

Predictors of physical strain of walking in adults with spastic cerebral palsy - A cross-sectional study

Sandra Linnea Klund-Hansen

sandralli@oslomet.no

OsloMet – Oslo Metropolitan University

Terje Gjøvaag

OsloMet – Oslo Metropolitan University

Arve Opheim

Sunnaas sykehus

Eivind Lundgaard

OsloMet – Oslo Metropolitan University

Espen Ingvald Bengtson

Sunnaas sykehus

Linda Rennie

OsloMet – Oslo Metropolitan University

Research Article

Keywords: Adults Cerebral palsy, Gait biomechanics, GDI, Physical strain, Spasticity, VO2peak

Posted Date: July 6th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10066099/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Introduction

Adults with spastic cerebral palsy often experience elevated physiological demands during walking. Physical strain, defined as oxygen consumption when walking at preferred speed expressed as a percentage of maximal aerobic capacity, quantifies how close everyday walking is to the individuals maximal capacity. Determinants of physical strain in adults with CP are insufficiently understood.

Methods

In this cross-sectional study, 89 ambulant adults (49 female) with spastic cerebral palsy (Gross Motor Function Classification System I-III) underwent cardiopulmonary exercise testing to measure maximal aerobic capacity and oxygen consumption during preferred walking speed on a treadmill, and physical strain was calculated. Three-dimensional gait analysis with force plates quantified gait biomechanics, including Gait Deviation Index, peak ankle power and mean support moment. Neuromotor impairments were assessed using composite scores of the Modified Ashworth Scale (spasticity) and the Selective Control Assessment of the Lower Extremity (selective motor control). Associations with physical strain were examined using bivariate and multiple linear regression with physical strain as a dependent variable and clinical and biomechanical variables as possible predictors.

Results

In the bivariate analysis, age, Gross Motor Function Classification System level, spasticity, gait deviation index and peak ankle power were significantly associated with physical strain (all $p < 0.001$). In the multiple regression model ($F(8,80) = 5.88$, $p < 0.001$), only age ($B = 0.320$, $CI\ 95\% = 0.144-0.496$, $p < 0.001$) and spasticity (MAS composite score; $B = 1.475$, $CI\ 95\% = 0.013-2.893$, $p = 0.048$) independently predicted higher physical strain, explaining about 30% of its variance ($R^2 = 0.37$, adjusted $R^2 = 0.307$, $p < 0.001$). Gait deviations, peak ankle power, mean support moment and selective motor control did not contribute independently after adjustment.

Conclusion

In ambulant adults with spastic cerebral palsy, older age and greater spasticity independently predict higher physical strain of walking. The effect sizes were moderate, however, in this population even a small increase in an already elevated physical strain could be of clinical importance. Targeting spasticity in treatment and preserving aerobic capacity should be a focus in interventions for reducing physical strain of walking in adults with CP.

Introduction

Adults with cerebral palsy (CP) often experience increasing walking difficulties with age [1], elevated physical strain during walking [2] and higher levels of fatigue [3]. Increased physical strain implies that even walking at a slow pace could represent a relative strenuous activity for adults with CP, which limits functional walking capacity and participation in daily activities [2, 4]. Therefore, improvements in walking function are a central rehabilitation goal for persons with CP [5].

Physical strain, defined as the relative oxygen consumption required to walk, expressed as a percentage of maximal oxygen uptake ($\%VO_{2peak}$) [4]. Which factors that influence physical strain in the adult CP population is not yet fully understood, however, research on related areas show that oxygen cost of walking ($VO_2 \text{ mL kg}^{-1} \text{ m}^{-1}$) in children with CP is associated with impaired selective motor control, increased spasticity [6] and greater gait deviations [7]. In healthy older adults, diminished muscle and tendon function and consequently reduced ankle push-off power [8], increases the oxygen demand of walking at the preferred walking speed [9, 10]. Further, walking deterioration has been proposed to be accompanied with a shift in propulsion strategy from ankle to hip in healthy elderly [11] and maximal oxygen uptake (VO_{2peak}) normally decrease with increasing age, and VO_{2peak} are commonly higher in men than women [12]. In adults with CP, gait pattern deviations increase earlier in life compared with the general population [1, 13], despite CP being considered a non-progressive condition [14]. This is particularly clear in individuals with bilateral spastic CP [13, 15].

As physical strain of walking depends on both the oxygen consumption during walking at a given speed and VO_{2peak} [4], an important question arises: to what extent do the predictors of VO_{2peak} and of oxygen cost of walking, as identified in children with CP and in healthy older adults, also retain explanatory power for physical strain of walking in adults with CP? To the authors knowledge, there are no studies investigating possible predictors of physical strain in adults with CP.

In the present study, we investigated if age, neuromotor impairments (spasticity and selective motor control) and gait biomechanics (propulsion strategy, support mechanisms and gait deviations) are predictors of physical strain during walking in adults with CP. Identifying determinants of physical strain may help target interventions aimed at improving walking capacity and daily function in adults with CP.

Methods

Design and setting

This cross-sectional study forms part of the “Cost of Walking in Adults with Cerebral palsy” (COWAC) study [16] and took place in the south-eastern health care region of Norway.

