for previous gait studies in children with DCD (Deconinck et al., 2006; Rosengren et al., 2009). The treadmill speed for each participant was scaled to the leg length with the following equation:

$$Fr = \frac{\mathrm{v}^2}{\mathrm{g} \cdot \mathrm{L}}$$

where v is velocity (m/s); g is the acceleration of gravity (m/s^2) and L is leg length (m). A Froude number (Fr) of 0.15 was used for a normal walking speed as in a previous study conducted by Deconinck et al. (2006). The treadmill speed was gradually increased to the desired velocity starting with a 10-minute familiarization trial, which was sufficient for treadmill habituation and reproduction of stride length (Van de Putte, Hagemeister, St-Onge, Parent, & de Guise, 2006). The participant then walked 2 more minutes to record kinetic and kinematic data using EMG, electrogoniometer and foot contact switches maintaining the pre- calculated speed. The children were instructed to walk as naturally as they would

normally do while looking ahead. The testing session ended with the treadmill speed gradually reduced to zero. All data was stored on the DataLOG for later offline analysis.

The maximal voluntary isometric contraction (MVIC) measured the four major leg muscles prior to walking on the treadmill. The MVIC value of each muscle was measured twice with a 1-minute recovery period between individual muscle tests. The children were instructed to exert their maximal strength against manual resistance for 5 seconds without any body movements. All muscle tests were performed in a seated position with designated knee joint angles pertaining to each test as specified in Dionisio, Almeida, Duarte, & Hirata (2008). The highest 1-second EMG signal was filtered with root mean square (RMS). The average EMG_{rms} value of the two trials of each leg muscle was selected as the representative MVIC value. The average MVIC value of each leg muscle was normalized to the RMS value of the MVIC of each leg muscle with the outcome expressed as %MVIC to enable comparison of muscle activity between individuals. The peak EMG_{rms} values (in %MVIC) of six consecutive strides were averaged for each leg muscle and gait phase (Diamond et al., 2014). The data recorded on the DataLOG were analyzed using Biometrics software (DataLOG PC Software Version 8.51). To determine the gait phases, the following methods were used. Foot contact switch signals were used to register heel strike and toe-off phases. In addition, as knee angle changes had a relatively low variability (Winter, 1991) and contributed considerably to stance (Hayot, Sakka, & Lacouture, 2013) and swing (Barrett, Besier, & Lloyd, 2007) phases, knee angle change was adopted to determine the remaining phases (i.e., loading response, mid stance, late stance, early swing, mid swing and late swing).

2.3. Statistical analyses

Because leg muscle activation during walking in children with DCD had not been extensively researched, a comparable study was used to estimate the sample size. In a study conducted by Chia et al. (2013) on the kinematics and kinetics of locomotion in children with and without DCD, effect sizes ranged from 0.6 to 0.8. Hence, an effect size of 0.6 was used to calculate the sample size for this study. Given that the statistical power was 80% with an alpha level of 5% (2-tailed), the minimum number of participants required to detect a between-groups significant difference was 45 for each group. G*Power version 3.1.0 (Franz Faul, Universität Kiel, Germany) was used for the sample size calculation.

Statistical analyses were processed and conducted using the Statistical Package for Social Science (SPSS) 23.0 software (SPSS Inc. Chicago, IL). The normality criterion was confirmed by using the Shapiro-Wilk test. The independent *t*-test (for continuous data) and chi-square test (for categorical data) were used to compare the demographic characteristics of the DCD and control groups. The multivariate analysis of variance (MANOVA) was used to investigate the between-group differences between the peak muscle activation of each muscle (rectus femoris, biceps femoris, tibialis anterior and gastrocnemius medialis) for each gait phase (heel strike, loading response, mid stance, late stance, toe-off, early swing, mid swing and late swing) and the DXA-derived lean mass variables (leg lean mass, leg total mass and ALMI). Using MANOVA avoids an inflation of type-I error associated with multiple comparisons. All tests were set at a two-tailed alpha level of 5%.

3. Results

3.1. Demographic characteristics

Table 1 illustrated the demographic characteristics of the participants. There were no significant differences in age, height, body weight, body mass index, leg length, physical activity level, EMG MVIC values of leg muscles, treadmill speed, or gender between the two groups. The MABC-2 percentile score and DCDQ total score were significantly lower in the DCD group ($p < 0.001$).

