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Children with cerebral palsy have larger Achilles tendon moment arms than typically developing children

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ABSTRACT

The effectiveness of the plantarflexor muscle group to generate desired plantarflexion moments is modulated by the geometry of the Achilles tendon moment arm (ATMA). Children with cerebral palsy (CP) frequently have reduced plantarflexion function, which is commonly attributed to impaired muscle structure and function, however little attention has been paid to the potential contribution of ATMA geometry. The use of musculoskeletal modelling for the simulation of gait and understanding of gait mechanics, rely on accuracy of ATMA estimates. This study aimed to compare 3D *in-vivo* estimates of ATMA of adults, children with CP and typically developing (TD) children, as well as compare 3D *in-vivo* estimates to linearly scaled musculoskeletal model estimates. MRI scans for eight children with CP, 11 TD children and nine healthy adults were used to estimate *in-vivo* 3D ATMA using a validated method. A lower limb musculoskeletal model was linearly scaled to individual tibia length to provide a scaled ATMA estimate. Normalised *in-vivo* 3D ATMA for children with CP was 17.2% ± 2.0 tibia length, which was significantly larger than for TD children (15.2% ± 1.2, $p = 0.013$) and adults (12.5% ± 0.8, $p < 0.001$). Scaled ATMA estimates from musculoskeletal models significantly underestimated *in-vivo* estimates for all groups, by up to 34.7%. The results of this study show children with CP have larger normalised 3D ATMA compared to their TD counterparts, which may have implications in understanding reduced plantarflexor function and the efficacy of surgical interventions whose aim is to modify the musculoskeletal geometry of this muscle group.

1. Introduction

The Achilles tendon and the plantarflexor muscle group play a crucial role in the functioning and regulation of gait mechanics. The impact of this is particularly highlighted when assessing the gait of children with cerebral palsy (CP). This neurodevelopmental movement disorder is related to a number of skeletal muscle-tendon unit impairments, with the plantarflexors being commonly affected. Impairments include spasticity, reduced selective motor control, reduced muscle volume and cross sectional area, and altered muscle histology (Graham et al., 2016). It is well known that children with CP have reduced plantarflexion strength (i.e. joint moment estimates) when compared to typically developing (TD) children (Eek et al., 2011; Wiley and Damiano, 1998). While the aforementioned impairments are contributing factors (Elder et al., 2003; Graham et al., 2016), current understanding of strength impairments among this population is incomplete. One contributing mechanism may be differences in three dimensional (3D) Achilles tendon moment arm (ATMA) geometry.

The moment arm of any musculo-tendon unit is understood as the "effectiveness" of skeletal muscle to generate a moment of force about an axis of rotation (Sherman et al., 2013), and thereby actuating the desired moment about a given joint to generate movement. The moment of force produced by a musculo-tendon unit is a product of the linear muscular force, and the 3D moment arm. The 3D moment arm is the perpendicular distance between the forces line of action and the axis of rotation. As such, alterations in either of these properties will influence the moment arm geometry. Training studies have shown that muscle hypertrophy increases the cross sectional area of the muscle belly, which in turn increases moment arm length (Maganaris et al., 1998, 2000; Sugisaki et al., 2010; Vigotsky et al., 2015). Children with CP have smaller gastrocnemius and soleus muscle volumes and cross sectional areas than TD children (Barrett and Lichtwark, 2010;

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Handsfield et al., 2016), so it is reasonable to expect that children with CP would have smaller ATMA. Another factor influencing the size of the ATMA is the location and orientation of the plantarflexion/dorsiflexion axis of rotation which is typically assumed to be the talocrural flexion/extension axis (Correa et al., 2011; Sheehan, 2010). The orientation and location of this axis is dictated by the bony geometry of the foot (Correa et al., 2011; Sheehan, 2010), and further modulated by muscle activation and loading (Maganaris et al., 1998, 2000). Children with CP commonly demonstrate bony deformities at the foot (Davids, 2010; Kedem, P. and Scher, D.M., 2015; Sees and Miller, 2013), as well as abnormal muscle tone and activation (Graham et al., 2016). To the authors' knowledge, only one study has assessed ATMA geometry among this population, finding normalised 2D ATMA of children with CP to be smaller than their TD peers (Kalkman et al., 2017). These results suggest that ambulatory paediatric CP populations also possess the mechanical disadvantage of short ATMA. Research has found substantial error, of up to 40%, can result from the use of 2D methods when compared to 3D measurement techniques, as a result of the moment arm being out of plane with the scan image (Hashizume et al., 2012). As such, further investigation into AMTA geometry in 3D will allow for better understanding of mechanical disadvantages, and facilitate more precise modelling, leading to improved clinical treatment plans for pathological populations.