Recruitment

Participants were recruited between September 2023 and June 2024 from three sources: individuals admitted for inpatient rehabilitation at a specialized rehabilitation hospital, respondents to public announcements distributed through the Norwegian Cerebral Palsy Association's website, and former participants from a previous intervention study [17] who had also participated in a recent long-term follow-up study [13].

Inclusion and exclusion criteria

Inclusion criteria were adults (age 18–65 years) with spastic CP, classified as GMFCS levels I–III, able to walk continuously for five minutes with or without a walking aid. To ensure cognitive function did not limit the understanding of participation and information during data collection, an inclusion criterion was that participants had completed normal schooling. Exclusion criteria included medical conditions affecting exercise performance (such as cardiac or pulmonary disease), orthopaedic surgery in the lower extremities within the past 18 months, Botulinum-toxin injections for lower limb spasticity within the last three months, or pregnancy. All participants received oral and written information about the study and provided written informed consent. The inclusion and exclusion process is presented in Figure 1.

Procedures

Demographic and clinical characteristics were recorded prior to testing, including age, sex, GMFCS-level [18], Body Mass Index (BMI) and spastic CP subtype (unilateral or bilateral).

Based on previous literature in children with CP and healthy older adults, potential predictors of physical strain were selected from demographic characteristics, clinical assessment and laboratory-based outcomes. We hypothesized that greater gait deviations, reduced peak ankle power and more pronounced neuromotor impairments would predict higher physical strain of walking.

Laboratory assessments

Oxygen consumption during walking at preferred walking speed ($\text{VO}_{2\text{walk}}$) and maximal aerobic capacity ($\text{VO}_{2\text{peak}}$) were measured using treadmill-based Cardiopulmonary Exercise Testing (CPET). Detailed description of the methods has been described previously [19]. $\text{VO}_{2\text{peak}}$ and $\text{VO}_{2\text{walk}}$ were expressed as $\text{VO}_2 \text{ mL kg}^{-1} \text{ min}^{-1}$. Cost of walking was calculated as oxygen consumption per unit distance ($\text{VO}_2 \text{ mL kg}^{-1} \text{ m}^{-1}$).

Physical strain was calculated as $(\text{VO}_{2\text{walk}}/\text{VO}_{2\text{peak}}) \times 100$ [4], with higher values indicating greater relative physiological demand.

Gait analysis

Three-dimensional gait analysis was performed using an eight-camera infrared motion capture system (Vantage cameras, Vicon Motion Systems, Oxford, UK). Ground reaction forces were synchronously measured using two embedded force plates (OR 6-7, AMTI, Watertown, USA). 16 retroreflective markers

were placed on the pelvis and lower limbs according to the conventional gait model (Plug-in-gait (PiG), Vicon Motion Systems, Oxford, UK). Participants walked barefoot at self-selected, comfortable walking speed along a 10-meter walkway until at least three valid trials with complete kinematic and kinetic data for each limb were obtained.

Marker trajectories and force plate data were processed using Vicon Nexus software (Vicon Motion Systems, Oxford, UK). Custom Python scripts (Python version 2.7) were used to compute gait variables and the Gait Deviation Index (GDI). For all gait variables, values from the left and right limbs were averaged for analysis.

The GDI [20] quantifies overall gait pattern deviations during walking, relative to a reference group without gait impairments and has demonstrated validity and reliability in both adults and children [20-22]. An unpublished local adult reference group data set (n = 50) was used in the calculations.

Peak ankle power (W kg^{-1}) was defined as the maximum ankle power generated during stance, normalised to body weight (kg).

The mean modified **support moment** (Nm kg^{-1}) was calculated according to Hof [23] and defined as the sum of the internal *extensor* (*ext*) moments at the hip, knee, and ankle during stance. The mean value across stance, normalised to body weight was used in statistical analysis.

Mean support moment =

$\frac{1}{2}$ (mean hip ext moment) + mean knee ext moment + $\frac{1}{2}$ (mean ankle ext moment) [23]

Clinical examination

Clinical assessments were performed bilaterally, and mean values of left and right sides were used.

Selective motor control was assessed using the Selective Classification Assessment of the Lower Extremity (SCALE) [24], which evaluates the ability to selectively activate muscles at the hip, knee, ankle, subtalar joint and toes. Scores range from 0 to 2 for each level, and are summarized up to 10 points for each side, where 10 points are the reference selective motor control [6].

Spasticity was assessed using a composite score of the Modified Ashworth Scale (MAS) [25] for hip adductors, knee flexors and -extensors and ankle plantar flexors. The MAS is an ordinal scale ranging from 0-4 (0, 1, 1+, 2, 3 and 4). The scores were recalculated to 0, 1, 1.5, 2, 3 and 4, respectively, for the analysis. After recoding, a composite score (0-16) was calculated, as done in earlier studies [26, 27], where higher scores represent more spasticity.

Statistics

All variables and residuals were assessed for normality using Shapiro-Wilk test, and by visual inspection of QQ-plots, and for heteroskedasticity using scatter plots. Normally distributed data are presented as

mean and standard deviation (SD) and non-normally distributed data as median and interquartile range (IQR). P-values less than 0.05 were considered statistically significant throughout all statistical tests.

