

Body composition, diet, and physical activity: a longitudinal cohort study in preschoolers with cerebral palsy^{1,2}

Stina Oftedal,^{3,4*} Peter SW Davies,⁴ Roslyn N Boyd,³ Richard D Stevenson,⁶ Robert S Ware,⁵ Piyapa Keawutan,^{3,7} Katherine A Benfer,³ and Kristie L Bell^{3,8}

³Queensland Cerebral Palsy and Rehabilitation Research Centre, University of Queensland Children's Health Research Center, ⁴Children's Nutrition Research Center, and ⁵School of Population Health, The University of Queensland, Brisbane, Australia; ⁶Division of Developmental Pediatrics, Department of Pediatrics, School of Medicine, University of Virginia, Charlottesville, VA; ⁷Department of Physical Therapy, Faculty of Allied Health Sciences, Thammasat University, Pathumthani, Thailand; and ⁸Dietetics and Food Services, Lady Cilento Children's Hospital, Children's Health Queensland, South Brisbane, Australia

ABSTRACT

Background: Altered body composition in children with cerebral palsy (CP) could be due to differences in energy intake, habitual physical activity (HPA), and sedentary time.

Objective: We investigated the longitudinal relation between the weight-for-age *z* score (WZ), fat-free mass (FFM), percentage of body fat (%BF), and modifiable lifestyle factors for all Gross Motor Function Classification System (GMFCS) levels (I–V).

Design: The study was a longitudinal population-based cohort study of children with CP who were aged 18–60 mo (364 assessments in 161 children; boys: 61%; mean \pm SD recruitment age: 2.8 ± 0.9 y; GMFCS: I, 48%; II, 11%; III, 15%; IV, 11%; and V, 15%). A deuterium dilution technique or bioelectrical impedance analysis was used to estimate FFM, and the %BF was calculated. Energy intake, HPA, and sedentary time were measured with the use of a 3-d weighed food diary and accelerometer wear. Data were analyzed with the use of a mixed-model analysis.

Results: Children in GMFCS group I did not differ from age- and sex-specific reference children with typical development for weight. Children in GMFCS group IV were lighter-for-age, and children in GMFCS group V had a lower FFM-for-height than those in GMFCS group I. Children in GMFCS groups II–V had a higher %BF than that of children in GMFCS group I, with the exception of orally fed children in GMFCS group V. The mean %BF of children with CP classified them as overfat or obese. There was a positive association between energy intake and FFM and also between HPA level and FFM for children in GMFCS group I.

Conclusions: Altered body composition was evident in preschool-age children with CP across functional capacities. Gross motor function, feeding method, energy intake, and HPA level in GMFCS I individuals are the strongest predictors of body composition in children with CP between the ages of 18 and 60 mo. *Am J Clin Nutr* 2017;105:369–78.

Keywords: body composition, body fat, cerebral palsy, energy intake, fat-free mass, habitual physical activity, preschool children, sedentary behavior

INTRODUCTION

The nutritional status and adequacy assessments in children with cerebral palsy (CP)⁹ are important. Children with CP are frequently described as having altered growth and compromised nutritional status (1–3). Malnutrition, as indicated by short stature, low fat stores, and low muscle mass, has been correlated with poorer health status and decreased societal participation in children with moderate-to-severe motor disability (4). In contrast, risk of overweight and obesity is an increasing concern across the spectrum of functional impairment in children with CP (5, 6). Obesity is of particular concern for children with CP because of long-term health risks associated with adiposity in general and also because of the potential for increased impairment to functional mobility that is related to being obese (7, 8). Obesity can also increase the caregiver burden and has been linked to increased fracture rate (9).

Compared with children with typical development (TD), children with moderate-to-severe gross motor disability have been shown to have lower fat-free mass (FFM) (3, 10, 11) and either a lower (3, 10) or similar (11, 12) percentage of body fat

¹Supported by the National Health and Medical Research Council (NHMRC) (grants 465128 and 569605). SO is supported by a Children's Hospital Foundation PhD scholarship and an Australian Postgraduate Scholarship. RNB is supported by the NHMRC (research fellowship 1105038).

²Supplemental Figure 1 is available from the "Online Supporting Material" link in the online posting of the article and from the same link in the online table of contents at <http://ajcn.nutrition.org>.

*To whom correspondence should be addressed. E-mail: stinaoftedal@gmail.com.

⁹Abbreviations used: BIA, bioelectrical impedance analysis; BMIZ, BMI-for-age *z* score; BW, birth weight; CA, corrected age; CP, cerebral palsy; cpm, counts per minute; DDS, Dysphagia Disorder Survey; DDT, deuterium dilution technique; FFM, fat-free mass; GA, gestational age; GMFCS, Gross Motor Function Classification System; HPA, habitual physical activity; OPD, oropharyngeal dysphagia; TBW, total body water; TD, typical development; WZ, weight-for-age *z* score; %BF, percentage of body fat.

Received May 9, 2016. Accepted for publication November 7, 2016.

First published online January 11, 2017; doi: 10.3945/ajcn.116.137810.



(%BF). In children with mild motor impairment, similar amounts of FFM (13, 14) and either a similar (11, 14) or higher (13) %BF have been shown compared with those of children with TD. Compared with children with CP with moderate-to-severe motor disability, children with mild motor disability have been shown to have more FFM and a lower %BF (11, 15). Children with severe motor disability who are fed via a gastrostomy tube may have similar (11) or less (16, 17) FFM and similar (11) or higher (16, 17) amounts of body fat compared with those of children who are fed orally.

The lack of agreement between previous studies might have been related to changes in body composition with age because some studies have included wider age ranges (3, 10, 13), whereas other studies have included only the preschool-age group (11). Differences in body-composition development over time could be a response to a combination of modifiable lifestyle factors such as dietary intake, both inadequate intake (18, 19) and overfeeding (17), habitual physical activity (HPA) levels, and sedentary time (20).

The aims of this study were to 1) investigate the longitudinal relation between anthropometric and body-composition measures and modifiable lifestyle factors across the spectrum of functional capacity [Gross Motor Function Classification System (GMFCS) levels I–V] and 2) compare anthropometric and body-composition measures between children with GMFCS I–V with those of age- and sex-specific reference children with TD.

METHODS

Children were recruited as part of the Queensland Cerebral Palsy Child Study of Motor Function and Brain Development and the Queensland Cerebral Palsy Child Study of Growth, Nutrition and Physical Activity in Brisbane, Australia, between April 2009 and March 2015 (21, 22). Written informed consent was obtained from each child's primary caregiver, and relevant institutional ethics were gained from relevant institutions (21, 22).

Participants

For the study of motor function and brain development, all Queensland-born children who were diagnosed with CP and were born between 1 January 2005 and 31 December 2009 were eligible for inclusion, whereas for the growth, nutrition, and physical activity study, children who were born between 1 September 2005 and 31 December 2009 were eligible for inclusion (21, 22). CP was defined as a group of permanent disorders of movement and posture that were attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain (23). Children with neurodegenerative diseases were excluded.