Internal muscle forces can be estimated when moment arms are known (scaled estimates or measured *in-vivo*) and when net joint moments can be calculated via inverse dynamics during gait or dynamometric assessments (Orendurff et al., 2002; Orendurff et al., 2005). Estimation of internal muscle forces, obtained through musculoskeletal modelling facilitate detailed exploration of muscle mechanics underpinning pathological movement patterns typically observed among CP populations (Delp et al., 2007; Hicks et al., 2009). While this computation method is powerful, it is not a common measurement approach within most clinical research settings (Correa et al., 2011; Hicks et al., 2009; Rezgui et al., 2013). Along with factors such as computational time and costs, one of the primary reasons for this is the sensitivity and specificity of the modelling parameters used to represent the clinical populations they are meant to measure. When *in-vivo* estimates of a participant's 3D moment arms are not available, it is common for researchers to linearly scale these musculoskeletal modelling parameters to accessible anthropometric information such as segment lengths. Most of the regression equations available in the literature are derived from small homogenous cadaveric populations (Arnold et al., 2010) meaning their suitability among pathological and/or paediatric populations is questionable (Correa et al., 2011; Hicks et al., 2009). Gastrocnemius muscle function has been found to be highly sensitive to changes in muscle-tendon architectural properties, including ATMA (Ackland et al., 2012), highlighting the importance of sensitive and participant specific musculoskeletal models. It is imperative to establish more accurate methods to correctly prepare and scale musculoskeletal models that are representative of paediatric and/or pathological populations to increase the specificity of the mechanical information derived.

The primary aim of this study is to compare *in-vivo* 3D ATMA estimates between adults, TD children and children with CP. It is hypothesised that adults will possess the largest 3D ATMA estimates, while children with CP will have the smallest. The secondary aim of this study is to compare 3D ATMA derived from an established musculoskeletal model linearly scaled to tibia length with participant specific *in-vivo* 3D ATMA for adults, TD children, and children with CP. It is hypothesised no differences in 3D ATMA estimates will be observed between methods for the adult population, however significant differences will be found between methods for both paediatric groups.

2. Methods

Eight children with spastic CP, aged 9.7 years (± 2.6), and a convenience sample of 11 TD children, aged 8.7 years (± 2.3), participated. Children with CP were classified as Gross Motor Functional Classification Scale level I (n = 4) and II (n = 4), with diplegia (n = 4) and hemiplegia (n = 4). No children with CP had previous orthopaedic surgery, seven had received their first injection botulinum toxin to the affected gastrocnemius within 6 months (mean 23.4 ± 1.5 weeks) prior to assessment while one remained toxin naïve. All children received physical therapy as part of their routine clinical care, and two participants utilised ankle foot orthoses as part of their clinical management. An existing dataset of 9 healthy adults was used (Clarke et al., 2015). Full participant characteristics can be found in Table 1. Informed consent was obtained from adults and parents of paediatric participants.

Participants had an MRI scan of the dominant (TD and adults) or most affected (CP) ankle at a comfortable resting angle, with the knee extended. For the paediatric groups, a 1.5 T whole body MRI unit (Siemens Medical Solutions, Erlangen, Germany) was used, employing a T1- weighted spin echo sequence; 256 × 256 matrix; 160 mm × 160 mm FOV; 3 mm slice thickness; and 3.3 mm slice gap. A mean of 18.2 slices were acquired, with a range of 16 – 20 slices. Adult scans were conducted in a 3 T whole body MRI unit (Philips Healthcare, Achieva, Netherlands). Adult scan parameters can be found at Clarke et al (2015).