Bivariate linear regression analyses were conducted to examine associations between each potential predictor and physical strain of walking (dependent variable). All possible predictors were entered into a multiple linear regression model and sex was included as a potential confounder. Multicollinearity was evaluated using variance inflation factors (VIF), with values below 10 considered acceptable [28].

All statistical analysis were performed using IBM SPSS statistics (version 31.0.0).

Results

Participants characteristics

Of the 100 participants included in the COWAC-study, five individuals with GMFCS level III were excluded due to missing kinetic data. Four additional participants did not complete cardiopulmonary testing because of high blood pressure (n=2) and early discharge (n=2). Two participants had missing spasticity data. Consequently, 89 participants were included in the final analysis (see flowchart of the inclusion process in Figure 1). Due to the small number of participants with GMFCS level III (n = 3), GMFCS levels II and III were pooled for statistical analyses. Participant characteristics are presented in Table 1.

Insert Table 1 approximately here.

Table 1. Descriptives of demographics, laboratory and clinical tests.

Variables	All (n = 89)	GMFCS I (n = 64)	GMFCS II and III (n = 25)
Sex (F/M), n	49/40	36/28	13/12
Age (years)	40 (12)	40 (11)	41 (13)
BMI	25.4 (4.4)	25.3 (4.4)	25.9 (4.4)
CP subtype (uni-/bilateral), n	58/31	51/13	7/18
Walking speed (m sec ⁻¹) *	1.12 (0.29)	1.22 (0.19)	0.84 (0.28)
VO ₂ peak (mL kg ⁻¹ min ⁻¹) *	36.0 (12.3)	36.6 (11.8)	32.5 (10.4)
VO ₂ walk (mL kg ⁻¹ min ⁻¹) *	17.4 (4.2)	17.2 (3.8)	18.1 (6.4)
O ₂ -cost (mL kg ⁻¹ m ⁻¹) *	0.25 (0.08)	0.23 (0.05)	0.35 (0.21)
Physical strain (%VO ₂ peak) *	49.3 (16.2)	45.7 (12.4)	58.0 (13.4)
SCALE composite score ^ (0-10) *	8.0 (2.5)	8.5 (2.4)	7.0 (2.8)
MAS composite score ^ (0-16) *	1.5 (2.6)	1.0 (1.8)	3.0 (3.0)
GDI ^ (0-100) *	79.4 (16.4)	82.0 (11.8)	66.5 (10.0)
Peak ankle power ^ (W kg ⁻¹)	3.4 (1.1)	3.8 (0.9)	2.4 (0.8)
Mean support moment in stance ^ (Nm kg ⁻¹)	0.42 (0.13)	0.42 (0.14)	0.44 (0.11)

Results are presented as mean (standard deviation) for normally distributed data. *Indicating non-normally distributed data, presented as median values (interquartile range).

^Variables are mean values of left and right leg. Abbreviations: BMI = Body Mass Index, CP = Cerebral Palsy, GDI = Gait Deviation Index, GMFCS = Gross Motor Function Classification System, MAS = Modified Ashford Scale, n = number of participants, SCALE = Selective Control Assessment of Lower Extremities, VO₂peak = Peak oxygen uptake, VO₂walk = Oxygen consumption during preferred walking speed.

Regression analyses

During the model building process, we noted a correlation coefficient, $r < 0.7$ (range 0.001-0.622) for all relationships. The GDI was associated with MAS ($r = -0.524$), SCALE ($r = 0.577$), peak ankle power ($r = 0.622$), GMFCS level ($r = -0.614$) and mean support moment ($r = -0.421$). The GMFCS level was associated with GDI ($r = -0.614$), peak ankle power ($r = -0.616$), MAS composite score ($r = 0.432$) and SCALE composite score ($r = -0.426$). These correlation values show weak-to-moderate correlations following Munro [29], and VIF statistics (range 1.0-3.1) for the multiple regression model are all below

the acceptable limit of 10 [28]. All VIF and tolerance values are provided in Supplementary table A and collinearity statistics in Supplementary table B.

Bivariate linear regression analyses showed that unadjusted age ($r = 0.356$, $r^2 = 0.127$), GMFCS-level ($r = 0.392$, $r^2 = 0.154$), MAS composite score ($r = 0.390$, $r^2 = 0.152$), GDI ($r = -0.406$, $r^2 = 0.165$) and peak ankle power ($r = -0.355$, $r^2 = 0.126$) significantly predicts physical strain of walking (all $p < 0.001$; Table 2). All potential predictors were entered into the multiple regression model.

Insert Table 2 approximately here.

Table 2. Bivariate and multiple linear regression analyses with physical strain of walking as dependent variable, and sex, age, GMFCS, SCALE, MAS, GDI, peak ankle power and Mean Support moment as independent variables.

