Longitudinal Growth, Diet, and Physical Activity in Young Children With Cerebral Palsy

Stina Oftedal, APD,[a,b] Peter S.W. Davies, PhD,[b] Roslyn N. Boyd, PT, PhD,[a] Richard D. Stevenson, MD,[c] Robert S. Ware, PhD,[d] Piyapa Keawutan, PT, MSc,[a,e] Katherine A. Benfer, MPH, PhD,[a] Kristie L. Bell, APD, PhD[a,f]

abstract

OBJECTIVES: To describe the longitudinal relationship between height-for-age *z* score (HZ), growth velocity *z* score, energy intake, habitual physical activity (HPA), and sedentary time across Gross Motor Function Classification System (GMFCS) levels I to V in preschoolers with cerebral palsy (CP).

METHODS: Children with CP (*n* = 175 [109 (62.2%) boys]; mean recruitment age 2 years, 10 months [SD 11 months]; GMFCS I = 83 [47.2%], II = 21 [11.9%], III = 28 [15.9%], IV = 19 [10.8%], V = 25 [14.2%]) were assessed 440 times between the age of 18 months and 5 years. Height/length ratio was measured or estimated via knee height. Population-based standards were used to calculate HZ and growth velocity *z*-score by age and sex categories. Feeding method (oral or tube) and gestational age at birth (GA) were collected from parents. Three-day ActiGraph and food diary data were used to measure HPA/sedentary time ratio and energy intake, respectively. Oropharyngeal dysphagia was rated with the Dysphagia Disorder Survey (part 2, Pediatric). Analysis was undertaken with mixed-effects regression models.

RESULTS: For GMFCS level I, height and growth velocity did not differ from population-level growth standards. Children in levels II to V were significantly shorter, and those in levels III to V grew significantly more slowly than those in level I. There was a significant positive association between HZ and GA at all GMFCS levels. Energy intake, HPA, sedentary time, Dysphagia Disorder Survey score, and feeding method were not significantly associated with either height or growth velocity once GMFCS level was accounted for.

CONCLUSIONS: Functional status and GA should be considered when assessing the growth of a child with CP. Research into interventions aimed at increasing active movement in GMFCS levels III to V and their efficacy in improving growth and health outcomes is warranted.

[a]*Queensland Cerebral Palsy and Rehabilitation Research Centre, The University of Queensland Child Health Research Centre, Brisbane, Australia;* [b]*Children's Nutrition Research Centre, and* [d]*School of Public Health, The University of Queensland, Brisbane, Australia;* [c]*Division of Developmental Pediatrics, Department of Pediatrics, University of Virginia School of Medicine, Charlottesville, Virginia;* [e]*Department of Physical Therapy, Faculty of Allied Health Sciences, Thammasat University, Pathumthani, Thailand; and* [f]*Dietetics and Food Services, Lady Cilento Children's Hospital, Children's Health, Queensland, South Brisbane, Australia*

Ms Oftedal conceptualized and designed the study, collected data, carried out the analysis, and drafted, revised, critically reviewed, and finalized the manuscript; Professors Davies, Boyd, and Stevenson, and Dr Bell conceptualized and designed the study and critically reviewed the manuscript; Dr Ware contributed to the analysis and interpretation of the data and critically revised the manuscript; Ms Keawutan and Dr Benfer contributed to the acquisition and analysis of data and critically revised the manuscript; and all authors approved the final manuscript for submission.

DOI: 10.1542/peds.2016-1321

WHAT'S KNOWN ON THIS SUBJECT: Children with cerebral palsy (CP), particularly with moderate to severe gross motor limitations, are typically shorter and grow more slowly than children with typical development. The influence of diet, physical activity, and sedentary time on growth in children with CP has not been elucidated.

WHAT THIS STUDY ADDS: Ambulatory status and gestational age at birth were significant predictors of height in children with CP, whereas only ambulatory status predicted growth velocity. Energy intake, habitual physical activity, and sedentary time did not explain additional variation in growth measures.

To cite: Oftedal S, Davies PS, Boyd RN, et al. Longitudinal Growth, Diet, and Physical Activity in Young Children With Cerebral Palsy. *Pediatrics.* 2016;138(4):e20161321