Children were seen at an interval of 12–18 mo from study entry. The mean \pm SD length between assessments was 16.0 \pm 5.8 mo. If children entered the study at a corrected age (CA) of 18 or 24 mo or at a CA of 30 or 36 mo, they were seen at a CA of 36 or 48 mo, respectively, for their second assessments. The third assessment point was at a CA of 60 mo. Some children entered the study late and had only 2 assessments (at CAs of 48 and 60 mo) or only 1 assessment (at a CA of 60 mo CA). Some children were seen at 4 occasions because they were participating in a substudy (24). The recruitment flowchart

(Supplemental Figure 1) outlines how the final study sample was obtained. The final study sample consisted of 161 children of whom 125 children (78%) participated on >1 occasion. In total, 364 assessments were included in the analysis. A full summary of available data is shown in Table 1. The distribution of GMFCS levels was representative of an Australian population with CP (24) but with a slightly higher proportion of children who were classified as GMFCS I (36% compared with 47%, respectively) and a lower proportion of children who were classified as GMFCS II (25% compared with 14%, respectively) (24). These differences were possibly due to the young age of the study population (25). The primary motor types were bilateral spasticity ($n = 87$), unilateral spasticity ($n = 53$), dyskinesia ($n = 9$), hypotonia ($n = 5$), and ataxia ($n = 4$). Although every effort was made to collect full data sets at each appointment, the feeding assessment for oropharyngeal dysphagia (OPD) scoring, the 3-d wear of the ActiGraph device (model GT3M/GT3X; ActiGraph), and the 3-d food diary were not always successfully completed because of either the compliance of the child or the time restraints of parents or caregivers.

TABLE 1

Participant recruitment numbers by age and characteristic¹

	Children, <i>n</i>	Repeated measurements available, <i>n</i>			
		1	2	3	4
Age recruited, mo					
18–24	50	10	9	28	3
30–36	64	7	13	44	—
48	30	2	28	—	—
60	17	17	—	—	—
Total children	161	36	50	72	3
Sex and anthropometric measures					
Sex	161	—	—	—	—
Height	161	36	50	72	3
Weight	161	36	50	72	3
Deuterium dilution measurement	92	26	32	35	2
Bioelectrical impedance analysis	66	10	18	37	1
Total body-composition data	161	36	50	72	3
Risk factors for poor growth					
Gestational age	161	—	—	—	—
Birth weight	111	—	—	—	—
GMFCS level	161	—	—	—	—
I	77	12	29	35	1
II	22	8	3	11	—
III	22	5	6	9	2
IV	17	8	3	6	—
V	23	3	9	11	—
Gastrostomy-tube fed	16	16	15	11	1
DDS score	146	49	51	45	1
Modifiable lifestyle factors					
ActiGraph device, ² HPA and TSS	84	36	33	15	0
Energy intake	132	48	24	57	3

¹Data are for the number of individual children recruited at the corresponding ages with 1, 2, 3, or 4 measurement occasions completed. DDS, Dysphagia Disorder Survey; GMFCS, Gross Motor Function Classification System; HPA, habitual physical activity; TSS, time spent sedentary.

²Model GT3M/GT3X (ActiGraph).

Anthropometric measures

Weight was measured to the nearest 100 g with the use of chair scales (Seca). Height and supine length were measured to the nearest completed millimeter with the use of a portable stadiometer (Shorr Productions). If accurate measures of length or height were not possible, height was estimated on the basis of knee height that was measured with the use of an anthropometer (Holtan Ltd.) and validated equations (26). If estimated height was required for one assessment point, it was used for the child's other assessment points as well to allow for consistency in comparisons. BMI (in kg/m^2) was calculated as weight divided by the square of height. Height-for-age z scores, weight-for-age z scores (WZs), and BMI-for-age z scores (BMIZs) were identified on the basis of age and sex from CDC growth charts to allow for comparison with age- and sex-specific reference children with TD (27).

Body composition

Total body water (TBW) was measured with the use of either the deuterium dilution technique (DDT) or a bioelectrical impedance analysis (BIA). Detailed descriptions of the procedures for the DDT and BIA are shown elsewhere (21). When both forms of measurement were available, TBW was measured with the use of the DDT because DDT is the gold standard. Children were given a single dose of deuterium in the form of water orally or via a feeding tube. A single baseline urine sample was collected before administration of the dose, and parents were asked to collect another sample ~ 5 h after the dose was given. Measurement of the isotopic enrichment of isotopes at this time enabled the calculation of the body water pool with the use of standard equations (28). Impedance (Ω) was collected with the use of a Bodystat 1500MDD device at 800 μA and a fixed frequency of 50 KHz. TBW was estimated from measurements of impedance and height or length with the use of equations that have been validated for the young CP population (29).

FFM was determined through the division of TBW by age and sex-specific hydration factors (30). FFM (kilograms) that was adjusted for height in the model as opposed to the FFM index (expressed as FFM/kg^2) was used because the FFM index did not normalize FFM for height in our population (31). Body fat was determined by subtracting the FFM value from the total body weight of the subject that gave the fat mass, which was converted to the %BF—i.e., $(\text{fat mass} \div \text{body weight}) \times 100$ —to account for weight differences between children (32). Age- and sex-specific reference values for FFM (kilograms) and the %BF were used to compare the body composition of children with CP with that of children with TD at the age of 60 mo (33, 34).

Risk factors for poor growth and development

The motor type of CP (e.g., spastic, dystonic, or hypotonic) and distribution (unilateral or bilateral) were determined by 2 independent physiotherapists at each assessment according to the methods of Sanger et al. (35) and the internationally accepted classification system of the European CP Register (36). The severity of activity limitation was classified with the use of the GMFCS (37). The GMFCS classifies children into 1 of 5 functional levels (I–V). Children who were classified as GMFCS I are expected to walk independently between 18 mo and 2 y of age,

whereas children who are classified as GMFCS II are not expected to be able to walk without any assistive devices until their fourth birthday (37). Children who are classified as GMFCS III are expected to be able to walk with an assistive device by their fourth birthday (34). Children who are classified as GMFCS IV may, at best, manage short distances with a walker under adult supervision but are not necessarily expected to achieve this skill (37). Children who are classified as GMFCS V are not expected to achieve any independent mobility skills (37). Gestational age (GA) and birth weight (BW) were reported by the parents (22). The severity of OPD was determined with the use of the Dysphagia Disorder Survey (DDS)—Pediatric Part 2 raw score, which is a valid and reliable measure of dysphagia (38, 39). The survey consists of a series of binary judgments of feeding competency on 8 ingestion functions for purees, chewable food, and fluids with a maximum impairment raw score of 22 (40). Children who are exclusively tube fed default to a raw score of 22 (40). The feeding method (oral compared with gastrostomy tube) was determined by parent report via a questionnaire.

Modifiable lifestyle factors

Energy intake (megajoules) was assessed with the use of a validated weighted 3-d food and fluid diary as described elsewhere (41). The 3-d average HPA [counts per minute (cpm)] and sedentary time as a percentage of the day was measured with the use of an ActiGraph device (model GT3M/GT3X; ActiGraph). The ActiGraph device and cutoffs for sedentary time have been validated in the young CP population as previously described (42, 43).

Procedures

Trained researchers took anthropometric measurements, performed BIA measurements, and dosed the deuterium. Anthropometric and BIA measures were taken in duplicate to reduce the chance of error. Two physiotherapists determined the GMFCS levels of participants, and a consensus was reached (22). The DDS score was determined by a pediatric speech pathologist via a videotaped, standardized mealtime assessment (39). Parents were supplied with food scales and a food diary with detailed instructions and weighed food and fluid intakes for 2 weekdays and 1 weekend day (41). The food diaries were analyzed with the use of FoodWorks software (version 7; Xyris). Parents were also given an ActiGraph device for their children to wear for 2 weekdays and 1 weekend day while completing a wear-time log (20).