Potential predictors	Bivariate analysis					Final Multiple model					
	B (SE)	95 % CI	B*	p	R ²	B (SE)	95 % CI	B*	p	R ²	Adj. R ²
									<0.001	0.370	0.307
Sex (F=1, M=2)	-1.490 (2.473)	-6.405; 3.424	-0.064	0.548	0.004						
Age (years)	0.348 (0.098)	0.154; 0.543	0.356	< 0.001	0.127	0.320 (0.088)	0.144; 0.496	0.327	<0.001		
GMFCS-level II/III	10.026 (2.523)	5.011; 15.041	0.392	< 0.001	0.154						
SCALE mean score (0-10)	-0.908 (0.746)	-2.391; 0.575	-0.129	0.227	0.017						
MAS composite score (0-16)	2.647 (0.670)	1.315; 3.979	0.390	< 0.001	0.152	1.475 (0.727)	0.013; 2.893	0.214	0.048		
GDI mean (0-100)	-0.400 (0.097)	-0.592; -0.208	-0.406	< 0.001	0.165						
Peak ankle power (W kg ⁻¹)	-3.817 (1.076)	-5.957; -1.678	-0.355	< 0.001	0.126						
Mean Support moment (Nm kg ⁻¹)	14.296 (9.125)	-3.842; 32.433	0.166	0.121	0.027						

All variables are mean values of left and right leg. *standardized B. Abbreviations: GDI = Gait Deviation Index; GMFCS = Gross Motor Function Classification System; MAS = Modified Ashford Scale; SCALE = Selective Control Assessment of Lower Extremities.

In the multiple regression model (Table 2), age ($B = 0.320$, $CI\ 95\% = 0.144-0.496$, $p < 0.001$) and MAS composite score ($B = 1.475$, $CI\ 95\% = 0.013-2.893$, $p = 0.048$) were independent predictors of physical strain of walking, after adjusting for sex, GMFCS level, SCALE, GDI, peak ankle power and mean support moment. The model ($F(8,80) = 5.88$, $p < 0.001$) explained about 30% of the variance in physical strain ($R^2 = 0.370$, adjusted $R^2 = 0.307$).

Discussions

This study examined whether predictors of oxygen cost of walking identified in children with CP and in healthy older adults also predict physical strain of walking in adults with CP and found that it's not always the case when variables are controlled for each other. When accounting for biomechanical and clinical variables, age and spasticity were the variables that independently predicted physical strain of walking. Gait-related variables explained the variation in physical strain only in unadjusted analyses.

Age as a predictor of physical strain

Age emerged as a significant predictor of physical strain. As physical strain measurements are relative to maximal aerobic capacity, which is known to decrease with age [30], this could indicate that age-related increases in physical strain are attributable to reductions in aerobic capacity. Even though the level of physical activity in ambulant adults with CP is similar to the general population [31], the level of physical activity is known to decrease with age [32]. Furthermore, physical activity is related to physical strain in adults with CP [2]. Less physical activity with increasing age will probably reduce VO_{2peak} and thereby the physical strain of walking at preferred speed will increase. Although the effect size of age was modest, corresponding to approximately 3% higher average physical strain per decade, the cumulative impact over adulthood may be clinically meaningful, particularly for individuals operating close to their maximal capacity.

The present findings support the notion that ageing itself contributes to increased physical strain, even after accounting for functional level, clinical measures and gait characteristics. This agrees with research on healthy elderly showing reduced maximum capacity and increasing energy demands of walking with higher age, with reduced force-generating capacity of the muscles as a possible reason [33]. Hence, changes in neuromuscular efficiency, motor coordination or secondary impairments associated with ageing with CP [34] may also contribute. Further, increased prevalence of comorbidities in persons with CP [35] may also play a role in increasing physical strain of walking with ageing in this population.

Spasticity affecting physical strain

Consistent with previous findings showing an association between clinically measured spasticity (MAS) and the oxygen cost of walking [6] in children, spasticity was identified as an independent predictor of physical strain in adults with CP. These results support the notion that spasticity contributes not only to impaired motor function, but also to the increase the energy demands of walking.

Although spasticity is widely recognized as a factor limiting walking function in individuals with CP [36], it is noteworthy that MAS, assessed during passive, non-weightbearing movement [25] was predictive of a dynamic gait-related outcome such as physical strain. This supports the causal framework proposed by Gill et al (2023) [6], suggesting that clinical measures of spasticity capture functionally relevant aspects of motor impairment. In contrast, Gagnat et al. (2023), reported that muscle co-contraction during walking, considered a measure of spasticity-related overactivity, was not predictive of cost of walking in children with CP [7]. This discrepancy raises the possibility that MAS, which reflects a combination of passive stiffness and velocity-dependent resistance, may provide a more comprehensive representation of the neuromuscular constraints influencing walking than isolated measures of muscle activation. Consequently, further investigation is warranted to clarify how different muscle properties contribute to physical strain during walking.

Despite the observed association, the effect size was modest, with a one-point higher mean MAS composite score corresponding to an approximate 1.5% higher physical strain. This relatively small effect of spasticity may explain the findings such as Zaino et al (2020), where selective dorsal rhizotomy

led to a significant reduction of spasticity, but did not significantly alter the cost of walking [37]. In that study, the reduction in MAS was approximately one point, which based on the current findings may have been insufficient to produce a measurable change in walking-related oxygen cost.