Scans were processed using Mimics software (version 16.0, Materialise, Leuven, Belgium). The Achilles tendon was defined using 7 manually digitised points. The talus was manually segmented and reconstructed, with a cylinder fitted to the reconstructed form. The bipolar axis of the cylinder was taken to represent the flexion/extension axis of the talocrural joint (Sheehan et al., 2007). 3D coordinates of the digitised Achilles tendon and the cylinder's bipolar axis were outputted and processed through a custom Matlab code to calculate *in-vivo* 3D ATMA. This method has been validated among adult populations (Alexander et al., 2017) and possesses established repeatability among TD children (Lum et al., 2015). Ankle joint angles measured from the MRI scans were operationally defined as the angle between the long axis of the tibia and the plantar surface of the hindfoot.

Tibia length for the paediatric groups were measured from lower leg coronal MRI. Tibial length was defined as the most superior lateral aspect of the tibial plateau, to the most inferior lateral aspect of the fibular notch. Coronal lower leg scans were not available for the adult sample, so Grieves Reference Length was used as a surrogate (Grieve et al., 1978).

Table 1
Participant demographics, tibia length and MRI derived joint angles $^{**}p < 0.01$ compared to other groups, $^{*}p < 0.05$ compared to other groups.

		Gender		Age (years)			Tibia length (mm)			MRI derived JA (*)		
	n	m	f	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max
Adult	9	5	4	**29.3****	**22.0**	**48.0**	**421.1****	**370.0**	**480.0**	−18.1	−11.7	−24.1
TD	11	5	6	8.7	5.5	11.8	274.0	215.4	329.7	−18.6	−6.7	−29.2
CP	8	7	1	9.7	5.8	13.6	275.5	240.9	328.7	−**32.0***	−**14.6**	−**53.6**

Musculoskeletal modelling was conducted in OpenSim (version 3.3, SimTK, Stanford, CA, USA) (Delp et al., 2007), using a lower limb model described by Arnold et al. (2010). The Arnold model defines the ankle joint as a revolute joint between the tibia and the talus with one degree of freedom (dorsiflexion/plantarflexion), and no wrapping surfaces or via points relating. All rigid bodies and musculoskeletal properties were manually scaled to tibial lengths. Following model scaling, the scaled moment arms were outputted at the same ankle joint angles measured from the MRI scans, as shown in Fig. 1 (blue closed circle). For the purposes of this comparison, the soleus moment arm was selected to represent the ATMA as it is mono-articular, thereby unaffected by knee positioning.

The soleus insertion onto the calcaneus body in OpenSim was adjusted in the anterior/posterior plane until the modelled moment arm was equal to the *in-vivo* ATMA at the experimentally measured joint angle (Fig. 1, red open circle). This allowed ATMA to be estimated over the model's plantar/dorsiflexion range of motion (Sherman et al., 2013). To compare ATMA across all participants, a predicted ATMA was taken at 20° plantarflexion, and normalised to tibia length. A joint angle of 20° plantarflexion was selected as it was similar to the mean joint angle measured, and an angle most children with CP can achieve despite presence of spasticity or contracture.

Participant age and tibia length, as well as MRI derived joint angle estimates were compared using a one-way ANOVA. To compare the absolute and normalised predicted ATMA at 20° plantarflexion between the three groups, a one-way ANOVA was used. To compare absolute *in-vivo* and linearly scaled 3D ATMA estimates between the three groups, a SPANOVA was used. All ANOVA's were followed up with *post-hoc* analysis conducted using Tukey's HSD. If a significant main effect for condition was found from the SPANOVA, two-tailed t-tests were conducted. All statistical analyses were performed in SPSS (version 23.0, IBM Analytics, Armonk, NY, USA), with an alpha of 0.05.

3. Results

There were significant main effects in age and tibia length ($p < 0.001$). *Post-hoc* analysis showed no significant differences in age ($p = 0.911$) or tibia length ($p = 0.994$) between TD and CP, while adults were significantly different than both paediatric groups for both variables ($p < 0.001$). A significant main effect in MRI derived joint angles was found ($p = 0.001$). *Post-hoc* analysis showed CP had significantly larger joint angles compared to both TD ($p = 0.003$) and adult ($p = 0.003$), but no significant difference was found between TD and adults ($p = 0.986$) (Table 1).