3.2. EMG-derived peak muscle activation in the leg during different phases of gait

Peak muscle activation (EMG_{rms}) in the right leg was observed throughout the eight gait phases. The results of MANOVA revealed a significantly lower gastrocnemius peak EMG_{rms} for the heel strike and late swing phases in the DCD group ($F_{1,101} = 4.659$, $p = 0.033$ and $F_{1,101} = 6.715$, $p = 0.011$ respectively). The early swing phase exhibited a lower biceps femoris ($F_{1,101} = 4.099$, $p = 0.046$) peak EMG_{rms} among this group of children. The remaining five gait phases revealed no significant differences in peak EMG_{rms} values between the two groups ($p > 0.05$) (Table 2).

3.3. DXA-derived lean mass and total mass

Although left and right leg total mass were similar ($p > 0.05$) between the two groups, the DCD group presented significantly lower left leg ($F_{1,101} = 5.240$, $p = 0.024$) and right leg ($F_{1,101} = 6.117$, $p = 0.015$) lean masses (Table 2). In addition, the children with DCD possessed a significantly lower ALMI ($F_{1,101} = 7.168$, $p = 0.009$) than that of the controls.

4. Discussion

This study was the first to investigate the leg lean mass, leg total mass, and peak leg muscle activation of walking in children with and without DCD. Our results supported our hypothesis where children with DCD had a less pronounced peak leg muscle activation during the heel strike, early swing and late swing phases when walking. Additionally, the children with DCD had an overall reduced leg lean mass and ALMI.

4.1. DXA-derived lean mass and total mass

This study found that children with DCD had a significantly less-pronounced leg lean mass and ALMI but their total leg mass was not significantly different to that of the controls. The link between muscle strength and muscle mass has long been sought. Children with DCD produce less power during isometric and isokinetic conditions, which some have suggested is related to muscle-fiber type distribution or hypotonia (Raynor, 2001). Previous studies have elucidated that lean mass is associated with muscle strength and peak twitch torque (Goodpaster et al., 2006; Mau-Moeller, Bruhn, Bader, & Behrens, 2015). Furthermore, leg lean mass has been revealed to substantially explain leg strength variance (Newman et al., 2003).

The lower lean mass in the legs and lower ALMI (0.31 kg/m^2) in children with DCD may not necessarily equate to a diagnosis of sarcopenia but rather imply that these children store relatively less muscle mass to support their body in daily activities compared to TD children. ALMI is used to determine the amount of lean muscle mass in non-trunk areas (peripheral extremities) relative to height. The lower the ratio, the less essential muscle mass there is in the peripheral extremities. In addition, a person with lower lean mass according to

the primary definition of ALMI is 4 to 7 times more likely to be weak than a person with higher lean mass (Cawthon et al., 2014). Although no previous study has reported lean mass measures in children with DCD, height and/or body size potentially influence the association between lean mass and muscle strength.

As expected, total leg mass was not significantly different between children with and without DCD, which agrees with a previous study on FFM (Cairney et al., 2011). We speculate that this may be due to the similarities in total mass and FFM, which both include a BMC component. Despite a study revealing a positive correlation between lean mass percentage and muscle performance (Stephenson et al., 2015), it is still unclear if the decrease in muscle strength relates to neural activation, muscle cross-sectional area or muscle mass. Further research is required to investigate the relationship between lean mass and muscle performance in children with DCD.

4.2. EMG-derived peak muscle activation in the leg during different phases of gait

Children with DCD presented a lower peak gastrocnemius medialis activation at the heel strike and late swing phases. Since gastrocnemius assists in knee flexion and contributes greatly with controlling peak knee flexion during walking (Goldberg, Anderson, Pandy, & Delp, 2004), our results agree with the less pronounced knee flexion angle at heel strike in children with DCD (Deconinck et al., 2006). Furthermore, gastrocnemius and tibialis anterior co-contract to enable correct foot positioning to prepare for the heel strike phase (Di Nardo et al., 2015). Findings compliment the greater variability of shank movements seen in children with DCD which Rosengren et al. (2009) postulated to be related to ankle control. For a normal gait, gastrocnemius activates before the heel strike phase to prepare for gait push-off (Winter, 1991). A less pronounced gastrocnemius activation from late swing to heel strike phase may affect the build-up of ankle power generation near the toe-off phase and the accuracy of foot position throughout the gait cycle.