Growth is a fundamental marker of health and well-being in all children.[1] Children with cerebral palsy (CP), particularly those with moderate to severe functional limitations, grow differently from children with typical development.[2,3] CP is defined as a group of permanent disorders of movement and posture attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain.[4] The etiology of poor growth in children with CP is hypothesized to include nutritional factors, physical factors, and factors related to the brain lesion itself.[5] Oropharyngeal dysphagia (OPD) is prevalent in up to 85% of children with CP across the spectrum of functional capacity.[6] Parent-reported difficulty with feeding and the resulting impact on dietary intake corresponds to decreased nutritional status as measured by height, weight, body fat stores, and muscle mass in children with moderate to severe CP, and the presence of a gastrostomy feeding tube has been associated with greater height and weight.[2,7] Physical factors related to poor growth include the physical impairment and lower levels of physical activity observed in children with CP that result in decreased weight bearing and mechanical stress on bones, leading to decreased bone formation and bone growth in children with CP.[8,9] Factors related to the brain lesion itself can affect growth directly, via negative neurotropic effects, or indirectly, via endocrine factors such as growth hormone deficiency.[10,11] The prevalence of preterm birth and low birth weight (BW) is higher in children with CP,[12] and these factors are commonly associated with shorter stature in typically developing children.[13] To our knowledge the influence of preterm birth on growth in children with CP has not been elucidated. However, a recent study identified being small for gestational age as a significant predictor of short stature in children with CP aged 0 to 5 years born at ≥36 weeks' gestation.[14]

Specific growth charts for children with CP have been developed to identify whether a child's pattern of growth is typical given his or her gross motor function.[2] These growth charts are descriptive, and although they do not necessarily depict desirable growth in this population, they may be used for early identification of nutritional or metabolic difficulties.[15] This article aims to investigate the longitudinal relationship between stature, growth velocity, energy intake, habitual physical activity (HPA), and sedentary time for each Gross Motor Function Classification System (GMFCS) level in children with CP aged 18 months to 5 years while controlling for OPD, gestational age at birth (GA), BW, and sex.

METHODS

Participants

Children with a diagnosis of CP born in Queensland between 2006 and 2009 were eligible to participate. Children entered the study from 18 months corrected age (CA), and measures were taken at either 18, 24, 30, or 36 months CA, then again at 36 or 48 months CA depending on the timing of their first assessment, and third at 60 months CA. Children entering the study aged >36 months did not have all 3 assessments. Some children were assessed on 4 occasions because of their participation in a separate substudy.[16]

Children were recruited as part of the Queensland CP Child Study of Growth, Nutrition and Physical Activity (n = 182)[17] in conjunction with Queensland CP Child Study of Motor Function and Brain Development (n = 227).[18] Queensland-born children with a diagnosis of CP were eligible for inclusion.[4] Children with a progressive or neurodegenerative lesion were excluded from the study.[19] Written informed consent was obtained from the child's primary caretaker.[17,18]

Growth Measures

Height or supine length was measured to the nearest completed millimeter on a portable stadiometer (Shorr Productions, Olney, MD). If direct measures of length or height were not available because of joint contractures, scoliosis, or involuntary movements making standing or lying straight difficult or impossible, height was estimated based on knee height and validated equations.[20] If estimated height was used for a child at 1 assessment, it was also used at all other available assessments to ensure consistency. Weight was measured to the nearest 100 g on chair scales (Seca, Hamburg, Germany). Two measures of height/length ratio and weight were taken, and the average was used. Height z-scores (HZ) and weight z-scores were identified based on age- and sex-based norms from children with typical development.[21]

To assess linear growth compared with expected age- and sex-specific growth over a defined time interval, growth velocity z-scores (GvZ) were calculated. If >2 measurement occasions were available for 1 child, growth velocity between each age gap was calculated if the interval was >0.8 years but <2.5 years.[22] The age used in the GvZ calculation was the midpoint age between the 2 assessments and will be referred to as age at GvZ calculation.

Risk Factors for Poor Growth

Children were classified on the GMFCS.[23] GA and BW were collected from parents during the initial physician interview.[17] Severity of OPD was determined from the Dysphagia Disorder Survey (DDS), Pediatric Part 2 raw score, a valid and reliable measure of dysphagia.[24–26] It consists

of a series of binary judgments of feeding competency on 8 ingestion functions for purée, chewable food, and fluids. Possible total scores range from 0 (no impairment) to 22 (profound impairment, including those fed exclusively via gastrostomy tube).[27] Feeding method (oral versus gastrostomy tube) was determined by parent report via questionnaire. Qualified dietitians, physiotherapists, and a pediatric speech pathologist assessed anthropometric measures, gross motor function, and mealtimes, respectively.

Modifiable Lifestyle Factors

Energy intake in megajoules was assessed with a validated weighed 3-day food and fluid diary.[16] Parents were supplied with food scales and a food diary with detailed instructions, and weighed food and fluid intake for 2 weekdays and a weekend day.[16] The food diaries were analyzed in FoodWorks 7 (Xyris Software, Brisbane, Australia). The 3-day average HPA (cpm) and sedentary time (percentage of the day) were measured on an ActiGraph activity monitor (model GT3M/GT3×; ActiGraph, Pensacola, FL), which has been validated in a young CP population.[28,29] Parents were given an ActiGraph for their child to wear for 2 weekdays and a weekend day while they completed a wear time log.[8]