Importantly, the clinical relevance of these changes remains difficult to interpret due to the lack of established benchmarks for minimal detectable change or minimal clinically important difference in physical strain among adults with CP. Establishing such thresholds is essential for determining whether observed changes reflect meaningful improvements in functional capacity and should be prioritized in future research.

Biomechanic measures

Although gait deviations and peak ankle power were associated with physical strain in bivariate analyses, they did not independently predict physical strain in the multiple regression model. This contrasts with findings for cost of walking in children with CP [7] and healthy elderly [8] and highlights the conceptual distinction between the oxygen cost of walking (per meter) and the physical strain (relative physiological effort) of walking. Differences in walking speed may partly explain this discrepancy. Greater gait deviations [38] and reduced push-off power are associated with reduced walking speed [39]. A lower walking speed usually reflects a lower oxygen consumption during walking [40], potentially reducing overall physical strain. However, as cost of walking is expressed as oxygen consumption per meter, it is highly dependent on walking speed and may not reflect the physical strain of walking [41] at slower speeds.

Even though GDI is associated with walking function [42] and cost of walking in children with CP [7], and both are related to daily activity [2, 38], GDI did not independently predict physical strain in this study. The use of a gait index, where several joint kinematics are included, might have made specific gait deviations less prominent [43]. Further, a mean of most and least affected leg is used in the calculations, which might have decreased the importance of the GDI, especially in this study where 65% of the sample have unilateral CP.

Mean support moment and peak ankle power were also considered potential predictors of physical strain as increased hip- and knee flexion [44] and a shift in propulsion strategy from ankle to hip [9, 10] has been reported to be important factors in cost of walking measurements. The lack of explanatory power of these biomechanical measures could be explained by the heterogeneity of gait patterns in the adult population. For example, flexed knee gait (crouch gait) is known to be an energy-demanding [36] gait pattern, which often reflect a large mean support moment [45] and reduced ankle push-off power [46]. However, a toe-walking pattern (jump-gait pattern) can also be energy-demanding due to the increased vertical movement of the body [47]. In this instance, the mean support moment would be small, as the leg is quite straight through stance phase and the peak ankle power is less affected, despite a shift in timing, because of the “jump-gait” pattern. This means that increased energy demand during walking possibly could be accompanied by both small and large mean support moments during walking.

Clinical implications

The present findings suggest that spasticity is an important therapeutic target to reduce the physical strain of walking in this population. Multimodal interventions that integrate management of spasticity and age-related and neuromotor impairments with strategies aimed at improving aerobic capacity may be particularly effective. Interventions targeting isolated gait features may have limited impact on overall physical strain, as they do not sufficiently address the broader physiological and neuromuscular contributors to physical strain of walking in adults with CP.

Limitations

The sample predominantly comprised high-functioning adults with CP (unilateral and GMFCS level I), which makes the results only generalizable to people with higher physical function and less to people with lower physical function. Further, the pooling of participants with GMFCS level II and III might have influenced the results. The inclusion of participants might have increased the difference between GMFCS levels I and II/III, as the level III participants might have affected the results of the GMFCS level II participants' function. However, excluding the three GMFCS level III participants able to walk without support, could have led to less variation in the data, and thereby minimizing the effect of the different functional and clinical measures.

The clinical measures, although widely used in the clinic, have limitations to its reliability [48]. To try to compensate for this, we used strict test protocols and two assessors for each assessment, where at least one was an experienced (> 10 years) clinician. Further, the use of composite scores may have limited the effect of these scores in the model, as the effect of specific muscles might have been reduced.

Furthermore, a mean of left and right leg was used for all measures. This could have reduced the effect of side-specific measures from the most affected leg, especially in those with unilateral CP. However, the mean score of both legs was used to retain a difference between those with unilateral and bilateral CP.

In addition, the interrelationship between kinetic, kinematic and clinical variables could have diminished the influence of the different independent variables. The effect of spasticity on GDI, peak ankle power and physical strain may moderate the independent effect of peak ankle power and GDI on physical strain. The VIF for the GDI is above 3, and the tolerance value 0.32, indicating that about 70% of the variance in GDI is accounted for in the other variables (See Tables 2 and 3 in the Supplementary files). However, the multicollinearity diagnostics did not show high variance proportions for GDI and other variables in the same dimensions, and it is therefore included in the multiple model. This may be explained by the GDI being influenced by a combination of the different clinical and kinetic variables, and the results should therefore be interpreted with caution.

Future directions

Future research should include longitudinal designs to capture changes in physical strain across adulthood, and to better understand the mechanisms underlying age-related increases in physical strain in this population. Future studies should also include dynamic measures of muscle activity (i.e. electromyography) and measurements of muscle-tendon force (i.e. shear wave tensiometer) to possibly help understand the role of neuromuscular control and load in determining physical strain. Further, studies investigating the contribution of physiologic properties of the cardiorespiratory system and muscle properties are warranted. Finally, studies examining the relationship between physical strain, perceived fatigue, and participation outcomes may provide further insight into clinically meaningful targets for interventions.