During the early swing phase, we found a less pronounced biceps femoris activation, which may suggest that children with DCD have an imbalance of muscle coactivations transitioning from stance to swing phase (Raynor, 2001). A less pronounced muscle activity may explain the decrease of knee flexion at mid-swing seen in children with DCD (Rosengren et al., 2009). An under-activation of the biceps femoris will lead to a proportionally greater activation at the rectus femoris and consequently a reduced knee flexion (Piazza & Delp, 1996). The transitional phase from stance to swing is crucial to prepare for forward progression or swing initiation propelling the body forward when walking. If this transitional phase is interrupted, the overall metabolic work will increase with less optimal coordination (Soo & Donelan, 2012). Several theories on the underlying mechanisms of the swing phase as either a passive ballistic model or an active control model contributed by ankle plantar flexors have been proposed (Winter, 1991; Meinders, Gitter, & Czerniecki, 1998). Despite the different schools of thought, the swing phase inevitably constitutes 10% to 15% of the net cost of walking (Gottschall & Kram, 2005; Umberger, 2010). The intricate muscle co-activations enable seamless fluidity and a non-strenuous gait cycle. Our results suggest that children with DCD have a less fine-tuned eccentric control of the biceps femoris to decelerate the leg after the toe-off phase. This could affect the reciprocal muscle activation pattern between the rectus femoris and biceps femoris with the goal of reducing muscle fatigue (Prilutsky, Gregor, & Ryan, 1998).

There was no peak muscle activation difference for the remaining gait phases in walking (loading response, mid stance, late stance, toe-off and mid swing), which may be explained by the lower demand of muscle activity and elastic energy (Lichtwark,

Bougoulias, & Wilson, 2007). Locomotion is an unconscious action that humans perform daily without feeling particularly strained. However, as speed increases, muscle activation moments increase respectively to produce a greater amount of work (Schwartz, Rozumalski, & Trost, 2008). Walking requires a relatively lower metabolic output and physical demand compared to running. The walking performance for children with DCD is characterized by shorter and more variable strides, velocity, acceleration and wider steps (Deconinck et al., 2006; Wilmut, Du, & Barnett, 2016). The gait differences are further translated to jogging/running, which ultimately results in a less efficient gait (Chia et al., 2013; Diamond et al., 2014). More specifically, a greater amount of work is produced during the stance phase in running (Lichtwark et al., 2007), which explains why children with DCD exhibit a lower peak knee extensor moment and angle during the stance phase in running but not in walking (Chia et al., 2013).

Ankle plantarflexion strength greatly influences one's ability to maintain a consistent step length (Judge, Davis & Õunpuu, 1996). Thus, less pronounced muscle activation may disrupt the consistency of cadence and velocity. The gait differences could be a manifestation of a neuromuscular problem (Raynor, 2001) and a safer walking strategy that children adopt to minimize gait unsteadiness. Gait phases with single limb support challenges dynamic balance and the ability to maintain center of mass (COM). From previous research, children with DCD exhibit a greater medio-lateral COM movement when stepping over an obstacle (Deconinck, Savelsbergh, De Clercq, & Lenoir, 2010). Moreover, delayed onset of the gastrocnemius and hamstrings are seen during unexpected perturbations (Fong et al., 2015). Gait deficits in children with DCD are not limited to locomotion but provide insight to static and dynamic balance ability. Our results further confirm that children with DCD exhibit a varied ankle control which may be a compensation to gait instability. Although their gait adaptations are comparable to the elderly population, by no means they receive the same treatments because there are distinctive gait adaptation differences between them (Diamond et al., 2014; Hausdorff et al., 1999). Gait deficits and a lower lean mass may contribute to the vicious cycle which accounts for the lower participation in physical activities, decrease in physical performance and lower self-efficacy in motor competence. The findings provide some pointers on what elements to incorporate in rehabilitation treatments. Phasic gastrocnemius strengthening should be included into interventions to improve ankle positioning and gait efficiency. Rehabilitation programs that aim for a more active lifestyle for children with DCD (Cairney et al., 2011) should integrate muscle-specific training components.