Analysis

Student's t tests and χ^2 tests were used to assess the association between clinical characteristics and study participation (more than once versus once only). Longitudinal analysis was undertaken via multilevel mixed-effects regression models, which account for repeat measurements within participants.[30] In all mixed-effects models a child's identification number was entered as a random effect. For the HZ analysis, the 5 GMFCS levels I to V were used. For the GvZ analysis 3 groupings (GMFCS I–II ambulatory, GMFCS III marginally ambulatory, and GMFCS IV–V nonambulatory) were used because of the small number of participants with >1 height measure point available in the GMFCS II and IV levels. Age-related changes were estimated, and variables significantly associated with the outcomes HZ and GvZ were identified. Variables investigated were sex, DDS score, gastrostomy tube feeding, BW, sedentary time, HPA, energy intake, and age terms (age at assessment, age at GvZ calculation, and GA). All continuous variables were centered at the lowest observed variable in the data set so as to not regress to, for example, a BW or GA of 0. Linear and quadratic age terms were used to investigate possible nonlinear relationships. When the models were built, first univariable analyses were conducted to identify variables significantly associated with the outcomes at the $P < .25$ level. Second, identified variables were entered into individual multivariable models that included GMFCS level as an explanatory variable. Third, all remaining variables significant at the $P < .05$ level after controlling for GMFCS level were included in a combined multivariable model. Variables were retained in the final model if they were statistically significant at $P < .05$ by the likelihood ratio test. Analyses were performed in Stata version 13.0 (Stata Corp, College Station, TX).

RESULTS

Participants

A total of 175 children participated in the CP child study, and in 147 (84%) of these GvZ could be calculated because they participated on >1 occasion. In total, 440 assessments were available for inclusion in the HZ analysis, and 241 pairs of growth measures were available for the GvZ analysis. Table 1 shows the number of children recruited at each age band and how many repeated measures were available. The distribution of GMFCS levels in the sample was representative of the Australian population of people with CP[12] but with a higher proportion of children classified as GMFCS I (47% vs 36%) and GMFCS III (16% vs 11%) and a lower proportion of children classified as GMFCS II (12% vs 25%). This difference could reflect the young age of the study population.[31] Participant characteristics by age group are displayed in Table 2.

Stature

The associations between candidate variables and HZ are reported in Table 3. Feeding method, GA, BW, DDS score, HPA, sedentary time, and energy intake were all identified as candidates for inclusion in a multivariable model including GMFCS level ($P < .25$). BW and GA both contributed significantly to the model at $P < .05$ when entered into a multivariate model that included GMFCS level, whereas HPA, sedentary time, DDS, feeding method, and energy intake did not and were excluded from additional testing. GA and BW were highly correlated ($r^2 = 0.83$, $P < .001$), and BW lost significance once GA was added to the combined model. GA had a quadratic effect. The final model for HZ includes interactions between GMFCS and age and GA.

The mixed-effects linear regression analysis of HZ (Table 4; model A) showed that as a group, children with CP at 18 months of age had an HZ significantly below 0 (ie, their mean HZ score was significantly lower than what is expected in a typically developing population). HZ did not significantly change between 18 months and 5 years. When the sample was stratified by GMFCS (Table 4; model B), HZ was not significantly different from 0 for GMFCS level I, but GMFCS levels II to V had an HZ significantly below that of GMFCS level I. In the final model the GA terms and the GMFCS by age at assessment term

were introduced (Table 4; model C). Preterm-born children in all GMFCS groups were significantly shorter than age- and sex-specific reference children with typical development. There is a significant, positive, curvilinear relationship between GA and HZ, so although HZ significantly increased for each week past 23 weeks a child was born, the increase diminished the closer to term a child was born (Table 4; model C). The interaction of GMFCS and age at assessment showed that HZ significantly declined between the ages of 18 months and 5 years in GMFCS levels IV and V, whereas GMFCS level I showed a slight but significant improvement in HZ each year.

Growth Velocity

The association between candidate variables and GvZ are reported in Table 3. The variables gastrostomy tube feeding, DDS score, sedentary time, and energy intake were identified for additional investigation in individual multivariable models ($P < .25$). None of the variables retained significance once GMFCS level was entered into the models. The mixed-effects analysis of GvZ (Table 4; model D) showed that as a group, the GvZ of children with CP at the age of 18 months was not significantly below 0, and GvZ was stable between the ages of 18 months and 5 years. When GMFCS levels were added to the model (Table 4; model E), the GvZ for children classified as GMFCS I to II was not significantly different from 0. The GvZ of GMFCS levels III and IV to V was significantly lower than that of GMFCS levels I to II (Table 4; model E). There was no change in GvZ between the ages of 18 months and 5 years in any GMFCS level (Table 4; model E).