Conclusions

Physical strain of walking in adults with CP was independently predicted by age and spasticity when controlled for gait deviations, peak ankle power, mean support moment, motor control and sex. The effect sizes (B-values) were moderate, however, in this population even a small increase in an already elevated physical strain could be of clinical importance. Targeting spasticity in treatment and preserving aerobic capacity should be a focus in interventions for reducing physical strain of walking in adults with CP.

This highlights that predictors of cost of walking in children with CP and healthy elderly, except for spasticity, do not predict physical strain of walking in adults with CP, possibly due to cost of walking being dependent on walking speed.

Declarations

Ethics approval and consent to participate

The study was approved by the Regional Committee for Medical and Health Research Ethics, South-East Norway (ref: 530205). Data handling clearance was obtained from the Norwegian Agency for Shared Services in Education and Research (ref: 655023).

Consent for publication

Not applicable.

Availability of data and materials

The dataset supporting the conclusions of this article is not publicly available due to Norwegian laws regarding data protection and research ethics.

Competing interests

The authors declare that they have no competing interests.

Funding

The study did not accept any external funding.

Author's contribution

SLKH and LR planned the study. SLKH was responsible for the data collection regarding clinical examination and biomechanical data, while EL was responsible for the data collection and extraction regarding the cardiopulmonary exercise testing on the treadmill. EIB and LR assisted in the data collection. EIB exported and calculated the biomechanical data. SLKH analyzed and interpreted the data. LR, AO and TG supervised SLKH in the analysis and interpretation of the data. SLKH was the main author of the manuscript, but all authors contributed with discussions and in editing of the final edition of the manuscript.

Acknowledgements

We want to acknowledge the contribution from the adults with CP participating in this study. Thank you for your time and effort in participating in the data collection. Further, we want to thank the staff at the Motion Laboratory and Clinical Physiological Laboratory at Sunnaas Rehabilitation Hospital. In addition, the major contribution of Marit Eline Spørck and Petra Ahlvin Nordby in the inclusion phase of this study. At last, we want to thank Sunnaas Rehabilitation Hospital, Oslo Metropolitan University and the Norwegian Cerebral Palsy Association for their support in this project.

Declaration of generative AI and AI assisted technologies in the writing process

During the preparation of this work, the authors utilized Sikt-KI-Chat and Microsoft 365 Copilot to enhance language and readability. The authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

References

1. Opheim, A., et al., *Walking function, pain, and fatigue in adults with cerebral palsy: a 7-year follow-up study*. Dev Med Child Neurol, 2009. **51**(5): p. 381-8.
2. Slaman, J., et al., *Physical strain of walking relates to activity level in adults with cerebral palsy*. Arch Phys Med Rehabil, 2013. **94**(5): p. 896-901.
3. Jahnsen, R., et al., *Fatigue in adults with cerebral palsy in Norway compared with the general population*. Dev Med Child Neurol, 2003. **45**(5): p. 296-303.
4. Balemans, A.C., et al., *Physical Strain: A New Perspective on Walking in Cerebral Palsy*. Arch Phys Med Rehabil, 2017. **98**(12): p. 2507-2513.
5. Sarmiento, C.A., et al., *Evolving rehabilitation needs across the lifespan: a mixed methods study with adults with cerebral palsy*. Disabil Rehabil, 2026. **48**(5): p. 1391-1406.

6. Gill, P.K., et al., *Causal modelling demonstrates metabolic power is largely affected by gait kinematics and motor control in children with cerebral palsy*. PLoS One, 2023. **18**(5): p. e0285667.
7. Gagnat, Y., et al., *The relation of energy cost of walking with gait deviation, asymmetry, and lower limb muscle co-activation in children with cerebral palsy: a retrospective cross-sectional study*. BMC Musculoskelet Disord, 2023. **24**(1): p. 111.
8. Boyer, K.A., et al., *Systematic review and meta-analysis of gait mechanics in young and older adults*. Exp Gerontol, 2017. **95**: p. 63-70.
9. Fallah, N. and O.N. Beck, *The metabolic cost of producing joint moments is greater at the hip than at the ankle*. J Exp Biol, 2025. **228**(2).
10. Pieper, N.L., et al., *The metabolic and mechanical consequences of altered propulsive force generation in walking*. J Biomech, 2021. **122**: p. 110447.
11. Lundh, S., S. Nasic, and J. Riad, *Fatigue, quality of life and walking ability in adults with cerebral palsy*. Gait Posture, 2018. **61**: p. 1-6.
12. Waters, R.L. and S. Mulroy, *The energy expenditure of normal and pathologic gait*. Gait Posture, 1999. **9**(3): p. 207-31.
13. Klund-Hansen, S.L., et al., *A 16-year follow-up of walking function, fatigue, and pain in adults aged 34–65 years with spastic cerebral palsy*. Journal of Rehabilitation Medicine, 2025. **57**: p. 43295.
14. Rosenbaum, P., et al., *A report: the definition and classification of cerebral palsy April 2006*. Dev Med Child Neurol Suppl, 2007. **109**: p. 8-14.
15. Tabard-Fougère, A., et al., *How Do Gait Outcomes Evolve in Adults with Spastic Cerebral Palsy Who Received Orthopedic Treatment in Childhood?* Children (Basel), 2026. **13**(1).
16. Klund-Hansen, S., et al., *Cost of walking in adults with Cerebral palsy (COWAC) – a study protocol and case presentation*. Gait & Posture, 2023. **106**: p. S101.
17. Maanum, G., et al., *Effects of botulinum toxin A in ambulant adults with spastic cerebral palsy: a randomized double-blind placebo controlled-trial*. Journal of Rehabilitation Medicine, 2011. **43**(4): p. 338-347.
18. McCormick, A., et al., *Stability of the Gross Motor Function Classification System in adults with cerebral palsy*. Dev Med Child Neurol, 2007. **49**(4): p. 265-9.
19. Sandra Linnea, K.-H., et al., *Physical strain during walking in adults with cerebral palsy: A construct validity study*. Archives of Rehabilitation Research and Clinical Translation, 2026: p. 100617.
20. Schwartz, M.H. and A. Rozumalski, *The Gait Deviation Index: a new comprehensive index of gait pathology*. Gait Posture, 2008. **28**(3): p. 351-7.
21. Maanum, G., et al., *Face and construct validity of the Gait Deviation Index in adults with spastic cerebral palsy*. J Rehabil Med, 2012. **44**(3): p. 272-5.
22. Rasmussen, H.M., et al., *Gait Deviation Index, Gait Profile Score and Gait Variable Score in children with spastic cerebral palsy: Intra-rater reliability and agreement across two repeated sessions*. Gait Posture, 2015. **42**(2): p. 133-7.