The mean predicted absolute ATMA at 20° plantarflexion was 52.8 mm ± 5.62 for adults, 41.8 mm ± 5.85 for TD and 47.1 mm ± 3.50 for CP, with a significant main effect found ($p < 0.001$). *Post-hoc* analysis showed adults had significantly larger absolute ATMA compared to TD ($p < 0.001$), but no differences were found between adults and CP ($p = 0.082$) or between CP and TD ($p = 0.089$).

A significant main effect was found ($p < 0.001$) when normalised to tibia length; mean normalised ATMAs were 12.5%±0.8 for adults, 15.2%±1.2 for TD and 17.2%±2.0 for CP (Fig. 2). The *post-hoc* analysis showed normalised ATMA at 20° plantarflexion were smaller among adults compared to TD ($p < 0.001$), and CP ($p < 0.001$). CP was found to have significantly larger normalised ATMA at 20° plantarflexion, compared to TD ($p = 0.013$).

When comparing the *in-vivo* and scaled methods for estimating absolute ATMA, a main effect for group was observed ($p < 0.001$), as was a main effect for method ($p < 0.001$). The interaction of these two factors was also significant ($p < 0.001$) (Fig. 3). *Post-hoc* analysis showed the effect of ATMA method (*in-vivo* or scaled) was significantly different among adults compared to CP and TD ($p < 0.001$), while the effect of method was not different between CP and TD ($p = 0.705$).

Post-hoc testing revealed that absolute *in-vivo* 3D ATMAs were significantly larger than the linearly scaled 3D ATMA at the same MRI derived joint angles for all groups ($p < 0.001$). The mean difference was -5.8 mm for adults, −11.0 mm for TD and −15.7 mm for CP. This corresponds to a 10.7%, 26.2%, and 34.7% underestimation of *in-vivo* measurements for adults, TD and CP respectively.

4. Discussion

The primary aim of this study was to compare 3D ATMA estimates for adults, children with CP and their TD counterparts. It

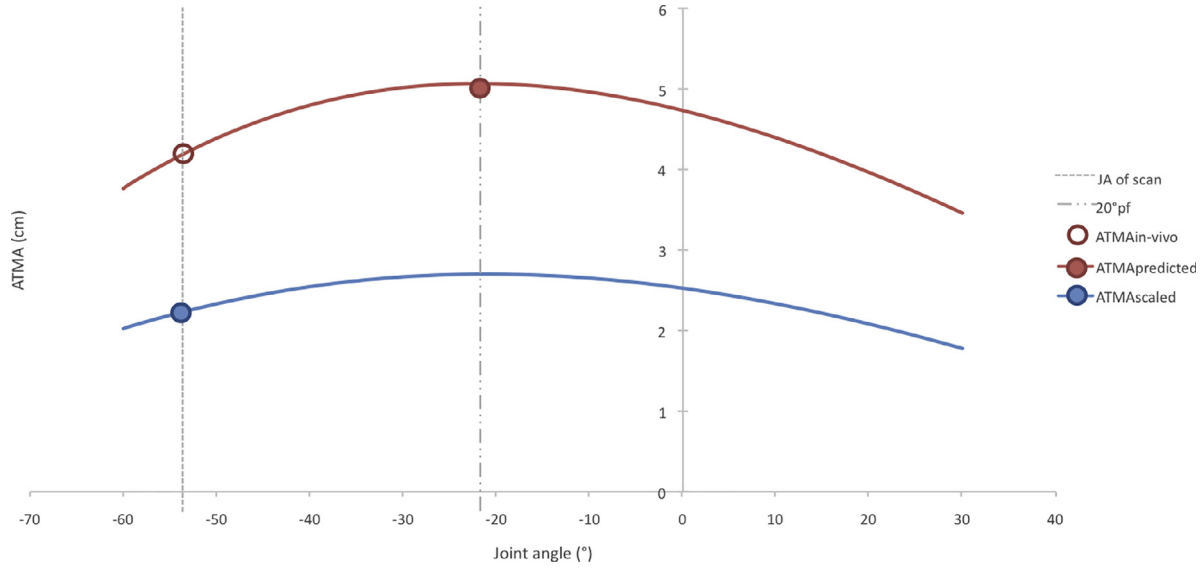

Fig. 1. Example output for one participant, showing the modelled ATMA derived from a linearly scaled musculoskeletal model (blue) and the subject-specific modelled ATMA derived from the in-vivo ATMA measured from MRI scans at the subject specific joint angles, as represented by a dotted line. The dashed line represents 20° of plantarflexion. The ATMA used for comparisons are indicated by circle. An open circle represents an in-vivo measurement, while a closed circle represents a modelled measurement. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