TABLE 1 Participant Recruitment Numbers by Age and Data Points Available by Covariable

	Children, *n*	Repeated Measurements Available, *n*[a]			
		1	2	3	4
Age recruited, mo					
18–24	63	5	7	44	7
30–36	69	7	14	48	—
48	30	9	21	—	—
60	13	13	—	—	—
Sum assessments	175	34	42	92	7
Anthropometric measures					
Height	175	29	38	101	7
Wt	175	29	38	101	7
Growth velocity	141	42	91	5	—
Risk factors for poor growth					
GA	175	—	—	—	—
BW	114	—	—	—	—
GMFCS level	175	32	42	94	7
Oral or gastrostomy tube fed	175	32	42	94	7
DDS score	158	55	53	48	2
Modifiable lifestyle factors					
ActiGraph (HPA and sedentary time)	94	44	35	15	0
Energy intake	140	45	37	59	5

[a] Number of individual children recruited at corresponding age with 1, 2, 3, or 4 measurement occasions completed.

TABLE 2 Participant Characteristics by Age Group

Assessment, Mean (SD)		18–24 Mo	30–36 Mo	48 Mo	60 Mo
Age, mo		22.3 (2.8)	34.6 (3.4)	48.1 (3.2)	61.3 (3.2)
Sample size, *n*		63	130	100	147
Boys, *n* (%)		40 (63)	81 (62)	64 (64)	89 (61)
Anthropometric measures, mean (SD)					
Height, cm		82.8 (5.3)	91.3 (4.5)	100.5 (5.0)	107.0 (5.8)
HZ[a]		−0.9 (1.6)	−0.5 (1.1)	−0.2 (1.2)	−0.4 (1.3)
GvZ		n/a	−0.2 (2.2)	−0.4 (1.7)	−0.4 (1.6)
Wt, kg		11.4 (2.4)	13.4 (1.9)	15.8 (2.3)	18.2 (3.3)
Wt-for-age *z*-score[a]		−0.5 (1.6)	−0.4 (1.3)	−0.2 (1.2)	−0.3 (1.4)
Risk factors for poor growth, mean (SD) or count (%)					
GA, wk[a]		33.5 (5.7)	34.7 (5.5)	35.4 (5.2)	34.6 (5.5)
BW, kg[a]		2.3 (1.1)	2.4 (1.1)	2.5 (1.1)	2.3 (1.1)
GMFCS, %[b]	I	25 (40%)	65 (50%)	48 (48%)	69 (47%)
	II	9 (14%)	8 (6%)	13 (13%)	28 (19%)
	III	14 (22%)	23 (18%)	13 (13%)	21 (15%)
	IV	5 (8%)	14 (11%)	11 (11%)	11 (7%)
	V	10 (16%)	20 (15%)	15 (15%)	18 (12%)
Gastrostomy-tube fed by GMFCS, %[b]	III	0 (—)	2 (8%)	0 (—)	1 (5%)
	IV	2 (40%)	3 (21%)	1 (9%)	1 (9%)
	V	5 (50%)	9 (45%)	8 (53%)	13 (72%)
DDS score		7.8 (7.3)	6.1 (7.3%)	5.4 (7.5)	4.5 (7.0)
Modifiable lifestyle factors, mean (SD)					
HPA, cpm		1201 (455)	987 (423)	1024 (578)	1105 (695)
Sedentary time, %		56 (11)	59 (14)	66 (18)	66 (16)
Energy intake, MJ		3.9 (0.9)	4.4 (1.3)	4.5 (1.2)	5.2 (1.5)

Continuous data presented as mean (SD); categorical data presented as frequency (%).
[a] *t* test: no significant difference between those with 1 or multiple assessments.
[b] χ^2 test: no significant difference between those with 1 or multiple assessments.

DISCUSSION

Children with CP who are able to ambulate independently (GMFCS I) did not differ from peers with typical development in terms of stature or growth velocity. This finding is in agreement with previous studies, but GMFCS levels I and II have not been explored separately.[2,14] In this study, children who were able to ambulate independently with limitations (GMFCS II) were on average shorter than children in GMFCS level I but had a similar growth velocity. The stature of children who were marginally

ambulant (GMFCS III) or nonambulant (GMFCS IV–V) was significantly lower than children in GMFCS level I at the age of 18 months, and they grew more slowly than children in GMFCS level I. Slower growth will therefore cause a discrepancy in stature that will become even more apparent with age for children with moderate to severe gross motor limitations, in agreement with previous studies.[2,3,32] Prematurity was a significant predictor of short stature at 18 months for all children with CP regardless of GMFCS level but was not a significant predictor of growth velocity. The effect of GA on growth status in the study population remained constant between the ages of 18 months and 5 years, indicating that children born prematurely did not catch up to their peers born closer to term in this period. It is common clinical practice to use CA until the age of 2 years in children born prematurely (GA <37 weeks). This is done under the assumption that they will have caught up by then, but children born very prematurely may never catch up or may do so at a much later age.[33,34] Feeding method or feeding difficulties as measured by OPD severity did not significantly predict stature or growth velocity. This finding is contrary to previous studies, but wider age ranges may have influenced results, because time since tube insertion and duration of feeding difficulties may have an influence on growth.[2,7]