This study had some limitations. First, the within-group variabilities of the gait outcomes were quite high. This could be attributable to the duration of the familiarization trial. Although treadmill habituation time is recommended to be 10 minutes (Van de Putte et al., 2006), children with DCD may require more time to familiarize with treadmill walking given that they may have difficulties with attention and motor learning (Fong et al., 2016). Second, our results were specific to treadmill walking and hence cannot be generalized to walking on level ground in daily life. Future research could investigate the muscle activity of over-ground walking in children with DCD. Third, previous findings revealed the gait asymmetries in children with DCD (Wilmut, Gentle, & Barnett, 2017) requiring bilateral EMG for future investigations. Finally, the relationships between gait parameters and lean mass were not explored in this study. Further research is necessary to unravel the relationship between lean mass and the intricacies of muscle activation during walking or locomotion in children with DCD so that specific gait training methods can be designed for this population.

5. Conclusions

Our study found that the biceps femoris and/or gastrocnemius medialis have a relatively lower activation level during the heel strike, early swing and late swing phases in children with DCD compared to TD children. This may affect foot placement and propulsion of the body when walking. Moreover, children with DCD had a significantly lower lean mass at the lower extremities and lower ALMI than controls. Therefore, future intervention should emphasize on both lifestyle modification and targeted muscle strengthening.

Acknowledgments

The authors would like to acknowledge the following schools for aiding the recruitment of participants: Si Yuan School of The Precious Blood, Heep Yunn Primary School, St Peter's Catholic Primary School, Emmanuel Primary School, Kowloon, SKH Tin Shui Wai Ling Oi Primary School, HKTA Yuen Yuen Institute Shek Wai Kok Primary School, Pun U Association Wah Yan Primary School, HHCKLA Buddhist Wisdom Primary School, and TWGHs Hok Shan School. The work described in this paper was partially supported by the Health and Medical Research Fund (13142081) of the Food and Health Bureau of Hong Kong.

Conflicts of interest

None declared.

References

APA, American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed., American Psychiatric Association, Washington, DC.

Barbero, M., Merletti, R., & Rainoldi, A. (2012). *Atlas of Muscle Innervation Zones – Understanding Surface Electromyography and Its Applications*, Springer, Milan, Italy.

Barrett, R. S., Besier, T. F., & Lloyd, D. G. (2007). Individual muscle contributions to the swing phase of gait: an EMG-based forward dynamics modelling approach. *Simulation Modelling Practice and Theory*, *15*(9), 1146–1155.

Biometrics Ltd. (2012). *EMG Sensor and DataLOG Operating Manual*, Biometrics Ltd., Newport, UK.

Biometrics Ltd. (2002). *Goniometer and Torsiometer Operating Manual*, Biometrics Ltd., Newport, UK.

Blanc, Y., Balmer, C., Landis, T., & Vingerhoets, F. (1999). Temporal parameters and patterns of the foot roll over during walking: normative data for healthy adults. *Gait and Posture*, *10*(2), 97–108.

Cairney, J., Hay, J. A., Faught, B. E., & Hawes, R. (2005). Developmental coordination disorder and overweight and obesity in children aged 9-14y. *International Journal of Obesity*, *29*, 369-372.

Cairney, J., Hay, J., Veldhuizen, S., & Faught, B. (2011). Assessment of body composition using whole body air-displacement plethysmography in children with and without developmental coordination disorder. *Research in Developmental Disabilities*, *32*(2), 830–835.

Cattuzzo, M. T., dos Santos Henrique, R., Nicolai Re, A. H., de Oliveira, I. S., Melo, B. M., de Sousa Moura, M., de Araujo, R. C., & Stodden, D. (2016). Motor competence and health related physical fitness in youth: a systematic review. *Journal of Science and Medicine in Sport*, *19*, 123–129.

Cawthon, P. M., Peters, K. W., Shardell, M. D., McLean, R. R., Dam, T. T. L., Kenny, A. M., et al. (2014). Cutpoints for low appendicular lean mass that identify older adults with clinically significant weakness. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *69*(5), 567–575.

Chia, L. C., Licari, M. K., Guelfi, K. J., & Reid, S. L. (2013). A comparison of running kinematics and kinetics in children with and without developmental coordination disorder. *Gait and Posture*, *38*(2), 264–269.

Deconinck, F. J. A., De Clercq, D., Savelsbergh, G. J. P., Van Coster, R., Oostra, A., Dewitte, G., & Lenoir, M. (2006). Differences in gait between children with and without developmental coordination disorder. *Motor Control*, *10*(2), 125–142.