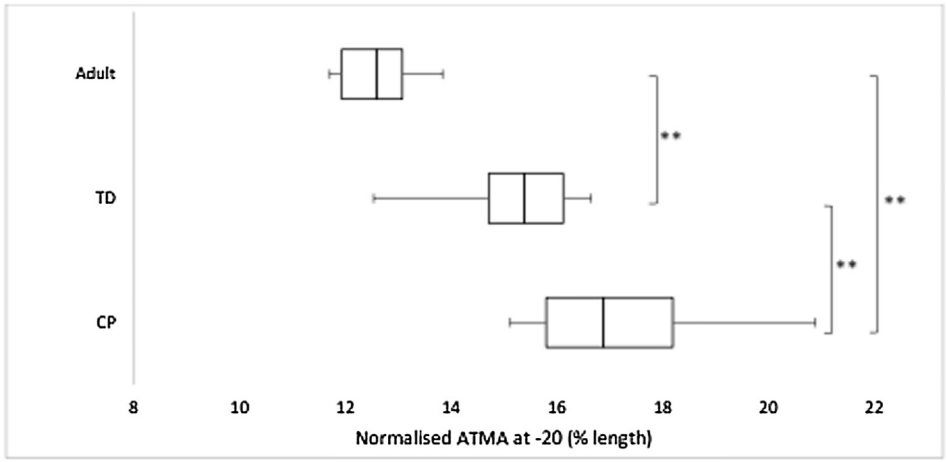

Fig. 2. Box plot showing normalised predicted ATMA for each group at 20° plantarflexion, **p < 0.01.

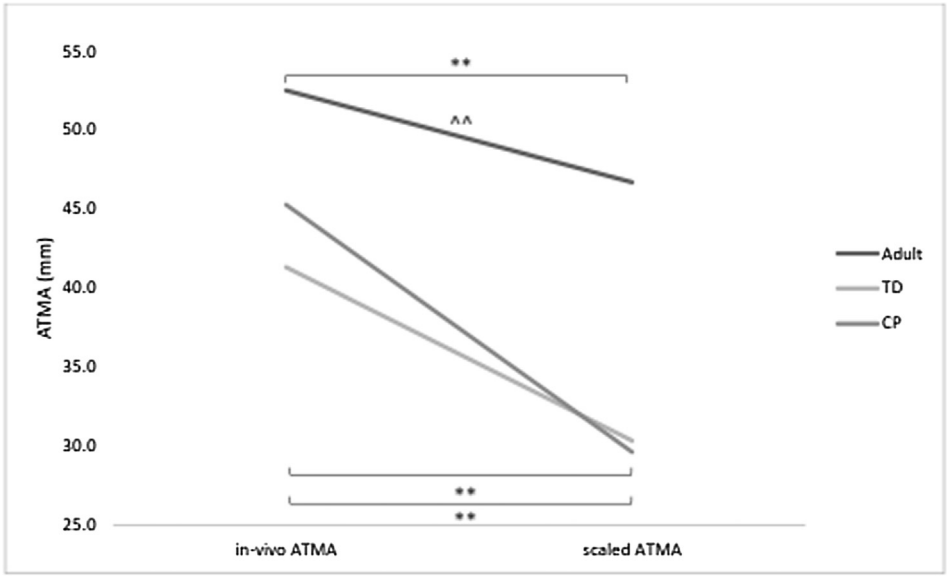

Fig. 3. . Relationship between in and vivo and scaled ATMA for each group **within group *t* test of p < 0.01, ^^ condition effect of p < 0.01.