Contrary to the findings reported in this article, a recent longitudinal study of children with CP aged 0 to 5 years born at ≥36 weeks' GA did not identify slower growth in the GMFCS III to V group.[14] This study did identify a significantly lower HZ in children classified as GMFCS III to V versus GMFCS I to II, but this difference lost significance once children with feeding difficulties were excluded from the sample. This result is in contrast to our findings, where feeding difficulties as measured by severity of OPD were not a significant predictor of HZ. The smaller sample size, reliance on height data collected via public health center records, and use of parent-reported feeding difficulties may explain the discrepancies between the findings of that study and the findings presented in this article.[14]

Using growth velocity compared with a static measure of stature allows better understanding of the factors operative during the growth interval.[35] The identification of a growth velocity slower than that observed in children with typical development may imply growth failure and be of more clinical value than height per se. The use of growth velocity should therefore be more accurate than stature when investigating factors contributing to slower growth in the CP population.[35]

Energy intake did not show a significant relationship to stature and growth velocity in our population. Linear growth has previously been found to slow down with age in children with CP independent of nutritional status,[32] and attempts to improve growth through gastrostomy feeding have also been shown to improve weight *z* score but not height *z* score.[36] Lower levels of physical activity and lack of weight bearing have been suggested as contributing factors to poor growth.[5,10] As in other studies of the CP population, children with milder motor disability were found to grow faster than those with moderate to severe motor disability.[2,3,32] A stepwise increase in sedentary time with decreasing gross motor function has been reported previously.[8] Although there was some evidence of possible relationships

TABLE 3 Univariable Correlations and Correlations Controlled for GMFCS for HZ and GvZ

	Univariable Analysis	*P*	Multivariable Analysis	*P*
HZ, mean (95% confidence interval)				
Age at assessment, linear term	0.03 (−0.02 to 0.08)	.20	0.03 (−0.02 to 0.08)	.69
Age at assessment, quadratic term	−0.03 (−0.08 to 0.01)	.13	−0.03 (−0.08 to 0.01)	.13
Male sex	−0.02 (−0.40 to 0.36)	.92	−0.003 (−0.35 to 0.34)	.69
Gastrostomy tube feeding	−0.30(−0.60 to 0.07)	.07[a]	−0.20 (−0.53 to 0.13)	.38
GA, wk, linear	0.05 (0.02 to 0.08)	.005[a]	0.05 (0.02 to 0.08)	.003[b,c]
GA, wk, quadratic	−0.01 (0.0003 to 0.004)	.001[a]	−0.01 (−0.02 to 0.003)	.004[b,c]
BW, kg	0.3 (0.2 to 0.5)	<.001[a]	0.3 (0.2 to 0.5)	<.001[b]
DDS score	−0.03 (−0.05 to −0.01)	.004[a]	−0.01 (−0.04 to 0.01)	.32
HPA (100 cpm)	0.02 (−0.002 to 0.05)	.07[a]	0.02 (−0.01 to 0.04)	.23
Sedentary time, %	−0.01 (−0.03 to −0.003)	.14[a]	−0.008 (−0.03 to 0.008)	.34
Energy intake, MJ	0.02 (−0.2 to 0.12)	.42	0.03 (−0.03 to 0.09)	.36
GvZ, mean (95% confidence interval)				
Age at GvZ calculation, linear	−0.06 (−0.30 to 0.18)	.64	−0.06 (−0.29 to 0.17)	.24
Age at GvZ calculation, quadratic	0.09 (−0.26 to 0.44)	.64	0.10 (−0.22 to 0.43)	.48
Sex (difference boys versus girls)	−0.12 (−0.57 to 0.34)	.61	−0.20 (−0.63 to 0.24)	.38
Gastrostomy tube feeding	−0.90 (−1.50 to −0.23)	.02[a]	−0.11 (−0.91 to 0.69)	.79
GA, wk	−0.01 (−0.05 to 0.03)	.54	−0.01 (−0.05 to 0.03)	.57
BW, kg	2.53×10^{-3} (−0.2 to 0.2)	.98	−0.1 (−0.2 to 0.2)	.85
DDS score	−0.06 (−0.09 to −0.03)	<.001[a]	−0.03 (−0.09 to 0.02)	.23
HPA (100 cpm)	0.02 (−0.04 to 0.09)	.42	−0.03 (−0.1 to 0.09)	.58
Sedentary time, %	−0.01 (−0.03 to 0.005)	.16[a]	−0.005 (−0.04 to 0.03)	.79
Energy intake, MJ	0.18 (0.007 to 0.37)	.04[a]	0.06 (−0.11 to 0.24)	.49

All multivariable analyses include GMFCS level as a covariable.
[a] Included in multivariable analysis.
[b] Significant once GMFCS included.
[c] Included in final model.