Deconinck, F. J. A., Savelsbergh, G. J. P., De Clercq, D., & Lenoir, M. (2010). Balance problems during obstacle crossing in children with Developmental Coordination Disorder. *Gait and Posture*, *32*(3), 327-331.

Diamond, N., Downs, J., & Morris, S. (2014). "The problem with running": Comparing the propulsion strategy of children with Developmental Coordination Disorder and typically developing children. *Gait and Posture*, *39*(1), 547–552.

Dionisio, V. C., Almeida, G. L., Duarte, M., & Hirata, R. P. (2008). Kinematic, kinetic and EMG patterns during downward squatting. *Journal of Electromyography and Kinesiology*, *18*(1), 134–143.

Di Nardo, F., Mengarelli, A., Maranesi, E., Burattini, L., & Fioretti, S. (2015). Assessment of the ankle muscle co-contraction during normal gait: A surface electromyography study. *Journal of Electromyography and Kinesiology*, *25*(2), 347-354.

Ferguson, G. D., Jelsma, D., Jelsma, J., & Smits-Engelsman, B. C. M. (2013). The efficacy of two task-oriented interventions for children with developmental coordination disorder: neuromotor task training and Nintendo Wii Fit training. *Research in Developmental Disabilities*, *34*(9), 2449-2461.

Fong, S. S .M., Ng, S. S .M., Guo, X., Wang, Y., Chung, R. C .K., Ki, W. Y., et al. (2015). Deficits in lower limb muscle reflex contraction latency and peak force are associated with impairments in postural control and gross motor skills of children with developmental coordination disorder: a cross-sectional study. *Medicine*, *94*(41).

Fong, S. S .M., Chung, J. W .Y., Cheng, Y. T .Y., Yam, T. T. T., Fong, D. Y .T., Cheung, C. Y., Chiu, H. C., Yuen, L., Yu, Y. E .T., Hung, Y. S., Macfarlane, D. J., & Ng, S. S .M. (2016). Attention during functional tasks is associated with motor performance in children with developmental coordination disorder: a cross-sectional study. *Medicine (Baltimore)*, *95*(37), e4935.

Fong, S. S .M., Lee, V. Y .L., Chan, N. N. C., Chan, R. S. H., Chak, W. K., & Pang, M. Y. C. (2011). Motor ability and weight status are determinants of out-of-school activity participation for children with developmental coordination disorder. *Research in Developmental Disabilities*, *32*(6), 2614–2623.

Goldberg, S. R., Anderson, F. C., Pandy, M. G., & Delp, S. L. (2004). Muscles that influence knee flexion velocity in double support: implications for stiff-knee gait. *Journal of biomechanics*, *37*(8), 1189-1196.

Goodpaster, B. H., Park, S. W., Harris, T. B., Kritchevsky, S. B., Nevitt, M., Schwartz, A. V., et al. (2006). The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *61*(10), 1059–1064.

Gottschall, J. S., & Kram, R. (2005). Energy cost and muscular activity required for leg swing during walking. *Journal of Applied Physiology*, *99*(1), 23–30.

Hausdorff, J. M., Zemany, L., Peng, C. K., & Goldberger, A. L. (1999). Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children. *Journal of Applied Pysiology*, *86*(3), 1040-1047.

Hayot, C., Sakka, S., & Lacouture, P. (2013). Contribution of the six major gait determinants on the vertical center of mass trajectory and the vertical ground reaction force. *Human Movement Science*, *32*(2), 279–289.

Henderson, S. E., Sugden, D. A. & Barnett, A. L. (2007). *Movement Assessment Battery for Children*, 2nd ed., Psychological Corporation Ltd., Sidcup, England.

Hologic Inc. (2015) *Horizon QDR Series User Guide.* Bedford, MA: Hologic Inc.

Judge, J. A., Davis, R. B., & Õunpuu, S. (1996). Step length reductions in advanced age: the role of ankle and hip kinetics. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 51*(6), M303-M302.

Kadesjö, B., & Gillberg, C. (1999). Developmental coordination disorder in Swedish 7-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *38*(7), 820–828.

Kagawa, T., Ohta, Y., & Uno, Y. (2011). State-dependent corrective reactions for backward balance losses during human walking. *Human Movement Science, 30*(6), 1210-1224).

Lichtwark, G. A., Bougoulias, K., & Wilson, A. M. (2007). Muscle fascicle and series elastic element length changes along the length of the human gastrocnemius during walking and running. *Journal of Biomechanics*, *40*(1), 157–164.