Statistics

A longitudinal analysis with the use of mixed-effects linear models was used to estimate age-related changes and to investigate the association between significant characteristics and the outcome variables WZ, BMIZ, FFM, and the %BF. These multilevel regression models allowed for the construction of separate trajectories for the 5 GMFCS levels. This approach accounted for both intraindividual variability and interindividual variability, which led to more-precise estimates of SEs and, thus, a greater statistical power than would have been provided by separate analyses for the individual levels (44). The mixed-model analysis also allowed for the inclusion of data from all available time points regardless of the number of times a child completed the assessments. In all mixed-effects models, a child's



identification number was entered as a random effect, and the covariance matrix was set to identity. Variables that were significantly associated with the outcomes WZ, BMIZ, FFM, and the %BF were identified. The GMFCS level (I–V) was included as a categorical variable. We investigated the variables of sex, DDS score, gastrostomy tube feeding, motor distribution (unilateral compared with bilateral), BW (kilograms), sedentary time (percentages), HPA (cpm), energy intake (megajoules), GA (weeks), and the age at assessment (years). The outcome variable FFM was also investigated for its relation to height (centimeters). Both linear and quadratic age-at-assessment terms were used to investigate possible nonlinear relations. All continuous variables were centered at the lowest observed variable in the data set so as not to regress outcome variables to, for example, a 0-kg BW or a GA of 0 wk.

Because of the large number of possible covariates, relations between covariables and outcome variables were first analyzed univariately and included in an additional analysis if P was <0.25 . Second, relations between the covariates and outcome variables were analyzed in a model that included the GMFCS level (I–V) and interaction terms between covariates (covariate \times covariate)

and age-by-covariate interactions (age terms \times covariate). Items that were significant at $P < 0.05$ were retained for investigation in the multivariable models. Third, all remaining variables and interactions were included in a combined multivariable model and were entered in a stepwise manner in the order of significance. Variables and interactions were retained in the final model if they remained significant at $P < 0.05$. All analyses were performed with the use of Stata v13.0 software (StataCorp LP).

RESULTS

Participants

There were no differences in GA, BW, WZ, or height-for-age z score or for the distribution of GMFCS levels, motor distribution, or sex between children who participated at only 1 time point and those who participated at multiple time points when their first assessments were compared (data not shown), which indicated that there was no systematic bias in the sampling in terms of anthropometric characteristics. Participant anthropometric measurements, outcome measurements, and covariates were

TABLE 2
Participant characteristics by age group¹

Participant characteristics	Age, mo			
	18–24	30–36	48	60
Sample size, n	56	99	82	127
Age, mo	22.5 \pm 2.6 ²	34.5 \pm 3.3	48.5 \pm 2.5	61.7 \pm 2.1
Boys, n (%)	36 (64)	64 (65)	50 (61)	78 (61)
Gestational age, ³ wk	34 \pm 6	35 \pm 5	35 \pm 5	34 \pm 6
Birth weight, ³ g	2327 \pm 1146	2476 \pm 1095	2472 \pm 1073	2328 \pm 1120
GMFCS, ⁴ n (%)				
I	22 (39)	53 (54)	40 (49)	63 (50)
II	8 (14)	7 (7)	10 (12)	22 (17)
III	11 (20)	14 (14)	12 (15)	15 (12)
IV	5 (9)	10 (10)	7 (8)	10 (8)
V	10 (18)	15 (15)	13 (16)	16 (13)
Gastrostomy-tube fed by GMFCS, ⁴ n (%)				
III	—	1 (7)	—	1 (7)
IV	2 (40)	1 (10)	1 (14)	—
V	5 (50)	6 (40)	7 (54)	11 (69)
DDS score	7.9 \pm 7.5	5.4 \pm 7.0	5.5 \pm 7.8	4.4 \pm 7.0
Anthropometric measures				
Height, cm	83.4 \pm 5.0	91.5 \pm 4.3	100.8 \pm 5.0	107 \pm 5.7
Height-for-age z score ³	−0.7 \pm 1.6	−0.4 \pm 1.1	−0.2 \pm 1.2	−0.4 \pm 1.2
Weight, kg	11.3 \pm 2.4	13.5 \pm 1.7	16.0 \pm 2.4	18.1 \pm 3.3
Weight-for-age z score ³	−0.6 \pm 1.6	−0.4 \pm 1.2	−0.2 \pm 1.3	−0.3 \pm 1.5
BMI	16.3 \pm 1.7	16.0 \pm 1.5	15.7 \pm 1.6	15.7 \pm 2.0
BMI-for-age z score ³	0.0 \pm 1.3	−0.2 \pm 1.3	−0.1 \pm 1.2	0.0 \pm 1.4
Fat-free mass	9.1 \pm 1.4	10.6 \pm 1.4	12.3 \pm 1.8	13.9 \pm 2.3
Body fat, %				
Orally fed	18.6 \pm 0.9	20.6 \pm 0.7	22.0 \pm 0.6	21.8 \pm 0.6
Tube fed	22.0 \pm 2.4	25.1 \pm 2.0	31.5 \pm 2.4	29.0 \pm 1.3
Modifiable lifestyle factors				
Habitual physical activity, cpm	1175 \pm 465	982 \pm 436	1007 \pm 583	1051 \pm 580
Sedentary time, %	56 \pm 11	59 \pm 15	67 \pm 18	66 \pm 17
Energy intake, MJ	3.9 \pm 0.9	4.3 \pm 1.2	4.6 \pm 1.1	5.3 \pm 1.5

¹ cpm, counts per minute; DDS, Dysphagia Disorder Survey; GMFCS, Gross Motor Function Classification System.

² Mean \pm SD (all such values).

³ With the use of the t test, there were no significant differences between one or multiple assessments.

⁴ With the use of the chi-square test, there were no significant differences between one or multiple assessments.

summarized by age group (**Table 2**) and GMFCS level (**Table 3**). All values were transformed to baseline age using the lowest observed participant age (1.42 y) via a mixed-model analysis to allow for comparisons. Covariables that showed significant relations to outcome measures at each step of the analysis process are shown in **Table 4**.

Weight, height, BMI, and comparison to age- and sex-matched standards (z scores)

Weights of children who were classified as GMFCS I did not significantly differ from those of age- and sex-matched reference children with TD (**Table 3**). Only children who were classified as GMFCS IV were significantly lighter than children who were classified as GMFCS I (**Table 3**). The change in WZ between the ages of 18 and 60 mo was nonsignificant and not different between GMFCS levels (**Table 5**, model B). These results indicated that weight gain was similar in all GMFCS levels and comparable with that of age- and sex-specific reference children with TD. BW was a significant predictor of the WZ, and all

children, regardless of the GMFCS level, who were born with low BW were significantly lighter than their peers who were born with a higher BW (**Table 5**, model C). The relation between BW and WZ was linear because the quadratic term was NS. There was no significant difference in the BMIZ between levels of gross motor function, and BMI-for-age was not significantly different from that of age- and sex-matched peers with TD (**Table 3**) and had a curvilinear relation with age (**Table 6**, model E).