was hypothesised that adults would have the largest ATMA, and children with CP would have smaller 3D ATMA than TD children, contributing to the documented reduced 'plantarflexor strength' (as measured by joint moments) (Eek et al., 2011; Wiley and Damiano, 1998). This hypothesis has been rejected with adults showing no difference to children with CP, who in turn demonstrate larger normalised 3D ATMA of 9% or 4.3 mm, compared to their TD counterparts. The secondary aim of this study, was to compare 3D ATMA derived from an established musculoskeletal model linearly scaled to tibia length with participant specific *in-vivo* 3D ATMA. As hypothesised, linear scaling of an adult musculoskeletal model did not demonstrate participant specificity for TD children or children with CP, with scaling underestimated *in-vivo* 3D ATMA estimates by 27.68% and 35.82% respectively. While scaled ATMA for adults was also significantly smaller than *in-vivo* ATMA, the effect of the ATMA method (*in*-vivo vs scaled) was significantly less than in either paediatric group and is comparable to the error range accepted in the literature of 10% (Arnold et al., 2000).

Literature suggests a peak plantarflexion moment of 0.86–1.11Nm/kgBW for children with CP during walking gait (Adolfsen et al., 2007; Baddar et al., 2002; Dallmeijer et al., 2011; Eek et al., 2011; Lyon et al., 2005). Based on a 4.3 mm longer ATMA in children with CP, the peak internal muscle force for a 30 kg child with CP would be 62N (10%) less than an appropriately matched TD child producing the same joint moment. Given the observed deficiencies in muscle structure among children with CP (Barrett and Lichtwark, 2010; Reid et al., 2014), a larger ATMA may in fact act as a mechanical compensation for muscle impairments, allowing children to maintain function. More research is required to understand gait mechanics and the role of ATMA among pathological paediatric populations as this has potential to impact surgical treatments that could affect 3D ATMA geometry.

The underestimation of ATMA as a result of linear scaling in musculoskeletal modelling will have significant implications on internal muscle force estimates. For a 30 kg child with CP, these underestimates would result in a 345N (52.83%) overestimation of internal plantarflexor muscle force, assuming a peak plantarflexion moment of 0.985 Nm/kgBW (Adolfsen et al., 2007; Baddar et al., 2002; Dallmeijer et al., 2011; Eek et al., 2011; Lyon et al., 2005). For a TD child of the same mass and plantarflexion moment, internal muscle forces would be overestimated by 259N or 36.21%. Linear scaling of adult musculoskeletal models, therefore, with intention of estimating internal muscle forces are not appropriate for paediatric populations.

This study relies on the assumption that the musculoskeletal model correctly modelled the 3D ATMA of a paediatric CP population through their full joint range of motion. These assumptions are, however, generally accepted as limitations within musculoskeletal modelling literature, including the assessment of children with CP (Barber et al., 2017; Kainz et al., 2018; Rezgui et al., 2013), where research typically calibrates the model using a single static posture to predict dynamic parameters. It was not feasible to measure ATMA at multiple joint angles within this study, and exploration of appropriateness of these assumptions specifically for children with CP is beyond the scope of this study. However, the results suggest further research into these assumptions would be a worthwhile pursuit, and allow for a better understanding of the relationship between ATMA and joint angle over the range of motion for this population. Such research would provide valuable data for informing musculoskeletal models for this unique population.

The findings of this study are in direct contrast to previously presented work by Kalkman et al. (2017) who found children with CP possessed smaller normalised 2D ATMAs when compared to their TD peers (Kalkman et al., 2017). When comparing the presented 3D ATMA estimates from the TD population (15.2%), they were in agreement with the published 2D ATMA estimates (16%). However, for the paediatric CP population in this study, normalised 3D ATMA were notably larger (17.2%) than the 2D estimates reported by Kalkman et al (14%). The participants in this study were all high functioning and could achieve independent ambulation (GMFCS I and II). Although data on bony deformities was not available for this sample, high rates or severity of musculoskeletal deformities may not be expected, as foot deformities are more prevalent for children of a higher GMFCS level (Kedem, M.P. and Scher, M.D., 2015; Rethlefsen et al., 2017). However, the rates and severity of deformities within the sample assessed by Kalkman et al., may contribute to the differences observed, though no information regarding GMFCS or functional capacity of the sample assessed by Kalkman et al is available.