TABLE 4 Mixed-Effects Regression of HZ and GvZ

		HZ			
Fixed Effects	GMFCS	HZ (Intercept at 18 Mo)	Age at Assessment Factor[a] (Rate of Change per Year)	GA Factors[b]	
				GA, Linear	GA, Quadratic
Unconditional model (model A)	I–V	−0.55 (−0.77 to −0.34)[c]	0.03 (−0.02 to 0.08)	n/a	
Growth model by GMFCS (model B)	I	−0.22 (−0.47 to 0.03)	0.03 (−0.02 to 0.08)	n/a	
	II[d]	−0.34 (−0.57 to −0.11)[e]			
	III[d]	−0.59 (−0.91 to −0.27)[e]			
	IV[d]	−1.04 (−1.45 to −0.64)[e]			
	V[d]	−0.66 (−1.11 to −0.20)[e]			
Final growth model by GMFCS and gestational age (model C)	I	−1.54 (−2.16 to −0.93)[c]	0.09 (0.02 to 0.15)[c]	0.25 (0.11 to 0.39)[c]	−0.01 (0.02 to −0.003)[c]
	II[d]	−0.44 (−0.84 to −0.03)[e]	0.04 (−0.09 to 0.18)		
	III[d]	−0.26 (−0.69 to 0.16)	−0.09 (−0.22 to 0.04)		
	IV[d]	−0.38 (−0.94 to 0.17)	−0.27 (−0.45 to −0.10)[e]		
	V[d]	−0.22 (−0.75 to 0.31)	−0.18 (−0.32 to −0.05)[e]		
		GvZ			
Fixed effects	GMFCS	GvZ (intercept at 18 mo)	Growth velocity age factor[f] (rate of change per year)		
Unconditional model (model D)	I–V	−0.23 (−0.66 to 0.22)	−0.06 (−0.30 to 0.18)	No other factors significantly add to the variance in GvZ	
Growth model by GMFCS (model E)	I–II	0.18 (−0.25 to 0.62)	−0.06 (−0.29 to 0.17)		
	III	−0.73 (−1.2 to −0.26)[e]			
	IV–V	−1.15 (−1.6 to −0.72)[e]			

[a] Age factor = age at assessments in years − 1.42 y.
[b] GA factor = GA in weeks − 23 wk.
[c] Different from 0: $P < .05$.
[d] GMFCS II–V is difference from GMFCS I.
[e] Different from GMFCS I: $P < .05$.
[f] Growth velocity age factor = age at growth velocity calculation in years − 2.17 y.

between HPA, sedentary time, and HZ, and also between sedentary time and GvZ (Table 3), these correlations between growth and measures of HPA and sedentary behavior are largely explained by the level of gross motor capacity. The differentiation of ambulant versus marginally ambulant and nonambulant may be sufficient for explaining the effect of HPA and sedentary behavior on growth. Validation and use of more sophisticated features of the ActiGraph such as an inclinometer to assess standing and sitting time could provide an even more detailed description of a child's daily activity level and therefore reveal relationships to growth. For example, length of time in a standing frame for children with severe motor disabilities could have an impact on their growth.[37]

The strengths of the current study include longitudinal prospective data collection of a representative population cohort of children with CP, all measured by trained study staff. Measures of HPA, sedentary time, energy intake, and OPD validated specifically for the young CP population were used.[16,25,28,29] A limitation of the study was missing data for physical activity, caused by the difficulty of collecting these data for some families. Because the sample sizes were fixed, post hoc power calculations were performed in PS Power and Sample Size Program (Version 3), with simplified assumptions and linear regression equations.[38] In the sample size of 94 children with activity data, the post hoc analysis shows that it would be possible to detect an increase in HZ of ≥0.08 for each 100-cpm increase in HPA with 80% power and 5% significance, which was of small magnitude, and it was therefore of low probability that a clinically significant relationship would go undetected.

CONCLUSIONS

When the growth of young children with CP is assessed in a clinical setting, both gross motor capacity and GA should be taken into consideration. Although children classified as GMFCS I to II appear to be growing at a rate similar to their peers with typical development, children classified as GMFCS III to V grow more slowly. These children also spend more time sedentary and have lower levels of HPA than peers classified as GMFCS I to II and those who are typically developing. More research into the causes and consequences of slower growth, novel interventions aimed at decreasing sedentary time and increasing HPA in GMFCS levels III to V, and their effect on growth and other health outcomes is warranted.