FFM, fat mass, and %BF

Children with CP had, on average, 8.5 kg FFM at 18 mo of age and gained 1.4 kg FFM/y until they were 60 mo old (**Table 7**, model F). Absolute FFM (kilograms) was significantly lower for children who were classified as GMFCS II–V than for children who were classified as GMFCS I (**Table 3**). However, relative to height, this relation remained significant only for GMFCS V children (**Table 3**), and FFM-for-height did not increase by age (**Table 7**, model H). Compared with age- and sex-specific

TABLE 3
GMFCS-level characteristics and comparisons regressed to baseline age (1.42 y) via a mixed-model analysis¹

	Value				
GMFCS level (n)	I (77)	II (22) ²	III (22) ²	IV (17) ²	V (23) ²
Descriptor					
Boys, n	52	12	20	12	13
Tube feeding, n	—	—	—	5	11
DDS score	2.8 (2.0, 3.7) ^{3,4}	1.3 (0.3, 2.4) ⁵	3.6 (2.5, 4.8) ⁵	8.9 (7.4, 10.4) ⁵	16.9 (15.6, 18.2) ⁵
Gestational age at birth, wk	35 ± 5 ⁶	35 ± 6	32 ± 6 ^{3,4}	36 ± 5	36 ± 5
Birth weight, kg	2.6 ± 1.1	2.2 ± 1.2	1.7 ± 0.8	2.4 ± 1.1	2.6 ± 1.1
Anthropometric measure					
Weight, kg	10.8 (10.2, 11.3)	−0.2 (−0.8, 0.5)	−0.8 (−1.5, 0.02)	−1.0 (−2.0, −0.1) ⁵	−1.1 (−2.0, −0.1) ⁵
Weight-for-age z score	−0.2 (−0.5, 0.1)	−0.1 (−0.4, 0.2)	−0.3 (−0.7, 0.04)	−0.7 (−1.2, −0.2) ⁵	−0.4 (−1.0, 0.1)
Height, cm	82.1 (81.1, 83.2)	−1.5 (−2.6, −0.5) ⁵	−2.8 (−4.2, −1.4) ⁵	−4.1 (−5.8, −2.4) ⁵	−3.2 (−5.1, −1.3)
Height-for-age z score	−0.1 (−0.4, 0.1)	−0.4 (−0.6, −0.1) ⁵	−0.8 (−1.1, −0.4) ⁵	−1.2 (−1.6, −0.8) ⁵	−0.7 (−1.1, −0.2) ⁵
BMI, kg/m ²	17.0 (16.4, 17.6)	0.1 (−0.3, 0.6)	0.2 (−0.4, 0.7)	0.02 (−0.7, 0.7)	−0.1 (−0.9, 0.6)
BMI-for-age z score	0.7 (0.2, 1.3) ⁴	0.2 (−0.2, 0.5)	0.1 (−0.4, 0.5)	0.1 (−0.4, 0.6)	−0.3 (−0.8, 0.2)
Body composition					
Fat-free mass, kg	9.1 (8.7, 9.6)	−0.8 (−1.3, −0.3) ⁵	−1.2 (−1.8, −0.6) ⁵	−1.5 (−2.2, −0.8) ⁵	−1.6 (−2.3, −0.9) ⁵
Fat-free mass-for-height, kg	5.7 (5.2, 6.3)	−0.2 (−0.7, 0.2)	−0.3 (−0.8, 0.1)	−0.4 (−0.9, 0.2)	−0.6 (−1.1, −0.1) ⁵
Body fat, %					
Girls, orally fed	16.8 (14.3, 19.3)	2.9 (1.0, 4.8) ⁵	2.1 (0.2, 4.1) ⁵	2.6 (0.3, 5.0) ⁵	2.2 (−0.2, 5.0)
Boys, orally fed, compared with girls			−2.8 (−4.2, −1.4) ⁷		
Tube fed (compared with orally fed boys and girls)			4.6 (1.9, 7.2) ⁸		
Dietary intake ⁹					
Energy intake, MJ	4.0 (3.7, 4.3)	−0.1 (−0.5, 0.3)	−0.2 (−0.6, 0.2)	−0.6 (−1.1, −0.03) ⁵	−1.2 (−1.7, −0.8) ⁵
Activity monitoring ¹⁰					
Habitual physical activity, cpm	1397 (1282, 1514)		−412 (−571, −254) ^{2, 11}	−991 (−1143, −839) ^{2, 11}	
Time spent sedentary, %	46 (42, 50)		11 (6, 16) ^{2, 11}	27 (6, 16) ^{2, 11}	

¹ cpm, counts per minute; DDS, Dysphagia Disorder Survey; GMFCS, Gross Motor Function Classification System.

² Values for this level are for differences from GMFCS I/GMFCS I–II unless otherwise noted.

³ Mean; 95% CI in parentheses (all such values).

⁴ Different from zero.

⁵ Different from GMFCS I.

⁶ Mean ± SD (all such values).

⁷ Orally fed boys had a significantly lower percentage of body fat compared with girls in all GMFCS groups.

⁸ Tube-fed children had a significantly higher percentage of body fat compared with orally fed children in all GMFCS groups.

⁹ n for GMFCS levels: I = 69; II = 18; III = 23; IV = 12; V = 24.

¹⁰ n for GMFCS levels: I–II = 58; III = 17; IV–V = 19.

¹¹ Different from GMFCS I–II.



TABLE 4Univariable correlations and correlations controlled for GMFCS via a mixed-model analysis¹

	Univariable analysis	<i>P</i>	Adjusted for GMFCS	<i>P</i>
Weight-for-age <i>z</i> score				
Age at assessment	0.02 (−0.03, 0.08)	0.319	0.03 (−0.03, 0.08)	0.372 ²
Age at assessment ³	0.06 (0.01, 0.1)	0.026 ⁴	0.06 (0.002, 0.1)	0.044 ³
Age at assessment ²	−0.06 (−0.1, −0.004)	0.034 ⁴	−0.05 (−0.1, −0.001)	0.047 ³
Gestational age	0.07 (0.03, 0.1)	<0.001	0.06 (0.03, 0.1)	<0.001 ³
Gestational age ³	−0.01 (−0.02, −0.001)	0.033	−0.01 (−0.01, −0.002)	0.059
Birth weight, kg	0.4 (0.2, 0.6)	<0.001	0.4 (0.2, 0.6)	<0.001 ^{2,3}
Energy intake, MJ	0.04 (−0.02, 0.1)	0.215	NC	NC
BMI-for-age <i>z</i> score				
Age at assessment	0.03 (−0.04, 0.10)	0.382	0.02 (−0.05, 0.1)	0.539 ²
Age at assessment ³	0.1 (0.05, 0.2)	0.001	0.1 (0.04, 0.2)	0.002 ^{2,3}
Sex, boys compared with girls	−0.3 (−0.7, 0.1)	0.121	NC	NC
Birth weight, kg	<0.001	0.121	NC	NC
Fat-free mass controlled for height				
Age at assessment	1.4 (1.3, 1.5)	<0.001 ⁴	1.4 (1.3, 1.5)	<0.001 ^{2,3}
Age at assessment ³	0.1 (0.001, 0.2)	0.03 ⁴	0.1 (0.01, 0.2)	0.02 ³
Height	0.2 (0.2, 0.3)	<0.001 ⁴	0.2 (0.19, 0.24)	<0.001 ^{2,3}
Sex, boys compared with girls	0.7 (0.2, 1.2)	0.006 ⁴	0.6 (0.1, 1.0)	0.010 ³
Motor distribution, bilateral compared with unilateral	−0.42 (−0.73, −0.10)	0.009 ⁴	−0.31 (−0.67, 0.05)	0.090
Tube feeding	−0.9 (−1.4, −0.3)	0.003 ⁴	−0.5 (−1.1, 0.05)	0.071 ³
Gestational age	0.1 (0.04, 0.1)	<0.001 ⁴	0.08 (0.04, 0.1)	<0.001 ³
Gestational age ³	0.3 (0.1, 0.5)	0.01 ⁴	−0.01 (−0.02, −0.0003)	0.042 ³
Birth weight, kg	0.4 (0.2, 1.0)	<0.001 ⁴	0.4 (0.2, 0.6)	<0.001 ³
DDS score	−0.07 (−0.1, −0.03)	<0.001 ⁴	−0.02 (−0.06, 0.03)	0.501
HPA, 100 cpm	0.1 (0.04, 0.2)	0.001 ⁴	0.04 (−0.03, 0.1)	0.311
Sedentary time, %	−0.03 (−0.05, −0.01)	0.003 ⁴	−0.003 (−0.3, 0.02)	0.841
Energy intake, MJ	0.2 (0.06, 0.3)	0.004 ⁴	0.2 (0.1, 0.4)	0.001 ^{2,3}
Body fat, %				
Age at assessment	1.1 (0.6, 1.5)	<0.001 ⁴	1.1 (0.6, 1.6)	<0.001 ^{2,3}
Age at assessment ³	−0.5 (−1.0, −0.04)	0.032 ⁴	−0.6 (−1.1, −0.1)	0.013 ^{2,3}
Sex, boys compared with girls	−3.1 (−4.7, −1.6)	<0.001 ⁴	−3.0 (−4.4, −1.5)	<0.001 ^{2,3}
Motor distribution, bilateral compared with unilateral	1.26 (−0.29, 2.82)	0.112	NC	NC
Gastronomy-tube feeding	5.8 (3.5, 8.1)	<0.001 ⁴	4.9 (2.1, 7.7)	0.001 ^{2,3}
DDS score	0.3 (0.1, 0.4)	<0.001 ⁴	−0.1 (0.3, 0.2)	0.673
HPA, 100 cpm	−0.5 (−0.6, −0.2)	<0.001 ⁴	−0.03 (−0.3, 0.2)	0.797
Sedentary time, %	0.2 (0.1, 0.2)	<0.001 ⁴	0.002 (−0.1, 0.1)	0.965