Mau-Moeller, A., Bruhn, S., Bader, R., & Behrens, M. (2014). The relationship between lean mass and contractile properties of the quadriceps in elderly and young adults. *Gerontology*, *61*(4), 350–354.

Meinders, M., Gitter, A., & Czerniecki, J. M. (1998). The role of ankle plantar flexor muscle work during walking. *Scandinavian Journal of Rehabilitation Medicine, 30*(1), 39–46.

Murray, M. P., Spurr, G. B., Sepic, S. B., Gardner, G. M., & Mollinger, L. A. (1985). Treadmill vs. floor walking: kinematics, electromyogram, and heart rate. *Journal of Applied Physiology, 59*(1), 87-91.

Newman, A. B., Haggerty, C. L., Goodpaster, B., Harris, T., Kritchevsky, S., Nevitt, M., ... & Visser, M. (2003). Strength and muscle quality in a well-functioning cohort of older adults: The health, aging and body composition study. *Journal of the American Geriatrics Society*, *51*(3), 323–330.

Okely, A. D., Booth, M. L., & Chey, T. (2004). Relationship between body composition and fundamental movement skills among children and adolescents. *Research Quarterly for Exercise and Sport, 75*(3), 238-247.

Perry, M. C., Carville, S. F., Smith, I. C. H., Rutherford, O. M., & Newham, D. J. (2007). Strength, power output and symmetry of leg muscles: effect of age and history of falling. *European Journal of Applied Physiology, 100*(5), 553-561.

Piazza, S. J., & Delp, S. L. (1996). The influence of muscles on knee flexion during the swing phase of gait. *Journal of Biomechanics, 29*(6), 723–733.

Prilutsky, B. I., Gregor, R. J., & Ryan, M. M. (1998). Coordination of two-joint rectus femoris and hamstrings during the swing phase of human walking and running. *Experimental Brain Research, 120*(4), 479–486.

Raynor, A. J. (2001). Strength, power, and coactivation in children with developmental coordination disorder. *Developmental Medicine and Child Neurology*, *43*(10), 676–684.

Rosengren, K. S., Deconinck, F. J., DiBerardino, L. A., Polk, J. D., Spencer-Smith, J., De Clercq, D., & Lenoir, M. (2009). Differences in gait complexity and variability between children with and without developmental coordination disorder. *Gait and Posture*, *29*(2), 225–229.

Ridley, K., Ainsworth, B. E., & Olds, T. S. (2008). Development of a compendium of energy expenditures for youth. *International Journal of Behavioral Nutrition and Physical Activity*, *5*(1), 45.

Schwartz, M. H., Rozumalski, A., & Trost, J. P. (2008). The effect of walking speed on the gait of typically developing children. *Journal of Biomechanics*, *41*(8), 1639–1650.

Skinner, R. A., & Piek, J. P. (2001). Psychosocial implications of poor motor coordination in children and adolescents. *Human Movement Science*, *20*(1), 73–94.

Soo, C. H., & Donelan, J. M. (2012). Coordination of push-off and collision determine the mechanical work of step-to-step transitions when isolated from human walking. *Gait and Posture*, *35*(2), 292–297.

Stephenson, M. L., Smith, D. T., Heinbaugh, E. M., Moynes, R. C., Rockey, S. S., Thomas, J. J., & Dai, B. (2015). Total and lower extremity lean mass percentage positively correlates with jump performance. *The Journal of Strength and Conditioning Research*, *29*(8), 2167–2175.

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

Toombs, R. J., Ducher, G., Shepherd, J. A., & De Souza, M. J. (2012). The impact of recent technological advances on the trueness and precision of DXA to assess body composition. *Obesity*, *20*(1), 30–39.

Umberger, B. R. (2010). Stance and swing phase costs in human walking. *Journal of the Royal Society Interface*, *7*(50), 1329–1340.

Van de Putte, M., Hagemeister, N., St-Onge, N., Parent, G., & de Guise, J. A. (2006). Habituation to treadmill walking. *Bio-medical Materials and Engineering*, *16*(1), 43–52.

Wilmut, K., Du, W., & Barnett, A. L. (2016). Gait patterns in children with Developmental Coordination Disorder. *Experimental Brain Research*, *234*(6), 1747–1755.