23. Hof, A.L., *On the interpretation of the support moment*. Gait Posture, 2000. **12**(3): p. 196-9.
24. Fowler, E.G., et al., *Selective Control Assessment of the Lower Extremity (SCALE): development, validation, and interrater reliability of a clinical tool for patients with cerebral palsy*. Dev Med Child Neurol, 2009. **51**(8): p. 607-14.
25. Bohannon, R.W. and M.B. Smith, *Interrater reliability of a modified Ashworth scale of muscle spasticity*. Phys Ther, 1987. **67**(2): p. 206-7.
26. Tabard-Fougère, A., et al., *Are Clinical Impairments Related to Kinematic Gait Variability in Children and Young Adults With Cerebral Palsy?* Front Hum Neurosci, 2022. **16**: p. 816088.
27. Papageorgiou, E., et al., *Are spasticity, weakness, selectivity, and passive range of motion related to gait deviations in children with spastic cerebral palsy? A statistical parametric mapping study*. PLoS One, 2019. **14**(10): p. e0223363.
28. Field, A., *Discovering statistics using IBM SPSS statistics*. 4th ed. 2014, London: Sage publications Ltd.
29. Kellar, S.P. and E.A. Kelvin, *Munro's Statistical Methods for Health Care Research*. 6 ed. 2012: LWW.
30. Edvardsen, E., et al., *Reference values for cardiorespiratory response and fitness on the treadmill in a 20- to 85-year-old population*. Chest, 2013. **144**(1): p. 241-248.
31. van Rijssen, I.M., et al., *The 24-Hour Physical Activities in Adults With Cerebral Palsy and Their Adherence to the 24-Hour Movement Guideline*. Arch Phys Med Rehabil, 2025. **106**(5): p. 696-703.
32. Westerterp, K.R., *Changes in physical activity over the lifespan: impact on body composition and sarcopenic obesity*. Obes Rev, 2018. **19 Suppl 1**: p. 8-13.
33. Martin, P.E., D.E. Rothstein, and D.D. Larish, *Effects of age and physical activity status on the speed-aerobic demand relationship of walking*. J Appl Physiol (1985), 1992. **73**(1): p. 200-6.
34. Gravholt, A., et al., *Motor function and gait decline in individuals with cerebral palsy during adulthood: a narrative review of potential physiological determinants*. Eur J Appl Physiol, 2024. **124**(10): p. 2867-2879.
35. Hollung, S.J., et al., *Comorbidities in cerebral palsy: a patient registry study*. Dev Med Child Neurol, 2020. **62**(1): p. 97-103.
36. Gage, J.R., et al., *The Identification and Treatment of Gait Problems in Cerebral Palsy*. Vol. 2. 2009.
37. Zaino, N.L., et al., *Energy consumption does not change after selective dorsal rhizotomy in children with spastic cerebral palsy*. Dev Med Child Neurol, 2020. **62**(9): p. 1047-1053.
38. Wilson, N.C., et al., *Gait Deviation Index Correlates With Daily Step Activity in Children With Cerebral Palsy*. Arch Phys Med Rehabil, 2015. **96**(10): p. 1924-7.
39. Geertsen, S.S., et al., *Impaired gait function in adults with cerebral palsy is associated with reduced rapid force generation and increased passive stiffness*. Clin Neurophysiol, 2015. **126**(12): p. 2320-9.
40. Ralston, H.J., *Energy-speed relation and optimal speed during level walking*. Int Z Angew Physiol, 1958. **17**(4): p. 277-83.