¹ All values are means (95% CIs). NC values were not included in the multivariable analysis because *P* was >0.25. cpm, counts per minute; DDS, Dysphagia Disorder Survey; GMFCS, Gross Motor Function Classification System; HPA, Habitual Physical Activity; NC, not computed.

² Included in the final model.

³ Variable remained significant after GMFCS level was controlled for in analysis.

⁴ Considered for inclusion in the multivariable analysis.

reference children with TD, boys with CP at the age of 60 mo had FFM between the 9th and 25th percentiles (FFM: 14.1 kg; 95% CI: 13.6, 14.6 kg), and girls had FFM between the 25th and 50th percentiles (FFM: 13.5 kg; 95% CI: 12.8, 14.1 kg) (34). When the FFM of boys and girls who were classified as GMFCS I were assessed separately, because their height did not significantly differ from that of age- and sex-specific reference children (Table 3), their placements in these percentiles remained (data not shown) (34).

Energy intake was significantly related to FFM in all GMFCS levels, and for every additional 1 MJ consumed in excess of 1.9 MJ (i.e., the minimum observed in the sample), FFM increased by a mean of 0.2 kg/MJ (95% CI: 0.1, 0.4 kg/MJ) (Table 7, model I). The range of energy intake in the population was 1.9–9.5 MJ. The HPA level (cpm) was also significantly related to FFM, but only for children who were classified as GMFCS I (Table 7, model I). For each 100-cpm increase in the HPA level,

FFM increased by 0.09 kg (95% CI: 0.02, 0.2 kg) in GMFCS I children, but not in GMFCS II, III, and V children. The range in HPA levels for GMFCS I was 490–2560 cpm. Although no difference was shown between GMFCS I and IV children in regards to the effect of the HPA level on FFM (Table 7, model I), this result needs to be interpreted with caution. The small sample size of children who were classified as GMFCS IV with activity data (*n* = 5) meant that the power to detect a significant difference was small.

Children with CP had 17% body fat at 18 mo of age and gained, on average, 3.5%/y between 18 and 60 mo of age. There was a curvilinear relation with age so that the %BF gain lessened (−0.5%/y) as a child approached the age of 60 mo (Table 8, model J). Sex and feeding method were significant predictors of the %BF (Table 3). Boys had a significantly lower %BF than did girls (Table 3). The %BF of children who were classified as GMFCS I was significantly lower than in all other GMFCS



TABLE 5Mixed-model analysis: WZ¹

Fixed effect and GMFCS level	WZ (intercept at 18 mo)	Age, ² WZ/y	Birth weight, ³ WZ/kg birth weight
Unconditional model (model A)			
I-V	-0.4 (-0.6, -0.2) ⁴	0.03 (-0.03, 0.08)	—
Growth model by GMFCS (model B)			
I	-0.2 (-0.5, 0.1)	0.03 (-0.03, 0.08)	—
II ⁵	-0.1 (-0.4, 0.2)	0.03 (-0.03, 0.08)	—
III ⁵	-0.3 (-0.7, 0.04)	0.03 (-0.03, 0.08)	—
IV ⁵	-0.7 (-1.2, -0.2) ⁶	0.03 (-0.03, 0.08)	—
V ⁵	-0.4 (-1.0, 0.1)	0.03 (-0.03, 0.08)	—
Final growth model by GMFCS and birth weight (model C)			
I	-1.0 (-1.5, -0.5) ⁴	0.03 (-0.03, 0.1)	0.4 (0.2, 0.6) ⁴
II ⁵	0.1 (-0.2, 0.5)	0.03 (-0.03, 0.1)	0.4 (0.2, 0.6) ⁴
III ⁵	-0.2 (-0.7, 0.3)	0.03 (-0.03, 0.1)	0.4 (0.2, 0.6) ⁴
IV ⁵	-0.8 (-1.4, -0.3) ⁶	0.03 (-0.03, 0.1)	0.4 (0.2, 0.6) ⁴
V ⁵	-0.6 (-1.2, 0.001)	0.03 (-0.03, 0.1)	0.4 (0.2, 0.6) ⁴

¹ All values are means (95% CIs). GMFCS, Gross Motor Function Classification System; WZ, weight-for-age z score.

² Age factor = age at assessment in years - 1.42 y.

³ Birth-weight factor = birth weight in kilograms - 0.640 kg.

⁴ Different from zero, $P < 0.05$.

⁵ Different from GMFCS I.

⁶ Different from GMFCS I, $P < 0.05$.

groups except for in orally fed children who were classified as GMFCS V (Table 3). There was no significant difference in the %BF between children who were classified as GMFCS I and orally fed children who were classified as GMFCS V. When the %BF was compared with that of age- and sex-specific reference children with TD, all orally fed children with CP, regardless of the GMFCS level, had a %BF between the 90th and 95th percentiles at the age of 60 mo (Table 2), which classified them as overfat (33). This classification remained for all GMFCS levels when the levels were analyzed separately (data not shown). Tube-fed children who were classified as GMFCS IV and V had a %BF that was greater than the 95th percentile (Table 2), which classified them as obese (33).