Wilmut, K., Gentle, J., & Barnett, A. L. (2017). Gait symmetry in individuals with and without Developmental Coordination Disorder. *Research in developmental disabilities, 60*, 107-114.

Wilson, B. N., Kaplan, B. J., Crawford, S. G., & Roberts, G. (2007). The developmental coordination disorder questionnaire 2007 (DCDQ'07), Retrieved from http://www.dcdq.ca.

Winter, D.A. (1991) *The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological* 2nd ed., Ont., Canada: Waterloo Biomechanics.

Wuang, Y. P., Su, J. H., & Su, C. Y. (2012). Reliability and responsiveness of the movement assessment battery for children – second edition test in children with developmental coordination disorder. *Developmental Medicine and Child Neurology, 54*(2), 160-165.

Tables

Table 1: Characteristics of the participants

	DCD (n = 51)	Control (n = 52)	*p* value
Age (years)	7.95 ± 1.04	8.02 ± 1.00	0.755
Sex			0.391
Male (n, %)	38 (74.5)	34 (65.4)	
Female (n, %)	13 (25.5)	18 (34.6)	
Height (cm)	125.26 ± 8.67	127.34 ± 7.65	0.200
Body weight (kg)	24.40 ± 5.09	25.60 ± 4.93	0.230
Body mass index (kg/m^2)	15.42 ± 1.94	15.57 ± 1.87	0.676
Leg length (cm)	63.82 ± 6.86	66.02 ± 4.92	0.064
MABC-2 test (percentile score)	8.10 ± 5.78	47.77 ± 22.03	<0.001*
DCDQ total score	43.35 ± 12.91	55.62 ± 11.21	<0.001*
Physical activity level (MET hours/week)	9.78 ± 8.71	13.51 ± 12.35	0.080
Comorbid conditions (n, %)			
ADHD	3 (5.9)	---	
ASD	6 (11.8)	---	
EMG MVIC values (μV)			
Quadriceps	1.34 ± 0.48	1.30 ± 0.51	0.666
Hamstring	1.42 ± 0.44	1.58 ± 0.43	0.069
Tibialis anterior	1.74 ± 0.59	1.76 ± 0.43	0.867
Gastrocnemius	0.93 ± 0.54	0.79 ± 0.48	0.192
Treadmill speed (km/hr)	1.24 ± 0.25	1.31 ± 0.24	0.141

Means ± standard deviations are presented unless otherwise specified.

*Significant difference at $p < 0.05$.

DCD: developmental coordination disorder; MABC-2: Movement Assessment Battery for Children 2nd edition; DCDQ: developmental coordination disorder questionnaire; MET: metabolic equivalent; ADHD: attention deficit hyperactivity disorder; ASD: autism spectrum disorder; EMG: electromyography; MVIC: maximal voluntary isometric contraction

Table 2: Comparison of outcome measures between the DCD and control groups

	DCD (n = 51)	Control (n = 52)	Mean Difference[a]	95% Confidence Interval	$F_{1,101}$	*p* value
Muscle peak EMG_{rms} (%MVIC) during different phases of gait						
Heel strike						
Rectus femoris	11.98 ± 10.48	12.26 ± 8.81	0.28	-3.584, 3.920	0.008	0.929
Biceps femoris	16.40 ± 8.55	14.15 ± 8.68	-2.25	-5.583, 1.087	1.788	0.184
Tibialis anterior	12.35 ± 8.29	10.80 ± 9.80	-1.55	-5.063, 1.966	0.764	0.384
Gastrocnemius medialis	15.24 ± 12.94	21.66 ± 17.24	6.42	0.520, 12.335	4.659	0.033*
Loading response						
Rectus femoris	10.09 ± 9.13	10.53 ± 7.35	0.44	-2.945, 3.458	0.025	0.874
Biceps femoris	17.05 ± 9.49	15.80 ± 11.78	-1.25	-5.443, 2.800	0.404	0.526
Tibialis anterior	8.52 ± 8.14	6.09 ± 5.54	-2.43	-5.192, 0.179	3.428	0.067
Gastrocnemius medialis	19.12 ± 17.24	25.17 ± 22.58	6.05	-1.684, 13.787	2.408	0.124
Mid stance						
Rectus femoris	3.03 ± 3.12	2.93 ± 2.50	-0.10	-1.130, 1.057	0.004	0.948
Biceps femoris	8.53 ± 6.02	8.92 ± 6.47	0.39	-1.733, 4.359	0.731	0.395
Tibialis anterior	8.42 ± 9.13	8.06 ± 7.73	-0.36	-3.615, 2.900	0.047	0.828
Gastrocnemius medialis	31.94 ± 22.20	41.05 ± 40.09	9.11	-3.366, 21.590	2.099	0.151
Late stance						
Rectus femoris	3.94 ± 4.50	3.81 ± 3.96	-0.13	-1.779, 1.499	0.029	0.866
Biceps femoris	5.26 ± 6.23	6.20 ± 7.42	0.94	-1.713, 3.596	0.495	0.483
Tibialis anterior	15.14 ± 10.30	16.13 ± 11.47	0.99	-3.232, 5.212	0.216	0.643
Gastrocnemius medialis	8.71 ± 13.32	9.00 ± 10.76	0.29	-4.401, 4.966	0.014	0.905
Toe-off						
Rectus femoris	4.46 ± 3.07	6.04 ± 7.10	1.58	-0.605, 3.649	2.014	0.159
Biceps femoris	5.63 ± 6.02	6.02 ± 7.06	0.39	-2.150, 2.934	0.093	0.760
Tibialis anterior	18.04 ± 12.26	17.03 ± 10.89	-1.01	-5.498, 3.470	0.201	0.655
Gastrocnemius	5.39 ±	8.23 ±	2.84	-0.696,	2.536	0.114