41. Gjovaag, T., et al., *Assessment of aerobic capacity and walking economy of unilateral transfemoral amputees*. Prosthet Orthot Int, 2014. **38**(2): p. 140-7.
42. Malt, M.A., et al., *Correlation between the Gait Deviation Index and gross motor function (GMFCS level) in children with cerebral palsy*. J Child Orthop, 2016.
43. Barton, G.J., et al., *A gait index may underestimate changes of gait: a comparison of the Movement Deviation Profile and the Gait Deviation Index*. Comput Methods Biomech Biomed Engin, 2015. **18**(1): p. 57-63.
44. Noorkoiv, M., et al., *Predictors of Walking Efficiency in Children With Cerebral Palsy: Lower-Body Joint Angles, Moments, and Power*. Phys Ther, 2019. **99**(6): p. 711-720.
45. McNee, A.E., et al., *Lower limb extensor moments in children with spastic diplegic cerebral palsy*. Gait Posture, 2004. **20**(2): p. 171-6.
46. Ebrahimi, A., et al., *American Society of Biomechanics Clinical Biomechanics Award 2021: Redistribution of muscle-tendon work in children with cerebral palsy who walk in crouch*. Clin Biomech (Bristol), 2023. **102**: p. 105871.
47. Opheim, A., et al., *Balance in relation to walking deterioration in adults with spastic bilateral cerebral palsy*. Phys Ther, 2012. **92**(2): p. 279-88.
48. Nourizadeh, M., et al., *Methods of muscle spasticity assessment in children with cerebral palsy: a scoping review*. J Orthop Surg Res, 2024. **19**(1): p. 401.

Figures

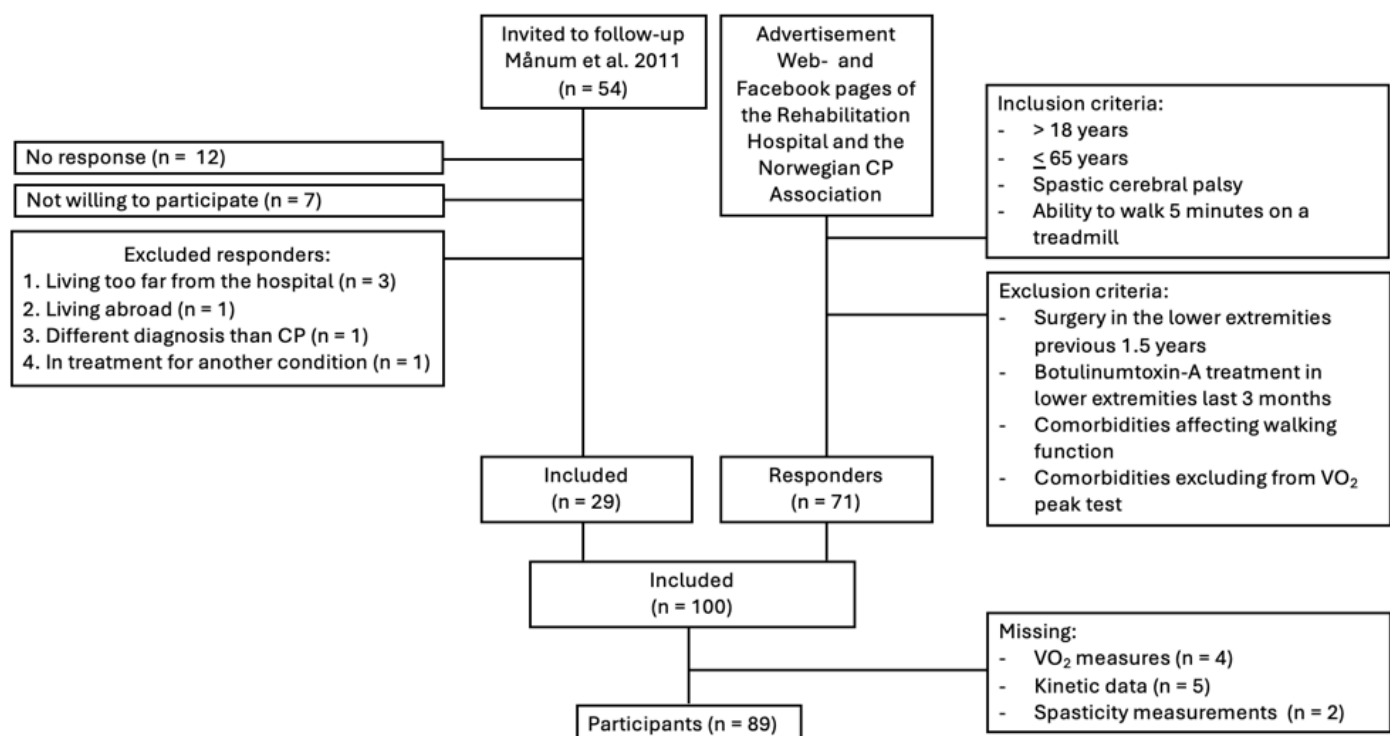


Figure 1

Inclusion and exclusion process

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Supplementaryfiles.docx](#)