DISCUSSION

Altered body composition was evident from an early age in this population of children with CP. Data showed that, on average, children with CP at all GMFCS levels had excess amounts of body fat and below-average FFM (kilograms) compared with those of age- and sex-specific reference children with TD. However, body weight and weight relative to height (BMI) were similar to those of age- and sex-specific reference children with TD as evidenced because the WZ and BMIZ did not differ significantly from zero. BMI has been shown to misclassify a large proportion of children in the CP population with moderate-to-severe motor limitations as having adequate or insufficient body fat stores when they have excessive body fat (45). Findings from

TABLE 6Mixed-model analysis: BMIZ¹

Fixed effect and GMFCS level	BMI-for-age z score (intercept at 18 mo)	Age, ² BMIZ/y	
		Age	Age ³
Unconditional model (model D)			
I-V	0.4 (0-0.01, 0.74)	-0.5 (-0.9, -0.2) ³	0.1 (0.05, 0.2) ³
Growth model by GMFCS (model E)			
I	0.4 (-0.1, 0.8)	-0.5 (-0.9, -0.2) ³	0.1 (0.04, 0.2) ³
II ⁴	0.2 (-0.2, 0.6)	-0.5 (-0.9, -0.2) ³	0.1 (0.04, 0.2) ³
III ⁴	0.1 (-0.4, 0.5)	-0.5 (-0.9, -0.2) ³	0.1 (0.04, 0.2) ³
IV ⁴	0.0 (-0.5, 0.5)	-0.5 (-0.9, -0.2) ³	0.1 (0.04, 0.2) ³
V ⁴	-0.3 (-0.8, 0.2)	-0.5 (-0.9, -0.2) ³	0.1 (0.04, 0.2) ³

¹ All values are means (95% CIs). BMIZ, BMI-for-age z score; GMFCS, Gross Motor Function Classification System;

² Age factor = age at assessment in years - 1.42 y.

³ Different from zero, $P < 0.05$.

⁴ Different from GMFCS I.



TABLE 7Mixed-model analysis: FFM¹

Fixed effect and GMFCS level	FFM (intercept at 18 mo), kg	Age, ² FFM/y	Height, ³ FFM/cm	Energy intake, ⁴ FFM/MJ	HPA level, ⁵ FFM/100 cpm
Unconditional model (model F)					
I–V	8.5 (8.2, 8.9)	1.4 (1.3, 1.5) ⁶	—	—	—
Growth model by GMFCS (model G)					
I	9.1 (8.7, 9.6)	1.4 (1.3, 1.5) ⁶	—	—	—
II ⁷	−0.8 (−1.3, −0.3) ⁸	1.4 (1.3, 1.5) ⁶	—	—	—
III ⁷	−1.2 (−1.8, −0.6) ⁸	1.4 (1.3, 1.5) ⁶	—	—	—
IV ⁷	−1.5 (−2.2, −0.8) ⁸	1.4 (1.3, 1.5) ⁶	—	—	—
V ⁷	−1.6 (−2.3, −0.9) ⁸	1.4 (1.3, 1.5) ⁶	—	—	—
Growth model by GMFCS and height (model H)					
I	5.7 (5.2, 6.3)	−0.3 (−0.6, 0.2)	0.2 (0.2, 0.3) ⁸	—	—
II ⁷	−0.2 (−0.7, 0.2)	−0.3 (−0.6, 0.2)	0.2 (0.2, 0.3) ⁸	—	—
III ⁷	−0.3 (−0.8, 0.1)	−0.3 (−0.6, 0.2)	0.2 (0.2, 0.3) ⁸	—	—
IV ⁷	−0.4 (−0.9, 0.2)	−0.3 (−0.6, 0.2)	0.2 (0.2, 0.3) ⁸	—	—
V ⁷	−0.6 (−1.1, −0.1) ⁸	−0.3 (−0.6, 0.2)	0.2 (0.2, 0.3) ⁸	—	—
Final growth model by GMFCS, height and HPA level (model I)					
I	3.6 (2.6, 4.7)	−0.6 (−0.9, −0.3) ⁶	0.3 (0.2, 0.3) ⁶	0.2 (0.1, 0.4) ⁶	0.09 (0.02, 0.2) ⁶
II ⁷	4.9 (0.4, 9.3) ⁸	−0.6 (−0.9, −0.3) ⁶	0.3 (0.2, 0.3) ⁶	0.2 (0.1, 0.4) ⁶	−0.4 (−0.3, −0.1) ⁸
III ⁷	2.5 (1.1, 3.8) ⁸	−0.6 (−0.9, −0.3) ⁶	0.3 (0.2, 0.3) ⁶	0.2 (0.1, 0.4) ⁶	−0.2 (−0.4, −0.1) ⁸
IV ⁷	1.8 (−0.5, 4.2)	−0.6 (−0.9, −0.3) ⁶	0.3 (0.2, 0.3) ⁶	0.2 (0.1, 0.4) ⁶	−0.3 (−0.6, 0.08)
V ⁷	1.3 (0.1, 2.5) ⁸	−0.6 (−0.9, −0.3) ⁶	0.3 (0.2, 0.3) ⁶	0.2 (0.1, 0.4) ⁶	−0.7 (−1.0, −0.3) ⁸

¹ All values are means (95% CIs). cpm, counts per minute; FFM, fat-free mass; GMFCS, Gross Motor Function Classification System; HPA, habitual physical activity.

² Age factor = age at assessment in years − 1.42 y.

³ Height factor = height at assessment in centimeters − 68.3 cm.

⁴ Megajoules factor = energy intake − 1.92 MJ.

⁵ HPA-level factor = cpm/100: per 100-cpm increase in HPA, FFM increased by values noted.

⁶ Different from zero, $P < 0.05$.

⁷ Different from GMFCS I.

⁸ Different from GMFCS I, $P < 0.05$.

the current cohort suggest that BMI performs poorly in identifying excess body fat stores in children with mild motor limitations.

Previous studies have identified malnutrition as being prevalent in children with moderate-to-severe motor disability (3, 10). This outcome was not evident in the current population studied; rather, the current study suggests that children with CP are at

risk of overweight and obesity, which has been reported previously with the use of BMI classifications (5, 6). It is possible that an increased awareness of risks associated with malnutrition and changes in the clinical management of children with CP have led to more-prompt nutritional supplementation and, therefore, the avoidance of malnutrition in this younger

TABLE 8Mixed model analysis: %BF¹

Fixed effect and GMFCS level	%BF (intercept at 18 mo)	Age, ² %BF/y		Sex (0 = girl, 1 = boy)	Tube fed (0 = no, 1 = yes)
		Age	Age ³		
Unconditional model (model J)					
I–V	17.3 (14.9, 19.5)	3.5 (1.3, 5.8) ³	−0.5 (−1.0, −0.05) ³	—	—
Final growth model incl. sex and tube feeding status (model K)					
I	16.8 (14.3, 19.3)	4.1 (1.8, 6.3) ³	−0.7 (−1.1, −0.2) ³	−2.8 (−4.2, −1.4) ³	4.6 (1.9, 7.2) ³
II ⁴	2.9 (1.0, 4.8) ⁵	4.1 (1.8, 6.3) ³	−0.7 (−1.1, −0.2) ³	−2.8 (−4.2, −1.4) ³	4.6 (1.9, 7.2) ³
III ⁴	2.1 (0.2, 4.1) ⁵	4.1 (1.8, 6.3) ³	−0.7 (−1.1, −0.2) ³	−2.8 (−4.2, −1.4) ³	4.6 (1.9, 7.2) ³
IV ⁴	2.6 (0.3, 5.0) ⁵	4.1 (1.8, 6.3) ³	−0.7 (−1.1, −0.2) ³	−2.8 (−4.2, −1.4) ³	4.6 (1.9, 7.2) ³
V ⁴	2.2 (−0.2, 5.0)	4.1 (1.8, 6.3) ³	−0.7 (−1.1, −0.2) ³	−2.8 (−4.2, −1.4) ³	4.6 (1.9, 7.2) ³

¹ All values are means (95% CIs). GMFCS, Gross Motor Function Classification System; incl., including; %BF, percentage of body fat.