ABBREVIATIONS

BW: birth weight
CA: corrected age
CP: cerebral palsy
DDS: Dysphagia Disorder Survey
GA: gestational age at birth
GMFCS: Gross Motor Function Classification System
GvZ: growth velocity *z* score
HPA: habitual physical activity
HZ: height-for-age *z* score
OPD: oropharyngeal dysphagia

Accepted for publication Jul 6, 2016

Address correspondence to Stina Oftedal, APD, QCPRRC, Centre for Children's Health Research Level 6, 62 Graham St, South Brisbane, QLD 4101, Australia. E-mail: s.oftedal@uq.edu.au

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

FINANCIAL DISCLOSURE: Dr Bell has consulted for Danone and received speaker fees and expenses related to an advisory board meeting for development of a future study protocol; the other authors have indicated they have no financial relationships relevant to this article to disclose.

FUNDING: Funded by the National Health and Medical Research Council (NHMRC), grants 465128 and 569605. Ms Oftedal is supported by a Children's Hospital Foundation PhD scholarship and Australian Postgraduate Scholarship. Professor Boyd receives an NHMRC Research Fellowship (1105038).

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

REFERENCES

1. Stevenson RD, Conaway M, Chumlea WC, et al; North American Growth in Cerebral Palsy Study. Growth and health in children with moderate-to-severe cerebral palsy. *Pediatrics*. 2006;118(3):1010–1018
2. Day SM, Strauss DJ, Vachon PJ, Rosenbloom L, Shavelle RM, Wu YW. Growth patterns in a population of children and adolescents with cerebral palsy. *Dev Med Child Neurol*. 2007;49(3):167–171
3. Krick J, Murphy-Miller P, Zeger S, Wright E. Pattern of growth in children with cerebral palsy. *J Am Diet Assoc*. 1996;96(7):680–685
4. Rosenbaum P, Paneth N, Leviton A, et al. A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol Suppl*. 2007;109:8–14
5. Kuperminc MN, Stevenson RD. Growth and nutrition disorders in children with cerebral palsy. *Dev Disabil Res Rev*. 2008;14(2):137–146
6. Benfer KA, Weir KA, Bell KL, Ware RS, Davies PS, Boyd RN. Oropharyngeal dysphagia in preschool children with cerebral palsy: oral phase impairments. *Res Dev Disabil*. 2014;35(12):3469–3481
7. Fung EB, Samson-Fang L, Stallings VA, et al. Feeding dysfunction is associated with poor growth and health status in children with cerebral palsy. *J Am Diet Assoc*. 2002;102(3):361–373
8. Oftedal S, Bell KL, Davies PS, Ware RS, Boyd RN. Sedentary and active time in toddlers with and without cerebral palsy. *Med Sci Sports Exerc*. 2015;47(10):2076–2083
9. Chad KE, Bailey DA, McKay HA, Zello GA, Snyder RE. The effect of a weight-bearing physical activity program on bone mineral content and estimated volumetric density in children with spastic cerebral palsy. *J Pediatr*. 1999;135(1):115–117
10. Stevenson RD, Roberts CD, Vogtle L. The effects of non-nutritional factors on growth in cerebral palsy. *Dev Med Child Neurol*. 1995;37(2):124–130
11. Shim ML, Moshang T Jr, Oppenheim WL, Cohen P. Is treatment with growth hormone effective in children with cerebral palsy? *Dev Med Child Neurol*. 2004;46(8):569–571
12. Australian Cerebral Palsy Register. *Report of the Australian Cerebral Palsy Register, Birth Years 1993–2006*. Allamby Heights, NSW: Australian Cerebral Palsy Register; 2013
13. Euser AM, de Wit CC, Finken MJ, Rijken M, Wit JM. Growth of preterm born children. *Horm Res*. 2008;70(6):319–328
14. Strand KM, Dahlseng MO, Lydersen S, et al. Growth during infancy and early childhood in children with cerebral palsy: a population-based study [published online ahead of print March 15, 2016]. *Dev Med Child Neurol*. 10.1111/dmcn.13098
15. Brooks J, Day S, Shavelle R, Strauss D. Low weight, morbidity, and mortality in children with cerebral palsy: new clinical growth charts. *Pediatrics*. 2011;128(2). Available at: www.pediatrics.org/cgi/content/full/128/2/e299
16. Walker JL, Bell KL, Boyd RN, Davies PS. Validation of a modified three-day weighed food record for measuring energy intake in preschool-aged children with cerebral palsy. *Clin Nutr*. 2013;32(3):426–431
17. Boyd RN, Jordan R, Pareezer L, et al. Australian Cerebral Palsy Child Study: protocol of a prospective population based study of motor and brain development of preschool aged children with cerebral palsy. *BMC Neurol*. 2013;13(1):57
18. Bell KL, Boyd RN, Tweedy SM, Weir KA, Stevenson RD, Davies PS. A prospective, longitudinal study of growth, nutrition and sedentary behaviour in young children with cerebral palsy. *BMC Public Health*. 2010;10(1):179
19. Badawi N, Watson L, Petterson B, et al. What constitutes cerebral palsy? *Dev Med Child Neurol*. 1998;40(8):520–527
20. Stevenson RD. Use of segmental measures to estimate stature in children with cerebral palsy. *Arch Pediatr Adolesc Med*. 1995;149(6):658–662
21. Kuczmarski R, Ogden C, Grummer-Strawn L. *CDC Growth Charts: United States Advance Data From Vital and Health Statistics, no. 314*. Hyattsville, MD: National Center for Health Statistics; 2000
22. Tanner JM, Davies PSW. Clinical longitudinal standards for height and height velocity for North American children. *J Pediatr*. 1985;107(3):317–329
23. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. *Dev Med Child Neurol*. 1997;39(4):214–223