medialis	5.74	11.50		6.360		
Early swing						
Rectus femoris	4.18 ± 3.52	3.83 ± 3.88	-0.35	-1.813, 1.057	0.273	0.602
Biceps femoris	4.35 ± 3.14	6.07 ± 5.28	1.72	0.035, 3.407	4.099	0.046*
Tibialis anterior	19.85 ± 11.90	17.37 ± 8.96	-2.48	-6.548, 1.593	1.457	0.230
Gastrocnemius medialis	4.80 ± 4.14	6.11 ± 5.00	1.31	-0.474, 3.083	2.118	0.149
Mid swing						
Rectus femoris	4.90 ± 4.69	4.37 ± 3.03	-0.53	-2.088, 0.963	0.535	0.466
Biceps femoris	11.41 ± 7.85	10.77 ± 6.61	-0.64	-3.445, 2.169	0.203	0.653
Tibialis anterior	10.19 ± 7.60	8.64 ± 7.88	-1.55	-4.541, 1.451	1.046	0.309
Gastrocnemius medialis	7.15 ± 8.14	8.71 ± 10.25	1.56	-2.028, 5.148	0.744	0.390
Late swing						
Rectus femoris	9.20 ± 7.03	9.67 ± 7.41	0.47	-2.435, 3.169	0.067	0.796
Biceps femoris	15.80 ± 8.65	14.88 ± 12.13	-0.92	-5.004, 3.166	0.199	0.656
Tibialis anterior	11.48 ± 8.48	9.48 ± 12.89	-2.00	-6.230, 2.229	0.880	0.350
Gastrocnemius medialis	12.85 ± 10.89	20.80 ± 16.14	7.95	1.863, 14.032	6.715	0.011*
DXA-derived lean (muscle) mass and total mass						
Left leg lean mass (g)	2337.49 ± 676.41	2607.85 ± 512.53	270.36	36.074, 504.649	5.240	0.024*
Left leg total mass[b] (g)	3989.49 ± 1333.91	4423.63 ± 1032.76	434.14	-24.058, 892.333	3.533	0.063
Right leg lean mass (g)	2380.58 ± 707.69	2691.16 ± 559.52	310.58	61.479, 559.692	6.117	0.015*
Right leg total mass[b] (g)	4094.41 ± 1380.01	4562.96 ± 1071.20	468.55	-5.944, 943.049	3.837	0.053
Appendicular lean mass index (kg/m^2)	3.88 ± 0.70	4.19 ± 0.48	0.31	0.082, 0.550	7.168	0.009*

Means ± standard deviations are presented unless otherwise specified.

*Significant difference at $p < 0.05$ [a]Mean difference: control subtract DCD group; [b]Total mass = fat mass + lean mass + bone mineral content

EMG_{rms}: $Electromyography_{root\ mean\ square}$; MVIC: maximal voluntary isometric contraction; df: degrees of freedom; DXA: dual energy X-ray absorptiometry