² Age factor = age at assessment in years − 1.42 y.

³ Different from zero, $P < 0.05$.

⁴ Different from GMFCS I.

⁵ Different from GMFCS I, $P < 0.05$.



population of children with CP. Additional longitudinal studies should assess risk factors for developing excess body fat stores in children with CP as they enter school age.

Energy intakes and HPA levels had significant relations with body composition in this study. Increased energy intake was associated with higher FFM, and a greater HPA level was associated with higher FFM but only in children who were classified as GMFCS I. It was not possible to say with confidence which direction the relation between the HPA level, energy intake, and FFM takes. It might be a plausible hypothesis that children who have a higher HPA level will have greater FFM and, therefore, greater energy intake because of a higher amount of metabolically active tissue and greater energy requirements. In a state of energy balance, energy intake equals total energy expenditure (46). The weighed food diaries that were used in this study have been validated against total energy expenditure as measured by the gold-standard doubly labeled water technique (47). Higher energy expenditure could indicate higher activity levels, and consequently, the HPA level could be a predisposing explanatory factor in the relation between FFM and energy intake. The relation between the HPA level and FFM was significant for GMFCS I children, and the more active children had significantly more FFM than did the least-active children even after energy intake was controlled for.

BW was a stronger predictor of weight status (WZ) than was GA. This finding indicated that, along with preterm-born children, those children who were born closer to or at term but who were small for GA also had lower weight than did children who were born within the normal weight range for their GA. This result is in concordance with previous studies of non-CP populations (48, 49) and with a recent longitudinal study of children with CP from birth to the age of 60 mo who were born ≥ 36 wk of gestation (50). The effect of BW on weight status remains constant between the ages of 18 and 60 mo, indicating that children who are born prematurely or small for GA do not catch up to their peers who are born closer to term or at a GA-appropriate weight in this period. It is common clinical practice to use the CA until the age of 2 y in children who are born prematurely (GA < 37 wk) (51), but premature or small-for-GA infants might never catch up or only do so at a much later age (52, 53).

A potential limitation of this study is that only 50% of children completed the activity monitoring. Because the sample size was fixed, post hoc power calculations were performed with simplified assumptions with the use of linear regression equations (54). In the sample size of 94 children with activity data, the SD for FFM was 1.95 kg, the SD for HPA was 4.95 cpm, and the partial correlation between the 2 variables was 0.10 after controlling for the effects of GMFCS and height. The post hoc analysis showed that an increase in FFM ≥ 0.11 kg for each 100-cpm increase would have been detected with 80% power and 5% significance. This lowest detectable difference was of a relatively small magnitude, and therefore, there was low probability that a clinically significant relation between FFM and cpm would have gone undetected. Although the study was sufficiently powered to detect overall relations, it may have been underpowered to detect significant relations within and differences between GMFCS levels. The sample size of gastrostomy tube-fed children in GMFCS IV was also small, which limited the power to detect differences by feeding methods for this GMFCS level. Strengths of the study included the longitudinal data collection of a representative population of children with

CP and the use of measures of energy intake, sedentary time, and OPD that were validated in the young CP population; in addition, to ensure consistency, trained research staff carried out assessments, took measurements, and interpreted data.

In conclusion, in this study, we identify the altered body composition in the form of excess body fat stores and lower FFM across the spectrum of functional capacity in a preschool-age, population-based sample of children with CP. The findings of the current study need to be considered within the Australian context before extending them to other locations, because Australia is a high-resource country where children have access to extensive public health services. Gross motor function, feeding method, energy intake, and HPA level (for GMFCS I) are the strongest predictors of body composition in the current study population. BW is a predictor of weight status. There are differences in body composition between GMFCS levels that are often analyzed together (i.e., GMFCS I and II and GMFCS IV and V) as well as between gastrostomy tube- and oral-fed children; however, the clinical significance of these differences in body composition are still unknown. Future studies with a larger sample size should explore these differences and consequences in relation to health outcomes.

The authors' responsibilities were as follows—SO: collected the data, carried out the longitudinal data analysis, and drafted, revised, and had primary responsibility for the final content of the manuscript; SO, PSWD, RNB, RDS, and KLB: conceptualized and designed the study and critically reviewed the manuscript; PSWD, RNB, and KLB: supervised the data collection; RSW: assisted with and critically reviewed the longitudinal data analysis; PK and KAB: collected and analyzed the data and critically reviewed the manuscript; and all authors: read and approved the final manuscript. None of the authors reported a conflict of interest related to the study.

REFERENCES

1. Stevenson RD, Hayes RP, Cater LV, Blackman JA. Clinical correlates of linear growth in children with cerebral palsy. *Dev Med Child Neurol* 1994;36:135–42.
2. Stallings VA, Charney EB, Davies JC, Cronk CE. Nutritional status and growth of children with diplegic or hemiplegic cerebral palsy. *Dev Med Child Neurol* 1993;35:997–1006.
3. Stallings VA, Cronk CE, Zemel BS, Charney EB. Body composition in children with spastic quadriplegic cerebral palsy. *J Pediatr* 1995;126:833–9.
4. Samson-Fang L, Fung E, Stallings V, Conaway M, Worley G, Rosenbaum P, Calvert R, O'Donnell M, Henderson R, Chumlea W. Relationship of nutritional status to health and societal participation in children with cerebral palsy. *J Pediatr* 2002;141:637–43.
5. Hurvitz EA, Green LB, Hornyak JE, Khurana SR, Koch LG. Body mass index measures in children with cerebral palsy related to gross motor function classification: a clinic-based study. *Am J Phys Med Rehabil* 2008;87:395–403.
6. Rogozinski BM, Davids JR, Davis RB, Christopher LM, Anderson JP, Jameson GG, Blackhurst DW. Prevalence of obesity in ambulatory children with cerebral palsy. *J Bone Joint Surg Am* 2007;89:2421–6.
7. Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord* 1999;23(Suppl 2):S2–11.
8. Hills AP, King NA, Armstrong TP. The contribution of physical activity and sedentary behaviours to the growth and development of children and adolescents: implications for overweight and obesity. *Sports Med* 2007;37:533–45.
9. Stevenson RD, Conaway M, Chumlea WC, Rosenbaum P, Fung EB, Henderson RC, Worley G, Liptak G, O'Donnell M, Samson Fang L. Growth and health in children with moderate to severe cerebral palsy. *Pediatrics* 2006;118:1010–8.
10. Arrowsmith FE, Allen JR, Gaskin KJ, Somerville H, Birdsall J, Barzi F, O'Loughlin EV. Nutritional rehabilitation increases the resting energy expenditure of malnourished children with severe cerebral palsy. *Dev Med Child Neurol* 2012;54:170–5.