24. Calis EA, Veugelers R, Sheppard JJ, Tibboel D, Evenhuis HM, Penning C. Dysphagia in children with severe generalized cerebral palsy and intellectual disability. *Dev Med Child Neurol*. 2008;50(8):625–630

25. Benfer KA, Weir KA, Bell KL, Ware RS, Davies PS, Boyd RN. Validity and reproducibility of measures of oropharyngeal dysphagia in preschool children with cerebral palsy. *Dev Med Child Neurol*. 2015;57(4):358–365

26. Sheppard JJ, Hochman R, Baer C. The Dysphagia Disorder Survey: validation of an assessment for swallowing and feeding function in developmental disability. *Res Dev Disabil*. 2014;35(5):929–942

27. Sheppard JJ. *Dysphagia Disorders Survey and Dysphagia Management Staging Scale (Adult and Pediatric Applications): User's Manual*. Australian Edition. Ryde, NSW: The Centre for Developmental Disability; 2003

28. Oftedal S, Bell KL, Davies PS, Ware RS, Boyd RN. Validation of accelerometer cut points in toddlers with and without cerebral palsy. *Med Sci Sports Exerc*. 2014;46(9):1808–1815

29. Keawutan P, Bell K, Oftedal S, Davies P, Boyd R. Validation of accelerometer cut-points in preschool children with cerebral palsy. *Pediatr Phys Ther*. 2016, In press

30. Singer JD, Willett JB. *Applied Longitudinal Data Analysis: Modeling Change and Event Occurrence*. Oxford, England: Oxford University Press; 2003

31. Gorter JW, Ketelaar M, Rosenbaum P, Helders PJ, Palisano R. Use of the GMFCS in infants with CP: the need for reclassification at age 2 years or older. *Dev Med Child Neurol*. 2009;51(1):46–52

32. Stevenson RD, Hayes RP, Cater LV, Blackman JA. Clinical correlates of linear growth in children with cerebral palsy. *Dev Med Child Neurol*. 1994;36(2):135–142

33. Casey PH, Kraemer HC, Bernbaum J, Yogman MW, Sells JC. Growth status and growth rates of a varied sample of low birth weight, preterm infants: a longitudinal cohort from birth to three years of age. *J Pediatr*. 1991;119(4):599–605

34. Hack M, Schluchter M, Cartar L, Rahman M, Cuttler L, Borawski E. Growth of very low birth weight infants to age 20 years. *Pediatrics*. 2003;112(1 pt 1). Available at: www.pediatrics.org/cgi/content/full/112/1/e30

35. Samson-Fang L, Stevenson RD. Linear growth velocity in children with cerebral palsy. *Dev Med Child Neurol*. 1998;40(10):689–692

36. Arrowsmith F, Allen J, Gaskin K, Somerville H, Clarke S, O'Loughlin E. The effect of gastrostomy tube feeding on body protein and bone mineralization in children with quadriplegic cerebral palsy. *Dev Med Child Neurol*. 2010;52(11):1043–1047

37. Pin TW. Effectiveness of static weight-bearing exercises in children with cerebral palsy. *Pediatr Phys Ther*. 2007;19(1):62–73

38. Dupont WD, Plummer WD Jr. Power and sample size calculations for studies involving linear regression. *Control Clin Trials*. 1998;19(6):589–601

Longitudinal Growth, Diet, and Physical Activity in Young Children With Cerebral Palsy

Stina Oftedal, Peter S.W. Davies, Roslyn N. Boyd, Richard D. Stevenson, Robert S. Ware, Piyapa Keawutan, Katherine A. Benfer and Kristie L. Bell

Pediatrics 2016;138;
DOI: 10.1542/peds.2016-1321 originally published online September 7, 2016;

PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Longitudinal Growth, Diet, and Physical Activity in Young Children With Cerebral Palsy

Stina Oftedal, Peter S.W. Davies, Roslyn N. Boyd, Richard D. Stevenson, Robert S. Ware, Piyapa Keawutan, Katherine A. Benfer and Kristie L. Bell

Pediatrics 2016;138;
DOI: 10.1542/peds.2016-1321 originally published online September 7, 2016;