Although comparison between the studies is limited by lack of clarity around the sample assessed, the observed differences in ATMA for the paediatric CP population may in part be to the computational method used to estimate ATMAs, rather than differences in the population itself. The 2D tendon excursion method used has been shown to result in measurement errors up to 40% among healthy adult populations (Hashizume et al., 2012). Three factors have been shown to contribute to these measurement errors: (1) the flexion/extension axis is not orthogonal to the sagittal plane; (2) the sagittal plane selected for analysis; and (3) visual identification of anatomical landmarks used in the measurement. While the second and third proposed source of error would uniformly affect all participants regardless of pathology, the orientation of the flexion/extension axis, given the deformities frequently seen among children with CP, would represent a significant source of error for this population, and may explain the differences found between previous research and results presented here. Further research should be conducted to directly compare reliable robust 3D methodologies, such as that employed in this study (Alexander et al., 2017), and 2D methods among children with CP.

While this study is, to the authors' knowledge, the first to compare linear scaling to *in-vivo* ATMA measurements among children with CP, previous research has compared linear scaling to subject specific MRI musculoskeletal models for this population. Correa et al. (2011) found a linearly scaled model resulted in a 2.4% (± 3.9%) overestimation of ATMA compared to MRI derived musculoskeletal model (an individualised model based on subject specific bone geometry and muscle insertions) for four children with CP. This suggests ATMA estimated from the MRI based musculoskeletal model would not correlate with the *in-vivo* measurements, however important differences between the studies must be considered. The current study directly measured *in-vivo* ATMA using a static, comfortable joint angle, without voluntary muscle activation or external force applied. The previous research compared the AMTA obtained from modelling single leg stance phase during gait. Given the computational expense of a full MRI derived lower limb model, simple *in-vivo* measurements hold strong appeal, and further research comparing the *in-vivo* measurements at MRI derived models would be beneficial.

While the mechanisms for altered ATMA cannot be determined from the current study, potential mechanisms are worth considering. Two factors that influence the ATMA are the tendon line of action, and the joint axis orientation and location. Research suggests a muscle with a smaller cross sectional area would have smaller 3D ATMA (Maganaris et al., 1998, 2000; Sugisaki et al., 2010; Vigotsky et al., 2015), due to a shift in the tendon line of action associated with reduced muscle bulk. Indeed, the smaller muscles of children with CP (Barrett and Lichtwark, 2010; Reid et al., 2014) was put forward as an explanation for the smaller 2D ATMA found by Kalkman et al. (2017). However, the this finding was not upheld in 3D ATMA measurements, suggesting proximal muscle properties are not contributing to the increased 3D ATMA via the tendon line of action. Abnormal forces acting on the calcaneus, including reduced external forces due to lack of heel strike at foot contact (Falso et al., 2005; Park et al., 2006; Pauk et al., 2010), and altered muscular forces due to spasticity, impaired neural activation, contracture and co-contraction, can result in an deformity of the bone (Davids, 2010; Kedem, P. and Scher, D.M., 2015; Woods et al., 2004), and may impact on tendon insertion and therefore line of action. Similarly, bony abnormalities of the foot may also impact on the second critical factor in ATMA length: the location of the joint axis. Pes planovalgus, a common foot deformity in children with CP, sees the lateral column of the foot shorten both functionally and structurally relative to the medial column (Kedem, P. and Scher, D.M., 2015), and may result in rotation or translation of the talocrural axis. Indeed, the contrasting findings between the 3D ATMA results of this study, and the previously reported 2D ATMA results of children with CP (Kalkman et al., 2017) provide support for the importance of joint axis as potential mechanism for altered ATMA geometry. Further research into the mechanisms behind altered ATMA, as well as the impact various surgical interventions targeting the foot and ankle may have, is required.

This study presents two critical findings. Firstly, children with CP were found to have significantly larger normalised 3D ATMA when compared to TD children. This has implications for the understanding of muscle function and torque generation at the ankle. Secondly, as hypothesised, linearly scaled musculoskeletal models cannot replicate *in-vivo* measured ATMA in TD children or children with CP.

Future research examining the relationship between ATMA and JA for these populations is warranted for developing appropriate musculoskeletal models, facilitating their use in a clinical setting.

5. Ethics

This study was approved by the Princess Margaret Hospital (#2013085), University of Western Australia (#RA/4/1/6780), University of New South Wales (#09179) and University of Sydney (#12192) Human Research Ethics Committees.

Conflict of interest

The authors have no relevant conflicts of interest to report. The results of this study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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