11. Walker JL, Bell KL, Stevenson RD, Weir KA, Boyd RN, Davies PS. Differences in body composition according to functional ability in preschool-aged children with cerebral palsy. *Clin Nutr* 2015;34:140–5.
12. Kuperminc MN, Stevenson RD. Growth and nutrition disorders in children with cerebral palsy. *Dev Disabil Res Rev* 2008;14:137–46.
13. van den Berg-Emons HJG, Saris WHM, de Barbanson DC, Westertorp KR, Huson A, van Baak MA. Daily physical activity of schoolchildren with spastic diplegia and of healthy control subjects. *J Pediatr* 1995;127:578–84.
14. Bell KL, Davies PSW. Energy expenditure and physical activity of ambulatory children with cerebral palsy and of typically developing children. *Am J Clin Nutr* 2010;92:313–9.
15. Finbråten AK, Martins C, Andersen GL, Skranes J, Brannsether B, Juliusson PB, Syversen U, Stevenson RD, Vik T. Assessment of body composition in children with cerebral palsy: a cross-sectional study in Norway. *Dev Med Child Neurol* 2015;57:858–64.
16. Rieken R, van Goudoever JB, Schierbeek H, Willemssen S, Calis EA, Tibboel D, Evenhuis HM, Penning C. Measuring body composition and energy expenditure in children with severe neurologic impairment and intellectual disability. *Am J Clin Nutr* 2011;94:759–66.
17. Sullivan PB, Alder N, Bachlet AM, Grant H, Juszczak E, Henry J, Vernon-Roberts A, Warner J, Wells J. Gastrostomy feeding in cerebral palsy: too much of a good thing? *Dev Med Child Neurol* 2006;48:877–82.
18. Sullivan PB, Juszczak E, Bachlet AM, Lambert B, Vernon-Roberts A, Grant HW, Eltumi M, McLean L, Alder N, Thomas AG. Gastrostomy tube feeding in children with cerebral palsy: a prospective, longitudinal study. *Dev Med Child Neurol* 2005;47:77–85.
19. Stallings VA, Zemel BS, Davies JC, Cronk CE, Charney EB. Energy expenditure of children and adolescents with severe disabilities: a cerebral palsy model. *Am J Clin Nutr* 1996;64:627–34.
20. Oftedal S, Bell KL, Davies PS, Ware RS, Boyd R. Sedentary and active time in toddlers with and without cerebral palsy. *Med Sci Sports Exerc* 2015; 47:2076–83.
21. 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;10:179.
22. Boyd RN, Jordan R, Pareezer L, Moodie A, Finn C, Luther B, Arnfield E, Pym A, Craven A, Beall P, 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;13:57.
23. Rosenbaum P, Paneth N, Leviton A, Goldstein M, Bax M. A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol Suppl* 2007;109:8–14.
24. Australian Cerebral Palsy Register Group. Report of the Australian Cerebral Palsy Register, birth years 1993–2003. April 2009. Available from: <https://www.cpreregister.com/pubs/PublicationsAndOtherResources.aspx>.
25. Gorter JW, Ketelaar M, Rosenbaum P, Hadders 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;51:46–52.
26. Stevenson RD. Use of segmental measures to estimate stature in children with cerebral palsy. *Arch Pediatr Adolesc Med* 1995;149:658–62.
27. Kuczmarski R, Ogden C, Grummer-Strawn L. CDC growth charts: United States. *Adv Data* 2000;314:1–27.
28. Halliday D, Miller A. Precise measurement of total body water using trace quantities of deuterium oxide. *Biomed Mass Spectrom* 1977;4:82–7.
29. Bell KL, Boyd RN, Walker JL, Stevenson RD, Davies PS. The use of bioelectrical impedance analysis to estimate total body water in young children with cerebral palsy. *Clin Nutr* 2013;32:579–84.
30. Fomon SJ, Haschke F, Ziegler EE, Nelson SE. Body composition of reference children from birth to age 10 years. *Am J Clin Nutr* 1982;35:1169–75.
31. Wells JC, Cole TJ. Adjustment of fat-free mass and fat mass for height in children aged 8 y. *Int J Obes Relat Metab Disord* 2002;26:947–52.
32. Wells JC, Fewtrell MS. Measuring body composition. *Arch Dis Child* 2006;91:612–7.
33. Laurson KR, Eisenmann JC, Welk GJ. Body fat percentile curves for U.S. children and adolescents. *Am J Prev Med* 2011;41(4 Suppl 2):S87–92.
34. Wells JC, Williams JE, Chomtho S, Darch T, Grijalva-Eternod C, Kennedy K, Haroun D, Wilson C, Cole TJ, Fewtrell MS. Body-composition reference data for simple and reference techniques and a 4-component model: a new UK reference child. *Am J Clin Nutr* 2012; 96:1316–26.
35. Sanger TD, Delgado MR, Gaebler Spira D, Hallett M, Mink JW. Classification and definition of disorders causing hypertonia in childhood. *Pediatrics* 2003;111:e89–97.
36. Surveillance of Cerebral Palsy in Europe. Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. Surveillance of Cerebral Palsy in Europe (SCPE). *Dev Med Child Neurol* 2000;42:816–24.
37. 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: 214–23.
38. 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;50:625–30.
39. 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; 57:358–65.
40. Sheppard JJ. Dysphagia disorders survey and dysphagia management staging scale (adult and pediatric applications): user's manual. Australian edition. Ryde (Australia): The Centre for Developmental Disability; 2003.
41. 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;32:426–31.
42. 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;46:1808–15.
43. Keawutan P, Bell K, Oftedal S, Davies P, Boyd R. Validation of accelerometer cut-points in children with cerebral palsy aged 4 to 5 years. *Pediatr Phys Ther* 2016;28:427–34.
44. Singer JD. Using SAS PROC MIXED to fit multilevel models, hierarchical models, and individual growth models. *J Educ Behav Stat* 1998;23:323–55.
45. Kuperminc MN, Gurka MJ, Bennis JA, Busby MG, Grossberg RI, Henderson RC, Stevenson RD. Anthropometric measures: poor predictors of body fat in children with moderate to severe cerebral palsy. *Dev Med Child Neurol* 2010;52:824–30.
46. McArdle WD, Katch FI, Katch VL. Exercise physiology: energy, nutrition and human performance. (Philadelphia): Lippincott Williams & Wilkins; 2001.
47. Schoeller DA, Fjeld CR. Human energy metabolism: what have we learned from the doubly labeled water method? *Ann Rev Nutr* 1991;11: 355–73.
48. Hediger ML, Overpeck MD, Kuczmarski RJ, McGlynn A, Maurer KR, Davis WW. Muscularity and fatness of infants and young children born small- or large-for-gestational-age. *Pediatrics* 1998;102:E60.
49. Hediger ML, Overpeck MD, McGlynn A, Kuczmarski RJ, Maurer KR, Davis WW. Growth and fatness at three to six years of age of children born small- or large-for-gestational age. *Pediatrics* 1999;104:e33.
50. Strand KM, Dahlseng MO, Lydersen S, Ro TB, Finbraten AK, Jahnsen RB, Andersen GL, Vik T. Growth during infancy and early childhood in children with cerebral palsy: a population-based study. *Dev Med Child Neurol* 2016;58:924–30.
51. Canadian Paediatric Society. A health professional's guide for using the new WHO growth charts. *Paediatr Child Health* 2010;15:84–98.
52. 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;119:599–605.
53. 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:e30–8.
54. Dupont WD, Plummer WD. Power and sample size calculations for studies involving linear regression. *Control Clin Trials* 1998;19:589–601.